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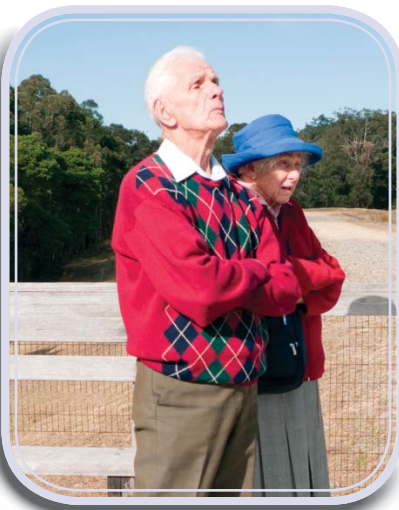
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VACCINE PREVENTABLE DISEASES IN AUSTRALIA, 2005 TO 2007



National Centre for Immunisation Research and
Surveillance of Vaccine Preventable Diseases

VACCINE PREVENTABLE DISEASES IN AUSTRALIA, 2005 TO 2007

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Executive summary

This report is the fifth in the biennial series of national reports on vaccine preventable diseases in Australia, bringing together the three most important national sources of routinely collected data about vaccine preventable diseases. This report adds data from January 2006 to December 2007 for notifications collected by the National Notifiable Diseases Surveillance System (NNDSS), from July 2005 to June 2007 for hospitalisation records in the AIHW National Hospital Morbidity Database, and from January 2005 to December 2006 for recorded deaths in the AIHW National Mortality Database. Jurisdictional notification data on two diseases for which national notification data are unavailable (varicella-zoster in South Australia and rotavirus in the Northern Territory) are also included.

The general trend towards improved control of disease and improved vaccination coverage is evident, particularly in the childhood years. Detailed results are presented in 16 individual chapters. The rates of notifications, hospitalisations and deaths for 10 more common diseases included in the National Immunisation Program (NIP) are summarised in the Table below.

Diseases with long-standing vaccination programs

During 2006–2007, there were no notified cases of diphtheria, and 1 imported case of poliomyelitis. A very low incidence of tetanus continued, affecting mainly older adults. A low incidence of invasive *Haemophilus influenzae* type b (Hib) disease (in young children), measles and rubella also continued in the 2 years under review. For measles, a relatively larger number of cases were reported in 2006, but this was predominantly associated with an outbreak in a community opposed to immunisation. Most, but not all, other outbreaks in the period were

Table: Notification, hospitalisation and death rates of 10 vaccine preventable diseases over two periods,* Australia, 2002 to 2007†

Disease‡	Notifications		Hospitalisations		Deaths	
	Average annual rate (per 100,000) 2003–2005	Average annual rate (per 100,000) 2006–2007	Average annual rate (per 100,000) July 2002–June 2005	Average annual rate (per 100,000) July 2005–June 2007	Average annual rate (per 100,000) 2003–2004	Average annual rate (per 100,000) 2005–2006
<i>H. influenzae</i> type b (age <5 years)§	0.6	0.8	0.9	0.6	–	–
Hepatitis B	1.5	1.4	0.9	0.8	0.06	0.09
Influenza¶	16.9	32.7	15.3	10.8	0.25	0.13
Measles	0.3	0.3	0.2	0.1	–	–
Meningococcal disease	2.3	1.5	3.6	2.5	0.12	0.08
Mumps	0.7	2.1	0.2	0.3	<0.005	<0.005
Pertussis	41.5	39.1	2.2	2.1	<0.005	0.01
Pneumococcal disease**	10.5	7.0	5.2	3.5	0.10	0.05
Rubella	0.2	0.2	0.1	<0.05	–	–
Tetanus	<0.05	<0.05	0.1	0.1	–	<0.005

* Data from the former period have been reported in the fourth report of this series.¹ Data from the latter period are reported in this current report in the following chapters.

† Notifications where the date of diagnosis was between January 2003 and December 2007; hospitalisations where the date of separation was between July 2002 and June 2007; deaths where the death was recorded between January 2003 and December 2006.

‡ See Chapter 4 for case definitions for individual vaccine preventable disease.

§ Invasive *Haemophilus influenzae* type b disease for notifications. Includes *Haemophilus meningitis* only for hospitalisations and deaths.

|| Includes acute hepatitis B only for notifications, hospitalisations and deaths.

¶ Note the limitations of notification systems and coding for influenza hospitalisation and death data, which may grossly underestimate the true disease burden due to influenza.

** Includes pneumococcal meningitis and septicaemia only for hospitalisations and deaths.

linked to overseas acquired cases. For rubella, despite low numbers of reported infections, 2 cases of congenital rubella syndrome were reported. While there are high levels of rubella immunity in the general population, immigrant women from some countries without an established rubella vaccination program, and Indigenous women in some communities, have been identified as having lower immunity and therefore being at higher risk of infection in pregnancy.

The increasing trend of mumps notifications since 2004 continued in 2006 and especially in 2007, predominantly in adolescents and young adults, many of whom were born at a time of relatively low vaccination coverage and a single-dose schedule, and grew up during a period of reduced circulation of wild virus. While the largest numbers came from New South Wales, significant outbreaks occurred in 2007 in Indigenous communities in the Northern Territory and the Kimberley region of Western Australia.

Pertussis remains both the disease most difficult to control and for which data are most difficult to interpret. The period 2006–2007 saw lower notification and hospitalisation rates than the previous 3-year period, which included an epidemic. There was a substantial decrease in notifications among adolescents, following the commencement of school-based vaccination in 2003–2004. However, there were marked increases in notification rates in adults and hospitalisation rates in the elderly, some of which may be attributable to false positive results from serology testing, an issue detected and rectified in 2006. Strategies to reduce pertussis, especially in young infants, are being actively considered.

For almost all these diseases for which vaccination programs have been well established, disease incidence has remained low. Elimination of endogenous transmission in Australia has been achieved and maintained for some diseases, like poliomyelitis and measles, and may be nearly achieved for rubella. However, there remains an ongoing risk of importation of these diseases acquired overseas. Ongoing high vaccination coverage and effective surveillance are still required.

Diseases with universal vaccination programs commencing in the last decade

At both national and jurisdictional levels, notification and hospitalisation rates of acute hepatitis B were stable over the 2006–2007 period, following the decline since 2001. This previously observed decline in notifications was most marked in young adults aged 15–29 years, who nevertheless remained the age group with the highest notification rates. This decline was most likely related to declining rates of intravenous drug use since 2000 and adolescent catch-up programs that commenced from the late 1990s. While a targeted program for high-risk infants began in the 1980s, the universal infant program was not implemented until 2000. The impact of this program will become more evident in the near future.

For both invasive meningococcal disease and invasive pneumococcal disease, substantial decreases in notifications, hospitalisations and deaths since implementation of the universal childhood programs with catch-up (2003 for the meningococcal program and 2005 for the pneumococcal program) were sustained with further decreases in the 2 most recent years analysed. This was predominantly due to a decrease in disease caused by the specific serogroup (meningococcal serogroup C) or serotypes (pneumococcal serotypes 4, 6B, 9V, 14, 18C, 19F, 23F) targeted by each conjugate vaccine. While the decrease in incidence of both these diseases was mainly seen in the target age groups for vaccination, herd immunity effects were also evident in other age groups not eligible for vaccination. The remaining challenges include the prevention of serogroup B meningococcal disease in infants following development of a vaccine effective against serogroup B disease, control of pneumococcal disease in older and at-risk people, and control of invasive disease caused by other serotypes, especially the emerging serotype 19A.

Diseases with vaccination programs targeted to specific population subgroups or settings

During this reporting period, annual seasonal influenza vaccination was recommended for all Australians aged ≥ 65 years, all Aboriginal and Torres Strait Islander people aged ≥ 15 years, and all individuals aged ≥ 6 months who were predisposed to severe influenza or its complications, with the intention of protecting those who were more vulnerable to severe outcomes from influenza during each season. Australians aged ≥ 65 years, and Aboriginal and Torres Strait Islander people who were aged ≥ 50 years or who had chronic medical conditions that predispose them to severe influenza, were eligible for annual vaccination under the NIP. The overall as

well as age-specific influenza notification rates were substantially higher in 2007 than in any previous year since national notification data became available in 2001, but hospitalisation data for the latter half of 2007 were unavailable at the time of this analysis. Although increased testing might partly explain the increase in notifications, the co-circulation of two strains of virus, A/H3N2 and A/H1N1, both of which had experienced some antigenic drift, as well as an increase in reported deaths due to influenza, including several young children, are unusual. Notwithstanding potential biases due to differences in documenting influenza notifications or hospitalisations across different age groups, it is noteworthy that the rates of notification and hospitalisation in children <5 years of age in Australia were considerably greater than in the elderly, although morbidity was predominantly in this latter age group.

Australia implemented the National Q Fever Management Program in various jurisdictions at different times during 2001 to 2006, targeting people at highest risk of occupational and environmental exposure to Q fever. While Q fever notification and hospitalisation rates declined to record low levels in 2005, no further decline occurred in 2006–2007, with the suggestion of slight increases in some jurisdictions. Multiple factors, including natural environmental factors, would have contributed. Limited availability of additional data, especially on risk factors and vaccination status, renders risk assessment and disease control difficult.

In Australia, hepatitis A generally occurs sporadically with periodic epidemic peaks related to outbreaks, although it has been endemic in the Indigenous population, especially among Indigenous children, in more remote areas. Notification and hospitalisation rates continued to decline in the 2-year review period, continuing the decline seen since the peaks in the late 1990s. Expanding from an immunisation program for Indigenous children in north Queensland, a program targeting Indigenous children aged 12–24 months in four jurisdictions (the Northern Territory, Queensland, South Australia and Western Australia) with catch-up (up to age 5 years) commenced in 2005. While this report focuses on patterns in the total population rather than Indigenous people in particular, data by jurisdiction are consistent with a substantial impact from this program.

Diseases with recent universal vaccination programs and limited national surveillance data

For varicella, both national hospitalisation and South Australian notification data suggest an early impact of the varicella childhood immunisation program which commenced in November 2005. The impact is most marked in children aged 12–23 months, as expected, but is also seen in children aged 24–47 months. This needs to be confirmed over time and in more age groups as vaccine coverage rises. The epidemiology of herpes zoster, as reflected in the national hospitalisation data and notification data from South Australia, does not appear to have changed. Specific data on herpes zoster are limited.

For rotavirus, publicly funded vaccine for infants was available in the Northern Territory from October 2006 and nationally from July 2007. Hospitalisation data available for this report, up to June 2007, only cover the pre-vaccine period, except for a 9-month period in the Northern Territory. These showed substantial year-to-year variation. The lower notification rate for rotavirus in 2007, post vaccine introduction, observed in the Northern Territory is encouraging, but more data are required to be conclusive.

Future surveillance priorities

While demographic data are generally complete, the completeness and consistency of some important fields in the notification records are more variable. These fields include Indigenous status, vaccination status and serogroup/subtype. National collection of additional clinical and/or laboratory data ('enhanced' data), as occurs currently for Hib and pneumococcal notifications, would be valuable for some other important diseases, such as pertussis and meningococcal disease, to facilitate better targeted control measures. Also, the range of diseases and information required is likely to continue to grow, with the introduction of additional vaccination programs like rotavirus and varicella in recent years and the expansion of existing programs. The use of complementary data sources, like emergency department presentations, general practice sentinel surveillance and Australian Paediatric Surveillance Unit data, for surveillance of vaccine preventable diseases should be explored. For example, linkage of the Australian Childhood Immunisation Register to morbidity and mortality data would greatly enhance the quality of vaccination status data for children as well as eliminating the resource-intensive re-collection of the information.

Future vaccination priorities

Although the National Immunisation Program has been very effective in controlling many vaccine preventable diseases, high vaccination coverage in all the existing vaccination programs needs to be maintained. Alternative settings to primary health care for reaching specific population groups, such as school-based programs or pre-travel vaccinations, should continue to be developed. For some diseases for which higher morbidity rates are primarily due to serogroups/serotypes not contained in the available vaccine, such as meningococcal or pneumococcal disease, use of vaccines with extended coverage as they become available would be beneficial. For some other diseases (e.g. influenza, Q fever and pertussis), morbidity occurs mainly in age groups or risk groups outside those recommended for vaccination or in those too young to be vaccinated. The cost-effectiveness of expanding eligibility of existing vaccines, such as influenza for young children and boosters of pertussis vaccine for adults, will require consideration over the coming years.

Reference

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1. Introduction

This is the fifth national surveillance report on the morbidity and mortality from vaccine preventable diseases (VPDs) in Australia. The first (1993 to 1998) was published in 2000, the second (1999 to 2000) in 2002, the third (2001 to 2002) in 2004, and the fourth (2003 to 2005) in 2007.¹⁻⁴ The overall progressive decline in the incidence of all the childhood VPDs continues. Most striking has been the very substantial decline in the numbers of deaths from these diseases since the pre-vaccination era, despite the Australian population increasing almost 3-fold (Table 1.1), and the close association of declines in individual disease mortality with the introduction of specific vaccination programs.⁵ However, continual effective surveillance for vaccine preventable diseases is still important, as there are recently introduced vaccines for which effectiveness is yet to be demonstrated, and remaining challenges like outbreaks of measles, mumps or pertussis, particularly in infants too young to be vaccinated.

Table 1.1: Number of deaths from diseases commonly vaccinated against, Australia, 1926 to 2005,* by decade

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
1996–2005	0	17	7	0	0	19,310,000

* Sources: Feery B. One hundred years of vaccination. *N S W Public Health Bull* 1997;8(8–9):61–63; Feery B. Impact of immunisation on disease patterns in Australia. *Med J Aust* 1981;2(4):172–176. Deaths recorded for 1966–1975 and 1996–2005 updated with data from the AIHW National Mortality Database.

† Excludes deaths from subacute sclerosing panencephalitis.

■ Indicates decade in which community vaccination started for the disease.

The past two decades 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 in 1996⁶
- the Seven Point Plan in 1997 (this included the Measles Control Campaign in the later part of 1998)⁷
- initiatives to encourage and remind parents to have their children immunised on time – currently referred to as the Maternity Immunisation Allowance initiative⁸
- the General Practice Immunisation Initiative in 1998⁹
- implementation of new national notifiable diseases definitions, daily data updates to NNDSS and online data publication in 2004
- new vaccination programs across the age spectrum (see Appendices 6.4 and 6.5 for details):
 - for infants and children
 - *Haemophilus influenzae* type b (Hib)
 - hepatitis B
 - meningococcal C disease
 - pneumococcal disease
 - varicella-zoster virus
 - rotavirus

- for adolescents and young adult women
 - hepatitis B
 - pertussis
 - varicella
 - human papillomavirus
- for older people
 - influenza
 - pneumococcal disease
- specific programs for Aboriginal and Torres Strait Islander people
 - influenza
 - pneumococcal disease
 - hepatitis A

Despite their limitations, data from routinely collected datasets remain a valuable information source especially with respect to trends over time. This fifth report uses similar methods to the previous four reports in the series, bringing together all major data sources at the national level relevant to VPDs and vaccination – notifications, coded hospitalisations and death certificates. This report is unique as a source of systematically analysed hospitalisation data on key VPDs, which gives an index of more severe disease presentations compared to notified cases.

The diseases covered in this report include those for which vaccines were funded nationally for children during the review period (diphtheria, invasive Hib disease, acute hepatitis B, invasive pneumococcal disease, measles, meningococcal [C] disease, mumps, pertussis, poliomyelitis, rubella, tetanus and varicella), those for which vaccines were available but only publicly funded or recommended for specific population groups with high risk (hepatitis A, influenza and Q fever), and rotavirus, for which new vaccines became available in 2006. Data potentially relevant for human papillomavirus (HPV) disease, against which there has been a vaccine since mid 2007, are not included in this report. This report does not include tuberculosis, for which reports are found elsewhere,^{10,11} or diseases that are currently of limited national public health significance in Australia, such as Japanese encephalitis and yellow fever.

This and the previous four reports, all compiled by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS), provide evidence of the impact of various vaccination policies over the past 16 years, as listed in Appendix 6.4, on the epidemiology of key vaccine preventable diseases in Australia and a baseline against which further initiatives can be evaluated.

With the publication in 2009 of national vaccination coverage data for 2007, together with the time trends of national data up to 2007,¹² and the plan for publication of national vaccination coverage reports annually from 2009, this report does not include national vaccination coverage data. The reports for 2007 and 2008 are available in *Communicable Diseases Intelligence*.^{12,13}

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2. Methods

Vaccine preventable diseases data

Three main sources of routinely collected data were used for this surveillance report on vaccine preventable diseases in Australia. Disease notification data were obtained from the Office of Health Protection's NNDSS; hospitalisation data were from the Australian Institute of Health and Welfare (AIHW) National Hospital Morbidity Database; and mortality data were from the AIHW National Mortality Database (unpublished data).

For a recent analysis of vaccine preventable diseases surveillance data and vaccination coverage focusing on Indigenous Australians, please refer to the report *Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia, 2003 to 2006*.¹

Notifications

The NNDSS was established in its current form in 1991, and includes de-identified information about cases of vaccine preventable diseases reported by state and territory authorities in Australia. Each of the eight jurisdictional health departments collect notifications of communicable diseases under their respective public health legislation. Prior to September 2007, there were no national legislative provisions for jurisdictions to report to NNDSS, although all jurisdictions voluntarily did so, with de-identified data electronically renewed daily or several times a week from jurisdictions as of 2007. In September 2007, the National Health Security Act 2007² received royal assent. This Act provides a legislative basis for and authorises the exchange of health information between jurisdictions and the Commonwealth, and includes the establishment of the National Notifiable Disease List.³ The National Health Security Agreement⁴ that followed, which was signed by Health Ministers in April 2008, established operational arrangements to formalise and enhance existing surveillance and reporting systems.⁵ Data quality of the NNDSS is continually monitored by the Office of Health Protection and the National Surveillance Committee (NSC), a jurisdictional committee comprised of surveillance and data managers. There is a continual process of reviewing the national consistency of communicable disease surveillance on a daily, fortnightly and quarterly basis.

Historically, state and territory notification criteria were based on the National Health and Medical Research Council (NHMRC) surveillance case definitions.⁶ However, application of these definitions differed among jurisdictions, with some using the 1994 NHMRC case definitions (e.g. South Australia and Western Australia) and others using their own modified definitions (e.g. New South Wales and Victoria). (See also Appendix 6.6 for case definitions in use prior to 2004.) In September 2003, a new set of national case definitions for notifiable diseases reported to NNDSS was endorsed by the Communicable Diseases Network Australia,⁷ with nearly all jurisdictions implementing the new definitions in January 2004 (New South Wales commenced in August 2004). Invasive pneumococcal disease and laboratory-confirmed influenza became notifiable to the NNDSS in 2001. Varicella and herpes zoster became nationally notifiable in 2006 in all Australian jurisdictions except New South Wales. However, data was not received from all notifying states until early 2008. Rotavirus infection is not notifiable to NNDSS, although it became notifiable in the Northern Territory in 1994, and laboratory notifiable in Queensland and Western Australia in 2005 and 2006, respectively.

The data collected by the NNDSS are frequently updated by jurisdictions. For this report, data extracted from the NNDSS as at 4 August 2008 were examined (except for pneumococcal disease for which data extracted on 5 December 2008 were analysed). Data were checked and cleaned where apparent errors were detected through consultation with appropriate surveillance staff in states and territories. There would be minor variations with NNDSS data referred to in the 2006 and 2007 Australia's Notifiable Disease Status reports (the annual reports of the NNDSS) and the biennial AIHW publication *Australia's Health 2008*,^{5,8,9} since different data versions were used for analysis. Disease notification data for cases with a date of diagnosis between 1 January 2006 and 31 December 2007 (2 years) are included in this report. Notifications with onset dates between 1 January 1993 and 31 December 2005 have been reported previously.¹⁰⁻¹³ It should be noted that historical notification data included in this report have been updated from previous reports.

In this report, notification data are reported and presented by the 'date of diagnosis'. Previous reports on data prior to 2005 analysed notification data by date of onset (if the date of onset from the clinical history was collected and was available), or the specimen collection date for laboratory-confirmed cases. As of mid 2005, a 'date of diagnosis' field was generated for every NNDSS record. For each notification record, a date of diagnosis

is derived from the date of onset, or, where not supplied, the earliest date recorded among these fields: date of specimen, date of notification, or date when the notification was received (the only mandatory date field). This algorithm applies to all diseases collected by the NNDSS except for 'hepatitis B, unspecified' and 'hepatitis C, unspecified' (not included in this report), where the onset date would not contribute to assigning the date of diagnosis.

The variables extracted for analysis for every vaccine preventable disease in this report are: the disease, the date of diagnosis, age at onset, sex, and state or territory of residence. Comments on other fields, including their completeness, are made where relevant to a specific disease. These fields include whether the case met the definition of a confirmed case, the serogroup/serotype of the causal organism, the vaccination status (and its validation status) of the case, and whether mortality was reported.

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 (the process by which an admitted patient completes an episode of care by being discharged, dying, transferring to another hospital, or changing type of care).¹⁴ The two most recent (financial) years for which hospitalisation data are available (1 July 2005 to 30 June 2007) are included in this report. Cases with hospital separation dates between 1 July 1993 and 30 June 2005 (12 years) have been reported previously.¹⁰⁻¹³ For trend analysis, this report presents some of these previously analysed historical data for years prior to and including 2004/2005 together with updated data for the 2 years 2005/2006 and 2006/2007.

As hospitalisation data for the reporting periods are defined by the date of separation of a hospitalisation episode, analyses by most variables such as age and sex are grouped by the financial year within which the hospital separation occurred. The exception is with analysis of seasonal trends by month (secular trend), when available data (based on hospital separation dates within the reporting period) are presented and reported by the month of hospital admission (as distinct from hospital separation).

Data for each reported disease are extracted based on the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification (ICD-10-AM). Eligible records of hospital separations include those with the code(s) of interest listed as the principal diagnosis (the diagnosis established after study to be chiefly responsible for occasioning an episode of admitted patient care, an episode of residential care or attendance at a health care establishment) or as any other diagnosis for that episode of hospitalisation. The proportion of hospital separations where the disease of interest has been coded as the principal diagnosis is reported for each disease. For hepatitis B disease, only hospitalisations with acute hepatitis B coded as the principal diagnosis are included, consistent with the approach taken in previous reports. Where the ICD-10-AM code for a disease specifies a severe manifestation of the disease (e.g. measles encephalitis), the number and type of these episodes with these diagnoses are reported as complications of the disease.

The variables extracted for analysis include: date of admission, financial year of separation, age on admission, sex, state or territory of residence, length of stay (LOS), and diagnosis (principal and other diagnoses – up to 31 diagnoses could be recorded for each admission) coded using the relevant edition of ICD-10-AM for the collection period. In addition, the mode of separation (whether death was the outcome of that hospitalisation episode) has been specifically analysed for hospitalisations due to meningococcal disease, as this measure is considered to be an important indicator of this disease. Hospitalisation episodes with fatal outcome are also reported as supplementary information for a few other diseases (e.g. invasive pneumococcal disease). For hospitalisation records where data on the jurisdiction of residence are missing, the jurisdiction in which the hospitalisation occurred is used to replace the jurisdiction of residence datum. This substitution was required in 0.6% (214/35,859) of all the records of hospitalisation in 2005/2006 and 2006/2007 analysed for this report.

Appendix 6.3 summarises the hospitalisation data by disease, year and jurisdiction. Regarding hospitalisation data in this publication, all Australian jurisdictions except New South Wales, Queensland and South Australia have required suppression for all cells with counts of less than five (but non-zero). In these tables, the exact count in each of these cells has been replaced by the symbol '<5' for these jurisdictions. Data in additional selected cells have to be suppressed (denoted 'n.p.') to prevent back calculation (by subtraction from row or column totals) of the suppressed counts of data from the five jurisdictions.

Deaths

Death data were obtained from the AIHW National 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 death data published by the ABS 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, this report only analyses records in which the disease of interest is recorded as the underlying cause of death, consistent with previous reports. Deaths analysed in this report are the deaths registered in the two calendar years of 2005 and 2006 (not necessarily the year in which the death occurred). The variables extracted for each death record are: underlying cause of death, age, year death was reported, sex, and state or territory in which the death was recorded.

Calculations

All rates are calculated using the mid-year estimated resident populations, released by the Australian Bureau of Statistics in December 2007, as population denominator. 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. The reported rate estimates for the populations not stratified by age groups (i.e. all ages together) are crude rates that have not been age-standardised.

For hospitalisation data, the mid-year population estimate for the first half of the financial year is used as the denominator; for example, the 2005 mid-year population estimate is used to calculate rates for 2005/2006. (This is consistent with previous reports in this series.) For notification and death data, the mid-year population estimates for the corresponding calendar year are used as the denominator population. Averages are calculated for rates of notifications and hospitalisations and for bed-days of hospitalisation episodes per year. The median (rather than average) and range are used to describe the distribution of notifications and hospitalisations per month, and the length of stay per hospitalisation episode, as these data are not normally distributed. Where there are ≤ 3 hospitalisation episodes recorded per age/disease category in the national hospitalisation data, the median length of stay data are 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 – hospitalisation bed days; length of stay of hospitalisation; proportion of hospitalisations with disease attributed as the principal diagnosis over any one of the diagnoses; complications; mortality; by age categories standardised across different reported diseases, as in previous reports
- age and sex distribution – by age groups and sex as relevant for each particular disease
- geographical distribution – by state or territory by year, as shown in Appendices 6.2 and 6.3; for hospitalisation data, some jurisdictions required suppression of hospitalisation data for those cells where there were fewer than 5 cases
- vaccination status of notified cases and laboratory serogroup/serotype information, as relevant
- comments – commentary and discussion on the presented data

Vaccination coverage data

Previous reports in this series have included a section on childhood vaccination coverage. However, vaccination coverage reports are now published separately – *Immunisation coverage annual report 2007*¹⁵ and *Immunisation coverage annual report 2008*.¹⁶

Notes on interpreting data

Vaccine preventable diseases data in general

Comparison between the notification, hospitalisation and death data should be made with caution since these datasets differ in their purposes of data collection, reporting mechanisms, accuracy, timeliness and period of reporting.

In this report, in order to provide the most recent information available, and to accommodate the varied reporting formats, data of different time periods (although all being the available data for the most recent 2 years) have been selected for review from each dataset. As there are no unique identifying codes to link records for the same individual across these datasets, and due to differences in defining a case and in the completeness and the accuracy of the data in each dataset, it is not possible to analyse deaths and hospitalisations as subsets of notifications.

For some diseases, there are no specific ICD codes that correspond to the particular disease condition of interest. This will limit the validity of comparisons between notification and hospitalisation or death data. Examples include invasive pneumococcal disease and invasive *Haemophilus influenzae* type b disease. The methods and algorithms used to select surrogate ICD codes to match the notification case definitions are explained in the relevant disease chapters and in the notes on interpreting hospitalisation data in the following section.

The rates presented in this report are crude rates and may be confounded by differences in the population (e.g. age structure, 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 versus 20.5 per 100,000 age-standardised; hepatitis B crude rate 6.8 per 100,000 versus 5.7 per 100,000 age-standardised). The Northern Territory is the jurisdiction with a population age structure most different from other jurisdictions. In view of this, and to maintain consistency with previous reports in this series, this report continues to report using crude rates. It is also important to note that high disease rates may be observed even with small absolute numbers of cases in jurisdictions with small populations (e.g. the Australian Capital Territory, Tasmania, and the Northern Territory), and a small change in the numbers may result in a relatively large change in rates.

To assist with interpreting data on the proportion of disease in various age groups, the proportions of the estimated Australian population in various age groups used in the standard tables and an additional age grouping used in some chapters presented in this report are tabulated in Tables 2.1 and 2.2.

Notification data

A major limitation of the notification data is that they represent only a proportion of all the cases occurring in the community, due to under-reporting. This proportion may vary between diseases, over time, and across jurisdictions. An infectious disease that is diagnosed by a laboratory test is more likely to be notified than if it is diagnosed only on clinical grounds. Data accuracy may also vary among jurisdictions due to the use of different case definitions for surveillance (prior to adoption of the national case definitions) and varying reporting requirements and mechanisms by medical practitioners, hospitals and laboratories. While in three jurisdictions $\geq 95\%$ of notifications originated from laboratories only, 43%–59% of notifications in three other states originated from both doctors and laboratories.⁵ Under-reporting of notifiable diseases by doctors and from hospitals has been documented in Australia.^{17–19}

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 (2005/2006, 2006/2007), there were approximately 7.3 million and 7.6 million separations in each financial year, respectively.^{14,20} Almost all public and private hospitals were included in each of these periods.

Table 2.1: Proportions of the Australian population in the age groups used in standard disease data tables, by year

Year	Age group					Total
	0–4 yrs	5–14 yrs	15–24 yrs	25–59 yrs	60+ yrs	
2005	6.3%	13.4%	13.8%	48.9%	17.5%	100.0%
2006	6.3%	13.2%	13.9%	48.8%	17.8%	100.0%
2007	6.3%	13.1%	13.9%	48.5%	18.2%	100.0%

Table 2.2: Proportions of the Australian population in the age groups used in some specific disease data tables or figures, by year

Year	Age group					Total
	0–4 yrs	5–9 yrs	10–19 yrs	20–59 yrs	60+ yrs	
2005	6.3%	6.6%	13.7%	55.9%	17.5%	100.0%
2006	6.3%	6.5%	13.6%	55.8%	17.8%	100.0%
2007	6.3%	6.4%	13.5%	55.6%	18.2%	100.0%

The AIHW performs logical validations on the ICD-10-AM coded data; for example, for sex- and age-specific diagnoses. Coding audits and coding quality improvement activities are variously performed at hospital level and/or state and territory level, and in some states also enhanced by using software such as PICQ (Performance Indicators for Coding Quality) developed by the National Centre for Classification in Health (NCCCH).²¹ Generally, states and territories consider that coding of the hospitalisation data in recent years has been of high quality.¹⁴

Some variation in hospital access, admission practices and record coding may occur between regions and over time and this may 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 among coders²² and hospitals performing coding audits and other quality initiatives to assess the quality of the coded data (M Cumerlato, NCCCH, personal communication, September 2009). The National Clinical Coder Workforce Survey of over 1,000 Australian coders in 2002 found that just over half of clinical coders held tertiary qualifications, and 10% of them had no formal coding education. About two-thirds of coders reported undertaking regular quality assurance activities relevant to clinical coding.²²

In 1998/1999, most states and territories began using ICD-10-AM and, since 1999/2000, all jurisdictions use the new classification. This change may impact on the sensitivity and specificity of some diagnostic codes relevant to this report, especially with respect to historical trend analyses. The NCCCH updates the ICD-10-AM every 2 years, under the guidance of the Australian Coding Standards Advisory Committee.^{23,24}

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 errors and may either occur along the patient pathway (e.g. level of details documented in medical records, clinicians' experience) or along the paper trail (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]).²⁵ A Canadian study based on four teaching hospitals showed the sensitivity of the validity of coding of hospital discharge data to range from 9.3% to 83.1% using International Statistical Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM) and 12.7% to 80.8% using ICD-10 codes, and varied with the conditions assessed.²⁶ A study of pertussis in children's hospitals in Sydney noted that, while 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.²² 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.²⁷

For a few rare diseases, such as acute poliomyelitis, tetanus and diphtheria, as indicated in the relevant disease chapters, some of the hospitalisation episodes or deaths that have been coded to be due to these diseases are likely to be coding errors or the coding error could be related to inaccurate documentation, as suggested by the short lengths of stay of the hospitalisation episodes and the lack of notification of that disease to public health authorities.

The ICD codes of diagnosis chosen for analysis of a disease should accurately reflect the condition of interest. For some diseases, such as Hib 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, for this report, only the ICD code of G00.0 (*Haemophilus meningitis*) was selected as the indicator for hospitalisation due to *H. influenzae* type b disease, as other codes, including those of *H. influenzae* pneumonia, *H. influenzae* septicaemia, *H. influenzae* infection and acute epiglottitis, are considered insufficiently specific. Wood et al have 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.²⁸ Generally, codes are most likely to reflect the disease accurately when the disease can be clearly defined with observable signs and symptoms, when information about the patient is documented by highly qualified physicians, when the coders are experienced and have full access to clinical information while assigning the codes, and if the codes are not new.²⁵ For each disease in this report, the ICD code(s) that have been selected to constitute the indicator for hospitalisation due to the disease are listed in the 'case definition' box on the first page of each disease chapter.

It must be noted that in the AIHW hospitalisation database, there is one record for each hospital admission episode. This means that there will be separate records for each re-admission or inter-hospital transfer. This is unlikely to have a major impact on the numbers reported for most of the diseases reviewed in this report, as they are mostly acute diseases. It should also be noted that it is difficult to gauge the relative importance of hospitalisations where the coded disease of interest was not the principal diagnosis but was recorded as an additional or secondary diagnosis for that hospitalisation episode. This indicates that the condition might be a co-morbidity.

Hospitalisations represent the more severe end of the morbidity spectrum of a disease, and the extent to which ICD-coded hospitalisation data can reflect the burden of the disease of interest varies with diseases. The general limitation of this data source is that hospitalisation may be affected by variations in admission practices and the availability of and access to hospitals should also be noted.¹⁴

Death data

Mortality data are reported and analysed by the year of registration rather than by year of death. This avoids problems associated with 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,²⁹ the bulk of which are deaths that occurred in December of that calendar year.

In this report, only the death records in which the disease of interest was recorded as the underlying cause of death (i.e. the single disease that initiated the train of morbid events leading directly to death) are reported. Hence, deaths where the disease of interest was a contributing cause of death are not included. The extent of underestimation due to this limitation varies with different diseases.

The problems associated with the accuracy of ICD coding used for hospital separations, discussed above, may also be relevant for the mortality data. In Australia, information on the cause of death is reported routinely for every death on a standard Medical Certificate of Cause of Death completed by a medical practitioner or a coroner. The person completing the certificate must nominate the underlying (principal) cause of death and any associated conditions.²⁹ The accuracy in ascertaining the cause of death may 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)³⁰ and inaccuracy in cause of death certification, compared to the gold standard of autopsy findings, has been documented,³¹⁻³⁴ although the studies were mainly based on non-infectious conditions. A recent meta-analysis estimated that at least one-third of deaths may be misclassified on death certificates and half of autopsies produced findings unsuspected before death (although the leading discrepant diagnoses were pulmonary embolism, cardiovascular disease, pneumonia and infections at other sites).³⁵ Studies have found that infectious diseases being the missed or discordant diagnosis when comparing clinical and autopsy diagnoses were not uncommon, although vaccine

preventable diseases were not specifically identified.^{36,37} In the case of pertussis and tetanus, studies have documented that deaths due to these diseases, which can be otherwise identified through disease surveillance systems and hospitalisation records, sometimes go unrecorded on death certificates.^{38,39}

In addition, newer versions of the ICD codes were used in more recent times, as necessitated by the increasing precision in identifying conditions required as medical understanding grew. The number of causes of death recorded by the ABS increased from 187 in 1907 to around 2,850 in 2000.²⁹ Thus, despite comprehensive mapping algorithms, which attempt to take into account changing disease classification over time, caution is required in interpreting trends in these mortality data. Australia adopted the use of the Automated Coding System (ACS) and introduced ICD-10 codes for processing deaths registered from 1 January 1997. Causes of death were classified by ICD-9 for deaths registered from 1979 to 1996 in Australia.²⁹ As a result, there could be some discontinuity in the underlying causes of death series between 1996 and 1997. 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

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3. Vaccine preventable diseases

3.1 Diphtheria

Diphtheria is an acute toxin-mediated systemic disease caused by the bacterium *Corynebacterium diphtheriae*. Infection remains localised to the throat or skin and disease is mainly due to local inflammation with membrane formation and/or systemic toxæmia. Pharyngeal diphtheria presents with a membranous inflammation of the upper respiratory tract, which may be extensive enough to cause airway obstruction. Damage to other organs including the myocardium, nervous system and kidneys, caused by the organism's exotoxin, may complicate pharyngeal or cutaneous diphtheria.^{1,2} Non-toxigenic *C. diphtheriae* usually causes mild throat or skin infection, which is occasionally complicated by invasive disease including endocarditis or septic arthritis. *Corynebacterium ulcerans*, a bacterium found in cattle and cats, can also express diphtheria toxin and cause a zoonotic infection in humans that is similar to diphtheria.³⁻⁶

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

National definition from January 2004:⁷

Both confirmed and probable cases are notifiable.

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

OR

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

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

OR

Clinical evidence as above and an epidemiological link to a confirmed case (probable case).

Hospitalisations and deaths

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

Notifications, hospitalisations and deaths

There were no notifications of diphtheria in 2006 or 2007. For the 2-year period 2005/2006 to 2006/2007, there were 49 hospitalisations coded as diphtheria. Most were cutaneous (A36.3; n=31), or classified non-specifically as other (A36.8; n=9) or unspecified (A36.9; n=8) diphtheria. Slightly more males than females were hospitalised with diphtheria (male:female ratio 1.5:1). The highest average annual rate of hospitalisation coded as diphtheria occurred in the Northern Territory (6.2 per 100,000) although diphtheria was not the principal diagnosis of any of these hospitalisation episodes. There was 1 death coded as due to diphtheria (unspecified) in a young adult male (age group 30–35 years) in 2006. However, there were no notifications or hospitalisations coded as due to diphtheria in the relevant jurisdiction in 2006.

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,⁸ but these are not classified as diphtheria in the absence of relevant symptoms. In the absence of any notifications during the period 2006–2007, the 49 hospitalisations, with only one having diphtheria as the principal diagnosis, were presumably non-toxigenic or culture negative suspected diphtheria cases or coding errors. It is very likely that the higher rate of hospitalisations coded as

diphtheria in the Northern Territory reflects the detection of non-toxigenic types endemic in the region. The death coded as due to diphtheria in 2006 is likely to be a coding error as the relevant jurisdiction has confirmed that there were no notified cases in 2006.

Diphtheria is still a global problem with 15 countries in Asia, Africa, the Middle East, the Caribbean and Europe reporting 10 or more cases of diphtheria to the World Health Organization in 2007;⁹ a total of 4,190 cases were reported globally in 2007. Notably, more than 3,000 cases were reported in 2007 from India,⁹ where diphtheria remains endemic.¹⁰ Five recent studies of diphtheria cases in different regions of India have each documented lack of immunisation as a major risk factor.^{10–14} The large outbreak of diphtheria in the newly independent states of the former Soviet Union in the 1990s¹⁵ underscored the risk of diphtheria returning when high vaccination coverage in children (who are critical vectors of respiratory transmission) is not maintained. A specific diphtheria surveillance network has recently been established in Europe.¹⁶

In countries with high childhood vaccination coverage against diphtheria, such as Australia, the United Kingdom (UK), Germany, the United States of America (USA) and Canada, cutaneous lesions are the most common manifestation of *C. diphtheriae* infection. Cutaneous infection may be caused by local circulating non-toxigenic strains¹⁷ (which can also cause invasive disease, including bacteraemia, endocarditis and septic arthritis, particularly in persons with risk factors such as homelessness, alcoholism or diabetes)¹⁸ or by imported toxigenic types due to overseas travel.^{19,20} Cutaneous *C. diphtheriae* infection (due to toxigenic or non-toxigenic strains) may be difficult to diagnose due to a low index of suspicion,^{21,22} may cause chronic infection, and may serve as a reservoir for ongoing transmission with greater efficiency than respiratory infection.^{17,19} 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.^{23,24} Australian serosurveillance data indicate that, while childhood protection is excellent (>99%), waning immunity in adults has resulted in a potentially susceptible population with travel the most likely source of exposure.²⁵ Australians travelling to countries where diphtheria remains a problem, including countries in the Asia–Pacific region such as India, Pakistan, Indonesia, the Philippines, Nepal, Bangladesh and Vietnam,⁹ should ensure that they are protected against diphtheria through booster immunisation.²⁶

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3.2 *Haemophilus influenzae* type b 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. Based on their capsular polysaccharide, *H. influenzae* can be further characterised into six types designated a to f; there are also non-typeable strains. Hib has most often been associated with invasive disease, and before Hib vaccines became available, caused at least 95% of invasive disease due to *H. influenzae* in children.¹⁻³ Prior to the introduction of vaccination, the most common manifestation of invasive Hib disease globally was meningitis, with children aged <18 months most at risk.^{1,2} 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.⁴ Survivors of Hib meningitis commonly had neurological sequelae such as deafness and intellectual impairment.¹⁻³ Epiglottitis was the other major category of infection with particularly high rates observed among non-Indigenous Australians, most often occurring in children aged >18 months. Other manifestations of Hib disease include cellulitis, septic arthritis, pneumonia, pericarditis, osteomyelitis and septicaemia.

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

Invasive Hib infection – national definition from January 2004:⁵

Only confirmed cases are notifiable.

- a) Isolation of *Haemophilus influenzae* type b 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 considered insufficiently specific for invasive *H. influenzae* type b disease.

Secular trends

During the 2 years from January 2006 to December 2007, a total of 39 invasive Hib infections were notified. The average annual notification rate was 0.09 per 100,000 population (Table 3.2.1). A median of 1 case (range 0–6) was notified per month (Figure 3.2.1). There were 25 hospitalisations (average annual rate 0.06 per 100,000) recorded as *Haemophilus meningitis*, with a median of 0.5 cases (range 0–5) hospitalised per month.

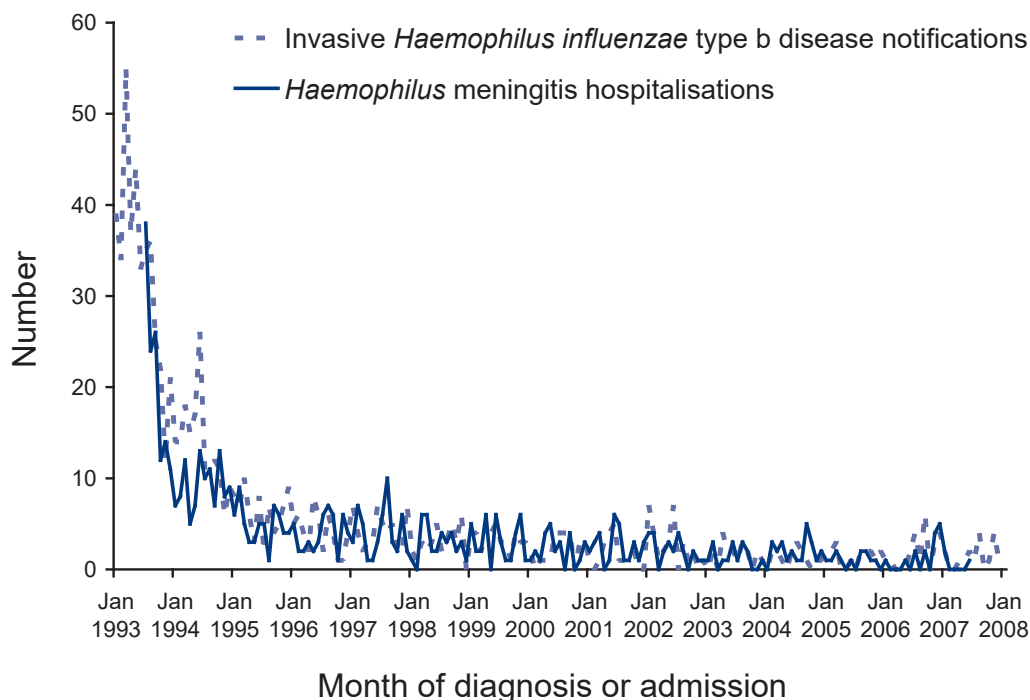
Severe morbidity and mortality

Hib morbidity and mortality data are drawn from three data sources: *Haemophilus meningitis* hospitalisation data, the AIHW National Mortality Database and NNDSS (Table 3.2.1).

Notification and hospitalisation rates by age group were broadly concordant, with the exception of the 5–24 years age group in whom no hospitalisations due to *Haemophilus meningitis* were reported between July 2005 and June 2007 despite notifications over this period. The total number of hospital bed days recorded for patients with this diagnostic code was 322 (average 161 bed days per year). The overall median length of stay for hospitalisations with a principal diagnosis of *Haemophilus meningitis* was 10.5 days.

Mortality data can be drawn from these three databases. However, these datasets are not linked and data are available from differing time periods – the AIHW National Mortality Database data from 2005–2006, the hospitalisation data from 2005/2006–2006/2007, and NNDSS from 2006–2007. Two deaths attributable to Hib were reported from these data sources during these overlapping reporting periods. In 2005, the death of a person aged ≥85 years due to *Haemophilus meningitis* was recorded in the AIHW National Mortality Database. This

Figure 3.2.1: *Haemophilus influenzae* type b notifications and *Haemophilus meningitis* hospitalisations for all ages, Australia, 1993 to 2007,* by month of diagnosis or admission



* Notifications where the date of diagnosis was between January 1993 and December 2007; hospitalisations where the date of admission was between July 1993 and June 2007.

Table 3.2.1: *Haemophilus influenzae* type b notifications, *Haemophilus meningitis* hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	<i>Haemophilus influenzae</i> type b notifications 2 years (2006–2007)		<i>Haemophilus meningitis</i> hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission (days)		<i>Haemophilus meningitis</i> deaths 2 years (2005–2006)	
	n	Rate [‡]	n	(§)	Rate [‡]	(§)	Median	(§)	n	Rate [‡]
0–4	21	0.80	15	(14)	0.58	(0.54)	11.0	(10.0)	0	–
5–14	4	0.07	0	(0)	–	(–)	–	(–)	0	–
15–24	2	0.03	0	(0)	–	(–)	–	(–)	0	–
25–59	5	0.02	5	(4)	0.02	(0.02)	10.0	(9.5)	0	–
60+	7	0.09	5	(4)	0.07	(0.06)	13.0	(16.5)	1	0.01
All ages	39	0.09	25	(22)	0.06	(0.05)	11.0	(10.5)	1	<0.005

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

record appeared to correspond to the only case of *Haemophilus meningitis* hospitalisation that resulted in death in 2005/2006. Among the invasive Hib disease cases notified to NNDSS in 2006 and 2007, there was 1 death in an Indigenous female infant in 2006.

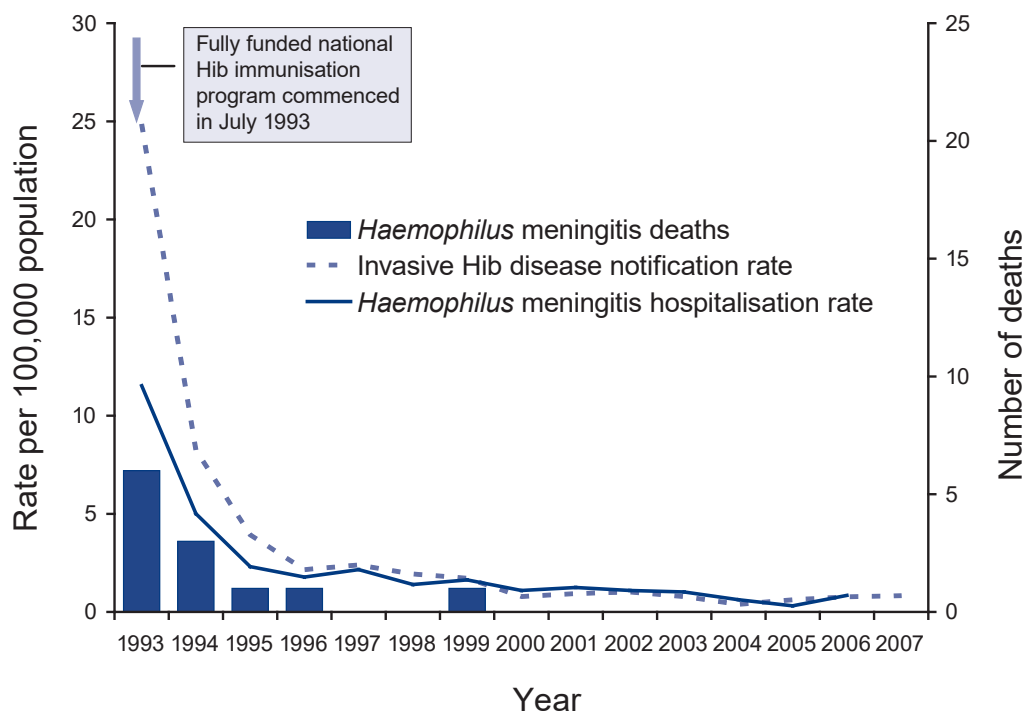
Age and sex

Discrepancies in case ascertainment between data sources were apparent but also expected given that only notification data are specific for serotype b. The rate of hospitalisations coded as *Haemophilus meningitis* was higher in males than in females, with a male:female ratio of 3.2:1. However, the rate of notifications to the NNDSS for invasive Hib disease was higher in females than in males with a male:female ratio of 0.63:1. These ratios were dominated by those from the 0–4 years age group which represented most of the notifications.

In children aged 0–4 years, there were 21 notifications of invasive Hib disease, accounting for 53.8% (21/39) of all notifications. Sixty-seven per cent (15/25) of all *Haemophilus meningitis* hospitalisations were in this age group with no reported deaths (Table 3.2.1). Ten of the 21 notifications and 9 of the 15 hospitalisations occurred in infants aged <1 year. Three of the 21 notifications and 4 of the 15 hospitalisations occurred in infants aged 1 to <2 years. For most age groups, the age-specific notification rates approximated the age-specific *Haemophilus meningitis* hospitalisation rates.

Since 1993 and after the introduction of universal childhood Hib immunisation, all measures of invasive Hib disease in children aged 0–4 years, who previously had the highest disease incidence, have progressively fallen, though less steeply in recent years (Figure 3.2.2). In this age group, the number of cases notified annually has decreased from approximately 27 in the late 1990s to remain at between 5 and 13 cases annually from 2000 to 2007. It should be noted that there are a number of caveats around the available death data. Between 1993 and 2007, NNDSS recorded 5 deaths as due to invasive Hib disease in the 0–4 years age group, whereas 12 deaths were coded as due to *Haemophilus meningitis* in the AIHW National Mortality Database for this age group over the same time period.

Figure 3.2.2: *Haemophilus influenzae* type b notification and *Haemophilus meningitis* hospitalisation rates and numbers of deaths* for children aged 0–4 years, Australia, 1993 to 2007†



* Hospitalisations and deaths coded as *Haemophilus meningitis* for the period up to June 2007 (hospitalisations) and December 2006 (deaths).

† Notifications where the date of diagnosis was between January 1993 and December 2007; hospitalisations where the date of separation was between July 1993 and June 2007; deaths where the death was recorded between January 1993 and December 2006.

Vaccination status

Completion of the vaccination status field in NNDSS was expected for all notifications of invasive Hib in subjects born after 31 December 1987. Twenty-six of 27 cases (96.3%) who were born after this date had this field completed; however, 2 of these 26 were entered as 'unknown'. Among the 24 cases whose vaccination status was known, 19 had this validated from the Australian Childhood Immunisation Register or written records, accounting for all cases classified as fully (n=12) or partially vaccinated (n=2), and 5 of the 10 cases classified as non-vaccinated.

Of the 14 cases notified in children aged 1–<10 years in 2006 and 2007, 2 (14%) were in partially vaccinated children aged 1 year, 3 (21%) were in non-vaccinated children aged 1–2 years, and vaccination status was unknown for 1 case (7%) aged 5 years. The other 8 notifications were in children who were reported as fully vaccinated; their median age was 3 years.

There were 10 notifications in infants aged <1 year, of whom 6 were in infants aged <6 months. Of these 6 notifications, 1 case was reported as fully vaccinated for age, 4 as unvaccinated, and vaccination status was unknown for 1 case.

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 remained small (Appendices 6.2 and 6.3).

Comment

This report is the second in the series of NCIRS national reports on vaccine preventable diseases that excludes hospitalisations recorded as epiglottitis as a measure of Hib disease. This is because a previous 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%) a result of incorrect coding.⁶ Hospitalisation data now includes *Haemophilus meningitis* only, and, although type-specific hospitalisation data are still not available, these cases, even when *Haemophilus meningitis* is the primary diagnosis, could in fact be due to non-type b disease or non-typeable *H. influenzae*. (See Methods section for further details regarding the limitations of the data sources utilised.)

There have been three eras of Hib vaccination in Australia.³ During the first era from 1993 until June 2000, all children resident in the Northern Territory received 2 doses of a vaccine containing the Hib component conjugated to an outer membrane protein derived from *Neisseria meningitidis* (PRP-OMP) at 2 and 4 months of age, with a booster at 12 months of age, whereas all children in the remaining states and territories received 3 doses of a vaccine containing the Hib component conjugated to a mutant diphtheria toxin (HbOC) at 2, 4 and 6 months of age, with a booster at 18 months of age. In 2000, the second era began in Australia with all children receiving 2 doses of PRP-OMP vaccine at 2 and 4 months of age, with a booster at 12 months of age. The third era commenced during this review period, in November 2005. All children resident in the Northern Territory, Queensland, Victoria and South Australia and Indigenous children in Western Australia were administered PRP-OMP vaccine with 2 doses at 2 and 4 months of age and a booster dose at 12 months of age, while all children in New South Wales, the Australian Capital Territory and Tasmania and non-Indigenous children in Western Australia received a vaccine containing the Hib component conjugated to a tetanus toxoid protein (PRP-T) with 3 doses administered at 2, 4 and 6 months of age and a booster dose at 12 months of age.³

Vaccination status data indicate that, consistent with the very high immunisation coverage reported for Hib vaccine (approaching 95%), many of the 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 25 years of age. An assessment of preventable cases of invasive Hib disease in vaccine eligible children between July 2000 and December 2005 reported that the proportion of cases identified as preventable did not differ significantly

between Indigenous and non-Indigenous children.³ Furthermore, this report indicated that approximately 60% of invasive Hib disease cases were preventable as they occurred among those who were either not immunised or not fully immunised.³

Incidence of invasive Hib disease in Australia may not decrease much more than the very low incidence now reached, as it is consistent with the lowest disease rates reported internationally.^{7,8} The UK experienced a resurgence of Hib infections 8 years following introduction of an accelerated primary schedule, with disease control re-established following a national catch-up immunisation campaign and the addition of a routine 4th (booster) dose in the 2nd year of life.^{9,10} This experience highlights the importance of continued post-licensure surveillance of vaccine preventable diseases. The rarity of invasive Hib disease re-emphasises the importance of laboratory confirmation of all suspected cases, ideally by typing with polymerase chain reaction (PCR) in a reference laboratory.

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3.3 Hepatitis A

Hepatitis A is caused by the hepatitis A virus (HAV), an RNA virus classified within the genus hepatovirus of the picornavirus family. There is only one human HAV serotype.^{1–3} Hepatitis A infection causes an acute inflammatory disease of the liver and can produce either asymptomatic or symptomatic infection. Clinical manifestations of symptomatic infection vary from mild anicteric illness to fulminant hepatic failure. Severity tends to increase with age and case fatality is normally low (0.1%–0.3%).⁴ HAV infection typically has a sudden onset of symptoms that can include fever, anorexia, malaise, nausea, and abdominal discomfort followed by jaundice and dark urine.^{5,6} The likelihood of having symptoms with HAV is related to age. Only 10%–50% of infections acquired before the age of 5 years are symptomatic, while 70%–95% of infected adults show clinical symptoms.⁶

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

National definition from January 2004:⁷

Both confirmed and probable cases are notifiable.

- a) Detection of anti-hepatitis A virus IgM antibody, in the absence of recent vaccination (confirmed case); *or*
- b) Detection of hepatitis A virus by nucleic acid testing (confirmed case); *or*
- c) Clinical hepatitis (jaundice and/or bilirubin in urine) without a non-infectious cause *and* an epidemiological link to a laboratory-confirmed case (probable case).

Hospitalisations and deaths

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

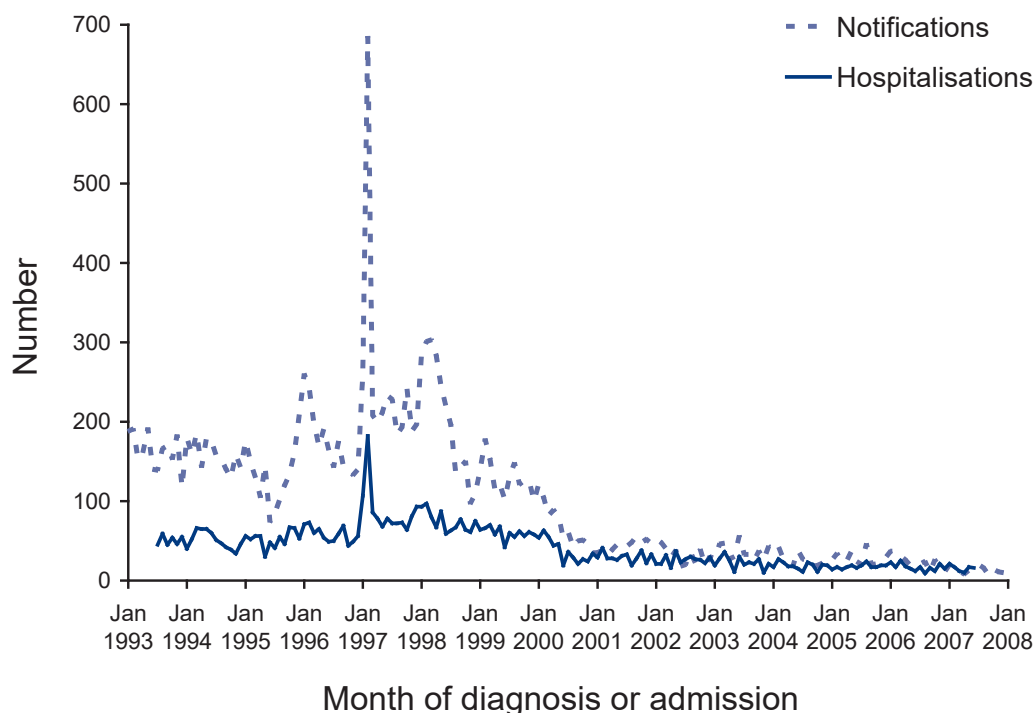
Secular trends

There were 445 hepatitis A notifications in the period January 2006 to December 2007 (average annual notification rate 1.1 per 100,000) (Table 3.3.1). Of these 445 notifications, 428 (96%) were confirmed cases, and 17 (4%) were probable cases. A median of 18 cases (range 7–37) were notified per month. There were 408 hospitalisations in the period July 2005 to June 2007 (average annual hospitalisation rate 1.0 per 100,000) with a median of 17 admissions (range 9–25) per month. The notification rate for hepatitis A infection in Australia has declined over recent years, from 1.8 per 100,000 population reported in 2003–2005⁸ to 1.1 per 100,000 population in 2006–2007, the lowest levels recorded since national data have been collated for notifications in the NNDSS (January 1991) and for hospitalisations (July 1993) (Figure 3.3.1). The large point source and community epidemics documented in the 1990s have not returned.

There was no apparent seasonality in notifications or hospitalisations.

Severe morbidity and mortality

In the review period July 2005 to June 2007, there were 2,617 hospital bed days (average 1,309 per year) recorded for patients with an ICD-10-AM code for hepatitis A. Overall, hepatitis A was the principal diagnosis in 55% of hospitalisations where hepatitis A was recorded (224 cases, average annual rate 0.5 per 100,000), declining from 95% in those aged 5–14 years to 42% in those aged ≥ 60 years. The median length of stay was longer for hospitalisations in those aged ≥ 60 years than for younger age groups (Table 3.3.1). There were no admissions recorded as hepatic coma (ICD-10-AM code B15.0) in the period of this report. In 2005–2006, hepatitis A was recorded as the underlying cause of 3 deaths (Table 3.3.1), 2 of them in people aged > 80 years and 1 in a person aged 44 years.

Figure 3.3.1: Hepatitis A notifications and hospitalisations, Australia, 1993 to 2007,* by month of diagnosis or admission


* Notifications where the date of diagnosis was between January 1993 and December 2007; hospitalisations where the date of admission was between July 1993 and June 2007.

Table 3.3.1: Hepatitis A notifications, hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Notifications 2 years (2006–2007)		Hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission (days)		Deaths 2 years (2005–2006)	
	n	Rate [‡]	n	(§)	Rate [‡]	(§)	Median	(§)	n	Rate [‡]
0–4	38	1.4	12	(7)	0.5	(0.3)	4.0	(2.0)	0	–
5–14	97	1.8	39	(37)	0.7	(0.7)	2.0	(2.0)	0	–
15–24	77	1.3	56	(41)	1.0	(0.7)	2.0	(2.0)	0	–
25–59	193	1.0	220	(105)	1.1	(0.5)	4.0	(4.0)	1	<0.005
60+	40	0.5	81	(34)	1.1	(0.5)	6.0	(4.5)	2	0.03
All ages	445	1.1	408	(224)	1.0	(0.5)	3.0	(3.0)	3	0.01

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

Age and sex distribution

Notification and hospitalisation rates for all age and sex groups continued to fall compared with previous years (Figures 3.3.2 and 3.3.3). In particular, the notification rate for males 15–34 years of age continued to decrease from the high rates in the latter part of the 1990s (Figure 3.3.2). For notifications, decreased rates in 2006–2007 occurred predominantly among people aged <20 years and 50–59 years. For hospitalisations, the decrease was most marked in the 0–9 years age group.

