Effect of Inactivated Poliovirus Vaccine Campaigns, Pakistan, 2014–2017

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Routine immunization with ≥1 dose of inactivated poliovirus vaccine (IPV) in all countries using oral poliovirus vaccine (OPV) was recommended by the World Health Organization (WHO) in November 2012, before the global withdrawal of the serotype 2 component from OPV (1). IPV has also been used since 2014 in mass campaigns to help interrupt wild poliovirus transmission and stop serotype 2 vaccine-derived poliovirus (VDPV2) outbreaks. The IPV supply was severely constrained during 2016–2017; only 2 manufacturers supply the United Nations Children’s Fund, and their failure to produce the expected bulk product has meant that only about half the awarded quantities were supplied (2). As a result of these unplanned reductions in IPV supply, countries have delayed the introduction of IPV to routine immunization or faced stockouts, and mass campaigns with IPV in response to VDPV2 are no longer recommended by WHO (3). Nonetheless, where possible, IPV continues to be used in mass campaigns for outbreak response; for example, Pakistan, Afghanistan, Nigeria, and Syria all used IPV in mass campaigns in 2017.

Given that IPV supply constraints are likely to continue until at least the end of 2018, it is crucial that available IPV be optimally allocated between routine immunization and mass campaigns. We recently published estimates of the impact of OPV mass campaigns with and without the inclusion of IPV in Nigeria and Pakistan during January 2014–April 2016 (4). These estimates demonstrated...
a reduction in the incidence of poliomyelitis and detection of poliovirus in the environment after campaigns that used IPV in Nigeria but not in Pakistan, where statistical power was limited. We have now updated these estimates in Pakistan for January 2014–October 2017, thereby including a longer period of surveillance and additional campaigns during a period when wild-type 1 poliovirus has been circulating (online Technical Appendix, https://wwwnc.cdc.gov/EID/article/24/11/18-0050-Techapp1.pdf). We find evidence of an impact of campaigns that used IPV alongside OPV (bivalent, trivalent, or monovalent) on the incidence of poliomyelitis caused by wild-type poliovirus (incidence rate ratio [IRR] for 90 days after compared with before the campaign, IRR 0.62, 90% bootstrap CI 0.23–1.14), and a significant impact on the detection of this virus in environmental samples (prevalence ratio [PR] 0.63, 90% CI 0.47–0.81) (Figure; online Technical Appendix Table). The effect of campaigns using only bivalent OPV was less than the effect of campaigns that included IPV (IRR for poliomyelitis 0.79 [90% CI 0.64–0.98] and PR for environmental detection 0.92 [90% CI 0.83–1.00] for the 90 days after compared with before the campaign); this difference was statistically significant for detection of poliovirus in the environment (bootstrap p values 0.239 comparing the IRRs and 0.005 comparing the PRs for campaigns with and without IPV). We did not update estimates for Nigeria because only 2 campaigns using IPV occurred during April 2016–October 2017, in areas with very limited VDPV2 detection.

Several caveats relate to this analysis, reflecting its observational nature, reliance on routinely collected data, and lack of randomization. Campaigns that included IPV may have been implemented with different standards and, potentially, greater coverage, although data supporting this assertion have not been presented. It is often assumed that campaigns including IPV would have lower coverage because IPV must be administered by trained healthcare staff from fixed points rather than in house-to-house campaigns (5). Furthermore, these findings may not apply to more recent serotype-2 vaccine-derived poliovirus outbreaks, which have occurred in countries without recent use of a serotype-2–containing oral vaccine, thereby limiting boosting of mucosal immunity by IPV to older cohorts.

In conclusion, these updated estimates from Pakistan provide support for including IPV in mass campaigns with OPV to reduce poliovirus transmission, in agreement with results from Nigeria. Intradermal administration of a 1/5 fractional dose may allow dose sparing during these campaigns while maintaining comparable immunogenicity (6). These findings are informing discussions about the role of IPV in stopping the last remaining chains of wild-type 1 poliovirus transmission, responding to VDPV2 outbreaks, and protecting children who have not received vaccine containing serotype 2.
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References

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Enterovirus D68 Surveillance, St. Louis, Missouri, USA, 2016

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A fall 2016 outbreak of enterovirus D68 infection in St. Louis, Missouri, USA, had less effect than a fall 2014 outbreak on hospital census, intensive care unit census, and hospitalization for a diagnosis of respiratory illness. Without ongoing surveillance and specific testing, these cases might have been missed.

The largest known outbreak of enterovirus D68 (EV-D68) occurred in the United States in 2014 (1). Severe respiratory illnesses increased in fall of 2014, corresponding to a period when EV-D68 was present in the community, at St. Louis Children’s Hospital (St. Louis, Missouri, USA) and elsewhere in the United States (1,2). Multiple reports suggested that the predominant virus was from clade B1, although some viruses from clades B2 were also detected (3–5). During 2015, there were few reports of EV-D68 circulating in the United States (6); however, in 2016, EV-D68 reappeared in multiple US locations (New York, Colorado); virus sequences suggested that the predominant virus was from clade B3 (4,7). We also documented EV-D68 activity in St. Louis in 2016. Sequencing of viruses from 2 patients tested in the St. Louis Children’s Hospital virology laboratory revealed clade B3 with 99% identity to the clade B3 virus from New York (8). Our goal with this study was to determine if the 2016 outbreak had caused an increase in hospital census or increase in patients admitted with respiratory diagnosis, as was seen during the 2014 outbreak.

During August 7, 2016, through December 16, 2016, we used a previously described EV-D68–specific PCR to test 5%–10% of enterovirus/rhinovirus–positive samples submitted each week to the St. Louis Children’s Hospital diagnostic virology laboratory. The samples had been obtained from patients seen at the hospital’s emergency department or clinics or admitted to the inpatient units and had been routinely tested by a FilmArray Respiratory Panel (BioFire, Salt Lake City, UT, USA) (9). Samples were selected by laboratory staff without regard to patient characteristics and were deidentified before EV-D68 testing. We obtained inpatient and intensive care unit (ICU) census data for all patients (not limited to those with a