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A Mathematical Model and Analysis of Cholera Dynamics

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Abstract
A member of the Vibrionaceae family, Vibrio cholerae is a dangerous bacterium. The human illness cholera is caused by the bacterium Vibrio cholerae. As a means of understanding the dynamics of cholera epidemics, we constructed a model. people who are vulnerable, people who are infectious, humans who have recovered, and the environment that bacteria need to multiply make up the model. The presence of both the disease-free and endemic equilibrium points was examined in our study. The jacobian matrix method was used to calculate the reproduction number. When the reproduction number was smaller than one, it was discovered that the disease-free equilibrium was locally asymptotically stable. Using sensitivity analysis, we were able to identify the parameter that was most affected by the basic reproduction number. The epidemic model's system of differential equations was modeled numerically and compared to the qualitative solutions in order to draw conclusions. According to the results, the number of vulnerable humans in the system reduces as the infected population grows.

Keywords: Cholera model,equilibrium points, reproduction number, sensitivity analysis, stability analysis.

1. Introduction

The bacterium Vibrio cholerae is the causative agent of cholera. A person may get cholera by ingesting food or water contaminated with Vibrio cholerae or by drinking contaminated water or other liquids [15]. The illness has a high mortality rate, particularly in cases when the epidemic is unnoticed and medical treatment is delayed. Whenever cholera breaks out, it disproportionately affects women and children. Globally, there are an estimated 1.4 to 4.3 million cases of cholera and 28000 to 142000 deaths due to the epidemic each year, according to a report by the World Health Organization. However, the exact number of cases cannot be obtained due to limitations in the monitoring system and a lack of clarity regarding different geographical areas. World Health Organization data shows that there were 33,421 recorded cases and 542 fatalities in Tanzania from 2015 to 2018 (based on the same source). During this time, 86% of all cholera cases occurred in Tanzania Mainland. Among these instances, it was found that 11.4% were youngsters less than five years old and elderly adults [3].
In their study, the scientists looked at the possibility of combining the next-generation matrix approach with cholera bacteriophage therapy [6]. Results demonstrate that therapy and bacteriophage integration decreased environmental cholera bacteria. To further

examine the role of media coverage in cholera transmission, the authors of [10] created an epidemic model based on child death rates as they relate to media coverage of the disease. A lack of public knowledge about the spread of cholera and effective methods to prevent it led to a decline in cholera cases in the community, according to their results. Incorporating the environmental component with V. Cholerae concentration in the water supply, researchers in Ghana used the Codeco compartmental (SIR) basic model to study the dynamic behavior of a fractional order of cholera. By using Ruth-Hurwitz stability criteria, we looked at the disease's local stability analysis. Without comprehensive and consistent surveillance, V. cholera might linger in the afflicted area forever or be brought in by asymptomatic immigrants, as happened in Haiti with the Napasesse peacekeepers. The significance of these findings for clinical medicine in the context of human-environment cholera sickness was shown by these findings. In addition, the model was expanded to include both human-to-human and environment-to-human transmission, suggesting that laboratory tests were the only reliable indicators of cholera outbreaks. While it was determined that cholera control measures and interventions should be a key towards risk reduction of transmission, it was noted that several critical and required factors were not explicitly addressed in the case of transmission. [4]. Between 41% and 95% of Zimbabweans fell victim to the cholera pandemic, and many made full recoveries, according to the authors' investigation on the disease in [2]. They focused on environmental factors and human-to-human transmission. Cholera is a water-borne illness, and in their mathematical model of environmental and epidemiological data, the authors of [5] took this into account. Disease dynamics in bacterial and phage populations, as well as cholera epidemiology, were the main points of discussion. It seemed from the results that the cholera epidemic was attributable to the reservoir's phage load.

A lot has been accomplished to improve the battle against the cholera outbreak, according to the public report [9], thanks to the cooperation with other partner organizations. However, it seemed that the focus was on therapy in the search for an antibiotic to cure cholera. First and foremost in the battle against cholera sickness is the ability to limit the cause. It was possible to reduce the severity of the cholera epidemic in the afflicted regions by taking the initiative and receiving support for the national response plan. Plus, it was a template for how to handle such public health crises in the future. According to some evaluations, the lack of access to clean water and sanitation, as well as the water supply's inability to treat water adequately at its source, was the primary cause contributing to the region's alarmingly high infection rate. By modifying the model from [2], which had included additional metrics, the authors in [8] created a mathematical model for the cholera transmission dynamic and placed an emphasis on public health decision making. The need of public health policymakers, epidemiologists, and modelers working together to achieve conceptual harmony was emphasized. Tanzania, and Dar es Salaam in particular, have conducted similar research on cholera outbreaks, but they have seldom used modeling tools. The training of future engineers and medical professionals, as well as researchers in related fields, relies heavily on mathematical models of the physical processes underlying human-observed natural phenomena.

