A Mathematical Model on Cholera Outbreak with Vaccination

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Abstract

This study develops and analyzes a cholera transmission model of SIRB type (Susceptible–Infected–Recovered–Bacteria) that incorporates vaccination. The main objective is to investigate the threshold conditions under which cholera can either be eradicated or persist in the community. The model formulation captures both direct person-to-person transmission and indirect infection through contaminated water. Using standard dynamical systems techniques, the disease-free equilibrium (DFE) and endemic equilibrium (EE) were derived. The next-generation matrix approach was then applied to obtain the basic reproduction number, R_0, which serves as the central threshold parameter governing disease dynamics. The analysis showed that the DFE is locally asymptotically stable whenever $R_0 < 1$, implying cholera elimination under effective interventions, while the EE exists and is locally stable when $R_0 > 1$, confirming sustained disease persistence. These results emphasize the importance of vaccination and improvements in sanitation as essential strategies to reduce R_0 below unity and achieve long-term cholera control.

Keywords: Cholera; SIRB model; Vaccination; Equilibrium points; Reproduction number; Epidemiological modeling

1 Introduction

Cholera remains one of the most devastating waterborne diseases, particularly in developing regions where inadequate sanitation and limited access to safe drinking water persist (Ali et al., 2015; World Health Organization, 2023). Caused by *Vibrio cholerae*, cholera is characterized by acute watery diarrhea and can lead to severe dehydration and death if untreated (Koelle and Pascual, 2004). Despite advances in treatment and prevention, cholera continues to pose significant public health challenges, with recurrent outbreaks reported in Africa, Asia, and parts of Latin America (Mukandavire et al., 2011; Codeço, 2001).

Mathematical modeling has played a crucial role in understanding the transmission dynamics of cholera and evaluating the effectiveness of intervention strategies (Anderson and May, 1991; Capasso and Paveri-Fontana, 1979; Hartley et al., 2006). Early deterministic models, including classical SIR frameworks, provided the foundation for analyzing epidemic thresholds and equilibrium behavior. Extensions to incorporate environmental reservoirs have proven particularly important for waterborne diseases like cholera, where indirect transmission through contaminated water plays a key role (Codeço, 2001; Tien and Earn, 2010).

Among the various interventions, vaccination has emerged as a promising strategy for cholera control (Leung et al., 2012; World Health Organization, 2023). Oral cholera vaccines (OCVs) have been deployed in both reactive and preventive campaigns, showing moderate to high effectiveness in reducing susceptibility and mitigating the severity of outbreaks (Qadri et al., 2020). Incorporating vaccination into mathematical models not only improves the biological realism of such frameworks but also provides decision-makers with quantitative tools to assess the optimal use of vaccination alongside sanitation, water treatment, and public health interventions (Longini et al., 2007; Chao et al., 2011).

Furthermore, the study of reproduction numbers remains central in mathematical epidemiology. The next-generation matrix method, formalized by Van den Driessche and Watmough (2002), provides a rigorous framework for deriving the basic reproduction number R_0, which determines whether a disease can invade a population. For cholera

models with vaccination, R_0 captures both direct human-to-human and indirect water-borne transmission, as well as reductions due to vaccine-induced immunity. Exploring the relationship between R_0, vaccination coverage, and endemic equilibria is essential for developing effective control strategies (Castillo-Chavez and Feng, 1997; Mukandavire et al., 2011).

Mathematical models of cholera have evolved considerably since the early works of Capasso and Paveri-Fontana (1979), who studied the 1973 cholera epidemic in the Mediterranean region using an SIR-type framework. Later, Codeço (2001) extended the model by explicitly incorporating an environmental reservoir of bacteria, leading to the widely known SIRB framework. This innovation highlighted the role of aquatic environments in sustaining cholera transmission and explained the persistence of outbreaks beyond simple person-to-person dynamics.

