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Mathematical Modelling and Analysis of the Dynamics of Cholera

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Abstract

Vibrio cholerae is a pathogenic bacteria belonging to the family Vibrionaceae. Vibrio cholerae is the causative agent of Cholera disease in humans. We developed an epidemic model for the dynamics of cholera infections. The model consist of four compartments; the susceptible humans, infectious humans, the recovered humans and the environment that serves as a breeding ground for the bacteria. We conducted an analysis on the existence of all the equilibrium points; the disease free equilibrium and endemic equilibrium. The reproduction number was computed by using jacobian matrix approach. Disease free equilibrium was found to be locally asymptotically stable if the the reproduction number was less than one. The most sensitive parameter to the basic reproduction number was determined by using sensitivity analysis. Numerical simulations of the system of differential equations of the epidemic model was carried out for interpretations and comparison to the qualitative solutions. The findings showed that as the number of infectious population increases, the number of susceptible human decreases in the system.

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

1. Introduction

Cholera is a disease caused by bacteria known as Vibrio cholerae. Cholera enters into a person through drinking of contaminated water and drinks or consumption of food contaminated with V. Cholerae [15]. The disease leads to rapid loss of lives especially when the outbreak is not discovered in time and when immediate medical intervention is not provided. The most affected group with cholera outbreak are women and children. According to the World Health Organization report, globally, the actual number of cholera cases even though cannot be obtain due to lack of clear monitoring system with regard to different geographical areas and limitations in surveillance system to access full information but rather it is estimated that each year there are about 1.4 to 4.3 million cases of cholera with 28000 to 142000 deaths due to cholera epidemic. From the same source of information, WHO confirmed that, between August 2015 to January, 2018 about 33,421 cases were reported and left 542 deaths in Tanzania. Within this period Tanzania Mainland accounted 86 percent of the total cases as cholera disease. It was further discovered that among these cases 11.4 percent were children below five years old and old people [3].

The work done authors in [6] investigated cholera bacteriophage combined with treatment using the next generation matrix method. The analysis of the findings shows that the incorporation of both bacteriophage and treatment reduced cholera bacteria in the environment. Furthermore, authors in [10] developed an epidemic model on child mortality for cholera transmission by media coverage to see the impact of media to cholera transmission. Their findings showed a decrease of cholera infections in the population due to lack of public awareness on cholera spread and appropriate measures to limit it. [1] carried out a research on the dynamic behavior of a fractional order of cholera model in Ghana using Codeco compartmental (SIR) basic model with incorporation of the environmental component with concentration of V. Cholerae in water supply. The local stability analysis of the disease was investigated through Ruth-Hurwitz stability conditions.

There is a high chance for V. Cholera to lurk indefinitely in the affected environment or imported from asymptomatic immigrants as it was in Haiti with the Napasesse peacekeepers, if no systematic thorough monitoring. This finding, proved the clinical medical importance on observations; human in Society-environment cholera disease. Further the model was extended based on human to human and environment to human transmission where measures also seemed to be limited to laboratory test as determinant of cholera outbreak. On the case of transmission pointed out that some of important and necessary elements were not clearly addressed, though concluded that in cholera control measures and interventions should be a key towards risk reduction of transmission. [4]. Authors in [2], investigated cholera outbreak in Zimbabwe focusing on human-to- human transmission and the environment, the discovery played a big part about (41-95) percent had cholera epidemic and many recovered. Authors in [5] employed a mathematical model of epidemiological and environmental observations of cholera outbreak as a waterborne disease. The focus was on the epidemiological of cholera and population dynamics of bacteria and phage. The outcome suggested that the increase of cholera outbreak was

attributed to the amount of phage in the reservoir.

According to the available report [9], with the collaboration with other partner Organization a lot has been done to enhance the fight for cholera epidemic endeavor. But it seemed that the emphasis was put to treatment looking for cholera antibiotic. The important thing is to be able contain the cause as the first priority in the fight of cholera disease. Through initiative and support established on national cholera response plan addressing cholera outbreak challenge in the affected areas showed improved ability to calm the outbreak. And this served as a model to respond to future public heath attention In the region, according to some assessment, the main factor associated with a severe spread of infection was the limited availability of safe water and sanitation, also water supply lacked authentic capacity of adequate water treatment from the sources of water.

