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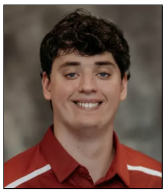
Waterborne Disease Dynamics with a River

*Jackson Leeper, Chad Westphal**



Jackson Leeper worked on this paper as part of a summer research internship between his Sophomore and Junior years at Wabash College. He graduated in 2025 with a double major in Computational Mathematics and Computer Science and hopes to use the analytical tools and the thought process in this research to conduct research in a career of data science.

Chad Westphal is a Professor of Mathematics and Computer Science at Wabash College. His research is primarily in numerical analysis and has enjoyed working with smart and ambitious students in various projects at the intersection of deterministic and stochastic modeling, making good use of tools from dynamical systems, probability, and network analysis.

Abstract

A wide range of waterborne diseases spread through a population through both human-to-human interaction and water-to-human interaction. In this paper, we propose a deterministic compartment model to simulate the transmission of a waterborne pathogen through a population whose common water source is a river with both upstream and downstream access points. This allows for a distinction between drinking and shedding behavior with respect to the upstream and downstream water sources. We consider the effectiveness of several intervention methods with respect to two metrics: the basic reproductive number, R_0, in the epidemic phase and the steady-state infected population fraction, i_∞, in the endemic phase. Using both local and global sensitivity analysis techniques, the relative effectiveness of interventions are demonstrated, leading to a clearer understanding of how to prioritize efforts to either prevent an epidemic or to reduce the endemic level of disease in a population.

Keywords: Compartmental Model, Waterborne Disease, Cholera, Basic Reproductive Number, Parameter Sensitivity
AMS Subject Classification: 92D30, 34C60

*__Corresponding author:__ westphac@wabash.edu

1 Introduction

Waterborne diseases are a present day threat to the health of many people worldwide. Around 2 billion people rely on water sources that may be contaminated with fecal matter, and many diseases, including cholera, dysentery, polio, and others, cause an estimated $485,000$ diarrheal deaths each year [13]. In the U.S., around 7.2 million people get sick from waterborne pathogens per year [6]. It is thus important to understand the dynamics of waterborne diseases and the effectiveness of intervention methods for populations with shared water sources that may become infected.

We consider a compartment model similar to work in [7, 10, 12, 14, 16, 15]. These authors mainly use a SIWR model, which is a common framework for waterborne diseases. The SIWR approach typically includes a single point source for water. In [7], Fung discusses the idea of a river with a flow but his model for cholera transmission only has people getting infected from one source of water. The model we propose in this paper represents a more general approach for waterborne diseases with a flowing water source. Our model includes coupled upstream and downstream water categories. This allows our system to model two scenarios: (1) a river with both upstream and downstream access points, or (2) a recirculating water source with a filter.

We include a wide range of possible interventions through a set of tuneable parameters to effectively study the relative merits of policies designed to prevent or eliminate the transmission of the disease. Similar to [12], we include both symptomatic and asymptomatic infected categories into our model to allow for a distinction in human behavior between those aware or unaware of their ability to spread the disease. Detailed analysis on interventions for related models can be found in [2, 1, 12, 11].

The organization of this paper is as follows. In section section 2 we give a detailed overview of our mathematical model and the relevant assumptions of the disease transmission. We define a set of model parameters and their assumed ranges, and provide an initial solution of the model for a set of default baseline parameters. In section section 3 we use analytical methods to describe key features in an outbreak scenario. We use the next-generation matrix approach [4] to derive the basic reproductive number, R_0, providing insight into the beginning of an outbreak. We also investigate equilibrium solutions of the model to define the steady state level of infection in the endemic state, i_∞ [9]. In section section 4 we perform parameter sensitivity analysis to understand how to effectively decrease R_0 and i_∞ through combinations of interventions. In section section 5 we close with a discussion of the main results and conclusions from both mathematical modeling and public policy points of view.

2 Mathematical Model

We develop our model using ideas similar to those in [7, 10, 12, 14, 16, 15], which focus on aspects of waterborne disease dynamics. We note that, while this model is not limited to a specific disease, cholera transmission is largely consistent with the main ideas we pursue here. See [14, 12] for examples of cholera models. The the key feature of these models are the ability of the disease to spread through both human-to-human and human-to-water interactions. Figure 1 below illustrates our compartment model.

