

8 Poliomyelitis

8.1 Introduction

Although a 3500-year-old Egyptian stele depicts a man with the characteristic features of poliomyelitis (polio), the first written description of polio as a distinct disease was by Michael Underwood in 1789. The epidemiology of polio changed from endemic to periodic epidemics, starting in Sweden and Norway in the late 19th century and then affecting other industrialised countries. These changes were presumably due to improvements in hygiene increasing the average age of infection, which is more likely to be symptomatic in older children and adults.

In 1908 the association of polio with a specific infectious agent was recognised by Landsteiner and Popper. Salk introduced his inactivated vaccine in 1955 and Sabin the attenuated live virus vaccine in 1960. With the introduction of effective vaccines, the devastating epidemics that had swept through Western Europe, the United States (US), Australia and New Zealand every two to three years disappeared.

Global poliomyelitis eradication

In 1988 the World Health Organization (WHO) estimated an annual world toll of about 350,000 cases of paralytic polio, and the World Health Assembly set the target of global polio eradication by the year 2000. In 2000, 722 cases were confirmed worldwide and the new target set for eradication was 2005. Polio continued to occur in the six countries where it remained endemic: Nigeria, India, Pakistan, Afghanistan, Niger and Egypt. Then in October 2005¹ WHO reported not only that there had been cases in the six endemic countries, but also outbreaks in 10 countries that had been polio free. These countries, which were reinfected in late 2004/05, were Somalia, Indonesia, Yemen, Angola, Ethiopia, Chad, Sudan, Mali, Eritrea and Cameroon. Yemen and Indonesia were reinfected after there had been no cases of wild polio since 1995 and 1996.

The spread across Africa through to Indonesia is thought to be a result of the failure of immunisation programmes in Nigeria, where immunisation against polio ceased in parts of the country for 12 months in 2003/04, as well as the failure to maintain high immunisation coverage in other countries. The Advisory Committee on Polio Eradication to WHO met in 2005 and estimated that polio could be eliminated within the next six months everywhere except Nigeria, where elimination will not be achieved for at least 12 months. A monovalent oral polio vaccine (mOPV1) will be used instead of the usual trivalent oral polio vaccine. The monovalent vaccine builds immunity faster, and its use in Egypt and some areas of India successfully stopped polio transmission. Poliovirus 2 has been eliminated since 1999, and type 3 is limited to three areas of focal transmission.

Until eradication throughout the world is confirmed there is a risk of polio returning to New Zealand. Polio vaccination will continue worldwide until the WHO authorises cessation. This is unlikely to occur in the foreseeable future.

The Americas were certified polio free in 1994. The Western Pacific, which includes New Zealand, was the second region to be certified polio free in October 2000, with no indigenous polio cases since March 1997. This is an impressive achievement given that there were over 6000 cases notified in the Western Pacific region in 1990 at the beginning of the programme. For a region to gain certification as polio free, all countries must provide details of their immunisation programmes, the disease surveillance programmes, information on immunisation delivery and coverage, and reports on the country's systems for identification, laboratory testing and diagnosis of cases of acute flaccid paralysis (AFP) over at least three years.

In New Zealand, poliomyelitis is a notifiable disease. Institute of Environmental Science and Research (ESR) laboratories perform reference testing for the poliovirus, and AFP is investigated and reported by paediatricians to the New Zealand Paediatric Surveillance Unit at the University of Otago.

For information on outbreaks caused by the vaccine virus, see section 8.3.

8.2 The illness

Humans are the only reservoir of the poliovirus and infection is more common in young children. The virus is transmitted by the faecal–oral route or by pharyngeal secretions.

Poliomyelitis is the acute illness following infection of the gastrointestinal tract with one of three types of poliovirus: types 1, 2 and 3. The virus is highly neurotropic and its primary effect occurs in the neurones of the spinal anterior horn or the motor ganglia of the brain stem. Infection may be clinically inapparent in up to 95 percent of infections, or range in severity from a non-paralytic fever to viral meningitis and flaccid paralysis.

