Water, Environmental Surveillance and Molecular Epidemiology of Poliovirus in India

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Abstract

Poliomyelitis has appeared in epidemic form, become endemic on a global scale and has been reduced to near elimination, all within the span of documented medical history. Environmental surveillance of poliovirus (PV) means monitoring of PV transmission in human populations by examining environmental specimens supposedly contaminated by human faeces. The rationale for surveillance is based on the fact that PV-infected individuals, whether presenting with disease symptoms or not, shed large amounts of PV in the faeces for several weeks. As the morbidity: infection ratio of PV infection is very low, this fact contributes to the sensitivity of PV surveillance, which under optimal conditions can be better than that of the standard acute flaccid paralysis (AFP) surveillance. The World Health Organization (WHO) has included environmental surveillance of PV in the new Strategic Plan of the Global Polio Eradication Initiative for years 2010–2012 to be increasingly used in PV surveillance, supplementing AFP surveillance.

Keywords

Epidemiology · Polio · Polio vaccine virus · Public health · Surveillance

Introduction

Clean water is essential for life. Throughout the world, millions of people do not have access to microbiologically safe water for drinking, cooking and other essential purposes. It is estimated that one-sixth of the world's population do not have access to improved sources of drinking water (WHO 2010). Enteric viruses are the important causative agents of human diseases that transit easily to water environments due to varied human activity. They are usually present in insufficiently treated drinking water, groundwater, rivers and seas. Impurities from human households are a main source of water contamination. Enteroviruses may cause a wide variety of pathological symptoms and enteroviral infections

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that affect especially young children. Enteroviral epidemics are predominantly waterborne; therefore, water contamination poses an absolute threat to human health (Nwachuku and Gerba 2006).

Success of the Global Polio Eradication Initiative

A multidecade effort of Global Polio Eradication Initiative (GPEI) launched by WHO in 1988 has reduced the global polio incidence by >99 % and intensified its efforts to eradicate the circulation of wild poliovirus (WPV) in the four endemic countries by the end of 2012. India, which was among the world's four endemic countries besides being Pakistan, Afghanistan and Nigeria, responsible for the transmission of WPV, has experienced a year without reporting a single case for the first time. The last case of wild poliovirus type 1 (WPV1) was reported in the state of West Bengal on 13 January 2011. The states of Uttar Pradesh and Bihar in northern region have been the focus against polio because of the high population density and poor sanitation and have complicated the efforts to break the transmission cycle.

With 741 cases of polio reported in the year 2009 to one in 2011, India has seen a massive drop in the number of polio cases. Since November 2009, no cases of WPV1 have been reported in the states of Uttar Pradesh and Bihar, both of which were polio endemic and no cases of WPV3 have been reported since April 2010. But seven cases of vaccine-derived polio were seen in India in 2011. By contrast, Pakistan and Afghanistan have reported three and nine cases, so far in 2011, respectively, and Nigeria still has all the three PV strains in circulation.

It is because of the massive efforts of Indian health authorities that have made possible India free of polio for 1 year. In 2011, more than 900 million doses of oral PV were administered to Indian children and more than 172 million children were vaccinated twice on national immunization days (NIDs) every year. About 120 million children were vaccinated during an additional seven sub-national immunization days.

The WHO's executive board declared the completion of its polio eradication efforts a programmatic emergency for public health and urged the handful of countries where polio still exists to declare a national public health emergency and also called for certification-standard surveillances to identify the emergence of circulating vaccine-derived PVs, and adequate funding to interrupt wild PV transmission globally, which they believe can be achieved by the end of 2013 (Kew 2012; Kaura and Abraham 2012).

Primary Strategies for Achieving this Goal

1. **High Routine Immunization**: To immunize every child aged <1 year with at least 3 doses of oral poliovirus vaccine (OPV). Paralytic polio can be caused by any of 3 closely related strains (serotypes) of PV. Trivalent OPV (OPV3) provides immunity against all 3 types. Three routine OPV doses should be received by infants at ages 6, 10 and 14 weeks.
2. **National Immunization Days**: Conduct Pulse Polio Immunization (PPI) programme by providing additional OPV doses to every child aged <5 years at intervals of 4–6 weeks. The aim of NIDs/PPI is to "flood" the community with OPV within a very short period of time, thereby interrupting transmission of virus throughout the community. Intensification of the PPI programme is accomplished by the addition of extra-immunization rounds, adding a house-to-house "search and vaccinate" component in addition to providing vaccine at a fixed post.
3. **Surveillance of AFP**: To identify all reservoirs of wild PV transmission. This includes AFP case investigation and laboratory investigation of stool specimens collected from AFP cases, which are tested for PVs in specialized laboratories.

