*Epidemiol. Infect.* (1995), **115**, 289–298



# Poliomyelitis in the Netherlands: a review of population immunity and exposure between the epidemics in 1978 and 1992

H. C. RÜMKE[1], P. M. OOSTVOGEL[2]*, G. VAN STEENIS[3] AND A. M. VAN LOON[2]

[1] *Laboratory for Clinical Vaccine Research,* [2] *Laboratory of Virology, and WHO Collaborative Centre for Reference and Research on Poliomyelitis,* [3] *Laboratory for the Control of Biological Products, National Institute of Public Health and Environmental Protection (RIVM), PO Box 1, 3720 BA Bilthoven, The Netherlands*

(*Accepted 23 March 1995*)

## SUMMARY

An overview of serological and virological studies on poliomyelitis in the Netherlands between two epidemics in 1978 and 1992 is given. Three unvaccinated patients acquired poliomyelitis abroad. In the Netherlands vaccination coverage with quadruple DPT–IPV vaccine is very high. The strong immunogenicity of inactivated poliovirus vaccine was confirmed in a cohort of children, reflected in age-stratified antibody profiles of the population. Adults born in the pre vaccination era appeared in general protected, but 10–25% of persons born between 1930 and 1945 lacked neutralizing antibodies. Revaccination induced a booster type of antibody response in 75–90% of such persons, indicating immunological memory and protection.

Virological studies on adopted children from other countries, patients with indications for viral examination, and river waters showed that the Netherlands was regularly exposed to polio virus (PV), without signs of indigenous transmission. Persons found to carry PV or their close contacts had travelled to a PV endemic country. Most of 557 isolates were vaccine-derived, only 8% were wild type viruses. Despite their presence, up to 1992 the well-known susceptibles for PV in the Netherlands were shielded by the herd immunity of the Dutch population.

## INTRODUCTION

In the Netherlands, two outbreaks of poliomyelitis were experienced in 1978 and 1992/3, despite a very high vaccination coverage [1–4]. This paper gives an overview of the epidemiology of polio in the Netherlands in the years between these two epidemics, and the results of serological and virological studies. These data give the background for the most recent epidemic in 1992/3.

## POLIO VACCINATION AND COVERAGE

Inactivated poliovaccine (IPV) is used in the Netherlands for routine immunizations of children, while live oral poliovirus vaccine (OPV) is used only

* Present address: Westeinde Hospital, The Hague, the Netherlands.

during polio epidemics. Childhood immunizations are fully integrated in the primary health care system [5]. Vaccination against poliomyelitis began in 1957 with a 4-year national campaign in which IPV was given to over 90% of children born after 1945. Since 1962, a quadruple DPT—IPV vaccine against diphtheria, pertussis, tetanus and poliomyelitis has been used [6, 7]. From 1965 onwards, the vaccination schedule included 4 doses of DPT–IPV vaccine at 3,4,5 and 11 months of age, followed by 2 doses of DT–IPV at 4 and 9 years of age. Since the introduction of the microcarrier culture technique for cultivation of viruses in 1978 [8], the polio components of DPT–IPV and DT–IPV have been: (killed) PV type 1 (PV1) (Mahoney strain), PV2 (MEF 1 strain) and PV3 (Saukett strain) at a concentration of 40–4–7·5 D-antigen units, respectively per dose.

For many years now, the vaccination coverage has been very high, and currently the national coverage in infants receiving at least three doses of DPT–IPV is 97%. In some municipalities (35 in 1992) or villages, however, vaccination coverage is below 90%, with some even as low as 60%. These are concentrated in an area stretching as a belt from the south-west to the north-east of the country, where there are several communities who refuse vaccinations for their children and themselves on religious grounds [5]. Many of these municipalities were affected by polio during the recent epidemics [1–4]. Initiatives to increase acceptance of vaccination in these groups in our country have not succeeded. In addition, 400000–500000 children and adults are not vaccinated for other reasons, and live scattered all over the country. They appear to have multiple reasons for not being vaccinated, such as ignorance with the health system, socio-ethnic problems in immigrant populations, 'illegal' residency, or objections against vaccinations because of nature-based attitudes towards life [5].