Notification rates tended to be lower in older age groups, while the reverse was true for hospitalisation rates. The highest notification rate was seen in males and females aged 5–9 years and the highest hospitalisation rate in males and females aged 50–59 years. Among persons aged <40 years, notifications exceeded hospitalisations, whereas the reverse was true in those ≥40 years of age and was particularly pronounced in the ≥60 years age group (Figure 3.3.4).

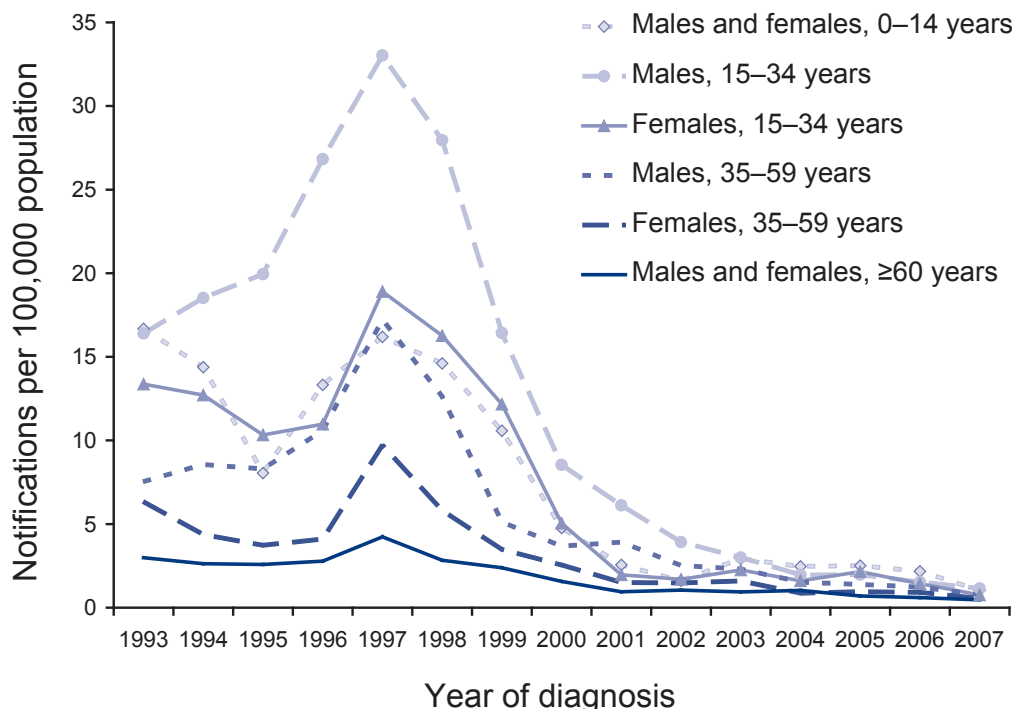
The sex ratio differed between age groups (Figure 3.3.2), with a marked male excess for those aged 15–59 years in earlier years, not seen in those aged <15 years or ≥60 years. For the two reporting years, the overall male:female ratio was 1.2:1 for both notifications and hospitalisations. Male predominance was reported mainly in those aged 15–59 years for notifications, and in those aged 35–59 years for hospitalisations. Two of the three reported deaths were in males.

Geographical distribution

Notification and hospitalisation rates for hepatitis A infection varied by jurisdiction, being higher in the Northern Territory, New South Wales and Western Australia than elsewhere during the respective reporting periods. The highest notification and hospitalisation rates were reported in the Northern Territory (8.2 per 100,000 population and 6.5 per 100,000 population, respectively). Rates in the other jurisdictions ranged from 0.4 to 2.1 per 100,000 for notifications and 0.5 to 1.1 per 100,000 for hospitalisations (see also Appendices 6.2 and 6.3).

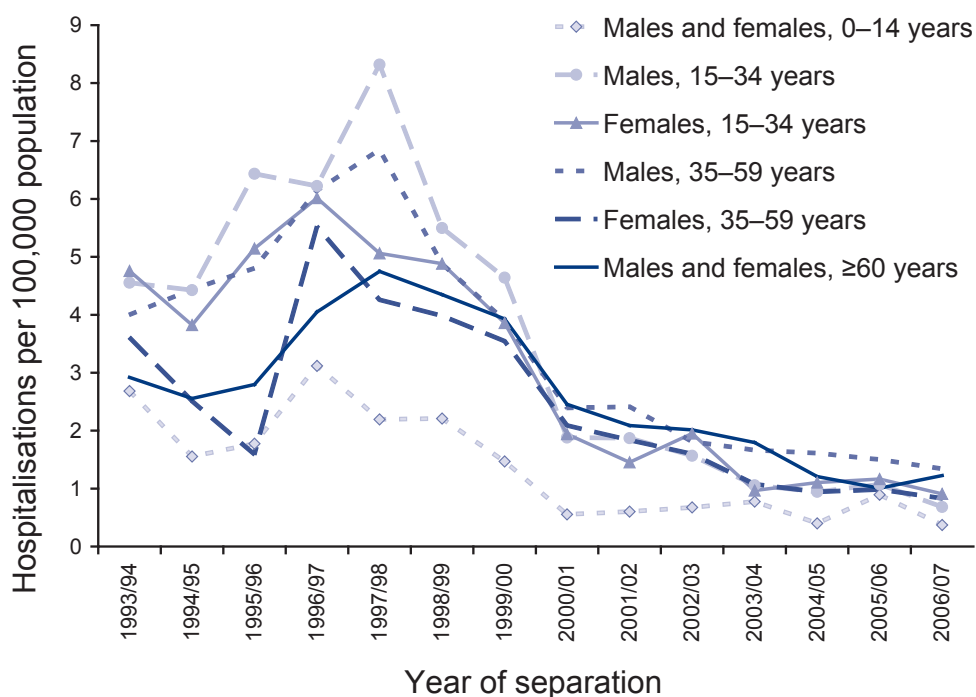
There were decreasing notification rates in all jurisdictions from 2003–2005 to 2006–2007 (Figure 3.3.5), particularly notable in the Northern Territory (Appendix 6.2). There were also decreasing hospitalisation rates in the Northern Territory, South Australia, Victoria and Western Australia from 2005/2006 to 2006/2007 (Appendix 6.3).

Figure 3.3.2: Hepatitis A notification rates, Australia, 1993 to 2007,* by age group, sex and year of diagnosis



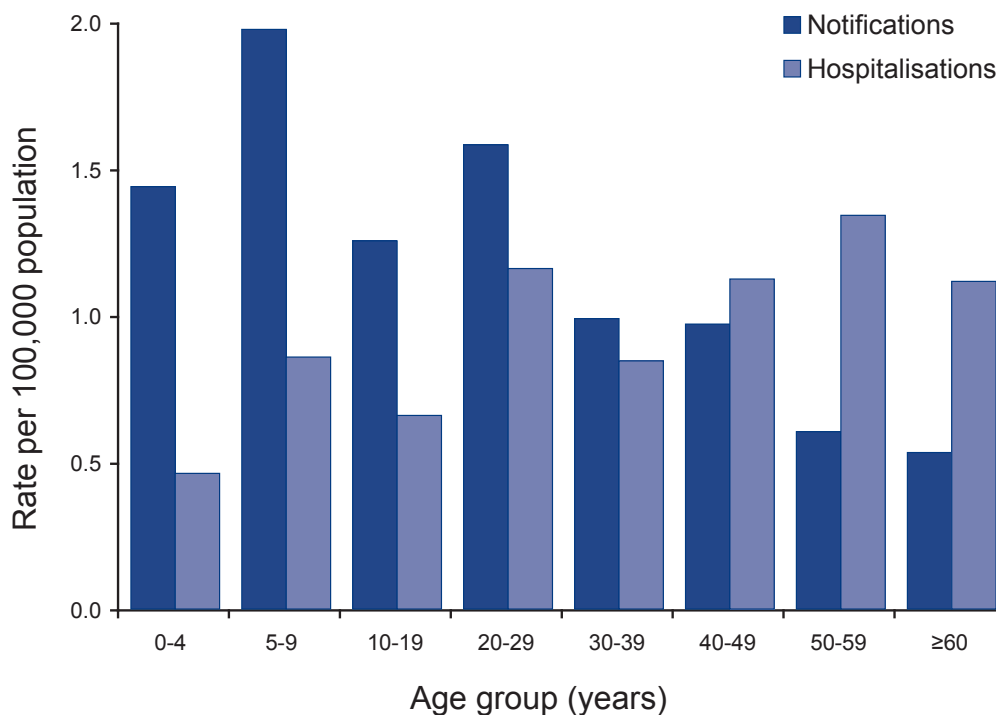
* Notifications where the date of diagnosis was between January 1993 and December 2007.

Figure 3.3.3: Hepatitis A hospitalisation rates, Australia, 1993/1994 to 2006/2007,* by age group, sex and year of separation

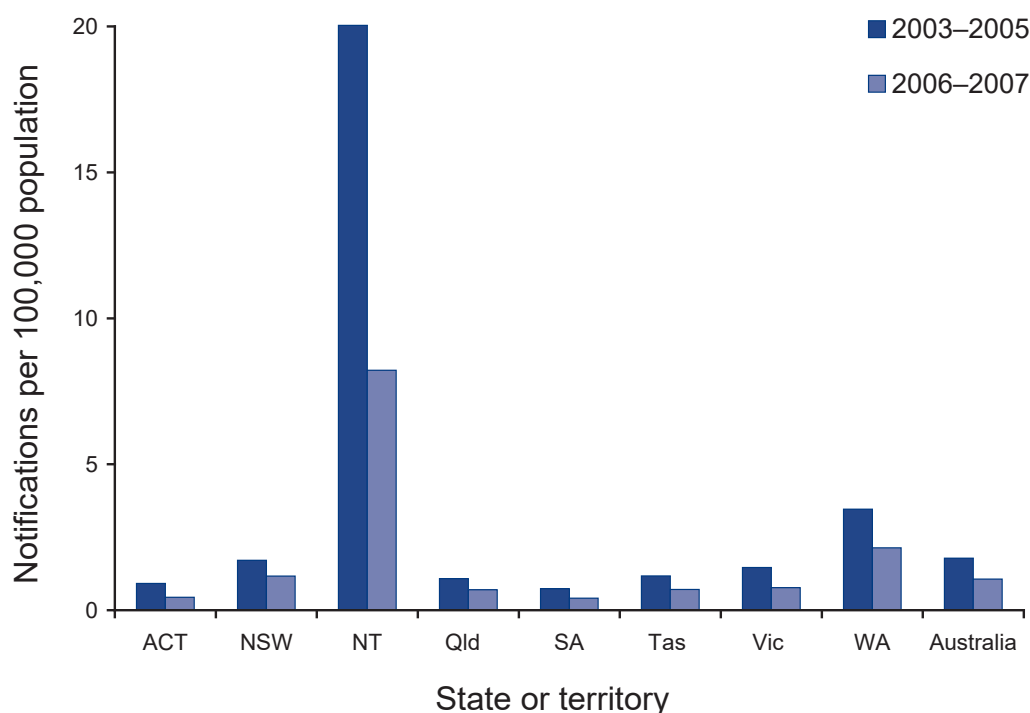


* Hospitalisations where the date of separation was between July 1993 and June 2007.

Figure 3.3.4: Hepatitis A notification rates, Australia,* 2006 to 2007 and hospitalisation rates, 2005/2006 to 2006/2007, by age group



* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007.

Figure 3.3.5: Hepatitis A notification rates, Australia, 2006–2007* compared with 2003–2005,† by state or territory

* Notifications where the date of diagnosis was between January 2006 and December 2007.

† Notifications where the date of diagnosis was between January 2003 and December 2005.

Vaccination status

Vaccination status was reported for 169 (38%) of the 445 notifications on the NNDSS in 2006–2007. There were 162 (36% of 445) who were unvaccinated, and 7 (2% of 445) who were partially vaccinated. No notified cases were fully vaccinated, and none of the partially vaccinated cases were <7 years of age. The vaccination status was unknown in 36 (57%) of 63 notified cases who were aged <7 years, and was unknown or missing in 226 (59%) of the 382 notified cases aged ≥7 years in the current reporting period; the vaccination status of 14 (4% of 382) cases was categorised as ‘not applicable’.

Comment

Hepatitis A infection is a worldwide problem associated with low levels of personal hygiene and sanitation.² Australia and other developed countries have low endemicity of hepatitis A infection; cases occur sporadically with epidemic peaks associated with outbreaks.⁹ Hepatitis A notification and hospitalisation rates over the last 5 years have been low with a downward trend. Prior to that, there were substantial declines in notifications and hospitalisations, from the peaks observed during the 1990s which were due to a large outbreak associated with consumption of contaminated oysters in February 1997¹⁰ and large community-wide epidemics, mainly among men who have sex with men (MSM) and injecting drug users (IDUs).^{11,12} The decrease in numbers of notifications and hospitalisations 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.¹³

In recent years, an increasing proportion of cases with hepatitis A infection have reported travel to countries where hepatitis A is endemic.^{14–16} The promotion of vaccination of travellers is the most efficient way to prevent secondary cases and reduce the burden of disease in Australia.¹⁷ Although there are currently limited data on the vaccination status of notified cases, none was fully vaccinated, with most being unvaccinated. Given the high vaccine efficacy reported, it is reasonable to assume that the majority of notified cases with unknown vaccination status were unvaccinated.

Higher risk of hepatitis A infection has been reported for various population groups, including household contacts of a case, MSM, sewage workers, IDUs, those attending child care centres, homeless individuals and Indigenous Australians.^{2,11,13,15,16,18–27} The epidemiology of hepatitis A differs significantly for the Aboriginal and Torres Strait Islander population, in whom it has been endemic. Among non-Indigenous Australians, as in other developed countries, adolescents and young adults have a lower seroprevalence than older adults.¹¹ In contrast, hospitalisation and notification rates are higher among Indigenous Australians, with rates in Indigenous children aged <5 years over 20 times as high as those of non-Indigenous children in the same age group.²⁸ The rate of hospitalisation is likely to be underestimated, due to the known under-identification of Indigenous Australians in the hospitalisation data. This greater disease burden in Indigenous children has been particularly pronounced in more remote areas.^{18,19,29} During 2003–2006, there was 1 death due to hepatitis A in a person aged >50 years identified as Indigenous.²⁸

Prevention of hepatitis A infection is effectively achieved by hepatitis A vaccination.^{30–32} In Australia, vaccination is recommended for selected at-risk groups and occupations.³⁰ In 1999, an immunisation program commenced for Indigenous children aged 18 months living in north Queensland, with catch-up vaccination up to the 6th birthday. Data indicated that this program had a significant impact on reducing hepatitis A across the community.¹⁸ This program was expanded in 2005 to include all Indigenous children aged ≤5 years in the Northern Territory, Queensland, South Australia and Western Australia.³³ In the expanded program, children aged <2 years received the vaccine in the primary program and children aged 2–5 years in the catch-up program which finished in 2007.³⁴ As the hepatitis A vaccination program has been running in north Queensland since 1999, most Indigenous children >2 years of age have now been immunised against hepatitis A in this region. Therefore, children ≤2 years of age in this region are currently provided with the free vaccine. This program appears to have contributed to the declining trend in notifications from these regions in the current reporting period (Figure 3.3.5). Similar trends have been observed in the USA where the number of hepatitis A cases decreased substantially following the staged implementation of vaccination of all infants and children in the country.^{14,35}

More hospitalisations and deaths due to complications of hepatitis A infection have been reported in the elderly compared with younger adults,^{36,37} but the hospitalisation data for people aged ≥60 years in the current report should be interpreted with caution. The majority of hospitalisations in this age group had principal diagnoses 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.³⁸ In contrast, among the hepatitis A hospitalisations in young children aged 5–14 years (95%), there was a high proportion with hepatitis A as the principal diagnosis; this emphasises that hepatitis A infection in young children, although regarded as generally asymptomatic, can require hospitalisation.

Enhanced surveillance (including more complete case information, such as risk factors and vaccination status) is needed to fully evaluate the hepatitis A vaccination program in Australia, to assess both the impact of new vaccination initiatives and the need for expansion of vaccination programs.

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3.4 Hepatitis B

Acute infection with hepatitis B virus (HBV), a hepadnavirus, 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.^{1,2} 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.¹ Of people chronically infected with HBV, without therapeutic intervention 15%–40% develop cirrhosis of the liver and/or hepatocellular carcinoma.^{3,4}

HBV transmission occurs by percutaneous or permucosal exposure to infective body fluids such as blood, semen and vaginal secretions.¹ 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.¹ The summary below is restricted to acute, or newly acquired, hepatitis B. Reviews of the burden of disease related to chronic hepatitis B infection in Australia have been published elsewhere.^{3,5–7}

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

National definition* for newly acquired hepatitis B from January 2004:⁸

Only confirmed cases are notifiable.

- 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 (anti-HBc IgM), 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 health 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 previous reports, hospitalisations were included only where the relevant ICD code was the principal diagnosis.

Deaths

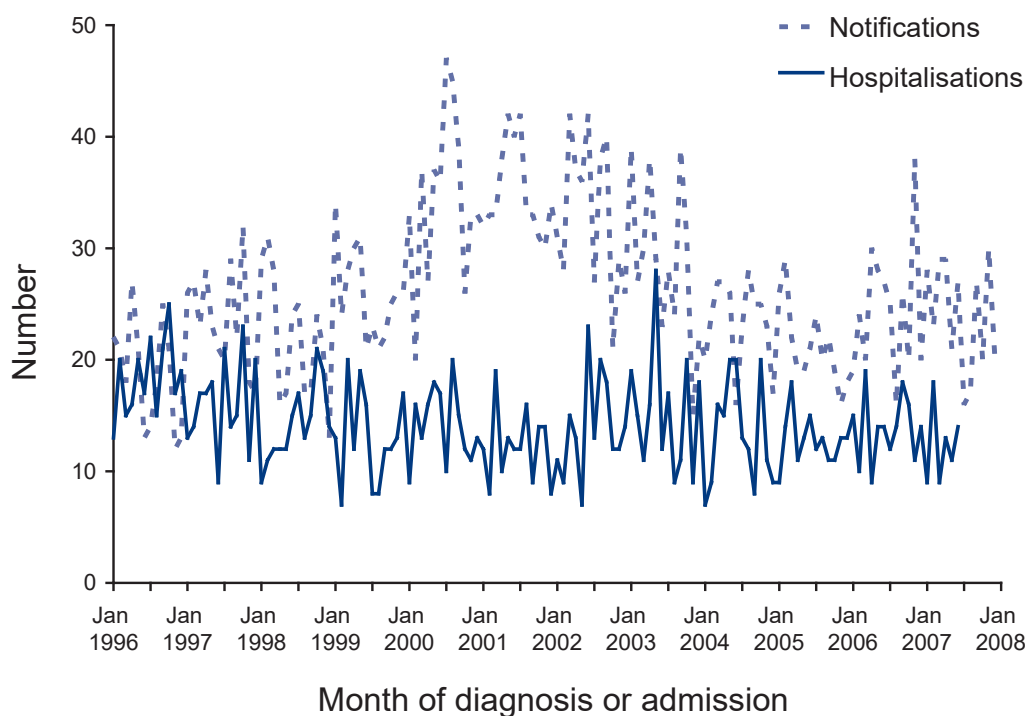
The ICD-10 code B16 (acute hepatitis B) was used to select deaths from acute hepatitis B.

Secular trends

In the 2 years from January 2006 to December 2007, there were 580 notifications (average annual rate 1.4 per 100,000) with a median of 24.5 notifications per month (range 16–38) (Figure 3.4.1; Table 3.4.1). The national annual notification rate had an upward trend between 1997 and 2001, peaked in 2001 at 2.2 per 100,000, before declining to 1.2 per 100,000 in 2005 and 1.4 per 100,000 in 2007 (Appendix 6.2).

In the period from July 2005 to June 2007, there were 315 hospitalisations with a principal diagnosis of acute hepatitis B (average annual rate 0.77 per 100,000) with a median of 13 hospitalisations per month (range 9–19). Nearly all (98%; 310/315) of these hospitalisations were coded as 'acute hepatitis B without delta-agent and without hepatic coma' (ICD-10-AM code B16.9). Hospitalisations have generally declined since 1993, stabilising at a national rate of 0.7–0.8 per 100,000 since 2003 (Appendix 6.3).

Figure 3.4.1: Acute hepatitis B notifications, and hospitalisations with a principal diagnosis of acute hepatitis B, Australia, 1996 to 2007,*† by month of diagnosis or admission



* In contrast to previous reports, this figure only includes data from 1996 onwards as, prior to July 1994, hospitalisations for acute hepatitis B could not be distinguished from hospitalisations for chronic hepatitis B infection, and it was not until 1996 that 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.

† Notifications where the date of diagnosis was between January 1996 and December 2007; hospitalisations where the date of admission was between January 1996 and June 2007.

Table 3.4.1: Acute hepatitis B notifications, hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Notifications 2 years (2006–2007)		Hospitalisations† 2 years (July 2005–June 2007)		LOS‡ per admission (days) Median	Deaths 2 years (2005–2006)	
	n	Rate§	n	Rate§		n	Rate§
0–4	5	0.19	0	–	–	0	–
5–14	8	0.15	2	0.04	n.p.	0	–
15–24	120	2.06	43	0.75	4.0	0	–
25–59	419	2.07	248	1.24	4.0	21	0.10
60+	28	0.37	22	0.30	8.0	18	0.25
All ages	580	1.39	315	0.77	4.0	39	0.09

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† 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.

n.p. Not published.

Severe morbidity and mortality

For patients with a principal diagnosis of acute hepatitis B, 1,843 hospital bed days were recorded over the 2-year reporting period between July 2005 and June 2007 (916 in 2005/2006 and 927 in 2006/2007). The median length of stay was 4 days, with longer stays for adults aged ≥ 60 years (Table 3.4.1). There was only one case of hepatic coma, aged 25–59 years, recorded in 2006/2007, among hospitalisations with a principal diagnosis of acute hepatitis B.

There were 39 deaths from acute hepatitis B recorded in the AIHW National Mortality Database in the 2 years between January 2005 and December 2006, 30 in males and 9 in females. All deaths occurred in those aged ≥ 25 years. Approximately half occurred in those aged 25–59 years and half in those aged ≥ 60 years. The number of deaths in 2006 was almost double that in 2005 (25 vs 14 by date of recording, and 23 vs 16 by actual date of death). The underlying cause of death in 2 of these 39 cases was acute hepatitis B (without delta agent) with hepatic coma (ICD-10 code B16.2); both these deaths were recorded in 2006. The remaining 37 cases were without hepatic coma (ICD-10 code B16.9). In contrast, only 4 deaths in notified cases were recorded during 2005 and 2006 and none in 2007.

Age and sex distribution

In 2006–2007, the peak acute hepatitis B notification rate was in the 25–29 years age group (average annual rate 3.7 per 100,000), followed by the 20–24 years age group (average annual rate 2.8 per 100,000) (Figure 3.4.2). Historically, notification rates have consistently been highest in older adolescents and young adults aged 15–29 years. Between 2001 and 2005, notification rates declined in this age group, particularly among persons 15–19 years of age, where, for the first time in 2005, rates fell below 1 per 100,000. Since 2005, there has been a continued decline in the rates among persons 20–24 years of age, but not among persons 15–19 and 25–29 years of age, with notification rates of 1.2 per 100,000 and 3.8 per 100,000, respectively, in 2007. Rates have remained fairly stable in the other age groups. As in previous years, there were more male than female notifications in almost all age groups in 2006 and 2007, with an overall male:female ratio of 1.7:1.

As in previous years, during the period July 2005 to June 2007, rates for hospitalisations with a principal diagnosis of acute hepatitis B varied between males and females (Figure 3.4.3). Males had almost double the hospitalisation rate of females (an overall male:female ratio of 1.9:1). Among males, hospitalisation rates were highest in the 25–29 years age group (2.1 per 100,000). Among females, the rates were highest in the 35–39 years age group (1.1 per 100,000). Among persons 15–24 years of age there was little gender disparity in hospitalisation rates.

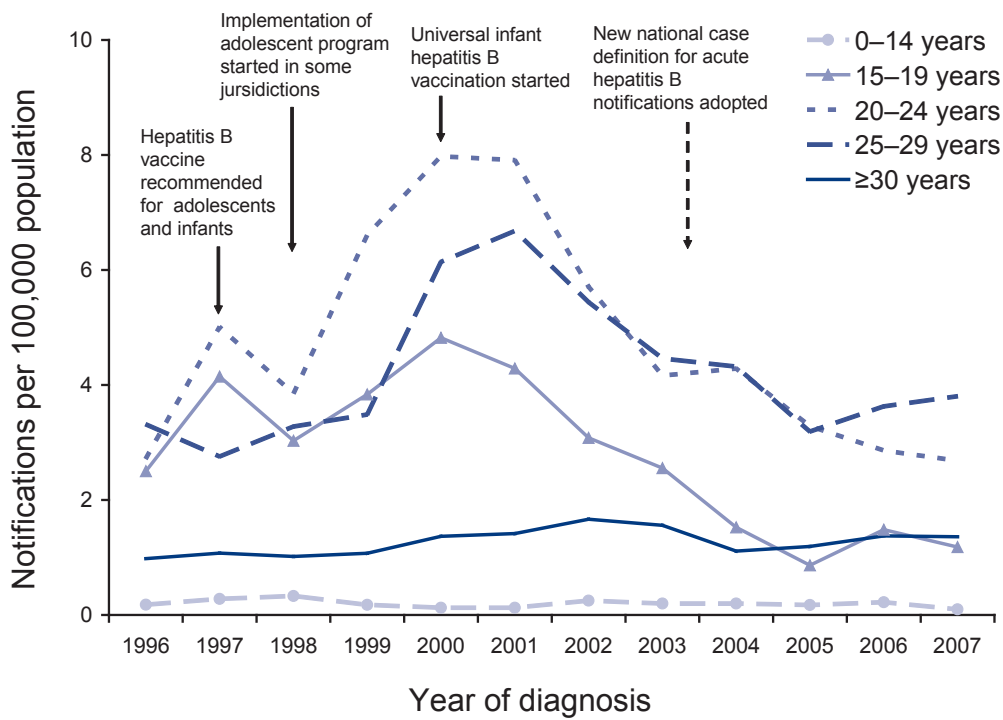
Geographical distribution

During the period January 2006 to December 2007, Victoria recorded the highest number of notifications ($n=191$; 33% of total notifications), followed by Queensland ($n=114$; 20% of total notifications), with the Northern Territory having the highest average annual notification rate at 4.5 per 100,000. The Australian Capital Territory and Western Australia had the next highest notification rates (2.8 and 2.2 per 100,000, respectively), while rates were 1.8 per 100,000 or less in the other jurisdictions (Appendix 6.2). Rates in the Australian Capital Territory doubled from 1.8 per 100,000 ($n=6$ cases) in 2006 to 3.8 per 100,000 ($n=13$ cases) in 2007. For the years 2005/2006 to 2006/2007, the Northern Territory had the highest annual average hospitalisation rate for acute hepatitis B (1.4 per 100,000), followed by Victoria (1.0 per 100,000) (Appendix 6.3).

Vaccination status

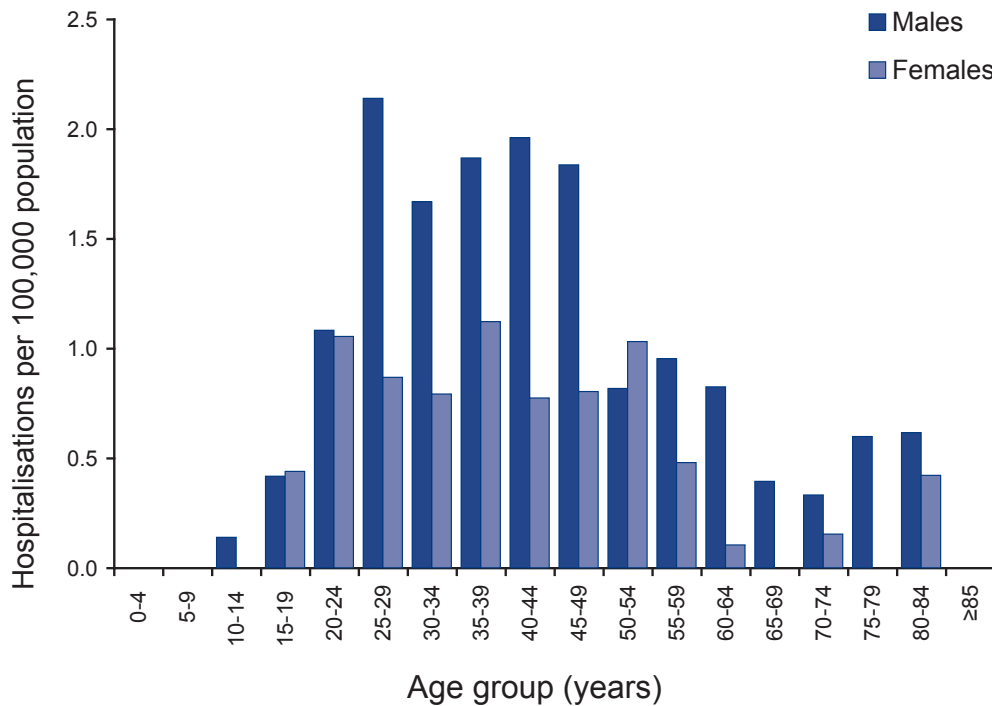
Universal infant hepatitis B immunisation was introduced in Australia in May 2000, and high school programs of hepatitis B immunisation for older children and adolescents were introduced at varying times in different jurisdictions. Over the review period, there were 14 cases of acute hepatitis B in children born since 1 May 2000, with 5 cases notified in 2006–2007. One, which was notified in 2005, was recorded as Indigenous. One of the 5 cases notified in 2006–2007 had received 4 doses of vaccine (validated); the vaccination status was recorded as “unknown” or “missing” in the remaining 13 of these 14 cases on NNDSS.

Figure 3.4.2: Acute hepatitis B notification rates, Australia, 1996 to 2007,* by age group and year of diagnosis



* Notifications where the date of diagnosis was between January 1996 and December 2007.

Figure 3.4.3: Acute hepatitis B hospitalisation rates, Australia, 2005/2006 to 2006/2007,* by age group and sex



* Hospitalisations where the principal diagnosis was acute hepatitis B and the date of separation was between July 2005 and June 2007.

Comment

Since 2004, only confirmed cases of newly acquired hepatitis B have been notified. This change in requiring the absence of prior evidence of hepatitis B infection in a confirmed case could have partially contributed to less notifications since 2004; however, the declining trend of notifications started before this change.⁹ 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 years age group. The decline in notifications in those aged 15–29 years since 2001 is consistent with the marked reduction in injecting drug use since the heroin drought and estimated declines in hepatitis C incidence.¹⁰ The rapid decline in notifications from 2003 to 2005 seems to have plateaued in 2006 and 2007. The slight increase in notifications in this reporting period may partly be due to more complete case follow-up; as a result, cases that were previously reported as unspecified hepatitis B were able to be confirmed as newly acquired.¹¹ Several reports note high rates of chronic hepatitis B carriage in immigrants and refugees from both Asia–Pacific countries and sub-Saharan Africa.^{12,13} However, notification data from Victoria since 2006 report that approximately 70%–80% of newly acquired notifications were from those born in Australia, and only a few cases were notified in Aboriginal and Torres Strait Islander people.^{14–18}

The source of exposure for hepatitis B in 2006 was reported in South Australia, Victoria and the Australian Capital Territory. The proportion of incident hepatitis B notifications associated with injecting drug use has not increased, remaining stable at approximately 51% from 2002 to 2006.¹¹ Injecting drug use was the main risk factor identified in notification data from Victoria in 2006–2007.^{14–18}

The first Australian children received hepatitis B vaccines as infants in the late 1980s. Adolescent hepatitis B catch-up immunisation programs for children aged 10–13 years were introduced from 1997 and implemented at different times by jurisdictions (New South Wales 1999, the Northern Territory 1998 [catch-up program only], Tasmania 1998, Victoria 1998, South Australia 1999, Western Australia 2002, and the Australian Capital Territory 1999).¹⁹ This program may be responsible for the continuing low rates in those aged <25 years. In a national serosurvey in 2002, the prevalence of hepatitis B surface antibody detection among those aged 12–17 years was 45.5%, nearly 2-fold higher than the prevalence seen in the serosurvey of 1996–1999 (28.5%). Prevalence of hepatitis B surface antibody detection was significantly higher in those states and territories that had implemented school-based programs.²⁰ This cohort was aged 17–22 years in 2006–2007 and their vaccine-induced immunity to hepatitis B is likely to account for some of the recent decrease in notifications in adolescents.

The effect of the universal infant hepatitis B immunisation policy on the reported incidence of acute hepatitis B in the 15–25 years age group will start to become apparent from 2015 onwards (which is 15 years after the universal program started). In countries with a high burden of hepatitis B, such as Taiwan and Italy, universal hepatitis B vaccination programs have had a profound impact on the incidence of chronic infection and hepatocellular carcinoma.^{21,22} Fourteen cases of hepatitis B were notified to NNDSS for children born since 1 May 2000, with 5 cases in 2006–2007, and nearly all (93%; 13/14) had vaccination status recorded as “unknown”. Reporting of cases according to age and vaccination status will become more important as the vaccinated cohort ages, and will assist in measuring the impact of vaccination and the duration of immunity. Data on the duration of immunity following infant vaccination in low endemic countries is sparse.

Notification rates were substantially higher than hospitalisation rates in all age groups in 2006–2007. The 39 deaths recorded in 2005 and 2006, all aged ≥ 25 years, increased from an annual average of approximately 10 since 2001. Misclassification remains a potential problem, as only 2 of the 39 deaths recorded for 2005 and 2006 had acute hepatitis B with hepatic coma (ICD-10 codes B16.0 [with delta agent] or B16.2 [without delta agent]) recorded as the underlying cause of death. This is probably due to misclassification of some chronic hepatitis B cases, particularly those with hepatic flare who had detectable anti-hepatitis B core immunoglobulin M antibodies (anti-HBc IgM), as acute hepatitis B infection. The mortality rate in those aged 25–59 years has more than doubled from 0.04 per 100,000 in 2003–2004⁹ to 0.10 per 100,000 in 2005–2006, notwithstanding limitations of the data and possible misclassifications of chronic HBV deaths.

The current prevalence of chronic HBV infection reflects historical transmission patterns and possibly the recent arrival of immigrants from high endemic countries. Data from Victoria on the country of birth of persons with newly acquired hepatitis B infection suggests approximately 20% of cases were born outside Australia. Over the next decade the full impact of immunisation policies implemented from the 1990s should be reflected in trends in chronic infection and reductions in hepatitis B related complications, such as liver cirrhosis and hepatocellular carcinoma.^{5,6,23}

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3.5 Influenza

Influenza virus causes annual epidemics of respiratory disease, mainly spread by droplet transmission. The disease is often indistinguishable clinically from that caused by other respiratory viruses. Asymptomatic influenza infection is well documented in serologic studies. Typical symptomatic cases have abrupt onset of fever and cough (most common), but malaise, myalgia, sore throat and headache are also prominent. Complications of influenza infection include pneumonia, otitis media and exacerbation of chronic medical conditions.^{1,2} In areas with temperate climate, influenza epidemics usually occur during the winter months, causing an increase in hospitalisations for pneumonia, exacerbation of chronic diseases and contributing to increased mortality, particularly among the elderly and people with high-risk underlying conditions. In areas with tropical climate, the seasonality patterns are more variable.^{3,4}

There are three types of influenza viruses that infect humans, types A, B and C, of which types A and B are clinically important. Type A influenza viruses can be subtyped according to the antigenic differences of two surface glycoproteins, haemagglutinin (e.g. H1, H2, etc) and neuraminidase (e.g. N1, N2, etc).¹ Both influenza A and influenza B viruses undergo frequent changes in their surface antigens, thus evading natural immunity acquired from previous infections. Cumulative stepwise genetic changes (point mutations) of the virus occur regularly and these mutations may result in antigenic variation, known as antigenic drift, which is responsible for seasonal outbreaks and smaller epidemics of influenza. Pandemics of influenza are caused by strains that have undergone dramatic or major genetic changes (called antigenic shift when a novel haemagglutinin is introduced), which might include reassortment (mixing) with influenza viruses that primarily infect other animal species.¹

Case definitions

Notifications

Laboratory-confirmed influenza was a nationally notifiable disease in all jurisdictions except South Australia during this reporting period. Although in South Australia influenza was not a notifiable disease, laboratory reports were collected and sent to NNDSS, and laboratory-confirmed influenza became notifiable in May 2008. Implementation of influenza notification occurred nationally in all other jurisdictions during 2001, except Tasmania (2002).

A laboratory-confirmed influenza infection is one in which an influenza virus is identified by cell culture, nucleic acid amplification testing, or influenza antigen testing, from appropriate respiratory tract specimens; OR a recent infection demonstrated by serological methods including IgG seroconversion (a significant [≥ 4 -fold] increase in antibody level) or a single high (IgG) titre to the influenza virus.

(The criteria for the national case definitions were revised and implemented in 2008, after the period covered by this report. The revisions consisted of specifying that the influenza virus antigen detection has to be performed by a laboratory, and that the high titre qualifying for notification has to be performed by complement fixation test [CFT] or by haemagglutination inhibition [HAI]).⁵

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 2006 to December 2007, there were 13,655 notifications of laboratory-confirmed influenza, giving an average annual rate of 32.7 per 100,000 (Table 3.5.1). Notifications varied considerably between the 2 years, with 3,252 in 2006 (annual rate 15.7 per 100,000) but 10,403 in 2007 (annual rate 49.5 per 100,000). Nationally, there was a clear seasonal distribution of notifications with peaks in August in both years, as in most previous years since 2001. The median number of notifications per month was 626 (range 244–1,327) in 2005, 542 (range 204–1,058) in 2006, but 1,238 (range 407–5,094) in 2007 during the months from June to October for each of these 3 years.

Between July 2005 and June 2007, there were 4,444 hospitalisations coded as influenza (an average annual rate of 10.8 per 100,000) (Table 3.5.1), with a higher number of hospitalisations (2,593) recorded in 2005/2006. There was a clear seasonal pattern nationally, with obvious increases over the winter months (Figure 3.5.1). Hospitalisations peaked in August in both 2005 and 2006. The median number of admissions per month was 92.5 (range 44–689) over the 2 years. During the months from June to October for these 2 years, the median number of admissions per month was 317.5 (range 130–689), with a peak of 689 in August 2005. Data on hospitalisation rates for the winter season in 2007, during which notification rates were higher than previous years since influenza became nationally notifiable, were not available at the time of preparing this report. The median duration of a seasonal epidemic (when the fortnightly hospitalisation count exceeded the mean count of the inter-epidemic period by ≥ 2 standard deviations) was 10 fortnights (20 weeks) in the years 1999–2006 (range 9–12 fortnights), based on virologically confirmed influenza hospitalisations.

Table 3.5.1: Influenza notifications, hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Notifications 2 years (2006–2007)		Hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission (days)		Deaths 2 years (2005–2006)	
	n	Rate [‡]	n	(§)	Rate [‡]	(§)	Median	(§)	n	Rate [‡]
0–4	2,904	110.0	1,241	(914)	47.8	(35.2)	2.0	(2.0)	5	0.19
5–14	1,971	35.9	383	(282)	7.0	(5.1)	2.0	(2.0)	2	0.04
15–24	1,751	30.1	494	(332)	8.7	(5.8)	1.0	(1.0)	1	0.02
25–59	4,869	24.0	1,354	(898)	6.7	(4.5)	2.0	(1.0)	7	0.03
60+	2,159	28.8	972	(534)	13.4	(7.4)	6.0	(5.0)	40	0.55
All ages	13,655	32.7	4,444	(2,960)	10.8	(7.2)	2.0	(2.0)	55	0.13

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

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

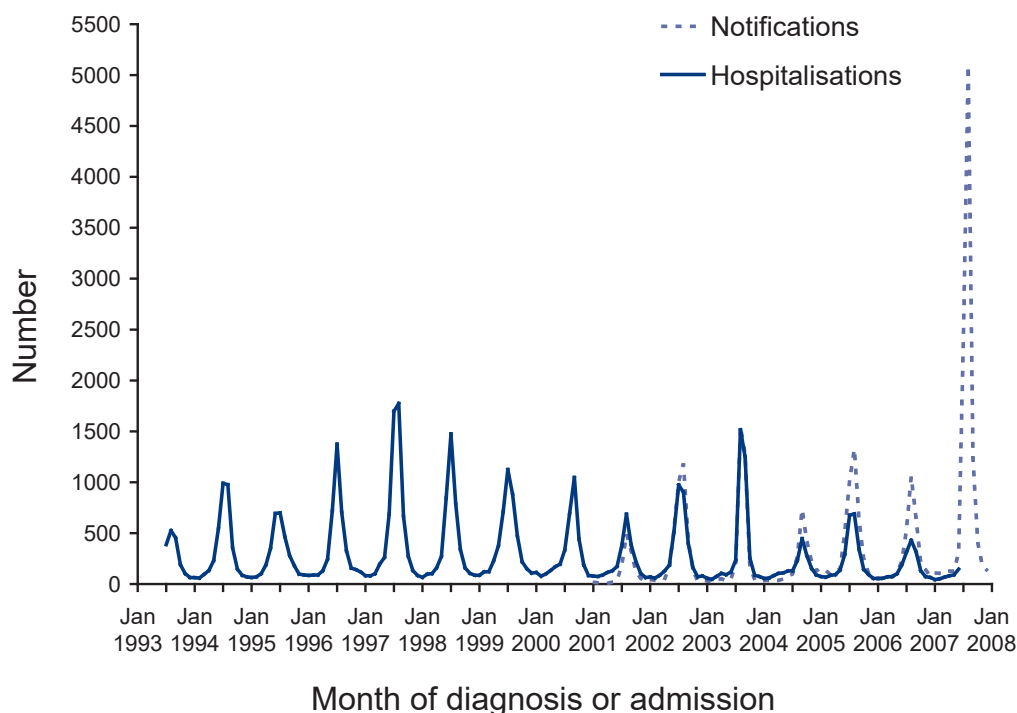
§ Principal diagnosis (hospitalisations).

|| Includes 1 case with unknown age.

Severe morbidity and mortality

A total of 30,823 hospital bed days with an ICD-10-AM code for influenza were recorded over the reporting period from July 2005 to June 2007. The median length of stay was much longer in people aged ≥ 60 years (6 days) compared with younger age groups (1 or 2 days) (Table 3.5.1). Influenza was the principal diagnosis for 67% of the hospitalisations where influenza was one of the separation diagnoses. There were substantially more bed days due to influenza in 2005/2006 compared with 2006/2007 (18,199 vs 12,624). Over these 2 reporting years, hospitalisations where the virus was identified (ICD-10-AM code J10) contributed to 44.5% (1,979/4,444) of all influenza hospitalisations, but 55.3% (17,043/30,823) of hospital bed days. The median length of stay was longer for hospitalisations where the virus was identified compared with those where the virus was not identified (ICD-10-AM code J11) overall (3 days vs 1 day), and for all age groups including those with longer length of stay (3.0 vs 1.5 days for those aged 0–4 years, 4 vs 1 days for those aged 25–59 years, and 9 vs 4 days for those aged ≥ 60 years).

From January 2005 to December 2006, influenza was recorded as the underlying cause of death in 55 cases in the AIHW National Mortality Database. Of these, 40 (73%) were aged ≥ 60 years (rate 0.55 per 100,000), and 5 (9%) were aged 0–4 years (rate 0.19 per 100,000) (Table 3.5.1). Three of the 5 deaths (60%) in children aged < 5 years were coded as death due to influenza with virus identified (ICD-10 code J10), whereas only 8 of the 50 deaths (16%) occurring at other ages were coded as death due to influenza with virus identified. Although there is no requirement for clinical information to be entered on NNDS for influenza cases (and whether death

Figure 3.5.1: Influenza hospitalisations and notifications,* Australia, 1993 to 2007, by month of diagnosis or admission

* Notifications where the month of diagnosis was between January 2001 and December 2007; hospitalisations where the month of admission was between July 1993 and June 2007. Note that the Northern Territory, Queensland, Tasmania and Victoria did not notify influenza for the complete year in 2001.

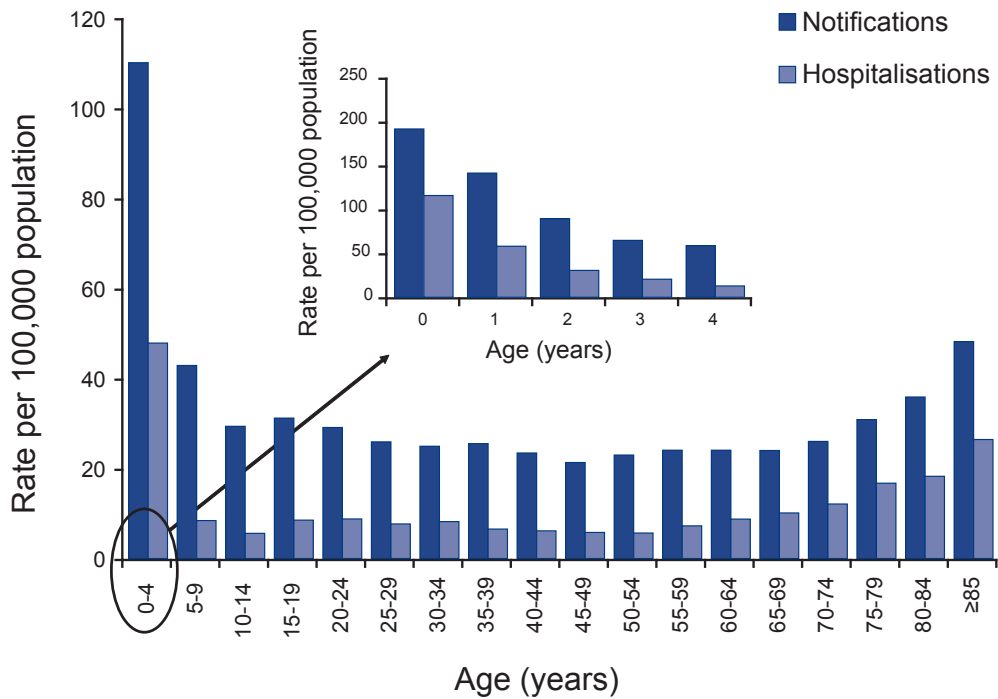
was the outcome was not recorded in 69% of notifications in this reporting period), 22 deaths were recorded on NNDSS among influenza notifications in 2006–2007 (5 deaths in 2006, 17 deaths in 2007); these included 1 death in 2006 and 5 deaths during 2007 in children aged <5 years, 3 of which were in Western Australia.

Age and sex distribution

Similar to previous years, during the period January 2006 to December 2007, the notification rate of laboratory-confirmed influenza was substantially higher among children aged <5 years (110.0 per 100,000) than in other age groups (Table 3.5.1, Figure 3.5.2 and Figure 3.5.3). Among those aged <5 years, the highest rate of notifications was in those <1 year of age, and the rate declined progressively with every year of increase in age. There were year-to-year variations in notification rates from 2002 to 2007, but the trends were similar for all age groups. The substantial increase in notifications during 2007 was not restricted to any particular age group but occurred across the age spectrum (Figure 3.5.3). Although the absolute increase in notification rates was most marked in the 0–4 years age group in 2007, the rate ratio (3.3), compared with the 2006 rates, was similar to other age groups <50 years, decreasing to 2.7 for those aged 50–64 years and 2.4 for those aged ≥65 years.

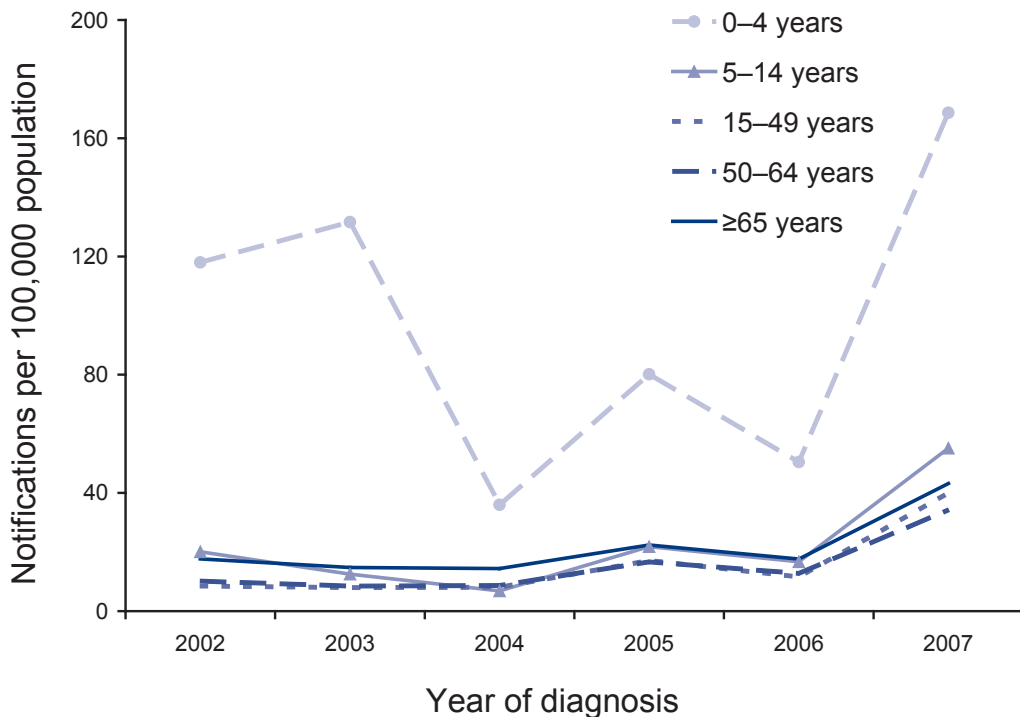
From July 2005 to June 2007, the hospitalisation rate was also highest in children aged <5 years (47.8 per 100,000), and was substantially higher than that of other age groups (Table 3.5.1, Figure 3.5.2). Hospitalisation rates were progressively higher with increasing age among people aged ≥50 years, ranging from 5.7 per 100,000 for those aged 50–54 years to 26.4 per 100,000 for those aged ≥85 years (Figure 3.5.2). Among children aged <5 years, the hospitalisation rate was highest among infants (annual average of 115.9 per 100,000 population aged <1 year, ranging from 95.9 per 100,000 in 2006/2007 to 136.7 per 100,000 in 2005/2006), and progressively lower with increasing age (Figure 3.5.2). The proportion of hospitalisations for which the virus was identified (ICD-10-AM code J10) progressively decreased with increasing age group from 0–4 years (84%) to 25–29 years (13%). The proportion of hospitalisations for which the virus was identified was 23% and 34%, respectively, for those aged 30–59 years and ≥60 years.

Figure 3.5.2: Influenza notification rates, Australia, 2006 to 2007 and hospitalisation rates 2005/2006 to 2006/2007,* by age group



* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007.

Figure 3.5.3: Annual influenza notification rates, Australia, 2002 to 2007,* by age group and year of diagnosis



* Notifications where the date of diagnosis was between January 2002 and December 2007.

Notification rates were higher among males than females at both ends of the age spectrum (those aged <15 years and ≥70 years), but the overall male to female ratio was close to unity (1:1.03). A similar pattern in the male to female ratio was also observed for hospitalisation rates (higher among males compared with females in those aged <15 years and ≥60 years), with the overall male to female ratio also close to unity (1:1.02).

Geographical distribution

There was a wide variation by jurisdiction in the average notification rate in the 2-year review period of 2006–2007, ranging from 11.7 per 100,000 in South Australia (where influenza only became officially notifiable in 2008) to 18.5 per 100,000 in New South Wales to 76.6 per 100,000 in Queensland (see also Appendix 6.2). Crude average hospitalisation rates were similarly varied, with the highest rate reported in Western Australia (16.2 per 100,000 population, n=660) and the lowest in the Australian Capital Territory (4.4 per 100,000, n=29) for the 2-year review period of July 2005–June 2007 (see also Appendix 6.3). The variation among jurisdictions was greater in notification rates than in hospitalisation rates (rate ratio 6.5 for notification rates and 3.7 for hospitalisation rates between the jurisdictions with the highest and the lowest rates).

For all jurisdictions, notification rates were higher in 2007 compared with 2006, and the hospitalisation rates higher in 2005/2006 than 2006/2007.

Influenza virus types and subtypes

The great majority of the influenza notifications reported to the NNDSS recorded the influenza type (13,095/13,665, 95.8%). Of those where the influenza type was known, most (85.9%) were type A (n=11,245), 13.5% (n=1,764) were type B, and both A and B were documented in 0.7% of notifications (n=86). Subtype or strain information of influenza type A was recorded in 1,218 notifications (8.9% of all notifications, 10.8% of type A influenza). Of these notifications where subtype was known, 30.6% were of subtype A/H1 and 69.4% were of subtype A/H3. Queensland and Victoria reported most of the notifications where subtyping was documented. More comprehensive Australian data from the WHO Collaborating Centre for Reference and Research on Influenza on the influenza subtypes for the respective years of this review period have been published in the annual reports of the National Influenza Surveillance Scheme (see *Comment* section below).^{6,7}

Comment

The seasonal patterns seen in both the notifications of laboratory-confirmed influenza (since 2001) and the hospitalisations with influenza virus identified (ICD-10-AM code J10) were similar, with trends of annual variation corresponding to each other. Notification and hospitalisation rates are substantially higher in children aged <5 years, and in those aged <1 year in particular, compared with all other age groups, even people aged ≥85 years. This is likely to reflect the patterns of health care use, and, in particular, diagnostic testing for respiratory viruses, which is much more common in the young age group.

It should be noted that, although notification data are a specific, but not sensitive, indicator of disease incidence, and hospitalisation data are an indicator of more severe cases, neither of these data sources is adequate for assessment of the true population burden due to influenza. The likelihood of diagnosing influenza by laboratory tests in a patient with respiratory or other symptoms compatible with influenza varies with the clinical threshold for obtaining a diagnostic specimen, the quality of the specimen, and other context factors. In addition, the role of influenza in exacerbating chronic disease in the elderly is minimally reflected in these surveillance data, especially in notifications data and data on the underlying cause of death. In a retrospective population-based study conducted in Sydney, excess hospitalisation rates attributable to influenza in children aged <18 years were estimated to be 2–11 times higher than the hospitalisation rate calculated from hospitalisations where influenza was coded as the principal discharge diagnosis, depending on the chosen estimation method and the age group.⁸ A more recent prospective cohort study conducted in a paediatric hospital in Sydney found that only 43% of the principal discharge diagnoses of hospitalisations of children aged <5 years with laboratory-confirmed influenza were coded as influenza; other diagnoses included lower respiratory tract infection, pneumonia and febrile convulsion.⁹ Deaths and hospitalisations coded as due to influenza are widely acknowledged to substantially underestimate the true number of deaths and hospitalisations attributable to influenza.^{10–13}

There was a marked increase in notifications of laboratory-confirmed influenza in 2007. This is likely to be a result of significant antigenic drift of the circulating strains of the influenza virus away from those that predominated in the previous few years and vaccine strains, against which a greater level of immunity would be expected in the population. While the majority of the types of influenza virus in the notified cases are reported, available information on subtypes is limited. Instead, additional information on antigenic drift is available from an alternate source, the WHO Collaborating Centre for Reference and Research on Influenza in Melbourne, Victoria.

Analysis by this Centre of 657 viable viral isolates or clinical specimens from Australian laboratories in 2006 identified 402 (61.2%) to be A(H3N2) strains, 24 (3.6%) to be A(H1N1) strains, and 231 (35.2%) to be influenza B strains. Those 2006 A(H3) strains were mostly antigenically similar to the 2006 vaccine strain A/New York/55/2004. Among the small number of A(H1) strain isolates, there was a drift away from the 2006 vaccine strain A/New Caledonia/20/99. The great majority (93.5%) of the 231 influenza B viruses analysed were antigenically related to the 2006 vaccine strain B/Malaysia/2506/2004 (B/Victoria/2/87-lineage viruses).⁶

However, in 2007, many more influenza virus isolates or specimens were received by the WHO Influenza Centre from Australian laboratories, and the proportional distribution of the influenza subtypes differed substantially from that of 2006. Of the 1,406 viable isolates, 826 (58.7%) were identified as A(H3N2) strains, 483 (34.4%) as A(H1N1) strains, and 97 (6.9%) as influenza B strains. The 2007 Australian A(H3N2) viruses were antigenically similar to either the 2007 vaccine strain A/Wisconsin/67/2005 (which was different from the dominant A/H3 strain in 2006) or the newly emergent variant A/Brisbane/10/2007. There was also significant antigenic drift in A(H1N1) viruses away from the 2007 vaccine strain A/New Caledonia/20/99. Among the relatively small number of influenza B viruses isolated in 2007, only 21% were antigenically related to the 2007 vaccine strain B/Malaysia/2506/2004 (B/Victoria/2/87-lineage viruses), while the remaining 79% were closely related to emergent B/Florida/7/2004-like viruses (B/Yamagata-lineage).⁷

Moreover, although A(H3N2) viruses constituted the majority (58.7%) of the Australian isolates analysed in 2007, co-circulating A(H1N1) viruses constituted a substantial proportion (34.4%). This was the highest annual proportion of A(H1N1) viruses found in Australia since 2001, when A(H1N1) was the predominant strain. The extensive co-circulation of two influenza A strains might have contributed to the higher incidence of influenza in 2007.⁷

The increase in the number of reported deaths among notified cases in 2007, including 5 deaths in children aged <5 years, is also consistent with an overall true increase in disease incidence. Although the reporting of death as an outcome of laboratory-confirmed influenza in the notification data is incomplete, it is likely that a notified case that died of influenza would be captured, especially in young children. The Australian Government Department of Health and Ageing, collating information from multiple surveillance sources, identified a total of 7 deaths caused by influenza in Australian children aged <5 years in 2007.⁷ Surveillance based on influenza-like illness syndrome and absenteeism revealed a higher case incidence compared with previous years.⁷

The increase in notifications in 2007 is also likely to be partly related to increased diagnostic testing, possibly prompted by influenza deaths in young children being widely reported in the media.¹⁴ However, the increase in notifications was not restricted to young children but occurred in all age groups. At the time of preparing this report, the corresponding hospitalisation and death data for the winter season in 2007 were not yet available.

Apparent differences in notification and hospitalisation rates between jurisdictions should be assessed with caution as they may primarily reflect differences in likelihood and availability of virological testing or in coding practices. Hospitalisation data referred to in this report are based on discharge coding and it is possible that some of those hospitalisations coded with the less specific set of codes for influenza without virological confirmation (i.e. ICD-10-AM J11 codes) may be due to other respiratory pathogens such as respiratory syncytial virus (RSV), parainfluenza virus, picornavirus, adenovirus or coronavirus.¹⁵⁻¹⁷

Annual seasonal influenza vaccination is currently recommended for all Australians aged ≥ 65 years, all Aboriginal and Torres Strait Islander people aged ≥ 15 years, and all individuals aged ≥ 6 months who are predisposed to severe influenza or its complications, for example, those with chronic medical conditions such as chronic pulmonary or cardiovascular disease, pregnant women, residents of nursing homes and other long-term care facilities, and homeless people and those providing care to them.¹⁸ From 1999, publicly funded seasonal influenza vaccine has been available for Australians aged ≥ 65 years, and for Aboriginal and Torres Strait Islander people who are aged ≥ 50 years or who have chronic medical conditions that predispose them to severe influenza. From January 2010, eligibility for free vaccine, has been extended to include all Aboriginal and Torres Strait Islander

people aged 15–49 years, all individuals aged ≥ 6 months who have a medical condition that predisposes them to severe influenza, and pregnant women.¹⁹ In Western Australia influenza vaccine has been available free for all children aged 6 months to 5 years since 2008.²⁰ The latest published estimations of influenza vaccination uptake, from the Adult Vaccination Survey conducted by the AIHW, were 79.1% in Australians aged ≥ 65 years for 2004,²¹ and 77.5% for 2006, which were not significantly different statistically.⁶

There have been progressively widening indications for influenza immunisation in children in the USA, where the Advisory Committee on Immunization Practices (ACIP) in 2003 recommended routine annual influenza vaccination of healthy American children aged 6–23 months^{22,23} based on the high burden of illness.^{12,24,25} In 2006, the ACIP extended its recommendation to include children up to the age of 5 years,²⁶ and, more recently, to include all children to the age of 18 years commencing 2008/2009.²⁷ Australian data in this report, as well as those published in previous studies, suggest a similarly significant burden of illness of more severe influenza in young children.^{8,28} The Australian Technical Advisory Group on Immunisation has been deliberating on influenza vaccination for children aged from 6 months up to 5 years.²⁹ In addition to disease burden, other aspects, such as vaccine efficacy and safety for recipients of this age group, the feasibility of implementation, and community acceptability of a universal influenza vaccination program for healthy children, need to be considered.^{30,31}

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3.6 Measles

Measles is an acute and highly communicable disease caused by a morbillivirus. The clinical picture includes a prodromal fever followed by a generalised maculopapular rash that often begins on the face. There are also frequently Koplik spots on the buccal mucosa, conjunctivitis, coryza and cough. Complications include otitis media, pneumonia and encephalitis. Subacute sclerosing panencephalitis (SSPE) occurs very rarely as a late sequel of infection by the wild-type virus but not following vaccination.¹ Complications and deaths are rare, but occur more commonly in developing countries, in children aged <5 years and adults, and in persons with malnourishment or immune deficiencies.²

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

National definition from January 2004:³

Both confirmed and probable cases are notifiable.