2. Model description and formulation

The model divides the total human and population at any time (t)into four sub-populations (compartments) with respect to their disease status in the system. The total population, represented by $N(t\ )$, is divided into sub-populations of Susceptible humans (S), Infectious humans (I) and Recovered vector (R) and the environment (V). The total population becomes: $N\ (t) = S(t) + I(t) + R(t)$, where $N_{(0)} = S_{(0)}$.

$(S(t\ ))$; these are the population that are at risk of developing an infection from the Cholera disease, $(I\ (t\ ))$; this compartment consists people that are showing the symptoms of the Cholera disease, $(R(t\ ))$; these include those who have recovered from the disease and got temporal immunity.

The Susceptible humans are recruited into the population at a rate Ω. Susceptible humans acquire the disease through ingestion of contaminated foods and water. Contact with infectious humans at a rate α. Individuals recover from the disease at a rate β. Humans who are infected with Cholera die at a rate ω and the recovered humans may loose immunity and return to the susceptible compartment at a rate γ. The natural death rate of the entire human compartments is μ. Infectious humans contaminate the environment at a rate σ and the environment infect humans with the bacteria at a rate of η.

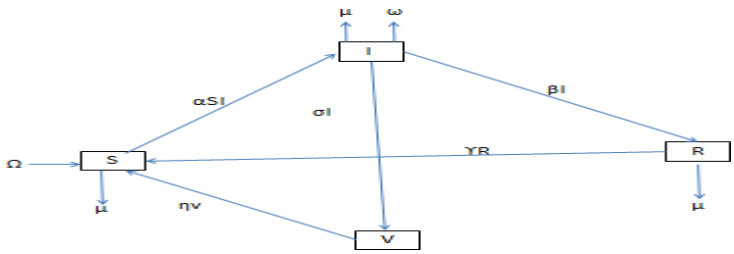


Figure 2.1: Model flow chart showing the compartments.

The following system of differential equations are obtained from the model in Figure 2.1

$$\left.\begin{aligned} \frac{dS}{dt} &= \Omega - \alpha SI - \mu S + \eta V + \gamma R \\ \frac{dI}{dt} &= \alpha SI - \mu I - \omega I - \sigma I - \beta I \\ \frac{dR}{dt} &= \beta I - \mu R - \gamma R \\ \frac{dV}{dt} &= \sigma I - \eta V \end{aligned}\right\} \tag{1}$$

3. Positivity and boundedness of solutions

The dynamic system is uniformly bounded in the proper subset $① \subset R^3_+$, under consideration that the total human population at any time t is given by;

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

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$$

$$\frac{dN}{dt} = \Omega - \alpha SI - \mu S + \gamma R + \alpha SI - \mu I - \omega I - \sigma I - \beta I + \beta I - \gamma R - \mu R$$

$$\frac{dN}{dt} = \Omega - \mu S - \mu I - \omega I - \sigma I - \mu R$$

In the absence of infection, there are no recovery. Then, initially $N(0) = S(0)$

$$\frac{dN}{dt} = \Omega - \mu S$$

$$\frac{dN}{dt} = \Omega - \mu N$$

Solving the differential equation, by separation of variables

$$\frac{dN}{\Omega - \mu N} = dt$$

Integrating the differential equation

$$\int \frac{dN}{\Omega - \mu N} \leq \int dt$$

$$-\frac{1}{\mu} In\,|\Omega - \mu N| \leq t + A$$

$$In\,|\Omega - \mu N| \geq -\mu(t + A)$$

$$\Omega - \mu N \geq \ell^{-\mu(t+A)} = \ell^{-\mu t}\ell^{-\mu A}$$

where $\ell^{-\mu A} = C$

$$\Omega - \mu N \geq C\ell^{-\mu t}$$

At $N(0) = N_0 \Rightarrow t = 0, N = N_0$

$$(\Omega - \mu N_0) = C$$

$$(\Omega - \mu N) \geq (\Omega - \mu N_0)\,\ell^{-\mu t}$$

By rearranging and simplifying

$$\frac{\blacktriangle}{\mu} - N \geq \frac{(\Omega - \mu N_0)}{\mu}\ell^{-\mu t}$$