We assume that people infected with the disease may be symptomatic (denoted I_s) or asymptomatic (denoted I_a) and that people show symptoms with probability

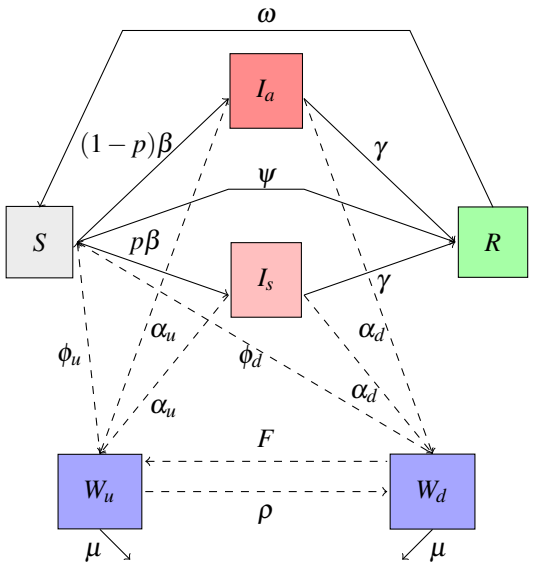


Figure 1: Compartment model illustration for system (1).

p. A person susceptible to the disease (denoted S) may become infected through an interaction with someone from either infected category with contact rate parameters β_s and β_a. This mode of transmission is typically through bodily fluids. A susceptible person may also become infected by interacting with water from either the upstream or downstream source with contact rate parameters ϕ_u and ϕ_d. The likelihood of infection from ingesting water depends on the relative pathogen concentration (denoted W_d and W_u). We assume a natural death rate of the water pollutant of μ. We also assume a uniform flow from upstream to downstream that transports water into and out of each water source with rate ρ. Once a person becomes infected, they shed waste into each water source with rate parameters α_u and α_d. If we assume $\alpha_u = \alpha_d$ and $\phi_u = \phi_d$, then interactions with the two water sources are symmetric and only the flow provides a distinction between upstream and downstream.

We typically assume that our water source is similar to a flowing river, where the flow is simply from upstream to downstream. We also include the possibility of a different configuration that models a water source with a recirculation filter. Under this assumption, water from the downstream source is filtered and returned to the upstream source. The effectiveness of filtration is denoted with the parameter F, where $F = 0$ represents a perfect filter (i.e., uncontaminated water is returned upstream), $F \in (0,1)$ represents an imperfect filter, and $F = 1$ represents a return of unfiltered water. Thus, $F = 0$ also corresponds to the default river configuration with no possibility of pollutant recirculation.

After being infected for some period of time, determined by parameter γ, the individual becomes recovered (denoted R). While in this category, an individual is immune to the disease. However, this immunity may not be permanent and how long the immunity lasts is determined by the parameter ω. Once the immunity is gone, the

individual becomes susceptible again. We also include the possibility of intervention through vaccination, where the susceptible population is vaccinated at a rate determined by the parameter ψ. Under vaccination, susceptible individuals are moved directly to the recovered/removed compartment, where they remain immune for the same time period as one newly recovered from an infection would.

We denote the total number of people in the model by $N = S + I_s + I_a + R$, which remains constant over time. The water compartments behave differently since they are not a part of the population. Both W_u and W_d are calibrated to represent a relative concentration scaled with $W_u, W_d \in [0,1]$. A value of 0 represents uncontaminated water, and 1 corresponds to a maximum concentration equivalent to pure wastewater. Under this scaling, the parameters α_u and α_d can be interpreted as the increase in pollutant concentration from one infected person in one day, when shedding into initially uncontaminated water. Shedding into maximally contaminated water does not increase the concentration. Likewise, the parameters ϕ_u and ϕ_d can be interpreted as the probability of infection for one susceptible individual who ingests the average daily amount of water that is maximally contaminated. It should be noted that these parameters reflect an overall assumption on the size of the water source as well as an average rate of interaction between people and water.

The ODE system for our model is given by

$$\begin{aligned}
\frac{dS}{dt} &= -(\beta_s I_s + \beta_a I_a)S/N - \phi_u S W_u - \phi_d S W_d + \omega R - \psi S \\
\frac{dI_a}{dt} &= (1-p)((\beta_s I_s + \beta_a I_a)S/N + (\phi_u W_u + \phi_d W_d)S) - \gamma I_a \\
\frac{dI_s}{dt} &= p\left((\beta_s I_s + \beta_a I_a)\,S/N + (\phi_u W_u + \phi_d W_d)S\right) - \gamma I_s \\
\frac{dR}{dt} &= \gamma(I_a + I_s) - \omega R + \psi S \\
\frac{dW_u}{dt} &= \alpha_u(1-W_u)(I_a + I_s)/N - \mu W_u - \rho W_u + F\rho W_d \\
\frac{dW_d}{dt} &= \alpha_d(1-W_d)(I_a + I_s)/N - \mu W_d + \rho W_u - \rho W_d,
\end{aligned} \tag{1}$$

which is well-posed when closed by a set of nonnegative initial conditions consistent with $N = S + I_s + I_a + R$ and $W_u, W_d \in [0,1]$ and nonnegative parameters. Over time, human populations will each remain nonnegative with constant N. Water concentrations each remain bounded in $[0,1]$.

