

# Mathematical Modelling of the Dynamics and Control of Communicable Diseases with Emphasis on Cholera Epidemics

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DOI: <https://doi.org/10.38177/ajast.2023.7412>

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Article Received: 17 October 2023

Article Accepted: 19 December 2023

Article Published: 28 December 2023

## ABSTRACT

Cholera remains a disease of public health importance, associated with high morbidity and mortality. This research provides a mathematical approach on the effective control and possible eradication of *Vibrio cholera* causing menace. This was done using a deterministic SPIRB mathematical model, where we defined S-susceptible, P-protected, I-infected, R-recovered and B-bacteria. We carried out mathematical analyses on the model, such as the invariant region analysis, non-negativity of solutions, equilibrium points, basic reproduction number, stability analysis, sensitivity analysis and bifurcation analysis. All solutions of the SPIRB model are positive and there exists an invariant region for the model. Although, the disease-endemic equilibrium of the model is globally asymptotically stable, the disease-free equilibrium of the model is not globally stable but locally asymptotically stable. It was also revealed that the recruitment rate into the susceptible class, the probability that each contact is effective enough to cause infection, the contact rate with contaminated environment, the average contribution of each infected individual to the pathogen population of *Vibrio cholera*, and the progression rate of protected individuals to the susceptible class are the most sensitive parameters. We showed via the bifurcation analysis that if the progression rate of protected individuals to the susceptible class can be kept very close to zero, say between zero and unity, then the disease-free equilibrium can be kept stable. Numerical simulations were conducted to show the effects of some parameters on the classes.

**Keywords:** Modeling; Epidemics; Diseases; Cholera; Control; Communicable Simulation.

## 1. Introduction

Cholera is an example of a bacterial disease whose primary mode of infection is indirect; which is caused when individuals ingest fecal-contaminated water containing the bacteria *V. cholera*. Transmission between humans and reservoirs of pathogens implies that disease transmission includes an indirect route other than human-to-human contact. In essence, Cholera is an infection of the small intestine caused by the gram-negative bacterium, *Vibrio cholera*. Untreated individuals suffer severely from diarrhea and vomiting. The disease can cause a rapid dehydration and electrolyte imbalance, and can lead to death. Outcomes behind the disruption of water and sanitation systems or the displacement of populations to inadequate and overcrowded camps can increase the risk of cholera transmission. Cholera remains a global threat to public health and a key indicator of lack of social development. Recently, the reemergence of cholera has been noted in parallel with the ever-increasing size of vulnerable populations living in unsanitary conditions. Education, which is a key tool in disease control, is often overlooked. It requires investment in people rather than in biomedical interventions, but it has the potential to lead to enormous benefits for relatively low cost. Conversely, a lack of information can have a severe impact on worsening the spread of the disease.

Understanding the fundamental mechanism in the disease transmission is crucial for effective prevention and intervention strategies against a cholera outbreak. To this effect, mathematical modeling provides a unique approach to gain basic insights into the dynamics of infectious diseases. Mathematical modeling can predict the dynamics of explosive epidemics often associated with cholera outbreaks. In an effort to gain deeper understanding

of the complex dynamics of cholera, several mathematical models have been published. In the work of [1] a model to study the 2008–2009 cholera outbreaks in Zimbabwe was proposed. The model explicitly considered both human-to-human and environment-to-human transmission pathways. The results in this work demonstrated the importance of the human-to-human transmission in cholera epidemics, especially in such places as Zimbabwe, a landlocked country in the middle of Africa. Two formulated models one without the control term and the other with control terms that are time-dependent was examined by [2]. Since the bacteria population grows at a density-dependent rate and the probability of catching cholera depends on the concentration of *v. cholera* in aquatic environment, then it behooves us to know the carrying capacity of the organism. The logistic growth term does that and is non-linear with respect to the bacteria population and makes the model more realistic since the population growth of bacteria is non-linear. More recently, [3], in their model, studied the effects of medical resources in cholera transmission, they fitted their model to the Yemen cholera outbreak during 2017–2018.

This article mathematically presents the dynamics of cholera disease and provides scientific information that will guide in the control and possible eradication of Cholera.

A continuous drastic loss of body fluids leads to dehydration, thus neglecting treatment as soon as the case occurs, and accelerates the death of the infected person within hours [4]. There are environmental factors that play a great role in the propagation of cholera infection.

Nevertheless, relevance has to be given to the environmental matrix in which the disease spreads into disease-free regions [5] together with consideration on individual mobility and travelers carrying the disease in long-distance journeys. Susceptible people traveling on a daily basis may contact the disease in destination sites and take the disease back to the possibly uninfected communities where they regularly live. At the same time, infected individuals not showing severe symptoms can carry the illness releasing bacteria via their feces [6]. Finally, symptomatic infected individuals locally increase the bacteria concentration, which is, then, spread along the hydrological network.

Experiments for spreading of infectious disease in individual are unethical. Epidemiologists and other researchers use mathematical modeling and numerical simulation for scientific understanding about the dynamics and preventive method of an infectious disease, for determining sensitivities, changes of parameter values, and to forecasting in the report of [7-16]. The models are based on cohorts, namely, susceptible, infected, and recovered, for individual population incorporated with some preventive measures such as treatment, vaccination, chlorination, hygienic, and sanitation through education. Treatment is more recommended when cases occur, that is, a short-time plan of controlling and eradicating the disease. Education is the most recommended option in the disease controlling and eradicating from the community whenever. Education is a long-term plan of controlling and eradicating the disease, especially in creating awareness of water treatment, sewage removal, food safety, personal hygiene, and environment sanitation as reported by [17-19], Nguyen et al. (2014) and Kumar, Kumar and Nilam (2020). It is an undeniable truth that there are individual populations who have awareness on the adequate preventive measures of cholera diseases and compliance them. In our best knowledge, those populations are not considered as one cohort of individuals in addition to susceptible, infected, and recovered individual cohorts in order to analyze the dynamics, control, and eliminate cholera. Other similar report could be found in [20-23].

Despite the effort of the previous authors as reported above, there is the need to analyze qualitatively the controlling mechanisms of cholera epidemics in a community by executing adequate control and preventive measures, hence presents the dynamics of cholera disease and provide scientific information that will guide the control mechanisms.

## 2. Mathematical Formulation

We divided the total human population at time -  $t$ , denoted by  $N(t)$ , according to the infection status into:  $S(t)$  - susceptible,  $P(t)$  - protected individuals,  $I(t)$  - infected,  $R(t)$  - recovered. Moreover,  $B(t)$  is the concentration of *Vibrio cholerae* in an aquatic environment at time  $t$ . The birth rate or immigration rate of individuals into the susceptible class is  $\alpha$ . Recovered individuals also increases the susceptible class at a rate of  $(1 - q)\phi$ , where  $q$  is the rate of compliance to hygienic regulations such as non-ingestion of cholera bacterium, and no contact with cholera patients, and  $\phi$  is the loss rate of immunity of recovered individuals. Individuals from the protected class also increase the susceptible class at the rate  $(1 - q)\chi$ , where  $\chi$  is progression rate of protected individuals to the susceptible cohort. The susceptible individuals either die from suffering at a rate of  $\delta$  (where  $\delta$  is the sum of the natural death rate and population-dependent death rate) or move to an infectious cohort by acquiring cholera through contact with the aquatic reservoir at a rate of  $\beta\epsilon(1 - q)\frac{B}{B + k}$  (where  $\beta$  is the probability that each contact is effective enough to cause infection,  $\epsilon$  is the contact rate with contaminated environment and  $k$  is the concentration of vibrio cholera in water) or move to the Protected cohort ( $P$ ) at a rate of  $bq$  (where  $b$  is progression rate of susceptible individuals to the protected cohort due to the preventive method).