As $t \to \infty$, the population size $N \to \frac{\blacktriangle}{\mu}$

And this implies that;

$0 \leq N < \frac{\Lambda}{\mu}$

and $N \leq \frac{\Lambda}{\mu}$

Therefore,

$$\Omega_t = (S, I, R) \in R^3 \quad +$$

$$: S + I + R \leq \frac{\Lambda}{\mu}$$

The solution set of the dynamic system of the equations in the model is bounded in the region such that $\Delta = \Omega_S + \Omega_I + \Omega_R$.This implies that the dynamic system of the model in the region is well-posed.

The solution of the system remains positive at any point in time ,if the initial values of all the variables are positive.

Theorem: Considering the dynamic system region such that;

$$\Phi = \{(S(t), I(t), R(t)) \in R_+^3 : S(0) > 0, I(0) > 0, R(0) > 0\},$$

then the solution of $\{(S(t), I(t), R(t)\}$ are positive for $t \geq 0$.

4. Disease free equilibrium

At disease free equilibrium, $I = R = V = 0$

$$\frac{dS}{dt} = \Omega - \alpha SI - \mu S + \eta V + \gamma R$$

$$\Omega - \alpha SI - \mu S + \eta V + \gamma R = 0$$

$$S^* = \frac{\Lambda}{\mu}$$

The disease free equilibrium;

$$S^*, 0, 0, 0 = \frac{\Lambda}{\mu}, 0, 0, 0 \tag{2}$$

5. Basic reproduction number

This is a threshold parameters that govern the spread of a disease in the population. whether the disease would persist or die out with time in the system. The basic reproduction number gives or tells the state of disease with time. The basic reproductive number is obtained by computing the Jacobian of the system at the disease free equilibrium by posing the condition that all eigenvalues of the corresponding characteristic equation must have negative real parts[12, 11].

Considering the Jacobian matrix of the system of differential equations;

$$J\left(S^*, I^*, V^*, R^*\right) = \begin{pmatrix} \frac{\partial S}{\partial S} & \frac{\partial S}{\partial I} & \frac{\partial S}{\partial V} & \frac{\partial S}{\partial R} \\ \frac{\partial I}{\partial S} & \frac{\partial I}{\partial I} & \frac{\partial I}{\partial V} & \frac{\partial I}{\partial R} \\ \frac{\partial V}{\partial S} & \frac{\partial V}{\partial I} & \frac{\partial V}{\partial V} & \frac{\partial V}{\partial R} \\ \frac{\partial R}{\partial S} & \frac{\partial R}{\partial I} & \frac{\partial R}{\partial V} & \frac{\partial R}{\partial R} \end{pmatrix}$$

$$J\left(S^*, I^*, V^*, R^*\right) = \begin{pmatrix} -\alpha I - \mu & -\alpha S & 0 & \gamma \\ \alpha I & \alpha S - (\mu + \omega + \sigma + \beta) & 0 & 0 \\ 0 & \sigma & -\eta & 0 \\ 0 & \beta & 0 & -(\gamma + \mu) \end{pmatrix} \quad (3)$$

The Jacobian matrix at disease free equilibrium is given by the relation;

$$J\left(\frac{\Lambda}{\mu}, 0, 0, 0\right) = \begin{pmatrix} -\mu & -\frac{\alpha\Lambda}{\mu} & 0 & \gamma \\ 0 & \frac{\alpha\Lambda}{\mu} - (\mu + \omega + \sigma + \beta) & 0 & 0 \\ 0 & \sigma & -\eta & 0 \\ 0 & \beta & 0 & -(\gamma + \mu) \end{pmatrix} \quad (4)$$

The determinant of the Jacobian matrix at free equilibrium;

$$\begin{vmatrix} -\mu - A & -\frac{\alpha\Lambda}{\mu} & 0 & \gamma \\ 0 & \frac{\alpha\Lambda}{\mu} - (\mu + \omega + \sigma + \beta) - A & 0 & 0 \\ 0 & \sigma & -\eta - A & 0 \\ 0 & \beta & 0 & -(\gamma + \mu) - A \end{vmatrix} = 0$$

Where A is the eigenvalues.

