Communicable and vaccine-preventable conditions under surveillance by the APSU: 2005 update

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Background

The Australian Paediatric Surveillance Unit (APSU) conducts national active surveillance of rare diseases of childhood, including infectious and vaccine preventable diseases, genetic disorders, childhood injuries and mental health conditions. Studies of communicable and vaccine-preventable diseases are supported by the Australian Government Department of Health and Ageing through its communicable diseases program. This report is a summary of surveillance results for communicable and/or vaccine preventable diseases studied through the APSU in 2005.

In 2005, eight communicable or vaccine preventable conditions were studied:

- acute flaccid paralysis (AFP);*
- congenital cytomegalovirus infection;
- congenital rubella infection;
- perinatal exposure to HIV and HIV infection;
- neonatal herpes simplex virus infection;
- hepatitis C virus infection;
- non-tuberculous *Mycobacterium* infection; and
- neonatal group B *Streptococcus* infection.†

Methods

APSU study protocols are developed with collaborating investigators and/or institutions and the objectives and chief investigators for each study are listed in Table 2. The methodology used to conduct surveillance is described in detail elsewhere.[1,2]

The APSU aims to provide epidemiological information that is representative of the Australian population and maximal case ascertainment is a high priority. Despite a representative mailing list (93% of all paediatricians in active clinical practice in Australia participate in monthly surveillance) and high monthly response rates, complete case ascertainment is unlikely. This is particularly relevant in remote communities where children have limited access to paediatricians. However, for most conditions studied by the APSU no other national data are available to estimate completeness of ascertainment. APSU encourages the use of complementary data sources where available and reporting by a range of specialists to maximize cases identified. Reported rates for conditions ascertained through the APSU therefore represent a minimum estimate for these conditions in the relevant Australian populations.

Results

In 2005, 1,148 clinicians participated in the monthly surveillance of 14 conditions, (including the 8 listed above), with an overall monthly response rate of 93 per cent. Questionnaire return rate is greater than 80 per cent for most studies. Table 1 shows the number of confirmed cases reported in 2005 and for the whole study period and the reported rate per 100,000 population.[3]

APSU data contribute significantly to the national surveillance effort, providing valuable information for clinicians, policy makers and the community. The APSU is often the only source of national data that includes clinical and/or laboratory details and data on both in-patients and out-patients. The key findings for studies undertaken in 2005 are summarised in Table 2.

Further information on the above studies may be obtained by contacting the APSU: website www.apsu.org.au Telephone 02 9845 3005; email: apsu@chw.edu.au, or the Principal Investigator for each study.

* Although the aim of this surveillance is to identify AFP due to poliomyelitis or associated with polio vaccination, there are many non-infectious causes of AFP.

† The study of neonatal group B *Streptococcus* infection commenced in July 2005.

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References

1. Elliott, E, Cronin P, Rose D, Zurynski Y (Eds). APSU Annual Report 2004. Australian Paediatric Surveillance Unit, 2005; ISSN:1443–3524.
2. Zurynski Y, Cronin P, Elliott E. Communicable and vaccine-preventable conditions under surveillance by the APSU: 2004 update. *Commun Dis Intell* 2005; 29:407–411.
3. Australian Bureau of Statistics. *Australian Demographic Statistics*, 3101.0, March Quarter; 2005.
4. Kelly H, Brussen KA, Lawrence A, Elliott E, Pearn J, Thorley B. Polioviruses and other enteroviruses isolated from faecal samples of patients with acute flaccid paralysis in Australia, 1996–2004. *J Paediatr Child Health* 2006;42:370–376.
5. Thorley B, Brussen KA, Elliott J, Kelly HA. Vigilance is required for Australia to remain polio free. *Med J Aust* 2006;184:474–475.
6. Munro SC, Trincado D, Hall B, Rawlinson WD. Symptomatic infant characteristics of congenital cytomegalovirus disease in Australia. *J Paediatr Child Health* 2005;41:449–452.
7. McDonald AM, Li Y, Cruickshank MA, Elliott EJ, Kaldor JM, Ziegler JB. Use of interventions for reducing mother-to-child transmission of HIV in Australia. *Med J Aust* 2001;174:449–452.
8. Elliott E, Jones CA, Mews C, Hardiker W, Kesson A, Polis S, *et al*. Reported risk factors for hepatitis C virus infection in Australian children: results of national surveillance. *J Paediatr Child Health* 2005;41:S3.
9. Hardiker W, Elliott E, Jones CA. The silent infection: should we be testing for perinatal hepatitis C and, if so, how? *Med J Aust* 2006;184:54–55.
10. Blyth CC, Palasanthiran P, Best E, Jones C, Daley AJ, Burgner D, *et al.* Non-tuberculous mycobacterial infection: results from a national surveillance study. Abstract – Royal Australasian College of Physicians Congress, 8–11 May 2006, Cairns, Australia. *J Paediatr Child Health* 2006. In press.