## HISTORY AND INCIDENCE OF POLIOMYELITIS

Poliomyelitis is a notifiable disease in the Netherlands. The polio immunization campaign from 1957–60 was launched directly after the large polio epidemic in 1956, followed by a strong decline of reported polio cases (Fig. 1). Between 1961 and 1991, 293 cases were notified (PV1: 269; PV2: 1; PV3: 23) [1, 6, 7]. Since 1965, all cases of poliomyelitis occurred in unvaccinated persons, with one exception; a boy who in 1966 had received only one dose of DPT–IPV as an infant, and caught the disease in 1968 [1, 6]. Between 1961 and 1971 small local outbreaks caused by PV1 occurred in communities with low vaccination coverage because of religious objections. In 1978, a PV1, and in 1992 a PV3 epidemic occurred [1–4]. These epidemics affected several provinces, and were not limited to municipalities with a low vaccination coverage, but occurred specifically among persons belonging to a few distinct orthodox protestant denominations that reject vaccinations. These groups had strong social coherence with many intensive contacts within their groups, and lived relatively isolated from the general population. From all over the country most of their children attended one of their four secondary schools [2, 4]. This pattern of life facilitated transmission of PV within the group which included members of the religious groups in other countries [9, 10]. Members of these groups living in municipalities with a high vaccination coverage were also affected. During the last two epidemics in 1978 and 1992 the spread of PV appeared to be limited to the above mentioned risk groups [2, 4]. In

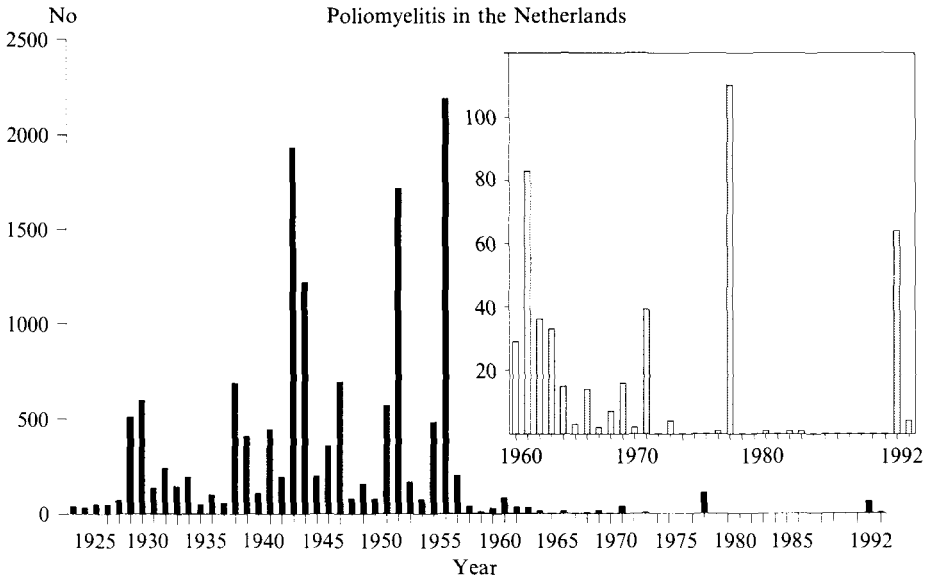


Fig. 1. Annually reported numbers of poliopatients in the Netherlands (data from the Chief Medical Officer). The insert box presents data from 1960 onwards on a larger scale.

non-vaccinated persons belonging to other groups, even those with an almost similar orthodox protestant background and living in the same area, polio patients did not occur, with only a single exception during the most recent epidemic (a 61-year old Roman-Catholic man) [4].

From 1958–84, 11 imported sporadic polio cases were notified. There have been no more imported cases since then. As killed poliovirus vaccine is used exclusively, cases of vaccine-associated paralytic poliomyelitis have not occurred.

Surveillance of PV circulation in the Netherlands in the 1960s and 1970s indicated that endemic transmission probably had ceased [1], though during the epidemic in 1978 a serosurvey among school children born between 1962 and 1968 demonstrated antibodies to PV1, as well as to PV3. The PV3 antibodies were neither explained by vaccinations nor by the epidemic [2]. It was thus inferred that type 3 virus had circulated silently between 1968 and 1978.