By determining the eigenvalues and selecting the dorminant eigenvalue, the basic reproduction is obtained as;

The determinants are as follows;

$$\frac{\alpha\blacktriangle}{\mu} - (\mu + \omega + \sigma + \beta).$$

Therefore, the basic reproduction number is given by the relation.

$$R_0 = \frac{\alpha\blacktriangle}{\mu} - (\mu + \omega + \sigma + \beta) \tag{5}$$

6. Endemic equilibrium

By setting the system of equations to zero and evaluating the state variables, the endemic equilibrium points would be in the form;

$$(EE) = S^*, I^*, V^*, R^*$$

From;

$$\frac{dS}{dt} = \alpha SI - (\mu + \omega + \sigma + \beta) I = 0$$

$$S^* = \frac{(\mu + \omega + \sigma + \beta)}{\alpha}$$

Also;

$$\frac{dR}{dt} = \beta I - (\gamma + \mu) R = 0$$

$$I = \frac{(\gamma + \mu) R}{\beta}$$

$$I^* = \frac{(\gamma + \mu) R^*}{\beta}$$

$$\Omega - \alpha SI - \mu S + \gamma R = 0$$

$$\blacktriangle - (\alpha I + \mu) S + \gamma R = 0$$

$$\gamma R = (\alpha I + \mu) S - \blacktriangle$$

$$R^* = \frac{(\alpha I^* + \mu) S^* - \blacktriangle}{\gamma}$$

$$\sigma I - \eta V = 0$$

$$V^* = \frac{\sigma I^*}{\eta},$$

$$V^* = \frac{\sigma}{\eta}\frac{(\gamma + \mu) R^*}{\beta}$$

Therefore, the endemic equilibrium state S^*, I^*, V^*, R^*

$$\frac{(\mu + \omega + \sigma + \beta)}{\alpha}, \frac{(\gamma + \mu) R^*}{\beta}, \frac{(\alpha I^* + \mu) S^* - \blacktriangle}{\gamma}, \frac{\sigma}{\eta}\frac{(\gamma + \mu) R^*}{\beta}$$

6.1. Stability of the endemic equilibrium

Considering the Lyaponuv function defined as;

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

The derivative of L along the solution of the system is directly;

$$\frac{dL}{dt} = \frac{S - S^*}{S} \frac{dS}{dt} + \frac{I - I^*}{I} \frac{dI}{dt} + \frac{V - V^*}{V} \frac{dV}{dt} + \frac{R - R^*}{R} \frac{dR}{dt}$$

$$\frac{dL}{dt} = \left\{ \frac{S - S^*}{S} \left[\Omega - \alpha SI - \mu S + \gamma R \right] + \frac{I - I^*}{I} \left[\alpha SI - (\mu + \omega + \sigma + \beta) I \right] + \frac{V - V^*}{V} \left[\sigma I - \eta V \right] + \frac{R - R^*}{R} \left[\beta I - (\gamma + \mu) R \right] \right\}$$

By expansion and simplification;

$$\frac{dL}{dt} = \left\{ \begin{array}{l} \Omega - \alpha SI - \mu S + \gamma R - \frac{\Omega S^*}{S} + \alpha S^* I + \mu S^* - \frac{\gamma R S^*}{S} + \alpha SI \\ -\alpha S^* I + (\mu + \omega + \sigma + \beta) \frac{IS^*}{S} + \sigma I - \eta V - \frac{\sigma IS^*}{S} \\ + \frac{\eta V S^*}{S} + \beta I - (\gamma + \mu) R - \frac{\beta IS^*}{S} + \frac{(\gamma + \mu) S^* R}{S} \end{array} \right\}$$

Let,

$$\frac{dL}{dt} = P - Q,$$

where P are the positive terms and Q are the negative terms, such that;

$$P = \Omega + \mu S^* + (\mu + \omega + \beta) \frac{IS^*}{S} + \frac{\eta V S^*}{S} + \beta I + \frac{\mu S^* R}{S}$$

and,

$$Q = \mu S + \frac{\Omega S^*}{S} + (\mu + \omega) I + \eta V + \mu R + \frac{\beta IS^*}{S} + (\gamma + \mu) R$$

If $P < Q$, then $\frac{dL}{dt} \leq 0$. $\frac{dL}{dt} = 0$, if and only if $S = S^*, I = I^*, V = V^*$, and $R = R^*$.