Table 1 summarizes the parameters in our model, the permissible range of each, and the default values and units that we use for numerical simulations.

We note that while this model is not specifically calibrated by data on a specific disease, it shares the basic structure and transmission dynamics with cholera, typhoid, and polio. Some of the parameters used in this paper are chosen empirically since they represent a combination of human behavior (e.g., drinking and shedding rates α_u, α_d, ϕ_u, ϕ_d, and vaccination rate ψ) and geographically specific conditions (e.g., river flow rate ρ and filter parameter F). Other parameters are more directly aligned with the particular disease (e.g., transmission rates β_a, β_s, recovery rate γ, pathogen death rate μ, symptomatic probability p, and waning immunity rate ω). In these cases our

Parameter	Range	Default Value
β_a	$[0,\infty)$	0.25 day^{-1}
β_s	$[0,\infty)$	0.2 day^{-1}
γ	$[0,\infty)$	0.1 day^{-1}
α_u	$[0,\infty)$	0.15 day^{-1}
α_d	$[0,\infty)$	0.15 day^{-1}
ϕ_u	$[0,\infty)$	0.2 day^{-1}
ϕ_d	$[0,\infty)$	0.2 day^{-1}
ω	$(0,\infty)$	0.02 day^{-1}
ρ	$[0,\infty)$	0.05 day^{-1}
p	$[0,1]$	0.4
μ	$[0,\infty)$	0.08 day^{-1}
ψ	$[0,\infty)$	0 or 0.01 day^{-1}
F	$[0,1]$	0

Table 1: Ranges, default values, and units of parameters in the model.

parameter selection and associated control ranges were done to be consistent with work in the related literature [7, 15, 16, 12, 2, 8].

Figure 2 shows the solution to system (1) with default parameters and initial conditions $S = 100, I_a = I_s = R = 0, W_u = 0.5, W_d = 0$. We see that this scenario results in an active epidemic phase lasting approximately $40-60$ days, leading into an endemic phase with a consistent portion of the population infected. We also note that each water source remains contaminated in the endemic phase, with a slightly higher asymptotic pollutant level downstream.

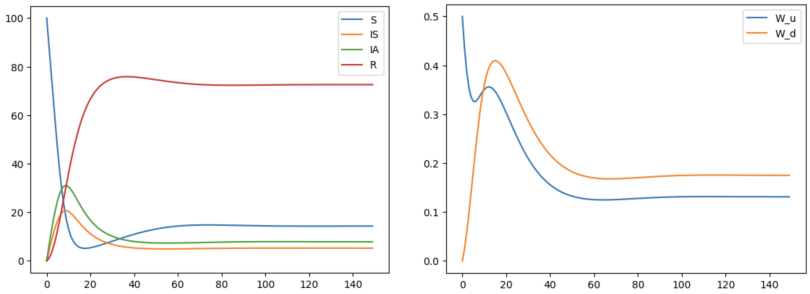


Figure 2: Solution of system (1), human populations (left) and water concentrations (right), for 150 days after an initial water contamination is introduced.

3 Analytical Results

In this section we consider equilibrium solutions and the basic reproductive number for our model. Understanding these concepts analytically provides a basis for predicting the dynamics of an outbreak scenario and gives insights toward potential intervention efforts.

System (1) has two physically relevant equilibrium solutions for parameters in the permissible range. The disease-free equilibrium represents the state with no pathogen present. The second equilibrium solution corresponds to the endemic phase of the disease outbreak. This solution depends on parameter values and is algebraically cumbersome in its general form. Using a value of $N = 100$ and default parameter values from Table 1 results in the equilibrium solution values in Table 2 below. Note that the endemic equilibrium values correspond to the horizontal asymptotes of Figure 2.

	S	I_a	I_s	R	W_u	W_d
Disease-Free Equilibrium	100	0	0	0	0	0
Endemic Equilibrium	12.09	8.19	5.46	74.26	0.13	0.17

Table 2: Values of the two equilibrium solutions with default parameters and $N = 100$.

The stability of the equilibrium solutions can help predict how the system will evolve over time. When parameters are in the permissible range, the endemic solution will be stable. The stability of the disease-free equilibrium solution depends on parameter values and is the basis for deriving the basic reproductive number.

The basic reproduction number, R_0, is the epidemiological concept of how many new infections are generated from each infected individual in the early stages of a potential outbreak. We use the next-generation matrix approach to derive an analytical determination of R_0. A detailed description of this method can be found in [4].

