TECHNICAL APPENDIX for "Global transmission of live polioviruses: Updated dynamic modeling of the polio endgame"

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TECHNICAL APPENDIX

Any references to tables and figures in the text below not preceded by "A" refer to the tables and figures in the main paper. New tables and figures that appear in the appendix include an "A" followed by a number. The appendix uses numbers for the references and includes references at the end of the text (before the tables and figures). Although the development of this model builds on nearly 20 years of experience modeling polio and numerous prior publications, this comprehensive appendix includes all of the assumptions and inputs required to fully describe the model presented in the main text and alternative variations of the model (e.g., other populations, stochastic versions). Section A1 provides an introduction to polioviruses and poliovirus vaccines for those unfamiliar with polio. Section A2 provides details about the previously-developed differential equation-based poliovirus transmission and OPV evolution (DEB) model that we use to characterize transmission. Section A3 provides details about the global model. Section A4 provides an overview of how we use the results from the DEB model to characterize and value health economic outcomes. Section A5 provides details about alternative ways that we formulate the DEB model to address questions for specific areas (e.g., countries) and how we convert the deterministic model into a stochastic formulation to characterize the confidence about no circulation

A1. Poliovirus and vaccines

This section provides a brief introduction to polioviruses, poliovirus vaccines, and acronyms used to describe them. Those familiar with poliovirus and vaccines can skip directly to appendix A2.

A1.1. Polioviruses

Comprised of short sequences of ribonucleic acid (RNA), enteroviruses remain stable and survive in acidic conditions like those in the human gastrointestinal tract. Polioviruses (PVs) are human enteroviruses in the *Picornaviridae* family that exist in three stable forms (i.e., serotypes 1, 2 and 3), also called PV1, PV2, and PV3, which transmit independently in populations. With humans representing the only reservoir for poliovirus transmission, stopping transmission in all humans leads to eradication. Transmission of polioviruses in human populations typically involves fecal-oral spread, but oropharyngeal or (oral-to-oral) spread may also occur and represent the dominant route of transmission in populations with high levels of hygiene and sanitation. Live polioviruses may survive and reproduce in the throat and gut and cause infection. Polioviruses may replicate in and destroy motor neurons in the central nervous system, which may present clinically as paralyzed limbs, respiratory arrest, and in rare cases lead to death. The three poliovirus serotypes differ sufficiently such that infection with one serotype does not prevent infection with another serotype, and immunity to one serotype does not yield

significant immunity to the others. However, after the first infection with any live poliovirus serotype, individuals appear to benefit from immunological protection from paralysis, although immune individuals can become asymptomatically infected and participate in the transmission of virus (albeit with lower chances of becoming infected and passing the virus on to others compared to fully susceptible people). In fully susceptible individuals (i.e., those not yet immune to the virus) approximately 1 in 200 infections with a WPV results in paralytic poliomyelitis (or paralytic polio), although the paralysis-to-infection ratio (PIR) varies by serotype (i.e., (1/200 or 0.005 for serotype 1, 1/2000 or 0.0005 for serotype 2, and 1/1000 or 0.001 for serotype 3) [1-3]. Prior to the introduction of poliovirus vaccines, paralysis from polio occurred with seasonal outbreaks that traumatized communities. As of 2019, WPV serotype 1 (WPV1) continues to endemically circulate in parts of 2 countries (i.e., Afghanistan and Pakistan) [4]. The last reported case of WPV serotype 2 (WPV2) occurred in 1999 in northern India, and the Global Commission for the Certification of Poliomyelitis Eradication declared WPV2 eradicated in September 2015 [5]. The last reported case of WPV serotype 3 (WPV3) occurred in 2012 in northeast Nigeria [6].

A1.2. Poliovirus vaccines

Intensive research efforts in the USA led to the development of two poliovirus vaccines introduced in the 1950s and 1960s [7], which differ significantly with respect to their mode of administration, costs, risks, and the protection they provide from infection. The live oral polio vaccine (OPV) serves as the primary vaccine for the control and eradication of polio. Using OPV offers several benefits, including low-cost of production and ease of delivery. As a vaccine containing a live, attenuated virus, OPV causes an infection in vaccine recipients, who can spread their infections to other people, which provides or boosts immunity. Infection with OPV induces a mucosal, intestinal immune response and provides good protection from re-infection, but its use comes with some risks. First, OPV causes very rare vaccine-associated paralytic polio (VAPP) in approximately 1 in a million vaccine recipients (with paralysis rates for serotype 3> 2>1). This represents a much smaller risk than infection with WPV, however, after WPV elimination, the risk of VAPP from using OPV becomes observable and difficult to accept. Second, in populations that use OPV with low coverage and thus maintain low population immunity, circulating OPV can continue to infect individuals and over time lose its attenuating mutations to become OPV-related viruses. These OPV-related viruses can continue to evolve, and may become circulating vaccine-derived polioviruses (cVDPVs) that behave like WPVs and can cause outbreaks. Third, some individuals with B-cell primary immunodeficiencies (PIDs) may also become infected with OPV or an OPV-related virus and experience long-term infections that evolve to immunodeficiency-associated vaccine-derived polioviruses (iVDPVs), which could potentially reintroduce transmission and trigger outbreaks after OPV cessation.

After the initial introduction of OPV in the 1960s, all national immunization programs used OPV and manufacturers produced and licensed trivalent OPV (tOPV), which contained all three serotypes. In addition to delivering OPV in their routine immunization (RI) program, some countries also used OPV in supplemental immunization activities (SIAs). Prior to 2005 and in the context of global use of tOPV, serotype 1 OPV caused the largest cVDPV outbreaks [8]. However, global efforts focused on stopping the transmission of WPV1 led to the use of monovalent OPV (mOPV) for serotype 1 (mOPV1) in some SIAs starting in 2005, then for

serotype 3 (mOPV3) starting in 2007, and later bivalent OPV (bOPV, containing serotypes 1 and 3) starting in 2010. These SIAs resulted in gaps in population immunity to serotype 2 transmission in some high-risk populations and to a significant increase in the size and number of serotype 2 cVDPV (cVDPV2) outbreaks [9]. Live polioviruses (LPVs) include OPV, OPV-related virus (i.e., an OPV virus transmitting through the population and losing its attenuating mutations as it evolves, but still less than fully reverted) or vaccine-derived poliovirus (VDPV), fully-reverted poliovirus (FRPV), and WPVs.

Similar to OPV, inactivated poliovirus vaccine (IPV) offers life-long protection to vaccine recipients from paralysis in the event of infection. As an inactivated (killed vaccine), IPV induces mainly humoral immunity, which does not significantly reduce the probability, duration, or infectiousness of fecal infections. Thus, although IPV protects the vaccine recipient from paralysis, it does not transmit to others, circulate in the population, or evolve. In addition, IPV requires injection by trained health workers, and it costs much more than OPV to produce and administer. Manufacturers produce and license only a trivalent form of IPV. After successfully eliminating national indigenous transmission of WPVs, most high-income countries transitioned their routine immunization schedules from OPV-only to a sequential schedule of IPV followed by OPV and then to IPV-only. The sequential IPV/OPV schedule gives vaccine recipients the benefit of protection from VAPP by exposing them to IPV first, while still inducing intestinal mucosal immunity. Using an IPV-only schedule for vaccination eliminates the opportunity to create new VDPVs. However, because of the transmission potential of LPVs in some populations and the properties of IPV, using IPV alone does not provide enough population immunity to stop transmission in poor hygiene settings due to its inability to spread to contacts or provide as much protection from subsequent participation in asymptomatic transmission as OPV.

A1.3. Acronyms and abbreviations

α	seasonal amplitude
AFP	acute flaccid paralysis
bOPV	bivalent OPV
CEI	cumulative effective (infectiousness-weighted) infections
COV1or2	RI coverage with 1 or 2 non-birth doses
cVDPV	circulating VDPV
D	average number of DALYs per polio case
DALY	disability-adjusted life-year
DEB	differential equation-based
DES	discrete-event simulation
DPT1	first dose of diphtheria toxoid, tetanus toxoid and pertussis vaccine
DPT3	third dose of diphtheria toxoid, tetanus toxoid and pertussis vaccine
dt_x	detection threshold in period x
E*	exportation threshold
EPI	effective proportion infectious
EPI*	transmission threshold
ES	environmental surveillance
ESP	effective susceptible proportion
FC_{AO}	cumulative, discounted financial costs associated with the alternative policy

FC_{RC}	cumulative, discounted financial costs associated with the reference case
FRPV	fully reverted PV
GPEI	Global Polio Eradication Initiative
HI	high income
ICER	incremental cost-effectiveness ratio
INB	incremental net benefit
IPV	inactivated poliovirus vaccine
iVDPV	immunodeficiency associated VDPV
IVIG	intravenous immunoglobulin
κ	age-group preferential mixing strength
LI	low income
LMI	lower middle income
LPV	live poliovirus
mOPV	monovalent OPV
nOPV	new OPV
OPV	oral poliovirus vaccine
OPV1	serotype 1 OPV
OPV2	serotype 2 OPV
OPV3	serotype 3 OPV
oSIA	outbreak response SIA
pd	seasonality peak day
PEF	poliovirus essential facilities
$P_{effective}$	effective introduction probability
PID	primary immunodeficiency
PIM	potentially infectious materials
PIR	paralysis-to-infection ratio
PMA	preferential mixing area
POL3	RI coverage with 3 or more non-birth doses
p^{oro}	proportion of transmissions via the oropharyngeal route
PP_{AO}	cumulative, discounted number of polio cases with the alternative policy
PP_{RC}	cumulative, discounted number of polio cases with the reference case
P_{RM}	repeated missed probability
P_{RR}	repeated reached probability
pSIA	planned, preventive SIA
PV	poliovirus
Q_{ES}	ES quality
R_0	basic reproduction number
RC	reference case
RI	routine immunization
RNA	ribonucleic acid
S	average societal economic costs per polio case
SAGE	Strategic Advisory Group of Experts on Immunization
SIA	supplementary immunization activities
SIA_x	number of planned, and the preventive SIA rounds in period x
sil	SIA impact level
T	average treatment costs per polio case

T_0	beginning of analytical time horizon
TC	true coverage
tcp_x	total catchment percent of the population covered by ES in period x
T_{end}	end of the analytical time horizon
T_{ES}	ES start year
T_{GPEI}	GPEI launch year (1988)
T_{IPV}	IPV use start year
tOPV	trivalent OPV
trl	OPV take rate level
UMI	upper middle income
UN	United Nations
UNICEF	United Nations Children's Fund
VAPP	vaccine-associated paralytic polio
VDPV	vaccine-derived poliovirus
WHO	World Health Organization
WPV	wild poliovirus
WPV1	serotype 1 WPV
WPV2	serotype 2 WPV
WPV3	serotype 3 WPV

A2. Differential-equation based transmission and OPV evolution model

We previously developed a differential equation-based poliovirus transmission and OPV evolution (DEB) model [3, 10]. This section discusses the components, inputs, comprehensive structure, and setting-invariant model inputs, and the process used to fit the model inputs to reproduce poliovirus transmission and OPV evolution in actual settings [3, 10]. Although the updated global model in the main text does not change any of the structure or fixed inputs for the DEB model compared to the prior global model [11], for completeness, we include the details about this model in this appendix. (Those familiar with the DEB model from prior work [11] can skip directly to appendix A3, noting that we updated inputs to reflect current data (e.g., population data, etc.)).

A2.1. Dynamic model

Modeling the transmission of infectious diseases requires tracking the progression of infections over time in a population by solving sets of differential equations used to describe the population and events. The model starts at an initial time (T_{Init}) and typically uses numerical integration techniques to move the population through time one small finite time step (Δt) at a time. The choice of numerical integration method (e.g., the Euler method) influences the nature and size of errors introduced in the numerical integration process, but modelers can minimize the impacts of these errors by using small time steps.

A2.2. Demographics, aging, and age group mixing

Since the transmission of infectious occurs in populations, setting up the population represents the first step in formulating the model. We use the UN World Population Prospects [12] and divide the modeled population into 7 age groups (0-2 months, 0.25-1 year, 1-4 years, 5-9 years, 10-14 years, 15-39 years, and 40 or more years, see Table 1) at T_{Init}. We calculate the effective birth and mortality rates based on the number of surviving infants and the population age distribution in the UN World Population Prospects [12] ignoring sex differences. The effective birth rate equals the number of surviving infants divided by the total population size. We calculate effective birth rates based on surviving infants to account for the real difference in death rates between young infants and children aged 1 year. To calculate effective mortality rates (μ_a), we first allocate annual estimates of one-year-wide age ranges provided in the data to the model age groups (except for the two first age groups for which we ignore mortality since our use of surviving infants already accounts for infant mortality). Next, we calculate μ_a for year *yr* and age group *a* using the formula:

$$\mu_a = \left(N_a(yr) - N_a(yr+1) + \frac{N_{a-1}(yr)}{w_{a-1}} - \frac{N_a(yr)}{w_a}\right) / N_a(yr),$$

where N_a represents the number of people in age group *a* and $w_a$represents the width of age group *a* (in years). We do not explicitly model emigration or immigration, but by using the UN World Population Prospects [12] to exactly reproduce the population estimates by age group, we account for changes in the population from events other than aging, which in some instances implies negative μ_a values. In those instances, the model implicitly adds people to each stock in proportion to the number of people in that stock at the given time, thus providing a somewhat

unbiased method to introduce people into the model to match the estimated population sizes by age group over time. For each time step, individuals conceptually age one Δt, which means that some fraction of the population in each age group needs to transition from a younger age group into the next older age group. We calculate aging rates equal to $1/w_a$ for all age groups (except the last one, which captures all ages over 39 years) and age the population appropriately according to these rates.

We capture the preferential mixing of individuals by age groups by creating 3 mixing age groups of 0-4 years, 5-14 years, and 15 or more years. We use $\kappa(a_m)$ to represent the proportion of contacts in mixing age group a_m reserved for other individuals in the mixing age group a_m, while the remaining proportion $1-\kappa(a_m)$ of contacts gets evenly distributed over all other mixing age groups, including age group a_m [13, 14]. We calculate the normalized age mixing matrix $M(a_m,b_m)$ using the following formula [13]:

$$M(a_m, b_m)(t) = \kappa(a_m)1_{\{a_m=b_m\}} + \frac{(1-\kappa(a_m))(1-\kappa(b_m))NM_{b_m}(t)}{\sum_{c_m=0}^{na_m-1} NM_{c_m}(t)(1-\kappa(c_m))},$$

where $NM_{c_m}(t)$ represents the number of people in mixing age group c_m at time t and the indicator function $1_{\{condition\}}$ equals 1 if the condition holds or 0 otherwise. Given that NM_{c_m} depends on time, the model recalculates the mixing matrix at each time step of the model.

A2.3. Transmission structure (immunity states, infection stages, transition rates, and waning stages)

The DEB model [3] tracks each poliovirus serotype separately (i.e., it contains three serotype indices) and moves people between demographic age groups according to the nature of their immunity for each serotype. The selection of immunity states for the model followed a literature expert review [9, 15] and elicitation process [16] that suggested the need for 8 immunity states and explicit consideration of waning immunity to capture poliovirus transmission dynamics in the population. The 8 immunity states include: maternal immunity (for infants), full susceptibility (i.e., no immunity), and 6 different types of immunity induced by infection with an LPV (including OPV) and/or vaccination with IPV, and accounting for different numbers of doses or prior infections. Figure A1 shows the 8 different immunity states and uses arrows to indicate the flows that move individuals in and out of them [3]. We refer to the immunity state that follows the transition event (i.e., a vaccine dose or infection) as "recent" and we model waning of immunity relative to these recent immunity states. The model assumes that active immunity from prior infection or any immunization results in permanent protection from polio (paralytic disease), but only partial protection from subsequent infection and participation in transmission, depending on the nature of the induced immunity (i.e., IPV-induced vs. LPV-induced or both) and time since the last exposure (i.e. waning stage). For convenience, we refer to individuals with any IPV- or LPV-induced immunity as partially infectible, which distinguishes them from fully susceptible individuals, and we refer to the individuals in both groups collectively as infectible.

Tables 1 and 2 summarize key inputs related to the DEB model that remain constant across all populations or vary as indicated. For the 8 immunity states, the model assumes different levels

of relative susceptibility to (re)infection (σ) by serotype (see Table 2). Conceptually, the model uses transition rates (e.g., aging rates) to determine the proportion of one group that transfers to another group per unit of time. We assume a quarter of a year (i.e., 365/4 days) as the average time for infants in the maternal immunity state to become fully susceptible (ρ_{MI}, in days), and a 7-day delay (φ) [1] between the receipt of an IPV dose and the development of an immune response that moves the individuals to the next IPV immunity state.

Generally, deterministic transmission models assume that the durations of the latent and infectious periods distribute exponentially (i.e., they equal the reciprocal of the transition rates of leaving these states). Due to the memory-less nature of exponential distributions, this assumption makes the rate of leaving a state independent of the time previously spent in this state. Although this assumption violates the true nature of infections at the individual level (i.e., on average most people clear infections after many days and few do so after one day of infectiousness), the population sizes change according to these rates due to the homogeneous mixing and continuous divisibility of the population. We characterize infection with an LPV using multiple stages to ensure more appropriate behavior in this type of model (i.e., gamma distributions for overall durations of infectiousness instead of exponential distributions) [17]. In addition, we consider both latent and infectious periods of the infection such that the model separately tracks 6 oropharyngeal and 6 fecal infection stages (i.e., 2 latent (r) and 4 infectious (s) stages each, with varying degrees of infectiousness). This approach results in appropriately different dynamics for oropharyngeal and fecal-oral transmission. We assume a 3-day latent period for fecal-oral (ξ^{fec}) or oropharyngeal (ξ^{oro}) transmission (each split into 2 equal stages). We assume different durations of infectiousness for fecal-oral transmission (γ^{fec}, in days) and different relative fecal infectiousness (π^{fec}) for each serotype and recent immunity state (see Table 2). Similarly, we also assume different durations of oropharyngeal infectiousness (γ^{oro}, in days) and relative oropharyngeal infectiousness (π^{oro}) for the recent immunity states, but we assume no serotype differences for this route (see Table 2). Finally, we assign different relative weights to the 4 stages of infectiousness compared to the average weight over the infectious period (θ_k, k=0, …, r+s-1, in this case r+s-1=2+4-1=5) (see Table 1).

To characterize waning, we assume 5 stages of waning immunity (nw) divided evenly according to the assumed average time required to reach the last waning stage (ρ, in days), for which we assume 4 years (i.e., 4x365 days) for serotypes 1 and 2, and 3 years (i.e., 3x365) for serotype 3. We calculate waning rates for all waning stages except the last one. The waning rate equals $(nw-1)/\rho$. Table 2 lists the assumptions for each immunity state for the relative susceptibility (σ) for the last waning stage, for which we assume no serotype differences, the duration of fecal infectiousness (γ^{fec}, in days) and relative fecal infectiousness (π^{fec}) of the last waning stage by serotype, and the duration of oropharyngeal infectiousness (γ^{oro}, in days) and relative oropharyngeal infectiousness (π^{oro}) of the last waning stage for which we assume no serotype differences. We calculate σ, γ, *and* π of a given immunity state i and waning stage w using the formulas:

$$\sigma_{i,w} = \sigma_{i,nw-1} - (\sigma_{i,nw-1} - \sigma_{i,0}) \times ((nw-1-w)/(nw-1))^z,\ w = 1,\ldots,\ nw\text{-}1,$$

$$\gamma_{i,w,e} = \gamma_{i,nw-1,e} - (\gamma_{i,nw-1,e} - \gamma_{i,0,e}) \times ((nw-1-w)/(nw-1))^z,\ w = 1,\ldots,\ nw\text{-}1,$$

$\pi_{i,w,e} = \pi_{i,nw-1,e} - (\pi_{i,nw-1,e} - \pi_{i,0,e}) \times ((nw - 1 - w)/(nw - 1))^z$, $w = 1,\ldots, nw\text{-}1$,
where e represents the mode of transmission (fecal-oral or oropharyngeal) and z_w represents the shape parameter.

To model the process of infection, contacts between individuals in infected and infectible states determine the proportion of the infectible people that becomes infected each Δt. The infection transmission rate depends on the (weighed) number of infectious people in the population and a proportionality constant (β), which in turn relates directly to the basic reproductive number (R_0). The R_0 represents a theoretical summary measure of transmissibility defined as the average number of secondary infections caused by the introduction of one infectious person into an entirely susceptible population. We include seasonality by oscillating R_0 according to a sine function characterized by the average R_0 (R_0^{ave}), a peak day (pd), and an amplitude (α) using the following formula:

$$R_0(t) = R_0^{ave}\left(1 + \alpha \times \sin\left(\left(t - \frac{pd}{365}\right) \times 2\pi + \frac{\pi}{2}\right)\right).$$

To simplify the equations, we calculate β using the average net duration of the infectious period taking into account mortality (rather than the full expression of a multi-stage infection process [37]), using the following formula:

$$\beta(t) = (1/\gamma + \mu^{ave})R_0(t),$$

where γ represents the average duration of infection for a fully susceptible individual and μ^{ave} represents the average effective mortality rate.

We calculate the force of infection, defined as the rate at which individuals acquire an infection, using the formula:

$$\lambda(t) = EPI\ (t) \times \beta,$$

where *EPI* represents a shorthand notation for the effective proportion of infectious individuals. To include the effect of age mixing on the force of infection, we calculate the prevalence of infection weighted by the relative contribution to transmission of individuals by immunity state for each virus strain and each mixing age group [3]. Here, EPI depends on the mode of transmission (fecal-oral or oropharyngeal) and the relative infectiousness in each infection stage for individuals depending on their prior immunity state. Therefore, the force-of-infection from a given virus strain to an individual in mixing age group a_m equals:

$$\lambda_{a_m}(t) = \beta \sum_{b_m=1}^{na_m-1} EPI_{b_m}\ (t)M(a_m, b_m)(t),$$

where, na_m represents the number of mixing age groups and the mixing matrix M does not depend on the virus strain [3].

For any outbreak, we consider those individuals who acquired an infection during the outbreak as fully protected from re-infection with the outbreak virus and remove them from participation

in transmission during the outbreak after they recover from their infection and transition out of an infectious state.

We demonstrate the multistage processes for infectiousness and waning by introducing a group of individuals into the first stage of a given process in the model, following their progression through the multiple stages, and recording how their average properties change from the assumed to implied levels. In Figures A2 to A5 we compare the implied distributions in the model (without the effect of mortality or any other flows that depend on the population-specific situation, e.g., vaccination) to our assumptions for discrete stages in the process from the expert assessment [16]. The infectiousness curves (Figures A2 and A3) and waning curves (Figure A4) focus on serotype 1 only, because serotypes 2 and 3 yield similar values, with the exception that the waning curves for serotype 3 follow the same patterns but they increase more rapidly due to the assumed shorter time to reach the last waning stage (see Table 1).

Figure A2 presents the distributions for the duration of fecal and oropharyngeal infectiousness implied by the 2 latent and 4 infectious stages in the model. Figure A3 shows the average level of infectiousness for a population infected at time 0 with two sets of assumptions: constant vs. empirically fitted relative infectiousness (ratios of 1:1:1:1 vs. 3:10:3:1 by infectious stage, respectively). We rescaled the curves in Figure A3 (the areas under the curves remain fixed) for the comparison (i.e., the relative infectiousness compared to fully susceptible individuals ($\pi_{i,w,e}$) and differences in duration of infectiousness ($\gamma_{i,w,e}$) lead to more variation between immunity states in the unscaled infectious curves). The better match of varied levels of infectiousness by infectious stage to the expert assessments motivated our assumptions about infectiousness over time (i.e., 3:10:3:1 x 4 = 12:40:12:4, and 3+10+3+1 =17, leading to 12/17, 4/17, 12/17, and 4/17, for the 4 stages of infectiousness, respectively, as shown in Table 1).

Figure A4 characterizes waning by showing the average relative contribution to transmission compared to fully susceptible individuals, which we calculate as a product of relative susceptibility, relative duration infectiousness, and relative infectiousness by immunity state and as a function of time since entering that immunity state without further infection or vaccination. The expert assessment process yielded significant uncertainty about waning, and consequently we considered a very wide range for the input values [16]. During the model fitting process, we found that the multistage waning process produces slightly slower implied waning than suggested by the average of the experts, but the results converge to the model input values due to steady-state error [18].

A2.4. OPV evolution and paralytic case incidence

In addition to transitions between immunity states and waning, the model tracks OPV evolution through 20 successive reversion stages (h), from stage 0, which represents Sabin OPV infection, to stage 19 (h-*1*), which represents a fully-reverted poliovirus (FRPV, equivalent to WPV or cVDPV). An OPV-related virus in any stage can transmit in the population and lead to paralysis in fully susceptible individuals. For each serotype, we assume different average times to reach the last reversion stage (ε, in days) [10], different paralysis-to-infection ratios (PIRs) for fully susceptible individuals infected with OPV (PIR_0), and PIRs for fully susceptible individuals

infected with FRPV ($PIR_{h\text{-}1}$), and we also assume a relative PIR for maternally immune infants compared to fully susceptible individuals ($RPIR_{MI}$) (Table 1).

We assume that the relative R_0 values compared to homotypic WPVs (τ) increase with the reversion stage, until they reach the R_0 equivalent to homotypic WPVs at the last reversion stage (stage 19). Table 1 provides our assumptions about the relative R_0 of OPV vs. FRPV (τ_0) by serotype and the relative R_0 for serotypes 2 and 3 compared to serotype 1. We assumed a linear increase by reversion stage for τ input values, which we modeled via the multistage process that leads to more gradual effective values as it approaches the maximum input. We calculate the R_0 of the different reversion stages using the formula:

$$R_{0,j}(z_r) = \begin{cases} \tau_0 R_{0,h}, \ j = 0 \\ R_{0,h-1} - (R_{0,h-1} - R_{0,0}) \times ((h-1-\mathrm{j})/(h-1))^{z_r}, \ j = 1, \dots, h-1' \end{cases}$$

where $R_{0,h-1}$ represents R_0 equivalent to homotypic WPVs at the last reversion stage, and z_r represents the shape parameter.

While we focus on modeling transmission, the model tracks and counts paralytic cases. Similar to the behavior with R_0, we assume that the PIR increases with the reversion stage, until it reaches the value of the homotypic WPV at the last reversion stage (stage 19). As a result of infection, fully susceptible and maternally immune individuals can become a recipient VAPP case when infected with stage 0 virus (OPV) during vaccination, a non-recipient VAPP case due to infection with a virus of any reversion stage except FRPV (i.e., OPV-related viruses), or a cVDPV case or WPV case when infected with stage 19 virus (FRPV). We assumed an S-shaped function with the log transformation after averaging the natural-scale of PIR for each model stage. We assume the lowest PIR for serotype 1 OPV, and the highest PIR for serotype 1 FRPV (compared to serotypes 2 and 3. This leads to a relatively higher effective PIR for serotype 1 compared to serotype 3 (or serotype 2), measured in Figure A5 as approximately 85 (or 32) days older or more diverged for serotype 3 (or 2). We calculate the PIR of a given reversion stage using the formula:

$$PIR_j(z_p) = \exp(PIR_{h-1} - (PIR_{h-1} - PIR_0) \times ((h-1-j)/(h\ -1))^{z_p}), \ j = 1, \dots, h-1,$$

where PIR_{h-1} represents the PIR for the homotypic WPV (i.e., at the last reversion stage), and z_p represents the shape parameter. We compute the incidence of paralytic cases ($PP_{a,j}$) in a age group a due to virus strain j from the force-of-infection as follows:

$$PP_{a,j}(t) = \lambda_{a,j}(t) PIR_j\left(\sigma_{1,0} PI_{0,1,0}(t) RPIR_{MI} 1_{\{a=0\}} + PI_{a,0,0}(t) RPIR_a^{age}\right).$$

Figure A5 shows the inputs by discrete reversion stage, with dots indicating the values of the 20 reversion stages, compared to the results of the multistage process for τ and PIR as a function of the virus age. The differences between the input values and the results occur due to the dispersion of values during viral progression through the stages for both τ and PIR.

A2.5. Immunization

We model immunization using two mechanisms: (i) the effective vaccination *coverage* (*evc*) for RI and (ii) the effective vaccination *rate* (*evr*) for SIAs. The *evc* describes continuous activities that target individuals as they reach specific ages according to an age-dependent immunization schedule. The *evr* describes pulse (focused in time) activities targeting individuals in specified age groups.

RI

In the model, we assume that RI occurs at fixed ages that correspond to age groups (e.g., birth, exactly 3 months), driven by the vaccination schedules (i.e., OPV-only, OPV+IPV, IPV/OPV, IPV-only). We use effective vaccination coverage (*evc*) of OPV and IPV (evc^{OPV} and evc^{IPV}) to direct flows of routinely vaccinated individuals from their current immunity state to the next one according to the schedule, coverage, and vaccine -used as a function of time and the appropriate "take" rate for the vaccine in that population (see Table A3). We assume that any child immunized by RI effectively takes either with OPV or IPV, but not to both at the same time ($evc^{\mathrm{OPV}} - evc^{\mathrm{IPV}} \leq 1$), and we allow individuals who receive OPV+IPV (simultaneously) to first take the OPV dose and those who remain susceptible to potentially take the IPV dose. We assume that IPV take rates (tr^{IPV}) incorporated into the calculation of the evc^{IPV} account for any effect of maternal antibodies on the per-dose take rate, while OPV take rates (tr^{OPV}) do not. Thus, we multiply the evc^{OPV} by relative susceptibility for maternally immune infants who receive OPV at birth. We assume that IPV boosts already primed or immune individuals in other immunity states at the same rate as fully susceptible individuals.

