TRANSMISSION DYNAMICS OF CHOLERA MODEL WITH CONTROL MEASURES

BY

ODO, Chika Emelda PhD/SPS/2017/936

DEPARTMENT OF MATHEMATICS, FEDERAL
UNIVERSITY OF TECHNOLOGY,

MINNA, NIGERIA.

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ABSTRACT

Cholera is an infectious intestinal disease that occurs as a result of poor sanitation and lack of basic education in its transmission. It is characterized by profuse vomiting and severe diarrhea when an individual eats food or drinks water contaminated with the *Vibrio cholerae*. In this present work, a dynamic mathematical model that explicitly simulates the transmission mechanism of cholera by considering the role of control measures and the environment in transmitting the disease is developed. The model comprises two populations: the human population and the bacteria population and the number of awareness programs driven by disease prevalence and media coverage. The nextgeneration method is used to compute the basic reproduction number, $_0$. Moreover, the sensitivity indices of each parameter with respect to $_0$ of the model are computed. To show the impact of the model parameters, the model equations were solved over a specific time period using the Homotopy Perturbation Method. Necessary conditions of the optimal control problem were analysed using Pontryagin's maximum principle with control measures such as awareness campaigns, vaccination of susceptible individuals, treatment of infected individuals and treatment of water bodies used to optimize the objective function. The results revealed that both the Disease-Free and Endemic Equilibria are shown to be locally and globally stable for $_0$ values less than unity and unstable otherwise. The model simulations confirm the significant role played by control measures (education, vaccination, therapeutic treatment and treatment of water bodies) and the bacteria in the environment in the transmission dynamics as well as reducing the spread of cholera. The optimal control results revealed that the best strategy for controlling cholera is the application of all control measures and this is attained when $_{c_0}$ 0.08. The research also affirmed that the worst-case scenario occurs when there is no any control strategy for the epidemic. This research revealed that the effective reproduction number $_{c_0}$ tends to zero $_{c_0}$ 0 as the human-to-environment contact rate tends to zero 0. This implies that Cholera will die out in the shortest time when control activities are taken to curb the human-to-environment contact which will eventually lead to reducing the spread of Cholera diseases. The research also revealed that at a hygiene-conscious rate, $_1$ 0.75, $_2$ 0.75 the effective reproduction number was minimal while the hygieneconscious rate, $_1$ 0.25, $_2$ 0.25 resulted in a high effective reproduction number. This implies that an increase in the hygieneconscious rate of Cholera will result in a low rate of Cholera infection among the human population. As the awareness rate is varies from 0.25 to 0.75 the effective reproduction number tends to zero. This is therefore indicating that the increase in awareness programmes (covering villages and market squares) will reduce greatly the spread of Cholera diseases.

TABLE OF CONTENTS

LIST OF FIGURES

Figure **Page**

LIST OF TABLES

Table Page

1.0 INTRODUCTION 1.1 Background to the Study

The cholera epidemic is a fatal waterborne disease-causing diarrhea, dehydration, and vomiting in an individual (WHO, 2019; WHO, 2018c). It is caused by a bacterium called *Vibrio cholerae*. Cholera is transmitted through ingesting contaminated drinks and food, contact with cholera patient's faeces, and touching vomit and corpse killed by the bacterium without using protective agents (WHO, 2019; WHO, 2018c; Panja, 2019). The incubation period of cholera is less than 24 hours to 5 days. The infection is frequently asymptomatic. Not more than 25% of the infected persons become symptomatic; of these, 10–20% experience severe disease (WHO, 2019). A continuous drastic loss of body fluids leads to dehydration, thus neglecting treatment as soon as the case occurs will accelerate the death of the infected person within hours (Lilje *et al*., 2015).

The disease has two modes of transmission: direct and indirect transmission. Direct transmission (human–human) is very uncommon as compared to indirect (environment–human) which occurs by ingesting contaminated food or water (Brachman and Abrutyn, 2009; CDC, 2004). An estimated 100,000–120,000 deaths are due to cholera every year in the world with only a small proportion being reported to World Health Organization (WHO) (Chaignat, 2014).

There are environmental factors that play a great role in the propagation of cholera infection. As *Vibrio cholerae* can move in the aquatic environment, every alteration in the hydrological cycle has a probability to affect the pathogenic concentration in water. The rain and its seasonal behaviour, droughts and floods, can increase or decrease the transmission process (Codeco, 2001).

Nevertheless, relevance has to be given to the environmental matrix in which the disease spreads into disease-free regions (Bertuzzo *et al*. 2008; Bertuzzo *et al*. 2010) 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 faeces (Mari *et al*., 2012). Finally, symptomatic infected individuals locally increase the bacteria concentration, which is, then, spread along the hydrological network.

Experiments for the spreading of infectious disease in individuals are unethical. Epidemiologists and other researchers use mathematical modelling and numerical simulation

for scientific understanding of the dynamics and preventive method of infectious disease, for determining sensitivities, changes of parameter values, and forecasting (Codeco, 2001;

Ayoade *et al*., 2018; Ratchford and Wang, 2019; Nyabadza *et al*., 2019; Eustace *et al*., 2018; Kwasi-Do *et al*., 2020). The models are based on cohorts, namely, susceptible, infected, and recovered, for individual populations incorporated with some preventive measures such as treatment, vaccination, chlorination, hygiene, and sanitation through education. Treatment is more recommended when cases occur, that is, a short-term plan for controlling and eradicating the disease. Education is a long-term plan for controlling and eradicating the disease, especially in creating awareness of water treatment, sewage removal, food safety, personal hygiene, and environment sanitation (Panja, 2019; Kumar *et al*., 2020).

This present work focuses on the development and analysis of a dynamic mathematical model that explicitly simulates the transmission mechanism of cholera by taking into account the role of control measures and the environment in the transmission of the disease.

1.2 Statement of the Problem

Cholera remains a significant threat to public health in the developing world, with cyclic outbreaks occurring twice per year in endemic areas (Jensen *et al*., 2006). For instance, on the 6th of September 2018, a cholera outbreak in Harare was declared by the Ministry of Health and Child Care (MoHCC) of Zimbabwe (WHO, 2018a). As of 15 September 2018, 3621 cumulative suspected cases, including 71 confirmed cases, and 32 deaths had been reported (case fatality ratio: 0.8%); of these, 98% (3564 cases) were reported from the densely populated capital Harare (WHO, 2018a). The City of Harare is facing a plethora of challenges, notably due to insufficient safe water supplies, frequent sewer pipe bursts, uncollected refuse and rampant illegal vending (WHO, 2018a). This has negatively impacted on public health in the city exposing residents to diarrhoeal disease outbreaks, an upsurge in

typhoid fever cases and sporadic outbreaks of cholera (WHO, 2018a). As of the year 2018, the disease has also claimed more than 67 lives in Zambia and Malawi did report some cases of cholera in Lilongwe.

In Nigeria, cholera is an endemic and seasonal disease, occurring annually mostly during the rainy season and more often in areas with poor sanitation, with the first series of cholera outbreaks reported between 1970 and 1990. Major epidemics also occurred in 1992, 19951996, and 1997. The Federal Ministry of Health reported 37,289 cases and 1,434 deaths between January and October 2010, while a total of 22,797 cases of cholera with 728 deaths and case-fatality rate of 3.2% were recorded in 2011. Outbreaks were also recorded in 2018 with the Nigeria Centre for Disease Control (NCDC) reporting 42,466 suspected cases including 830 deaths with a case fatality rate of 1.95% from 20 out of 36 States from the beginning of 2018 to October 2018 (NCDC, 2019). In 2016, a record of 768 cases of cholera was recorded in Nigeria (WHO, 2018b).

Thus, the cholera tragedy continues to devastate disadvantaged countries and communities. Hence, the needs for this research to analyse qualitatively the controlling mechanisms of cholera epidemics in a population by implementation of adequate preventive measures.

1.3 Significant of the Study

The dynamics of cholera involve multiple interactions between the human host, the pathogen, and the environment (Hethcote, 2000), which contribute to both direct (human-tohuman) and indirect (environment-to-human) transmission pathways.

Understanding the fundamental mechanism in the disease transmission is crucial for effective prevention and intervention strategies against a cholera outbreak. To this effect, mathematical modelling provides a unique approach to gain basic insights into the dynamics of infectious diseases. Therefore, by exploring the potential effects of disease-control strategies such as water chlorination, mathematical modeling can predict the dynamics of explosive epidemics often associated with cholera outbreaks.

Mathematical modeling enables us to characterize the general and specific behavior of the systems analytically and to understand which aspect contribute the most to the observed dynamics as well as making policy decisions for preventive measures and control strategies.

Hence, the need for this research work.

1.4 Justification of the Study

The application of differential equations in the transmission dynamics of infectious diseases have been extensively used in several papers (Bolarin and Bamigbola, 2014; Bolarin and Abdullahi, 2016; Ocheche, 2013; Wang and Modnak, 2011). The analysis of these models predicted and suggested several control strategies for the control and eradication of the infections (diseases).

Some of the control strategies being suggested were early detection and reporting, vaccination and mass immunization, therapeutic treatment and good sanitation practices. It is therefore important that adequate attention is paid to stopping the spread of such diseases by using effective control strategies and measures.

This research work will help to consolidate previous works on cholera and its complications, by incorporating hygiene consciousness and awareness programme driven by media

coverage to our system of differential equations to investigate their effects in control of the disease.

1.5 Scope and Limitation of the Study

This research work focuses on the mathematical modeling of the dynamics of Cholera transmission and control strategy taking into consideration the hygiene consciousness and awareness programme and it is limited to mathematical analysis of the disease.

1.6 Aim and Objectives of the Study

The aim of this research is to develop and analyze mathematical model of the dynamics of cholera transmission incorporating hygiene consciousness and awareness programme as

control strategies.

The objectives of the study are to:

1. Establish the existence of the disease-free equilibrium and endemic equilibrium of the model.

2. Analyse the condition for local stability of the disease-free equilibrium and endemic equilibrium.

3. Analyse the condition for global stability of the disease-free equilibrium and endemic equilibrium.

4. Carry out the sensitivity analysis of the model using Nigeria Demographic data.
5. Obtain the analytical solution of the model using the Homotopy perturbation method (HPM).
6. Carry out a graphical simulation of the solutions of the model equations using MAPLE 17 software.
7. Perform optimal control analysis of the model.

1.7 Definition of Terms

Analytical solution: This involves framing the problem in a well understood form and calculating the exact solution.

Basic Reproduction number: This is the average number of secondary cases of disease made by a typical infectious person (during his infectious period) in a wholly (completely) susceptible population.

Disease-Free Equilibrium Point: The disease-free equilibrium point is the steady-state solutions determined when there is no disease ($I = 0$).

Effective Reproduction number: This is the average number of secondary cases of disease made by a typical infectious person (during his infectious period) in a wholly (completely) susceptible population where control measure such as treatment, vaccination, quarantining,

etc.

Equilibrium: This is the state of rest of a body. A state of balance between opposing forces or actions that is either static (as in a body acted on by forces whose resultant is zero) or dynamic (as in a reversible chemical reaction when the rates of reaction in both directions are equal).

Endemic Equilibrium Point: The endemic equilibrium points are steady-state solutions determined when $I \neq 0$, and there exist at least one endemic equilibrium point.

Global Stability: This means that the attracting basin of trajectories of a dynamical system is either the state space or a certain region in the state space which is the defining region of the state variables of the system.

Homotopy Perturbation Method: This is a semi-analytical technique for solving linear as well non-linear ordinary/partial differential equations.

Incidence: This is the occurrence, rate or frequency of a disease, crime or other undesirable thing, for example, an increase incidence of cholera.

Infected: This is the compartment used for persons who have cholera infections. **Local Stability:** If points near an equilibrium tend to move towards the equilibrium over time, the equilibrium is said to be locally stable.

Mathematical Model: A mathematical model is a description of a system using mathematical concepts and language.

Numerical simulation: This is a calculation that is run on a computer following a program that implements a mathematical model for a physical system. Numerical simulation are required to study the behavior of systems whose mathematical models are too complex to provide analytical solutions as in most nonlinear systems.

Optimal control: This is a condition of dynamics systems that satisfy design objectives and is achieved with control laws that execute following defined optimality criteria.

Prevalence: This is the proportion of a particular population found to be affected by a medical condition at a specific time.

Recovered: These are group of persons who have been treated and recovered from the cholera illness.

Sensitivity Analysis: This is the study of how the uncertainty in the output of a mathematical model or system can be divided and allocated to different sources of uncertainty in its inputs.

Stable equilibrium: This is the state of a system such that when slightly moved tends to come back to its original state of rest or in other word, an equilibrium is considered stable if the system always returns to it after small disturbance.

Susceptible: These are individuals who are not yet infected but can still be infected.

Unstable equilibrium: If the system moves away from the equilibrium after small disturbance, then the equilibrium is unstable.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Overview of Cholera Diseases

Cholera is a disastrous water-borne infectious disease that is caused by the bacterium vibrio cholera (V. cholera). It is a very serious problem in many developing countries due to inadequate access to safe drinking water supply, improper treatment of reservoirs and improper sanitation.

In 2019, the Norwegian Refugee Council (NRC) reported that the overcrowded displacement of camps coupled with a lack of basic sanitation facilities and hygiene will cause another cholera outbreak in northeast Nigeria if action is not taken to prevent it and if the camps are not decongested and sanitation facilities improved, cholera will inevitably return, and vulnerable displaced people will bear the brunt of the epidemic again (NCDC, 2019).

For instance, 466 people are sharing one latrine at one of the displacement camps in the state of Borno, according to the Humanitarian Office for the Coordination of Humanitarian Affairs (OCHA). This is nine times above the agreed humanitarian standards, which is set at 50 people per latrine in emergency situations. As a result of lack of sanitation, people choose to defecate in the open, exacerbating an already vulnerable situation and increasing the likelihood of the spread of disease (NCDC, 2019).

In 2019, about 400,000 people need emergency shelter in the northeast Nigeria, just over 2,288 people have been reached with 24% safe water been made accessible to the total number of people in need (NCDC, 2019).

2.1.1 Signs and symptoms of cholera disease

The vast majority of the time, the symptoms of cholera is hard to distinguish from diarrhea caused by many other possibilities. The Bacteria will be present in an infected person's feces for 7 - 14 days, however (WHO, 2014), around 1 in 10 cases display the characteristic signs and symptoms of cholera. They are extreme diarrhea, nausea and vomiting, and dehydration. With diarrhea, the infected person may lose as much as a litre of fluid an hour. The Diarrhea from cholera has a pale, milky Appearance, and for this reason, diarrhea from cholera is generally called "rice - water stool." Nausea and vomiting may last for several hours at a time. Dehydration Causes electrolyte imbalance, which can lead to muscle spasms and shock, specifically hypovolemic shock. Signs and symptoms for cholera dehydration also include irritability, lethargy, sunken eyes, dry mouth, extreme thirst, dry and shriveled skin, little or no urine output, low blood pressure, and an irregular heartbeat (Mayo Clinic Staff, 2014).

2.1.2 Key Tests for Identification of cholera

The main test for identification for V. cholera is serologic identification, searching for the presence of 01 serotype antigens. This Can be shown by using an agglutination test. Most serotypes of cholera that do not Cause epidemic or pandemic outbreaks also do not produce cholera toxin (CDC & P, 2014). V. cholera samples are generally taken from stool samples of infected persons. The stool will have the Appearance of "rice - water stool (Finkelstein, 2014). It will have a white, pasty layer similar to water after it has been used to boil rice. The stool will also have a fishy odor. The above are quick methods for identification, which is important since cholera can turn deadly very rapidly. Another Test is noting whether the serotype produces cholera toxin. Only the three pathogenic serotypes will (CDC & P, 2014).

2.1.3 Control/Treatment of cholera diseases

Anyone thought to be suffering from cholera should be treated with Oral Rehydration Solution (ORS), which contains salts, sugar, and electrolytes. It is dissolved in water and administered orally throughout the period of infection. Rehydration is the key to overcoming cholera (CNN Library, 2014).

2.1.4 Prevention/Vaccines against cholera diseases

Cholera is a disease spread primarily by poor sanitation. The Best way to combat cholera is to improve waste management, water treatment, and food preparation. Currently, there are three vaccines for cholera available. One is Dukoral, which is World Health Organization

(WHO) prequalified, and the other is ShanChol and Euvichol, which is licensed in India. Two doses of ShanChol and Euvichol provide protection against cholera for three years while a single dose provide short-time protection. The two are currently available for mass vaccination campaign through the global OCV stockpile, which is supported by Gavi, the vaccine alliance. Dukoral is the third vaccine available. The vaccines are both administered orally, two times (six weeks apart). Their effectiveness lasts for about two years (Mayo Clinic Staff, 2014). The Vaccines may take several weeks for their benefits to begin taking shape in a person, so vaccination should not replace standard prevention and control measures (CDC & P, 2013).

Currently, the research into cholera vaccines is still in progress. For example to see if one dose of vaccine is sufficient instead of two doses.

2.1.5 History of cholera diseases

The Word cholera comes from the Greek 'khole' Meaning 'illness from bile.' The First notable reports specifically referencing cholera come from John Snow of London, England, and Filippo Pacini of Florence, Italy. Both Reports come from 1854. Pacini was the first to identify *V. Cholera* as the ctiologic agent of cholera, though his discovery was not widely known until Robert Koch Publicized his own independent research in1884. John Snow did not discover the cause of cholera, but he did impart knowledge on how to stop a local outbreak (Davis, 2014). There have been seven epidemic outbreaks of cholera since 1817. The First six outbreaks were caused by the classical 01 biotype. The Seventh outbreak was caused by the ElT or 01 Biotype (Finkelstein, 2014).

2.2 Review of Related Works

In an effort to gain deeper understanding of the complex dynamics of cholera, several mathematical models have been published. For example, Codeco (2001) proposed a model that explicitly accounted for the environmental component, i.e., the *V. cholera* concentration in the water supply, into a regular SIR epidemiological model. The incidence (or, the infection force) was modeled by a logistic function to represent the saturation effect. Hartley *et al*. (2006) extended Codeco's work to include a hyper infectious state of the pathogen, representing the "explosive" infectivity of freshly shed *V. cholerae*, based on the laboratory observations (Merrell *et al*., 2002). This model was rigorously analyzed in (Liao and Wang,

2011). Joh *et al*. (2009) modified Codeco's model by a threshold pathogen density for infection with a careful discussion on human environment contact and in-reservoir pathogen

dynamics. Mukandavire *et al*. (2011) proposed a model to study the 2008–2009 cholera outbreaks in Zimbabwe. The model explicitly considered both human-to-human and environment-to-human transmission pathways. The results in their 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. Moreover, Tien and Earn (2010) published a water-borne disease model which also included the dual transmission pathways, with bilinear incidence rates employed for both the environment-to-human and human-to-human infection routes. No saturation effect was considered in Tien and Earn's work. A rigorous global stability analysis was conducted in (Tian and Wang, 2011) for many of the aforementioned models. In addition, Neilan *et al*. (2010) modified the cholera model proposed by Hartley *et al*. (2006) and added several control measures into the model. They consequently analyzed the optimal intervention strategies and conducted numerical simulation based on their model. No human-to-human infection route is considered in their work.

Liao and Wang (2011) considered three types of controls: vaccination, therapeutic treatment (including hydration therapy, antibiotics, etc.), and water sanitation but he did not incorporate the role of education control strategy in their model, also they did not consider a logistic growth of *vibrio* cholera.

Ochoche (2013) developed a mathematical model for the control of cholera transmission dynamics using water treatment as a control strategy. The model was designed by dividing the system into compartments leading to corresponding differential equations. The model was built on the assumption that cholera is contracted only through the ingestion of contaminated water. Conditions were derived for the existence of the disease free and endemic equilibrium. He proved that the disease free equilibrium is locally asymptotically stable under prescribed conditions on the given parameters.

Sani *et al.* (2013) worked on a deterministic mathematical model on cholera dynamics and some control strategies. In their study, a system of four differential equations with two control measures which are therapeutic treatment and sanitary measures were considered.

Madubueze *et al.* (2015) considered the bifurcation and stability analysis of the dynamics of cholera model with controls. The existence of backward bifurcation is investigated in their work and the numerical simulation performed revealed that combine control measures will help to reduce the spread of cholera in the human population. Pransenjit and Debasis (2012) worked on the qualitative analysis of a cholera bacteriophage model. In their work, they concluded that by using phage as a biological control agent in endemic areas, one may also influence the temporal dynamics of cholera epidemic while reducing the excessive use of chemicals. Adewale *et al.* (2015) worked on the mathematical analysis of the effect of growth rate of vibrio-cholera in the dynamical spread of cholera. In their work, they developed a mathematical model that incorporated phage virus which serves as a biological control of cholera bacteria in the population; they concluded that phage virus plays a vital role in reducing the spread of cholera in the population.