We first compute the Jacobian of the right-hand side of system 1, evaluated at the disease-free equilibrium, given by

$$J = \begin{pmatrix} -\psi & -\beta_a & -\beta_s & \omega & -\phi_u N & -\phi_d N \\ 0 & (1-p)\beta_a - \gamma & (1-p)\beta_s & 0 & (1-p)\phi_u N & (1-p)\phi_d N \\ 0 & p\beta_a & p\beta_s - \gamma & 0 & p\phi_u N & p\phi_d N \\ \psi & \gamma & \gamma & -\omega & 0 & 0 \\ 0 & \alpha_u/N & \alpha_u/N & 0 & -\mu-\rho & F\rho \\ 0 & \alpha_d/N & \alpha_d/N & 0 & \rho & -\mu-\rho \end{pmatrix}. \quad (2)$$

Now consider the rows and columns of that correspond to the infectious components of the system, I_a, I_s, W_u, W_d, and define the resulting 4×4 matrix as $\tilde{J}$. Splitting this into transmission (T) and transition (Σ) components gives $\tilde{J} = T + \Sigma$ with

$$T = \begin{pmatrix} (1-p)\beta_a & (1-p)\beta_s & (1-p)\phi_u N & (1-p)\phi_d N \\ p\beta_a & p\beta_s & p\phi_u N & p\phi_d N \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad (3)$$

and

$$\Sigma = \begin{pmatrix} -\gamma & 0 & 0 & 0 \\ 0 & -\gamma & 0 & 0 \\ \alpha_u/N & \alpha_u/N & -\mu-\rho & F\rho \\ \alpha_d/N & \alpha_d/N & \rho & -\mu-\rho \end{pmatrix}. \tag{4}$$

These matrices generally represent the rates of infection into and out of the overall system. The next-generation matrix $K = -T\Sigma^{-1}$ thus represents a sense of the ratio of these rates. The basic reproductive ratio is given by the spectral radius of K and provides an intuitive epidemic threshold. A value of $R_0 > 1$ indicates a phase of exponential growth in the infected components while $R_0 < 1$ indicates exponential decay in the infected components. For our system we may express the overall reproductive ratio by

$$R_0 = \frac{(1-p)\beta_a + p\beta_s}{\gamma} + \frac{(\mu+\rho)(\alpha_u\phi_u + \alpha_d\phi_d) + \rho(\alpha_u\phi_d + F\alpha_d\phi_u)}{\gamma(\mu^2 + 2\mu\rho + (1-F)\rho^2)}. \tag{5}$$

The first term in R_0 accounts for human-human transmission and the second term accounts for human-water transmission. Using the default values for parameters described in Table 1 results in a default value of

$$R_0 = 7.8,$$

which combines a human-human and human-water components of 2.3 and 5.5, respectively. Further, we note that using $F = 1$ increases the value to $R_0 = 9.8$.

To compare with specific infectious diseases, we note that the basic reproductive number for cholera is 1.7-2.6, polio is 5˜7, measles is 12˜14, and pertussis is 12–17 [5, 3]. The basic reproductive number is a metric that represents conditions present at the beginning of a potential outbreak scenario, and incorporates biological, behavioral, and environmental factors. As such, specific diseases may have different effective R_0 values in different situations. For example, [3] highlights this variation as well as the challenge in estimating the basic reproductive number from measurable epidemiological data.

The analytical formula for R_0 provides a way to understand how the system parameters affect the dynamics of an outbreak. In the next section, we explore this in detail.

4 Parameter Sensitivity and Interventions

A primary motivation in the development of a mathematical model for a disease is to be able to design meaningful intervention strategies. To this end, we focus on two metrics that can help determine the effectiveness of interventions. The first metric is the basic reproductive number, R_0. This quantity provides insight to the beginning of a potential outbreak, addressing the question, “will an epidemic occur in the near future?" The second metric we consider is defined by

$$i_\infty = \lim_{t\to\infty}(I_a + I_s)/N, \tag{6}$$

which provides insight to the possibility of an endemic phase, addressing the question, “will the disease persist in the population over a long time period?" We consider 7 intervention strategies with the goal of reducing R_0 below a value of 1 and to minimize i_∞. As described in section 3, with no interventions, using the default parameters leads to values of $R_0 = 7.8$ and $i_\infty = 0.137$. Each intervention is treated as a unitless constant

that modifies a parameter in the original ODE system (1). Each of the interventions and the range of values considered are summarized in Table 3 below.