The Protected class is also increased from the recovered class at a rate of  $q\phi$ . The infected individuals recover from the disease at a rate of  $\tau$ . The death rate of  $\delta$  also occurs in the Protected class, the Infected class, and the Recovered class. Disease-induced death occurs in the infected class at a rate of  $\eta$ . The concentration ( $B$ ) of *Vibrio cholerae* in the aquatic environment is increased by natural recruitment at a rate of  $\varphi$ , and also by infected humans at a rate of  $\psi$ , where  $\psi$  the average contribution of each infected individual to the pathogen population. The concentration is reduced by loss of bacteria into the infected class at a rate of  $(1 - \theta)\sigma$ . The model is given as system 2.1 and the schematic diagram is shown in figure 2.1.

$$\left. \begin{aligned} \frac{d}{dt}S(t) &= \alpha + (1 - q)\chi P + (1 - q)\phi R - \left( \beta\epsilon(1 - q)\frac{B}{B + k} + bq + \delta \right) S \\ \frac{d}{dt}P(t) &= bqS + q\phi R - (\delta + (1 - q)\chi)P \\ \frac{d}{dt}I(t) &= \beta\epsilon(1 - q)\frac{B}{B + k}S - (\tau + (1 - \theta)\psi + \delta + \eta)I \\ \frac{d}{dt}R(t) &= \tau I - ((1 - q)\phi + q\phi + \delta)R \\ \frac{d}{dt}B(t) &= \varphi + (1 - \theta)\psi I - \lambda B \end{aligned} \right\} \quad (2.1)$$

Subject to the following conditions:

$$S(0) \geq 0, P(0) \geq 0, I(0) \geq 0, B(0) \geq 0.$$

$$N(t) = S(t) + P(t) + I(t) + R(t), \quad N(0) = S(0).$$

### 2.1. Model Assumptions

The model is based on the following assumptions.

- [1] Every individual in the susceptible class has equal chance of being infected.
- [2] The disease does not confer permanent immunity on recovered individuals. Recovered persons, if not protected can be re-infected again.
- [3] Infected individuals can recover through treatment, or die due to the disease or natural death.
- [4] The time scale is considered in such a way that natural births and deaths are not neglected.
- [5] Protected individuals, who now turn down compliance to hygienic regulations, become susceptible, before a possible infection.

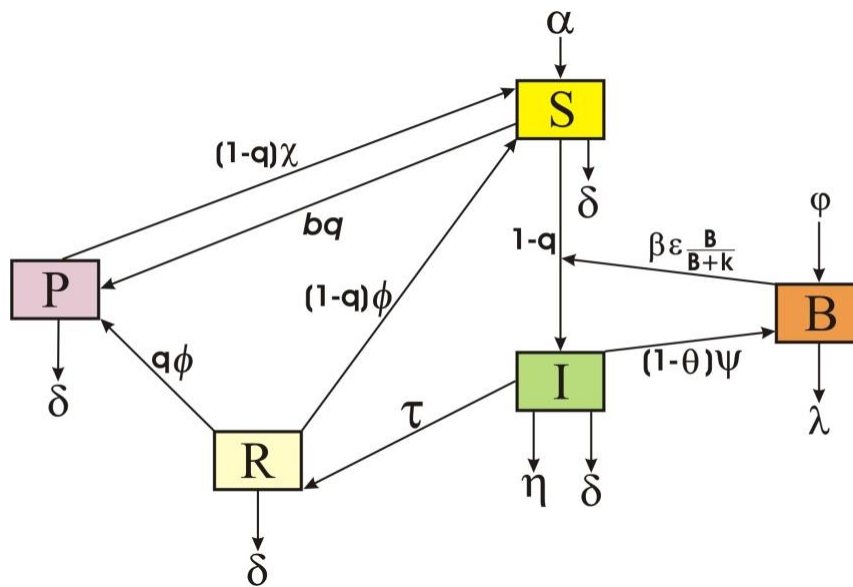


Figure 2.1. Schematic diagram

### 3. Basic Mathematical Analyses of the Model

In this section, we establish the non-negativity of solutions, the feasible region, determining the equilibrium points, the basic reproduction, and analyzing the stability of the disease-free equilibrium point.

#### 3.1. Positivity or non-negativity of Solutions

*Theorem 3.1 (Positivity of Solution):* Suppose  $\Gamma = \{(S, P, I, R, B) \in \mathbb{R}^5: S(0) > 0, P(0) > 0, I(0) > 0, R(0) > 0, B(0) > 0\}$ , then the solution set  $\{S, P, I, R, B\}$  is positive for all  $t \geq 0$ .

*Proof:* Observe that from the first equation,

$$\frac{dS(t)}{dt} = \alpha + (1 - q)\chi P + (1 - q)\phi R - \left( \beta\epsilon(1 - q)\frac{B}{B + k} + bq + \delta \right) S,$$

$$\frac{dS(t)}{dt} \geq - \left( \beta\epsilon(1 - q)\frac{B}{B + k} + bq + \delta \right) S.$$

Suppose  $S(0) = S_0 > 0$ , we obtain the solution

$$S(t) \geq S_0 e^{-\left(\beta \epsilon (1-q) \frac{B}{B+k} + bq + \delta\right)t} > 0 \quad \forall t \geq 0. \quad (3.1)$$

Similarly,

$$P(t) > 0, I(t) > 0, R(t) > 0, B(t) > 0 \quad \forall t \geq 0. \quad (3.2)$$

This completes the proof. It follows that all solutions of the model are non-negative.

### 3.2. Feasible Region for System Solutions

*Theorem 3.2 (Feasible Region):* The sets

$$\Gamma_1 = \{(S, P, I, R) \in \mathbb{R}_+^4 : 0 \leq S + P + I + R = N \leq \alpha/\delta\} \quad (3.3)$$

$$\Gamma_2 = \{B \in \mathbb{R}_+ : 0 \leq R \leq \varphi/\lambda\} \quad (3.4)$$

are feasible solution sets for the model 2.3.

*Proof:* We recall that the total human population size at time  $t$  is given by

$$N(t) = S(t) + P(t) + I(t) + R(t).$$

$$\frac{dN(t)}{dt} = \alpha - \delta N - \eta I \leq \alpha - \beta N.$$

Solving for  $N(t)$  gives  $N(t) \leq \frac{\alpha}{\delta} + ce^{-\delta t}$ . As  $t \rightarrow \infty$ , we obtain

$$N(t) \leq \frac{\alpha}{\delta}. \quad (3.5)$$

Now, considering the bacterial class

$$\frac{d}{dt} B(t) = \varphi + (1 - \theta)\psi I - \lambda B \leq \varphi - \lambda B.$$

Solving for  $B(t)$  gives  $B(t) \leq \frac{\varphi}{\lambda} + ce^{-\lambda t}$ . As  $t \rightarrow \infty$ , we obtain

$$B(t) \leq \frac{\varphi}{\lambda}. \quad (3.6)$$

Therefore, the threshold human population level is  $\frac{\alpha}{\delta}$ , and threshold bacterial population level is  $\frac{\varphi}{\lambda}$ . It follows that the feasible solution sets of the model remain in the regions:  $\Gamma_1 = \{(S, P, I, R) \in \mathbb{R}_+^4 : 0 \leq S + P + I + R = N \leq \alpha/\delta\}$  and  $\Gamma_2 = \{B \in \mathbb{R}_+ : 0 \leq R \leq \varphi/\lambda\}$  respectively. Observe that if the population is higher than the threshold level, the population reduces to the carrying capacity. If the population is less than the threshold level, then the solutions of the model remain in the invariant region for all  $t > 0$ . Therefore, the regions  $\Gamma_1$  and  $\Gamma_2$  are positively invariant. This completes the proof.

### 3.3. Disease Equilibrium Points

*The Disease-free Equilibrium Point (DFEP)*

We obtain the disease-free equilibrium  $E_0$  by equating the right-hand side of the model to zero and solving the resulting algebraic system of equations. First, we set  $I = R = B = 0$ , and solve to obtain

$$\mathbb{E}_0 = (S, P, I, R, B)_0 = \left( \frac{((1-q)\chi + \delta)\alpha}{\delta(\delta + (1-q)\chi + bq)}, \frac{\alpha bq}{\delta((b-\chi)q + \chi + \delta)}, 0, 0, 0 \right) \quad (3.7)$$

