ONLINE APPENDIX FOR: Duintjer Tebbens RJ, Thompson KM. Costs and benefits of including inactivated in addition to oral poliovirus vaccine in outbreak response after cessation of oral poliovirus vaccine use. MDM Policy & Practice 2017;2:1-13.

A1. Description of the differential equation-based poliovirus transmission and OPV evolution model (DEB model)

The differential equation-based poliovirus transmission and OPV evolution model (DEB model) [1] tracks the movement of people between demographic age groups (grouped into mixing age groups that mix preferentially amongst themselves), and for each serotype between oropharyngeal and intestinal infection stages (resulting in potential oropharyngeal and fecal-oral transmission, respectively), immunity states, and waning stages. Figure A1 provides an overview of the model structure based on prior work.[1] Figure A1a depicts the immunity states with the flows that move individuals in and out of them and Figure A1b details how effectively vaccinated or infected individuals progress through different stages of infection and, in the event of infection with OPV, through OPV evolution stages. The model assumes that active immunity from prior vaccination or infection results in permanent protection from polio (disease), but only partial protection from subsequent infection and participation in transmission, depending on the nature of immunity (IPV-induced vs. LPV-induced or both) and time since the last exposure (i.e. waning stage). The model includes 5 waning stages, 6 fecal-oral and 6 oropharyngeal infection stages (2 latent and 4 infectious, with varying degrees of infectiousness), and also accounts for a delay between IPV receipt and development of the immune response that moves individuals to the next IPV immunity state. In Figure A1a, we note that the model assumes identical properties for "IPV and LPV" and "≥ 2 LPV infections" and that the recent waning stages of these immunity states represent the highest degree of immunity to transmission in the model. The model further tracks OPV evolution by moving individuals infected with the OPV parent strain (stage 0) through 20 successive reversion stages that can each transmit and that come with increasing paralysis-to-infection ratios and relative basic reproduction numbers (R_0 values) compared to homotypic WPVs. The last reversion stage (stage 19) represents fully-reverted VDPVs with assumed paralysis-to-infection ratio and R_0 equivalent to homotypic WPVs. For WPVs or any OPV reversion stage, the DEB model mimics die-out by setting the force-of-infection for the given strain to 0 whenever its effective prevalence of infections resides below a calibrated threshold of 5 per million people. Consequently, OPV-related viruses can only continue to transmit and thus evolve to cVDPVs through successive infections when low enough population immunity to transmission permits circulation of the OPV viruses introduced in the population through vaccination. We fixed the die-out process, model structure, and numerical model inputs that characterize them across all populations we modeled and Table A1 includes the corresponding generic model inputs.

A2. DEB model calibration

Figure A2 summarizes the results of the model calibration process, based on prior work.[1] With the generic model inputs from Table A1 fixed, we compared our model behavior against i) data on children with non-polio acute flaccid paralysis who reported no receipt of OPV for northern India (modeled separately for Western Uttar Pradesh (WUP) and Bihar) and northwest (NW) Nigeria; ii) data on polio incidence and die-out of endemic WPV transmission for all situations

and serotypes (shown in Figure A2 for WPV1 and WPV3 in northern India and northwest Nigeria and for all 3 WPV serotypes in the USA); iii) data from WPV importation outbreak behavior in the Netherlands, Tajikistan, and Albania; iv) data on age distributions of cases for all situations in which meaningful data was available (shown in Figure A2 for the Netherlands, Tajikistan, and Albania); v) available serogical data on the effect of secondary OPV immunity in the USA and Cuba (not shown); vi) indigenous emergence of cVDPVs (shown in Figure A2 for northern India, NW Nigeria (both serotype 2), Haiti, and Madura in Indonesia (both serotype 1); and vii) no indigenous emergence of cVDPVs in all other situations and serotypes (die-out of serotype 1 OPV-related viruses shows in Figure A2 for Cuba and Haiti). We subsequently applied the model to successfully reproduce the asymptomatic transmission of an imported WPV1 in Israel in 2013.[2]

A3. Characterization of simultaneous IPV and OPV use during oSIAs

As mentioned in the main paper, the immunity state structure of the DEB model assumes that successful vaccination with IPV from any recent or waned LPV immunity state brings those individuals back to the highest immunity state (i.e., the same properties as 2 or more recent homotypic LPV infections, see Online Appendix A1). Although we assume higher inherent average per-dose take rates for mOPV2 and the serotype 2 component of tOPV vs. IPV in northwest Nigeria (i.e., 70% vs. 63%, respectively,),[3, 4] the model assumes that individuals with prior LPV-induced immunity possess reduced susceptibility to subsequent LPV infections, including OPV vaccinations, but that this reduced susceptibility does not affect the probability of IPV boosting. Therefore, for individuals with prior immunity, we multiply the effective rate of vaccination (i.e., the fraction of individuals targeted who receive a successful vacccine dose per unit time) for OPV by the relative susceptibility associated with the current immunity state of the individual, while we do not multiply the effective vaccination rate for IPV by the relative susceptibility.[1] For example, an individual with a typical relative susceptibility of 0.5 due to partially waned immunity from prior OPV doses will receive an intestinal immunity boost from a subsequent mOPV2 dose with a probability of take of $0.7\times0.5=0.35$ (after accounting for relative susceptibility), while the same individual would receive an intestinal immunity boost from a subsequent IPV dose with a probability of take of 0.63.

As mentioned in the main paper, we assume that co-administration of IPV and OPV (OPV+IPV) does not reduce the impact of OPV. Thus, for any OPV+IPV SIA, the proportion of fully susceptible OPV+IPV recipients that takes to OPV remains $cov \times tr_{opv}$, where cov denotes the appropriate SIA coverage and tr_{opv} the average per-dose take rate of OPV. The remaining OPV+IPV recipients that do not take to OPV may still take to IPV, so that the proportion of OPV+IPV recipients that takes to IPV equals $(cov- cov \times tr_{opv}) \times tr_{ipv}$, where tr_{ipv} denotes the average per-dose take rate of IPV. Ignoring in- and outflows of fully susceptible individuals due to non-vaccine processes (e.g., aging, mortality),[1, 3] accomplishing these proportions requires proportional outflows due to IPV and OPV take:

$$evr_{tot} = -\ln(1- cov \times (tr_{opv} + (1 - tr_{opv}) \times tr_{ipv}))/d$$
$$evr_{opv} = evr_{tot} \times cov \times tr_{opv}/(cov \times (tr_{opv} + (1 - tr_{opv}) \times tr_{ipv}))$$
$$evr_{ipv} = evr_{tot} \times (cov- cov \times tr_{opv}) \times tr_{ipv} /(cov \times (tr_{opv} + (1 - tr_{opv}) \times tr_{ipv}))$$

where evr_{tot} = total effective vaccination rate due to OPV+IPV
evr_{opv} = effective vaccination rate due to OPV
evr_{ipv} = effective vaccination rate due to IPV
d = duration of the SIA

For individuals with pre-existing maternal, IPV-induced, or LPV-induced immunity, the equations differ somewhat due to the multiplication of the evr values by the relative susceptibility in the model flows for OPV but not in those for IPV.[1, 3] The proportion effectively vaccinated with OPV equals $OPV_{vacc} = 1 - (1 - cov \times tr_{opv})^{Srel}$, where Srel denotes the relative susceptibility of the immunity state.[1] The proportion effectively vaccinated with IPV equals $IPV_{vacc} = (cov\text{-}OPV_{vacc}) \times tr_{ipv}$. The effective vaccination rates for individuals with pre-existing immunity equal:

$evr_{tot} = -\ln(1 - (OPV_{vacc} + IPV_{vacc}))/d$
$evr_{opv} = evr_{tot} \times OPV_{vacc} / (OPV_{vacc} + IPV_{vacc})$
$evr_{ipv} = evr_{tot} \times IPV_{vacc} / (OPV_{vacc} + IPV_{vacc})$

In the event of unequal IPV and OPV coverage or fractional SIAs that unevenly target less than the entire population with IPV and OPV, for simplicity we assume complete overlap between OPV and IPV use, i.e., that the proportion who receives OPV+IPV equals the lesser of the fraction or coverage for the two vaccines. With this assumption, multiplication of branches and addition of identical boxes on the right in Figure A3 yields the proportions that receive no vaccine, OPV-only, IPV-only, and OPV+IPV during any given OPV+IPV SIA. For these proportions, we then again use proportional allocation of the total proportion that gets vaccinated following the logic of the above equations to determine the aggregate OPV and IPV effective vaccination rates for all groups combined as a result of an SIA that involved both OPV and IPV.

A4. Updated NW Nigeria model results

Nigeria introduced IPV in routine immunization co-administered with the third non-birth OPV dose between late February and June 2015, prioritizing high-risk areas in the north.[5] We assume an average IPV routine immunization introduction date of March 15, 2015. Table A2 shows the updated SIA schedule for northwest Nigeria, based on actually conducted activities in 2014-2016 and planned activities for 2016-2017. The last column in Table A2 shows the assumed relative SIA coverage in the under-vaccinated subpopulation compared to the general population both for the updated base case and for the modified case that results in a cVDPV2 outbreak after OPV2 cessation. For the general population, we assume true SIA coverage of 85% and repeated missed probability of 0.85 (i.e., coverage for previously missed children of 15%) for all SIAs in Table A1.

Figure A4 shows the net reproduction numbers (R_n values) for the prior [4, 6, 7] and updated northwest Nigeria model, including the modified case for serotype 2 without any outbreak response. R_n equals one minus the mixing-adjusted effective immune proportion (EIPM), multiplied by the seasonally varying and serotype-specific R_0 of WPV and VDPV (i.e., fully-reverted OPV-related virus in stage 19).[8] We show R_n values for this update because the most recent previous analysis of the model showed the R_n values, because R_n values provide an appropriately scaled comparison between serotypes, and because the absence of large numbers of

cases makes polio incidence comparisons not meaningful.[4] The addition of IPV to routine immunization and two small SIAs in 2015 results in a negligible change in R_n values for all three serotypes (not shown), while the change in SIA schedules substantially alters the R_n behavior. For serotypes 1 and 3, the change in SIA schedule only slightly changes the behavior during 2014-2015 (Figure A4a and c). However, the currently planned 2 annual bOPV SIAs for 2016 and 2017 (Table A2) result in a substantial increase in serotype 1 and 3 R_n values (i.e., lower population immunity to transmission) compared to the previously assumed high frequency of SIAs going forward. Without further improvements in the coverage for the under-vaccinated subpopulation, this leads to a cVDPV1 outbreak in early 2019, as shown in Figure A4a by the decrease in R_n in 2019 due to natural immunity from the outbreak. For serotype 2, the difference between the previous model update and the new base case shows the clear effect on R_n values of tOPV intensification for SIAs, starting in the first half of 2015 (Table A2), which prevents the cVDPV2 outbreak that would otherwise occur. The modified case effectively offsets this gain with the deterioration of the relative SIA coverage in the under-vaccinated subpopulation in the second half of 2015 and early 2016 (Table A2), which results in the cVDPV2 outbreak.

For completeness, Figure A5 shows the incidence curve for the updated model results with no oSIAs to respond to the cVDPV2 outbreak.

A5. Breakdown of population immunity by subpopulation with or without IPV added to oSIA2

Figure A6 breaks down the EIPM by subpopulation for the comparison of No IPV to IPV added during oSIA1. The subpopulation-specific EIPM accounts for mixing between mixing age groups but not between subpopulations, while the overall EIPM for both subpopulations adjusts for mixing between age groups and subpopulations.[8] Clusters of individuals with higher ability to participate in transmission (e.g., the first mixing age group of 0-4 year olds, which includes all children born since OPV2 cessation, and the under-vaccinated subpopulation, which includes significantly more missed children before the oSIAs than the general population) disproportionally influence the overall EIPM. Consequently, before oSIA1, the overall EIPM for both subpopulations remains much closer to that for the initial outbreak population (i.e., the under-vaccinated subpopulation) than to that for the general population despite the 9-fold smaller size of the initial outbreak subpopulation. The curves for No IPV and IPV added during oSIA1 still overlap at this point because the strategy change starts at oSIA1. After oSIA1, which targets only the initial outbreak population, the addition of IPV results in a notably higher EIPM in the initial outbreak population for IPV added during oSIA1 than for No IPV, with no difference in the general population. Without vaccination during oSIA1 in the general population, its EIPM becomes the lowest subpopulation-specific EIPM and almost entirely determines the overall EIPM. Following oSIA2, the use of mOPV2 in the entire population makes the difference between the two curves for the initial outbreak subpopulation much smaller, with still no difference between IPV added during oSIA1 and No IPV in the general population. The EIPM in the general population continues to drive the EIPM for both subpopulations due to its size, such that the overall EIPMs for both subpopulations remains very close for both oSIA strategies. The behavior in Figure A6 explains the negligible difference between the overall EIPM for No IPV and IPV added during oSIA1 in Figure 1b, despite the notable difference in incidence in Figure 1a.

A6. Impact of IPV take rate assumptions

Figure A7 shows the effect of average per-dose take rate assumption for IPV on the impact of adding IPV use during outbreak mOPV2 oSIAs. Changing the IPV take rate does not prevent the cVDPV2 outbreak, but slightly alters its course because of the use of IPV during routine immunization, which remains very low in northwest Nigeria (i.e. approximately 14% coverage with the third non-birth routine immunization dose).[4] Consequently, the outbreaks in Figure A4 differ even for No IPV use during the oSIAs, with an overall earlier outbreak for lower IPV take rate. Not surprisingly, the area between the curves for No IPV and IPV added during oSIA2 becomes larger for a higher IPV take rate, although even with 95% IPV take the benefit remains only incremental to the already large reduction accomplished by mOPV2 use during oSIA2.