Constant	Range	Purpose	Modified Parameter
c_1	$[0,1]$	Hygiene	$c_1\beta_s \to [0.0, 0.2]$
c_2	$[1,3]$	Medical Treatment	$c_2\gamma \to [0.1, 0.6]$
c_3	$[1,3]$	Water Treatment	$c_3\mu \to [0.08, 0.24]$
c_4	$[0,2]$	Shedding Behavior	$c_4\alpha_u, c_4\alpha_d \to [0.0, 0.3]$
c_5	$[0,2]$	Drinking Behavior	$c_5\phi_u, c_5\phi_d \to [0.0, 0.4]$
c_6	$[0,2]$	Vaccination	$c_6\psi \to [0.0, 0.02]$
c_7	$[0,2]$	Flow	$c_7\rho \to [0.0, 0.1]$

Table 3: Purpose and Default values of Interventions

The resulting modified ODE system is given by

$$\begin{aligned}
\frac{dS}{dt} &= -(c_1\beta_s I_s + \beta_a I_a)S/N - (2-c_5)\phi_u S W_u - c_5\phi_d S W_d + \omega R - c_6\psi S \\
\frac{dI_a}{dt} &= (1-p)((c_1\beta_s I_s + \beta_a I_a)S/N + (2-c_5)\phi_u S W_u + c_5\phi_d S W_d) - \gamma I_a \\
\frac{dI_s}{dt} &= p((c_1\beta_s I_s + \beta_a I_a)S/N + (2-c_5)\phi_u S W_u + c_5\phi_d S W_d) - c_2\gamma I_s \\
\frac{dR}{dt} &= \gamma(I_a + c_2 I_s) - \omega R + c_6\psi S \\
\frac{dW_u}{dt} &= (2-c_4)\alpha_u(1-W_u)(I_a+I_s)/N - c_3\mu W_u - c_7\rho W_u + F c_7\rho W_d \\
\frac{dW_d}{dt} &= c_4\alpha_d(1-W_d)(I_a+I_s)/N - c_3\mu W_d + c_7\rho W_u - c_7\rho W_d.
\end{aligned} \tag{7}$$

Assumptions for each of the interventions are given in the following:

- The parameter $c_1 \in [0,1]$ reduces the contact rate between the susceptible and symptomatic infected populations. Public health efforts can educate people to be aware of the danger in contact with people who are showing symptoms of the disease. The number range of this parameter would decrease the β_s value to simulate this intervention. We assume no change in the contact rate with the asymptomatic population.
- The parameter $c_2 \in [1,3]$ represents efforts to administer medical treatment to people showing symptoms of the disease, which will decrease the average recovery time for these individuals. The number range of this parameter would increase the γ value to simulate this intervention. We assume no change in the recovery rate for the asymptomatic population.
- The parameter $c_3 \in [1,3]$ increases the death rate of the pathogen in each water source which reflects efforts to apply a water treatment routine. The number range of this parameter would increase the μ value to simulate this intervention.

- The parameters $c_4, c_5 \in [0,2]$ represent modifications in how people utilize the water sources for drinking and shedding. At the default values of $c_4 = c_5 = 1$, people will access the upstream and downstream water sources for both drinking and shedding with equal rates. Choosing either value in the range $[0,2]$ represents a proportional shift in usage from purely upstream (0) to purely downstream (1). For example, $c_4 = 1.5$ and $c_5 = 0.2$ indicates that the population is primarily shedding in the downstream water source and drinking from the upstream source.

- The parameter $c_6 \in [0,2]$ represents an effort to administer a vaccination routine to the population. When a vaccine is considered, we assume a default deployment rate of $\psi = 0.01 day^{-1}$. Choosing $c_6 = 0$ is consistent with no vaccination and $c_6 = 2$ is consistent with a vaccination campaign that innoculates 2% of the susceptible population per day, starting at the beginning of an outbreak.

- The parameter $c_7 \in [0,2]$ represents a modification of the flow rate of the water from upstream to downstream. The number range of this parameter would increase or decrease the μ value to simulate this intervention.

We first consider the implementation of each intervention in isolation. Figure 3 shows how each intervention affects the value of R_0, where the other parameters are held constant at default values.

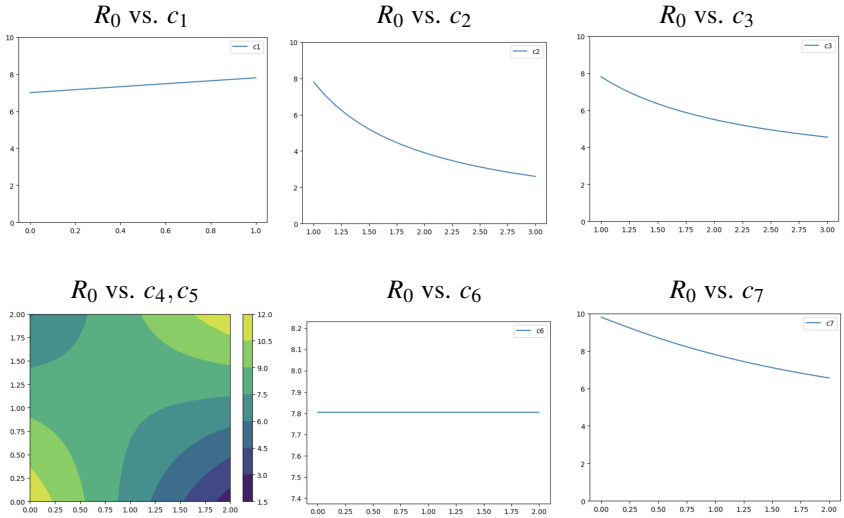


Figure 3: Control parameter local sensitivity for R_0.