## ANTIBODY RESPONSE AFTER POLIOVACCINATION

Results of two studies indicate that the routine vaccinations build a strong immunity against poliomyelitis. The first study included a cohort of children mainly from health care workers recruited by advertising in (para)medical journals. They were followed during the 9 years over which vaccinations were offered (Table 1A). The study was done by RIVM, in cooperation with the Netherlands Institute of Preventive Healthcare, Leiden. A total of 150 children were enrolled between 1979 and 1983. Blood samples were taken before and after each vaccination. After three DPT–IPV vaccinations, nearly all children had neutralizing antibodies. After the revaccinations at about 1.4 and 9 years, all children had antibodies, with increasingly high titres.

The second study was done in Rotterdam in 1989, jointly with the Municipal

https://doi.org/10.1017/S0950268800058416 Published online by Cambridge University Press

Table 1. *Neutralizing poliovirus antibody formation during routine immunizations in the Netherlands*

| Age | Vaccination status* | Type 1 | | Type 2 | | Type 3 | |
|---|---|---|---|---|---|---|---|
| | | %3+† | gmt‡ | %3+ | gmt | %3+ | gmt |
| | | A. Cohort study | | | | | |
| 3 months ($n$ = 125) | Pre vaccination | 71·2 | 4·1 | 69·8 | 4·1 | 64·5 | 3·6 |
| 6 months ($n$ = 118) | Post dose 3 of DPT–IPV | 96·6 | 8·7 | 94·9 | 7·3 | 94·1 | 7·5 |
| 11 months ($n$ = 110) | Pre dose 4 of DPT–IPV | 90·9 | 6·7 | 75·5 | 4·9 | 77·1 | 5·2 |
| 15 months ($n$ = 114) | Post dose 4 of DPT–IPV | 100 | 10·4 | 99·1 | 9·3 | 96·5 | 9·0 |
| 4 years ($n$ = 97) | Pre dose 5 of DT–IPV | 100 | 8·9 | 97·9 | 7·2 | 93·5 | 6·3 |
| 4 years ($n$ = 98) | Post dose 5 of DT–IPV | 100 | 12·4 | 100 | 11·6 | 100 | 11·2 |
| 9 years ($n$ = 97) | Pre dose 6 of DT–IPV | 100 | 11·0 | 100 | 9·8 | 99 | 8·6 |
| 9 years ($n$ = 102) | Post dose 6 of DT–IPV | 100 | 12·8 | 100 | 12·7 | 100 | 12·6 |
| | | B. Rotterdam | | | | | |
| 9 years ($n$ = 214) | Pre dose 6 of DT–IPV | 99·1 | 9·6 | 99·5 | 8·4 | 99·1 | 7·7 |
| 9 years ($n$ = 146) | Post dose 6 of DT–IPV | 100 | 11·3 | 100 | 11·0 | 100 | 9·9 |

* DPT–IPV: vaccine against diphtheria, pertussis, tetanus and poliomyelitis: DT–IPV: vaccine against diphtheria, tetanus and poliomyelitis.

† %3+: Percentage of children with neutralizing titre 8 and higher.

‡ gmt: geometric mean titre (2 log value).

Health Service, in 9-year-old children who were randomly invited from the population register. Blood samples were taken before and 3 months after their sixth DT–IPV vaccination (Table 1B). Almost all pre-vaccination sera were positive against the three types of PV, probably resulting from the previous five vaccinations. After vaccination all children were antibody-positive, and the titres were considerably higher, with an average fourfold increase. In these sera, neutralizing activity against the inducing vaccine strain (Saukett) was found to be comparable to that against the type 3 virus strain that caused the epidemic in 1992/3 [4, 11]. It is assumed that all those who have been immunized according to the national immunization program are well immune against PV.