Table A1: Generic inputs of the DEB model,[1, 3] and situation-specific model inputs for northwest Nigeria not modified since prior updates [1, 3, 4, 6-8] (see Table A2 for model inputs that we updated)

a) Generic model inputs

Model input (symbol)	Best estimate	Source
Relative susceptibility (σ) of recent immunity states (for PV1;PV2;PV3)		[9, 10]
- Maternally immune	0.78;0.79;0.77	
- 1 successful IPV	0.91;0.92;0.90	
- 2 successful IPV	0.80;0.80;0.79	
- ≥ 3 successful IPV	0.72;0.72;0.71	
- 1 LPV infection	0.42;0.43;0.41	
- ≥ 2 LPV infections	0.21;0.22;0.20	
- IPV and LPV	0.21;0.22;0.20	
Duration of latent period (ξ^{fec} or ξ^{oro}, in days)	~ 3[a]	[9, 10]
Duration of fecal infectiousness (γ^{fec}, in days) of recent immunity states (for PV1;PV2;PV3)		[9, 10]
- Fully susceptible	28.0;27.8;28.3	
- Maternally immune	24.6;24.6;24.6	
- 1 successful IPV,	24.5;24.4;24.7	
- 2 successful IPV	21.1;20.8;21.3	
- ≥ 3 successful IPV	18.0;17.7;18.2	
- 1 LPV infection	11.6;10.5;10.5	
- ≥ 2 LPV infections	10.1;8.9;8.9	
- IPV and LPV	10.1;8.9;8.9	
Duration of oropharyngeal infectiousness (γ^{oro}, in days) of recent immunity states (no serotype differences)		[9, 10]
- Fully susceptible	13.4	
- Maternally immune	11.9	
- 1 successful IPV	9.9	
- 2 successful IPV	6.6	
- ≥ 3 successful IPV	6.1	
- 1 LPV infection	5.0	
- ≥ 2 LPV infections	3.7	
- IPV and LPV	3.7	
Relative fecal infectiousness (π^{fec}) of recent immunity states (for PV1;PV2;PV3)		[9, 10]
- Maternally immune	0.96;0.96;0.95	
- 1 successful IPV	0.92;0.92;0.91	
- 2 successful IPV	0.70;0.69;0.68	
- ≥ 3 successful IPV	0.61;0.59;0.59	
- 1 LPV infection	0.39;0.43;0.43	
- ≥ 2 LPV infections	0.20;0.23;0.23	
- IPV and LPV	0.20;0.23;0.23	
Relative oropharyngeal infectiousness (π^{oro}) of recent immunity states (no serotype differences)		[9, 10]
- Maternally immune	0.68	
- 1 successful IPV	0.30	
- 2 successful IPV	0.17	
- ≥ 3 successful IPV	0.12	
- 1 LPV infection	0.33	
- ≥ 2 LPV infections	0.21	
- IPV and LPV	0.21	
Number of infection stages		
- Latent period (r)	2	

- Infectious period (s)	4	
Relative weight of infection stages, compared to average weight over the infectious period ($\theta_j, j=0,\ldots,r+s-1$)		[9, 10]
- Infection stage 0 and 1 (latent stages)	0	
- Infectious stage 2	12/17	
- Infectious stage 3	40/17	
- Infectious stage 4	12/17	
- Infectious stage 5	4/17	
IPV immunity delay (φ, in days)	7	[11]
Number of waning stages (nw)	5	
Shape of waning function (z_w)	5	[9, 10]
Average time to reach last waning stage (ρ, in days)		[9, 10]
- Type 1&2	4×365	
- Type 3	3×365	
Average time for maternal immunes to wane to fully susceptible (ρ_{MI}, in days)	0.25×365	[9, 10]
Relative susceptibility (σ) for last waning stage (no serotype differences)		[9, 10]
- 1 successful IPV	1.0	
- 2 successful IPV	1.0	
- ≥ 3 successful IPV	1.0	
- 1 LPV infection	0.8	
- ≥ 2 LPV infections	0.7	
- IPV and LPV	0.7	
Duration of fecal infectiousness (γ^{fec}, in days) of last waning stage (for PV1;PV2;PV3)		[9, 10]
- 1 successful IPV	26.6;26.4;26.9	
- 2 successful IPV	25.2;25.0;25.5	
- ≥ 3 successful IPV	23.8;23.6;24.1	
- 1 LPV infection	14.0;13.9;14.1	
- ≥ 2 LPV infections	11.4;11.4;11.6	
- IPV and LPV	11.4;11.4;11.6	
Duration of oropharyngeal infectiousness (γ^{oro}, in days) of last waning stage (no serotype differences)		[9, 10]
- 1 successful IPV	11.4	
- 2 successful IPV	6.7	
- ≥ 3 successful IPV	6.6	
- 1 LPV infection	6.7	
- ≥ 2 LPV infections	4.0	
- IPV and LPV	4.0	
Relative fecal infectiousness (π^{fec}) of last waning stage (no serotype differences)		[9, 10]
- 1 successful IPV	0.95	
- 2 successful IPV	0.9	
- ≥ 3 successful IPV	0.85	
- 1 LPV infection	0.5	
- ≥ 2 LPV infections	0.3	
- IPV and LPV	0.3	
Relative oropharyngeal infectiousness (π^{oro}) of last waning stage (no serotype differences)		[9, 10]
- 1 successful IPV	0.43	
- 2 successful IPV	0.25	
- ≥ 3 successful IPV	0.13	
- 1 LPV infection	0.5	
- ≥ 2 LPV infections	0.3	
- IPV and LPV	0.3	
Number of reversion stages (h)	20	

Shape of reversion function with respect to: - R_0 (z_r) - ln(PIR) (z_p)	 1 2.5	
Average time to reach last reversion stage (ε, in days) (for PV1;PV2;PV3)	620.5; 408; 620.5	[3]
Paralysis-to-infection ratio for fully susceptible individuals infected with OPV (PIR_0) (for PV1; PV2;PV3)	0.26×10^{-6}; 1.2×10^{-6}; 1.8×10^{-6}	
Paralysis-to-infection ratio for fully susceptible individuals infected with FRPV (PIR_{h-1}) (for PV1; PV2;PV3)	0.005; 0.0005; 0.001	[1, 11, 12]
Relative R_0 of OPV vs. FRPV (τ_0) (for PV1; PV2; PV3)	0.37;0.55;0.25	[1, 9, 10]
Effective infectious proportion below which we assume 0 force-of-infection (transmission threshold *EPI**)	5/1,000,000	
Relative PIR for maternally immunes compared to fully susceptible individuals ($RPIR_{MI}$)	0.5	
Ratio of R_0 by serotype in the same setting (PV1:PV2:PV3)	1:0.9:0.75	[3]
Average incubation period (δ, in days)	10	[11, 13]
Demographics for all situations	Time series 1950-2100	[14]

Acronyms: CDC = (U.S.) Centers for Disease Control and prevention; cVDPV = circulating vaccine-derived poliovirus; DEB = differential equation-based FRPV = fully-reverted poliovirus; GPLN = Global Polio Laboratory Network; IPV = inactivated poliovirus vaccine; LPV = live poliovirus; OPV = oral poliovirus vaccine; PIR = paralysis-to-infection ratio; PV(1,2,3) = poliovirus (type 1, 2, or 3, respectively); R_0 = basic reproductive number; UN = United Nations; USA = United States of America; VAPP = vaccine-associated paralytic poliomyelitis; VP1 = viral protein 1; WPV(1,2,3) = wild poliovirus (type 1, 2, or 3, respectively)
Notes: [a] Mean estimates obtained from experts and used in the model for the different immunity states, serotypes, and excretion modes vary between 2.85 and 3.37 days

b) Model inputs specific for northwest Nigeria (based on original model [1] and subsequently updated model [3, 4, 6-8] to reflect evolving vaccination choices