- 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 immunoglobulin (Ig) G seroconversion or significant increase in IgG antibody level or a 4-fold 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 8 days to 8 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 receipt of measles-containing vaccine 8 days to 8 weeks prior to testing (confirmed case); *or*
- f) A clinical illness characterised by a generalised maculopapular rash lasting at least 3 days, with 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 characterised by a generalised maculopapular rash lasting at least 3 days, with fever of at least 38°C at the time of rash onset and either cough, coryza, conjunctivitis or Koplik spots, together with the detection of measles-specific IgM antibody by a laboratory which is not an approved reference laboratory, except if the case has received a measles-containing vaccine 8 days to 8 weeks prior to testing (probable case).

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code B05 (measles) was used to identify hospitalisations and deaths. SSPE, a very rare late sequel of measles infection, was not included in these analyses.

Secular trends

In the 2-year review period of 2006 and 2007, there were 137 notified cases of measles, an average annual notification rate of 0.33 per 100,000 population (Table 3.6.1). Of these 137 cases, 128 (93%) were confirmed cases, and only 9 (7%) probable cases (all notified in 2006). The number of measles notifications in 2006 (n=125) was high compared to both the previous year (n=10) and to 2007 (n=12). The large increase in 2006 was driven by a multi-jurisdictional measles outbreak in March to May of that year, which is described further below (see *Geographical distribution*). The median number of notifications per month in 2006 was 2 (range 0–59) and in 2007 was 1 (range 0–3).

In the period from July 2005 to June 2007, there were 56 hospitalisations with the ICD-10-AM code B05 (measles). This equates to an average annual hospitalisation rate of 0.14 per 100,000 population. Since a decline in the mid-1990s, annual hospitalisation rates have been at a fluctuating low level, and, in 2006/2007, was the lowest on record at 17 separations, a rate of 0.08 per 100,000. In 2006/2007, the 5-year age groups with the highest rates of hospitalisations were the 0–4 and 25–29 years age groups (0.38 and 0.43 per 100,000, respectively). As with notifications, the peak in the number of hospitalisations per month occurred during the autumn months of March (n=8), April (n=16) and May (n=6) 2006 (Figure 3.6.1). The median number of hospitalisations per month was 1 (range 0–16) in the period July 2005 to June 2007.

Table 3.6.1: Measles notifications, hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Notifications 2 years (2006–2007)		Hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission (days)		Deaths 2 years (2005–2006)	
	n	Rate‡	n	(§)	Rate‡	(§)	Median	(§)	n	Rate‡
0–4	33	1.25	16	(14)	0.62	(0.54)	1.0	(1.0)	0	–
5–14	40	0.73	5	(5)	0.09	(0.09)	2.0	(2.0)	0	–
15–24	23	0.40	10	(10)	0.18	(0.18)	2.0	(2.0)	0	–
25–59	40	0.20	25	(23)	0.12	(0.11)	2.0	(2.0)	0	–
60+	1	0.01	0	(0)	–	–	–	(–)	0	–
All ages	137	0.33	56	(52)	0.14	(0.13)	2.0	(2.0)	0	–

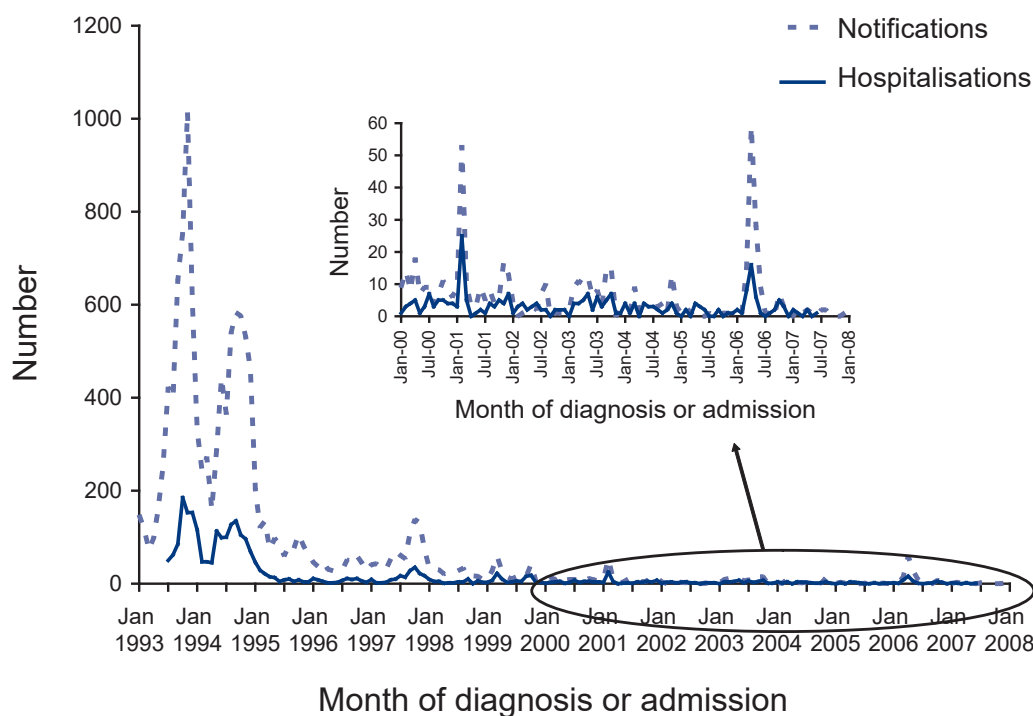
* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

Figure 3.6.1: Measles notifications and hospitalisations, Australia, 1993 to 2007,* by month of diagnosis or admission



* Notifications where the date of diagnosis was between January 1993 and December 2007; hospitalisations where the date of admission was between July 1993 and June 2007.

Severe morbidity and mortality

In the 2-year period from July 2005 to June 2007, hospital separations for measles accounted for 138 hospital bed days. The median length of stay was 2 days, with little variation across the age groups (Table 3.6.1). Of the 56 hospitalisations, 52 (93%) had measles recorded as the principal diagnosis. Complications arising from measles infection were recorded for 9 (16%) separations, of which 5 were coded as having measles complicated

by pneumonia and 4 as having complications other than pneumonia, otitis media, encephalitis or meningitis (Table 3.6.2). All hospitalisations coded with measles complicated by pneumonia were aged either <5 years or 25–59 years.

There were no deaths from measles recorded in the AIHW National Mortality Database in 2005 or 2006 (Table 3.6.1).

Table 3.6.2: Selected indicators of severe morbidity* for hospitalised cases of measles, Australia, 2005 to 2007,* by age group

Age group (years)	Measles complicated by pneumonia		Measles with complications other than pneumonia, otitis media, encephalitis or meningitis		Measles without complications	
	n	% Total†	n	% Total†	n	% Total†
0–4	2	12.5	2	12.5	12	75.0
5–14	0	–	1	20.0	4	80.0
15–24	0	–	1	10.0	9	90.0
25–59	3	12.0	0	–	22	88.0
60+	0	–	0	–	0	–
All ages	5	8.9	4	7.1	47	83.9

* Based on National Hospital Morbidity data where the date of hospital separation was between July 2005 and June 2007.

† % of total in the age group.

Age and sex distribution

In 2006–2007, the highest notification rate for measles was seen in the <5 years age group, and the notification rates were progressively lower with increasing age (Table 3.6.1, Figure 3.6.2).

Notification rates for all age groups returned to very low levels in 2007 after the resurgence in 2006. Most notably, notifications in children aged <5 years decreased from 2.22 per 100,000 population in 2006 to 0.30 per 100,000 in 2007 (Figure 3.6.2). Additionally, in 2007, for the first time since the Measles Control Campaign in 1998, there were no cases recorded in adults aged ≥ 35 years. In both 2006 and 2007, the notification rate for the 0–4 years age group returned to being the highest of all groups, after the previous 3 years in which the 20–34 years age group had the highest rate (Figure 3.6.2).

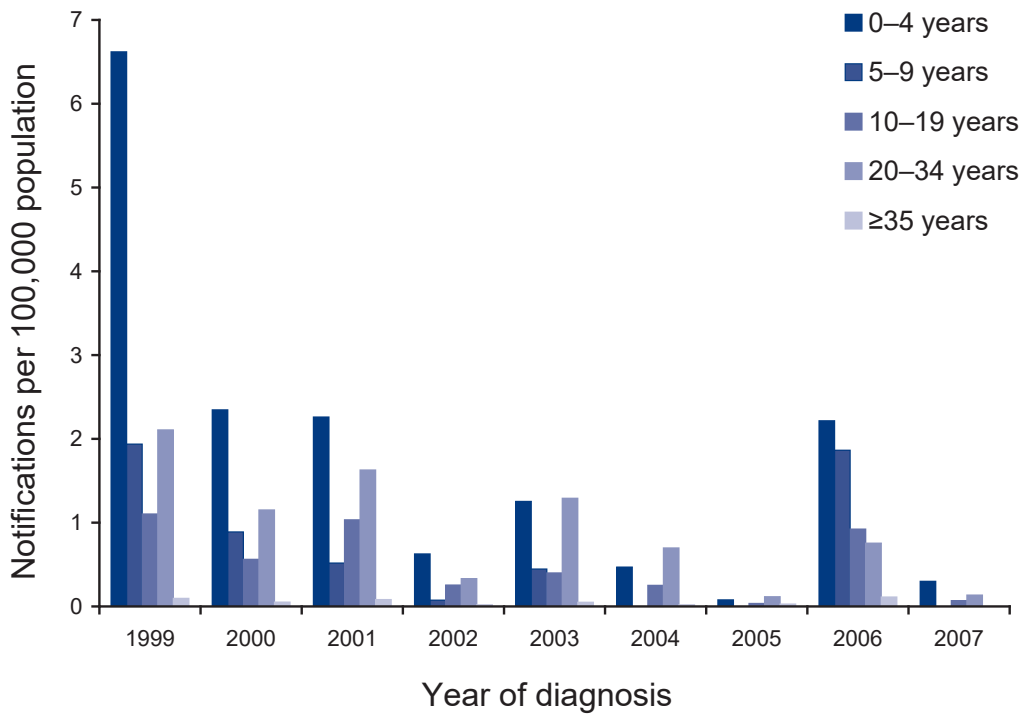
From July 2006 to June 2007, hospitalisation rates were the lowest on record for the 0–4 years (0.38 per 100,000) and ≥ 35 years (0.00 per 100,000) age groups (Figure 3.6.3). No hospitalisations were recorded for persons aged ≥ 35 years. Hospitalisation rates for both the 5–9 years and 10–19 years age groups also decreased over the review period July 2005 to June 2007, returning to levels comparable to those observed in the previous 3 years. Hospitalisation rates for the 0–4 years age group have consistently been the highest of all age groups since reporting began, and, since 2000/2001, persons 20–34 years of age have had the second highest hospitalisation rate (Figure 3.6.3).

Over the 2-year period 2006–2007, there were slightly more notifications for females than males (male:female ratio 1:1.1). However, more males were hospitalised than females (male:female ratio 1.3:1).

Geographical distribution

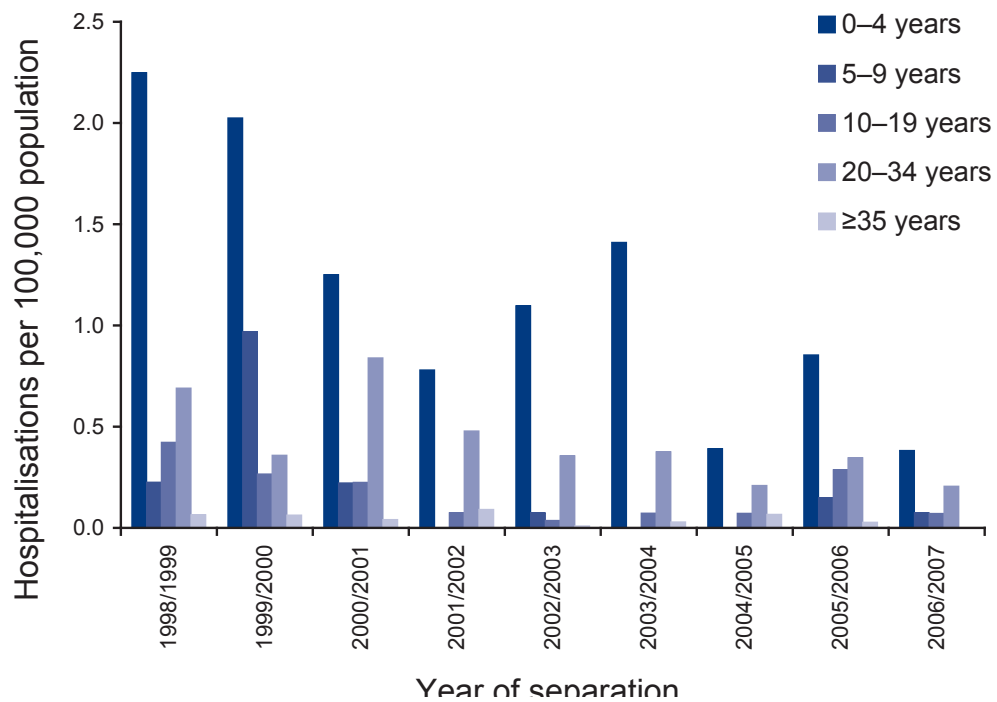
There were 125 measles notifications in 2006, of which 82 (66%) occurred as a result of a multi-jurisdictional outbreak associated with an Indian spiritual group that toured Australia from March to May 2006. Only 7% of the cases in that outbreak were fully immunised against measles, while two-thirds were unimmunised.⁴ Another separate outbreak consisting of 3 primary cases and 8 secondary cases was linked to an Emergency Department in New South Wales on 1 March 2006. The index case was believed to be a sick traveller; however, this could not be confirmed.⁵ One case was imported from Europe in March 2006 but no related secondary transmission

Figure 3.6.2: Measles notification rates, Australia, 1999 to 2007,* by age group and year of diagnosis



* Notifications where the date of diagnosis was between January 1999 and December 2007.

Figure 3.6.3: Measles hospitalisation rates, Australia, 1998/1999 to 2006/2007,* by age group and year of separation



* Hospitalisations where the date of separation was between July 1998 and June 2007.

was detected, despite enhanced surveillance measures implemented at the time for the 18th Commonwealth Games in Melbourne.⁶ Several other Victorian cases had a recent history of overseas travel; however, some additional cases were identified where travel outside the state during the incubation period had not occurred.^{7–10} In 2006–2007, 17 cases were recorded in NNDSS as acquired overseas. For a further 18 cases, this information was unknown or missing. The remaining 102 cases were recorded as locally acquired, but it is unknown how many of these were linked to an imported case, as this information is not routinely collected at national level.

Measles cases were notified from all jurisdictions, except the Northern Territory, during the period 2006–2007. The rate of notification over the reporting period was highest in Tasmania with 1.12 per 100,000 population (n=11), followed by Western Australia with 0.74 per 100,000 (n=31). In both states, these rates were more than twice the national rate of 0.33 per 100,000. The increased notification rates in most jurisdictions during the review period were largely due to the multi-jurisdictional outbreak described above.

There was little variation during 2006–2007 between jurisdictional rates of hospitalisation for those regions with hospitalised cases (Appendix 6.3).

Measles typing and vaccination status

Measles virus genotype was recorded in NNDSS for 20 cases (15%) notified during the reporting period 2006–2007. The majority of these were D8, the genotype confirmed as the cause of the 2006 multi-state outbreak imported by the Indian spiritual group.⁴ Other genotypes noted in NNDSS included D4 and D5, and the case in Victoria in March 2006 was identified as genotype B3, most likely contracted during travel in Germany.⁶

Vaccination status is expected to be completed in NNDSS for all notifications of measles in people born after 31 December 1969. Overall, 89% (116/130) of cases who were born after this date and notified in the review period had this field completed. Of the 123 cases aged ≥ 1 year among these 130 notified cases, 14 were reported as fully vaccinated for age, of which 5 were aged 1–3 years and thus had received only 1 dose of measles-containing vaccine; a further 9 were recorded as partially vaccinated for age, and 86 as unvaccinated. The remaining 7 of the 130 cases were aged < 1 year and were thus most likely too young to have received any dose of measles-containing vaccine. Vaccination status was validated by written records in 43% (6/14) of the cases reported to be fully vaccinated, 20% (2/10) of partially vaccinated cases, and 28% (26/92) of cases whose vaccination status was recorded as unvaccinated or 'not applicable'.

Comment

In the 2-year review period 2006–2007, with the exception of the outbreak between March and May 2006, measles notifications and hospitalisations remained at very low rates. Measles accounted for only 12 notifications in 2007 and 17 hospitalisations between July 2006 and June 2007. The overall long-term downward trend is similar to that seen in other countries with high vaccination coverage. Endemic measles has been eradicated in the USA since 2000^{11,12} and transmission has been interrupted in most other countries across the Americas.¹³ Indigenous measles was declared to be eliminated in England and Wales following an intensive school-based vaccination program in 1994.¹⁴ However, since that time, vaccination coverage in the UK decreased significantly, mostly associated with the controversy regarding the safety of the measles-mumps-rubella (MMR) vaccine. As a result the proportion of susceptible children aged 2–4 years has been estimated to be 27% in 2004/2005,¹⁵ a level sufficient to support the continuous endemic spread of measles.¹⁶ Despite all the gains made in measles control in recent decades, outbreaks continue to occur in unvaccinated groups throughout Europe¹⁷ and in the USA.¹⁸

Notwithstanding the progress made in countries with high vaccination coverage, travellers remain at increased risk of measles exposure in many countries where high vaccination coverage has not been achieved and endemic measles transmission still occurs. In 2007, global measles-containing vaccine coverage was estimated to be 82%. However, this varied significantly geographically, with coverage in the Western Pacific region at over 90%.¹⁹ The global measles disease burden continues to decrease but there were still an estimated 279,000 cases in 2007 and 242,000 deaths in 2006.¹⁹ Despite the progress made in improving measles surveillance activities by many countries, these figures are still likely to be an underestimate.²⁰

Where endemic measles has been eliminated, enhanced surveillance including laboratory confirmation is required and recommended by the World Health Organization (WHO).²¹ Therefore, in Australia, laboratory evidence or linkage to a chain of transmission that includes a laboratory-confirmed case is now required in the case definition for notification and this is the first review period for which this has been consistent at a national level for the whole period.

The WHO Regional Committee for the Western Pacific set a target date of 2012 for elimination of endemic measles transmission in the region.²² Mathematical modelling of vaccine coverage and data generated from the two previous serosurveys in Australia have demonstrated that the effective reproductive number R (the average number of secondary cases produced by a typical primary case in a given population) is less than 1 and this is predicted to remain the case until at least 2012.^{23,24} Consistently high 2-dose vaccine coverage, serological evidence of very high population immunity and a high proportion of cases imported or linked to an imported case, as well as sustained maintenance of an R of less than 1, indicate that endemic transmission has already been eliminated in Australia since 2005.²² Continued monitoring is still required, however, as Australia has not yet achieved a residual population susceptibility of 5% following completion of the 2-dose schedule, which is required for long-term sustained measles elimination.²³ Furthermore, vaccine coverage varies geographically and between age groups. Thus certain regions or birth cohorts with low proportions of immune individuals may allow some sustained transmission of measles if it is imported into that cohort, although secondary transmission in the vaccinated population would be extremely limited.²⁴

The current 2-dose measles vaccine schedule began in 1998 following the mass vaccination of primary school aged children as part of the Measles Control Campaign.²⁵ Since then, consistently high vaccination coverage prior to school entry has been maintained and, as discussed above, this has contributed to the elimination of endemic measles transmission in Australia.^{22,26} However, the high level of vaccine coverage in children and reduced exposure to naturally circulating measles virus have left a residual cohort of susceptible young adults born in the late 1970s and early 1980s, when measles vaccine was first introduced but coverage was low. To improve immunity in this birth cohort, the young adult MMR vaccination campaign was undertaken during 2001.²⁵ However, a serosurvey conducted in 2002 indicated that immunity in the young adult cohort did not significantly improve.²³ It also indicated Australia had reached WHO age-specific susceptibility targets in children <10 years of age but not in older age groups, with a relatively high proportion (9%) of targeted young adults remaining susceptible.²⁷ This was reflected in the notification data from 2003–2005 when the notification rate for 20–34 year olds was the highest of all age groups. However, in the current review period, the 0–4 years age group has again returned to having the highest notification rate.

Despite the elimination of endemic transmission, Australia remains at risk of measles importation from countries where measles is still endemic.²⁶ The source case of most measles outbreaks had a recent overseas travel history and molecular genotyping of measles isolates provided confirmation of imported strains,²⁸ but some outbreaks have not had a source case identified.

In summary, the notification data show a relative decrease in measles cases in young adults and that very low rates of background measles cases have been maintained. However, the potential for relatively large outbreaks, such as the one that occurred in early 2006, still exists and ongoing efforts to increase uptake of the MMR vaccine need to be maintained to decrease residual susceptibility to below 5%. Children aged <5 years have the highest rates of measles infection and hospitalisation of any age group, suggesting that high coverage, including better timeliness and completeness of childhood vaccinations among preschool aged children, should remain an important goal of Australia's measles control strategy if we are to avoid a return of endemic measles transmission such as that currently occurring in England.¹⁵

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3.7 Meningococcal disease

Meningococcal disease is defined for surveillance purposes as the isolation of the bacterium *Neisseria meningitidis* from cerebrospinal fluid, blood and other normally sterile sites including skin lesions. Clinical manifestations include meningitis, meningococcaemia without meningitis (which varies in presentation from fulminant to chronic), a mixture of meningitis and septicaemia, 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 laboratory evidence. This includes the identification of Gram-negative intracellular diplococci or meningococcal antigen in, for example, blood or cerebrospinal fluid; the identification of nucleic acid from *N. meningitidis* in body fluids; or demonstration of a serological response to *N. meningitidis*.^{1,2}

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

National definition from January 2004:^{*3}

Both confirmed and probable cases are notifiable. Confirmed cases require either laboratory definitive evidence, or laboratory suggestive evidence together with clinical evidence. Probable cases require specified clinical evidence only (as below).

- 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 *N. meningitidis*; or
 - Positive polysaccharide antigen test in cerebrospinal fluid with other laboratory parameters consistent with meningitis.[‡]
- c) Clinical evidence (for notification of confirmed cases, together 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.

* The national case definition was revised and endorsed by CDNA in October 2007. Time of implementation of the revised case definition varied among different jurisdictions following the revision.

† 'Detection of meningococcus from a normally sterile site by nucleic acid testing' is considered laboratory definitive evidence in the revised case definition of October 2007.

‡ 'Positive polysaccharide antigen test in cerebrospinal fluid with other laboratory parameters consistent with meningitis' is no longer accepted as laboratory suggestive evidence in the revised case definition of October 2007.

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

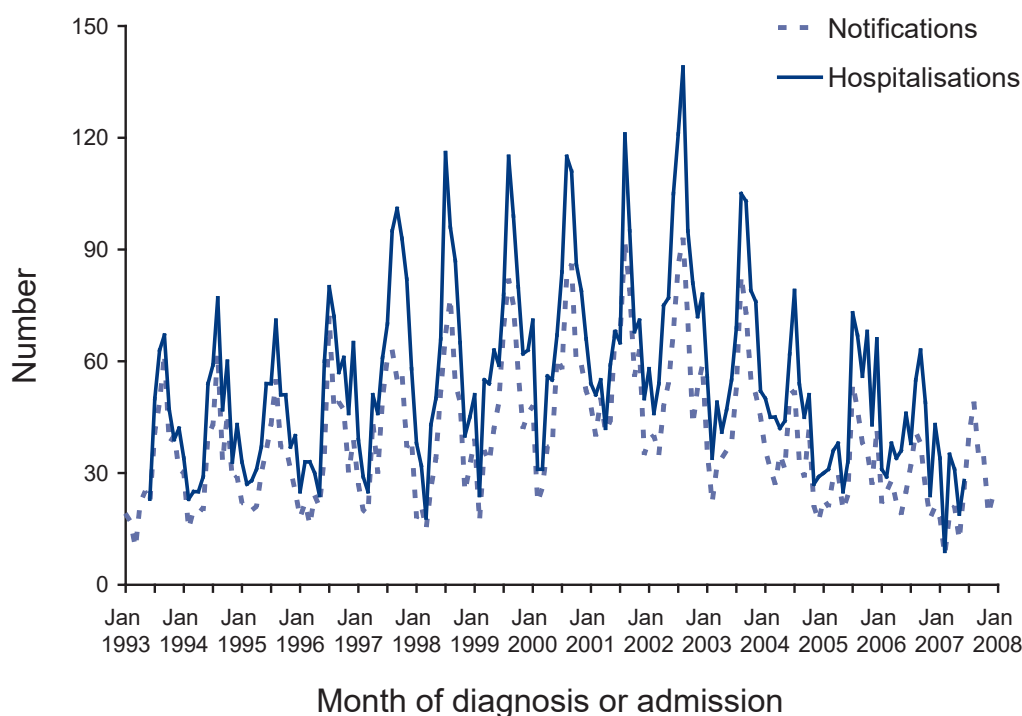
The ICD-10 code used to identify deaths recorded in the AIHW National Mortality Database was A39 (meningococcal infection).

Secular trends

There were 622 notifications of meningococcal disease in the 2 years 2006–2007, an average annual notification rate of 1.5 per 100,000 population (Table 3.7.1). A median of 24.5 cases was notified each month, with a range of 8–49 cases. Between July 2005 and June 2007, there were 1,024 episodes of hospitalisation recorded as ICD-10-AM code A39 (average annual rate 2.5 per 100,000), and a median of 38 admissions (range 9–73) per month. Coinciding with the introduction of the national meningococcal C immunisation program in January 2003,⁴ both the notification and hospitalisation rates for meningococcal disease decreased each year, down from a peak in 2002 (Figure 3.7.1). The notification rate for meningococcal disease of any serotype decreased by 57% from 3.5 cases per 100,000 in 2002 to 1.5 cases per 100,000 in 2007 (Appendix 6.2), while the hospitalisation rate for all meningococcal disease decreased by 53% from 4.5 per 100,000 in 2002/2003 to 2.1 per 100,000 in 2006/2007 (Appendix 6.3).

A clear seasonal pattern was apparent, with the highest number of notifications and hospitalisations occurring between June and September each year (Figure 3.7.1).

Figure 3.7.1: Meningococcal notifications and hospitalisations, Australia, 1993 to 2007,* by month of diagnosis or admission



* Notifications where the date of diagnosis was between January 1993 and December 2007; hospitalisations where the date of admission was between July 1993 and June 2007.

Severe morbidity and mortality

A total of 7,482 hospital bed days (average 3,741 days per year) were recorded for patients with an ICD-10-AM code of A39, of which 48% were coded as meningococcal meningitis (A39.0) and 36% were coded as septicaemia (A39.1–A39.4, not A39.0). The proportion of hospitalisations where a meningococcal disease code was the principal diagnosis varied from 94% of cases among the 0–14 year age group to 86% of cases for those aged 15–59 years and 62% for those aged ≥ 60 years. The median length of hospital stay was 5 days and increased with age (Table 3.7.1).

Table 3.7.1: Meningococcal notifications, hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Notifications 2 years (2006–2007)		Hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission (days)		Deaths 2 years (2005–2006)	
	n	Rate [‡]	n	(§)	Rate [‡]	(§)	Median	(§)	n	Rate [‡]
0–4	222	8.4	381	(359)	14.7	(13.8)	4.0	(4.0)	15	0.58
5–14	63	1.1	141	(133)	2.6	(2.4)	2.0	(3.0)	2	0.04
15–24	180	3.1	242	(213)	4.2	(3.7)	5.0	(5.0)	3	0.05
25–59	116	0.6	182	(151)	0.9	(0.8)	7.0	(6.0)	7	0.03
60+	41	0.5	78	(48)	1.1	(0.7)	11.0	(8.0)	5	0.07
All ages	622	1.5	1,024	(904)	2.5	(2.2)	5.0	(5.0)	32	0.08

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006 on the AIHW National Mortality Database.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

Information about deaths was available from three different sources for overlapping time periods. All show an overall downward trend.

- Death certificate data: In 2005 and 2006 there were 32 deaths (0.08 per 100,000 population) with meningococcal disease recorded as the underlying cause of death (Table 3.7.1). Twenty-five (78%) were coded as septicaemia (A39.1–A39.4, not A39.0).
- Hospitalisation data: Of the 1,024 hospital separations during the period July 2005–June 2007, 24 (2.3%) were recorded as dying in hospital. The proportion of meningococcal infection hospitalisations with death as the outcome was lowest in the 5–24 year age group (1%) and highest in those aged ≥ 60 years (6%). Case fatality rates were higher among patients coded as having meningococcal septicaemia (A39.1–A39.4, not A39.0; 4.1%) compared with those coded as having meningococcal meningitis (A39.0; 1.6%).
- Notifications: Twenty-one deaths were recorded among the 622 cases of meningococcal disease notified to NNDSS for the 2 years 2006–2007 (case fatality rate of 3.4%). The age group distribution of the 12 notified cases who died in 2006 was comparable to that of the 12 deaths in 2006 in the AIHW National Mortality Database.

Age and sex distribution

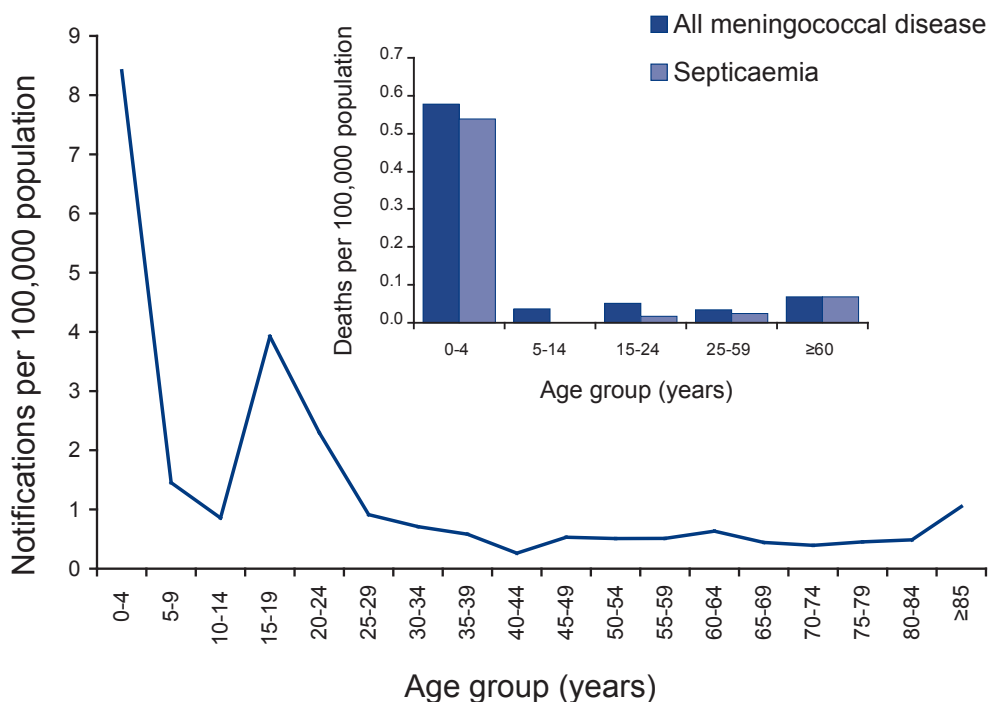
The highest meningococcal disease notification, hospitalisation and death rates occurred among children aged < 5 years (Table 3.7.1), and the highest rates within this age group were among those < 1 year of age (19.4 notifications, 34.4 hospitalisations and 2.03 deaths per 100,000 population). There was a second peak in notification rates (Figure 3.7.2) and hospitalisation rates (Figure 3.7.3) in the 15–19 year age group (3.9 and 5.0 per 100,000, respectively); rates were also elevated in the 20–24 year age group, but were lower in those ≥ 25 years of age (Table 3.7.1, Figures 3.7.2 and 3.7.3).

Overall, there was a slight predominance of male cases (male:female rate ratio 1.1:1). This varied across age groups with generally higher rates in males aged 0–34 years and females aged ≥ 60 years.

Geographical distribution

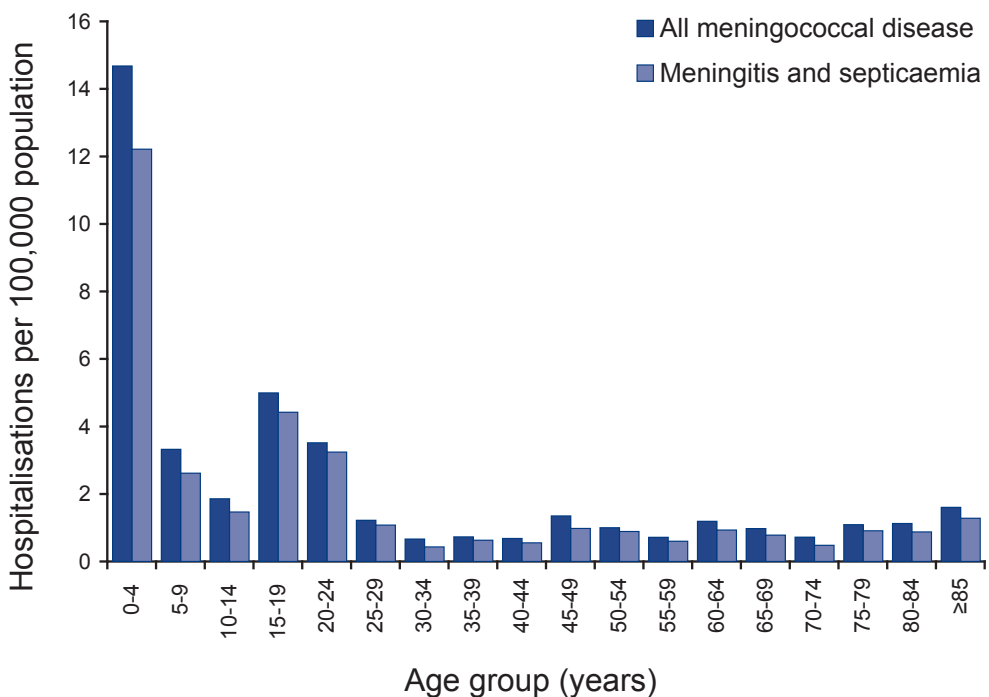
The pattern of notification and hospitalisation rates for meningococcal disease varied across the country. The highest notification and hospitalisation rates were in the Northern Territory (2.8 notifications per 100,000 population in both 2006 and 2007, and 5.3 hospitalisations per 100,000 population in 2005/2006). The lowest notification rates occurred in Tasmania and Western Australia in 2006–2007 (both were 1.0 per 100,000 population) (see also Appendix 6.2), while the lowest hospitalisation rate occurred in the Australian Capital Territory in 2005/2006–2006/2007 (1.7 per 100,000 population) (see also Appendix 6.3).

Figure 3.7.2: Meningococcal disease notification and death rates, Australia, 2005 to 2007,* by age group



* Notifications where the date of diagnosis was between January 2006 and December 2007; deaths where the death was recorded between January 2005 and December 2006 on the AIHW National Mortality Database.

Figure 3.7.3: Meningococcal disease hospitalisation rates, Australia, 2005/2006 to 2006/2007,* by age group



* Hospitalisations where the date of separation was between July 2005 and June 2007.

Notification rates were slightly lower in most states and territories in 2007 compared with 2006 (Appendix 6.2). Hospitalisation rates decreased between 2005/2006 and 2006/2007 across all jurisdictions (Appendix 6.3).

Meningococcal serogrouping and vaccination

Meningococcal serogroup information was recorded for 83% of the 622 notified cases in 2006–2007 (Figure 3.7.4). There were 431 notified cases (1.0 per 100,000 population) of serogroup B meningococcal disease and 43 cases (0.1 per 100,000 population) of serogroup C disease. Between 2002 (before the national meningococcal C vaccination program commenced) and 2007, serogroup C disease notifications decreased by 92% from a peak of 225 in 2002 to 19 in 2007 (1.15 to 0.09 per 100,000 population) (Figure 3.7.4). By comparison, serogroup B disease notifications decreased by 27% from a peak in 2002 of 294 to 213 cases in 2007 (1.5 to 1.1 per 100,000 population). As a proportion of total notifications for meningococcal disease, serogroup C decreased from 33% in 2002 to 9% in 2007, while, over the same period, the proportion of serogroup B notifications increased from 40% to 70%; those where serogroup information was not available decreased slightly from 21% to 17%.

Much of the decrease in serogroup C meningococcal disease notifications occurred among the age groups included in the national meningococcal C vaccination program during 2003–2006 (Figure 3.7.5a). A decrease in serogroup C notifications was also evident among the 20–24 year age group, which includes some people born during or after 1984 and eligible for free vaccine, as well as adults aged ≥25 years who were not included in the program (Figure 3.7.5b). Serogroup C notifications among children aged <1 year remained low in 2006–2007, with an average of 2 notifications per year (0.7 cases per 100,000 population), compared with an average of 38 serogroup B notifications per year (14.0 notifications per 100,000 population).

In 2006–2007, meningococcal C vaccination status was recorded for 35 (81%) of the 43 notified cases of serogroup C disease and 15 of the 16 cases aged ≤19 years. There were just three reports of vaccine failure in children, aged 3 years, 14 years and 15 years at disease onset.

Figure 3.7.4: Meningococcal disease notifications, Australia, 1991 to 2007, by serogroup and year of diagnosis

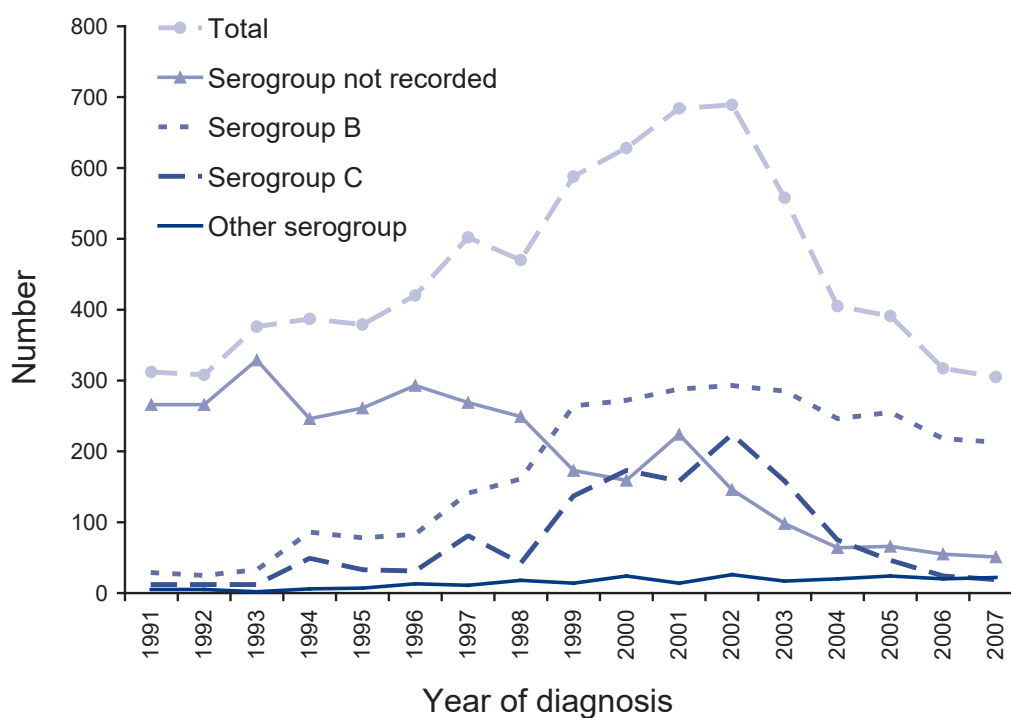
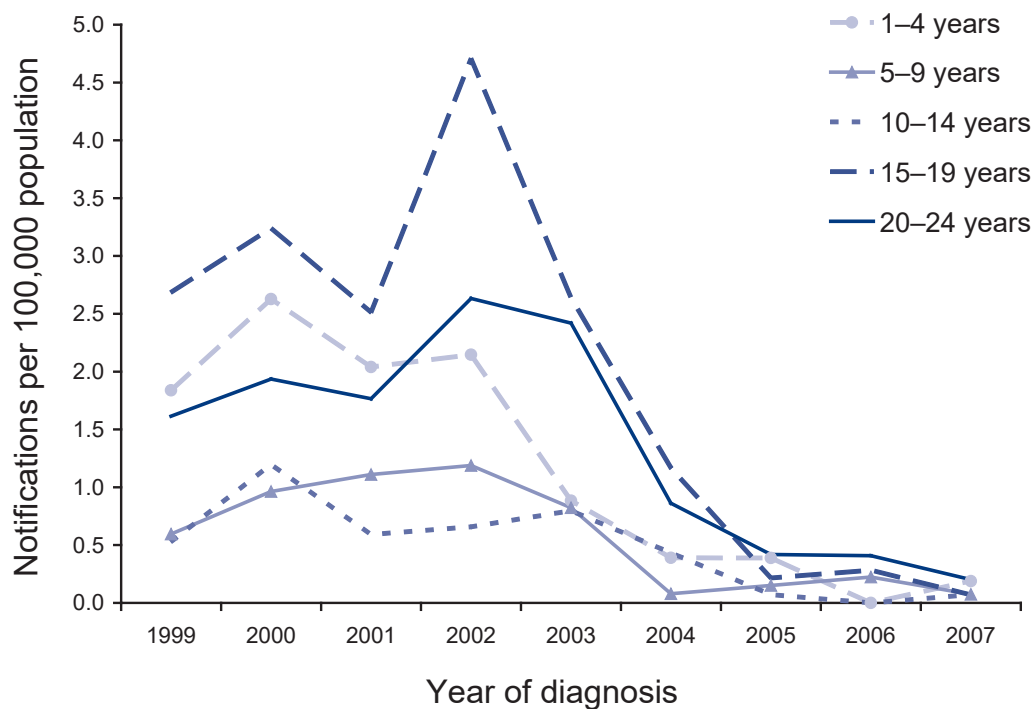
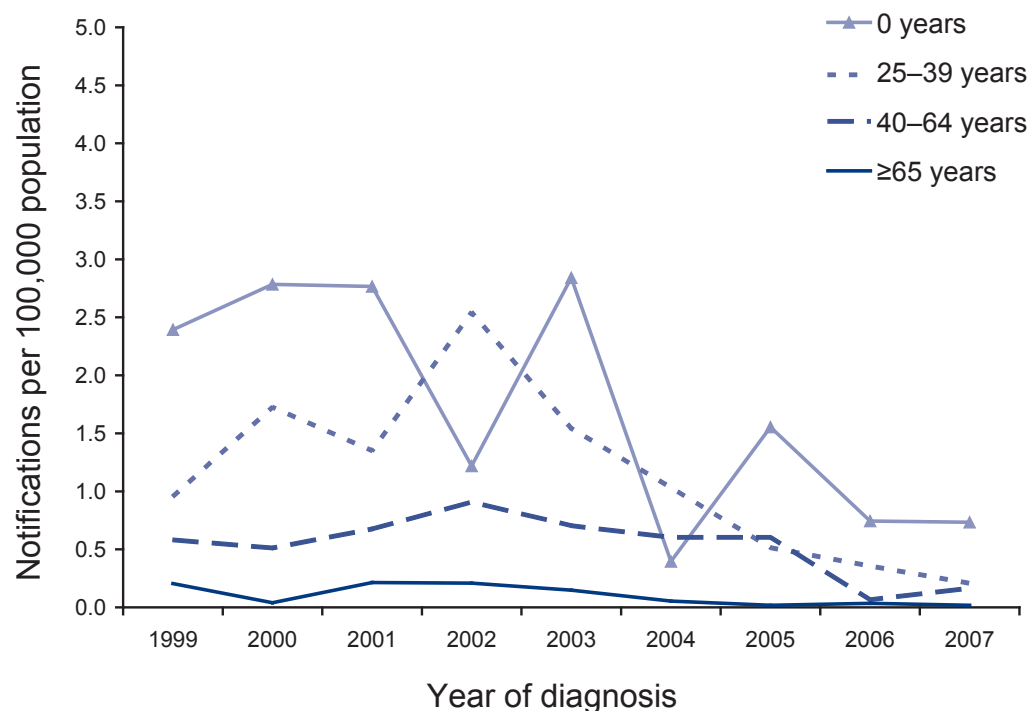


Figure 3.7.5: Meningococcal serogroup C disease notification rates, Australia, 1999 to 2007, by age group* and year of diagnosis

(a)

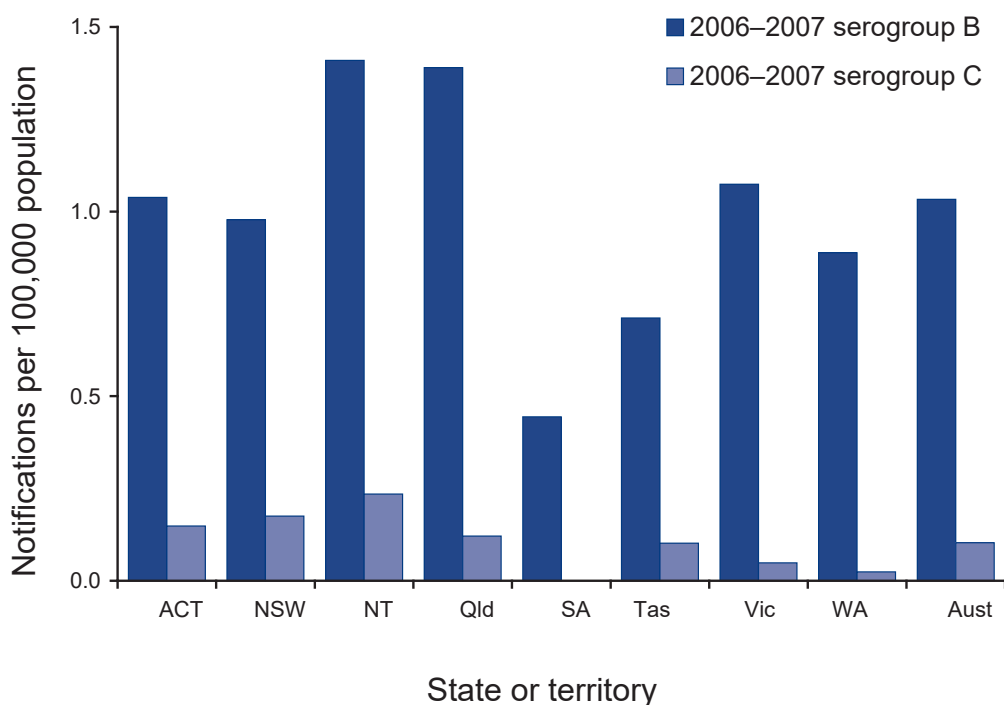


(b)



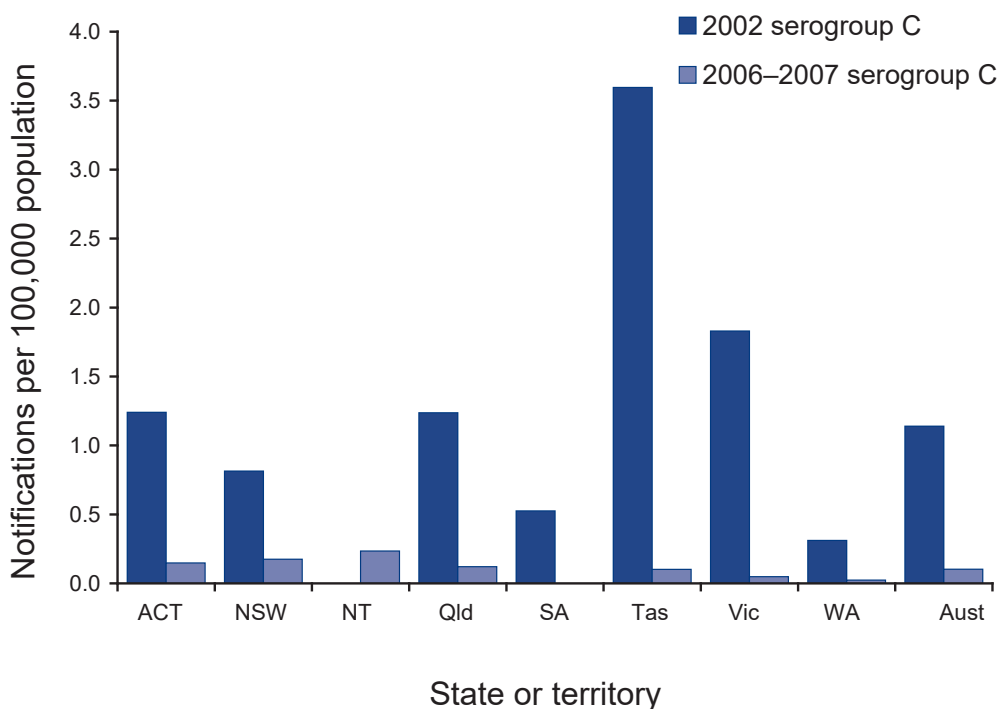
* Figure 3.7.5a shows the age groups included in the meningococcal C vaccination program from 1 January 2003. Figure 3.7.5b shows other age groups. (Note: Cases aged 20–24 years in 2007 include those born during or after 1984 and included in the national meningococcal C vaccination program.)

Figure 3.7.6: Meningococcal disease notification rates, Australia, 2006 to 2007,* by state or territory and serogroup



* Notifications where the date of diagnosis was between January 2006 and December 2007.

Figure 3.7.7: Meningococcal serogroup C disease notification rates, Australia, 2006–2007* compared with 2002,† by state or territory



* Notifications where the date of diagnosis was between January 2006 and December 2007.

† Notifications where the date of diagnosis was between January 2002 and December 2002.

During 2006–2007, there was some heterogeneity between jurisdictions in the relative notification rates of serogroup B and serogroup C meningococcal disease (Figure 3.7.6), with the ratio of serogroup B to serogroup C varying from 37:1 in Western Australia to 5.5:1 in New South Wales. Following the introduction of the national meningococcal C vaccination program in January 2003, there was a significant reduction in serogroup C notification rates across most states and territories, with the largest reductions in Tasmania and Victoria (Figure 3.7.7).

Comment

The incidence of meningococcal disease in Australia increased continuously between 1991 and 2002. Since 2003, there has been a marked and sustained decrease in meningococcal disease notifications, hospitalisations and deaths⁵ following the introduction of the routine and catch-up meningococcal C vaccination programs for those born after 1983.

The largest reduction in notifications and deaths occurred for serogroup C disease and those where serogroup information was not available (suggesting many were serogroup C). Over the same period, notifications for serogroup B have declined slightly, presumably related to known natural variations in meningococcal disease epidemiology over time.^{5,6} Notifications for other serogroups, mainly W₁₃₅ and Y, have remained relatively stable over the 2002–2007 period. Reassuringly, these data, and those reported elsewhere for Australia and internationally, show that there has been no evidence of serogroup replacement following mass meningococcal C immunisation programs.^{7–10}

The decrease in serogroup C disease notifications occurred across Australia and among all 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 adults not included in the national vaccination program, while rates among children <1 year of age remained very low. This suggests protection across all age groups through herd immunity, primarily as a consequence of the mass immunisation program and, to a lesser extent, from a reduction in natural exposure to pharyngeal infection/colonisation with serogroup C.^{11–14}

Between 2003 and 2007, there were six recorded cases of vaccine failure in Australia, including the three reported here.^{15,16} Four were aged 1–4 years and two were adolescents. A similar low rate of vaccine failure among young children has been observed in The Netherlands,¹⁷ which, like Australia, routinely immunises children in the 2nd year of life. In contrast, the UK has reported considerably higher levels of vaccine failure, particularly among those immunised during the first 6 months of life.^{18,19} A recent serologic study in the UK reported that persistence of bactericidal antibody was correlated with age at vaccination, with higher levels of protection 5 years after receipt of conjugate meningococcal vaccine among children aged ≥10 years at the time of vaccination compared with younger age groups.²⁰ This finding suggests that the low number of vaccine failures seen among Australian adolescents is related to a strong immunogenic response in those vaccinated through mass school- and community-based immunisation programs during 2003 and 2004 as well as herd immunity. Ongoing surveillance and reporting of the vaccination status of cases of meningococcal serogroup C disease is essential to monitor vaccine effectiveness over the longer term and inform Australian meningococcal C immunisation policy.

Currently, as reported here and by the National Neisseria Network,⁷ the great majority of the burden of invasive meningococcal disease in Australia is now due to serogroup B disease, which contributes approximately 70% of meningococcal disease cases. Those at particular risk of meningococcal serogroup B disease are infants <1 year of age and, as reported elsewhere, Indigenous Australians.^{21,22} The high burden of serogroup B meningococcal disease in these risk groups emphasises the importance of early recognition and appropriate clinical management of disease and the need for the development of a vaccine to reduce the significant morbidity and mortality. Several candidate serogroup B vaccines are under investigation;²³ however, it will be some time yet before a universal serogroup B vaccine appropriate for use in Australia becomes available for consideration.

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3.8 Mumps

Mumps is an acute viral disease caused by a paramyxovirus. The classical disease is characterised by fever, as well as swelling and inflammation of one or more salivary glands, most commonly the parotid glands. However, up to 30% of cases will not have salivary gland involvement. It is a multi-system infection, with up to 10% of cases developing aseptic meningitis and 30% of post-pubertal males experiencing epididymo-orchitis.¹

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

National definition from January 2004:²

Only confirmed cases are notifiable. 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 4-fold 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 other salivary glands lasting 2 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

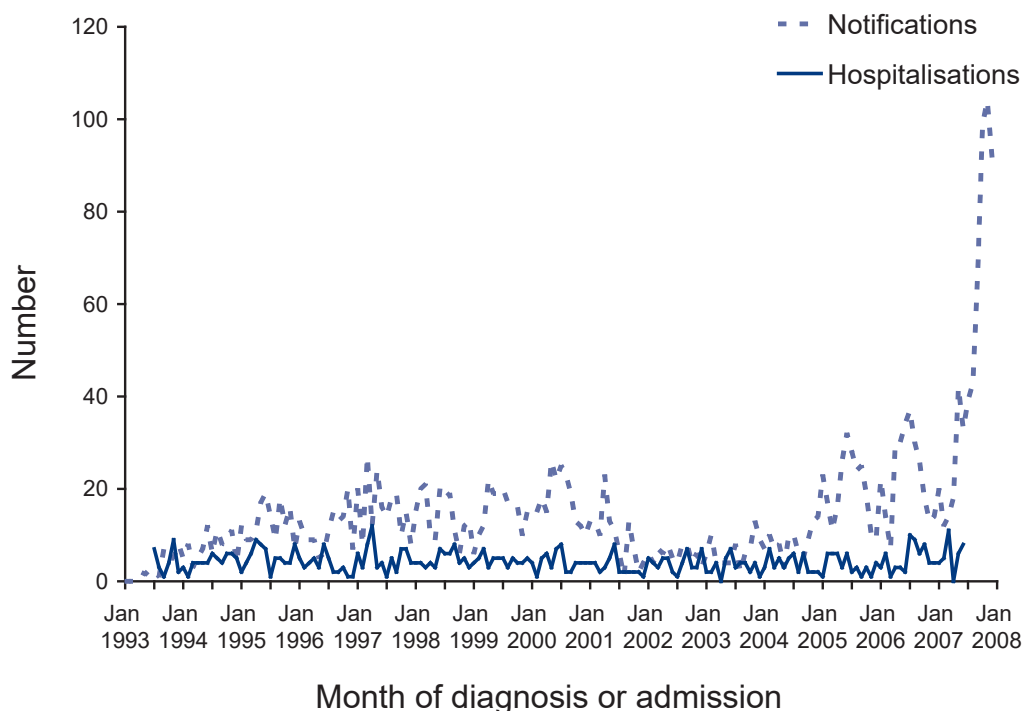
The epidemiology of mumps in Australia during this reporting period was characterised by a substantial increase in notifications in the latter half of 2007. This resulted particularly from outbreaks of mumps around two Indigenous communities in the Northern Territory, together with increased notifications from Western Australia (where some of the cases were linked to the Northern Territory outbreaks) and, to a lesser extent, New South Wales. Those affected were predominantly young adults and adolescents.

During the 2 years from January 2006 to December 2007, there were 857 notifications of mumps (an average annual notification rate of 2.1 per 100,000) (Table 3.8.1). Although 2006 continued the recent trend of increasing notifications (275 notifications, rate 1.3 per 100,000), it was 2007 that was particularly remarkable with 582 notifications, at a rate of 2.8 per 100,000. To put this in perspective, there were 163 more notifications in 2007 alone than in 2003–2005 combined, and the notification rate in 2007 was about 4 times the average annual rate for 2003–2005.³ The median number of notifications per month during the period from January 2006 to April 2007 was 19 (range 7–37) with the peak in July 2006, in contrast to 55 per month (range 33–104) from May to December 2007 with the peak in November 2007 (Figure 3.8.1). It is generally recognised that the peak incidence of mumps in temperate climates typically occurs in winter and spring. This pattern has largely been maintained during the 2006–2007 period with the number of cases during the winter and spring months being either close to the median or well above it.

From July 2005 to June 2007, there were 107 hospitalisations coded as being due to mumps (average annual rate of 0.26 per 100,000), of which 94 (88%) had mumps listed as the principal diagnosis (Table 3.8.1). During this period there was a median of 4 admissions per month (range 0–11), compared with a median of 3.5 admissions per month in the preceding 2-year period.³ Seventy-five of the 107 hospitalisations occurred in the 12 months from July 2006 to June 2007, with a median of 6 admissions per month (range 0–11) during this period, compared with a median of 3 admissions per month (range 1–6) from July 2005 to June 2006. At the time of preparation

of this report, hospitalisation data for the latter half of 2007, when there was a substantial increase in mumps notifications, were not yet available for comparison. During the period when data are available for both, hospitalisation rates mirrored the fluctuating trends in notification rates (Figure 3.8.1) on a much smaller scale.

Figure 3.8.1: Mumps notifications and hospitalisations, Australia,* 1993 to 2007,† by month of diagnosis or admission



* 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.

† Notifications where the date of diagnosis was between January 1993 and December 2007; hospitalisations where the date of admission was between July 1993 and June 2007.

Severe morbidity and mortality

There were 343 hospital bed days (average 171.5 per year) recorded for patients with an ICD-10-AM code for mumps. Complications arising from mumps infection were recorded for 22 hospitalisations (20.6%). As in the past, the most commonly reported complication was orchitis. There were 14 (13%) hospitalised cases coded with orchitis; 13 of these were between 15 and 59 years of age (Table 3.8.2). There were 2 hospitalisations coded as neurological complications (encephalitis or meningitis) and 1 as pancreatitis, all of which occurred in cases aged 15–59 years. Five patients were listed as having ‘other complications’; unfortunately, these are not specified. As with the neurological complications and orchitis, nearly all the cases with ‘other complications’ occurred in the adolescent and adult age groups. None of these hospitalisations had multiple mumps complication codes. The median length of stay in hospital was 1 day, but adults aged ≥ 25 years had a longer median LOS compared with younger age groups (Table 3.8.1), especially cases aged ≥ 60 years who had a median LOS of 6 days. Adults in the 25–59 years age group (which constitute about 49% of the Australian population) accounted for 48.6% of the hospitalisations, compared with 38.4% during the preceding 3-year period.³ Adults over 15 years of age (constituting about 80% of the total population) accounted for 78.5% of hospitalisations, which was not substantially different from the figure in the preceding 3 years of 75.4%.³ Mumps was recorded as the underlying cause of death in one adult (aged ≥ 60 years) in the period 2005–2006 in the AIHW National Mortality Database.

