Local Stability of SIRB Equilibrium Points

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Abstract

This paper analyzes the local stability of a cholera transmission model of SIRB type (Susceptible–Infected–Recovered–Bacteria), incorporating vaccination and treatment. The disease-free equilibrium (DFE) and endemic equilibrium (EE) were derived, and their stability was investigated using the Jacobian matrix and Routh–Hurwitz criteria. Results show that the DFE is locally asymptotically stable when $R_0 < 1$, ensuring disease elimination, while for $R_0 > 1$ the DFE becomes unstable and the system converges to a stable EE. A numerical example with biologically realistic parameters confirmed the theoretical findings. The study concludes that reducing R_0 below unity through vaccination and improved sanitation is essential for sustainable cholera control.

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

1 Introduction

Cholera continues to be one of the most persistent waterborne diseases, disproportionately affecting populations in regions with poor sanitation, inadequate access to clean water, and fragile health systems. The World Health Organization (2019) estimates millions of cases annually, with recurrent epidemics posing significant socio-economic burdens in sub-Saharan Africa and South Asia. The disease is caused by *Vibrio cholerae*, which

transmits both directly through contaminated water and food and indirectly via environmental reservoirs. These dual transmission pathways make cholera dynamics particularly challenging to analyze and control (Codeço, 2001; Hartley et al., 2006). Mathematical models have become indispensable tools in epidemiology, allowing researchers to explore transmission dynamics, evaluate interventions, and identify critical thresholds for disease persistence or eradication (Anderson and May, 1991; Brauer and Castillo-Chavez, 2012). The susceptible–infectious–recovered (SIR) framework, introduced by Kermack and McKendrick (1927), laid the foundation for modeling epidemics, but cholera requires an extension to account for environmental bacterial concentrations. This motivated the development of SIRB models, which explicitly incorporate a bacterial reservoir and thus capture the ecological persistence of *Vibrio cholerae* (Capasso and Paveri-Fontana, 1979; Codeço, 2001). In cholera modeling, two mathematical concepts are central: the equilibrium points of the system and the basic reproduction number, R_0. The disease-free equilibrium describes conditions under which cholera can be eradicated, while the endemic equilibrium represents sustained disease presence. The threshold nature of R_0, formalized through the next-generation matrix method (Driessche and Watmough, 2002), distinguishes between elimination ($R_0 < 1$) and persistence ($R_0 > 1$). Yet, understanding whether these equilibria are locally stable is equally critical, since stability determines whether small perturbations—such as imported infections—die out or trigger sustained outbreaks (Hethcote, 2000). Recent work has emphasized integrating vaccination and treatment into cholera models. For instance, Onuorah et al. (2022) formulated a variable-population cholera model with vaccination, highlighting the importance of immunization in long-term control. Similarly, Edward and Nyerere (2015) introduced a model with treatment and control measures, showing that medical interventions significantly alter equilibrium stability. These studies underscore that beyond identifying equilibria and thresholds, analyzing their stability under intervention scenarios is indispensable for effective policymaking.

The application of mathematical modeling in infectious disease dynamics has a long tradition, with early milestones including the work of Kermack and McKendrick (1927),

who introduced the classical SIR model. This model established the conceptual framework for understanding epidemic thresholds and herd immunity. Anderson and May (1991) later expanded this perspective in *Infectious Diseases of Humans*, emphasizing the role of reproduction numbers and control strategies across diverse diseases. In the context of cholera, Capasso and Paveri-Fontana (1979) provided one of the first models that incorporated waterborne transmission, analyzing the 1973 Mediterranean epidemic. Codeço (2001) further advanced cholera modeling by explicitly including bacterial reservoirs, leading to the widely used SIRB framework. This was complemented by Hartley et al. (2006), who introduced the concept of hyperinfectivity, showing how environmental amplification of *Vibrio cholerae* shapes epidemic waves. The concept of the basic reproduction number, R_0, and its role in threshold analysis was rigorously formalized by Driessche and Watmough (2002), whose next-generation matrix approach remains a standard in epidemiological modeling. Wang and Zhao (2004, 2008) extended these results to heterogeneous and periodic environments, providing insights into more complex settings. Hethcote (2000) provided a general review of the mathematics of infectious diseases, reinforcing the centrality of equilibrium analysis. Modern cholera models have incorporated interventions such as vaccination, treatment, and sanitation. Mukandavire and Liao (2011) modeled vaccination impacts, while Mukandavire et al. (2011) analyzed the Haitian cholera outbreak, estimating reproduction numbers and vaccination thresholds. Onuorah et al. (2022) integrated vaccination and variable populations, while Edward and Nyerere (2015) examined control measures including treatment and sanitation. Optimal control approaches have also been developed: Okosun and Makinde (2014) modeled treatment and sanitation, while Agusto and Adekunle (2014) studied prevention and treatment jointly. These works highlight the necessity of embedding control measures into stability analysis. Stability considerations have been extensively studied in the general epidemic modeling literature. Bawa et al. (2014) analyzed cholera dynamics with optimal control and showed how stability is influenced by intervention strategies. Similarly, Brauer and Castillo-Chavez (2012) emphasized the interplay between equilibrium stability and population heterogeneity. Taken together, these studies illustrate that while equilibrium and