Wang and Modnak (2011) presented and analyzed a cholera epidemiological model with control measures incorporated. This model was extended from the one proposed in Mukandavire *et al*. (2011) by including the effects of vaccination, therapeutic treatment, and water sanitation. Equilibrium analysis is conducted in the case with constant controls for both epidemic and endemic dynamics. Optimal control theory was applied to seek costeffective solution of multiple time-dependent intervention strategies against cholera outbreaks. Their model equations are:

$$\frac{dS}{dt} = \Lambda N - \beta_h SI - \beta_e S - (\nu_t + \mu)S + \gamma B + \kappa K \quad (2.1)$$

$$\frac{dI}{dt} = \beta_h SI + \beta_e S - (\alpha_t + \mu + \delta)I \quad (2.2)$$

$$\frac{dR}{dt} = (\alpha_t + \mu)I - (\mu + \nu_t)R \quad (2.3)$$

$$\frac{dB}{dt} = \delta I - (\mu + \gamma + w_t)B \quad (2.4)$$

Edward and Nyerere (2015) formulated a mathematical model that captures some essential dynamics of cholera transmission with public health educational campaigns, vaccination, sanitation and treatment as control strategies in limiting the disease. The reproduction numbers with single and combined controls are computed and compared with each other to assess the possible community benefits. Numerical simulation shows that in a unique control strategy, treatment yields the best results followed by education campaign, then sanitation and vaccination being the last. Furthermore, they noted that the control of cholera is very much better when they incorporated more than one strategy, in two controls the results were

better than one strategy, and in three control strategies the results were far better than in two control strategies. Further simulations with all four interventions showed the best results among all combinations attained before. They performed sensitivity analysis on the key parameters that drive the disease dynamics in order to determine their relative importance to disease transmission and prevalence. Their model equations are:

$$\frac{dS}{dt} = \Lambda_1(1-\varepsilon_e)S\left(\frac{B}{K}\right) - \mu R - \mu_h S \quad (2.5)$$

$$\frac{dI}{dt} = \Lambda_1(1-\varepsilon_e)S\left(\frac{B}{K}\right) - \delta I - \mu_h I \quad (2.6)$$

$$\frac{dR}{dt} = \delta I - \mu S - \mu_h R \quad (2.7)$$

$$\frac{dB}{dt} = b\left(1-\frac{B}{K}\right)B - \mu(1-\varepsilon_e)I - \mu_p - w \quad (2.8)$$

Yang (2017) proposed two differential equation-based models to investigate the impact of awareness programs on cholera dynamics. The first model represents the disease transmission rates as decreasing functions of the number of awareness programs, whereas the second

model divides the susceptible individuals into two distinct classes depending on their awareness/unawareness of the risk of infection. He studied the essential dynamical properties of each model, using both analytical and numerical approaches. He found that the two models, though closely related, exhibit significantly different dynamical behaviors. Namely, the first model follows regular threshold dynamics while rich dynamical behaviors such as backward bifurcation may arise from the second one. His results highlighted the importance of validating key modeling assumptions in the development and selection of mathematical models toward practical application. His model equations are:

Model 1:

$$\frac{dS}{dt} = \Lambda N - \beta_1 M SI - \beta_2 M S - \frac{B}{K} S - \mu R \quad (2.9)$$

$$\frac{dI}{dt} = \beta_1 M SI + \beta_2 M S - \frac{B}{K} I - (\mu + \gamma) I \quad (2.10)$$

$$\frac{dR}{dt} = \gamma I - \frac{B}{K} R - \mu R \quad (2.11)$$

$$\frac{dB}{dt} = \alpha M I - \mu B \quad (2.12)$$

$$\frac{dM}{dt} = \square\square\square\square I \square\square\ M \qquad (2.13)$$

Model 2: $dS_u\ N\ {}_1S_uI\ {}_2S_u B \square\square S_uM\ \square\square 1\square\ p\square\square R\ \square\ kS_a\ \square\square\ S_u$

(2.14)

$\square\square$

$B\ \square\ \ dt\ \ K$

$dS_a\ S_uM\ \square\square_1\square_1S_aI\ \square\square_2\square_2S_a B\ \ \square\ p\square R\square\square k\ \square\square\square S_a \qquad (2.15)$

$\square\square$

$dt\ \ B\square\ K$

$dt\ \square\square_1\square S_u\ \square\square_1S_a\ \square I\ \square\square_2\square S_u\ \square\square_2S_a\ \square_{B\ \square\ K}\ \square\square\square\square\square I$

(2.16)

$$\frac{dR}{dt} = \square\square I\ \square\square\square\square\square\ R \qquad (2.17)$$

$$\frac{dB}{dt} = \square\square I\ \square\square\ B \qquad (2.18)$$

$$\frac{dM}{dt} = \square\square\square\square I\ \square\square\ M \qquad (2.19)$$

2.3 Summary of Review and Gap to Fill

In reviewing the above literatures, we observed that several works have been done on the dynamics of Cholera and control strategy. Some authors worked on the dynamics of Cholera and control strategy without considering the hygiene conscious compartment. Others concentrated on vaccination, therapeutic treatment and water sanitation but ignored awareness programme.

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In view of the above, this research work modified the models of Wang and Modnak (2011); Edward and Nyerere (2015); Yang (2017) by adding:

(i) hygiene conscious compartment

(ii) and awareness programme (covering villages and market squares) compartment as a control strategy and also consider that *vibrio* bacteria grow logistically. So we have four types of controls: vaccination, therapeutic treatment (including hydration therapy, antibiotics, just to mention a few), water sanitation and awareness programme.

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 Mathematical Formulation

The cholera model developed in (Wang and Modnak, 2011; Edward and Nyerere, 2015; Yang, 2017) is a combined system of human populations and the environmental component (SIR-B), with the environment-to-human transmission represented by a logistic (or Michaelis-Menten type) function and the human-to-human transmission by the standard mass action law. We now extend these models by adding vaccination, treatment, hygiene consciousness, awareness programme, and water sanitation.

The total human population is divided into six compartments depending on the epidemiological status of individuals. These compartments include: Susceptible Unaware individuals, $S_u(t)$, Susceptible Aware individuals, $S_a(t)$, Hygiene Conscious individuals, $H(t)$, Symptomatically Infected individuals, $I(t)$, Vaccinated individuals, $V(t)$ and Recovered individuals, $R(t)$. We assume that the total population is non-constant, which is a reasonable assumption for a relatively short period of time and for low-mortality diseases such as cholera. The concentration of the *vibrios* in the environment (that is contaminated water) is denoted by $B(t)$ and the number of the awareness programs driven by disease prevalence and media coverage is denoted by $M(t)$. Vaccination reduces the risk of infection by a factor $f \in [0,1]$ and the efficacy of the vaccine is $(1-f)$. Let a constant Λ stands

for the number of newborn babies and immigrants into the population, then f are recruitment rate into vaccinated class while $(1-f)\Lambda$ are the recruitment rate into susceptible class.

Individuals in the S_a compartment have lower chances of contracting the disease than those in S_u. Unaware individuals may switch to the aware group due to the involvement with the awareness programmes, and aware individuals may lose the awareness of cholera after a period of time. Furthermore, the susceptible population increases due to the incoming of newborns and immigrants, loss of vaccination and loss of immunity of individuals at the rates $(1-f)\Lambda$, θ_1 and respectively. On the other hand, the susceptible population decreases due to the infection and vaccination strategy.

Concentration of *Vibrio Cholerae* in food and water that yields 50% chance of catching cholera disease is denoted by K, γ is the rate at which infected people recovered from cholera disease, w is the loss rate of *Vibrio Cholerae* in the environment, ξ is the contribution of each infected person to the population of *Vibrio Cholerae* in the environment.

The total human population size $N = S_u + S_a + E + I + V + R$. For the unaware compartment S_u, the direct and indirect transmission rates are represented by β_1 and β_2, respectively, which are assumed to be constant at all times. For the aware compartment S_a, the disease transmission rates are lower and are given by $b\beta_1$ and $b\beta_2$, respectively, where $0 < b < 1$

$0 < b_1, b_2 < 1$. For the hygiene conscious compartment H, the disease transmission rates are lower and are given by $c_1\beta_1$ and $c_2\beta_2$, respectively, where $0 < c_1, c_2 < 1$. The human contribution rate Λ is also assumed to be constant. Unaware individuals gain knowledge of the disease and enter the S_a class through interacting with the awareness programs at a rate

σ. Meanwhile, aware individuals become unaware of the disease over time and enter the S_u class at a rate q. In addition, recovered individual go back to the S_a and S_u classes, at the fractions p and $(1-p)\gamma$, respectively. Hygiene conscious individual go back to the S_a and S_u classes, at the fractions d and $(1-d)\gamma$, respectively. Also, vaccinated individual go back to the S_a and S_u classes, at the fractions r and $(1-r)\theta$, respectively.

3.1.1 Model assumptions

The formulation of our model is guided by the following assumptions:

1. The total population of individuals is not constant.

2. Controls are implemented continuously.

3. Vaccination is introduced to the susceptible population at rate of θ_2.

4. Therapeutic treatment is applied to the infected individuals at a rate of a.

5. Water sanitation leads to the death of *vibrios* at a rate of w.

6. The number of the awareness programs driven by disease prevalence and media coverage covered both villages and market squares.

7. On recovery, there is temporary immunity.

The above description leads to the compartmental diagram in Figure 3.1. The parameters and variables indicated in Figures 3.1 are described in Tables 3.1 and 3.2.

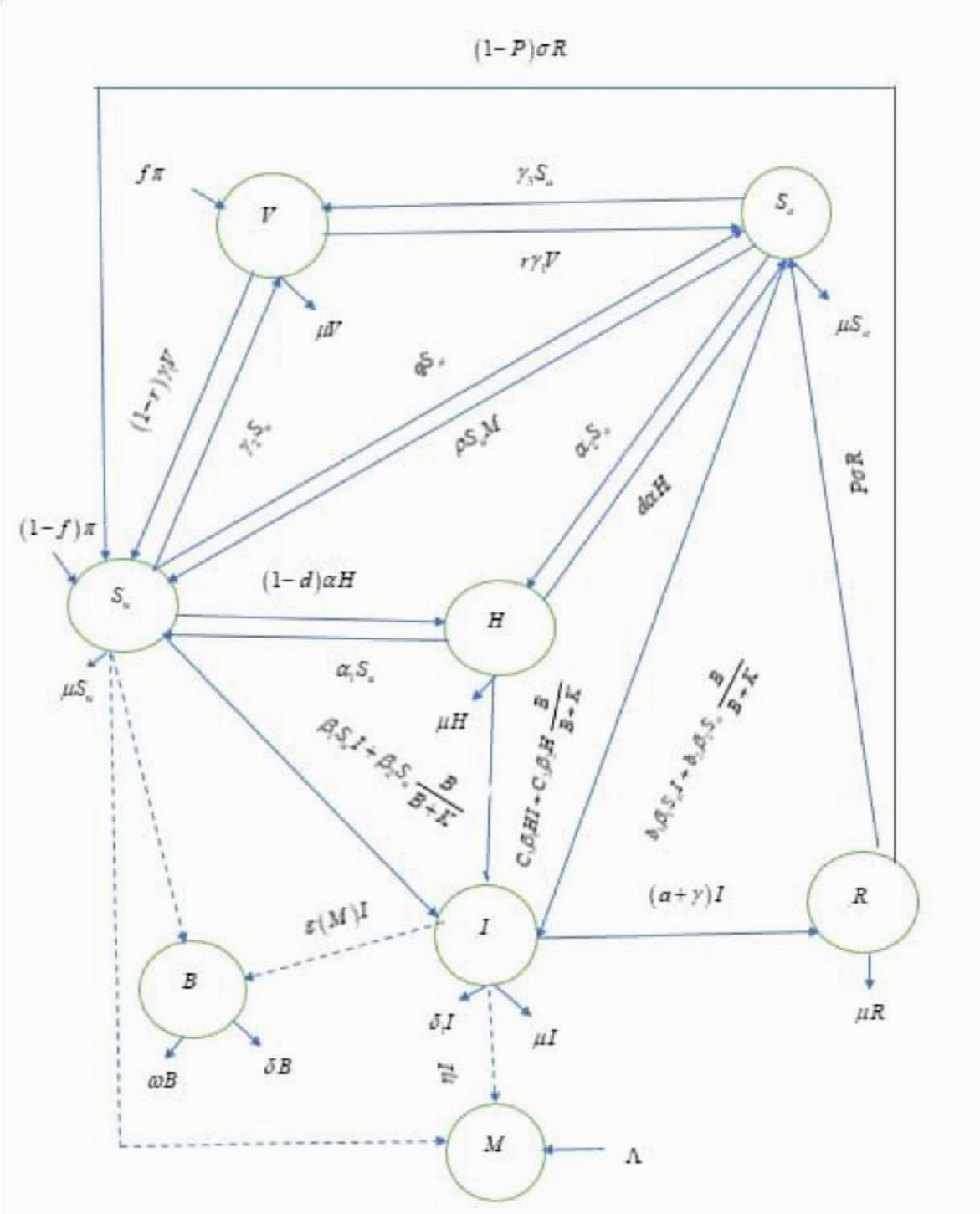


Figure 3.1: Schematic Diagram of the Model

Table 3.1: Description of Variables

Variables	Definitions
$S_u(t)$	Susceptible unaware individuals
$S_a(t)$	Susceptible aware individuals
$H(t)$	Hygiene conscious individuals
$I(t)$	Symptomatically infected individuals
$V(t)$	Vaccinated individuals
$R(t)$	Recovered individuals
$B(t)$	The concentration of the *vibrios* in the environment (that is contaminated water)
$M(t)$	Number of the awareness programs driven by disease prevalence and media coverage

Table 3.2: Description of parameters

Parameters	Definitions
$\Lambda_1(1-f)$	Recruitment rate into the susceptible unaware class
α_2	Rate at which the susceptible unaware humans are vaccinated
α_3	Rate at which the susceptible aware humans are vaccinated
α_1	Vaccination loss rate
γ	Rate at which the recovered humans are susceptible
$f\Lambda$	Recruitment rate into the vaccinated class
μ	Natural death rate

δ_1 Disease induced death rate

w Rate at which water sanitation leads to the death of *vibrios*

N Total population

β_1 Human to human transmission rate

β_2 Environm ent to human transmission rate

α Infected human shedding rate

γ_1 Rate at which the susceptible unaware humans are hygiene conscious

γ_2 Rate at which the susceptible aware humans are hygiene conscious

ε Hygiene conscious loss rate

b_1 Proportion of direct disease transmission rate for the aware compartment

b_2 Proportion of indirect disease transmission rate for the aware compartment

c_1 Proportion of direct disease transmission rate for the hygiene conscious compartment

c_2 Proportion of indirect disease transmission rate for the hygiene conscious compartment

d Proportion of hygiene conscious class r

Proportion of vaccinated individual p Proportion of recovery class

θ Awareness programme stimulated rate

μ Natural death rate of *vibrios* from environment

η Growth rate of number of awareness programmes

a	Therapeutic treatment rate
K	Carry capacity for *V. cholera*
$\square$	Recovery rate of infected humans
$\square$	Awareness programme decay rate
$\square$	Rate at which susceptible unaware humans are aware
q	Rate at which susceptible aware humans loss their awareness

3.1.2 The model equations

From the assumptions, descriptions and the compartment diagram in Figure 3.1, we formulate the following system of differential equations:

$$\square \frac{dS_u}{dt} \square 1\square f \square\square\square_1 S_u I \square\square_2 S_u B \square B K \square\square S_u M \square\square 1\square p\square\square R \square qS_a \square\square 1\square r\square\square \square \quad (3.1)$$

$$S_{u0} \square 1\square d\square\square H \square\square_2 \square\square_1 \square\square\square S_u, S_u \square 0\square\square$$

$$\frac{dS_a}{dt} S_u M \square b_1\square_1 S_a I \square b_2\square_2 S_a B \square p\square R \square r\square_1 V \square d\square H \square \quad (3.2)$$

$$\frac{dH}{dt} \square q \square\square_3 \square\square_2 \square\square\square S_a, \quad S_a \square 0\square\square S_{a0}$$

dt □□$_1S_u$ □□$_2S_a$ □c_1□$_1HI$ □c_2□$_2H$ B □ K □□□□□□H,

—— ——— H□0□□ H_0 (3.3)

dI B

□□$_1$□S_u □b_1S_a □ c_1H□I □□$_2$□S_u □b_2S_a □ c_2H□ □□a □□□□$_1$ □□□I, I□0□□

B □ K

I_0(3.4) dt

—

———

dV

□ f□□□$_2S_u$ □□$_3S_a$ □□□$_1$ □□□V,

—— V□0□□ V_0 (3.5)

dt

dR

□ □□□ a□I □□□□□□R,

—— R□0□□ R_0 (3.6)

dt

dB

□□I □□□□ w□B,

—— B□0□□ B_0 (3.7)

dt

dM

□□□□I □□M, M□0□□ M (3.8)

——

dt 0

3.2 Basic Properties of Model

For the special case when the rates of all three control parameters are positive constants, i.e. $\alpha_2 > 0$, $a > 0$, $w > 0$, we will examine the positivity and invariant region of the solutions of equations (3.1) – (3.8).

3.2.1 Positivity of solution

Since the model monitors human population, we need to show that all the state variables remain non-negative for all times.

Theorem 3.1: Let

$$\Omega = \{(S_u, S_a, I, R, V, H, B, M) \in \mathbb{R}^8_+ : S_u(0) \geq 0, S_a(0) \geq 0, I(0) \geq 0, R(0) \geq 0,$$

$$V(0) \geq 0, H(0) \geq 0, B(0) \geq 0, M(0) \geq 0\}$$

then the solutions $(S_u(t), S_u(t), I(t), R(t), V(t), H(t), B(t), M(t))$ of the system of equations

(3.1) – (3.8) are positive for all $t > 0$. **Proof:**

Consider equation (3.1)

$$\frac{dS_u}{dt} = \Lambda(1-f) + \alpha_1 S_u I + \alpha_2 S_u B + \beta_B K - \alpha S_u M - (1-p)\beta_R R + qS_a - (1-r)\alpha_1 V \quad (3.9)$$

$$\frac{dS_u}{dt} \geq -\alpha S_u M$$

i.e. $\frac{dS_u}{S_u} \geq -\alpha dH$, $S_u(0) > 0$ $\Rightarrow S_u \geq \Lambda(1-f) + \alpha_2 \alpha_1 S_u$

(3.10)

$$\frac{dS_u}{dt} \geq \frac{dS}{dt}$$

$$\frac{dS_u}{dt} = \alpha_2 - \alpha_1 S_u - (1-f)\beta_1 S_u \quad (3.11)$$

Solving, we have $S_u = \frac{\alpha_2}{\alpha_1 + (1-f)\beta_1} + ce^{-(\alpha_1 + (1-f)\beta_1)t}$ (3.12)

$$S_u = \frac{\alpha_2}{\alpha_1 + (1-f)\beta_1}$$

Taking initial condition, we get

$$c = S_{u0} - \frac{\alpha_2}{\alpha_1 + (1-f)\beta_1}$$

$$S_u = \frac{\alpha_2}{\alpha_1 + (1-f)\beta_1} + \left(S_{u0} - \frac{\alpha_2}{\alpha_1 + (1-f)\beta_1}\right)e^{-(\alpha_1 + (1-f)\beta_1)t} \quad (3.13)$$

Consider equation (3.2) $\frac{dS_a}{dt} = S_u(\beta_1 S_a + \beta_2 S_a) - \gamma S_a$

$$\frac{dS_a}{dt} = \alpha_3 - \alpha_2 S_a, \quad S_a(0) = S_{a0}$$

i.e. $\frac{dS_a}{dt} = \alpha_3 - \alpha_2 S_a$

$$\frac{dS_a}{\alpha_3 - \alpha_2 S_a} = dt \quad (3.15)$$

$$\frac{dS_a}{\alpha_3 - \alpha_2 S_a} = dt \quad (3.16)$$

Solving, we have

$$S_a(t) = c_1 e^{(-q-\beta_3-\beta_2-\mu)t} \tag{3.17}$$

Taking initial condition, we get

$$S_a(t) = S_{a0} e^{(-q-\beta_3-\beta_2-\mu)t} \geq 0 \tag{3.18}$$

Consider (3.4)

$$\frac{dI}{dt} = \beta_1(S_u + b_1 S_a + c_1 H)I + \beta_2(S_u + b_2 S_a + c_2 H)B - KI \tag{3.19}$$

$$\alpha_a = \alpha_1, I(0) = I_0$$

i.e.

$$\frac{dI}{dt} = (\alpha_a - \alpha_1)I \tag{3.20}$$

$$\frac{dI}{I} = (\alpha_a - \alpha_1)dt \tag{3.21}$$

Solving, we have

$$I(t) = c_2 e^{(\alpha_a - \alpha_1)t} \tag{3.22}$$

Taking initial condition, we get

$$I(t) = I_0 e^{(\alpha_a - \alpha_1)t} \geq 0 \tag{3.23}$$

Similarly, it can be shown that

$$R(t) = R_0 e^{(\cdots)t} \geq 0 \tag{3.24}$$

$$f = \frac{\quad}{\quad} \qquad f = \cdots_1 \cdots_t \quad 0 \tag{3.25}$$

$$V(t) = \cdots_1 \cdots V_0 = \cdots_1 \cdots e$$

$$\frac{\quad}{\quad}$$

$$(t) = H_0 e^{(\cdots)t} \qquad H\,0 \tag{3.26}$$

$$(t) = B_0 e^{(\cdots)t} \qquad B\,0 \tag{3.27}$$

$$(t) = \frac{}{v} \quad M_0 \quad e^{vt} \qquad M\,0 \tag{3.28}$$

$$\frac{}{} \qquad \frac{}{}$$

for all time $t \geq 0$.

3.2.2 Invariant region

Theorem 3.2: Let $(S_u(t), S_a(t), I(t), R(t), V(t), H(t), B(t), M(t))$ be the solution of system (3.1)

– (3.8) with initial conditions $(S_u \quad 0), S_a$ $(0), I(0), R(0), V(0), H(0), B(0), M(0))$. The compact

$$\frac{\quad}{S_u, S} \quad \frac{,I}{,V, a} \quad \frac{}{,R} \quad H,B,M \quad \cdots \quad ,W_H \quad \quad ,W_B \quad \quad ,\cdots$$

set, □□□□ □□1 □□□ □□1 □□□□□□ w□ □□ is positively

□ □W_M □ 1v □□□□ □□ □□□□1 □□□□□ □□□ □□

invariant and attract all solution in □^{8}□ .

Proof: We follow the proof given by Mushanyu *et al*. (2018). Consider,

$$W\square t\square\square \square W_H, W_B, W_M \square\square \square S_u, S_a, I, R, V, H, B, M\square. \tag{3.29}$$

The time derivative of $W\square t\square$ is given by

$$dW\square t\square \square \square dW_H, dW_B, dW_M \square\square\square\square dS_u \square dS_a \square dI \square dR \square dV \square dH, dB, dM \square \square$$

$$dt \quad \square dt \quad dt \quad dt \square \square dt \quad dt \quad dt \quad dt \quad dt \quad dt\ dt\ dt \square \tag{3.30}$$

$$\square \square\square\square\square W_H \square\square_1 I, \square I \square\square\square\square w\square B, \square\square\square I \square vM\square$$

This gives

$$dW_H \; W\,I\,W\,W\,0 \text{ for } W \square \tag{3.31}$$

□□□□ □□ □□□□ □□ □

□

$$\frac{dW_B}{dt} = I\,\nu_B\,w_B W_B - \mu_B W_B - w_B W_B = 0 \text{ for } W_B \tag{3.32}$$

$$\frac{dW_M}{dt} = I\,\nu_M\,W_H - \nu_M W_M - \mu_M W_M = 0 \text{ for } W_M = 1 - \nu \tag{3.33}$$

with $\mu_1 - \mu_H - \mu_B > 0$ and $\mu_B + w_B > 0$.

From (3.31) – (3.33), we have $\frac{dW}{dt} \geq 0$ which implies that Ω is a positive invariant set. We also note that by solving (3.31) – (3.33), we have

$$0 \leq W_H, W_B, W_M \leq \frac{\mu_1}{\mu_H + \mu_B + \mu_M + w_B} + \varepsilon, \quad \varepsilon > 0 \tag{3.34}$$

as $t \to \infty$ and hence Ω is an attractive set.

3.3 Model Analysis

The model system (3.1) – (3.8) is analysed qualitatively to get insights into its dynamical features which give better understanding of the impact of control strategies on the transmission dynamics of *Vibrio* cholera virus.