## AGE-STRATIFIED ANTIBODY STATUS OF THE POPULATION

To assess the antibody status of the Dutch population against poliomyelitis (and other infectious diseases) national sero-epidemiological studies were done in 1980 and 1985. General practitioners of the Dutch 'NIVEL' Sentinel System, which covers about 1% of the population, collected sera from health persons, visiting their practice. Only age and sex were recorded [12, 13]. A total of 1475 sera could be examined (798 in 1980, 677 in 1985). Results are presented as PV antibody prevalence (neutralizing antibody titre $\geqslant 8$), and geometric mean titre by year of birth (Fig. 2). Nearly all of those born after 1945 had antibody titres against PV1, and to a lesser extent, but still over 90%, against PV2 and PV3. Presumably, the antibodies in this group resulted from previous vaccinations, whereas the contribution of immunization by endemic virus is very small. Nearly all those born before 1930 had antibodies. In this group, immunity most probably resulted from contact with endemic PV. A part of the group born between 1930 and 1945 lacked circulating neutralizing antibodies: 10% against PV1, and 22 and 25% respectively against PV2 and PV3, had titres below 8. Geometric mean titres

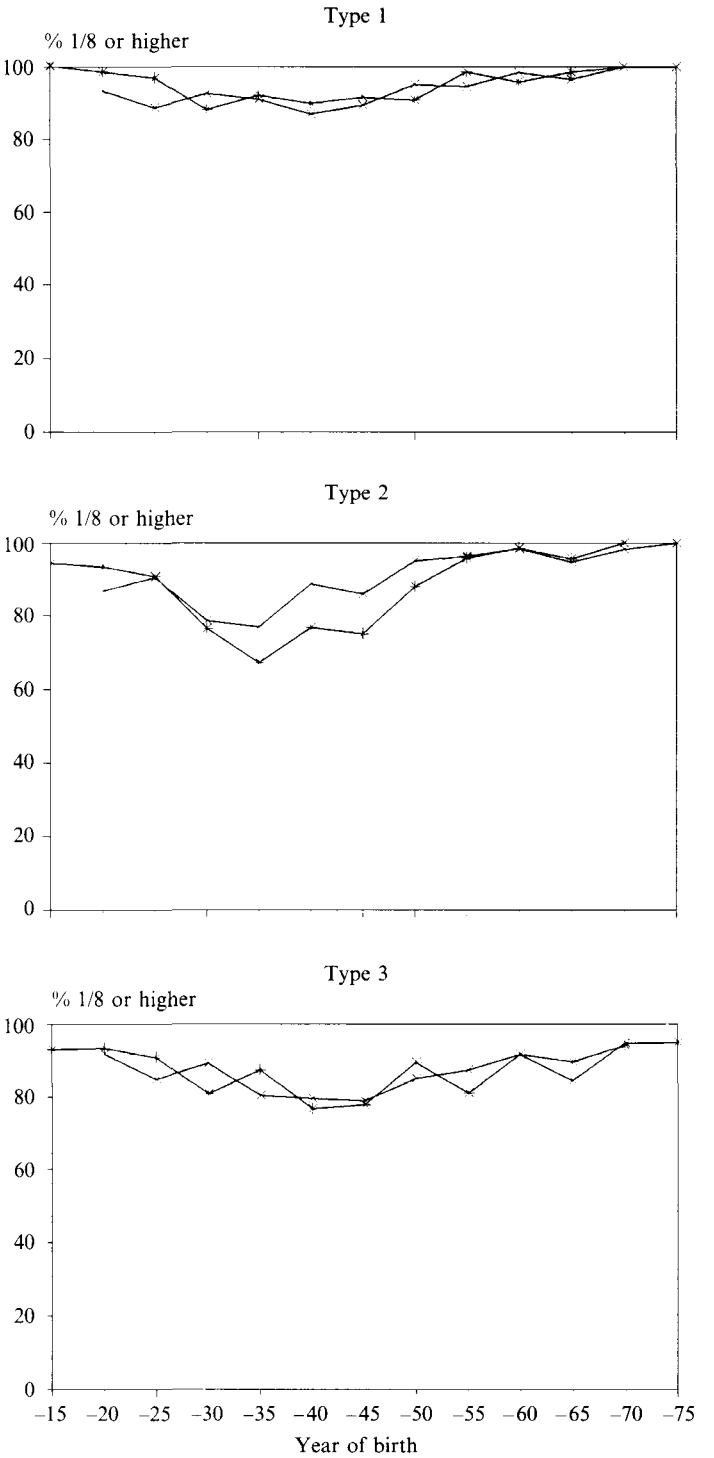


Fig. 2. Neutralizing antibodies against poliovirus in 1475 sera from the Dutch population. Percentages of persons with neutralizing antibody titres of 8 or higher are shown per type of poliovirus by year group of birth. Results from 1980 are indicated by [*], from 1985 by [×].