Table 3.8.1: Mumps notifications, hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Notifications 2 years (2006–2007)		Hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission (days)		Deaths 2 years (2005–2006)	
	n	Rate [‡]	n	(§)	Rate [‡]	(§)	Median	(§)	n	Rate [‡]
0–4	19	0.7	9	(9)	0.35	(0.35)	1.0	(1.0)	0	–
5–14	95	1.7	14	(13)	0.26	(0.24)	1.0	(1.0)	0	–
15–24	200	3.4	19	(18)	0.33	(0.32)	1.0	(1.0)	0	–
25–59	525	2.6	52	(46)	0.26	(0.23)	1.5	(1.0)	0	–
60+	18	0.2	13	(8)	0.18	(0.11)	6.0	(5.5)	1	0.01
All ages	857	2.1	107	(94)	0.26	(0.23)	1.0	(1.0)	1	<0.005

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

Table 3.8.2: Indicators of severe morbidity* for hospitalised cases of mumps, Australia, 2005 to 2007,* 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	–	1	11.1	0	–	0	–
5–14	0	–	0	–	0	–	1	7.1
15–24	1	5.3	2	10.5	0	–	0	–
25–59	1	1.9	11	21.2	1	1.9	2	3.8
60+	0	–	0	–	0	–	2	15.4
All ages	2	1.9	14	13.1	1	0.9	5	4.7

* Based on National Hospital Morbidity data where the date of hospital separation was between July 2005 and June 2007.

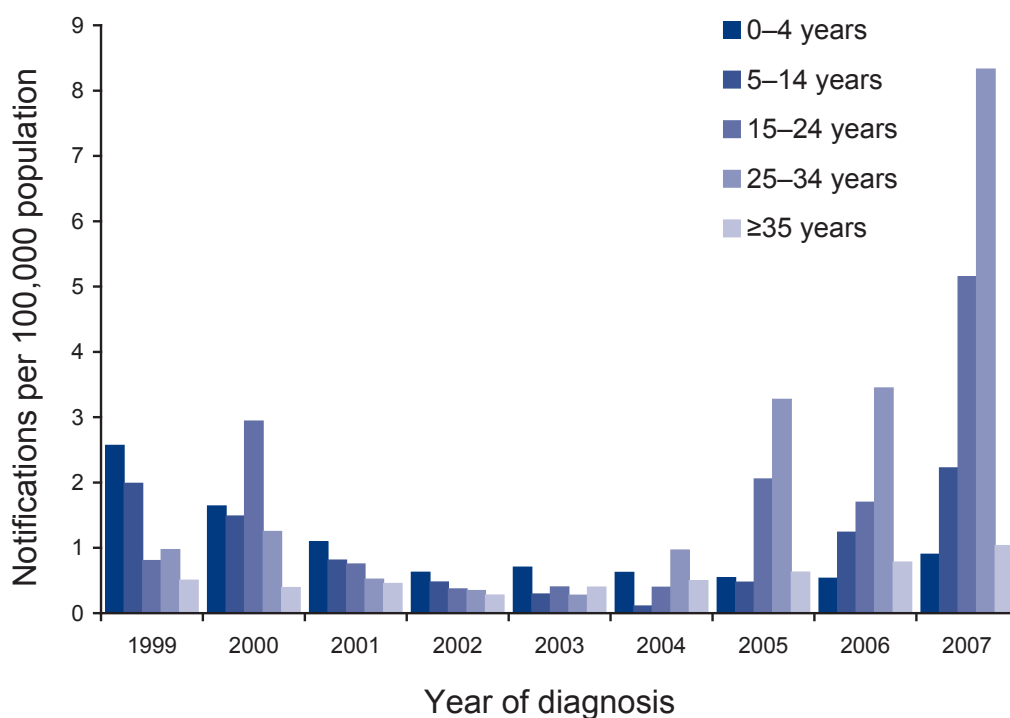
† % of total in the age group.

Age and sex distribution

Since 2002, relatively low notification rates have continued in the 0–4 years and the ≥ 35 years age groups. Since 2005, there have been progressive increases in notification rates for most age groups, compared with 2002–2004, particularly in those aged 15–34 years. The increases in notification rates were most notable in 2007, and affected all age groups (Figure 3.8.2). The majority of notifications in 2006–2007 occurred in the 15–34 years age group. The proportion of the total notifications accounted for by this age group, who constituted approximately 28% of the total Australian population, has increased from 50.4% of the total (211/419) during 2003–2005 to 63.4% of all cases (543/857) in the 2006–2007 period.

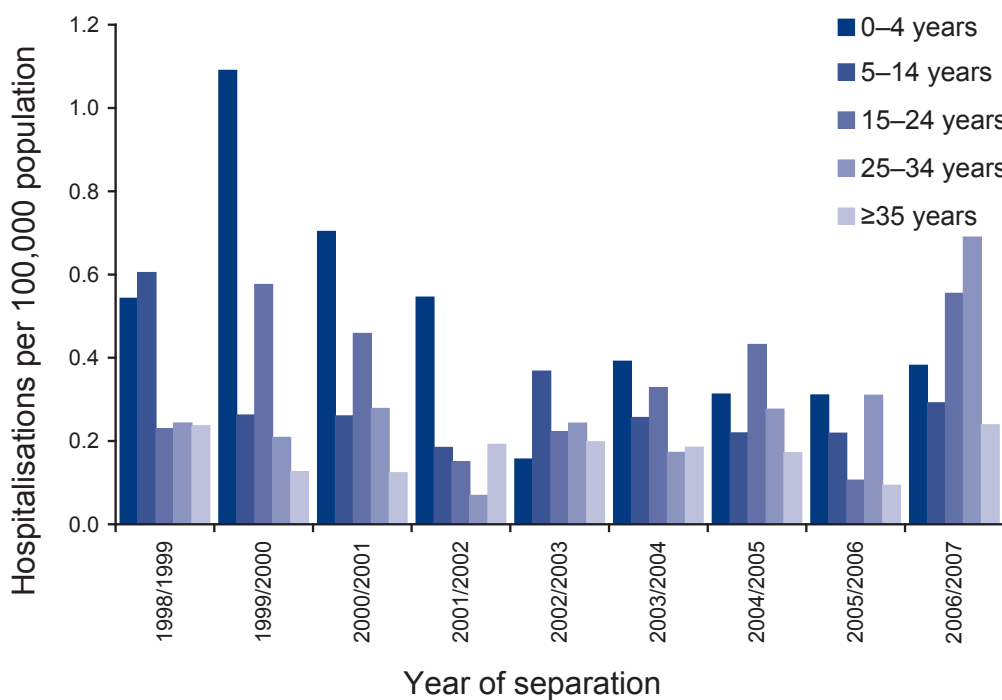
Hospitalisation rates in 2005/2006, compared with the previous 3 years, were lower for those aged 15–24 years and ≥ 35 years, and were comparable among other age groups. However, in 2006/2007, there was an increase in the hospitalisation rates for all age groups, in particular those aged 15–34 years, compared with 2005/2006 (Figure 3.8.3). Over the 2-year period between July 2005 and June 2007, the highest hospitalisation rates were observed in those aged 25–29 years (0.54 per 100,000) and 30–34 years (0.46 per 100,000). The overall male:female ratio was 1.2:1 for notifications and 1.7:1 for hospitalisations over the 2-year review period. The male predominance in notification rates occurred mainly in those aged 20–39 years, with a male:female ratio of 1.3:1 (ranging from 1.2:1 in those aged 25–29 years to 1.6:1 in those aged 20–24 years).

Figure 3.8.2: Mumps notification rates, Australia, 1999 to 2007,* by age group and year of diagnosis



* Notifications where the date of diagnosis was between January 1999 and December 2007.

Figure 3.8.3: Mumps hospitalisation rates, Australia, 1998/1999 to 2006/2007,* by age group and year of separation



* Hospitalisations where the date of separation was between July 1998 and June 2007.

Geographical distribution

The Northern Territory reported a mumps notification rate of 27.0 per 100,000 in 2007, which was dramatically higher than any of the other states or territories over the 2-year period, although it only accounted for 10% of the total notifications in 2007 (58/582). On a lesser scale, Western Australia also experienced a marked increase in the notification rate in 2007 of 5.2 per 100,000 compared with 0.8 per 100,000 in 2006. New South Wales accounted for the bulk of the notifications in 2006–2007 with 478 cases (55.8%), at an increased rate in 2007 (4.7 per 100,000) compared with 2006 (2.3 per 100,000) and 2005 (1.6 per 100,000) (Appendix 6.2).

In Western Australia, the majority of mumps notifications in 2007 were teenage and young adult Aboriginal people living in the Kimberley region, epidemiologically linked to a mumps cluster in the Northern Territory.^{4,5} Among notified mumps cases in Western Australia between July 2007 and June 2008 that occurred or were epidemiologically linked to the Kimberley region (which comprised 84% [153/183] of all notified cases in this state during this period), 92% were Indigenous Australians. Most of these mumps patients (82%; 126/153) were aged 5–29 years, with 22% (34/153) aged 15–19 years. Among the Kimberley Indigenous population, notification rates were highest in those aged 15–19 years, with a rate of 1,816 per 100,000 population.⁵ In the Northern Territory in 2007, despite notifications being found territory-wide, highest notification rates occurred within two geographically defined communities and their outstations, at a remarkable rate of 3,700 per 100,000. Over 90% of notifications in the Northern Territory were in Indigenous people, with the majority occurring in the 15–19 years age group.⁶

Thirty-two notifications of mumps during 2006–2007 were acquired overseas (3.7%). The geographic origin of 76 notifications (8.9%) was unknown.

No cases were hospitalised in the Australian Capital Territory or the Northern Territory during the 2-year review period. The remaining states had similar hospitalisation rates, ranging from 0–0.2 per 100,000 in 2005/2006 and 0.1–0.5 per 100,000 in 2006/2007 (Appendix 6.3).

Vaccination status

During the 2006–2007 period, there were 371 notifications of individuals born after 31 December 1980. Among the 357 cases who were aged ≥ 4 years (who would have received 2 doses of mumps-containing vaccine as per current recommendations), 111 (31%) were reported as ‘fully vaccinated’, 23 (6%) as ‘partially vaccinated for age’ and 102 (29%) as ‘unvaccinated’; vaccination status was unknown or missing for the remaining 121 cases (34%). The vaccination status was validated in only 92 (25%) of these 371 records. Of the 92 cases with vaccination status validated, 72 (78%) had been fully vaccinated, 16 (17%) partially vaccinated, 2 were unvaccinated and 2 had an unknown status.

The Northern Territory examined mumps notifications in the 2007 outbreak using an expanded ‘outbreak’ case definition. Of those in whom immunisation status could be verified, 12/61 (20%) were fully immunised and 31/61 (51%) were partially immunised. It is important to note that the definition for ‘partially immunised’ included those individuals who had received 2 doses of vaccine but with the first dose having been given under 12 months of age (21/31).⁶

Comment

There was a considerable rise in mumps notifications during 2003–2005 and this trend has shown no indication of slowing over the 2006–2007 period; in fact, 2007 provided the largest number of annual notifications since mumps became notifiable in all states and territories in 1996.⁷

While higher rates of other vaccine preventable diseases have frequently been reported in Aboriginal and Torres Strait Islander people, this has rarely been the case for mumps until the dramatic increases in notification rates in the Northern Territory and Western Australia in 2007. This is likely to be multifactorial in origin with factors such as overcrowding and Indigenous-specific immunisation policies (see below).

Once again, almost 90% of notifications could be attributed to adolescents and adults. Those adults aged 20–34 years accounted for over 50% of cases. This finding comes in the light of recent data highlighting that many Australians born in the late 1970s and early 1980s are particularly susceptible to mumps due to not having received 2 doses of the vaccine. The recommendation arising from these data is that adults aged 25–30 years of age should receive a second dose of MMR vaccine at an opportune occasion, such as just prior to overseas travel.⁷

Despite the increase in notifications over 2006–2007, the rates of hospitalisation and documented complications of mumps seem to have remained fairly stable. This is somewhat surprising since severe complications of mumps, such as orchitis, can occur in up to 30% of post-pubertal males with the infection; however, it may be that such cases were managed in the outpatient setting and therefore not documented in hospitalisation data. Another plausible explanation is that mumps cases and the associated complications were never notified because they had not been identified. This is because the clinical case definition for mumps makes parotid or other salivary gland involvement mandatory; yet, paradoxically, 10%–30% of cases of mumps do not have the classical parotitis and therefore would not meet the clinical case definition and would not be reported unless there was laboratory confirmation or an epidemiological link to a confirmed case.¹

Mumps continues to be a problem in other developed nations although notifications appear to be declining in the USA and the UK. In 2006 in the USA, over 5,000 cases of mumps were notified. Similar to Australia, adolescents and young adults were the age group predominantly infected in the USA with the highest age-specific rates occurring in the 18–24 years age group. Many of these were college students. The median age for mumps notifications in the USA in 2006 was 22 years.⁸ There was a marked decline in notifications in the USA in 2007.⁹ Although the reasons for this are not certain, it may partly reflect (particularly at a college level) a combination of fewer susceptible persons as a result of outbreaks over the previous years and increased immunisation among susceptible persons in response to these outbreaks. England and Wales have experienced a decline in mumps notifications in 2006 and 2007 with 4,420 and 1,476 confirmed cases (by laboratory reporting or detection of salivary IgM), respectively, following a peak of >43,300 cases in 2005. Mirroring the trend in Australia and the USA, the bulk of the notifications in England and Wales in 2006 and 2007 occurred in adolescents and young adults aged 15–24 years.¹⁰

Despite the push to vaccinate those young adults who may only be partially immune to mumps, a worrying finding is that mumps cases are occurring even in those individuals who are fully vaccinated. In other words, it is not just an infection of those partially vaccinated or unvaccinated. At least 72 notifications in Australia over 2006–2007 occurred in these fully vaccinated individuals; the number may well be higher but the vaccination status of others was not validated and therefore cannot be reliably assessed. The Northern Territory data for mumps outbreaks in two communities in 2007 raise another important issue, namely the effect of the timing of childhood vaccination on the efficacy of the mumps vaccine. From 1984–1998 in the Northern Territory, Indigenous infants received their first dose of mumps vaccine at the age of 9 months, in contrast to non-Indigenous infants who received their first dose at 12 months of age. This policy was put in place to counter Indigenous children's increased susceptibility to measles.¹¹ In two Northern Territory communities that reported mumps outbreaks in 2007, 21/61 cases in whom the immune status was known occurred in individuals who had received 2 doses of mumps vaccine but with the first dose at <12 months of age. This raises the possibility that immunising infants against mumps with the first dose at age <12 months might have been less effective than immunising them for the first time at 12 months of age. In Western Australia in 2007, almost half of the notifications occurred in those who had been vaccinated twice.⁴ This pattern is not unique to Australia. Examination of mumps cases from Iowa in the USA in 2006 found that 49% of cases had received 2 or more doses of MMR vaccine, 14% had received 1 dose, while 30% had an unknown vaccination status.⁸ Even more strikingly, in a mumps outbreak in 2005–2006 in Czechoslovakia, 87% of 15–19 year old cases had received 2 doses of vaccine.¹² The implication of these findings is that even ensuring that all susceptible individuals receive 2 doses of MMR vaccine is no guarantee of protection from mumps. There are potentially a number of reasons for appropriately vaccinated individuals developing mumps, namely administration of ineffective vaccine initially (e.g. by breaching the cold chain), waning immunity, and failure of cross-neutralisation between vaccine strains and wild-type genotypes.¹

In Australia in 2006–2007, only about 4% of notified mumps cases were imported. This indicates that local transmission of mumps is well established in the community. However, the occurrence of imported cases indicates that mumps can be a travel-related infection, as can measles and meningococcal disease, especially for susceptible adolescents and young adults travelling overseas from Australia. These travellers can, in turn, infect other susceptible individuals on their return to Australia. Furthermore, it may be possible for a fully immunised traveller to be exposed to a strain of mumps overseas that will not induce cross-neutralisation with the local vaccine strain, thereby leading to clinical infection and infectivity.

One case of hospitalisation due to mumps orchitis was reported in a boy aged 0–4 years (Table 3.8.2). Although mumps orchitis is regarded as a manifestation of post-pubertal males, rare occurrences in young children have been reported.¹³

In summary, Australia has seen a large increase in mumps notifications, in 2007 in particular. Mumps has now become a disease primarily affecting adolescents and young adults, although all age groups are still affected. It also now appears that immunisation with 2 doses of childhood vaccine is not a guarantee of immunity, and a booster program targeting at-risk age groups needs to be considered. During late 2007, Indigenous communities in the Northern Territory and Western Australia experienced mumps outbreaks. These factors underline the importance of ongoing surveillance, including the Indigenous status and vaccination status of people reported with mumps, to enable monitoring of incidence in Indigenous populations and vaccine effectiveness in a range of settings.

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3.9 Pertussis

Pertussis (whooping cough), caused by the *Bordetella pertussis* bacterium, is an acute illness, involving the respiratory tract. The typical illness begins with an irritating cough that becomes paroxysmal, lasts for 1–2 months or longer and may be associated with post-tussive vomiting. Paroxysms are characterised by repeated violent coughs, followed by a characteristic crowing or high-pitched inspiratory whoop. Infants less than 6 months old, adolescents and adults often have fewer classical symptoms without paroxysms or whoop.¹

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

National definition from January 2004:²

Both confirmed and probable cases are notifiable.

Confirmed cases are those with definitive laboratory evidence; or laboratory suggestive evidence together with clinical evidence; or clinical evidence together with an established epidemiological link to a confirmed case with laboratory evidence.

- a) Laboratory definitive evidence
 - Isolation of *Bordetella pertussis* from a clinical specimen or detection of *B. pertussis* by nucleic acid testing.
- b) Laboratory suggestive evidence
 - Seroconversion or significant increase in antibody level or 4-fold 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 2 or more weeks; *or*
 - Paroxysms of coughing or inspiratory whoop or post-tussive vomiting.
 - **Probable cases** require clinical evidence only. The clinical evidence required is
 - A coughing illness lasting 2 or more weeks; *AND*
 - Paroxysms of coughing or inspiratory whoop or post-tussive vomiting.

Hospitalisations and deaths

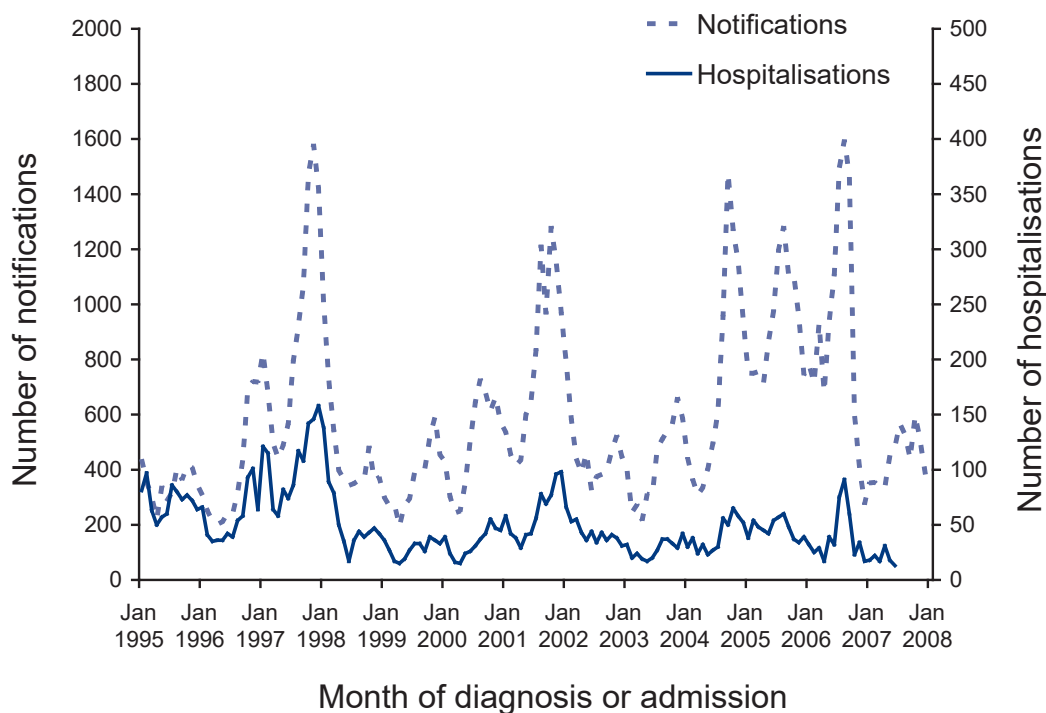
The ICD-10-AM/ICD-10 code A37 (whooping cough) was used to identify hospitalisations and deaths.

Secular trends

There were 16,321 notifications of pertussis received by the NNDSS with dates of onset in the 2 years between January 2006 and December 2007 (Table 3.9.1). Of these 16,321 notifications, 13,880 (85%) were confirmed cases, and 2,441 (15%) were probable cases. A median of 547 cases were notified each month (range 275–1,596). Epidemic peaks have occurred every 3–4 years since national notifications became available in 1991. 2006 was an epidemic year with 10,992 notifications, dropping to 5,329 notifications in 2007. In 2006, the national notification rate was 53.1 per 100,000, the third highest national rate recorded since 1993, after 1997 (58.1 per 100,000) and 2005 (54.9 per 100,000). A clear seasonal pattern was apparent, with the highest number of notifications in the late winter and summer months (between August and February) each year between 1995 and 2007 (Figure 3.9.1).

Hospitalisations followed a similar pattern to notifications. The median number of pertussis hospitalisations per month was 32 (range 13–91) and the average annual national hospitalisation rate was 2.1 per 100,000 for this reporting period, similar to the previous 3 years 2002/2003 to 2004/2005 (2.2 per 100,000).

Figure 3.9.1: Pertussis notifications and hospitalisations, Australia, 1995 to 2007,* by month of diagnosis or admission



Note varying scales between notifications and hospitalisations.

* Notifications where the date of diagnosis was between January 1995 and December 2007; hospitalisations where the date of admission was between January 1995 and June 2007.

Table 3.9.1: Pertussis notifications, hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Notifications 2 years (2006–2007)		Hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission (days)		Deaths 2 years (2005–2006)	
	n	Rate [‡]	n	(§)	Rate [‡]	(§)	Median	(§)	n	Rate [‡]
0–4	596	22.6	340	(284)	13.1	(10.9)	2.0	(2.0)	1	0.04
5–14	765	13.9	31	(19)	0.6	(0.3)	2.0	(2.0)	0	–
15–24	1,503	25.8	20	(10)	0.4	(0.2)	4.0	(2.0)	0	–
25–59	9,145	45.1	227	(130)	1.1	(0.6)	4.0	(4.0)	0	–
60+	4,310	57.5	264	(151)	3.6	(2.1)	7.0	(6.0)	2	0.03
All ages	16,321	39.1	882	(594)	2.1	(1.4)	4.0	(3.0)	3	0.01

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

|| Includes 2 cases with unknown age.

Severe morbidity and mortality

From July 2005 to June 2007, there were 882 episodes of hospitalisation with a pertussis diagnosis (ICD-10-AM code A37) (457 in 2005/2006 and 425 in 2006/2007). The median length of stay per hospital admission was 4 days (Table 3.9.1) and the total number of bed days for the period was 6,214. Of the 882 hospitalisations, 594 (67%) had a principal diagnosis of pertussis (average annual rate 1.4 per 100,000). The discharge diagnosis code A37.0 (*B. pertussis*) was recorded for 439 (50%) hospitalisations and was the principal diagnosis for 298 (68%) of these. *Bordetella parapertussis* (A37.1) was recorded for 9 hospitalisations, and other *Bordetella* species (A37.8) for 16. The remaining 417 (47%) hospitalisations were coded as whooping cough (organism unspecified – A37.9), which was the principal diagnosis in 285 (68%) cases.

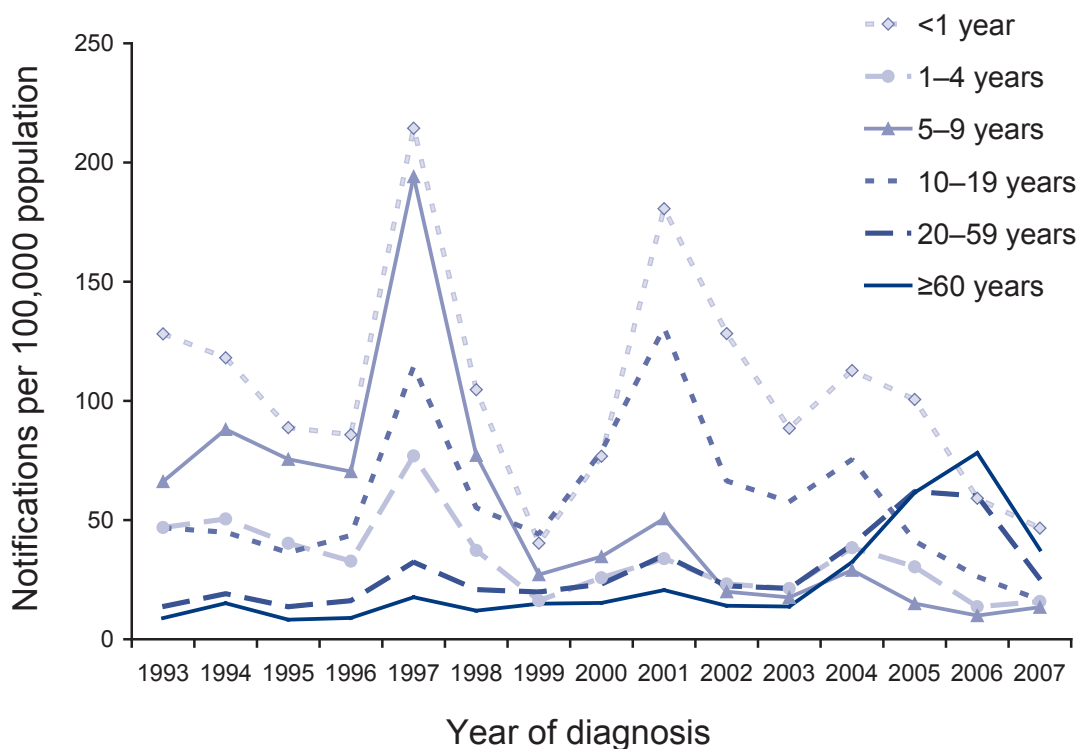
For the 2 years 2005–2006, 3 deaths were recorded where pertussis was the underlying cause (Table 3.9.1). While all the 3 deaths were recorded in 2006, 2 of these 3 recorded deaths in fact occurred in late December 2005, with one 1 month of age and two 93 and 94 years of age. Between 1993 and 2004, there were 18 deaths with pertussis recorded as the underlying cause of death. All but two were younger than 12 months of age; six occurred in 1997.³⁻⁶

Age and sex distribution

The highest notification rates were seen in infants aged <1 year (Figure 3.9.2), with annual average rates of 59.1 per 100,000 in 2006 and 46.6 per 100,000 in 2007. In the 2-year review period, infants aged <1 year accounted for 1.8% of all notifications (n=286) but 34% of hospitalisations (n=300). The average hospitalisation rate for infants was 57.0 per 100,000 in this reporting period compared with 88.1 per 100,000 for the previous 3 years, 2002/2003–2004/2005 (see also Figure 3.9.3).⁶

The lowest notification rates for both years were in the 5–9 years age group. The lowest hospitalisation rates in 2005/2006 and 2006/2007 were in the 5–9 and 10–19 years age groups, respectively.

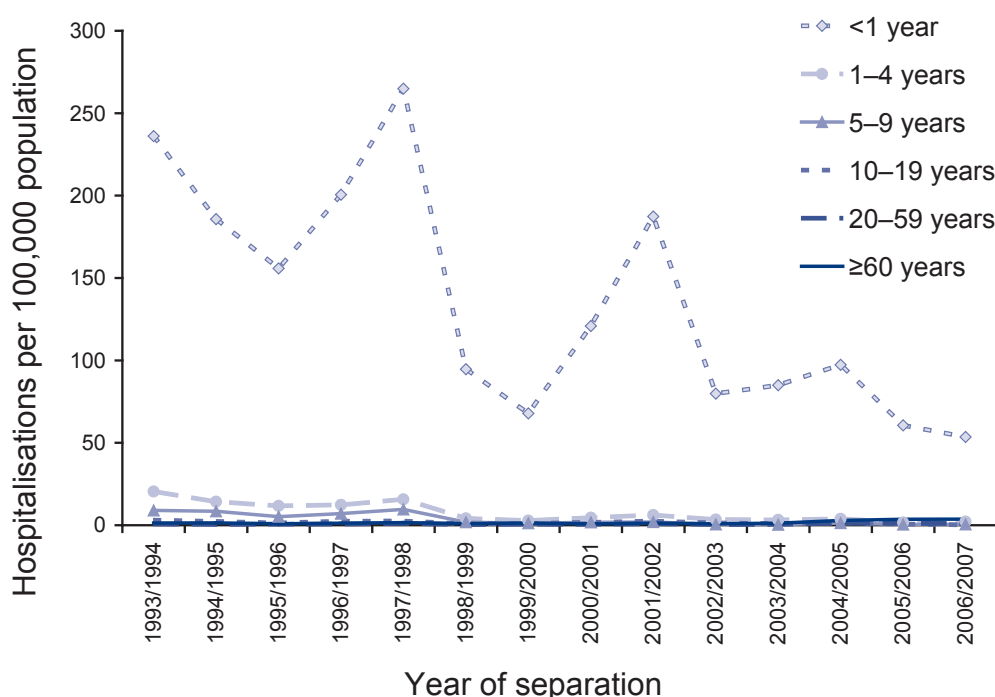
Figure 3.9.2: Pertussis notification rates, Australia, 1993 to 2007,* by age group and year of diagnosis



* Notifications where the date of diagnosis was between January 1993 and December 2007.

The 10–19 years age group accounted for 7.4% of all pertussis notifications in 2006–2007 ($n=1,211$) and 2.7% of all hospitalisations ($n=24$). The 10–19 years age group had very high notification rates in 2003 (57.6 per 100,000) and 2004 (75.4 per 100,000) but rates started declining in 2005 (41.2 per 100,000) (Figure 3.9.2). In 2006 and 2007, there was a further decline in the notification rate for this age group to 26.3 and 16.6 per 100,000, respectively, with the rate remaining below that of people aged 20–59 years and ≥ 60 years (Figure 3.9.2). In both 2006 and 2007, the notification rate was higher in the 15–19 years age group than in the 10–14 years age group (34.1 vs 18.4 per 100,000 and 19.2 vs 13.9 per 100,000, respectively). In 2005/2006, the hospitalisation rate was lower in the 15–19 years age group than in the 10–14 years age group (0.1 vs 1.3 per 100,000), while in 2006/2007 it was 0.3 per 100,000 in the 15–19 years age group with no hospitalisations in the 10–14 years age group.

Figure 3.9.3: Pertussis hospitalisation rates, Australia, 1993/1994 to 2006/2007,* by age group and year of separation

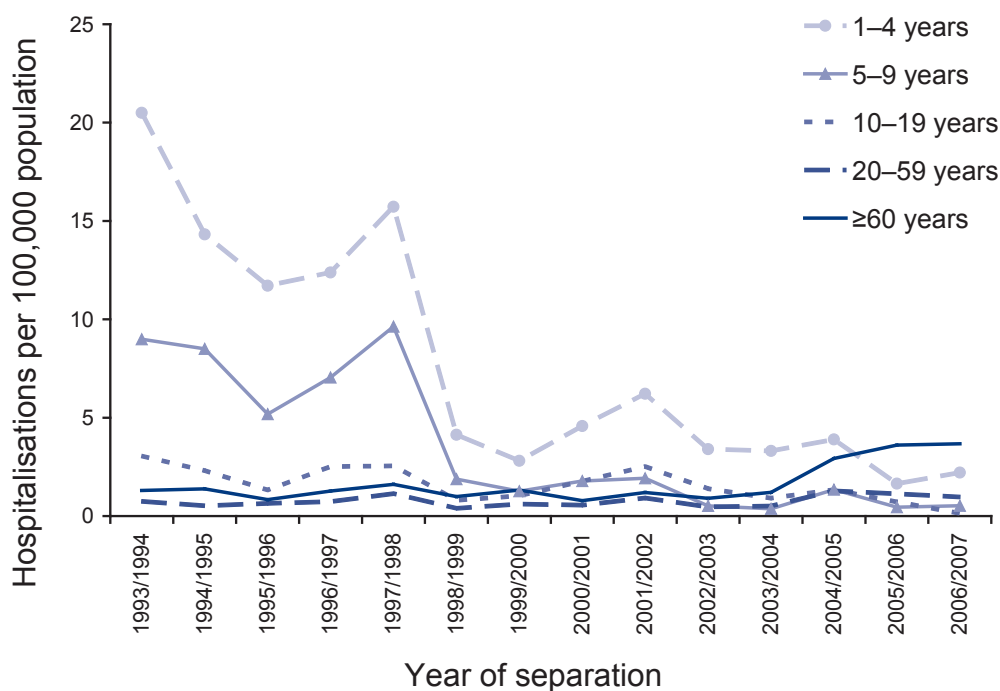


* Hospitalisations where the date of separation was between July 1993 and June 2007.

Adults aged 20–59 years, who constitute approximately 56% of the total Australian population, accounted for 60.6% of notifications ($n=9,888$) and 27.3% of hospitalisations ($n=241$), with an average annual hospitalisation rate of 1.0 per 100,000 compared with 0.8 per 100,000 for the previous 3 years 2003–2005.⁶ The notification rates in all adults aged ≥ 20 years continued to rise in 2006, reaching a record high of 64.4 notifications per 100,000, but dropped substantially to 28.3 notifications per 100,000 in 2007. The proportions of notifications in these age groups have increased from 35%–45% between 1993 and 1998 to 87% in 2006–2007 (Figure 3.9.2). Older persons aged ≥ 60 years, constituting about 18% of the total population, accounted for 26.4% of notifications ($n=4,310$) and 29.9% of hospitalisations ($n=264$). The hospitalisation rate for this age group reached a record high of 3.6 per 100,000 during this reporting period (Figure 3.9.4).

The overall male:female ratio was 1:1.5 for notifications and 1:1.3 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 10–19 years, where the male:female ratio was 1.9:1.

Figure 3.9.4: Pertussis hospitalisation rates, Australia, 1993/1994 to 2006/2007,* by age group (excluding <1 year) and year of separation



* Hospitalisations where the date of separation was between July 1993 and June 2007.

Geographical distribution

Periodic epidemics of pertussis occur in Australia at intervals of 3–4 years. However, the frequency and length of the epidemic cycles vary between jurisdictions, particularly in the less populated and/or geographically isolated regions, such as the Northern Territory, Tasmania and Western Australia. During the period of review, there was a large variation in notification (Appendix 6.2) and hospitalisation rates (Appendix 6.3) between regions and years. South Australia experienced a pertussis epidemic in 2006 with a notification rate of 138.9 per 100,000 population. The Australian Capital Territory (77.2 per 100,000) and New South Wales (72.1 per 100,000) also experienced elevated notification rates in 2006. However, there was a significant drop in notification rates during 2007 across most states and territories.

Vaccination status

Completion of the vaccination status field in NNDSS was expected for all pertussis notifications aged <15 years. For the period 2006–2007, 76% of the notifications aged 0–14 years had information about vaccination status. Missing data on vaccination status varied by age group, ranging from 16% for notifications aged 4–9 years, to 0% for those aged 6–11 months. The percentages of notifications fully and partially vaccinated for age were calculated for children aged <9 years with a known vaccination status. The proportion of cases reported to be fully vaccinated for age rose from 42% in infants <6 months of age to 81% in those aged 1–4 years (Table 3.9.2). Within the 0–5 months age group, 5/83 notifications recorded as ‘fully vaccinated for age’ were in infants <2 months of age who were not expected to have received any vaccine doses.

Comment

Pertussis continues to be one of the most difficult vaccine preventable diseases to control in Australia. Following on from the epidemic period in 2005, the number of cases was again elevated in 2006, with many jurisdictions also having an apparent epidemic. This may be, in part, due to errors in diagnosis as, in October 2006, Australia’s largest distributor of pertussis serology kits announced a major revision in the cut-off for reporting of a positive

Table 3.9.2: Vaccination status of notified pertussis cases aged 0 months to 9 years, Australia, 2006 to 2007,* by age group

Age group	Vaccination status (2006–2007)			
	Fully vaccinated for age		Partially vaccinated for age	
	n	%†	n	%†
0–5 months	83	42	11	6
6–11 months	50	72	3	4
12–47 months	177	81	4	2
4–9 years	217	68	6	2

* Notifications where the date of diagnosis was between January 2006 and December 2007.

† Percentage of those within the age group with a known vaccination status.

test. Subsequent revision resulted in a sharp decline in pertussis notifications in the last months of 2006. This led to concern about the validity of all notifications for 2006. However, re-testing of the majority of specimens during this period was not possible, so validation of the notifications could not be performed. These issues with the specificity of serologic diagnosis primarily affected those aged >20 years.

There has been a downward trend in the notification rate among 10–19 year olds since 2002 and a sharper decline in the rate since 2005 which continued during 2006 and 2007. This is likely to reflect the impact of the adult-formulated diphtheria, tetanus and acellular pertussis vaccine (dTpa), recommended in September 2003 and funded since January 2004 as a booster for adolescents, and improved coverage of the preschool age booster dose of DTPa following its replacement of whole-cell pertussis vaccine. The lower notification rate in 2006–2007 for the 10–14 years age group compared with the 15–19 years age group is probably due to the eligibility for a DTPa preschool booster in the former cohort group. Interpretation of the impact of the adolescent booster within these smaller age groups is complicated by differing delivery among states and territories and over time. As Australian school-based dTpa programs mature, and successive cohorts are vaccinated in future years, pertussis in adolescents would be expected to become well controlled, as occurred in 5–9 year olds following the introduction of the preschool booster.

In this reporting period, pertussis was primarily a problem in two broad age groups: first, infants who had the highest notification and hospitalisation rates, particularly those <4 months of age who are too young to have received 2 or more doses of DTPa; and second, people aged ≥ 20 years, who account for the majority of pertussis notifications, at least in part related to the increased use of serological testing to diagnose pertussis in this age group,⁷ and increased diagnostic awareness of pertussis in adults. Hospitalisations in adults are most likely to be related to complications. Although severe morbidity and mortality are less likely, adults are a significant reservoir of infection and increased circulation of pertussis can facilitate transmission to susceptible infants who are too young to be vaccinated.^{8–11}

The recent increase in the incidence and burden of pertussis notifications in persons aged ≥ 60 years warrants further investigation. As with parents, grandparents are an important source of pertussis transmission to infants.⁸ It is also unclear whether the morbidity of pertussis in older people is greater, or if complications are more likely to occur, but it is of note that 4 of 11 pertussis deaths in the past 7 years have been recorded in people aged ≥ 80 years.⁶

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3.10 Pneumococcal disease

Pneumococcal disease is caused by the bacterium *Streptococcus pneumoniae* (pneumococcus). Pneumococci can be isolated from the upper respiratory tract in children and, less frequently, adults, and can spread directly from the nasopharynx to the respiratory tract which may cause otitis media, sinusitis or pneumonia. Pneumococci are also able to enter the bloodstream to cause invasive disease which may manifest as meningitis, pneumonia, septicaemia without focal infection or, less commonly, infection of other sites such as the pleura, the peritoneum or the joints. Invasive pneumococcal disease (IPD) is diagnosed by detection of *S. pneumoniae* in the blood, cerebrospinal fluid or other sterile sites. In the absence of a sterile site isolate, a presumptive diagnosis of pneumococcal pneumonia may be based on a sputum isolate of *S. pneumoniae* and/or clinical or radiological features, such as characteristic chest x-ray appearance or prompt response to antibiotic therapy.¹

Historically, worldwide and in Australia, IPD has predominantly affected the very young, the very old, indigenous populations, and people with certain chronic diseases or high-risk conditions.^{2,3} In Australia, vaccination with the 23-valent pneumococcal polysaccharide vaccine (23vPPV) has been funded nationally since 1999 for Aboriginal and Torres Strait Islander adults aged ≥ 50 years, and Aboriginal and Torres Strait Islander people aged 15–49 years with high-risk conditions.⁴ A nationally funded vaccination program using the 7-valent pneumococcal conjugate vaccine (7vPCV) commenced in 2001 for all Aboriginal and Torres Strait Islander infants and young children, and non-Indigenous Australian children with certain high-risk conditions.⁵ Since 2005, 7vPCV has been publicly funded for all Australian infants (with a catch-up program for children aged < 2 years), and 23vPPV has been publicly funded for all adults aged ≥ 65 years.⁶

Case definitions

Notifications⁷

Only confirmed IPD cases are notifiable. Notification has occurred since 1995 in the Northern Territory, since 1997 in Queensland and since January 2001 Australia wide. Confirmation of IPD is through:

- a) Isolation of *Streptococcus pneumoniae* from a normally sterile site by culture; 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, these two codes were considered to be a proxy for IPD); 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 lastly, those with neither of these codes but with code J13 were counted as 'pneumococcal pneumonia without meningitis or septicaemia'. A combined category of 'Pneumococcal disease' included all hospitalisations with one or more of these pneumococcal codes.

Deaths

ICD-10 codes G00.1, A40.3 and J13 were used to select deaths attributed to pneumococcal disease.

Secular trends

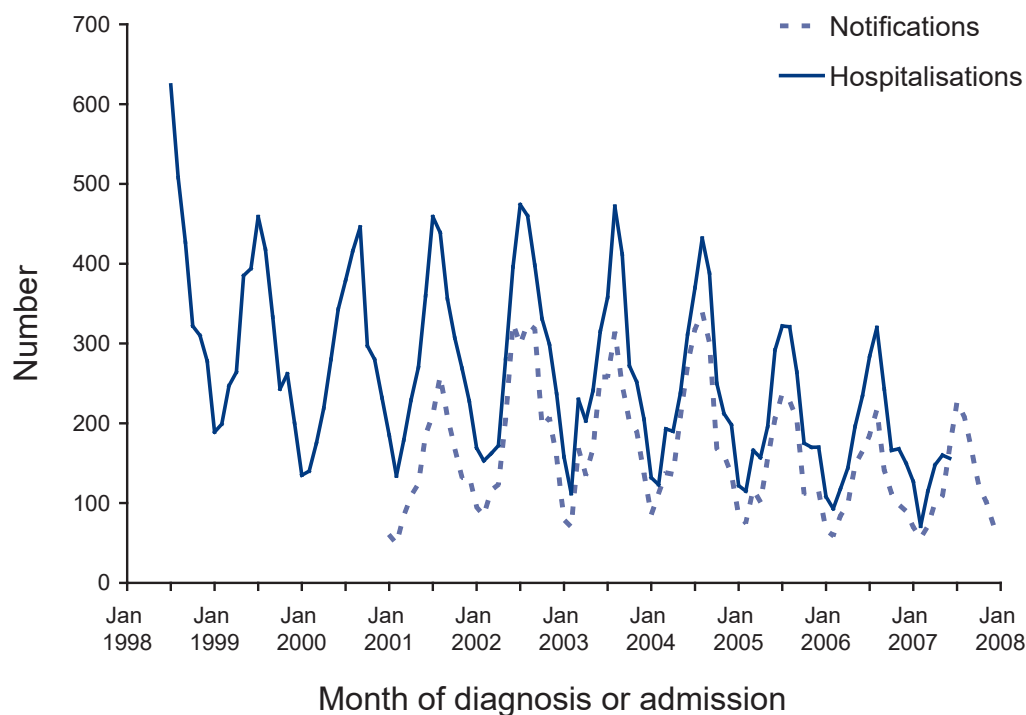
Notification rates and hospitalisation rates for pneumococcal disease decreased in 2006 and 2007 compared with previous years in all age groups. In 2006–2007, there were a total of 2,937 notifications of IPD, 1,464 in 2006 and 1,473 in 2007, with an average annual notification rate of 7.0 per 100,000 for the 2 years (Table 3.10.1). Compared with the average national notification rate in 2002–2004 (11.8 per 100,000 population), there was a 40% decrease in the IPD notification rate in 2006–2007 (Table 3.10.2).

Between July 2005 and June 2007, there were 4,515 hospital separations coded as pneumococcal meningitis, septicaemia or pneumonia, with 2,326 in the 12 months July 2005 to June 2006, and 2,189 in the 12 months July 2006 to June 2007. The average annual rate of hospitalisation was 11.0 per 100,000 (Table 3.10.1). This is a 35% decrease compared with the average annual hospitalisation rate in July 2002–June 2004 (16.8 per 100,000) (Table 3.10.3).

An obvious seasonality with winter peaks was observed for both IPD notifications and hospitalisations for pneumococcal disease during their respective 2-year reporting periods (Figure 3.10.1). This seasonality was similar to that observed in previous years prior to the introduction of the universal infant pneumococcal conjugate vaccine program in 2005. However, both the peak and trough incidences in the post-vaccine introduction period were lower compared with the previous period. The median number of IPD notifications and pneumococcal disease hospitalisations in the winter months (May to October) of 2006, which was within the reporting period of both the notification and hospitalisation data, was 156.5 and 238.5 per month, respectively. This compared with 286.5 per month for notifications and 341 per month for hospitalisations in the winter of 2004. The median number of notifications per month for IPD in the summer months (November to April) of 2005/2006 and 2006/2007 was 87, and that of hospitalisations with a pneumococcal disease code was 136 per month. These compared with 136.5 notifications per month and 191.5 hospitalisations per month for the summer of 2003/2004.

Pneumococcal pneumonia was recorded as the principal diagnosis in 67.6% (2,090/3,092) of hospitalisations where ‘pneumococcal pneumonia without pneumococcal meningitis or septicaemia’ was one of the diagnoses for the hospitalisation. The median number of hospitalisations per month for ‘pneumococcal pneumonia without meningitis or septicaemia’ was 114 (range 54–222), while for pneumococcal meningitis or septicaemia it was 51 (range 17–109). For the months of May–October in 2005 and 2006, the median number of hospitalisations per month for ‘pneumococcal pneumonia without meningitis or septicaemia’ was 168.5, while that for pneumococcal meningitis or septicaemia was 87.5.

Figure 3.10.1: Pneumococcal disease notifications and hospitalisations, Australia, 1998 to 2007,* by month of diagnosis or admission



* Notifications where the date of diagnosis was between January 2001 and December 2007; hospitalisations where the date of admission was between July 1998 and June 2007. Hospitalisations include pneumonia, meningitis and septicaemia.

Severe morbidity and mortality

Between July 2005 and June 2007, a total of 49,945 hospital bed days were recorded for hospital separations with ICD-10-AM codes corresponding to pneumococcal meningitis, septicaemia or pneumonia, with an average of 24,973 hospital bed days per year. The average number of bed days was shorter in this reporting period than the period from July 2002 to June 2005.⁸ The overall median length of stay for hospitalisations coded with any of these codes increased with age (Table 3.10.1). For pneumococcal meningitis, the overall median length of hospital stay was 11 days, compared with 7 days for ‘septicaemia without meningitis’ and 6 days for ‘pneumonia without meningitis or septicaemia’.

Of the 48 reported pneumococcal deaths in 2005–2006 in the AIHW National Mortality Database, the underlying cause of death was recorded as pneumococcal meningitis in 10 (21%), pneumococcal septicaemia in 11 (23%), and pneumococcal pneumonia in 27 (56%) cases. The mortality rate was greatest in adults ≥ 60 years of age (0.36 per 100,000 population) for all sites of infection, followed by children aged < 5 years (0.23 per 100,000 population) (Table 3.10.1). Of all 2,937 IPD notifications reported to NNDSS in 2006 and 2007, death was reported in 249 (8.5%) cases, but whether death eventuated was unknown for 1,093 (37%) cases. For the reporting year 2006, for which both notification data and death registry data in the AIHW National Mortality Database are available, there were 130 deaths among the notified cases recorded in the NNDSS, and 25 deaths recorded in the registry. Two of the 24 recorded deaths that actually occurred in 2006 were in children aged < 2 years and 12 were in people aged ≥ 65 years. Of the 130 IPD deaths reported in NNDSS in 2006, 4 were in children aged < 2 years and 85 were in people aged ≥ 65 years.

Table 3.10.1: Pneumococcal disease notifications, hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Notifications (IPD) 2 years (2006–2007)		Hospitalisations (pneumococcal disease) 2 years (July 2005–June 2007)				LOS [†] per admission (days)		Deaths (pneumococcal disease) 2 years (2005–2006)			
	n	Rate [‡]	n [§]	(M/S)	Rate ^{‡,§}	(M/S) ^{‡,}	Median [§]	(M/S)	n [§]	(M/S)	Rate ^{‡,§}	(M/S) ^{‡,}
0–4	443	16.8	343	(184)	13.2	(7.1)	4.0	(5.0)	6	(5)	0.23	(0.19)
5–14	142	2.6	135	(73)	2.5	(1.3)	4.0	(4.0)	1	(1)	0.02	(0.02)
15–24	120	2.1	158	(41)	2.8	(0.7)	4.0	(5.0)	2	(1)	0.04	(0.02)
25–59	1,042	5.1	1,683	(510)	8.4	(2.5)	6.0	(7.5)	13	(8)	0.06	(0.04)
60+	1,188	15.9	2,196	(615)	30.3	(8.5)	8.0	(9.0)	26	(6)	0.36	(0.08)
All ages	2,937 [¶]	7.0	4,515	(1,423)	11.0	(3.5)	6.0	(8.0)	48	(21)	0.12	(0.05)

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

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

§ Pneumococcal meningitis, septicaemia or pneumonia.

|| M/S = pneumococcal meningitis or septicaemia (proxy for invasive pneumococcal disease).

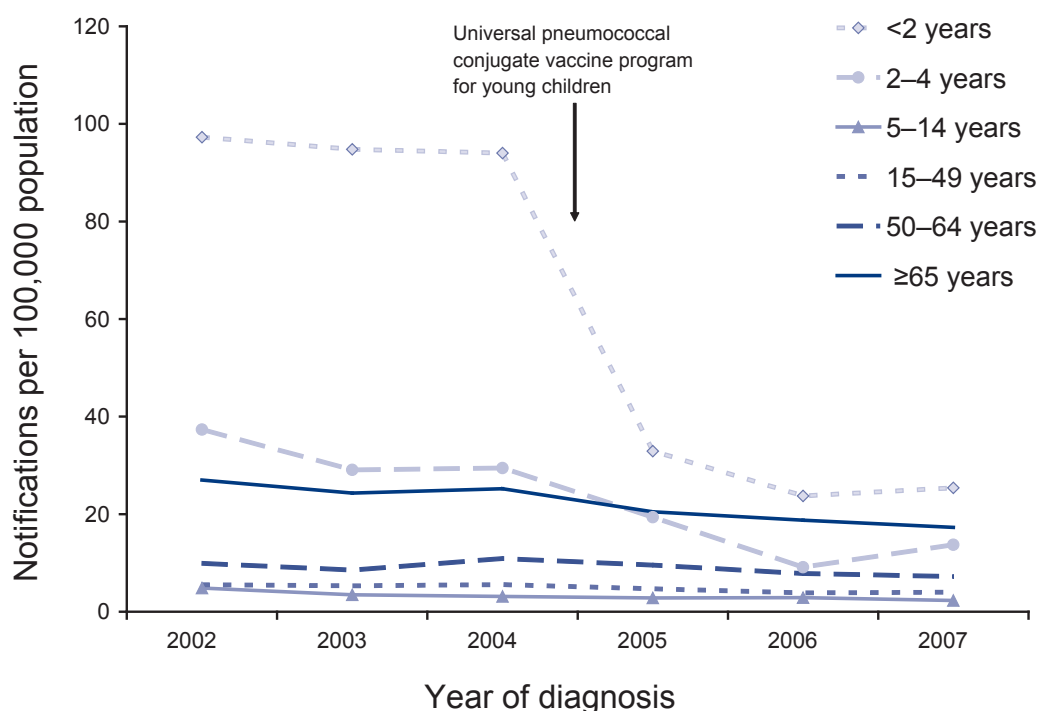
¶ Includes 2 cases with unknown age.

Age and sex distribution

In both 2006 and 2007, the age distribution of notified IPD cases was in line with previous data, with highest notification rates in the elderly and young children (Table 3.10.1). Among adults aged ≥ 65 years, as age increased, so did the notification rate. The highest rates were in those aged ≥ 85 years (33.9 per 100,000 population) compared with those aged 80–84 years (23.1 per 100,000 population) and 75–79 years (19.1 per 100,000 population). Rates were highest in children aged 12–23 months (28.7 per 100,000 population) compared with all other 12-month age groups under 5 years.

Figure 3.10.2 clearly shows the decrease in reported IPD notification rates among children < 2 years of age since introduction of universal 7-valent pneumococcal conjugate vaccination for young children in 2005. Average annual notification rates decreased by 74% between 2002–2004 and 2006–2007 in the age groups targeted for vaccination (children aged < 2 years) (Table 3.10.2). There was a decrease of 64% in the notification rate, to 11.4 per 100,000 population, in children aged 2–4 years, most of whom would also have been eligible for vaccination. In the same period, there were also decreases in notification rates in age groups not targeted by the childhood vaccination program, by 28% in those aged 15–49 years, 23% in those aged 50–64 years, and by 29%, to 18.0 per 100,000 population, in those aged ≥ 65 years.

Between July 2005 and June 2007, hospitalisation rates for pneumococcal disease were greater in adults aged ≥ 60 years (30.3 per 100,000 population) than in children aged < 5 years (13.2 per 100,000 population) (Table 3.10.1). For all ages, the rate of hospitalisation for ‘pneumococcal pneumonia without meningitis or

Figure 3.10.2: Pneumococcal disease notification rates, Australia, 2002 to 2007,* by age group and year of diagnosis


* Notifications where the date of diagnosis was between January 2002 and December 2007.

Table 3.10.2: Average annual notification rates and percentage change in notification rates of invasive pneumococcal disease, Australia, 2006–2007 compared with 2002–2004, by age group

Age group (years)	Average rate 2002–2004*	Average rate 2006–2007†	Change‡ (%)
<2	95.4	24.6	–74
2–4	31.9	11.4	–64
5–14	3.8	2.6	–32
15–49	5.5	3.9	–28
50–64	9.8	7.5	–23
65+	25.5	18.0	–29
All ages	11.8	7.0	–40

* Average age-specific rate per 100,000 population. Notifications where the date of diagnosis was between January 2002 and December 2004.

† Average age-specific rate per 100,000 population. Notifications where the date of diagnosis was between January 2006 and December 2007.

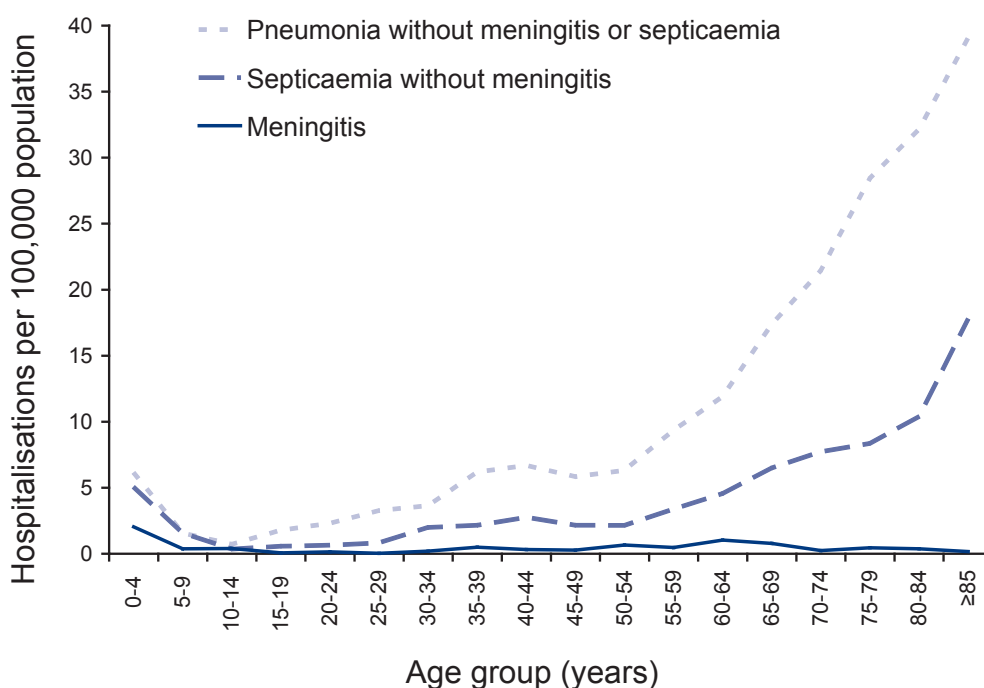
‡ Percentage change in the average notification rate of 2006–2007 compared with the average notification rate of 2002–2004.

septicaemia' was greater than for pneumococcal meningitis or pneumococcal septicaemia without meningitis (Figure 3.10.3). For pneumonia (without meningitis or septicaemia) and septicaemia (without meningitis), there was a small peak among children aged <5 years, and a larger peak in the elderly age groups. The hospitalisation rate for 'pneumonia without meningitis or septicaemia' peaked at 39.1 hospitalisations per 100,000 population in the ≥85 years age group, and the peak for 'septicaemia without meningitis' was 17.8 per 100,000 population in the same age group. In contrast, the age distribution of hospitalisation rates for pneumococcal meningitis showed a peak only among young children, with the highest rate in children aged <5 years (2.0 per 100,000 population) (Figure 3.10.3).

Average hospitalisation rates of pneumococcal disease in all age groups decreased in the period July 2005 to June 2007 compared with the period July 2002 to June 2004. There were decreases of 72% for those aged <2 years, 58% for those aged 2–4 years and 33% for those aged 50–64 years (Table 3.10.3). During the period July 2005 to June 2007, the ≥65 years age group had the highest hospitalisation rate (34.9 hospitalisations per 100,000). For hospitalisation rates of pneumococcal ‘pneumonia without meningitis or septicaemia’, the proportional change between July 2005–June 2007 and July 2002–June 2004, as well as the relative age group distribution, were similar to those of hospitalisations for pneumococcal disease (Table 3.10.3).

For the 2-year reporting periods, both IPD notification and pneumococcal disease hospitalisation rates were higher in males than in females for most age groups and overall (male:female ratio 1.3:1 for both notification and hospitalisation rates).

Figure 3.10.3: Pneumococcal meningitis, septicaemia and pneumonia hospitalisation rates, Australia, 2005/2006 to 2006/2007,* by age group



* Hospitalisations where the date of separation was between July 2005 and June 2007.

Geographical distribution

The average annual notification rate of IPD of 28.7 per 100,000 population in the Northern Territory in 2006–2007 was about 4 times that of the national rate (7.0 per 100,000) and the highest rate in any jurisdiction, as was the case in previous years. The average annual notification rates ranged from 5.4 to 8.0 in other jurisdictions in 2006–2007 (Appendix 6.2).

A similar distribution was seen in the hospitalisation rates. In 2005/2006–2006/2007, the average annual hospitalisation rate for pneumococcal disease (pneumococcal meningitis, septicaemia or pneumonia) of 42.4 per 100,000 in the Northern Territory was 3.9 times that of the national rate of 11.0 per 100,000 population. Hospitalisation rates in other jurisdictions ranged between 8.2 and 11.8 per 100,000 population. In the same period, the hospitalisation rate for pneumococcal pneumonia without meningitis or septicaemia in the Northern Territory (24.2 per 100,000) was 3.2 times that of the national average rate (7.5 per 100,000; range 5.9–8.0 per 100,000 for other jurisdictions). The average annual hospitalisation rate for pneumococcal meningitis or septicaemia (a proxy for IPD) in the Northern Territory (18.2 per 100,000) was about 5 times that of the national rate (3.5 per 100,000), while that of other jurisdictions ranged between 2.4 and 3.8 per 100,000 (Appendix 6.3).

Table 3.10.3: Average annual hospitalisation rates and percentage change in hospitalisation rates for pneumococcal disease and for pneumococcal pneumonia without meningitis or septicaemia, Australia, 2005/2006–2006/2007 compared with 2002/2003–2003/2004, by age group

Age group (years)	Average rate 2002/2003–2003/2004*		Average rate 2005/2006–2006/2007†		Change‡ (%)	
	Pneumococcal disease§	Pneumococcal pneumonia	Pneumococcal disease§	Pneumococcal pneumonia	Pneumococcal disease§	Pneumococcal pneumonia
<2	71.8	26.0	20.1	8.3	–72	–68
2–4	20.4	10.3	8.6	4.6	–58	–55
5–14	4.0	2.4	2.5	1.1	–38	–54
15–49	8.5	6.3	6.1	4.3	–27	–32
50–64	19.0	13.8	12.8	8.9	–33	–36
65+	49.1	35.7	34.9	25.4	–29	–29
All ages	16.8	11.4	11.0	7.5	–35	–34

* Average age-specific rate per 100,000 population. Hospitalisations where the date of separation was between July 2002 and June 2004.