The largest invariant set in $S^*, I^*, V^*, R^*) \in ① : \frac{dL}{dt} = 0$ is a singleton of E^*, where E^* is the endemic equilibrium.

This implies that the endemic equilibrium is globally asymptotically stable [7, 13].

7. Sensitivity of analysis

In this section, we perform sensitivity analysis to determine the contribution of each parameter to the basic reproduction number. This analysis determines the level of contribution of each parameter value to the reproduction number [14].

The basic reproductive number;

$$R_0 = \frac{\alpha\blacktriangle - \mu(\mu + \omega + \sigma + \beta)}{\mu}$$

Sensitivity index of the model parameter is given by the relation;

$$S^{R0}_{X} = \frac{\partial R_0}{\partial X} * \frac{X}{R_0} \tag{6}$$

where X represents any parameter in the model.

For α :

$$S^{R0}_{\alpha} = \frac{\partial R_0}{\partial \alpha} * \frac{\alpha}{R_0},$$

$$S^{R_0}_{\alpha} = \frac{\blacktriangle}{\mu} * \frac{\alpha\mu}{\alpha\blacktriangle - \mu(\mu + \omega + \sigma + \beta)}$$

$$S^{R_0}_{\alpha} = \frac{\alpha\blacktriangle}{\alpha\blacktriangle - \mu(\mu + \omega + \sigma + \beta}$$

For μ :

$$S^{R0}_{\mu} = \frac{\partial R_0}{\partial \mu} * \frac{\mu}{R_0},$$

$$S^{R_0}_{\mu} = -\frac{\alpha\blacktriangle + \mu^2}{\mu^2} * \frac{\mu^2}{\alpha\blacktriangle - \mu(\mu + \omega + \sigma + \beta}$$

$$S^{R_0}_{\mu} = -\frac{\alpha\blacktriangle + \mu^2}{\alpha\blacktriangle - \mu(\mu + \omega + \sigma + \beta}$$

For ω :

$$S^{R0}_{\omega} = \frac{\partial R_0}{\partial \omega} * \frac{\omega}{R_0},$$

$$S^{R0}_{\omega} = -\frac{\omega\mu}{\alpha\blacktriangle - \mu(\mu + \omega + \sigma + \beta}$$

For σ :

$$S^{R0}_{\sigma} = \frac{\partial R_0}{\partial \sigma} * \frac{\sigma}{R_0},$$

$$S^{R0}_{\sigma} = -\frac{\sigma\mu}{\alpha\blacktriangle - \mu(\mu + \omega + \sigma + \beta}$$

For β :

$$S^{R0}_{\beta} = \frac{\partial R_0}{\partial \beta} * \frac{\beta}{R_0},$$

$$S_{\beta}^{R0} = -\frac{\beta\mu}{\alpha\blacktriangle - \mu(\mu + \omega + \sigma + \beta}$$

For $\blacktriangle$:

$$S_{\blacktriangle}^{R0} = \frac{\partial R_0}{\partial \blacktriangle} * \frac{\blacktriangle}{R_0},$$

$$S_{\blacktriangle}^{R_0} = \frac{\alpha\mu}{\mu(\alpha\blacktriangle - \mu(\mu + \omega + \sigma + \beta)}$$

$$S_{\blacktriangle}^{R0} = \frac{}{(\alpha\blacktriangle - \mu(\mu + \omega + \sigma + \beta)}$$

Table 1: Sensitivity analysis of the parameter values.

Parameter	Sensitivity Index
α	0.263839
μ	-1.062007
Ω	0.263839
ω	-0.956416
σ	-2.3985
β	-1.1992

Table 1 shows the contribution of each parameter to the basic reproduction number. Given that the reproduction number is less than unity, an increase in the contact rate and human recruitment rates by 10% would increase cause an increase in the basic reproduction number by 2.6%. Moreover, increasing the recovery rate by 10% would reduce the basic reproduction number by 11%. However, decreasing the recovery rate by 10% would increase the reproduction number by 11%.