https://doi.org/10.1017/S0950268800058416 Published online by Cambridge University Press

paralleled antibody prevalence figures, antibodies against PV1 being higher than to PV2 and PV3.

A study in 1986 in the city of Utrecht (Municipal Health Service) among 668 persons born before 1945 randomly selected from the population register, confirmed the findings described above [14]. Moreover, it was found that among persons with a lower socio-economic status both antibody prevalence and mean titres appeared to be higher [14].

To determine whether absent or low titres of antibodies imply that persons are not protected against challenge with PV, we examined a group of 337 persons born between 1901 and 1960 (municipality workers, groups of elderly citizens, RIVM employees). Persons with serum neutralizing antibody titre below 8 were given DT–IPV vaccine. Before vaccination, 24 persons lacked antibodies against PV1. Within 1 week after vaccination, 22 (92%) showed an at least fourfold titre rise. For PV2 and PV3, these figures were 25 of 29 (89%) and 24 of 29 (83%), respectively. These rapid booster reactions demonstrate that more than 80% of these antibody-negative persons most probably had been primed, and were therefore immune.

## ISOLATION OF POLIOVIRUS

All PV strains isolated in Dutch virus laboratories are sent to the Laboratory of Virology of the RIVM for intratypic differentiation with cross-absorbed intratype-specific antisera, used in neutralization tests or enzyme immunoassays [18]. From 1979–90, 895 PV isolates were submitted for intratypic differentiation. Their number decreased over the years. Isolates were obtained from (i) adopted children, (ii) patients with various symptoms requiring viral examination of faeces, and (iii) environmental samples.

(i) Between 1979 and 1989 557 PV strains were isolated from 5868 faecal samples from adopted children who had recently entered the country. The majority of samples were collected by the Amsterdam Municipal Health Service, as partly described earlier [1]. Most of the isolates (92%) were characterized as Sabin virus strains, but 44 (8%) were wild PV strains (Table 2).

(ii) Between 1985 and 1990, 49 PV strains were isolated by virus laboratories in faecal samples from patients: 21 were found to be wild strains (15 PV1, 6 PV3) and 28 were Sabin-like virus strains (Table 2). These isolates came always from households with a history of international travel. The three 'imported' polio patients (see above) were also included in this group. The other patients had no clinical signs of poliomyelitis. Between 1985 and 1990 out of over 46000 faecal cultures wild PV was isolated in only 23 faecal samples.

(iii) Between 1979 and 1989, we received strains from water samples from the rivers Rhine and Meuse for further typing, as part of a study into the quality of drinking water. Large samples of water were concentrated, and a selection of viruses found through the plaque method were typed [16]. From 58 water samples, 289 PV strains were isolated, of which 13 were wild type (PV1, 11; PV2 and PV3: 1). The wild type isolates were found in 1981–3. Water samples from the river 'Drentse Aa', that originates in the Netherlands, never yielded PV, vaccine nor wild.

Table 2. *Isolations of poliovirus in fecal samples of adopted children and patients with indication for viral diagnostics*

