Commentary

Oral Polio Vaccine Is Unsafe for the World and Should Be Replaced with Inactivated Poliovirus Vaccine Globally

T Jacob John[1], Dhanya Dharmapalan[2], Robert Steinglass[3], Norbert Hirschhorn[4]

1. Independent researcher; 2. Apollo Hospitals, Chennai, India; 3. Independent immunization advisor, USA; 4. Independent consultant, USA

The excessive and prolonged reliance on Sabin oral polio vaccine (OPV) for global polio eradication by the World Health Organisation has critically impeded the mission's success, in part due to persistent safety challenges associated with OPV. For individual children there has always been the risk of vaccine-associated paralytic polio (VAPP). For the community, the increasing risk has been genetic reversal of live vaccine viruses to neuro-virulent 'vaccine-derived polioviruses' (VDPVs) which cause polio far more frequently than VAPP and to 'circulating VDPVs' (cVDPVs) that are essentially wild polioviruses. As long as OPV is widely used, polio will not be eradicated. The way forward is to introduce the inactivated poliovirus vaccine (IPV) that is completely safe and exquisitely efficacious when given as 3-dose schedule during infancy, plus one or two boosters. Once IPV is made routine, OPV should be withdrawn country by country. Hexavalent combination vaccines including IPV are ideal, as separate injections are avoided. The sooner OPV is globally replaced with IPV, the faster global polio eradication can be achieved.

Corresponding author: T Jacob John, tjacobjohn@yahoo.co.in

Introduction

In the USA, 3 doses of oral polio vaccine (OPV, Sabin) had near-100% vaccine efficacy (VE). Dr. Albert Sabin taught that the vaccine poliovirus would spread among children as efficiently as wild poliovirus (WPV) and immunize even unvaccinated children, enhancing community level 'vaccine effectiveness'[1][2][3]. Since vaccine poliovirus was directly derived from WPV, shown to be not neuro-virulent in monkey

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spinal cord injection, and was infectious by oral inoculation, Sabin was justified to expect that attenuation was confined to neuro-virulence, without affecting transmission efficiency[4].

Soon after the OPV was rolled out in the USA, post-marketing surveillance showed a serious adverse event following immunization (sAEFI), viz., polio in vaccinated children, and in unvaccinated children in contact with vaccinated children[5]. They were classified as 'vaccine-associated paralytic polio' (VAPP) and 'contact VAPP', respectively[6]. Clearly, residual neuro-virulence had survived in vaccine poliovirus.

Contact VAPP proved that vaccine poliovirus was transmissible between children and also that transmitted virus can either immunize or cause polio. Direct evidence of vaccine poliovirus transmission within families of vaccinated children was also available[4]. The rarity of contact VAPP proved that vaccine poliovirus transmission in the community was thankfully infrequent. Contrary to Sabin's expectation, any increase in vaccine effectiveness was minimal. An unvaccinated 'third party' getting sAEFI was unprecedented in vaccinology and it has no specific term in medical literature.

Despite the sAEFI, OPV was chosen for the national immunization programme in the USA in 1966, replacing the Salk injectable inactivated poliovirus vaccine (IPV) that was already in use and had very high VE for 3 doses without sAEFI[7][8]. Apparently, the Government accepted the risks of OPV at a time when the incidence of polio was at its lowest level[8]. Most probably the expectation was that WPV would very soon be eliminated due to the putative enhancement of vaccine effectiveness. That did not happen. Eventually WPV stopped circulating in the USA only in 1979, taking 13 more years[9]. The 'experiment' had failed; vaccine poliovirus did not spread passively to a meaningful extent and in the process ~100 children had developed VAPP[10].

A decision to reverse the vaccine choice would have been wise and urgent, but it was delayed until 1999 when IPV replaced OPV in the USA. From 2000, USA has remained totally polio-free under exclusive IPV use, except for one polio case in 2022 caused by an imported virulent, mutated vaccine poliovirus, called 'circulating vaccine-derived poliovirus' (cVDPV)[9]. Our call is for WHO to replicate the USA decision in all OPV-using countries by replacing OPV with IPV.