Table 1. Confirmed cases identified for 2005 and for the total study period

Condition	Date study commenced	Questionnaire response for total study period (%)	Number of confirmed cases		Reported rate for total study period (per 10^5 per annum)
			2005	Total study period	
Acute flaccid paralysis	March 1995	90	30*	368*	0.86‡
Congenital cytomegalovirus	Jan 1999	66	9	57	3.25§
Congenital rubella (with defects)	May 1993	96	NIL	50	0.10‡
Perinatal exposure to HIV;	May 1993	90	13	266	8.08§
HIV infection			5	45	1.99§,‖
Neonatal herpes simplex virus infection	Jan 1997	95	9	78	3.45§
Hepatitis C virus infection	Jan 2003	80	7	31	0.26‡
Non-tuberculous mycobacteria	July 2004	80	24†	44†	0.47
Neonatal B group *Streptococcus* infection	July 2005	56	30	30	¶

* All reported cases that have been classified by the Polio Expert Committee were 'non-polio AFP' according to WHO criteria.

† Includes confirmed and probable cases.

‡ Based on population of children aged ≤-15 years as estimated by the Australian Bureau of Statistics.[3]

§ Based on number of births as estimated by the Australian Bureau of Statistics.[3]

‖ All HIV infections except for one (source of infection unknown) resulted from perinatally acquired HIV.

¶ The study of neonatal groups B *Streptococcus* infection commenced in July 2005. Due to the short surveillance period of only 6 months, a rate is not reported.

Table 2. Results summary

Condition and principal investigator	Objectives	Key findings
Acute flaccid paralysis (AFP) Prof Heath Kelly, Victorian Infectious Diseases Reference Laboratory	To determine the notification rate of AFP in children aged <15 years. To determine whether AFP is caused by poliovirus infection and if so, whether it is a wild, vaccine, or vaccine-derived strain of poliovirus. To determine other causes and the clinical picture of AFP in Australia.	Decreased notification rates resulted in failure to reach the WHO AFP surveillance target of 1 case per 10^5 aged <15 years per annum. The primary diagnoses for AFP remain Guillain-Barré syndrome and transverse myelitis.[4] There were 3 cases of AFP due to infant botulism in 2005. Only 19 per cent of cases had adequate faecal specimen collection—well below the 80 per cent WHO target. An outbreak of 303 cases of wild poliovirus was recorded in Indonesia. Sixteen countries reported importations of wild poliovirus. Continued surveillance is required to detect imported cases and keep Australia polio free.[5]
Congenital cytomegalovirus (cCMV) infection Prof William Rawlinson, Virology Division, Department of Microbiology, Prince of Wales Hospital, Sydney	The study aims to determine: • the incidence of congenital and suspected congenital CMV infection; • the presenting features and clinical spectrum of disease due to congenital CMV; • the genotypes of CMV which cause congenital disease; • current therapy for congenital CMV infection; and • the epidemiology of congenital CMV prior to trials of vaccines and antivirals.	cCMV continues to be the most common infectious cause of malformations in Australia. cCMV infection was not associated with maternal illness in approximately one third of cases, and should be considered regardless of maternal history. cCMV remains under-diagnosed. Although most cases are diagnosed by urine culture; use of PCR for urinary screening for CMV may increase diagnostic yield.[6] Universal neonatal hearing screening programs may also help identify new cases.
Congenital rubella (with defects) A/Prof Cheryl Jones, The Children's Hospital at Westmead & Discipline of Paediatrics & Child Health, University of Sydney	To document the incidence of congenital rubella infection. To determine the vaccination status of mothers of infected infants. To monitor the effectiveness of the current vaccination program.	There were no cases of congenital rubella reported in 2005. As the risk of congenital rubella remains, particularly among immigrant women born in countries with poorly developed vaccination programs, such women should have serological testing for rubella after arrival in Australia, and vaccination when appropriate. Travel to rubella endemic counties in the first trimester by women with no prior rubella immunity poses a risk of congenital rubella to the fetus.
Perinatal exposure to HIV and HIV infection Ann McDonald, National Centre in HIV Epidemiology and Clinical Research	To identify new cases of perinatal exposure to HIV, paediatric HIV infection, and AIDS. To describe the pattern of perinatal exposure to HIV in Australia. To monitor the perinatal HIV infection transmission rate and use of interventions for reducing the risk of mother-to-child transmission. To describe the natural history of paediatric HIV infection.	In 2005, 13 cases of perinatal exposure were reported. The main source of infection for the mother was through heterosexual contact with a high risk partner.[7] Six reported cases of HIV infection in children were newly diagnosed in 2005, including 5 cases of perinatally-acquired HIV infection and 1 case of HIV infection acquired in a high HIV prevalent country in sub-Saharan Africa. The 5 cases of perinatal HIV infection were all born in Australia. These cases were reported through national surveillance for newly diagnosed HIV infection. Although the mother's HIV infection was diagnosed prenatally in 2 cases, interventions such as elective caesarean, avoidance of breast feeding and anti-viral therapies were not used. Antenatal diagnosis of the mother's HIV infection and use of interventions is required to minimise the risk of mother-to-child HIV transmission.