3.3.1 Disease free equilibrium (DFE)

The disease-free equilibrium (DFE) point is state where there is absence of cholera infection in the population. The disease free equilibrium of model system (3.1) – (3.8) is obtained by setting

$$\frac{dS_u}{dt} = \frac{dS_a}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = \frac{dV}{dt} = \frac{dH}{dt} = \frac{dB}{dt} = \frac{dM}{dt} = 0, \quad (3.35)$$

and in the absence of disease, $I = B = 0$ so that:

From equation (3.5), we have

$$V = \frac{\Lambda_1 f}{\mu_1 + \mu_2} S_u^0 = \frac{\Lambda_1 f}{\mu_1 + \mu_3} S_a^0 = 0 \quad (3.36)$$

and from equation (3.8),

we have

$$M = 0 \quad (3.37)$$

and

from (3.3),

H_0 ... $BCAC_{11}$ ... $BA_{11}CC$ ... $CA_{11}BB_{00}$ ... (3.38)

$CBAB_{11}$

From (3.6), we have

$$R^0 = 0 \tag{3.39}$$

Substituting (3.36) – (3.39) into (3.1) – (3.2) and solve simultaneously yields

S_{u0} ... $BCAC_{11}$ ... $BA_{11}CC$

S_{a0} ... $CBAB_{11}$ ... $CA_{11}BB_{00}$

H_0 ... BAC_0C_{11} ... $AB_{11}CC$ ... $CA_{11}BB_{00}$ ... , (3.40)

$CBAB_{11}$

V_0 □□□□ f □ □□ □ □$_2$ □ □□□ BAC_0C_{11}□□$AB_{11}CC$ □□□□□ □□$_1$□□$_3$□□□□□□$CBAB_{11}$ □□$CA_{11}BB_{00}$ □□□□□□□□□□ □ □

□ □$_1$□□ □$_1$□□ □

$$M_0 \square \square \square \square v \square$$

where

A □ □□□□□□□□$_1$ □□1□ f □□□□, B_0 □ □□□□□□□□□$_1$ □□$_2$ □□□□□□$_1$□□$_1$ □□□□□, C □ □□□□□□□□□$_1$ □□$_3$ □□□□□□$_2$□□$_1$ □□□□□, A_1 □ rf□v□$_1$□□□□□, B_1 □ □□□□□□□v□ad□$_1$ □ r□$_1$□$_2$□□□□□□$_1$ □□□□, C_1 □ □□□□□□□□r□$_1$□$_3$ □□□$_2$ □□□□□$_1$ □□□□□□□□□d□$_2$ □□□$_3$ □ q□□□□□□□□

Hence DFE is

□$S_{v0},S_{a0},I_0,R_0,V_0,H_0,B_0,M_0$□□

□ □□ f□ □ □$_2$ □□ AC_1 □ A_1C □□□□□ □ □

□

□□ □ AC_1 □ A_1C □□, □□□ □ $CBAB_{11}$ □□$CA_{11}BB_{00}$ □ □□□, 0, 0, □□ □□□$_1$□□$_3$□□□□ AB□□$_{11}$□□□ A□$_1B$□□□$_0B$□□ $_0C_1$ □ B_1C □□ □□□,□ □ □ □ (3.41)

□□□ B_0C_1 □ B_1C □□

□ □□ □□$_1$ □□□□□□CB_1 □C_1B_0 □□ □□ □

□

□□

□ □□□□□ □□□□$_1$□□□□ AC_1 □ A_1C □□□ □$_2$ □ □□$CBAB_{11}$ □□$CA_{11}BB_{00}$ □□□□□□□□□, 0, □ v □ □ □ □

□ B_0C_1 □ B_1C □□ □□□□□□

3.3.2 Basic reproduction number, R_0

The basic reproduction number denoted by R_0 is the average number of secondary infections caused by an infectious individual during his or her entire period of infectiousness (Diekmann *et al.*, 1990). The basic reproduction number is an important non-dimensional quantity in epidemiology as it sets the threshold in the study of a disease both for predicting its outbreak and for evaluating its control strategies. Thus, whether a disease becomes persistent or dies out in a community depends on the value of the reproduction number, R_0.

Furthermore, stability of equilibria can be analyzed using R_0. If $R_0 < 1$ it means that every infectious individual will cause less than one secondary infection and hence the disease will die out and when $R_0 > 1$, every infectious individual will cause more than one secondary infection and hence the disease will invade the population. A large number of R_0 may indicate the possibility of a major epidemic. For the case of a model with a single infected class, R_0 is simply the product of the infection rate and the mean duration of the infection.

Since the infection components in this model are I and B, then from equation (3.4) and (3.7)

$$F_i = \begin{bmatrix} \beta_1 S_u & \beta_1 S_a & c_1 H \\ \beta_2 S_u & \beta_2 S_a & c_2 H \\ 0 & 0 & 0 \end{bmatrix} \begin{matrix} I \\ B \\ K \end{matrix} \quad (3.42)$$

Partial differentiation of F_i with respect to I and B gives the new infection matrix

$$F = \begin{bmatrix} \beta_1 S_{u0} & \beta_1 S_{a0} & c_1 H_0 \\ \beta_2 S_{u0} & \beta_2 S_{a0} & c_2 H_0 \end{bmatrix} \quad (3.43)$$

On the other hand,

$$\frac{dI}{dt} = \beta_1 S_1 I_1 + \beta_2 S_2 I_2 - (\gamma + \mu + \alpha)I$$

$$\frac{dB}{dt} = \alpha I - (\mu + \omega)B$$

V_i (3.44)

Partial differentiation of V_i with respect to I and B gives the transition matrix

$$\begin{bmatrix} \beta_1 S_1 & \beta_2 S_2 \\ 0 & 0 \end{bmatrix}$$

$$\begin{bmatrix} \gamma + \mu + \alpha & 0 \\ -\alpha & \mu + \omega \end{bmatrix}$$

V (3.45)

It follows that

$$\begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix}$$

$$V^{-1} \begin{bmatrix} \beta_1 S_1 & \beta_2 S_2 \\ 0 & 0 \end{bmatrix} \quad (3.46)$$

$$\begin{bmatrix} \frac{1}{\gamma + \mu + \alpha} & 0 \\ \frac{\alpha}{(\gamma + \mu + \alpha)(\mu + \omega)} & \frac{1}{\mu + \omega} \end{bmatrix}$$

$$\begin{bmatrix} \frac{\beta_1 S_1}{\gamma + \mu + \alpha} & \frac{\beta_2 S_2}{\gamma + \mu + \alpha} \\ \frac{\alpha \beta_1 S_1}{(\gamma + \mu + \alpha)(\mu + \omega)} & \frac{\alpha \beta_2 S_2}{(\gamma + \mu + \alpha)(\mu + \omega)} \end{bmatrix}$$

It follows that the next generation matrix is given by

FV^{-1} =

$$K(a S_{u0} + b S_{a0}) + c H_0 + w(a S_{u0} + b S_{a0}) + c H_0 \quad (3.47)$$

The spectral radius for FV^{-1} gives the effective reproduction number (basic reproduction number with controls) denoted by R_0^c which is given by

$$R_0^c = \frac{K(a S_{u0} + b S_{a0}) + w(a S_{u0} + b S_{a0})}{1 - K(c H_0) - w(c H_0)}$$

$$R_0 = \frac{K(a S_{u0} + b S_{a0}) + w(a S_{u0} + b S_{a0})}{1 - K(c H_0) - w(c H_0)} \quad (3.48)$$

Substituting

$$S_{u0} = \frac{BAC_0C_{11} + AB_{11}CC}{CA_{11}BB_{00}}, \quad S_{a0} = \frac{BAC_0C_{11} + AB_{11}CC}{CBAB_{11}}$$

$$H_0 = \frac{BAC_0C_{11} + AB_{11}CC}{CBAB_{11}(CA_{11}BB_{00})}$$

into equation (3.48) we get

□□ □2□ □□ AC_1 □ A_1C □ □ □b

R_{0c} □ 1 □□□□ □_____ _____□□□□B_0C_1 □B_1C□□□□□□ □□$_1b_1$ □ K□□$_2$

□$_2$□w□□□□□□□□□$CBAB_{11}$ □□$CA_{11}BB_{00}$ □□□□□□ □ □ □ (3.49)

_____ _____

□□$_1$ □ K□□□w□□□

_____

□a□□□□$_1$ □□□□□□□ c □$_2c_2$□ □□□ □$_1$ □□ AC_1 □ A_1C □□□ □$_2$ □□□$CBAB_{11}$ □□$CA_{11}BB_{00}$

□□□□□□□□ □ □□ □□□□$_{1\,1}$ □ K□□□w□□□□□□□□□□□□□□B_0C_1 □B_1C□□

□□□□□□

_____ _____ _____ _____ _____

When there is no any intervention we have: □$_2$ □□$_3$ □ a □ w □□$_1$ □□$_2$ □ □ □ 0 . Thus, the basic reproduction number for system (3.1) – (3.8) is:

□□ □$_2$□ □□ AC□

R_0 □ 1 □□ □□□ □ K□□□ w□□□□ □□□ $B_{1\,0}$□□CA_1□$_1C$□□□□□ □ □□□□□$_1b_1$ □

K□□□$_2b$□$_2$□w□□□□□□□□□□ CA_{11}□□BB_{00}□□ □□□□ □□ □□ , (3.50)

□ $_1$

_____ _____

□ □

_____

□□□□$_1$ □ □□□□□□□□ □□c □ K□□$_2c_2$□w□ □□□ □$_1$ □□ AC_1□ □ A_1C□□□ □ □$_2$

□□□□ CA_{11}□□BB_{00}□□ □□□□□□□□□ □ □ □

_____ _____ _____ _____ _____

$_{1\,1}$ □□□ □□□ □□□□ B_0□C_1□ □□ □□□ □□□

□□ □□

_____ _____

where

A = [illegible] B_0 = [illegible], C = [illegible],

A_1 = [illegible], B_1 = 0, C_1 = [illegible] which provides a measurement for the disease risk during a cholera outbreak. The first term in R_0 comes from the direct (human to human) transmission route, and the second term represents the contribution from the indirect (environment to human) transmission route.

3.3.2.1. Analysis of R_0^c with unique control strategy

In this sub-section, we use effective reproduction number in equation (3.49) to compute reproduction numbers for individual control strategy (intervention). The similar approach was done by Stephen *et al*. (2014a, 2014b) and Nyerere *et al*. (2014a, 2014b).

If vaccination is the only control that is [illegible] 0, a [illegible] w = 0 in (3.49) then the basic reproduction number with vaccination only is given by:

R_0^V = 0.0549287856 4

If awareness campaign is the only control that is [illegible] 0, [illegible] w 0 in (3.49) then the basic reproduction number with awareness campaign only is given by:

R_0^A = 0.8178062310

If treatment is the only control, we have a = 0, [illegible] w = 0 in (3.49) then the basic reproduction number with treatment only is given by:

R_0^T = 0.2504395594

If sanitation is the only control that is $w = 0, a = \beta_2 = \beta_3 = 0$ in (3.49) then the basic reproduction number with sanitation only is given by:

$R_0^S =$ 0.03973953413

3.3.2.2. Analysis of R_0^c with two control strategies

In this section, we further analyse the effective reproduction number in equation (3.49) by computing reproduction numbers for the combination of two control strategies

(interventions).

If the combination of vaccination and awareness campaign is the only intervention that is $\beta_2 = \beta_3 = 0, a = w = 0$ in (3.49) then the basic reproduction number with vaccination and education campaign only is given by:

$R_0^{VA} =$ 0.3132754649

If the combination of vaccination and treatment is the only intervention that is $\beta_2 = \beta_3 = 0, a = 0, w = 0$ in (3.49) then the basic reproduction number is given by:

$R_0^{VT} =$ 0.0989874770 2

If the combination of vaccination and water sanitation is the only intervention that is $\beta_2 = \beta_3 = 0, w = 0, a = 0$ in (3.49) then the basic reproduction number is given by:

R_0^{VS} = 0.05476273250

If the combination of treatment and awareness campaign is the only intervention that is $\psi_1 = 0, \psi_2 = 0, \psi_3 \neq 0$ in (3.49) then the basic reproduction number is given by:

R_0^{TA} = 0.5398014031

If the combination of water sanitation and awareness campaign is the only intervention that is $w = 0, \psi_2 = 0, \psi_3 \neq 0$ in (3.49) then the basic reproduction number is given by:

R_0^{SA} = 0.8175555096

If the combination of water sanitation and treatment is the only intervention that is $w = 0, a = 0, \psi_2 = 0, \psi_3 = 0$ in (3.49) then the basic reproduction number is given by:

R_0^{ST} = 0.2503089462

3.3.2.3. Analysis of R_0^c with three control strategies

Lastly, we analyse the effective reproduction number in (3.49) by computing reproduction numbers for the combination of three control strategies (interventions).

If the combination of water sanitation, treatment and awareness campaign is the only intervention that is $w = 0, a = 0, \psi_3 = 0$ in (3.49) then the basic reproduction number is given by:

R_0^{STA} = 0.5396913686

If the combination of water sanitation, vaccination and awareness campaign is the only intervention that is $w = 0, \psi_2 = 0, \psi_3 = 0$ in (3.49) then the basic reproduction number is given by:

R_0^{SVA} = 0.3176535300

If the combination of treatment, vaccination and awareness campaign is the only intervention that is $\lambda_3 = 0, \gamma_2 = 0, w = 0$ in (3.49) then the basic reproduction number is

given by:

R_0^{TVA} = 0.2133539861

If the combination of treatment, vaccination and water sanitation is the only intervention that is $\lambda_3 = 0, \gamma_2 = 0, w = 0$, in (3.49) then the basic reproduction number is given

by:

R_0^{TVS} = 0.1002509183

3.4 Stability Analysis of the Disease-Free Equilibrium Point

Here, we investigate the local and global stability of the disease-free equilibrium point,

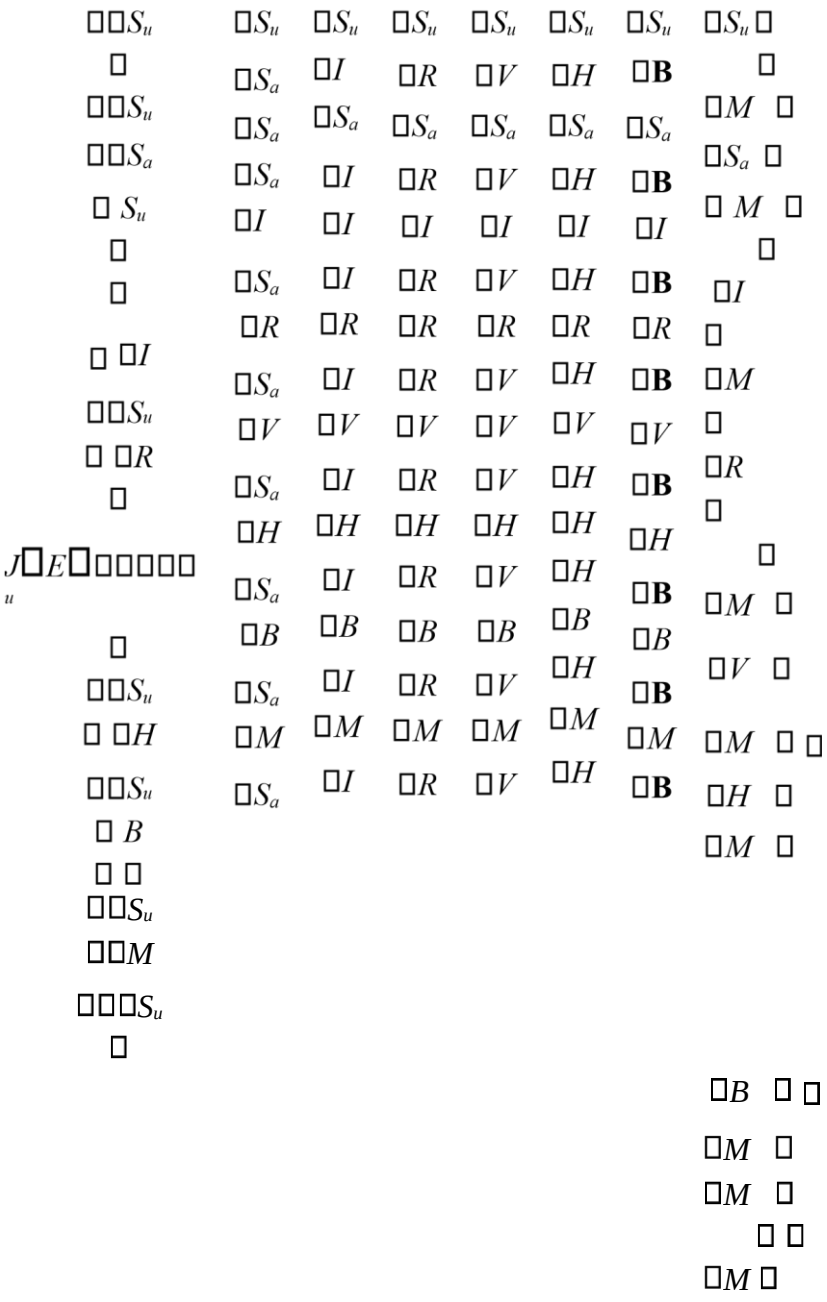
$E_0 = (S_{u0}, S_{a0}, I_0, R_0, V_0, H_0, B_0, M_0)$.

3.4.1 Local stability analysis of the disease-free equilibrium point

Theorem 3.3: If $c_0 < 1$, then the disease free equilibrium (DFE) point E^0 of the dynamical system (3.1) – (3.8) is locally asymptotically stable, and if $c_0 > 1$, then E^0 is unstable.

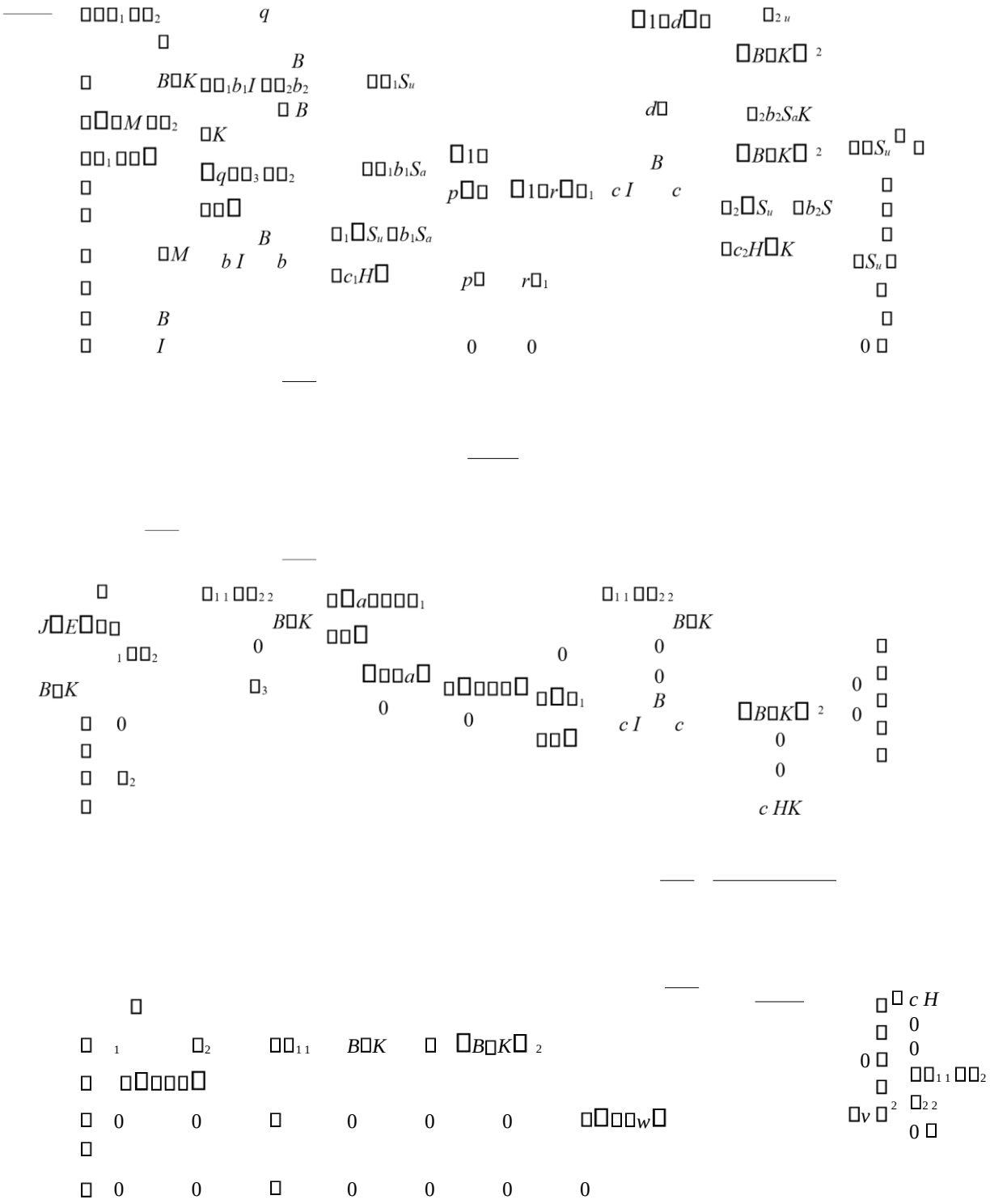
Proof: We linearize model system (3.1) – (3.8) by computing its Jacobian matrix, $J(E)$. The Jacobian matrix is computed by differentiating each equations in the system with respect

to the state variables $\{S_u, S_a, I, R, V, H, B, M\}$ as follows:



i.e.

$$\frac{B}{I} \quad \frac{}{} \quad \frac{}{SK}$$

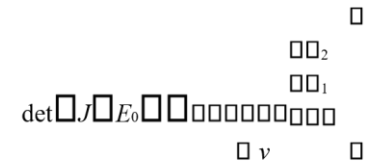


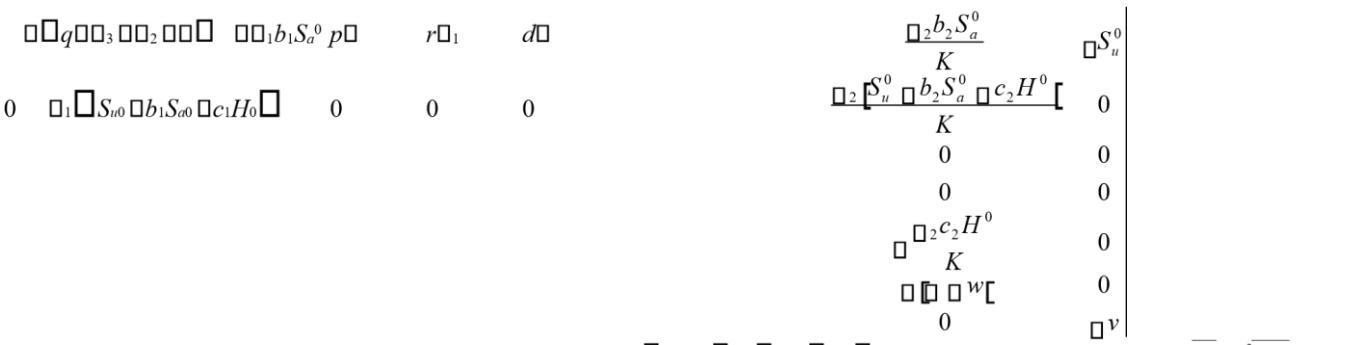
The Jacobian matrix at disease free equilibrium is given by the relation;

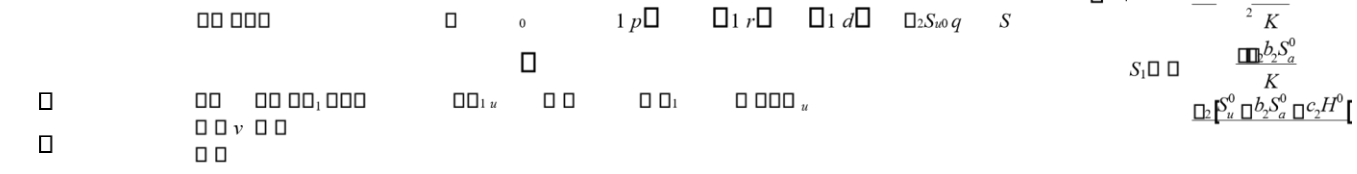
that 0.