Symptoms include fever, headache, gastrointestinal disturbances, malaise, stiffness of the neck and back, and pain in the limbs, back and neck, with or without paralysis. In children who develop paralysis, the illness may be biphasic, the initial phase of one to three days duration being indistinguishable from other viral infections. The patient appears to recover, only to be struck down abruptly two to five days later with meningism, followed by paralysis. In adults and adolescents the illness presents with a gradual onset of paralysis and pain without the early symptoms.

Asymptomatic people with the infection will shed the virus in their stool and through poor hygiene spread the infection to others. Infection rates may be as high as 100 percent in households where there are non-immune young children, although paralysis may occur in only 0.1–2 percent of infected individuals. Paralysis is more common in adults, occurring in up to 1 in 75 cases of infection. Case fatalities from paralytic polio vary from 2–10 percent and increase markedly with age.

The incubation period for poliomyelitis is commonly 7–14 days for paralytic disease, with a reported range of 3–21 days. The risk of transmission of infection is greater

for 7–10 days prior to and following the onset of symptoms. The virus persists in the pharynx for approximately one week and in the faeces for three to six weeks or longer, particularly in the immune suppressed. The diagnosis may be confirmed by isolation of the virus from two faecal specimens taken at least 24 hours apart in the first 14 days after the onset of paralysis. Serological tests are available, although virus isolation is required to confirm the diagnosis.

The post-polio syndrome occurs some 30–40 years after poliomyelitis. The cause is not known but is probably related to the ageing or death of nerves and muscles that were compensating for the original damage. Patients experience muscle pain and exacerbation of existing muscle weakness. The risk of developing post-polio syndrome is greater in women than in men, and the risk increases with time since the episode of acute polio.²

Vaccine associated paralytic poliomyelitis (VAPP)

After receiving the OPV most infants excrete the polio vaccine virus for about six weeks. Their family and other contacts are exposed to the vaccine virus and the contacts may then excrete the virus in faeces. There is a small risk that the vaccine virus causes VAPP (see sections 8.1, 8.3 and 8.6) in a vaccine recipient or non-immune contact. VAPP presents with AFP from 7 to 30 days after vaccination in the recipient and from 7 to 60 days in the contact of a vaccine recipient. The immune suppressed are more likely to suffer VAPP, whether they acquire infection directly or as a contact.

8.3 Epidemiology

Before poliovirus vaccines were available, cases of poliomyelitis occurred sporadically and in epidemics in industrialised countries of temperate zones. Cases were more common in the summer and autumn but with some variation from year to year. In tropical countries, where the virus still circulates, there is no seasonal pattern.

Characteristically, poliomyelitis is a disease of young children and adolescents. However, with improvements in living standards a greater number of cases have occurred in older individuals, with an associated higher frequency of paralytic disease. Paralytic disease is a particular risk in early adult life. In countries where polio was endemic, most children acquired antibodies to all three subtypes by five years of age and most paralytic disease occurred in children under three years of age. As the disease became rare because of the effective vaccines and immunisation programmes, only sporadic outbreaks occurred in those groups not reached by the programmes, whether because of socioeconomic circumstance or because of specific religious beliefs. This has been shown by outbreaks of poliomyelitis in unimmunised groups in the Netherlands in 1993, and in Israel.^{3,4} More recently there have been outbreaks of acute paralysis in Egypt, the Dominican Republic, Haiti, the Philippines and Madagascar associated with the sustained circulation of vaccine derived fully neurovirulent strains of poliovirus when population immunity was low.^{5,6,7,8}

In 2005 the US reported poliovirus infections⁹ with a vaccine derived poliovirus (VDPV) of type 1 in four unvaccinated children of an Amish community, whose members were mostly unvaccinated. These were the first identified infections since 2000, when the US switched from OPV to IPV. The VDPV was identified in an immune deficient infant, and subsequent screening of community members found the virus in three asymptomatic unvaccinated siblings of another household. Analysis of the VDPV suggested that because of differences between the virus found in the children and the usual oral vaccine derived strain, this VDPV had been replicating and circulating for up to two years, and was likely to have originated from a visitor from a country where OPV is still used.