In the model, a birth dose of OPV directs a proportion (evc^{OPV}) of fully susceptible or maternal immune newborns into the first latent LPV stage of the first age group as they enter that age group. For OPV-only or OPV+IPV RI schedules, the model uses the effective RI coverage to move a fraction evc^{OPV} of the aging flow of infants in the first age group in all immunity states except maternally immunes into their first latent LPV stage of the second age group. For an IPV-only schedule, the effective RI coverage comes in upon aging of infants from the first to the second age group, and moves a fraction evc^{IPV} of the aging flow of all immunity states except maternally immunes into their IPV-exposed (IPVE) state, which represents the state corresponding to the brief period (average duration φ) after receipt of IPV before the recipient benefits from full protection from disease and acquires all of the attributes of the next IPV state. . For countries that use an IPV/OPV sequential schedule, for simplicity the model delivers the IPV doses to the first age group, and the OPV doses to the second age group. This effectively protects all infants that would take according to this schedule from paralysis. For all RI schedules, the remaining unvaccinated fraction $1-evc^{OPV}-evc^{IPV}$ follows the regular aging path into the next age group of the same immunity state. For the maternally immune infants who receive a routine dose at 3 months, the fraction evc^{OPV} (or evc^{IPV}) of the aging flow moves into the first latent LPV stage (or into their IPVE state) of the next age group for previously fully susceptible individuals, without any effects of residual maternal immunity on "take" (i.e., we do not multiply by relative susceptibility).

We characterize the effective vaccination coverage of an OPV-only schedule as:
$evc_0^{OPV} = relbd \times POL3 \times tr_1^{OPV}$ at birth and

$evc_1^{OPV} = POL3 \times tr_3^{OPV} + (1 - POL3) \times COV1or2 \times tr_1^{OPV} + (1 - POL3) \times COV1or2 \times tr_2^{OPV}$ at 3 months,
where $tr_x^{OPV} = 1 - (1 - tr^{OPV})^x$ is the cumulative take rate after *x* doses, *relbd* is the relative coverage with birth dose compared to non-birth RI coverage with 3 doses, *COV1or2* is the RI coverage with 1 or 2 non-birth doses and POL3 is the RI coverage with 3 or more non-birth doses.

We characterize the effective vaccination coverage of the OPV+IPV schedule as the OPV-only schedule using:
$evc_0^{OPV} = relbd \times POL3 \times tr_1^{OPV}$ at birth and
$evc_1^{OPV} = POL3 \times tr_3^{OPV} + (1 - POL3) \times COV1or2 \times tr_1^{OPV} + (1 - POL3) \times COV1or2 \times tr_2^{OPV}$ at 3 months,
and
$evc_1^{IPV} = (1 - POL3) \times COV1or2 \times (tr_1^{OPVPV} - tr_1^{OPV}) + (1 - POL3) \times COV1or2 \times (tr_2^{OPVPV} - tr_2^{OPV}) + POL3 \times tr_3^{OPVIPV}$ at 3 months,
where $tr_x^{OPVIPV} = 1 - (1 - tr_d^{OPV}) \times (1 - tr^{IPV})$ is the take rate for a simultaneous OPV and IPV third-dose.

We characterize the effective vaccination coverage of the IPV/OPV sequential schedule as:
$evc_0^{IPV} = evc_0^{IPV}(IPV1) + evc_0^{IPV}(IPV2)$ at birth, where
$evc_0^{IPV}(IPV1) = (1 - POL3) \times COV1or2 \times tr^{IPV} + 2 \times (POL3 + (1 - POL3) \times \mathrm{COV1or2}) \times (1 - tr^{IPV}) \times tr^{IPV}$ and $evc_0^{IPV}(IPV2) = (POL3 + (1 - POL3) \times COV1or2) \times tr^{IPV} \times tr^{IPV}$ and $evc_1^{OPV} = POL3 \times tr_2^{OPV}$ at 3 months.

We characterize the effective vaccination coverage in an IPV-only schedule as:
$evc_1^{IPV} = evc_1^{IPV}(IPV1) + evc_1^{IPV}(IPV2) + evc_1^{IPV}(IPV3)$ at 3 months, where
$evc_1^{IPV}(IPV1) = (1 - POL3) \times COV1or2 \times tr^{IPV} + 2 \times (1 - POL3) \times COV1or2 \times (1 - tr^{IPV}) \times tr^{IPV} + 3 \times POL3 \times (1 - tr^{IPV}) \times (1 - tr^{IPV}) \times tr^{IPV}$, $evc_1^{IPV}(IPV2) = (1 - POL3) \times COV1or2 \times tr^{IPV} \times tr^{IPV} + 3 \times POL3 \times (1 - tr^{IPV}) \times tr^{IPV} \times tr^{IPV}$, and $evc_1^{IPV}(IPV3) = POL3 \times tr^{IPV} \times tr^{IPV} \times tr^{IPV}$.

SIAs

In the model, SIAs move individuals in a targeted age group to the appropriate group of partially infectible individuals at a rate determined by the one-dose take rate, coverage, and duration of the SIA. We developed a characterization of SIAs that allows direct specification of the true coverage of individual SIA rounds, the probability of children repeatedly receiving or missing doses, extraction of the number of administered doses, and the number of distributed doses after accounting for wastage (i.e., the fraction of doses distributed to the field, but is not administered). Our approach focuses on conditional probabilities of receiving a SIA dose, which only depends on receiving a dose in the previous round. Specifically, we define:

- true coverage (*TC*) of a SIA round as the fraction (between 0 and 1) of the targeted population that receives a dose in a given round, calculated as $TC = ND \times (1 - w)/N$, where *N* equals the target population size, *ND* equals the number of distributed does, and *w* equals the wastage factor,

- repeated missed probability (P_{RM}) as the conditional probability that a targeted individual does not receive a dose in a round, conditional on the individual not receiving a dose in the previous round despite falling into the targeted population for that round,
- repeated reached probability (P_{RR}) as the conditional probability that a targeted individual receives a dose in a round, given the same individual received a dose in the previous round.

Since P_{RM} and P_{RR} must together produce the *TC*, the ability to describe any two of these three inputs leads to sufficient characterization of an SIA of two consecutive rounds. Figure A6 shows a probability tree of receiving a dose in two subsequent rounds, representing the fraction of targeted individuals who: b1, receive a dose in two consecutive rounds; b2, receive a dose in the first but not in the second round; b3, receive a dose in the second but not in the first round; and b4, do not receive a dose in either round. Taking *TC1* and *TC2* as the true coverage of rounds 1 and 2, respectively, the total fraction receiving a dose in round 2 equals:
$TC2 = b1 + b3 = TC1 \times P_{RR} + (1 - TC1) \times (1 - P_{RM})$,
where P_{RR} and P_{RM} both relate to round 2. Therefore:

$$P_{RR} = (TC2 - (1 - TC1) \times (1 - P_{RM}))/TC1.$$

In order to use this characterization in the model, we track the population fraction receiving a dose in the most recent round from each immunity state. However, for simplicity, we do so only for individuals who did not yet acquire active immunity (i.e., fully susceptible and maternally immune individuals (FSMI)). We divide all FSMIs within the target age range into three categories:

- new children fraction (*ncf*) - fraction of all targeted children born after the previous SIA round who receive a dose in the current round with probability *TC*.
- reached children fraction (*rcf*) - fraction of all targeted children who received a dose in the previous SIA round but remained FSMI due to failure to take and who receive a dose in the current round with probability P_{RR}.
- missed children fraction (*mcf*) - fraction of all targeted children who did not receive a dose in the previous SIA round and who receive a dose in the current round with probability $1\text{-}P_{RM}$.

We use average coverage for all FSMIs (cov_{FSMI}) to determine the vaccination rates for all targeted FSMIs, where:

$$cov_{FSMI} = TC \times ncf + P_{RR} \times rcf + (1 - P_{RM}) \times mcf.$$

To calculate *ncf*, *rcf*, and *mcf* (by age), we track age-dependent stocks of new fully susceptible ($NFS_a(t)$) and new maternally immune ($NMI_a(t)$) individuals (bound by the same in- and outflows as corresponding fully susceptible (FS_a) and maternally immune (MI_a) individuals in the model), and we accumulate new FSMIs from newborns once any SIA round finishes. Consequently, at the beginning of the current SIA (t_{curr}), the fraction *NC* in age group *a* equals
$ncf_a = (NFS_a(t_{curr}) + NMI_a(t_{curr}))/(FS_a(t_{curr}) + MI_a(t_{curr}))$.

To distribute remaining *FSMI*s into *mcf* and *rcf,* we track those fractions from the previous round. Since *mcf* and *rcf* are part of fully susceptible or maternally immune individuals (bound

by the same fractional outflows between subsequent rounds), the fractions remain intact. Therefore, for age group *a,* the appropriate take rate for the vaccine used during the previous round (*tr*), and the relative susceptibility of the respective immunity state (σ_i) fractions equal: $mcf/(\text{mcf} + rcf) = (1 - cov_{FSMI})/(1 - tr \times cov_{FSMI})^{\sigma_i}$ and $rcf/(\text{mcf} + rcf) = 1 - mcf/(\text{mcf} + rcf)$.

We compute the coverage for all individuals with actively-acquired immunity (cov_{Imm}) based on the condition that the overall SIA coverage equals *TC*, thus for the fully susceptible or maternally immune proportion (*fsmi*) of the target population:

$$cov_{Imm} = (TC - fsmi \times cov_{FSMI})/(1 - fsmi).$$

We calculate effective vaccination rates for FSMIs as:

$$evr_{FSMI} = -\ln(1 - cov_{FSMI} \times tr)/d$$

and for immune individuals as:

$$evr_{Imm} = -\ln(1 - cov_{Imm} \times tr)/d,$$

where *d* represents duration of the SIA. The effective vaccination rates change over time according to the dates and assumed *TC* and P_{RM} for each SIA, but remain constant for the duration of each round. The model uses the same mechanics to implement both preventive or planned SIAs (pSIAs) and reactive outbreak response SIAs (oSIAs).

Incidence of paralytic cases due to OPV immunization

We compute recipient VAPP incidence from the sum of all OPV infections in fully susceptible and maternally immune individuals due to evc_a^{OPV} and evr_a^{OPV} and using PIR_0. For vaccine given at time of aging from maternally immune to fully susceptible (i.e., at 3 months of age), we assume the relative PIR for maternally immunes still applies.

A2.6. Schematic of progression through infection and reversion stages

For LPV infections, individuals from immunity state *i* and age group *a* ($PI_{a,i}$) move through the different stages of infection according to their relative susceptibility (σ_i) and the force of infection of age group *a* for the virus strain *j* ($\lambda_{a,j}$) they receive. All individuals infected with a LPV progress through both oropharyngeal and fecal infection paths, and while infected they can infect other individuals due to their oropharyngeal and/or fecal-oral poliovirus excretion. Specifically, we assume individuals with a fecal infection also become infectious via oropharyngeal excretions (i.e., relative susceptibility to oropharyngeal infection depends directly on relative susceptibility to fecal infection). As shown in Table 2, we assume higher rates of infectiousness for fecal excretion than oropharyngeal excretion in most immunity states by assuming longer durations and higher relative infectiousness compared to fully susceptible individuals for fecal than oropharyngeal infections. However, these assumptions do not preclude the possibility of oropharyngeal transmission representing the dominant route of transmission in

some settings or subpopulations, because the model assumes situation-specific proportions of transmission via the oropharyngeal route (p^{oro}) and fecal-oral route ($1- p^{oro}$). The force-of-infection by excretion mode accounts for differences in duration using the following:

$$\lambda(t) = \sum_{e=0}^{1} EPI(t)p_e\left(1/\gamma_{0,0,e} + \mu^{ave}(t)\right)R0_j(t),$$

where p_e represents the proportion of transmission via excretion mode e, and $\gamma_{0,0,e}$ represents the average duration of infection for a fully susceptible individual excreting via mode e. The relative infectiousness only accounts for disproportionate effects of immunity on each excretion mode for each immunity state relative to fully susceptible individuals. We model the oropharyngeal infection process as a "co-flow" [18] to preserve the correct population size (i.e., we do not take oropharyngeal infections out of the stock *PIi* or let them recover into the next LPV state, and we do not double count individuals in these states in the population). While individuals remain fully protected from homotypic reinfection while still fecally infectious to others, they become partially infectible again according to the relative susceptibility of the next LPV state that they enter after they recover from their fecal infection. IPV vaccinated individuals from immunity state i and age group a ($PI_{a,i}$) move to the appropriate IPV-exposed state ($IPVE_{a,i}$), which represents the state corresponding to the brief period (average duration φ) after receipt of IPV before the recipient benefits from full protection from disease and acquires all of the attributes of the next IPV state.

Figure A7 provides a schematic of how infected or effectively OPV vaccinated individuals move through different stages of infection, including infection with OPV-related viruses that transmit in populations that use OPV and become immune following successful IPV vaccination. For simplicity, Figure A7 uses v_a^{ipv} (or v_a^{opv}) to indicate the force of IPV (or OPV) vaccination to age group a, which includes both RI and SIAs, although the model separately accounts for delivery of doses through both mechanisms and for the probability of take of the vaccine as appropriate [16, 19]. Figure A7 shows how IPV vaccinated individuals from immunity state i and age group a ($PI_{a,i}$) move to their respective $IPVE_{a,i}$ according to the v_a^{ipv} on the left. As noted above, the $IPVE_{a,i}$ represents the brief period (average duration φ) after receipt of IPV before movement into the next IPV state and full protection from disease. With oropharyngeal transmission at the top and fecal-oral transmission at the bottom, Figure A7 shows how individuals infected with an LPV move from immunity state i and age group a ($PI_{a,i}$) through different stages of infection according to their relative susceptibility (σ_i), the force of infection of age group a and virus strain j ($\lambda_{a,j}$), and the v_a^{opv}. OPV infections can occur as a result of contact with other OPV infectious individuals ($\lambda_{a,0}$) and/or through receipt of vaccine according to the v_a^{opv}, or as a result of contact with LPV infectious individuals ($\lambda_{a,j}$, $j = 1, \ldots, h$). The progression of individuals infected with a LPV through fecal or oropharyngeal infection paths (i.e., $FI_{a,i,j,1}$ and $OI_{a,i,j,1}$ respectively) shows movement through infection stages according to rates indicated by the number of latent (r) and infectious (s) stages, and average duration of the fecal (oropharyngeal) latent and infectious periods for immunity state i, ξ_i^{fec} (ξ_i^{oro}) and γ_i^{fec} (γ_i^{oro}) respectively. Individuals move through reversion stages according to rates indicated by number of reversion stages (h) and average time to reach last reversion stage (ε). We assume that individuals remain fully protected from reinfection while still infectious to others. After recovering, individuals enter the next LPV immunity state and acquire all attributes of that state.

A2.7. Die-out, thresholds and importations

In the DEB model, which tracks the individuals in populations by accounting for the fraction of the population in each immunity state, the model allows for fractional individuals, which can imply less than one person infected as infections die-out. To simulate die out in the DEB, we assume that the force-of-infection goes to 0 in a population when the effective infectiousness-weighted proportion of the population infectious with that poliovirus drops below 5 per million people (i.e., the transmission threshold EPI^*) (Table 1) [3]. Although actual die-out behavior depends on chance and local heterogeneity, this simplified approach reproduced WPV die-out times consistent with observations in a broad range of settings [3, 10, 20, 21]. We model die-out by setting the force-of-infection for the virus strain and mixing age group to 0.

To model importations (introductions), we move a fraction equal to the EPI* (see Table 1) of all fully susceptible individuals in each age group to the first infectious stage, increasing the effective prevalence of infections of the population (EPI). If the fraction of fully susceptible individuals in an age group is less than EPI*, we move a fraction of individuals in each immunity state i equal to EPI* divided by the relative contribution to combined fecal-oral and oropharyngeal transmission for the immunity state i to the first infectious stage for that immunity state [3, 16, 22].

A2.8. Surveillance triggers for outbreak response

We model AFP surveillance by accumulating the incidence of polio cases in each subpopulation resulting from (i) effective importations or indigenous cVDPV emergences before serotype-specific OPV cessation or (ii) any LPV (i.e., all OPV-related viruses) after serotype-specific OPV cessation. Once the cumulative incidence per 10 million people reaches more than the subpopulation-specific detection threshold (i.e., 1, 2, 3, … polio cases, depending on the population-specific conditions), we trigger outbreak response. After the completed outbreak response, the model clears the cumulative detected incidence and restarts the accumulation of polio cases until any new detection occurs, or every 6 months without any outbreak response.

We model the probability of finding poliovirus in a sewage sample by ES (P_{ES}) using a "system-wide" approach [23], for which we describe the probability of detecting poliovirus in any sampling site in the subpopulation given the total catchment percent of the population covered by ES sites (tcp), the ES quality (Q_{ES}), and the effective prevalence of infections (EPI), using formula $P_{ES} = tcp^{-Q_{ES} \times ln(EPI)}$.

A2.9. Calibration of the generic model inputs

We calibrated the generic model inputs by fitting the model to the observed data for a wide range of epidemiological situations. The DEB model needs to appropriately estimate the incidence of paralytic cases of each serotype, the die-out of live poliovirus transmission, and the development of cVDPVs in places where they occur but no development of them where they do not occur.

Taken directly from prior work [3, 21], Figure A8 provides the model results (with the generic model inputs from Table 1 and Table 2 fixed) compared to the actual experience from multiple

diverse settings. Specifically, we compared the behavior of the model to: (i) data for northern India (modeled separately for Western Uttar Pradesh (WUP) and Bihar) and for northwest (NW) Nigeria for children with non-polio acute flaccid paralysis who reported no receipt of OPV; (ii) data on polio incidence and die-out of endemic WPV transmission for all situations and serotypes (WPV1 and WPV3 results for northern India and northwest Nigeria and for all 3 WPV serotypes in the USA shown in Figure A8); (iii) data from the Netherlands, Tajikistan, and Albania for WPV importation outbreak behavior and (iv) the age distributions of cases (also done for all other situations with appropriate data, not shown); (v) serological data for the USA (shown in Figure A8) and Cuba (not shown) on the effect of secondary OPV immunity; (vi) indigenous emergence of cVDPVs (shown in Figure A8 for serotype 2 for northern India and NW Nigeria and serotype 1 for Haiti and Madura, Indonesia; (vii) no indigenous emergence of cVDPVs in all other situations and serotypes (Figure A8 shows die-out of serotype 1 OPV-related viruses for Cuba and Haiti); and (viii) reproduction of asymptomatic transmission (detected by ES) of an imported WPV1 into Israel in 2013. The model consistently approximates the dynamics and interruption of live poliovirus transmission [3, 10, 20, 21]. In addition, the model simulates the persistence of OPV-related viruses and their evolution to fully transmissible and neurovirulent cVDPVs, and shows cVDPV outbreaks for conditions in which they occurred (e.g., in Hispaniola [24] and Nigeria [25]) and no cVDPV outbreaks for settings where they did not occur despite OPV use and cessation (e.g., in Cuba [26] and the USA [27]) [3].

A2.10. Limitations

As with all models, the choice of model structure affects the results. All DEB models come with inherent challenges in characterizing aggregate-level behavior, which can lead to unrealistic distributions and/or steady-state errors implicit in aging chains and delay processes that ignore arrival times in stocks. Random timing and numbers of transitions between stocks due to stochastic variability in the populations represented by a stock occur around their population averages (i.e. variability within stocks) and uncertainty exists about these averages. The fractional rate-based processes in DEB models that drain stocks mean that these stocks cannot go to absolute zero, while in the real world some stocks can deplete completely (e.g. populations can reach zero prevalence of an infection and inventories can become completely empty). Our use of a transmission threshold as the criterion for die-out rather than absolute 0 total infected individuals represents a simplified construct of the complex dynamics of transmission die-out.

The model uses simplified realistic populations to show the spectrum of possible outcomes and demonstrate the effect of changing population-specific assumptions, but real populations include more heterogeneity and more complicated poliovirus exposure and vaccination histories. Our consideration of a limited number of subpopulations represents a simplification of the complex and changing mixing patterns and heterogeneity that exists in the real world and in real populations. The way that we characterize the importance of the model construct of an under-vaccinated subpopulation may only partially capture some of the heterogeneity that exists in the population. The choice of the number of stages for OPV evolution influences the flows between reversion stages and the timing of when the prevalence in an individual reversion stage drops below the transmission threshold due to transitions between reversion stages. Our assumptions about a multi-stage infection process with variable infectiousness for each infection stage also

affects the kinetics of prevalence and die-out. Random events play a role in OPV evolution and the emergence of cVDPVs, and the DEB model does not account for micro-level dynamics that impact transmission in real populations.

Despite careful calibration of the generic model, other model structures and inputs could potentially reproduce the uncertain real-world values of inputs and the epidemiological experience and evidence equally well or better. We do not perform additional uncertainty or sensitivity analyses that change any of the assumptions of the poliovirus transmission and OPV evolution model, because this would reduce its consistency with observed behavior in the modeled specific situations unless we recalibrate the entire DEB model. Recalibration of the entire DEB model for each new set of input assumptions for such an exercise would take a prohibitively long amount of time. We previously performed extensive sensitivity and uncertainty analyses that inform our understanding of the inputs that matter for policy [16, 28, 29] and we have and will continue to perform specific input- and policy-specific analyses to address these topics [10, 30-34].

Due to limited experience with IPV in settings that allow fecal-oral transmission, significant uncertainty remains about the impact of IPV-induced immunity on asymptomatic fecal-oral poliovirus transmission. Our model assumes significantly lower potential to participate in poliovirus transmission for those with LPV-induced compared to those with IPV-only-induced immunity. We remain somewhat uncertain about the impact of IPV-only on poliovirus transmission in different settings, the extent to which waning of immunity and the relatively simple age-mixing structure affects transmission, the uncertain speed of OPV evolution within populations, and the ability of the model to capture die-out.

A2.11. Equations

This section provides the list of indices and symbols used in the equations of the generic DEB model used to characterize poliovirus transmission and OPV evolution as introduced in our prior work and repeating some of the symbols defined above [3, 31]. The indices used in this section may differ from those used above, because the previous sections presented general concepts that simplified or omitted some concepts and/or model components. This section provides the full equations that account for all of the model components.

Indices:

a = situation-specific age group ($a = 0, \ldots, na-1$, where na depends on the situation and age group 0 is always from 0-2 months, inclusive; note that maternally immunes only exist in age group 0)
a_m = mixing age group ($a_m = 0, \ldots, na_m-1$, where typically $na_m = 3$ with $a_m = 0$ (0-4 years), 1 (5-14 years), 2 (15 or more years))
$A(a)$ = mixing matrix age group that situation-specific age group a belongs to (i.e., $A(a) = a_m$ if situation-specific age group a belongs to mixing age group a_m)
$c(a_m)$ = cut-off for mixing age group a_m = first situation-specific age group included in mixing age group a_m, where we define $c(na_m) = na_m$
e = excretion and transmission mode ($e = 0$ (fecal) or 1 (oropharyngeal))

fa = first situation-specific age group for which we assume immunes can give birth to children with maternal immunity
i = immunity state (i = 0, …, ni, where ni = 8 and i = 0 (fully susceptible), 1 (maternally immune), 2 (1 successful IPV), 3 (2 successful IPV, 4 (≥ 3 successful IPV), 5 (1 LPV infection), 6 (≥ 2 LPV infections) ,7 (IPV and LPV))
j = virus strain (j = 0 (OPV), 1, …, h-2 (OPV-related), h-1 (FRPV), h (WPV), where h=20)
k = infection stage (k = 0 (first latent stage), r-1(last latent stage), r (first infectious stage), …, r+s-1 (last infectious stage), where r = 2 and s = 4)
la = last situation-specific age group for which we assume immunes can give birth to children with maternal immunity
w = waning stage (w = 0 (recent), …, nw -1, where nw = 5; note that fully susceptibles and maternally immunes only exists in waning stage 0)

Symbols for state variables:

$IPVE_{a,i,w}$ = successfully IPV-vaccinated individuals from immunity state i, age group a, and waning stage w that have not yet acquired the properties of the next IPV state (i.e., IPV-exposed individuals)
$LI_{a,i,w,j,k,e}$ = individuals from immunity state i, age group a, and waning stage w infected with live virus strain j and residing in infection stage k of excretion mode e (i.e., live-virus-infected individuals)
$PI_{a,i,w}$ = partially infectible individuals in immunity state i, age group a, and waning stage w

Other symbols:

α = seasonal amplitude of R_0 (for any strain) (i.e., difference from the average R_0 at peak or through, relative to the average R_0)
b = birth rate [1/(people × days)]
$\gamma_{i,w,e}$ = total duration of infectious period (in all infectious stages) for immunity state i, waning stage w, and excretion mode e [days]
$EPI_{a,j}$ = effective proportion infectious to situation-specific age group a with virus strain j
$EPIM_{am,j,e}$ = effective proportion infectious to mixing age group m_a with virus strain j with respect to excretion mode e
EPI^* = effective proportion infectious below which we assume 0 force-of-infection (i.e., the transmission threshold)
evc_a^{IPV} (evc_a^{OPV}) = effective vaccination coverage with IPV (OPV) = fraction of the population receiving an effective IPV (OPV) dose upon entering situation-specific age group a (i.e., a dose that takes if given to a fully susceptible individual)
evr_a^{IPV} (evr_a^{OPV}) = effective IPV (OPV) vaccination rate = fraction of the population in situation-specific age group a receiving an effective IPV (OPV) dose per day (i.e., a dose that takes if given to a fully susceptible) [1/day]
ε = average time to reach last reversion stage [days]
φ = IPV immunity delay [days]
θ_k = relative infectiousness weight of infection stage k
$\kappa(a_m)$ = proportion of potentially infectious contacts of individuals in mixing age group a_m reserved for individuals within the same mixing age group

$\lambda_{a,j}$ = force-of-infection to situation-specific age group a due to virus strain j [1/days]
mf = fraction of newborns born with maternal immunity
μ_a = fraction of people in situation-specific age group a that die (or emigrate) per day [1/days]
μ^{ave} = fraction of all people that die (or emigrate) per day [1/days]
$M(a_m,b_m)$ = mixing matrix containing normalized mixing coefficients for potentially infectious contacts of individuals in mixing age group a_m with individuals in mixing age group b_m
$MNI_{a,i}$ = inflow for partially infectible individuals in situation-specific age group a and immunity state i due to individuals moving to the next IPV state after leaving the IPVE state [people/days]
$MNL_{a,i}$ = inflow for partially infectible individuals in situation-specific age group a and immunity state i due to individuals moving to the next LPV state after recovering from infection [people/days]
N = total population size [people]
N_a = total population size in age group a [people]
NM_{a_m} = total population size in mixing age group a_m [people]
$\pi_{i,w,e}$ = relative infectiousness for immunity state i, waning stage w, and excretion mode e
$R0_j$ = R_0 for virus strain j (as a function of time)
$R0^{ave}_j$ = average R_0 for virus strain j based on functional form for relative R_0 by reversion stage (see methods)
p_e = proportion of transmissions via excretion mode e ($p_0 = 1\text{-}p^{oro}$ and $p_1 = p^{oro}$)
pd = seasonal peak day for R_0 [day number in each year]
ρ = average time to reach the last waning stage [days]
$\sigma_{i,w}$ = relative susceptibility for immunity state i in waning stage w
τ = relative R_0 of OPV vs. homotypic FRPV and WPV
w_a = width of age group a (with $w_0 = \rho_{MI} = 0.25\times365$ days (Table 1)) [days]
$\xi_{i,w,e}$ = duration of latent period for immunity state i, waning stage w, and excretion mode e [days]
z_p = shape parameter for the relationship between PIR and reversion stage
z_r = shape parameter for the relationship between R_0 and reversion stage
z_w = shape parameter of the waning functions

We note that the equations included here appear as introduced in our prior work [3, 31]. We use the characteristic function:

$$1_{\{c1,c2,\dots\}} = \begin{cases} 1, & c1, c2, \dots = TRUE \\ 0, & otherwise \end{cases}$$

and we omit dependence on serotype to simplify the equations. The next 3 equations give the full set of differential equations to model the flows in Figure A7.