det J E0

det J E0

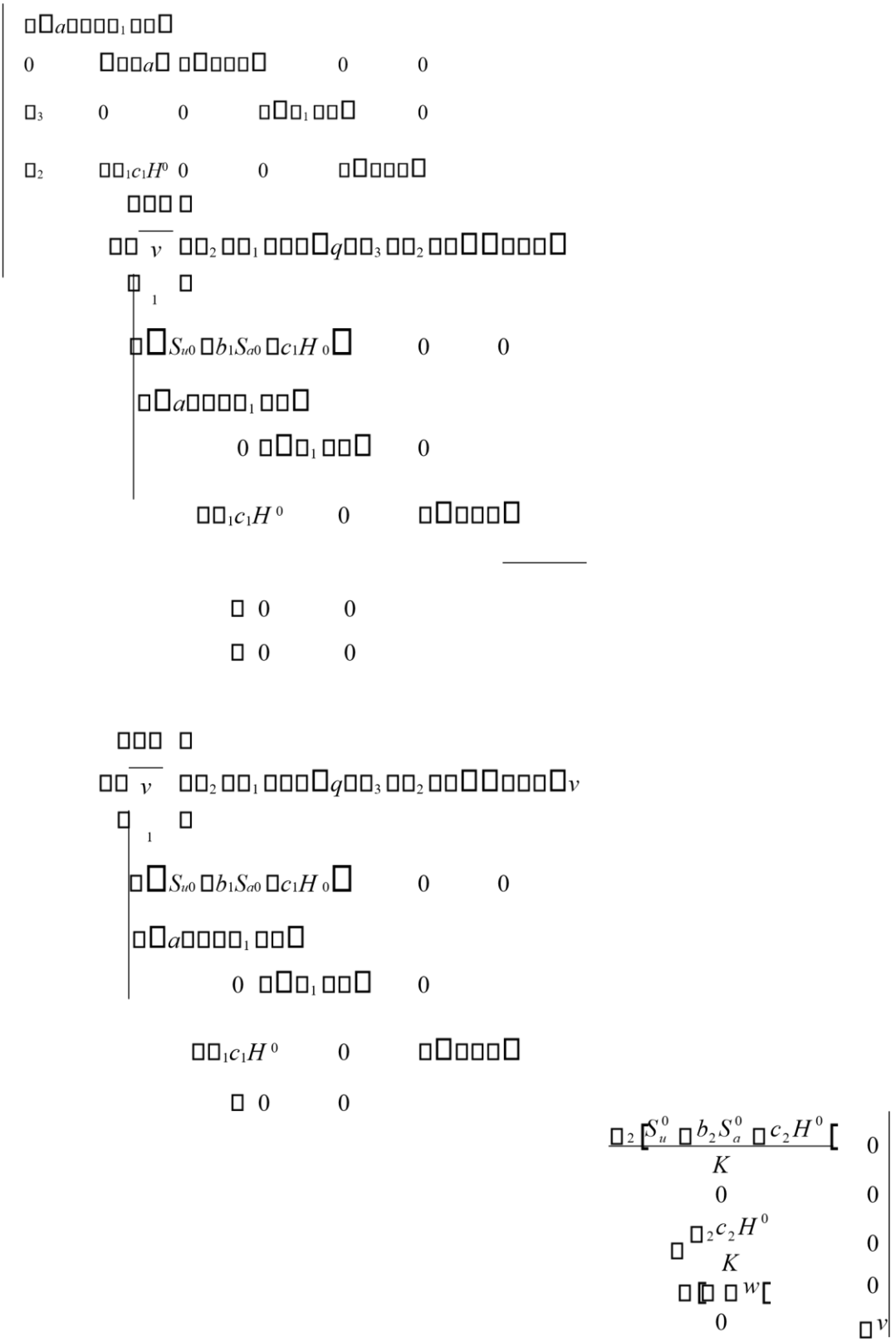


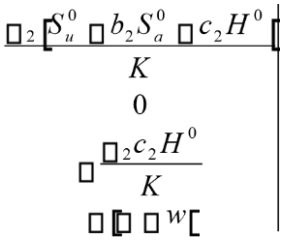


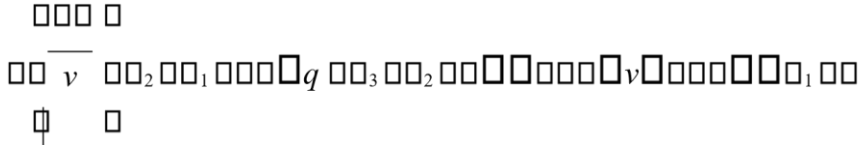


(3.53)

If a 1 1 S u 0 b1 S a 0 c1 H 0 0 then tr J E 0 0 so we only need to show







3.4.2 Global stability analysis of the disease-free equilibrium point

Theorem 3.4: If $\mathcal{R}_0 < 1$, the disease-free equilibrium (DFE) point E^0 of the dynamical system (3.1) – (3.8) is globally asymptotically stable.

Proof: Define a Lyapunov function:

$$L = \left(\frac{N_{Su}}{N_{Su}} - 1 - \ln\frac{S_{u0}}{S_u}\right) + \left(\frac{N_{Sa}}{N_{Sa}} - 1 - \ln\frac{S_{a0}}{S_a}\right) + \left(\frac{N_H}{N_H} - 1 - \ln\frac{H_0}{H}\right) + \frac{c_{0c}}{c_2}I + \frac{c_2}{S_{u0}K + bc_2S_{a0}w + c_2H_0}B \quad (3.55)$$

Then, the derivative of L along solutions of system (3.1) – (3.8) is

□□□□1□ HH_0 □□□□ $dHdt$ □□$_{c0}$ K□b□□$_{2}S$□$_{a0}w$□□c_2H_0□$dBdt$ (3.56)

$dIdt$ □ □$_2$□S_{u0}

□

□ S_0 □□ ——B

□□□1 □ S_{uu} □□□□□□□ □□11□□ df□□□□H□□□$_1$□□$S_{u2}I$□□□□$_{12}$□S□$_u$ □BS_u□ K

□□S_uM □□1□ p□□R □ qS_a □□1□ r□□$_1V$ □□ □□□□ □ □

——

□□1□ SS_{a0} □□□□□S_uM □b_1□$_1S_aI$ □b_2□$_2S_a$ B □ p□R □ r□$_1V$

□ d□H □□q □□$_3$ □□$_2$ □□□S_a □□□

□

——

□ $_a$□□ B □ K □

□ H^0□□ B □

□□1□ H □□□□□□$_1S_u$ □□$_2S_a$ □c_1□$_1HI$ □c_2□$_2{}^H$ ——B □ K □□□□□□H □ □□

——

□

□□a □□□□$_1$ □□□I

□□□c0 □1□□□□1□Su □b1Sa □c1H□I □□2□Su □b2Sa □c2H□_____B □□□

□ B □ K □

□□ □1□ f □□□□R □□1V □□H □□1Su □□2Sa □□□2 □□1 □□□Su □ □ □

□□_____□1□ f □□Su0 □ □□2 □□1 □□□Su0 □ □ □□

□ S u □

□□ □3 □□2 □□□Sa □□q □□3 □□2 □□□Sa0 □□□□□H □□□□□H0 □□Su0M □□

□□□□1□S0 □b1Sa0 □c1H0□□ K□□2□□w□ □Su0 □b2Sa0 □c2H0□□□□I □

u

□ □ □

—1— 1−□B □ Su0 □□1□ p□□R □ qSa □□1□r□□1V

□□1□d□□H□□

□2□Su0 □b2Sa0 □c2H0□□ □ □ □

□ B □ KK □ Su

Sa0 □□SuM □ p□R □ qSa □ r□1V □ d□H□□ H0 □□1Su □□2Sa□□□c0□a □□□□1 □□□I

—

—

Sa H

□□□c0 □1□□□□1□Su □b1Sa □c1H□I □□2□Su □b2Sa □c2H□ B □□□

—

□ B □ K □

□□□1□ f □□□□R □□1V □□H □□1Su □□2Sa □□□2 □□1 □□□Su □ □1□ f □□S u0 □ □ □

—

□ Su □

□□□ □□ □□□ Su0 □□3 □□2 □□□Sa □□q □□3 □□2 □□□Sa0 □□□□□H □ □□

□ 2 1

$$H^0 \quad S_u^0 M$$

$$c_0 a \quad _1 \quad I \quad _2 S_u^0 \quad b_2 S_a^0 \quad c_2 H^0 \quad 1 \quad 1 \quad B \quad B \quad K K$$

$$SS_{uu0} \quad 1 \quad p \quad R \quad qS_a \quad 1 \quad r \quad _1 V \quad 1 \quad d \quad H \quad SS_{aa0} \quad qSS_{au} M$$
$$r \quad _1 Vp \quad R \; d \quad H$$

$$_1 \qquad c \; a \quad _1 \quad I$$

$H_0 \qquad S_u \quad _2 S_a \quad _0$

$$H$$

That is $$c_0 \quad 1 \quad _1 S_u \quad b_1 S_a \quad c_1 H I \quad _2 S_u \quad b_2 S_a \quad c_2 H \quad B \quad \frac{dL}{dt}$$ (3.57)

$$B \quad K$$

^{c} 1, we get $\frac{dL}{dt}$ 0 which implies that the disease-free equilibrium E^0 of system (3.1) If $_0$

– (3.8) is globally asymptotically stable.

3.5 Existence of Endemic equilibrium (EE) Point

We proceed to investigate the endemic equilibrium (EE) point of model equations (3.1) – (3.8). The endemic equilibrium point is a positive steady state solution where the disease persists in the population. In this case, the infected variables are non-zero. For simplicity, we denote

$\square_{2\square} \; \square \; \square\square_2 \; \square\square_1 \; q_\square \square \square q\square\square_3 \square\square_2 \square\square\square, \square_\square \square \square\square\square\square, a_\square \square \square a\square\square\square\square_1 \square\square\square,$

$\square\square\square,$

$\square_{1\square} \square \square\square_1 \square\square\square, \square_\square \square \square\square\square\square, \square_w \square \square\square\square w\square, \square_a \square \square\square\square a\square$

The endemic equilibrium satisfies

$$N \square \quad \square S_u \; \square \; S_a \; \square \; H \square V \quad I \qquad R \tag{3.58}$$

From (3.6),

$$R \square \frac{\square^a \quad I}{\square_\square} \tag{3.59}$$

From (3.7),

$$\square \frac{\square I \quad B}{\square_w} \tag{3.60}$$

From (3.8),

$$\square \frac{\square\square\square I}{v} \qquad \frac{M}{\qquad} \tag{3.61}$$

From (3.5),

□ ——————— *f* □□□ $_2S_u$ □□□ $_3S$

V □1□ *a* (3.62)

From (3.4),

□□*I* □ □2*B* □*Su* □ □□*b*1□1*I* □ *b*2□2*B* □□*Sa* □ □□*c*1□1*I* □ *c*2□2*B* □□*H* □ □

(3.63)

□ 1 □

——— *a I*

——— ———

□ *B* □ *K* □ □ *B* □ *K* □ □ *B* □ *K* □

From (3.2),

M □

□□□□ □2□□□ *Su* □□□□*b*1□1*I* □

R □□*f*□ 1□ (3.64)

—— —— *bB*2□□2*KB* □ *q*□□□13□□□□*Sa* □ *d*□*H* □□*p*□

———

——

□ □1 □ □

From (3.3),

□1*Su* □□2*Sa* □ □ □*c*1□1*I* □ *c*2□2*B* □□□ □□*H* □

——— □ *B* □ *K* □ 0 (3.65)

Since I > 0, substituting equations (3.59) - (3.62) into (3.63) - (3.65) yields

A 0 , ———————————————*A*5*I*5 □ *A*4*I*4 □ *A*3*I*3 □ *A*2*I*2 □ *A*1*I* □

□ *Sa* □ *H* □*V* □ *B*5 5 *B*4*I*4 □ *B*3*I*3 □ *B*2*I*2 □ *B*1*I* □ *B*0 *I* □

(3.66

) *Su*

where

d□ n_3n_6 □□$_1$ □ n_3□□$_2$ □□ □□n_0 □

□□□□□ □□□□ n_7□$_2$ □ n_6□$_3$□□□ □ d□□□$_1$□$_3$ □□$_2$□$_2$□□ □□□ □ □

□□ □□□□$_1$□$_3$ □ n_1□$_2$□□□ □ n_3□□$_1$□$_3$ □□$_2$□$_2$□□f □□

□□□$_2n_7$ □ n_2n_6 □□$_2$ □ n_2□□□ □□d□n_2 □□□ n_7 □1□n_4 □ n_2□□$_1$ □□ □

□□□□$_2$□□□$_2d$□□□□1□ n_6□n_4 □□$_2$□ □□□□□□ □□ $_2$
□□ fn_0

□□□□□□□□□$_2$□$_3$ □ n_2□$_2$□□ n_4□□$_1$□$_3$ □□$_2$□$_2$□ □□□
□
□ □□□ n_6 □ n_7□□□ □□d□□ n_7□□$_1$ □□□ d□□ n_6□□$_2$□□□□^{2} □ □ □

□□□□□$_1n_7$ □ n_1n_6 □□$_1$ □ n_1□□□ □□n_1d□□ n_3n_7 □ n_1 □ n_3□□$_1$ □□ □ □

A_0 □□□□□□□□□□□□$_1$ □ □ f□□□ □ □ ^{3}

□□ □□□□□ □□n_5□ n_1□$a_{□}$□ n_8P□□□$_1$ □ n_1□□□$_{□}$□□□n_6□ n_7□$a_{□}$□ n_8P□□□$_1$

□□$_2$□□n_3□□ □ □ □

□□□□□□□$_1n_1$ □□$_2n_5$□$a_{□}$□ n_8P□□□□□$_1n_1$ □□$_1$□$_2$□

□□

□□ □□n_6□ n_7□$a_{□}$□ □

□□□□□ n_2 □3n_6 □3n_7□$a_{□}$□ n_8P□□n_2 □□$_2$□□$_{□}$□□□n_8P□□□$_1$ □□$_2$□□□□n_4 □

□□ □

□$_{□}$□$^{□}$□ □□□3d□□ n_2 □3n_7□□$_1$ □3□$_2$□□ d□□ n_6□□$a_{□}$□ n_8P□□n_2□$_1$ □□$_2$□$_2$□

□□

□□

□ □□□□□□□ f □□□□ □ n_1□n_5 □□$_1$□□$_{□}$□ n_3□□□□□□□□$n_{16}n_1$□□1□nn_{51}□□$_2$□□$_1$□n_7

□1□□□□□□□□□□□□□ □ □

□ □ □ □ □

□ □□□□□□□$_2n_1$ □□$_3n_5$□$a_{□}$□ n_8P□□□$_3$□$_1$ □□$_2n_1$□□□$_{□}$□ □□ $_2$

□ □□ n_0□

□□ □□□□□ n_3□□□□$_2n_7$ □□$_3n_6$□$a_{□}$□ n_8P□□□$_3$□$_1$ □□$_2$□$_2$□□ □ □

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□ □□ □

□ □□□□□□3n_6 □□$_2$ □3□n_1 □□n_5 □□$_1$□n_2 □3□$_1$□n_7 □1□□□$_{□}$□ □

□ □□□ □

□ □□□□□n_2 □3n_7 □3□□$_1$ □□□3n_6 □3□□$_2$ □□□1□ n_6□n_2 □□ □

□□ □□□□□□□□□□ □$_2$□n_7 □1□ □□□n_3 □□□ f□□

□ □□□□□□□$_1n_1$ □□$_2n_5$ □□□1□ n_6□n_1 □□$_1$□n_7 □1□□n_4 □ □ □

□□ □□□□ 3□d□□1□□□□□$_1n_1$ □□$_2$□$_1$□ □

A_1 □ □□□□□□□□□□□ n_2□$_2$ □3n_7□$_2$ □3n_6□$_3$□$a_{□}$□ n_8P□□□□$_2$□$_3$ □ n_2□$_2$□□□$_{□}$□

□□□ n_0□□

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□n_0□$_{□2}$

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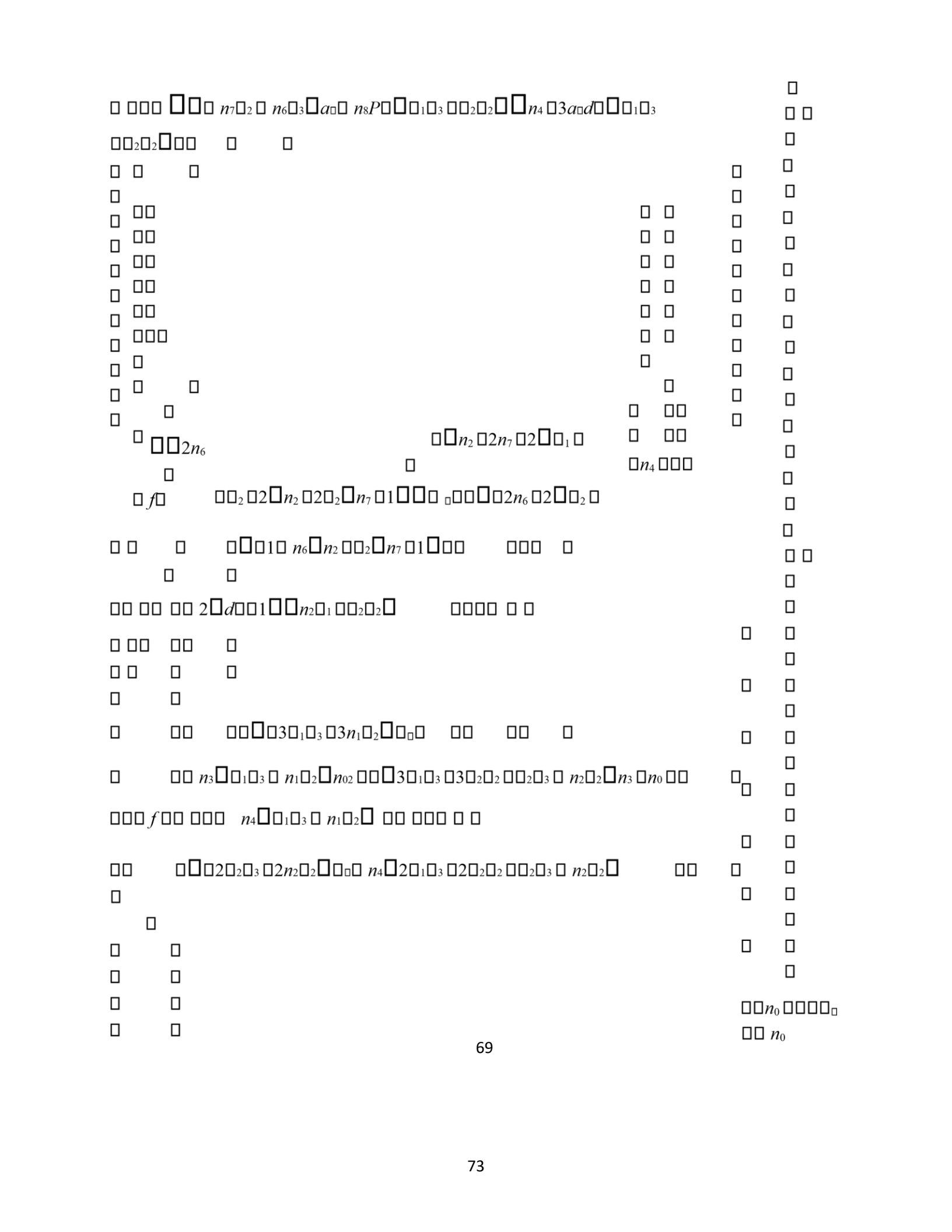