*The Endemic Equilibrium Point (FEP)*

The model admits an endemic equilibrium  $\mathbb{E}_e = (S, P, I, R, B)_e$  when  $I_e > 0, B_e > 0$ .  $\mathbb{E}_e$  is also obtained by equating the right-hand side of the model (2.1) to zero and solving the resulting system. The endemic equilibrium is obtained as:

$$S = -((-\delta + \chi(q-1))(\delta + \phi)(-\delta + (\theta-1)\psi - \eta - \tau)\alpha) / (-\delta^4 + (\chi(q-1) - \phi - bq - \Lambda + (\theta-1)\psi - \eta - \tau)\delta^3 + (-(-\phi - \Lambda + (\theta-1)\psi - \eta - \tau)(q-1)\chi + (-bq - \Lambda + (\theta-1)\psi - \eta - \tau)\phi + ((\theta-1)\psi - \eta - \tau)(bq + \Lambda))\delta^2 + (-((-\Lambda + (\theta-1)\psi - \eta - \tau)\phi + \Lambda((\theta-1)\psi - \eta - \tau))(q-1)\chi + ((-\tau\Lambda + ((\theta-1)\psi - \eta - \tau)b)q + \Lambda((\theta-1)\psi - \eta))\phi)\delta - \Lambda(-q\tau + (\theta-1)\psi + b\tau - \eta)\chi(q-1)\phi).$$

$$P = (\alpha(-\delta^2q + q(-\phi + (\theta-1)\psi - \eta - \tau)\delta + (((\theta-1)\psi - \eta - \tau)q - \Lambda\tau)\phi)b) / (-\delta^4 + (-\phi + (-b + \chi)q - \chi - \Lambda + (\theta-1)\psi - \eta - \tau)\delta^3 + (((-b + \chi)q - \chi - \Lambda + (\theta-1)\psi - \eta - \tau)\phi + ((\Lambda + (1-\theta)\psi + \eta + \tau)\chi + ((\theta-1)\psi - \eta - \tau)b)q + (-\Lambda + (\theta-1)\psi - \eta - \tau)\chi + ((\theta-1)\psi - \eta - \tau)\Lambda)\delta^2 + (((\Lambda + (1-\theta)\psi + \eta + \tau)\chi - \Lambda\tau + ((\theta-1)\psi - \eta - \tau)b)q + (-\Lambda + (\theta-1)\psi - \eta - \tau)\chi + ((\theta-1)\psi - \eta)\Lambda)\phi - ((\theta-1)\psi - \eta - \tau)\chi(q-1)\Lambda)\delta - \phi\chi(q-1)(-q\tau + (\theta-1)\psi + b\tau - \eta)\Lambda).$$

$$I = ((-\delta + \chi(q-1))\Lambda\alpha(\delta + \phi)) / (-\delta^4 + (\chi(q-1) - \phi - bq - \Lambda + (\theta-1)\psi - \eta - \tau)\delta^3 + (-(-\phi - \Lambda + (\theta-1)\psi - \eta - \tau)(q-1)\chi + (-bq - \Lambda + (\theta-1)\psi - \eta - \tau)\phi + (bq + \Lambda)((\theta-1)\psi - \eta - \tau))\delta^2 + (- (q-1)((-\Lambda + (\theta-1)\psi - \eta - \tau)\phi + \Lambda((\theta-1)\psi - \eta - \tau))\chi + \phi((-\tau\Lambda + b((\theta-1)\psi - \eta - \tau))q + ((\theta-1)\psi - \eta)\Lambda))\delta - \chi\Lambda(q-1)\phi(-q\tau + (\theta-1)\psi + b\tau - \eta)).$$

$$R = ((-\delta + \chi(q-1))\tau\Lambda\alpha) / (-\delta^4 + (\chi(q-1) - \phi - bq - \Lambda + (\theta-1)\psi - \eta - \tau)\delta^3 + (-(-\phi - \Lambda + (\theta-1)\psi - \eta - \tau)(q-1)\chi + (-bq - \Lambda + (\theta-1)\psi - \eta - \tau)\phi + (bq + \Lambda)((\theta-1)\psi - \eta - \tau))\delta^2 + (- (q-1)((-\Lambda + (\theta-1)\psi - \eta - \tau)\phi + \Lambda((\theta-1)\psi - \eta - \tau))\chi + \phi((-\tau\Lambda + b((\theta-1)\psi - \eta - \tau))q + ((\theta-1)\psi - \eta)\Lambda))\delta - \chi\Lambda(q-1)\phi(-q\tau + (\theta-1)\psi + b\tau - \eta)).$$

$$\Lambda = \beta\epsilon(1-q) \frac{B}{B+k}$$

**3.4. The Basic Reproduction Number ( $R_0$ )**

The basic reproduction number is the average number of secondary infections caused by a single infectious individual in an entirely susceptible population during his/her infective period. We obtain this using the next generation matrix approach. Suppose  $X$  is the set of the infected classes, then we can write

$$X(t) = (I(t), B(t)), \quad X'(t) = \mathcal{F}(t) - \mathcal{V}(t),$$

where

$$\mathcal{F} = \left[ \begin{array}{c} \beta\epsilon(1-q) \frac{B}{B+k} S \\ \phi \end{array} \right] \text{ and } \mathcal{V} = \left[ \begin{array}{c} (\tau + (1-\theta)\psi + \delta + \eta)I \\ -(1-\theta)\psi I + \lambda B \end{array} \right].$$

The Jacobian matrices  $F$  and  $V$  of the matrices  $\mathcal{F}$  and  $\mathcal{V}$  respectively, evaluated at the disease-free equilibrium are:

$$F = \begin{pmatrix} 0 & \frac{\beta \epsilon k (-1+q)((-1+q)\chi - \delta)\alpha}{(\delta + (1-q)\chi + bq)\delta k} \\ 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} \tau + (1-\theta)\psi + \delta + \eta & 0 \\ -(1-\theta)\psi & \lambda \end{pmatrix}.$$

$$V^{-1} = \begin{pmatrix} \frac{1}{-\psi\theta + \delta + \eta + \psi + \tau} & 0 \\ -\frac{(1-\theta)\psi}{(-\psi\theta + \delta + \eta + \psi + \tau)\lambda} & \frac{1}{\lambda} \end{pmatrix}.$$

$$FV^{-1} = \begin{bmatrix} \frac{\alpha(-1+q)(-1+\theta)(\delta - (-1+q)\chi)\beta\epsilon\psi}{k\lambda(\tau + (1-\theta)\psi + \delta + \eta)(\delta + (1-q)\chi + bq)\delta} & \frac{\beta \in (-1+q)((-1+q)\chi - \delta)\alpha}{(\delta + (1-q)\chi + bq)\delta k\lambda} \\ 0 & 0 \end{bmatrix}$$

The eigenvalues of  $FV^{-1}$  are:

$$0 \text{ and } \frac{\alpha(-1+q)(-1+\theta)((1-q)\chi + \delta)\beta \in \psi}{k\lambda(-\psi\theta + \delta + \eta + \psi + \tau)(\delta + (1-q)\chi + bq)\delta}.$$

Therefore, it follows that the spectral radius and hence  $R_0$  is given by:

$$R_0 = \frac{\alpha + (1-q)\chi \frac{\alpha}{\delta}}{(\delta + (1-q)\chi + bq)} \frac{\frac{(1-q)\beta\epsilon}{k}}{(\psi(1-\theta) + \delta + \eta + \tau)} \frac{(1-\theta)\psi}{\lambda}. \quad (3.8)$$

The basic reproduction number shows that an individual enters into an entirely susceptible population at a rate of  $\alpha$ , or the individual may come from the protected class at a rate of  $(1-q)\chi$ . This rate is moderated by the threshold population level  $\frac{\alpha}{\delta}$ . Such individual becomes infected at a rate of  $\frac{(1-q)\beta\epsilon}{k}$ . The total time spent by this individual both in the susceptible class, protected class and the infectious class is  $\frac{1}{(\delta + (1-q)\chi + bq)(\tau + (1-\theta)\psi + \delta + \eta)}$ . Vibrio cholerae is introduced into the bacterial class by this infected individual at a rate of  $(1-\theta)\psi$ . The total time spent by this Vibrio cholerae in the bacterial cohort is  $\frac{1}{\lambda}$ .

### 3.5. Stability Analysis of the Disease-free Equilibrium

In this section we consider the stability of the equilibrium points. The local stability guarantees that as long as the initial values of the class sizes are within the basin of attraction of the disease-free equilibrium point (DFE), the DFE prevails.

#### Local Stability of the Disease-free Equilibrium

The Jacobian matrix, which is evaluated at the disease-free equilibrium, is given by

$$J(E_0) = \begin{bmatrix} -bq - \delta & (1-q)\chi & 0 & (1-q)\phi & -\frac{((q-1)\chi - \delta)\beta\alpha(q-1)\epsilon}{\delta k(\delta + (1-q)\chi + bq)} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(1-\theta)\psi - \eta - \tau - \delta & 0 & \frac{((1-q)\chi + \delta)\beta\alpha(1-q)\epsilon}{\delta k(\delta + (1-q)\chi + bq)} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-\theta)\psi & 0 & -\lambda \end{bmatrix}. \quad (3.9)$$

**Theorem 3.3:** The disease-free equilibrium ( $\mathbb{E}_0$ ) is locally asymptotically stable if the effective reproduction number  $R_0 < 1$ , otherwise it is unstable.