Authors in [8] developed a mathematical model for the transmission dynamic of cholera and stressed on public health decision making through modification of the model in [2] which had incorporated other measures. The emphases was put on the collaboration between the policy-makers, epidemiologists and modelers in public health to ensure there harmonization of ideas. Analogous studies have been underway in Tanzania particular in Dar es salaam on cholera outbreak but rarely using modeling techniques. Mathematical modeling of physical systems of a humanly natural phenomenon is practically essential to development of experts in engineering field and health practitioners with a direct application in the field of health science and other discipline.

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.

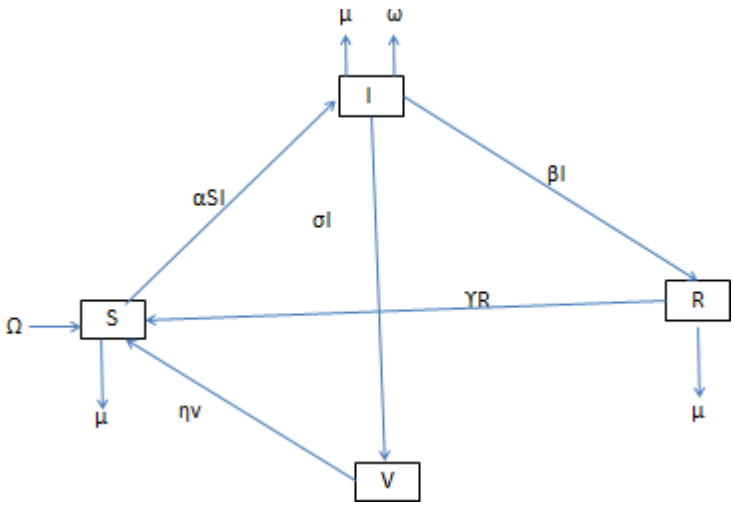

Figure 2.1: Model flow chart showing the compartments.

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 $\Theta \subset R_+^3$, 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 $N\,(0) = N_0 \Longrightarrow t = 0, N = N_0$

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

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

By rearranging and simplifying

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

As $t \to \infty$, the population size $N \to \dfrac{\Omega}{\mu}$

And this implies that;

$0 \leq N < \dfrac{\Omega}{\mu}$ and $N \leq \dfrac{\Omega}{\mu}$

Therefore,

$$\Theta_t = \left\{(S, I, R) \in R_+^3 : S + I + R \leq \frac{\Omega}{\mu}\right\}$$

The solution set of the dynamic system of the equations in the model is bounded in the region such that $\Delta = \Theta_S + \Theta_I + \Theta_R$.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;

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

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

4. Disease free equilibrium

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

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

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

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

The disease free equilibrium;

$$\left(S^*, 0, 0, 0\right) = \left(\frac{\Omega}{\mu}, 0, 0, 0\right) \tag{2}$$

4.1. Stability of the disease free equilibrium

Proposition 4.1. Disease free equilibrium point is locally asymptotically stable if the basic reproduction number (R_0) is less than one ($R_0 < 1$) and unstable if the basic reproduction number is greater than one ($R_0 > 1$).

Proof. The disease free equilibrium is given by; $\left(\frac{\Omega}{\mu}, 0, 0, 0\right)$ has the basic reproductive number;

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

such that; $R_0 < 0$

$$\frac{\alpha\Omega}{\mu} - (\mu + \omega + \sigma + \beta) < 0$$

$$\frac{\alpha\Omega}{\mu(\mu + \omega + \sigma + \beta)} < 1$$

where,

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

and $R_0 < 1$. ■

Given that $R_0 < 1$, therefore,the disease free equilibrium point is locally asymptotically stable.