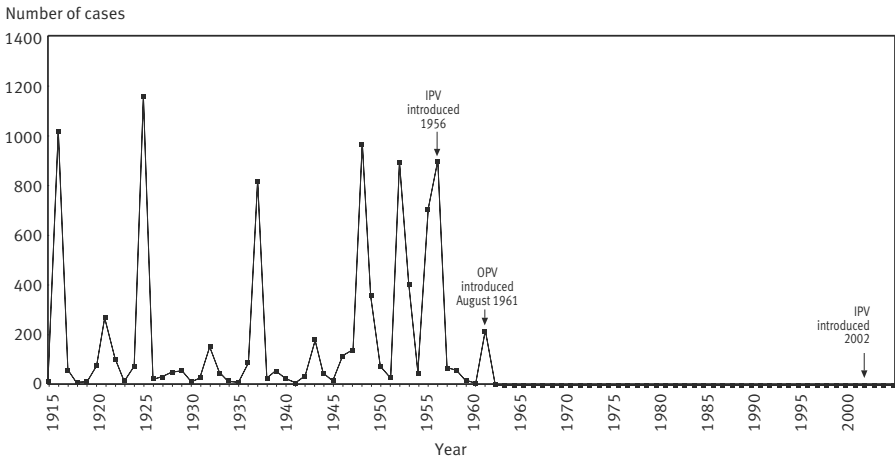
Intensive polio immunisation campaigns throughout the world have been successful in reducing cases of paralytic poliomyelitis. The global eradication programme uses OPV. As indigenous polio is eliminated from a country the risk remains that paralytic disease may be imported from a country where the wild virus still circulates. However, with the success of the global immunisation programmes, infection with the wild virus is now uncommon and localised to specific countries, so that the risk of VAPP is now higher than the risk of imported wild virus disease. This has led countries such as the US and New Zealand, and Australia from 1 November 2005, to change from OPV to IPV to eliminate the risk of VAPP. The risk of importing wild type or neurovirulent oral vaccine derived strains means that maintaining high vaccine coverage is essential.^{10,11}

New Zealand epidemiology

Deaths from polio were reported in official statistics from 1908, and notifications show large epidemics (about 1000 cases) in 1916, 1925 and 1937. Polio epidemics became more frequent and prolonged during 1948/49, 1952/53 and 1955/56. The use of the Salk (inactivated) vaccine delayed the next epidemic until 1961; this epidemic was halted by the use of the Sabin (oral) vaccine.

Following mass immunisation campaigns with OPV in 1961 and 1962 there have been no cases of poliomyelitis from indigenously acquired wild type poliovirus in New Zealand. It appears likely that poliovirus circulation was effectively stopped by these campaigns because of the high population coverage achieved (see Figure 8.1).

Figure 8.1: Numbers of cases of poliomyelitis, 1915–2004



Since 1962 there have been four definite laboratory confirmed cases of VAPP and two probable cases of VAPP identified in New Zealand (see Table 8.1). Two cases were notified in 1970, one in a vaccinee and the other in an unimmunised contact. A probable case notified in 1977 may not have been polio as no virus was isolated and the child was reported to have made a ‘good recovery’. There were two notifications in 1990, one of a child notified on suspicion who was subsequently diagnosed as having Guillain-Barré syndrome. The 1990 case reported as VAPP was an unimmunised adult contact with a high titre on serological testing, although no stool specimens were taken to support this diagnosis. The two most recent cases in 1998 were a child who had received their second dose of OPV, and an unimmunised mother following her infant’s first dose of the vaccine.¹² The number of cases in New Zealand was higher than would be expected from the estimated risk of VAPP in the US.

Table 8.1: Cases of VAPP in New Zealand, 1962–2000, confirmed and probable cases

Year	Number of notified cases of VAPP in vaccine recipient	Number of notified cases of VAPP in contacts of a vaccine recipient	Number notified but not VAPP	Total probable VAPP*	Total number of laboratory confirmed cases of VAPP
1970	1	1			2
1977	1			1**	
1990		1	1***	1	
1998	1	1			2
Total 1962–2000				2	4

* No laboratory confirmation but clinical course consistent.