† Average age-specific rate per 100,000 population. Hospitalisations where the date of separation was between July 2005 and June 2007.

‡ Percentage change in the average hospitalisation rate of 2005/2006–2006/2007 compared with the average hospitalisation rate of 2002/2003–2003/2004.

§ Pneumococcal meningitis, septicaemia and/or pneumonia

|| Pneumococcal pneumonia without meningitis or septicaemia.

Pneumococcal typing

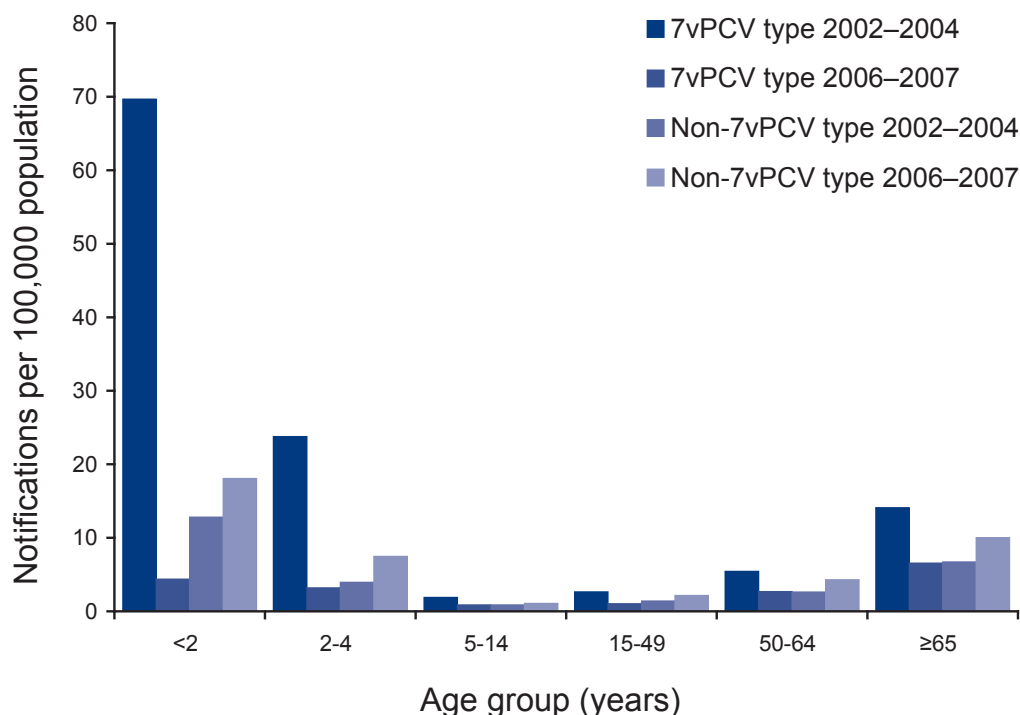
Data on the pneumococcal serotypes of notified IPD cases during 2006–2007 were available for 89% (2,601/2,937) of all notified cases, compared with 81% (5,722/7,050) of all notified cases during 2002–2004, the 3 years prior to introduction of the universal 7vPCV program. Of the 335 notifications in 2006–2007 with unknown or missing serotype information, the majority (222/335, 66%) were aged 5–64 years, the age groups where enhanced surveillance for IPD notifications was not implemented in some jurisdictions. The proportion of notified cases in 2006–2007 with unknown or missing serotype information in those aged <5 years, 5–64 years and ≥65 years were 7.9%, 14.7% and 11.5%, respectively.

Thirty-five per cent (918) of all serotyped cases during 2006–2007 were caused by serotypes contained in the 7vPCV, compared with 73% in 2002–2004. During 2006–2007, among children targeted by the program (i.e. aged <2 years), 20% (47/241) of serotyped cases were caused by serotypes in the 7vPCV compared with 85% (1,049/1,242) in 2002–2004.⁸

The notification rate of IPD caused by 7vPCV serotypes in 2006–2007 decreased overall by 68%, to 2.2 per 100,000, when compared with that in 2002–2004. The rate of IPD caused by 7vPCV serotypes decreased in age groups targeted by the vaccination program, as well as in groups not targeted by the program, but the decrease was greatest among children <5 years of age. Among children <2 years of age, there was a 94% decrease (from 69.7 per 100,000 population to 4.4 per 100,000 population) (Figure 3.10.4, Table 3.10.4).

For most age groups, in 2006–2007 compared with 2002–2004, there were increases in notification rates of IPD caused by serotypes that are contained in the 23vPPV but not in the 7vPCV (23v-non-7v serotypes), and serotypes not included in either vaccine (non-23vPPV serotypes) (Table 3.10.4). The percentage increases in average annual notification rates caused by serotypes not contained in the 7vPCV ranged between 24% and 89% among different age groups (Table 3.10.4). The greatest absolute increases in the average annual notification rates of IPD caused by non-7vPCV serotypes were seen in children aged <2 years, children aged 2–4 years, and adults aged ≥65 years (Figure 3.10.4). The increases in rates were 5.2, 3.5 and 3.3 cases per 100,000 population, respectively. Overall, the increase in non-23vPPV serotypes was less than that in 23v-non-7v serotypes (Table 3.10.4).

Figure 3.10.4: Notification rates of IPD cases with serotypes contained in the 7-valent pneumococcal conjugate vaccine (7vPCV),* versus notification rates for other non-7-valent serotypes,† Australia, 2006–2007 compared with 2002–2004, by age group



* Serotypes contained in the 7-valent pneumococcal conjugate vaccine: 4, 6B, 9V, 14, 18C, 19F, 23F.

† All IPD cases with known serotypes and age caused by serotypes not contained in the 7-valent pneumococcal conjugate vaccine.

Table 3.10.4: Percentage change in the average annual notification rates* of invasive pneumococcal disease cases, Australia, 2006–2007 compared with 2002–2004, by age group and serotype category

Age group (years)	Serotype category				
	7v† (%)	23v-non-7v‡ (%)	Non-23v§ (%)	All non-7v combined	Serotypes unknown/missing
<2	–94	+71	–10	+41	–83
2–4	–87	+131	+29	+89	–82
5–14	–54	+55	–46	+24	–40
15–49	–60	+65	+22	+53	–50
50–64	–51	+56	+93	+63	–68
65+	–53	+46	+57	+49	–69
All ages	–68	+62	+36	+55	–64

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

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

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

§ Serotypes not contained in either vaccine.

|| All other serotypes except the 7 serotypes contained in the 7-valent pneumococcal conjugate vaccine.

In children <5 years of age the predominant 23v-non-7v serotype in 2006–2007 was 19A (28.9% of IPD notifications, average annual rate 4.8 per 100,000 population), and the predominant non-23vPPV serotype was 6A (6.5%, average annual rate 1.1 per 100,000 population). The notification rate for serotype 19A in 2006–2007 was 2.3 times that in 2002–2004, but the notification rate for serotype 6A decreased by 37% in 2006–2007 compared with 2002–2004.

For adults aged ≥ 65 years, the rate of IPD caused by 7vPCV serotypes decreased by 53% in 2006–2007 compared with 2002–2004, while 23v-non-7v and non-23vPPV serotypes increased, by 46% and 57%, respectively (Table 3.10.4). In those aged ≥ 65 years there was a broader distribution of serotypes, with the predominant 23v-non-7v serotypes being serotype 3 (9.5% of IPD notifications), 19A (7.8%) and 22F (8.3%), while 6A was again the predominant non-23vPPV serotype (7.1%). Notification rates of these serotypes increased between 2002–2004 and 2006–2007 by 35%, 95%, 55% and 16%, respectively.

Vaccination status

From 1 January 2005, children born from 1 January 2003 were eligible for a full course of the funded 7vPCV and catch-up programs. Of 336 eligible children aged >6 months at disease onset and reported to NNDSS in 2006–2007 as IPD cases, data on vaccination status were available for 281 (84%). Of those, 220 (78% of 281) were reported to be fully vaccinated, and this was validated by vaccination records for 215 (98% of 220). Of the 220 fully vaccinated cases, 22 (10%) had 7vPCV serotypes, 143 (65%) had 23vPPV serotypes and 37 (17%) had other non-23vPPV serotypes. The vaccination status was validated by written confirmation for all (22 of 22) 7vPCV serotype cases, 139 (97% of 143) 23vPPV serotype cases and 36 (97% of 37) of the other non-23vPPV serotype cases. A status of being partially vaccinated was reported for 34 (12% of 281) cases, and 27 (10% of 281) were reported to be unvaccinated.

Among notified IPD cases aged ≥ 65 years in 2006–2007, vaccination status was reported for 563 of 981 cases (57%). Twenty-six per cent (253/981) were reported as fully vaccinated, 6% (57/981) as partially vaccinated and 26% (253/981) as unvaccinated, while vaccination status information was not available for 418 (43%).

Comment

The substantial impact of the universal 7vPCV program on Australian hospitalisation and notification rates of pneumococcal disease is highlighted in this report. Between 2002–2004 and 2006–2007, there were decreases in the overall average annual notification rate (from 11.8 to 7.0 per 100,000 population), notification rates in all age groups, and notification rates of IPD caused by 7vPCV serotypes. Hospitalisation data from 2005/2006–2006/2007, compared with previous years, showed decreased overall average annual rates (from 16.8 per 100,000 in 2002/2003–2003/2004 to 11.0 per 100,000), and decreased rates in all age groups, and for the various clinical manifestations of pneumococcal disease.

Reduction in the IPD notification rate is most pronounced in the target age group of the universal 7vPCV program, those <2 years of age, especially with respect to IPD caused by 7vPCV serotypes. As has been observed in other countries,^{9–11} the 7vPCV vaccination program has had a protective effect on unimmunised as well as immunised age groups in Australia, with decreases in notification and hospitalisation rates observed in age groups both targeted and not targeted by the 7vPCV program. This effect is due to herd immunity mediated by the universal 7vPCV program and subsequent reductions in carriage of pneumococci^{12,13} and in transmissions to the unvaccinated.¹⁰ In addition to herd immunity effects, it is possible that catch-up vaccination with 7vPCV in children aged <2 years when the 7vPCV program was initially implemented in 2005 would have contributed to decreased IPD rates in children aged 2–4 years in 2006–2007.

Concerns about replacement disease by serotypes not contained in the 7-valent vaccine have been expressed overseas¹⁴ and in Australia.^{15–17} In the USA, increases in non-7-valent serotypes have been documented post implementation of the national vaccination program.^{9,18} However, overall in the USA, the decreased disease rates attributable to 7vPCV outweigh the smaller increases in non-vaccine serotypes.¹⁰ An exception to this is one region of Alaska, where increases in serotype 19A have been greater than the decreases in 7vPCV serotypes, resulting in a net increase in total IPD rates in Alaskan Natives after implementation of their vaccination program.¹⁹ In the data presented here and also reported from Victoria,¹⁶ the increases in notifications of non-vaccine serotypes in Australia to date appear similar to the situation seen in the general US population. Continued monitoring of this situation is clearly important.

The 23vPPV has been shown to be effective against IPD in older adults who did not have high prevalence of risk factors,^{20–22} but its effectiveness may be poorer in populations with chronic disease or high-risk conditions,²² including Indigenous populations.²³ The effectiveness of the 23vPPV against nasopharyngeal carriage and pneumococcal pneumonia has not been established.²² The decreases seen in hospitalisation and notification rates in those aged ≥ 65 years in 2006–2007, compared with 2002–2004, are largely due to decreases in IPD caused by serotypes contained in the 7vPCV, implying a herd immunity effect. The proportion of the decrease attributable to universal adult 23vPPV vaccination is not clear. In this age group, there were increased rates of IPD caused by both non-23vPPV serotypes and 23v-non-7v serotypes. The increase in rates of IPD caused by non-23vPPV serotypes may reflect the extent of ‘natural’ increase due to serotype replacement in the community after implementation of the 7vPCV program. The comparatively smaller increase in the rate of IPD caused by non-23vPPV serotypes in this age group suggests that, overall, there might be a modest protective effect achieved by the 23vPPV in those aged ≥ 65 years.

This report documents lower hospitalisation rates after the implementation of the universal 7vPCV program in Australia, shorter lengths of hospital stays, and fewer deaths recorded as due to pneumococcal meningitis, septicaemia or pneumonia. Overall, hospitalisation rates for pneumococcal disease (including pneumococcal pneumonia without meningitis or septicaemia) were higher than IPD notification rates. The hospitalisation codes used to identify pneumococcal disease were less specific than the case definition used for notification data (which only includes invasive disease), as they include pneumococcal pneumonia without bacteraemia, and microbiological detection of *S. pneumoniae* is not mandatory for assigning diagnosis in the hospitalisation data. As expected, hospitalisation rates for pneumococcal meningitis or septicaemia, used as a proxy indicator for IPD, were lower than IPD notification rates for all age groups. In addition to protection against IPD, 7vPCV may protect against other pneumococcal infections including non-bacteraemic pneumococcal pneumonia and otitis media.^{14,24,25} The observed decreases in the rate of pneumococcal pneumonia hospitalisation are a potential indicator of the vaccine’s effectiveness against non-invasive pneumococcal infection.

The total number of pneumococcal disease deaths reported to the AIHW National Mortality Database halved in 2005–2006 compared with the previous 2 years. The age distribution of the deaths was unchanged, with the majority occurring in the elderly. Even though these deaths reported to the registry included the broader category of all pneumococcal pneumonia, in 2006 there were 5.4 times as many pneumococcal deaths reported through NNDSS as through registry reports (which are based only on the underlying cause of death, not including other contributing causes of death). This suggests that pneumococcal pneumonia as the underlying cause of death is under-reported in registry data. The crude case-fatality rate from the notification data in NNDSS (8.5%) falls in the range of case-fatality rates reported from elsewhere in the vaccine era; between 5% and 27%.^{11,26} However, the validity of outcome data in NNDSS should be interpreted with caution because of poor completeness.

Higher rates of IPD have been reported in Aboriginal and Torres Strait Islander young children and adults than in non-Indigenous Australians in several regions of Australia.^{27–29} The completeness of identification of Indigenous status in the national notification and hospitalisation datasets is highly variable across different jurisdictions, in particular among age groups that are not subjected to enhanced IPD surveillance in some jurisdictions. This limits the usefulness of epidemiological analysis of national data on pneumococcal disease by Indigenous status. A report on notification and hospitalisation data among Aboriginal and Torres Strait Islander people by various age groups, from selected jurisdictions with acceptable completeness of Indigenous status identification in records over the period 2003–2006, has been published.⁴ While a detailed analysis of IPD in Indigenous people is not the subject of this chapter, previous analyses have shown substantial reductions in 7vPCV-type IPD, but continuing high rates of non-vaccine type IPD, similar to levels seen prior to widespread vaccination.^{4,15} Serotypes causing infection in indigenous populations worldwide have been more diverse than those causing infection in non-indigenous populations, meaning that there is less protection provided by currently available conjugate vaccines for indigenous people.³ Conjugate vaccines with extended serotype valencies or novel protein-based pneumococcal vaccines (which are under preliminary development) may potentially contribute to achieving further reductions in disease, particularly in Indigenous Australians.

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3.11 Poliomyelitis

Poliomyelitis (polio) is caused by an enterovirus, poliovirus. Infection involves the gastrointestinal tract, and may progress to the central nervous system, resulting in paralysis. Acute flaccid paralysis (AFP) occurs in less than 1% of infections. More than 90% of infections are asymptomatic or are associated with a non-specific fever. About 10% are associated with a minor illness characterised by fever, headache, malaise, nausea and vomiting. Paralysis is usually asymmetric, the maximum extent of which is usually reached within 3–4 days of disease onset. Any paralysis still present after 60 days is likely to be permanent.¹ Although wild poliovirus transmission has ceased in the majority of countries in the world, the threat of importation to Australia continues.

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 (>1 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).²

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

National definition from January 2004:³

Both confirmed and probable cases are notifiable. 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: 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

There was 1 confirmed notification of acute polio in a 22-year old male student from Pakistan in July 2007, who acquired wild-type poliovirus (type 1) infection while visiting his family in Pakistan.^{4–6} He was hospitalised upon his return to Melbourne but, as his hospital separation date was after the end of June 2007, he is not included in the present analysis of hospitalisation data. From July 2005 to June 2007, there were 8 hospitalisations with a recorded diagnosis of acute poliomyelitis. Of these, 6 were coded as acute unspecified poliomyelitis (A80.9), 1 as acute paralytic poliomyelitis (other and unspecified) (A80.3) and 1 as acute non-paralytic poliomyelitis (A80.4). This latter hospitalisation was of a young male who was the only case where poliomyelitis was coded as the principal diagnosis. Six of the 8 separations recorded as acute poliomyelitis were among persons aged ≥50 years. No hospitalisations were recorded in those aged <15 years. There were no deaths coded as due to poliomyelitis in 2005 or 2006.

Comment

Although Australia and the Western Pacific Region were declared polio-free in October 2000,⁷ the imported case of polio notified in 2007 is a reminder that, unless global polio eradication is achieved, ongoing vigilance in polio surveillance and maintenance of high levels of vaccination coverage are critical in maintaining successes in polio control achieved to date. 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 infection due to indigenous wild-type poliovirus. Some hospitalisations could represent cases of AFP where polio could not be excluded, but most are likely to be adults with late effects of polio rather than acute cases, as indicated by the age distribution of the hospitalisations. However, imported cases of polio can include persons of any age, as exemplified by the 2007 importation event involving a young adult who had been fully immunised as a child.⁶ The current hospitalisation data suggest an ongoing improvement in coding practices compared to earlier periods where there were many more hospitalisations in older age groups miscoded as acute poliomyelitis.

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 poliovirus infection, cases of VAPP, and outbreaks of circulating vaccine-derived polioviruses. In Australia, surveillance of AFP in children <15 years of age is coordinated through the Victorian Infectious Diseases Reference Laboratory in collaboration with the Australian Paediatric Surveillance Unit. AFP cases are notified and stool specimens are referred to the Australian National Poliovirus Reference Laboratory for testing for polioviruses and other enteroviruses. Cases are referred to the Polio Expert Committee for a determination as to the cause of the AFP. In 2006, 48 eligible cases were received and 43 of these had sufficient information available for review and were classified as non-polio AFP.⁸ In 2007, there were 27 eligible cases and 26 were classified as non-polio AFP. The remaining case could not be discarded as non-polio AFP based on the available information and was reported to WHO as polio-compatible.⁹ The onset date of this case was not consistent with the case of hospitalisation for which the principal diagnosis was classified as acute non-paralytic poliomyelitis.

The WHO target for surveillance of AFP in a polio-free country (1 notified case of non-polio AFP per 100,000 children aged <15 years) has only been intermittently achieved in Australia (in 2000, 2001, 2004 and 2006). The rates in 2006 and 2007 were 1.1 and 0.65 per 100,000, respectively.^{8,9} The WHO target of faecal sampling from 80% of AFP cases has never been achieved, with the 52% sampling proportion achieved in 2007 the highest to date (previously 19%–36%, with a proportion of 21% in 2006).^{8–11}

The global aim to eradicate polio by 2000 has proven elusive, prompting ongoing discussion and debate about the feasibility and desirability of elimination.^{12,13} In 2006 and 2007, endemic transmission of wild-type poliovirus remained constrained to four countries (Afghanistan, India, Nigeria and Pakistan) and the strategies used to control outbreaks following importations in 2005 seemed successful, with a dramatic fall in these cases. There were 1,997 and 1,315 wild poliovirus cases reported to WHO in 2006 and 2007, respectively.¹⁴ A total of 12 countries reported cases caused by wild poliovirus in 2007. The Global Polio Eradication Initiative Strategic Plan 2009–2013 will have five main objectives (interrupt wild poliovirus transmission; ensure sustainable surveillance for polioviruses; achieve certification and containment of wild polioviruses; prepare for VAPP and VDPV elimination and the post-OPV era; plan for restructuring of the Global Polio Eradication Initiative for the VAPP/VDPV Elimination Phase).¹⁵ Updates on the global polio situation are available at the Global Polio Eradication Initiative website (www.polioeradication.org).

In November 2005, inactivated poliovirus vaccine (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. With the replacement of OPV with IPV in Australia, incidental detection of polioviruses in faecal specimens should no longer occur, with the last routine isolation of Sabin-like polioviruses from submitted specimens occurring in 2006.⁸ Future poliovirus isolations will, therefore, require full investigation.¹¹ 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 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.¹⁶ Travellers should be reminded to ensure that they are vaccinated against polio.¹⁷

In 2008, the Australian Government Department of Health and Ageing released *An acute flaccid paralysis and poliomyelitis response plan for Australia*.¹⁸ The plan outlines the routine surveillance procedures for AFP cases in Australia, focusing on children <15 years of age. The plan also acts as a guide for the investigation of and response to a case of polio in a person of any age.

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3.12 Q fever

Q fever is a zoonotic disease caused by *Coxiella burnetii*. It has been identified in a wide range of wild and domestic animal hosts, including arthropods, birds, rodents, marsupials and livestock, but the most important reservoirs as sources for human infections are cattle, sheep and goats. *C. burnetii* can withstand harsh environmental conditions including desiccation, and are shed in the urine, faeces, milk and particularly birth products of infected animals. Humans become infected primarily by inhaling aerosols contaminated by *C. burnetii*. Occupations with higher exposure risks include abattoir and farm workers and veterinarians. Windborne spread and indirect exposures in a contaminated environment account for non-occupational infections.^{1,2}

Q fever may present with acute or chronic clinical manifestations, and there is increasing acceptance of an association with long-term sequelae, in particular the post Q fever fatigue syndrome.^{1,3,4} A high proportion of infected persons are asymptomatic or only experience a self-limiting febrile illness. Although Q fever infections generally respond to antibiotic treatment, they may result in significant morbidity, including pneumonitis, hepatitis, myocarditis, chronic endocarditis, and occasional fatality. Recrudescence may occur, especially in pregnant or immunocompromised persons.^{1,2}

A whole-cell formalin-inactivated vaccine against Q fever, the Q-VAX™ developed in Australia, has been available since 1989. In the early 1990s, 90%–95% of the vaccine produced was purchased by abattoirs, but the usage was low before the manufacturer began to actively promote the vaccine in 1994.⁵ A National Q Fever Management Program, which was funded by the Australian Government, was implemented from 2001 in Queensland, South Australia, Victoria and Western Australia, and from 2002 in the Australian Capital Territory, New South Wales and Tasmania. (The Northern Territory opted out of the program as, until 2002, there had been no cases of Q fever recorded since 1991.⁶) The program consisted of systematic delivery of a subsidised Q fever vaccination service to targeted populations with a high risk of environmental exposure who did not show evidence of pre-existing immunity to Q fever (identified by clinical and laboratory screening tests). The program was delivered in two phases. Phase 1 targeted abattoir workers, workers in the meat and livestock industry, and sheep shearers. Phase 2 of the program expanded to include sheep, dairy and beef cattle farmers, their employees, and family members working on farms. The national program was concluded at different times between June 2004 and December 2006 in different jurisdictions.⁷

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

National definition from January 2004:⁸

Only confirmed cases are notifiable. Confirmed cases require either laboratory definitive evidence, or laboratory suggestive evidence together with 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.

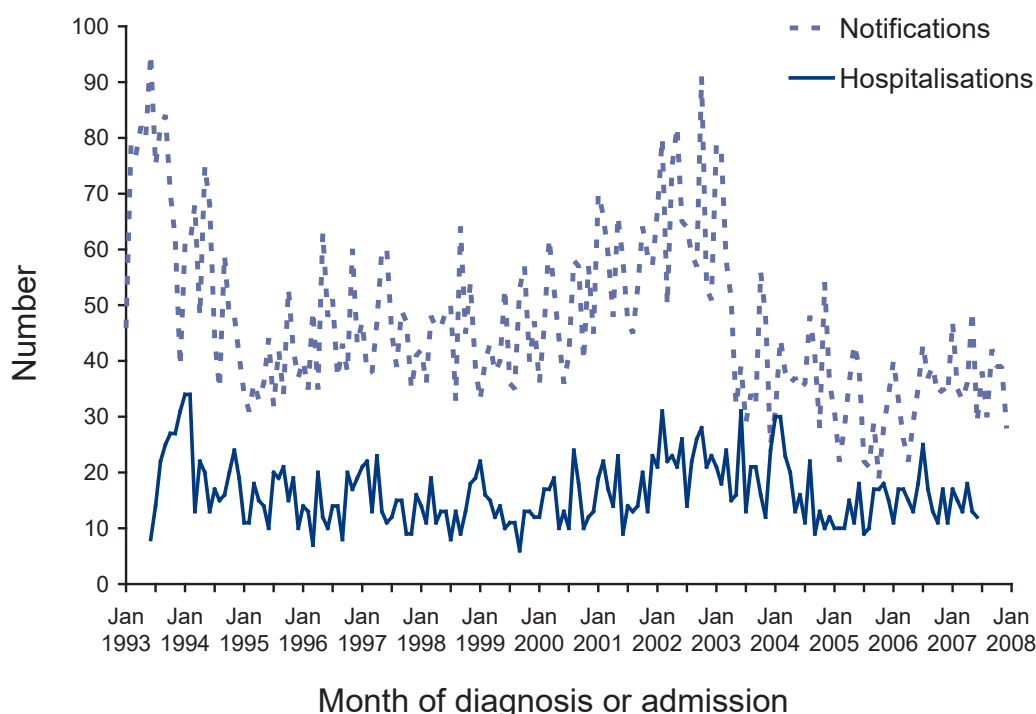
Hospitalisations and deaths

The ICD-10-AM/ICD-10 code A78 was used to identify hospitalisations and deaths.

Secular trends

From January 2006 to December 2007, there were 852 cases of Q fever notified to the NNDSS (average annual rate 2.04 per 100,000). There were 360 hospitalisations coded with a diagnosis of Q fever (average annual rate 0.88 per 100,000) from July 2005 to June 2007. The progressive decline in the overall notification and hospitalisation rates observed in the 3 years following a peak in 2002⁹ did not continue over the 2 years under review (Figure 3.12.1). The notification rates were 1.72 (95% CI 1.55–1.91), 1.97 (95% CI 1.78–2.17) and 2.12 (95% CI 1.93–2.32) per 100,000 in 2005, 2006 and 2007, respectively (see also Appendix 6.2). The hospitalisation rates were 0.77, 0.85 and 0.90 per 100,000 in 2004/2005, 2005/2006 and 2006/2007, respectively (see also Appendix 6.3). Over the 2-year review period, the median number of notifications per month was 35 (range 22–49), and the median number of hospitalisations per month was 15 (range 9–25). The total number of Q fever notifications and hospitalisations over the most recent 4 years for which data are available are shown by month of the year in Figure 3.12.2. There were considerable month-to-month variations in the number of notifications and hospitalisations with no consistent seasonal pattern.

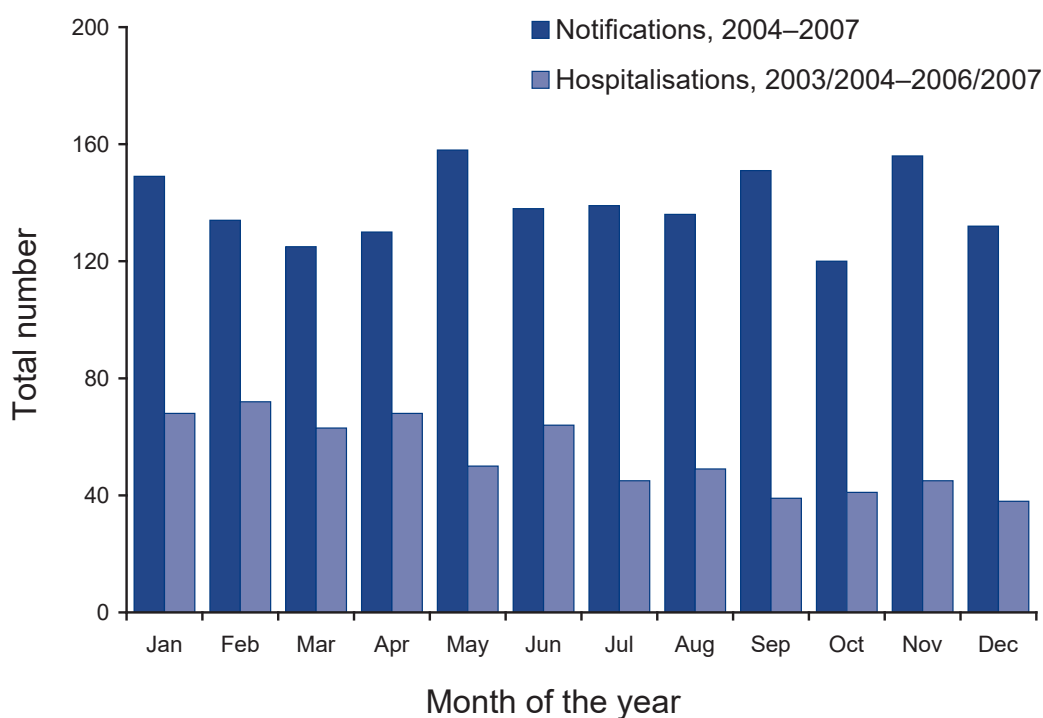
Figure 3.12.1: Q fever notifications and hospitalisations, Australia, 1993 to 2007,* by month of diagnosis or admission



* Notifications where the date of diagnosis was between January 1993 and December 2007; hospitalisations where the date of admission was between July 1993 and June 2007.

Severe morbidity and mortality

In the period July 2005 to June 2007, hospital separations for Q fever accounted for a total of 2,262 hospital bed days (1,065 in 2005/2006 and 1,197 in 2006/2007) with an annual average of 1,131 bed days per year; this was less than for the period July 2002 to June 2005 (1,269 bed days per year).⁹ The median length of stay was 4 days. There were no recorded hospitalisations among children aged <5 years and only 7 hospitalisations (5 with Q fever as the principal diagnosis) among children aged 5–14 years during this period. However, the median length of stay of this latter age group was longest (median 7 days, range 2–14 days), irrespective of whether Q fever was the principal diagnosis (Table 3.12.1). Overall, Q fever was the principal diagnosis for 80% (288/360) of all hospitalisations with any separation diagnosis of Q fever. This proportion does not differ significantly across different age groups.

Figure 3.12.2: Q fever notifications and hospitalisations, Australia, for the most recent 4 years,* by month of the year


* Notifications where the date of diagnosis was between January 2004 and December 2007; hospitalisations where the date of admission was between July 2003 and June 2007.

Table 3.12.1: Q fever notifications, hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Notifications 2 years (2006–2007)		Hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission (days)		Deaths 2 years (2005–2006)	
	n	Rate [‡]	n	(§)	Rate [‡]	(§)	Median	(§)	n	Rate [‡]
0–4	4	0.15	0	(0)	–	(–)	–	(–)	0	–
5–14	29	0.53	7	(5)	0.13	(0.09)	7.0	(7.0)	0	–
15–24	94	1.62	28	(23)	0.49	(0.40)	3.5	(3.0)	0	–
25–59	587	2.89	246	(204)	1.23	(1.02)	4.0	(4.0)	0	–
60+	138	1.84	79	(56)	1.09	(0.77)	6.0	(5.0)	0	–
All ages	852	2.04	360	(288)	0.88	(0.70)	4.0	(4.0)	0	–

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

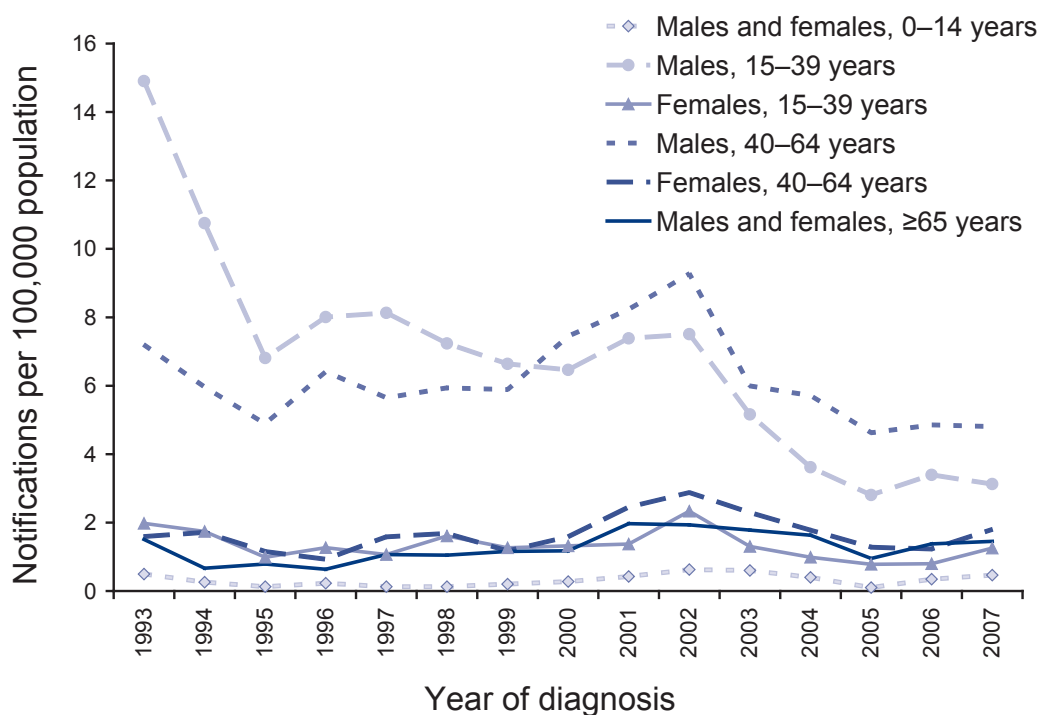
There were no deaths with an underlying cause coded as Q fever recorded in 2005–2006 in the AIHW National Mortality Database. There was 1 death, a female aged >75 years, among the notified cases reported to NNDSS in 2007, but, consistent with the mortality database, none in 2005 or 2006.

Age and sex distribution

During the years 2006 and 2007, the highest average annual notification rates were seen in adult males aged 40–64 years (4.8 per 100,000), followed by males aged 15–39 years (3.3 per 100,000); this higher rate in males aged 40–64 years compared with other age/gender groups has been observed since 2000 (Figure 3.12.3). Notification rates were considerably lower in females aged 40–64 years and people aged ≥ 65 years (1.5 and 1.4 per 100,000, respectively) and even lower in females aged 15–39 years and children aged < 15 years (1.0 and 0.4 per 100,000, respectively).

In the 10 years prior to 2008, annual Q fever notification rates peaked in 2002 in all age groups except those aged ≥ 65 years (which peaked in 2001), and declined progressively until 2005 inclusive. Excluding children aged < 15 years, in whom there were only a small number of cases, the percentage decreases across different age/gender groups were quite similar, and ranged from 51% to 67% when the rate in 2005 was compared with 2002. A modest increase in notification rates was observed in 2006–2007 compared with 2005 for all age/gender groups, but the increases were not statistically significant (Figure 3.12.3). A greater proportional increase was observed in those aged ≥ 65 years and females aged 15–39 years compared with other adults.

Figure 3.12.3: Q fever notification rates, Australia, 1993 to 2007,* by age group, sex and year of diagnosis



* Notifications where the date of diagnosis was between January 1993 and December 2007.

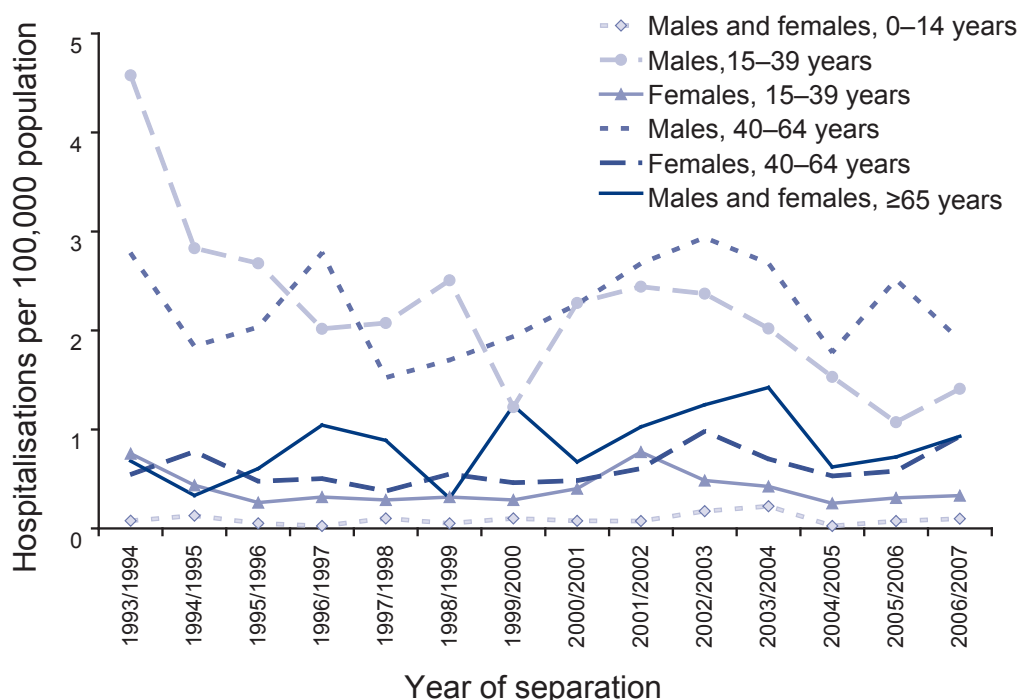
For Q fever hospitalisation rates in 2005/2006–2006/2007, the differentials among various age/gender groups were similar to those for notification rates, with the highest rates seen in adult males aged 40–64 years (2.2 per 100,000), followed by males aged 15–39 years (1.2 per 100,000) (Figure 3.12.4).

There has been a progressive increase in the median age of Q fever notifications from 1993 to 2007 for male cases (Figure 3.12.5). The increase in the median age from 30 years in 1993 to 44 years in 2007 is substantially greater than that seen in the general Australian male population, which increased from 32.4 years in 2003 to 36.9 years in 2007.¹⁰ For notified female cases, the median age increased from 36 years to a peak of 46 years in 2004 and decreased to 41 years in 2007, while the median age of the general female population increased from

33.3 years in 1993 to 37.1 years in 2004 and 37.6 years in 2007. The median age in females tended to be higher than males prior to 1999, but has become similar to that of males since then. A similar progressive increase in the median age of Q fever hospitalisations from 1998/1999 to 2006/2007 was also observed.

There was a clear male predominance for both notification and hospitalisation rates over the 2-year review period, with the male:female ratios being 2.9:1 and 3.6:1, respectively. This was consistent with previous observations since the 1990s, although the male:female ratio for notification rates was lower than that observed in the previous reporting period of 2003–2005 (3.3:1).⁹

Figure 3.12.4: Q fever hospitalisation rates, Australia, 1993/1994 to 2006/2007,* by age group, sex and year of separation



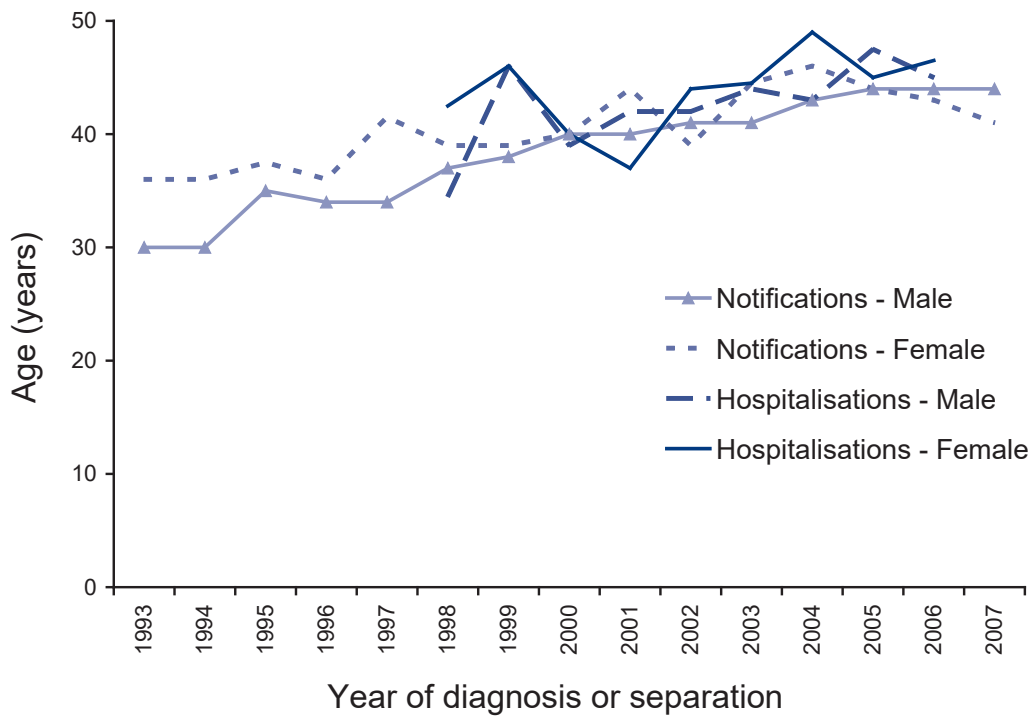
* Hospitalisations where the date of separation was between July 1993 and June 2007.

Geographical distribution

Over the respective 2-year review periods, notification and hospitalisation rates were highest in Queensland, as in previous years. This was followed by New South Wales, when jurisdictions with <5 notifications or hospitalisations were excluded (Appendix 6.2 and Appendix 6.3). Together, these two states contributed 85% (724/852) of the notifications and 81% (293/360) of the hospitalisations for Q fever during the respective 2-year review periods. Excluding the jurisdictions with <5 notifications or hospitalisations, the proportion of hospitalisations relative to notifications was lower in the two states that reported higher overall rates (Queensland, New South Wales) than in the two states that reported lower overall rates (South Australia, Victoria).

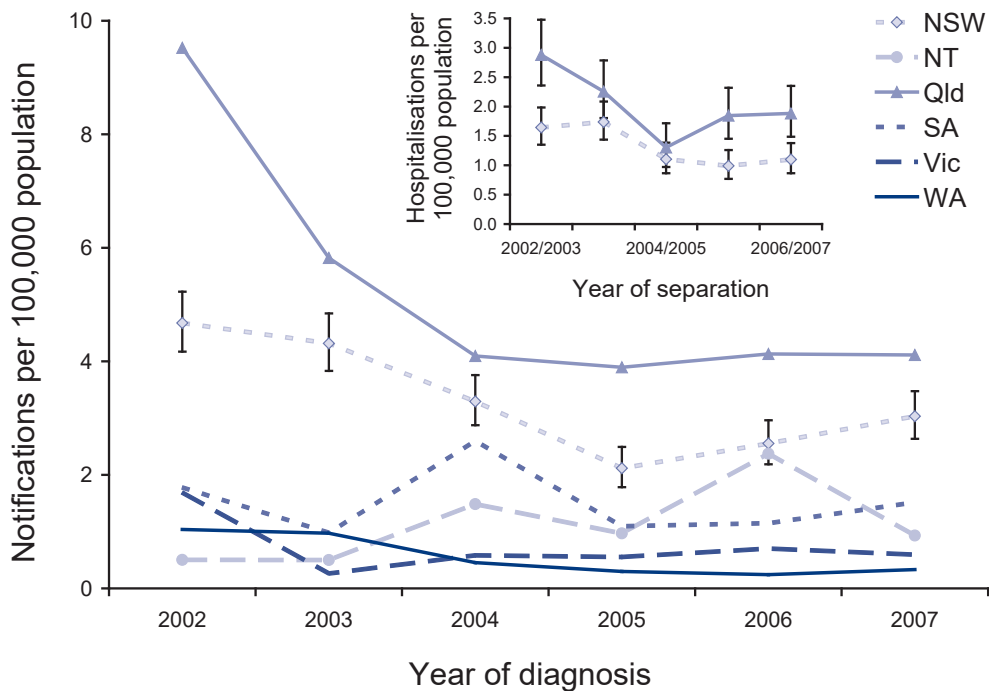
In Queensland and New South Wales, notification rates declined to a trough in 2005 (Figure 3.12.6). While the rates in Queensland remained relatively stable in 2006 and 2007, the notification rates were higher in New South Wales in 2006 and 2007 (2.6 per 100,000 [95% CI 2.2–3.0] and 3.0 per 100,000 [95% CI 2.6–3.5], respectively) compared with 2005 (2.1 per 100,000 [95% CI 1.8–2.5]). For most other jurisdictions, the notification rates remained relatively stable for these 2 reporting years, and there were no significant trends over the 6-year period 2002–2007. The exception was the Northern Territory where Q fever notification rates progressively increased from 2002 to 2006, but decreased in 2007; however, these rates were derived from a very small number of cases and a small population denominator.

Figure 3.12.5: Median age of Q fever notifications and hospitalisations, Australia, 1993 to 2007,* by sex and year of diagnosis or separation



* Notifications where the date of diagnosis was between January 1993 and December 2007; hospitalisations where the date of separation was between July 1998 and June 2007; hospitalisation data for each of the financial years are plotted according to the year in which the financial year began.

Figure 3.12.6: Average annual Q fever notification and hospitalisation rates in selected jurisdictions, Australia, 2002 to 2007,* by year of diagnosis or separation



* Notifications where the date of diagnosis was between January 2002 and December 2007; hospitalisations where the date of separation was between July 2002 and June 2007.

In the two states with more substantial numbers of hospitalisations (Queensland and New South Wales), the magnitude of relative change over time in hospitalisation rates was less than notification rates from 2002 to 2007. In Queensland, hospitalisation rates rose in 2005/2006 and 2006/2007 after a significant decrease in 2004/2005 compared with the preceding 2 years, although rates still remained lower (with marginal statistical significance) compared with 2002/2003. In New South Wales the hospitalisation rates in these 2 recent years remained significantly lower compared with 2003/2004 (Figure 3.12.6).

Vaccination status

Of the 852 notifications of Q fever reported to NNDSS in 2006–2007, 11 (1.3%) were reported to have been vaccinated, but only 3 had their vaccination status validated. The vaccination status was unknown or missing in 302 cases (35.4% of total). These vaccination status proportions were very similar for the cases among males aged 20–69 years (1.2% vaccinated, 36.7% unknown or missing).

Comment

The incidence of Q fever notifications in Australia remains relatively high compared with other countries that report Q fever, which are predominantly in Europe.² In Europe, Q fever is notified often in the context of outbreaks,^{11–13} although outbreaks may be prolonged and become entrenched in certain regions of a country,¹² as exemplified by an ongoing outbreak in The Netherlands.^{14,15} In Europe in the 3 years from 2005 to 2007, notification rates were <0.5 per 100,000 in countries with higher incidence (France, Germany, the UK, Spain, Bulgaria, Greece, Cyprus),^{16–18} except for countries where significant outbreaks occurred (rate 1.03 per 100,000 in The Netherlands and 4.6 per 100,000 in Slovenia in 2007).¹⁸ For all countries, the reported rates of Q fever are likely to be an underestimate of the true disease incidence, as a high proportion of those infected are either asymptomatic or would have mild and non-specific clinical manifestations.²

One earlier French study reported significant seasonal prevalence in May, June and July (spring and early summer) over the years 1982–1990,¹⁹ supposedly attributed to the ‘outside’ lambing season with heavy environmental contamination with the causal bacteria.² However, the peak season of reported Q fever in Europe varied from June–July to September–October in the 3 years from 2005 to 2007.^{16–18} In contrast, the Australian data do not suggest any apparent seasonality in Q fever notifications in earlier reports⁵ or in recent years (Figure 3.12.1 and Figure 3.12.2). This may reflect, at least in part, differences in the epidemiology in Australia, including the relative contribution of sporadic cases and of various transmission routes and environment of the reported cases.

The decline in rates of Q fever in Australia from 2002 to 2005 is likely to reflect the substantial impact of the National Q Fever Management Program (NQFMP), especially among younger adult males, consistent with the demographic profile of abattoir workers.⁷ However, the progressive decrease in the overall notification rates (and, to a lesser extent, the hospitalisation rates) in the 3 years following 2002 did not continue in the 2-year period under review (2006–2007 for notifications, 2005/2006–2006/2007 for hospitalisations). This trend needs to be monitored closely, while recognising that the number of cases was relatively small in some age groups, and that there are inherent limitations and variations in diagnosis and reporting of Q fever. There may also be differences in trend between different jurisdictions or regions.

The levelling, or possibly reversal, of the decreasing trend in the 2 years under review coincided with the cessation of the NQFMP. Since the conclusion of the NQFMP, anecdotal reports suggest that most well-established abattoirs have maintained access to Q fever vaccination for their new staff. However, occupational exposure still continued to contribute to a considerable proportion of cases during and after the cessation of the NQFMP,^{20–24} due to limitations of the program in reaching the more dispersed at-risk populations with potential exposure to Q fever in the farming environment. The vaccine manufacturer announced in late 2005 the cessation of production of the Q fever vaccine^{25,26} until further production could commence in a new vaccine manufacturing facility with the support of the Australian Government.²⁷ This might possibly have impacted on the supply and general availability of the vaccine some time after 2006, and contributed to some occupationally acquired cases who would otherwise have been vaccinated. However, data on the occurrence or extent of any shortage are not available. Building work of a new facility for manufacturing the Q fever vaccine commenced in May 2007, and the facility was scheduled to be operational in 2009.²⁸

The more rapid increase in the median age of Q fever notifications from a lower age to a higher age compared with the increase in median age of the general male population, and the diminishing gender difference in the median age of notifications in more recent years, suggest a progressively lower relative incidence in younger males, the dominant age/gender of workers in high-risk occupations. This may also suggest that exposure in environments other than the traditional high-risk occupational environment or non-occupational exposure may become more important both in absolute and relative terms. A recent analysis of New South Wales notifications showed a significant decline in the proportion of notifications in the 'Abattoir/Meat worker' occupational group and an increase in the 'Farmer/Livestock' occupational group in 2005–2007 compared with previous periods.²⁴ In addition, as would be expected, the proportion of cases that reported community exposure rather than occupational exposure was increasing following the implementation of the NQFMP²⁰ in which the main targets for vaccination were people with high occupational exposure risks. The extent of the absolute increase in disease rates in those with non-occupational (community) exposures is unknown. Control of non-occupational exposure to Q fever is more challenging, and the benefits of using the Q fever vaccine in those environments would be more equivocal. The existing Q fever vaccine formulation has been shown to be efficacious and effective from several published studies conducted in high-risk settings. However, there is a lack of information on its effectiveness in other settings.²⁹

Males aged 15–64 years continued to have the highest rates of Q fever notification and hospitalisation, which reflects the demographic profile of the workforce in the at-risk occupations. However, it should be noted that the percentage decrease in notification rates across different age/gender groups, excluding children aged <15 years in whom there were only a small number of cases, were quite similar when the rate in 2005 was compared with 2002. In addition, greater proportional increases in notification rates in 2006–2007, compared with 2005, were observed in those aged ≥ 65 years and females aged 15–39 years compared with other adults. This suggests that factors other than Q fever vaccination, such as local drought conditions, movements of livestock and livestock slaughter rates, and other environmental factors, might also contribute to the variation in the observed rates. This poses further challenges to the control of Q fever.

The majority of notified Q fever cases in 2006–2007 were unvaccinated. However, it is not possible to ascertain the proportion who would have been considered to be at high risk of exposure and thus should have been recommended vaccination. A similar proportion of cases among males aged 20–69 years were unvaccinated. This suggests that either a significant proportion of those at high occupational risk remained unvaccinated, or that the great majority of notified cases in males aged 20–69 years were acquired through occupational settings with lower risk, where the risks were less well recognised, or through community non-occupational exposure.

Since the early 1990s, southeast Queensland and northeast New South Wales have been the areas that report the highest Q fever incidence in Australia.⁵ The current data continue to reflect this geographic distribution of the disease. Q fever appears to be emerging in the Northern Territory in the past few years. The first notified case of Q fever in the Northern Territory (at least since availability of electronic records in 1991) occurred in March 2002.³⁰ Noting that only a small number of notified cases occurred within a comparatively small population, the Q fever notification rate in the Northern Territory increased from 2002 to 2006 and exceeded that of several other jurisdictions of lower incidence (Figure 3.12.6). Occupational exposures were not identified in these cases, and clustering of cases was not observed.⁶ The rate increase was considered likely to represent a true increase, although increased testing and improved diagnosis might have contributed.³¹ Q fever notification rates decreased in 2007 in the Northern Territory; this might have resulted from changes in natural environmental factors that affected disease transmission, but further careful monitoring is warranted.

As the symptomatology of Q fever tends to be non-specific, and diagnostic practices may vary among patients of different gender and various geographic, occupational and age groups, notification data are likely to be underestimating the true burden of Q fever. Hospitalisation data are only a measure of more severe cases. Notification data in the NNDSS core dataset currently do not contain information on the occupation, probable route of exposure, or immune status of the notified cases. The vaccination status was unknown or missing in more than one-third of all notified cases in 2006–2007, a period after the conclusion of the NQFMP when reporting of vaccination status would be expected to have improved. Information on likely exposure of some notified cases is available, with varying degrees of detail, only from some jurisdictional reports based on enhanced surveillance data.^{20–24,32–34} More complete national surveillance data on exposure factors, occupations and immune/vaccination status of cases are highly desirable to facilitate better understanding of the epidemiology and control of Q fever in Australia.

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3.13 Rotavirus

Rotavirus is a non-enveloped virus that is the major cause of acute gastroenteritis in young children and infants. Infection can be asymptomatic, cause mild to moderate gastroenteritis, or severe gastroenteritis with dehydration requiring hospitalisation.¹ Virtually all children worldwide are infected with rotavirus by 5 years of age, but severe disease occurs most commonly in those aged 6 months to 2 years.^{2,3} Each year >500,000 deaths occur in children <5 years of age worldwide, with >80% of deaths occurring in developing countries.⁴ However, disease does occur in all age groups.¹ Rotaviruses are primarily spread by faecal–oral transmission. Infection with rotavirus confers protection against subsequent serious disease.⁵ Rotaviruses are typed based on two surface proteins, VP7 ('G', glycoprotein) and VP4 ('P', protease sensitive protein). Viruses that contain either G1, 2, 3, 4 or 9 (and either P4 or P8) are the five most common virus types currently circulating in Australia.⁶

Two vaccines for the prevention of rotavirus gastroenteritis became available in Australia in 2006. Both products are oral live attenuated vaccines for use in infants, in either a 2-dose course at 2 and 4 months of age (Rotarix[®], a live attenuated human rotavirus), or a 3-dose course at 2, 4, and 6 months of age (RotaTeq[®], a pentavalent human-bovine reassortant rotavirus). Rotavirus vaccination commenced in the Northern Territory in October 2006, and was funded for all Australian infants under the National Immunisation Program from July 2007. Rotarix[®] is used in the Northern Territory, the Australian Capital Territory, New South Wales and Tasmania. RotaTeq[®] is used in Victoria, South Australia and Queensland. Western Australia changed from using Rotarix[®] to RotaTeq[®] in May 2009. Overall, it is estimated from pre-licensure studies that vaccination prevents around 70% of rotavirus gastroenteritis of any severity and 85%–100% of cases of severe gastroenteritis in immunised infants/children.^{7,8}

Case definitions

Hospitalisations and deaths

The ICD-10-AM/ICD-10 code A08.0 (rotaviral enteritis) was used to identify hospitalisations and deaths.

Northern Territory notification data

Northern Territory notification data for the period January 2006 to December 2007 are included in this report. Historical notification data is published elsewhere.⁹ 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'.

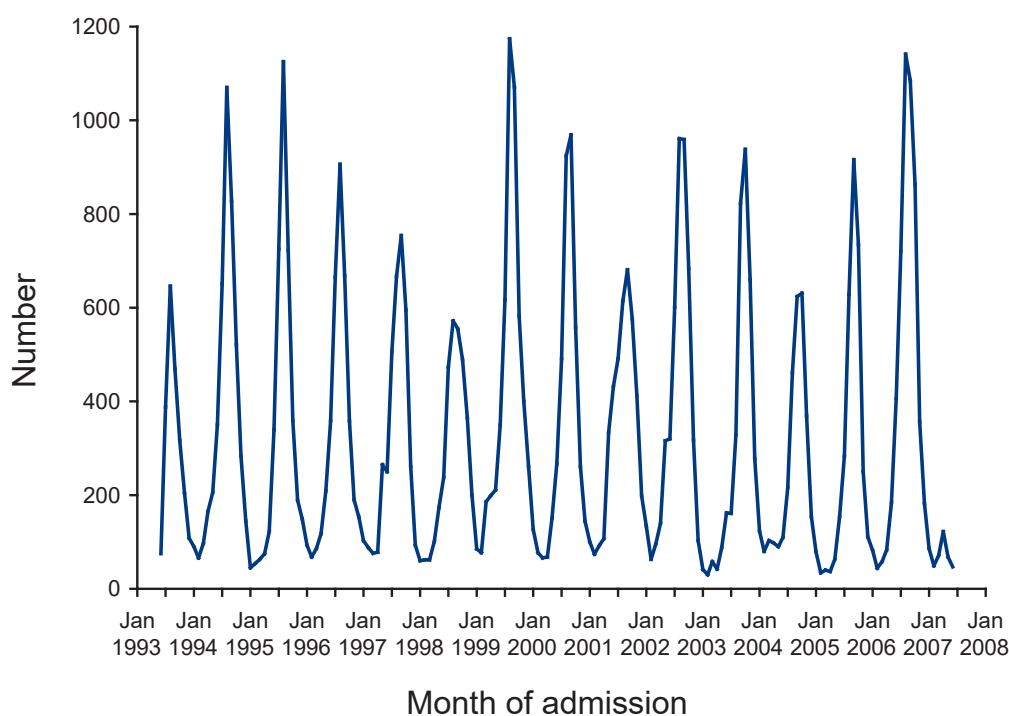
Secular trends

National notification data for rotavirus are not available for reporting, as rotavirus was not nationally notifiable during the reporting period of 2006–2007. (Notification data from the Northern Territory are reported in a later section of this chapter.)

In the 2-year review period (2005/2006 to 2006/2007), there were 8,602 hospitalisations for rotavirus (average annual rate of 20.9 per 100,000 population). Rotavirus was recorded as the principal diagnosis in 88% of these hospitalisations (Table 3.13.1). There were more hospitalisations recorded in the 2nd year of the review period (n=3,745 in 2005/2006, n=4,857 in 2006/2007). Overall, hospitalisation rates for this review period were higher in each age group, compared with the previous 3-year period (2002/2003–2004/2005).¹⁰ During that period, the number of hospitalisations for rotavirus gastroenteritis was greatest in 2002/2003 (n=4,071) (Appendix 6.3). These observations are consistent with known fluctuations in rotavirus disease activity between years.¹¹ 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 current review period was consistent with this observation, with a low of 44 hospitalisations in February 2006 and a peak of 1,141 hospitalisations in August 2006 (Figure 3.13.1).

Severe morbidity and mortality

The number of bed days and median length of stay were calculated only for those hospitalisations with a principal diagnosis of rotavirus. There were a total of 18,308 bed days recorded for rotavirus (average 9,154 per year). The median LOS was 2 days.

Figure 3.13.1: Rotavirus hospitalisations, Australia, 1993/1994 to 2006/2007,* by month of admission

* Hospitalisations where the date of admission was between July 1993 and June 2007.

Table 3.13.1: Rotavirus hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission [‡] (days) Median	Deaths 2 years (2005–2006)	
	n	(*)	Rate [§]	(*)		n	Rate [§]
0–4	7,591	(6,747)	292.4	(259.9)	2.0	0	–
5–14	682	(623)	12.4	(11.4)	2.0	0	–
15–24	35	(23)	0.6	(0.4)	2.0	0	–
25–59	95	(60)	0.5	(0.3)	2.0	0	–
60+	199	(107)	2.7	(1.5)	4.0	2	0.03
All ages	8,602	(7,560)	20.9	(18.4)	2.0	2	<0.005

* Hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

‡ Principal diagnosis (hospitalisations).

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

The AIHW National Mortality Database records indicate a total of 16 cases with the underlying cause of death due to rotavirus over the last 17 years (1990–2006). Seven of the deaths occurred in children <5 years of age, three in those aged 5–69 years, and six in adults aged ≥70 years. Only 3 deaths have been recorded since 2001. In the 2-year period of this report, only 2 deaths were reported, both in very elderly adults >90 years of age.