$$
\begin{aligned}
A_2 = &- \Bigg(-n_0 \Big(-n_3 \Big(\big(n_5 - n_1 \big) a_\mu + n_8 P \sigma \big(\beta_1 - n_1 \big) \Big) n_0^2 + \Big(\Big(\big(n_2 - 3 n_6 + 3 n_7 \big) a_\mu \\
&+ n_8 P \sigma \big(n_2 - \beta_2 + 3 \alpha_1 - 3 \alpha_2 \big) \Big) n_3 + \Big(\big(-n_5 + n_1 \big) a_\mu - n_8 P \sigma \big(\beta_1 - n_1 \big) \Big) n_4 + \Big(\big(\\
&-3 n_5 + 3 n_1 \big) \alpha_\mu + 3 n_5 \alpha_2 - 3 \alpha_1 n_1 \Big) a_\mu + 3 P n_8 \sigma \Big(\big(-\beta_1 + n_1 \big) \alpha_\mu + \beta_1 \alpha_2 - \alpha_1 n_1 \Big) \Big) \\
&n_0 + \Big(\big(n_2 - 2 n_6 + 2 n_7 \big) a_\mu + n_8 P \sigma \big(n_2 - \beta_2 + 2 \alpha_1 - 2 \alpha_2 \big) \Big) n_4 + \Big(\big(2 n_2 - 3 n_6 \\
&+ 3 n_7 \big) \alpha_\mu - 2 n_2 \alpha_1 + \big(3 d \alpha - 3 n_7 \big) \alpha_1 + 3 \alpha_2 \big(-d \alpha + n_6 \big) \Big) a_\mu - 2 \Big(\big(-n_2 + \beta_2 \big) \alpha_\mu \\
&+ n_2 \alpha_1 - \beta_2 \alpha_2 \Big) P n_8 \sigma \Big) \gamma_\mu^2 + \Bigg(\Big(f n_1 \big(n_5 - \beta_1 \big) \pi + \big(-\gamma_2 n_1 + \gamma_3 n_5 \big) a_\mu \\
&+ n_8 P \sigma \big(\beta_1 \gamma_3 - \gamma_2 n_1 \big) \Big) n_3 n_0^3 + \Big(f \Big(\Big(\big(3 n_6 - \beta_2 + 3 \alpha_1 - 3 \big) n_1 + \big(n_5 - \beta_1 \big) n_2 + \big(\\
&-3 n_7 + 3 \big) \beta_1 - 3 n_5 \alpha_2 \big) n_3 + n_1 \big(n_5 - \beta_1 \big) \big(n_4 + 3 \alpha_\mu \big) \Big) \pi + \Big(\big(-n_2 \gamma_2 - 3 n_7 \gamma_2 \\
&+ 3 n_6 \gamma_3 \big) a_\mu - P n_8 \sigma \big(n_2 \gamma_2 - 3 \alpha_2 \gamma_2 - \gamma_3 \big(\beta_2 - 3 \alpha_1 \big) \big) \Big) n_3 + \Big(\big(-\gamma_2 n_1 + \gamma_3 n_5 \big) a_\mu \\
&+ n_8 P \sigma \big(\beta_1 \gamma_3 - \gamma_2 n_1 \big) \Big) \big(n_4 + 3 \alpha_\mu \big) \Big) n_0^2 + \Bigg(2 f \Big(\Big(\Big(n_6 - \frac{\beta_2}{2} + \alpha_1 - 1 \Big) n_2 + \Big(-\frac{3}{2} \\
&+ \frac{3 n_7}{2} \Big) \alpha_1 + \big(-n_7 + 1 \big) \beta_2 - \frac{3 \alpha_2 \big(n_6 - 1 \big)}{2} \Big) n_3 + \Big(\Big(n_6 - \frac{\beta_2}{2} + \alpha_1 - 1 \Big) n_1 \\
&+ \Big(\frac{n_5}{2} - \frac{\beta_1}{2} \Big) n_2 + \big(-n_7 + 1 \big) \beta_1 - n_5 \alpha_2 \Big) n_4 + \Big(\Big(-\frac{3}{2} + \frac{3 n_6}{2} - \beta_2 \Big) n_1 + \big(n_5 \\
&- \beta_1 \big) n_2 - \frac{3 \beta_1 \big(n_7 - 1 \big)}{2} \Big) \alpha_\mu + \frac{3 \big(d \alpha - 1 \big) \big(-\alpha_1 n_1 + \beta_1 \alpha_2 \big)}{2} \Big) \pi + \Big(\big(-n_2 \gamma_2 \\
&- 2 n_7 \gamma_2 + 2 n_6 \gamma_3 \big) a_\mu - P n_8 \sigma \big(n_2 \gamma_2 - 2 \alpha_2 \gamma_2 - \gamma_3 \big(\beta_2 - 2 \alpha_1 \big) \big) \Big) n_4 + \Big(\big(-2 n_2 \gamma_2 \\
&- 3 n_7 \gamma_2 + 3 n_6 \gamma_3 \big) \alpha_\mu - 3 d \alpha \big(\alpha_1 \gamma_3 - \alpha_2 \gamma_2 \big) \Big) a_\mu - 2 n_8 P \alpha_\mu \sigma \big(-\beta_2 \gamma_3 + n_2 \gamma_2 \big) \Big) n_0 \\
&+ f \Big(\Big(\big(n_6 - \beta_2 + \alpha_1 - 1 \big) n_2 + \big(n_7 - 1 \big) \alpha_1 + \big(-n_7 + 1 \big) \beta_2 - \alpha_2 \big(n_6 - 1 \big) \Big) n_4 + \Big(\big(n_6 \\
&- \beta_2 - 1 \big) n_2 - \beta_2 \big(n_7 - 1 \big) \Big) \alpha_\mu - \big(d \alpha - 1 \big) \big(n_2 \alpha_1 - \beta_2 \alpha_2 \big) \Big) \pi \Big) \gamma_\mu - 2 f \Bigg(\\
&- \frac{3 n_3 \big(\beta_1 \gamma_3 - \gamma_2 n_1 \big) n_0^2}{2} + \Bigg(\Big(n_2 \gamma_2 - \frac{3 \alpha_2 \gamma_2}{2} - \gamma_3 \Big(\beta_2 - \frac{3 \alpha_1}{2} \Big) \Big) n_3 - \Big(n_4 \\
&+ \frac{3 \alpha_\mu}{2} \Big) \big(\beta_1 \gamma_3 - \gamma_2 n_1 \big) \Big) n_0 + \Big(\frac{n_2 \gamma_2}{2} - \frac{\alpha_2 \gamma_2}{2} - \frac{\gamma_3 \big(\beta_2 - \alpha_1 \big)}{2} \Big) n_4 \\
&+ \frac{\alpha_\mu \big(-\beta_2 \gamma_3 + n_2 \gamma_2 \big)}{2} \Big) \pi \Bigg)
\end{aligned}
$$

$$
\begin{aligned}
A_3 = -2 \Bigg(& \Bigg(\frac{3 n_3 \left(\left(n_5 - n_1 \right) a_\mu + n_8 P \sigma \left(\beta_1 - n_1 \right) \right) n_0^2}{2} + \Bigg(\Bigg(\Bigg(-n_2 + \frac{3 n_6}{2} - \frac{3 n_7}{2} \Bigg) a_\mu \\
& - \left(n_2 - \beta_2 + \frac{3 \alpha_1}{2} - \frac{3 \alpha_2}{2} \right) P n_8 \sigma \Bigg) n_3 + \Bigg(\Bigg(-n_4 + \frac{3 \alpha_1}{2} - \frac{3 \alpha_\mu}{2} \Bigg) n_1 + n_5 \Bigg(-\frac{3 \alpha_2}{2} \\
& + \frac{3 \alpha_\mu}{2} + n_4 \Bigg) \Bigg) a_\mu + \Bigg(\Bigg(-n_4 + \frac{3 \alpha_1}{2} - \frac{3 \alpha_\mu}{2} \Bigg) n_1 + \beta_1 \Bigg(-\frac{3 \alpha_2}{2} + \frac{3 \alpha_\mu}{2} \\
& + n_4 \Bigg) \Bigg) P n_8 \sigma \Bigg) n_0 + \Bigg(\Bigg(-\frac{n_2}{2} + \frac{n_6}{2} - \frac{n_7}{2} \Bigg) n_4 + \Bigg(-\frac{n_2}{2} + \frac{n_6}{2} - \frac{n_7}{2} \Bigg) \alpha_\mu + \frac{n_2 \alpha_1}{2} \\
& + \left(\frac{n_7}{2} - \frac{d \alpha}{2} \right) \alpha_1 - \frac{\alpha_2 \left(-d \alpha + n_6 \right)}{2} \Bigg) a_\mu \\
& - \frac{P n_8 \sigma \left(\left(n_2 - \beta_2 + \alpha_1 - \alpha_2 \right) n_4 + \left(n_2 - \beta_2 \right) \alpha_\mu - n_2 \alpha_1 + \beta_2 \alpha_2 \right)}{2} \Bigg) \gamma_\mu^2 \\
& + \Bigg(\frac{3 \left(f n_1 \left(n_5 - \beta_1 \right) \pi + \left(-\gamma_2 n_1 + \gamma_3 n_5 \right) a_\mu + n_8 P \sigma \left(\beta_1 \gamma_3 - \gamma_2 n_1 \right) \right) n_3 n_0^2}{2} \\
& + \Bigg(f \Bigg(\Bigg(\Bigg(-\frac{3}{2} + \frac{3 n_6}{2} - \beta_2 + \frac{3 \alpha_1}{2} \Bigg) n_1 + \left(n_5 - \beta_1 \right) n_2 + \left(\frac{3}{2} - \frac{3 n_7}{2} \right) \beta_1 \\
& - \frac{3 n_5 \alpha_2}{2} \Bigg) n_3 + \left(n_5 - \beta_1 \right) n_1 \left(n_4 + \frac{3 \alpha_\mu}{2} \right) \Bigg) \pi + \Bigg(\Bigg(-n_2 \gamma_2 + \frac{3}{2} n_6 \gamma_3 - \frac{3}{2} n_7 \gamma_2 \Bigg) a_\mu \\
& - P \left(n_2 \gamma_2 - \frac{3 \alpha_2 \gamma_2}{2} - \gamma_3 \left(\beta_2 - \frac{3 \alpha_1}{2} \right) \right) n_8 \sigma \Bigg) n_3 + \Big(\left(-\gamma_2 n_1 + \gamma_3 n_5 \right) a_\mu \\
& + n_8 P \sigma \left(\beta_1 \gamma_3 - \gamma_2 n_1 \right) \Big) \left(n_4 + \frac{3 \alpha_\mu}{2} \right) \Bigg) n_0 + \frac{1}{2} \Big(f \Big(\Big(\left(n_6 - \beta_2 + \alpha_1 - 1 \right) n_2 \\
& + \left(n_7 - 1 \right) \alpha_1 + \left(-n_6 + 1 \right) \alpha_2 - \beta_2 \left(n_7 - 1 \right) \Big) n_3 + \Big(\left(n_6 - \beta_2 + \alpha_1 - 1 \right) n_4 + \left(n_6 \\
& - \beta_2 - 1 \right) \alpha_\mu + \left(-d \alpha + 1 \right) \alpha_1 \Big) n_1 + \Big(\left(n_5 - \beta_1 \right) n_2 + \left(-n_7 + 1 \right) \beta_1 - n_5 \alpha_2 \Big) n_4 \\
& + \Big(\left(n_5 - \beta_1 \right) n_2 - \beta_1 \left(n_7 - 1 \right) \Big) \alpha_\mu + \beta_1 \alpha_2 \left(d \alpha - 1 \right) \Big) \pi \Big) + \Bigg(\Bigg(\frac{1}{2} n_6 \gamma_3 - \frac{1}{2} n_7 \gamma_2 \\
& - \frac{1}{2} n_2 \gamma_2 \Bigg) n_4 + \left(\frac{1}{2} n_6 \gamma_3 - \frac{1}{2} n_7 \gamma_2 - \frac{1}{2} n_2 \gamma_2 \right) \alpha_\mu - \frac{d \alpha \left(\alpha_1 \gamma_3 - \alpha_2 \gamma_2 \right)}{2} \Bigg) a_\mu \\
& - \frac{\left(\left(n_2 \gamma_2 - \alpha_2 \gamma_2 - \gamma_3 \left(\beta_2 - \alpha_1 \right) \right) n_4 + \alpha_\mu \left(-\beta_2 \gamma_3 + n_2 \gamma_2 \right) \right) P n_8 \sigma}{2} \Bigg) \gamma_\mu \\
& - \frac{1}{2} \Big(f \Big(-3 n_3 \left(\beta_1 \gamma_3 - \gamma_2 n_1 \right) n_0 + \left(n_2 \gamma_2 - \alpha_2 \gamma_2 - \gamma_3 \left(\beta_2 - \alpha_1 \right) \right) n_3 - \left(\beta_1 \gamma_3 \\
& - \gamma_2 n_1 \right) \left(n_4 + \alpha_\mu \right) \Big) \pi \Big) \Big)
\end{aligned}
$$

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$$g(I) = N - bI - B_5 I^5 - B_4 I^4 - B_3 I^3 - B_2 I^2 - B_1 I - B_0 \quad (3.68)$$

Then at the endemic equilibrium we have

$$h(I) = g(I), \; I \in \left[0, \frac{N}{b}\right] \quad (3.69)$$

Since $A_i > 0 \; (i = 0,1,2,3,4,5)$, it is straightforward to see $h(0) < 0$ and $h(I) > 0$.

We further make the following assumption:

$$bB^{i} \qquad \_\_\_ \qquad (3.70)$$

$$N \quad B_{i\square 1}$$

It is worth noting that $b \square\square$ 1. Here we introduce this condition to facilitate our analysis that follows.

Based on assumption (3.70), we obtain

$$g\square\square\square I\square\square\square \quad \square \ \square \ \square \ \square \ \square \ \square \ \square \ \square \ \square \ \square \ \square \ \square \ \square \ \square$$

$$\square bB_1\square\square 0 \quad 30bB_5I_4 20 \quad NB_5 bB_4 \quad I_3 12 \quad NB_4 bB_3 \quad I_2 6 \quad NB_3 bB_2 \quad I\ 2$$

$$NB_2$$

Now we denote $\square\square I\square\square\ h\square\square I\square\square\ g\square\square I\square$. Then $\square\square I\square$ is increasing since

0. $\square\square\square I\square\square\ h\square\square\square I\square\square\ g\square\square\square I\square\square$
(3.71)

In addition, notice that $R_0^c = \frac{}{} g^{0} B_0 N$. Hence, the following results can be obtained:

$$h(0) A_0$$

(1) If $R_0^c < 1$, then $h(I)$ and $g(I)$ have a unique intersection in .

(2) If $R_0^c > 1$, then there are two possibilities:

(i) If $(0) < 0$, these two curves have no intersection in ;

(ii) If $(0) > 0$, there is a unique intersection in .

(3) If $R_0^c = 1$, then there are three possibilities:

(i) If $h(I) < g(I)$ for all $I > 0$, then there is no intersection in ;

(ii) If there exists $I^* > 0$ such that $h(I^*) = g(I^*)$ and $h'(I^*) = g'(I^*)$, then there is a unique intersection in ;

(iii) Otherwise, there are two intersections in .

3.6 Stability Analysis of the Endemic Equilibrium Point

Here, we investigate the local and global stability of the endemic equilibrium point,

$E^* = (S_u^*, S_a^*, I^*, R^*, V^*, H^*, B^*, M^*)$.

3.6.1 Local stability analysis of the endemic equilibrium point

Theorem 3.5: If $R_0 > 1$, the endemic equilibrium (EE) point E^* of the dynamical system

(3.1) – (3.8) is locally asymptotically stable.

Proof: The Jacobian matrix of the system (3.1) – (3.8) at E^* is given by

$$J(E^*) = \begin{bmatrix} P & P_1 & -a & 0 & 0 & P_2 & Q_3 & 0 \\ 0 & 0 & a & 0 & 0 & 0 & 0 & 0 \\ \gamma_2 & \gamma_3 & 0 & 0 & -(\gamma_1+\mu) & 0 & 0 & 0 \\ \gamma_1 & \gamma_2 & -c_1\gamma_1 H^* & 0 & 0 & -P_2 & -Q_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & -w & 0 & -v \end{bmatrix} \quad (3.72)$$

where

$$P = -\gamma_1 I^* - \gamma_2 B^* B^* K, \quad P_1 = b_1\gamma_1 I^* + b_2\gamma_2 B^* B^* K, \quad P_2 = c_1\gamma_1 I^* + c_2\gamma_2 B^* B^* K,$$

$$Q = \frac{\beta B^{*2} S_u^* K}{K}, \quad Q_1 = \frac{\beta b B^{*2} S K_a^* K}{K^2}, \quad Q_2 = \frac{\beta c B^{*2} H K^* K}{K^2} \text{ and } Q_3 = \frac{\beta_2 S_u^* \beta b B^{*2} S K_a^* K + \beta c^2 H^* K}{2}$$

A necessary and sufficient condition for local asymptotic stability is for the real part of the eigenvalue to be in the negative half plane (Lewnard *et al*., 2014). Thus, it can be shown that

$J(E^*)$ given by (3.68) has eigenvalues which all have a negative real part.

In what follows, the characteristic equation of $J(E^*)$ is of the form $|J(E^*) - \lambda I| = 0$ given

by:

$$P M^* \beta_2 \beta_1 \left(\frac{\mu}{\mu+\alpha}\right)\left(\frac{\alpha}{\mu+\alpha+\gamma}\right)^3 P^{\frac{1}{2}} q \left(\frac{\mu}{\mu+\alpha}\right)\left(\frac{\alpha}{\mu+\alpha+\gamma}\right) v \beta_2 P_2 \lambda^3, \qquad (3.73)$$

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0$$

where a_0

$$a_1 = a\left(\frac{\mu}{\mu+\alpha}\right)\left(\frac{\alpha}{\mu+\alpha+\gamma}\right)^2 \beta_1 \left(\frac{\mu}{\mu+\alpha}\right)\left(\frac{\alpha}{\mu+\alpha+\gamma}\right) S_u^* + b_1 S_a^*$$

$$c_1 H^* \left(\frac{\mu}{\mu+\alpha}\right)\left(\frac{\alpha}{\mu+\alpha+\gamma}\right) w \quad a_2 = \left(\frac{\mu}{\mu+\alpha}\right)\left(\frac{\alpha}{\mu+\alpha+\gamma}\right) w a\left(\frac{\mu}{\mu+\alpha}\right)\left(\frac{\alpha}{\mu+\alpha+\gamma}\right)^2 \beta_1 \left(\frac{\mu}{\mu+\alpha}\right)\left(\frac{\alpha}{\mu+\alpha+\gamma}\right) S_u^* + b_1 S_a^* + c_1 H^* \left(\frac{\mu}{\mu+\alpha}\right)\left(\frac{\alpha}{\mu+\alpha+\gamma}\right) Q_3$$

The Routh-Hurwitz criterion (Nisbet and Gurney, 1982) requires

$a_1 > 0$, $a_2 > 0$ and $a_1 a_0 a_2 > 0$ (3.74) as the necessary and sufficient conditions for the locally asymptotical stability; i.e., all roots of the polynomial (3.73) have negative real parts. Note that at the endemic equilibrium, using equations of (3.4) and (3.7) yields

$$a = \mu_1 + (\mu_1 + b_1 S_{u^*} + b_1 S_{a^*} + c_1 H_*)(\mu_2 + \frac{S_{I^*}}{K^2}(S_{a^*} + w c_2 H_*)) \quad (3.75)$$

From equation (3.75) we can easily obtain

$$a > \mu_1 + (\mu_1 + b_1 S_u^* + b_1 S_a^* + c_1 H^*) > 0,$$

$$a w > a(\mu_1 + (\mu_1 + Q_3 + b_1 S_u^* + b_1 S_a^* + c_1 H^*)) + w > 0$$

Using the fact that all model parameters as well as P, P_1, P_2, Q, Q_1, Q_2 and Q_3 are positive, it is then straight forward to observe that all the inequalities in (3.74) hold.

3.6.2 Global stability analysis of the endemic equilibrium point

Let $E^* = (S_u^*, S_a^*, I^*, R^*, V^*, H^*, B^*, M^*)$ denote an endemic equilibrium (EE) of model (3.1) – (3.8). To establish the global stability of E^*, we make the following assumptions:

0 □□1□ $\frac{}{I}$ I_* □ □ $\frac{}{}$ □□1□ $M^{MI}{}_*^*$ I □ □□ □□ (3.76)

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for 0 □ I □ N and M^0 □ M □ $M_{\max}$, and

□ B □□ B_*
□ □□ □ $\frac{}{}$
□ $\frac{}{}$

0 □□1□ $\frac{B}{B}$ □$_*\frac{K}{}$ □□□□1□ $\frac{B_*}{}$ $B\frac{}{K}$ □ □□
(3.77)

$\frac{}{}$

□□ $\frac{}{B_*}$ □ K □□□□ B □ K □ □
for M^0 □ M □ $M_{\max}$ and 0 □ B □ $B_{\max}$.

Theorem 3.7: Suppose that (i) assumptions (3.76) and (3.77) are satisfied; (ii) □ is constant.

If □$_0$1, □he endemic equilibrium (EE) point E^* of the dynamical system (3.1) – (3.8) is globally asymptotically stable.

Proof: We define a Lyapunov function:

□ S_{u^*} □□dx $_S$□ S^*□ $_H$
L □ □$_{SS\,u^*u}$□□1□ x □□ □□$_{Sa^*a}$□□□1□ x_a □□□dx□□$_{H^*}$□□□□1□ Hx^* □□□□dx□□$_{II^*}$□□□□1□
$I\,x^*$ □□□□dx□ (3.78) □

$\frac{}{}$

$\frac{}{}$

$\frac{}{}$
$\frac{}{}$

□$_2$□S_{u^*} □I $b_2S_{a^*}$ □ c_2H^*□$B_{*\,B^*}$□ □1□ B^* □□□dx□□$_1$□S_{u^*} □ b_1□$SI_{a^{**}}$□ c_1H^*□I^* □$_{MM^*}$
□□□□1□ Mx^* □□ □□dx

$\frac{}{}$

Then, the derivative of L along solutions of system (3.1) – (3.8) is $\frac{dL}{dt} = \left(1 - \frac{1}{S_u^* S_a^*}\right) \frac{dS_u}{dt} + \left(1 - \frac{1}{S_a^*}\right) \frac{dS}{dt_a} + \left(1 - \frac{1}{HH^*}\right) \frac{dH}{dt} + \left(1 - \frac{1}{II^*}\right) \frac{dI}{dt}$ (3.79)

$\left(1 - \frac{1}{S_u^*}\right) \frac{dS_u}{dt} = \frac{1}{S_u^*} \left(-b_2 S_u^* S_a^* Ib^* BS^* + Kc_2 H^* B^* - \frac{1}{BB^*} \frac{dB}{dt}\right) + \frac{1}{S_u^*} \left(b_1 SI_a^* - c_1 H^* I^* - \frac{1}{MM^*} \frac{dM}{dt}\right)$

By direct calculations, we have that:

$\left(1 - \frac{1}{S_u^*}\right) \frac{dS_u}{dt} = \left(1 - \frac{1}{S_u^* I^*}\right) \left(1 - \frac{1}{SS_{uu}^*}\right) \left(1 - \frac{1}{SS_u^* II^*}\right) \left(-b_2 S_u^* B^* B^* K - \left(1 - \frac{1}{SS_{uu}^*}\right) \left(1 - \frac{1}{SS_u^* BB^* B^* KK}\right)\right)$

$\frac{dS_u}{dt}$

$S_{u^*}M^{*}\ 1\ SS_{uu^*}\ SS_{u^*u}MM^{*}\ 1\ 2\ S_{u1}\ S_{u^*}\ S_u\ 2\ 1$

$p\ R^{*}\ 1\ SS_{uu^*}\ 1\ RR^{*}$ (3.80)

$^{*}1\ S_{u^*}\ 1\ V^{V}{}_{*}\ 1\ d\ H^{*}\ 1\ S_{S_u^u}{}^{*}\ 1\ H^{H}{}_{*}$

$qS_a^{*}\ 1\ S_{S_u^u}{}^{*}\ 1\ S^{S}{}_{u}{}^{a}{}_{*}$

$1\ r\ _1V\ \ S_u$

B

$^{*}I^{*}\ 1\ S^{*}\ \ S\ I\ \ B^{*}$

$_1S_u\ S_{uu}\ 1\ S_{u^*u}I^{*}\ _2S_{u^*}B^{*}\ K\ 1\ SS_{uu^*}\ 1\ SS_{u^*u}BB^{*}B\ \ ^{*}KK$

B

$1\ SS_{a^*}\ dSdt_a\ b_1\ _1S_{a^*}I^{*}\ 1\ SS_{aa^*}\ 1\ SS_{a^*a}II^{*}\ b_2\ _2S_{a^*}B^{*}B\ ^{*}K$

$1\ SS_{aa^*}\ 1\ SS_{a^*a}BB^{*}B\ \ ^{*}KK$

$_a$

$S_{a^*}\ 1\ S\ M\ q$

$S_u^{*}M^{*}\ 1\ S\ S\ ^{*}{}_{u}M^{*}\ _3S\ _a\ _2\ S_{a^*}\ S_a\ _2\ p\ R^{*}\ 1\ SS_{aa^*}$

$1\ RR^{*}$ (3.81)

$_a\ \ _u$

$r\ _1V^{*}\ 1\ SS^{a^*}\ 1\ VV_{*}\ d\ H^{*}\ 1\ SS_a^a{}^{*}\ 1\ HH_{*}$

$_a$

B

$b_1\ _1S_{a^*}I^{*}\ 1\ S_{a^*}\ 1\ S^{S}{}_{a^*}{}^{a}I^{I}{}_{*}\ b_2\ _2S_a^{*}B^{*}B\ ^{*}K\ 1\ S_{S_a^a}{}^{*}\ 1$

$SS_{a^*}{}^{a}B^{B}{}_{*}B\ \ ^{*}{}^{K}K$

S_a

(3.82)

(3.83)

and

(3.84)