$$\frac{dPI_{a,i,w}(t)}{dt} = 1_{\{a=0\}}b(t)N(t)\Big(1_{\{i=0\}}\big(1-mf(t)\big)\big(1-evc_0^{IPV}(t)-evc_0^{0PV}(t)\big)$$
$$+ 1_{\{i=1\}}mf(t)\Big(1-evc_0^{IPV}(t)-\sigma_{1,0}evc_0^{0PV}(t)\Big)\Big)$$
$$+ 1_{\{a>0\}}\Big(1-evc_{a-1}^{IPV}(t)-\sigma_{i,w}evc_{a-1}^{0PV}(t)\Big)\Big(PI_{a-1,i,w}(t)$$
$$+ 1_{\{a=1,i=0\}}PI_{0,1,0}(t)\Big)\frac{1}{w_{a-1}} + 1_{\{w=0\}}\Big(MNI_{a,i}(t)+MNL_{a,i}(t)\Big)$$
$$+ 1_{\{w>0,i>1\}}PI_{a,i,w-1}(t)\frac{nw-1}{\rho}$$
$$-\Bigg\{\mu_a(t)+\frac{1_{\{a<na-1\}}}{w_a}+\sum_{j=0}^{h}\sigma_{i,w}\lambda_{a,j}(t)+\sigma_{i,w}evr_a^{OPV}(t)+evr_a^{iPV}(t)$$
$$+ 1_{\{w<nw-1,i>1\}}\frac{nw-1}{\rho}\Bigg\}PI_{a,i,w}(t)$$

$$\frac{dIPVE_{a,i,w}(t)}{dt} = 1_{\{a=0\}}b(t)N(t)evc_0^{IPV}(t)\Big(1_{\{i=0\}}\big(1-mf(t)\big)+1_{\{i=1\}}mf(t)\Big)$$
$$+ 1_{\{a>0\}}\Big(\Big(1-\sigma_{i,w}evc_{a-1}^{OPV}(t)\Big)IPVE_{a-1,i,w}(t)$$
$$+ 1_{\{a=1,i=0\}}\Big(1-evc_0^{OPV}(t)\Big)IPVE_{0,1,0}(t)$$
$$+ 1_{\{i=0\}}evc_{a-1}^{IPV}(IPV1)(t)\Big(PI_{a-1,0,0}(t)+1_{\{a=1\}}PI_{0,1,0}(t)\Big)$$
$$+ 1_{\{i=2\}}evc_{a-1}^{IPV}(IPV2)(t)\Big(PI_{a-1,0,0}(t)+1_{\{a=1\}}PI_{0,1,0}(t)\Big)$$
$$+ 1_{\{i=3\}}evc_{a-1}^{IPV}(IPV3)(t)\Big(PI_{a-1,0,0}(t)+1_{\{a=1\}}PI_{0,1,0}(t)\Big)$$
$$+ 1_{\{i=2\}}evc_{a-1}^{IPV}(IPV1)(t)PI_{a-1,2,w}(t)$$
$$+ 1_{\{i=3\}}\big(evc_{a-1}^{IPV}(IPV2)(t)+evc_{a-1}^{IPV}(IPV3)(t)\big)PI_{a-1,2,w}(t)$$
$$+ 1_{\{i\geq 3\}}evc_{a-1}^{IPV}(t)PI_{a-1,i,w}(t)\Big)\frac{1}{w_{a-1}} + 1_{\{w>0,i>1\}}IPVE_{a,i,w-1}(t)\frac{nw-1}{\rho}$$
$$+ evr_a^{IPV}(t)PI_{a,i,w}(t)$$
$$-\Bigg\{\mu_a(t)+\frac{1_{\{a<na-1\}}}{w_a}+\sum_{j=0}^{h}\sigma_{i,w}\lambda_{a,j}(t)+\sigma_{i,w}evr_a^{OPV}(t)$$
$$+ 1_{\{w<nw-1,i>1\}}\frac{nw-1}{\rho}+\frac{1}{\varphi}\Bigg\}IPVE_{a,i,w}(t)$$

$$\frac{dLI_{a,i,w,j,k,e}(t)}{dt}$$
$$= 1_{\{a=0,j=0,k=0\}} b(t)N(t)evc_0^{OPV}(t)\left(1_{\{i=0\}}\left(1-mf(t)\right)+1_{\{i=1\}}\sigma_{0,1}mf(t)\right)$$
$$+ 1_{\{a>0\}}\Big(\big(LI_{a-1,i,w,j,k,e}(t)+1_{\{a=1,i=0\}}LI_{0,1,0,j,k,e}(t)$$
$$+ 1_{\{j=0,k=0\}}\sigma_{i,w}evc_{a-1}^{OPV}(t)IPVE_{a-1,i,w}(t)$$
$$+ 1_{\{a=1,i=0,j=0,k=0\}}evc_0^{OPV}(t)IPVE_{0,1,0}(t)+1_{\{j=0,k=0\}}\sigma_{i,w}evc_{a-1}^{OPV}(t)PI_{a-1,i,w}(t)$$
$$+ 1_{\{a=1,i=0,j=0,k=0\}}evc_0^{OPV}(t)PI_{0,1,0}(t)\Big)\frac{1}{w_{a-1}}$$
$$+ 1_{\{k=0\}}\left(1_{\{j=0\}}evr_a^{OPV}(t)+\lambda_{a,j}(t)\right)\sigma_{i,w}PI_{a,i,w}(t)$$
$$+ 1_{\{k=0\}}\left(1_{\{j=0\}}evr_a^{OPV}(t)+\lambda_{a,j}(t)\right)\sigma_{i,w}IPVE_{a,i,w}(t)$$
$$+ 1_{\{0<k\leq r\}}\frac{r}{\xi_{i,w,e}}LI_{a,i,w,j,k-1,e}(t)+1_{\{k>r\}}\frac{s}{\gamma_{i,w,e}}LI_{a,i,w,j,k-1,e}(t)$$
$$+ 1_{\{0<j<h\}}\frac{(h-1)}{\varepsilon}LI_{a,i,w,j-1,k,e}(t)$$
$$-\Bigg\{\mu_a(t)+\frac{1_{\{a<na-1\}}}{w_a}+1_{\{k<r\}}\frac{r}{\xi_{i,w,e}}+1_{\{k\geq r\}}\frac{s}{\gamma_{i,w,e}}$$
$$+ 1_{\{j<h-1\}}\frac{(h-1)}{\varepsilon}\Bigg\}LI_{a,i,w,j,k,e}(t)$$

The next 2 equations present the flows between immunity states (Figure A1).

$$MNI_{a,i}(t) = 1_{\{i=2\}}\left(IPVE_{a,0,0}(t)+1_{\{a=0\}}IPVE_{0,1,0}(t)\right)/\varphi$$
$$+\sum_{w=0}^{nw-1}\Big(1_{\{i=3\}}IPVE_{a,2,w}(t)+1_{\{i=4\}}\left(IPVE_{a,3,w}(t)+IPVE_{a,4,w}(t)\right)$$
$$+ 1_{\{i=7\}}\left(IPVE_{a,5,w}(t)+IPVE_{a,6,w}(t)+IPVE_{a,7,w}(t)\right)\Big)/\varphi$$

$$MNL_{a,i}(t) = \sum_{j=0}^{h}1_{\{i=5\}}\left(LI_{a,0,0,j,r+s-1,0}(t)/\gamma_{0,0,0}+1_{\{a=0\}}LI_{0,1,0,j,r+s-1,0}(t)/\gamma_{1,0,0}\right)s$$
$$+\sum_{w=0}^{nw-1}\Big(1_{\{i=6\}}\left(LI_{a,5,w,j,r+s-1,0}(t)/\gamma_{5,w,0}+LI_{a,6,w,j,r+s-1,0}(t)/\gamma_{6,w,0}\right)$$
$$+ 1_{\{i=7\}}\left(LI_{a,2,w,j,r+s-1,0}(t)/\gamma_{2,w,0}+LI_{a,3,w,j,r+s-1,0}(t)/\gamma_{3,w,0}\right.$$
$$\left.+ LI_{a,4,w,j,r+s-1,0}(t)/\gamma_{4,w,0}+LI_{a,7,w,j,r+s-1,0}(t)/\gamma_{7,w,0}\right)\Big)s$$

The following equations give the transitional quantities, introduced to further simplify the notation.

$$N(t) = \sum_{a=0}^{na-1} N_a(t)$$

$$N_a(t) = \left(PI_{0,1,0}(t) + IPVE_{0,1,0}(t) + \sum_{j=0}^{h} \sum_{k=0}^{r+s-1} LI_{0,1,0,j,k,0}(t) \right) 1_{\{a=0\}} + PI_{a,0,0}(t)$$
$$+ IPVE_{a,0,0}(t) + \sum_{j=0}^{h} \sum_{k=0}^{r+s-1} LI_{a,0,0,j,k,0}(t)$$
$$+ \sum_{i=2}^{ni-1} \sum_{w=0}^{nw-1} \left(PI_{a,i,w}(t) + IPVE_{a,i,w}(t) + \sum_{j=0}^{h} \sum_{k=0}^{r+s-1} LI_{a,i,w,j,k,0}(t) \right)$$

$$mf(t) = \frac{\sum_{a \geq fa}^{la} \sum_{i=3}^{ni-1} \sum_{w=0}^{nw-1} PI_{a,i,w}(t)}{+ \sum_{a \gg fa}^{la} N_a(t)}$$

$$\lambda_{a,j}(t) = 1_{EPI_{a,j}(t) \geq EPI^*} \sum_{e=0}^{1} EPIM_{A(a),j,e}(t) p_e \left(1/\gamma_{0,0,e} + \mu^{ave}(t)\right) R0_j(t)$$

$$EPI_{a,j}(t) = \sum_{e=0}^{1} EPIM_{A(a),j,e}(t) p_e$$

$$EPIM_{a_m,j,e}(t) = \sum_{k=r}^{r+s-1} \sum_{b=c(a_m)}^{c(a_m+1)} \left(LI_{0,1,w,j,k,e}(t) \pi_{1,0,e} M(a_m, 0)(t) 1_{\{b=0\}} \right.$$
$$+ \left(LI_{a,0,w,j,k,e}(t) \pi_{0,0,e} \right.$$
$$\left. \left. + \sum_{i=2}^{ni-1} \sum_{w=0}^{nw-1} LI_{a,i,w,j,k,e}(t) \pi_{i,w,e} \right) M\left(a_m, A(b)\right)(t) \right) \frac{\theta_k}{NM_{a_m}(t)}$$

$$M(a_m, b_m)(t) = \kappa(a_m) 1_{\{a_m = b_m\}} + \frac{\left(1 - \kappa(a_m)\right)\left(1 - \kappa(b_m)\right) NM_{b_m}(t)}{\sum_{c_m=0}^{na_m-1} NM_{c_m}(t)\left(1 - \kappa(c_m)\right)}$$

$$NM_{a_m}(t) = \sum_{b=c(a_m)}^{c(a_m+1)} N_b(t)$$

$$\mu^{ave}(t) = \sum_{a=0}^{na-1} \mu_a(t)\, N_a(t)/N(t)$$

$$R0_j(t) = R0_j^{ave}\left(1 + \alpha \times \sin\left(\left(t - \frac{pd}{365}\right) \times 2\pi + \frac{\pi}{2}\right)\right)$$

$$R0_j^{ave} = R0_{h-1}^{ave} - (R0_{h-1}^{ave} - R0_0^{ave}) \times ((h-1-j)/(h-1))^{zr}, \quad j = 1, \ldots, h\text{-}1$$

$$R0_{h-1}^{ave} = \tau R0_h^{ave}$$

$$R0_0^{ave} = \tau R0_h^{ave}$$

$$\sigma_{i,w} = \sigma_{i,nw-1} - (\sigma_{i,nw-1} - \sigma_{i,0}) \times ((nw-1-w)/(nw-1))^{z}, \quad w = 1, \ldots, nw\text{-}1$$

$$\gamma_{i,w,e} = \gamma_{i,nw-1,e} - (\gamma_{i,nw-1,e} - \gamma_{i,0,e}) \times ((nw-1-w)/(nw-1))^{z}, \quad w = 1, \ldots, nw\text{-}1$$

$$\pi_{i,w,e} = \pi_{i,nw-1,e} - (\pi_{i,nw-1,e} - \pi_{i,0,e}) \times ((nw-1-w)/(nw-1))^{z}, \quad w = 1, \ldots, nw\text{-}1$$

$$\xi_{i,w,e} = \xi_{i,0,e}$$

A3. Global model structure

We use a similar approach to the one reported in a prior analysis that aimed to optimally manage polio endgame risks from 2013-2052 [11]. However, as discussed in the main paper, we updated the model to reflect the demographic, epidemiological, and programmatic experience through the end of 2019, and the actual planned GPEI future activities. These changes make the model sustain continued WPV1 transmission through the end of 2019 and push all of the polio endgame timelines forward. In addition, the updated model accounts for the insufficient tOPV intensification that occurred in some countries prior to the April-May 2016 tOPV-bOPV switch, the resulting challenges with cVDPV2s, and the use of mOPV2 for outbreak response since the switch. Although an analysis of options for outbreak response [35] using the earlier model [11] highlighted the need for aggressive, large, and high-quality outbreak response SIAs using the appropriate OPV (i.e., in this case mOPV2), the actual scope, coverage, and quality of outbreak response SIAs since the tOPV-bOPV switch have varied which increases the risks of needing to restart the use of OPV2. In addition, although prior work clearly indicated marginal (if any) benefits and showed no economic justification for IPV use in outbreak response [36], in some cases countries and the GPEI used IPV for outbreak response. Remarkably, this use occurred despite insufficient IPV supply to introduce IPV into RI in all countries before the tOPV-bOPV switch and up through the end of 2019. Since the development of the prior model [11], the GPEI also pushed for a change to the recommended future minimum IPV schedule to include 2 doses of IPV in all countries for at least 10 years after globally-coordinated cessation of the last serotype of OPV [37]. In addition, the GPEI began transition activities, despite not completing polio eradication, and this decreased financial investments in some programs and generally decreased spending on preventive SIAs, which previous modeling demonstrated play an important role in maintaining high population immunity up until future globally-coordinated bOPV cessation. While downsizing in these areas, the GPEI expanded surveillance activities by adding ES in some areas at relatively higher risk of transmission. All of these differences and others motivated the global model update. The following sections describe the assumptions for the updated global model.

A3.1. Stratification of the population into blocks and preferential mixing areas (PMAs)

Table A1 summarizes the high-level model stratification of the global population using the 2019 revision of the UN World Population Prospects [12], by 2019 World Bank income level [38], and polio vaccine use as of October 2018 [39] for all 200 countries with available data. We assign 72 blocks to the different combinations of income level and polio vaccine use at T_0 as shown in Table A1. In some instances, we assign some less-populated countries to a block with a higher or lower income level recognizing the historical changes in income level that occurred over time or due to geographic proximity to countries that used the same polio vaccine in 2019 (and possibly experienced similar transmission conditions). The World Bank income level of some countries changed over time, notably for two very large countries (i.e., China and India), and we characterize these countries in their current income level. As described in the main text, we also group the blocks into 9 preferential mixing areas (PMAs) (see Table 1).

A3.2. Characteristics of inputs for subpopulations

Table A2 provides the assumptions for the 72 blocks for the assumptions that generally do not vary by block. As suggested by Table A2, for some blocks within multi-block areas that correspond to large countries, we assume identical properties as indicated by the use of single rows for multiple blocks. As noted in the main text, we use several model inputs to characterize the variability in conditions relevant to poliovirus transmission, including: the basic reproduction number (R_0), its seasonal amplitude (α) and peak day (*pd*), the age-group preferential mixing strength (κ), the proportion of transmissions via the oropharyngeal route (p^{oro}), the OPV take rate level (*trl*), the IPV use start year (T_{IPV}). The second to last column in Table A2 refers to inputs provided in the top of Table A3, which provides the assumed OPV take rates, inputs for SIA impact levels, and impact of outbreak response SIAs (oSIAs). We allow for different assumptions for the subpopulations within three HI blocks (43, 44, 67), which leads to more than one row for the block in Table A2. Specifically, in one subpopulation in each of those blocks we assume a slightly higher R_0 and lower p^{oro} (compared to all other subpopulations), which allows us to explore the impacts of heterogeneity that results from relatively lower-performing areas of blocks with otherwise current high IPV-only coverage and on poliovirus spread following an importation. We assume positive correlations between higher R_0, lower α, p^{oro}, and *tr*.

Table A4 summarizes the immunization inputs for all of the 720 subpopulations (i.e., 10 subpopulations per block), noting when subpopulations within the block share the same values by including more than one subpopulation number in the subpopulation column. Immunization inputs include assumptions about the RI coverage with 1 or 2 non-birth doses (*COV1or2*), the RI coverage with 3 or more non-birth doses (*POL3*), the SIA impact level (*sil,* characterized by the true coverage and repeated missed probability), the number of planned, and the preventive SIA rounds as a function of conditions and time (SIA_x, where 1 = (all-WPV-elimination, OPV2-cessation), 2 = (OPV2-cessation, T_0), 3 = [T_0, T_0+3), 4 = [T_0+3, T_0+6)), the detection threshold as a function of time (dt_x, x=1, 2, 3, 4, where 1 = [T_0-49, T_0), 2 = [T_0, T_0+6), 3 = [T_0+6, T_0+9), 4 = [T_0+9, T_{end})). The values for SIA impact level refer to inputs provided in the middle of Table A3. To approximate RI coverage in different populations at T_0, we only used a discrete set of *POL3* values (i.e., 0.1, 0.3, 0.6, 0.9, and 0.98), chosen such that average coverage in each block corresponds to the estimated average value for that block. We assumed no changes in coverage levels over time going forward (i.e., static RI coverage from T_0 -2 to T_{end}).

Table A5 summarizes the surveillance inputs for all of the 720 subpopulations for the ES start year (T_{ES}), the ES quality (Q_{ES}), and the total catchment percent of the population covered by ES as a function of time (tcp_x, x = 1, 2, 3, where 1 = [T_{ES}, T_0+6), 2 = [T_0+6, T_0+9), 3 = [T_0+9, T_{end})).

We assume that some inputs that vary by subpopulation (i.e., *COV1or2*, *POL3*, *sil*, and SIA_x in Table A4 and dt_x, T_{ES}, Q_{ES}, *and* tcp_x in Table A5) may also vary over time due to their dependence on risk management policies (i.e., SIA_x, dt_x, *and* tcp_x). These assumptions allow us to account for real heterogeneity in RI, SIAs, and surveillance within blocks, and policy changes post OPV2 cessation. We assume positive correlations between higher *POL3*, higher SIA impact, lower *dt,* and lower Q_{ES} within blocks.

While we use a relatively limited (albeit large) set of assumptions that allowed us to simulate global incidence relatively well, we noted some important epidemiological events that led to changes or disruptions in immunization and motivated changes in the assumptions in Table A4

for some subpopulations at specific times. Notably, to represent the last reservoirs of indigenous WPV transmission that resemble the real-world final reservoirs of WPV transmission, we allow sustained poliovirus transmission to continue despite frequent SIAs by assuming that one LI block (i.e., 32) and three LMI blocks (i.e., 34, 47, 48) each contain one chronically under-vaccinated subpopulation and one LMI block (i.e., 8) contains two chronically under-vaccinated subpopulations with much lower *POL3*, SIA impact, and higher dt_x compared to other subpopulations in the blocks. Moreover, we assumed a temporary lack of any vaccination in one of two chronically under-vaccinated subpopulations in LMI block (i.e., 8) from 2003.5 (T_0-15.5) to 2004.5 (T_0-14.5), followed by partial increase from 2014.5 (T_0-14.5), and original schedule from 2005.9 (T_0-13.1) (see notes in Table A8a). To ensure timely elimination of transmission of individual WPV serotypes in three of those blocks, we assumed temporary improvement in the chronically under-vaccinated subpopulations for the one LI block (i.e., 32) and one LMI block (i.e., 34) in the second half of 2010 (T_0-8.3 to T_0-8), and continuous improvements in the chronically under-vaccinated subpopulations in two LMI blocks (i.e., 47, 48) since 2010 (T_0-9). To represent realistic reservoirs in which importations of WPV led to outbreaks post elimination of indigenous transmission, we assumed a temporary decrease in number of planned SIA (SIA_1 in Table A4) in one subpopulation of one LI block (i.e., 1) since 2010 (T_0-9), two subpopulation of one LI block (i.e., 3) since 2008 (T_0-11), and 2013 (T_0-6), three LMI blocks (i.e., 13, 33, and 7) since 2004 (T_0-15), and 2004 (T_0-15), and 2008 (T_0-11), following by regular schedule after finished outbreak response. To represent realistic reservoirs in which cVDPV emergences occurred after OPV2 cessation, we assumed a decrease in vaccination quality in one subpopulation of one UMI block (i.e., 36) since 2012 (T_0-7.1) and in two LI blocks (i.e., 1 and 3) since 2015.7 (T_0-3.3) and 2015.5 (T_0-3.5) (see notes in Table A4), until OPV2 cessation. Moreover, we included additional block-wide SIA vaccination with mOPV2 in one LMI block (i.e., 8) in January 2017 (T_0-2) and January 2019 (T_0) representing realistic post-OPV2 cessation use of mOPV2 in African region that led to new cVDPV2 emergences (see notes in Table A8c).

A3.3. Model run-up and assumptions about planned, preventive SIA (pSIA) schedules in blocks that use OPV+IPV or IPV/OPV

Considering the high computational demands and highly variable polio vaccination histories around the world, we used a simplified run-up. Specifically, we start the model by introducing a single infection in each (fully susceptible) subpopulation, and we allow the model to establish endemic equilibrium without population growth or vaccination by running it for 25 years without seasonal fluctuation and then 25 years with seasonal fluctuations, which is equivalent to starting the model at T_0-119. Next, we turn on population growth and we allow the model to run for additional 10 years, until the first set of blocks start RI (i.e., from T_0-69 to T_0-59). To apply more realistic routine vaccination levels, we estimated relative POL3 coverage using WHO/UNICEF Estimates of National Immunization Coverage (WUENIC) for 1980-2018 [40], with all unknown years assumed to linearly increase from 1980 (i.e., using a midpoint value between 0% and the first reported coverage estimate) for years prior to the first reported value. Table A6 presents relative coverage for each block for 1980-2018 (relative to the 2019 estimates for 2018). We introduce tOPV RI in 1980, 1980, 1970, and 1960 (T_0-39, T_0-39, T_0-49, and T_0-59) for LI, LMI, UMI, and HI blocks respectively. We assume a linear increase from 0% of the subpopulation-specific POL3 up to block specific relative coverage of the subpopulation-specific POL3 in 1980 (i.e., T_0-39) in UMI and HI blocks (see column 2 in Table A6). We use block-

specific yearly relative coverage of the subpopulation-specific POL3 from 1980 (i.e., T_0-39) up to 2018 (i.e., T_0-1), followed by 100% of the subpopulation-specific POL3 for the rest of the time horizon. To estimate the implied POL3 and COV1or2 coverage for each subpopulation over time, we multiply the appropriate RI coverage estimate in columns 6 and 7 in Table A4 by the estimated values in Table A6 for each block. We introduce tOPV pSIAs in the LI, LMI, and UMI blocks in 1993, 1993, and 1990 (i.e., T_0-26, T_0-26, and T_0-29), respectively, according to the subpopulation-specific vaccination assumptions. We include the period of mOPV use for some pSIAs in endemic countries in 2005-10 by introducing mOPV1 in 2005 (i.e., T_0-14) and mOPV3 in 2007 (i.e., T_0-12), and we introduce bOPV for SIAs in 2010 (i.e., T_0-9) by switching some SIAs from tOPV to bOPV.

The model allows for IPV introduction at different times for each block based on its T_{IPV} (Table A2). The model assumes that at the time of IPV introduction, RI in the block shifts from an OPV-only schedule to the schedule indicated in column 5 of Table A2. This assumption represents a simplification of the RI schedules used by countries over time, which included more than one shift in some cases (e.g., HI countries generally shifted from OPV-only to IPV/OPV and then to IPV-only).

The model assumes that WPV elimination occurs in a block when the WPV prevalence of infections of all serotypes in all subpopulations falls below the transmission threshold (EPI*, see appendix A2.7). We assume that WPV importations can occur, cause outbreaks, and delay the achievement of global WPV eradication, however, they do not reverse the WPV elimination status for a block. Table A7 includes a set of manual introductions of WPV1 and WPV3 in blocks that represent locations that historically experienced large outbreaks (over 20 AFP cases) due to an importation of WPV (post elimination of indigenous transmission), which contributed to global poliovirus incidence (i.e., during the late 2000s, more paralytic cases occurred in non-endemic counties than in endemic ones [41]). Table A7 also includes a set of manual introductions of cVDPV2 in blocks that represent locations that recently experienced large outbreaks (over 20 AFP cases) due to an importation of cVDPV2.

Table A8 summarizes the assumptions for pSIAs in OPV-using blocks. Table A8a provides assumptions for the number of pSIA rounds before elimination of all WPVs in a block. Table A8b provides assumptions for pSIA schedules in the pre-OPV2 cessation era (depending on the WPV elimination status and RI coverage). Table A8c provides assumptions for pSIA schedules in the post-OPV2 cessation era (depending on the WPV elimination status and the number of pSIAs of the subpopulation indicated in Table A4). In UMI blocks the SIA schedule consists of two annual tOPV rounds until WPV elimination. In LI and LMI blocks, we based the SIA schedule on the average number of pSIAs used in endemic countries of a given income level over time until WPV elimination, except in one LMI block that performed fewer pSIAs consistently below the average. The vaccine choice for each round depends on the time period (i.e., only tOPV until the introduction of mOPV1 and mOPV3, then predominantly tOPV with some mOPV1 and mOPV3 in endemic areas until introduction of bOPV, then predominantly bOPV in endemic areas with some tOPV until OPV2 cessation, then bOPV with one IPV round in subpopulations 1 and 2 of the remaining endemic countries until T_0-1 after OPV2 cessation, and then only bOPV after T_0-1 until WPV elimination). Once a block reaches WPV elimination status, the model begins to use the RI coverage-dependent on the post-WPV-elimination

schedule (see the SIA_1 column in Table A4). All blocks that use IPV-only at T_0 achieved WPV elimination before switching to IPV-only (at which time they also stopped any possible pSIAs). For the remaining bocks, the choice of vaccine depends on the time period, with tOPV-only initially, predominantly bOPV with some tOPV in lower RI coverage blocks after bOPV introduction, predominantly tOPV (intensification) with some bOPV in the first quarter of 2016 (i.e., the OPV2 cessation year), and bOPV-only between OPV2 cessation and OPV13 cessation.

Figure A10 presents the results for the burn-in period of the model relative to T_{GPEI}. In the pre-vaccination era (i.e., before 1960, years T_{GPEI} -38 to T_{GPEI}-28) the global WPV incidence of all serotypes oscillates around 400,000 cases per year. Once RI begins in the HI blocks (i.e., 1960 or T_{GPEI}-28), followed by UMI blocks (i.e., 1970 or T_{GPEI}-18), the incidence of all serotypes starts to slowly decrease to around 230,000 cases per year when RI introduction occurs in all remaining areas (i.e., 1980 or T_{GPEI}-8). The incidence further decreases to around 170,000 cases at T_{GPEI} as RI coverage increases.

A3.4. Modeling virus (re)introductions using the probability of an effective introduction function ($P_{effective}$)

We model effective virus (re)introductions using the probability of an effective introduction function ($P_{effective}$) of the mixing-adjusted net reproduction number (R_n). We define R_n as the average number of secondary infections generated by a single infection that accounts for population immunity, equal to the R_0 multiplied by the age-mixing-adjusted effective susceptible proportion (ESPM) [32]. We calculated ESPM as the dominant eigenvalue of the effective susceptibility matrix $ESP(a_m, b_m) = M(a_m, b_m)(t) \times ESP(a_m)$, where $M(a_m, b_m)$ represents the normalized preferential age mixing matrix (see section A2.2) and $ESP(a_m)$ represents the effective susceptible proportion in mixing age group a_m which equals:

$$ESP(a_m) = \left(\sum_{i=0}^{ni-1} \sum_{w-0}^{nw-1} \sigma_{i,w} \sum_{e=0}^{2} \frac{p_e \sum_{b=c(a_m)}^{c(a_m+1)} N_{b,i,w}(t) \pi_{i,w,e} \gamma_{i,w,e}}{\gamma_{0,0,w}} \right) \Big/ NM_{a_m}(t).$$

Therefore, $P_{effective}$ depends on subpopulation-, serotype-, and reversion-stage-specific differences in R_0, [3, 10] as well as time- and subpopulation-dependent seasonality, vaccination policies, and LPV exposure expressed by ESPM. Figure A9 shows the $P_{effective}$ for which we assume a logarithmic increase for $R_n>1$ (i.e., high population immunity), with values of 0.2 when $R_n=1$ (i.e., threshold level population immunity) and 0.5 when $R_n\geq5$ (i.e., very low population immunity). Since every potential poliovirus introduction event may or may not establishes transmission, we use a random uniform number (U) to determine whether the introduction is effective. When an effective introduction occurs (i.e., $U<P_{effective}(R_n)$), the model generates exactly enough initial infections in the receiving subpopulation to exceed the transmission threshold and potentially lead to circulation (see appendix A2.7).

A3.5. Risk of long-term immunodeficiency-associated vaccine-derived poliovirus (iVDPV) excreters

We previously developed a discrete-event simulation (DES) model of long-term iVDPV excreter prevalence [42] to characterize iVDPV prevalence over the time horizon. The model depends on

the timing of OPV cessation of each serotype, and we updated these and other assumptions in an updated iVDPV model [43]. Each stochastic iteration of the global model uses a corresponding stochastic realization of the DES model to create random, potential iVDPV introductions into the general population, as well as randomly generated contacts with the general population for each active long-term iVDPV excreter after serotype-specific OPV cessation. We use R_0 as a proxy to estimate the general population contact rate. Specifically, we assume that primary immunodeficiency disease (PID) patients who survive long enough to become long-term excreters mix much less with others than typical immunocompetent individuals in the general population, effectively leading to R_0 values of 1–4 (consistent with relatively good hygiene and limited mixing for continued survival). Also, we assume only 5% of their contacts involve non-close contacts (most contacts within same household individuals who possess sufficient immunity to prevent further spread due to their ongoing exposure to the long-term excreter). Therefore, we assume a range of 0.05-0.2 contacts per 30 days (i.e., average times of approximately 150–600 days between potential contacts that may lead to an iVDPV infection) with the general population for a long-term excreter (Table A9). For each individual long-term excreter, we draw a random contact rate from this range using a uniform distribution. Based on the draw, we randomly determine the time between general population contacts and include these as potentially effective iVDPV introductions, which become censored by if the excreter dies, recovers from infection, or the analytical time horizon ends (i.e., the next contact occurs after T_{end}). We assume the iVDPV enters the general population at reversion stage 10, if successful. Specifically, iVDPV excreter contacts with the general population may or may not lead to effective introductions (depending on microlevel dynamics and chance, see section A2.7), for which we apply the $P_{effective}$ to determine whether the introduction establishes transmission (see section A3.4). Faced with substantial uncertainty about iVDPV risks in the absence of evidence, we previously assumed (conservatively) that introductions from iVDPV excreters would enter the population at stage 19 [3], and we demonstrated the significance of this assumption in a sensitivity analysis that assumed introduction at stage 10 [44]. The lack of evidence of iVDPV2 transmission associated with iVDPV excreters within the first 3.5 years after OPV2 cessation led us to change the baseline assumption to introduction at stage 10, although we emphasize that considerable uncertainty about the risks of iVDPV excreters and how to best manage and model them remains.

