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A Tale of Two Viruses: Why Smallpox was Eradicated and Polio Persists

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Abstract

The smallpox and poliomyelitis (polio) viruses were, at a time, one of the largest threats to global public health killing millions until global eradication campaigns were put into effect. Vaccination led to the eradication of smallpox and the elimination of polio for most of the world. However, polio continues to persist at endemic levels in Pakistan and Afghanistan. We developed ODE models of smallpox and polio to explore differences in transmission dynamics and determine if the underlying biology has made poliomyelitis more difficult to eradicate. Our model analysis shows there are multiple factors which should allow polio to have a lower threshold for eradication than smallpox: a lower threshold for herd immunity, and vaccines that are more effective at reducing infections and deaths. Thus, our model analysis leads us to conclude that the persistence of polio is due to the persistence of inadequate vaccination rates in the remaining polio-endemic countries.

Keywords: differential equation model, polio, smallpox, biomathematics, vaccination

1 Introduction

The smallpox and poliomyelitis (polio) viruses were, at a time, one of the largest threats to global public health. Millions of individuals were killed or debilitated by these viruses until global eradication campaigns were put into effect. The polio and smallpox viruses share the biological traits of being transmitted human-to-human and of not having any non-human animal reservoirs Other human diseases such as SARS, Ebolavirus, and influenza A have animal reserviors which means that even if these disease is not currently present witin human populations, they can reside in animal populations which have the potential to transmit the disease to humans. Thus, total eradication of smallpox and polio requires only that each disease be completely eliminated within humans. Both smallpox and polio have been targets of global vaccination campaigns. However, while smallpox has been eliminated, polio persists in a few populations globally (though it has been eliminated in many countries).

Smallpox can be traced back to ancient Egypt, where it was consistently spread along trade routes killing every three out of ten infected individuals. Until the World Health Organization (WHO) began their global smallpox eradication campaign in 1959, individual countries had been left to control and manage smallpox outbreaks. Within a span of twenty years through mass vaccination campaigns and surveillance the WHO led the effort to successfully eradicate smallpox in 1980 [9]. The same level of success has not been seen for polio, an infectious disease that paralyzes 1/200 individuals who are infected and kills 5–10% of those who are paralyzed. Global eradication efforts began for polio in 1988 when the world faced an estimated 350,000 cases and have been bolstered since through the 2022–2026 Polio Eradication Strategy [24]. Despite continual attention, funding, and technological advances that were not present during the smallpox eradication campaign, 20% of the world still live in regions that are at risk of endemic polio [10]. Specifically, wild-poliovirus currently exists in Afghanistan and Pakistan, while vaccine-derived poliovirus is present in large numbers in Middle and Western Africa, Yemen, Afghanistan, and Pakistan [24]. Before addressing the question of why polio continues to persist while smallpox was quickly eradicated, the biology of both viruses needs to be understood.

1.1 Biology of smallpox

Smallpox is an infectious disease that is caused by the variola virus, mainly through the variola major strain. It is transmitted through coughing and sneezing and enters the body via respiratory and oral mucus where it then travels to the lymph nodes to enter the blood stream. Smallpox can also be spread through contact with contaminated objects [19]. When an individual contracts the variola virus, they first develop flu-like symptoms. This phase of the infection lasts 2–10 days. Next the individual

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enters the early rash stage of smallpox, where red spots begin to appear in their mouth and throat. An individual will remain in this stage for 2–4 days before progressing to the last stage of infection where the individual develops pustules over their body. The infected individual remains contagious until all of the scabbed over pustules fall off, which takes about 28 days [5]. There is no treatment for smallpox, it can only be prevented via vaccination [6]. The smallpox vaccine is made from vaccinia, a poxvirus that is similar to smallpox but not as deadly. An individual cannot contract smallpox from the vaccine as it is not derived from the variola virus. The smallpox vaccine was typically administered in 2 doses with the potential for a booster [7]. While smallpox outbreaks are no longer a prominent threat to the world, stockpiles of the vaccine still exist in the United States and Russia [19].

1.2 Biology of poliomyelitis

Polio is caused by a poliovirus that enters the blood stream through lymphoid tissue. It is spread through droplets or fecal contamination of eating utensils, food, water or hands. After an individual is infected by poliovirus one of four routes are possible: an individual can (1) be asymptomatic, (2) have minor symptoms like fever and chills, (3) develop non-paralyzing polio, or (4) develop paralyzing polio. In an individual that develops paralyzing polio, the central nervous system becomes infected by the virus and neuronal cells are killed which leads to the loss of muscle strength. Adults are more likely to develop paralyzing polio [4]. After an individual is infected there are no treatment options; therefore, vaccination against polio is crucial. There are two vaccines available against polio, inactivated poliovirus vaccine (IPV) and oral poliovirus vaccine (OPV). IPV consists of a killed poliovirus, provides a high rate of immunity, and is administered intramuscularly. The duration of IPV immunity is unknown and re-infection by wild-type poliovirus is still possible allowing IPV vaccinated individuals to potentially infect non-vaccinated individuals. OPV produces lifelong immunity and is the cheaper and easier option of the two vaccines as it is administered orally in tablet form. Since the OPV vaccine virus replicates in the intestine, it is able to spread via faecal matter which can allow a passive immunization of unvaccinated people in areas with poor hygiene and sanitation. However, it is possible for the OPV vaccine-virus to mutate into the strain that causes paralysis (this strain is known as circulating vacine-derived poliovirus, cVDPV) [10]. Different variations of cVDPV are present depending on the version of OPV in use. For example, in 2016 trivalent OPV (tOPV) was discontinued by the WHO and replaced with bivalent OPV (bOPV) because of a related cVDPV outbreak [11]. The environmental spread of an OPV-derived poliovirus through contamination of water sources is possible. In the United States, IPV is the only form of polio vaccination and is administered in 4 doses in children between the ages of 2 months and 6 years [8].

2 Model Description

The proposed models simulate the transmission dynamics of poliomyelitis and smallpox respectively within populations where vaccination can occur. Each model divides a population into subpopulations (referred to as states) based on disease or vaccination status (i.e., there are subpopulations for susceptible individuals, infected individuals, vaccinated individuals, etc.). The model equations describe the rates at which individuals leave or enter each state. For both models it is assumed that the time in between doses of the vaccine are minimal so there are not different states for each dose of a vaccine.