(3.85)

$$^{*}I^{*}\square\square\square 1\square\ MM^{*}\square\square\square\square\square\square\square\ II^{*}\square\ MM^{*}\square\ \square\ \square$$

$$c_1\square_1 H$$

$$\square$$

As a result, we get

$$\square \qquad B \qquad \square$$

$$dL\ \square\square S\ {}_{*}I{*}\square\square\square 1\square\ \overline{SS_{uu*}}\ \square\square\square\square\square\square\square\square 1\square\ \overline{SS_{u*u}II_{*}}\ \square\square\square\square\square\square_2 S_{u*}\ \overline{B*B\square_{*}K}$$
$$\square\square\square\square 1\square\ SS_{uu*}\ \square\square\square\square\square\square\square\square 1\square\ SS_{u*u}\ BB*B\square\square\ {}_{*}KK\ \square\ \square\ \square\ \square\square\ \square\ dt\ {}_{1\,u}\ \square$$

$$\square$$

$$\square$$

$$\square \qquad B \qquad \square$$

$$b_1\square_1 S_a\ {}^{*}I^{*}\square\square\square 1\square\ SS^{a}{}_{a}{}^{*}\ \square\square\square\square\square\square\square\square 1\square\ \overline{SS_{a^{*}}{}^{a}II_{*}}\ \square\square\square\square\square b_2\square_2 \overline{S_a^{*}\ B*B\square^{*}K}\ \square\square\square 1\square\ SS^{a}{}_{a}{}^{*}$$
$$\square\square\square\square\square\square\square 1\square\ SS_{a^{*}}{}^{a}\underline{BB*B}\square\square\ {}_{*}KK\ \square\ \square\ \square\ \square\ \square\square$$

$$\square$$

□

□ *B* □

*c*1□1*H**I*□□□1□ *HH** □□□□□□□1□ *HHI***I** □□□□ *c*2□2*H** *B***B*□* *K* □□□□1□
*HH** □□□□□□□□ □1□ *HH** *BB***B*□ **KK* □ □□ □ □ □ □

□ *B* □

□1*Su***I**□□□□ 1□ *II** □□□□□□□ *SSu***uII** □ *II** □□□□□□2*Su** *B***B*□* *K*
□□□□1□ *II**
□□□□□□□□ *SSu***u*
*BB***B*□□ **KK* □ *II** □
□□ □ □ □ □

□ *B* □

*b*1□1*Sa***I**□□□1□ *II* * □□□□□□ *SSa***aII** □ *II** □□□□□*b*2□2*Sa** *B***B*□* *K* □□□□1□ *II** □□□□□□□□
*SSa***aBB***B*□□ **KK* □ *II** □ □□ □ □ □

□ □□

□

□ *B* □

*I** □ *c*1□1*H***I**□□□ □ 1□ *I* □□□□□□ *HHI***I** □ *II** □□□□ *c*2□2*H**

*B***B*□* *K* □□□□1□ *II** □□□□□□□□ *H** *BB***B*□ **KK* □ *II** □ □□ □ □ □

□

□2*Su** *B* **B** *K* □□□1□ *BB* * □□□□□ *II** □ *BB** □□□□*b*2□2*Sa** *B***B** *K* □□□1□ *BB** □□□ □□ *II** □ *BB** □ □□
□ (3.86)

□ □ □□ □ □ □ □□

*B**

*c*2□2*H** *B* * *K* □□□□1□ *BB** □□□□□□□ *II** □ *BB** □□□□1*Su***I**□□□1□ *MM** □□□□□□ *II** □
*MM** □□ □ □

□

*b*1□1*S* **I**□□□□ 1□ *MM** □□□□□□□ *II** □ *MM** □□□□ *c*1□1*H***I**□□□□1□ *MM** □□□□□□□ *II** □ *MM**
□ □ □

For the function $v(x) = 1 - x + \ln x$, we know that $x > 0$ leads to $v(x) \le 0$. And if $x = 1$, then $v(x) = 0$. Note that:

□₁Sᵤ*I*□□□1□ SSᵤ* □□□ □□1□ S*ᵤI I*
□□□□□□₁Sᵤ*I*□□□□1□ I I* □□□□□□□□
S Sᵤ*ᵤ I* □ I I* □ □□□ □ □
□ ᵤ □□ Sᵤ

□₁Sᵤ*I*□□□2 □ SSᵤᵤ* □ II* □ SSᵤᵤ*III**I □ II* □□ □ □□
□

□₁Sᵤ*I*□□□ □□□□1□ II* □□□□□□□□1□ MMI**I □□□□□□ 3□ SSᵤᵤ* □ SSᵤᵤ*III**I MMI**I II*
□ MM* □ □ □ □□ (3.87) □

□₁Sᵤ*I*□□ □□□□□ SSᵘᵤ* □1□□□□□□□□□ SSᵤᵘ*III*I □1□□□□□□□□□ MMI*I
□1□□□□□ II* □ MM* □ □ □ □ □

□ □

□₁Sᵤ*I*□□□ MM* □ ln□□□ MM* □□□□
II* □ ln□□□ II* □□□□ □□ □ □

b ₁□₁Sₐ*I*□□□1□ SSₐₐ* □□□□□□□□1□ SSₐ*ₐII*
□□□□□b₁□₁Sₐ*I*□□□□1□ II* □□□□□□□□ SSₐ*ₐII* □
II* □□ □ □□ □

b₁□₁Sₐ*I*□□□2 □ SSₐₐ* □ II* □ SSₐₐ*III**I
□ II* □□□ □ □ □

b₁□₁Sₐ*I*□□□□□□□1□ II* □□□□□□□ MI* □ Sₐ* SₐII* MI* I M □□ □ (3.88)
□ 1□ *I □□□□ 3□ Sₐ □ Sₐ*I*I □ M *I □ I* □ M *
□□ M

□ □

□1*Sa*I*□□□ □□□□ SS*a*a* □1□□□□□□□□ SS*a*a*III*I □1□□□□□□□□□ *b*1 *MMI*I* □ MM* □ □ □□ □ II* 1

*b*1□1*Sa*I*□□□ M M* □ ln□□ MM* □□□□ II* □ ln□□□ II* □□□□ □ □ □ □ □

*c*1□1*H*I*□□□ 1□ *HH** □□□□□□□1□ *HHI*J* □□□□

*c*1□1*H*I*□□□□1□ *II** □□□□□□□ *HHI*I* □ *II** □ □□ □ □

c_1 $_1H$ $*I*$ 2 $HH*$ $II*$ $HHII*I**I$ $II*$

$*I*$ I

c_1 $_1H$ 1 $I*$ 1 $MMI**I$ 3 $HH*$ $HHII*I**I$ $MMI**I$ $MM*$ (3.89)

c_1 $_1H^*I^*$ $HH*$ 1 $HHII*I^*I$ 1 $MMI*^*I$ 1 $II*$ $MM*$

$_1H*I*$ $MM*$ ln $MM*$ $II*$ ln $II*$

c_1

Moreover, we can obtain

□ B □ □ B □

* □ Su* □□□□1 Su B □

□1□

□2Su* B *B □ K □ S u □□□□ □ Su* B*B□□ *KK □□□□□□□2Su* B*B□* K □□□□1□ II*

□□□□□□□□□□ SSu*u BB*B□□*KK □ II* □ □ □ □ □□

□

□ B B * □

B* □□2 S* Su I I □ □□

□2Su* B * □ K □□ □ Suuu □ B B*B□*KK □ Su* BB *□B*KK I □ I* □ □ □ (3.90)

□ □ □

B*

□2Su* B* □ K □□□□ BB* □ ln□□ BB* □□□ II* □ ln□□□ II* □□□ □ □□ □

□ □

□ B □ □ B □

B* □ Sa* □□□□1 Sa B

□ □1□

b2□2Sa* B* □ K □□ Sa □□□□ □ Sa* B*B□□ *KK □□□□□b2□2Sa* B*B□* K □□□□1□ II*

□□□□□□□□□□SSa*aBB*B□□*KK □ II* □ □□ □ □ □

□

□ B B * □

* B* □□2 S* Sa I I □ □□ (3.91) b2□2S a B* □ K □ □ Saa □ BB□*K □ □B*K I* □

B □

Sa* I

□□ B * □ K B * □ K □ □

* B B□ * □ B ln□□ B* □□□ II* □ ln□□□II* □□□□ □ □□

b2□2Sa * K □□□B* □ □B □

□ B □ □ B □

B*

c2□2H* B* □ K □□□□1□ HH* □□□□□□□□□1□ HH* BB*□B□ *KK □□□□□□□c2□2H* B*B□* K

□□□□1□ II* □□□□□□□□□HH* BB*□B□ *KK □ II* □ □ □ □ □

$*I*\square\square \quad M^* \square\square I \quad M\square$

$\square_1 S_u \square 1\square \overline{M\square\square} \overline{\square\square} \overline{I*\square M*} \square\square\square b_1\square_1 S_a*I*\square\square\square\square 1\square MM^* \square\square\square\square\square\square\square II*\square MM*\square\square\square$

$\square$

$\square \overline{M^*\square^{\square}I} \overline{M\square} \quad *I*\square\square\square I\square M\square M^*I\square 1\square\square$

S_u

$c_1\square_1 H^*I^*\square\square\square 1\square M\square\square\square\square I*\square M*\square\square\square\square_1 \square I* \quad M* \quad MI* \quad \square\square\square$

$b_1\square_1 S_a*I*\square\square\square I\,I*\square MM*\square MMI^**I \quad \square 1\square\square\square\square\square c_1\square_1 H*I*\square\square\square\square II*\square MM*\square MMI**I\square 1\square$
$\square\square\square\square$ (3.94)

$\square_1 S_u^* I^*\square\square\square II*\square\ln\square\square\square II*\square\square\square MM*\square\ln\square\square^{\square} MM*\square\square\square\square\square\square b_1\square_1 S_a^* I^*\square\square\square^{\square}$

$II*\square\ln^{\square}\square\square II*^{\square}\square\square\square MM*\square\ln\square^{\square} MM*\square^{\square}\square\square\square\square^{\square}\square\square c_1\square_1 H*I*\square\square\square\square II*$

$\square\ln\square\square\square II*\square\square\square\square \underline{MM*}\square\ln\square\square\square \underline{MM*}\square\square\square\square\square\square$

Consequently, we gain

$dLdt \quad \square\square S*I*\square\square\square MM*\square\ln\square\square\square MM*\square\square\square\square II*\square\ln\square\square\square II*\square\square\square\square\square\square\square\square b_1\square_1 S_a*I*\square\square\square\square MM*$
$\square\ln\square\square\square MM*\square\square\square\square II*\square\ln\square\square\square II*\square\square\square\square\square\square$

$\square$

$\square\, c_1\square_1 H^*I^*\square\square\square\square \quad MM*\square\ln\square\square MM*\square\square\square\square II*\square\ln\square\square II*\square\square\square\square\square\square\square\square_2 S_u^* B*B\square^* K\square\square\square BB*$
$\square\ln\square\square^{\square} BB*\square\square^{\square}\square II*\square\ln\square\square^{\square} II*\square\square^{\square}\square\square^{\square}$

$\square B \quad \square B\square\square$

$\square \quad \square\ln$

$b_2\square_2 S_a* \overline{B*B\square^*K\square\square\square\square} \overline{B^{\overline{B}_*}\square\square} \quad B\square I\square I\square\square *B_*\square B_*\square\square B_*\square\square\square\square\square\ln\square *$
$\square\square I*\square\ln\square\square I*\square\square\square\square\square\square c_2\square_2 H\,B*\square K\square\square\square II* \quad \square\ln\square\square\square II*\square\square\square\square\square\square\square$
$B\square$

$\square$

$$\left(1-\frac{I^*}{I^*+\alpha_1}\right)\left(\frac{I}{I^*}-\frac{I}{I}\right)$$

$$B^*S_uB^*K\left(\frac{II^*}{I^*+\alpha_1}\right)\left(\frac{I}{I^*}-\frac{I}{I}\right)\left(\frac{B}{B^*}-1\right)-\frac{B^*}{B}\left(\frac{I}{I^*}-\frac{I}{I}\right)$$

$$\left(1-\frac{I^*}{I^*+\alpha_1}\right)\ln\left(\frac{I}{I^*}\right)-\left(\frac{I}{I^*}-\frac{I}{I}\right)$$

$$\left(1-\frac{I^*}{I^*+\alpha_1}\right)\ln\left(\frac{I^*+\alpha_1}{I+\alpha_1}\right)+\left(\frac{I^*+\alpha_1}{I+\alpha_1}-1\right)$$

$$\alpha_2 B^*\left(1-\frac{II^*}{MM^*}\right)-\ln\left(\frac{II^*}{MM^*}\right)+\left(\frac{II^*}{MM^*}-1\right)+b_2\alpha_2S_aB^*\left(1-\frac{K}{BB^*}\right)-\ln\left(\frac{K}{BB^*}\right)+\left(\frac{K}{BB^*}-1\right)$$

$$\left(1-\frac{B^*}{B}\right)\left(\frac{I}{I^*}-\frac{I}{I}\right)\left(\frac{B}{B^*}-1\right)$$

$$c_2\alpha_2H^*B^*\left(1-\frac{K}{I^*}\right)\left(1-\frac{I^*}{I}\right)\ln\left(\frac{I}{I^*}\right)+\left(\frac{B}{B^*}-1\right)\ln\left(\frac{B}{B^*}\right)+\alpha_1S_uI^*\left(1-\frac{II^*}{MM^*}\right)\ln\left(\frac{II^*}{MM^*}\right)+\left(\frac{II^*}{MM^*}-1\right)$$

$$b_1\alpha_1S_a^*I^*\left(1-\frac{II^*}{MM^*}\right)\ln\left(\frac{II^*}{MM^*}\right)+\left(\frac{II^*}{MM^*}-1\right)+c_1\alpha_1H^*I^*\left(1-\frac{II^*}{MM^*}\right)\ln\left(\frac{II^*}{MM^*}\right)+\left(\frac{II^*}{MM^*}-1\right)\leq 0 \quad (3.95)$$

One can see that the largest invariant subset, where $\frac{dL}{dt}=0$, is E^*. By LaSalle's Invariance Principle (LaSalle, 1979), E^* is globally asymptotically stable when $\mathcal{R}_c<1$.

3.7 Sensitivity Analysis of Model Parameters

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 (Osman *et al*., 2018).

The effective reproductive (basic reproductive number with controls);

$$\mathcal{R}_c=\frac{\alpha_2\left(\alpha_0+b\right)}{\left(\alpha_1+K\right)\left(\alpha_0+w\right)\left(\alpha_0+S_u\right)}\left(\frac{\alpha_1b_1}{\left(K+\alpha_2\right)\left(w+S_{a0}\right)}\right)<1$$

□0 □ □a □□□□1 □□ □ □□□□□□□ □1c1 □ K□□□2c□2□w□□□□□H 0 □□□ □
(3.96)

□

_____

□

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

□□c X

S X□c0□ 0 □
(3.97)

____ ___
X □c0

□

where *X* represents any parameter in the model.

For □

□1 □ □c0

□

For □2: □□1

□c0 □□ c 0 □2

S

□□c (3.99)

□c0 ____ ___0 □1

S (3.98)

____ ___

$$\square_2 \quad \square \qquad \square_{c0} \qquad S$$

(3.100)

For $\square$:

$$\square \qquad \square\square_2$$

$$\square_{c0} \qquad \square\square_{c0} \qquad \square$$

$$\square \qquad \square \qquad \square_{c0} \qquad S$$

(3.101)

For K:

$$\square \qquad \square\square$$

$$\square_{c0} \qquad \square\square_{c0} \qquad K$$

$$\square \quad {}_K\square K \quad \square\square_{c0}$$

For :

$$\square \qquad \square \qquad \square_{c0}$$

For w:

$$\square \qquad \square\square$$

$$S_w^{\square_{c0}} \square^0 \square \qquad \square\square^c \qquad w$$

$$\square w \qquad \square_0$$

(3.103)

For $\square$:

$$\square\square^c \qquad \square$$

$$S_{\square}^{\square_{c0}} \square^0 \square \qquad \square$$

(3.104)

$$\square\square^c$$

$$\square_{c0} \qquad \square^0 \qquad \square \qquad S$$

(3.102)

$$\square\square \quad \square_0$$

For $\square_1$:

$$\square_{\square_{1c0}} \square\square \quad \square_1 \qquad (3.105)$$

$$\square^{\ 0}\square \qquad c$$

$$\square\square_1 \quad \square_0$$

For $\square_2$:

$$\square_{c0} \quad \square\square_{\ c\,0} \ \square_2$$

$$c \qquad (3.106)$$

$$\_\_\_\_ \quad \_\_\_$$

$$\_\_\_\_ \quad \_\_\_$$

$$\square_2 \ \square \qquad \square_{c0}$$

For a : $\square$

$$\square\square_2$$

$$S^{\square_{c0}} \quad \square\square_{c\,0} \quad a \qquad (3.107)$$

$$\_\_\_\_ \quad \_\_\_$$

$$_a\square \quad a \ \square\square_{c0}$$

$$\square$$

For $\square$

$$c$$

$$\square \ \square \qquad \square_{\ c}$$

$$\square\square \quad \square_0$$

For $\square$:

$$\square_{c0} \qquad \square\square_{c\,0} \quad \square$$

$$\square\square \qquad S \qquad (3.109)$$

$$S^{\square_{c0}} \_\_\_\_ \quad \_\_\_^{0} \ \square \qquad (3.108)$$

For β_1:

$$\frac{\partial \Lambda_{c0}}{\partial \beta_1} = \frac{\partial \Lambda_{c0}}{\partial \beta_0} \frac{\partial \beta_0}{\partial \beta_1} = \frac{\partial \Lambda_{c0}}{\partial \beta_0} S \quad (3.110)$$

$$\frac{\partial \Lambda_{c0}}{\partial \beta_0} = \frac{\partial \Lambda_{c0}}{\partial \beta_0}$$

$$\frac{\partial \Lambda_{c0}}{\partial \beta_0} = \frac{\partial \Lambda_{c0}}{\partial \beta_0}$$

$$\frac{\partial \Lambda_{c0}}{\partial \beta_0} = \frac{\partial \Lambda_{c0}}{\partial \beta_0}$$

And similarly for other parameters.

Table 3.3: Sensitivity Index of the Parameter Values

Parameters	Sensitivity Index
β_1	0.2788515937

$$\beta_1 \quad c$$

$$\beta_1 \quad \beta_0$$

For β:

$$S_{\beta}^{c} \quad \beta_{c0} \quad \beta_0 \qquad c \qquad (3.111)$$

For f: f

$$\beta_{c0} \quad S_f^{c} \qquad (3.112)$$

	β_2	0.7211484063
	β	0.7211484063
	K	-0.7211484063
	β	-0.7125972390
	w	-0.008551166870
-	β	0.6568105028
	β_1	0.4052468096
-	β_2	0.4004237081
	β_3	0.01972919981
	a	-0.5573687407

-	β	0.4628099174
-	β	0.1665709271
-	β_1	0.009208972845
	f	-0.03050679816
	β	1.00000000000
0	β	
-	β	0.06762473393
0	β	
	v	-0.04620627709
	β	-0.06762473392
	q	0.00004732727241
β_1		-0.4031770561
β_2		-0.03417063174

Table 3.3 shows the sensitivity indices of the effective reproduction number with respect to parameters. Thus, the negative indices are $\beta K,\beta,w,\beta,\beta_2,a,\beta,\beta_1, f ,\beta,\beta,\beta,\beta,\beta_1,\beta_2\beta$ indicating that they had a negative impact on the transmission of disease as the values of the

parameter are increasing. The positive indices are $\Box\Box_1,\Box_2,\Box,\Box_1,\Box_3,\Box,q\Box$ indicating that they had a great impact on the transmission of disease as the values of the parameter are increasing. This indicates that, to eradicate cholera, stakeholders should devise strategies and implement it to reduce these parameters.

Table 3.4: Values for Variables used for the Graphical Presentation

Variables	Values per year	Source
S_u $\Box 0\Box$	100,423,617	E10
S_a $\Box 0\Box$	43,038,693	E9
H $\Box 0\Box$	49,596,906	E7
$\Box$ $0\Box$ I	42,466	E4
V $\Box 0\Box$	5,244,305	E6
R $\Box 0\Box$	41,636	E5
B $\Box 0\Box$	275,000	Assumed
M $\Box 0\Box$	100	Assumed
N	198,387,623	E1

Table 3.5: Values for Parameters used for the Graphical Presentation

Parameters	Value	Description	Unit	Source
$\Box$	1	0.011 Human to human transmission rate	/day	Wang and Modnak (2011)
$\Box$	2	0.075 Environment to human transmission	/day	Mukandavire *et al*. (2011)

		rate		
□	0.033	Decay rate of vibrios	/day	Sun *et al*. (2017)
□		Infected human shedding rate	Cells/ml/day	Lemos-Paiao *et al*. (2016)
K	2000000	Concentration of vibrio in the environment	Cells/ml	Merrell *et al*. (2002)

□

□$_1$

□$_2$

□$_3$

w	0.0004	Disinfection rate	/year	Assumed
0.018	Natural death rate	/year	E11	
0.075	Vaccination loss rate	/year	Assumed	
0.3366	/year	E14		

Vaccination rate for unaware humans

0.6733	Vaccination rate for	/year	E15

□

□

aware humans

a	1.1	Treatment rate	/day	Assumed
0.98	Recovery rate	/day	E13	
0.01158	/year	Assumed		

Hygiene conscious loss rate

□^{1}	0.0195	Death rate (due to infection)	/day	NCDC (2019); E12
f	0.75	Proportion		Assumed
7588327	Recruitment rate	/day	E3	
		0.002	/year	Neilan *et al*. (2010)

□		Immunity waning rate		
0.03	□	Awareness growth	/day	Assumed rate
	0.01	Awareness	/day	Assumed
□		stimulated rate		
v	0.03	Awareness decay rate	/day	Assumed
□	0.95	Awareness rate	/day	Assumed
q	0.0003	Awareness loss rate	/year	Assumed
		□1	0.1 /day	Assumed

Hygiene conscious rate for unaware humans

	□2	0.3	Hygiene conscious	/day Assumed
		rate for aware humans		
r	0.4	Proportion of vaccinated individual		Assumed
d	0.45	Proportion of hygiene conscious class		Assumed
p	0.75	Proportion of recovery class		Assumed
b_1	0.0003	Proportion of direct disease transmission rate for the aware compartment		Assumed
c_1	0.001	Proportion of direct disease transmission rate for the hygiene conscious compartment		Assumed

b_2	0.4	Proportion of indirect disease transmission rate for the aware compartment	Assumed
c_2	0.0002	Proportion of indirect disease transmission rate for the hygiene conscious compartment	Estimated

See appendix, for the estimation of variables and parameters values shown in Tables 3.4 and 3.5, used in graphical presentation.