4. **"Mopping-up" Immunization**: When poliovirus transmission has been reduced to well-defined and focal geographical areas, intensive house-to-house, child-to-child immunization campaigns are conducted over a period of days to break the final chains of virus transmission.

Cross-Border Polio Spread: A Threat to India

India has been polio free for over a year. But the big danger now is a cross-border threat. In 2011, there were 198 cases in Pakistan and 80 cases in Afghanistan. Given the porous border, this increases India's chances of a cross-border polio spread. Last year, China reported a polio outbreak after a gap of 10 years and the virus had spread from Pakistan. Therefore, if a polio-free country becomes re-infected, the virus can spread like wildfire. This is precisely why the WHO has declared polio eradication in the Pakistan region as an "emergency". Hence, the government had made a policy that every Pakistani traveller, irrespective of age, or of vaccination status, must take a dose of OPV before travelling to India. Accordingly, India recently had set up vaccination booths at Chakdabagh (Poonch) and at Kaman (Baramulla) of Jammu and Kashmir, at Munabao railway station in Rajasthan's Barmer district and at Wagah border and Attari railway station in Punjab to administer polio drops to all children below 5 years, coming in from Pakistan. This was done to control the biggest threat of import of the virus.

Poliomyelitis: The Disease

Poliomyelitis, or polio, is a life-threatening acute paralytic disease caused by PV, a member of the genus *Enterovirus* in the family Picornaviridae (Hovi et al. 2004). PVs are transmitted from person to person following excretion in faeces and pharyngeal secretions, mainly via the hand-to-hand-to-mouth route. Because the PV receptor is only expressed on cells of humans and a few sub-human primate species, there are no known extra-human reservoirs (Racaniello 2006). Following infection, the virus replicates in the gastrointestinal tract and may cause viremia (Sabin 1956). Occasionally, the virus then invades the central nervous system and destroys lower motor neurons, causing a clinically distinctive flaccid paralysis without permanent sensory loss (Nathanson 2008). Like other RNA viruses, PVs exist as mixtures of microvariants, called quasi-species (Mulders et al. 1999). This is caused by the error-prone, virus-encoded RNA polymerase, which lacks proof-reading activity, resulting in a rapid accumulation of mutations upon replication (Mulders et al. 1999; Hovi et al. 2004). An additional mode of generating divergence between PVs and other enteroviruses (EVs) is their ability to recombine with other serotypes (intertypic recombinants) or with another genome of the same serotype (intratypic recombinants) (Mulders et al. 1999; Hovi et al. 2004). During replication in humans and upon transmission between hosts, some of the mutations are enriched, which has resulted in numerous genetic lineages within each serotype of PV that cocirculate worldwide (Mulders et al. 1999).

To date, there are three PV serotypes, designated type 1, type 2 and type 3, which were originally distinguished from the other EVs by neutralization with serotype-specific antisera and the propensity to cause paralytic illness (Georgopoulou et al. 2000) (Fig. 2.1).

Epidemiology

The disease of poliomyelitis has a long history. The first example may even have been more than 3,000 years ago. An Egyptian stele dating from the 18th Egyptian dynasty (1580–1350 BCE) shows a priest with a deformity of his leg characteristic of the flaccid paralysis typical of poliomyelitis. The first known clinical description of poliomyelitis is attributed to Michael Underwood, a British physician, who in 1789 reported observing an illness which appeared to

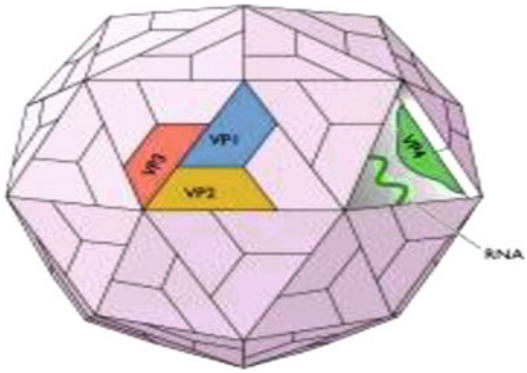

Fig. 2.1 Poliovirus serotypes. Three immunologically distinct types of PV, PV1, PV2 and PV3 have been recognized. Strains which cause severe paralysis are known as wild poliovirus (PV1 wild, PV2 wild and PV3 wild). Sabin PV1, PV2 and PV3 strains are used as efficient vaccine known as vaccine-derived poliovirus. Adapted from http://www.wenliang.myweb.uga.edu

target primarily children and left those afflicted with residual debility of the lower extremities. Initial outbreaks in Europe were documented in the early nineteenth century, and outbreaks in the United States were first reported in 1843. However, it was not until the early twentieth century that the number of paralytic poliomyelitis cases reached epidemic proportions (De Jesus 2007). The polio is in circulation in India since ages, especially in Uttar Pradesh and Bihar because of poor sanitation and high population density. If one looks at the picture of different villages of Uttar Pradesh and Bihar, old paralytic cases of AFP can easily be found. This indicates that the poliovirus has survived and circulated in these areas for years. This circulating virus has made victim of polio cases time to time, depending on the immunity in general population.