Table 2. Results summary, *continued*

Condition and principal investigator	Objectives	Key findings
Neonatal herpes simplex virus infection (HSV) A/Prof Cheryl Jones, Herpes Virus Research Unit, The Children's Hospital at Westmead & Discipline of Paediatrics & Child Health, University of Sydney	To determine the incidence of neonatal HSV infection in Australia, its mortality and morbidity. To determine its mode of presentation e.g. localised, disseminated or complicated by encephalitis or pneumonitis and mode of transmission. To determine whether there is delay between presentation, diagnosis and initiation of treatment.	Over half of neonatal HSV infections in Australia are caused by HSV type 1, in contrast to the USA where HSV type 2 predominates. Typical herpetic lesions of the skin, eye or mouth were not evident in half of infants identified with neonatal HSV infection, which makes early diagnosis difficult. Disseminated HSV infection in the newborn may be associated with the early onset of pneumonitis in infants (in whom the chest X-ray may be normal). This is highly lethal unless antiretroviral therapy is initiated.
Hepatitis C virus infection (HCV) A/Prof Cheryl Jones, The Children's Hospital at Westmead & Discipline of Paediatrics & Child Health, University of Sydney	To determine the reported incidence of newly diagnosed HCV infection in Australian children. To describe the clinical presentation, investigation and management of newly diagnosed HCV infection in Australian children. To document the presence of known risk factors for HCV infection in an Australian paediatric population. To determine the prevalence of co-infection with hepatitis B virus (HBV) and/or human immunodeficiency virus in Australian children with newly diagnosed HCV infection.	Perinatal transmission is the main source of HCV infection in Australian children. In the APSU study infants at risk were born to mothers who used IV drugs, had invasive procedures overseas or had tattoos.[8] Most HCV-infected children are clinically asymptomatic with mildly elevated liver function test at diagnosis. However, HCV induced chronic liver disease and liver failure have been reported among children. Given that 1–2 per cent of Australian women of child-bearing age are infected with HCV, the reported rates of infection are lower than predicted. This may be due to the lack of a consistent approach to identifying children with HCV infection.[9]
Non-tuberculous *Mycobacterium* infection (NTMI) Dr Pamela Palasanthiran, Paediatric Infectious Diseases Specialist, Department of Immunology and Infectious Diseases, Sydney Children's Hospital Randwick, NSW	To estimate the incidence of newly diagnosed NTM infection in children seen by child health specialists in Australia. To describe the epidemiology and spectrum of disease and document known risk factors. To describe diagnostic investigations used in Australia; frequency of use of skin testing and the clinical utility of the test, including differential skin testing. To describe the management of NTM in Australia and the response to treatment.	This infection most often presents as lymphadenitis predominantly in immunocompetent children. *Mycobacterium avium intracellulare* and *Mycobacterium fortuitum* are the most common organisms isolated in Australian children. Surgery is the most commonly offered therapy and in NTMI lymphadenitis complete excision is associated with a lower risk of relapse. There is marked heterogeneity in the antimicrobials and course prescribed. Despite therapy, relapse occurs in about 20 per cent of cases.[10]
Neonatal and infant *Streptococcus agalactiae* (group B streptococcus – GBS) sepsis Prof Lyn Gilbert, Centre for Infectious Diseases and Microbiology, Institute for Clinical Pathology and Medical research, Westmead Hospital, Westmead NSW	To determine: • the current incidence of early and late onset neonatal GBS infection; • the incidence of maternal and infant risk factors; • the proportion of early onset GBS infection in infants of women who have been given intrapartum antibiotic prophylaxis; • short-term mortality and morbidity of early and late onset GBS infection; and • the distribution of GBS genotypes between isolates.	Preliminary results only, as the surveillance period is only 6 months. Over half of the reported cases have been early onset at less than 8 days of age. The number of notifications received so far is consistent with other available data.