

## Poliomyelitis

### POLIOMYELITIS VACCINES FOR AUSTRALIAN CHILDREN: INFORMATION FOR IMMUNISATION PROVIDERS

#### Disease and epidemiology

- Poliomyelitis (polio, infantile paralysis) is caused by a virus that has three serotypes: PV1, PV2 and PV3.
- Person-to-person spread of polio virus is by the faecal–oral route. Typical manifestations of poliomyelitis are caused when the virus spreads to infect and replicate in the cells of the central nervous system.
- The characteristic and most severe form of clinical manifestation of polio infection is ‘paralytic polio’ that usually presents as asymmetrical permanent paralysis of the legs.
- Australia was declared polio free by the World Health Organization in 2000.

#### Who should be vaccinated

- Inactivated polio vaccine (IPV) is recommended and funded under the National Immunisation Program (NIP) for routine immunisation of Australian children as a primary sequence of 3 doses at 2, 4 and 6 months of age and a booster at 4 years of age.
- Unvaccinated adults are recommended a schedule of 3 doses at 1–2 month intervals. A booster dose is recommended for adults travelling to areas or countries where poliomyelitis is epidemic or endemic and for healthcare workers, including laboratory workers, in possible contact with poliomyelitis cases.
- A booster dose every 10 years is desirable for those exposed to a continuing risk of polio infection.

#### Vaccines

- Since 2005, inactivated polio vaccine (IPV) is used for all doses of polio vaccine in Australia.
- IPV-containing combination vaccines licensed for use in Australia for children 2 months to 8 years of age are Infanrix hexa, Infanrix Penta, Infanrix-IPV and Quadracel.
- IPV-containing combination vaccines recommended for use in persons aged  $\geq 8$  years are Adacel Polio and Boostrix-IPV.
- IPOL, which only contains inactivated poliovirus strains, can be administered to persons from 2 months of age.

## The disease

Poliomyelitis, often called polio or infantile paralysis, is an infectious disease caused by a virus. This virus is a member of the enterovirus subgroup of the Picornaviridae family and has three serotypes: PV1, PV2 and PV3.

Immunity to one serotype of the virus does not provide significant protection against the other serotypes.<sup>1,2</sup>

Polio virus transmission is by faecal–oral or occasionally oral–oral routes. Once the virus enters the body through the mouth it multiplies in the oropharynx and the small intestine. In the gastrointestinal tract, the virus invades the local lymphoid tissues and, in a minority of cases, then enters the bloodstream and spreads to the central nervous system. The virus may also spread to the central nervous system along the peripheral nerves. The incubation period for polio infection is usually between 7 and 14 days but may range from 2 to 35 days. By 3–5 days after exposure, the virus can be isolated in the blood, throat and faeces. The virus continues to be excreted in the stools for several weeks after infection.<sup>1,2</sup>

Clinical manifestations of poliomyelitis can vary and they are categorised according to severity. A large majority (up to 95%) of polio infections are inapparent or asymptomatic. However, even those infected who don't show any symptoms shed the virus in their stools and, therefore, are able to transmit the virus to others. In polio endemic areas, persons with inapparent infections, particularly children, act as the main reservoir of polio infection.

Rarely (in less than 1% of polio infections), the virus invades and damages or completely destroys the motor neurons of anterior horn cells of the spinal cord and brain stem. This form, known as **paralytic polio**, is the most severe and typical manifestation of poliomyelitis. Depending on the extent of central nervous system damage, paralytic polio is classified into spinal, bulbar and bulbospinal forms. Of these, spinal polio accounts for about 80% of all paralytic polio cases. It is characterised by asymmetrical paralysis, commonly of the legs. Weakness of muscles innervated by cranial nerves is the main feature of bulbar polio.<sup>1,3</sup>

About a tenth of patients who develop paralytic polio can die without appropriate respiratory support when their respiratory muscles are paralysed. In the past, patients with respiratory muscles affected by polio were immobilised inside negative pressure mechanical ventilators to regulate their breathing and keep them alive. These huge metal cylinders that were called 'iron lungs', over the years, became a symbol of polio.<sup>4</sup>

Most patients, even with paralytic polio, recover completely and, in most others, muscle function returns to some degree. However, paralysis or weakness that persists 12 months after the onset is usually permanent.