In May 1988, during the World Health Assembly, Minister of Health of all member states of the WHO voted to launch global goal to eradicate polio. As a result of this, GPEI started and estimated that global incidence of polio decreased by more than 99 % with three WHO regions (Americas, Western Pacific and Europe) being certified polio-free (Sutter et al. 2001). Intensive polio eradication programme in the South-East Asia Region (SEAR), with the use of tOPV, led to the substantial decrease in the number of polio cases. By 2001, PV circulation in India was limited primarily to northern states of Uttar Pradesh and Bihar, with 268 cases reported nationwide (Mukherji et al. 2005). However, a major resurgence occurred in 2002 with 1,600 cases nationwide (Mukherji et al. 2005; Sathyamala et al. 2005), of which majority of cases, that is, 1,363 (85 %) were from Uttar Pradesh and Bihar only (Sathyamala et al. 2005). This resurgence was attributed to the decline in OPV coverage in critical areas with vaccination coverage of children in 15 % houses in some districts (Mukherji et al. 2005). Thus, a large number of children were missed in areas with high population density, resulting in a very large birth cohort of susceptible individuals in areas of poor sanitation.

Molecular Epidemiology of Wild Poliovirus Circulation in India

The role of molecular surveillance in eradication initiatives of poliomyelitis has proved to be an extremely powerful tool for assessing the transmission pathways, monitoring quality of the national immunization programme, assessing vaccine coverage and monitoring the success of eradication strategies. The poliovirus has three serotypes PV1, PV2 and PV3, and sufficient genetic clusters (John et al. 2011) have been observed in each serotype. The genotype and cluster, and lineage and sub-lineage are the operational taxonomic units for molecular surveillance. For all practical purposes, VP1-906 nt has been sequenced and compared (Martin 2011). It is essential to know the indigenous baseline genotype/cluster/lineage circulating in different states of India and to monitor the changes with accelerated efforts for elimination of these lineages. Currently, the poliovirus wild-type 1 has three clusters with multiple lineages circulating in different parts of India, while wild-type 3 has four clusters with few lineages. P2 wild poliovirus has already been eradicated from India (Barrett 2009). Surveillance for poliomyelitis is a dynamic process, and continuous

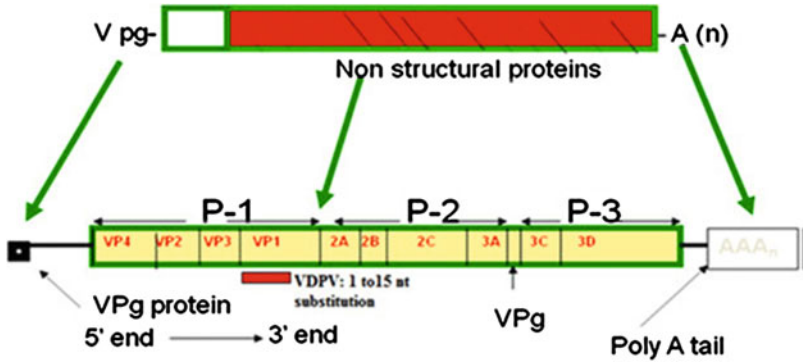

Fig. 2.2 Structure of poliovirus. Poliovirus genome consists of a single molecule of single-stranded RNA, *7,500 nucleotides long. It acts like mRNA in infected cell and is translated into a single large polypeptide. This polypeptide is cleaved by virus-coded enzymes into capsid proteins (VP1, VP2, VP3 and VP4) and non-structural proteins which include proteases and RNA-dependent RNA polymerase

monitoring of appearance and disappearance of different lineages in any given region is imperative. The most effective and useful strategy of strain surveillance is crossing over from endemic to relatively non-endemic area. Depending on the effective and optimal immunization coverage, circulation of indigenous lineage may persist or disappear. The close similarity of sequences within each lineage indicates good quality of surveillance. Some of the wild PV strains may disappear without surfacing for more than 2 years (silent transmission) or may appear as importation from other countries. It is a barometer of current status of polio eradication which helps in refining the strategies to achieve the goal in shortest possible time (Fig. 2.2).