Age and sex distribution

Across all age groups, slightly more males were hospitalised with rotavirus (male:female ratio 1.15:1). The vast majority of rotavirus hospitalisations occurred in those <5 years of age (n=7,591, 88%) (Table 3.13.1). The age distribution of hospitalisations in children <5 years of age can be seen in Table 3.13.2. The highest rate of hospitalisation occurred in those aged 12–23 months, closely followed by those aged 6–11 months. The median length of stay for hospitalisations with rotavirus as principal diagnosis was 2 days for all the age groups <5 years. Figure 3.13.2 shows the trends in hospitalisation rates by age over time in children <5 years of age.

Table 3.13.2: Rotavirus hospitalisations, Australia, 2005/2006 to 2006/2007,* by age group (<5 years)

Age group (months)	Hospitalisations 2 years (July 2005–June 2007)			
	n	(†)	Rate‡	(†)
0–5	793	(574)	301.3	(218.1)
6–11	1,385	(1,178)	526.3	(447.6)
12–23	2,808	(2,537)	542.5	(490.1)
24–35	1,541	(1,459)	299.7	(283.7)
36–47	687	(643)	133.4	(124.8)
48–59	376	(355)	72.0	(67.0)
All ages	7,591§	(6,747)§	292.4	(259.9)

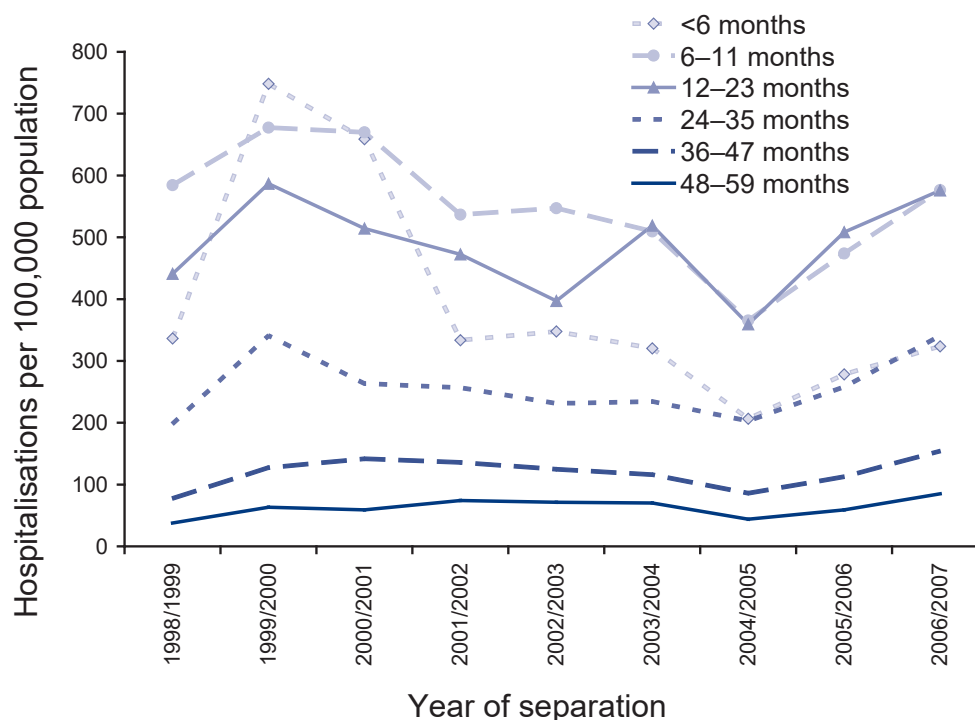
* Hospitalisations where the date of separation was between July 2005 and June 2007.

† Principal diagnosis (hospitalisations).

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

§ Including 1 case of hospitalisation aged <5 years with unknown age in months.

Figure 3.13.2: Rotavirus hospitalisation rates, Australia, 1998/1999 to 2006/2007,* by age group (<5 years) and year of separation



* Hospitalisations where rotavirus was recorded as either a principal or any other diagnosis and the date of separation was between July 1998 and June 2007.

Hospitalisations recorded in infants <6 months of age appear to have declined since 2001/2002, from peak rates observed in 1999/2000 and 2000/2001, whereas other age groups have continued to have fluctuating but similar hospitalisation rates over 9 years.

Geographical distribution

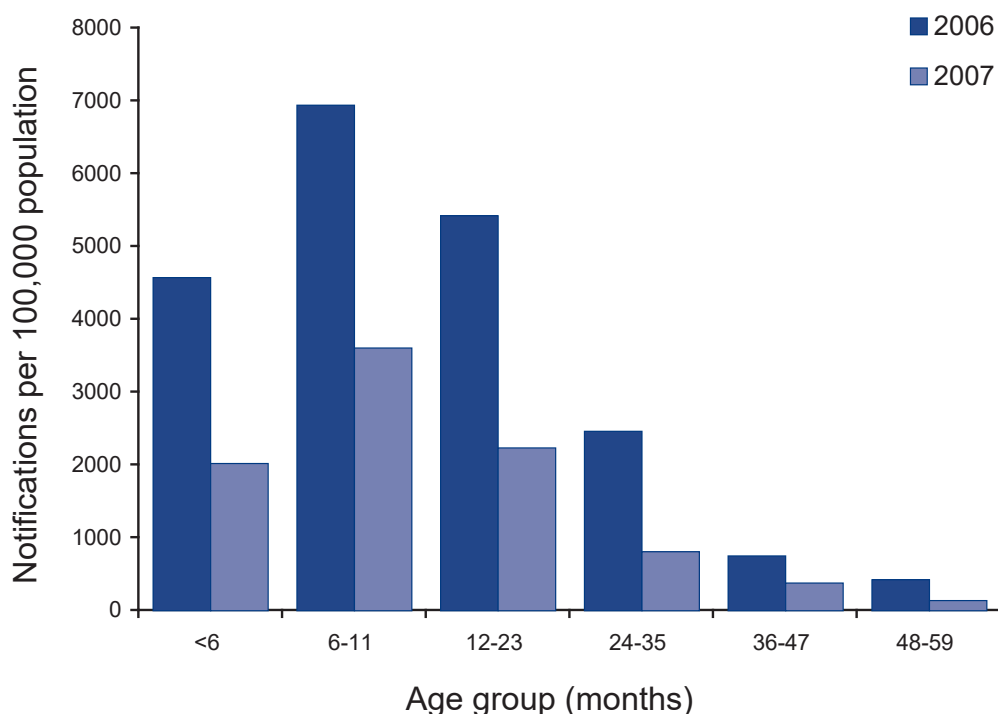
Over the period 2005/2006 to 2006/2007, the Northern Territory recorded over 5 times the Australian average rate of hospitalisation for rotavirus gastroenteritis (Northern Territory rate: 101.4 per 100,000 population) (Appendix 6.3). Victoria had the lowest rate of hospitalisation (11.4 per 100,000 population), about 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.^{2,12}

Northern Territory notification data, rotavirus

A total of 846 cases were notified from January 2006 to December 2007 among residents of the Northern Territory, an average annual rate of 198.8 per 100,000 population. In 2006, there were more than twice as many notifications (n=590; annual rate 280.1 per 100,000 population) as in 2007 (n=256; annual rate 119.1 per 100,000 population). This represents a 57% decline in the notification rate in the Northern Territory in 2007 compared with 2006. The male:female ratio among notified cases was 1:1.

The rate of notification (198.8 per 100,000) for rotavirus was considerably higher than the hospitalisation rate (101.4 per 100,000). The peak age group of notified cases from the Northern Territory (6–11 months) (Figure 3.13.3) was younger than that of hospitalised cases from all of Australia.

Figure 3.13.3: Rotavirus notification rates, the Northern Territory, 2006 and 2007,* by age group (<5 years)



* Notifications where the date of diagnosis was between January 2006 and December 2007.

Comment

Prior to the national rotavirus immunisation program which commenced in July 2007, rotavirus was responsible for several thousand hospitalisations with either a principal or secondary diagnosis of rotavirus in Australia annually. The primary disease burden is in those <5 years of age, with hospitalisations most common in the first 2 years of life.

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 always conducted. Clinical guidelines for the management of acute uncomplicated gastroenteritis do not recommend routine stool testing for confirmation of the aetiological agent.¹³ Several international studies have shown that measurement of rotavirus hospitalisation rates utilising the specific rotavirus code underestimated the true number of rotavirus-associated hospitalisations, and that the sensitivity of the rotavirus coded hospitalisations was only 25%–47%.^{14–16} Two Australian studies have estimated the burden of rotavirus hospitalisations, imputed from the proportion of acute gastroenteritis hospitalisations attributable to rotavirus, using distinct methodologies. Both studies, conducted a decade apart, estimated that rotavirus was responsible for approximately 10,000 hospitalisations annually in Australia, prior to vaccine introduction.^{12,17} In addition, the number of emergency department visits for rotavirus was estimated to be approximately 21,500 annually.¹⁷ These estimations are likely to be a more accurate picture of the true disease burden.

Compared with other developed countries, prior to the rotavirus immunisation program, Australia had a relatively high rate of hospitalisation, with an estimated 1 in 27 children hospitalised by 5 years of age.¹² In Europe it was estimated that 1 in 50 children had been hospitalised by the age of 5 years, although estimates varied by country and study methods.¹⁸ 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.^{19,20}

Rotavirus hospitalisation rates were higher in this review period in comparison with those reported for all age groups in the previous 3 years. This may reflect a greater awareness of rotavirus disease and possible increased testing or coding associated with the imminent availability of two new rotavirus vaccines. In addition, rotavirus has increasingly been recognised as a cause of morbidity in elderly patients, including in settings such as aged care facilities.^{21,22} Of note, the 2 rotavirus deaths recorded in this review period were in persons >90 years of age. Alternatively, higher hospitalisation rates may reflect the natural seasonal fluctuations seen in the severity of rotavirus disease.

The Australian Rotavirus Surveillance Program was initiated in June 1999 to monitor changes in the distribution of rotavirus serotypes over time. Serotype G1 continues to be the most frequently reported serotype worldwide and has been the most common Australian serotype for all but 2 years since 1999.^{23,24} From July 2001 to June 2003, serotype G1 was replaced by G9 as the most dominant serotype,^{25,26} followed by a decline in G9 prevalence in subsequent years.^{6,23,26,27} It appears likely that both rotavirus vaccines are effective against the G9 serotype, as well as the majority of other serotypes detected in Australia.^{7,8}

Hospitalisation and notification data from the Northern Territory emphasise 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.⁹ These epidemics are thought to result from the relative isolation of remote communities with a lack of immunity against circulating strains, which then see a rapid spread of infection upon reintroduction of the virus.^{9,28,29} Rotavirus immunisation of all infants in the Northern Territory began in October 2006, 8 months earlier than the national program. The 57% decline in rotavirus notifications observed in the Northern Territory in 2007, compared with 2006, may be an early impact of vaccination in that jurisdiction, although substantial year-to-year variation in incidence has been observed prior to vaccine introduction.²⁹ Confirmation of this trend through ongoing observations will be required.

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3.14 Rubella

Rubella is caused by the rubella virus (family togaviridae). It is usually a mild febrile viral disease characterised by a non-confluent maculopapular rash, conjunctivitis, coryza, headache, nausea, and post-auricular, suboccipital and cervical lymphadenopathy. However, subclinical infection occurs in up to 50% of cases. Arthralgia and arthritis may also occur, particularly in post-pubertal females. More severe disease manifestations, such as encephalitis, haemorrhage and Guillain-Barré syndrome, may also rarely occur. Rubella is of public health significance because, when acquired in the first trimester of pregnancy, it is associated with spontaneous abortion, or, in survivors, with abnormalities of the congenital rubella syndrome (CRS) in up to 80% of cases. These include cataract, retinopathy, deafness, heart defects and neurological deficit.¹

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

Rubella – national definition from January 2004:²

Both confirmed and probable cases are notifiable. 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 ≥ 4 -fold 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.

Congenital rubella syndrome (CRS) – national definition from January 2004:²

Both confirmed and probable cases are notifiable. A confirmed case requires laboratory definitive evidence and clinical evidence. A probable case requires laboratory suggestive evidence (either maternal or infant) and clinical evidence.

- a) Laboratory definitive evidence
 - Isolation of rubella virus from the infant; *or*
 - Detection of rubella virus in the infant by nucleic acid testing; *or*
 - Detection of rubella-specific IgM antibody in the serum of the infant and confirmation of the result in a reference laboratory.
- b) Laboratory suggestive evidence
 - Isolation of rubella virus from the mother; *or*
 - Detection of rubella virus in the mother by nucleic acid testing; *or*
 - IgG seroconversion or a significant increase in antibody level or a ≥ 4 -fold rise in titre to rubella virus in paired sera of the mother tested in parallel; *or*
 - Detection of rubella-specific IgM antibody in the mother in the absence of recent rubella vaccination (must be confirmed in a reference laboratory); *or*
 - Detection of rubella-specific IgM antibody in the blood of the infant using capture enzyme-linked immunosorbent assay; *or*
 - Infant rubella-specific antibody that persists at a higher level and for a longer period than expected from passive transfer of maternal antibody (rubella titre that does not drop at the expected rate of a 2-fold dilution per month).

c) Clinical evidence

- A live or still born infant with any of the following compatible defects: cataracts, congenital glaucoma, congenital heart disease, hearing defects, microcephaly, pigmentary retinopathy, mental retardation, purpura, hepatosplenomegaly, meningoencephalitis, radiolucent bone disease.

Hospitalisations and deaths

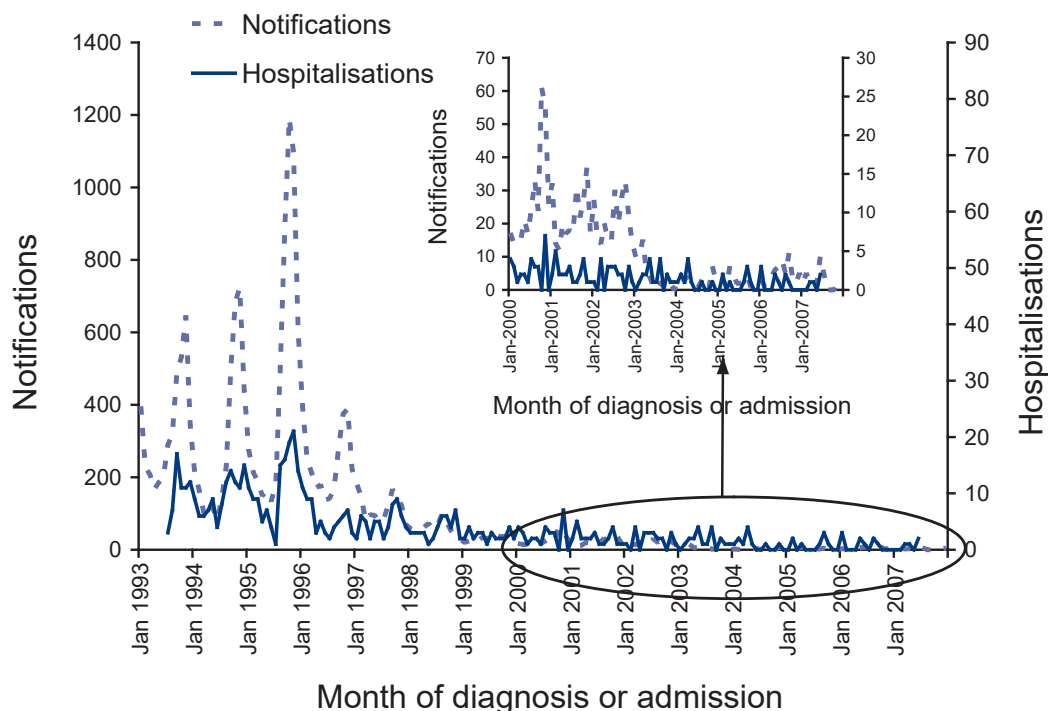
The ICD-10-AM/ICD-10 code B06 (rubella [German measles]) was used to identify hospitalisation and deaths. However, hospitalisations coded as congenital rubella syndrome (ICD-10-AM code P350) are not included in this report, as all were in cases aged >10 years in which secondary diagnosis codes suggested long-term sequelae or complications of the condition.

Secular trends

During 2006–2007, there were 94 notified cases of rubella, of which 77 (82%) were confirmed and 17 (18%) were probable cases. Overall, the average annual notification rate was 0.23 per 100,000 population (Table 3.14.1). The annual number of rubella notifications has remained relatively stable over the last 5 years following the marked decline in the late 1990s and early 2000s (Figure 3.14.1). The 59 notifications in 2006 (0.29 per 100,000) was higher compared with the 31 in the previous year (0.15 per 100,000) and 35 in 2007 (0.17 per 100,000). The median number of notifications per month over the review period was 3.5 (range 0–11). Notifications peaked in late winter and early spring in 2006 and mid winter in 2007, which contrasted slightly to the mid spring peak in activity observed earlier in the decade.³

Nine confirmed cases of CRS have been notified to the NNDSS since 2002, with none in 2006 and two diagnosed in 2007. Both of these cases were diagnosed at age <1 year. The Australian Paediatric Surveillance Unit (APSU) also operates a surveillance system for CRS in Australia. Each month, the APSU contacts approximately 1,250 clinicians requesting them to report children who were newly diagnosed with a range

Figure 3.14.1: Rubella notifications and hospitalisations, Australia, 1993 to 2007,* by month of diagnosis or admission



Note varying scales between notifications and hospitalisations.

* Notifications where the date of diagnosis was between January 1993 and December 2007; hospitalisations where the date of admission was between July 1993 and June 2007.

of uncommon conditions, including CRS. A brief follow-up questionnaire is then sent to the clinician by the CRS study investigator, requesting further de-identified information for each new case.⁴ The APSU identified 8 confirmed cases of CRS between 2002 and 2007. However, all of these occurred prior to 2005.⁴⁻⁸

A very low rate of rubella hospitalisation has been maintained with only 18 hospitalisations coded as being due to rubella between July 2005 and June 2007, an average annual rate of 0.04 per 100,000 population.

Severe morbidity and mortality

For the period July 2005 to June 2007, 57 hospital bed days were recorded for patients with an ICD-10-AM code for rubella. Of the 18 hospital separations, 9 (50%) had a principal diagnosis of rubella (average annual rate 0.02 per 100,000). The median length of stay in hospital was 2 days for all hospitalisations and 1 day for those with a principal diagnosis of rubella (Table 3.14.1). There were no deaths with rubella recorded as the underlying cause in 2005 or 2006.

Table 3.14.1: Rubella notifications, hospitalisations and deaths, Australia, 2002 to 2007,* by age group

Age group (years)	Notifications 2 years (2006–2007)		Hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission (days)		Deaths 2 years (2005–2006)	
	n	Rate [‡]	n	(§)	Rate [‡]	(§)	Median	(§)	n	Rate [‡]
0–4	8	0.30	9	(7)	0.35	(0.27)	1.0	(1.0)	0	–
5–14	4	0.07	1	(0)	0.02	(–)	n.p.	(–)	0	–
15–24	20	0.34	3	(1)	0.05	(0.02)	n.p.	(n.p.)	0	–
25–59	60	0.30	4	(1)	0.02	(<0.005)	2.5	(n.p.)	0	–
60+	2	0.03	1	(0)	0.01	(–)	n.p.	(–)	0	–
All ages	94	0.23	18	(9)	0.04	(0.02)	2.0	(1.0)	0	–

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

n.p. Not published.

Complications arising from rubella infection were recorded for 4 (22% of 18) hospitalisations (Table 3.14.2). One of the 4 hospitalisations with complications was recorded in a child <5 years of age, while the remaining 3 were in adults aged 25–59 years. This is comparable to the previous 3 years, during which complications were recorded for 5 of 44 hospitalisations with rubella, including one aged <5 years.⁹

Age and sex distribution

The significant decrease in rubella notifications, and to a lesser extent hospitalisations, achieved earlier in the decade has been maintained, with a low incidence of both across age and sex categories over recent years (Figures 3.14.2 and 3.14.3). Adults aged 25–59 years accounted for almost two-thirds of the rubella notifications for the 2-year review period 2006–2007 (Table 3.14.1). Notification rates were highest in the 25–29 years age group, who accounted for over a quarter (26/94, 28%) of all cases (average annual rate 0.91 per 100,000). Males predominated in this latter age group (male:female ratio 1.8:1) and were the only group where the notification rate was over 1 per 100,000 (Table 3.14.3). The declining rates of rubella in children and higher proportions in adults have led to an increase in the median age of both notified and hospitalised cases since the Measles Control Campaign (in which the MMR vaccine was used) in 1998.⁹ This trend continued with a median age of notified cases of 27 years during the review period, which represented an increase from the median age of 25 years over the previous review period (2003–2005).

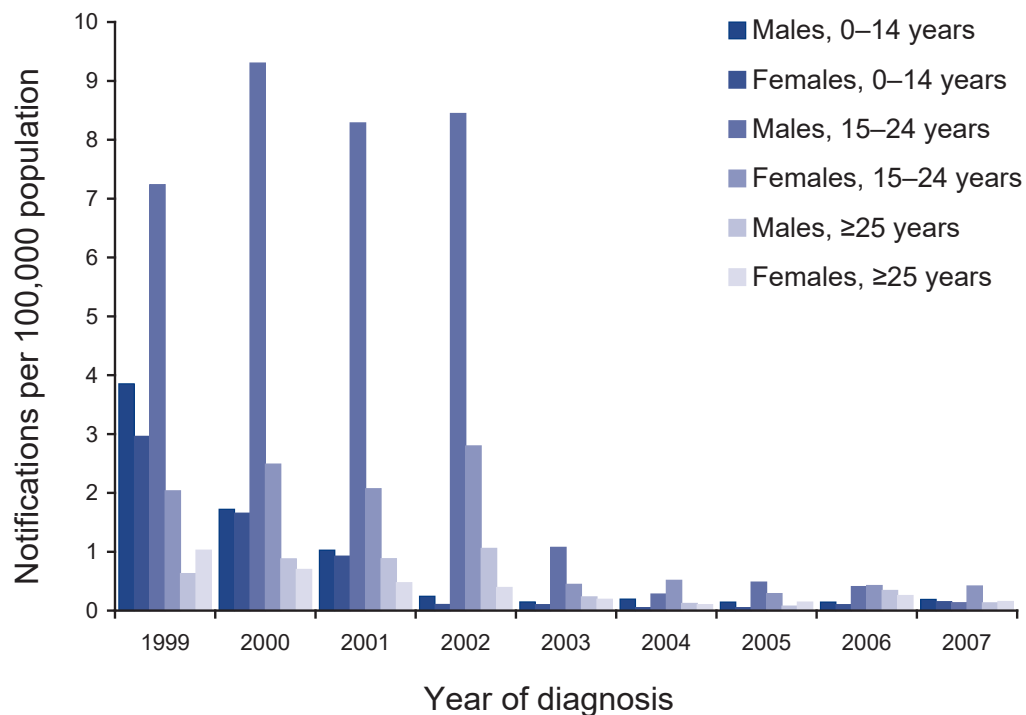
Table 3.14.2: Indicators of severe morbidity* for hospitalised cases of rubella, Australia, 2005 to 2007,* by age group

Age group (years)	Rubella with neurological complication		Rubella with other complications		Rubella without complications	
	n	% Total†	n	% Total†	n	% Total†
0–4	0	–	1	11.1	8	88.9
5–14	0	–	0	–	1	100
15–24	0	–	0	–	3	100
25–59	1	25.0	2	50.0	1	25.0
60+	0	–	0	–	1	100
All ages	1	5.6	3	16.7	14	77.8

* Based on National Hospital Morbidity data where the date of hospital separation was between July 2005 and June 2007.

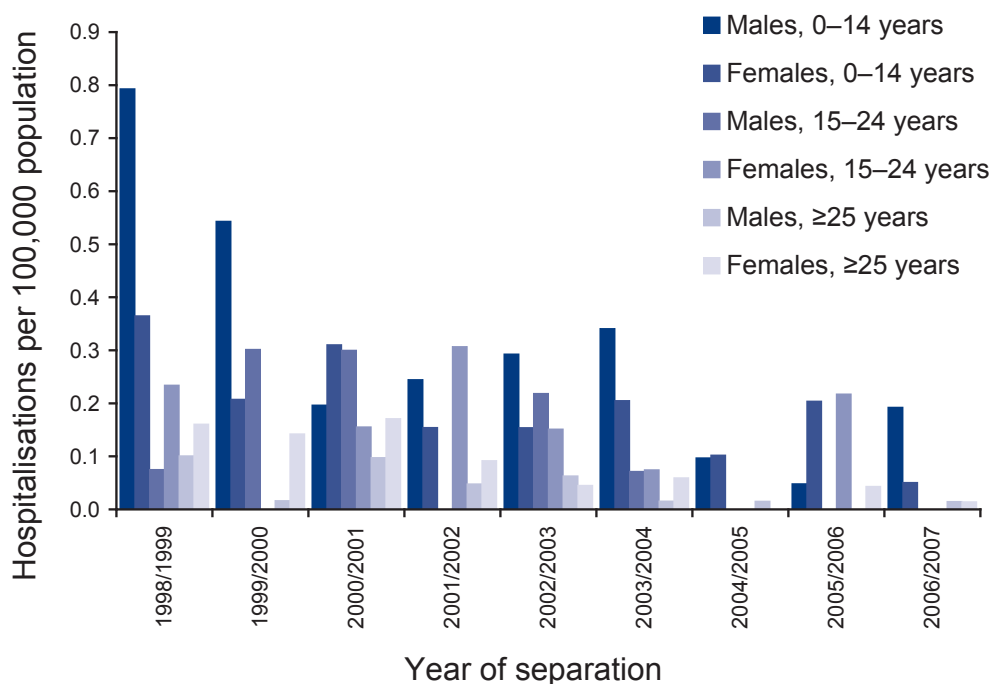
† % of total in the age group.

The overall male:female ratio of notified cases was 1:1 for the 2-year review period. There were 35 notifications of rubella in females of child-bearing age (15–44 years) in 2006–2007, resulting in an average annual rate of 0.4 per 100,000, which is comparable to the previous review period. Those aged 25–44 years were the cohort in which females but not males were recommended to receive rubella vaccine at age 10–14 years since 1971 before the implementation of the adolescent MMR vaccination for both sexes in 1994–1996 and the Measles Control Campaign in 1998. In this cohort, the male:female ratio was 1.3:1. In particular, the male:female ratio was 1.7:1 among those aged 25–29 years, although the number of notifications was small (Table 3.14.3). The male:female ratio among those aged <25 years was 0.84:1.

Figure 3.14.2: Rubella notification rates, Australia, 1999 to 2007,* by age group, sex and year of diagnosis


* Notifications where the date of diagnosis was between January 1999 and December 2007.

Figure 3.14.3: Rubella hospitalisation rates, Australia, 1998/1999 to 2006/2007,* by age group, sex and year of separation



* Hospitalisations where the date of separation was between July 1998 and June 2007.

Table 3.14.3: Rubella notifications and hospitalisations, Australia, 2005 to 2007,* by sex and selected age groups of interest

Age group (years)	Notifications 2 years (2006–2007)				Hospitalisations 2 years (July 2005–June 2007)			
	Male		Female		Male		Female	
	n	Rate [†]	n	Rate [†]	n	Rate [†]	n	Rate [†]
<1	2	0.72	1	0.38	4	1.48	2	0.78
1–4	2	0.19	3	0.29	1	0.09	2	0.20
15–19	0	–	5	0.36	0	–	1	0.07
20–24	8	0.53	7	0.48	0	–	2	0.14
25–29 [‡]	16	1.11	9	0.64	0	–	2	0.14
30–34	6	0.41	6	0.40	0	–	0	–
35–39	5	0.32	6	0.39	1	0.07	0	–
40–44	0	–	2	0.13	0	–	0	–

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007.

[†] Average annual age-specific rate per 100,000 population.

[‡] There was 1 notification where gender was not reported.

For the 2 years from July 2005 to June 2007, children aged 0–4 years continued to have the highest hospitalisation rate (average annual rate 0.35 per 100,000). However, two-thirds (6/9) of the hospitalisation episodes in this age group were in children too young to be vaccinated (<1 year of age) (Table 3.14.3). While there were twice as many males in this age group, the overall male:female ratio for rubella hospitalisations was 0.5:1.

Geographical distribution

The majority of cases in 2006–2007 were notified from New South Wales ($n=45$; rate 0.33 per 100,000) and Queensland ($n=26$; rate 0.31 per 100,000), analogous to the previous review period. Notifications were highest in New South Wales in 2006 ($n=37$; rate 0.54 per 100,000), with the majority of cases in south eastern and central Sydney in those aged 15–44 years.¹⁰ No source was identified for this increase in notifications. Victoria notified 13 cases during the review period, including a cluster of 3 cases in one family in 2006.¹¹ Five cases were notified in Western Australia, three in South Australia and two in the Australian Capital Territory. No rubella notifications were recorded in Tasmania or the Northern Territory in either 2006 or 2007 (Appendix 6.2).

There were 8 notified cases (9%) in the NNDSS recorded as being acquired overseas (age range 22–38 years) and 55 (59%) recorded as locally acquired; no information was available for the remaining 31 cases.

Of the 2 cases of CRS in 2007 that were notified to the NNDSS, one was recorded as acquired overseas and the other as not imported.

The geographical distribution of rubella hospitalisations reflected that of the notifications (see Appendix 6.3). However, there were too few hospitalisations in each jurisdiction to identify any trends.

Vaccination status

Vaccination status is required in the NNDSS for all notifications of rubella in women of child-bearing age (i.e. 15–44 years). Although this field was completed for 34 of 35 cases in this category, the vaccination status was recorded as 'unknown' for 16 (47%). Of those women with known vaccination status, the majority were unvaccinated (12/18; 67%), one was partially vaccinated (i.e. had received 1 dose of rubella-containing vaccine), and five (28%) were reported as fully vaccinated (i.e. had received 2 doses). Two of the five reported as fully vaccinated had valid written records. The vaccination status of the partially vaccinated woman was also validated.

Of the 9 notified cases in 2006–2007 who were aged <7 years, 6 were aged 1–3 years, and 3 were aged 4–7 years. Of those 6 cases aged 1–3 years, 2 had been vaccinated with 1 dose of rubella-containing vaccine (validated), and 2 were reported to be unvaccinated. Of the 3 cases aged 4–7 years, 1 had received 2 vaccine doses (validated), 1 was reported to be unvaccinated, and the vaccination status of the remaining case was unknown.

Overall, vaccination status was recorded for 55 cases, of which 11 (6 validated) were fully vaccinated, 4 (1 validated) partially vaccinated, and 40 unvaccinated.

The vaccination status of the mothers of the 2 notified cases of CRS in 2007 was not available from the NNDSS.

Comment

The successful control of rubella in Australia over the last decade through mass school-based MMR vaccination has been sustained, and rubella notification and hospitalisation rates continue to remain at very low levels. Young men no longer appear to be at greater risk for rubella infection as previous surveillance and serology data have indicated.^{3,12} In fact, the male:female ratio in persons aged <25 years in 2006–2007 was about 1:1. However, despite these achievements, some challenges still remain.

Evidence suggests that Indigenous women in the Northern Territory have inadequate immunity to rubella.¹³ Serological evidence from women of child-bearing age also indicates that migrants from countries without an established rubella vaccination program, particularly Asia, South America and sub-Saharan Africa, are at greater risk of not being immune to rubella compared with the general Australian population.^{14,15} A study in Victoria found that self-reported rubella vaccination prior to arrival in Australia for adult migrants from East Africa was less than 1%.¹⁶ In the majority of cases (89%), vaccination status was unknown. Serological testing of African refugees living in Melbourne in the first half of 2005 demonstrated that 32% of those aged <15 years and 4% of those aged ≥ 15 years were not immune to rubella.¹⁷ Targeted rubella screening and vaccination of high-risk groups remains an important component of ongoing rubella control.

The epidemiology of CRS in Australia from 1992 to 1997 has been described and discussed by Sullivan et al (1999).¹⁸ More recently, 6 of the 8 cases of CRS identified by the APSU since 2002 were born to immigrant mothers who had incomplete vaccination or unvaccinated mothers who had travelled to rubella prevalent countries during pregnancy.^{5,6,19} The other 2 CRS cases were born to Australian-born mothers who missed school-based MMR immunisation.^{5,19} Of the 2 CRS cases in 2007 notified to NNDSS, 1 was recorded as acquired overseas and the other as not imported. Details on the vaccination status of the mothers of these cases were not available. The identification of different cases of CRS at different times from the APSU and the NNDSS, which utilise different reporting sources and mechanisms, indicates that both systems are complementary and important for surveillance of CRS in Australia.

Endemic transmission of rubella in the USA was declared to be eliminated in October 2004, on the basis of evidence showing high levels of vaccine coverage and population immunity, and low disease incidence over a number of years.^{20,21} Rubella genotyping was also an important tool in the USA elimination effort as it could be demonstrated that local strains were no longer circulating.²² The WHO strategic plan for Europe has set a target of <1 case of CRS per 100,000 live births and <1 case of rubella per 100,000 population across the region by 2010.²³

Despite the progress made in countries with high vaccination coverage, travellers remain at risk of rubella exposure in many other countries where high vaccination coverage has not been achieved. By the end of 2006, 123 countries, covering 27% of that year's birth cohort, included a rubella vaccine in their immunisation schedule.²⁴ Globally, there were 85,158 cases of rubella and 206 CRS cases notified to the WHO in 2007, of which 46% and 83%, respectively, were in the Western Pacific region.²⁵ However, these figures are an underestimate as a high proportion of cases are asymptomatic and many countries do not undertake routine surveillance for rubella. The potential for global rubella control continues to improve on the back of intensive measles elimination efforts and the opportunity to increase coverage of combined vaccines. Individual countries must evaluate the burden of disease caused by rubella through appropriate surveillance mechanisms and assess the cost-effectiveness of introducing rubella vaccine into national immunisation programs, either targeted at adolescent and adult females or universal vaccination of all children.²⁶ Although only 8 cases in the review period were identified in NNDSS as being imported from overseas, this information was missing for a further 31 cases.

The low level of rubella notifications in Australia, combined with serology data demonstrating that <5% of both women of child-bearing age and children aged 2–14 years are seronegative,²⁷ indicate that the potential for endemic transmission in this country is very limited. In addition, the high level of immunity to rubella and sustained low disease incidence indicates that Australia is moving towards rubella elimination. However, despite this, 55 cases during the review period were recorded in the NNDSS as 'not imported'. In order for Australia to eliminate endogenous rubella transmission, a strong regional approach must be taken, similar to the Pan American approach that resulted in the elimination of rubella in the USA.²⁸ This approach involved intensive mass immunisation campaigns that achieved high immunity and low incidence of both rubella and CRS across the Americas.²⁹ Rubella genotyping, which is not routinely available in Australia, may also facilitate a better understanding of circulating strains and assist in determining if indigenous strains have been eliminated. Nevertheless, maintaining high coverage and improved timeliness of MMR vaccination among children should remain an important goal of Australia's rubella elimination strategy.

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3.15 Tetanus

Tetanus is an acute disease resulting from the formation of exotoxin by *Clostridium tetani*, an anaerobic bacterium that grows at the site of injury and produces toxin with local and systemic neuromuscular effects. Tetanus spores are ubiquitous in the environment and can contaminate all types of wounds. 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 over 80%, with the highest rates in infants and the elderly.¹

Case definitions

Notifications

See Appendix 6.6 for pre-2004 definition

National definition from January 2004:²

Only confirmed cases are notifiable. 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 other apparent causes.

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 6 notifications of tetanus in the period January 2006 to December 2007 (an average annual notification rate of 0.01 per 100,000) (Table 3.15.1). Notifications for tetanus remained stable in 2006 and 2007, with 3 notifications annually. Between July 2005 and June 2007, there were 36 hospitalisations coded as tetanus (an average annual rate of 0.09 per 100,000). Hospitalisations for tetanus also remained relatively stable between 2005/2006 and 2006/2007; however, there has been a downward trend over the past decade (Figure 3.15.1).

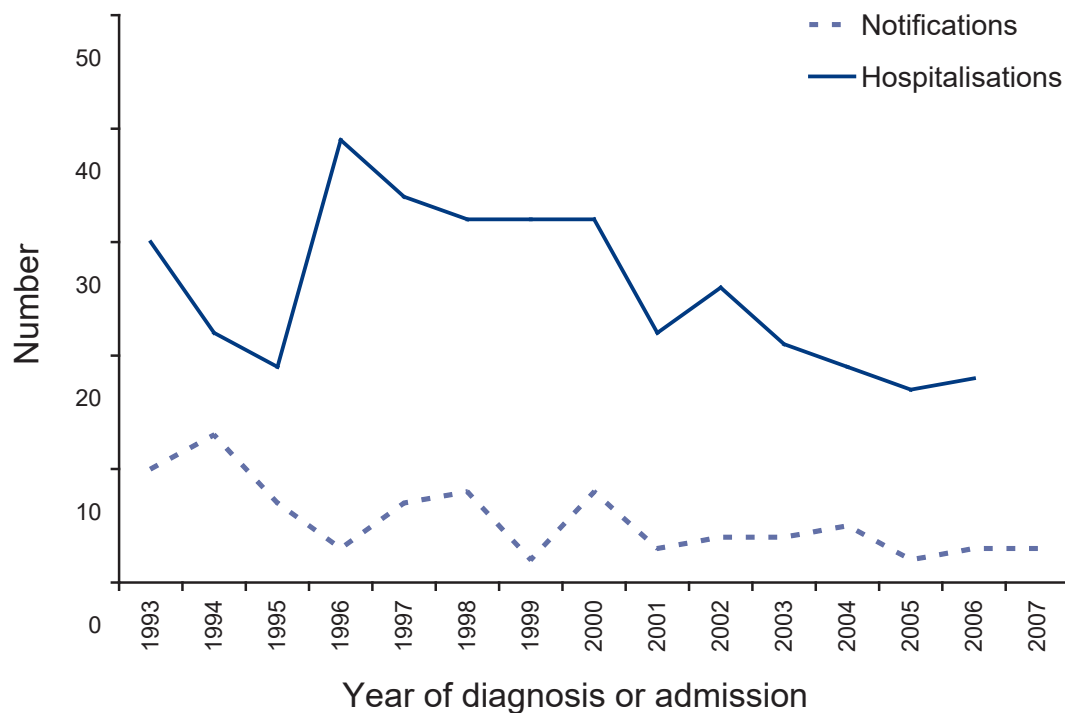
Severe morbidity and mortality

A total of 616 hospital bed days (average 308 per year) were recorded for patients with an ICD-10-AM code for tetanus over the 2 years July 2005 to June 2007. None of these were coded as obstetric tetanus (A34). Of the 36 separations, 20 (56%) had tetanus recorded as the principal diagnosis. The median length of stay in hospital was 3 days and varied with age. Adults aged ≥ 60 years had longer median lengths of stay (Table 3.15.1). In the review period 2005–2006, there was 1 death in the AIHW National Mortality Database with tetanus recorded as the underlying cause, in a person aged 87 years.

Age and sex distribution

The majority of both the notified (5/6; 83%) and hospitalised (17/36; 67%) cases were aged ≥ 60 years. The youngest notified and hospitalised cases were aged 15–24 years. There was no difference in gender among notified cases, but there were fewer hospitalised female patients, with a male:female ratio of 1.3:1. In the ≥ 70 years age group, 69% of the hospitalised cases (11/16) were males.

For both notifications and hospitalisations, rates increased with increasing age (Figure 3.15.2). Males aged ≥ 70 years had the highest average annual hospitalisation rate (0.67 per 100,000).

Figure 3.15.1: Tetanus notifications and hospitalisations, Australia, 1993 to 2007,* by year of diagnosis or admission


* Notifications where the date of diagnosis was between January 1993 and December 2007; hospitalisations where the date of admission was between July 1993 and June 2007. Hospitalisation data for each financial year are plotted according to the year in which the financial year began.

Table 3.15.1: Tetanus notifications, hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Notifications 2 years (2006–2007)		Hospitalisations 2 years (July 2005–June 2007)				LOS [†] per admission (days)		Deaths 2 years (2005–2006)	
	n	Rate [‡]	n	(§)	Rate [‡]	(§)	Median	(§)	n	Rate [‡]
0–4	0	–	0	(0)	–	(–)	–	(–)	0	–
5–14	0	–	0	(0)	–	(–)	–	(–)	0	–
15–24	1	0.02	2	(2)	0.04	(0.04)	n.p.	(n.p.)	0	–
25–59	0	–	17	(11)	0.08	(0.05)	2.0	(2.0)	0	–
60+	5	0.07	17	(7)	0.23	(0.10)	20.0	(16.0)	1	0.01
All ages	6	0.01	36	(20)	0.09	(0.05)	3.0	(2.0)	1	<0.005

* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

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

§ Principal diagnosis (hospitalisations).

n.p. Not published.

Geographical distribution

Notification and hospitalisation rates varied over time and between states and territories (Appendices 6.2 and 6.3). However, there were too few cases in each jurisdiction to identify any trends.

Vaccination status

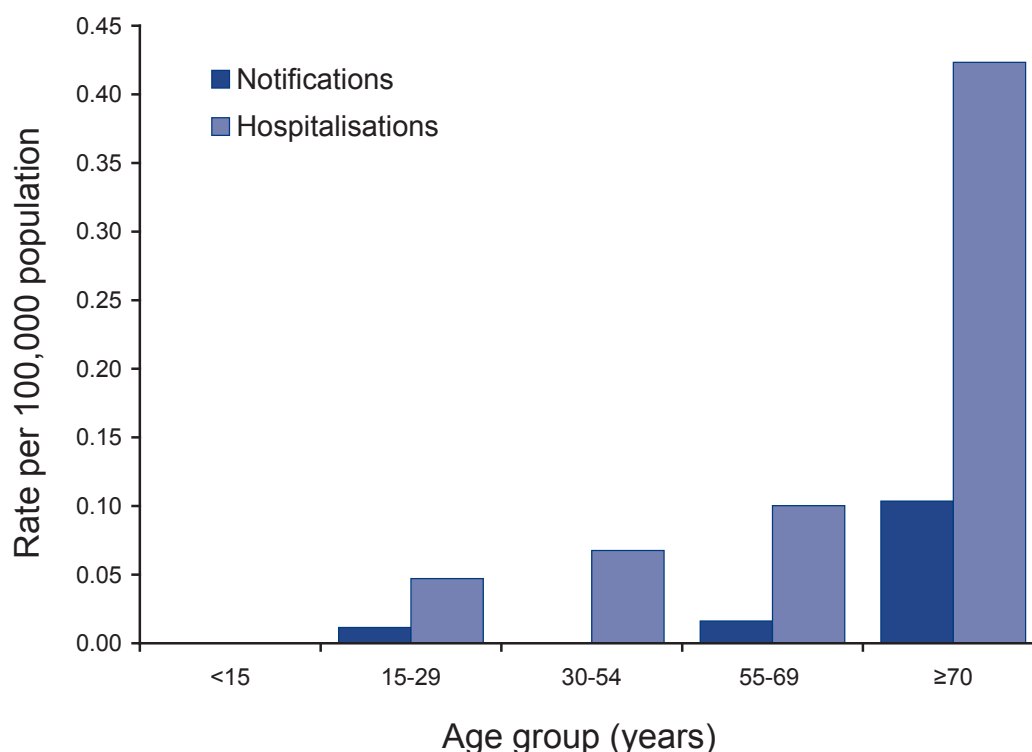
Two-thirds (4/6) of notified cases in the NNDSS in 2006–2007 were reported to be unvaccinated. A case aged 10–19 years was recorded as partially vaccinated for age. The final case had an unknown vaccination status.

Comment

There has been a downward trend in tetanus hospitalisation rates, which remain higher than notification rates. It is likely that this discrepancy is primarily due to under-reporting, as well as multiple hospital admissions for one case and coding errors.³ Coding errors may have resulted from misclassification of other conditions as tetanus, especially where tetanus was not the 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,⁴ 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 is also noted in Canada and Switzerland.^{5,6} The major impetus for tetanus immunisation in adults is injury,⁵ but tetanus occurs in cases with trivial or no known injury and the definition of a ‘tetanus prone’ wound is unclear.^{7,8} International serosurveys and the Australian National Serosurvey have shown progressively lower levels of tetanus antibody in older age groups, particularly in women.^{9–13} 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 on the disease burden in Australia. The current tetanus notification rate in Australia is similar to that in other developed countries.^{7,14–16} A tetanus booster is

Figure 3.15.2: Tetanus notification and hospitalisation rates, Australia, 2005 to 2007,* by age group



* Notifications where the date of diagnosis was between January 2006 and December 2007; hospitalisations where the date of separation was between July 2005 and June 2007.

recommended at the age of 50 years unless a booster has been documented within the previous 10 years.¹⁷ While 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, review of tetanus immunisation status at the time of annual influenza vaccination is clearly appropriate. Recent tetanus cases among injecting drug users in the United Kingdom and the USA demonstrate a younger population at risk from this preventable disease.^{15,18,19} Therefore, maintenance of immunity in young adults, through the scheduled booster dose at age 15–17 years, is also important.

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3.16 Varicella-zoster virus infection

The varicella-zoster virus (VZV) is so named because it causes two distinct illnesses: varicella (chickenpox), following primary infection, and herpes zoster (shingles), following reactivation of latent virus. Varicella is a highly contagious infection with an incubation period of 10–21 days, most commonly 14–16 days, after which a characteristic rash appears. Acute varicella may be complicated by secondary bacterial skin infections, haemorrhagic complications, cerebellitis, encephalitis, and viral and bacterial pneumonia.^{1,2} About 5% of infections are subclinical.³

In unvaccinated populations, varicella is primarily a childhood illness with more than 80%–90% of the population in temperate countries developing clinical or serological infection by adolescence.^{4,5} Varicella is generally a benign, self-limiting illness in children, but morbidity and mortality rates are higher in adults,⁶ at the extremes of ages, and in the immunocompromised.⁷ Varicella also causes congenital disease that can severely affect newborns of non-immune women.⁸ Varicella-zoster virus has been responsible for a significant disease burden, including hospitalisations and deaths, in Australia and New Zealand.⁹ Routine use of varicella vaccine in childhood was first recommended in Australia in 2003, and, in November 2005, varicella vaccination was funded under the National Immunisation Program for all children using a single dose schedule at 18 months of age, and in a school-based catch-up program at 10–13 years of age for those with no history of disease or previous vaccination.

Herpes zoster or shingles is a sporadic disease, caused by reactivation of latent VZV in sensory nerve ganglia. It is usually self-limiting and is characterised by severe pain with dermatomal distribution, sometimes followed by post-herpetic neuralgia which can be chronic and debilitating in the elderly.^{10,11} Although herpes zoster can occur at any age, most cases occur after the age of 50 with the incidence of complications also increasing with age.¹² However, children infected in utero or those who acquire varicella before the age of 1 year, and patients on immunosuppressive drugs or infected with human immunodeficiency virus, are also at increased risk of herpes zoster.^{13–15} A new herpes zoster vaccine which is over 60% effective in reducing the burden of herpes zoster and post-herpetic neuralgia¹⁶ has been available on the private market in Australia since 2008. The zoster vaccine is formulated from the same VZV strain (Oka-derived) as the licensed varicella (chickenpox) vaccines but is of higher potency (at least 14 times greater).

Case definitions

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 notification data

Varicella, herpes zoster and unspecified VZV-related disease have been notifiable in South Australia since 2002, and became nationally notifiable in 2006 in all Australian jurisdictions except New South Wales. However, data was not received from all notifying states until early 2008. The methods of reporting vary among states and territories.

South Australian notification data are included in this report. Cases notified in South Australia meet the following case definition: Clinical diagnoses of chickenpox or herpes zoster by a medical practitioner, and/or laboratory diagnoses of varicella-zoster virus infection. Due to the dual notification system in South Australia, laboratory reports are initially classified as 'Varicella virus (unclassified)' until the corresponding medical notification, if available, allows further classification of the disease as either chickenpox or herpes zoster.

Secular trends, varicella and herpes zoster

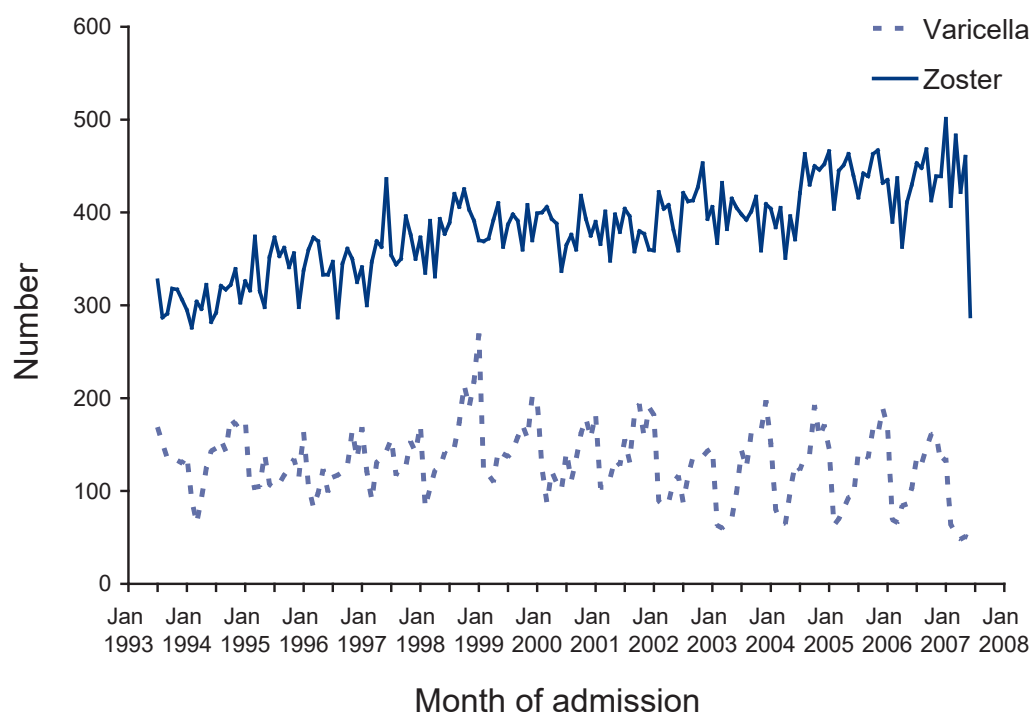
National notification data on varicella-zoster virus infections are not available for the reporting period of January 2006 to December 2007. (Notification data from South Australia are reported in a later section of this chapter.)

There were 2,787 hospitalisations (average annual hospitalisation rate 6.8 per 100,000) for varicella between July 2005 and June 2007. During this period, the median number of varicella hospitalisations per month was 132.5 (range 48–189) (Figure 3.16.1). The overall population rate and median monthly admissions were similar to that of the period July 2002 to June 2005 (rate 7.2 per 100,000; median 124 with range 60–197).¹⁷

Figure 3.16.1 shows that there were considerably more hospitalisations for herpes zoster than varicella. There were 10,506 hospitalisations (average annual hospitalisation rate 25.6 per 100,000 for all herpes zoster and 10.7 per 100,000 for herpes zoster as a principal diagnosis) between July 2005 and June 2007. The median number of hospitalisations per month with herpes zoster as one of the discharge diagnoses was 438 (range 363–501, excluding the last month of reporting with incomplete admission data) (Figure 3.16.1). The rate and median monthly admissions were similar to the period July 2002 to June 2005 (rate 25.0 per 100,000; median 410 with range 310–465).¹⁷

There is some pattern of disease peaks for varicella hospitalisations, particularly in more recent years, with most hospitalisations occurring between June and January (Figure 3.16.1).

Figure 3.16.1: Varicella and herpes zoster hospitalisations, Australia, 1993/1994 to 2006/2007,* by month of admission



* Hospitalisations where the date of admission was between July 1993 and June 2007.

Severe morbidity and mortality, varicella

For hospitalisations with an ICD-10-AM code for chickenpox, 15,375 hospital bed days (average 7,688 per year) were recorded during the 2 years July 2005 to June 2007. Of the 2,787 varicella hospitalisations, 1,907 (68%) had a principal diagnosis of varicella (average annual rate 4.6 per 100,000) (Table 3.16.1). Complications arising from varicella infection were recorded for 897 hospitalisations (32%). Of all varicella hospitalisations, 136 (4.9%) were coded as having varicella encephalitis or meningitis, and 249 (8.9%) were coded as having varicella pneumonia (Table 3.16.2). There were 9 hospitalisation records where both varicella pneumonia and varicella encephalitis/meningitis were coded as diagnoses. Although the hospitalisation rate was highest in the youngest age group (age 0–4 years), hospitalisations coded as chickenpox in people aged ≥ 60 years had the longest median length of stay (Table 3.16.1). The hospitalisation rate for varicella in children aged 0–4 years (34.9 per 100,000) declined significantly in comparison with the period July 2002 to June 2005 (42.1 per 100,000).¹⁷

Among the 24 hospitalisations with a varicella code where death was the mode of separation during the period July 2005 to June 2007, varicella was the principal diagnosis for 7 (all had either varicella encephalitis or varicella pneumonia, together with underlying medical conditions in other diagnostic codes [data not shown]). All but one of them were aged ≥ 60 years. For the majority of the remaining 17 varicella hospitalisations resulting in death, the principal or other diagnoses included underlying immunocompromising medical conditions, in particular malignancies, and the age ranged from 33–92 years (median 61 years).

There were 10 deaths recorded with varicella as the underlying cause in the AIHW National Mortality Database in the calendar years 2005 and 2006; 8 (80%) of them were people aged ≥ 60 years. In children aged 0–4 years, 1 death was recorded during this period; this was a decline in comparison with the period 2003–2004, during which there were 3 recorded deaths in this age group.¹⁷

Table 3.16.1: Varicella hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Hospitalisations 2 years (July 2005–June 2007)				LOS† per admission (days)		Deaths 2 years (2005–2006)	
	n	(‡)	Rate§	(‡)	Median	(‡)	n	Rate§
0–4	905	(631)	34.9	(24.3)	2.0	(2.0)	1	0.04
5–14	468	(324)	8.5	(5.9)	2.0	(2.0)	0	–
15–24	260	(182)	4.6	(3.2)	2.0	(2.0)	0	–
25–59	929	(653)	4.6	(3.3)	3.0	(2.0)	1	<0.005
60+	225	(117)	3.1	(1.6)	7.0	(6.0)	8	0.11
All ages	2,787	(1,907)	6.8	(4.6)	2.0	(2.0)	10	0.02

* Hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

‡ Principal diagnosis (hospitalisations).

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

Table 3.16.2: Selected indicators of severe morbidity* for hospitalised cases of varicella, Australia, 2005 to 2007,* by age group

Age group (years)	Varicella encephalitis or meningitis		Varicella pneumonia†	
	n	% of cases	n	% of cases
0–4	23	2.5	24	2.7
5–14	27	5.8	20	4.3
15–24	17	6.5	19	7.3
25–59	54	5.8	175	18.8
60+	15	6.7	11	4.9
All ages	136	4.9	249	8.9

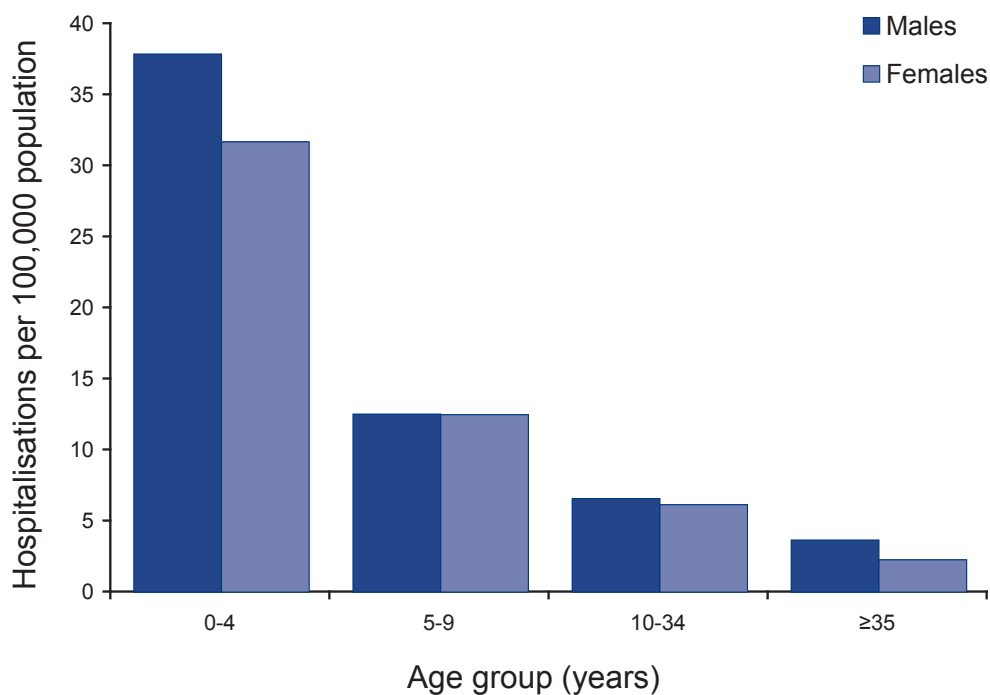
* Based on National Hospital Morbidity data where the date of separation was between July 2005 and June 2007.

† Nine hospitalisations with varicella pneumonia also had diagnostic codes of varicella encephalitis or meningitis.

Age and sex distribution, varicella

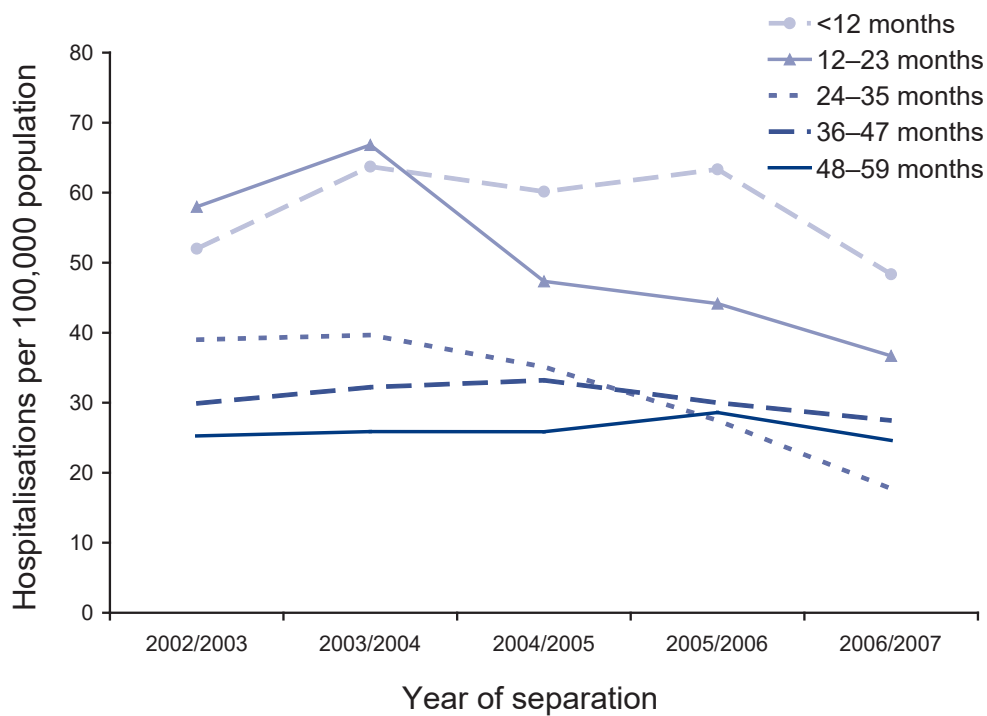
The highest rate of varicella hospitalisations continued to occur in the youngest age groups, especially the 0–4 years age group (Table 3.16.1; Figure 3.16.2), although the rate was reduced in this age group compared with the period July 2002 to June 2005. Within the 0–4 years age group (Figure 3.16.3), there was a sharp decrease in hospitalisations in the 12–23 months age group beginning in 2004/2005, a period which included the time when universal varicella immunisation at 18 months of age became recommended. This decreasing trend continued in 2005/2006 and 2006/2007. The hospitalisation rate decreased in infants aged <12 months during 2006/2007, but remained higher than in those aged 12–23 months, and was similar to the rate in infants <12 months of age in 2002/2003. Hospitalisation rates also declined in children aged 24–35 months over the 2-year period 2005/2006–2006/2007. The overall male:female ratio of hospitalisations was 1.2:1. Males predominated in all age groups, except young adults aged 15–29 years and elderly people aged ≥ 85 years, where there was a slight female predominance (data not shown). Of the 10 varicella deaths recorded in the AIHW National Mortality Database in 2005–2006, 9 were males.

Figure 3.16.2: Varicella hospitalisation rates, Australia, 2005/2006 to 2006/2007,* by age group and sex



* Hospitalisations where the date of separation was between July 2005 and June 2007.