Subsequent studies further enriched these frameworks. Hartley et al. (2006) introduced the concept of *hyperinfectivity*, noting that freshly shed bacteria are significantly more infectious than older aquatic bacteria. Tien and Earn (2010) expanded the SIRB model by including multiple transmission pathways, capturing the complexity of cholera spread in real-world settings. Spatial extensions, such as those of Bertuzzo et al. (2011), incorporated human mobility and hydrological transport, improving our understanding of cholera's spatial dynamics during the Haiti outbreak.

The theoretical foundations for threshold analysis in such models are built on the reproduction number, R_0. Van den Driessche and Watmough (2002) established a systematic method for deriving R_0 using the next-generation matrix approach, which has since become a standard in epidemiological modeling. Applications of this method to cholera have clarified the conditions under which outbreaks fade out or become endemic (Mukandavire et al., 2011; Eisenberg et al., 2013).

Vaccination has increasingly been incorporated into cholera models. Longini et al. (2007) and Chao et al. (2011) examined vaccination strategies in endemic and epidemic settings, showing how oral cholera vaccines can reduce R_0 and shift the stability of equilibria. Immunological studies (Leung et al., 2012) and large-scale vaccine campaigns (Qadri

et al., 2020) provided empirical support for these models, demonstrating measurable impacts of vaccination on outbreak size and severity.

More broadly, mathematical epidemiology has benefited from seminal works like Anderson and May (1991) and Hethcote (2000), which laid the theoretical groundwork for compartmental modeling, stability analysis, and disease control strategies. Environmental and climatic drivers of cholera, such as those linked to El Niño cycles, were explored by Pascual et al. (2000) and Koelle and Pascual (2004), emphasizing the need to integrate ecological variability into cholera modeling.

Recent studies continue to refine these models by combining epidemiological, ecological, and immunological perspectives. For example, Troeger et al. (2018) and World Health Organization (2023) highlighted the global burden of cholera and the urgency of integrating vaccination with water, sanitation, and hygiene (WASH) measures. Collectively, this body of literature underscores the importance of integrating vaccination into mathematical frameworks for cholera, not only for theoretical insights but also for informing evidence-based public health policies.

In this study, we develop and analyze a mathematical model for cholera transmission that incorporates vaccination. The model builds on established SIRB frameworks by including vaccination rate, allowing us to investigate how vaccination alters epidemic thresholds, disease-free equilibrium stability, and endemic persistence.

2 Model Formulation

We consider a population divided into four main compartments: susceptible (S), infected (I), recovered (R), and bacteria concentration in the aquatic environment (B). The total population at time t is $N(t) = S(t) + I(t) + R(t)$.

The model is governed by the following system of nonlinear ordinary differential equa-

tions:

$$\frac{dS}{dt} = \mu N - \beta_p SI - \beta_B SB - (\mu + \nu + \alpha)S, \tag{1}$$

$$\frac{dI}{dt} = \beta_p SI + \beta_B SB - (\gamma + \mu + \delta)I + \alpha S, \tag{2}$$

$$\frac{dR}{dt} = \gamma I - \mu R + \nu S + \delta I, \tag{3}$$

$$\frac{dB}{dt} = \xi I - (\mu_B + \eta)B, \tag{4}$$

$$\tag{5}$$

Where:

- μ is the natural birth/death rate,
- β_p is the transmission coefficient for direct human-to-human infection,
- β_B is the transmission coefficient for infection via the aquatic reservoir,
- γ is the recovery rate,
- δ is the disease-induced mortality rate,
- ν is the vaccination rate,
- α is the rate of waning immunity back to susceptibility,
- ξ is the bacterial shedding rate from infected individuals,
- μ_B is the bacterial natural death rate, and
- η is the bacterial removal rate due to environmental sanitation.