| | Adopted children | | | Dutch patients | | |
|---|---|---|---|---|---|---|
| | | Type of virus | | | Type of virus | |
| Year | Total | Vaccine | Wild type 1/2/3 | Total | Vaccine | Wild type 1/2/3 |
| 1979 | 1006 | 37 | 6/6/10 | n.a.* | 0 | 4/–/3 |
| 1980 | 970 | 27 | 5/–/– | n.a. | 1 | 1/–/1 |
| 1981 | 813 | 43 | –/5/– | n.a. | 2 | 1/–/2 |
| 1982 | 765 | 56 | –/–/– | n.a. | 0 | 1/–/– |
| 1983 | 657 | 81 | –/2/5 | n.a. | 2 | 4/–/– |
| 1984 | 384 | 64 | –/–/1 | n.a. | 0 | 3/–/– |
| 1985 | 467 | 57 | –/–/– | 5805 | 5 | 1/–/– |
| 1986 | 373 | 78 | –/–/1 | 6782 | 9 | –/–/– |
| 1987 | 229 | 36 | –/–/– | 7552 | 2 | –/–/– |
| 1988 | 204 | 31 | –/–/3 | 8446 | 7 | –/–/– |
| 1989 | n.a.† | 3 | –/–/– | 8746 | 0 | –/–/– |
| 1990 | | | | 8764 | 0 | –/–/– |
| Total | 5868 | 513 | 11/13/20 | 46095 | 28 | 15/–/6 |

* Numbers not available.
† Project was ended in 1989.

## DISCUSSION

After the introduction of vaccination in 1957, the reported incidence of poliomyelitis and the prevalence of PV in the Netherlands declined drastically. All recent epidemics in the Netherlands have affected non-vaccinated patients. Vaccination coverage exceeds 97% of the younger population. We have shown that antibody prevalence in the vaccinated generations is very high. Vaccine-induced antibodies apparently persist for decades; the younger generations born after 1950 are hardly exposed to circulating wild PV. Similar observations have been made in Sweden [17]. Vaccinees appear therefore well protected against poliomyelitis for a very long period of time after vaccination, presumably lifelong. Our studies indicate that most inhabitants born before 1950 are protected against PV1, and to a lesser extent against PV2 and PV3. Even in persons with antibody titres below 8, a secondary immune reaction after vaccination was found in over 80% of such seemingly unprotected individuals, indicating immunologic memory. This will most likely protect them against disease upon challenge with wild virus.

Between the epidemics in 1978 and 1992, only three non-vaccinated patients with poliomyelitis were notified, and they acquired the disease abroad. We have shown the presence of wild PV since 1979 in persons with a history of travelling abroad or their (household) contacts. Such asymptomatic carriers can shed PV for weeks after infection and are considered as the prime source for an epidemic. Evidence of indigenous virus transmission was not found, and thus any PV occurring in the Netherlands probably is imported by travellers from countries where PV is still endemic. In Holland, travellers to PV-endemic regions are advised to be (re)vaccinated with IPV or DT–IPV, to reinforce systemic and gut immunity and thus to reduce the chance of importation.

The presence of PV in river water is no necessarily a sign of indigenous transmission, nor a threat to public health. The vaccine-like viruses presumably originated from neighbouring countries (Germany, Belgium), where live polio vaccine is given in the immunization programs. It is unclear where the wild viruses came from; they could have been imported into the neighbouring countries, and through the sewage systems finally have ended up in the rivers passing through our country.

Most of the imported PV will not cause outbreaks because of the high level of population immunity. In the epidemics of 1978 and 1992, the exact source of virus introduction was not known, but we assumed that a direct importation into the susceptible groups with very coherent social structures had lead to these confined epidemics, in which the virus was transmitted through narrow but international streams. Virus typing results suggested that in 1978 a relation existed between the Dutch epidemic and an epidemic in North America and Canada, and a virus isolate in Turkey in 1977 [9]. Again, in 1993 virus strains were also isolated in (family) contacts in North America and Canada (10). In these epidemics, with one single exception, no cases were found among the large numbers of persons that did not belong to the above mentioned communities and were not vaccinated for similar reasons, even when they lived in the same area but within another social environment. Either these persons were adequately protected by herd immunity, or they had not been exposed as a consequence of their segregated way of life.

Herd immunity was observed in IPV-vaccinated communities in the United States in 1959 [18]. However, it is known that IPV-recipients can excrete PV after challenge with wild or vaccine virus [19, 20], and may contribute to faecal spread. Mucosal immunity to PV shortly after vaccination with live oral PV vaccine (OPV) appears stronger than after vaccination with IPV [20]. Some studies have indicated that the extent of intestinal virus carriage (virus titres and duration of excretion) is lower in persons with high pre-challenge titres of serum neutralizing antibodies [18, 21–230, but others could not confirm this [19].