We also use the updated DES model [43] to create new iVDPV excreters as a result of any post-OPV cessation outbreak response SIAs that use OPV. The DES model [42, 43] assigns each individual born with a pre-disposition of developing a PID an iVDPV excreter potential acquisition status and thus only some PIDs can acquire a long-term iVDPV infection upon OPV exposure after PID onset. All PID patients pre-determined to potentially acquire long-term iVDPV excretion (if infected and surviving for any time after OPV cessation) could develop long-term excretion as a result of any OPV use in their subpopulation in the post-OPV cessation era. Consequently, the corresponding stochastic realization of the DES model [42, 43] records events that affect the ability of becoming a newly-generated iVDPV excreter for all individuals meeting those criteria (i.e., times of PID onset, PID diagnosis, any intravenous immunoglobulin (IVIG) treatment start or lapse, death, and contacts with the general populations). In the event of post-cessation OPV use in a subpopulation, we pre-determine random characteristics of any potential new OPV infections by generating a set of random numbers to characterize 10 possible exposures to OPV after OPV cessation (i.e., ten uniform random numbers to compare against the

probability of infection given exposure, ten random durations of an infection to apply in the event a new infection, ten uniform random numbers to determine the time of VAPP onset in the event of a new infection, and one uniform random number to determine whether fatal VAPP occurs in the event of VAPP onset prior to the pre-determined non-VAPP related time of death and prior to recovery from the new infection). We recycle the random numbers in the very unlikely event of more than ten OPV exposures post-OPV cessation. In case of post-OPV cessation OPV use for outbreak response, we calculate the probability of infection given exposure for each alive, clinical, not-yet-infected PID patient (based on pre-determined PID events and infection probabilities). Thus, depending on chance, any OPV exposure may or may not lead to new infection. Thus, we model it by comparing the random uniform number of the corresponding exposure to the probability of infection given exposure. Once a new infection occurs, we use the infection duration of the corresponding exposure and determine whether (fatal) VAPP occurs by comparing the appropriate random uniform number to the appropriate probability.

A3.6. Post OPV cessation risks, blocks with vaccine production sites and non-vaccine producing Poliovirus Essential Facilities (PEFs)

Table A9 provides estimates for the non-cVDPV risks, which we categorize as occurring from a containment breach from a poliovirus essential facility (PEF) leading to release of an LPV, release from a facility holding potentially infectious material (PIM) or another unintentional release of an LPV, intentional release of an LPV, or any unreturned OPV use. We assume the highest risk of unreturned OPV use occurs within the first year of vaccine withdrawal (i.e., serotype-specific OPV cessation or vaccine withdrawal after oSIAs), with significantly lower chances in the second year (see Table A9). Also, we assume that blocks characterized by worse program performance (i.e., lower RI or pSIA intensity) present a more likely location for unreturned OPV use (see block list in Table A9). We assume that the use of mOPV for outbreak response after homotypic OPV cessation further extends the potential use of unreturned OPV.

The assumed rate of release from IPV production sites remains 1 per 5 years (Table A9). For each release we randomly select the IPV production site that releases either WPV (i.e., from a Salk-IPV facility) or Sabin OPV virus (i.e., from a Sabin-IPV facility). We base the distribution of IPV production sites on expected or assumed potential IPV producing facilities (see Table A10a).

We assume an overall rate of 1 per 40 years (i.e., 0.025 per year) for other releases that do not involve IPV production (Table A9). We divided these other releases into (i) accidental releases from non-vaccine producing PEFs (i.e., PEFs that do not manufacture or distribute vaccine assuming these facilities account for 80%, or an absolute rate of 0.02 per year)), (ii) other unintentional releases from facilities holding PIMs (assuming that they account for 95% of the remaining 20%, or an absolute rate of 0.00475 per year), and (iii) intentional releases (assuming they account for the remainder, or an absolute rate of 0.00025 per year). For each release from non-vaccine producing PEFs we randomly select the site and the virus type released (i.e., Sabin OPV, an OPV-related virus, or a FRCV that represents a WPV or cVDPV). We base the distribution of non-IPV producing PEFs on the WHO list of facilities expected to apply for PEF status (see Table A10b).

Non-cVDPV releases of poliovirus may or may not lead to effective introductions (see section A2.7). Therefore, we apply the $P_{effective}$ to determine whether the introduction establishes transmission (see section A3.4).

A3.7. Limitations

Specific limitations of the integrated global model include our conceptual and structural characterization of global variability using 720 subpopulations and our simplified modeling constructs that simulate die-out, OPV evolution, waning of immunity to transmission, and effective poliovirus introduction into the DEB model that characterizes poliovirus transmission and OPV evolution. Specifically, we characterize global variability and mixing using a finite number of subpopulations (which only approximates the true variability in the population and global mixing patterns). The model assumes spatially-homogeneous (age-heterogeneous) mixing in subpopulations of approximately 10.7 million people each, which implies faster spread than more heterogeneous mixing. We off-set the potential impacts of faster spread to some degree using a relatively low assumed rate of exportations between subpopulations.

The speed of poliovirus spread between populations in the absence of any recent prior LPV exposure represents a significant uncertainty, which we previously explored by varying the threshold (i.e., E*) to trigger potentially effective exportations and for which we found substantial impacts on the ability to control outbreaks after OPV cessation [11]. The value of E* interacts directly with the assumed relationship between population immunity to transmission and the probability of an effective introduction, all of which remain uncertain. Real exportations represent stochastic events, with chance determining the actual path of viral transmission. Different assumptions about the speed of spread between populations will imply different requirements for the aggressiveness of the outbreak response and vaccine needs from outbreak response stockpiles.

Although the model captures the possibility of exportations of OPV use during an outbreak response to other subpopulations, it does not account for the potentially higher probability of exportation of OPV at the borders between the targeted and non-targeted population that may mix more intensely. Thus, in outbreak response the global model may underestimate the local risks of mOPV exportation following its use in outbreak response after OPV cessation. The model appears to capture the kinetics of poliovirus transmission adequately in spite of the simplifications inherent in the assumptions of spatially-homogeneous mixing within subpopulations. However, uncertainties about the kinetics of outbreaks and vaccine virus spread between populations will affect the expected consequences of rare poliovirus reintroductions and the ability of different vaccines to control outbreaks and/or seed new outbreaks.

We base our results on a limited number of model iterations, and additional iterations may lead to the realization of some other sequences of events that we did not yet observe, with many iterations required to observe relatively rare events. Our estimates of future inputs and policies (for prospective modeling) come with inherent uncertainties associated with projection (e.g., time series for vaccine coverage, prices, wastage, etc.) and uncertainty in these estimates may significantly impact the overall results, which suggests the need for future evaluation of these

assumptions. Our assumptions about the RC maintaining constant RI over time represents an unrealistic path, since in reality RI tends to vary year-to-year, but this assumption provides a basis for comparisons.

In the real world, the availability of different quantities and formulations of vaccines as a function of time determines what can and will actually be used. The true number of paralytic cases that would occur with use of the existing poliovirus vaccines will further depend on the actual impact of IPV on transmission in different settings and on the proportion of vaccine recipients protected from poliomyelitis disease as potentially more immunogenic IPV vaccines become available.

Many uncertainties and stochastic events limit our ability to predict what will actually happen in the unprecedented post-OPV era and which may lead to a wide range of potential consequences. Besides the outbreak response strategy itself, the rate of decrease in population immunity to transmission with IPV-only routine immunization and the assumed frequency of poliovirus exportations to other subpopulations and blocks determine the ability to control outbreaks and prevent mOPV from starting new VDPV outbreaks.

Uncertainties remain about the risks of cVDPVs, iVDPVs, and potential unintentional or intentional releases of LPVs after OPV cessation that could reintroduce LPV transmission. The potential benefits and uses of poliovirus antiviral drugs currently under development and the potential for screening efforts to identify asymptomatic iVDPV excreters for treatment also remain uncertain. Modeling the kinetics of VDPVs in a differential-equation-based model also may not account for stochasticity and dynamics at the individual level, although we hope that it captures the average behavior of poliovirus transmission and population immunity in large populations. Insufficient data and uncertainty exist to model the properties of each PID defect in the combined oPID category, but important differences certainly exist, and more serious forms of PID (e.g., SCID) may imply a greater probability of long-term poliovirus excretion, but also lead to reduced survival, particularly in developing countries. Significant uncertainty also exists about the fraction of PIDs treated with IVIG as a function of time and the impact of treatment on their survival.

Numerous uncertainties and information gaps influence the nature and quality of information available from poliovirus surveillance. For example, the future of AFP surveillance remains uncertain. Also, in our characterization of ES, we found many challenges in developing the methods and model inputs for the global model, and the results of the simulations remain sensitive to the approach to model ES and the assumptions about the distribution of sites. The quality of information about the available data on watershed population of ES sampling sites remain incomplete and, in some cases, inconsistent with expectations for a high-quality ES system. The methods for sample collection and virus concentration, and the ability to recover and identify poliovirus from concentrated ES samples, represent finer levels of detail than we capture with our abstract model as part of site quality.

A4. Valuation of health and economic outcomes

We use the global model to characterize health and economic outcomes consistent with standard guidelines for health and economic modeling analyses [45, 46]. We use a societal perspective to estimate all benefits and costs regardless of who pays or receives them. We use a 3% discount rate [45, 46] for future costs, and report the health outcomes (i.e., polio cases) with and without discounting. We express all monetary amounts in 2019 net present values, using 2019 US dollars ($). We calculate incremental economic outcomes for the alternative option (AO) compared to the reference case (RC) using different metrics including: (i) the incremental cost-effectiveness ratios (ICER) in $ per prevented (paralytic) polio case (i.e., $ICER_{case}$), (ii) the ICER in $ per averted disability-adjusted life-years (DALYs) (i.e., $ICER_{DALY}$) [47] and the incremental net benefits (INBs, in $) as follows:

$$ICER_{case}\ (AO\ vs.RC) = \frac{(FC_{AO} - FC_{RC}) - T \times (PP_{RC} - PP_{AO})}{PP_{RC} - PP_{AO}}$$

$$ICER_{DALY}\ (AO\ vs.RC) = \frac{(FC_{AO} - FC_{RC}) - T \times (PP_{RC} - PP_{AO})}{D \times (PP_{RC} - PP_{AO})}$$

$$INB\ (AO\ vs.RC) = (T + S) \times (PP_{RC} - PP_{AO}) - (FC_{AO} - FC_{RC})$$

where
FC_{RC} = the cumulative, discounted financial costs associated with the reference case;
FC_{AO} = the cumulative, discounted financial costs associated with the alternative policy;
PP_{RC} = the cumulative, discounted number of polio cases with the reference case;
PP_{AO} = the cumulative, discounted number of polio cases with the alternative policy;
T = the average treatment costs per polio case;
D = the average number of DALYs per polio case;
S = the average societal economic costs per polio case.

We report the ICERs by income level and the INBs both by income level and as a global aggregate. For ICERs, we use "cost-saving, life-costing" (CSLC), "cost-saving, life-saving" (CSLS), and "dominated" labels to describe ICERs with negative incremental costs and negative prevented cases, negative incremental costs but positive prevented cases, and positive incremental costs but negative prevented cases, respectively [48].

In the context of economic analyses, in some cases we make some assumptions that may completely or partly cancel out in the incremental outcomes, but imply underestimation of the absolute values of non-incremental costs (e.g., the exclusion of global programmatic costs for both two different immunization policies).

A5. Alternative formulations

We use the generic DEB model described in section A2 as a base for poliovirus transmission modeling of different situations and specific populations. We used this approach to calibrate the model inputs as described in Section A2.9. Section A3 describes how we use the generic structure for global model of 72 blocks with 10 equally-sized subpopulations each and 7 age groups. However, in the context of modeling specific countries or geographical regions we use appropriate population-specific demographic and immunization information, as well as age and population structure. We also use alternative formulations for mixing between subpopulations [3, 10, 20, 21] to capture the nature of un- and under-vaccinated groups in some specific populations. In addition, in some cases we convert the deterministic model into a stochastic form [23, 49]. For completeness, we include these alternate formulations of the DEB model in the following subsections.

A5.1. Mixing between subpopulations

In situations in which we divide the total modeled population into *hsp* interacting subpopulations (e.g., an under-vaccinated subpopulation (*usp*) and *hsp-1* in the general subpopulation (*gsp*)). We assume the subpopulations mix preferentially, and we define p_{within} as the proportion of contacts within the subpopulation for individuals in one of the *hsp* subpopulations. All within-subpopulation contacts of individuals in the un(der)vaccinated subpopulation equal p_{within}, while $1 - p_{within}$ represents the proportion of their contacts with the general subpopulation. All within-subpopulation contacts of individuals in any *hsp*-1 subpopulations within the general subpopulation equal p_{within}, while the remaining $1 - p_{within}$ contacts outside that subpopulation divide into (*hsp*-2)/(*hsp*-1) contacts with individuals in other hypothetical subpopulations belonging to the general subpopulation, and 1/(*hsp*-1) contacts with members of the under-vaccinated subpopulation. Therefore, the force-of-infection in the under-vaccinated subpopulation equals (see section A2.11 for the list indices and symbols):

$$\lambda_{a,j}^{usp}(t) = 1_{EPI_{a,j}(t)\geq EPI^{*}} \sum_{e=0}^{1} \Big(EPIM_{A(a),j,e}^{usp}(t) p_{within} + EPIM_{A(a),j,e}^{gesp}(t)(1 - p_{within}) \Big)\, p_e\big(1/\gamma_{0,0,e} + \mu^{ave}(t)\big) R0_j(t)$$

and the force-of-infection for the general population equals:

$$\lambda_{a,j}^{gsp}(t) = 1_{EPI_{a,j}(t)\geq EPI^{*}} \sum_{e=0}^{1} \Big(EPIM_{A(a),j,e}^{usp}(t)\,(1 - p_{within})/(hsp - 1) + EPIM_{A(a),j,e}^{gsp}(t)(p_{within} + (1 - p_{within})(hsp - 2)/(hsp - 1)) \Big)\, p_e\big(1/\gamma_{0,0,e} + \mu^{ave}(t)\big) R0_j(t)$$

A5.2. Stochastic transformation and probability of undetected circulation

When focusing on modeling the probability of undetected poliovirus circulation, we transform the deterministic DEB model to a stochastic one. To do so, we first round the fractional number of individuals to integers for all stocks in the deterministic model at the time of the transformation. Specifically, we draw a random uniform number for each stock to determine whether we will round the fractional number in that stock up or down to the nearest integer. For each stochastic iteration we use a fixed time step of 0.5 day. For each time step, we calculate all transition rates that change the current state of the model. Next, for all calculated transition rates, we draw a random number from the Poisson distribution with an expected value (and variance) equal to the transition rate times the fixed time step. Each random Poisson draw returns the expected number of individuals to follow the given transition rate, with the condition that the sum off all transitions cannot exceed the size of the stock. We use random uniform numbers to determine whether infection events lead to paralytic cases, and we track the effective proportion of infectious individuals excreting the virus, which ES could detect. Also, we use situation-specific probabilities of detecting paralytic cases via AFP surveillance, and situation-specific characteristics of the ES system that may identify positive sewage isolates that indicate the presence of transmission. To estimate the confidence about the absence of circulation in the absence of detected cases and/or positive sewage isolates, we focus on detected-event-free periods (DEFPs). We use following metrics to describe the results ([49], p.6.):

- POE – "the probability of eradication defined as the fraction of stochastic iterations in which die-out occurs"
- DEFP – "the detected-event-free period defined as the time in months since the last detected case (AFP) or positive isolate (environmental surveillance)"
- CNC – "confidence about no circulation given the DEFP approximated as (1 – the number of DEFPs equal to t months with ongoing WPV/cVDPV circulation, divided by all DEFPs of t months)"
- CNCx% – "the time when the confidence about no circulation exceeds x% (i.e., CNC95%, CNC99%)"
- TUC – "the time of undetected circulation after the last detected-event (for those iterations in which extinction occurs)"
- TUCx% – "the xth percentile of the TUC (i.e., TUC95%, TUC99%)"

The CNCx% metric characterizes confidence about no WPV/cVDPV circulation as a function of time without observed events. The TUCx% metric estimates how long we might expect silent to continue WPV/cVDPV circulation after the last detected event given extinction.

A5.3. Limitations

Stochastic models use non-negative integer values for stocks and randomly draw transitions between stocks (e.g., by randomly determining the exact time intervals between transitions that occur in the system or use a fixed time step to randomly determine the number of transitions that occur in the system during this time step (e.g. using Poisson draws)).

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Table A1: Distribution of the global population as of 2019 [12] in hundreds of millions by 2019 World Bank income level [38] and polio vaccine use as of 2018 [39] covering 200 countries with available data. Numbers in parentheses indicate the number of corresponding epidemiological blocks in the global model.

	Polio vaccine use at T_0			
Income level	**OPV+IPV**	**IPV/OPV**	**IPV-only**	**Total blocks**
LI	6.94 (6)	0.11 (0)	0 (0)	6
LMI	29.45 (27)	0.78 (1)	0 (0)	28
UMI	2.30 (19→0)	23.89 (7→26)	0.73 (1)	27
HI	0.09 (0)	1.19 (0)	10.82 (11)	11
Total blocks	52	8	12	72

Acronyms: HI, high-income; IPV, inactivated poliovirus vaccine; LMI, lower middle-income; LI, low-income; OPV, oral poliovirus vaccine; UMI, upper middle-income); T_0, beginning of analytical time horizon (i.e., January 1, 2019)

Table A2: Summary of transmission model inputs for the 72 blocks in the global poliovirus transmission model (list of symbols provided at bottom of table)

Preferential mixing area (PMA)	Block	Sub-popul-ation(s)	Income level	Polio vaccine at T_0	R_0	α	pd	K	p^{oro}	trl	T_{IPV}
Africa	1	1-10	LI	OPV+IPV	10	0.15	0	0.35	0.3	3	-3
Africa	2	1-10	LI	OPV+IPV	11	0.1	180	0.35	0.3	3	-4
Africa	3	1-10	LI	OPV+IPV	10	0.15	180	0.35	0.3	3	-4
Africa	4	1-10	LI	OPV+IPV	9	0.2	0	0.35	0.3	3	-2
Africa	5	1-10	LI	OPV+IPV	11	0.1	120	0.4	0.3	3	-3
Africa	6	1-10	LMI	OPV+IPV	9	0.2	0	0.4	0.3	2	-1
Africa	7	1-10	LMI	OPV+IPV	8	0.15	0	0.4	0.3	4	-3
Africa	8	1-10	LMI	OPV+IPV	8	0.1	120	0.4	0.3	3	-4
Africa	9	1-10	LMI	OPV+IPV	9	0.05	120	0.4	0.3	3	-4
Africa	10	1-10	LMI	OPV+IPV	8	0.1	120	0.4	0.3	4	-3
Africa	11	1-10	UMI	OPV+IPV	7	0.25	300	0.35	0.6	5	-4
Australasia	12	1-10	LMI	OPV+IPV	7	0.15	180	0.4	0.5	5	-4
Australasia	13	1-10	LMI	OPV+IPV	8	0.2	120	0.35	0.3	3	-3
Australasia	14	1-10	LMI	OPV+IPV	7	0.2	120	0.4	0.5	4	-3
Australasia	15	1-10	LMI	OPV+IPV	6	0.2	120	0.4	0.6	5	-3
Australasia	16	1-10	HI	IPV-only	5	0.2	60	0.45	0.8	7	-11
Australasia	17	1-10	HI	IPV-only	4	0.2	120	0.45	0.9	8	-11
China and neighbors	18	1-10	UMI	OPV+IPV	7	0.4	180	0.5	0.6	6	-4
China and neighbors	19	1-10	UMI	OPV+IPV	6	0.35	180	0.5	0.8	7	-4
China and neighbors	20	1-10	UMI	OPV+IPV	8	0.35	180	0.5	0.6	5	-4
China and neighbors	21-31	1-10	UMI	OPV+IPV	7	0.35	180	0.5	0.6	6	-4
East and Central Asia	32	1-10	LI	OPV+IPV	11	0.2	180	0.35	0.3	2	-4
East and Central Asia	33	1-10	LMI	OPV+IPV	8	0.5	60	0.3	0.8	2	-1
East and Central Asia	34	1-10	LMI	OPV+IPV	11	0.15	180	0.35	0.3	2	-4
East and Central Asia	35	1-10	UMI	OPV+IPV	7	0.2	60	0.35	0.6	5	-4
East and Central Asia	36	1-10	UMI	IPV/OPV	7	0.25	60	0.35	0.6	5	-10
East and Central Asia	37	1-10	UMI	IPV/OPV	7	0.2	120	0.35	0.6	5	-11
Europe	38	1-10	LMI	IPV/OPV	7	0.5	60	0.3	0.8	2	-6
Europe	39-40	1-10	UMI	IPV/OPV	6	0.5	180	0.45	0.8	6	-11
Europe	41	1-10	HI	IPV-only	6	0.4	180	0.45	0.8	6	-11
Europe	42	1-10	HI	IPV-only	5	0.4	240	0.4	0.9	7	-19
Europe	43	1	HI	IPV-only	6	0.2	180	0.35	0.6	7	-19
Europe	43	2-10	HI	IPV-only	5	0.2	180	0.35	0.8	7	-19
Europe	44	1-9	HI	IPV-only	5	0.2	180	0.35	0.8	7	-19
Europe	44	10	HI	IPV-only	6	0.2	180	0.35	0.6	7	-19
Europe	45	1-10	HI	IPV-only	5	0.4	240	0.4	0.8	7	-19
Europe	46	1-10	HI	IPV-only	5	0.35	240	0.4	0.9	7	-19
India	47-48	1-10	LMI	OPV+IPV	13	0.2	180	0.35	0.3	1	-4
India	49	1-10	LMI	OPV+IPV	12	0.2	180	0.35	0.3	2	-4
India	50-56	1-10	LMI	OPV+IPV	11	0.15	240	0.35	0.3	3	-4
India	57-58	1-10	LMI	OPV+IPV	10	0.1	240	0.35	0.3	3	-4
Latin America and Caribbean	59	1-10	LMI	OPV+IPV	8	0.05	180	0.3	0.3	3	-4
Latin America and Caribbean	60	1-10	UMI	OPV+IPV	8	0.2	240	0.35	0.6	5	-4

Latin America and Caribbean	61	1-10	UMI	OPV+IPV	7	0.1	0	0.35	0.6	5	-3
Latin America and Caribbean	62	1-10	UMI	IPV/OPV	8	0.1	300	0.35	0.5	4	-7
Latin America and Caribbean	63	1-10	UMI	IPV/OPV	8	0.05	300	0.35	0.5	4	-7
Latin America and Caribbean	64	1-10	UMI	IPV-only	7	0.05	120	0.35	0.6	5	-11
North America	65	1-10	HI	IPV-only	4	0.4	240	0.35	0.9	7	-19
North America	66	1-10	HI	IPV-only	5	0.3	240	0.35	0.8	7	-19
North America	67	1-9	HI	IPV-only	5	0.05	180	0.35	0.8	7	-19
North America	67	10	HI	IPV-only	6	0.05	180	0.35	0.6	7	-19
South Asia	68	1-10	LMI	OPV+IPV	12	0.3	180	0.3	0.3	1	-4
South Asia	69	1-10	LMI	OPV+IPV	13	0.1	180	0.3	0.3	1	-4
South Asia	70	1-10	LMI	OPV+IPV	8	0.15	180	0.4	0.3	4	-2
South Asia	71	1-10	UMI	OPV+IPV	7	0.1	240	0.35	0.6	5	-4
South Asia	72	1-10	UMI	IPV/OPV	7	0.15	0	0.35	0.6	5	-10

Model input symbols: [3, 33] R_0, average annual basic reproduction number for WPV 1; α, seasonal amplitude of R_0, defined as the "proportional change in R_0 due to seasonality" [3, p. 717]; pd, peak day of R_0; κ, strength of preferential mixing between age groups, defined as the "proportion of contacts reserved for individuals within the same mixing age group" [3, p. 717]; p^{oro}, proportion of transmissions via oropharyngeal route; trl, OPV take rate level (see Table A4); T_0, January 1, 2019; T_{IPV}, year (relative to T_0) when IPV is introduced to the block
Acronyms: HI, high-income; IPV, inactivated poliovirus vaccine; LMI, lower middle-income; LI, low-income; OPV, oral poliovirus vaccine; UMI, upper middle-income

Table A3: SIA impact and OPV take rate levels used in Table A2 for the take rate level and in Table A4 for the SIA impact level, generalized from situation-specific values used in prior work [3, 10, 21, 33]

Take rate level	Average per-dose take rate for:							
	tOPV1	tOPV2	tOPV3	mOPV1	mOPV2	mOPV3	bOPV1	bOPV3
1	0.35	0.60	0.27	0.45	0.60	0.45	0.42	0.42
2	0.40	0.65	0.32	0.52	0.65	0.52	0.50	0.50
3	0.45	0.70	0.35	0.60	0.70	0.60	0.54	0.54
4	0.50	0.72	0.40	0.70	0.75	0.65	0.60	0.60
5	0.55	0.73	0.45	0.80	0.85	0.75	0.70	0.70
6	0.60	0.74	0.50	0.85	0.90	0.80	0.75	0.75
7	0.65	0.75	0.55	0.90	0.95	0.85	0.80	0.80
8	0.70	0.80	0.60	0.95	0.98	0.90	0.85	0.85

pSIA impact level	True coverage	Repeated missed probability
1	0.15	0.95
2	0.35	0.95
3	0.50	0.80
4	0.80	0.70
5	0.95	0.50

R_0	oSIA impact[a]			
	RI coverage (POL3) > 0.50		RI coverage (POL3) <0.50	
	True coverage	Repeated missed probability	True coverage	Repeated missed probability
4	0.90	0.50	0.90	0.50
5	0.90	0.50	0.90	0.50
6	0.90	0.50	0.90	0.50
7	0.70	0.80	0.70	0.80
8	0.70	0.80	0.70	0.80
9	0.70	0.80	0.70	0.80
10	0.80	0.70	0.70	0.80
11	0.80	0.70	0.70	0.80
12	0.80	0.70	0.80	0.70
13	0.80	0.70	0.80	0.70

Acronyms: bOPV(1,3), bivalent OPV (serotype 1 and serotype 3 component, respectively); mOPV(1,2.3), monovalent OPV (containing serotype 1, serotype 2, and serotype 3, respectively); OPV, oral polio vaccine; oSIA, outbreak response SIA; POL3, coverage with 3 or more non-birth RI doses; pSIA, planned, preventive SIA; R_0, average annual basic reproduction number for WPV 1; RI, routine immunization; SIA, supplemental immunization activity; tOPV(1,2,3), trivalent OPV (serotype 1, serotype 2, and serotype 3 component, respectively)
Footnote: [a] oSIA impact set equal to pSIA impact post OPV2 cessation, and one lever lower if OPV no longer available and IPV is used instead.