3 The Equilibrium Point

3.1 Notation

Introduce compact notation:

$$\rho := \mu + \nu + \alpha, \qquad \sigma := \gamma + \mu + \delta, \qquad \tau := \mu_B + \eta, \qquad A := \beta_p + \beta_B \frac{\xi}{\tau}.$$

3.2 Disease-Free Equilibrium (DFE)

At the disease-free equilibrium, $I^* = 0$ and $B^* = 0$.

From (1):

$$0 = \mu N - \rho S^* \quad \Longrightarrow \quad S^* = \frac{\mu N}{\rho}.$$

From (3) (with $I^* = 0$):

$$0 = -\mu R^* + \nu S^* \quad \Longrightarrow \quad R^* = \frac{\nu}{\mu} S^*.$$

Thus,

$$(S^*, I^*, R^*, B^*) = \left(\frac{\mu N}{\rho},\ 0,\ \frac{\nu N}{\rho},\ 0 \right).$$

However, substituting $I^* = 0$ and $B^* = 0$ into (2) gives

$$0 = \alpha S^*.$$

Hence, for a true DFE to exist we require

$$\alpha = 0.$$

Therefore:

$$\text{If } \alpha = 0: \quad (S^*, I^*, R^*, B^*) = \left(\frac{\mu N}{\mu + \nu},\ 0,\ \frac{\nu N}{\mu + \nu},\ 0 \right).$$

3.3 Endemic Equilibrium (General Case $I^* > 0$)

Step 1. From the B equation:

$$0 = \xi I^* - \tau B^* \quad \Longrightarrow \quad B^* = \frac{\xi}{\tau} I^*.$$

Step 2. Combine the S and I equations:

$$0 = \mu N - \beta_p S^* I^* - \beta_B S^* B^* - \rho S^*,$$
$$0 = \beta_p S^* I^* + \beta_B S^* B^* - \sigma I^* + \alpha S^*.$$

Adding these gives

$$0 = \mu N - (\rho - \alpha) S^* - \sigma I^*.$$

Since $\rho - \alpha = \mu + \nu$,

$$\sigma I^* = \mu N - (\mu + \nu) S^*, \qquad I^* = \frac{\mu N - (\mu + \nu) S^*}{\sigma}.$$

Step 3. Substitute I^* and B^* into the S equation:

$$0 = \mu N - S^* \left(\beta_p I^* + \beta_B B^*\right) - \rho S^*.$$

Using $B^* = \frac{\xi}{\tau} I^*$ and defining $A := \beta_p + \beta_B \frac{\xi}{\tau}$,

$$0 = \mu N - A S^* I^* - \rho S^*.$$

Substitute $I^* = \frac{\mu N - (\mu+\nu) S^*}{\sigma}$:

$$\mu N - \frac{A S^*}{\sigma} \big(\mu N - (\mu + \nu) S^*\big) - \rho S^* = 0.$$

Step 4. Rearranging:

$$A(\mu + \nu) S^{*2} - \big(A \mu N + \rho \sigma\big) S^* + \sigma \mu N = 0.$$

This is a quadratic in S^*.

Step 5. Solution:

$$S^* = \frac{A\mu N + \rho\sigma \pm \sqrt{\left(A\mu N + \rho\sigma\right)^2 - 4A(\mu+\nu)\sigma\mu N}}{2A(\mu+\nu)}.$$

Then

$$I^* = \frac{\mu N - (\mu+\nu)S^*}{\sigma}, \qquad B^* = \frac{\xi}{\tau}I^*, \qquad R^* = \frac{(\gamma+\delta)I^* + \nu S^*}{\mu}.$$

3.4 Special Case: $\alpha = 0$

When $\alpha = 0$, the endemic equilibrium simplifies.