** Child made a good recovery.

*** Final diagnosis Guillain-Barré syndrome.

In 1976 there was one case of imported poliomyelitis. An infant who arrived in New Zealand, having become ill in Tonga, had wild poliovirus. This case is not included in Table 8.1 above because the child had become ill outside New Zealand.

There has been no case of VAPP detected since the change in the National Immunisation Schedule (Schedule) to IPV in 2002.

History of the New Zealand Immunisation Schedule

Limited supplies of the Salk vaccine (IPV) became available in 1956 and immunisation initially targeted eight and nine year old children.¹³ As supplies improved, immunisation was extended to include all 5 to 10 year olds, then children 11 to 15 years of age, with approximately 80 percent coverage. By 1960 immunisation was offered to everyone between six months and 21 years of age (with three doses of vaccine).¹⁴

The Sabin vaccine (OPV) was introduced in August 1961, initially for children up to 12 months of age; eight months later it was made available to all school children. On completion of this programme in September 1962 the vaccine was offered to adolescents and adults.¹⁵

In 1967 OPV was given with diphtheria, tetanus and whole cell pertussis (DTwP) vaccine at three, four, five and 18 months of age. The four-month dose was dropped in 1971 when the DTwP dose was dropped from the Schedule. An extra dose of polio vaccine was added at five years of age in 1980, based on serological data, which showed decreased immunity to poliovirus types 1 and 3 in school entrants.

In 1996, as part of the National Immunisation Schedule changes, the three dose primary series was moved to the first year of life, with OPV given at six weeks, three

months and five months of age. The booster dose was moved to 11 years of age to be given at the same time as the measles, mumps and rubella (MMR) and adult tetanus diphtheria (Td) vaccines. In 2001 the Schedule was changed to give the fourth dose of OPV at four years of age at the same time as the second dose of MMR. Students aged between 5 and 10 years in 2001, who did not receive the fourth dose of polio vaccine at four years of age, are offered a dose at 11 years; this will continue until the end of 2007. From 2002 IPV replaced OPV for all doses (see section 8.4).

Change to inactivated polio vaccine on the National Immunisation Schedule in 2002

The Sabin live attenuated vaccine is given orally as oral polio vaccine (OPV). Because of the advantages of oral administration, OPV has been used in most countries of the world to control poliomyelitis. However, as the wild poliovirus infection becomes uncommon in a population through high immunisation coverage and low exposure, the risk of adverse events following the vaccine must be weighed against the decreased risk of the disease. Following OPV there is a small risk of vaccine associated paralytic poliomyelitis (VAPP) in the vaccine recipient or a non-immune contact. It has been estimated in the US that the risk is one case of VAPP per 2.4 million doses of OPV distributed. The risk is higher after the first dose of vaccine and estimated to be one case of VAPP for every 750,000 children vaccinated¹⁶ (see section 8.2). VAPP presents with acute flaccid paralysis typical of poliomyelitis, and the vaccine virus may be isolated from faecal specimens from cases.

In 2006 the Schedule will continue to be three doses of DTaP-IPV in the first year of life, and a booster at age four years. Those children who have not received four doses of polio vaccine will be offered IPV with the dTap, as dTap-IPV (BOOSTRIX®-IPV) at 11 years of age in 2006/07. Beyond 2007 it is expected that dTap will be offered at age 11 years (as all children should then have received four doses of polio vaccine by age four years). Note that if the dose of IPV at 11 years is the fifth dose, this is not expected to increase reactivity and may be safely given.

8.4 Vaccines

Since February 2002 IPV has been the publicly funded poliovirus vaccine on the National Immunisation Schedule. Vaccines available are as follows.

1 Inactivated polio vaccine (IPOL, Sanofi Pasteur/MSD)

IPV contains three strains of poliovirus (40D antigen units of the Mahoney, 8D units of the MEFl, and 32D antigen units of the Saukett strains), inactivated by formaldehyde and containing phenoxylethanol as a preservative. The viruses are highly purified and grown in cultures of VERO cells, a continuous line of monkey kidney cells. Trace amounts of neomycin, streptomycin, polymyxin B and bovine serum albumin may be present. This IPV vaccine is an 'enhanced potency' form of IPV. It is a different formulation with a greater antigenic content than the IPV introduced by Jonas Salk.