Poliovirus Vaccines

Protective immunity against poliomyelitis is conferred through immunization or natural PV infection. Immunity is PV serotype specific (Ghendon and Robertson 1994). Protection against infection is associated with both circulating antibodies in the blood and secretory antibodies in the gut and upper respiratory tract, which prevent the spread of PV to the CNS (Ghendon and Robertson 1994; Wood et al. 2000; CDC 2002). PV is the only EV for which a vaccine is available (Zaoutis and Klein 1998). In 1955, the first successful vaccine against poliomyelitis was developed by Jonas Salk, known as inactivated poliovirus vaccine or IPV (CDC 2002). In 1963, Albert Sabin developed an oral, live attenuated poliovirus vaccine (OPV) that contained all three PV serotypes (Wood et al. 2000; CDC 2002). After its introduction, the OPV was adopted rapidly worldwide as the vaccine of choice (CDC 2002). Virologists were of the opinion that Salk's vaccine (IPV) could not provide long-lasting protection and that this could only be achieved with the Sabin's live attenuated version, which had sufficient immunogenicity to provide protection (Blume and Geesink 2000). However, none of these vaccines (IPV and OPV) can provide 100 % immunity against infection or re-infection with PV (Wood et al. 2000; Zaoutis and Klein 1998; Blume and Geesink 2000).

Inactivated Poliovirus Vaccine

The IPV is very effective in inducing circulating antibodies in the blood, thus preventing PV in the gut from entering and replicating in the CNS (Wood et al. 2000). The use of IPV in several Northern European countries (Denmark, Finland, Sweden, The Netherlands) succeeded in

effectively eliminating wild-type PV circulation (WHO 2003a, b). The IPV provides protection for many years after a complete series; however, this duration of immunity is not certain (Wood et al. 2000; CDC 2002). Although IPV is very effective in inducing circulating antibodies against PV for individual protection, it is less effective than OPV in inducing mucosal immunity at replication sites in the gastrointestinal tract (Wood et al. 2000). The IPV stimulates production of serum antibodies in the bloodstream, which cannot prevent the PV from initially multiplying in the intestine (Pelczar et al. 1993). However, these serum antibodies do prevent PV in the bloodstream from reaching the spinal cord and causing paralysis (Pelczar et al. 1993). The OPV produces long-lasting mucosal immunity by stimulating the formation of secretory IgA antibodies in the intestine and also serum antibodies in the bloodstream (Pelczar et al. 1993). Thus, mucosal immunity restricts viral replication following exposure to PV and is important for community protection (Wood et al. 2000). The intestinal secretory antibodies can prevent the primary intestinal infection by neutralizing the infectivity of virulent PV strains that a person may encounter later (Pelczar et al. 1993).

Oral Poliovirus Vaccine

The trivalent OPV (tOPV) contains live attenuated strains of all three serotypes of PV. These attenuated PV strains replicate in the human gut, inducing mucosal immunity that inhibits replication of the virus in the gastrointestinal tract (Wood and Thorley 2003; CDC 2002). A single dose of OPV produces immunity to all three PV vaccine strains in about 50 % of the recipients, and three doses of OPV will produce immunity in 95 % of the recipients (CDC 2002). The OPV has been found to confer longer-lasting immunity, so that repeated boosters are not necessary and act quickly, immunity being achieved in a matter of days (Wood and Thorley 2003; Blume and Geesink 2000).

Repeated OPV not only potentiates the immune response of the child but may produce immune paralysis. The immunogenicity of OPV varies region to region; the seroconversion rate and protection in children of Bihar and Uttar Pradesh in India are below the Indian standard (Vashishtha 2009; Paul 2007). The herd effect of OPV is also quite low in developing countries like India (Paul 2007). Vaccine viruses are less infectious than their wild counterparts, and spread to non-immune children (contact immunization) is another reason. Both of these factors are weak in children of Uttar Pradesh and Bihar. Therefore, virtually every child must be vaccinated with repeated doses of OPV to ensure personal protection, but it is difficult to achieve where primary immunization is weak. If the immunization rates fall after achieving high level of immunity in the polio-free community, risk of large outbreaks increases rapidly among the growing cohorts of non-immune children. The routine immunization with OPV will no longer outweigh the burden of diseases either due to paralysis caused by OPV (vaccine-associated paralytic polio), or due to outbreak caused by circulating vaccine-derived poliomyelitis (cVDPV) (Heymann et al. 2006a, b). The mucosal immunity induced by OPV in India varies by location, serotype and vaccine formulation (Grassly et al. 2009). The present discrepancies in tOPV versus IPV are debatable based on scientific merit and demerit, and advantage versus disadvantage of their use in two highly populated states of the country (Blume 2005). The tOPV/monovalent OPV (mOPV) has reduced the circulation of wild PV from major part of the country without making significant impact on their circulation in 107 sub-districts of Uttar Pradesh and Bihar. Therefore, the success of tOPV cannot be ignored, but its small amount inherent problem of the vaccine needs to be addressed. It is difficult to maintain the high level of immunity in the community with tOPV because of seroconversion, GI immunity and sustaining the high level of antibody for protection. The polio eradication stands for eradication of wild as well as vaccine strain from the community. Therefore, prolonged use