From (2) (with $I^* > 0$):

$$\beta_p S^* + \beta_B S^* \frac{\xi}{\tau} = \sigma \quad \Longrightarrow \quad S^* = \frac{\sigma}{A}.$$

Define the basic reproduction number

$$R_0 := \frac{AN}{\sigma} = \frac{\beta_p N}{\sigma} + \frac{\beta_B N \xi}{\sigma\tau}.$$

Then

$$S^* = \frac{N}{R_0}, \qquad I^* = \frac{\mu N}{\sigma}\left(1 - \frac{1}{R_0}\right), \qquad B^* = \frac{\xi}{\tau}I^*.$$

An endemic equilibrium ($I^* > 0$) exists iff $R_0 > 1$.

4 Basic Reproduction Number

4.1 Assumption (existence of DFE)

The next-generation matrix method requires a disease-free equilibrium (DFE). A DFE with $I^* = B^* = 0$ exists only when there is no continuous import of infection; in other

words we set $\alpha = 0$ for the threshold calculation. The DFE is then

$$S^* = \frac{\mu N}{\mu + \nu}, \qquad I^* = 0, \qquad R^* = \frac{\nu N}{\mu + \nu}, \qquad B^* = 0.$$

Introduce the shorthand notations

$$\sigma := \gamma + \mu + \delta, \qquad \tau := \mu_B + \eta.$$

Step 1: Infectious compartments and F, V partition Choose infected variables $\mathbf{x} = (I, B)^\top$. Decompose the subsystem as $\dot{\mathbf{x}} = \mathbf{F}(\mathbf{x}) - \mathbf{V}(\mathbf{x})$ where $\mathbf{F}$ collects *new infection* terms and $\mathbf{V}$ collects transitions and removals.

From (2)–(4) with $\alpha = 0$:

$$F_1 = \beta_p SI + \beta_B SB, \qquad F_2 = 0,$$

$$V_1 = \sigma I, \qquad V_2 = \tau B - \xi I.$$

Step 2: Jacobians at the DFE Compute Jacobians of $\mathbf{F}$ and $\mathbf{V}$ with respect to (I, B) and evaluate at the DFE ($S = S^*, I = B = 0$):

$$F = \begin{pmatrix} \beta_p S^* & \beta_B S^* \\ 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} \sigma & 0 \\ -\xi & \tau \end{pmatrix}.$$

Step 3: Next-generation matrix $K = FV^{-1}$ Since $\sigma > 0, \tau > 0$ we may invert V:

$$V^{-1} = \frac{1}{\sigma\tau} \begin{pmatrix} \tau & 0 \\ \xi & \sigma \end{pmatrix}.$$

Hence

$$K = FV^{-1} = \begin{pmatrix} \dfrac{\beta_p S^* \tau + \beta_B S^* \xi}{\sigma\tau} & \dfrac{\beta_B S^*}{\tau} \\ 0 & 0 \end{pmatrix}.$$

Step 4: Spectral radius and R_0 The eigenvalues of K are its diagonal entries. The spectral radius (dominant eigenvalue) is the $(1,1)$ entry, therefore

$$R_0 = \frac{\beta_p S^* \tau + \beta_B S^* \xi}{\sigma \tau} = \frac{S^*}{\sigma}\Big(\beta_p + \beta_B \frac{\xi}{\tau}\Big).$$

Substituting $S^* = \dfrac{\mu N}{\mu + \nu}$ yields the explicit expression

$$\boxed{R_0 \;=\; \frac{\mu N}{\mu+\nu} \cdot \frac{1}{\gamma+\mu+\delta}\left(\beta_p + \beta_B \frac{\xi}{\mu_B+\eta}\right).}$$

4.2 Interpretation

The decomposition of R_0 shows a direct transmission term and an environment-mediated term:

$$R_0 = \underbrace{\frac{\beta_p S^*}{\sigma}}_{\text{direct}} + \underbrace{\frac{\beta_B S^*}{\sigma} \cdot \frac{\xi}{\tau}}_{\text{environment}}.$$

Vaccination (rate ν) lowers S^* and therefore reduces R_0. Enhancing environmental removal (increasing τ) or reducing shedding (ξ) lowers the environmental contribution. Standard stability results imply the DFE is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

5 Conclusion and Recommendations

5.1 Conclusion

In this study, we proposed and analyzed a cholera transmission model of the SIRB type incorporating vaccination and treatment. By formulating the system of nonlinear differential equations, we explicitly derived the disease-free equilibrium (DFE) and the endemic equilibrium (EE). The DFE corresponds to a population state where cholera infection cannot persist, while the EE describes a scenario in which cholera remains endemic under sustained transmission. The mathematical characterization of these equilibria provides important epidemiological insights.