*Proof:* The disease-free equilibrium is locally asymptotically stable if and only if all roots of the characteristic polynomial of the Jacobian matrix have negative real parts. The characteristic polynomial of the Jacobian matrix  $J(\mathbb{E}_0)$  is given by

$$\text{Det}(J(\mathbb{E}_0) - \Omega I) = 0. \quad (3.10)$$

$$a_1\Omega^5 + a_2\Omega^4 + a_3\Omega^3 + a_4\Omega^2 + a_5\Omega = 0. \quad (3.11)$$

where

$$a_1 = -1, a_2 = -\delta(bq + 2\delta + (1 - \theta)\psi + \lambda + \eta + \tau),$$

$$a_3 = \frac{1}{(\delta + (b - \chi)q + \chi)k\delta} \left( -\delta^4 k - 2k \left( (b - \frac{1}{2}\chi)q + \frac{1}{2}\chi - \frac{1}{2}(1 - \theta)\psi + \lambda + \frac{1}{2}\eta + \frac{1}{2}\tau \right) \delta^3 - k(b(b - \chi)q^2 + ((b - 2\lambda - \eta - \tau - (1 - \theta)\psi)\chi + 2((1 - \theta)\psi + \frac{3}{2}\lambda + \eta + \tau)b)q + ((1 - \theta)\psi + 2\lambda + \eta + \tau)\chi + \lambda((1 - \theta)\psi + \eta + \tau))\delta^2 + (\epsilon\psi\beta\alpha(1 - q)(1 - \theta) - ((b - \chi)q + \chi)(b((1 - \theta)\psi + \lambda + \eta + \tau)q + \lambda((1 - \theta)\psi + \eta + \tau))k)\delta + \alpha\beta\chi\psi\epsilon(1 - q)^2(1 - \theta) \right),$$

$$a_4 = -\frac{1}{(\delta + (b - \chi)q + \chi)k\delta} \left( (\delta^3 k\lambda + ((b - \chi)q + \chi + (1 - \theta)\psi + \eta + \tau)k\lambda\delta^2 - (((b - \chi)q + \chi)\lambda((1 - \theta)\psi + \eta + \tau)k + \epsilon\psi\beta\alpha(1 - q)(1 - \theta))\delta - \alpha\beta\psi\epsilon(1 - q)^2(1 - \theta))(bq + \delta) \right), \quad a_5 = 0.$$

Solving the characteristic polynomial, we obtain the following result:

$$\Omega_1 = -(bq + \delta), \quad \Omega_2 = 0, \quad \Omega_3 = 0,$$

$$\Omega_4 = -\frac{1}{2} \frac{1}{(\delta + (1 - q)\chi + bq)k\delta} \left( +\delta^3 k + k((1 - \theta)\psi + (1 - q)\chi + bq + \lambda + \eta + \tau)\delta^2 + ((1 - q)\chi + bq)k((1 - \theta)\psi + (\eta + \tau + \lambda))\delta - \left( (\delta + (b - \chi)q + \chi)k\delta \left( \delta^4 k + k((b - \chi)q + \chi + 2(1 - \theta)\psi + 2(\eta + \tau - \lambda))\delta^3 + 2k((1 - \chi)((1 - \theta)\psi + (\eta + \tau - \lambda))^2 k + 4\epsilon\psi\beta\alpha(1 - q)(1 - \theta))\delta + 4\alpha\beta\chi\psi\epsilon(1 - q)^2(1 - \theta) \right) \right)^{1/2} \right),$$

$$\Omega_5 = -\frac{1}{2(\delta + (1 - q)\chi + bq)k\delta} \left( \delta^3 k + k((1 - \theta)\psi + (1 - q)\chi + bq + \lambda + \eta + \tau)\delta^2 + ((1 - q)\chi + bq)k((1 - \theta)\psi + (\eta + \tau + \lambda))\delta + \left( (\delta + (b - \chi)q + \chi)k\delta \left( \delta^4 k + k((b - \chi)q + \chi + 2(1 - \theta)\psi + 2(\eta + \tau - \lambda))\delta^3 + 2k((1 - \chi)((1 - \theta)\psi + (\eta + \tau - \lambda))^2 k + 4\epsilon\psi\beta\alpha(1 - q)(1 - \theta))\delta + 4\alpha\beta\chi\psi\epsilon(1 - q)^2(1 - \theta) \right) \right)^{1/2} \right).$$

We observe that in order to obtain a positive value for the square root function, the following two conditions must hold:

$$b > \chi, \quad \eta + \tau > \lambda.$$

The first condition simply states that the progression rate of susceptible individuals to the protected cohort must be greater than the progression rate of protected individuals back into the susceptible class. The second condition simply states the rate at which infected humans leave the infected compartment must be greater than the rate at which bacterial exit the bacteria cohort. Furthermore, for the root  $\Omega_4$  to have negative real part, the following must hold:

$$\begin{aligned} & + \delta^3 k + k((1 - \theta)\psi + (1 - q)\chi + bq + \lambda + \eta + \tau)\delta^2 + ((1 - q)\chi + bq)k((1 - \theta)\psi + (\eta + \tau + \lambda))\delta \\ & - \left( (\delta + (b - \chi)q + \chi)k\delta \left( \delta^4 k + k((b - \chi)q + \chi + 2(1 - \theta)\psi + 2(\eta + \tau - \lambda)) \right. \right. \\ & \left. \left. - \chi)q + \chi) \left( (1 - \theta)\psi + (\eta + \tau - \lambda) \right)^2 k + 4\epsilon\psi\beta\alpha(1 - q)(1 - \theta) \right) \delta \right. \\ & \left. + 4\alpha\beta\chi\psi\epsilon(1 - q)^2(1 - \theta) \right)^{1/2} > 0. \end{aligned}$$

From the expression for  $R_0$  given as equation (3.11), we obtain

$$\chi = \frac{(kR_0((-1 + \theta)\psi + \delta + \eta + \tau)(bq + \delta)\lambda - \epsilon\psi\beta\alpha(-1 + q)(-1 + \theta))\delta}{(-1 + q)(kR_0((-1 + \theta)\psi + \delta + \eta + \tau)\delta\lambda - \epsilon\psi\beta\alpha(-1 + q)(-1 + \theta))}. \quad (3.12)$$

Substituting equation (3.12) into the preceding inequality above, we obtain  $R_0 < 1$ . This completes the proof.

#### Global Stability of the Disease-Endemic Equilibrium

In order to investigate the global asymptotic stability of the disease-free equilibrium, an appropriate Lyapunov function can be constructed, but we shall employ the method introduced by Carlos and Song (2009). Here, we rewrite the model (2.1) in the following form

$$\begin{cases} \frac{dX}{dt} = L(X, Z) \\ \frac{dZ}{dt} = M(X, Z), \quad M(X, 0) = 0 \end{cases} \quad (3.13)$$

where  $X = (S, P, R)$  denotes the uninfected individuals and  $Z = (I, B)$  denotes the infected individuals. Therefore, an equilibrium point would be denoted by  $E = (X, Z)$ . It follows that the disease-free equilibrium ( $E_0$ ) is represented as  $E_0 = (X^*, 0)$  where  $X^* = \left( \frac{((1 - q)\chi + \delta)\alpha}{\delta(\delta + (1 - q)\chi + bq)}, \frac{abq}{\delta((b - \chi)q + \chi + \delta)}, 0 \right)$ . Now the disease-free equilibrium is globally asymptotically stable if the following two conditions are satisfied:

$$C1: \quad \text{For } \left. \frac{dX}{dt} \right|_{Z=0} = L(X, 0),$$

$$X^* = \left( \frac{((1 - q)\chi + \delta)\alpha}{\delta(\delta + (1 - q)\chi + bq)}, \frac{abq}{\delta((b - \chi)q + \chi + \delta)}, 0 \right)$$

is globally asymptotically stable.

$$C2: \quad \frac{dZ}{dt} = D_Z M(X^*, 0)Z - \widehat{M}(X, Z), \text{ where } \widehat{M}(X, Z) \geq 0 \text{ for all } (X, Z) \in \Gamma.$$

$\Gamma$  is the region where the model is biologically feasible, and  $D_Z M(X^*, 0)$  is known as the Metzler matrix with nonnegative off-diagonal elements.

*Theorem 3.4:* The equilibrium point  $E_0 = (X^*, 0)$  of the system (3.13) is globally asymptotically stable if  $R_0 < 1$ , and conditions (C1) and (C2) are satisfied.