2.1 Smallpox model

The following smallpox transmission model is influenced by a model proposed by Kaplan et al. [12]. The Kaplan et al. model is designed to simulate contact tracing and queuing for vaccination in the case of a deliberate smallpox attack. The model we present here uses Kaplan's et al. configuration of the multiple stages of infection, and adds separate states for recovered, successfully vaccinated, and unsuccessfully vaccinated. Figure 1 shows a flow diagram representation of the model depicting the rates of transition between model states. Individuals enter the population in the susceptible state (S) at a (population dependent) natural birth rate of Π. Susceptible individuals become infected at a density dependent rate through interactions with infectious individuals, and move to an infectious state (I_1) where individuals have flu-like symptoms. Note, the density dependent transmission rate increases when there are either more infectious individuals or more susceptible individuals. The I_1 infectious individuals progress to another infectious state (I_2) where individuals develop a smallpox rash. The I_2 infectious individuals then progress to a final infectious state (Q) where individuals develop sores and pustules. We assume that individuals in the Q state are visibly ill with smallpox and self quarantine (i.e., we assume their number of contacts is drastically reduced). Individuals remain in the final infectious state Q until they either recover (progressing to the state R) or die from the disease (progressing to the state D). When vaccination campaigns are in place, individuals are vaccinated at a rate of ν with a smallpox vaccine with efficacy ε. Individuals that are successfully vaccinated progress from the susceptible state to a vaccinated protected state (V_P), while individ-

uals for whom the vaccine was not effective progress to a vaccinated but susceptible state (S_V) and can move into the I_1 state if infected. All individuals in the S, I_1, I_2, V_P, R, and S_V states are removed from the population at a (population dependent) natural death rate μ. This model assumes a homogeneously mixing population and an insignificant death rate from vaccines. Additionally, there is no natural death rate when in the quarantined, symptomatic state (Q), because we assume if an individual dies in this state it is from smallpox.

The smallpox model is given by

$$S'(t) = \Pi - \beta S(t)\big(I_1(t) + I_2(t)\big) - \nu S(t) - \mu S(t), \tag{1.1}$$

$$S_V'(t) = \nu(1-\varepsilon)S(t) - \beta S_V(t)\big(I_1(t) + I_2(t)\big) - \mu S_V(t), \tag{1.2}$$

$$I_1'(t) = \beta\big(S(t) + S_V(t)\big)\big(I_1(t) + I_2(t)\big) - r_1 I_1(t) - \mu I_1(t), \tag{1.3}$$

$$I_2'(t) = r_1 I_1(t) - r_2 I_2(t) - \mu I_2(t), \tag{1.4}$$

$$Q'(t) = r_2 I_2(t) - \gamma Q(t), \tag{1.5}$$

$$V_P'(t) = \nu\varepsilon S(t) - \mu V_P(t), \tag{1.6}$$

$$R'(t) = (1-\delta)\gamma Q(t) - \mu R(t), \tag{1.7}$$

$$D'(t) = \delta\gamma Q(t). \tag{1.8}$$

Descriptions, values, and references for the model parameters are given in Table 1.

2.2 Poliomyelitis model

The following poliomyelitis transmission model is influenced by a model proposed by Denes et al. [10] and Sun et al. [20]. The Dene et al. model is designed to simulate the potential spread of polio in a European country where IPV is currently used when interacting with an arriving refugee population which is assumed to receive vaccination using OPV. The Sun et al. model simulates the transmission dynamics of cholera, explicitly modeling the amount of cholera within the local water system. The model we present here uses the Denes et al. model configuration without interacting populations, and adds separate states for vaccination via IPV and OPV, and an additional state for explicitly modeling the amount of poliovirus within local water systems (similar in approach to the Sun et al. model. Figure 2 depicts a flow diagram representation of the model showing the rates of transition between model states. Individuals enter the population in the susceptible state (S) at a (population dependent) natural birth rate of Π. Susceptible individuals can become infected at a density dependent rate through inter-

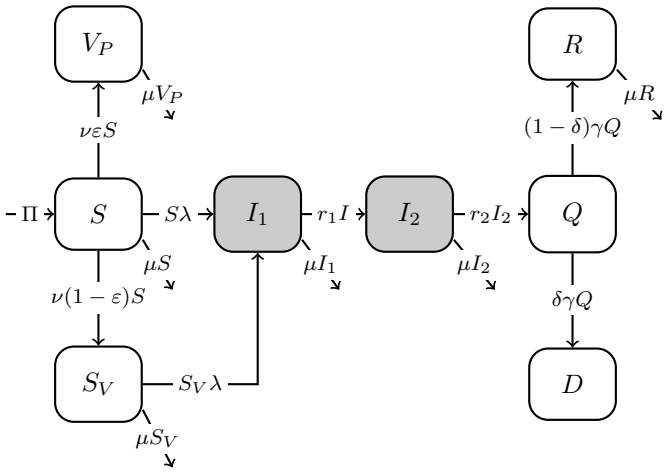

Figure 1: Flow diagram of the smallpox model given by Equations (1.1)–(1.8), where $\lambda = \beta(I_1 + I_2)$. Infectious states are grey.

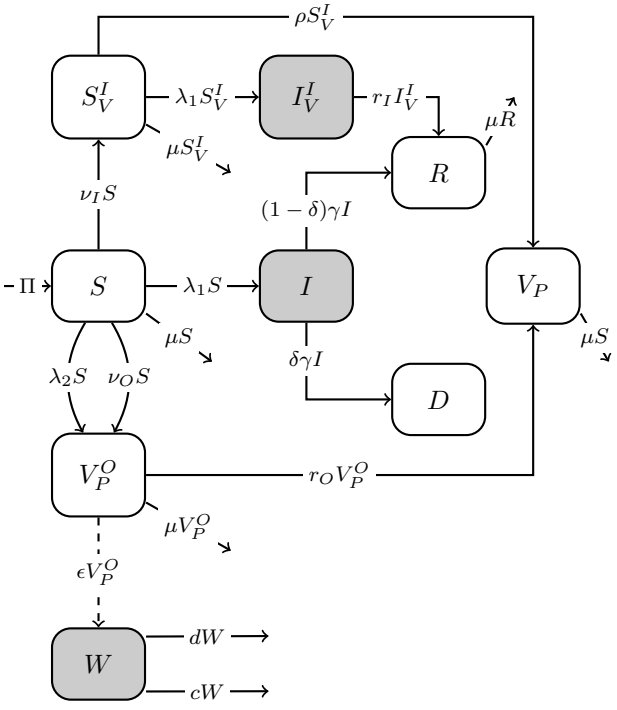

Figure 2: Flow diagram of the poliomyelitis model given by Equations (2.1)–(2.11), where $\lambda_1 = \lambda_h + \theta\lambda_w$ and $\lambda_2 = (1-\theta)\lambda_w$. Infectious states are grey. The dashed line indicates the flow the virus particle, not the movement of individuals.

Table 1: Parameters for smallpox model given by Equations (1.1)–(1.8).