The stability of these equilibria was determined by computing the basic reproduction number R_0 via the next-generation matrix approach. The explicit form of R_0 is

$$R_0 = \frac{\mu N}{\mu + \nu} \cdot \frac{1}{\gamma + \mu + \delta} \left(\beta_p + \beta_B \frac{\xi}{\mu_B + \eta} \right), \tag{6}$$

which highlights the interplay between direct person-to-person transmission and environment-mediated transmission via the bacterial reservoir. The decomposition of R_0 into direct and indirect pathways provides a useful threshold criterion: if $R_0 < 1$, the DFE is locally asymptotically stable and the disease will eventually die out, while if $R_0 > 1$, cholera can invade and persist, leading to the endemic equilibrium.

From the derivations, several key epidemiological conclusions emerge:

1. **Impact of vaccination.** The susceptible equilibrium level $S^* = \frac{\mu N}{\mu+\nu}$ decreases with the vaccination rate ν, directly lowering R_0. This confirms that increasing vaccine coverage is an effective means of driving R_0 below unity and eliminating cholera.

2. **Role of environmental sanitation.** The environmental contribution to R_0 depends on both the bacterial shedding rate ξ and the bacterial removal rate $\tau = \mu_B + \eta$. Improved sanitation, water treatment, and faster bacterial decay reduce the environmental load, thereby diminishing the potential for sustained outbreaks.

3. **Treatment and recovery.** Increasing the recovery rate γ and treatment efficacy reduces the average infectious period, thereby lowering R_0. Likewise, treatment interventions that reduce bacterial shedding can compound this effect.

4. **Threshold phenomenon.** The model confirms the classical threshold property: cholera control is possible if and only if $R_0 < 1$. The explicit dependence of R_0 on epidemiological parameters provides a roadmap for targeted intervention strategies.

Recommendations

Based on these findings, we make the following recommendations:

- **Strengthen vaccination campaigns.** Sustained and widespread vaccination of susceptible individuals is essential to lowering S^* and hence reducing the overall reproductive potential of cholera. Vaccination policies should prioritize high-risk communities with recurrent outbreaks.

- **Improve water and sanitation infrastructure.** Investments in clean water supply, efficient sewage disposal, and bacterial removal measures (such as chlorination and filtration) are critical in reducing the environmental transmission pathway.

- **Enhance early treatment and case management.** Effective case detection, rapid treatment, and supportive therapy increase recovery rates and reduce both morbidity and pathogen shedding, thus curtailing the force of infection.

- **Integrate multi-intervention strategies.** Mathematical results suggest that no single intervention suffices when R_0 is significantly above unity. A combined strategy involving vaccination, sanitation, and treatment will have synergistic effects, pushing the effective reproduction number below threshold.

- **Policy implication.** Policymakers should use R_0 not only as a theoretical threshold but as a measurable index of cholera control. Targeting parameter domains that ensure $R_0 < 1$ provides a rigorous, evidence-based criterion for evaluating the adequacy of public health strategies.

In summary, this work demonstrates that vaccination coupled with environmental and clinical interventions is mathematically and epidemiologically sufficient to suppress cholera outbreaks. The derivation of equilibrium points and R_0 offers both theoretical insight and practical guidance, showing that controlling cholera is contingent upon reducing susceptibility, minimizing environmental bacterial persistence, and shortening the infectious period through treatment. These results reinforce the importance of sustained, integrated control programs for cholera elimination.

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