*Proof:* Observe that

$$\frac{dX}{dt} = L(X, Z) = \begin{bmatrix} \alpha + (1-q)\chi P + (1-q)\phi R - \left( \beta\epsilon(1-q)\frac{B}{B+k} + bq + \delta \right) S \\ bqS + q\phi R - (\delta + (1-q)\chi)P \\ \tau I - ((1-q)\phi + q\phi + \delta)R \end{bmatrix}, \quad (3.14)$$

$$\frac{dZ}{dt} = M(X, Z) = \begin{bmatrix} \beta\epsilon(1-q)\frac{B}{B+k}S - (\tau + (1-\theta)\psi + \delta + \eta)I \\ \varphi + (1-\theta)\psi I - \lambda B \end{bmatrix}, \quad (3.15)$$

$$\left. \frac{dX}{dt} \right|_{Z=0} = L(X, 0) = \begin{bmatrix} \alpha + (1-q)\chi P + (1-q)\phi R - (bq + \delta)S \\ bqS + q\phi R - (\delta + (1-q)\chi)P \\ -((1-q)\phi + q\phi + \delta)R \end{bmatrix}. \quad (3.16)$$

We can see that  $X^* = \left( \frac{((1-q)\chi + \delta)\alpha}{\delta(\delta + (1-q)\chi + bq)}, \frac{abq}{\delta((b-\chi)q + \chi + \delta)}, 0 \right)$  is the only equilibrium point of the system (3.13). We now solve (3.13) for  $S(t)$  and  $P(t)$ .

$$\left. \frac{d}{dt} S(t) \right|_{Z=0} = \alpha + (1-q)\chi P + (1-q)\phi R - (bq + \delta)S,$$

$$\Rightarrow \quad \left. \frac{d}{dt} S(t) \right|_{Z=0} \leq (1-q)\chi P - (bq + \delta)S.$$

$$\begin{aligned} \therefore S(t) &\leq \frac{(1-q)\chi P}{bq + \delta} + e^{-(bq + \delta)t} \leq \frac{(1-q)\chi \alpha b q}{\delta((b-\chi)q + \chi + \delta)(bq + \delta)} + e^{-(bq + \delta)t} \\ &< \frac{((1-q)\chi + \delta)\alpha}{\delta(\delta + (1-q)\chi + bq)} + e^{-(bq + \delta)t}. \end{aligned}$$

$$\left. \frac{d}{dt} P(t) \right|_{Z=0} = bqS + q\phi R - (\delta + (1-q)\chi)P \leq bqS - (\delta + (1-q)\chi)P.$$

$$\begin{aligned} \therefore P(t) &\leq \frac{bqS}{\delta + (1-q)\chi} + e^{-(\delta + (1-q)\chi)t} = \frac{\alpha b q}{\delta(\delta + (1-q)\chi + bq)} + e^{-(\delta + (1-q)\chi)t} \\ &= \frac{\alpha b q}{\delta((b-\chi)q + \chi + \delta)} + e^{-(\delta + (1-q)\chi)t}. \end{aligned}$$

Therefore, as  $t \rightarrow \infty$ , we obtain that  $S(t) \rightarrow \frac{((1-q)\chi + \delta)\alpha}{\delta(\delta + (1-q)\chi + bq)}$ , and  $P(t) \rightarrow \frac{\alpha b q}{\delta((b-\chi)q + \chi + \delta)}$ . This implies global convergence of  $X = (S, P, R)$ . Hence  $X^* = \left( \frac{((1-q)\chi + \delta)\alpha}{\delta(\delta + (1-q)\chi + bq)}, \frac{\alpha b q}{\delta((b-\chi)q + \chi + \delta)}, 0 \right)$  is globally asymptotically stable for the system  $\left. \frac{dX}{dt} \right|_{Z=0}$ . We now obtain  $D_Z M(X^*, 0)Z$ .

$$D_Z M(X', 0) = \begin{bmatrix} (-1 + \theta) \psi - \eta - \tau - \delta & \frac{\alpha(-1+q)\epsilon((1+q)\chi - \delta)\beta}{k((1-q)\chi + \delta + bq)\delta} \\ (-\theta + 1) \psi & -\lambda \end{bmatrix}$$

By the condition (C2), we have

$$M(X, Z) = \left[ \frac{-((S\delta^2 + (S(1-q)\chi + S bq - \alpha)\delta - \alpha\chi(1-q))k - ((1-q)\chi + \delta)\alpha B)(1-q)\beta\epsilon B}{((1-q)\chi + \delta + bq)k(B+k)\delta} \right]_{-\varphi}$$

Observe that the condition  $\widehat{M}(X, Z) \geq 0$  for all  $(X, Z) \in \Gamma$  is not satisfied. We have established that the condition  $R_0 < 1$  holds, and so does the condition (C1) hold, but the condition (C2) fails to hold; therefore, the disease-free equilibrium of the model (2.3) is not globally asymptotically stable. This completes the proof.

### Stability Analysis of the Disease-Endemic Equilibrium

We shall establish this result using a Lyapunov function. This is shown in theorem 3.5.

*Theorem 3.5:* If  $R_0 \geq 1$ , the disease-endemic equilibrium is globally asymptotically stable.

*Proof:* Let  $\mathbb{E}_e = (S^*, P^*, I^*, R^*, B^*)$ . We define the following Lyapunov function:

$$L(S^*, P^*, I^*, R^*, B^*) = \left( S - S^* - S^* \ln \frac{S}{S^*} \right) + \left( P - P^* - P^* \ln \frac{P}{P^*} \right) + \left( I - I^* - I^* \ln \frac{I}{I^*} \right) \\ + \left( R - R^* - R^* \ln \frac{R}{R^*} \right) + \left( B - B^* - B^* \ln \frac{B}{B^*} \right).$$

Calculating the derivative of  $L$  along the solution of (2.3), we obtain

$$\frac{dL}{dt} = \left( 1 - \frac{S^*}{S} \right) \frac{dS}{dt} + \left( 1 - \frac{P^*}{P} \right) \frac{dP}{dt} + \left( 1 - \frac{I^*}{I} \right) \frac{dI}{dt} + \left( 1 - \frac{R^*}{R} \right) \frac{dR}{dt} + \left( 1 - \frac{B^*}{B} \right) \frac{dB}{dt}.$$

Therefore,

$$\frac{d}{dt} L = \left( 1 - \frac{S^*}{S} \right) \left( \alpha + (1-q)\chi P + (1-q)\phi R - \left( bq + \frac{\beta\epsilon(1-q)B}{B+k} + \delta \right) S \right) + \left( 1 - \frac{P^*}{P} \right) \left( bqS + b\phi R - (\delta + (1-q)\chi)P \right) + \left( 1 - \frac{I^*}{I} \right) \left( \frac{\beta\epsilon(1-q)BS}{B+k} - (\tau + (1-\theta)\psi + \delta + \eta)A \right) + \left( 1 - \frac{R^*}{R} \right) \left( \tau A - ((1-q)\phi + q\phi + \delta)R \right) + \left( 1 - \frac{B^*}{B} \right) \left( \varphi + (1-\theta)\psi A - \lambda B \right).$$

Observe that  $\frac{dL}{dt} = 0$  if and only if  $S = S^*$ ,  $P = P^*$ ,  $I = I^*$ ,  $R = R^*$ , and  $B = B^*$ .  $\frac{dL}{dt} < 0$  is true if  $S < S^*$ ,  $P < P^*$ ,  $I < I^*$ ,  $R < R^*$ , and  $B < B^*$ . It follows that the largest compact invariant set in  $(S, P, I, R, B \in \Gamma: \frac{dL}{dt} = 0)$  is the singleton set  $\mathbb{E}_e$  which is the endemic equilibrium of the system (2.1). Therefore, by LaSalle's invariant principle as given in Ana and James (1976), the disease-endemic equilibrium is globally asymptotically stable in  $\Gamma$ .