Model input (symbol)	**Best estimate**	**Source**
Number of subpopulations	2	
Relative population size compared to all of Nigeria	0.256	[15]
Number of age groups	10	
Number of mixing age groups	3	
Year when model run-up starts	1970	
Year when R_0 seasonality starts	1980	
Year when die-out first allowed	1980	
Average basic reproductive number (R_0) (PV1)	7.5	[3]
Proportional change in R_0 due to seasonality (α)	0.1	
Day of seasonal peak in R_0	100 (April 10)	
Proportion of contacts reserved for individuals within the same mixing age group (κ)	0.4	
Proportion of potentially infectious contacts of individuals in subpopulation 1 that are with other individuals in subpopulation 1 (p_{within})	0.88	[11]
Proportion of transmissions via oropharyngeal route (p^{oro})	0.3	[9]
Per-dose take rate (tr) (PV1;PV2;PV3) - tOPV - mOPV - bOPV	 0.45;0.7;0.35 0.6;NA;0.6 0.54;NA;0.54	
Coverage with 3 or more doses by 1 year of age, and partial coverage	DTP coverage time series 1984-2012	[16-18]
Characterization of routine tOPV vaccination from 1984 - Doses at birth - Cumulative effect of 3 primary non-birth doses at ages (months)	 1 3	[3, 7, 19]

- Relative coverage in under-vaccinated subpopulation 1984-2011 - Relative coverage in under-vaccinated subpopulation 2012 - Relative coverage in under-vaccinated subpopulation 2013 - Relative coverage in under-vaccinated subpopulation 2014+	0 0.2 0.25 0.3	
Characterization of OPV campaigns from 1996-2014	See Duintjer Tebbens et al. (2014)[3]	[3]
Characterization of OPV campaigns from 2014-2020	See Table A2	

Acronyms: bOPV = bivalent OPV; mOPV = monovalent OPV; NA = not applicable; OPV = oral poliovirus vaccine; PV1,2,3 = poliovirus type 1, 2, and 3, respectively; SIA = supplemental immunization activity; tOPV = trivalent OPV

Notes: * Age groups marked with an asterisk indicate age groups that count towards determining the fraction of newborns that received maternal antibodies, based on the immune fraction in those age groups

[a] Information about SIA history as reported to WHO and elsewhere[20-29]

Table A2: Updated SIA assumptions for the northwest Nigeria model based on the actual vaccination activities. SIA assumptions used in most recent previous model update [6] given in parentheses, if different

Activity	Vaccine	Fraction of northwest Nigeria targeted	Duration	Relative SIA coverage in the under-vaccinated subpopulation, given as base case value; value for modified case that leads to a cVDPV2 outbreak after OPV2 cessation
2014 SIAs (month/day)				
- 1/25	bOPV	1	4	0.20; 0.20
- 3/1	bOPV	1	4	0.20; 0.20
- 3/22[a]	bOPV	0.021	4	0.20; 0.20
- 4/12	bOPV	1	4	0.20; 0.20
- 5/24	bOPV	1	4	0.20; 0.20
- 6/19 (6/24)	bOPV	1	3 (4)	0.20; 0.20
- 7/12[a]	bOPV	0.15	4	0.20; 0.20
- 8/9	tOPV	1	4	0.20; 0.20
- 8/21[a]	bOPV	0.29	4	0.20; 0.20
- 9/20	bOPV	1	4	0.20; 0.20
- 10/11[a]	bOPV	0.041	4	0.20; 0.20
- 11/1	tOPV	0.92 (1)	4	0.20; 0.20
- 12/13	bOPV	0.90 (1)	4	0.20; 0.20
- 12/17[a]	tOPV	0.18	4	0.20; 0.20
2015 SIAs (month/day)[b]				
- 1/24 (1/25)	bOPV	1	4	0.20; 0.20
- 3/1[a]	tOPV+IPV[c]	0.079	4	0.20; 0.20
- 3/14 (3/1)	tOPV	1	4	0.20; 0.20
- 4/25 (4/12)	tOPV (bOPV)	1	4	0.20; 0.20
- 4/25[a]	IPV[c]	0.036	4	0.20; 0.20
- 6/19 (6/21)	bOPV	1	3 (4)	0.20; 0.20
- 7/7[a]	tOPV	0.17	4	0.20; 0.15
- 7/27	tOPV	1	3	0.20; 0.10
- 8/18 (8/9)	tOPV	0.17 (1)	4	0.20; 0.08
- 9/15 (9/20)	tOPV (bOPV)	1	3 (4)	0.20; 0.06
- 10/17 (11/1)	tOPV	1	3 (4)	0.20;0.05
- 12/17 (12/13)	bOPV	1	3 (4)	0.20;0.20[d]
2016 SIAs (month/day)[e]				
- 1/19	tOPV (bOPV)	1	4	0.20;0.04
- 2/27[a]	tOPV	1	4	0.20;0.03
- 3/19 (3/1)	tOPV	1	4	0.20;0.02
- 5/14 (5/24)	bOPV	1	4	0.20;0.20[d]
- 10/15[a]	bOPV	1	4	0.20;0.20[d]
2017 SIAs (month/day)[e]				
- 2/1 (1/25)	bOPV	1	4	0.20;0.20[d]
- 10/15 (11/1)	bOPV	1	4	0.20;0.20[d]

[a] Activity not included in previous model update

[b] Excludes one previously assumed full-scale bOPV SIA on 5/24/2015 that did not take place

[c] IPV targeted only children aged 14 weeks (March round) or 6 months (April round) to 59 months

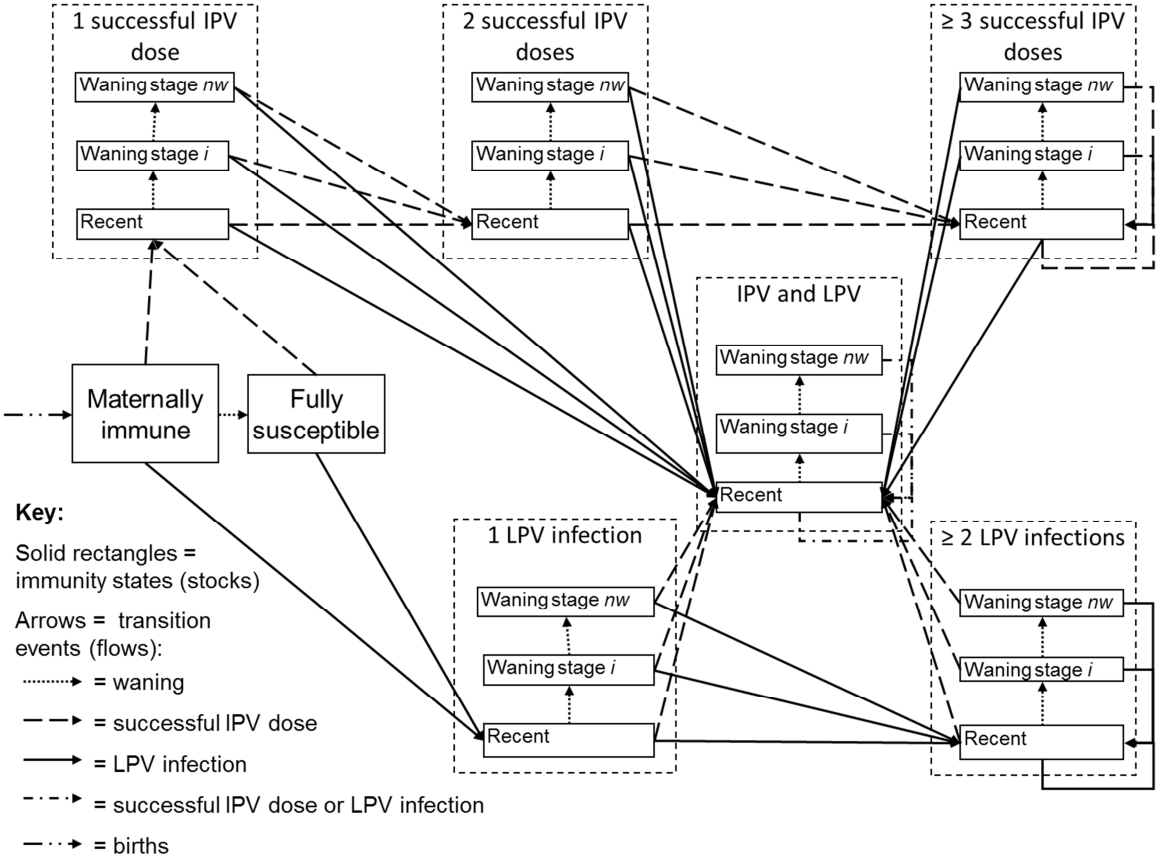
[d] Not modified due to lack of influence of bOPV SIAs on serotype 2 results