of tOPV will invite innumerable problems like frequent importation of wild strains, occurrence of circulating cVDPV's and vaccine-associated paralytic poliomyelitis (VAPP) and circulation of Sabin strains in the community (Modlin 2010; Heymann et al. 2006a, b; Estivariz et al. 2011).

The frequent importation has been observed from polio endemic countries to relative non-endemic countries as well as interstate within India. In India, frequent importation has been observed to neighbouring areas like Nepal to Bihar, Bihar to Bangladesh, Uttar Pradesh to Mumbai and other nearby states (Andrus et al. 2001). The importation of virus has been observed mainly in those countries from where the OPV vaccine coverage is low in general population after eradication.

The effective use of the OPV by many countries involved in the global PEI has nearly achieved elimination of wild-type PV circulation. However, maintenance of high immunization coverage is crucial to protect against imported wild-type PVs and to prevent person-to-person transmission of OPV-derived viruses (Buttinelli et al. 2003). It is important that all countries maintain a high-quality AFP surveillance system and that a global strategy is developed for the cessation of OPV immunization after global certification of polio eradication (Buttinelli et al. 2003) (Fig. 2.3).

Vaccine-Derived Polioviruses

A variety of OPV-derived viruses can be isolated from OPV recipients and their contacts (WHO 2004). The extent of sequence divergence of the VP1 capsid gene from Sabin PV strains can be used as a "molecular clock" to estimate the duration of PV replication (WHO 2004). A constant rate of accumulation of synonymous nucleotide substitutions is assumed to exist, and for the PV, genome rates of approximately 1–2 % change per year have been proposed (Kew et al. 1998). All clinical and environmental PV isolates that are related to OPV strains are VDPVs (WHO 2004). Derivatives of Sabin OPV strains have been classified into two broad categories for programmatic reasons: "OPV-like viruses" and "vaccine-derived polioviruses (VDPVs)" (WHO 2004).

The vast majorities of vaccine-related isolates are "OPV-like" and have close sequence relationships (>99 % VP1 sequence identity) to the original OPV strains (WHO 2004). Immunologically normal OPV recipients are known to excrete PVs for an average of 3–4 weeks. Short excretion periods and high population immunity normally limit the person-to-person spread of these OPV-like viruses (WHO 2004). Rare "VDPV" isolates show <99 % VP1 sequence identity to the parental Sabin PV vaccine strains,

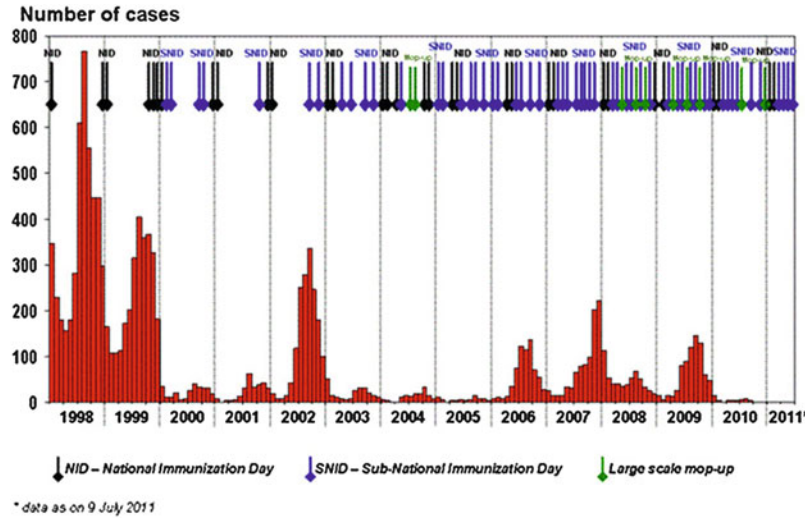

Fig. 2.3 Monthly incidence of wild poliovirus cases in India with national, sub-national and large-scale immunization. Adapted from National Polio Surveillance Project (NPSP), India

and the extent of their genetic changes indicates prolonged replication. Up to date, two categories of VDPV isolates have been identified: immunodeficient VDPVs (iVDPVs) and circulating VDPVs (cVDPVs) (WHO 2004).