Parameter	Units	Value	Ref	Definition
β	1/(ppl·day)	10^{-6}	[13]	Transmission rate
ε	—	0.975	[12]	Vaccine efficacy
$1/r_1$	days	$[2, 10]$	[5]	I_1 infectiousness period
$1/r_2$	days	$[2, 4]$	[5]	I_2 infectiousness period
$1/\gamma$	days	28	[5]	Recovery period
δ	—	0.3	[12]	Proportion infectious that die from smallpox
μ	1/day	*population dependent*		Natural death rate
Π	ppl/day	*population dependent*		Birth rate
ν	1/day	*policy dependent*		Vaccination rate

actions with infectious individuals and move to an infectious state (I). The density dependent transmission rate increases when there are either more infectious individuals per contaminated water sources or more susceptible individuals. The I infectious individuals either recover from polio (progressing to state R) or die from the disease (progressing to state D).

When OPV vaccination campaigns are in place, individuals are vaccinated at a rate of ν_O and progress to an immune state (V_P^O). In this state, they are able to shed the virus through fecal matter at a rate of ϵ and infect water sources. The model assumes that individuals vaccinated with OPV cannot directly infect susceptible individuals, but that suspectible individuals may become infected through contact with infected water sources (W) which is measured as a concentration of the number of virions per mL. OPV vaccinated indivdiuals (V_P^O) shed virus for $1/r_O$ days after which they progress to state V_P. When the polio virus has entered local water sources, it will naturally decay (and be removed from the water) at a rate of d. Additionally, if water treatment/disinfection measures are in place, the disinfection removes virons from the water at a rate of c (which occurs in addition to the natural decay of the virons). However, while polio virions are in the water, a proportion θ of individuals who consume polio-contaminated water will also become infected and move the infectious state (I). In this case, individuals are infected with cVDPV, however, we include both cVDPV and the wild-type strain infections in the infectious state (I). The remaining $1-\theta$ proportion will gain immunity and move to the OPV vaccinated and protected state (V_P^O).

When IPV vaccination campaigns are in place, individuals are vaccinated at a rate of ν_I and progress to a vaccinated but susceptible state (S_V^I). IPV vaccinated but susceptible individuals are either not exposed to polio and move into the vaccinated protected state (V_P) at a rate of ρ, or become infected at a density dependent rate through interactions with infected individuals and move into a vaccination, infectious state (I_V^I). The I_V^I infectious individuals recover from polio at a rate of r_I, progressing to state R. If both IPV and OPV vaccinations are present, individuals in the IPV vaccinated but susceptible state (S_V^I) can also be infected through consumption of polio-contaminated water.

All individuals in the S, R, S_V^I, V_P and V_P^O states are removed from the population at a (population dependent) natural death rate μ. There is no natural death rate in the infectious state (I), because we assume that if an individual dies in this state, it is from polio.

The poliomyelitis model is given by

$$S'(t) = \Pi - \big(\lambda_h(t) + \lambda_w(t)\big)S(t) - \mu S(t) - (\nu + \nu_o)S(t), \quad (2.1)$$

$$I'(t) = \big(\lambda_h(t) + \theta\lambda_w(t)\big)S(t) - \gamma I(t) - \mu I(t), \quad (2.2)$$

$$S_V^{I\,\prime}(t) = \nu S(t) - \big(\lambda_h(t) + \lambda_w(t)\big)S_V^I(t) - \rho S_V^I(t) - \mu S_V^I(t), \quad (2.3)$$

$$I_V^{I\,\prime}(t) = \big(\lambda_h(t) + \theta\lambda_w(t)\big)S_V^I(t) - r_1 I_V^I(t), \quad (2.4)$$

$$V_P'(t) = \rho S_V^I(t) + r_2 V_P^O(t) - \mu V_P(t), \quad (2.5)$$

$$R'(t) = (1-\delta)\gamma I(t) + r_1 I_V^I(t) - \mu R(t), \quad (2.6)$$

$$V_P^{O\,\prime}(t) = (1-\theta)\lambda_w(t)\big(S(t) + I_V^I(t)\big) + \nu_o S(t) - r_2 V_P^O(t) - \mu V_P^O(t), \quad (2.7)$$

$$W'(t) = \epsilon V_P^O(t) - (c+d)W(t), \quad (2.8)$$

$$D'(t) = \delta\gamma I(t), \quad (2.9)$$

where

$$\lambda_h(t) = \frac{\beta_h\big(I(t)+I_V^I(t)\big)}{S(t)+I(t)+S_V^I(t)+I_V^I(t)+V_P(t)+R(t)+V_P^O(t)}, \quad (2.10)$$

$$\lambda_w(t) = \frac{\beta_w W(t)}{\kappa + W(t)}. \quad (2.11)$$

Equation (2.10) accounts for transmission from the infectious classes (I and V_C^I) and Equation (2.11) accounts for transmission from contaminated water sources (W). Descriptions, values, and references for the model parameters are given in Table 2.

3 Methods

Solutions to the smallpox and polio models, given by Equations (1.1)–(1.8) and Equations (2.1)–(2.11), respectively, were generated using the `NDSolveValue` function in Mathematica, Version 13.0.1.0. All simulations start with five infectious individuals ($I_1(0) = 5$ for the smallpox model; $I(0) = 5$ for the polio model). Lastly, all simulations of the smallpox model, Equations (1.1)–(1.8), were simulated over 500 days, and all simulations of the polio model, Equations (2.1)–(2.11), were simulated over 1400 days. The respective time horizons of 500 and 1400 days were selected as a wide enough time period such that all simulations would return to the disease-free state (i.e., less than 1 infectious individual) by the end of the simulation.

For each model, there are some parameters for which there are a range of estimated values, some parameters which are population dependent, and some parameters which are policy dependent. The population dependent parameters are the initial population size $S(0)$, the birth rate (Π) and natural death rate (μ). All simulations were run assuming a total population size of $S(0) = 659{,}862$ people to simulate the population of Sargodha, Pakistan which is currently classified as a polio endemic area [24, 18]. Correspondingly, the birth rate (Π) and natural death rate (μ) were set to values representing the birth and natural death rates in Pakistan in 2017 ($\Pi = 7.4 \times 10^{-5}$ people/day; $\mu = 1.7 \times 10^{-5}$ people/day) [16]. In our model analysis, we chose to focus on the impact of varying the rate or proportion of the population that is vaccinated. Thus, non-policy dependent parameters for which there exists a range of estimated values were set to a value within the range given in Table 1 (for smallpox) or 2 (for polio). Specifically, for the smallpox model given by Equations (1.1)–(1.8), we set to $1/r_1 = 6$ and $1/r_2 = 3$; all other parameter values are given in Table 1. For the polio model given by Equations (2.1)–(2.11), we set $\beta_w = 10^{-3}$, $\epsilon = 0.001$, $d = 0.05$, $\kappa = 10^4$, $1/\gamma = 35$, $1/r_1 = 23.45$, $\delta = 0.000375$, $\theta = 0.005$; all other parameter values are given in Table 2.