[e] Excludes six previously assumed bOPV SIAs in April, June, August, November, and December that did not take place

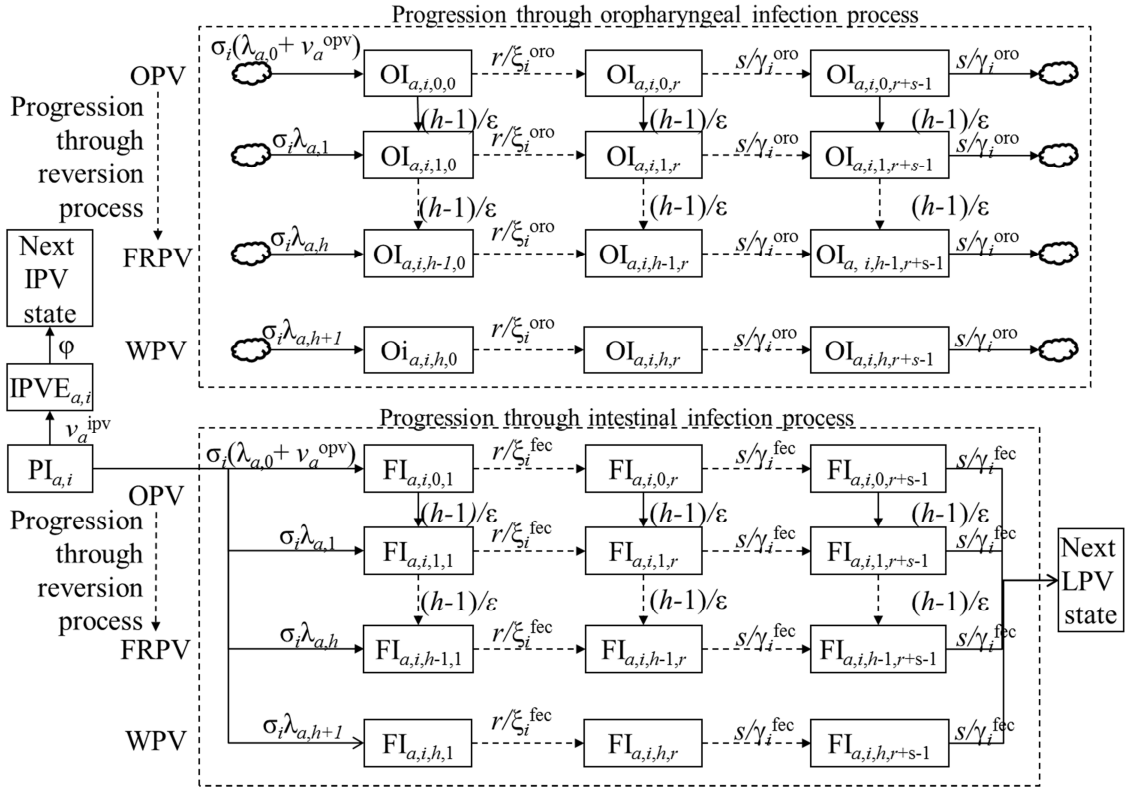
[f] Excluding outbreak response SIAs; in the absence of data on expected SIAs, previous model update assumed continuation of expected schedule for 2015 and 2016 of 9 SIAs in February, March, April, May, June, August, September, October, and December.

Figure A1: Schematic of the DEB model structure, adopted from Duintjer Tebbens et al. (2013)[1, p. 706]

(a) Immunity states and flows between them due to epidemiological events



(b) Progression through infection and reversion stages



"Acronyms: FRPV = fully-reverted poliovirus; IPV = inactivated poliovirus vaccine; OPV = oral poliovirus vaccine; WPV = wild poliovirus**; Symbols:** $PI_{a,i}$ = partially infectible in age group *a* and immunity state *I*; $IPVE_{a,i}$ = IPV-exposed individual from immunity state *i* and age group *a*; $FI_{a,i,j,k}$ ($OI_{a,i,j,k}$) = individual in age group a from immunity state *i*, infected with virus strain *j* and in fecal (oropharyngeal) infection stage *k*; $\lambda_{a,j}$ = force-of–infection to age group *a* for virus strain *j;* ν_a^{ipv} (ν_a^{opv}) = force-of-IPV(OPV)-vaccination to age group *a* as a result of routine and supplementary immunization; σ_i = relative susceptibility for immunity state *i*; ξ_i^{fec} (ξ_i^{oro}) = average duration of the fecal (oropharyngeal) latent period for immunity state *i*; γ_i^{fec} ($\gamma_i^{oro)}$ = average duration of the fecal (oropharyngeal) infectious period for immunity state *i*; φ = IPV immunity delay; *h* = number of reversion stages; *r* = number of latent stages; *s* = number of infectious stages" [1, p. 706]

Figure A2: Summary results from the model calibration process, adapted from Duintjer Tebbens et al. (2013)[1]