Immunodeficient Vaccine-Derived Polioviruses

The potential for prolonged replication of PV vaccine stains in patients with B-cell immunodeficiencies has been recognized for many years (WHO 2004). The first iVDPV isolates to be characterized with modern molecular techniques were from patients with defects in antibody production (generally CVID or X-linked agammaglobulinemia) (Kew et al. 1998; Sutter and Prevots 1994; Yang et al. 2003). Some iVDPV isolates are highly divergent (−90 % VP1 sequence identity to the parental Sabin PV vaccine strain), suggesting that the chronic PV infections had persisted for 10 years or more (Kew et al. 1998; Sutter and Prevots 1994; Yang et al. 2003). Eighteen chronic iVDPV excretors were detected worldwide through the end of 2002, although this number may be an underestimate in the absence of systematic screening of immunodeficient patients (WHO 2004). So far, all reports of persistent iVDPV infections have been from countries with high or intermediate levels of development, where the rates of OPV coverage are high and where the survival times of immunodeficient patients may be extended by their access to appropriate clinical management (Yang et al. 2003). Currently, there is no clear evidence of spread of iVDPV from immunodeficient patients to the wider community (Yang et al. 2003; WHO 2004) (Fig. 2.4).

Circulating Vaccine-Derived Polioviruses

In regions of low OPV coverage, a VDPV may result from transmission of Sabin PV vaccine strains from one immunized individual to another and accumulation of sufficient mutations to adopt wild-type PV characteristics of neurovirulence and transmissibility (Wood and Thorley 2003). A VDPV may cause an outbreak of poliomyelitis, and if there is evidence of person-to-person transmission, based on epidemiological and phylogenetic studies, it is defined as a circulating VDPV (cVDPV) (Yang et al. 2003; Wood and Thorley 2003). According to scientific reports, any PV that is circulating will eventually recombine with another related EV and that recombination is an indicator of circulation rather than a step in the increased ability to transmit from person to person (WHO 2004). All cVDPVs but none of the iVDPVs described in scientific reports thus far appear to be recombinants with EVs closely related to PVs (Yang et al. 2003; Kew et al. 2004). The possible role of recombination in the phenotypic reversion of OPV is unclear. Recombination with EVs appears to be an indicator of circulation, as the cVDPVs in Hispaniola and Egypt had participated in successive rounds of recombination during the outbreaks (Yang et al. 2003; Kew et al. 2004).

Several outbreaks of poliomyelitis due to cVDPV have been documented (Wood and Thorley 2003). A type 2 vaccine-related PV circulated in Belarus following local cessation of OPV use from 1963 to 1966 (Kew et al. 2004). *An* outbreak of type 3 poliomyelitis in Poland in 1968 was associated with PV strains derived from the USOL-D-bac vaccine (Martin et al. 2000). In Egypt between 1983 and 1993, 32 cases of paralytic disease from a cVDPV type 2 were reported, including many retrospective cases (CDC 2001). Polio cases attributed to cVDPV type 1 have been found in Haiti, the Philippines and the Dominican Republic during 2000 and 2001 (Kew et al. 2004; Wood and Thorley 2003). The small cluster of cases marked the first polio outbreak in the Western Hemisphere in more than 9 years (WHO 2000). There have been 19 reports of AFP in the Dominican Republic and one in Haiti (WHO 2000; Dove 2001). Though AFP can be caused by conditions other than polio, laboratory tests confirmed that a cVDPV type 1 was involved in recent outbreaks (Dove 2001). In Hispaniola (the