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;

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

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

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

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

The determinant of the Jacobian matrix at free equilibrium;

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

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;

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

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

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

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;

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

From;

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

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

Also;

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

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

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

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

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

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

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

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

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

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

Therefore, the endemic equilibrium state $\left(S^*, I^*, V^*, R^*\right)$

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

6.1. Stability of the endemic equilibrium

Considering the Lyaponuv function defined as;

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

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

$$\begin{aligned} \frac{dL}{dt} &= \left(\frac{S - S^*}{S}\right)\frac{dS}{dt} \\ &\quad + \left(\frac{I - I^*}{I}\right)\frac{dI}{dt} \\ &\quad + \left(\frac{V - V^*}{V}\right)\frac{dV}{dt} + \left(\frac{R - R^*}{R}\right)\frac{dR}{dt} \end{aligned}$$

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

By expansion and simplification;

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

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,

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

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 $\left\{S^*, I^*, V^*, R^*) \in \Theta : \frac{dL}{dt} = 0\right\}$ 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;

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

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

$$S_X^{R_0} = \frac{\partial R_0}{\partial X} * \frac{X}{R_0} \tag{6}$$

where X represents any parameter in the model.

For α :

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

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

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

For μ :

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

$$S_\mu^{R_0} = -\left(\frac{\alpha\Omega + \mu^2}{\mu^2}\right) * \left(\frac{\mu^2}{\alpha\Omega - \mu(\mu + \omega + \sigma + \beta}\right)$$

$$S_\mu^{R_0} = -\frac{\left(\alpha\Omega + \mu^2\right)}{\alpha\Omega - \mu(\mu + \omega + \sigma + \beta}$$

For ω :

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

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

For σ :

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

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

For β :

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

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

For Ω :

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

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

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

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

Numerical simulation was carried out to show the impact of the model parameters by employing fourth order Range-Kutta scheme on the system of differential equations in figure 2.1 using a software. The systems of differential equations were solved over a specific period of time period using Range-Kutta fourth order scheme. The parameter values used in the simulations are found in the table 2. The simulation was done within three months period of the cholera epidemic.

8.1. Population dynamics

We performed the numerical simulations of the system of differential equations of the susceptible humans, recovered humans and the infectious humans to determine the changes in the various populations of these compartments with time. There seems to be a continuous decrease in the number of susceptible humans as the number of the infectious population increases with time. Our findings showed an inverse relationship between the

Table 2: Parameter values used in the simulation.

Parameter	Value	Reference
Ω	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.

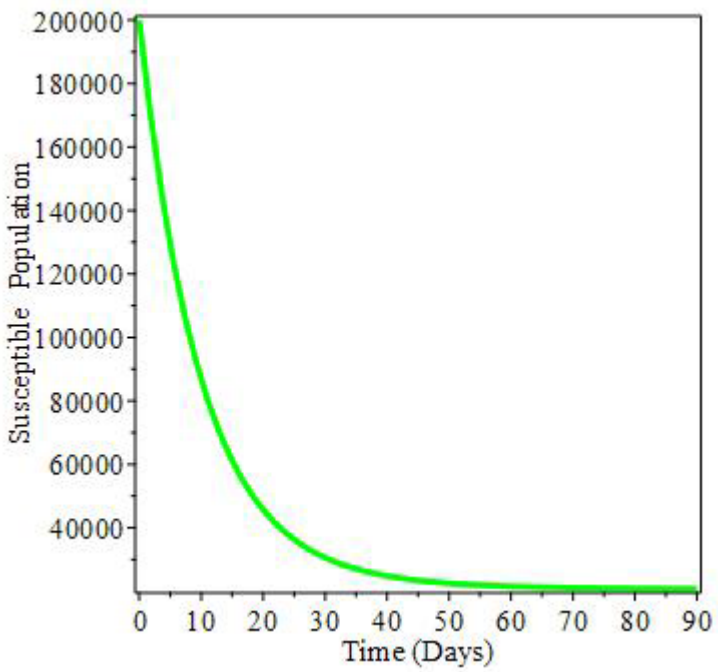

Figure 8.1: Susceptible population.

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.