Figure 3.16.3: Varicella hospitalisation rates, Australia, 2002/2003 to 2006/2007,* by age group (0–4 years) and year of separation



* Hospitalisations where the date of separation was between July 2002 and June 2007.

Geographical distribution, varicella

For the period 2005/2006–2006/2007, the Northern Territory had the highest average annual hospitalisation rate (12.7 per 100,000), with all other states recording average annual rates between 4.8 and 8.0 per 100,000. Hospitalisation rates were lowest in the Australian Capital Territory, South Australia and Tasmania (average annual rate 4.8, 4.8 and 4.9 per 100,000, respectively) (see also Appendix 6.3).

South Australian surveillance data, varicella

Figure 3.16.4 shows the notifications of varicella in South Australia by month of notification from January 2002 to December 2007. A total of 1,474 cases of chickenpox were notified in the period January 2006 to December 2007, an average annual rate of 46.8 per 100,000. Figure 3.16.5 shows the notifications by gender and age group for the current review period.

The highest rate of varicella notifications continued to occur in the youngest age groups, especially the 0–4 years age group (Figure 3.16.5), although there was a decline in the overall rate of notifications in the 0–4 years age group during 2006–2007 (306.9 per 100,000 during 2006–2007 compared with 377.1 per 100,000 during 2003–2005).

Severe morbidity and mortality, herpes zoster

For patients with an ICD-10-AM code for herpes zoster, 122,987 hospital bed days (average 61,494 per year) were recorded. Of the 10,506 herpes zoster hospitalisations, 4,410 (42%) had a principal diagnosis of herpes zoster (average annual rate 10.7 per 100,000) (Table 3.16.3). Complications arising from herpes zoster infection were recorded for 46% (4,783 of 10,506) of all hospitalisations with a herpes zoster diagnosis, and 58% (2,557 of 4,410) of hospitalisations where the principal diagnosis was herpes zoster. Of all the 10,506 herpes zoster hospitalisations, 169 (1.6%) were coded as having zoster encephalitis or meningitis, 105 (1.0%) were coded as having disseminated herpes zoster, and 1,172 (11.2%) were coded as having ocular complications (Table 3.16.4); <4% of the 4,783 hospitalisations with zoster complications had multiple diagnostic codes for zoster complications in their records. By far the greatest number of hospitalisations was in the oldest age group, who also had the longest median length of stay. There were 34 deaths recorded with herpes zoster as the underlying cause in the calendar years 2005 and 2006; 33 of them were people aged ≥ 60 years. The highest death rate was also recorded in people aged ≥ 60 years.

Table 3.16.3: Herpes zoster hospitalisations and deaths, Australia, 2005 to 2007,* by age group

Age group (years)	Hospitalisations 2 years (July 2005–June 2007)				LOS† per admission (days)		Deaths 2 years (2005–2006)	
	n	(‡)	Rate§	(‡)	Median	(‡)	n	Rate§
0–4	56	(41)	2.2	(1.6)	3.0	(3.0)	0	–
5–14	179	(109)	3.3	(2.0)	3.0	(2.0)	0	–
15–24	179	(91)	3.1	(1.6)	2.0	(2.0)	1	0.02
25–59	1,812	(841)	9.0	(4.2)	4.0	(3.0)	0	–
60+	8,280	(3,328)	114.2	(45.9)	7.0	(5.0)	33	0.46
All ages	10,506	(4,410)	25.6	(10.7)	6.0	(4.0)	34	0.08

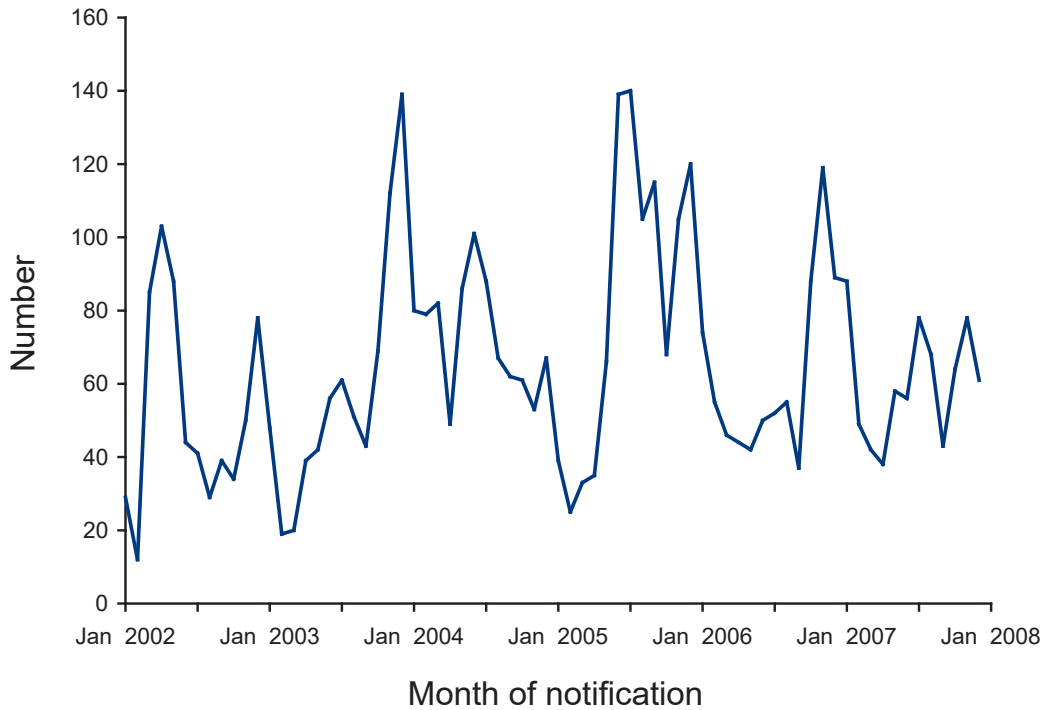
* Hospitalisations where the date of separation was between July 2005 and June 2007; deaths where the death was recorded between January 2005 and December 2006.

† LOS = length of stay in hospital.

‡ Principal diagnosis (hospitalisations).

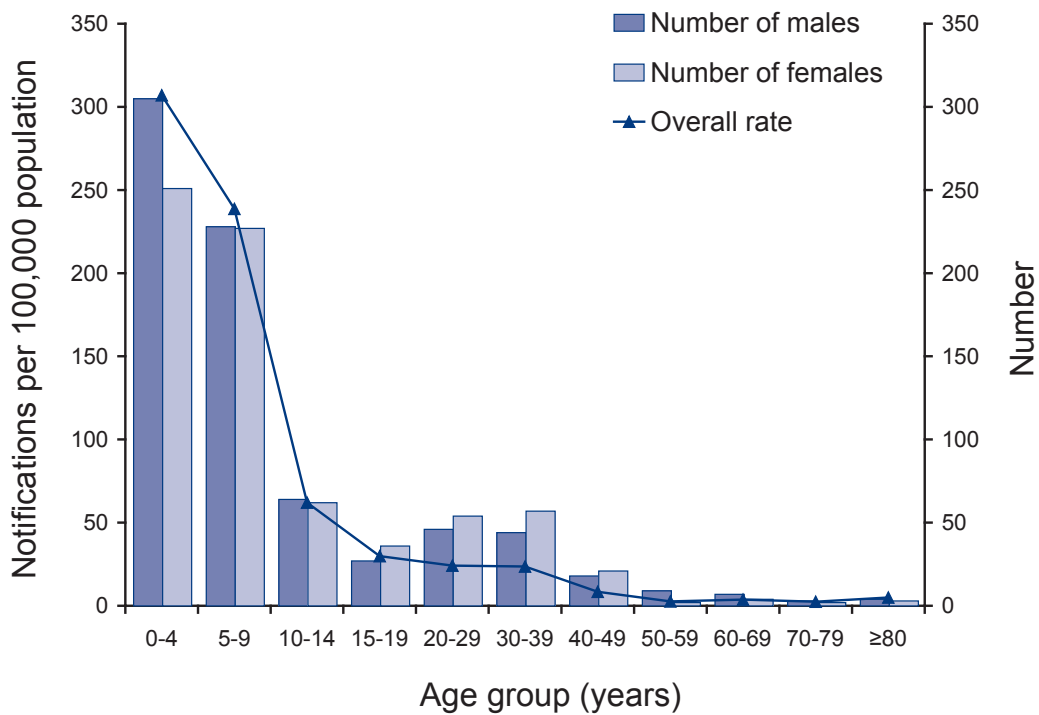
§ Average annual age-specific rate per 100,000 population.

Figure 3.16.4: Varicella notifications, South Australia, 2002 to 2007,* by month of notification



* Notifications where the date of notification was between January 2002 and December 2007.

Figure 3.16.5: Varicella notifications and rates, South Australia, 2006 to 2007,* by age group and sex



* Notifications where the date of notification was between January 2006 and December 2007.

Table 3.16.4: Indicators of severe morbidity* for hospitalised cases of herpes zoster, Australia, 2005 to 2007,* by age group

Age group (years)	Zoster encephalitis or meningitis†		Zoster with other nervous system involvement (without encephalitis or meningitis)†		Disseminated zoster†		Ocular complications of herpes zoster†	
	n	% of cases	n	% of cases	n	% of cases	n	% of cases
0–4	4	7.1	2	3.6	2	3.6	13	23.2
5–14	5	2.8	10	5.6	1	0.6	26	14.5
15–24	9	5.0	16	8.9	2	1.1	20	11.2
25–59	58	3.2	306	16.9	33	1.8	215	11.9
60+	93	1.1	2,419	29.2	67	0.8	898	10.8
All ages	169	1.6	2,753	26.2	105	1.0	1,172	11.2

* Based on National Hospital Morbidity data where the date of separation was between July 2005 and June 2007.

† A small proportion of hospitalisations had multiple diagnostic codes for zoster complications.

Age and sex distribution, herpes zoster

The highest number and rate of herpes zoster hospitalisations occurred in the oldest age groups, especially in the ≥ 60 years age group, where the rate was over 114 per 100,000 (Table 3.16.3). Across all ages, the male:female rate ratio of hospitalisations was 0.7:1. The male:female rate ratio for deaths due to herpes zoster was 0.7:1.

Geographical distribution, herpes zoster

For the period 2005/2006–2006/2007, South Australia had the highest crude average annual hospitalisation rate for herpes zoster (32.6 per 100,000), followed by Tasmania (27.7 per 100,000). The Northern Territory and the Australian Capital Territory had the lowest rates at around 20.6 per 100,000 (Appendix 6.3).

South Australian surveillance data, herpes zoster

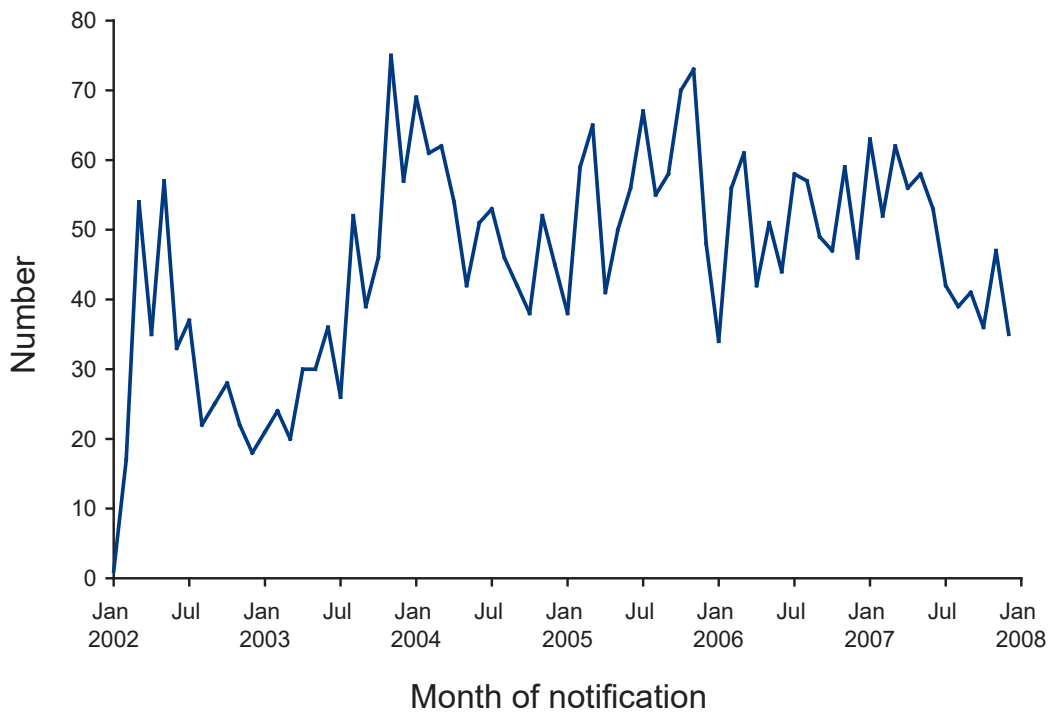
Figure 3.16.6 shows the notifications of herpes zoster by month from January 2002 to December 2007. A total of 1,188 cases were notified during 2006–2007, an average annual rate of 37.7 per 100,000. Figure 3.16.7 shows the notifications by gender and age group for the current review period.

Comment

The burden of disease caused by primary infection and reactivation of VZV is substantial. Severe herpes zoster occurs predominantly in the elderly, resulting in more hospitalisations than severe varicella, which is most common in young children, even when only the principal diagnosis is considered. The median length of stay for herpes zoster is also longer than for varicella, by 4 days.

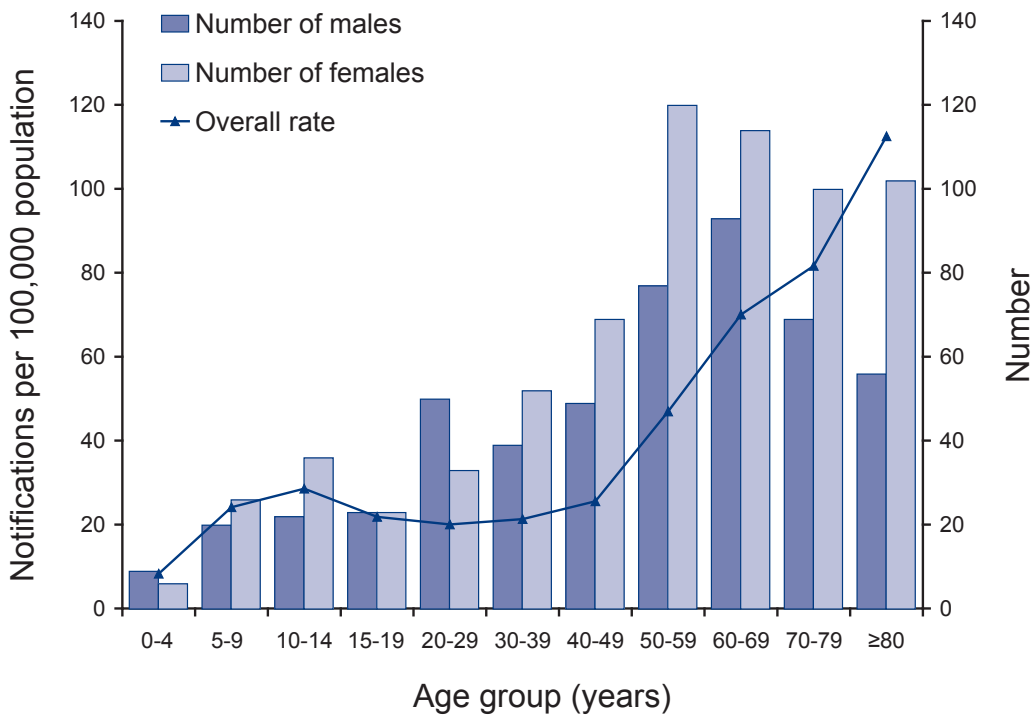
Although most varicella hospitalisations were in the youngest age group (905 episodes with a rate of 34.9 per 100,000 among those aged 0–4 years), hospitalisations coded as chickenpox in people aged ≥ 60 years (rate 3.1 per 100,000) had the longest median length of stay of 7 days (6 days for those with varicella as the principal diagnosis). Hospitalisations of patients aged ≥ 60 years coded as primary varicella infection may be more likely from re-activation of VZV, rather than primary disease, due to the prevalence of underlying medical conditions in this age group. The observation that the majority of varicella hospitalisations that eventuated in death occurred in older persons with underlying immunocompromising medical conditions supports this. The finding that the Northern Territory has the lowest overall hospitalisation rates for herpes zoster and the highest for varicella presumably reflects the younger age structure of the population of the Northern Territory.

Figure 3.16.6: Herpes zoster notifications, South Australia, 2002 to 2007,* by month of notification



* Notifications where the date of notification was between January 2002 and December 2007.

Figure 3.16.7: Herpes zoster notifications and rates, South Australia, 2006 to 2007,* by age group and sex



* Notifications where the date of notification was between January 2006 and December 2007.

During 2005/2006–2006/2007, 32% of hospitalised varicella cases had at least one recorded complication. For herpes zoster hospitalisations, 46% of those with zoster as any one of the diagnoses, and 58% of hospitalisations where zoster was the principal diagnosis, had at least one complication. This routine data is in agreement with a more detailed study that found that over 50% of hospitalisation episodes with a principal diagnosis of herpes zoster had documented complications, the majority of which were neurological.¹⁸ In that study, 16% had ophthalmic zoster, which is a serious complication that threatens vision.¹⁸

Early data suggested an impact of vaccine use in children aged 1–4 years in 2004/2005, the period during which vaccination was recommended but not funded.⁹ This trend continued in 2005/2006 and 2006/2007, particularly in children aged 18–23 months, whose hospitalisation rate became lower than that of the <1 year age group, and in those aged 24–35 months, of whom some would have been eligible for the funded vaccine. Varicella hospitalisations do not appear to have declined substantially in other age groups, including those <12 months of age. This suggests there is not yet evidence of a herd immunity effect, which was observed in the USA from approximately 5 years after commencement of their universal immunisation program in 1995. The decline in the incidence of varicella in the USA, using active surveillance, was in all age groups, but most marked in those aged 1–4 years.¹⁹

In 1952, Hope-Simpson proposed the hypothesis that exposure to varicella may boost immunity against herpes zoster.²⁰ There is increasing evidence to support that hypothesis, with two observational studies showing lower rates of herpes zoster in groups who have been exposed to varicella.^{21,22} If exposure to wild varicella provides boosting and protection against activation of herpes zoster, universal infant varicella vaccination and the subsequent decline in wild varicella may result in an increase in herpes zoster incidence among those previously infected.²³ Mathematical modelling has also suggested that widespread infant varicella vaccination might result in a significant increase in the incidence of herpes zoster, possibly over a 40-year period.²³ An Australian study, performed to assess the potential impact of universal varicella vaccination based on this hypothesis, suggested that total morbidity due to varicella and herpes zoster in Australia would decrease for the first 7 years of a population program, but, for 8–51 years after vaccination commenced, total morbidity was predicted to be higher than pre-vaccination levels.²⁴ However, this model assumed 90% vaccination coverage and 93% vaccine effectiveness. These predictions might not be correct, particularly given that overall vaccine coverage and effectiveness are now estimated to be less than that originally used in the model. 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.²⁵

The South Australian notification data show a declining notification rate among those aged 0–4 years, consistent with the decline in hospitalisations reported in this age group nationally. The reduction in reported cases may also have been on the background of an increase in the tendency to report cases which often occurs due to increasing awareness of disease among clinicians following introduction of recommendations for vaccination. Varicella cases were more frequently detected than herpes zoster cases through the South Australian surveillance system, in contrast to the national hospitalisation data. The gender-specific and age-specific notification data from South Australia show a similar epidemiology to the hospitalisation data, suggesting that hospitalisation data provides a useful measure of trends in varicella and herpes zoster.

The decline in hospitalisations and notifications in young children who are in the cohort eligible for funded varicella vaccine suggests an early impact of the vaccination program. This needs to be confirmed over time and in more age groups as vaccine coverage rises. Additionally, trends in disease epidemiology will need to be reviewed through notification data from other Australian states and territories, and via other mechanisms, such as reporting to the Australian Paediatric Surveillance Unit of data on neonatal, congenital and severe varicella infections.

The epidemiology of herpes zoster, as reflected in the national hospitalisation data and notification data from South Australia, does not appear to have changed over this review period. Surveillance for herpes zoster across all jurisdictions, together with ongoing monitoring of hospitalisation trends over time, should assist in identifying any changes in disease burden associated with ongoing use of the varicella vaccine and with the anticipated increase in uptake of the zoster vaccine over time in older Australians.

Acknowledgement: Dr Ann Koehler, Communicable Diseases Control Branch, Department of Health, South Australia, for provision of the South Australian notification data for varicella-zoster virus infections.

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4. Vaccination coverage

Previous reports in this series have included a section on childhood vaccination coverage in Australia.

Australian national vaccination coverage reports are now published separately – please refer to *Immunisation coverage annual report 2007*,¹ and *Immunisation coverage annual report 2008*.²

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5. Discussion

The period January 2006 to December 2007 was marked by continuing gains in the control of vaccine preventable diseases and further expansion of the National Immunisation Program (NIP) to include vaccines against rotavirus and human papillomavirus.

These vaccine policy and program changes represent a large investment in public health, and put Australia's funded immunisation programs at the forefront internationally, especially when the level and rapidity of population coverage are considered. This investment is set to increase even further in coming years, as new vaccines capable of inducing significant disease reductions in Australia, such as those against herpes zoster and invasive meningococcal disease due to serogroup B, become available. Like other industrialised countries, Australia faces the dual challenges of maintaining both high immunisation coverage and public confidence in immunisation, while making 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 routine data sources (notifications, hospitalisations and mortality) presented in this report and in the complementary reports on vaccination coverage^{1,2} provide an ongoing picture of progress across the spectrum of Australian immunisation activity.

A summary of the relative morbidity and mortality due to the diseases reported in the 3 years prior to the current report (a 3-year period within 2002–2005 that varied with different data sources) is shown in Table 5.1 and for the 2 years that followed (within 2005–2007) in Table 5.2. While the limitations of the data sources for notifications, hospitalisations and deaths should be borne in mind (see Chapter 2, Methods), and are especially evident for rare diseases or diseases for which a specific diagnostic test is lacking, 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 <5 years of age (the main target of the current childhood program), ongoing reductions in relative disease burden continued over the 2-year period under review. Hospitalisations due to measles and rubella have continued to decrease. There were substantial decreases in notifications and hospitalisations for meningococcal disease and pneumococcal disease in this age group in particular, but also in other age groups to a lesser extent. There was also a significant decline in hospitalisation rates for varicella disease in young children, although the vaccination program was only implemented in late 2005. Although both notification and hospitalisation rates for pertussis remain high overall, both decreased in this review period in children aged <5 years and particularly in adolescents, for whom a new immunisation program was introduced in 2004. Hospitalisation due to pertussis continues to be a significant burden in infants too young to be fully immunised. The notification and hospitalisation rates of mumps remained similar to the previous review period for children aged <5 years, but increased in young adults, attributable to an extended outbreak.

With respect to hospitalisations, influenza, varicella, meningococcal disease, pertussis and pneumococcal disease accounted for the largest numbers in those aged <5 years, and influenza and varicella outside this age group. The vaccine preventable diseases most frequently recorded as the underlying cause of death were influenza in adults and meningococcal disease in children aged <5 years.

The implications of these data are discussed below.

Table 5.1: Average annual morbidity and mortality from selected vaccine preventable diseases in Australia for 3 years within 2002–2005*

Disease†	Notifications 2003–2005 (average number)		Notification rate/100,000 (average rate)		Hospitalisations 2002/03–2004/05 (average number)		Hospitalisation rate/100,000 (average rate)		Deaths‡ 2003–2004 (average number)		Death rate/100,000 (average rate)	
	0–4 yrs	All ages	0–4 yrs	All ages	0–4 yrs	All ages	0–4 yrs	All ages	0–4 yrs	All ages	0–4 yrs	All ages
<i>Haemophilus influenzae</i> type b§	7.7	10.3§	0.6	0.3§	11.7	13.0§	0.9	0.3§	0.0	0.0	–	–
Hepatitis A	31.3	358.3	2.5	1.8	9.0	251.7	0.7	1.3	0.0	0.0	–	–
Hepatitis B¶	2.0	291.3	0.2	1.5	0.0	172.3	–	0.9	0.0	11.0	–	0.06
Influenza	1,057.7	3,395.0	83.7	16.9	1,038.7	3,038.7	82.1	15.3	2.5	50.5	0.20	0.25
Measles	7.7	49.3	0.6	0.3	12.3	31.3	1.0	0.2	0.0	0.0	–	–
Meningococcal disease	142.3	451.7	11.3	2.3	219.0	711.7	17.3	3.6	6.0	23.0	0.48	0.12
Mumps	8.0	139.7	0.6	0.7	3.7	46.0	0.3	0.2	0.0	0.5	–	<0.005
Pertussis	562.7	8,345.0	44.5	41.5	254.7	439.7	20.1	2.2	0.5	1.0	0.04	<0.005
Pneumococcal (invasive)**	570.7	2,101.0	45.2	10.5	277.0	1,038.3	21.9	5.2	5.5	19.0	0.44	0.10
Poliomyelitis††	0.0	0.0	–	–	0.3	1.7	<0.05	<0.05	0.0	0.0	–	–
Rubella	4.0	38.7	0.3	0.2	7.0	14.7	0.6	0.1	0.0	0.0	–	–
Tetanus	0.0	3.7	–	<0.05	0.0	22.0	–	0.1	0.0	0.0	–	–
Varicella	NN	NN	NN	NN	532.3	1,427.0	42.1	7.2	1.5	5.5	0.12	0.03
Zoster	NN	NN	NN	NN	36.0	4,975.0	2.8	25.0	0.0	19.0	–	0.10

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

† See Chapter 4 for case definitions for individual vaccine preventable disease.

‡ Includes only deaths with disease classified as the underlying cause of death.

§ Data for *Haemophilus influenzae* disease include only cases aged 0–14 years.

¶ Invasive *Haemophilus influenzae* type b disease for notifications. For hospitalisations and deaths, only includes *Haemophilus meningitis* cases.

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

** Includes pneumococcal meningitis and septicaemia only for hospitalisation and death data.

†† Principal diagnosis only for hospitalisations.

NN Not notifiable

Table 5.2: Average annual morbidity and mortality from selected vaccine preventable diseases in Australia for 2 years within 2005–2007

Disease†	Notifications 2006–2007 (average number)		Notification rate/100,000 (average rate)		Hospitalisations 2005/06–2006/07 (average number)		Hospitalisation rate/100,000 (average rate)		Deaths‡ 2005–2006 (average number)		Death rate/100,000 (average rate)	
	0–4 yrs	All ages	0–4 yrs	All ages	0–4 yrs	All ages	0–4 yrs	All ages	0–4 yrs	All ages	0–4 yrs	All ages
<i>Haemophilus influenzae</i> type b§	10.5	12.5§	0.8	0.3§	7.5	7.5§	0.6	0.2§	0.0	0.0	–	–
Hepatitis A	19.0	222.5	1.4	1.1	6.0	204.0	0.5	1.0	0.0	1.5	–	0.01
Hepatitis B¶	2.5	290.0	0.2	1.4	0.0	157.5	–	0.8	0.0	19.5	–	0.09
Influenza	1,452.0	6,827.5	110.0	32.7	620.5	2,222.0	47.8	10.8	2.5	27.5	0.19	0.13
Measles	16.5	68.5	1.3	0.3	8.0	28.0	0.6	0.1	0.0	0.0	–	–
Meningococcal disease	111.0	311.0	8.4	1.5	190.5	512.0	14.7	2.5	7.5	16.0	0.58	0.08
Mumps	9.5	428.5	0.7	2.1	4.5	53.5	0.4	0.3	0.0	0.5	–	<0.005
Pertussis	298.0	8,160.5	22.6	39.1	170.0	441.0	13.1	2.1	0.5	1.5	0.04	0.01
Pneumococcal (invasive)**	221.5	1,468.5	16.8	7.0	92.0	711.5	7.1	3.5	2.5	10.5	0.19	0.05
Poliomyelitis††	0.0	0.5	–	<0.05	0.0	0.5	–	<0.05	0.0	0.0	–	–
Rubella	4.0	47.0	0.3	0.2	4.5	9.0	0.4	<0.05	0.0	0.0	–	–
Tetanus	0.0	3.0	–	<0.05	0.0	18.0	–	0.1	0.0	0.5	–	<0.005
Varicella	NA	NA	NA	NA	452.5	1,393.5	34.9	6.8	0.5	5.0	0.04	0.02
Zoster	NA	NA	NA	NA	28.0	5,253.0	2.2	25.6	0.0	17.0	–	0.08

* Notification data, National Notifiable Diseases Surveillance System, January 2006–December 2007; hospitalisation data, Australian Institute of Health and Welfare (AIHW) National Hospital Morbidity Database, July 2005–June 2007; death data, AIHW National Mortality Database, January 2005–December 2006.

† See Chapter 4 for case definitions for individual vaccine preventable disease.

‡ Includes only deaths with disease classified as the underlying cause of death.

§ Data for *Haemophilus influenzae* disease include only cases aged 0–14 years.

|| Invasive *Haemophilus influenzae* type b disease for notifications. For hospitalisations and deaths, only includes *Haemophilus meningitis*.

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

** Includes pneumococcal meningitis and septicaemia only for hospitalisation and death data.

†† Principal diagnosis only for hospitalisations.

NA National notification data not available

5.1 Diseases with long-standing vaccination programs

Poliomyelitis, tetanus, diphtheria, pertussis, measles, rubella, mumps, invasive *Haemophilus influenzae* type b disease

Poliomyelitis, tetanus and diphtheria

Australia and the Western Pacific region have been declared polio free, but the overseas acquired case in 2007 highlights the ongoing need for high vaccination coverage and improved active surveillance for acute flaccid paralysis until global eradication 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. Tetanus continues to occur at a very low but declining rate with an average of 3 cases per year in 2006–2007 compared with 4 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 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 among travellers.

Pertussis

Pertussis remains a disease that is difficult to control and whose data are difficult to interpret. The period 2006–2007 saw lower notification and hospitalisation rates than the previous 3-year period which included an epidemic. Notification rates decreased in children and adolescents, with the greatest decrease seen in adolescents following the commencement of school-based vaccination in 2003–2004. However, there were marked increases in notification rates in adults and hospitalisation rates in the elderly. It remains unclear how much of these increases was due to false positive results from serology testing, an issue that was detected and rectified in 2006. There are limited data on morbidity and mortality from pertussis in older persons internationally, but the continuing high notification and hospitalisation rates, as well as 2 deaths, seen in Australia suggests that immunisation against pertussis as well as tetanus is an important consideration in persons >60 years of age. Currently immunisation is only recommended in the context of potential contact with young infants.³

Measles and rubella

During 2006–2007, measles and rubella notifications and hospitalisations continued at very low levels, similar to those in the previous 2–3 year period. A relatively large number of cases were reported in 2006 predominantly associated with an outbreak in a community opposed to immunisation. Most, but not all, other outbreaks in the period were linked to overseas acquired cases. This is still consistent with the elimination of endemic transmission in Australia as these outbreaks were not sustained. Unlike in previous reports, young adults did not have the highest notification rate during this study period. However, more data are required before their continued status as a susceptible age group can be ruled out. The maintenance of high 2-dose vaccine coverage in pre-school children remains the cornerstone of measles control, but increased emphasis on travel vaccination and monitoring of imported cases may require more attention in the future. For rubella, despite low numbers of reported infections, 2 cases of congenital rubella syndrome were reported. While there are high levels of rubella immunity in the general population, immigrant women from some countries, and Indigenous women in some communities, have been identified as having lower immunity and being therefore at higher risk of infection in pregnancy. The laboratory identification and typing of measles isolates is now a critical component of surveillance to demonstrate the absence of circulating endemic strains. This is likely to be required for rubella in the future when the goal of elimination is adopted.

Mumps

A considerable increase in mumps notifications was seen in 2006 and especially in 2007, predominantly in adolescents and young adults, many of whom were born at a time of relatively low vaccination coverage, a single-dose schedule, and reduced circulation of wild virus. While the largest numbers came from New South Wales, outbreaks were also seen in Indigenous communities in the Northern Territory and the Kimberley region of Western Australia, where a reduced response to an earlier dose given at 9 months of age in the Northern Territory in the 1980s and 1990s appears to be a contributing factor. However, as well as reduced immunity due to these factors, a substantial minority of Indigenous and non-Indigenous cases had received 2 doses of vaccine, at 12 months of age and in adolescence. This had also been observed in the USA and the UK previously,

where epidemics had peaked earlier than in Australia. This is probably attributable to lesser and less sustained antibody responses to mumps antigen compared with measles and rubella and may warrant consideration of an additional booster dose, at least in some settings.

Invasive *Haemophilus influenzae* type b disease

The virtual disappearance of invasive *Haemophilus influenzae* type b (Hib) disease among children aged <5 years has been an ongoing success story for vaccination with continued year-on-year falls in notifications of invasive Hib disease during 2006–2007. 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.

5.2 Diseases with universal vaccination programs commencing in the last decade

Hepatitis B, meningococcal disease, pneumococcal disease

Hepatitis B

At both national and jurisdictional levels, notification and hospitalisation rates for acute hepatitis B were stable over the 2006–2007 period. This followed an increase in notification rates between 1993 and 2001 and a subsequent decline, while hospitalisations had been steady since 1999. The previously observed declines in notifications were most marked in young adults aged 15–29 years. This decline may be partly related to the lower rates of intravenous drug use since 2000, consistent with trends in hepatitis C virus notifications,⁴ but the adolescent catch-up programs that commenced from the late 1990s are likely to also play a role in maintaining low levels of disease in young adults. An even greater and more uniform impact can be expected from universal infant vaccination which commenced in 2000.

Meningococcal disease

Substantial decreases in meningococcal notifications, hospitalisations and deaths were seen from 2003 to 2005, following the commencement in 2003 of the national routine vaccination program at 12 months of age and catch-up program for those aged <19 years. The decreases were seen in all target age groups (those aged <19 years in 2003), as well as in young adults outside the target age group, providing some evidence of continuing herd immunity. Those lows were sustained in the 2006–2007 review period. The reduction in notifications occurred in serogroup C disease, while notifications for serogroup B and other serogroups, mainly W₁₃₅ and Y, remained relatively stable. 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.⁵ However, availability of a universal serogroup B vaccine appropriate for use in Australia is still some way off.

Pneumococcal disease

The 2006–2007 period covered in this report includes the second and third years of the implementation of funded 7-valent pneumococcal conjugate vaccine (7vPCV) for all Australian infants. This period saw an overall decrease of 40% in invasive pneumococcal disease (IPD) notifications and a 35% decrease in pneumococcal disease hospitalisations, compared to the 3 years immediately before the introduction of the program. Decreases were most marked in vaccinated age groups (72%–74% reductions), but also occurred in non-vaccinated age groups, with decreases of 20%–40% in adults. This has been seen in some other countries, and suggests herd immunity through the prevention of nasopharyngeal carriage. Increases in non-7vPCV serotypes were seen, in particular a 130% increase in serotype 19A. This is similar to observations in the USA, where there have been substantial increases in serotype 19A, but the overall IPD rate remains substantially lower in the general population than in the pre-vaccine era.⁶

In the elderly, the impact of funded 23-valent pneumococcal polysaccharide vaccine (23vPPV), also from 2005, is less clear. Decreases in IPD have been limited to 7vPCV serotypes, while non-7vPCV serotypes have increased, including those in the 23vPPV. However, increases in 23vPPV serotypes have been less than non-23vPPV serotypes, suggesting a possible impact of the 23vPPV. Estimates of 23vPPV effectiveness against IPD have been satisfactory in the otherwise healthy elderly,⁷ although lower than for the 7vPCV in children.

While Aboriginal and Torres Strait Islander people are not a separate focus of this report, IPD rates have been substantially higher, and non-vaccine serotypes more prevalent, in this population than in the non-Indigenous population.⁸ In the USA, significant serotype replacement has been observed among Alaskan Natives, a population with high incidence of invasive pneumococcal disease.⁹ Serotype replacement has not been documented in Australian Indigenous people so far, but should be closely monitored. New recently licensed 10- and 13-valent conjugate pneumococcal vaccines may make further contributions to pneumococcal disease control in the future in both Indigenous and non-Indigenous Australians.

5.3 Diseases with vaccination programs targeted to specific population subgroups or settings

Influenza, Q fever, hepatitis A

Influenza

During this reporting period, annual seasonal influenza vaccination was recommended for all Australians aged ≥ 65 years, all Aboriginal and Torres Strait Islander people aged ≥ 15 years, and all individuals aged ≥ 6 months who were predisposed to severe influenza or its complications, with the intention of protecting those who were more vulnerable to severe outcomes from influenza during each season. Australians aged ≥ 65 years, and Aboriginal and Torres Strait Islander people who were aged ≥ 50 years or who had chronic medical conditions that predispose them to severe influenza, were eligible for annual vaccination under the NIP. The 2006–2007 period was notable for a substantially higher notification rate in 2007 than in any previous year since notifications became available in 2001. Hospitalisation data were not available for all of 2007 at the time of this analysis. With only one source of data it is difficult to determine the extent to which the increase reflects the relative severity of that influenza season. However, there are reasons to suspect that 2007 was a more severe influenza season than usual. The co-circulation of two common strains of virus, A/H3N2 and A/H1N1 (both antigenically different to the strains of the previous season), is unusual, as is an increase in reported deaths due to influenza, including seven in young children. However, an increase in testing for influenza after the reported child deaths is also expected to have contributed to the substantial increase in that year. The child deaths, three of which were in Western Australia, were followed by the decision to offer funded influenza vaccine to all children in Western Australia aged < 5 years from the following influenza season. Influenza vaccine has been recommended and funded for US children aged 6–23 months since 2003,^{10,11} and, since 2006, for children up to the age of 5 years.¹² In 2008, the US recommendation was further expanded to include all children aged 6 months to 18 years, to be commenced from the 2008/09 influenza season.¹³ Notwithstanding bias due to variable rates of documenting notifications or hospitalisations across different age groups, it is noteworthy that notification and hospitalisation rates in children < 5 years of age in Australia were approximately 4 times greater than in the elderly aged ≥ 60 years.

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) was implemented in various jurisdictions at different times between 2001 and 2006. The program 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). By 2005, Q fever notification and hospitalisation rates declined to record low levels. There has been no further decline during the 2006–2007 period, with some evidence of a slight increase. Limited data on vaccination status of cases, as well as the absence of risk factor data on routine notifications, make it difficult to assess the relative importance of a range of non-program and program factors potentially affecting disease rates. Relevant non-program factors include variations in drought conditions, livestock slaughtering patterns, and the role of less high-risk exposure occupations and settings. With respect to program factors, the extent to which a pause in vaccine manufacture during the reporting period affected availability and use of Q fever vaccine and whether this contributed to the small changes in Q fever notifications and hospitalisations observed is unclear.

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 continued to decline in the 2-year period examined in this report, a decline which has continued since the peaks in the late 1990s associated with a large foodborne outbreak and an epidemic in homosexual men and injecting drug users. Notification and hospitalisation rates are now at levels lower than any observed since the collection of current

data sources began in the early 1990s. 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 12–24 months in the Northern Territory, Queensland, South Australia and Western Australia. While this report is focused on patterns in the total population rather than Indigenous people in particular, a substantial impact from this program is expected, similar to that seen in north Queensland¹⁴ and targeted programs in the USA.¹⁵

5.4 Diseases with recent universal vaccination programs and limited national surveillance data

Varicella-zoster, rotavirus

Varicella-zoster

An early impact of the national varicella immunisation program appears to be evident in both national hospitalisation and South Australian notification data. The decline in varicella hospitalisations commenced from 2004, when vaccine was available but not funded, and continued after it was added to the National Immunisation Program in November 2005. The impact is most marked in children aged 12–23 months, but is also seen in children aged 24–47 months. This needs to be confirmed over time and in more age groups as vaccine coverage rises. Additionally, trends in disease epidemiology will need to be reviewed through notification data from other Australian states and territories, and via other mechanisms, such as reporting to the Australian Paediatric Surveillance Unit of data on neonatal, congenital and severe varicella infections.

The epidemiology of herpes zoster, as reflected in the national hospitalisation data and notification data from South Australia, does not appear to have changed over this review period. Studies based on modelling have predicted an increase in herpes zoster as the circulation of wild varicella infection declines following vaccination.¹⁶ In light of this and with the anticipated increase in uptake of the zoster vaccine over time in older Australians, more comprehensive monitoring of trends in zoster occurrence, particularly trends in age-specific hospitalisations, would be valuable for evaluation of the impacts of both the varicella program in children and any subsequent zoster program.

Rotavirus

In the pre-vaccine era rotavirus infection was the cause of approximately 10,000 hospitalisations per year in Australia.^{17,18} International estimates of the sensitivity of coding for rotavirus among gastroenteritis hospitalisations range from 25% to 47%.^{19–21} The data presented here for Australia are within that range. Publicly funded vaccine was available in the Northern Territory from October 2006 and nationally from July 2007. Therefore, the hospitalisation data presented here, up to June 2007, cover only the pre-vaccine period, with the exception of a 9-month period in the Northern Territory. The data show year-to-year variation in the pre-vaccine era, higher in 2006–2007 than the previous year. Notification data are available for this period only from the Northern Territory, and they show a lower rate in 2007 compared to 2006, possibly due to vaccine introduction, but more data are required before a firm conclusion can be drawn. Immunisation of infants in the first 6 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 is important to encourage timeliness of vaccination in order to ensure a maximal impact upon disease. Sufficient data should be available in the near future to provide a clearer picture of vaccine impact.

5.5 Vaccine preventable disease notification rates compared with other industrialised countries

The comparison of notification rates of five selected vaccine preventable diseases in Australia with Canada, the USA, New Zealand, England and Wales are shown in Table 5.3. Comparison should be cautious, as there were outbreaks of diseases at different times across the different countries, impacting particularly on some diseases, such as measles and mumps, across any one year. In addition, the tabulated rates are not age-standardised.

Rates of invasive Hib disease were comparably low in all countries, a reflection of the striking success of programs with this vaccine. The measles notification rate in Australia has been decreasing in the past decade from 1.7 per 100,000 in 1998 to <1 per 100,000 in 2007. Even though the crude incidence rate of measles is lower in North America than in Australia, there is evidence that Australia has satisfied multiple criteria that justify the formal

declaration of elimination of endemic transmission of measles.²² The mumps notification rate in the USA was also lower than the rates in New Zealand, Australia, and England and Wales. In contrast, the estimated incidences of measles and mumps were both high for England and Wales in comparison to the rest of the selected countries. This is consistent with the known outbreaks in that country, affecting susceptible individuals who were either unimmunised or partly immunised, especially older teenagers and young adults who, in the UK, received the measles–rubella vaccine, not MMR vaccine, in the school-based catch-up program conducted in the mid 1990s.^{23–26} MMR vaccination coverage has been lower in the UK than in the other countries mentioned above. The proportion of children aged 2–4 years who were susceptible to measles was estimated to have increased to 27% in 2004/2005,²⁷ and the incidence of measles in England and Wales for the period 2005–2008 was 35.0 per 100,000 population (95% CI 34.4–35.6) for children <15 years of age and 103.6 per 100,000 population (95% CI 99.7–107.4) for those <1 year of age.²⁸ Rubella notification rates were low in Australia, New Zealand, Canada and the USA. For pertussis, notification rates in Australia are high compared with all other countries. Although comparisons are difficult because of differences in case definition and in availability and use of laboratory tests (such as serology and PCR tests), the available data suggest that Australia has a uniquely high pertussis disease burden, as reflected in hospitalisation rates and seroepidemiologic studies.²⁹

5.6 Future surveillance priorities

This report demonstrates again the value of routinely collected data in monitoring the impact of Australia’s National Immunisation Program. Hospitalisation and death data from the Australian Institute of Health and Welfare (AIHW) National Hospital Morbidity Database and National Mortality Database provide important information on more severe disease, while the National Notifiable Diseases Surveillance System (NNDSS) of the Communicable Diseases Network Australia provides data that include less severe cases that are often more specific due to the common use of laboratory confirmation. National standardisation of NNDSS data, including case definitions and field codes, continues to expand. While general demographic data are generally complete, the completeness of some important fields in the notification records is more variable, such as

Table 5.3: Most recent* published notification rates† per 100,000 population for frequently notified vaccine preventable diseases, by country of residence

Disease	Australia	New Zealand ³⁰	USA ³¹	Canada ³²	England & Wales ^{33,34}
Hib disease	0.1	0.4	0.9	0.2	0.1
Measles	0.1	0.6	<0.05	<0.05	6.8
Mumps	2.8	1.7	0.3	0.1	13.3
Rubella	0.2	0.3	<0.05	<0.05	2.0
Pertussis	25.4	7.9	3.5	8.8	2.0

* Australia 2007; New Zealand 2007; USA 2007; Canada 2004; England and Wales 2007 (calculated from published number of notifications and population).

† Notification rates are not age-standardised.

Indigenous status, vaccination status and serogroup/subtype. This is often a reflection of the limited resources available to follow up cases to collect information, resources which are focused on particular diseases and/or age groups and geographic regions. ‘Enhanced’ surveillance data (additional clinical and/or laboratory data) are collected nationally for Hib and pneumococcal notifications, but not for other important diseases such as pertussis or meningococcal disease.

Two vaccines became available under the NIP shortly before (varicella vaccine) or within (rotavirus vaccine) the period covered by this report. For both varicella and rotavirus, notification status varied by jurisdiction, and there were insufficient data at national level to merit inclusion for this time period. Indeed, laboratory-based notification of cases has significant limitations for both these diseases, and other sources of routinely collected data, in particular emergency department presentations, have been harnessed in recent years to provide useful epidemiologic information on a number of infectious diseases. Especially with validation, emergency department presentations could be a valuable source of trends post program introduction for varicella, rotavirus and influenza.

Augmentation of routinely collected data by other more active surveillance systems is also likely to continue and increase. These systems currently include the Australian Paediatric Surveillance Unit, the Paediatric Active Enhanced Disease Surveillance pilot study, and general practice sentinel surveillance through the Australian Sentinel Practice Research Network. In the future, data quality and quantity, as well as efficiency of collection, could be improved through data linkage, by using information already collected on individuals from a range of sources. For example, linkage of the Australian Childhood Immunisation Register to morbidity and mortality data would greatly enhance the quality of vaccination status data for children as well as eliminating the resource-intensive re-collection of the information. Other electronic sources are likely to become important in the future as well, such as electronic health records, and increased availability of data linkage across multiple data sources offers great potential.

5.7 Future vaccination priorities

Table 5.2 summarises measures of morbidity and mortality for selected diseases for which there are vaccines currently on the NIP. Data for the previous period are summarised for comparison in Table 5.1. These tables reflect a highly effective NIP in general, with very low levels of morbidity and/or mortality for diseases like invasive Hib disease, measles, poliomyelitis, rubella and tetanus. For other diseases, higher morbidity rates are primarily due to serogroups/serotypes not contained in the available vaccine (meningococcal, pneumococcal) or in age groups outside those targeted (influenza). For mumps and measles, low vaccination coverage in previous years of people who are now young adults has resulted in higher incidence in this age group. The success of school-based programs in Australia is reflected by their impact on adolescent pertussis disease. The difficulties in reaching older age groups who have left school are well understood, and more creative solutions are needed to engage this population. These could include a greater emphasis on vaccination of people who intend to travel overseas, as the risk of acquiring diseases such as poliomyelitis, diphtheria, measles and mumps overseas is greater than at home. The possible incremental benefits and cost-effectiveness (which is beyond the scope of this report) arising from expanding eligibility of existing vaccines, for example influenza for all young children, regular boosters of pertussis vaccine for adults, and Q fever vaccine for those in moderate risk occupations or settings, should be considered.

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Appendix 6.1 Charts of historical notification data of selected vaccine preventable diseases in Australia

Figure 6.1.1: Diphtheria, 1917 to 2007*

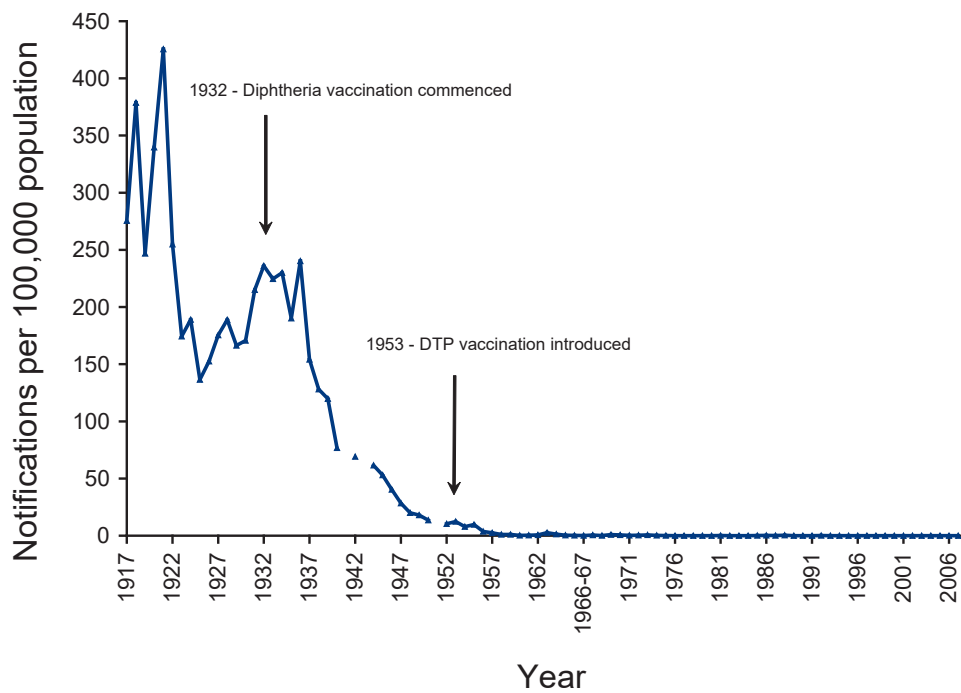
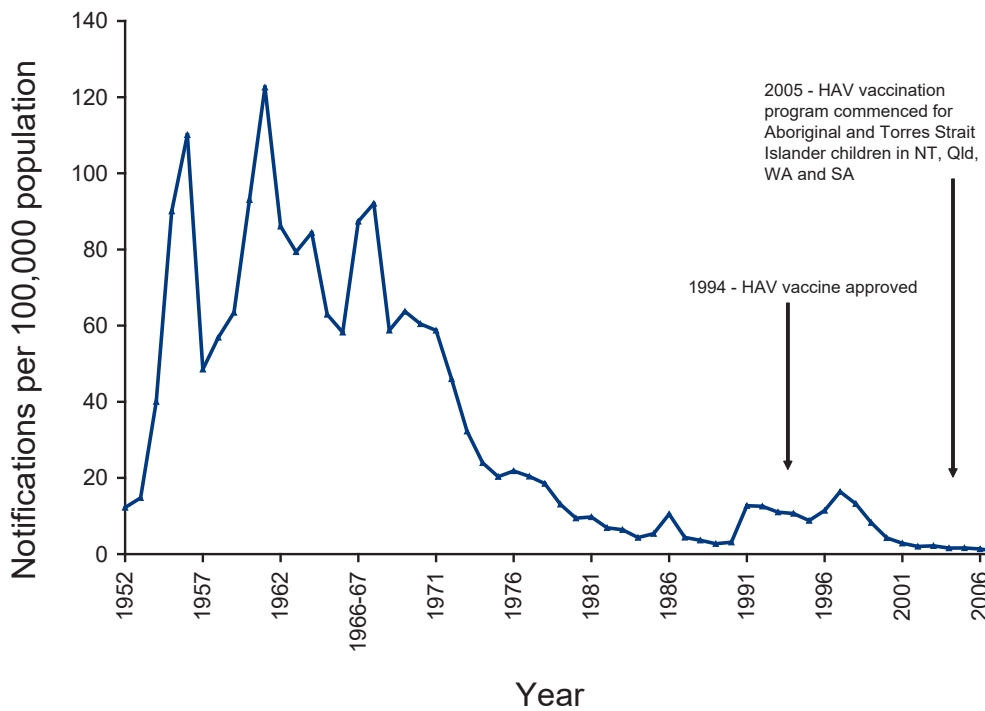


Figure 6.1.2: Hepatitis A, 1952 to 2007*



* Data are reported by calendar year from 1917–1963 and 1970–2007 and by financial year from 1963/1964–1969/1970. Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17(11):226–236.

Figure 6.1.3: Measles, 1917 to 2007*

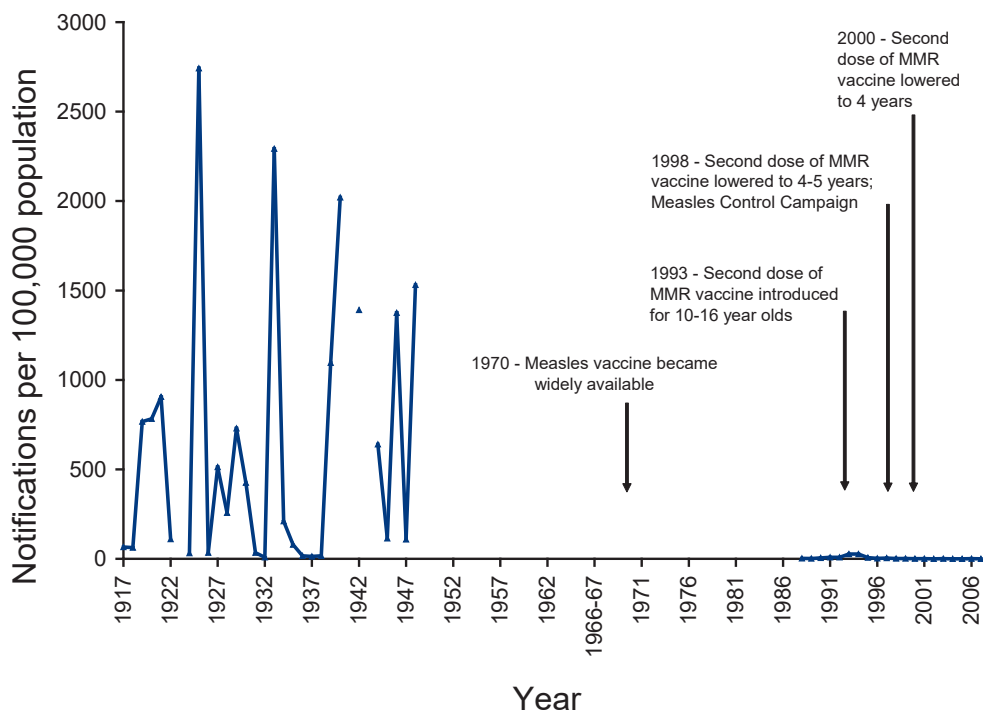
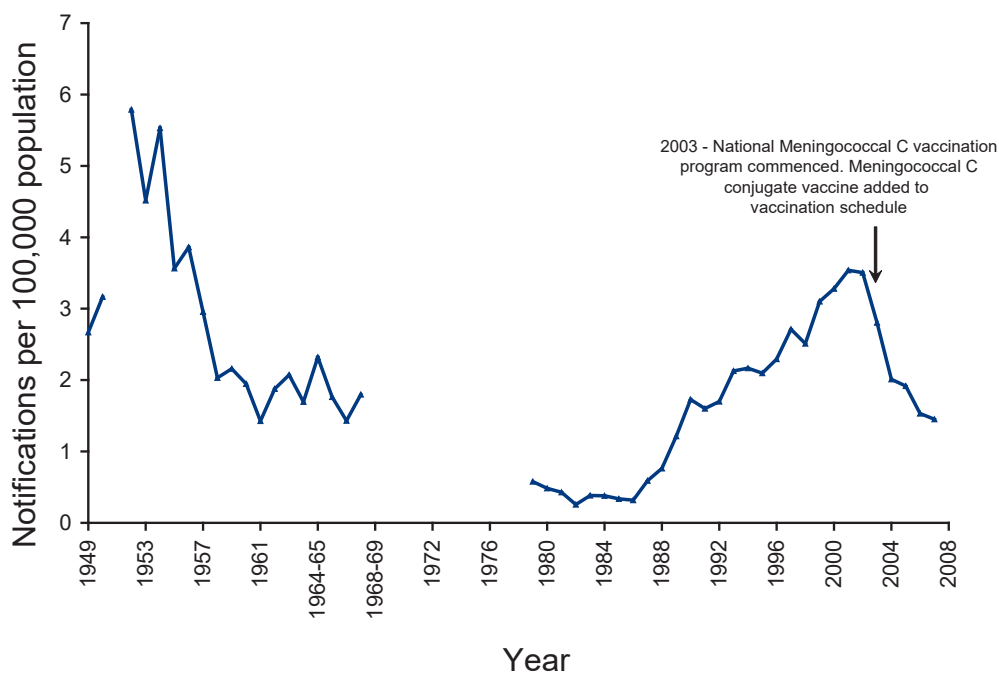


Figure 6.1.4: Meningococcal disease (invasive), 1949 to 2007*



* Data are reported by calendar year from 1917–1963 and 1970–2007 and by financial year from 1963/1964–1969/1970. Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17(11):226–236.

Figure 6.1.5: Mumps, 1932 to 2007*

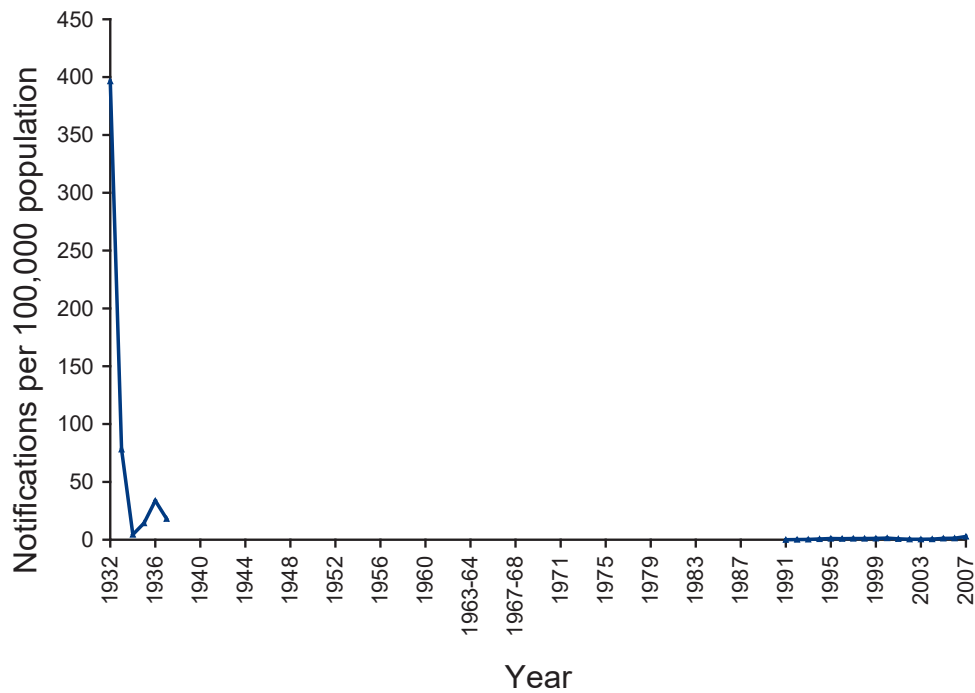
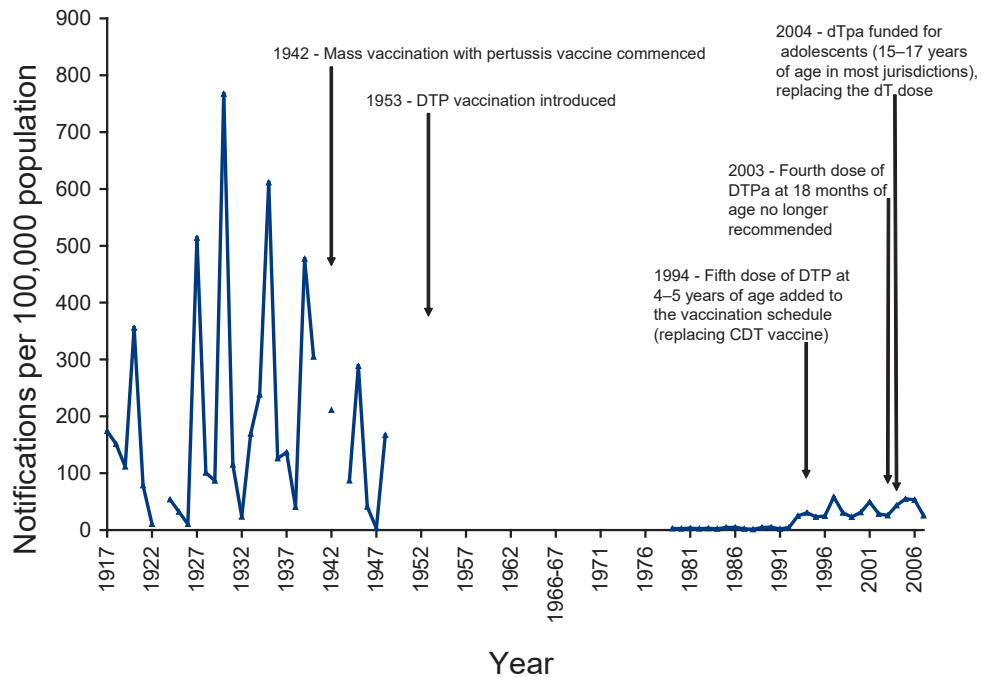


Figure 6.1.6: Pertussis, 1917 to 2007*



* Data are reported by calendar year from 1917–1963 and 1970–2007 and by financial year from 1963/1964–1969/1970. Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17(11):226–236.