3.8 Model Solution Via Homotopy Perturbation Method (HPM)

Homotopy perturbation method (HPM) was first proposed by He and was successfully applied to various engineering problems (He, 2006). We apply Homotopy-perturbation to equations (3.1) – (3.8), where details can be found in He (2006). We construct a homotopy in the form:

$$\frac{dS^{u}}{dt} = h\left[\frac{dS}{dt}_{u} - \left(\frac{1}{2}S_{u}\right)I - \left(\frac{1}{2}S_{u}\right)B - BK - \left(\frac{1}{2}S_{u}\right)M - \left(\frac{1}{2}\right)\left(\frac{1}{2}\right)\left(\frac{1}{2}S_{u}\right)\left(\frac{1}{2}\right)\right] \quad (3.113)$$

$$\left(1-\frac{h}{2}\right)\frac{dI}{dt} = \left(1-\frac{p}{2}\right)R - \left(\frac{qS_{a}}{2}\right)\left(1-\frac{r}{2}\right)V - \left(1-\frac{d}{2}\right)H - \left(1-\frac{f}{2}\right)\left(\frac{0}{2}\right)$$

$$\left(1-\frac{h}{2}\right)\frac{dt^{a}}{dt} = h\left(\frac{1}{2}\right) - \left(\frac{qdt}{3}\right)\left(\frac{1}{2}\right)\left(\frac{1}{2}\right)^{a}\left(\frac{1}{2}\right)\left(\frac{S^{2}_{a}}{2}\right)^{2}$$

$$p^{a} - BR - rK\left(\frac{1}{2}\right)V - d^{u}H\left(\frac{1}{2}\right) = 0 \quad (3.114)$$

$$\left(1-\frac{h}{2}\right)\frac{dI}{dt} = h\left(\frac{1}{2}\right) - \left(\frac{dta}{2}\right) - \left(\frac{1}{2}\right)\left(\frac{S_{1u}}{2}\right) - b_{1}\left(\frac{SI_{a}}{2}\right) - c_{1}H\left(\frac{I}{2}\right) - \left(\frac{1}{2}\right)\left(\frac{S_{u}}{2}\right) - b_{2}S_{a} - c_{2}H\left(\frac{B}{2}\right)$$

$$K\left(\frac{0}{2}\right) = 0 \quad (3.115)$$

□1□ $\overline{h}$□dR □ $\overline{h}$□ ^{dR} □□□□ a□I □□□□□□R□□□

(3.116) dt □ dt □ 0

□1□ h□dV □ h□ ^{dV} □ f□□□$_2S_u$ □□$_3S_a$ □□□$_1$ □□□V □□□

(3.117) dt □ dt □ 0

□1□ h□dH □ h□ ^{dH} □□□$_1S_u$ □□$_2S_a$ □ c_1□$_1HI$ □ c_2□$_2H^B$ □□□□□□H □□□

(3.118) dt □ dtB □ K □ 0

dS □ dS_a b S I b S B S M□

$$\left(1+h\right)\frac{dB}{dt}+h\frac{dB}{dt}+I+w\left(B-1\right)=0 \tag{3.119}$$

$$\left(1+h\right)\frac{dM}{dt}+h\frac{dM}{dt}+I+M-1=0 \tag{3.120}$$

Let

$$\begin{aligned}
S_u &= S_{u0}+hS_{u1}+h^2S_{u2}+\ldots \\
S_a &= S_{a0}+hS_{a1}+h^2S_{a2}+\ldots \\
I &= I_0+hI_1+h^3I_2+\ldots \\
R &= R_0+hR_1+h^2R_2+\ldots \\
V &= V_0+hV_1+h^2V_2+\ldots \\
H &= H_0+hH_1+h^2H_2+\ldots \\
B &= B_0+hB_1+h^2B_2+\ldots \\
M &= M_0+hM_1+h^2M_2+\ldots
\end{aligned} \tag{3.121}$$

Substituting (3.121) and (3.122) into equations (3.113) – (3.120) and collecting the coefficient of power of h , we have: h^0:

$$M = M_0 + hM_1 + h^2M_2 + \ldots$$

and

$$\begin{aligned}
S_u &= S_{u0} + hS_{u1} + h^2S_{u2} + \ldots \\
S_a &= S_{a0} + hS_{a1} + h^2S_{a2} + \ldots \\
I &= I_0 + hI_1 + h^2I_2 + \ldots \\
R &= R_0 + hR_1 + h^2R_2 + \ldots \\
V &= V_0 + hV_1 + h^2V_2 + \ldots \\
H &= H_0 + hH_1 + h^2H_2 + \ldots
\end{aligned} \tag{3.122}$$

$$\frac{dS_{u0}}{dt} = 0 \tag{3.123}$$

$$\frac{dS_{a0}}{dt} = 0 \tag{3.124}$$

$$\frac{dI_0}{dt} = 0 \tag{3.125}$$

$$\frac{dR_0}{dt} = 0 \tag{3.126}$$

$$\frac{dV_0}{dt} = 0 \quad (3.127)$$

$$\frac{dH_0}{dt} = 0 \quad (3.128)$$

$$\frac{dB_0}{dt} = 0 \quad (3.129)$$

$$\frac{dM_0}{dt} = 0 \quad (3.130)$$

h^1:

$$\frac{dS_{u1}}{dt} = K_1 - K_2 \quad (3.131)$$

$$\frac{dK}{dt} = K\left[\quad\right] - K\left[\quad\right] \qquad (3.132)$$

$$p R_0\left[\quad\right] - \left[\quad\right] \varepsilon_1 V_0 \left[\quad\right] - d\left[H_0 \quad\right] 0$$

$$dI_1 \left[\right] \left[\right] {}^{1}\left[S_{u0} \left[\right] b_1 S_{a0} \left[\right] c_1 H_0\right] I_0 \left[\right] \left[\right] {}_2\left[S_{u0} \left[\right] b_2 S_{a0} \left[\right] c_2 H_0\right] B_0 \left[\right] \left[\right] 1\left[\right] B_0 \left[\right] \left[\right] \left[\right]$$

$$\frac{dK}{dt} = K\left[\right] K\left[\right] \qquad (3.133)$$

$$\left[a \left[\right] \left[\right] \left[\right] \left[\right] {}_1 \left[\right] \left[\right] \left[I_0 \right] \left[\right] 0$$

$$dR_1 \left[\right] \left[\right] \left[\right] a\left[I_0 \right] \left[\right] \left[\right] \left[\right] \left[\right] \left[R_0 \right] \left[\right] 0 \qquad (3.134)$$

$$dt\, dV_1 \left[\right] \quad f\left[\right] \left[\right] \left[\right] {}_2 S_{u0} \left[\right] \left[\right] {}_3 S_{a0} \left[\right] \left[\right] \left[\right] {}_1 \left[\right] \left[\right] \left[V_0 \right] \left[\right] 0 \qquad (3.135)$$

$$dt\, dH_1 \left[\right] \left[\right] S_{u0} \left[\right] \left[\right] {}_2 S_{a0} \left[\right] c_1 \left[\right] {}_1 H_0 I_0 \left[\right] c_2 \left[\right] {}_2 H_0 B_0 \left[\right] {}^{1}\left[\right] B_0 \left[\right] \left[\right] \left[\right] \left[\right] \left[\right] \left[H_0 \right] \left[\right] 0 \qquad (3.136)$$

$$\frac{dK}{dt} = K\left[\quad\right] \quad K\left[\quad\right]$$

$$dB_1 \left[\right] \left[\right] I_0 \left[\right] \left[\right] \left[\right] w\left[B_0 \right] \left[\right]$$

$$\frac{}{dt} \qquad 0 \qquad (3.137)$$

$$\frac{dM_1}{dt} = I_0 \times M_0 \times h^2 : \qquad 0 \qquad (3.138)$$

$$dS_{u2} \; {}_1S_{u1}I_0 \square\square_1S_{u0}I_1 \square\square_2S_{u1}B_0 \square\square 1\square B_0 \square\square\square\square_2S_{u0}B_1 \square\square 1\square \qquad 2B_0 \square \; S_{u1}M_0 \square$$

$$\square\square \qquad \_\_ \qquad \_\_ \qquad \_\_ \qquad \_\_\_\_ \square\square\square K$$

$$dt \qquad K \square \quad K \square \quad K \square \qquad \square \qquad (3.139)$$

$$\square S_{u0}M_1 \square\square_2 \square\square_1 \square\square\square S_{u1} \square\square 1\square p\square\square R_1 \square qS_{a1} \square\square 1\square r\square\square_1V_1 \square\square 1\square$$

$$d\square\square H_1 \square 0$$

$$\_\_ \qquad \_\_\_\_$$

$$dt \square \quad {}_1\square_{1\,a1\,0} \square \; {}_1\square_{1\,a0\,1} \square \; {}_2\square_{2\,a1}K \square\square \square K \square\square\square \; {}_2\square_{2\,a0}K \square\square \square K \square\square\square \qquad (3.140)$$

$$\square S_{u1}M_0 \square\square S_{u0}M_1 \square\square q \square\square_3 \square\square_2 \square\square\square S_{a1} \square p\square R_1 \square r\square_1V_1 \square d\square H_1 \square 0 \; dI$$

$$_2 \square\square \; {}^1 \square S_{u1} \square b_1S_{a1} \square c_1H_1\square I_0 \square\square_1\square S_{u0} \square b_1S_{a0} \square c_1H_0\square I_1 \square dt$$

$$\square_2\square S_{u1} \square b_2S_{a1} \square c_2H_1\square\overline{B}_0 \square\square 1\square B_0 \square\square\square\square_2\square S_{u0} \square b_2S_{a0} \square c_2H_0\square B_1 \square\square 1\square 2\,B_0 \square\square\square \qquad (3.141)$$

$$\_\_ \qquad \_\_\_\_$$

$$K \square \quad K \square \quad K \square \quad K \square$$

$$\square a \square\square\square\square_1 \square\square\square I_1 \square 0$$

$$\_\_\_\_$$

$$dS_{u2} \; b\,S\,I\,b\,S\,I\,b\,S\,B^{\,1}\square\square B_0 \quad \square \quad b\,S\,B\square 1\,\underline{2B_0}\square \quad \_\_$$

$$\_\_\_\_ \qquad \_\_\_\_ dR_{\,2} \square \; \square\square\square \; a\square I_1 \square \; \square\square\square\square\square R_1 \square \quad 0$$

$$\_\_\_\_$$

$$\_\_\_\_$$

$$_1dV_{\,2} \square\square \; {}^2S_{u1} \square\square_3S_{a1} \square\square\square_1 \square\square\square V_1 \square \qquad (3.143)$$

$$dt \qquad \_\_\_\_$$

(3.142) *dt*

$$dH_2 \square\square_1 S_{u1} \square\square_2 S_{a1} \square c_1 \square_1 H_1 I_0 \square\square c_1 \square_1 H_0 I_1 \square c_2 \square_2 H_1 B_0 \square\square 1 \square B_0 \square\square\square$$

$$dt \qquad K \square \quad K \square \qquad (3.144)$$

$$c_2 \square_2 H_0 B^{\overline{1}} \square 1 \square 2 \overline{B^0} \square \square\square\square\square\square\square H_1 \square 0$$

$$K \square \quad K \square$$

$$\frac{dB_2}{dt} \square\square I_1 \square \square\square\square w \square B_1 \square \quad 0 \qquad (3.145)$$

$$\frac{dM_2}{dt} \square\square I_1 \square\square M_1 \square \quad 0 \qquad (3.146)$$

Solving equation (3.123) – (3.146), we obtain

$$\square t \square\square \quad \lim_{h \square 1} \square S_{u0} \square t \square\square \quad h S_{u1} \square t \square\square \quad h^2 S_{u2} \square t \square\square ... \square\square \quad S_{u0} \quad \square \quad A_0 t \quad \square \quad A_8 \qquad (3.147)$$

$$S_u \qquad\qquad t^2$$

$$\square t \square\square \quad \lim_{h \square 1} \square S_{a0} \square t \square\square \quad h S_{a1} \square t \square\square \quad h^2 S_{a2} \square t \square\square ... \square\square \quad S_{a0} \quad \square \quad A_1 t \quad \square \quad A_9 \qquad (3.148)$$

$$S_a \qquad\qquad t^2$$

$$\square^{2} I_2 \square t \square \square \ldots \square \square I_0 \square A_2 t \square \quad A^{10} t^2 \qquad (3.149)\ I \square t \square \square \lim_h$$

$$\square I_0 \square t \square \square h I_1 \square t \square \square h \qquad {}^{1}$$

$$\frac{}{2}$$

$$\square t \square \square \lim_{h \square 1} \square R_0 \square t \square \square h R_1 \square t \square \square h^2 R_2 \square t \square \square \square \square R_0 \square A_3 t \square A$$

(3.150)

$$R \qquad \frac{}{2} \qquad {}^{11} t^2$$

$$V \square t \square \square \lim_{h \square 1} \square V_0 \square t \square \square h V_1 \square t \square \square h^2 V_2 \square t \square \square \square \square V_0 \square A_4 t \square A_{12} t^2$$

(3.151)

$$\frac{}{2}$$

$$H \square t \square \square \lim_{h \square 1} \square H_0 \square t \square \square h H_1 \square t \square \square h^2 H_2 \square t \square \square \square \square H_0 \square A_5 t \square A_{13} t^2$$

(3.152) 2

$$\frac{}{}$$

$$\square t \square \square \lim_{h \square 1} \square B_0 \square t \square \square h B_1 \square t \square \square h^2 B_2 \square t \square \square \square \square B_0 \square A_6 t \square A$$

(3.153)

$$B \qquad \frac{}{2} \qquad {}^{14} t^2$$

$$\square t \square \square \lim_{h \square 1} \square M_0 \square t \square \square h M_1 \square t \square \square h^2 M_2 \square t \square \square \square \square M_0 \square A_7 t \square A$$

(3.154) 2

$$M \qquad \frac{}{} \qquad {}^{15} t^2$$

Where

□□□□ [1] B^0 □1□ B^0 □□□□□$S_{u0}M_0$ □□□$_2$ □□$_1$ □□□S_{u0} □□1□ p□□R_0 □ qS_{a0} □□ $S_{u0}I_0$
□□$_2S_{u0}$ □

A_0 □□ K □ K □ □,

□□ □□1□ r□□$_1V_0$ □□1□ d□□H_0 □□1□ f □□ □□

□□□b $_1$□$_1S_{a0}I_0$ □b_2□$_2$ $\overline{B_0}$ □1□ $\overline{B_0}$ □□□□$S_{u0}M_0$ □□q □□$_3$ □□$_2$ □□□S_{a0} □□□

S_{a0} □

A_1 □□ K □ K □ □

□□ p□R_0 □ r□$_1V_0$ □ d□H_0 □ □

□□□$_1$□S_{u0} □b_1S_{a0} □ B^0 □1□ B^0 □□□□□

c_1H_0□I_0 □□□$_2$□S_{u0} □b_2S_{a0} □ c_2H_0□ □

A_2 □□ K □ K □ □,

□□ □a □□□□$_1$ □□□I_0 □□

A_3 □□□□□a□I_0 □□□□□□R_0□, A_4 □□f□□□$_2S_{u0}$ □□$_3S_{a0}$ □□□$_1$ □□□V_0□,

A_5 □□□□□□$_1S_{u0}$ □□$_2S_{a0}$ □c_1□$_1H_0I_0$ □c_2□$_2H_0$ $\overline{B}K_0$ □□□1□ B K_0
□□□□□□□□□□H_0□□□□ , □

A_6 □□□□M □I_0 □□□□ w□B_0□, A_7 □□□□□I_0 □□M_0□,

□□□□ [1] B^0 □1□ B^0 □□ □□$_2S_{u0}A^6$ □□1□ 2B^0 □□ □□A_0M_0 □ □

A_0I_0 □□$_1S_{u0}A_2$ □□$_2A_0$ □

A_8 □ □ K □ K □ K □ K □ □,

□□ □$S_{u0}A_7$ □ □□$_2$ □□$_1$ □□□A_0 □ □1□ p□□A_3 □ qA_1 □ □1□ r□□$_1A_4$ □ □1□

d□□A_5 □ □ □□□b □ A I □b □S I □b □ A B_0 □1□ B_0 □□ □b_2□$_2S_{a0}A_6$ □□1□ 2B_0

□□ □ □A_0M_0□ □

1 1 1 0 1 1 a 0 1 2 2 1

A_9 K K K K

$S_{u0}A_7$ q A_1 p A_3 r A_4 d A_5

A_0 b_1A_1 c_1A_5 I_0 S_{u0} b_1S_{a0} c_1H_0 A_2

A_{10} A_0 b_2A_1 c_2A_5 BK_0 1 BK_0 S_{u0} b_2S_{a0} c_2H_0 AK_6 1 2 KB_0

a A_2

A_{11} a A_2 A_3, A_{12} A_0 A_1 A_4,

A_0 A_1 $c_1A_5I_0$ $c_1H_0A_2$ $c_2A_5B_0$ 1 B_0 c_2H_0 A_6 1 $2B_0$

A_{13} K K K K,

A_5

A_{14} M A_2 w A_6, A_{15} A_2 A_7

3.9 Optimal Control Analysis

In this section, we discussed the optimal control of the model by minimizing the spread of cholera using the control parameters u_1,u_2,u_3 and u_4. Let u_i 1, for i 1,2,3,4, be linear

functions. The control measures u_1, u_2, u_3 and u_4 are very effective when u_1 u_2 u_3 u_4 1 and not effective when u_1 u_2 u_3 u_4 0. The control measure

u_1 : is the awareness campaign given to individuals to help reduce cholera; u_2 : is the vaccination strategy that can improve the immunocompetence of susceptible individuals; u_3 : is the treatment strategy that aims at increasing the recovery rate of infected individuals; u_4 :is the sanitation strategy (treatment of water bodies) that aims at killing vibrio in contaminated water.

Thus, the optimal control system now reads:

$$dt \quad \Lambda_1 \quad S_u \quad b_1 S_a \quad c_1 H \quad I \quad \Lambda_2 \quad S_u \quad b_2 S_a \quad c_2 H \quad B \quad K \quad u_3 \quad t \quad \Lambda_1 \quad I$$

$$dV$$

$$f \quad u_2 \quad t \quad S_u \quad u_2 \quad t \quad S_a \quad \Lambda_1 \quad V \quad dt \quad dR$$

$$dt \quad u_3 \quad t \quad I \quad R \quad dB \quad I$$

$$u_4 \quad t \quad B \quad dt$$

$$\frac{dM}{dt} \quad I \quad M \qquad (3.155)$$

Subject to the initial conditions

$$0 \quad S_{u0}, S_a \quad 0 \quad S_{a0}, H \quad 0 \quad H_0, I \quad 0 \quad I_0, V \quad 0 \quad V_0, R \quad 0 \quad R_0, B \quad 0 \quad S_u \quad 0 \quad M_0$$

B_0,

M

Considering the use of bounded Lebesgue measurable control (Kwasi-Do *et al*. (2020)), we define the objective function to be minimized as

$$J \quad \int_{0}^{t_F} \quad \Lambda_1 I \quad \Lambda_2 B \quad 1 \quad u_1,u_2,u_3,u_4 \; 2 \; a_1 u_1^2 \quad \frac{1}{2} a_2 u_2^2 \quad \frac{1}{2} a_3 u_3^2 \quad \frac{1}{2} a_4 u_4^2 \quad dt$$

$$0, t_F$$

(3.156) where is the time period of intervention. The dynamics of the controls to minimize the objective function is given by system (3.155). The associated basic reproduction number for system (3.155) is given as:

where

We minimize the infectious human population (I) and bacteria (B), as well as the cost involved in implementing the control measures u_1, u_2, u_3 and u_4. The functional objective

includes the social cost which relates to enlightening susceptible individuals $a_1 u_1^2$,

$1 - u_1$, treating infected individuals $a_3 u_3^2$ and vaccinating susceptible individuals $a_2 u_2$

2 2

1 ². The quantity $\frac{1}{2}$ represents the cost associated with

treatment of water bodies a_4u_4 2

minimizing the infected human population, and ² represents the cost associated with minimizing the bacteria population in the environment. The costs corresponding to I and

1 ², 1 $a_2u_2^2$, 1

$a_3u_3^2$ and 1 $a_4u_4^2$ are $_2B$ are linear, whereas the cost

control functions a_1u_1

2 2 2

2

nonlinear. The Lagrangian of the optimal control problem is given by

$$\_\_{}_1I \_\_{}_2B \_ \frac{1}{2} a_1u_1^2 \_\_ \frac{1}{2} a_2u_2^2 \_ \frac{1}{2} a_3u_3^2 \_ \frac{1}{2} a_4u_4^2 \_ \quad (3.158)$$

L

2 2 2 2

To determine the Lagrangian minimum value, the Hamiltonian, H^*, for the control problem is defined as

$$* \quad I \square\square_2 B \square 1 a_1 u_{12} \square\square 1 a_2 u_{22} \square 1 a_3 u_{32} \square 1 a_4 u_{42} \square v_{Su} dS_u \square \quad v_{Sa} dS_a \square$$
$$\square\square_1 \qquad\qquad\qquad\qquad\qquad\qquad\qquad\qquad\qquad\qquad\qquad\qquad H$$

$$-$$

$$-$$

$$-$$

$$-$$

$$\frac{dR}{dt}\frac{dB}{dt}\frac{dM}{dt}\underline{v_H} \square v_I \frac{\quad}{\quad} \square v_V \frac{\quad}{\quad} \square v_R \frac{\quad}{\quad} \square v_B \quad \square \quad v_M \quad \overline{2}\,\overline{2} \quad 2 \quad 2 \quad dt \quad dt \quad \overline{\quad} \quad \overline{\quad} \quad (3.159)\ dH \quad dI \quad dV$$

where v_{Su}, v_{Sa}, v_H, v_I, v_V, v_R, v_B, and v_M are the adjoint variables. The differential equations of the adjoint variables are obtained by taking the partial derivatives of the Hamiltonian equation, that is, equation (3.162), with respect to the state variables which gives

□ □ □□ 1□ *f* □□□□1*SuI* □□2*Su* *B* □*u*1□*t*□*SuM* □□ □ □

□ □ *B*□ *K* □ □

□*vSu* □□1□ *p*□□*R*□*qSa* □□1□*r*□□1*V* □□1□*d*□□*H* □ □□ □

□ □ □ □

□ □□□*u*2□*t*□□□1 □□□*Su* □□ □ □

□ □ □

□ □

□*vSa* □□□*u*1□*t*□*SuM* □*b*1□1*SaI* □*b*2□2*Sa B*□*BK* □ *p*□*R*□□□□□ □

□

□

dvdtSu □□*HSu** □□ □□*Su* □□□ □*vH* □□□□□□ *r*□1*S*1*Vu* □□□*d*□2*SHa*

□□□*cq*1□□1*HIu*2□*t*□□*c*□2□□22*H*□□*B*□□*SBaK* □□□□□ □□ □□ □ □

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□ □□□□□□*H* □□ □

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□ □

□□*vI* □□□□1□*Su* □*b*1*Sa* □*c*1*H*□*I* □□2□*Su* □*b*2*Sa* □*c*2*H*□*B*□*BK* □□□□□

□ □

□□ □□ □u_3□t□□□□□$_1$ □□□I □□ □ □

□ □□□□u_3□t□□I □□ □

□ □

□□ *vv*$_{VB}$

□□□fI□□□□□u_2□□tu□S_4□$_{ut}$□□□Bu_2□□□t□vS_{Ma}□□□□□□□$_1$□□I

□□□□VM□□□v_R□□□□□□□R □□□□□□□ □ (3.160)