3.1 Vaccination policy

To understand the impact of different vaccination policies on both smallpox and poliomyelitis outbreaks, simulations of each model were generated for a variety of different vaccination policies including (1) no vaccination (a baseline case), (2) vaccinating during an outbreak (at various rates), and (3) having vaccinated various portions of the population prior to an outbreak. Vaccination during an outbreak occurs at a rate of ν in the smallpox model, and at rates ν_O for OPV and ν_I for IPV in the polio model. When vaccination has occurred prior to an outbreak we assume a proportion v of the initial population of 659,862 people is in the vaccinated and protected state V_P at time $t = 0$ while the remainder is susceptible, i.e., $V_P(0) = v \times 659{,}862$ and $S(0) = (1 - v) \times 659{,}862$.

Uncertainty analysis. Uncertainty analysis is a systematic exploration of how model states or outputs change over the set parameter space. Uncertainty analysis is employed to determine the various possible model outcomes when either the values of model parameters are not exactly known or when a range of potential strategies (e.g., vaccination strategies) is to be explored [2]. For the smallpox and polio models given in Equations (1.1)–(1.8) and (2.1)–(2.11), respectively, we used uncertainty analysis to examine the number of infections and cumulative deaths over time given a range of potential vaccination rates.

In the smallpox model, the vaccination rate (ν) was varied over the range $\left[\frac{0.10}{365}, \frac{0.95}{365}\right]$ using 100 unique parameter values evenly spaced at intervals of $\Delta\nu = \frac{0.95/365 - 0.10/365}{100-1}$ (i.e., $\nu = \frac{0.10}{365}, \frac{0.10}{365} + \Delta\nu, \frac{0.10}{365} + 2\Delta\nu, \ldots, \frac{0.95}{365}$). A value of $\nu = \frac{0.10}{365}$ would represent a low vaccination rate of 10% of the unvaccinated population per year being vaccinated. A value of $\nu = \frac{0.95}{365}$ would represent an ambitious vaccination campaign where 95% of the unvaccinated population is vaccinated within a single year. The smallpox model, Equations (1.1)–(1.8), was simulated over 500 days for each value of ν.

In the polio model, the vaccination rates of both OPV and IPV were varied. However, the cost of administering IPV vaccines is about twice that of administering OPV vaccines in lower income, lower middle income, and upper middle income countries [22]. To compare IPV and OPV vaccination strategies of similar cost to administer, we compare OPV vaccination rates to IPV vaccination rates which are half the OPV vaccination rate. Thus, in the polio model, the vaccination rates for both IPV (ν_I) and OPV (ν_O) were varied over the ranges $\left[\frac{0.05}{365}, \frac{0.475}{365}\right]$ and $\left[\frac{0.10}{365}, \frac{0.95}{365}\right]$, respectively, using 100 unique parameter values evenly spaced at intervals of $\Delta\nu_I = \frac{0.475/365 - 0.05/365}{100-1}$ and $\Delta\nu_O = \frac{0.95/365 - 0.10/365}{100-1}$, respectively. The polio model, Equations (2.1)–(2.11), was simulated over 1400 days for each value of ν_I for IPV vaccination strategies and for each value of ν_O for OPV vaccination strategies.

Table 2: Parameters for poliomyelitis model given by Equations (2.1)–(2.11).

Parameter	Units	Value	Ref	Definition
β_h	1/(ppl·day)	0.1	[17]	Human transmission rate
β_w	1/day	10^{-2}	[1]	Water transmission rate
ϵ	virions/(mL·day·ppl)	$[10^{-5}, 1]$	[1]	Virus shed rate of infected
d	1/days	$[10^{-5}, 1]$	[1]	Natural decay rate of virus
κ	virions/mL	$[10^3, 10^5]$	[3, 1]	Half capacity of water to carry virus
$1/\gamma$	days	$[20.3, 49.7]$	[21]	Non-vaccinated recovery rate
$1/r_I$	days	[11.9, 35]	[21]	IPV vaccinated time to immunity
$1/r_O$	days	14	[14]	OPV vaccinated time to immunity
$1/\rho$	days	7	[21]	Time to IPV full immunity
δ	—	[0.00025, 0.0005]	[10]	Proportion infectious that die from polio
θ	—	[0.001, 0.01]	—	Proportion water exposures that result in infection
μ	1/day	*population dependent*		Natural death rate
Π	ppl/day	*population dependent*		Birth rate
ν_I	1/days	*policy dependent*		IPV vaccination rate
ν_O	1/days	*policy dependent*		OPV vaccination rate
c	1/days	*policy dependent*		Disinfection rate of water

Sensitivity analysis. Sensitivity analysis is used to quantify the impact of changes in model parameters to the model outcomes. This provides the ability to find the independent behavior of each parameter even if their effects on the model outcomes are correlated [15].

For each model, we examined the impact of changes in vaccination rates during the outbreak and the proportion of the population vaccinated prior to an outbreak on the percentage of deaths averted over the entire simulation as compared to the baseline case of no vaccination. Let t_f be the final day of the simulation ($t_f = 500$ for smallpox simulations and $t_f = 1400$ for polio simulations) and $\hat{D}(t_f)$ be the cumulative number of deaths in the scenario with no vaccination prior or during an outbreak. Then, the percentage of deaths averted for each simulation is calculated as

$$\% \text{ Deaths Averted} = \frac{\hat{D}(t_f) - D(t_f)}{\hat{D}(t_f)} \times 100. \qquad (3)$$

For the smallpox model, the percentage of deaths averted is calculated from the results of simulations given $\nu = \frac{0.10}{365}, \frac{0.15}{365}, \ldots, \frac{0.95}{365}$ and given the proportion of the population vaccinated prior to the outbreak being $0.10, 0.15, \ldots, 0.95$. For the polio model, the percentage of deaths averted is calculated for OPV vaccination rates of $\nu_O = \frac{0.10}{365}, \frac{0.15}{365}, \ldots, \frac{0.95}{365}$ (assuming an OPV only vaccination strategy) and for comparible (in cost) IPV vaccination rates of $\nu_I = 0.5\nu_O$ (assuming an IPV only vaccination strategy). Additionally, the percentage of deaths averted is calculated from the results of simulations given the proportion of the population vaccinated prior to the outbreak being $v = 0.10, 0.15, \ldots, 0.95$. In this case, we assume vaccinated individuals are fully protected and start in the V_P state. Thus, the only difference between OPV and IPV vaccination strategies is the cost. Again, to compare strategies of similar expense, we assume the proportion vaccinated prior to an outbreak with IPV to be half that which is vaccinated prior to an outbreak with OPV.