In low and middle income countries (LMICs), especially in the tropical/subtropical zone, OPV has sub-optimal and variable VE, ranging from 30% to 65% for 3 doses[11][12][13]. Consequently, many vaccinated children had, and continue to have, 'vaccine-failure' polio due to WPV in LMICs[14][15].

Low VE is due to low vaccine virus 'take' rate and sero-conversion[16]. Low take rate meant that vaccine poliovirus transmission would also be far less frequent in LMICs than in the USA, an insight apparently

missed by many. Thanks to the WHO-designed Expanded Programme on Immunization (EPI, see below), all LMICs were using the trivalent OPV (tOPV). In 1988 the global call for polio eradication by the WHO allowed 12 years to reach the finish line, apparently assuming that the USA experience would be repeated in LMICs[17]. However, WHO seems to have neither analyzed the nuances of the USA's experience nor investigated if vaccine poliovirus transmission enhanced vaccine effectiveness beyond the low VE in LMICs.

Relying on transmission for high vaccine effectiveness of OPV, as a public health tactic for rapid elimination of WPV in LMICs, was fanciful and inconsistent with experience[18][19]. In the USA even with near-100% VE, tOPV had not stopped WPV circulation rapidly.

Faced with continuing WPV circulation in LMICs, wherein 3-dose routine tOPV coverage in children had surpassed 80%, WHO implemented supplementary immunization in many LMICs without an upper limit for the number of doses, in an attempt to match the 3-dose VE in the USA[18][19]. The safety of repeated OPV doses in communities in which the ratio of immune and non-immune children was unknown was not investigated. Although we can understand GPEI's consternation at the unbudging persistence of WPV circulation despite Herculian efforts, we must point out that community-wide distribution of 20 and more doses per child was an unprecedented experimental procedure. Today we know that it was not a safe procedure for the community at large, as mass campaigns with OPV led to polio outbreaks caused by cVDPV (See below)[20][21].

Repeated mass campaigns have also raised suspicions about the motives of international actors and resistance from parents and communities, especially in Afghanistan and Pakistan – countries in which WPV type 1 continues to circulate even now, defying all attempts by GPEI to stop it. It is well known that vaccines are given in a limited and pre-determined number of doses, and in clinics, but when repeated many times, in an ad-hoc manner, we cannot expect full compliance, year after year, decade after decade.

Moreover, for the WHO, reliance on repeated mass campaigns with OPV has resulted in a second target virus for eradication, namely cVDPV, which is essentially WPV, but iatrogenic in origin. WPV had sired vaccine poliovirus in cell cultures in Sabin's laboratory, and in turn vaccine poliovirus began to sire WPV in the community under unimaginably huge numbers of replications akin to laboratory test tube replications, in the present century.

Historically, WPV-2 was eliminated in Afghanistan and Pakistan, through many rounds of OPV campaigns by or before 1999. Similarly, during 2019 to 2021, Afghanistan and Pakistan had several

hundreds of polio cases caused by cVDPV-2 that were stopped by tOPV campaigns. This proves that low coverage was not the critical weak link but very low vaccine efficacy of OPV against WPV-1 that has much higher transmission efficiency than cVDPV-2.

As long as live infectious polioviruses continue to be introduced into the community on a large scale through OPV (in routine childhood vaccination and mass campaigns), WHO will not be able to eradicate polio. The purpose of this paper is to elaborate on such problems caused by the continued use of OPV in LMICs and on the logic of our assertion that global polio eradication is contingent upon the transition from OPV to IPV in all LMICs.

Benefit-risk balance of OPV in the Expanded Programme on Immunization (EPI)

In the 1970s and 1980s, as EPI was being accepted by all LMICs, the annual incidence of polio was about 1.6 per 1000 pre-school children (birth to 4 years) or 1600/million.WPV type 1 would cause one case per 160 infections, or 6 cases in 1000 children[22][23]. WPV types 2 and 3 would cause one case each in 1000 children – for a total of 8 cases/1000 children; that is how we derived 1.6/1000 child-years/year. The risk of VAPP was one per 150,000 preschoolers given OPV, or 6.7/million[24]. During 1974 to 1988, the benefit-risk balance of OPV was highly favorable.