We are convinced that polio epidemics in our country are due to failure to vaccinate, regardless of the type of vaccine used. Epidemics appear to occur in countries using OPV, not only in non-immunized pockets [10, 24], but also in the general population [25–27]. In these countries the average vaccination rates in susceptible groups were lower than in the Netherlands (70–90%), but in our country the rates among the orthodox reformed communities were very low. Herd immunity to PV is not solely dependent on the type of poliovaccine used, but on the over-all immunity level, socio-economic and hygienic conditions as well. It is questionable whether the additional use of OPV contributes further to herd immunity in the general population and will prevent epidemics in non-vaccinated pockets.

In 1988 the World Health Assembly adopted a resolution for the global eradication of poliomyelitis, and a plan of action for eradication of poliomyelitis by the year 2000 was formulated [28]. So far the eradication programme appears successful, bearing completely upon OPV, being cheap and easy to administer during routine and mass vaccination campaigns [29]. In some countries however, there may be a role for IPV (alone or additional to OPV), based upon epidemiological and practical arguments [30, 31]. The global programme will

reduce the circulation of wild PV and thereby the chance of importations and the threat to non-vaccinated susceptible groups.

ACKNOWLEDGEMENT

The authors express their gratitude to Albert Ras, Dick Jut, Bert Weitering, Arie Franke en Piet Poelstra for their virus typing and titrations of neutralizing antibodies.

REFERENCES

1. Bijkerk H. Surveillance and control of poliomyelitis in the Netherlands. Rev Infect Dis 1984; **6**: S451–6.
2. Schaap GJP, Bijkerk H, Coutinho RA, Kapsenberg JG, Van Wezel AL. The spread of wild poliovirus in the well-vaccinated Netherlands in connection with the 1978 epidemic. Prog Med Virol 1984; **29**: 124–40.
3. CDC. Poliomyelitis – Netherlands, 1992. MMWR 1992; **41**: 775–8.
4. Oostvogel PM, Van Wijngaarden JK, Van der Avoort HGAM, et al. Poliomyelitis outbreak in an unvaccinated community in the Netherlands, 1992–93. Lancet 1994; **344**: 665–70.
5. Verbrugge HP. The national immunization program of the Netherlands. Pediatrics 1990; **86**: S1060–3.
6. Hofman B. Poliomyelitis in the Netherlands 1958–69: the influence of a vaccination programme with inactivated poliovaccine. Bull WHO 1972; **46**: 735–45.
7. Cohen H, Hofman B, Brouwer R, Van Wezel Al, Hannik ChA. Combined inactivated vaccines. Series Immunobiol Standard 1973; **22**: 133–41.
8. Van Wezel AL, Van Steenis G, Hannik ChA, Cohen H. New approach to the production of concentrated and purified inactivated polio and rabies tissues culture vaccines. Develop Biol Standard 1978; **41**: 159–68.
9. Furesz J, Armstrong RE, Contreras G. Viral and epidemiological links between poliomyelitis outbreaks in unprotected communities in Canada and the Netherlands. Lancet 1978; **ii**: 1248.
10. CDC. Isolation of wild type poliovirus type 3 among members of a religious community objecting to vaccination. Alberta, Canada, 1993. MMWR 1993; **42**: 337–9.
11. Oostvogel PM, Van der Avoort HGAM, Mulders MN, Rümke HC, Van Steenis G, Van Loon AM. Virologische en serologische aspecten van de Nederlandse polio-epidemie in 1992. Ned Tijdschr Geneeskd 1993; **137**: 1404–6.
12. Conijn-van Spaendonck MAE, Hannik ChA, Bijkerk H, et al. Immuunstatus in 1980 van de Nederlandse bevolking vanaf 10 jaar tegen poliomyelitis, rubella en morbilli; een serologisch onderzoek. Ned Tijdschr Geneeskd 1984; **128**: 1884–7.
13. Van der Veer M, Van Noorle Jansen LM, Nagel J, Van Steenis G, Plantinga AD, Rümke HC. Antistofpatronen in een doorsnede van de Nederlandse bevolking. Onderzoek peilstations in 1980 en 1985. January 1993. RIVM report 927901007.
14. Welp EAE, Van Steenbergen JE, Rümke HC, Van Steenis G. Beschermingsgraad tegen poliomyelitis van Nederlanders geboren voor 1945 en van allochtonen in de stad Utrecht, en het verband met de sociaal-economische status. Ned Tijdschr Geneeskd 1993; **137**: 1388–91.
15. Van Loon AM, Ras A, Poelstra P, Mulders M, Van der Avoort H. Intratypic differentiation of polioviruses. In: Measles and poliomyelitis, Kurstak E, ed. Vienna: Springer Verlag, 1993: 359–69.
16. Van Olphen M, Kapsenberg JG, Van de Baan E, Kroon WA. Removal of enteric viruses from surface water at eight waterworks in the Netherlands. Appl Environ Microbiol 1984; **47**: 927–32.
17. Böttiger M. Polio immunity to killed vaccine: an 18-year follow-up. Vaccine 1990; **8**: 443–5.
18. Marine WM, Chin TDY, Gravelle CR. Limitations of fecal and pharyngeal poliovirus excretion in Salk-vaccinated children. A family study during a type 1 poliomyelitis epidemic. Am J Hyg 1962; **76**: 173–95.