4 Results

We generated solutions to the smallpox and polio models given by Equations (1.1)–(1.8) and Equations (2.1)–(2.11), respectively. For all simulations of each model, we recorded the number of infections over the duration of the outbreak, the cumulative number of deaths over the duration of the outbreak, and the percent deaths averted (as given by Equation (3)) at the final time of the simulation. For each model, we consider scenarios with (1) no vaccination (a baseline case), (2) vaccination occurring during the outbreak (at various rates), and (3) vaccination (of various proportions of the population) having occurred prior to the outbreak. Cumulative infections and deaths, outbreak duration, and the percent deaths averted for the no vaccination scenarios, scenarios with 50%/yr vaccination rates (or 25%/yr equivalent for IPV vaccination rates), and scenarios with 50% of the population vaccinated prior to an outbreak (or 25% of the population vaccinated with IPV) are summarized in Table 3.

Table 3: Smallpox and polio cumulative infections and deaths, percent averted deaths, and outbreak duration given by Equations (1.1)–(1.8) and Equations (2.1)–(2.11) model outputs assuming various vaccination (vax) strategies and, in the case of polio, water treatment strategies.

	Vaccination Strategy	% Vax @ $t = 0$	Vax Rate (%/yr)	Cumulative Infections	Cumulative Deaths	% Deaths Averted	Duration (day)
Smallpox	No vax	0	0	657 874	197 333	—	405
	Vax during outbreak	0	50	640 561	192 140	2.63	405
	Vax prior to outbreak	50	0	309 309	92 780	52.98	409
Polio ($c = 0.1$)	No vax	0	0	635 646	238	—	698
	Vax during outbreak (OPV)	0	50	459 499	172	27.71	728
	Vax during outbreak (IPV)	0	25	555 482	207	13.03	711
	Vax during outbreak (OPV+IPV)	0	25+12.5	507 640	190	20.33	719
	Vax prior to outbreak (OPV)	50	0	233 265	87	63.30	1307
	Vax prior to outbreak (IPV)	25	0	447 381	168	29.62	840
Polio ($c = 0.9$)	No vax	0	0	635 646	238	—	698
	Vax during outbreak (OPV)	0	50	463 440	174	27.09	729
	Vax during outbreak (IPV)	0	25	555 482	207	13.03	711
	Vax during outbreak (OPV+IPV)	0	25+12.5	510 013	191	19.96	720
	Vax prior to outbreak (OPV)	50	0	233 265	87	63.30	1307
	Vax prior to outbreak (IPV)	25	0	447 381	168	29.62	840

4.1 Smallpox results

No vaccination (baseline case). For the smallpox model (Equations (1.1)–(1.8)), when no vaccination is administered prior to or during an outbreak, the duration of the outbreak (i.e., the first day upon which $I_1(t) + I_2(t) + Q(t) < 1$) is 405 days. In this case, the cumulative number of infections is 657,874 (almost the entire population), and the total number of deaths due to smallpox is 197,333 over the entire time horizon (500 days). Thus, using the notation of Equation (3), $\hat{D}(500) = 197{,}333$. This is the baseline number of deaths used to calculate the percent of deaths averted in the scenarios where vaccination is used. In Figure 3, this scenario with no vaccination is shown as a dashed line in each of the graphs, showing the number of infections over time (top row) and the graphs showing the cumulative deaths over time (middle row).

Vaccination during an outbreak. We examined smallpox simulations where no one has immunity to smallpox prior to an outbreak and vaccination only occurs once an outbreak has started. Vaccination occurs at a rate of ν which has units of 1/day, and is varied over $\nu \in \left[\frac{0.10}{365}, \frac{0.95}{365}\right]$. However, we report results using measures of a yearly vaccination rate $\nu \times 365$ which is thus varied from a modest 10%/yr up to an ambitious 95%/yr. When the yearly vaccination rate is 50% (i.e., 50% of the susceptible population is vaccinated within one year), then there are 640,561 cumulative infections, 192,140 cumulative deaths, 2.63% of deaths are averted, and the duration of the outbreak is the same as the no vaccination scenario: 405 days (see Table 3). The left column in Figure 3 shows the number of infections over time (top row, solid line) and cumulative deaths over time (middle row, solid line) when the vaccination rate is 50%. Given the speed with which smallpox is spread through the population (99.7% of individuals becoming infected in the first 50 days), a vaccination rate of 50% of the susceptible population vaccinated in one year is insufficient to cause much of a reduction in the number of infections or cumulative deaths over time. The middle column of Figure 3 shows the range of infections over time (top row), cumulative deaths (middle row), and percent deaths averted (bottom row) as the yearly vaccination rate is varied from 10% to 95%. Even at a vaccination rate of 95% per year (i.e., $\nu = 0.95/365$), there are still a cumulative total of 624,978 infections, 187,466 deaths, and only 5.00% deaths averted.

Vaccination prior to an outbreak. We also examined smallpox simulations where a certain portion of the initial susceptible population $S(0)$ is vaccinated prior to an outbreak, thus moving them to either the V_P state in which they have immunity or the S_V state where they remain susceptible. Given a smallpox vaccine efficacy of