That scene changed drastically in the 1990s once the goal of EPI was revised in 1988, from polio control to global polio eradication, with a target year of 2000[17]. Under eradication mode, the risk of polio due to WPV would progressively decline towards zero. As that risk would reach 6.7/million, OPV's benefit-risk balance would no longer be favorable. Therefore, WHO logically had to plan the transition from OPV to IPV prior to, or at the latest by, 1999 to meet the eradication target of 2000. Eradication would not be achieved with OPV since VAPP alone would account for ~800 cases annually in the birth cohort of 120 million babies in LMICs[23].

The consequences of continuing OPV beyond 1999

The WHO continued the policy of exclusive use of tOPV into the twenty-first century, disregarding the logic stated above. No justification was ever offered. The innumerable polio outbreaks caused by cVDPV type 2, as enumerated below, are taking place in a world in which the original natural WPV type 2 was globally eradicated in 1999, according to WHO data[20].

Large-scale public health interventions that diverge from evolving scientific, logical, and ethical considerations can have far-reaching and unintended consequences. In 2000, after over seven years of the absence of WPV circulation in the Caribbean, and with the continued use of OPV, an outbreak of VAPP occurred in the Dominican Republic and adjacent Haiti in the Hispaniola Island; the outbreak continued in 2001 and was controlled with campaigns of tOPV[20]. During the twentieth century, VAPP used to be sporadic, but then it became epidemic. The vaccine virus had mutated and regained neuro-virulence and transmission efficiency, thus becoming as pathogenic as the parent WPV. The virologists who investigated the genetic mutation coined the terms 'vaccine-derived poliovirus' (VDPV) for virus which had mutated appreciably away from the gene sequence of Sabin OPV, and cVDPV for the fully feralized vaccine poliovirus that had become genotypically and phenotypically identical to WPV[20].

The quintessential lessons are that vaccine poliovirus had sired cVDPV and that cVDPV and WPV differ only in their past pathways. OPV was derived directly from WPV by altering laboratory culture conditions and not genetically engineered for irreversible gene sequence for the sake of safety. The reverse process in community circulation resulted in reversion to WPV in the form of cVDPV. WPV, vaccine poliovirus, VDPV and cVDPV are variants in one species and can move in either direction on the scale of virulence, given the right external conditions.

Apparently, WHO polio experts implementing the mission of the 'global polio eradication initiative' (GPEI) since 1988, faced a tension in 2000-2001. The policy-makers seem to have misread the Hispaniola episode as a one-off event, not as the harbinger of a new sinister iatrogenic problem for the world in the new century – consequently the exclusive use of tOPV was continued. That was unwise and reckless. The foundation for GPEI's eventual shortcomings were laid during this period.

The GPEI virologists, on the other hand, seem to have recognised the reality and introduced molecular testing of every poliovirus isolate, from children with acute flaccid paralysis and from sewage surveillance, in GPEI's global network of polio laboratories [21]. The reversion of vaccine poliovirus to VDPV and WPV (aka cVDPV) has identifiable genetic mutations, and vaccine progeny viruses in children and sewage can be classified through molecular virology analysis as vaccine poliovirus (designated Sabin-like) with <0.1% mutations in VP1 region; VDPV with 0.1 to 0.6% mutations for type 2 and up to 1% mutations for types 1 and 3; and cVDPV with >0.6% mutations for type 2 and >1% mutations for types 1 and 3[21].

Thus, since 2000, GPEI has been reporting polio due to WPV and cVDPV in various documents (as targets of eradication) but not polio due to vaccine poliovirus and VDPV, presumably as they were not considered GPEI's targets of eradication. The rationale seems to be that WPV and cVDPV circulate in the community and have to be eradicated by immunization, whereas polio due to VDPV and vaccine poliovirus that do not circulate would automatically disappear as OPV is discontinued after eradicating WPV and cVDPV.

Iatrogenic polio in the twenty-first century

Due to the foresight of GPEI virologists introducing molecular testing of poliovirus isolates, there are data on the numbers of polio cases due to Sabin-like virus (viz, VAPP), VDPVs and cVDPVs. However, GPEI does not divulge the numbers of VAPP or polio caused by VDPVs, hiding the bad news from the public. As said earlier, GPEI places in the public domain information on polio cases caused by WPVs and cVDPVs.