Persons with residual impairment following paralytic poliomyelitis can develop a condition called **post polio syndrome (PPS)**, after a period of prolonged stability (usually 30–40 years). It is characterised by exacerbation of existing muscle weakness coupled with development of weakness/paralysis in previously unaffected muscles. PPS is believed to be caused by degeneration, with age, of over sized motor units created during the recovery process of initial paralytic polio. It is rarely life threatening but has a slow, step-wise, unpredictable course. PPS is not an infectious process and persons who develop PPS do not shed poliovirus.<sup>5</sup>

## Epidemiology

As a result of vaccination, there has been a dramatic reduction in the incidence of poliomyelitis globally. However, the disease still remains endemic in four countries – Nigeria, India, Pakistan and Afghanistan. Other countries also have cases of wild-type poliomyelitis from time to time, due to importation.<sup>6</sup>

The World Health Organization (WHO) aimed to eradicate poliomyelitis by the year 2005. Although this was not successful, WHO is still hopeful that polio eradication will be achieved by 2010 or soon after, primarily through 'national polio days' where all children in a certain region are given oral polio vaccine (OPV) (<http://www.polioeradication.org>).

Poliomyelitis has been a notifiable disease in Australia since 1922. The highest recorded incidence of poliomyelitis in the country (39.1 per 100,000 population) was in 1938. The last polio epidemic in Australia was in 1961–1962.<sup>7</sup>

Mass vaccination against polio using intramuscular Salk inactivated polio vaccine (IPV-Salk) first started in Australia in 1956. In 1966, IPV-Salk was replaced by Sabin oral polio vaccine (OPV-Sabin) in the publicly funded immunisation program. OPV is particularly suited to provide mass protection against wild-type polio. As expected, good vaccine coverage with OPV over several years led to cessation of indigenous transmission of wild polio virus infections in Australia.<sup>8</sup>

The last reported case of locally acquired wild-type polio in Australia was in 1972. In 1986, there was a case of poliomyelitis reported in a 22 year old that was initially thought to be wild-type virus, but was later confirmed as a

vaccine-like strain.<sup>8-10</sup> In October 2000, Australia together with the other 36 countries in the Western Pacific Region was declared 'polio free' by the WHO. This certification of 'polio-free' status is confirmation of interruption of indigenous poliovirus transmission and containment of wild polio virus in the country.<sup>11</sup>

Until global eradication of polio is achieved, all countries are at risk of polio infection. In 2005, Indonesia had an imported case of polio, 10 years after the last case of indigenously acquired poliomyelitis was reported in the country. This imported case of polio caused a re-establishment of local transmission of the virus and resulted in a large polio epidemic, with more than 200 cases.<sup>12</sup>

With increased air travel around the world and recent outbreaks of polio in other countries, the importation of polio is a real threat to Australia. In July 2007, in Victoria, wild polio virus type 1 was isolated from a young man who had recently arrived from Pakistan. Due to the protective effect of high polio vaccine coverage in the country, the imported virus did not spread beyond the index case. This person suffered mild paralysis but has since fully recovered.<sup>6,13</sup>

Continuing the polio vaccination program to maintain the current high coverage rate and adequate surveillance for cases of acute flaccid paralysis remain the best defence against the continuing threat of polio infection.<sup>8,12,14,15</sup>

### **Surveillance for acute flaccid paralysis**

Acute flaccid paralysis (AFP) is a common clinical manifestation of poliomyelitis. AFP is defined as the acute onset of flaccid paralysis in one or more limbs or acute onset of bulbar paralysis. In the past, in Australia, acute poliomyelitis was the most common cause of AFP; now Guillain-Barré syndrome and transverse myelitis are the two leading causes of non-polio AFP. However, there are many other causes.

Active surveillance of AFP is a highly sensitive indicator of wild polio virus activity. Adequate surveillance for AFP in children aged 0–15 years is a criterion that needs to be satisfied for initial certification and maintenance of polio-free status by WHO. Australia continues to work towards the WHO targets for AFP detection (at least one case of AFP per 100,000 population aged <15 years) and adequate stool sample collection (2 samples within 14 days of onset of paralysis).

The Australian Paediatric Surveillance Unit (APSU) has conducted surveillance for AFP since 1993. APSU relies on reporting by ~1360 paediatricians around Australia. Recently, in collaboration with NCIRS, APSU developed

a hospital-based surveillance system in four children's hospitals in NSW, Victoria, South Australia and Western Australia. This system, Paediatric Active Enhanced Disease Surveillance (PAEDS), relies on active case identification by specialist surveillance nurses.

While it is highly unlikely that Australia has indigenous wild polio, all cases of acute flaccid paralysis need to be extensively investigated to ensure that the country remains polio free.<sup>15-17</sup> The Polio Expert Committee (PEC) reviews all cases of AFP and determines if a case is compatible with poliovirus infection after review of clinical data and laboratory results from virus culture. The PEC reports to the WHO Western Pacific Regional Office and these data are used to determine the polio-free status for Australia, which is reviewed each year by the Regional Commission for the Certification of the Eradication of Poliomyelitis in the Western Pacific Region, convened by WHO.