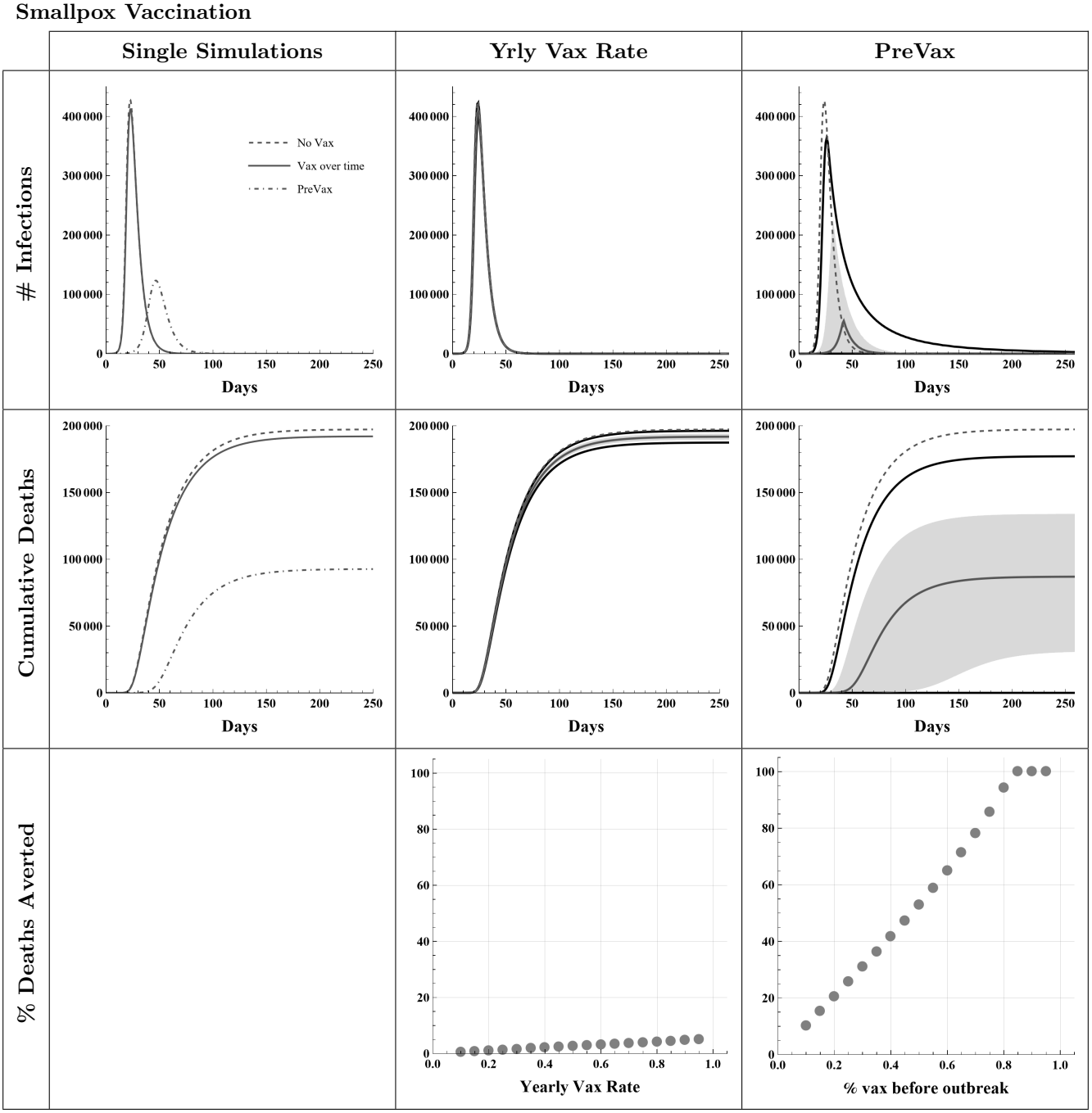

Figure 3: Results from smallpox simulations using different vaccination rates showing number of infections (top row), cumulative deaths (middle and bottom row), and percent deaths averted (bottom row). Results were calculated over 500 days, however, the temporal graphs in the top and middle rows show only 250 days. The left column shows results from scenarios with no vaccination (solid line; $\nu = 0$), with vaccination occurring over time (dashed line; $\nu = 0.5/365$), and a scenario that starts with 50% of the population already vaccinated (dotted line). The other columns show the range of results over a range of yearly vaccination rates $\nu \in [0.1/365, 0.9/365]$ (middle column) and a range of 10%–95% of the population vaccinated prior to an outbreak. The maximum and minimum results are shown respectively by the upper and lower solid black curves, the interquartile range shown as the shaded gray region, and the median is depicted by a solid dark grey line.

$\varepsilon = 0.975$, we assume that 97.5% of susceptible individuals who are vaccinated prior to an outbreak are moved to the V_P state, and only 2.5% are moved to the S_V state. When 50% of the susceptible population is vaccinated prior to an outbreak, there are 309,309 cumulative infections, 92,780 cumulative deaths, 52.98% of deaths are averted, and the duration of the outbreak is 409 days (see Table 3). The left column of Figure 3 shows the number of infections over time (top row, dot-dashed line) and cumulative deaths over time (middle row, dot-dashed line) when 50% of the susceptible population is vaccinated prior to an outbreak. The right column of Figure 3 shows the range of infections over time (top row), cumulative deaths (middle row), and percent deaths averted (bottom row) as the percent vaccinated prior to an outbreak is varied from 10% to 95%. Note at 85%, 90%, and 95% vaccinated prior to an outbreak almost 100% of deaths are averted with cumulative deaths of 14, 4, and 2, respectively.

4.2 Poliomyelitis results

In the polio model (Equations (2.1)–(2.11)), in addition to varying the vaccination rate and proportion vaccinated prior to an outbreak, we examine results for two different disinfection rates of water. Water contaminated with poliomyelitis virus can be disinfected through water treatment and purification processes. We examine results assuming a low disinfection rate of 10% of virions per day ($c = 0.1$) and a high disinfection rate of 90% of virions per day ($c = 0.9$).

No vaccination (baseline case). For the polio model (Equations (2.1)–(2.11)), when no vaccination is administered prior to or during an outbreak, the duration of the outbreak (i.e., the first day upon which $I(t) + I_V^I(t) < 1$) is 698 days for both the low and high disinfection rates ($c = 0.1$ and $c = 0.9$, respectively). For both disinfection rates, the total number of deaths due to polio is 238 over the entire time horizon (1400 days). Thus, using the notation of Equation (3), $\hat{D}(1400) = 238$. This is the baseline number of deaths used to calculate the percent of deaths averted in the scenarios where vaccination is used. Note that the baseline number of deaths during a polio outbreak are orders of magnitude smaller than the number of deaths during a smallpox outbreak because the death rate (δ) is orders of magnitude smaller ($\delta = 0.000375$ for polio; $\delta = 0.3$ for smallpox). Figure 4 shows the no vaccination scenario with disinfection rate $c = 0.1$ as a dashed line showing the number of infections over time (top row) and the cumulative deaths over time (middle row).

Vaccination during an outbreak. We examined polio simulations where no one has immunity to polio prior to an outbreak and vaccination only occurs once an outbreak has started. Vaccination with OPV occurs at a rate of ν_O and vaccination with IPV occurs at a rate of $\nu_I = 0.5\nu_O$. Both parameters have units of %/day, and ν_O is varied over $\nu_O \in \left[\frac{0.10}{365}, \frac{0.95}{365}\right]$. However, we report results using measures of a yearly vaccination rate $\nu_O \times 365$ which is thus varied from a modest 10%/yr up to an ambitious 95%/yr. In Figure 4, OPV simulations are shown in cyan while IPV simulations are shown in magenta.

OPV only. When the yearly vaccination rate of OPV is 50%/yr (with no IPV administered) and there is a low water disinfection rate of $c = 0.1$, then there are 459,499 cumulative infections, 172 deaths, 27.71% deaths averted, and the duration of the outbreak is 728 days (30 days longer than the no vaccination case). The left column in Figure 4 shows the number of infections (top) and cumulative deaths (middle) over time in this case as the cyan curve. If the water disinfection rate is increased to $c = 0.9$, then there are 463,440 cumulative infections, 174 deaths, 27.09% deaths averted, and the duration of the outbreak increases to 729 days. Compared to the lower water disinfection rate ($c = 0.1$), the high water disinfection rates increases cumulative infections by 3,941 and deaths by 2 people, and increases the duration of the outbreak by 1 day. The increase in the cumulative infections and deaths is due to the reduced passive immunity acquired through consumption of polio-contaminated water (since a higher water disinfection rate reduces amount of polio virions found in water). Given the similarities in temporal dynamics of the low and high water disinfection rates, only the low water disinfection rate ($c = 0.1$) is shown in Figure 4. However, a comparison of the results is shown in Table 3.