Table A4: Summary of immunization model inputs for the 720 subpopulations in the global poliovirus transmission model (list of symbols provided at bottom of table)

Preferential mixing area (PMA)	Block	Sub-popul-ation(s)	Income level	Polio vaccine at T_0	COV 1or2	POL 3	sil	SIA_1	SIA_2	SIA_3	SIA_4
Africa	1	1	LI	OPV+IPV	0.07	0.3[d]	3[d]	2[j]	2	2	2
Africa	1	2	LI	OPV+IPV	0.07	0.6	4	2	2	2	2
Africa	1	3-4	LI	OPV+IPV	0.07	0.9	4	1	2	2	2
Africa	1	5	LI	OPV+IPV	0.07	0.9	4	1	1	2	2
Africa	1	6	LI	OPV+IPV	0.07	0.9	4	1	1	2	1
Africa	1	7-8	LI	OPV+IPV	0.07	0.9	4	1	1	1	1
Africa	1	9	LI	OPV+IPV	0.07	0.9	4	1	0	0	0
Africa	1	10	LI	OPV+IPV	0.07	0.98	5	0	0	0	0
Africa	2	1	LI	OPV+IPV	0.23	0.3	4	2	2	2	2
Africa	2	2-5	LI	OPV+IPV	0.23	0.6	4	2	1	1	1
Africa	2	6	LI	OPV+IPV	0.23	0.9	4	2	1	1	1
Africa	2	7-10	LI	OPV+IPV	0.23	0.9	4	2	1	0	0
Africa	3	1	LI	OPV+IPV	0.30[e]	0.3[e]	3[e]	2[k]	3	2	2
Africa	3	2	LI	OPV+IPV	0.30	0.3	3	2[h]	2	2	2
Africa	3	3-4	LI	OPV+IPV	0.30	0.9	4	1	1	1	1
Africa	3	5-6	LI	OPV+IPV	0.30	0.9	4	1	1	1	0
Africa	3	7-8	LI	OPV+IPV	0.30	0.98	4	0	1	0	0
Africa	3	9-10	LI	OPV+IPV	0.30	0.98	4	0	0	0	0
Africa	4	1	LI	OPV+IPV	0.22	0.6	4	2	2	1	1
Africa	4	2	LI	OPV+IPV	0.22	0.6	4	2	2	0	0
Africa	4	3-6	LI	OPV+IPV	0.22	0.9	4	1	0	0	0
Africa	4	7-10	LI	OPV+IPV	0.22	0.98	4	0	0	0	0
Africa	5	1	LI	OPV+IPV	0.22	0.3	3	2	4	2	2
Africa	5	2-4	LI	OPV+IPV	0.22	0.6	3	2	4	2	2
Africa	5	5	LI	OPV+IPV	0.22	0.6	3	2	3	2	1
Africa	5	6-7	LI	OPV+IPV	0.22	0.9	4	2	2	1	1
Africa	5	8-9	LI	OPV+IPV	0.22	0.9	4	2	1	0	0
Africa	5	10	LI	OPV+IPV	0.22	0.9	4	2	0	0	0
Africa	6	1	LMI	OPV+IPV	0.10	0.6	3	3	0	0	0
Africa	6	2-5	LMI	OPV+IPV	0.10	0.98	4	0	0	0	0
Africa	6	6-10	LMI	OPV+IPV	0.10	0.98	5	0	0	0	0
Africa	7	1	LMI	OPV+IPV	0.15	0.3	2	1	3	2	2
Africa	7	2	LMI	OPV+IPV	0.15	0.3	2	1	2	2	2
Africa	7	3	LMI	OPV+IPV	0.15	0.3	2	1[i]	2	2	2
Africa	7	4	LMI	OPV+IPV	0.15	0.6	4	1	1	2	2
Africa	7	5	LMI	OPV+IPV	0.15	0.9	4	1	1	2	2
Africa	7	6-7	LMI	OPV+IPV	0.15	0.9	4	1	0	1	1
Africa	7	8-9	LMI	OPV+IPV	0.15	0.9	4	1	0	0	0
Africa	7	10	LMI	OPV+IPV	0.15	0.98	5	0	0	0	0
Africa	8	1	LMI	OPV+IPV	0.15	0.1	2	NA	6	4	3
Africa	8	2	LMI	OPV+IPV	0.15	0.1	2	NA	5	3	2
Africa	8	3-4	LMI	OPV+IPV	0.15	0.3	3	NA	5	3	2
Africa	8	5-6	LMI	OPV+IPV	0.15	0.6	3	NA	5	2	2

Africa	8	7-10	LMI	OPV+IPV	0.15	0.9	4	NA	4	1	1
Africa	9	1-2	LMI	OPV+IPV	0.15	0.3	4	3	5	2	2
Africa	9	3-5	LMI	OPV+IPV	0.15	0.6	4	3	5	2	2
Africa	9	6	LMI	OPV+IPV	0.15	0.6	4	3	4	2	2
Africa	9	7-8	LMI	OPV+IPV	0.15	0.6	4	3	4	2	1
Africa	9	9	LMI	OPV+IPV	0.15	0.9	4	1	4	2	1
Africa	9	10	LMI	OPV+IPV	0.15	0.9	4	1	4	1	1
Africa	10	1	LMI	OPV+IPV	0.10	0.6	4	3	1	0	0
Africa	10	2-3	LMI	OPV+IPV	0.10	0.9	4	1	1	0	0
Africa	10	4-5	LMI	OPV+IPV	0.10	0.9	4	1	0	0	0
Africa	10	6-10	LMI	OPV+IPV	0.10	0.98	5	0	0	0	0
Africa	11	1	UMI	OPV+IPV	0.26	0.6	3	3	1	1	1
Africa	11	2	UMI	OPV+IPV	0.26	0.9	4	1	1	1	1
Africa	11	3-4	UMI	OPV+IPV	0.26	0.98	5	0	1	1	1
Africa	11	5-10	UMI	OPV+IPV	0.26	0.98	5	0	0	0	0
Australasia	12	1-8	LMI	OPV+IPV	0.01	0.6	4	3	0	0	0
Australasia	12	9-10	LMI	OPV+IPV	0.01	0.9	5	1	0	0	0
Australasia	13	1	LMI	OPV+IPV	0.11	0.3	2	1[f]	1	1	1
Australasia	13	2	LMI	OPV+IPV	0.11	0.6	4	1	1	1	1
Australasia	13	3-7	LMI	OPV+IPV	0.11	0.9	4	1	1	1	1
Australasia	13	8	LMI	OPV+IPV	0.11	0.9	4	1	1	1	0
Australasia	13	9-10	LMI	OPV+IPV	0.11	0.98	5	0	0	0	0
Australasia	14-15	1-4	LMI	OPV+IPV	0.14	0.6	4	3	1	1	1
Australasia	14-15	5-10	LMI	OPV+IPV	0.14	0.9	4	1	1	1	1
Australasia	16	1	HI	IPV-only	0.09	0.9	4	1	0	0	0
Australasia	16	2-10	HI	IPV-only	0.09	0.98	5	0	0	0	0
Australasia	17	1-10	HI	IPV-only	0.00	0.98	5	0	0	0	0
China and neighbors	18	1-10	UMI	OPV+IPV	0.08	0.98	5	0	0	0	0
China and neighbors	19	1-10	UMI	OPV+IPV	0.00	0.98	5	0	0	0	0
China and neighbors	20	1-10	UMI	OPV+IPV	0.00	0.9	4	1	0	0	0
China and neighbors	21-31	1-10	UMI	OPV+IPV	0.00	0.98	5	0	0	0	0
East and Central Asia	32	1	LI	OPV+IPV	0.14	0.3	2[b]	NA	NA	7	4
East and Central Asia	32	2	LI	OPV+IPV	0.14	0.6	4	NA	NA	7	4
East and Central Asia	32	3	LI	OPV+IPV	0.14	0.6	4	NA	NA	6	4
East and Central Asia	32	4	LI	OPV+IPV	0.14	0.6	4	NA	NA	6	3
East and Central Asia	32	5-6	LI	OPV+IPV	0.14	0.6	4	NA	NA	6	2
East and Central Asia	32	7-10	LI	OPV+IPV	0.14	0.9	4	NA	NA	6	2
East and Central Asia	33	1	LMI	OPV+IPV	0.21	0.6	3	1[g]	0	0	0
East and Central Asia	33	2-10	LMI	OPV+IPV	0.21	0.98	5	0	0	0	0
East and Central Asia	34	1	LMI	OPV+IPV	0.16	0.3	2[b]	NA	8	6	4
East and Central Asia	34	2-4	LMI	OPV+IPV	0.16	0.6	3	NA	8	6	3
East and Central Asia	34	5-6	LMI	OPV+IPV	0.16	0.6	3	NA	8	6	2
East and Central Asia	34	7	LMI	OPV+IPV	0.16	0.9	4	NA	8	6	2
East and Central Asia	34	8-10	LMI	OPV+IPV	0.16	0.9	4	NA	8	6	1
East and Central Asia	35	1	UMI	OPV+IPV	0.09	0.6	4	3	3	3	2
East and Central Asia	35	2	UMI	OPV+IPV	0.09	0.9	4	1	3	3	2
East and Central Asia	35	3	UMI	OPV+IPV	0.09	0.98	5	0	2	0	0
East and Central Asia	35	4-10	UMI	OPV+IPV	0.09	0.98	5	0	0	0	0

East and Central Asia	36	1	UMI	IPV/OPV	0.10	0.9[c]	2[c]	1	2	2	2
East and Central Asia	36	2	UMI	IPV/OPV	0.10	0.9	2	1	2	2	2
East and Central Asia	36	3	UMI	IPV/OPV	0.10	0.98	4	0	1	1	0
East and Central Asia	36	4-10	UMI	IPV/OPV	0.10	0.98	5	0	0	0	0
East and Central Asia	37	1-10	UMI	IPV/OPV	0.25	0.98	5	0	0	0	0
Europe	38	1-2	LMI	IPV/OPV	0.20	0.3	4	3	0	0	0
Europe	38	3-5	LMI	IPV/OPV	0.20	0.6	4	3	0	0	0
Europe	38	6-7	LMI	IPV/OPV	0.20	0.9	5	1	0	0	0
Europe	38	8-10	LMI	IPV/OPV	0.20	0.98	5	0	0	0	0
Europe	39-40	1-2	UMI	IPV/OPV	0.00	0.9	4	1	0	0	0
Europe	39-40	3-10	UMI	IPV/OPV	0.00	0.98	5	0	0	0	0
Europe	41	1-5	HI	IPV-only	0.23	0.9	4	1	0	0	0
Europe	41	6-10	HI	IPV-only	0.23	0.98	5	0	0	0	0
Europe	42	1-5	HI	IPV-only	0.31	0.9	4	1	0	0	0
Europe	42	6-10	HI	IPV-only	0.31	0.98	5	0	0	0	0
Europe	43	1-5	HI	IPV-only	0.24	0.9	4	1	0	0	0
Europe	43	6-10	HI	IPV-only	0.24	0.98	5	0	0	0	0
Europe	44	1-4	HI	IPV-only	0.25	0.9	4	1	0	0	0
Europe	44	5-10	HI	IPV-only	0.25	0.98	5	0	0	0	0
Europe	45	1-8	HI	IPV-only	0.34	0.9	4	1	0	0	0
Europe	45	2-10	HI	IPV-only	0.34	0.98	5	0	0	0	0
Europe	46	1-2	HI	IPV-only	0.32	0.9	4	1	0	0	0
Europe	46	3-10	HI	IPV-only	0.32	0.98	5	0	0	0	0
India	47-48	1	LMI	OPV+IPV	0.14	0.3	2[a]	5	3	3	3
India	47-48	2-10	LMI	OPV+IPV	0.14	0.6	5	5	3	3	3
India	49-56	1-5	LMI	OPV+IPV	0.14	0.9	5	3	3	2	2
India	49-56	6-9	LMI	OPV+IPV	0.14	0.98	5	1	3	2	2
India	49-56	10	LMI	OPV+IPV	0.14	0.98	5	1	2	2	2
India	57-58	1-5	LMI	OPV+IPV	0.14	0.9	5	1	3	2	2
India	57-58	6-9	LMI	OPV+IPV	0.14	0.98	5	0	3	2	2
India	57-58	10	LMI	OPV+IPV	0.14	0.98	5	0	2	2	2
Latin America and Caribbean	59	1-2	LMI	OPV+IPV	0.22	0.6	3	3	0	0	0
Latin America and Caribbean	59	3-6	LMI	OPV+IPV	0.22	0.9	4	1	0	0	0
Latin America and Caribbean	59	7-10	LMI	OPV+IPV	0.22	0.98	5	0	0	0	0
Latin America and Caribbean	60	1-3	UMI	OPV+IPV	0.10	0.6	4	3	0	0	0
Latin America and Caribbean	60	4-10	UMI	OPV+IPV	0.10	0.9	5	1	0	0	0
Latin America and Caribbean	61	1	UMI	OPV+IPV	0.23	0.6	4	3	0	0	0
Latin America and Caribbean	61	2-9	UMI	OPV+IPV	0.23	0.9	5	1	0	0	0
Latin America and Caribbean	61	10	UMI	OPV+IPV	0.23	0.98	5	0	0	0	0
Latin America and Caribbean	62	1-2	UMI	IPV/OPV	0.14	0.6	4	3	0	0	0
Latin America and Caribbean	62	3-10	UMI	IPV/OPV	0.14	0.9	5	1	0	0	0
Latin America and Caribbean	63	1-2	UMI	IPV/OPV	0.12	0.6	4	3	0	0	0
Latin America and Caribbean	63	3-10	UMI	IPV/OPV	0.12	0.9	5	1	0	0	0
Latin America and Caribbean	64	1	UMI	IPV-only	0.09	0.6	4	3	0	0	0
Latin America and Caribbean	64	2-10	UMI	IPV-only	0.09	0.9	5	1	0	0	0
North America	65	1-6	HI	IPV-only	0.24	0.9	4	1	0	0	0
North America	65	7-10	HI	IPV-only	0.24	0.98	5	0	0	0	0
North America	66-67	1-5	HI	IPV-only	0.25	0.9	4	1	0	0	0

North America	66-67	6-10	HI	IPV-only	0.25	0.98	5	0	0	0	0
South Asia	68	1	LMI	OPV+IPV	0.26	0.6	3	5	1	0	0
South Asia	68	2	LMI	OPV+IPV	0.26	0.9	4	3	0	0	0
South Asia	68	3-10	LMI	OPV+IPV	0.26	0.98	5	1	0	0	0
South Asia	69	1	LMI	OPV+IPV	0.24	0.6	4	5	1	1	0
South Asia	69	2	LMI	OPV+IPV	0.24	0.9	4	3	1	0	0
South Asia	69	3-10	LMI	OPV+IPV	0.24	0.98	5	1	0	0	0
South Asia	70	1	LMI	OPV+IPV	0.07	0.9	4	3	1	0	0
South Asia	70	2-4	LMI	OPV+IPV	0.07	0.9	4	3	0	0	0
South Asia	70	5-9	LMI	OPV+IPV	0.07	0.9	4	1	0	0	0
South Asia	70	10	LMI	OPV+IPV	0.07	0.98	5	0	0	0	0
South Asia	71	1-10	UMI	OPV+IPV	0.33	0.98	5	0	0	0	0
South Asia	72	1-3	UMI	IPV/OPV	0.10	0.6	4	3	0	0	0
South Asia	72	4-7	UMI	IPV/OPV	0.10	0.9	5	1	0	0	0
South Asia	72	8-10	UMI	IPV/OPV	0.10	0.98	5	0	0	0	0

Model input symbols: COV1or2, RI coverage with 1 or 2 non-birth doses; POL3, RI coverage with 3 or more non-birth doses; sil, SIA impact level (see Table A4); SIA_x, number of planned, preventive SIAs per year in period x = 1, 2, 3, 4, where 1 = [all-WPV-elimination, OPV2 -cessation], 2 = (OPV2-cessation, T_0), 3 = [T_0, T_0+3), 4 = [T_0+3, T_0+6) ; T_0, January 1, 2019.

Acronyms: HI, high-income; IPV, inactivated poliovirus vaccine; LMI, lower middle-income; LI, low-income; OPV, oral poliovirus vaccine; SIA, supplemental immunization activity; UMI, upper middle-income

Footnote: [a] Assumed to increase to SIA impact level 4 in T_0-9 as a result of intensification of efforts to interrupt wild poliovirus transmission in these reservoirs; [b] Assumed to increase to SIA impact level 3 from T_0-8.3 to T_0-8 as a result of intensification of efforts to interrupt wild poliovirus transmission in these reservoirs; [c] Assumed to decrease to SIA impact level 1 and POL3 of 0.05 in T_0-7.1 as a result of political conflict; [d] Assumed to decrease to SIA impact level 1 and POL3 of 0.05 in T_0-3.3 as a result of lack of efforts post WPV elimination in the region; [e] Assumed to decrease to SIA impact level 1, POL3 of 0.05 and COV1or2 of 0.10 in T_0-3.5 as a result of lack of efforts post WPV elimination in the region; [f] Assumed to decrease to SIA_1=0 in T_0-15, [g] Assumed to decrease to SIA_1=0 in T_0-15; [h] Assumed to decrease to SIA_1=0 in T_0-11; [I] Assumed to decrease to SIA_1=0 in T_0-11; [j] Assumed to decrease to SIA_1=0 in T_0-9; [k] Assumed to decrease to SIA_1=0 in T_0-6.

Table A5: Summary of surveillance model inputs for the 720 subpopulations in the global poliovirus transmission model (list of symbols provided at bottom of table)

Preferential mixing area (PMA)	Block	Sub-popul-ation(s)	Income level	Polio vaccine at T_0	dt_1	dt_2	dt_3	dt_4	T_{ES}	Q_{ES}	tcp_1	tcp_2	tcp_3
Africa	1	1	LOW	OPV-only	3	5	13	30	-1.3	L	3	0	0
Africa	1	2-4	LOW	OPV-only	2	4	13	30	-1.3	M	3	3	0
Africa	1	5-7	LOW	OPV-only	2	4	13	30	-	-	0	0	0
Africa	1	8	LOW	OPV-only	2	4	13	30	-1.0	M	9	9	0
Africa	1	9	LOW	OPV-only	2	4	13	30	-	-	0	0	0
Africa	1	10	LOW	OPV-only	1	3	12	30	-	-	0	0	0
Africa	2	1-2	LOW	OPV-only	2	4	13	30	-1.9	M	2	2	0
Africa	2	3-4	LOW	OPV-only	2	4	13	30	-1.9	M	1	1	0
Africa	2	5-10	LOW	OPV-only	2	4	13	30	-	-	0	0	0
Africa	3	1	LOW	OPV-only	3	5	13	30	-1.3	L	4	0	0
Africa	3	2-3	LOW	OPV-only	3	5	13	30	-1.7	L	4	0	0
Africa	3	4	LOW	OPV-only	3	5	13	30	-	-	0	0	0
Africa	3	5	LOW	OPV-only	2	4	13	30	-	-	0	0	0
Africa	3	6	LOW	OPV-only	2	4	13	30	-5.3	M	5	5	0
Africa	3	7-9	LOW	OPV-only	2	4	13	30	-5.3	M	4	4	0
Africa	3	10	LOW	OPV-only	2	4	13	30	-	-	0	0	0
Africa	4	1-2	LOW	OPV-only	2	4	13	30	-2.5	M	10	10	0
Africa	4	3	LOW	OPV-only	1	3	12	30	-1.6	H	4	4	4
Africa	4	4-6	LOW	OPV-only	1	3	12	30	-	-	0	0	0
Africa	4	7	LOW	OPV-only	1	3	12	30	0.0	H	4	4	4
Africa	4	8-10	LOW	OPV-only	1	3	12	30	-	-	0	0	0
Africa	5	1	LOW	OPV-only	3	5	13	30	-3.2	L	10	0	0
Africa	5	2	LOW	OPV-only	2	4	13	30	-	-	0	0	0
Africa	5	3	LOW	OPV-only	2	4	13	30	-2.9	M	5	5	0
Africa	5	4	LOW	OPV-only	2	4	13	30	-2.8	M	4	4	0
Africa	5	5	LOW	OPV-only	2	4	13	30	-	-	0	0	0
Africa	5	6	LOW	OPV-only	1	3	12	30	-1.8	H	5	5	5
Africa	5	7	LOW	OPV-only	1	3	12	30	-1.3	H	4	4	4
Africa	5	8	LOW	OPV-only	1	3	12	30	0.0	H	2	2	2
Africa	5	9-10	LOW	OPV-only	1	3	12	30	-	-	0	0	0
Africa	6	1-5	LMI	OPV-only	2	4	9	20	-3.0	M	5	5	0
Africa	6	6-8	LMI	OPV-only	1	3	9	20	-3.0	H	5	5	5
Africa	6	9-10	LMI	OPV-only	1	3	9	20	-3.0	H	4	4	4
Africa	7	1-3	LMI	OPV-only	2	4	9	20	-2.1	M	3	3	0
Africa	7	4	LMI	OPV-only	2	4	9	20	-0.8	M	10	10	0
Africa	7	5	LMI	OPV-only	2	4	9	20	-	-	0	0	0
Africa	7	6	LMI	OPV-only	2	4	9	20	-1.0	M	10	10	0
Africa	7	7-8	LMI	OPV-only	2	4	9	20	-	-	0	0	0
Africa	7	9-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
Africa	8	1	LMI	OPV-only	3	5	10	20	-7.5	L	9	0	0
Africa	8	2	LMI	OPV-only	2	4	9	20	-7.5	M	9	9	0
Africa	8	3-10	LMI	OPV-only	2	4	9	20	-7.5	M	8	8	0
Africa	9	1-4	LMI	OPV-only	2	4	9	20	-7.5	M	8	8	0

Africa	9	5-8	LMI	OPV-only	2	4	9	20	-	-	0	0	0
Africa	9	9-10	LMI	OPV-only	2	4	9	20	-3.0	M	15	15	0
Africa	10	1	LMI	OPV-only	2	4	9	20	-3.8	M	2	2	0
Africa	10	2	LMI	OPV-only	2	4	9	20	-	-	0	0	0
Africa	10	3	LMI	OPV-only	2	4	9	20	-2.1	M	7	7	0
Africa	10	4	LMI	OPV-only	2	4	9	20	-	-	0	0	0
Africa	10	5	LMI	OPV-only	2	4	9	20	0.0	M	10	10	0
Africa	10	6-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
Africa	11	1	UMI	OPV-only	2	4	6	10	-1.4	M	4	4	0
Africa	11	2	UMI	OPV-only	2	4	6	10	-	-	0	0	0
Africa	11	3-4	UMI	OPV-only	1	3	5	10	-	-	0	0	0
Africa	11	5	UMI	OPV-only	1	3	5	10	0.0	H	5	5	5
Africa	11	6-10	UMI	OPV-only	1	3	5	10	-	-	0	0	0
Australasia	12	1	LMI	OPV-only	2	4	9	20	0.0	M	5	5	0
Australasia	12	2	LMI	OPV-only	1	3	9	20	0.0	H	5	5	5
Australasia	12	3-4	LMI	OPV-only	1	3	9	20	0.0	H	4	4	4
Australasia	12	5-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
Australasia	13	1	LMI	OPV-only	2	4	9	20	-0.3	M	4	4	0
Australasia	13	2-3	LMI	OPV-only	2	4	9	20	-2.0	M	2	2	0
Australasia	13	4-8	LMI	OPV-only	2	4	9	20	-	-	0	0	0
Australasia	13	9-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
Australasia	14	1-4	LMI	OPV-only	2	4	9	20	-2.0	M	1	1	0
Australasia	14	5-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
Australasia	15	1-4	LMI	OPV-only	2	4	9	20	-2.0	M	1	1	0
Australasia	15	5-9	LMI	OPV-only	1	3	9	20	-	-	0	0	0
Australasia	15	10	LMI	OPV-only	1	3	9	20	3.0	H	1	1	1
Australasia	16	1	HIGH	IPV-only	2	5	5	5	-1.3	M	1	1	0
Australasia	16	2	HIGH	IPV-only	1	5	5	5	3.0	H	1	1	1
Australasia	16	3-7	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
Australasia	16	8	HIGH	IPV-only	1	5	5	5	3.0	H	1	1	1
Australasia	16	9-10	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
Australasia	17	1-3	HIGH	IPV-only	1	5	5	5	-36	H	4	4	4
Australasia	17	4-5	HIGH	IPV-only	1	5	5	5	-36	H	3	3	3
Australasia	17	6	HIGH	IPV-only	1	5	5	5	3.0	H	1	1	1
Australasia	17	7-10	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
China and neighbors	18	1	UMI	OPV-only	1	3	5	10	-11	H	2	2	2
China and neighbors	18	2-10	UMI	OPV-only	1	3	5	10	-	-	0	0	0
China and neighbors	19	1	UMI	OPV-only	1	3	5	10	-11	H	1	1	1
China and neighbors	19	2-6	UMI	OPV-only	1	3	5	10	3.0	H	1	1	1
China and neighbors	19	7-10	UMI	OPV-only	1	3	5	10	-	-	0	0	0
China and neighbors	20	1	UMI	OPV-only	2	4	6	10	3.0	M	1	1	1
China and neighbors	20	2-10	UMI	OPV-only	2	4	6	10	-	-	0	0	0
China and neighbors	21-31	1	UMI	OPV-only	1	3	5	10	3.0	H	1	1	1
China and neighbors	21-31	2-10	UMI	OPV-only	1	3	5	10	-	-	0	0	0
East and Central Asia	32	1	LOW	OPV-only	3	5	13	30	-5.3	L	11	0	0
East and Central Asia	32	2	LOW	OPV-only	2	4	13	30	-5.3	M	10	10	0
East and Central Asia	32	3	LOW	OPV-only	2	4	13	30	-	-	0	0	0
East and Central Asia	32	4-6	LOW	OPV-only	2	4	13	30	-9.1	M	5	5	0

East and Central Asia	32	7-10	LOW	OPV-only	1	3	12	30	-9.1	H	5	5	5
East and Central Asia	33	1	LMI	OPV-only	2	4	9	20	-	-	0	0	0
East and Central Asia	33	2-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
East and Central Asia	34	1	LMI	OPV-only	3	5	10	20	-9.1	L	4	4	4
East and Central Asia	34	2-3	LMI	OPV-only	2	4	9	20	-9.1	M	4	4	0
East and Central Asia	34	4-6	LMI	OPV-only	2	4	9	20	-9.1	M	3	3	0
East and Central Asia	34	7	LMI	OPV-only	1	3	9	20	-9.1	H	3	3	3
East and Central Asia	34	8-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
East and Central Asia	35	1-2	UMI	OPV-only	2	4	6	10	-	-	0	0	0
East and Central Asia	35	3-4	UMI	OPV-only	1	3	5	10	-	-	0	0	0
East and Central Asia	35	5	UMI	OPV-only	1	3	5	10	-1.0	H	5	5	5
East and Central Asia	35	6-10	UMI	OPV-only	1	3	5	10	-	-	0	0	0
East and Central Asia	36	1	UMI	IPV/OPV	3	5	7	10	-1.0	L	16	0	0
East and Central Asia	36	2	UMI	IPV/OPV	2	4	6	10	-	-	0	0	0
East and Central Asia	36	3	UMI	IPV/OPV	2	4	6	10	-1.0	M	4	4	0
East and Central Asia	36	4	UMI	IPV/OPV	1	3	5	10	-1.0	H	4	4	4
East and Central Asia	36	5-10	UMI	IPV/OPV	1	3	5	10	-	-	0	0	0
East and Central Asia	37	1-10	UMI	IPV/OPV	2	4	6	10	-	-	0	0	0
Europe	38	1-5	LMI	IPV/OPV	2	4	9	20	-	-	0	0	0
Europe	38	6-7	LMI	IPV/OPV	1	3	9	20	-	-	0	0	0
Europe	38	8	LMI	IPV/OPV	1	3	9	20	3.0	H	1	1	1
Europe	38	9-10	LMI	IPV/OPV	1	3	9	20	-	-	0	0	0
Europe	39	1-2	UMI	IPV/OPV	2	4	6	10	-2.3	M	1	1	1
Europe	39	3-10	UMI	IPV/OPV	1	3	5	10	-	-	0	0	0
Europe	40	1	UMI	IPV/OPV	2	4	6	10	-2.3	M	1	1	1
Europe	40	2	UMI	IPV/OPV	2	4	6	10	3.0	M	1	1	1
Europe	40	3	UMI	IPV/OPV	1	3	5	10	3.0	H	1	1	1
Europe	40	4-10	UMI	IPV/OPV	1	3	5	10	-	-	0	0	0
Europe	41	1	HIGH	IPV-only	2	5	5	5	3.0	M	1	1	1
Europe	41	2-4	HIGH	IPV-only	2	5	5	5	-	-	0	0	0
Europe	41	5	HIGH	IPV-only	2	5	5	5	3.0	M	1	1	1
Europe	41	6	HIGH	IPV-only	1	5	5	5	3.0	H	1	1	1
Europe	41	7-10	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
Europe	42	1-5	HIGH	IPV-only	2	5	5	5	3.0	M	1	1	1
Europe	42	6-9	HIGH	IPV-only	1	5	5	5	3.0	H	1	1	1
Europe	42	9-0	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
Europe	43	1-5	HIGH	IPV-only	2	5	5	5	-	-	0	0	0
Europe	43	6-10	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
Europe	44	1	HIGH	IPV-only	2	5	5	5	-31	M	1	1	0
Europe	44	2-3	HIGH	IPV-only	2	5	5	5	-	-	0	0	0
Europe	44	4	HIGH	IPV-only	2	5	5	5	3.0	M	1	1	1
Europe	44	5-10	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
Europe	45	1-8	HIGH	IPV-only	2	5	5	5	-	-	0	0	0
Europe	45	9-10	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
Europe	46	1	HIGH	IPV-only	2	5	5	5	3.0	M	1	1	1
Europe	46	2	HIGH	IPV-only	2	5	5	5	-	-	0	0	0
Europe	46	3	HIGH	IPV-only	1	5	5	5	3.0	H	1	1	1
Europe	46	4-8	HIGH	IPV-only	1	5	5	5	-	-	0	0	0

Europe	46	9	HIGH	IPV-only	1	5	5	5	3.0	H	1	1	1
Europe	46	10	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
India	47-50	1	LMI	OPV-only	1	3	9	20	-2.8	H	7	7	7
India	47-50	2-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
India	51-54	1	LMI	OPV-only	1	3	9	20	-2.8	H	6	6	6
India	51-54	2-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
India	55-58	1-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
Latin America and Caribbean	59	1-2	LMI	OPV-only	3	5	10	20	-3.0	L	8	0	0
Latin America and Caribbean	59	3-7	LMI	OPV-only	2	4	9	20	-	-	0	0	0
Latin America and Caribbean	59	8	LMI	OPV-only	1	3	9	20	3.0	H	1	1	1
Latin America and Caribbean	59	9-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
Latin America and Caribbean	60	1-10	UMI	OPV-only	2	4	6	10	-	-	0	0	0
Latin America and Caribbean	61	1	UMI	OPV-only	2	4	6	10	-	-	0	0	0
Latin America and Caribbean	61	2-7	UMI	OPV-only	1	3	5	10	-	-	0	0	0
Latin America and Caribbean	61	8	UMI	OPV-only	1	3	5	10	3.0	H	1	1	1
Latin America and Caribbean	61	9-10	UMI	OPV-only	1	3	5	10	-	-	0	0	0
Latin America and Caribbean	62	1	UMI	IPV/OPV	2	4	6	10	3.0	M	1	1	1
Latin America and Caribbean	62	2-10	UMI	IPV/OPV	2	4	6	10	-	-	0	0	0
Latin America and Caribbean	63	1	UMI	IPV/OPV	2	4	6	10	3.0	M	1	1	1
Latin America and Caribbean	63	2-10	UMI	IPV/OPV	2	4	6	10	-	-	0	0	0
Latin America and Caribbean	64	1	UMI	IPV-only	2	4	6	10	3.0	M	1	1	1
Latin America and Caribbean	64	2-5	UMI	IPV-only	2	4	6	10	-	-	0	0	0
Latin America and Caribbean	64	6-10	UMI	IPV-only	1	3	5	10	-	-	0	0	0
North America	65	1-6	HIGH	IPV-only	2	5	5	5	3.0	M	1	1	1
North America	65	7-10	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
North America	66	1-5	HIGH	IPV-only	2	5	5	5	3.0	M	1	1	1
North America	66	6-10	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
North America	67	1-5	HIGH	IPV-only	2	5	5	5	3.0	M	1	1	1
North America	67	6-10	HIGH	IPV-only	1	5	5	5	-	-	0	0	0
South Asia	68	1	LMI	OPV-only	2	4	9	20	-2.8	M	8	8	0
South Asia	68	2	LMI	OPV-only	2	4	9	20	-1.0	M	5	5	0
South Asia	68	3-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
South Asia	69	1-2	LMI	OPV-only	2	4	9	20	-1.0	M	2	2	0
South Asia	69	3-10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
South Asia	70	1	LMI	OPV-only	2	4	9	20	1.0	M	3	3	3
South Asia	70	2-19	LMI	OPV-only	2	4	9	20	-	-	0	0	0
South Asia	70	10	LMI	OPV-only	1	3	9	20	-	-	0	0	0
South Asia	71	1-3	UMI	OPV-only	1	3	5	10	-2.0	H	2	2	2
South Asia	71	4	UMI	OPV-only	1	3	5	10	-2.0	H	1	1	1
South Asia	71	5-10	UMI	OPV-only	1	3	5	10	-	-	0	0	0
South Asia	72	1	UMI	IPV/OPV	2	4	6	10	0.0	M	6	6	6
South Asia	72	2-4	UMI	IPV/OPV	2	4	6	10	-	-	0	0	0
South Asia	72	5	UMI	IPV/OPV	2	4	6	10	0.0	M	3	3	0
South Asia	72	6-7	UMI	IPV/OPV	2	4	6	10	-	-	0	0	0
South Asia	72	8-10	UMI	IPV/OPV	1	3	5	10	-	-	0	0	0