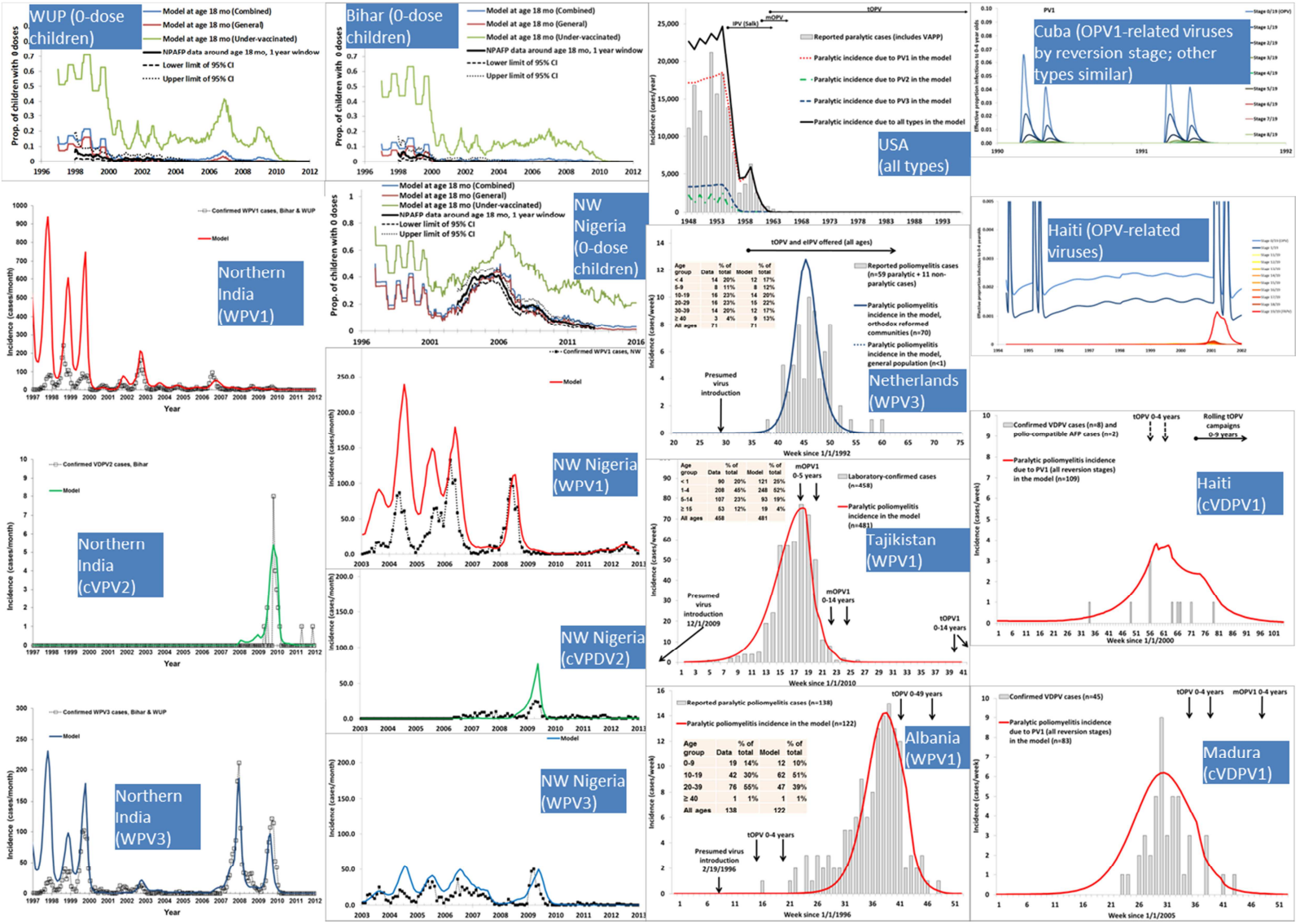
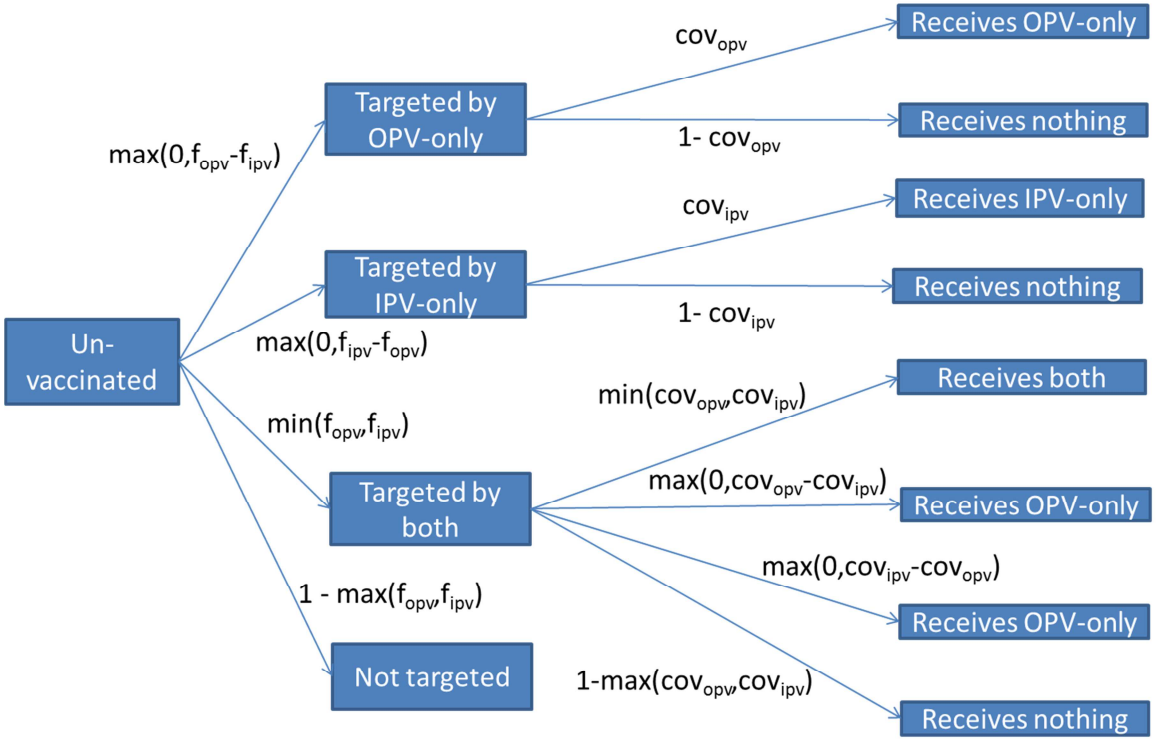


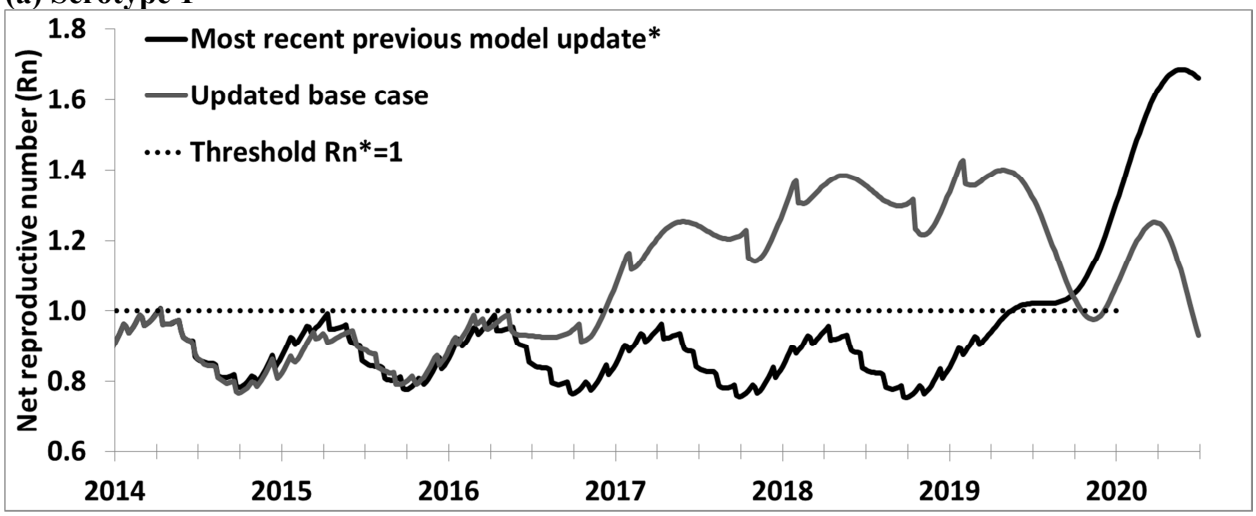
Figure A3: Possible pathways of IPV and OPV vaccination in the event of unequal OPV and IPV coverage values and/or fractions, assumed full OPV-IPV overlap