2 Diphtheria, tetanus, acellular pertussis and inactivated polio vaccine (DTaP-IPV, INFANRIX™-IPV, GSK)

DTaP-IPV is the Schedule vaccine for infants and children. The IPV in this combined vaccine is expected to provide protection equivalent to IPV alone. The IPV in INFANRIX™-IPV is also produced from a VERO cell line. (See section 6.4.)

3 Adult diphtheria, tetanus, acellular pertussis and inactivated polio vaccine (dTAp-IPV, BOOSTRIX®-IPV, GSK)

This vaccine is available as a booster against diphtheria, tetanus, pertussis, and polio for individuals from the age of seven years. The dTap-IPV will be the Schedule vaccine offered to children at age 11 years (school year 7). (See section 6.4.)

Efficacy of IPV

Virtually all infants will seroconvert after three doses of IPV, and more than 85 percent will seroconvert after two doses. The efficacy of IPV is more than 90 percent.¹⁷ Follow up studies show that following two or three doses in the first year of life and a booster in the second year, close to 100 percent show seropositivity four years later.

The need for any further boosters is not clear. Some experts believe immunological memory is established and no further doses are necessary because the vaccinated individuals develop an anamnestic response if further challenged. The response to future infection is likely to be the same.¹⁸

Efficacy of DTaP-IPV and dTap-IPV

One month after receipt of the three dose primary vaccination series with DTaP-IPV, the overall seropositivity for poliovirus serotypes 1, 2 and 3 was 99.5 percent. The immune response to the diphtheria, tetanus, acellular pertussis and IPV components of DTaP-IPV is similar to that for DTaP and IPV administered separately and it is therefore expected that the clinical protection of the DTaP component is comparable.

One month after dTap-IPV the immune responses to poliovirus were similar to the responses to IPV alone.

Dosage

Follow the manufacturer's directions. IPV is given by subcutaneous injection (0.5 mL). DTaP-IPV is given by intramuscular injection (0.5 mL). The dTap-IPV is also given by intramuscular injection (0.5 mL), preferably in the deltoid region. (See section 2.3 for needle sites and sizes.)

Oral poliomyelitis vaccine

OPV is no longer used in New Zealand. However, OPV will continue to be used in many countries because it remains the vaccine for the WHO Expanded Programme of Immunization. OPV contains poliovirus types 1, 2 and 3 grown either on monkey kidney or human diploid cells.

8.5 Recommended immunisation schedule

The recommended immunisation schedule is four doses of polio vaccine given at six weeks, three months, five months and four years of age, before school entry, as DTaP-IPV.

IPV is given as the vaccine dTap-IPV at age 11 years (school year 7). This will continue until 2007, when the children who were between 5 and 10 years of age in 2001 will have already received four doses of polio vaccine.

Preterm infants

Preterm infants who are still in hospital at six weeks of age should receive IPV as part of the usual childhood schedule.

Adults and children

Previously unimmunised individuals are given a primary course of three doses of IPV. The recommended interval is eight weeks between the first two doses, followed by the third dose approximately 12 months later. However, if necessary they may be given with a minimum of four weeks between doses.

If a course of vaccine is interrupted, it may be resumed without repeating prior doses. A booster may be given if 10 years has elapsed since the last dose and exposure is possible (eg, a traveller to an area where the virus circulates).

A combination of OPV and IPV is acceptable. Four doses, in any combination of OPV and IPV (given at least four weeks apart) by the time of school entry, is considered a complete vaccination series. This is particularly relevant when a child who was begun on a course of OPV in another country moves to New Zealand. It is not necessary in that situation to start the full IPV series, and it is acceptable to continue the series using IPV for the final doses.

Recommendations for other groups

Booster doses of IPV are recommended for:

- travellers to areas or countries where poliomyelitis remains endemic – a booster of IPV should be offered to these individuals if more than 10 years has elapsed since their last dose; where there is uncertainty about previous immunisation, a full course of IPV should be started (see Health Advice for Overseas Travellers, Ministry of Health, 1996)
- health care workers in direct contact with a case of poliomyelitis
- individuals at particular risk of exposure (eg, laboratory workers handling specimens, which may contain wild or vaccine derived polioviruses); a booster dose of IPV vaccine should be considered every 10 years in these individuals.