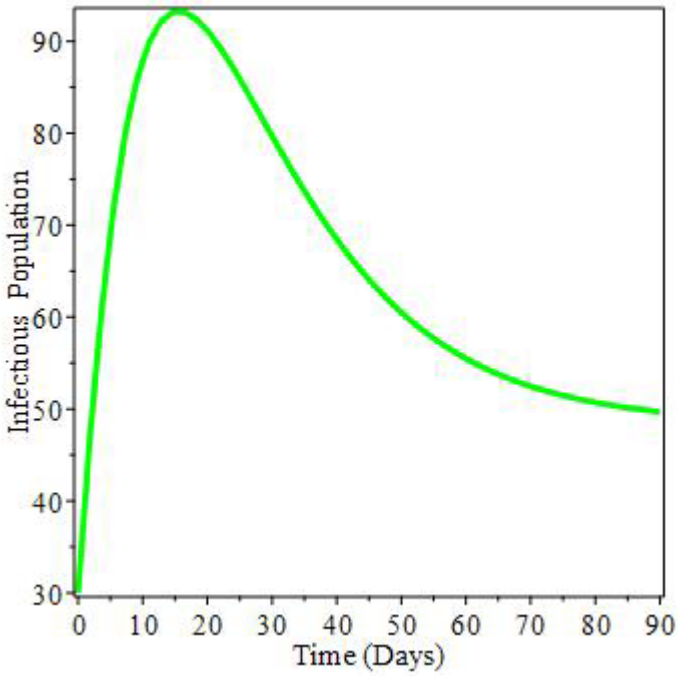

Figure 8.2: Infectious population.

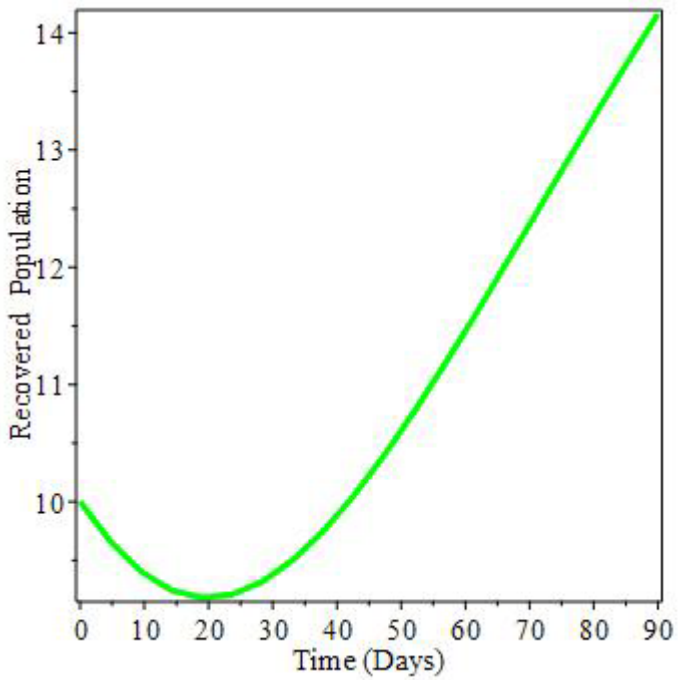

Figure 8.3: Recovered population.

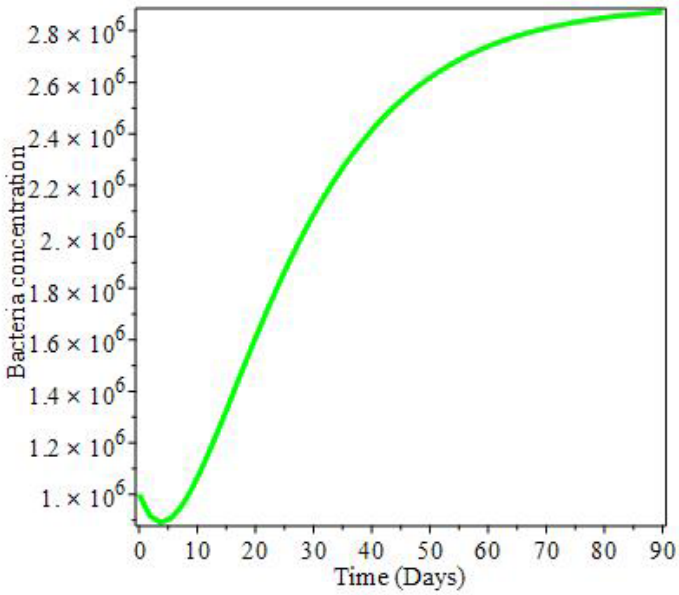

Figure 8.4: Bacteria concentration.