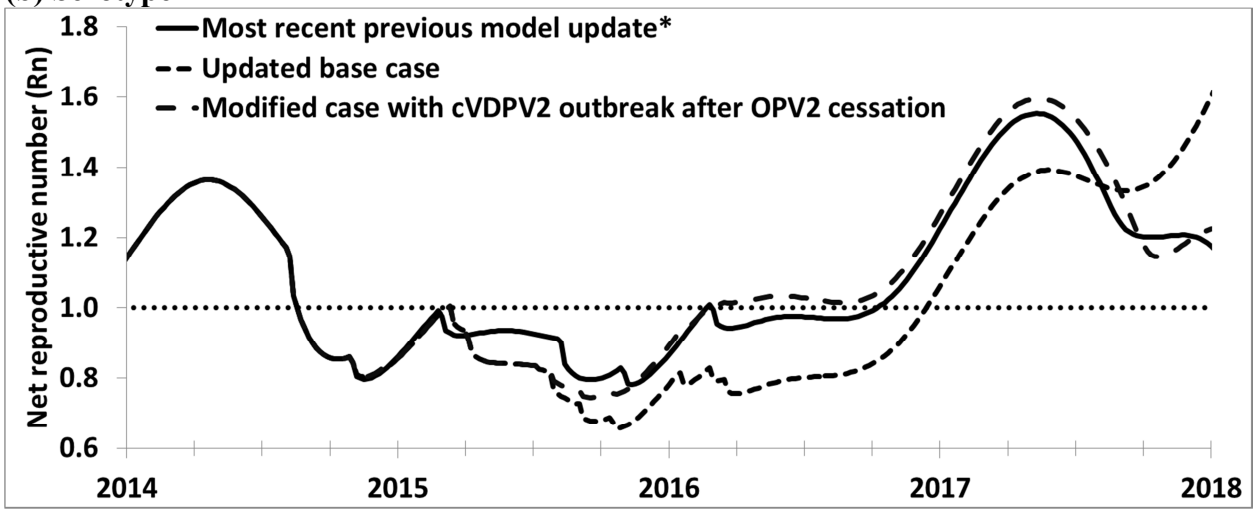
Notation: cov_{ipv} = coverage achieved with IPV; cov_{opv} = coverage achieved with OPV; f_{ipv}=fraction of target age group targeted with IPV; f_{opv}=fraction of target age group targeted with OPV

Figure A4: Updated model results based on revised SIA assumptions (Table A1) and addition of IPV for routine immunization from March 15, 2015, in terms of net reproduction numbers (R_n values). *As published in prior work [4, 6, 7]

(a) Serotype 1



(b) Serotype 2



(c) Serotype 3

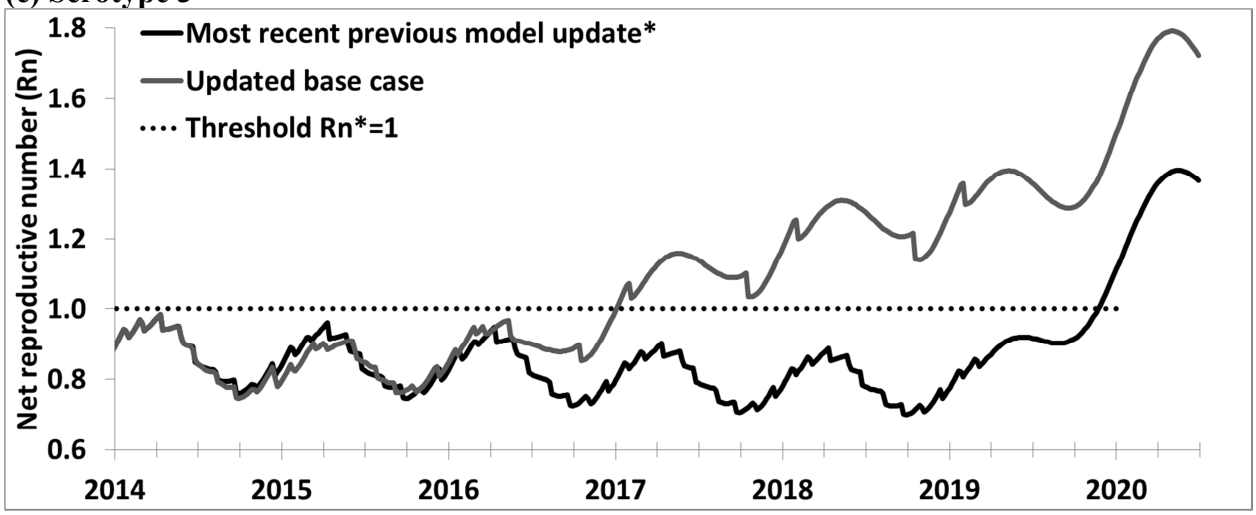


Figure A5: Outbreak curve in the absence of an outbreak response (full No oSIA curve from main paper figures)

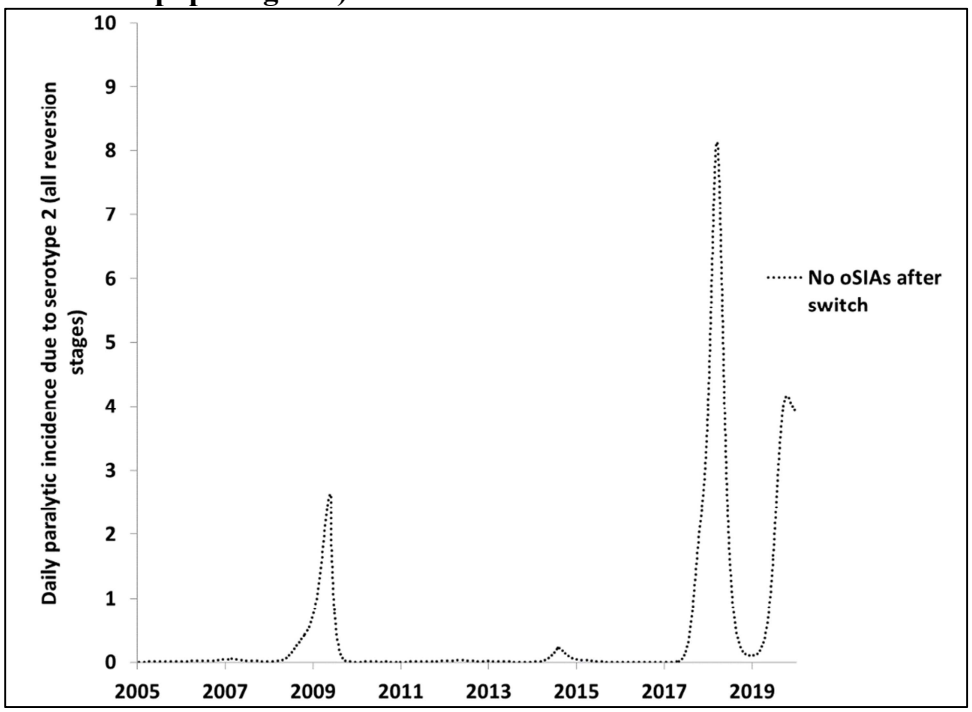


Figure A6: Breakdown of population immunity to serotype 2 transmission by subpopulation with or without IPV added to oSIA2.