8. Numerical results

Using a software program, we ran numerical simulations on the system of differential equations in figure 2.1 using a fourth-order Range-Kutta method to demonstrate how the model parameters affected the results. Over a predetermined time frame, the Range-Kutta fourth-order technique was used to solve the systems of differential equations. Table 2 contains the parameter values that were used in the simulations. This model was run during the first three months of the cholera pandemic.

8.1. Population dynamics

To find out how the varied populations of susceptible, recovered, and infectious individuals changed over time, we ran numerical simulations of their system of differential equations. As the infected population grows, the number of vulnerable persons seems to be steadily decreasing. We found that there was a negative correlation between the

Table 2: Parameter values used in the simulation.

Parameter	Value	Reference
▲	0.000096274	[10]
μ	0.00002537	[10]
ω	0.0004	[16]
β	5	[16]
σ	10	[10]
η	0.075	assumed
α	0.011	[16]
γ	0.002	assumed

susceptible and infectious populations as shown in figure 8.1. This could be attributed to the number of infectious and recovered populations having a direct relationships.

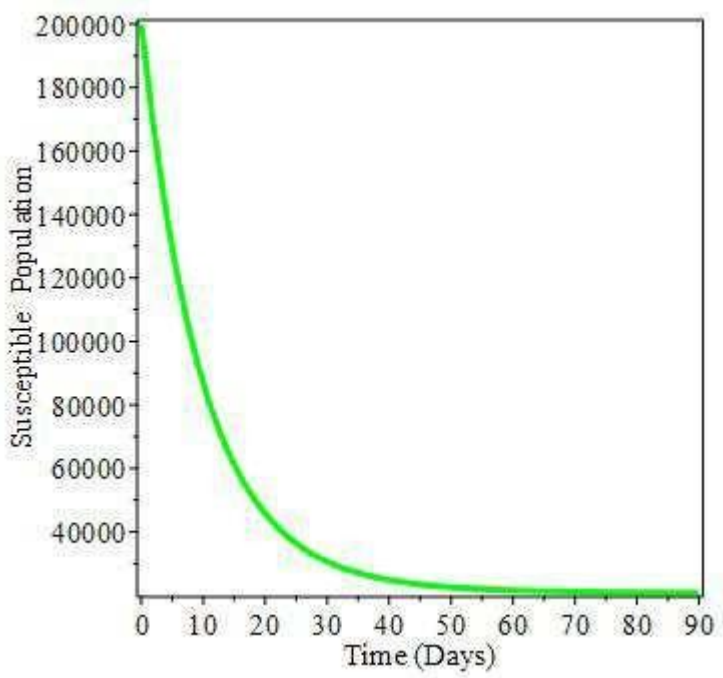


Figure 8.1: Susceptible population.

In figure 8.4, there has been an increase in the number of bacteria concentrations. This could be attributed to the infectious humans contributions to the pollution of the environment. The activities of humans continue to contaminate the environment. This could be the contributing factor of the exponential increase in the number bacteria in the environment as indicated in figure 8.4.

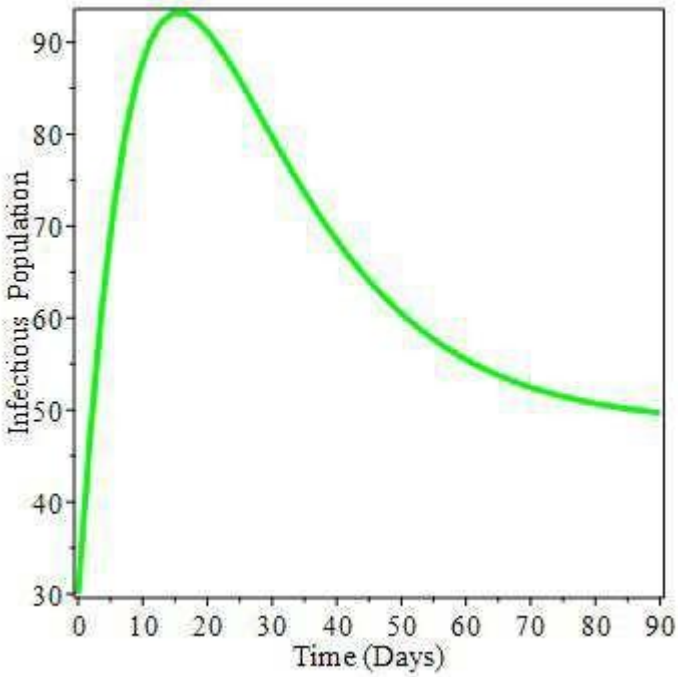


Figure 8.2: Infectious population.