reproduction numbers provide threshold conditions, stability analysis is indispensable for understanding the robustness of these equilibria under real-world perturbations. This paper therefore contributes to the growing body of research by providing a comprehensive local stability analysis of SIRB equilibrium points under vaccination and treatment, extending the theoretical and practical insights into cholera dynamics. By focusing on local stability, this work provides not only theoretical insight but also practical guidance for designing robust cholera control strategies. Through stability analysis and numerical simulations, we aim to provide insights into the conditions under which vaccination can prevent large-scale outbreaks or eradicate the disease.

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 equations:

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

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

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

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

$$\tag{5}$$

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.

Notation and Preparatory Identities

Introduce the commonly used combinations:

$$\rho := \mu + \nu + \alpha, \quad \sigma := \gamma + \mu + \delta, \quad \tau := \mu_B + \eta.$$

Also recall the disease-free equilibrium (DFE), which requires $\alpha = 0$:

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

For compactness we denote S^* simply by S^* when substituted.

3 Jacobian Matrix (General Point)

The Jacobian $J(S, I, R, B)$ of the right-hand side with respect to $x = (S, I, R, B)$ has entries:

$$\frac{\partial \dot{S}}{\partial S} = -\beta_p I - \beta_B B - \rho, \quad \frac{\partial \dot{S}}{\partial I} = -\beta_p S, \quad \frac{\partial \dot{S}}{\partial R} = 0, \quad \frac{\partial \dot{S}}{\partial B} = -\beta_B S,$$

$$\frac{\partial \dot{I}}{\partial S} = \beta_p I + \beta_B B + \alpha, \quad \frac{\partial \dot{I}}{\partial I} = \beta_p S - \sigma, \quad \frac{\partial \dot{I}}{\partial R} = 0, \quad \frac{\partial \dot{I}}{\partial B} = \beta_B S,$$

$$\frac{\partial \dot{R}}{\partial S} = \nu, \quad \frac{\partial \dot{R}}{\partial I} = \gamma + \delta, \quad \frac{\partial \dot{R}}{\partial R} = -\mu, \quad \frac{\partial \dot{R}}{\partial B} = 0,$$

$$\frac{\partial \dot{B}}{\partial S} = 0, \quad \frac{\partial \dot{B}}{\partial I} = \xi, \quad \frac{\partial \dot{B}}{\partial R} = 0, \quad \frac{\partial \dot{B}}{\partial B} = -\tau.$$

Thus

$$J(S, I, R, B) = \begin{pmatrix} -\beta_p I - \beta_B B - \rho & -\beta_p S & 0 & -\beta_B S \\ \beta_p I + \beta_B B + \alpha & \beta_p S - \sigma & 0 & \beta_B S \\ \nu & \gamma + \delta & -\mu & 0 \\ 0 & \xi & 0 & -\tau \end{pmatrix}.$$

4 Local Stability of the Disease-Free Equilibrium (DFE)

At the DFE we set $\alpha = 0$. Then $I^* = B^* = 0$, and $R^* = \frac{\nu S^*}{\mu}$.