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

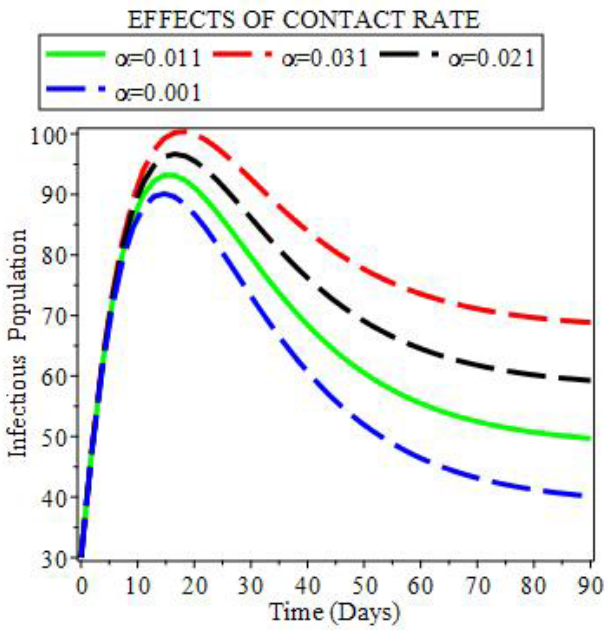

Figure 8.5: Effects of increasing or decreasing contact rate.

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.

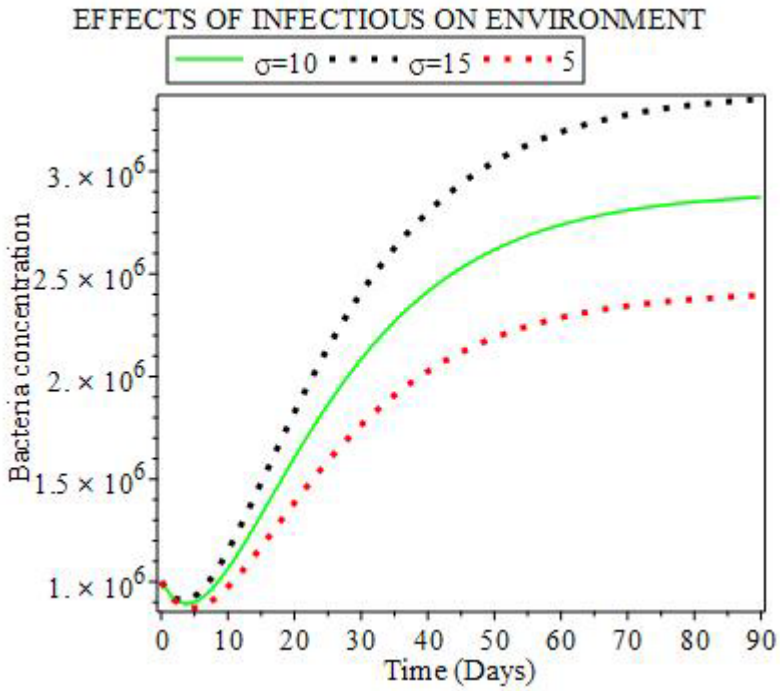

Figure 8.6: Effects of increasing or decreasing infectious contact with environment.