It is clear that in most cases, the reduction on R_0 is marginal, with two notable exceptions. Aggressively speeding up the recovery time for symptomatic individuals with $c_2 = 3$ can reduce the basic reproductive number to $R_0 = 2.6$. The contour plot in Figure 3 shows the dependence of R_0 on c_4 (horizontal axis) and c_5 (vertical axis) simultaneously. The coupled effects of shedding and drinking behavior reveal intuitive results. Points $(0,0)$ and $(2,2)$ correspond to using one water source exclusively for both shedding and drinking, resulting in an increase in the basic reproductive number

to $R_0 = 11.5$. At $(0,2)$, we have a value of $R_0 = 5.85$, where people are exclusively shedding upstream and drinking downstream. But the most advisable behavior is clearly at $(2,0)$, which results in $R_0 = 2.3$, and represents exclusively drinking from the upstream water source and shedding into the downstream source.

Next, we test each intervention in isolation for i_∞, as shown in Figure 4.

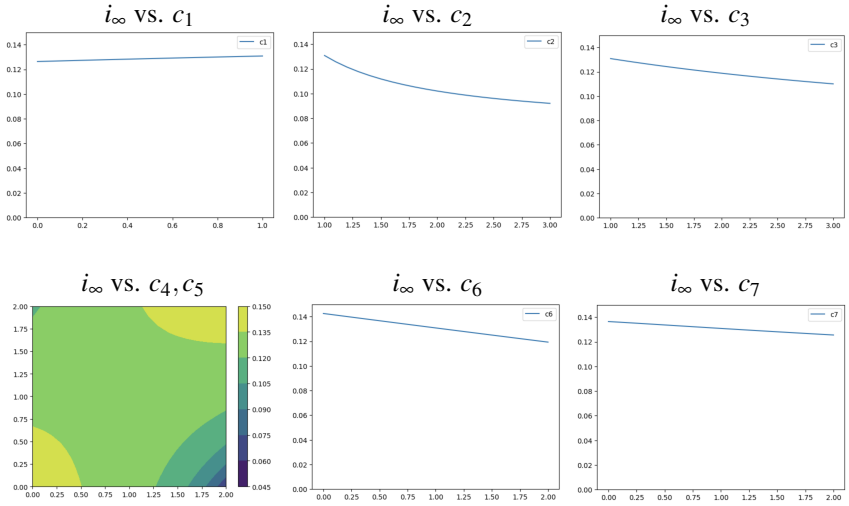


Figure 4: Control parameter local sensitivity for i_∞.

We again see that most interventions have only marginal benefit in reducing i_∞ in isolation. With similar reasoning to the sensitivity to R_0, choosing $(c_4, c_5) = (2,0)$ results in a reduction to $i_\infty = 0.05$.

One additional result of note is that c_6 affects i_∞, even though it does not affect R_0. Thus, implementing a vaccination routine at the onset of an outbreak isn't likely to prevent an epidemic, but it can be effective in reducing the persistence of the disease in the endemic period.

Now, rather than analyzing a rate of vaccine administration, we consider the question of a herd immunity threshold – what fraction of the total population should be vaccinated to ensure that a disease can not enter an epidemic phase? To answer this we reconsider the derivation of the basic reproductive number, but starting from a linearization about the point where $S = (1-V)N$ and $R = VN$, where $V \in [0,1]$ represents the initial fraction of the population immune to the disease. This results in a herd immunity threshold of

$$V = 1 - 1/R_0,$$

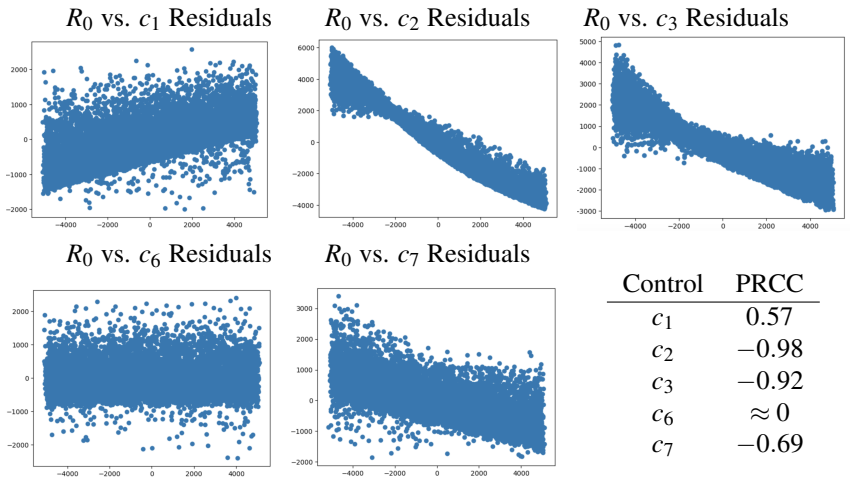
where R_0 is as computed in section 3. Using default values for parameters results in $V = 0.87$.