19. Benyesh-Melnick M, Melnick JL, Rawls WE, et al. Studies of the immunogenicity, communicability and genetic stability of oral poliovaccine administered during the winter. Am J Epidemiol 1967; **86**: 112–36.
20. Onorato IM, Modlin JF, McBean AM, Thoms ML, Losonsky GA, Bernier RH. Mucosal immunity induced by enhanced-potency inactivated and oral polio vaccines. J Infect Dis 1991; **163**: 1–6.
21. Glezen WP, Lamb GA, Belden EA, Chin TDY. Quantitative relationship of preexisting homotypic antibodies to the excretion of attenuated poliovirus type 1. Am J Epidemiol 1966; **83**: 224–37.
22. Howe H. The quantitation of poliomyelitis virus in the human alimentary tract with reference to coexisting levels of homologous serum neutralizing antibody. Am J Hyg 1962: **75**: 1–17.
23. Dick GWA, Danes DS, McAlister J, Briggs M, Nelson R, Field CMB. Vaccination against poliomyelitis with live virus vaccines. 7. Effect of previous Salk vaccination on virus excretion. BMJ 1961; **2**: 266–9.
24. Expanded Programme on Immunization. Poliomyelitis outbreak-1992. Malaysia. Wkly Epidemiol Rec 1993; **68**: 297–300.
25. Kim-Farley RJ, Rutherford G, Lichfield P, et al. Outbreak of paralytic poliomyelitis. Taiwan. Lancet 1984; **ii**: 1322–4.
26. Deming M, Jaiteh KO, Otten MW, et al. Epidemic poliomyelitis in The Gambia following the control of poliomyelitis as an endemic disease. Part II: The clinical efficacy of trivalent oral polio vaccine. Amer J Epidemiol 1992; **135**: 393–408.
27. Sutter RW, Patriarca PA, Brogan S, et al. Outbreak of paralytic poliomyelitis in Oman: evidence for widespread transmission among fully vaccinated children. Lancet 1991; **338**: 715–20.
28. Wright PF, Kim-Farley RJ, De Quadros CA, et al. Strategies for the global eradication of poliomyelitis by the year 2000. N Engl J Med 1991; **325**: 1774–9.
29. Hull HF, Ward NA, Hull BP, Milstien JB, de Quadros C. Paralytic poliomyelitis: seasoned strategies, disappearing disease. Lancet 1994; **343**: 1331–7.
30. Moriniere BJ, Van Loon FPL, Rhodes PH, et al. Immunogenicity of a supplemental dose of oral versus inactivated poliovirus vaccine. Lancet 1993; **341**: 1545–50.
31. Patriarca PA, Foege WH, Swartz TA. Progress in polio eradication. Lancet 1993; **342**: 1461–4.