## **Who should be vaccinated**

### **National Immunisation Program (NIP)**

#### **Children**

Children are recommended a primary course of 3 doses of an IPV-containing vaccine at 2, 4 and 6 months of age and a booster dose at 4 years of age. The recommended interval between 2 doses is 2 months, but, for catch-up, the minimum interval can be 1 month. In the Australian setting, a 3-dose primary schedule and a booster at 4 years of age provides adequate protection. Therefore, for those children who have received a complete course of polio vaccine during childhood, a further booster dose is not required later in life unless they are at increased risk of infection as below.

#### **Adults**

The schedule for unvaccinated adults is 3 doses administered at intervals of 1–2 months.

#### **Booster doses**

A booster dose is not required for fully vaccinated children or adults unless they are at increased risk of infection, such as

- travelling to areas or countries where poliomyelitis is epidemic or endemic (see <http://www.polioeradication.org> for more information on affected countries)
- healthcare workers, including laboratory workers, in possible contact with poliomyelitis cases.

For those exposed to a continuing risk of infection, booster doses are desirable every 10 years.

Diphtheria/tetanus/pertussis/IPV-combination vaccines for children or adults can be used where other antigens are also required.

## Vaccines

Inactivated poliomyelitis vaccine (IPV) was first licensed for use in 1955 and was used extensively from that time until the early 1960s. Oral polio vaccines were first introduced in 1962 as monovalent vaccines. Trivalent oral polio vaccine was first licensed in 1963 and this largely replaced IPV use. An enhanced potency IPV first became available in 1988. Over the next few decades, enhanced potency IPV gradually replaced OPV use in many countries. This change from OPV to IPV was implemented in Australia in November 2005.

### Switch to inactivated poliomyelitis vaccines

The worldwide use of live trivalent OPV resulted in the elimination of polio from many countries including Australia. OPV has several advantages over IPV in providing mass protection against the transmission of wild polio virus. OPV induced a good local immune response in the intestines, the primary site for polio virus multiplication. This provides local resistance to subsequent infection with wild-type polio virus and also reduces the risk of symptomless excretion of the virus. In addition, OPV confers community protection when vaccine virus is transmitted from recent vaccinees to their non-immunised contacts.<sup>18</sup>

However, OPV on rare occasions can cause paralytic polio, known as vaccine associated paralytic polio (VAPP), in primary vaccine recipients and their contacts.

After receiving OPV, the vaccine virus normally replicates in the gastrointestinal tract of healthy individuals, and may be present in the stools for up to 6 weeks. Each time a vaccine strain replicates in the gastrointestinal tract, random mutations may occur and give rise to an increase in neurovirulence of that strain. These mutations or reversions occur in almost all OPV recipients but very rarely cause paralytic disease. When a mutated vaccine virus regains the ability to cause disease, it may cause VAPP either in the vaccine recipient or their close contacts. This OPV-associated paralysis is identical to that caused by wild-type virus and may be permanent.<sup>1,19</sup>

The last reported case of VAPP in Australia occurred in an unvaccinated mother of a recently vaccinated infant in 1995.<sup>10</sup>

In comparison to OPV, the major advantage of IPV is that it does not contain live virus and, therefore, cannot cause VAPP.

In Australia, since naturally occurring paralytic polio no longer occurs, even the small risk of VAPP from OPV has been considered unacceptable. For this reason, a change to an all IPV immunisation schedule to replace OPV was implemented in November 2005. Prior to this date, IPV had been recommended but not routinely used.<sup>1,6,19</sup>

Similarly, the United States, the United Kingdom and New Zealand have also recently implemented a change to IPV-containing vaccines from OPV, and a number of European countries have always used IPV.<sup>5,18</sup>

### Vaccine names

There are a number of inactivated poliomyelitis virus vaccines licensed for use in Australia. These include IPOL, which only contains inactivated poliovirus strains and can be administered to all persons >2 months of age. This vaccine may not be routinely stocked but is available on request.

IPV-containing combination vaccines available for use in infants and young children <8 years of age are Infanrix hexa, Infanrix Penta, Infanrix-IPV and Quadracel. These vaccines are suitable for providing other vaccine antigens as well, recommended on the National Immunisation Program, particularly at 2, 4 and 6 months of age. The IPV-containing combination vaccines are equally effective in generating an immune response to poliovirus as IPOL. When combination vaccines are used, fewer injections need to be given at each visit than if IPOL is given separately. The IPV-containing combination vaccines used will vary between states and territories and more information can be obtained by contacting your State or Territory Health Department.

Children who had already commenced their immunisation schedule with OPV and require catch-up vaccination for poliomyelitis, or children who have recently arrived from a country which still administers OPV, can complete the schedule using IPOL or IPV-containing vaccine.

IPV-containing combination vaccines (diphtheria-tetanus-acellular pertussis-inactivated poliomyelitis) recommended for use in persons aged  $\geq 8$  years are Adacel Polio and Boostrix-IPV. The monovalent IPOL can also be administered if the use of combination vaccines is contraindicated.