### 3.6. Sensitivity Analysis

In this section, we analyze the sensitivity of the parameters of the basic reproduction number ( $R_0$ ). We employ the approach used by Kizito and Tumwiine (2018) to compute the sensitivity of the parameters of  $R_0$ . The sensitivity of a parameter, say  $\mu$ , of  $R_0$  is defined as

$$\zeta_{\mu}^{R_0} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0}. \quad (3.17)$$

The analysis revealed that the positively sensitive parameters of the basic reproduction number,  $R_0$ , are the recruited rate ( $\alpha$ ) into the susceptible class, the probability ( $\beta$ ) that each contact is effective enough to cause infection, the contact rate ( $\epsilon$ ) with contaminated environment, the average contribution ( $\psi$ ) of each infected individual to the pathogen population of *Vibrio cholera*, and the progression rate ( $\chi$ ) of protected individuals to the susceptible class. Thus, reducing the number of susceptible individuals, reducing or eliminating contact with contaminated environment, effectively restricting infected humans from adding to the pathogen population, and ensuring that protected individuals remain protected can greatly lower the value of the basic reproduction number ( $R_0$ ) and thereby increasing the stability of the disease-free equilibrium. Increasing the values of the positively sensitive parameters has the effect of increasing the value of the basic reproduction number ( $R_0$ ), which is not a desired condition. Amongst the negatively sensitive parameters are the death rate ( $\lambda$ ) of *Vibrio cholerae*, the rate ( $q$ ) of compliance to hygienic regulations, the rate ( $\theta$ ) of the compliance to sanitation by the infected cohort, the natural death rate ( $\delta$ ), the cholera induced death rate ( $\eta$ ), the progression rate ( $\tau$ ) of infected individuals to the recovered class due to treatment, and the progression rate ( $b$ ) of susceptible individuals to the protected cohort. Increasing the values of these negatively sensitive parameters, reduces the value of the basic reproduction number ( $R_0$ ), which is the desired condition.

### 4. Bifurcation Analysis

The proof is established based on the Centre Manifold Theorem as presented by Castillo-Chavez and Song (2004).

*Theorem 4.1:* Consider the following general system of ODEs with a parameter  $\phi$ .

$$\frac{dy}{dx} = f(y, \phi), f: \mathbb{R}^n \times \mathbb{R} \text{ and } f \in C^2(\mathbb{R}^n \times \mathbb{R}), \quad (4.1)$$

where 0 is an equilibrium point of the system (that is,  $f(0, \phi) \equiv 0$  for all  $\phi$  and assume:

A1:  $A = D_y f(0,0) = \left( \frac{\partial f_i}{\partial y_j} (0,0) \right)$  is the linearization matrix of the system (4.1) around the equilibrium point 0 with  $\phi$  evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts;

A2: Matrix A has a right eigenvector  $w$  and a left vector  $v$  (each corresponding to the zero eigenvalue).

Let  $f_k$  be the  $k^{\text{th}}$  component of  $f$  and

$$a = \sum_{i,j,k=1}^n v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (0,0), \quad (4.2)$$

$$b = \sum_{i,k=1}^n v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0), \quad (4.3)$$

The local dynamics of the system (4.1) around 0 is totally determined by the signs of  $a$  and  $b$ :

1.  $a > 0, b > 0$ . When  $\phi < 0$  with  $|\phi| \leq 1$ , 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when  $0 < \phi \ll 1$ , 0 is unstable and there exists a negative and locally asymptotically stable equilibrium.

2.  $a < 0, b < 0$ . When  $\phi < 0$  with  $|\phi| \ll 1$ , 0 is unstable; when  $0 < \phi \ll 1$ , 0 is locally asymptotically stable, and there exists a negative unstable equilibrium.

3.  $a > 0, b < 0$ . When  $\phi < 0$  with  $|\phi| \ll 1$ , 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when  $0 < \phi \ll 1$ , 0 is stable, and a positive unstable equilibrium appears.

4.  $a < 0, b > 0$ . When  $\phi$  changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.

Particularly, if  $a > 0$  and  $b > 0$ , then a backward bifurcation occurs at  $\phi = 0$ .

*Proof:* We set

$$S = x_1, P = x_2, I = x_3, R = x_4, B = x_5. \quad (4.4)$$

Thus, the model (2.3) becomes

$$\left. \begin{aligned} \frac{d}{dt}x_1 &= \alpha + (1-q)\chi x_2 + (1-q)\phi x_4 - \left( \beta\epsilon(1-q)\frac{x_5}{x_5+k} + bq + \delta \right) x_1 \\ \frac{d}{dt}x_2 &= bq x_1 + q\phi x_4 - (\delta + (1-q)\chi)x_2 \\ \frac{d}{dt}x_3 &= \beta\epsilon(1-q)\frac{x_5}{x_5+k}x_1 - (\tau + (1-\theta)\psi + \delta + \eta)x_3 \\ \frac{d}{dt}x_4 &= \tau x_3 - ((1-q)\phi + q\phi + \delta)x_4 \\ \frac{d}{dt}x_5 &= \varphi + (1-\theta)\psi x_3 - \lambda x_5 \end{aligned} \right\} \quad (4.5)$$

Now, the Jacobian of the system (4.5) evaluated at the disease-free equilibrium ( $E_0$ ) is given by:

$$J(E_0) = \begin{bmatrix} -bq - \delta & (1-q)\chi & 0 & (1-q)\phi & -\frac{((q-1)\chi - \delta)\beta\alpha(q-1)\epsilon}{\delta k(\delta + (1-q)\chi + bq)} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(1-\theta)\psi - \eta - \tau - \delta & 0 & \frac{((1-q)\chi + \delta)\beta\alpha(1-q)\epsilon}{\delta k(\delta + (1-q)\chi + bq)} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-\theta)\psi & 0 & -\lambda \end{bmatrix}.$$

Let  $\chi = \chi^*$  be the bifurcation parameter. When  $R_0 = 1$ , we obtain

$$\chi^* = \frac{\delta(\epsilon\psi\beta\alpha(1-q)(1-\theta) - ((1-\theta)\psi + \eta + \tau + \delta)(bq + \delta)k\lambda)}{(\delta((1-\theta)\psi + \eta + \tau + \delta)k\lambda - \epsilon\psi\beta\alpha(1-q)(1-\theta))(1-q)}.$$

From the characteristic equation  $|J(\mathbb{E}_0) - \Omega I| = 0$  of  $J(\mathbb{E}_0)$ , we obtain the eigenvalues:

$$\Omega_1 = -(bq + \delta), \Omega_2 = 0, \Omega_3 = 0, \Omega_4 = 0, \Omega_5 = -((1 - \theta)\psi + \eta + \tau + \delta + \lambda).$$

We observe that zero is a simple eigenvalue of  $J(\mathbb{E}_0)$  and other eigenvalues of  $A$  have negative real parts. The right eigenvector  $(g_1, g_2, g_3, g_4, g_5)^T$  of  $J(\mathbb{E}_0)|_{\chi=\chi^*}$  is given as:

$$\begin{aligned} g_1 &= 0.3142677561 g_2 + 0.004714016342 g_4 - 5.324473667 \cdot 10^{-16} g_5 > 0, \\ g_2 &= g_2 > 0, \\ g_3 &= 4.0000000000 \cdot 10^{-8} g_5 > 0, \\ g_4 &= g_4 > 0, \\ g_5 &= g_5 > 0. \end{aligned}$$

The left eigenvector  $(h_1, h_2, h_3, h_4, h_5)$  of  $J(\mathbb{E}_0)|_{\mu=\mu^*}$  is given as:

$$h_1 = 0, h_2 = h_2 > 0, h_3 = 0.9060794305 h_5 > 0, h_4 = h_4 > 0, h_5 = h_5 > 0.$$

We now compute  $a$  and  $b$  from equations (4.2) and (4.3) where:

$$f_1 = \alpha + (1 - q)\chi x_2 + (1 - q)\phi x_4 - \left( \beta \epsilon (1 - q) \frac{x_5}{x_5 + k} + bq + \delta \right) x_1,$$

$$f_2 = bq x_1 + q\phi x_4 - (\delta + (1 - q)\chi) x_2,$$

$$f_3 = \beta \epsilon (1 - q) \frac{x_5}{x_5 + k} x_1 - (\tau + (1 - \theta)\psi + \delta + \eta) x_3,$$

$$f_4 = \tau x_3 - ((1 - q)\phi + q\phi + \delta) x_4,$$

$$f_5 = \varphi + (1 - \theta)\psi x_3 - \lambda x_5.$$

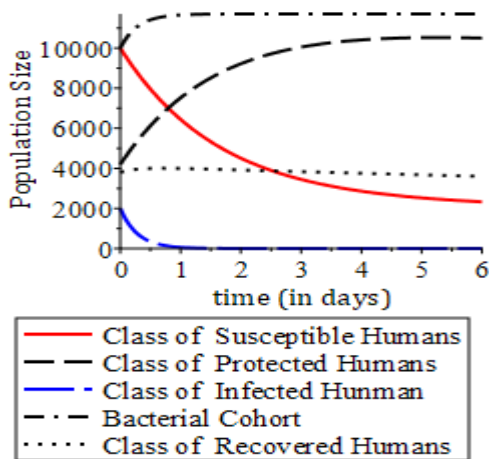
Considering only the non-zero components of  $(h_1, h_2, h_3, h_4, h_5)$ , we obtain:

$$\begin{aligned} a &= 0.9060794305 h_5 g_5 \left( 9.082338151 \cdot 10^{-17} g_2 + 1.362350723 \cdot 10^{-18} g_4 \right. \\ &\quad \left. - 3.388496581 \cdot 10^{-25} g_5 \right) > 0, \\ b &= -0.5 h_2 g_2 < 0. \end{aligned}$$