There is no evidence of the need for boosters, but they are recommended to reduce any possible risk from waning immunity in situations of increased risk of exposure.

Note: all immune suppressed individuals and their household contacts may receive IPV. OPV was contraindicated in the immune suppressed because of the risk of VAPP. There is no risk of VAPP with IPV.

8.6 Expected responses and adverse events following immunisation (AEFI)

IPV

Expected responses

A small proportion of individuals experience mild local symptoms following IPV. Erythema, induration and pain occur in 33, 1 and 13 percent of all vaccines, respectively, and symptoms of sleepiness, fussiness, crying and change in feeding have been noted in more than 5 percent of infants (manufacturer's data sheet for IPOL). There is no poliovirus excretion following IPV.

Adverse events following immunisation

Serious adverse events are very rare following administration of the IPV currently manufactured. More than 90 million doses have been used with no association with subsequent polio, Guillain-Barré Syndrome, anaphylaxis or other serious reaction. IPV contains streptomycin and neomycin, and hypersensitivity reactions to these are possible.

For adverse events after DTaP-IPV and dTap-IPV, see Pertussis chapter, section 6.6.

Any severe or unexpected reactions should be reported to CARM, PO Box 913, Dunedin, using the prepaid postcard HP3442 (see section 2.4) or via online reporting at <http://carm.otago.ac.nz>. If the patient or parent/caregiver does not consent to being identified, the report should be made without personal identification.

OPV

OPV is no longer used on the National Immunisation Schedule in New Zealand. VAPP following OPV is discussed above. There was concern after some batches of OPV produced before 1962 were found to be contaminated with SV40, a simian (monkey) virus, which causes cancer in rodents. Production was changed, and after 1963 SV40 was excluded from all vaccines. Long term studies of vaccine recipients and their offspring do not support any association of exposure to SV40 contaminated vaccine with human cancer.¹⁹

8.7 Contraindications

See section 1.9 for general contraindications for all vaccines. IPV is contraindicated if there is a history of an anaphylactic reaction to a previous dose of IPV or to the antibiotics streptomycin, neomycin or polymyxin.

During pregnancy

No adverse effects on the fetus have been reported following administration of polio vaccine during pregnancy, but immunisation should not be carried out during the first or second trimester unless there are compelling reasons to do so, such as planned travel to an endemic area. However, pregnant women are particularly susceptible to paralytic polio. If a pregnant woman plans to travel to an endemic area, then two doses should be administered four weeks apart prior to departure. If departure cannot be delayed to allow a four week gap then two doses should be given at the maximum possible interval, though protection cannot be guaranteed. If the available interval is less than two weeks, a single dose should be administered.

Use with other vaccines

IPV may be given at the same time as inactivated or attenuated virus vaccines.

8.8 Control measures

All cases of poliomyelitis should be notified immediately on suspicion to the local medical officer of health. Case investigation and surveillance for AFP will continue in New Zealand to monitor the successful eradication of polio. All cases of AFP should be immediately notified to the local medical officer of health and investigated as suspected poliomyelitis.

A stool sample needs to be taken within 14 days of onset to search for poliovirus. Serology should also be done. All paediatricians or physicians caring for any person less than 15 years of age with AFP should report the case to the New Zealand Paediatric Surveillance Unit (NZPSU) based at the University of Otago. The NZPSU is responsible for sending case investigation and follow up forms to clinicians to continue to monitor that New Zealand has eradicated polio and to provide information to the WHO.

There are no outbreak control measures recommended if the case is VAPP.

A single case of paralytic wild poliomyelitis would be a major public health emergency. Control measures would involve mass vaccination of all people in the immediate neighbourhood, regardless of a previous history of immunisation, except where there are genuine contraindications. In those who have not previously received vaccine, a full course of three doses should be given at monthly intervals. There would need to be a careful search for the source of the virus and for other potential cases.