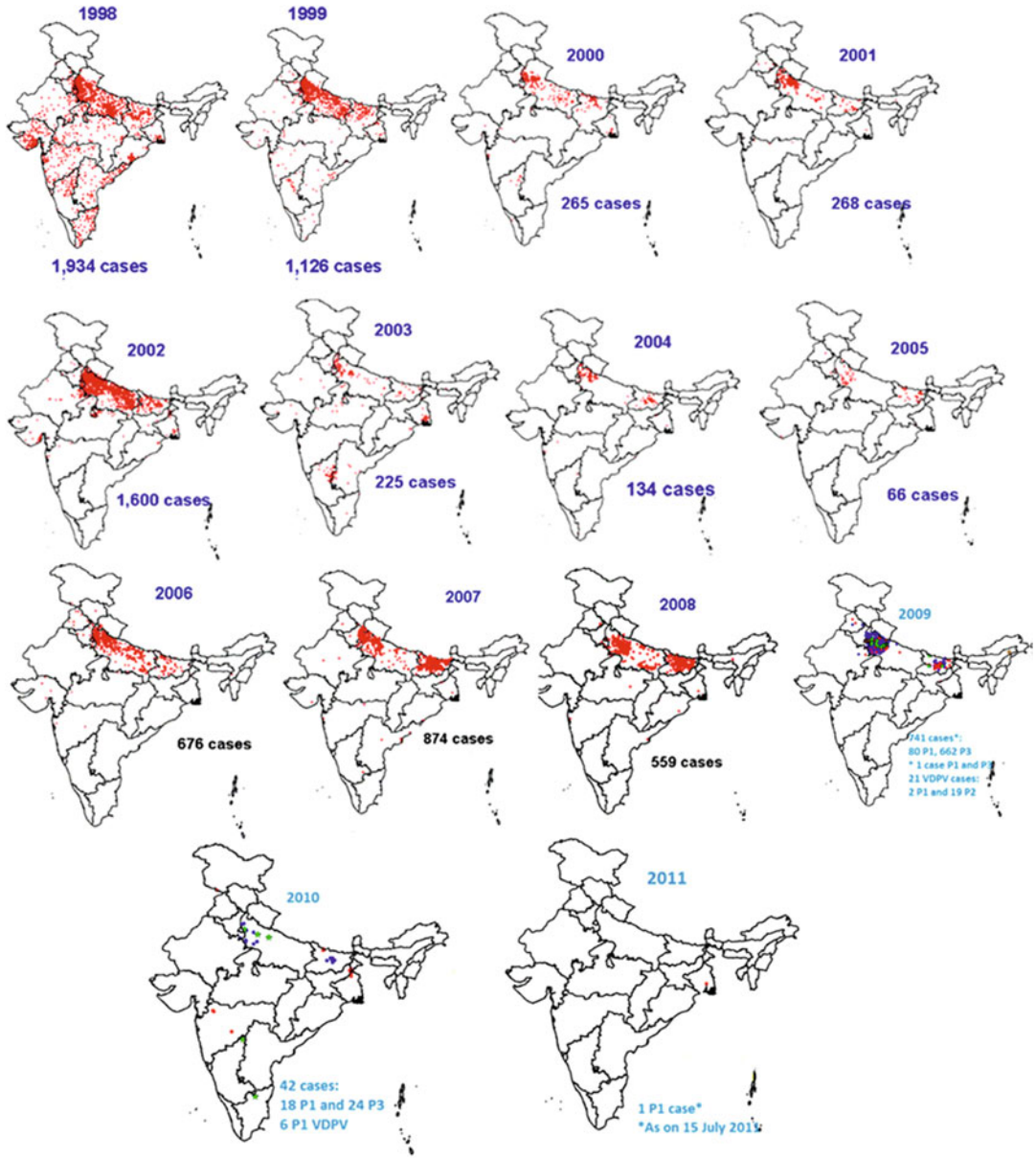

Fig. 2.4 Geographical distribution of wild poliovirus cases in India (1998–2011). Adapted from National Polio Surveillance Project (NPSP), India

Dominican Republic and Haiti) and the Philippines, the cVDPV had undergone recombination with NPEVs. This has been the first reliable report that a VDPV strain reverted to a virulent form and spread contagiously. The virus in these episodes showed more than 2 % genetic sequence difference from the parent Sabin PV vaccine strain (VP1 region of the genome) and probably circulated for more than 2 years before being detected (Kew et al. 2004). The outbreaks began when a VDPV infected inadequately vaccinated individuals, leading to the spread of the pathogenic virus (Dove 2001). Outbreaks of a similar kind have occurred more recently in the Philippines and Madagascar (WHO 2002; Rousset et al. 2003). In Madagascar, five cases

of AFP associated with cVDPV type 2 were reported and partial genomic sequencing indicated that two of the PV strains had been circulating for approximately 1 and 2.5 years, respectively (Rousset et al. 2003). Mass vaccination campaigns with OPV interrupted circulation of cVDPVs in Hispaniola and have been underway in the Philippines and Madagascar (Kew et al. 2004). A common factor to all cVDPV outbreaks has been low population immunity, consistent with low OPV coverage and the apparent absence of circulating indigenous wild-type PV of the same serotype. Other risk factors are typical for wild-type PV circulation and include crowding, high birth rates, poor hygiene and sanitation and tropical climate (Kew et al. 2004).