In 2001 three more children with polio due to cVDPV-1 were detected in the Philippines and in 2001 and 2002, five children with polio caused by cVDPV-2 were identified in Madagascar. Hispaniola, the Philippines and Madagascar, all islands/archipelago, seemed to have a high risk of evolution of vaccine poliovirus into cVDPVs, suggesting some ecological/socio-demographic influence on the evolution of cVDPVs. We believe that the time had come, now for the second time, in the third year of the new millennium, when GPEI had to urgently plan the transition from tOPV to IPV globally.

During the decade starting from 2000, the global scene became alarming, with vaccine poliovirus types 1, 2 and 3 genetically reverting to WPV-like cVDPVs in multiple countries (See Table). The emergence of cVDPV-2 signaled the re-introduction of WPV-2 of vaccine virus origin into the world -- capturing the niche vacated by the natural WPV-2. Disappointingly, WHO did not guide GPEI to withdraw OPV-2 globally, despite a strong request[25].

By 2014 the number of countries with polio outbreaks caused by cVDPV-2 swelled to 14 (See Table), signaling that potentially a 'point of no return' had been reached. Nature had given yet another warning and a way forward – use of the noninfectious IPV was unavoidable for polio eradication. Delaying its global application meant deliberately delaying polio eradication.

GPEI decided to drop type 2 from tOPV and instead use bivalent OPV (bOPV) containing only vaccine poliovirus types 1 and 3 in a globally synchronized 'tOPV to bOPV switch' in April of 2016[26]. Unfortunately, WHO did not insist on the introduction of a routine 3-dose IPV schedule in the EPI prior to

the switch. Instead, GPEI gave just one dose of IPV, assuring LMICs that this would mitigate any risk of the emergence of cVDPV-2, the basis of which was never clarified.

If for GPEI this recommendation was a 'cost-reduction' tactic, it was clearly too risky for the world -- 'penny wise, pound foolish'. Perhaps the rationale was an extrapolation from the early experience that a single dose of IPV could prevent VAPP in children subsequently given 3 doses of trivalent OPV. After all, cVDPV polio was VAPP in epidemic form. The reliance on a single dose of IPV was a strategic underestimation of community-level risk—arguably a short-term cost-saving approach with long-term global consequences. Even if the one dose prevented VAPP in vaccinated children, expecting that the community would be protected from vaccine virus transmission that leads to cVDPV emergence was unrealistically optimistic.

Had GPEI introduced a three dose-schedule of IPV into EPI at least 12 months prior to the tOPV to bOPV switch, there would have been a reasonable chance that the community risk could have been mitigated. As IPV coverage reached the then prevalent coverage rates of 3 doses of DPT vaccine or pentavalent vaccine (DPT plus Hib and Hepatitis B vaccines), in all LMICs, WHO could have transitioned from OPV to IPV globally, expediting the successful eradication of polio.

Lessons from the one crucial experiment, conducted in Byelorussia in the mid-1960s, in which tOPV was withdrawn in a well-vaccinated and polio-free population, to learn what would happen, was lost on the policy-makers[27]. Vaccine virus type 2 continued circulating in the community silently, and about one year later, began causing polio -- proving that the virus had regained both neurovirulence and transmission efficiency. If the virus was tested and defined by the current classification rule, it would have been cVDPV-2. This history was known widely, but WHO did not insist that GPEI should avoid repeating the mistake.

	Polio cases/No. of countries				
	2000-2004	**2005-2009**	**2010-2014**	**2015-2019**	**2019-2024**
cVDPV-1	26/3	51/2	3/2	26/6	386/7
cVDPV-2	5/1	370/6	308/14	382/19	2943/37
cVDPV-3	0	3/2	9/2	7/1	2/2

Table. Numbers of reported cases of polio caused by cVDPV types 1, 2 and 3, and the number of countries so affected, in quinquennial intervals from 2000 to 2024. The frequency of evolution to cVDPV was highest for vaccine poliovirus type 2, lowest for vaccine poliovirus type 3, and in between for vaccine poliovirus type 1, resulting in a ratio of 10:32:1 for types 1,2 and 3.