Although polio has been eradicated in the WHO Western Pacific Region, New Zealand will need to continue with high levels of IPV coverage. This is because of the small risk that polio may be imported from another region where polio remains endemic.

For more details on control measures, refer to *Control of Communicable Diseases Manual*.²⁰

References

- 1 Advisory Committee on Polio Eradication: WHO. 2005. New tools move polio eradication drive into final stage. News release WHO/49, 12 October 2005.
- 2 Ramlow J, Alexander M, LaPorte R, et al. 1992. Epidemiology of the post-polio syndrome. *Am J Epidemiol* 136: 769–86.
- 3 Oostvogel PM, van Wijngaarden JK, van der Avoort HGAM, et al. 1994. Poliomyelitis outbreaks in an unvaccinated community in the Netherlands, 1992–93. *Lancet* 344: 665–70.
- 4 Goldblum N, Gerichter CB, Tulchinsky, et al. 1994. Poliomyelitis control in Israel, the West Bank and Gaza Strip: changing strategies with the goal of eradication in an endemic area. *Bull WHO* 72: 783–96.
- 5 Kew O, Morris-Glasgow V, Landaverde M, et al. 2002. Outbreak of poliomyelitis in Hispaniola associated with circulating type 1 vaccine-derived poliovirus. *Science* 296: 356–9.
- 6 Yang CF, Naguib T, Yang SJ, et al. 2003. Circulation of endemic type 2 vaccine-derived poliovirus in Egypt from 1983–93. *J Virol* 77: 8366–77.
- 7 Shimizu H, Thorley B, Paladin FJ, et al. 2004. Circulation of type 1 vaccine-derived polioviruses in the Philippines in 2001. *J Virol* 78: 13512–21.
- 8 US Centers for Disease Control and Prevention. 2001. Public health dispatch: acute flaccid paralysis associated with circulating vaccine-derived poliovirus, Philippines 2001. *MMWR* 50 (40): 874–5.
- 9 US Centers for Disease Control and Prevention. 2005. Poliovirus infections in four unvaccinated children: Minnesota, August – October 2005. *MMWR* 54 (41): 1053–5.
- 10 Rousset D, Rakoto-Andranarivelo M, Razafindratsimandresy R, et al. 2003. *Emerg Infect Dis* 9: 885–7.
- 11 Huang QS, Greening G, Baker MG, et al. 2005. Persistence of polio vaccine virus after its removal from the immunisation schedule in New Zealand. *Lancet* 366: 396–6.
- 12 Edwards EA, Grant CC, Huang QS, et al. 2000. A case of vaccine-associated paralytic poliomyelitis. *J Paediatr Child Health* 36: 408–11.
- 13 Annual Report of the Director-General of Health. *Appendices to the Journals of the House of Representatives* 1957, H31: 18.
- 14 Report of the Department of Health. *Appendices to the Journals of the House of Representatives* 1960, H31: 7.
- 15 Adults now it's your turn! 1962. *Health* 14(3): 7.
- 16 US Centers for Disease Control and Prevention. 1997. Poliomyelitis prevention in the US: introduction of a sequential vaccination schedule of inactivated poliovirus vaccine followed by oral poliovirus vaccine: recommendations of the Advisory Committee on Immunization Practice. *MMWR* 46(RR3): 1–25.
- 17 Plotkin SA, Vidor E. 2004. Inactivated polio vaccine. In: SA Plotkin, WA Orenstein (eds). *Vaccines*. (4th edition). Philadelphia: WB Saunders Company.
- 18 Plotkin SA, Vidor E. 2004. Inactivated polio vaccine. In: SA Plotkin, WA Orenstein (eds) *Vaccines*. (4th edition). Philadelphia: WB Saunders Company.
- 19 Strickler HD, Rosenberg PS, Devesa SS, et al. 1998. Contamination of poliovirus vaccines with simian virus 40 (1955–1963) and subsequent cancer rates. *JAMA* 279: 292–5.
- 20 Heymann DL (ed). 2004. *Control of Communicable Diseases Manual* (18th edition). Washington: American Public Health Association.