9. Concluding remarks

In this paper, an epidemic model for the transmission dynamics of cholera was developed. The basic reproductive number was derived and established that our model has a globally stable infection free equilibrium whenever the basic reproductive number is less than one. We performed the sensitivity analysis of the basic reproductive number to each of the parameters to investigate the significance of each to the reproduction number. The analysis of the contribution of each parameter value to the reproduction number showed that an increase in the recruitment rate by ten percent, increases the basic reproduction

number as shown in table 1. This implies that, the infection would persist as the reproduction number would be greater than unity. Also, by increasing the contact rate by a percentage, it would increase the reproduction number as indicated in table 1. This would lead to the persistence of the infection. The numerical simulations showed that as the susceptible population decreases, there has been an increase in the total population of the infectious humans. The relationship between the susceptible human and the infectious humans are inversely proportional. 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 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. 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.

References

[1] Taofeek Abdulai. *Using Atanackovic and Stankovic Numerical Method to Investigate Fractional Order Cholera Model*. PhD thesis, 2015.

[2] Camilo J Acosta, Claudia M Galindo, John Kimario, Kesheni Senkoro, Honorathy Urassa, Climent Casals, Manuel Corachán, N Eseko, Marcel Tanner, Hassan Mshinda, et al. Cholera outbreak in southern tanzania: risk factors and patterns of transmission. *Emerging infectious diseases*, 7 (3 Suppl): 583, 2001.

[3] Jason R Andrews and Sanjay Basu. Transmission dynamics and control of cholera in haiti: an epidemic model. *The Lancet*, 377 (9773): 1248–1255, 2011.

[4] Dennis L Chao, M Elizabeth Halloran, and Ira M Longini. Vaccination strategies for epidemic cholera in haiti with implications for the developing world. *Proceedings of the National Academy of Sciences*, 108 (17): 7081–7085, 2011.

[5] Francisco J Luquero, Marc Rondy, Jacques Boncy, André Munger, Helmi Mekaoui, Ellen Rymshaw, Anne-Laure Page, Brahima Toure, Marie Amelie Degail, Sarala Nicolas, et al. Mortality rates during cholera epidemic, haiti, 2010–2011. *Emerging infectious diseases*, 22 (3): 410, 2016.

[6] Oluwole D Makinde, Daniel S Mgonja, and Estomih S Massawe. Computational modelling of cholera bacteriophage with treatment. 2015.

[7] Oluwole Daniel Makinde. Adomian decomposition approach to a sir epidemic model with constant vaccination strategy. *applied Mathematics and Computation*, 184 (2): 842–848, 2007.

[8] Janeth Libent Mghase. *Investigation on Risk Factors Influencing Cholera Outbreaks: A Case of Buguruni Ward in Ilala Municipality Dar es Salaam*. PhD thesis, The Open University of Tanzania, 2014.

[9] Melinda Moore, Philip Gould, and Barbara S Keary. Global urbanization and impact on health. *International journal of hygiene and environmental health*, 206 (4-5): 269–278, 2003.

[10] Musundi Beryl Onyuma. *modelling cholera transmission incorporating media coverage*. PhD thesis, MOI UNIVERSITY, 2017.

[11] Shaibu Osman and Oluwole Daniel Makinde. A mathematical model for co-infection of listeriosis and anthrax diseases. *International Journal of Mathematics and Mathematical Sciences*, 2018.

[12] Shaibu Osman, Oluwole Daniel Makinde, and David Mwangi Theuri. Stability analysis and modelling of listeriosis dynamics in human and animal populations. *Global Journal of Pure and Applied Mathematics*, 14 (1): 115–137, 2018a.

[13] Shaibu Osman, Oluwole Daniel Makinde, and David Mwangi Theuri. Mathematical modelling of transmission dynamics of anthrax in human and animal population. *Mathematical Theory and Modelling*, 2018b.

[14] Shaibu Osman, Oluwole Daniel Makinde, and David Mwangi Theuri. Stability analysis and modelling of listeriosis dynamics in human and animal populations. *Global Journal of Pure and Applied Mathematics*, 14 (1): 115–137, 2018c.

[15] Mercedes Pascual, Menno J Bouma, and Andrew P Dobson. Cholera and climate: revisiting the quantitative evidence. *Microbes and Infection*, 4 (2): 237–245, 2002.

[16] Jin Wang and Chairat Modnak. Modeling cholera dynamics with controls. *Canadian applied mathematics quarterly*, 19 (3): 255–273, 2011.