Basic lessons of virology and polio vaccinology

Poliovirus is a species in the genus Enterovirus, family Picornaviridae[28]. Poliovirus has three antigenic types, numbered 1 to 3. The gene sequences of vaccine poliovirus, VDPV and WPV for each type are one continuum. WPVs are the most 'fit' for infection in cell cultures and in humans and also for human-to-human transmission of infection. Vaccine polioviruses were developed in the laboratory by selecting out and propagating the least fit virus variants, and not by any genetic manipulation to prevent reversion to neuro-virulence, as has been done recently for designing the 'novel OPV'[29].

When vaccine polioviruses are passaged either in cell cultures or children-to-children, inevitably they will keep mutating to regain fitness – which includes neuro-virulence and transmission efficiency. All poliovirus isolates from any source are a mixture of mutants, mostly fit, some with very low fitness and others in between. OPV manufacturers restrict the number of cell-culture passages to the bare minimum in order to keep neuro-virulent variants to the minimum. Yet, every dose of OPV will contain a few neuro-virulent virus particles[30]. Thus, VAPP is like *Russian Roulette:* the child who gets infected first with a neuro-virulent virus is the one at risk of VAPP. We know this because VAPP occurs within one incubation period of polio – namely 1 to 4 weeks, which is far too short for reversion in vivo before infecting the spinal cord.

When an unsafe vaccine was prescribed for legitimate indication, for example the Pasteur-Semple animal brain rabies vaccine for post-exposure prophylaxis, its benefit-risk had to be determined to be favourable. When WPVs were rampant and polio outbreaks very frequent, benefit from OPV was much greater than risk, but it reversed in the twenty-first century with low prevalence of WPV. The continued use of tOPV, even after the eradication of WPV-2, suggests missed opportunities for timely policy adaptation and risk reassessment. For example, continuing type 2 vaccine poliovirus until 2016 in a world without type 2 WPV was reckless[31]. The sinister consequence was the veritable re-introduction of WPV-2, in the guise of cVDPV-2, proving that the action was indeed an error without any justification whatsoever[31].

The GPEI has repeated the same error after WPV-3 stopped circulating everywhere in 2012. Vaccine poliovirus-3 is known as the commonest cause of VAPP. How many children have developed VAPP since 2012? GPEI has not been counting, choosing instead to label VAPP as 'non-polio', contrary to science and medical ethics[32].

During 2008-2024, for every case of WPV-polio, there were about eight cVDPV-polio cases. The rationale for widely distributing vaccines against non-existent pathogens has never been stated. Indeed, WHO had been advised not to do so[25]. There appears to be a gap in critical oversight of GPEI decision-making, which merits closer examination in light of the scientific and ethical standards upheld by WHO. It is deeply concerning that despite generous philanthropic funding intended to eradicate polio, continued OPV use has inadvertently contributed to the re-emergence of vaccine-origin WPV type 2 and causing new polio outbreaks in dozens of countries including, less frequently, with types 1 and 3 (see Table)[32].

To break this vicious cycle, a globally coordinated and expedited withdrawal of OPV should be pursued, transitioning toward exclusive IPV use. The sooner OPV is replaced with IPV, the earlier we can eradicate polio globally..

Conclusion

The twin risks, sporadic and epidemic polio, make OPV clearly unsuitable for eradicating polio. The GPEI has the duty to protect children from polio and that duty is sacrosanct. All those who believe in equity, fairness and justice as essential in public health, ought to demand the global transition from OPV to IPV. Methods of doing so are not elaborated here; precedents in WHO regions and member countries indicate it can be achieved flexibly and quickly. Delays in transitioning away from OPV continue to expose children to preventable paralysis, underscoring the urgency of policy action. Not only the executive arm

of GPEI but also every one of its partners must be held accountable to fulfill GPEI's duty to stop causing paralytic polio in the name of its eradication. And WHO has a sacred duty to ensure that GPEI is held accountable to all United Nations member states, rich and poor.

Statements and Declarations

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No potential competing interests to declare.

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