Model input symbols: dt_x, detection threshold in period x = 1, 2, 3, 4, where 1 = [T_0-49, T_0), 2 = [T_0, T_0+6), 3 = [T_0+6, T_0+9), 4 = [T_0+9, T_{end})), defined as the cumulative incidence of paralytic polio cases per 10 million people required to detect an outbreak due to a poliovirus introduction in a subpopulation [3]; T_0, January 1, 2019; T_{ES}, year

when ES is introduced to the subpopulation relative to T_0; Q_{ES}, quality of ES; tcp_x, total ES coverage percentage in period x = 1, 2, 3, where 1 = [T_{ES}, T_0+6), 2 = [T_0+6, T_0+9), 3 = [T_0+9, T_{end})
Acronyms: ES, environmental surveillance; HI, high-income; IPV, inactivated poliovirus vaccine; LMI, lower middle-income; LI, low-income; OPV, oral poliovirus vaccine; UMI, upper middle-income

Table A6: Relative coverage for the 72 blocks the global poliovirus transmission model over time relative to T_0, based on 1980 to 2018 (WUENIC) estimates [40].

a) Years 1980 to 1998 (T_0-39 to T_0-20)

Preferential mixing area (PMA)	Block	-39	-38	-37	-36	-35	-34	-33	-32	-31	-30	-29	-28	-27	-26	-25	-24	-23	-22	-21	-20
Africa	1	17	23	25	28	36	45	48	55	56	58	57	40	44	47	35	43	39	37	37	45
Africa	2	3	4	4	5	5	8	9	22	22	36	68	29	18	38	79	79	76	76	56	37
Africa	3	24	28	33	37	40	48	51	59	65	66	68	71	74	76	80	81	77	73	74	76
Africa	4	49	53	49	51	52	56	63	71	76	76	77	79	83	83	84	83	83	82	83	81
Africa	5	11	13	14	15	16	17	23	28	34	44	58	54	54	56	56	56	56	57	56	56
Africa	6	60	60	60	60	72	88	84	85	91	94	91	80	83	85	87	92	94	92	96	100
Africa	7	21	24	28	31	36	42	45	52	62	75	92	73	73	79	70	71	73	73	88	75
Africa	8	9	11	12	14	15	28	38	50	63	82	98	68	75	50	77	59	45	36	56	54
Africa	9	8	9	14	18	20	31	41	52	63	79	87	64	67	49	69	59	49	43	60	59
Africa	10	17	25	28	33	34	39	50	60	61	62	68	65	72	73	74	80	81	79	79	81
Africa	11	27	31	35	40	43	48	53	63	72	82	84	84	80	79	77	78	82	82	76	79
Australasia	12	72	83	83	84	87	90	100	100	100	100	100	100	100	100	100	100	100	100	100	100
Australasia	13	15	15	18	24	31	44	63	67	72	76	80	82	83	85	87	89	91	92	93	95
Australasia	14	0	1	1	7	16	34	59	63	68	72	75	78	81	83	86	87	91	92	93	94
Australasia	15	0	1	1	7	16	34	59	63	68	72	75	78	81	83	86	87	91	92	93	94
Australasia	16	57	69	75	76	79	80	83	85	87	90	92	93	89	88	84	80	97	75	79	84
Australasia	17	53	69	72	75	80	80	81	72	73	89	83	86	86	87	87	87	95	76	75	84
China and neighbors	18	41	47	52	57	72	73	74	79	94	96	98	95	91	89	84	77	76	73	75	77
China and neighbors	19	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	20	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	21	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	22	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	23	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	24	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	25	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	26	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	27	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	28	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	29	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	30	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
China and neighbors	31	36	43	51	58	73	78	78	75	95	95	97	94	91	88	85	80	83	84	84	85
East and Central Asia	32	3	4	6	16	28	34	36	45	51	52	62	57	48	42	42	63	57	61	62	65
East and Central Asia	33	45	48	52	55	58	61	65	68	71	74	78	81	84	59	76	89	92	93	95	96
East and Central Asia	34	2	4	6	20	30	40	45	48	50	53	72	66	56	49	52	77	61	69	72	76
East and Central Asia	35	30	28	38	31	48	66	72	73	93	88	93	90	95	95	93	95	97	97	97	98
East and Central Asia	36	34	50	53	59	67	75	94	87	94	100	100	100	100	100	100	100	100	100	100	100
East and Central Asia	37	42	65	59	57	57	56	45	87	87	80	85	73	78	77	88	68	73	80	82	80

Europe	38	65	69	74	78	82	87	91	95	100	100	100	100	100	100	100	100	100	100	100	100
Europe	39	40	43	46	49	52	54	57	60	63	66	69	72	75	81	90	80	86	90	93	97
Europe	40	40	43	46	49	52	54	57	60	63	66	69	72	75	81	90	80	86	90	93	97
Europe	41	80	83	85	88	91	93	93	95	97	98	99	100	100	100	100	100	100	100	100	100
Europe	42	55	59	65	70	75	75	76	80	82	85	89	93	96	97	97	99	98	97	98	98
Europe	43	63	72	81	89	97	82	92	82	96	97	91	93	91	93	95	96	96	99	100	100
Europe	44	58	65	66	67	68	70	77	80	83	82	85	97	98	98	98	98	99	98	98	98
Europe	45	79	100	100	100	100	87	97	87	87	87	87	87	92	70	80	85	91	94	95	100
Europe	46	84	85	90	94	97	98	99	94	86	98	98	98	98	98	99	99	100	99	97	99
India	47	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	48	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	49	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	50	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	51	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	52	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	53	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	54	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	55	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	56	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	57	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
India	58	6	6	13	15	17	20	22	33	44	56	78	64	62	68	75	79	74	69	68	66
Latin America and Caribbean	59	40	43	51	49	50	52	66	60	73	76	81	81	82	88	88	90	86	89	90	90
Latin America and Caribbean	60	34	39	45	56	61	66	63	70	80	83	95	91	94	100	96	98	93	94	79	93
Latin America and Caribbean	61	54	60	66	67	71	77	76	82	84	88	91	87	88	88	91	94	92	94	93	98
Latin America and Caribbean	62	37	46	55	58	66	72	66	64	71	69	82	91	90	94	93	100	98	99	100	100
Latin America and Caribbean	63	44	56	67	72	81	79	69	69	69	67	79	93	85	90	89	97	92	95	100	100
Latin America and Caribbean	64	51	51	47	48	60	48	59	71	69	74	62	99	100	93	100	100	100	100	100	100
North America	65	84	86	88	91	93	94	97	99	100	98	96	93	91	95	100	99	100	99	100	100
North America	66	100	100	100	100	100	100	100	100	100	100	95	91	88	93	100	100	100	100	100	100
North America	67	100	100	100	100	100	100	100	100	100	100	95	91	88	93	100	100	100	100	100	100
South Asia	68	2	5	5	7	8	10	13	19	24	49	64	69	64	70	79	67	76	81	78	79
South Asia	69	2	2	4	5	5	7	10	15	21	51	80	74	71	77	85	78	85	86	86	81
South Asia	70	4	5	6	7	8	48	52	59	82	100	100	100	99	100	100	100	100	100	100	100
South Asia	71	50	53	50	50	54	63	68	77	82	86	94	92	87	91	95	98	96	97	98	100
South Asia	72	64	70	80	81	81	82	80	85	83	91	94	100	100	100	97	97	98	100	99	99

b) Years 1999 to 2017 (T_0-19 to T_0)

Preferential mixing area (PMA)	Block	-19	-18	-17	-16	-15	-14	-13	-12	-11	-10	-9	-8	-7	-6	-5	-4	-3	-2	-1	0
Africa	1	60	49	58	62	73	80	83	91	85	92	81	94	95	93	99	100	98	100	100	100
Africa	2	41	44	48	51	55	61	63	69	75	80	84	90	86	81	84	100	100	100	100	100
Africa	3	76	76	81	76	76	80	80	87	88	93	95	98	98	94	96	96	98	95	100	100
Africa	4	82	88	86	94	96	95	96	91	92	91	94	96	96	96	99	98	98	98	100	100
Africa	5	63	66	73	78	79	85	90	92	94	96	99	98	98	96	95	98	99	100	100	100
Africa	6	100	100	100	100	100	100	100	100	100	100	100	100	97	100	98	97	100	98	100	100
Africa	7	81	84	83	80	86	88	85	100	100	99	100	94	95	99	100	100	100	98	100	100
Africa	8	50	47	43	50	57	63	70	73	92	100	94	84	73	75	75	78	100	100	100	100
Africa	9	58	56	54	62	67	73	79	81	96	100	97	89	82	85	84	86	100	100	100	100
Africa	10	85	84	85	85	91	94	95	98	97	98	100	94	100	97	99	97	100	100	100	100
Africa	11	84	87	83	87	89	92	95	98	98	96	100	100	100	100	100	100	100	100	100	100
Australasia	12	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	92	100	100	100	100
Australasia	13	95	96	91	93	92	94	94	95	98	99	100	100	100	100	100	100	100	100	100	100
Australasia	14	94	96	88	89	89	91	91	92	97	98	100	100	100	100	98	98	100	100	100	100
Australasia	15	94	96	88	89	89	91	91	92	97	98	100	100	100	100	98	98	100	100	100	100
Australasia	16	89	96	96	98	99	98	98	98	98	97	97	98	97	97	97	97	99	100	100	100
Australasia	17	92	97	97	98	94	98	99	95	97	96	96	99	99	98	98	98	100	99	100	100
China and neighbors	18	79	81	82	83	84	86	93	94	97	98	98	99	99	98	98	99	99	100	100	100
China and neighbors	19	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	20	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	21	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	22	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	23	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	24	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	25	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	26	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	27	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	28	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	29	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	30	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
China and neighbors	31	85	86	86	86	87	87	93	93	97	100	100	100	100	100	100	100	100	100	100	100
East and Central Asia	32	66	72	75	80	86	85	81	78	78	76	78	89	90	89	92	96	100	100	100	100
East and Central Asia	33	96	96	96	95	97	96	94	95	96	96	97	99	99	100	100	100	100	100	100	100
East and Central Asia	34	78	81	84	88	90	84	78	72	70	69	69	84	85	86	92	96	100	100	100	100
East and Central Asia	35	98	95	97	96	96	90	91	91	94	97	96	98	96	95	96	94	96	100	100	100
East and Central Asia	36	100	100	100	100	100	100	100	100	100	100	100	100	99	99	99	99	99	100	100	100
East and Central Asia	37	86	89	79	69	86	91	91	97	97	97	98	98	98	100	97	98	100	97	100	100
Europe	38	100	100	100	100	100	100	100	100	100	100	100	100	100	100	79	80	71	100	100	100
Europe	39	98	98	100	98	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100

Europe	40	98	98	100	98	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100
Europe	41	100	100	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	99	100	100
Europe	42	98	97	98	98	98	97	98	98	98	99	100	100	100	100	100	100	100	100	100	100
Europe	43	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100
Europe	44	92	97	97	99	98	99	100	100	100	100	100	100	100	100	99	98	98	99	100	100
Europe	45	96	99	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100
Europe	46	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100
India	47	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	48	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	49	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	50	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	51	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	52	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	53	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	54	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	55	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	56	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	57	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
India	58	65	66	66	68	70	73	73	71	78	83	88	92	92	93	95	97	98	100	100	100
Latin America and Caribbean	59	92	91	93	90	94	97	98	98	100	100	100	100	100	100	98	97	99	97	100	100
Latin America and Caribbean	60	99	97	95	100	100	100	100	100	98	100	100	100	100	100	100	100	100	100	100	100
Latin America and Caribbean	61	95	96	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	99	100	100
Latin America and Caribbean	62	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100
Latin America and Caribbean	63	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100
Latin America and Caribbean	64	100	100	100	100	100	100	100	100	100	100	100	100	100	94	98	98	100	96	100	100
North America	65	99	99	98	100	100	100	100	100	100	100	99	99	100	100	100	100	100	100	100	100
North America	66	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100
North America	67	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100
South Asia	68	83	84	83	88	97	91	96	94	96	98	94	98	96	98	99	100	98	99	100	100
South Asia	69	86	84	85	87	96	89	93	95	96	98	97	95	94	92	98	99	99	99	100	100
South Asia	70	100	100	92	100	100	100	100	100	100	100	100	100	100	83	100	100	100	100	100	100
South Asia	71	100	98	98	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100
South Asia	72	98	96	95	95	98	100	100	100	100	100	97	99	97	100	100	100	99	100	100	100

Table A7: Assumptions for manual WPV1, WPV3 and cVDPV2 introductions

Time	Block	Subpopulation	Serotype
T_0-15.25	1	2	1
T_0-14.75	3	2	1
T_0-14.25	7	2	1
T_0-13.92	13	1	1
T_0-13.75	2	1	1
T_0-13.75	3	1	1
T_0-13.75	3	2	1
T_0-12.25	1	1	1
T_0-11.25	7	1	3
T_0-10.92	5	1	3
T_0-10.26	3	2	1
T_0-10.25	7	1	1
T_0-9.92	10	1	1
T_0-9.92	5	2	1
T_0-9.92	5	1	3
T_0-9.25	3	2	1
T_0-9.25	7	1	1
T_0-9.25	7	3	1
T_0-9.25	1	1	1
T_0-9.08	33	1	1
T_0-8.25	1	1	1
T_0-7.92	5	1	1
T_0-7.92	10	1	3
T_0-6.08	36	1	1
T_0-5.75	3	1	1
T_0-2.08	36	0	2
T_0+025	7	0	2
T_0+0.5	34	0	2

Acronyms: T_0, beginning of analytical time horizon (i.e., January 1, 2019); WPV, wild poliovirus

Table A8: Assumptions for planned, preventive SIAs in OPV-using blocks

a) Number pSIA rounds, before elimination of all WPVs in block

Time period	Income level	Number of SIAs
-69 ≤ year < -29	Any	0
-29 ≤ year < -26	LI, LMI	0, 0, 0
	UMI	2, 2, 2
	HI	0, 0, 0
-26 ≤ year < WPV elimination in block	LI[b]	1, 1, 2, 2, 2, 2, 4, 5, 5, 6, 6, 7, 7, 8, 6, 7, 8, 8, 6, 7, 7, 6, 7, 7, 6, 6, 4, 8, 6, …, 6
	LMI	1, 1, 2, 2, 2, 2, 4, 6, 5, 4, 6, 7, 6, 8, 6, 7, 6, 7, 7, 7, 7, 6, 8, 8, 8, 7, 6, 6, 6, …, 6
	LMI (block 8)[a]	0, 0, 0, 1, 3, 2, 2, 3, 5, 4, 5, 6, 6, 4, 4, 4, 5, 5, 6, 5, 6, 5, 6, 6, 6, 6, 4, 4, 4, …, 4
	UMI	2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, 2, …, 2
	HI	0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, …, 0

Acronyms: HI, high-income; LMI, lower middle-income; LI, low-income; pSIA, planned, preventive SIA, SIA, supplementary immunization activity; UMI, upper middle-income; WPV, wild poliovirus
Footnote: [a] Subpopulation 2 assumed to decrease to SIA_1=0, POL3=0, and COV1or2=0 between in T_0-15.5 and T_0-14.5, and POL3=0.05 and COV1or2=0.05 between in T_0-14.5 and T_0-13.1; [b] Subpopulation 1 assumed to decrease to SIA_1=0, between in T_0+0.1 and T_0+0.9.

b) Pre-OPV2 cessation

Time period	RI coverage (*POL3*)	SIA schedule showing vaccine (day(s) of year)
Before elimination of all WPVs in block[a]		
Year < -14	Any	tOPV (15, 45, 75, 105, 260, 290, 320, 350)
Year = -14	Any (block 8, sp 1)	tOPV (15, 45, 75, 105, 260, 290, 320); mOPV1 (350) (tOPV (15, 45, 75, 105, 260, 290, 320, 350))
Year = -13	Any (block 8, sp 1 and 2) (block 47, sp 1) (block 48, sp 1)	tOPV (45, 75, 105, 260, 290); mOPV1 (15, 320, 350) (mOPV (15, 45, 75, 105, 260, 290, 320, 350)) (tOPV (45, 75, 105); mOPV1 (15, 260, 290, 320, 350)) (tOPV (45, 75, 105); mOPV1 (15, 260, 290, 320, 350))
Year = -12	Any (block 8, sp 1) (block 47, sp 1) (block 48, sp 1)	tOPV (15, 45, 75, 105, 260); mOPV1 (290, 320, 350) (mOPV (15, 45, 75, 105, 260, 290, 320, 350)) (mOPV (15, 45, 75, 105, 260, 290, 320, 350)) (mOPV (15, 45, 75, 105, 260, 290, 320, 350))
Year = -11	Any (block 47, sp 1) (block 48, sp 1)	tOPV (75, 105, 260, 290); mOPV1 (15, 320, 350); mOPV3 (45) (tOPV (75, 105, 260); mOPV1 (15, 45, 290, 320, 350)) (tOPV (75, 105, 260); mOPV1 (15, 45, 290, 320, 350))
Year = -10	Any (block 8, sp 1 and 2) (block 47, sp 1) (block 48, sp 1)	tOPV (15, 75, 105, 260, 290); mOPV1 (45, 350); mOPV3 (320) (mOPV (15, 45, 75, 105, 260, 290, 350); mOPV3(320)) (mOPV (15, 45, 75, 105, 260, 290, 320, 350)) (mOPV (15, 45, 75, 105, 260, 290, 320, 350)
Year = -9	0.1 (sp 1)	tOPV (45, 320); bOPV (75, 105, 260, 290, 320, 350) (bOPV (15, 45, 75, 105, 260, 290, 320, 350))
	0.3	tOPV (320, 350); bOPV (15, 45, 75, 105); mOPV3 (260, 290)
	0.6	tOPV (45, 320); bOPV (15, 75, 105, 260, 290, 350)
	0.9 or 0.98	tOPV (15); bOPV (45, 75, 105, 260, 290, 320, 350)
-8 ≤ year < -4	0.1 (sp 1)	tOPV (45, 320); bOPV (15, 75, 105, 260, 290, 350) (bOPV (15, 45, 75, 105, 260, 290, 320, 350))
	0.3	tOPV (45, 320); bOPV (15, 75, 105, 260, 290, 350)
	0.6	tOPV (45, 320); bOPV (15, 75, 105, 260, 290, 350)

	0.9 or 0.98	tOPV (15); bOPV (45, 75, 105, 260, 290, 320, 350)
Year = -4	0.1	tOPV (45, 320); bOPV (15, 75, 105, 260, 290, 350) (tOPV (75, 290); bOPV (15, 105, 165, 200, 260, 350))
	0.3	tOPV (45, 320); bOPV (15, 75, 105, 260, 290, 350)
	0.6	tOPV (45, 320); bOPV (15, 75, 105, 260, 290, 350)
	0.9 or 0.98	tOPV (15); bOPV (45, 75, 105, 260, 290, 320, 350)
-3 ≤ year < OPV2 cessation time (after tOPV intensification)	0.1	tOPV (15, 75); bOPV (105, 165, 200, 260, 290, 350)
	0.3	tOPV (15, 45); bOPV (75, 105, 260, 290, 320, 350)
	0.6	tOPV (15, 45); bOPV (75, 105, 260, 290, 320, 350)
	0.9 or 0.98	tOPV (15); bOPV (45, 75, 105, 260, 290, 320, 350)
After elimination of all WPVs in block		
Year < -9 (before bOPV introduction)	0.1	tOPV (0, 40, 80, 140, 240, 300)
	0.3 (LI) 0.3 ($R_0 \leq 10$, non-LI) 0.3 ($R_0 > 10$, non-LI)	tOPV (0, 60) tOPV (0, 60, 120) tOPV (0, 40, 80, 140, 240)
	0.6 (LI) 0.6 ($R_0 \leq 10$, non-LI) 0.6 ($R_0 > 10$, non-LI)	tOPV (0, 60) tOPV (0, 60, 120) tOPV (0, 40, 80, 140, 240)
	0.9 ($R_0 \leq 10$) 0.9 ($R_0 > 10$, LI) 0.9 ($R_0 > 10$, non-LI)	tOPV (0) tOPV (0, 60) tOPV (0, 60, 120)
	0.98 ($R_0 \leq 10$) 0.98 ($R_0 > 10$)	No SIAs tOPV (0)
-9 ≤ year < -3 (before tOPV intensification)	0.1	tOPV (0, 40); bOPV (80, 140, 240, 300)
	0.3 (LI) 0.3 ($R_0 \leq 10$, non-LI) 0.3 ($R_0 > 10$, non-LI)	tOPV (0); bOPV (60) tOPV (0); bOPV (60, 120) tOPV (0, 40); bOPV (80, 140, 240)
	0.6 (LI) 0.6 ($R_0 \leq 10$, non-LI) 0.6 ($R_0 > 10$, non-LI)	tOPV (0); bOPV (60) tOPV (0); bOPV (60, 120) tOPV (0, 40); bOPV (80, 140, 240)
	0.9 ($R_0 \leq 10$) 0.9 ($R_0 > 10$, LI) 0.9 ($R_0 > 10$, non-LI)	tOPV (0) tOPV (0); bOPV (60) tOPV (0); bOPV (60, 120)
	0.98 ($R_0 \leq 10$) 0.98 ($R_0 > 10$)	No SIAs tOPV (0)
-3 ≤ year < OPV2 cessation time (after tOPV intensification)	0.1	tOPV (0, 40); bOPV (80, 140, 240, 300)
	0.3 (LI) 0.3 ($R_0 \leq 10$, non-LI) 0.3 ($R_0 > 10$, non-LI)	tOPV (0, 60) tOPV (0, 60); bOPV (120) tOPV (0, 40, 80); bOPV (140, 240)
	0.6 (LI) 0.6 ($R_0 \leq 10$, non-LI) 0.6 ($R_0 > 10$, non-LI)	tOPV (0, 60) tOPV (0, 60); bOPV (120) tOPV (0, 40, 80); bOPV (140, 240)
	0.9 ($R_0 \leq 10$) 0.9 ($R_0 > 10$, LI) 0.9 ($R_0 > 10$, non-LI)	tOPV (0) tOPV (0); bOPV (60) tOPV (0); bOPV (60, 120)
	0.98 ($R_0 \leq 10$) 0.98 ($R_0 > 10$)	No SIAs tOPV (0)

Acronyms: bOPV, bivalent OPV; LI, low-income; mOPV(1,3), monovalent OPV (containing serotype 1, and serotype 3, respectively); OPV, oral poliovirus vaccine; OPV13, serotype 1- and 3-containing OPV; OPV2, serotype-2-containing OPV; POL3, coverage with 3 or more non-birth RI doses; RI, routine immunization; SIA, supplemental immunization activity; tOPV, trivalent OPV; WPV, wild poliovirus

Footnote: [a] Annual frequency of SIAs starts at 2 and increases according to average number of pSIAs used given year in endemic countries of LI and LMI blocks until all WPV eliminated from the block, see Table A5a

c) Post-OPV2 cessation

Time period	Number of SIAs	SIA schedule showing vaccine (day(s) of year)
Before elimination of all WPVs in block[a]		
OPV2 cessation time ≤ year < -1	4	bOPV (15, 75, 105, 350)
	6	IPV[b](120); bOPV (15, 75, 105, 260, 290, 350)
	7	IPV[b](90); bOPV (15, 45, 75, 105, 290, 320, 350)
	8	IPV[b](90); bOPV (15, 45, 75, 105, 260, 290, 320, 350)
-1 ≤ year < OPV13 cessation time (after OPV2 cessation)	6	bOPV (15, 45, 75, 290, 320, 350)
	7	bOPV (15, 45, 75, 105, 290, 320, 350)
	8	bOPV (15, 45, 75, 105, 260, 290, 320, 350)
After elimination of all WPVs in block		
OPV2 cessation time ≤ year < OPV13 cessation time (after OPV2 cessation)	8	bOPV (0, 40, 80, 120, 160, 200, 240, 280)
	7	bOPV (0, 40, 80, 140, 200, 240, 280)
	6	bOPV (0, 40, 80, 160, 240, 280), mOPV2[c] (25)
	5	bOPV (0, 40, 80, 160, 240)
	4	bOPV (0, 40, 80, 240), mOPV2[d] (25)
	3	bOPV (0, 60, 120)
	2	bOPV (0, 60)
	1	bOPV (0)

Acronyms: IPV, inactivated poliovirus vaccine; bOPV, bivalent OPV; OPV, oral poliovirus vaccine; OPV13, serotype 1- and 3-containing OPV; OPV2, serotype-2-containing OPV; SIA, supplemental immunization activity; WPV, wild poliovirus

Footnote: [a] Annual frequency of SIAs starts at 2 and increases according to average number of pSIAs used given year in endemic countries of LI and LMI blocks until all WPV eliminated from the block, see Table A5a; [b] IPV round only in subpopulations 1 and 2 of remaining endemic blocks post-OPV2 cessation; [c] mOPV2 round only in block 8 in 2017 (T_0-2); [d] mOPV2 round only in block 8 in 2019 (T_0).

Table A9: Characterization of non-cVDPV post-OPV cessation risks

Model input	Value
Average time between contacts of long-term iVDPV excreters with the general population (days)	150-600
Reversion stage of iVDPV virus when introduced into general population	10
Global Poisson rate for release of unreturned OPV in blocks that use OPV at T_0 (1/year)	
- within 1st year post serotype specific cessation	4
- within 2nd year post serotype specific cessation	1
Poor-performing blocks assumed to be more likely to release unreturned OPV	1, 3, 5, 7, 8, 13, 32, 34
Weight of unreturned OPV releases by block performance	
- poor-performing blocks (1/block)	5
- other performing blocks (1/block)	1
Global Poisson rate for release from IPV production site (1/year)	0.2
Global Poisson rate for release from other PEF (1/year)	0.02
Global Poisson rate for release from PIM or other unintentional release (1/year)	0.004975
Distribution of PIM other unintentional releases by income level	
- LI	0.00
- LMI	0.01
- UMI	0.09
- HI	0.90
Global Poisson rate for other intentional release (1/year)	0.000025
Distribution of intentional releases by income level	
- LI, LMI, UMI	0.5
- HI	0.5

Acronyms: HI, high-income; IPV, inactivated poliovirus vaccine; LMI, lower middle-income; LI, low-income; OPV, oral poliovirus vaccine; PEF, polio essential facility; PIM, potentially infectious materials; UMI, upper middle-income

Table A10: Distribution of Polio Essential Facilities (PEFs) into specific blocks and subpopulations

a) IPV producing PEFs

Preferential mixing area (PMA)	Block	Sub-popul-ation(s)	Income level	Polio vaccine at T_0	Number of Salk-IPV facilities	Number of Sabin-IPV facilities
Australasia	15	9	LMI	OPV+IPV	0	1
Australasia	16	1	HI	IPV-only	0	2
Australasia	17	0	HI	IPV-only	0	1
Australasia	17	5	HI	IPV-only	0	1
China and neighbors	19	0	UMI	OPV+IPV	0	1
China and neighbors	19	1	UMI	OPV+IPV	0	1
China and neighbors	19	2	UMI	OPV+IPV	0	1
China and neighbors	19	3	UMI	OPV+IPV	0	1
China and neighbors	19	4	UMI	OPV+IPV	0	1
China and neighbors	19	5	UMI	OPV+IPV	0	1
East and Central Asia	35	4	UMI	OPV+IPV	0	2
Europe	40	0	UMI	IPV/OPV	0	1
Europe	42	6	HI	IPV-only	1	0
Europe	42	7	HI	IPV-only	0	1
Europe	46	0	HI	IPV-only	1	0
Europe	46	2	HI	IPV-only	1	0
Europe	46	3	HI	IPV-only	0	0
Europe	46	8	HI	IPV-only	2	0
North America	65	5	HI	IPV-only	1	0
South Asia	70	0	LMI	OPV+IPV	0	1

Acronyms: HI, high-income; IPV, inactivated poliovirus vaccine; LMI, lower middle-income; OPV, oral poliovirus vaccine; PEF, polio essential facility; UMI, upper middle-income

b) Non-IPV producing PEFs

Preferential mixing area (PMA)	Block	Sub-popul-ation(s)	Income level	Polio vaccine at T_0	Number of non-IPV PEFs
Australasia	16	1	HI	IPV-only	1
Australasia	16	7	HI	IPV-only	1
Australasia	17	5	HI	IPV-only	1
China and neighbors	20	0	UMI	OPV+IPV	1
China and neighbors	21	0	UMI	OPV+IPV	1
East and Central Asia	34	0	LMI	OPV+IPV	1
Europe	38	7	LMI	IPV/OPV	1
Europe	39	0	UMI	IPV/OPV	2
Europe	39	1	UMI	IPV/OPV	1
Europe	40	0	UMI	IPV/OPV	1
Europe	40	1	UMI	IPV/OPV	1
Europe	40	2	UMI	IPV/OPV	1
Europe	41	0	HI	IPV-only	1
Europe	41	4	HI	IPV-only	1
Europe	41	5	HI	IPV-only	1
Europe	42	0	HI	IPV-only	1
Europe	42	1	HI	IPV-only	1
Europe	42	2	HI	IPV-only	1
Europe	42	3	HI	IPV-only	1
Europe	42	4	HI	IPV-only	1
Europe	42	5	HI	IPV-only	1
Europe	42	6	HI	IPV-only	1
Europe	44	3	HI	IPV-only	3
Europe	46	0	HI	IPV-only	9
Europe	46	2	HI	IPV-only	5
Europe	46	8	HI	IPV-only	3
India	49	0	LMI	OPV+IPV	1
Latin America and Caribbean	59	7	LMI	OPV+IPV	1
Latin America and Caribbean	61	7	UMI	OPV+IPV	3
Latin America and Caribbean	62	0	UMI	IPV/OPV	1
Latin America and Caribbean	63	0	UMI	IPV/OPV	1
Latin America and Caribbean	64	0	UMI	IPV-only	1
North America	65	0	HI	IPV-only	1
North America	65	1	HI	IPV-only	1
North America	65	2	HI	IPV-only	1
North America	65	3	HI	IPV-only	1
North America	65	4	HI	IPV-only	1
North America	66	0	HI	IPV-only	1
North America	66	1	HI	IPV-only	1
North America	66	2	HI	IPV-only	1
North America	66	3	HI	IPV-only	1
North America	66	4	HI	IPV-only	1
North America	67	0	HI	IPV-only	1
North America	67	1	HI	IPV-only	1