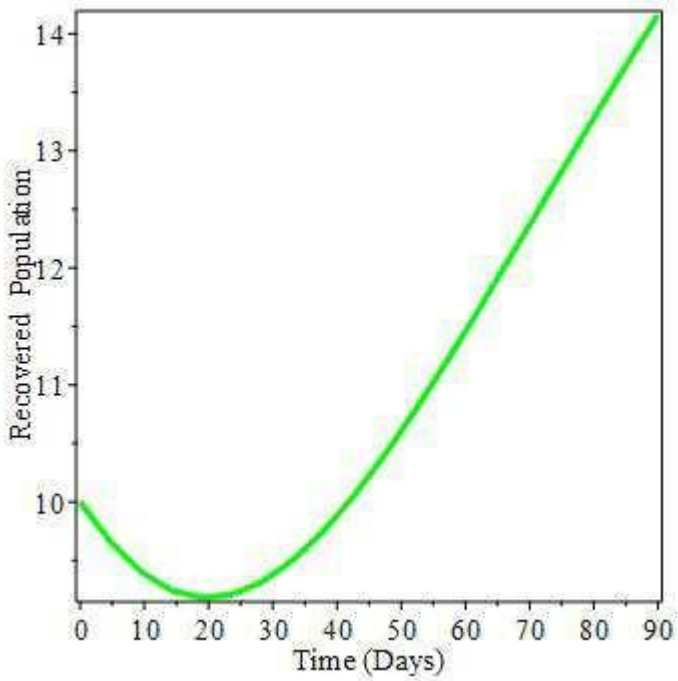


Figure 8.3: Recovered population.

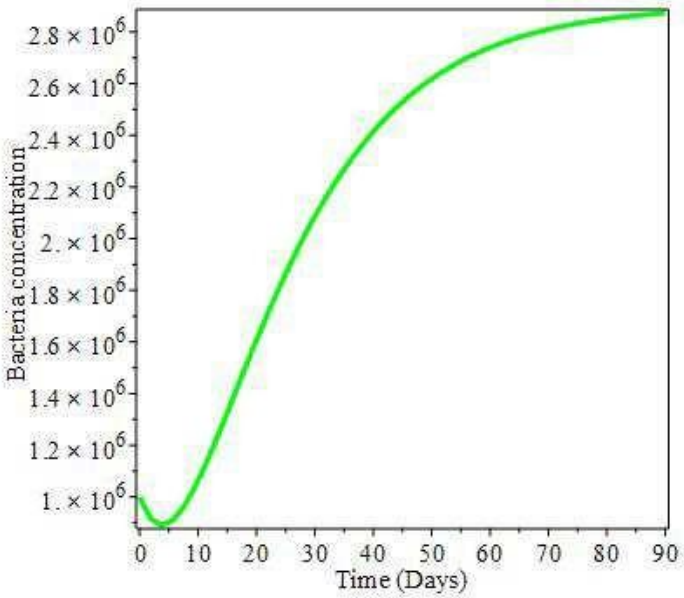


Figure 8.4: Bacteria concentration.

8.1.1 Effects of human contact rate

Numerical analysis of the rate of contact between the susceptible human populations and the infectious human population was conducted to see whether or not the contact rate contribute significantly to the epidemics of the cholera infections in the environment. The