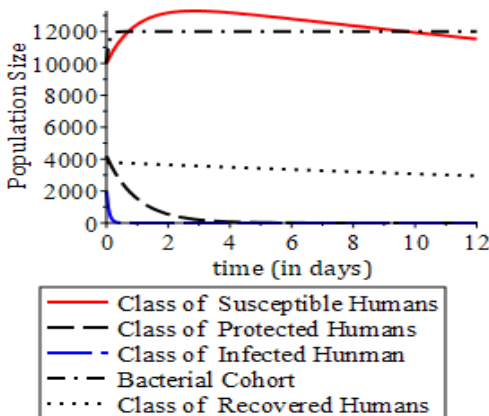
Therefore,  $a > 0, b < 0$ . When  $\chi < 0$  with  $|\chi| \ll 1$ , the disease-free equilibrium is unstable, and there exists a locally asymptotically stable negative equilibrium; when  $0 < \chi \ll 1$ , the disease-free equilibrium is stable, and a positive unstable equilibrium appears.

## 5. Numerical Simulations of the Model

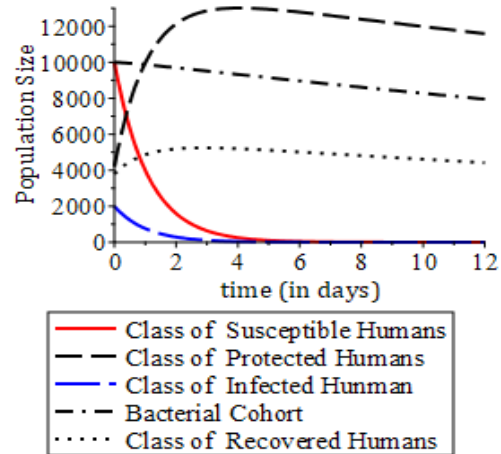
In addition to the values given in Table 1, we have used the following initial values:  $S(0) = 10000, P(0) = 4200, A(0) = 2000, R(0) = 3800, B(0) = 10000$ . The numerical simulations presented here were conducted using the *rkf45* solvers coded in Maple programming Language.



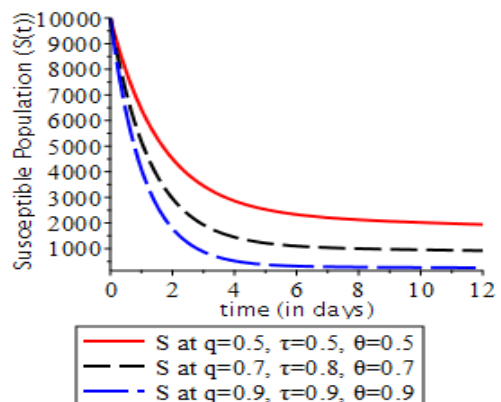
**Figure 5.1.** Compartmental population sizes obtained with all parameter values unaltered



**Figure 5.3.** Compartmental population sizes obtained by increasing the values of the positively sensitive parameters and lowering the values of the negatively sensitive parameters



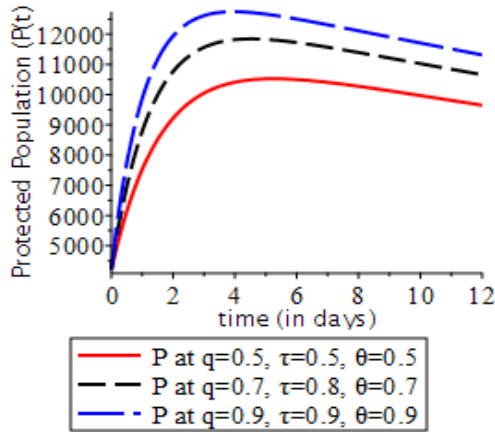
**Figure 5.2.** Compartmental population sizes obtained by lowering the values of the positively sensitive parameters and increasing the values of the negatively sensitive parameters



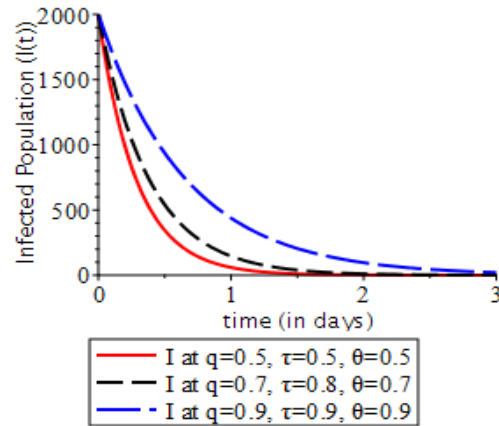
**Figure 5.4.** Effect of increased rate of compliance to hygienic regulations and rate of compliance to sanitation by the infected cohort, on the susceptible human population

### 5.1. Discussion of Results

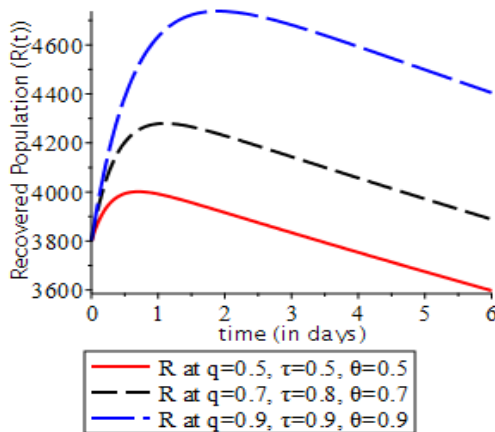
Figure 5.1 shows the simulation of our model given as system 2.1 with all parameter values unaltered. We observed that the susceptible population does not reduce to zero even within the first ten months of the disease outbreak. The Bacteria cohort increases within this time frame thereby adding to the force of infection. The Protected class increases, and there is a gradual reduction in the Recovered class. The infected population appears to be reducing but never reaching zero. This is not a desirable state. In figure 5.2, the simulation has been carried out by lowering the values of the positively sensitive parameters and at the same time increasing the values of the negatively sensitive parameters. Here, we observed that the population of individuals who are susceptible to the disease drops down towards zero within the first six months of the disease outbreak.



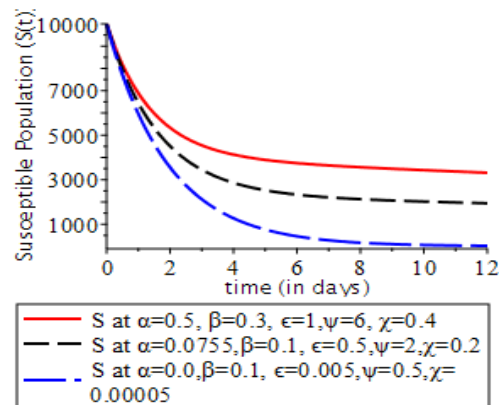
**Figure 5.5.** Effect of increased rate of compliance to hygienic regulations and rate of compliance to sanitation by the infected cohort, on the protected human population



**Figure 5.6.** Effect of increased rate of compliance to hygienic regulations and rate of the compliance to sanitation by the infected cohort, on the infected human population

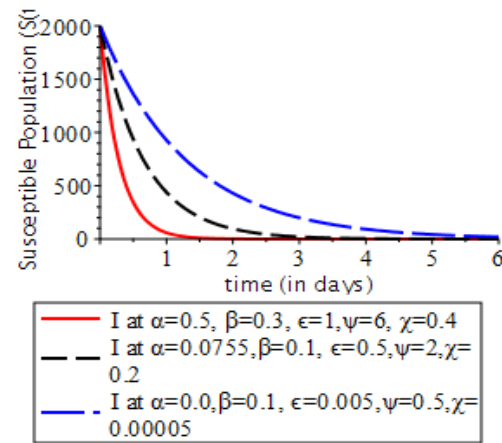
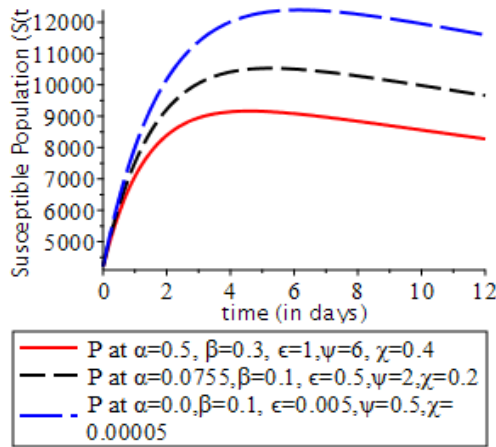