### Administration

IPV (IPOL) is administered by subcutaneous injection. IPV-containing combination vaccines are given by intramuscular injection.

## Vaccine efficacy/effectiveness

IPV is highly effective in producing immunity to polio virus and protection from paralytic poliomyelitis. After 2 doses of the vaccine, over 90% of recipients develop protective antibodies to all three types of the polio virus. After 3 doses, at least 99% of the recipients will have protection against the disease. Protection against paralytic disease correlates with the presence of antibodies against the polio virus.

IPV appears to produce less local gastrointestinal immunity than does OPV, so it is possible that persons who receive IPV are more readily infected with wild-type polio virus than OPV recipients.

The exact duration of protection with IPV is not known with certainty. However, evidence shows that IPV provides protection for many years after a complete course.

## Vaccine safety

Inactivated poliomyelitis vaccines can be safely administered to either persons with impaired immunity or to persons living with someone with impaired immunity.

IPOP and IPV-containing vaccines may cause erythema, pain, and induration at the injection site. Other symptoms reported following administration of IPOP or IPV-containing vaccines in young babies include fever, crying and decreased appetite.

## Contraindications/precautions

The only absolute contraindications to IPV (IPOP) or IPV-containing vaccines are anaphylaxis following a previous dose of the vaccine, or anaphylaxis following any component of the vaccine.

## References

1. Department of Health and Human Services, Centers for Disease Control and Prevention (CDC). Poliomyelitis. In: Atkinson W, Wolfe C, Hamborsky J, McIntyre L, eds. *Epidemiology and prevention of vaccine-preventable diseases*. 11th ed. Washington DC: Public Health Foundation; 2009: 231-243.
2. Poliomyelitis, acute. In: Heymann DL, ed. *Control of communicable diseases manual*. 19th ed. Washington, D.C.: American Public Health Association; 2008: 484-491.
3. Melnick JL. Current status of poliovirus infections. *Clinical Microbiology Reviews* 1996;9:293-300.
4. Drutz JE, Ligon BL. Polio: its history and its eradication. *Seminars in Pediatric Infectious Diseases* 2000;11:280-286.
5. Howard RS. Poliomyelitis and the postpolio syndrome. *BMJ* 2005;330:1314-1318.
6. Roberts JA, Grant KA, Ibrahim A, Thorley BR. Annual report of the Australian National Poliovirus Reference Laboratory, 2007. *Communicable Diseases Intelligence* 2008;32:308-315.
7. Hall R. Notifiable diseases surveillance, 1917 to 1991. *Communicable Diseases Intelligence* 1993;17:226-236.
8. Roche P, Spencer J. Polio eradication in Australia and the world [editorial]. *Communicable Diseases Intelligence* 2002;26:113-117.
9. Kennett ML, Brussen KA, Wood DJ, et al. Australia's last reported case of wild poliovirus infection. *Communicable Diseases Intelligence* 1999;23:77-79.
10. Burgess MA, McIntyre PB. Vaccine-associated paralytic poliomyelitis. *Communicable Diseases Intelligence* 1999;23:80-81.
11. D' Souza RM, Kennett M, Watson C. Australia declared polio free. *Communicable Diseases Intelligence* 2002;26:253-260.
12. Thorley BR, Brussen KA, Elliott EJ, Kelly HA. Vigilance is required for Australia to remain polio free. *Medical Journal of Australia* 2006;184:474-475.
13. Thorley B, Kelly H, Roberts J. Importation of wild poliovirus into Australia, July 2007. *Communicable Diseases Intelligence* 2007;31:299.
14. Conclusions and recommendations of the Advisory Committee on Poliomyelitis Eradication, November 2008. *Weekly Epidemiological Record* 2009;84:17-28.
15. D'Souza R, Kennett M, Antony J, Longbottom H, Elliott E. Acute flaccid paralysis surveillance in Australia progress report 1995-1998. *Communicable Diseases Intelligence* 1999;23:128-131.
16. Kelly H, Brussen KA, Lawrence A, et al. Polioviruses and other enteroviruses isolated from faecal samples of patients with acute flaccid paralysis in Australia, 1996-2004. *Journal of Paediatrics and Child Health* 2006;42:370-376.
17. Zurynski YA, Mahajan D, Elliott EJ. Australian Paediatric Surveillance Unit annual report, 2007. *Communicable Diseases Intelligence* 2008;32:430-434.
18. MacLennan C, MacLennan J. What threat from persistent vaccine-related poliovirus? *Lancet* 2005;366:351-353.
19. Inactivated poliovirus vaccine following oral poliovirus vaccine cessation. *Weekly Epidemiological Record* 2006;81:137-144.