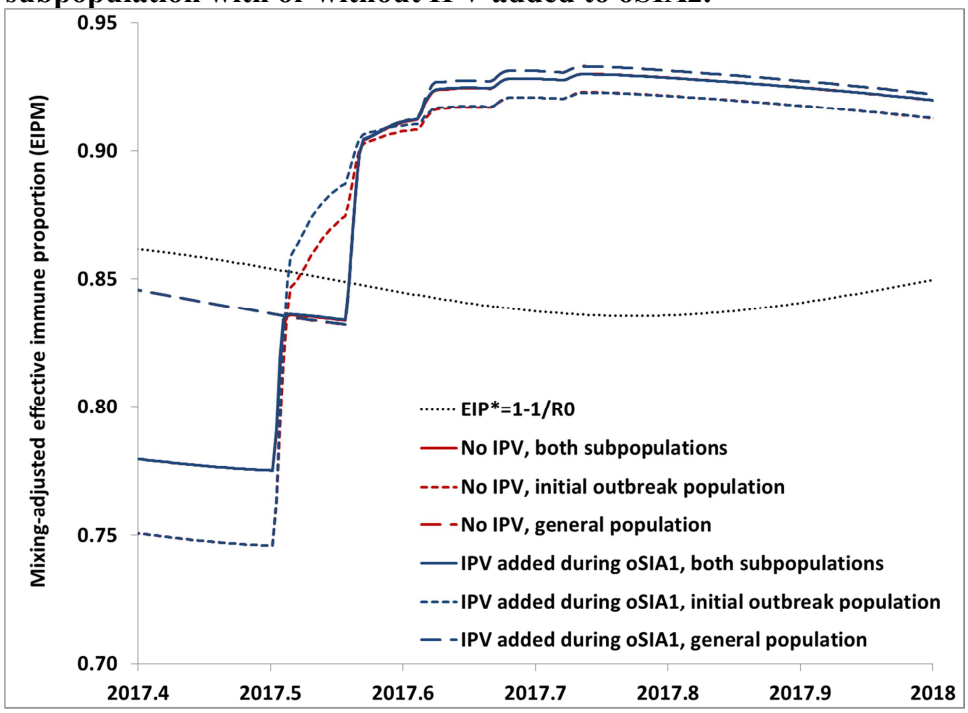
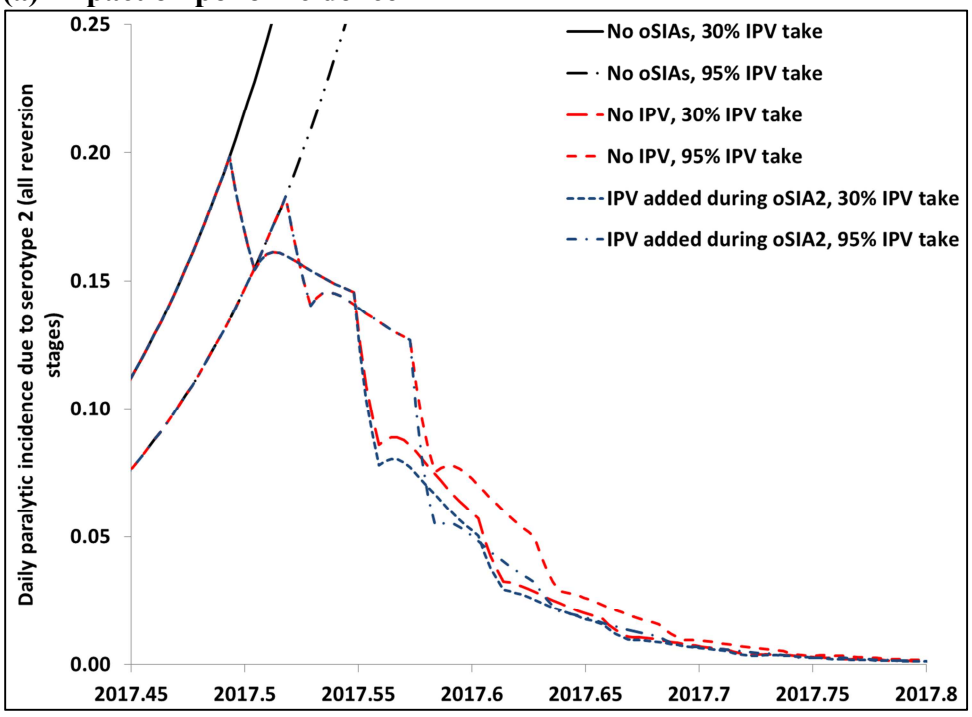
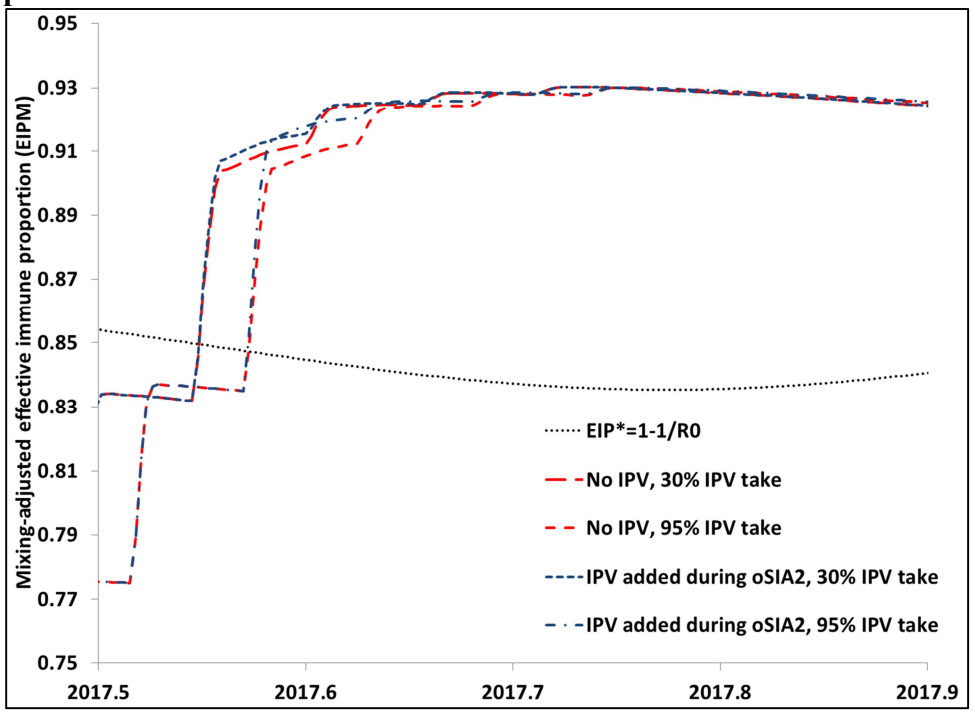


Figure A7: Effect of average per-dose take rate assumptions for IPV on the impact of adding of IPV use during outbreak mOPV2 oSIAs.

(a) Impact on polio incidence



(b) Impact on population immunity in comparison to the threshold effective immune proportion (EIP*) needed to stop transmission of serotype 2 wild or fully-reverted poliovirus



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