IPV only. When the yearly vaccination rate of IPV is 25%/yr (a rate which is comparable in administration cost to 50%/yr of OPV only), then for both the low and high water disinfection rates ($c = 0.1$ and $c = 0.9$, respectively) there are 555,482 cumulative infections, 207 deaths, 13.03% deaths averted, and the duration of the outbreak is 711 days (13 days longer than the no vaccination case). In the left column in Figure 4 the number of infections (top) and cumulative deaths (middle) over time is shown in this case as the solid magenta curve. When only IPV is administered, it makes biological sense that an increase in the water disinfection rate will have no impact because the poliomyelitis virus is only shed into the local water sources by individuals vaccinated with OPV in the V_P^O state (see Figure 2).

OPV & IPV. We additionally consider the possibility of a vaccination strategy with a combination of OPV

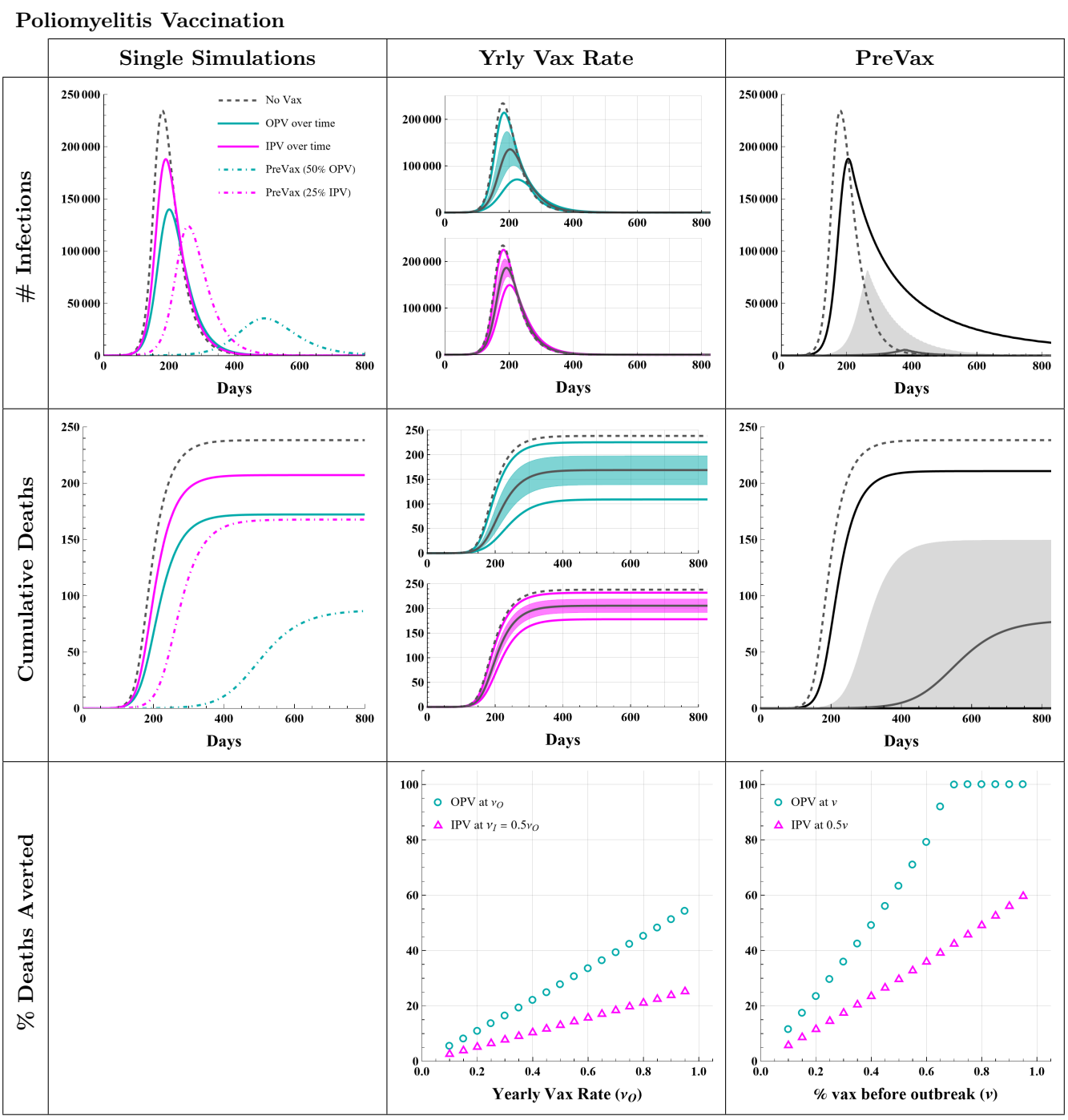

Figure 4: Results from poliomyelitis simulations using different vaccination rates showing number of infections (top row), cumulative deaths (middle and bottom row), and percent deaths averted (bottom row) assuming the lower disinfection rate of $c = 0.1$. Results were calculated over 1400 days, however, the temporal graphs in the top and middle rows show only 800 days. The left column shows results from scenarios with no vaccination (dashed line $\nu = 0$), with vaccination occurring over time (solid cyan line $\nu_O = 0.5/365$; solid magenta line $\nu_I = 0.25/365$), and a scenario that starts with a proportion of the population already vaccinated (dot dashed lines). The other columns show the range of results over a range of yearly vaccination rates $\nu_O \in [0.1/365, 0.9/365]$ and $\nu_I = 0.5\nu_O$ (middle column) and a range of $v = 10\%$–95% of the population vaccinated prior to an outbreak. The maximum and minimum results are shown respectively by the upper and lower solid curves, the interquartile range shown as the shaded region, the median is depicted by a solid dark grey line, and the dashed line show the no vaccination scenario for comparison.

and IPV. An OPV+IPV vaccination strategy which is comparable in administrative cost to a 50%/yr OPV vaccination rate or 25%/yr IPV vaccination rate is a combination of 25%/yr OPV and 12.5% IPV vaccination rates. For these vaccination rates, when there is a low water disinfection rate of $c = 0.1$, then there are 507,640 cumulative infections, 190 deaths, 20.33% deaths averted, and the duration of the outbreak is 719 days. The left column in Figure 4 shows the number of infections (top) and cumulative deaths (middle) over time in this case as the gray curve. If the water disinfection rate is increased to $c = 0.9$, then there are 510,013 cumulative infections, 191 deaths, 19.96% deaths averted, and the duration of the outbreak is 720 days. For both the low and high water disinfection rates, the combined use of OPV and IPV results in fewer cumulative infections and deaths than using only IPV, but more than when using only OPV (Table 3).