**Figure 5.7.** Effect of increased rate of compliance to hygienic regulations and rate of the compliance to sanitation by the infected cohort, on the recovered human population



**Figure 5.8.** Effect of reducing the values of  $\alpha$  (recruitment rate into the susceptible class),  $\beta$  (probability that each contact is effective enough to cause infection),  $\epsilon$  (contact rate with contaminated environment),  $\psi$  (average contribution of each infected individual to the pathogen population of *Vibrio cholera*) and  $\tau$  (progression rate of protected individuals to the susceptible class) on the susceptible human population

The Bacteria cohort reduces within this time frame thereby weakening to the force of infection. The Protected class greatly increases in an appreciable proportion, so also is an increment observed for the Recovered class. The population size of infected humans is now closer to zero, although this was not the case in the first five months. Thus, a positive result is achieved by implementing the extracts from the sensitivity analysis. Figure 5.3 is a simulation of the model done by increasing the values of the positively sensitive parameters and lowering the values of the negatively sensitive parameters.



**Figure 5.9.** Effect of reducing the values of  $\alpha, \beta, \epsilon, \psi$  and  $\tau$  on the protected human population **Figure 5.10.** Effect of reducing the values of  $\alpha, \beta, \epsilon, \psi$  and  $\tau$  on the infected human population

It is observed that the population of individuals who are susceptible to the disease increases in the first three months and then begins to drop down but never below a certain value (here 10,000) within the next ten months of the disease outbreak. The Bacteria cohort increases and then maintains a particular value throughout the remaining time duration thereby adding to the force of infection. Here, the protected class has dropped down to zero even within the first five months, and the Recovered class appeared to follow a uniform and linear decrement. The population size of infected humans appears to be close to zero. Figure 5.4 studied the effect of increased rate of compliance to hygienic regulations by the infected cohort, on the susceptible population. We observed that when the rate of compliance is high, the population of individuals who are susceptible to the disease, rapidly drops down within the first four months. In figure 5.5, where we studied the effect of increased rate of compliance to hygienic regulations by the infected cohort, on the protected class. The simulation revealed that increased rate of compliance to hygienic regulations, increases the population size of the protected class for the first three months, and thereafter, the population of the protected individuals begins to drop due to mortality and the magnitude of the force of infection.

Figure 5.6 shows the effect of increased rate of compliance to hygienic regulations by the infected class, on the infected human population. The simulation clearly revealed that a high compliance to hygienic regulations by infected individuals greatly reduces the number of infected individuals. Here, the effect of high compliance to hygienic regulations on the infected class lowered the number of infected individuals close to zero within the first five months. It is obvious that a total compliance is required. In figure 5.7, we observed the effect of increased rate of compliance to hygienic regulations on the recovered class. In the first three months, there is an increment in the population size, but thereafter, a reduction in size starts to take place. This reduction is due to mortality rates and loss of immunity. In figure 5.8, we examined the effect of reducing the values of  $\alpha$  (recruitment rate into the susceptible class),  $\beta$  (probability that each contact is effective enough to cause infection),  $\epsilon$  (contact rate with contaminated environment),  $\psi$  (average contribution of each infected individual to the pathogen population of *Vibrio cholera*) and  $\tau$  (progression rate of protected individuals to the susceptible class) on the susceptible human population. We observed that there is a reduction in the population size of the susceptible class when these parameters are lowered. It is revealed that a desirable size can be obtained within the first five months by

implementing this strategy. Figure 5.9 shows that this strategy increases the size of the protected class for about six months before a reduction can be noticeable. In figure 5.10 we also observed a reduction in the size of the infected class. It was also confirmed that this strategy increases the number of recovered individuals, and this can happen for about four months before a reduction is noticeable.

## 6. Conclusion

This study has constructed a deterministic SPIRB model based on certain realistic assumptions and used the model to study the dynamics of cholera disease. Epidemiological analysis has been performed on the disease model. Under certain conditions, the disease-free equilibrium is locally asymptotically stable but not globally asymptotically stable. The disease-endemic equilibrium is globally asymptotically stable. In this dissertation, we have executed some interesting numerical simulations of the system using the Maple programming software. The numerical simulations showed that wise perturbations of the highly sensitive parameters of our model can be harnessed in order to effectively control and/or possibly eliminate the cholera disease from a population within a given period of time. The study has: shown that for a deterministic SPIRB model, the parameters required in determining the disease-free equilibrium and the carrying-capacity of the population are the recruitment rate  $\alpha$  and the natural death rate  $\alpha$ ; analyzed the sensitivity of the parameters of the basic reproduction number and has shown that the recruitment rate ( $\alpha$ ) into the susceptible class, the probability ( $\beta$ ) that each contact is effective enough to cause infection, the contact rate ( $\epsilon$ ) with contaminated environment, the average contribution ( $\psi$ ) of each infected individual to the pathogen population of *Vibrio cholerae*, and the progression rate ( $\chi$ ) of protected individuals to the susceptible class are the most sensitive parameters; shown that for local stability of the disease-free equilibrium to be attained, the progression rate of susceptible individuals to the protected class must be greater than the progression rate of protected individuals back into the susceptible class, and the rate at which infected humans leave the infected compartment must be greater than the rate at which bacterial exit the bacteria cohort; when the progression rate ( $\chi$ ) of protected individuals to the susceptible class satisfies  $\chi < 0$  with  $|\chi| \ll 1$ , the disease-free equilibrium is unstable, and there exists a locally asymptotically stable negative equilibrium, and when  $0 < \chi \ll 1$ , the disease-free equilibrium is stable, and a positive unstable equilibrium appears; a total compliance to hygienic regulations such as non-ingestion of cholera bacterium, and no contact with cholera patients is a necessity for total elimination of the cholera disease from a given population.

**Table 1.** Parameter Descriptions and Values

Parameter	Description	Value	Source
$\alpha$	Recruitment rate into the susceptible class.	0.0755	Tsetimi, Ossaiugbo & Atonuje, 2022
$\psi$	The average contribution of each infected individual to the pathogen population of <i>Vibrio cholerae</i> .	10cells/mL/ day	Codecco, 2021
$\delta$	Natural death rate.	0.0182	Tsetimi, Ossaiugbo & Atonuje, 2022
$\beta$	The probability that each contact is effective enough to cause infection	0.1	Assumed
$\epsilon$	Contact rate with contaminated environment.	$2.89 \cdot 10^{-6}/\text{day}$	Olaniyi & Ogbonna, 2021

Parameter	Description	Value	Source
$K$	Concentration of vibrio cholera in water.	1,000,000,000	Codecco, 2001
$\eta$	Cholera induced death rate.	0.00008	WHO, 2012
$\lambda$	Death rate of <i>Vibrio cholerae</i> .	0.0000002	Assumed
$\tau$	Progression rate of infected individuals to the recovered class due to treatment.	0 – 1	Olaniyi & Ogbonna, 2021
$q$	Rate of compliance to hygienic regulations such as non-ingestion of cholera bacterium, and no contact with cholera patients.	0 – 1	Olaniyi & Ogbonna, 2021
$b$	Progression rate of susceptible individuals to the protected cohort.	0.6	Assumed
$\chi$	Progression rate of protected individuals to the susceptible class.	0.2	Assumed
$\phi$	Loss rate of immunity of recovered individuals.	0.003	Olaniyi & Ogbonna, 2021
$\varphi$	Birth rate of <i>Vibrio cholerae</i> .	0.003 per day	Codecco, 2001
$\theta$	Rate of the compliance to sanitation by the infected cohort.	0 – 1	Assumed

## Declarations

### Source of Funding

This study did not receive any grant from funding agencies in the public or not-for-profit sectors.

### Competing Interests Statement

The authors declare that there are no competing interests.

### Consent for Publication

The authors declare that they consented to the publication of their original research work.

### Authors' Contributions

Both the authors took part in data collection, literature review, analysis, and manuscript writing equally.

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