North America	67	2	HI	IPV-only	1
North America	67	3	HI	IPV-only	1
North America	67	4	HI	IPV-only	1
South Asia	72	0	UMI	IPV/OPV	1

Acronyms: HI, high-income; IPV, inactivated poliovirus vaccine; LMI, lower middle-income; OPV, oral poliovirus vaccine; PEF, polio essential facility; UMI, upper middle-income

Figure A1: Immunity states and flows between them due to epidemiological events (Source: Duintjer Tebbens et al. [3, p. 706])

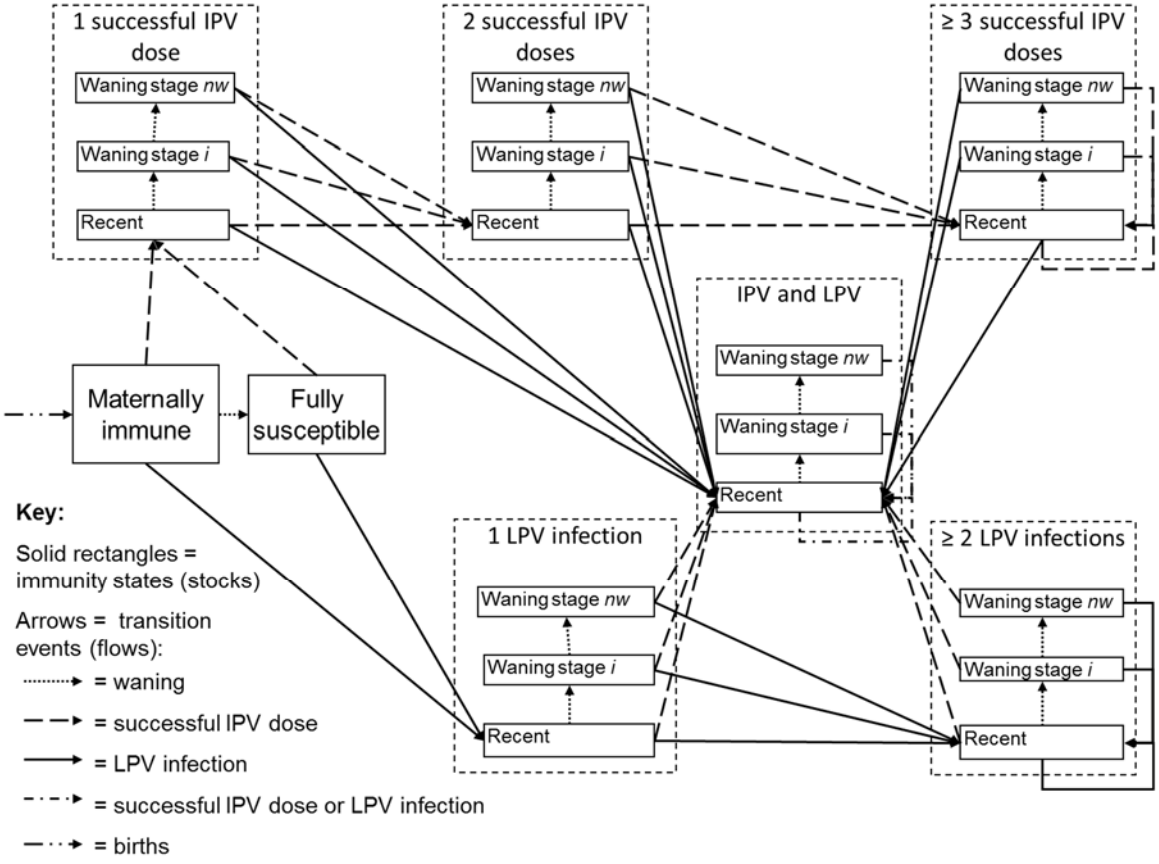
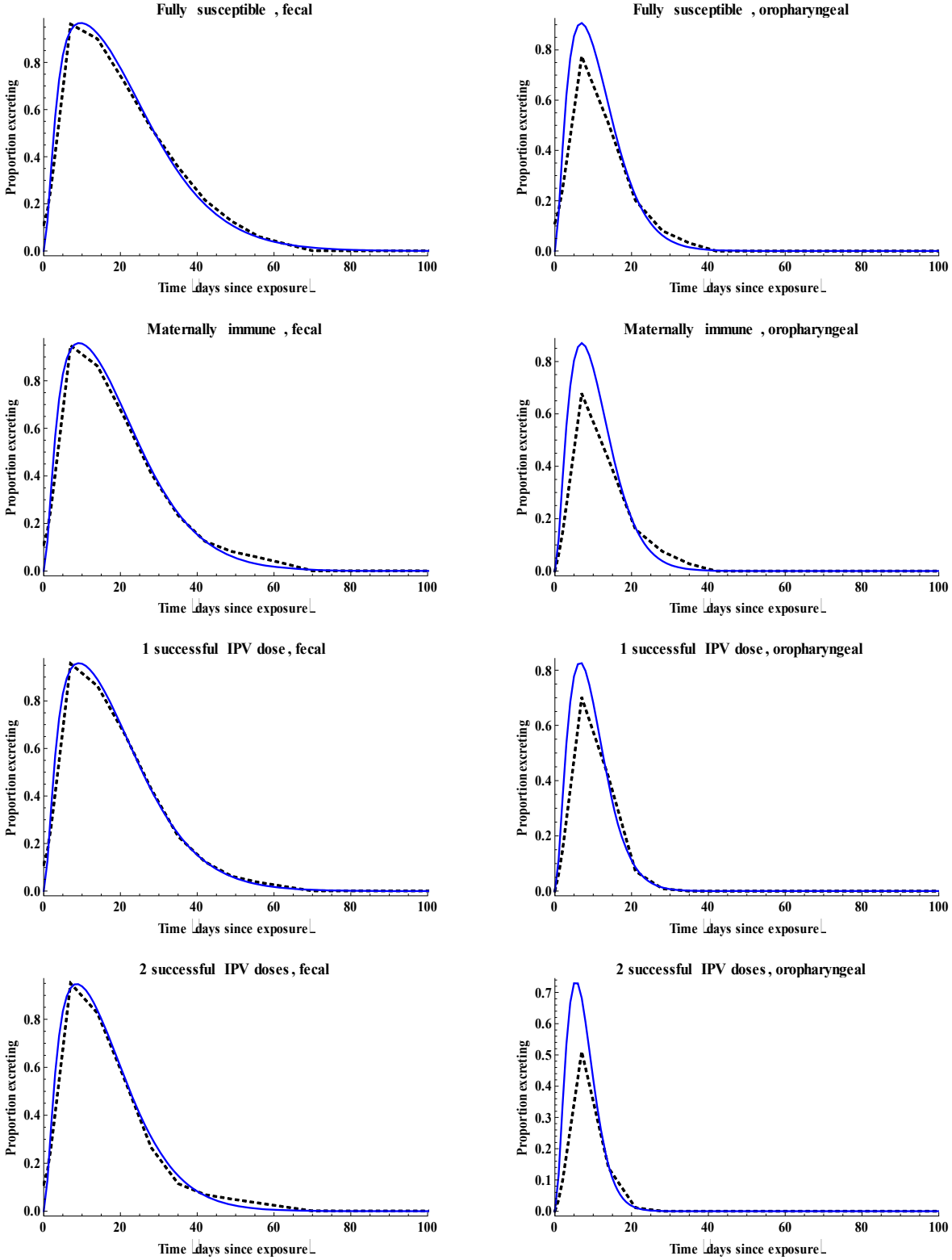
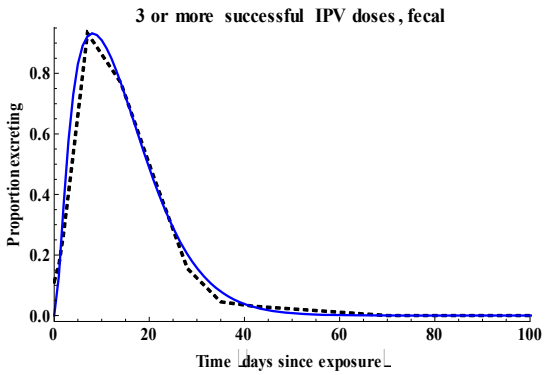
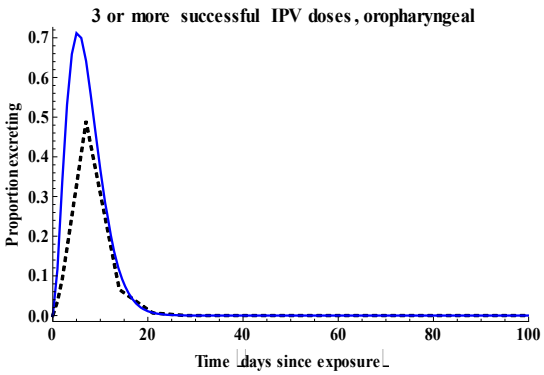
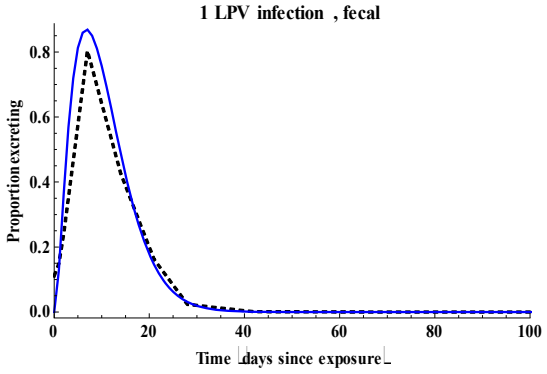


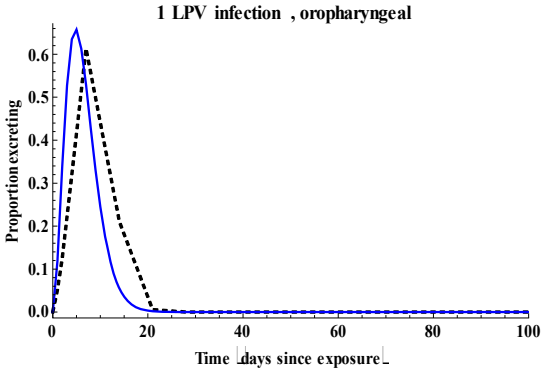
Figure A2: Distribution of the duration of fecal and oropharyngeal infectiousness in the model (solid blue line) and according to the means of expert assessments (black dashed line) [16], by immunity state.

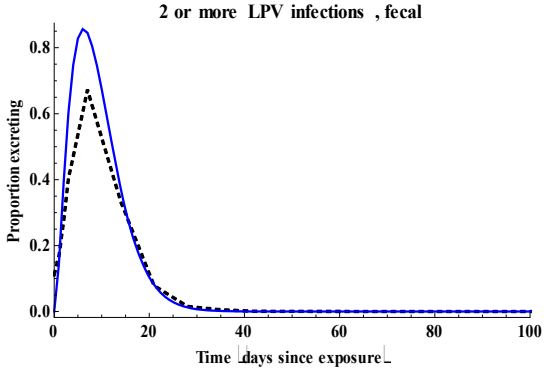


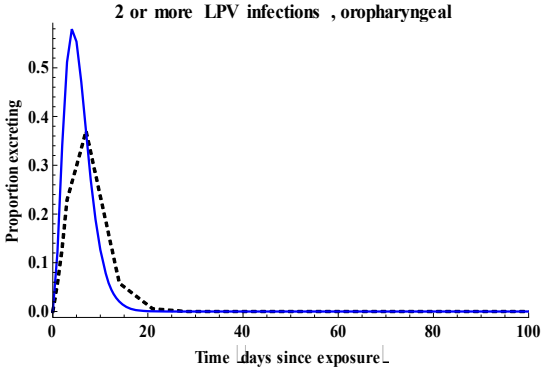

3 or more successful IPV doses, fecal
Proportion excreting
Time (days since exposure)

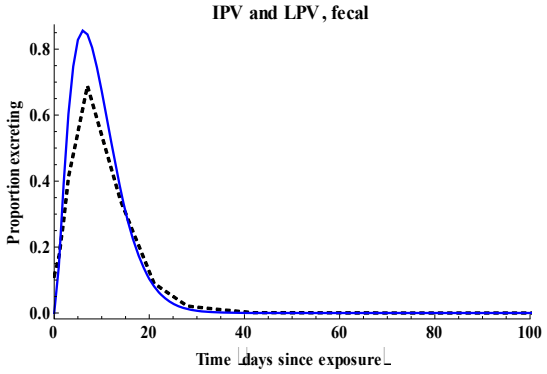

3 or more successful IPV doses, oropharyngeal
Proportion excreting
Time (days since exposure)


1 LPV infection , fecal
Proportion excreting
Time (days since exposure)


1 LPV infection , oropharyngeal
Proportion excreting
Time (days since exposure)


2 or more LPV infections , fecal
Proportion excreting
Time (days since exposure)


2 or more LPV infections , oropharyngeal
Proportion excreting
Time (days since exposure)


IPV and LPV, fecal
Proportion excreting
Time (days since exposure)

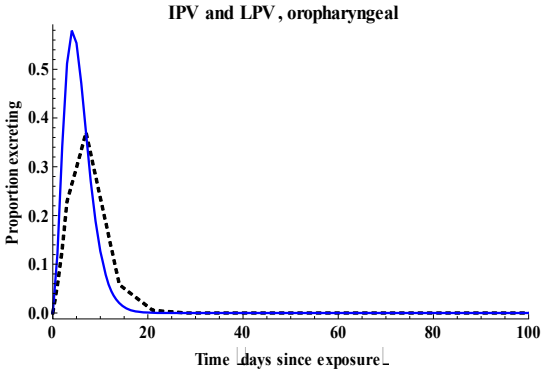
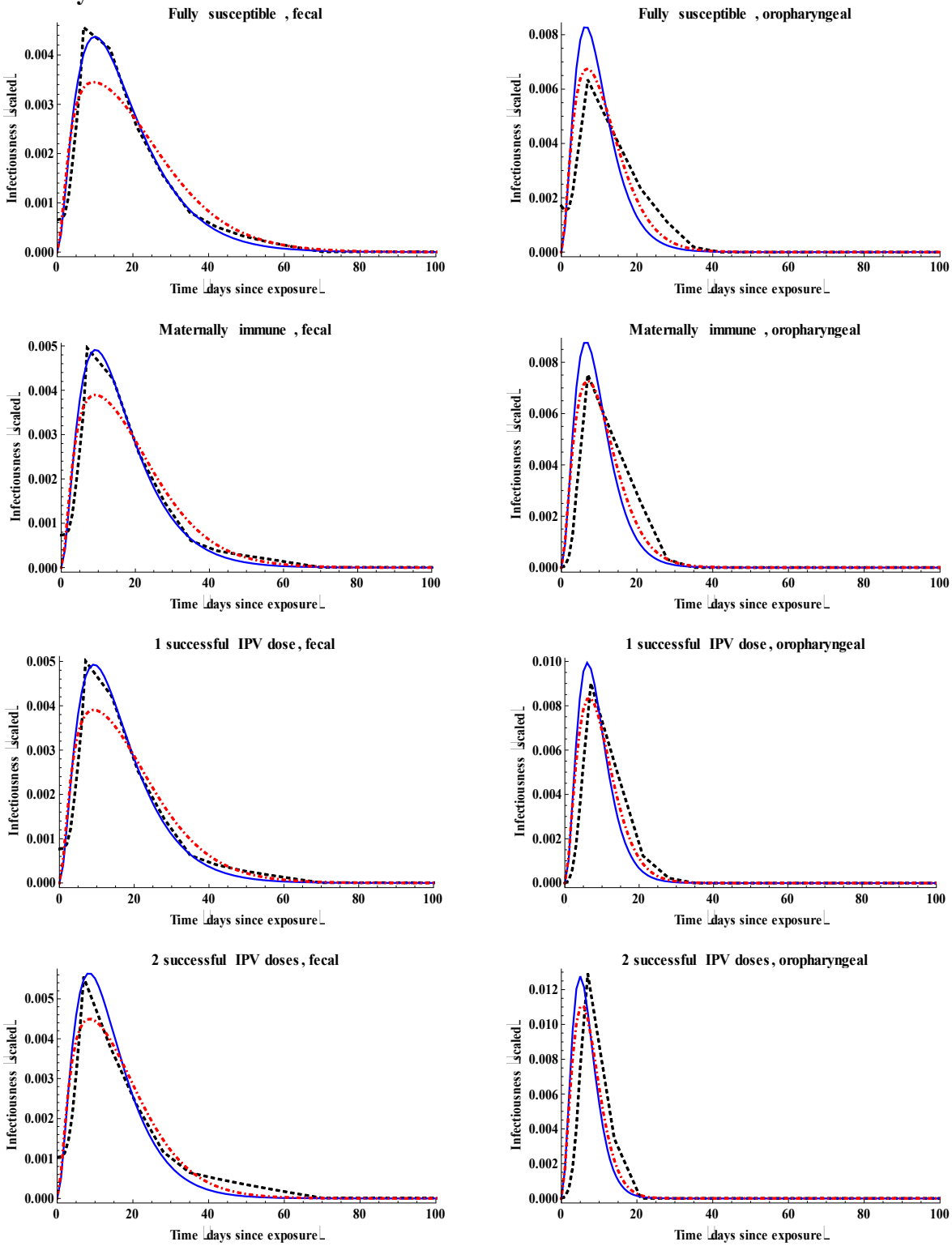
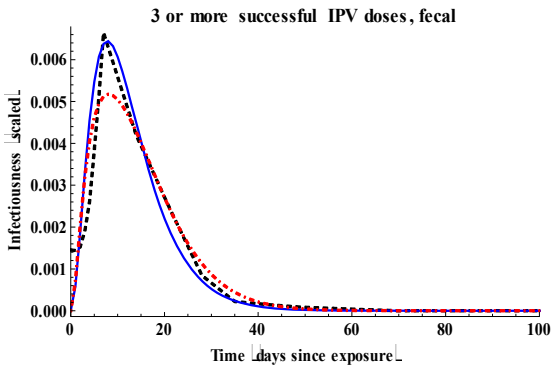
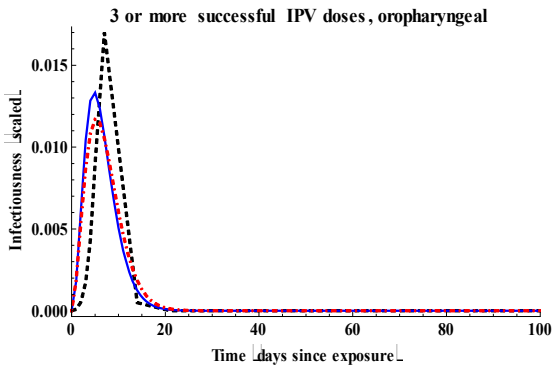
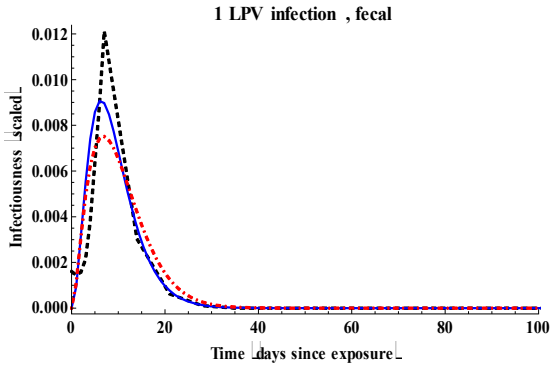

IPV and LPV, oropharyngeal
Proportion excreting
Time (days since exposure)

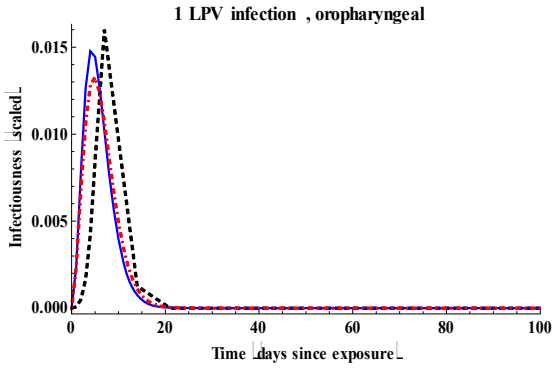
Figure A3: Average relative infectiousness for a population infected at time 0 according to expert assessments (dashed black line) [16], the model with constant infectiousness (dash-dotted red line), and the model with variable infectiousness by stage (solid blue line), by immunity state.

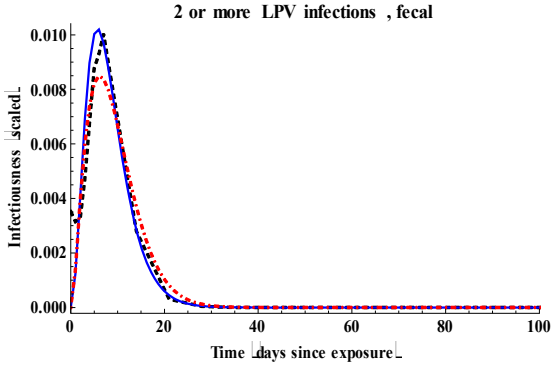


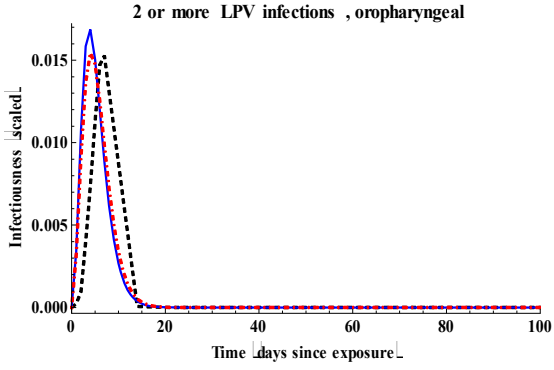

3 or more successful IPV doses, fecal
Infectiousness (scaled)
0.006
0.005
0.004
0.003
0.002
0.001
0.000
0
20
40
60
80
100
Time (days since exposure)

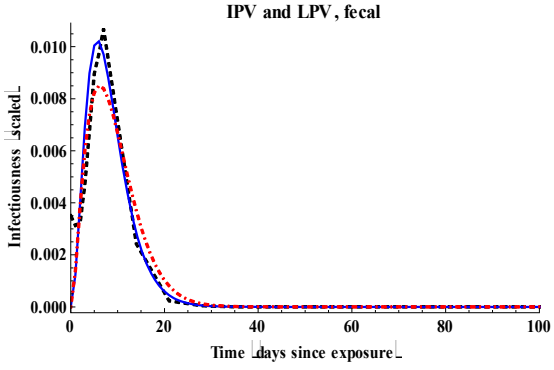

3 or more successful IPV doses, oropharyngeal
Infectiousness (scaled)
0.015
0.010
0.005
0.000
0
20
40
60
80
100
Time (days since exposure)


1 LPV infection , fecal
Infectiousness (scaled)
0.012
0.010
0.008
0.006
0.004
0.002
0.000
0
20
40
60
80
100
Time (days since exposure)


1 LPV infection , oropharyngeal
Infectiousness (scaled)
0.015
0.010
0.005
0.000
0
20
40
60
80
100
Time (days since exposure)


2 or more LPV infections , fecal
Infectiousness (scaled)
0.010
0.008
0.006
0.004
0.002
0.000
0
20
40
60
80
100
Time (days since exposure)


2 or more LPV infections , oropharyngeal
Infectiousness (scaled)
0.015
0.010
0.005
0.000
0
20
40
60
80
100
Time (days since exposure)


IPV and LPV, fecal
Infectiousness (scaled)
0.010
0.008
0.006
0.004
0.002
0.000
0
20
40
60
80
100
Time (days since exposure)