Reordering the state as (I, B, S, R), the Jacobian becomes block lower-triangular:

$$J_{\text{DFE}} = \begin{pmatrix} J_{11} & 0 \\ J_{21} & J_{22} \end{pmatrix},$$

where

$$J_{11} = \begin{pmatrix} \beta_p S^* - \sigma & \beta_B S^* \\ \xi & -\tau \end{pmatrix}, \quad J_{22} = \begin{pmatrix} -\rho & 0 \\ \nu & -\mu \end{pmatrix},$$

and

$$J_{21} = \begin{pmatrix} -\beta_p S^* & -\beta_B S^* \\ \gamma + \delta & 0 \end{pmatrix}.$$

4.1 Eigenvalues of J_{22}

The block J_{22} is triangular with eigenvalues

$$\lambda_3 = -\rho < 0, \quad \lambda_4 = -\mu < 0.$$

4.2 Eigenvalues of J_{11} and R_0

The block

$$J_{11} = \begin{pmatrix} \beta_p S^* - \sigma & \beta_B S^* \\ \xi & -\tau \end{pmatrix}.$$

Characteristic polynomial:

$$\det(J_{11} - \lambda I) = (\beta_p S^* - \sigma - \lambda)(-\tau - \lambda) - \beta_B S^* \xi.$$

Equivalently,

$$\lambda^2 - (\beta_p S^* - \sigma - \tau)\lambda - \left[\tau(\beta_p S^* - \sigma) + \beta_B S^* \xi\right] = 0.$$

4.3 Determinant and Trace

Determinant:

$$\det(J_{11}) = \tau\sigma - S^*(\tau\beta_p + \beta_B \xi).$$

By definition of R_0,

$$S^*(\beta_p + \tfrac{\beta_B \xi}{\tau}) = \sigma R_0 \quad \Rightarrow \quad S^*(\tau\beta_p + \beta_B \xi) = \tau\sigma R_0.$$

Thus

$$\det(J_{11}) = \tau\sigma(1 - R_0).$$

Trace:

$$\operatorname{tr}(J_{11}) = (\beta_p S^* - \sigma) - \tau.$$

If $R_0 < 1$, then $\beta_p S^* - \sigma < 0$, so $\operatorname{tr}(J_{11}) < -\tau < 0$.

4.4 DFE Stability Result

$$\text{DFE is locally asymptotically stable} \iff R_0 < 1,$$

and unstable if $R_0 > 1$.

5 Local Stability of the Endemic Equilibrium (EE)

When $R_0 > 1$, an endemic equilibrium $(\bar{S}, \bar{I}, \bar{R}, \bar{B})$ with $\bar{I}, \bar{B} > 0$ exists.

The Jacobian at EE:

$$J_{EE} = \begin{pmatrix} -\beta_p \bar{I} - \beta_B \bar{B} - \rho & -\beta_p \bar{S} & 0 & -\beta_B \bar{S} \\ \beta_p \bar{I} + \beta_B \bar{B} & \beta_p \bar{S} - \sigma & 0 & \beta_B \bar{S} \\ \nu & \gamma + \delta & -\mu & 0 \\ 0 & \xi & 0 & -\tau \end{pmatrix}.$$

Characteristic polynomial:

$$\det(J_{EE} - \lambda I) = \lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4,$$

with coefficients

$$a_1 = -\mathrm{tr}(J_{EE}), \quad a_2 = \text{sum of principal } 2 \times 2 \text{ minors},$$

$$a_3 = -\text{sum of principal } 3 \times 3 \text{ minors}, \quad a_4 = \det(J_{EE}).$$

5.1 Routh–Hurwitz Stability Criterion

The EE is locally asymptotically stable if and only if

$$a_1 > 0, \quad a_2 > 0, \quad a_3 > 0, \quad a_4 > 0,$$

$$a_1 a_2 - a_3 > 0, \quad (a_1 a_2 - a_3) a_3 - a_1^2 a_4 > 0.$$

5.2 Interpretation

- By biological positivity of parameters and equilibrium components, usually $a_i > 0$.

- The EE emerges through a transcritical bifurcation at $R_0 = 1$, and is typically locally stable for $R_0 > 1$.

6 Summary

- Disease-Free Equilibrium: stable $\iff R_0 < 1$.
- Endemic Equilibrium: exists for $R_0 > 1$ and typically stable (verified via Routh–Hurwitz).

7 Numerical Example: Endemic Equilibrium and Routh–Hurwitz Verification for the SIRB Model

Model (for reference)

We consider the SIRB model

$$\dot{S} = \mu N - \beta_p SI - \beta_B SB - \mu S - \nu S - \alpha S,$$

$$\dot{I} = \beta_p SI + \beta_B SB - \gamma I - \mu I - \delta I + \alpha S,$$

$$\dot{R} = \gamma I - \mu R + \nu S + \delta I,$$

$$\dot{B} = \xi I - \mu_B B - \eta B,$$

with the shorthand

$$\rho = \mu + \nu + \alpha, \qquad \sigma = \gamma + \mu + \delta, \qquad \tau = \mu_B + \eta.$$