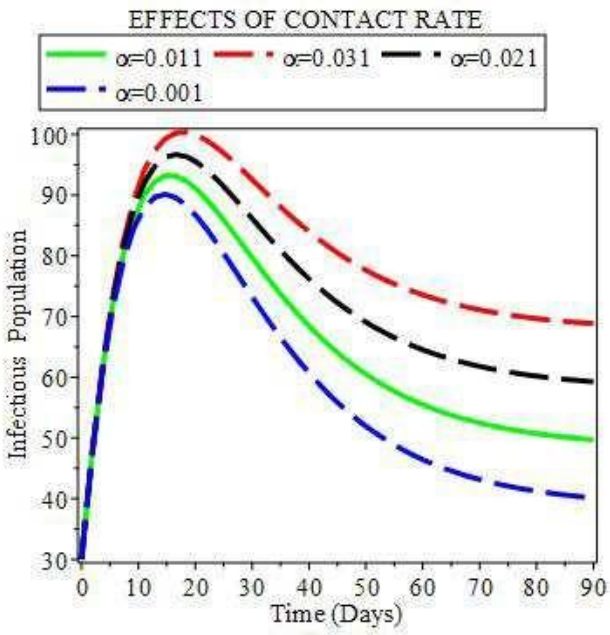


Figure 8.5: Effects of increasing or decreasing contact rate.

diagram in figure 8.5 shows a positive effects of the contact rate in disease transmission. An increase in human interactions contribute significantly to the spread of the cholera infections in the system.

8.1.2 Effects of infectious contact with environment

Simulation of the system of differential equations from the model in figure 2.1 showed a change in the concentration of the bacteria population in the environment. As infectious humans interact with the environment by activities that contaminate the environment, the bacteria responsible for the cholera infections increases in the environment.

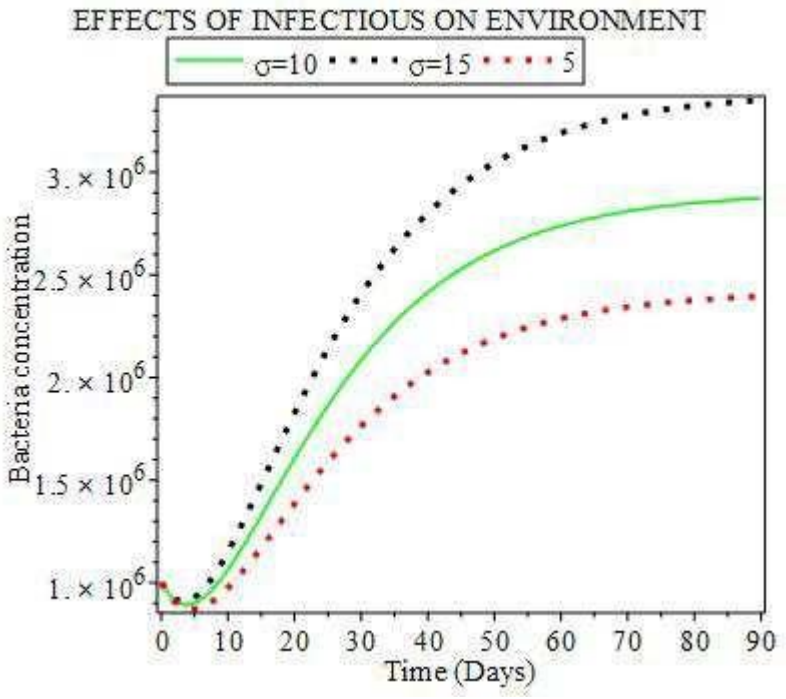


Figure 8.6: Effects of increasing or decreasing infectious contact with environment.

9. Concluding remarks

This work presents the development of an epidemic model that describes the dynamics of cholera transmission. We found that our model has an infection-free equilibrium that is globally stable whenever the basic reproductive number is less than one, and we also determined the basic reproductive number. To find out how each parameter affected the reproduction number, we ran a sensitivity analysis on the fundamental reproductive number. A ten percent rise in the recruitment rate improves the basic reproduction number, according to the study of the contribution of each parameter value to the reproduction number.

figure according to table 1. The re-production number would be larger than one, which means the infection would remain. Additionally, as shown in table 1, a percentage increase to the contact rate would result in an increase to the reproduction number. Because of this, the virus would remain dormant. According to the computer models, the overall number of infected persons has increased while the vulnerable population has decreased. There is a negative correlation between the number of vulnerable persons and the number of infected humans. To determine whether the contact rate substantially contributes to environmental cholera outbreaks, researchers analyzed the rate of contact between susceptible and infected people groups. A favorable impact of the contact rate on disease transmission is shown in figure 8.5's diagram. There is a strong correlation between the rise in human contact and the acceleration of the cholera epidemic. A shift in environmental bacterial concentration was shown by simulation of the model's system of differential equations (figure 2.1). The bacteria that cause cholera diseases are becoming more prevalent in the environment due to human activities that pollute it.

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