Environmental Surveillance of Poliovirus Circulation

Environmental surveillance has been used successfully in monitoring enteric virus circulation and assessing the extent or duration of epidemic PV circulation in specific populations (WHO 2002, 2004). The rationale for environmental surveillance is based on the characteristic PV excretion pattern. Infected individuals excrete PVs in faeces for periods up to several weeks, whether or not they are symptomatic, and therefore, large numbers of PVs may remain infectious in the environment for varying lengths of time, depending on the immediate conditions (WHO 2002, 2004). Wild-type PVs and cVDPVs have been detected in the environment even in the absence of reported cases of AFP, which is of major concern, since these PVs might be transmitted and continue to circulate in a non-immune population after the cessation of polio vaccination (Friedrich 2000). A study conducted by Divizia et al. (1999) confirmed the environmental circulation in Albania of recombinant PV strains (Sabin-like PV type 2/wild PV type 1), sustained by a massive immunization effort and by the presence in the environment of a PV type 1, isolated from a river 2 months before the first case of symptomatic AFP. An unusual highly diverged derivative of the Sabin PV type 2 strain was isolated from environmental samples during routine screening for wild-type PV in Israel (Shulman et al. 2000). The extensive genetic divergence of the isolate from its parental Sabin PV type 2 vaccine strain suggested that the virus had replicated in one or more individuals for approximately 6 years (Shulman et al. 2000). According to other studies, VDPVs (with 1.4 % nucleotide divergence from the vaccine strain) were isolated from sewage and river water in Japan within 3 months following OPV vaccination, and several of these VDPV type 1 and 3 isolates showed increased neurovirulence (Yoshida et al. 2002; Horie et al. 2002). More recently, two Sabin-like PVs were found by environmental surveillance 8 and 11 months after any OPV vaccine was used in New Zealand and showed 99.8 % as well as 99.9 % homology with Sabin PV type 2 vaccine strain in the VP1 region (WHO 2003a, b). This suggested that these PVs could have been excreted by recently vaccinated children (1 or 2 months) visiting from a country using OPV (WHO 2003a, b). Furthermore, a highly evolved VDPV type 3 strain harbouring a 13 % sequence drift from Sabin PV type 3 vaccine strain has been isolated from sewage in Estonia (Blomqvist et al. 2004). Research has shown that PV isolates in the environment are genetically and epidemiologically related to those circulating in the community (Divizia et al. 1999; Shulman et al. 2000). Thus, the properties of PV isolates from sewage and river water would reflect those of PVs excreted from humans after OPV immunization, and for susceptible individuals, VDPVs have the potential to be the causative agents of poliomyelitis (Yoshida et al. 2002). However, it is difficult to address the risk of infection from the environment, since there is little chance that individuals come into direct contact with raw sewage. In contrast, access to river water or any other water source (used by the community for domestic purposes) is easy, and therefore, susceptible individuals should be regarded as at greater risk of infection from such water sources (Yoshida et al. 2002). Nonetheless, although it is possible to eliminate

wild-type PV from the human community and environment, it will be difficult to eradicate poliomyelitis completely as long as OPV is not replaced by IPV (Yoshida et al. 2002). It is evident that environmental surveillance is still epidemiologically important, because the results of virus surveillance retrospectively reflect the properties of virus circulating in the community and it assesses the potential risk of infection from the environment as well as food (Divizia et al. 1999; Yoshida et al. 2002). The examination of composite human faecal samples through environmental surveillance links PV isolates from unknown individuals to populations served by the wastewater system (WHO 2004). In addition, environmental surveillance provides valuable information, particularly in urban populations where AFP surveillance is absent and where persistent PV circulation or reintroduction is suspected (WHO 2004).

Future Strategies

Eradication of polio is a public health programme of unprecedented magnitude, complexity and cost. Tremendous progress has been made in the global fight against poliovirus.

India has invested heavily to reach where we are today. There are innumerable risks associated with the future of polio eradication. Protecting the investment requires the successful completion of both wild and vaccine virus eradication and its sustenance. Plans to design a new vaccine policy for the future especially to systematically introduce IPV and to safely discontinue OPV after eradication of WPVs have to be made urgently. There must be a smooth gradual transition from the current pre-eradication vaccine strategies to post-eradication vaccine policy, to chalk out a clear strategy on how to deal with the issues like OPV cessation plans, global synchronization versus regional/national synchronization, duration of AFP surveillance, tackling of future outbreaks of both wild and vaccine viruses, role of IPV in controlling future outbreaks of cVDPVs and development of safe and affordable IPV.

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