Figure 6.1.7: Poliomyelitis, 1917 to 2007*

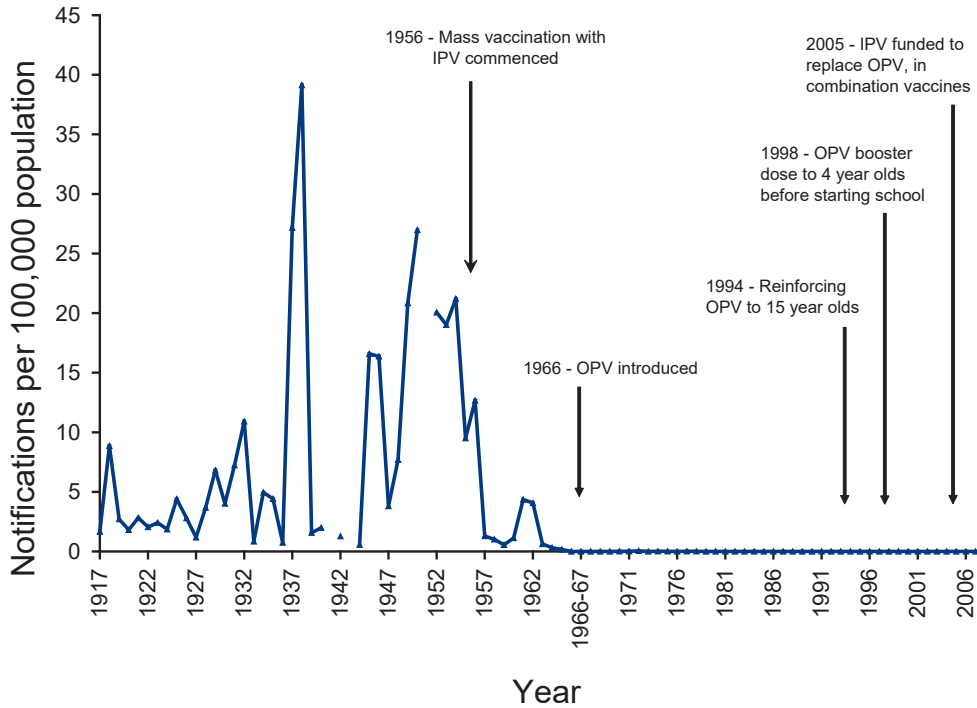
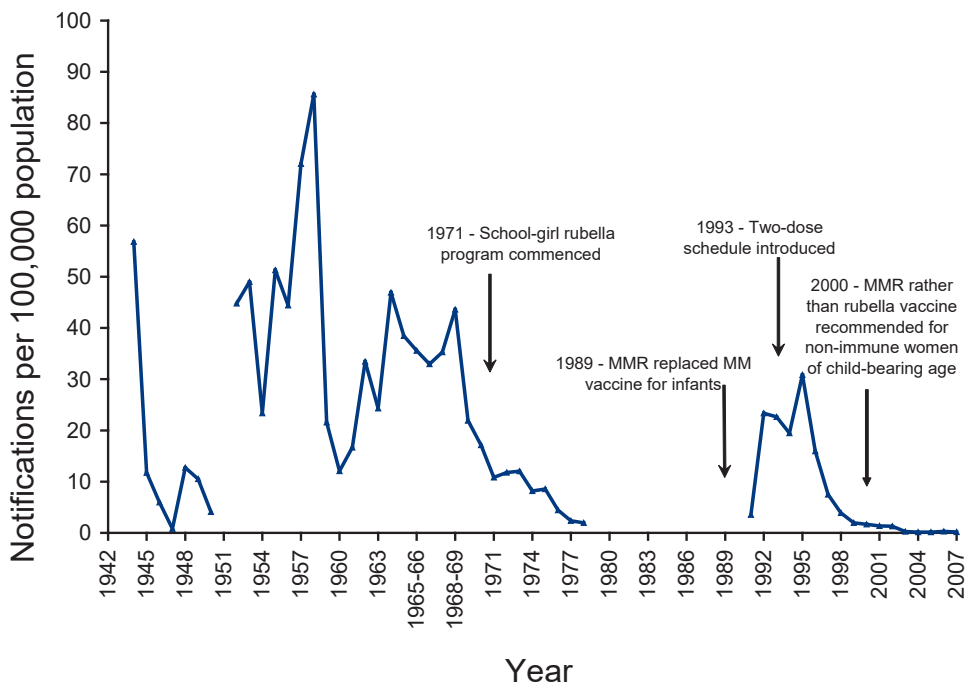
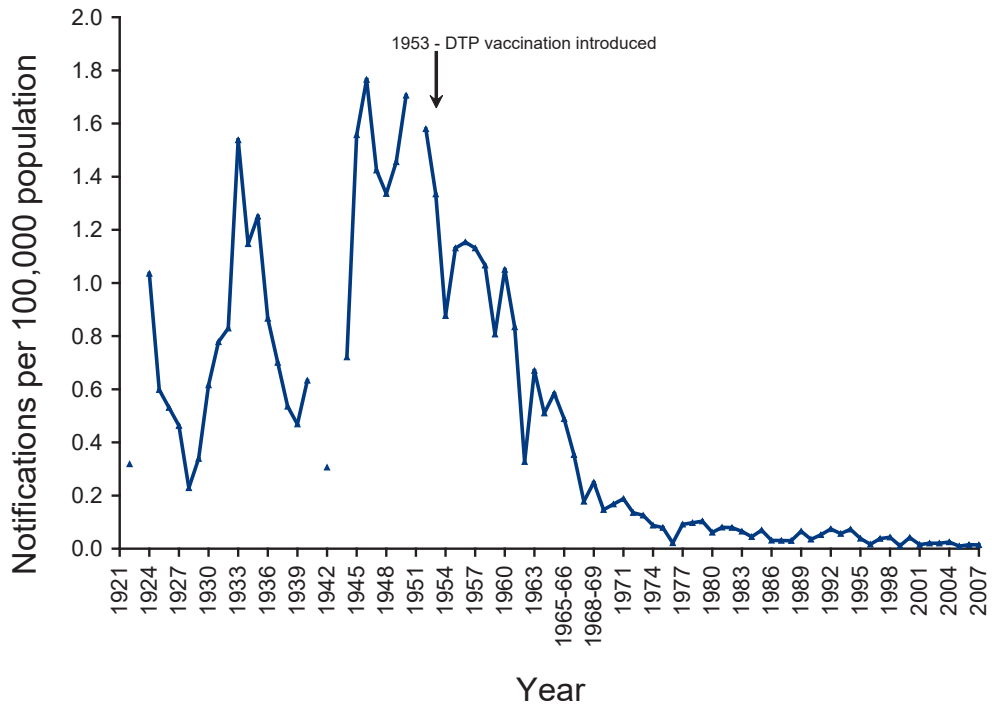


Figure 6.1.8: Rubella, 1942 to 2007*



* Data are reported by calendar year from 1917–1963 and 1970–2007 and by financial year from 1963/1964–1969/1970. Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17(11):226–236.

Figure 6.1.9: Tetanus, 1921 to 2007*



* Data are reported by calendar year from 1917–1963 and 1970–2007 and by financial year from 1963/1964–1969/1970. Source: Hall R. Notifiable disease surveillance, 1917 to 1991. *Commun Dis Intell* 1993;17(11):226–236.

Appendix 6.2 Notifications of selected vaccine preventable diseases, January 2002 to December 2007, by state or territory

Table 6.2: Notifications, January 2002 to December 2007, by state or territory and year

Disease*	Year	Number of notifications										Notification rate per 100,000 population									
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total		
Diphtheria	2002	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
	2003	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
	2004	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
	2005	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
	2006	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
	2007	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
	Total†	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
	Total	0	5	2	1	2	0	1	6	17	0	4	3.9	0.1	0.7	0	0.1	1.5	0.4		
<i>Haemophilus influenzae</i> type b meningitis (age <15 years only)	2002	0	4	2	3	1	0	1	1	12	0	3.9	0.4	0.3	0	0.1	0.2	0.3			
	2003	0	2	2	3	2	0	1	0	10	0	3.9	0.4	0.7	0	0.1	0	0.2			
	2004	0	4	1	2	0	0	2	0	9	0	3.9	0.4	0.7	0	0.1	0	0.2			
	2005	0	4	0	7	0	0	2	0	13	0	3.9	0.4	0.7	0	0.1	0	0.2			
	2006	0	4	2	2	1	0	1	2	12	0	3.9	0.4	0.7	0	0.1	0.5	0.3			
	2007	0	23	9	18	6	0	8	9	73	0	3.9	0.4	0.7	0	0.1	0.4	0.3			
	Total†	0	145	47	67	16	4	68	37	392	0	3.9	0.4	0.7	0	0.1	0.4	0.3			
	Total	8	124	43	48	13	14	89	95	431	2.5	2.2	23.6	1.8	1.1	0.8	1.4	1.9	2.0		
Hepatitis A	2002	5	137	14	27	11	1	71	57	319	1.5	1.9	21.5	1.3	0.8	2.9	1.8	4.9	2.2		
	2003	3	83	65	52	10	2	59	54	328	0.3	2.0	6.9	0.7	0.7	0.2	1.4	2.9	1.6		
	2004	1	95	30	31	8	4	44	68	281	0.9	1.2	31.5	1.3	0.6	0.4	1.2	2.7	1.6		
	2005	2	65	5	27	5	3	36	21	164	0.3	1.4	14.2	0.8	0.5	0.8	0.9	3.3	1.4		
	2006	20	649	204	252	63	28	367	332	1,915	0.6	0.9	2.3	0.6	0.3	0.6	0.7	1.0	0.8		
	2007	0	88	12	58	11	19	174	35	397	1.0	1.6	16.5	1.1	0.7	1.0	1.2	2.8	1.6		
	Total†	0	88	12	58	11	19	174	35	397	0	1.3	6.0	1.6	0.7	4.0	3.6	1.8	2.0		
	Total	0	74	15	39	10	10	152	45	345	0	1.1	7.5	1.0	0.7	2.1	3.1	2.3	1.7		
Hepatitis B (acute)	2002	5	53	8	52	8	17	110	28	281	1.5	0.8	4.0	1.3	0.5	3.5	2.2	1.4	1.4		
	2003	3	56	5	66	8	3	79	35	255	0.9	0.8	2.4	1.6	0.5	0.6	1.6	1.7	1.2		
	2004	6	53	11	50	7	9	107	50	293	1.8	0.8	5.2	1.2	0.4	1.8	2.1	2.4	1.4		
	2005	13	56	8	64	11	9	84	42	287	3.8	0.8	3.7	1.5	0.7	1.8	1.6	2.0	1.4		
	2006	27	380	59	329	55	67	706	235	1,858	1.4	0.9	4.8	1.4	0.6	2.3	2.3	2.0	1.5		
	2007	0	88	12	58	11	19	174	35	397	0	1.3	6.0	1.6	0.7	4.0	3.6	1.8	2.0		
	Total†	0	74	15	39	10	10	152	45	345	0	1.1	7.5	1.0	0.7	2.1	3.1	2.3	1.7		
	Total	5	53	8	52	8	17	110	28	281	1.5	0.8	4.0	1.3	0.5	3.5	2.2	1.4	1.4		

Table 6.2: Notifications, January 2002 to December 2007, by state or territory and year, continued

Disease*	Year	Number of notifications							Total	Notification rate per 100,000 population							Total		
		ACT	NSW	NT	Qld	SA	Tas	Vic		WA	ACT	NSW	NT	Qld	SA	Tas		Vic	WA
Influenza	2002	19	1,013	53	1,143	291	7	593	551	3,670	5.9	15.3	26.6	30.7	19.1	1.5	12.2	28.6	18.7
	2003	8	861	151	886	311	7	640	615	3,479	2.5	12.9	75.4	23.2	20.3	1.5	13.0	31.5	17.5
	2004	1	1,011	39	616	75	4	206	184	2,136	0.3	15.1	19.3	15.8	4.9	0.8	4.1	9.3	10.6
	2005	42	1,414	61	1,689	277	19	595	465	4,562	12.7	20.9	29.5	42.2	17.8	3.9	11.8	23.1	22.4
	2006	80	615	40	1,751	89	48	420	209	3,252	23.9	9.0	19.0	42.8	5.7	9.8	8.2	10.2	15.7
	2007	390	1,918	183	4,590	280	415	1,589	1,038	10,403	114.8	27.8	85.1	109.8	17.7	84.1	30.5	49.3	49.5
	Total†	521	5,819	474	9,532	1,032	493	3,450	2,511	23,832	39.1	21.4	56.8	58.9	16.5	25.2	16.9	30.8	29.0
	2002	0	8	0	8	2	0	14	0	32	-	0.1	-	0.2	0.1	-	0.3	-	-
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	0.2
2005	0	5	0	1	0	1	2	1	10	-	0.1	-	<0.05	-	0.2	<0.05	<0.05	<0.05	
2006	1	60	0	2	9	11	12	30	125	0.3	0.9	-	<0.05	0.6	2.2	0.2	1.5	0.6	
2007	0	4	0	4	1	0	2	1	12	-	0.1	-	0.1	0.1	-	<0.05	<0.05	0.1	
Total†	1	107	4	26	42	12	84	41	317	0.1	0.3	0.3	0.1	0.5	0.4	0.3	0.3	0.3	
Meningococcal disease	2002	6	217	9	122	30	26	211	68	689	1.9	3.3	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	5.9	2.1	0.8	3.7	1.6	2.0	2.0
	2005	8	139	11	62	26	10	89	46	391	2.4	2.1	5.3	1.5	1.7	2.1	1.8	2.3	1.9
	2006	5	107	6	68	20	5	85	21	317	1.5	1.6	2.8	1.7	1.3	1.0	1.7	1.0	1.5
	2007	3	112	6	75	15	5	69	20	305	0.9	1.6	2.8	1.8	0.9	1.0	1.3	0.9	1.5
	Total†	46	925	56	516	137	84	660	241	2,665	2.3	2.3	4.5	2.2	1.5	2.9	2.2	2.0	2.2
Mumps	2002	0	29	1	6	10	0	10	13	69	-	0.4	0.5	0.2	0.7	-	0.2	0.7	0.4
	2003	2	35	0	10	13	0	4	13	77	0.6	0.5	-	0.3	0.8	-	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	111	7	71	8	0	20	22	240	0.3	1.6	3.4	1.8	0.5	-	0.4	1.1	1.2
	2006	1	155	7	58	21	0	16	17	275	0.3	2.3	3.3	1.4	1.3	-	0.3	0.8	1.3
	2007	4	323	58	46	22	2	18	109	582	1.2	4.7	27.0	1.1	1.4	0.4	0.3	5.2	2.8
	Total†	11	718	73	208	78	2	71	184	1,345	0.6	1.8	5.9	0.9	0.8	0.1	0.2	1.5	1.1

Table 6.2: Notifications, January 2002 to December 2007, by state or territory and year, continued

Disease*	Year	Number of notifications							Total	Notification rate per 100,000 population							Total		
		ACT	NSW	NT	Qld	SA	Tas	Vic		WA	ACT	NSW	NT	Qld	SA	Tas		Vic	WA
Pertussis (all ages)	2002	55	2,012	37	1,853	473	37	868	234	5,569	17.1	30.3	18.6	49.8	31.1	7.8	17.8	12.2	28.3
	2003	357	2,772	5	716	232	133	626	255	5,096	109.7	41.5	2.5	18.8	15.1	27.8	12.7	13.1	25.6
	2004	124	3,568	27	1,035	994	37	871	2,097	8,753	37.9	53.2	13.4	26.5	64.5	7.7	17.5	105.8	43.5
	2005	315	5,804	92	1,776	1,510	33	1,155	516	11,201	95.4	85.9	44.5	44.3	97.2	6.8	22.9	25.6	54.9
	2006	258	4,916	96	2,172	2,179	41	1,066	264	10,992	77.2	72.1	45.6	53.1	138.9	8.4	20.8	12.8	53.1
	2007	95	2,090	27	1,535	375	25	1,049	133	5,329	28.0	30.3	12.6	36.7	23.7	5.1	20.2	6.3	25.4
	Total†	1,204	21,162	284	9,087	5,763	306	5,635	3,499	46,940	60.8	52.3	23.0	38.3	62.0	10.5	18.7	29.1	38.5
	Total†	36	1,296	36	474	144	30	353	474	2,843	29.2	49.9	33.9	30.4	26.6	16.5	18.8	61.6	36.7
Pneumococcal disease (invasive)*	2002	23	882	68	438	215	63	552	210	2,451	7.1	13.3	34.1	11.8	14.1	13.3	11.3	10.9	12.5
	2003	41	807	73	464	170	52	469	150	2,226	12.6	12.1	36.5	12.2	11.1	10.9	9.5	7.7	11.2
	2004	55	905	93	474	205	56	389	196	2,373	16.8	13.5	46.0	12.1	13.3	11.6	7.8	9.9	11.8
	2005	30	658	69	325	136	45	303	185	1,751	9.1	9.7	33.4	8.1	8.8	9.2	6.0	9.2	8.6
	2006	18	580	56	253	106	41	276	134	1,464	5.4	8.5	26.6	6.2	6.8	8.4	5.4	6.5	7.1
	2007	35	518	66	321	91	30	280	132	1,473	10.3	7.5	30.7	7.7	5.7	6.1	5.4	6.3	7.0
	Total†	202	4,350	425	2,275	923	287	2,269	1,007	11,738	10.2	10.7	34.4	9.6	9.9	9.9	7.5	8.4	9.6
	Total†	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Poliomyelitis	2002	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2003	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2004	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2005	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2006	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2007	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Total†	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Total†	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Q fever	2002	0	310	1	354	27	0	82	20	794	0	4.7	0.5	9.5	1.8	0	1.7	1.0	4.0
	2003	1	288	1	222	15	1	13	19	560	0.3	4.3	0.5	5.8	1.0	0.2	0.3	1.0	2.8
	2004	2	221	3	160	40	0	29	9	464	0.6	3.3	1.5	4.1	2.6	0	0.6	0.5	2.3
	2005	0	143	2	156	17	0	28	6	352	0	2.1	1.0	3.9	1.1	0	0.6	0.3	1.7
	2006	0	174	5	169	18	0	36	5	407	0	2.6	2.4	4.1	1.1	0	0.7	0.2	2.0
	2007	0	209	2	172	24	0	31	7	445	0	3.0	0.9	4.1	1.5	0	0.6	0.3	2.1
	Total†	3	1,345	14	1,233	141	1	219	66	3,022	0.2	3.3	1.1	5.2	1.5	<0.05	0.7	0.5	2.5
	Total†	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Table 6.2: Notifications, January 2002 to December 2007, by state or territory and year, continued

Disease*	Year	Number of notifications								Total	Notification rate per 100,000 population								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Rubella	2002	3	35	1	188	5	1	16	4	253	0.9	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	<0.05	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
	2006	0	37	0	12	2	0	6	2	59	-	0.5	-	0.3	0.1	-	0.1	0.1	0.3
	2007	2	8	0	14	1	0	7	3	35	0.6	0.1	-	0.3	0.1	-	0.1	0.1	0.2
	Total†	5	130	1	257	11	2	36	21	463	0.3	0.3	0.1	1.1	0.1	0.1	0.1	0.2	0.4
	Tetanus	2002	0	0	0	3	0	0	0	1	4	-	-	-	0.1	-	-	-	0.1
	2003	0	1	0	2	0	0	1	0	4	-	<0.05	-	0.1	-	-	<0.05	-	<0.05
	2004	0	0	0	3	2	0	0	0	5	-	-	-	0.1	0.1	-	-	-	<0.05
	2005	0	1	0	0	0	1	0	0	2	-	<0.05	-	-	-	0.2	-	-	<0.05
	2006	0	2	0	0	0	0	1	0	3	-	<0.05	-	-	-	<0.05	-	-	<0.05
	2007	0	2	0	0	0	1	0	0	3	-	<0.05	-	-	0.2	-	-	-	<0.05
	Total†	0	6	0	8	2	2	2	1	21	-	<0.05	-	<0.05	<0.05	0.1	<0.05	<0.05	<0.05

* See the corresponding disease chapter in Section 4 for case definitions.

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

‡ Invasive pneumococcal disease (IPD) notification data were based on the December 2008 dataset provided by NNDSS.

Appendix 6.3 Hospitalisations, July 2002 to June 2007, by state or territory and financial year of separation

Note: All Australian jurisdictions except New South Wales, Queensland and South Australia have required suppression for all cells with counts of less than 5 (but non-zero). In these tables, the exact count in each of these cells has been replaced by the symbol '<5' for these jurisdictions. Data in additional selected cells have to be suppressed (denoted 'n.p.') to prevent back calculation of the suppressed counts of data from the five jurisdictions.

Table 6.3: Hospitalisations, July 2002 to June 2007,* by state or territory and financial year of separation

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 disease‡ (age <15 years only)	02/03	0	3	0	2	2	0	0	7	14	-	0.2	-	0.3	0.7	-	-	1.7	0.4
	03/04	0	2	<5	6	4	0	0	<5	15	-	0.1	n.p.	0.8	1.4	-	-	n.p.	0.4
	04/05	0	2	<5	4	0	<5	<5	0	10	-	0.2	n.p.	0.5	-	n.p.	n.p.	-	0.2
	05/06	0	2	0	1	0	0	n.p.	n.p.	4	-	0.2	-	0.1	-	-	n.p.	n.p.	0.1
	06/07	0	1	<5	6	1	0	<5	0	11	-	0.1	n.p.	0.7	0.3	-	n.p.	-	0.3
	Total§	0	10	<5	19	7	<5	5	8	54	-	0.1	n.p.	0.5	0.5	n.p.	0.1	0.4	0.3
	Total	<5	112	24	61	29	n.p.	48	26	307	n.p.	1.7	12.0	1.6	1.9	n.p.	1.0	1.4	1.6
Hepatitis A	03/04	<5	87	n.p.	25	22	5	42	43	243	n.p.	1.3	n.p.	0.7	1.4	1.0	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
	05/06	<5	69	25	23	24	<5	55	24	224	n.p.	1.0	12.1	0.6	1.5	n.p.	1.1	1.2	1.1
	06/07	<5	74	<5	30	10	<5	46	18	184	n.p.	1.1	n.p.	0.7	0.6	n.p.	0.9	0.9	0.9
	Total§	10	414	77	177	93	16	248	128	1,163	0.6	1.2	7.6	0.9	1.2	0.7	1.0	1.3	1.2
	Total	<5	59	<5	25	4	6	75	20	194	n.p.	0.9	n.p.	0.7	0.3	1.3	1.5	1.0	1.0
	Total	0	50	<5	23	8	<5	62	18	169	-	0.7	n.p.	0.6	0.5	n.p.	1.3	0.9	0.8
Hepatitis B (acute) (principal diagnosis only)	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
	05/06	<5	57	<5	22	8	5	42	14	152	n.p.	0.8	n.p.	0.5	0.5	1.0	0.8	0.7	0.7
	06/07	<5	49	<5	17	15	<5	60	16	163	n.p.	0.7	n.p.	0.4	1.0	n.p.	1.2	0.8	0.8
	Total§	6	254	17	102	43	20	300	90	832	0.4	0.8	1.7	0.5	0.6	0.8	1.2	0.9	0.8
	Total	9	997	36	785	211	30	543	530	3,141	2.8	15.0	18.1	21.1	13.9	6.3	11.2	27.5	16.0
	Total	14	1,241	108	714	386	70	644	779	3,956	4.3	18.6	54.0	18.7	25.2	14.7	13.1	39.9	19.9
	Total	11	618	35	423	146	14	371	401	2,019	3.4	9.2	17.3	10.8	9.5	2.9	7.4	20.2	10.0
Influenza¶	05/06	17	720	21	590	263	30	511	441	2,593	5.1	10.7	10.2	14.7	16.9	6.2	10.1	21.9	12.7
	06/07	12	571	12	527	118	22	370	219	1,851	3.6	8.4	5.7	12.9	7.5	4.5	7.2	10.6	8.9
	Total§	63	4,147	212	3,039	1,124	166	2,439	2,370	13,560	3.8	12.3	20.8	15.6	14.6	6.9	9.8	23.8	13.5
	Total	0	11	0	0	2	<5	16	<5	32	-	0.2	-	-	0.1	n.p.	0.3	n.p.	0.2
	Total	<5	13	0	9	4	0	11	<5	39	n.p.	0.2	-	0.2	0.3	-	0.2	n.p.	0.2
	Total	<5	9	<5	1	3	<5	<5	<5	23	n.p.	0.1	n.p.	<0.05	0.2	n.p.	n.p.	n.p.	0.1
	Total	<5	24	0	0	4	0	5	<5	39	n.p.	0.4	-	-	0.3	-	0.1	n.p.	0.2
Measles	06/07	0	3	0	2	2	0	n.p.	<5	17	-	<0.05	-	<0.05	0.1	-	n.p.	n.p.	0.1
	Total§	<5	60	<5	12	15	<5	45	11	150	n.p.	0.2	n.p.	0.1	0.2	n.p.	0.2	0.1	0.1

Table 6.3: Hospitalisations, July 2002 to June 2007,* by state or territory and financial year of separation, 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	02/03	<5	277	10	195	40	n.p.	243	95	883	n.p.	4.2	5.0	5.2	2.6	n.p.	5.0	4.9	4.5
	03/04	19	250	14	152	57	24	189	64	769	5.8	3.7	7.0	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	5.9	2.2	1.7	3.7	1.7	2.4	2.4
	05/06	6	179	11	111	56	18	144	60	585	1.8	2.6	5.3	2.8	3.6	3.7	2.9	3.0	2.9
	06/07	n.p.	152	8	96	28	<5	113	35	439	n.p.	2.2	3.8	2.3	1.8	n.p.	2.2	1.7	2.1
	Total§	46	1,055	55	640	207	81	774	301	3,159	2.8	3.1	5.4	3.3	2.7	3.4	3.1	3.0	3.1
		02/03	0	19	<5	9	3	0	10	<5	45	-	0.3	n.p.	0.2	0.2	-	0.2	n.p.
Mumps	03/04	0	16	<5	9	1	<5	11	6	45	-	0.2	n.p.	0.2	0.1	n.p.	0.2	0.3	0.2
	04/05	0	15	n.p.	9	4	<5	12	7	48	-	0.2	n.p.	0.2	0.3	n.p.	0.2	0.4	0.2
	05/06	0	13	n.p.	7	0	0	9	<5	32	-	0.2	n.p.	0.2	-	-	0.2	n.p.	0.2
	06/07	0	37	0	11	4	<5	18	<5	75	-	0.5	-	0.3	0.3	n.p.	0.4	n.p.	0.4
	Total§	0	100	<5	45	12	<5	60	22	245	-	0.3	n.p.	0.2	0.2	n.p.	0.2	0.2	0.2
	02/03	<5	111	<5	131	29	6	57	22	359	n.p.	1.7	n.p.	3.5	1.9	1.3	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
Pertussis (all ages)	04/05	n.p.	179	13	87	89	<5	81	129	587	n.p.	2.7	6.4	2.2	5.8	n.p.	1.6	6.5	2.9
	05/06	<5	177	15	144	60	<5	40	17	457	n.p.	2.6	7.3	3.6	3.9	n.p.	0.8	0.8	2.2
	06/07	0	198	<5	95	63	<5	57	9	425	-	2.9	n.p.	2.3	4.0	n.p.	1.1	0.4	2.1
	Total§	17	806	34	533	260	31	308	212	2,201	1.0	2.4	3.3	2.7	3.4	1.3	1.2	2.1	2.2
	Total§	10	410	18	236	59	21	203	147	1,104	9.8	19.0	20.4	18.3	13.1	13.9	13.1	23.2	17.2
	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	22.5	4.2	5.1	2.3	5.2	4.4	5.2
Pneumococcal disease (invasive)††	04/05	15	336	45	142	71	24	222	114	969	4.6	5.0	22.3	3.6	4.6	5.0	4.5	5.7	4.8
	05/06	8	286	36	89	62	14	163	80	738	2.4	4.2	17.4	2.2	4.0	2.9	3.2	4.0	3.6
	06/07	8	219	40	102	56	17	180	63	685	2.4	3.2	19.0	2.5	3.6	3.5	3.5	3.1	3.3
	Total§	72	1,593	208	670	340	93	1,102	460	4,538	4.4	4.7	20.4	3.4	4.4	3.9	4.4	4.6	4.5

Table 6.3: Hospitalisations, July 2002 to June 2007,* by state or territory and financial year of separation, 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
Poliovirus**	02/03	n.p.	26	<5	3	0	0	6	0	39	n.p.	0.4	n.p.	0.1	-	-	0.1	-	0.2
	03/04	0	16	0	1	0	0	<5	<5	20	-	0.2	-	<0.05	-	-	n.p.	n.p.	0.1
	04/05	0	0	0	0	0	0	n.p.	n.p.	1	-	-	-	-	-	-	n.p.	n.p.	<0.05
	05/06	0	2	0	0	0	0	0	0	2	-	<0.05	-	-	-	-	-	-	<0.05
	06/07	n.p.	5	0	0	0	n.p.	0	0	6	n.p.	0.1	-	-	-	n.p.	-	-	<0.05
	Total§	<5	49	<5	4	0	<5	8	<5	68	n.p.	0.1	n.p.	<0.05	-	n.p.	<0.05	n.p.	0.1
	Total§	0	6	0	0	0	0	0	0	6	-	<0.05	-	-	-	-	-	-	<0.05
Q fever	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	5	238	-	1.7	n.p.	2.3	0.6	1.3	0.3	0.3	1.2
	04/05	0	74	<5	51	10	<5	14	<5	155	-	1.1	n.p.	1.3	0.6	n.p.	0.3	n.p.	0.8
	05/06	0	67	<5	74	10	0	18	<5	173	-	1.0	n.p.	1.8	0.6	-	0.4	n.p.	0.8
	06/07	n.p.	75	<5	77	12	0	16	5	187	n.p.	1.1	n.p.	1.9	0.8	-	0.3	0.2	0.9
	Total§	<5	441	11	395	58	n.p.	74	21	1,012	n.p.	1.3	1.1	2.0	0.8	n.p.	0.3	0.2	1.0
	Total§	91	1,710	199	900	337	49	472	313	4,071	28.2	25.8	99.8	24.2	22.2	10.4	9.7	16.3	20.7
Rotavirus	03/04	39	1,575	231	567	556	53	436	346	3,803	12.0	23.6	115.4	14.9	36.3	11.1	8.9	17.7	19.1
	04/05	45	972	163	571	413	116	344	235	2,859	13.7	14.5	80.6	14.6	26.8	24.0	6.9	11.9	14.2
	05/06	73	1,465	232	754	477	42	415	287	3,745	22.1	21.7	112.3	18.8	30.7	8.6	8.2	14.2	18.3
	06/07	64	1,839	191	1,076	553	116	750	268	4,857	19.1	27.0	90.7	26.3	35.3	23.7	14.6	13.0	23.5
	Total§	312	7,561	1,016	3,868	2,336	376	2,417	1,449	19,335	19.0	22.5	99.7	19.8	30.3	15.6	9.7	14.6	19.2
	02/03	<5	9	0	2	1	<5	5	<5	21	n.p.	0.1	-	0.1	0.1	n.p.	0.1	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
Rubella	04/05	0	1	0	0	0	0	<5	<5	5	-	<0.05	-	-	-	-	n.p.	n.p.	<0.05
	05/06	<5	4	0	1	0	0	<5	<5	11	n.p.	0.1	-	<0.05	-	-	n.p.	n.p.	0.1
	06/07	0	2	0	3	1	n.p.	n.p.	0	7	-	<0.05	-	0.1	0.1	n.p.	n.p.	n.p.	<0.05
	Total§	<5	23	0	8	4	<5	15	7	62	n.p.	0.1	-	<0.05	0.1	n.p.	0.1	n.p.	0.1
	Total§	<5	9	0	2	1	<5	5	<5	21	n.p.	0.1	-	0.1	0.1	n.p.	0.1	n.p.	0.1

Table 6.3: Hospitalisations, July 2002 to June 2007,* by state or territory and financial year of separation, 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**	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	2	<5	<5	0	17	-	0.1	-	0.1	0.1	n.p.	n.p.	-	0.1
	04/05	0	5	0	10	6	n.p.	n.p.	0	22	-	0.1	-	0.3	0.4	n.p.	n.p.	-	0.1
	05/06	0	11	0	0	0	<5	<5	0	17	-	0.2	-	-	-	n.p.	n.p.	-	0.1
	06/07	0	3	<5	12	2	0	n.p.	0	19	-	<0.05	n.p.	0.3	0.1	-	n.p.	-	0.1
	Total§	0	32	<5	33	13	<5	13	6	102	-	0.1	n.p.	0.2	0.2	n.p.	0.1	0.1	0.1
Varicella	02/03	6	428	18	244	91	34	307	130	1,258	1.9	6.5	9.0	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	13.5	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	8.9	7.1	7.3	9.7	6.3	9.9	7.3
	05/06	17	601	23	271	87	31	299	182	1,511	5.1	8.9	11.1	6.8	5.6	6.4	5.9	9.0	7.4
	06/07	15	438	30	298	63	17	270	145	1,276	4.5	6.4	14.2	7.3	4.0	3.5	5.3	7.0	6.2
	Total§	63	2,527	116	1,446	476	161	1,480	799	7,068	3.8	7.5	11.4	7.4	6.2	6.7	5.9	8.0	7.0
Zoster	02/03	57	1,619	26	857	487	176	1,222	448	4,892	17.7	24.4	13.0	23.1	32.0	37.2	25.1	23.3	24.9
	03/04	56	1,545	27	817	456	124	1,246	436	4,707	17.2	23.2	13.5	21.4	29.8	26.0	25.3	22.3	23.7
	04/05	33	1,971	36	951	459	115	1,275	487	5,327	10.1	29.4	17.8	24.3	29.8	23.8	25.6	24.6	26.5
	05/06	64	1,751	35	843	487	113	1,384	444	5,121	19.4	25.9	16.9	21.0	31.4	23.2	27.4	22.0	25.1
	06/07	73	1,788	51	882	529	157	1,405	500	5,385	21.8	26.2	24.2	21.6	33.7	32.0	27.4	24.3	26.0
	Total§	283	8,674	175	4,350	2,418	685	6,532	2,315	25,432	17.3	25.8	17.2	22.3	31.3	28.4	26.2	23.3	25.2

* Note that total hospitalisations potentially include re-admissions of the same case, inter-hospital transfer, and change of care type. Reported data may also contain possible coding errors.

† See the corresponding disease chapter in Section 4 for definition of ICD-10 codes used to define the disease for analysis.

‡ *Haemophilus influenzae* type b (Hib) hospitalisations include only those coded G00.0 (Hib meningitis). Code J05.1 (acute epiglottitis) used in some 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 substantially underestimated, since influenza may not be reported as the underlying cause or diagnosis in many patients who were diagnosed with the complications of influenza.

¶ Pneumococcal meningitis and septicaemia (proxy for invasive pneumococcal disease).

** Hospitalisations for rare diseases such as poliomyelitis and tetanus should be interpreted with caution due to possible coding errors. In addition, the observation that the majority of hospitalisations coded as acute poliomyelitis occurred in those of older age suggests that these are hospitalisations of adults with a past history of poliomyelitis.

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 6.4 Changes in vaccination practice in Australia 1992 to 2007* (including the Australian Standard Vaccination Schedule and National Immunisation Program schedule†)

* See also Gidding HF, Burgess MA, Kempe AE. A short history of vaccination and childhood vaccination practices in Australia. *Med J Aust* 2001;174(1):37–40. Detailed historical tables available at: <http://www.ncirs.edu.au/immunisation/index.php>

† The National Immunisation Program (NIP) schedule replaced the Australian Standard Vaccination Schedule (ASVS) for children in May 2005.

Table 6.4.1: Diphtheria, tetanus and pertussis vaccination practice in Australia, 1992 to 2007

Date	Intervention
1994	Fifth dose of DTPw at 4–5 years of age added to the recommended vaccination schedule (replacing child diphtheria-tetanus 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 dT be replaced with a routine booster dT dose at 50 years of age (unless a booster dT dose has been documented within last 10 years) DTPa-hepB vaccine included on childhood schedule (used in the Australian Capital Territory, New South Wales, the Northern Territory, Queensland and South Australia) 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 for adolescents replacing the diphtheria-tetanus dose – the eligible age group varied with different jurisdictions

Table 6.4.2: *Haemophilus influenzae* type b vaccination practice in Australia, 1992 to 2007

Date	Intervention
1992	First Hib vaccines (PRP-D, ProHIBit) licensed in Australia for vaccinating infants aged 18 months to 5 years
1993	Hib vaccine recommended as part of the childhood vaccination schedule for all children born from February 1993 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 vaccine recommended for all infants (administered separately or in combination with hepatitis B antigen)
2001	Combined DTPa-hepB-IPV-Hib vaccine approved HbOC (Hib-TITER) vaccine availability ceased
2002	Combined DTPa-IPV-Hib vaccines approved
2005	November: Combined DTPa-hepB-IPV-Hib (PRP-T) vaccines used in the Australian Capital Territory, New South Wales and Western Australia (for non-Indigenous children) and Tasmania; PRP-OMP containing vaccine(s) continues to be used in other jurisdictions including Indigenous infants in Western Australia

Table 6.4.3: Hepatitis A vaccination practice in Australia, 1992 to 2007

Date	Intervention
1994	Hepatitis A vaccine (formaldehyde inactivated HAV) approved in Australia for at-risk groups, 3 doses recommended
2005	Hepatitis A vaccination (2 doses) recommended and funded for Aboriginal and Torres Strait Islander children aged 12–24 months residing in the Northern Territory, Queensland, South Australia and Western Australia

Table 6.4.4: Hepatitis B vaccination practice in Australia, 1992 to 2007

Date	Intervention
1997	Vaccination recommended for adolescents aged 10–16 years (New South Wales, the Northern Territory, Queensland, South Australia and Tasmania) 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	South Australia 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)-hepB vaccine approved May: Universal infant vaccination included in childhood schedule with a birth dose of monovalent paediatric hepatitis B vaccine, followed by 3 doses as part of a combination vaccine schedule DTPa-hepB vaccine included on childhood schedule (used in the Australian Capital Territory, New South Wales, the Northern Territory, Queensland and South Australia) Hib(PRP-OMP)-hepB vaccine included on childhood schedule (used in Tasmania, Victoria and Western Australia) Preadolescent vaccination recommended at 10–13 years of age rather than 10–16 years of age Hepatitis B booster doses no longer recommended by the National Health and Medical Research Council
2001	Combined DTPa-hepB-IPV and DTPa-hepB-IPV-Hib vaccines approved

Table 6.4.5: Human papillomavirus vaccination practice in Australia, 1992 to 2007

Date	Intervention
2007	Funded immunisation against human papillomavirus for all Australian girls aged 12–13 years delivered through a school-based program since April 2007, with a temporary catch-up program through schools or primary care providers for females aged 13–26 years until December 2009

Table 6.4.6: Influenza vaccination practice in Australia, 1992 to 2007

Date	Intervention
Pre-1992	Annual vaccination recommended for individuals who were at particular risk of complications or death from influenza due to a limited number of specified conditions, including persons >65 years of age and residents of nursing homes and other chronic care facilities
1994	Annual vaccination recommended for individuals aged >65 years and Aboriginal and Torres Strait Islander adults aged >50 years Annual vaccination recommended to be considered for individuals with specified medical conditions that increased their risk of severe influenza or complications, for residents of nursing homes and other chronic care facilities, and for staff who care for immunocompromised patients and staff of nursing home and other chronic care facilities
1997	Annual vaccination recommended for individuals with specified medical conditions that increased their risk of severe influenza or complications, and for residents of nursing homes and other chronic care facilities In Victoria, influenza vaccine funded for all adults aged ≥65 years
1999	Funding provided for both the national Older Australian Flu program (for adults aged ≥65 years) and the National Indigenous Pneumococcal and Influenza Immunisation (NIPII) program (for Aboriginal and Torres Strait Islander adults aged ≥50 years and Aboriginal and Torres Strait Islander adults aged 15–49 years who had at least one of a range of underlying conditions that increased their risk of influenza or complications)
2000	List of underlying medical conditions and population groups for which annual vaccination was recommended was expanded – additional groups included pregnant women, healthcare workers and travellers under specified conditions
2003	List of underlying medical conditions and population groups for which annual vaccination was recommended was further expanded

Table 6.4.7: Measles, mumps and rubella vaccination practice in Australia, 1992 to 2007

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)
1994	National MMR vaccination catch-up program for both sexes in Year 6
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 the national 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 6.4.8: Meningococcal C vaccination practice in Australia, 1992 to 2007

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 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	Meningococcal C conjugate vaccine added to childhood vaccination schedule at 12 months of age National Meningococcal C Vaccination Program commenced January 2003

Table 6.4.9: Pneumococcal vaccination practice in Australia, 1992 to 2007

Date	Intervention
Pre-1992	Vaccination recommended for individuals with increased risk of pneumococcal disease or complications due to specified underlying medical conditions
1994	Vaccination recommended for Aboriginal and Torres Strait Islanders living in high-risk communities aged >50 years
1997	Vaccination recommended for all persons aged >65 years Vaccination recommended for all Aboriginal and Torres Strait Islanders aged >50 years
1998	In Victoria, pneumococcal vaccine funded for all adults aged ≥65 years and all Aboriginal and Torres Strait Islanders aged ≥50 years
1999	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 >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 The Northern Territory recommended 23-valent vaccine for all Aboriginal and Torres Strait Islander people aged ≥15 years 7-valent conjugate pneumococcal vaccine licensed in Australia
2001	Australian Government program for children at highest risk for invasive pneumococcal disease using the conjugate pneumococcal vaccination (all Aboriginal and Torres Strait Islander infants; all Australian children with specified 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 for all infants from 2 months of age at 2, 4 and 6 months of age (but funded only for children with increased risk of disease) List of high-risk medical conditions for which a child became eligible for the 7-valent conjugate pneumococcal vaccine was expanded
2005	January: Universal funded infant 7-valent pneumococcal conjugate vaccine (7vPCV) program replaced the previous targeted childhood program, with a catch-up program for children aged <2 years Universal 23-valent pneumococcal polysaccharide vaccine (23vPPV) for adults aged ≥65 years replaced previous subsidy through the PBS

Table 6.4.10: Polio vaccination practice in Australia, 1992 to 2007

Date	Intervention
1994	Recommendation for reinforcing dose of OPV to 15-year old adolescents
1998	4-year old OPV booster before starting school commenced (administered along with MMR and DTPa)
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 4 years of age
2005	November: IPV funded to replace OPV, in combination vaccines

Table 6.4.11: Rotavirus vaccination practice in Australia, 1992 to 2007

Date	Intervention
2006	Funded immunisation in the Northern Territory against rotavirus using Rotarix® at 2 and 4 months of age from October 2006
2007	Universal funded immunisation against rotavirus at 2 and 4 months of age (Rotarix®) or at 2, 4 and 6 months of age (Rotateq®) from July 2007

Table 6.4.12: Varicella vaccination practice in Australia, 1992 to 2007

Date	Intervention
2001	Varicella vaccine (Varivax) approved
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)

Appendix 6.5 Government funded national immunisation programs in Australia (up to December 2007)

Prior to 1988, the Australian Government provided childhood vaccines to states and territories for distribution to providers in the public sector. During the same time, live attenuated vaccines such as 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 the diphtheria-tetanus-pertussis (whole cell) vaccine (DTPw), by a pharmacist.

In July 1988, the Australian Government 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 Australian Government 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. 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 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 Hib vaccine was approved for use in children aged 18 months to 5 years of age. 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 Australian Government provided funds to states and territories for Hib vaccines.

In 1994, the Australian Government agreed to fund the purchase of a number of childhood vaccines (DTP, MMR, OPV) via Specific Purpose Payments (SPPs) to states and territories. Australian Government 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-pertussis (acellular) vaccine (DTPa) be used for the fourth and fifth doses of DTP vaccination. These became funded nationally in September 1997.

The 1998/1999 Australian (Commonwealth) Government Budget included an initiative to streamline all childhood vaccine funding from 1999/2000, 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 and influenza vaccines for Aboriginal and Torres Strait Islander Australians with risk factors or aged ≥ 50 years, and influenza vaccine for Australians aged ≥ 65 years, were also funded. Existing vaccine funding through the FAGs and HFGs were not adjusted, thereby freeing up resources of states and territories to purchase non-Commonwealth funded vaccines.

Federal funding to use DTPa for all five infant vaccination doses 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 vaccines 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. Australian Government 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 the hepatitis B vaccine was recommended by the NHMRC and was funded by the Australian Government. In 2001, the 7-valent pneumococcal conjugate vaccine (7vPCV) was provided at no cost to children in the following three categories: (1) all Aboriginal and Torres Strait Islander children aged <2 years; (2) in the Central Australian region, Indigenous children aged <5 years and non-Indigenous children aged <2 years; and (3) all children aged <5 years with medical risk factors predisposing them to a high incidence or severity of pneumococcal infection.

Meningococcal C conjugate vaccine was recommended and funded for all infants 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 publicly funded 7-valent pneumococcal conjugate vaccination program was extended to all Australian children.

In May 2005, the NIP schedule replaced the ASVS for children aged up to 6 years, with all recommended vaccines on the Schedule being publicly funded, including the varicella-zoster vaccine and inactivated poliomyelitis vaccine from November 2005. Hepatitis A vaccine was also recommended and funded under the NIP from November 2005 for Indigenous children aged <5 years living in jurisdictions with a high incidence of hepatitis A (the Northern Territory, Queensland, South Australia and Western Australia).

The HPV vaccine for all girls aged 12–13 years (with a time-limited catch-up program for females aged 13–26 years) and rotavirus vaccines for all infants were added to the NIP in April 2007 and July 2007, respectively.

Table 6.5.1 summarises the dates when each of the vaccines was provided at no cost to recipients in the public sector and through private medical practitioners, as outlined above. There are separate arrangements for the funding of vaccines not provided through the NIP, such as travel-related vaccines, vaccines listed on the Pharmaceutical Benefits Scheme (PBS) and those provided by employers.

Table 6.5.1: Dates when vaccines became available free of charge to recipients* in Australia, delivered in the public and private sectors, up to December 2007†

Vaccine†	Public sector delivery		Private sector delivery ^s	
	Australia	Exceptions	Australia	Exceptions
DTPw vaccine	1953		1994	WA 1988
Child diphtheria-tetanus vaccine (children aged 5–6 years or prior to school entry)	1975		1994	WA 1988
ADT vaccine (adolescent aged 15 or prior to leaving school)	1982		1994	WA 1988
DTPa boosters (infants aged 18 months and 4–5 years)	1997 September	Tas 1997 October Qld 1997 December	1997 September	Tas 1997 October Qld 1997 December
DTPa vaccine (infants aged 2, 4 and 6 months)	1999 February	NT 1997 August SA 1997 August Tas 1999 February Qld 1999 April	1999 February	NT 1997 August SA 1997 August Tas 1999 February Qld 1999 April
dTpa booster (adolescent dose, replaced ADT)	2004 January		2004 January	
Salk (inactivated poliomyelitis vaccine)	1956			
OPV vaccine	1966		1994	NSW 1966 Tas 1966 Qld (? 1998)
IPV (replacing OPV)	2005 November		2005 November	
Rubella vaccine (school girls aged 10–14 years and susceptible women post-partum)	1971			
Measles vaccine for infants aged 12 months	1975			
Combined measles-mumps vaccine replacing measles vaccine	1982			
MMR vaccine (infant dose)	1989		1994	NSW 1989 Qld 1989
MMR vaccine (adolescent dose)	1994	NSW 1994 females only; 1996 both sexes SA 1996	1994	WA 1993 SA 1996

Table 6.5.1: Dates when vaccines became available free of charge to recipients* in Australia, delivered in the public and private sectors, up to December 2007,† continued

Vaccine†	Public sector delivery		Private sector delivery§	
	Australia	Exceptions	Australia	Exceptions
Hep B vaccine (at-risk infants)	1987 November	NT 1988 January SA 1996	Not funded nationally	NSW 1987
Hep B vaccine (adolescent dose)	1998 January	Qld 1998 March Tas 1998 March NT 1998 April (with catch-up for children aged 6–16 years) NSW 1999 SA 1999	1998	Qld 1998 March Tas 1998 March NT 1998 April NSW 1999
Hep B vaccine (universal infant program)	2000 May	NT 1990 August	2000 May	NT 1993 August
Hib vaccines (infants born from February 1993)	1993 April		1993 April	
Hib vaccines (catch-up for children aged <5 years)	1993 July	WA 1993 January NT 1993 April	1993 July	WA 1993 January NT 1994
Influenza vaccine for all adults aged >65 years	1999 (2000: lower age limit changed from >65 to ≥65 years)	Vic 1997	1999 (2000: lower age limit changed from >65 to ≥65 years)	Vic 1997
Influenza vaccine for Aboriginal and Torres Strait Islander people aged >50 years or aged 15–50 years with risk factors (NIPPI program)	1999 (2000: lower age limit changed from >50 to ≥50 years)		1999 (2000: lower age limit changed from >50 to ≥50 years)	
23vPPV for Aboriginal and Torres Strait Islander people aged >50 years or aged 15–50 years with risk factors (NIPPI program)	1999 (2000: lower age limit changed from >50 to ≥50 years)	Vic 1998 for Indigenous adults aged ≥50 years NT 2000 for Indigenous adults aged ≥15 years	1999 (2000: lower age limit changed from >50 to ≥50 years)	Vic 1998 for Indigenous adults aged ≥50 years NT 2000 for Indigenous adults aged ≥15 years
23vPPV for all adults aged ≥65 years	2005	Vic 1998	2005	Vic 1998
7vPCV (at-risk children only, including Aboriginal and Torres Strait Islander children aged <5 years)	2001 (2003: list of at-risk conditions expanded)		2001 (2003: list of at-risk conditions expanded)	
7vPCV (universal infant program, with catch-up for all children aged <2 years)	2005 January		2005 January	
Conjugate meningococcal C vaccine (infants aged 12 months and catch-up aged 1–18 years)	2003 January		2003 January	

Table 6.5.1: Dates when vaccines became available free of charge to recipients* in Australia, delivered in the public and private sectors, up to December 2007,† continued

Vaccine†	Public sector delivery		Private sector delivery§	
	Australia	Exceptions	Australia	Exceptions
VZV vaccine (infants at age 18 months with school-based catch-up for children aged 10–13 years who have no history of varicella)	2005 November		2005 November	
Hepatitis A vaccine for Indigenous children living in jurisdictions with high disease incidence (NT, Qld, SA, WA)	2005 November	northern Queensland 1999	2005 November	
HPV vaccine (ongoing for girls aged 12–13 years, with a time-limited catch-up program for females aged 13–26 years until December 2009)	2007 April		2007 July	
Rotavirus vaccines (for all infants)	2007 July	NT 2006 October	2007 July	NT 2006 October

* Vaccines on the Australian Standard Vaccination Schedule and the current National Immunisation Program 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.

† This table focuses on the time when a particular vaccine first became available free of charge to recipients, grouped by vaccine antigens. For more details regarding change of vaccination practice in Australia 1992–2007, please refer to Appendix 6.4.

‡ Please refer to the text or the abbreviations list for abbreviations of the vaccines.

§ 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.

Acknowledgement: Australian Government Department of Health and Ageing.

Appendix 6.6 Case definitions of selected notifiable vaccine preventable diseases prior to 2004

In September 2003, new national case definitions for notifications reported to NNDSS were endorsed by the Communicable Diseases Network Australia,¹ with nearly all jurisdictions implementing the new definitions in January 2004 (New South Wales commenced in August 2004). Prior to the adoption of the national definitions, some jurisdictions used the 1994 NHMRC case definitions² (e.g. Tasmania and the Australian Capital Territory), some jurisdictions used modified definitions that were based on the NHMRC case definitions, and some others used definitions specific to the state (e.g. New South Wales) for some diseases.

This appendix highlights the 1994 NHMRC Surveillance Case Definitions² in use for notifiable diseases data prior to 2004 for the vaccine preventable diseases covered in this report, with the exception of those that became notifiable after 1994 (influenza, invasive pneumococcal disease, rotavirus and varicella/zoster). An unpublished report that reviewed the national notifiable diseases case definitions, prepared by Dr Sue Skull for the Communicable Diseases Network Australia and New Zealand in January 2001, identified considerable discrepancies from the respective NHMRC case definitions being used by various jurisdictions then, for each of the diseases included in this appendix. The variations included the exclusion of one or more of the NHMRC criteria, addition of extra alternative or mandatory criteria, modification of some of the criteria, and inclusion of suspected or presumptive cases in addition to confirmed cases. Further details about definitions in use previously may be found in earlier reports in this series.³⁻⁵

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.

Haemophilus influenzae type b

Notifications prior to 2004

a) An invasive 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: In Victoria, from 2002, notifications only included cases where Hib was laboratory confirmed.⁶

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, with or without elevated aminotransferase levels, and 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).

Note: Prior to 2004, the variations in case definition criteria among jurisdictions included the exclusion of one or more of the NHMRC criteria (e.g. other causes of acute hepatitis), addition of extra alternative criteria (e.g. HbsAg positive with a previous negative test in the last 12 months), inclusion but differentiation of acute, unspecified infection and chronic carrier cases, and the inclusion of suspected and presumptive cases.

Influenza

Notifications prior to 2004

The 1994 NHMRC Surveillance Case Definitions did not include influenza. Laboratory-confirmed influenza became a notifiable disease in 2001 in all jurisdictions except for South Australia, where laboratory-confirmed influenza became notifiable in May 2008; nevertheless, cases reported in South Australia prior to May 2008 were included in the NNDSS dataset. The case definition criteria for laboratory-confirmed influenza remained unchanged from 2001 to 2004.

Measles

Notifications prior to 2004

a) An illness characterised by all of the following features:

- a generalised maculopapular rash lasting 3 or more days, and
- a fever (at least 38°C if measured), and
- cough or coryza or conjunctivitis or Koplik spots

or

b) Demonstration of measles-specific IgM antibody

or

c) A 4-fold or greater change in measles antibody titre between acute and convalescent phase sera obtained at least 2 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

- a) Isolation of *Neisseria meningitidis* from a normally sterile site
or
- b) Detection of meningococcal antigen in joints, blood or cerebrospinal fluid (CSF)
or
- c) Detection of Gram negative intracellular diplococci in blood or CSF.

Note: The variations in case definition criteria among jurisdictions included the addition of extra alternative criteria (e.g. presence of clinical compatible illness in addition to laboratory criteria of diplococci isolated from skin and joints; culture from conjunctiva; positive nucleic acid test from cerebrospinal fluid, blood or normally sterile sites together with clinically compatible disease) and inclusion of suspected, presumptive and probable cases.

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 2 days or more without other apparent cause).

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

Pertussis

Notifications prior to 2004

- a) Isolation of *Bordetella 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 2 weeks or more with one of the following:
 - paroxysms of coughing, or
 - inspiratory whoop without other apparent causes, or
 - post-tussive vomiting
- d) An illness characterised by a cough lasting at least 2 weeks in a patient who is epidemiologically linked to a laboratory-confirmed case.

Invasive pneumococcal disease

Notifications prior to 2004

The 1994 NHMRC Surveillance Case Definitions did not include invasive pneumococcal disease. Invasive pneumococcal disease became a notifiable disease in 2001 in all jurisdictions, and the case definition criteria remained unchanged from 2001 to 2004.

Poliomyelitis

Notifications prior to 2004

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

Q fever

Notifications prior to 2004

- a) A 4-fold or greater change in serum (CF) antibody titre to Phase II antigen of *Coxiella burnetii*
or
- b) A 4-fold or greater change in enzyme-linked immunosorbent assay (ELISA) antibody titre to Phase I or Phase II antigens of *C. burnetii*
or
- c) An IgM fluorescent antibody titre of at least 1:160 during the convalescent phase of the illness (i.e. 10 days or more after onset)
or
- d) In chronic infections (e.g. endocarditis), elevated (CF) IgG or IgA titres to *C. burnetii* Phase I antigen
or
- e) Isolation of *C. burnetii* from a clinical specimen.

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 4-fold or greater rise in rubella antibody titre between acute and convalescent phase sera obtained at least 2 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; this led to an increase in the specificity of notifications.⁷

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.

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Abbreviations

7vPCV	7-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
ABS	Australian Bureau of Statistics
ACIP	Advisory Committee on Immunization Practices (USA)
ACT	Australian Capital Territory
ADT	Adult diphtheria-tetanus
AFP	Acute flaccid paralysis
AIHW	Australian Institute of Health and Welfare
APSU	Australian Paediatric Surveillance Unit
ASVS	Australian Standard Vaccination Schedule
aVDPV	Ambiguous vaccine-derived poliovirus
CRS	Congenital rubella syndrome
cVDPV	Circulating vaccine-derived poliovirus
DT	Diphtheria-tetanus (child formulation)
dT	Adolescent/adult diphtheria-tetanus
DTP	Diphtheria-tetanus-pertussis (child formulation)
DTPa	Diphtheria-tetanus-pertussis (acellular) (child formulation)
dTpa	Adolescent/adult diphtheria-tetanus-pertussis (acellular)
DTPw	Diphtheria-tetanus-pertussis (whole cell) (child formulation)
FAG	Finance assistance grant
HAV	Hepatitis A virus
HbOC	<i>Haemophilus influenzae</i> type b polysaccharide conjugated to non-toxic diphtheria CRM ₁₉₇ protein
HBsAg	Hepatitis B surface antigen
HBV	Hepatitis B virus
HFG	Hospital funding grant
Hib	<i>Haemophilus influenzae</i> type b
HPV	Human papillomavirus
ICD	International Classification of Diseases
ICD-9	International Classification of Diseases, 9th Revision
ICD-9-CM	International Classification of Diseases, 9th Revision, Clinical Modification
ICD-10	International Classification of Diseases, 10th Revision
ICD-10-AM	International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification
IgG	Immunoglobulin G
IgM	Immunoglobulin M
IDU	Injecting drug users
IPD	Invasive pneumococcal disease
IPV	Inactivated poliovirus vaccine
iVDPV	Immunodeficiency-associated vaccine-derived poliovirus
LOS	Length of stay (in hospital)
MenCCV	Meningococcal C conjugate vaccine
MMR	Measles-mumps-rubella
MSM	Men who have sex with men
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
NIPII	National Indigenous Pneumococcal and Influenza Immunisation (Program)
NNDSS	National Notifiable Diseases Surveillance System
n.p.	Not published
NQFMP	National Q Fever Management Program
NSW	New South Wales
NT	Northern Territory
OPV	Oral poliovirus vaccine
PBS	Pharmaceutical Benefits Scheme
PCR	Polymerase chain reaction
PHOFA	Public Health Outcome Funding Agreement
PRP-D	<i>Haemophilus influenzae</i> type b polysaccharide conjugated to diphtheria toxoid
PRP-OMP	<i>Haemophilus influenzae</i> type b polysaccharide conjugated to the outer membrane protein of <i>Neisseria meningitidis</i> vaccine
PRP-T	<i>Haemophilus influenzae</i> type b polysaccharide conjugated to tetanus toxoid
Qld	Queensland
<i>R</i>	Effective reproductive number
RSV	Respiratory syncytial virus
SA	South Australia
SSPE	Subacute sclerosing panencephalitis
Tas	Tasmania
VAPP	Vaccine-associated paralytic poliomyelitis
VDPV	Vaccine-derived poliovirus
aVDPV	Ambiguous vaccine-derived poliovirus
cVDPV	Circulating vaccine-derived poliovirus
iVDPV	Immunodeficiency-associated vaccine-derived poliovirus
Vic	Victoria
VPD	Vaccine preventable disease
VZV	Varicella-zoster virus
WA	Western Australia
WHO	World Health Organization