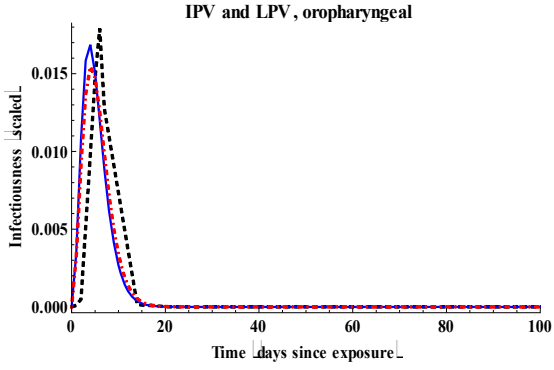
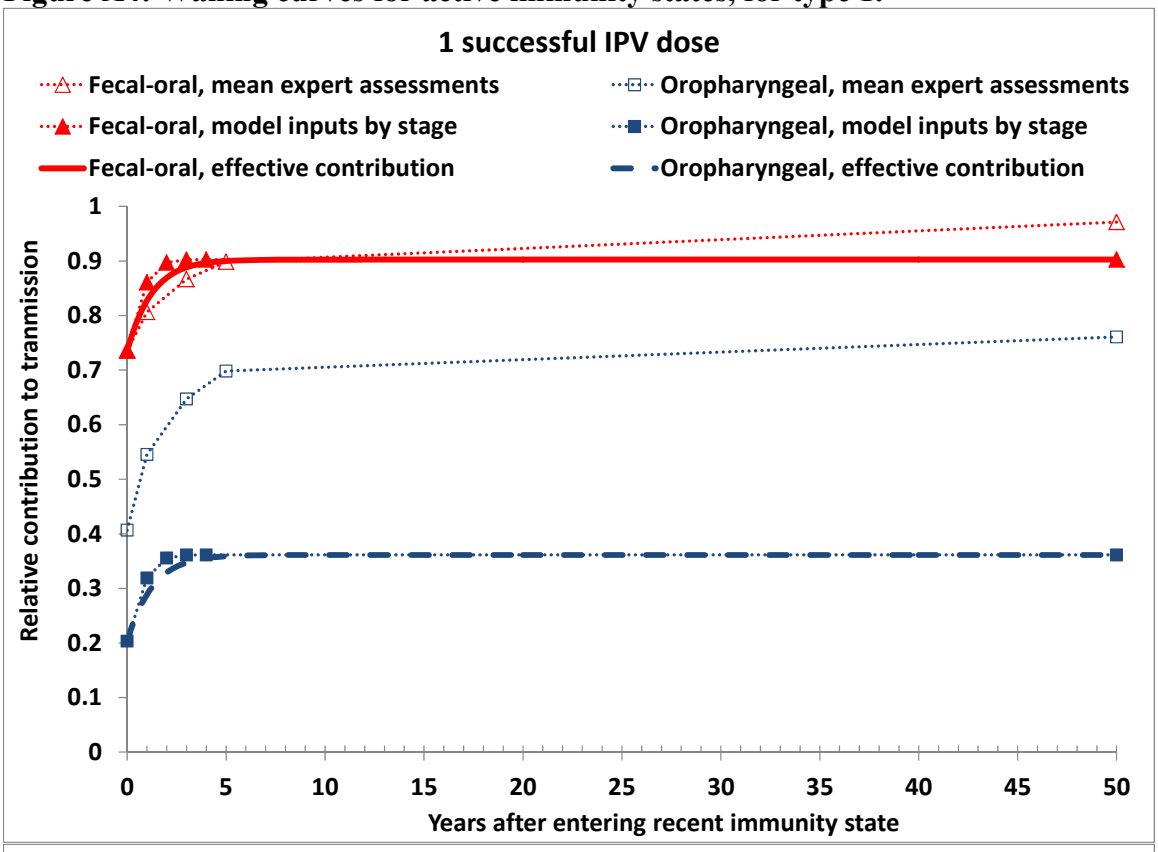
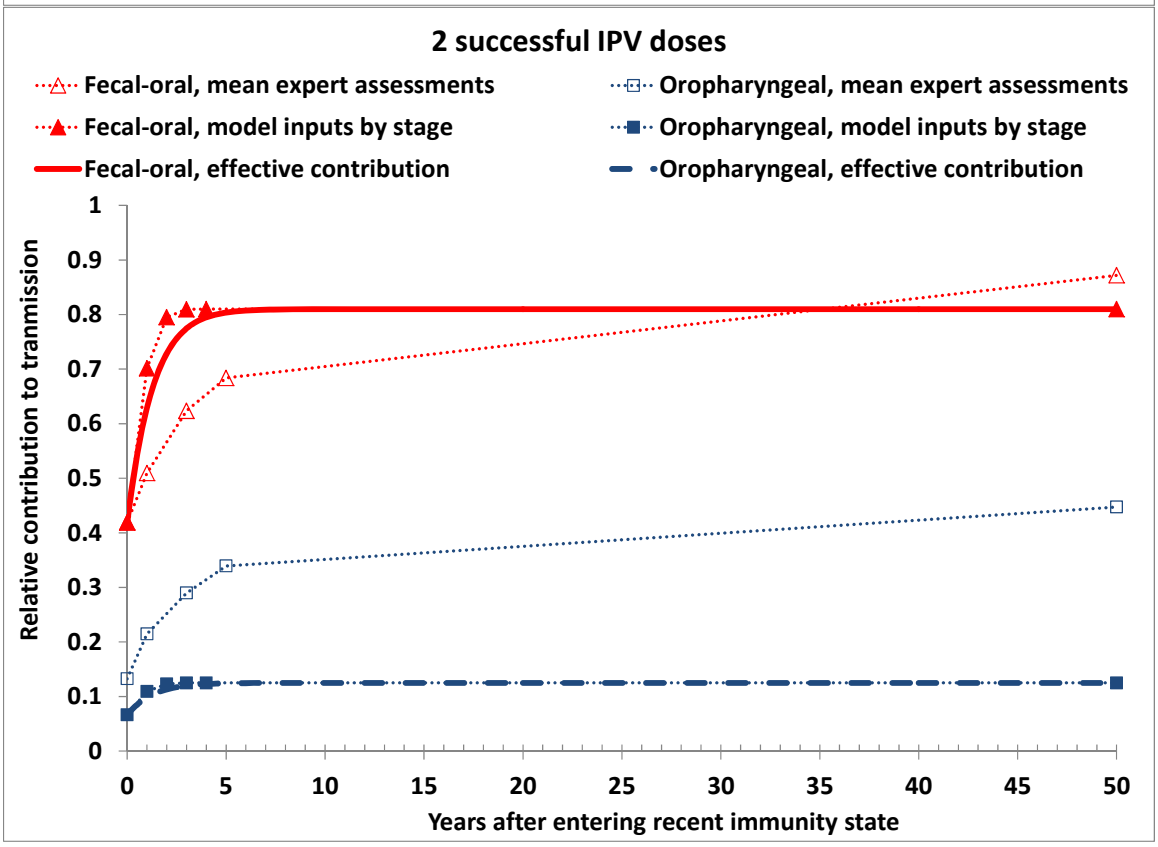
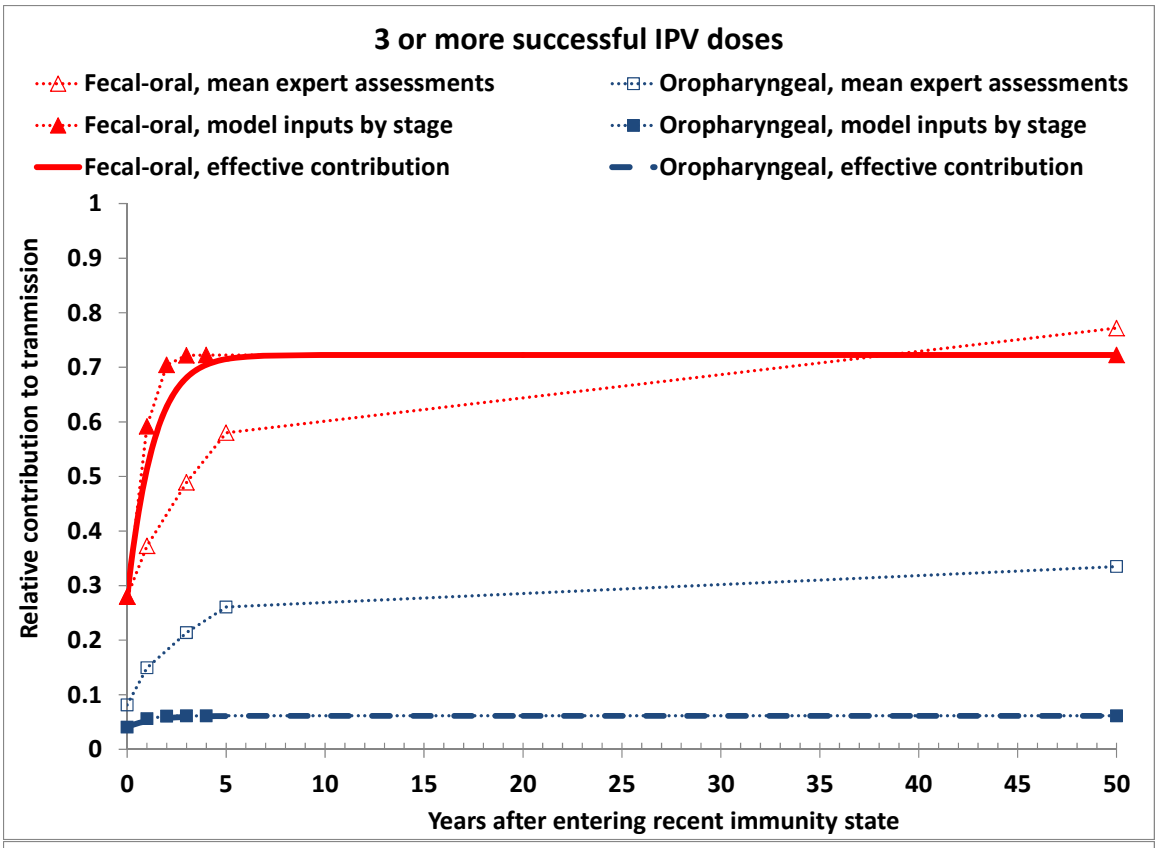
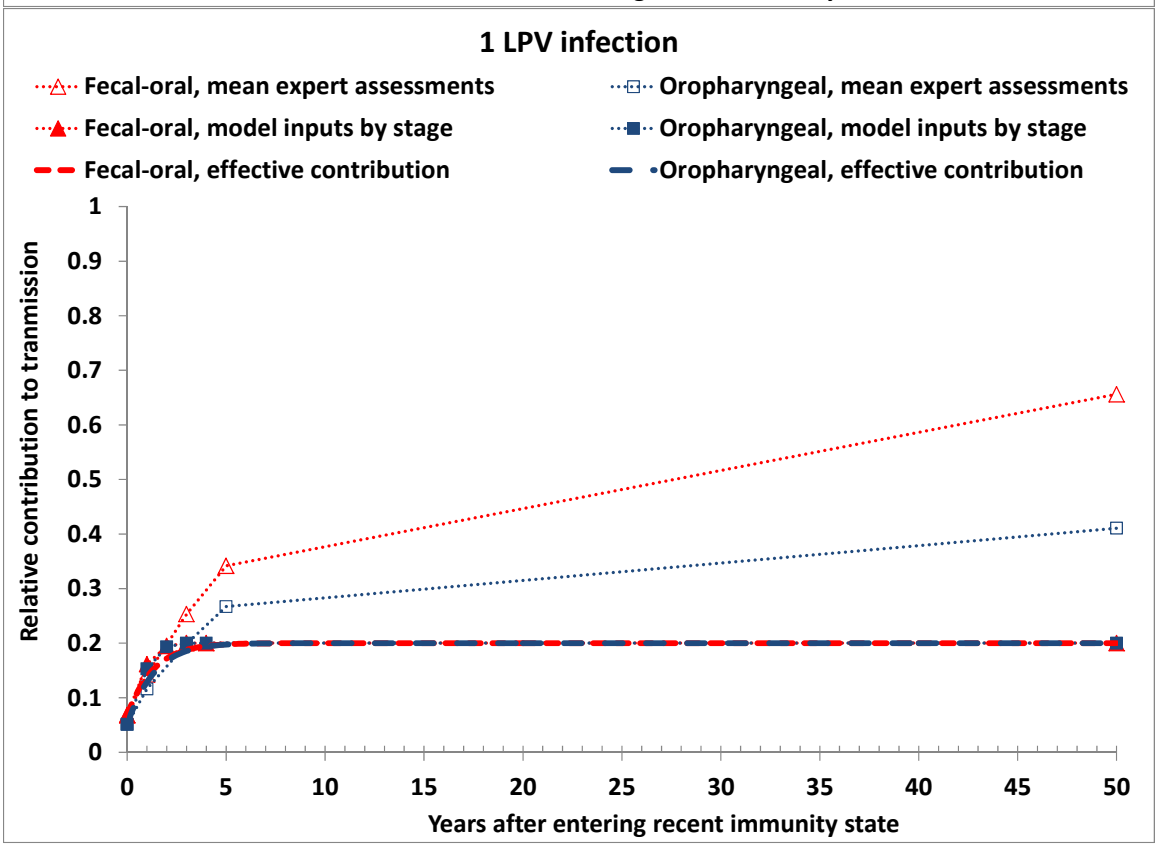

IPV and LPV, oropharyngeal
Infectiousness (scaled)
0.015
0.010
0.005
0.000
0
20
40
60
80
100
Time (days since exposure)

Figure A4: Waning curves for active immunity states, for type 1.






3 or more successful IPV doses
Fecal-oral, mean expert assessments
Fecal-oral, model inputs by stage
Fecal-oral, effective contribution
Oropharyngeal, mean expert assessments
Oropharyngeal, model inputs by stage
Oropharyngeal, effective contribution
Relative contribution to tranmission
0
0.1
0.2
0.3
0.4
0.5
0.6
0.7
0.8
0.9
1
0
5
10
15
20
25
30
35
40
45
50
Years after entering recent immunity state


1 LPV infection
Fecal-oral, mean expert assessments
Fecal-oral, model inputs by stage
Fecal-oral, effective contribution
Oropharyngeal, mean expert assessments
Oropharyngeal, model inputs by stage
Oropharyngeal, effective contribution
Relative contribution to tranmission
0
0.1
0.2
0.3
0.4
0.5
0.6
0.7
0.8
0.9
1
0
5
10
15
20
25
30
35
40
45
50
Years after entering recent immunity state

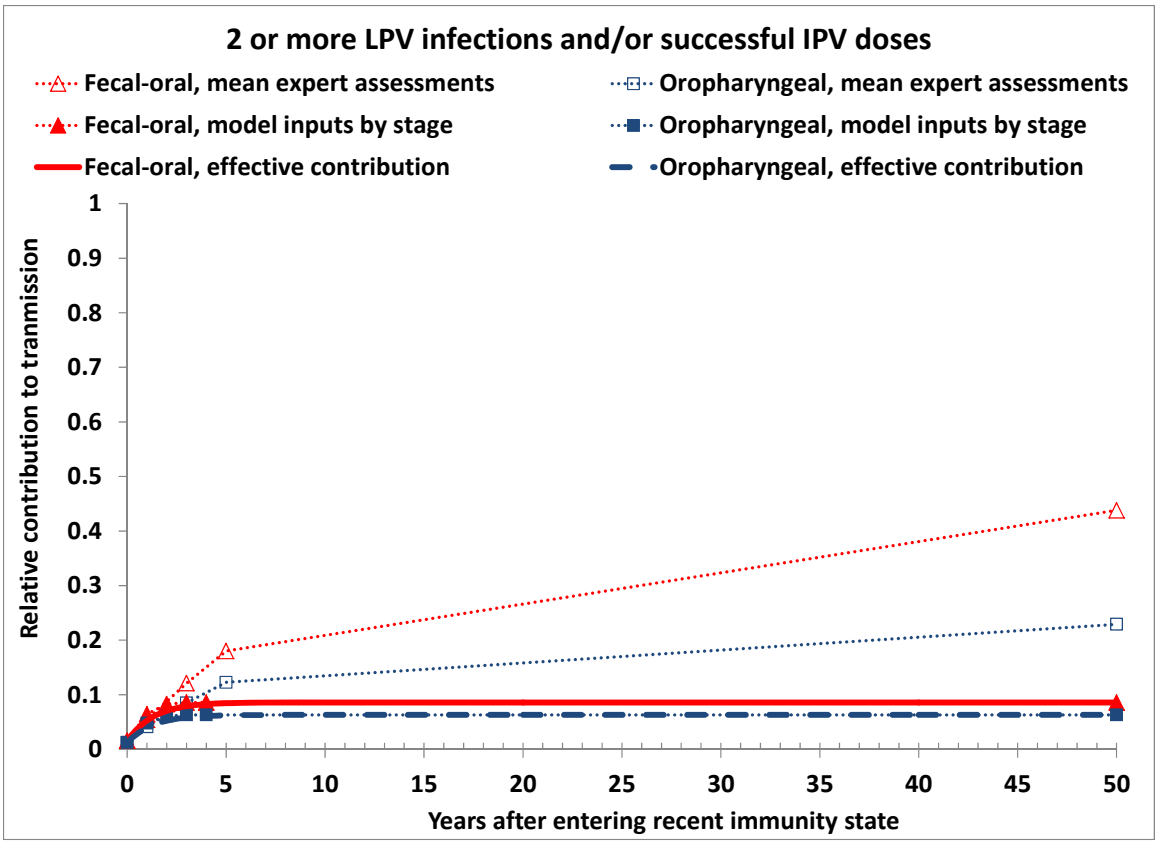
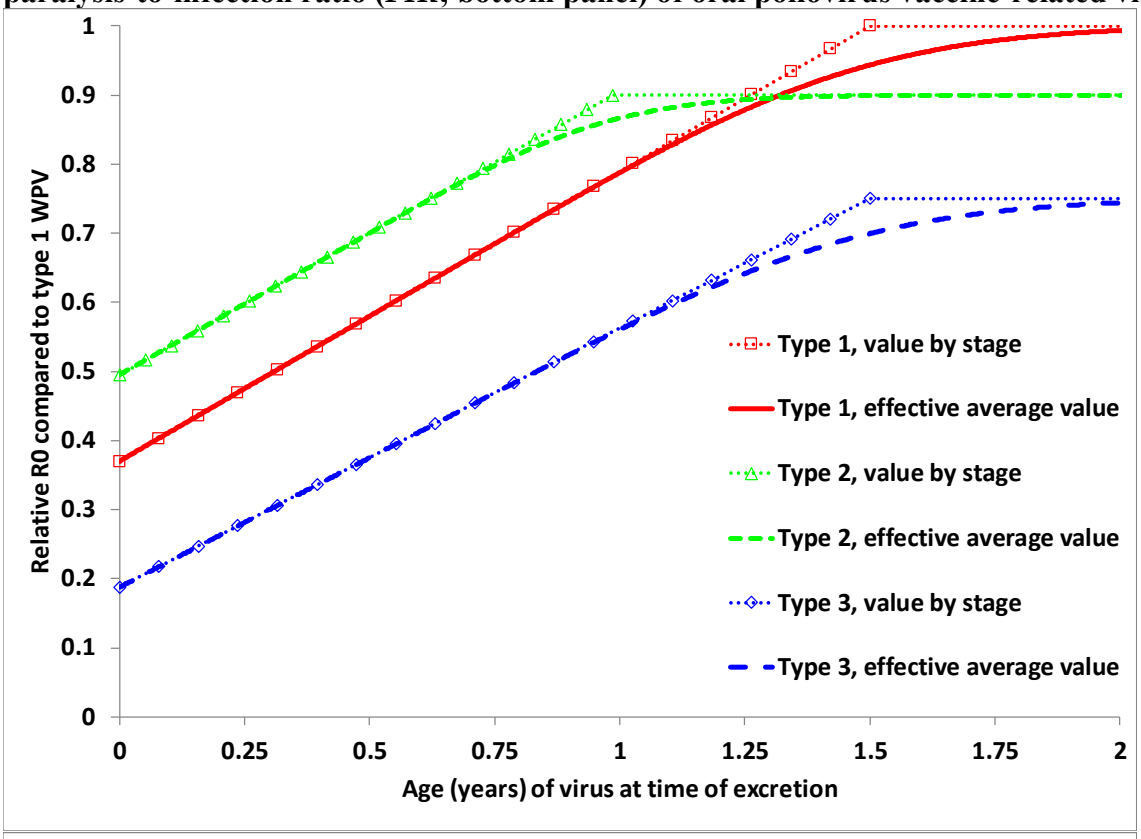

2 or more LPV infections and/or successful IPV doses
Fecal-oral, mean expert assessments
Oropharyngeal, mean expert assessments
Fecal-oral, model inputs by stage
Oropharyngeal, model inputs by stage
Fecal-oral, effective contribution
Oropharyngeal, effective contribution
Relative contribution to tranmission
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
5
10
15
20
25
30
35
40
45
50
Years after entering recent immunity state

Figure A5: Assumed effect of reversion on basic reproductive number (R_0, top panel), and paralysis-to-infection ratio (*PIR*; bottom panel) of oral poliovirus vaccine-related viruses



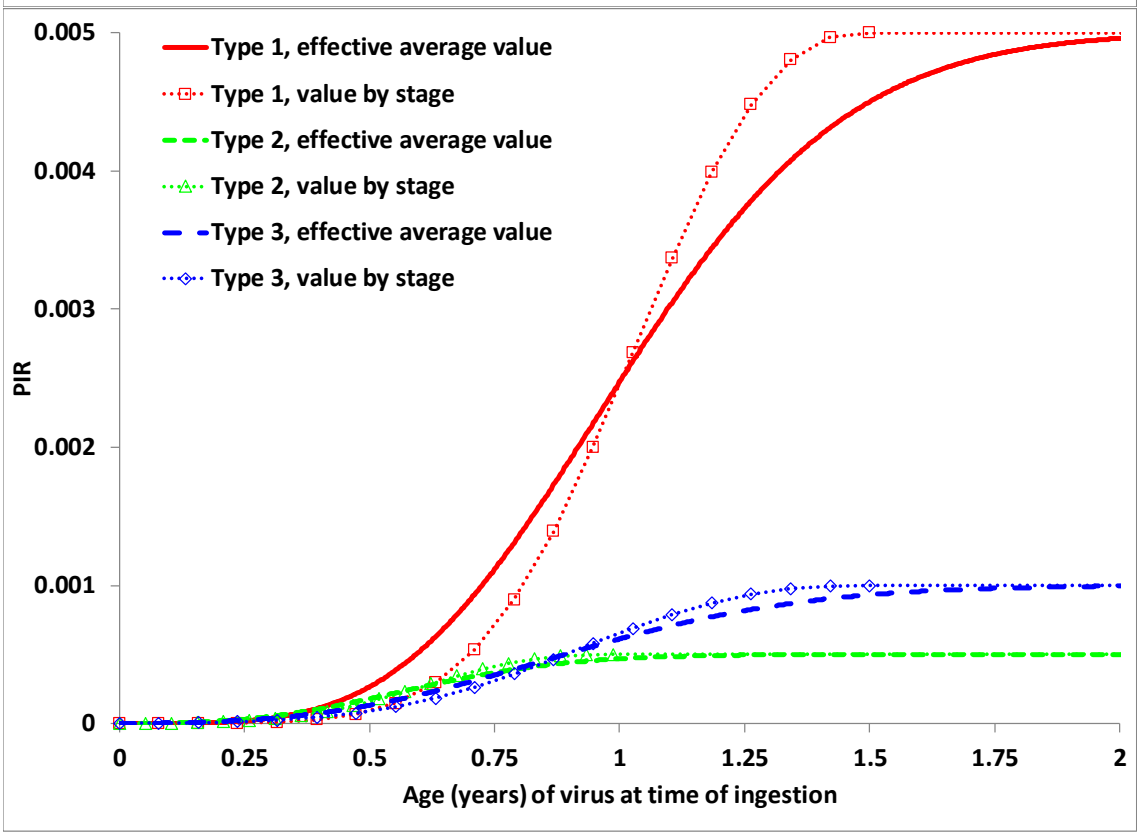


Figure A6: SIA tree showing the probabilities of receiving a dose in two subsequent rounds

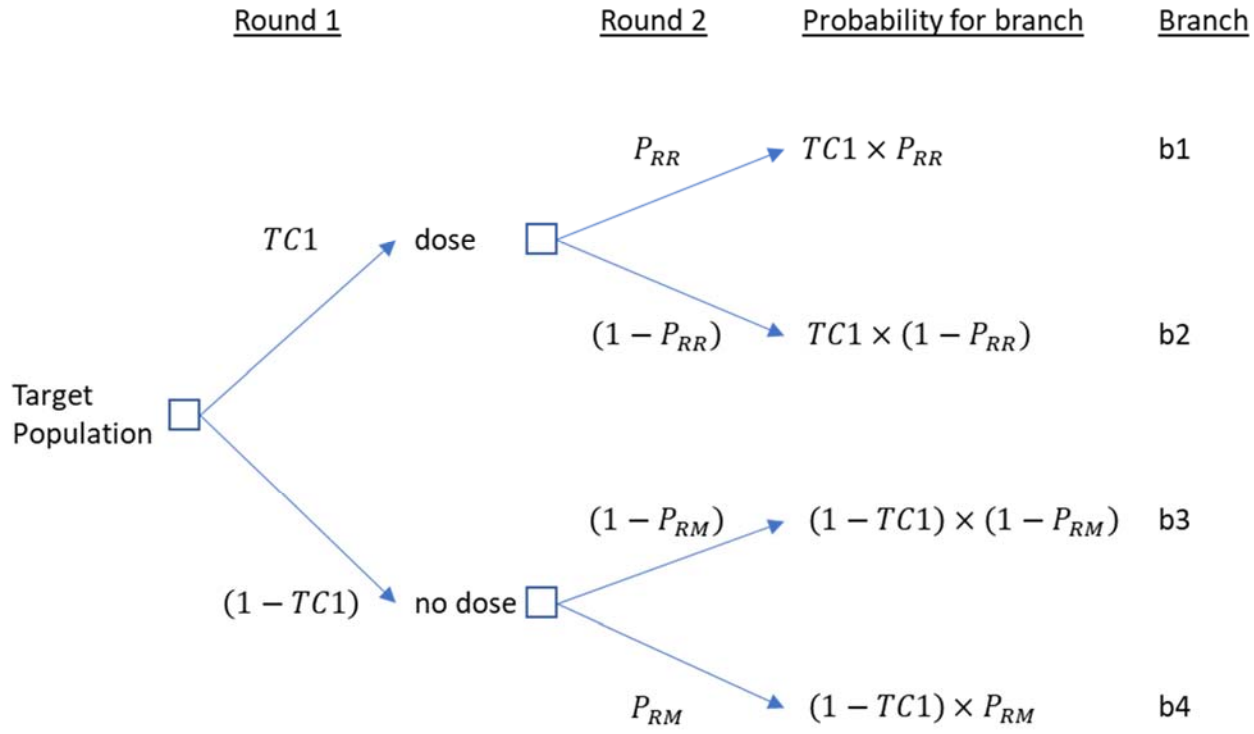
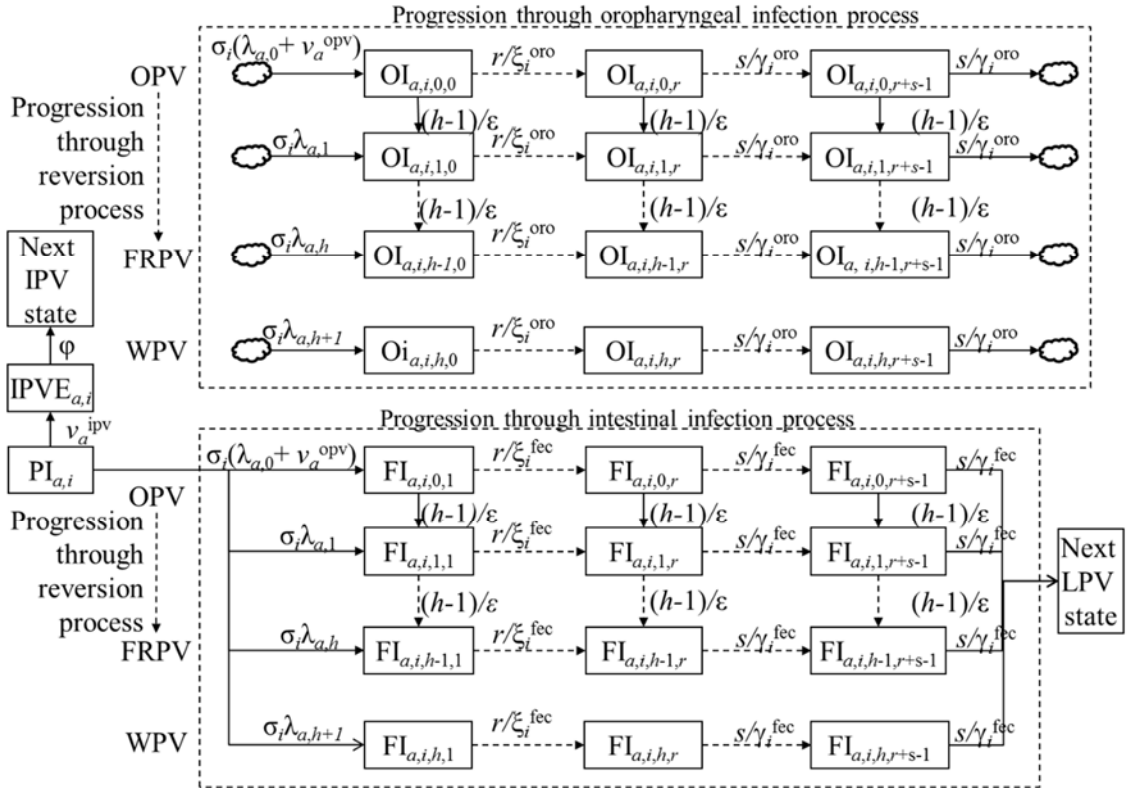


Figure A7: Progression through infection and reversion stages (Source: Duintjer Tebbens et al. [3, p. 706])



"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} (γ_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" [3, p. 706]

Figure A8: Summary results from the model calibration process, adapted from Duintjer Tebbens et al. (2013) [3]

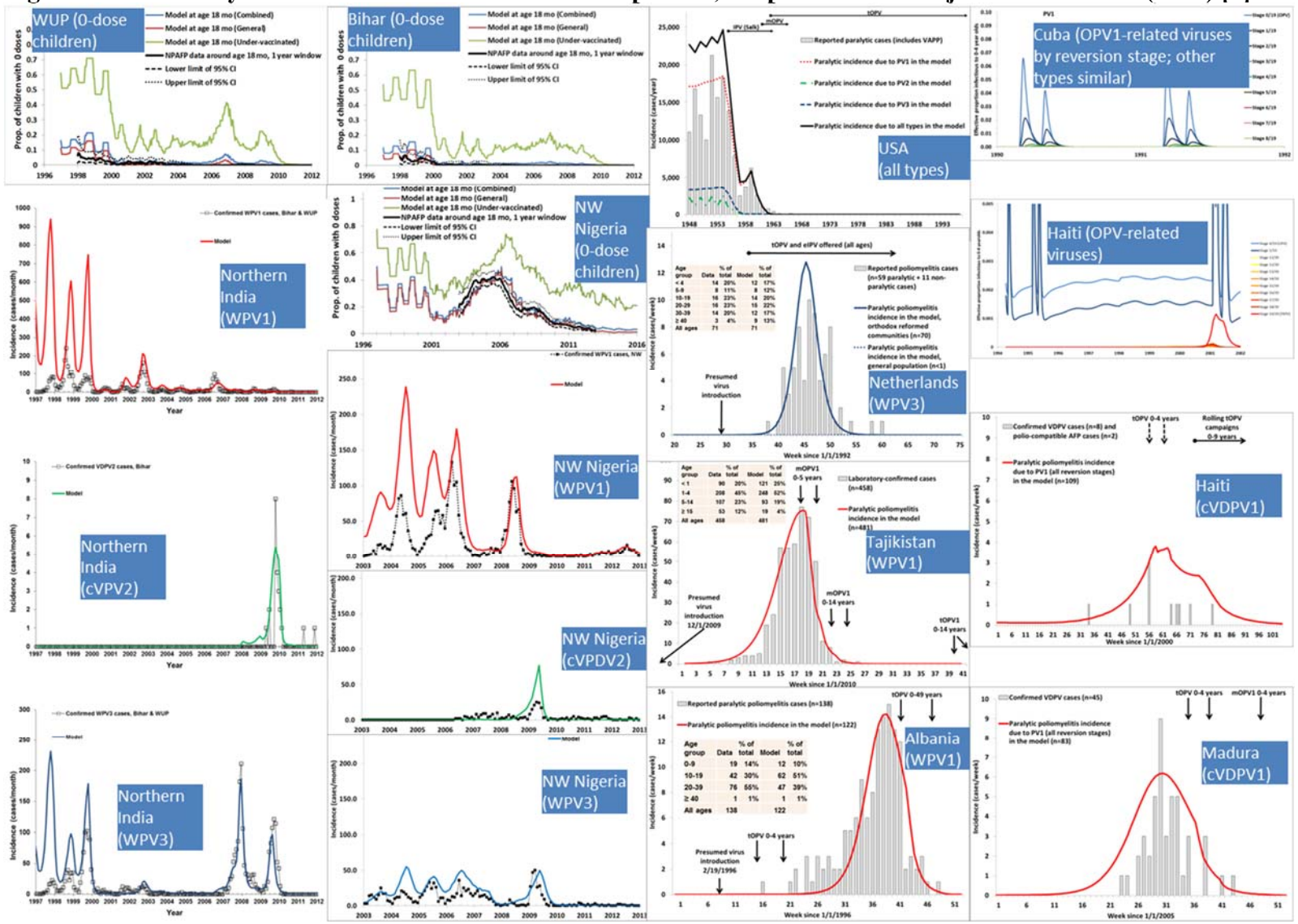


Figure A9: Probability of effective introduction function ($P_{effective}$)

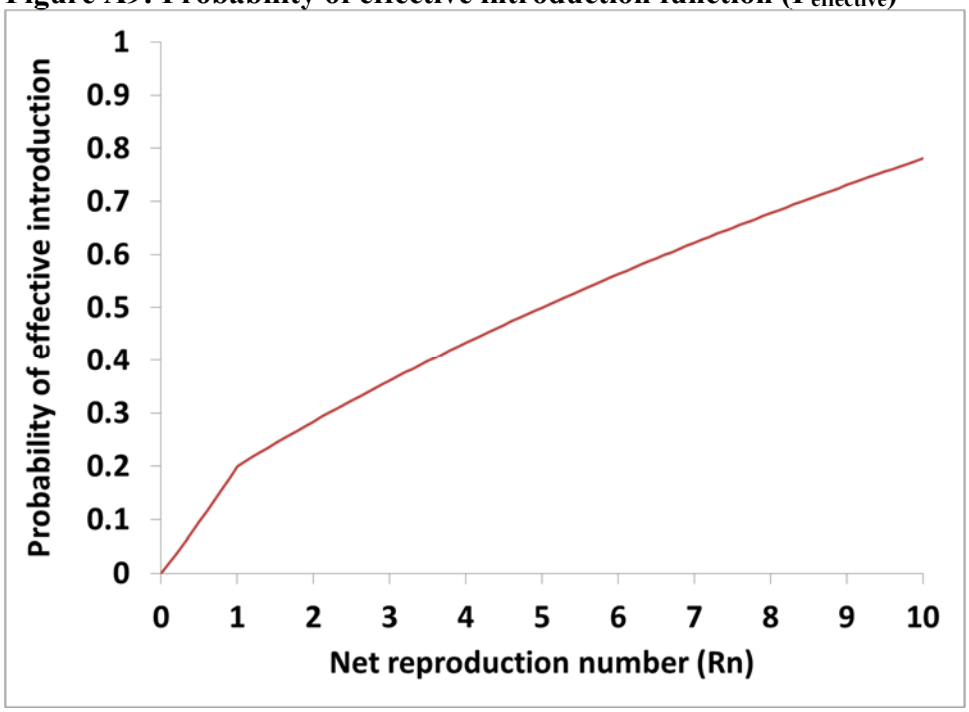


Figure A10: Modeled annual wild poliovirus (WPV) incidence during the 38-year burn-in period relative to the beginning of the analytic time horizon (T_{GPEI}, i.e., January 1, 1988) by serotype.