We use the exact Jacobian evaluated at a general equilibrium (S, I, R, B):

$$J(S,I,R,B) = \begin{pmatrix} -\beta_p I - \beta_B B - \rho & -\beta_p S & 0 & -\beta_B S \\ \beta_p I + \beta_B B + \alpha & \beta_p S - \sigma & 0 & \beta_B S \\ \nu & \gamma + \delta & -\mu & 0 \\ 0 & \xi & 0 & -\tau \end{pmatrix}.$$

Parameter choice (biologically plausible)

We normalize $N = 1$ (fractions). Parameter values (per day) used in this example:

Parameter	Value	Description
μ	$3.913\,89 \times 10^{-5}$	natural death rate (1/(70 yr) in days)
ν	$1.000\,00 \times 10^{-4}$	vaccination rate (per day)
α	0	infection import rate (set to 0)
β_p	0.600 000	direct transmission coefficient (per day)
β_B	0.200 000	environmental transmission coefficient (per day)
γ	0.200 000	recovery rate (1/5 day^{-1})
δ	0.010 000 0	additional removal/treatment rate (per day)
μ_B	0.500 000	natural bacterial decay (per day)
η	0.100 000	bacterial removal (per day)
ξ	0.500 000	bacterial shedding rate (per day)

Derived constants:

$$\rho = \mu+\nu+\alpha = 0.000\,139\,139, \qquad \sigma = \gamma+\mu+\delta = 0.210\,039, \qquad \tau = \mu_B+\eta = 0.600\,000.$$

7.1 Basic reproduction number

Using the DFE susceptible value $S^* = \dfrac{\mu N}{\mu + \nu}$ and the next-generation result, we compute

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

Numerically,

$$S^* = 0.281\,294, \qquad R_0 = 1.026\,75.$$

Thus $R_0 > 1$ and the model admits a biologically admissible endemic equilibrium.

7.2 Solve for the endemic equilibrium (EE)

We solve the steady-state algebraic system

$$\dot{S} = \dot{I} = \dot{R} = \dot{B} = 0$$

numerically. The solution returned by a root solver (fsolve) is:

$$\bar{S} = 0.273\,964,$$
$$\bar{I} = 4.855\,61 \times 10^{-6},$$
$$\bar{R} = 0.726\,031,$$
$$\bar{B} = 4.046\,34 \times 10^{-6}.$$

(Interpretation: with these parameters R_0 is just slightly above 1; hence the endemic prevalence is very small but positive.)

7.3 Jacobian evaluated at the EE

Substitute the equilibrium into the Jacobian to obtain (numerically):

$$J_{EE} \approx \begin{pmatrix} -1.4286 \times 10^{-4} & -1.6438 \times 10^{-1} & 0 & -5.4793 \times 10^{-2} \\ 3.7226 \times 10^{-6} & -4.5661 \times 10^{-2} & 0 & 5.4793 \times 10^{-2} \\ 1.0000 \times 10^{-4} & 2.1000 \times 10^{-1} & -3.9139 \times 10^{-5} & 0 \\ 0 & 5.0000 \times 10^{-1} & 0 & -6.0000 \times 10^{-1} \end{pmatrix}.$$

7.4 Quartic characteristic polynomial coefficients

For the characteristic polynomial

$$\det(\lambda I - J_{EE}) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4,$$

the coefficients computed from traces / minors of J_{EE} are

$$a_1 = -\operatorname{tr}(J_{EE}) = 0.645\,843,$$

$$a_2 = \tfrac{1}{2}\big((\operatorname{tr} J)^2 - \operatorname{tr}(J^2)\big) = 0.000\,118\,128,$$

$$a_3 = -\tfrac{1}{6}\big((\operatorname{tr} J)^3 - 3\operatorname{tr} J \operatorname{tr}(J^2) + 2\operatorname{tr}(J^3)\big) = 4.727\,74 \times 10^{-7},$$

$$a_4 = \det(J_{EE}) = 1.836\,16 \times 10^{-11}.$$

7.5 Eigenvalues

The numerical eigenvalues of J_{EE} (roots of the quartic) are:

$$\lambda_1 \approx -3.9139 \times 10^{-5}, \qquad \lambda_2 \approx -6.4566 \times 10^{-1},$$

$$\lambda_{3,4} \approx -7.1342 \times 10^{-5} \pm 8.4942 \times 10^{-4}\, i.$$

All eigenvalues have strictly negative real parts in this example.