While it can be valuable to see the sensitivity of R_0 and i_∞ to one intervention at a time, it is perhaps more realistic to consider the combined effects of multiple interventions. We use Latin Hypercube Sampling (LHS) and Partial Rank Correlation Coefficient (PRCC) analysis to gain a more global view of parameter sensitivity. We describe the LHS/PRCC approach briefly here, but a more detailed overview of can be

found in [11]. Importantly, we note that we focus this analysis on the combined effect of control parameters c_1, c_2, c_3, c_6 and c_7, excluding c_4 and c_5 since their effect on R_0 and i_∞ is nonmonotonic (a necessary assumption for PRCC since it is based on linear regression).

We start with a Latin Hypercube Sampling (LHS) of 10000 values, where each parameter chosen independently and uniformly random for each parameter in the ranges described in Table 3. This sampling produces 10000 corresponding values for R_0 and i_∞. We use the analytical result for R_0 in equation (5) and use numerical solutions of system (7) to determine $i\infty$.

With this data, Partial Rank Correlation Coefficient (PRCC) values are computed to estimate the sensitivity of R_0 and i_∞ to each variable, while taking the effect of the other variables into account. Scatter plots of the subsequent residual ranking and a chart of PRCC values are shown in Figures 5, 6 and 7. In all cases, the PRCC values are statistically significant at the $p = 0.001$ level.

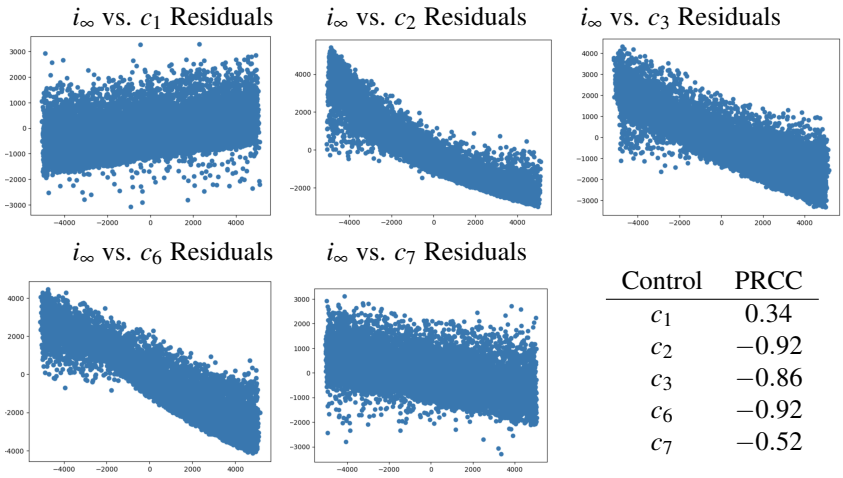


Control	PRCC
c_1	0.57
c_2	-0.98
c_3	-0.92
c_6	≈ 0
c_7	-0.69

Figure 5: LHS/PRCC results for global R_0 sensitivity.

These results show that the most effective overall interventions for reducing R_0 are c_2 (medical treatment) and c_3 (water treatment). To reduce the likelihood of an outbreak, this therefore suggests a policy that focuses resources on both reducing the time that individuals are infected as well as increasing the rate of contaminant removal. We also note that since R_0 is not dependent on c_6 the PRCC value is essentially zero and the plot of residual ranking shows no correlation.

The most effective overall interventions for reducing i_∞ are c_2 (medical treatment), c_3 (water treatment), and c_6 (vaccination). This suggests that in the endemic phase it remains important to focus on medical and water treatment, but also provides evidence for the effectiveness of implementing a vaccination routine to keep endemic infection levels as low as possible.



Control	PRCC
c_1	0.34
c_2	−0.92
c_3	−0.86
c_6	−0.92
c_7	−0.52

Figure 6: LHS/PRCC results for global i_∞ sensitivity.

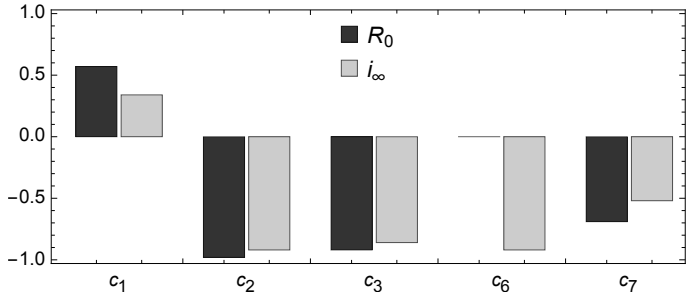


Figure 7: PRCC Values for R_0 and i_∞.