The middle column of Figure 4 displays the range of infections over time (top), cumulative deaths over time (middle), and percent deaths averted (bottom) over 1400 days when the OPV vaccination rates are varied from 10%/yr to 95%/yr (shown in cyan), and comparable cost IPV vaccination rates are varied from 5%/yr to 47.5%/yr (shown in magenta). The range in number of infections and cumulative deaths result from 100 simulations using parameter values $\nu_O = \frac{1}{2} \times \frac{0.10}{365}, \frac{1}{2} \times \left(\frac{0.10}{365} + \Delta\nu\right), \frac{1}{2} \times \left(\frac{0.10}{365} + 2\Delta\nu\right) + \cdots + \frac{1}{2} \times \frac{0.95}{365}$ where $\Delta\nu = \frac{0.95/365 - 0.10/365}{100-1}$, and $\nu_I = 0.5\nu_O$. Note, the resulting range in cumulative deaths varies from as high as 225 deaths (5.43% deaths averted) to as low as 109 deaths (54.17% deaths averted) when only OPV vaccination is used, and as high as 232 deaths (2.57% deaths averted) to as low as 178 deaths (25.2% deaths averted) when only IPV vaccination is used.

Vaccination prior to an outbreak. We also examined polio simulations where a proportion v of the initial population has been vaccinated prior to an outbreak, thus moving them to V_P state in which they have immunity. Note, since vaccination with OPV and IPV ultimately results in an individual moving to the V_P state, there is no distinction between the two vaccinations if the vaccination occurs prior to an outbreak. However, to compare the effectiveness of OPV vs IPV vaccination prior to an outbreak which have similar costs to administer, we compared an OPV pre-outbreak proportion of v to an IPV pre-outbreak proportion of $0.5v$. When 50% of the population is vaccinated prior to an outbreak (with either vaccine), then for both the low and high water disinfection rates ($c = 0.1$ and $c = 0.9$) there are 233,265 cumulative infections, 87 cumulative deaths, 63.30% deaths averted, and the duration of the outbreak is 1307 days, almost twice as long as when there is no vaccination (see Table 3). However, since IPV is twices as costly to administer than OPV, we compared the scenario of 50% vaccinated with OPV prior to an outbreak with 25% vaccinated with IPV prior to an outbreak. When only 25% of the population is vaccinated prior to an outbreak, then for both the low and high water disinfection rates there are 447,381 cumulative infections, 168 cumulative deaths, 29.02% deaths averted, and the duration of the outbreak is 840 days. The right column of Figure 4 shows the number of infections over time (top row), cumulative deaths over time (middle row), and percent deaths averted (bottomr row) for proportions $v = 0.1, 0.15, \ldots, 0.95$. Note, when 70% or more of the population is vaccinated prior to an outbreak, almost 100% of deaths are averted (>99.9% of deaths averted).

5 Conclusion

Ordinary differential equations were used to model smallpox and polio transmission with vaccination. The respective model outputs were used to compare how smallpox and polio spread through a population and the amount of vaccination needed to quell an outbreak. Both smallpox and polio are unique in that neither have animal reservoirs where the virus can live, so biologically, both viruses have the potential for eradication. Therefore, the goal of these models is to examine why smallpox has been successfully eradicated while polio has not.

Biologically, these models tell us that smallpox runs through a closed population 6.5 times faster than polio. This, combined with its higher death rate, makes it clear why smallpox was targeted for eradication first. However, the proportion of the population which needs to be immune in order to achieve herd immunity is higher for smallpox (at least 85%) than for polio (at least 70%). Thus, there is a lower threshold for eradication of polio than there was for smallpox which begs the question, why has the world yet to eradicate polio? Though polio has been eliminated in all but two countries (Pakistan and Afghanistan) through vaccination campaigns, global eradication has remained illusive.

The two polio vaccines, IPV and OPV, have different impacts on the virus's persistence in a population. IPV is administered via injection and can lead to herd immunity, but is almost twice as expensive to administer than OPV in lower, lower-middle, and upper-middle income countries. OPV is administered via an oral tablet, making it more economical and easier to transport. Furthermore, in areas with poor hygiene and sanitation, the use of OPV can lead to passive immunization of non-vaccinated individuals through the consumption of polio-contaminated water. However, it is possible that under these conditions the virus can mutate into the strain of polio that

causes paralysis (called circulating vaccine-derived poliovirus (cVDPV)). While the WHO still utilizes OPV in endemic areas, extra precautions and surveillance are in place to quell cVDPV outbreaks via the distribution of OPV variations like novel OPV type 2 (nOPV2) [23]. When comparing OPV and IPV vaccination strategies of similar administrative cost, our model results demonstrated that OPV vaccination strategies were more effective at reducing cumulative infections and deaths, though this was mostly due to the fact that twice as much OPV vaccine could be administered. Additionally, OPV was more effective in areas with poor sanitation (modeled as the low disinfection rate of water, $c = 0.1$) due to the increased capacity for passive immunity to be conferred through consumption of polio-contaminated water. These results, however, should not be interpreted as a lack of endorsement for improving hygienic and sanitary conditions as there are a variety of other pathogens that can also spread through unclean water which were not considered as a part of this study.

The results of our analysis show, that despite the fact that the OPV vaccinations create another possible route for transmission and IPV vaccinations do not provide full coverage for a short period of time, these vaccines are more effective than the smallpox vaccine (at comparable vaccination rates) at reducing cumulative infections and deaths. Combined with the fact that the threshold for herd immunity is lower in polio than for smallpox, we are left to conclude that the persistence of polio is due to the persistence of inadequate vaccination rates in the remaining polio-endemic areas. This conclusion aligns with the risk assessment in the executive summary of the Polio Endgame Strategy 2019–2023 [11], which states, "The greatest risks to reaching eradication are not a matter of science, biology or virology; they are instead a matter of reorienting efforts to current realities that impede the delivery of critical health interventions." A variety of factors including government will, funding, and vaccination distribution logistics contribute to the persistence of inadequate vaccination rates. Future modeling research should construct models that additionally include social interactions, funding structures, vaccination distribution, and movement between localities to simulate current polio vaccination and intervention measures. Agent-based modeling could be an ideal framework for assessing the potential impact of current eradication strategies and quantifying the potential time to eradication.

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Author Contributions

KGM conceived of the project's premise and in all other ways both authors contributed equally in this manuscript.

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