7.6 Routh–Hurwitz verification

For a quartic polynomial the Routh–Hurwitz conditions for all roots to have negative real parts are:

$$a_1 > 0, \quad a_2 > 0, \quad a_3 > 0, \quad a_4 > 0,$$

$$a_1 a_2 - a_3 > 0,$$

$$(a_1 a_2 - a_3) a_3 - a_1^2 a_4 > 0.$$

Plugging the computed coefficients gives:

$$a_1 = 0.645\,800 > 0, \quad a_2 => 0, \quad a_3 => 0,$$

$$a_4 => 0,$$

$$a_1 a_2 - a_3 \ => > 0,$$

$$(a_1 a_2 - a_3) a_3 - a_1^2 a_4 \ => > 0.$$

All Routh–Hurwitz inequalities are satisfied; therefore the endemic equilibrium is *locally asymptotically stable* for this parameter set (consistent with the negative eigenvalue real parts computed above).

7.7 Interpretation

- The chosen parameters produce $R_0 \approx 1.027 > 1$, so an endemic equilibrium exists. Because R_0 is only slightly above 1 the endemic prevalence is very small ($\bar{I} \approx 4.9 \times 10^{-6}$) in the normalized population.
- The Jacobian evaluated at the EE yields a quartic characteristic polynomial with coefficients all positive and satisfying the Routh–Hurwitz inequalities, and the eigenvalues all have negative real parts — hence the EE is locally asymptotically stable.
- Practically, this example shows how even a small overshoot above $R_0 = 1$ produces a small but persistent infection level: control efforts (raising vaccination ν, increasing environmental removal τ, reducing shedding ξ or transmission coefficients β_p, β_B) can move R_0 below unity and eliminate the disease (DFE stable).

8 Conclusion and Recommendation

8.1 Conclusion

In this study, we examined the local stability properties of the disease-free equilibrium (DFE) and the endemic equilibrium (EE) of a cholera SIRB (Susceptible–Infected–Recovered–Bacteria) model with vaccination and treatment. By employing rigorous mathematical analysis, we derived the equilibrium points and determined their stability conditions through the Jacobian matrix approach and the Routh–Hurwitz criteria. Furthermore, a worked numerical example with biologically realistic parameters confirmed the analytical results, providing concrete evidence of the model's dynamical behavior. The analysis revealed that the DFE is locally asymptotically stable whenever the basic reproduction number $R_0 < 1$. This implies that under such conditions, small perturbations from the disease-

free state will decay over time, leading to the eventual eradication of cholera. Biologically, this highlights the critical role of public health interventions—particularly vaccination, effective treatment, and environmental sanitation—in maintaining the system below the epidemic threshold. Conversely, when $R_0 > 1$, the DFE loses stability and the system gravitates towards the EE, indicating the persistence of cholera in the population. The endemic equilibrium point was also shown to be locally asymptotically stable under the Routh–Hurwitz conditions, provided the epidemiological parameters ensure that $R_0 > 1$. The stability of the EE implies that cholera will persist at a steady but non-negligible level in the population, with disease prevalence and bacterial concentration depending sensitively on the transmission rates and recovery parameters. The numerical example corroborated these theoretical findings, showing convergence towards the EE for realistic parameter values when $R_0 > 1$. From the derivations and the numerical evidence, it can be deduced that cholera control strategies must be directed at reducing R_0 below unity. This can be achieved through widespread vaccination, prompt treatment of infected individuals, improved access to clean water, and effective environmental sanitation measures to reduce bacterial contamination. Additionally, the analysis underscores that partial interventions that fail to bring R_0 below one may only stabilize cholera at endemic levels, without achieving eradication.

8.2 Recommendations

1. **Public Health Strategies:** Vaccination should be prioritized in endemic regions to reduce the susceptible population, coupled with timely treatment interventions to accelerate recovery and lower transmission.

2. **Environmental Measures:** Improving water quality, sanitation, and hygiene (WASH) infrastructure is essential to reduce bacterial concentration in the environment, thereby suppressing indirect transmission pathways.

3. **Mathematical Insights for Policy:** The threshold condition $R_0 < 1$ should guide health policymakers in designing effective cholera control programs. Resource

allocation should be aligned with strategies that guarantee reduction of R_0 below unity.

In conclusion, the local stability analysis of the SIRB model demonstrates that controlling cholera is mathematically achievable, but requires coordinated, multi-faceted interventions. The interplay of vaccination, and environmental management is key to achieving sustainable eradication, and the mathematical framework provided herein offers a valuable tool for designing and evaluating public health strategies.

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