5 Discussion and Conclusion

Water-based pathogens can have the potential to spread very rapidly in a population that uses a shared water source. Some common waterborne diseases like cholera and polio are highly infectious and can spread easily throughout a population without the proper precautions. Understanding the dynamics under dual transmission pathways is an important tool in assessing the relative merits of potential intervention methods and their effects on both the epidemic and endemic phases of an outbreak.

To illustrate the effectiveness of a strategic combination of parameters, consider the scenario with $c_2 = 3$, $c_4 = 2$, and $c_5 = 0$. Here we assume a medical treatment procedure where symptomatically infected people remain infectious for approximately 3.3 days instead of 10, and a policy where people exclusively obtain drinking water from the upstream source and direct wastewater exclusively in the downstream source. This results in $R_0 = 0.76$ and $i_\infty = 0.0$, which means that under these conditions the introduction of the pathogen to a new population is unlikely to result in an epidemic

and that any level of infection in the population should reduce to negligible values over time. The model here assumes a homogeneous and well-mixed population of people, which generally limits it to a relatively small scale. It is possible to generalize the approach here to include heterogeneous populations by implementing a network model. Within this framework it is also possible to further distinguish behavioral responses or interventions on individuals in subsets of populations which could lead to differences in threshold parameters or time scales in the dynamics.

Bibliography

[1] I.B. Augsburger, G.K. Galanthay, J.H. Tarosky, J. Rychta, and D. Taylor, *Voluntary vaccination may not stop monkeypox outbreak: A game-theoretic model*, PLoS Negl Trop Dis **16** (2002), no. 12, 1–29.

[2] O.C. Collins and K.J. Duffy, *Analysis and optimal control intervention strategies of a waterborne disease model: A realistic case study*, Journal of Applied Mathematics **2018** (2018), 1–14.

[3] PL Delamater, EJ Street, TF Leslie, Y. Yang, and KH Jacobsen, *Complexity of the basic reproduction number (r0).*, Emerg Infect Dis. **25** (2019), no. 1, 1–4.

[4] O. Diekmann, J.A. Heesterbeek, and M.G. Roberts, *The construction of next-generation matrices for compartmental epidemic models*, J R Soc Interface **7** (2010), no. 47, 873–885.

[5] M Doherty, P. Buchy, B. Standaert, C. Giaquinto, and D. Prado-Cohrs, *Vaccine impact: Benefits for human health.*, Vaccine **34** (2016), no. 52, 6707–6714.

[6] Center for Disease Control, *Waterborne disease in the United States*, (2023), https://www.cdc.gov/healthywater/surveillance/burden/index.html.

[7] I. C.-H. Fung, *Cholera transmission dynamic models for public health practitioners*, Emerg Themes Epidemiol **11** (2014), no. 1, 1–11.

[8] J.I. Irunde, J.Z. Ndendya, J.A. Mwasunda, and P.K. Robert, *Modeling the impact of screening and treatment on typhoid fever dynamics in unprotected population*, Results in Physics **54** (2023), 107120.

[9] G. Kozyreff, *Asymptotic solutions of the SIR and SEIR models well above the epidemic threshold*, medRxiv **9** (2022), no. 12, 1–15.

[10] M. Li, J. Ma, and P. van den Driessche, *Model for disease dynamics of a waterborne pathogen on a random network*, J Math Biol **71** (2015), no. 4, 961–977.

[11] S. Marino, I.B. Hogue, C.J. Ray, and D.E. Kirschner, *A methodology for performing global uncertainty and sensitivity analysis in systems biology*, J Theor Biol **254** (2008), no. 1, 178–196.

[12] R.L.M. Neilan, E. Schaefer, and H. Gaff, *Modeling optimal intervention strategies for Cholera*, Bull. Math. Biol. **72** (2010), no. 1, 2004–2018.

[13] World Health Organiztion, *Drinking-water*, Sept. 2023, https://www.who.int/news-room/fact-sheets/detail/drinking-water.

[14] S.L. Robertson, M.C. Eisenberg, and J.H. Tien, *Heterogeneity in multiple transmission pathways: modelling the spread of cholera and other waterborne disease in networks with a common water source*, Journal of Biological Dynamics **7** (2013), no. 1, 254–275, PMID: 24303905.

[15] N. Shah, *Deterministic mathematical model for dynamics of water borne diseases*, Advances in Research **2** (2014), 515–522.

[16] J.H. Tien and D.J.D. Earn, *Multiple transmission pathways and disease dynamics in a waterborne pathogen model*, Bull. Math. Biol. **72** (2010), no. 1, 1506–1533.