□

□$_{VB}$ □□$_I$ □

□

□ □ □ 1□ *f* □□□□1*SuI* □□2*Su* *B* □*u*1□*t*□*SuM* □□ □

□

———

□ □ *B*□ *K* □ □

□*vSu* □□1□ *p*□□*R*□*qSa* □□1□*r*□□1*V* □□1□*d*□□*H* □ □□ □

□ □ □ □

□ □□□*u*2□*t*□□□1 □□□*Su* □□ □ □

□ □ □

□ □

□*vSa* □□□*u*1□*t*□*SuM* □*b*1□1*SaI* □*b*2□2*Sa B*□*BK* □ *p*□*R*□□□□□ □ □

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□

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dv □*H** □ □ □

Sa□□ □□ □*Sa* □□□*vH* □□□□1*Su* □□2*Sa* □*c*1□1*HI* □*c*2□2*H B*□*BK* □□□□□ □ □ □

dt □*Sa*

□ □□□□□□*H* □□ □

□ □

□ □□1□*Su* □*b*1*Sa* □*c*1*H*□*I* □□2□*Su* □*b*2*Sa* □*c*2*H*□ *B* □□□ □ □

□*vI* □□*B*□ *K* □□

□□ □□*u*3□*t*□□□□□1 □□□*I* □□ □ □

□

□ □□□□*u*3□*t*□□□*I* □□ □

□*v* □*f u* □*t*□*S* *u* □*t*□*S* □ □*V*□ *v* □

V□□ 2 *u*□ 2 *a*□ □1 □□ □ *R*□□ □□□

□ □ □ □□

□*vB* □□*I* □

□

□ □□□□□□R □ □ (3.161)

□ v_B □□I □□□□u4□t□□B□□v_M□□□□I □□M □ □
□ □
□ □□ 1□ f□□□□1SuI □□2Su B □u1□t□SuM
□□□
□
□ □ B□ K □ □
□v_{Su} □□1□ p□□R□qSa □□1□r□□1V □□1□d□□H □ □
□□ □
dv H* □ □ □ □
H □ □□ □□□□u2□t□□□1 □□□Su □□□ □
□□
□ □
□ □□u1□t□SuM □b1□1SaI □b2□2Sa B □ □
p□R□□□ □
□v_{Sa}□ B□ K □□ □
□ □ □r□1V □d□H □□q□u2□t□□□2 □□□Sa □
□□ □
□ □
□
□ □ B □
□□
dt □H □H □v □□1Su □□2Sa □c1□1 HI □c2□2H B K □□□
□ □ □r□1V □d□H □□q□u2□t□□□2 □□□Sa □□ □

□ □ □ □□

□v_B □□I □
□

□v_V □f□□u_2□t□S_u □u_2□t□S_a □□□$_1$ □□□V□□v_R□□□□□□R

□□□□ 3.162)

□ (

□□□□□u_4□t□□B□□v_M□□□□I □□M□

□□ □□ 1□ f□□□□□$_1S_uI$ □□$_2S_u$ B □u_1□t□S_uM □□□ □ □

□

_____

□ □ B□ K □ □

□v_{Su} □□1□ p□□R□qS_a □□1□r□□$_1V$ □□1□d□□H □ □□ □

□ □ □ □

□ □□□u_2□t□□□$_1$ □□□S_u □□ □ □

___ ____ __ □ □ □

□ □

□ □□u_1□t□S_uM □b_1□$_1S_aI$ □b_2□$_2S_a$ B □ p□R□□□ □

_____

□ v_{Sa} □ B□ K □□ □

□□ □□r□$_1V$ □d□H □□q□u_2□t□□□$_2$ □□□S_a □□ □□

_____

_____

□ □ □ □□

□v_B □□I □ _____

□

dv H^*

$_I$

dt I I v_H $_1S_u$ $_2S_a$ c_1 $_1HI$ c_2 $_2H$ B BK

H

$_1$ S_u b_1S_a c_1H I $_2$ S_u b_2S_a c_2H B

v_I B K

u_3 t $_1$ I

u_3 t I

v f u t S u t S V v

$_V$ $_{2u}$ $_2$ $_a$ $_1$ $_R$

R (3.163)

v_B I u_4 t B v_M I M

1 f $_1S_uI$ $_2S_u$ B u_1 t S_uM

B K

v_{S_u} 1 p R qS_a 1 r $_1V$ 1 d H

u_2 t $_1$ S_u

dv

v_B I

$u_1 t S_u M b_1 {}_1 S_a I b_2 {}_2 S_a \quad B$

$p R$

$v_{S_a} \quad B \quad K$

$r {}_1 V \; d H \; q u_2 t {}_2 \; {}_2 S_a$

H_*

$V \quad S_u \; {}_2 S_a \; c_1 {}_1 HI \; c_2 {}_2 H \quad B$

$dt \quad V \quad V \; v \; {}_1 \quad B \quad K$

$B \quad K$

$v_{S_u} \; 1 \; p \; R \; q S_a \; 1 \; r \; {}_1 V \; 1 \; d \; H$

$u_2 t \; {}_1 \; S_u$

$v_{S_a} \; u_1 t S_u M b_1 {}_1 S_a I \; b_2 {}_2 S_a B \; BK \; p R$

$r {}_1 V \; d H \; q u_2 t {}_2 \; {}_2 S_a$

$v_B \; I$

□□ □□ dv^R □ H^*□ □ □□S □□ S □c □HI □c □ H B □□ □

dt □R □R□□ v_H□□ 1 u 2 a 1 1 2 2 B□ K □□□ □ □

□ □□□□□□□□H □□ □

□ □

□ □□□$_1$□S_u □b_1S_a □c_1H□I □□□$_2$□S_u □b_2S_a □c_2H□B □□□□ □ □
□v_I □□ B□ K □□

□ □□u_3□t□□□□□□$_1$ □□□I □□ □ □

□ □

□ □□□□□u_3□t□□I □□ □

□v □f u □t□S u □t□S □ □V□ v □

$_V$□□ $_2$ $_u$□ $_2$ $_a$□ □$_1$ □□ □ $_R$□□ □□□

□ □□□□□□R □ □ (3.165)

□ v_B □□I □□□□□u_4□t□□B□□v_M□□□□I □□M □ □

□ □

□ □□□1□ f□ □□□□$_1S_uI$ □□$_2S_u$ B □u_1□t□S_uM □□□

□ □

□ □ B□ K □ □

□v_{Su} □□1□ p□□R□qS_a □□1□r□□$_1V$ □□1□d□□H □ □

□ □ □

□ □□□u_2□t□□□$_1$ □□□S_u □□

□ □ □

□

□ □□u_1□t□S_uM □b_1□$_1S_aI$ □b_2□$_2S_a$ B □ p□R□□□

□v_{Sa}□ B□ K □□

□ □ r□$_1V$ □d□H □□q□u_2□t□□□$_2$ □□□S_a □□ □

dv H^* □ □

□ □ □ □□□

□v_B □□I □

□

$_B$ □□□ □□ □ □ □ $_1S_u$ □□ S_a □c_1□$_1HI$ □c_2□$_2H$ B □□□

dt □B □B□v □ $_2$ B K □

□v_V □f□□u_2□$_t$□S_u □u_2□$_t$□S_a □□□$_1$ □□□V□□v_R□□□□□□R

□□□□ (3.166)

□

□□□□u_4□$_t$□□B□□v_M□□□□I □□M □

□ □ □ □□

□v_B □□I □

□

□□ □□□ 1□ *f* □□□□1*SuI* □□2*Su* *B* □*u*1□*t*□*SuM* □□□ □ □

———

□ □ *B*□ *K* □ □

□*vSu* □□1□ *p*□□*R*□*qSa* □□1□*r*□□1*V* □□1□*d*□□*H* □ □□ □

□ □ □ □

□ □□□*u*2□*t*□□□1 □□□*Su* □□ □ □

□ □ □

□ □

□*vSa* □□□*u*1□*t*□*SuM* □*b*1□1*SaI* □*b*2□2*Sa B*□*BK* □ *p*□*R*□□□□□□ □

———

□

* □□ □□*r*□1*V* □*d*□*H* □□*q*□*u*2□*t*□□□2 □□□*Sa* □□ □□

dvdtM □□□□*HM* □□ □□*M* □□*vH* □□□□1*Su* □□2*Sa* □*c*1□1*HI* □*c*2□2*H B*□*BK* □□□□□ □ □ □

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□

□ □□□□□□*H* □□ □

□ □

□□*vI* □□□□1□*Su* □*b*1*Sa* □*c*1*H*□*I* □□2□*Su* □*b*2*Sa* □*c*2*H*□*B*□*BK* □□□□□ □ □

———

□□ □□ □*u*3□*t*□□□□1 □□□*I* □□ □ □

□ □□□□*u*3□*t*□□*I* □□ □

□□

Simplifying (3.163) – (3.167) yields

$$\frac{dv_{Su}}{dt} = -\frac{v_{Su}}{} + \frac{I}{} + \frac{1}{} - \frac{B}{K} - \frac{u_1 M}{} - \frac{v_{Sa} - v_{Su}}{} - \frac{u_2}{} - \frac{v_V - v_{Su}}{}$$

$$\frac{dv_{Sa}}{dt} = -\frac{v_{Sa}}{} + \frac{I}{} + \frac{b_1 - 1}{} + \frac{b_2 - 2}{} - \frac{B}{K} - \frac{q}{} - \frac{v_{Sa} - v_{Su}}{} - \frac{u_2}{} - \frac{v_V - v_{Sa}}{}$$

$$\frac{dv_H}{dt} = -\frac{v_H - v_{Sa}}{} + \frac{c_1 - 1}{} + \frac{I}{} + \frac{c_2 - 2}{} - \frac{B}{K} - \frac{d}{} - \frac{v_{Sa} - v_{Su}}{}$$

$$\frac{dv_I}{dt} = -\frac{v_I - v_{Su}}{} - \frac{1}{} - \frac{S_u}{} + \frac{b_1 - 1}{} - \frac{S_a}{} + \frac{v_I - v_{Sa}}{}$$

$$\frac{dv_H}{dt} = -\frac{c_1 - 1}{} - \frac{H}{} + \frac{v_I - v_H}{} - \frac{u_3}{} - \frac{v_R - v_I}{} - \frac{1}{}$$

□□□

—

□v_B □□v_M□□□$_1$ □□□v_I □ □ dv

dt $_V$ □□ r□$_1$□v_{Sa} □v_{Su} □□□$_1$□v_V □v_{Su} □□□v_V □ □ dv^R □□

p□□v_{Sa} □v_{Su} □□□□v_R □v_{Su} □□□v_R □ □ dt

□□□ dv_B □v_I □v_{Su} □□$_2S_u$ □B□KK□$_2$ □□v_I □v_{Sa} □b_2□$_2S_a$ □B□KK□

$_2$□ □ □ □

dt $_2$□

—— ————

————

□ K

□ □v_I □v_H □c_2□$_2H$ □B□ K□ $_2$ □□□□u_4□v_B □ (3.168)

————

dv_M □□ u_1S_u □v_{Sa} □v_{Su} □□□v_M □ □ dt □

——

Theorem 3.8: Given the optimal controls u_1,u_2,u_3 and u_4 . and the solutions

S_u ,S_a ,H,I,V,R,B,and M of corresponding state equation (3.168) which minimizes the objective function J u_1,u_2,u_3,u_4□over the region Ω, then there exist adjoint variables v_{Su}, v

$_{Sa}$, v_H, v_I, v_V, v_R, v_B, and v_M satisfying

dv_i □□ □H_* , i□□

(3.169) dt □i

—— —— S_u ,S_a ,H,I,V,R,B,M□

Thus, the optimal solutions u_1^* , u_2^* , u_3^* and u_4^* are given by u_1^*

□ min□1,max 0,□u_1 □□□

* □ minmin□□

(3.170)

□

u_3 □

u_4^* □ min□1,max□0,u_4□□□□ 2* 11,,maxmax

□□00,,uu_{32}□□□□□□ □

Proof: Differentiating the Hamiltonian with respect to the different control measures u_1,u_2,$u_3$and u_4 and equating them to zero, we obtained

□H^*

—— □ a_1u_1 □ □v_{Sa} □ v_{Su} □S_uM □ 0

(3.171)

□u_1

□H^* □ a_2u 2

□u_2 □ □v_V □ v_{Su} □S_u □ □v_V □ v_{Sa} □S_a □ 0

(3.172)

——

□H^*

□u_3 □ a_3u_3 □ □v_R □ v_I □I □

—— 0 (3.173)

□H^*

——□u_4 □ a_4u_4 □ v_BB □ 0 (3.174)

Making u_1,u_2,$u_3$and u_4 the subject from equations (3.171) - (3.174) respectively, gives

$$u_1 = \frac{\nu_{S_u}\nu a_{S1_a} S_u M}{\nu_V \nu_{S_u} S_u + \nu_V \nu_{S_a} S_a}$$

$$u_2 = \frac{a_2}{\nu_R \nu_I I}$$

$$u_3 = \frac{a_3 u_4 \nu_B B}{a_4}$$

(3.175)

Therefore, u_1,u_2,$u_3$and u_4 are used in MAPLE to make simulations.

CHAPTER FOUR

4.0 RESULTS AND DISCUSSION

4.1 Analysis of Results

In this thesis, an epidemic model for the transmission dynamics of cholera was formulated and analysed. The main objective of this study was to assess the impact of the incorporated control strategies on the transmission dynamics of the disease. Numerical simulations of

model system (3.1) – (3.8) are carried out using a set of parameter values given in Tables 3.4 and 3.5. MAPLE 17 version is used in the numerical simulations.

Since most of the parameters values were not readily available; we used data from literature and the missing data were estimated (Appendix). Tables 3.4 and 3.5 show the set of parameter values which were used.

4.2 Graphical Presentation of Results and Discussion

We performed the numerical simulations of the system of differential equations (3.1) – (3.8) of the susceptible unaware humans, susceptible aware humans, hygiene conscious humans, vaccinated humans, recovered humans, and the infectious humans to determine the changes in the various populations of these compartments with time.

4.2.1 Reproduction number graphs

Graphical representations showing the variations in reproduction numbers with respect to contact rate between the susceptible and the contaminated environment are provided in Figures 4.1 – 4.3.

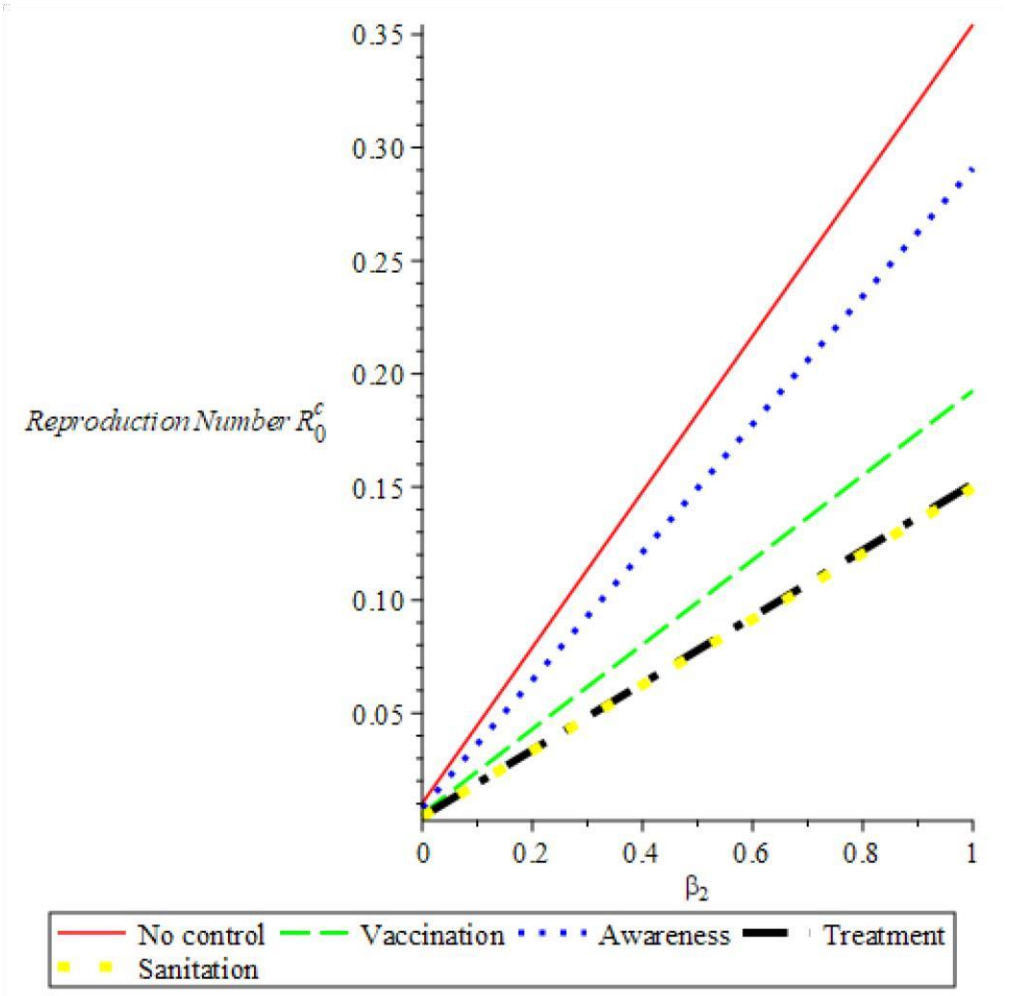

Figure 4.1: Variations of a mono-control reproduction number with respect to humanenvironment contact rate

Figure 4.1 shows that, $R_0^S \leq R_0^T \leq R_0^A \leq R_0$. We see from the figure that R_0 is worst case scenario, it occurs when there is no any control strategy for the epidemic. The basic reproduction number R_0 grows very sharp with respect to an increase in human-environment contact rate. Such an increase in R_0 above unity implies that there is a high eruption of cholera in the community.

The best case scenario is R_0^S which takes account for water sanitation. It is noted that treating water with chlorine plays an important role in combating cholera as compared to vaccination.

This is because addition of chlorine in water kills the *vibrio cholera* virus whereas vaccinating individual just boost immunity of an individual, it is clear from literature that no vaccine is perfect, the vaccines usually wanes with time and thus previously vaccinated individual might be easily infected with the disease if the vaccine has already expired (Andrews and Basu, 2011). Another concern about vaccination is coverage; it is practically not easy to attain mass vaccination because of several reasons; including financial constraints and infrastructure constraints. That governments opt to offer clean water to its individuals because it is not only cheaper but also is healthier than vaccination. In so doing they tend to limit the eruption of the disease.

The next to the best case scenario occurs at graph R_0^T, here treatment is the only intervention offered to infected individuals. It can be noticed that the reproduction number with treatment strategy is very small indeed less than unity, which means that the disease dies out. After treatment individuals recover, since recovery is not permanent the recovered individuals might become susceptible again to the disease.

The next to best case scenario after vaccination occurs at graph R_0^A, here awareness campaign is emphasised to individuals. This includes awareness on self-hygiene, importance of using toilets, drinking boiled water, humans not to contaminate water, use of oral salts to help already infected individuals, avoiding direct contact to infected individuals etc. It can be noticed from Figure 4.1 that awareness campaign is important because individual awareness about the disease limit the spread of the epidemic better.

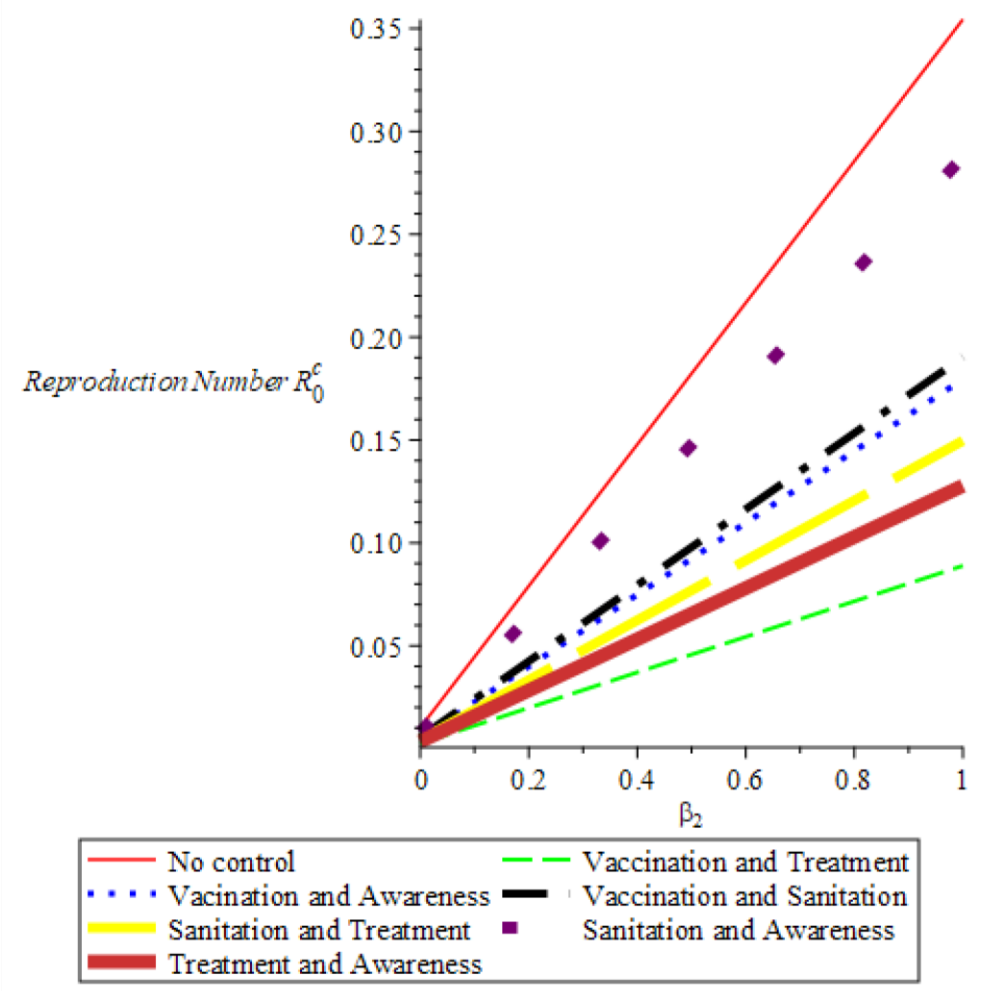

Figure 4.2: Variations of a bi-control reproduction numbers with respect to humanenvironment contact rate

Clearly, we observe in Figure 4.2 that there is a drastic reduction in disease as compared to Figure 4.1. Our simulations with bi-controls lead to the inequality:

$R_0^{VT} < R_0^{TA} < R_0^{ST} < R_0^{VA} < R_0^{VS} < R_0^{SA}$ as seen from Figure 4.2. It is obvious that R_0^{SA} is worst case scenario, it occurs when a combination of sanitation and awareness as control strategies to limit the epidemic cholera. It is at the peak, this implies that there is a high increase in

reproduction number with respect to human-environment contact rate. Such an increase results in the eruption of cholera in the community.

The best case scenario occurs at graph R_0^{VT}, here two control strategies namely vaccination and treatment are incorporated. The next to best case scenario occurs at graph R_0^{TA}, in which the combination of treatment and awareness were considered, followed by R_0^{ST} which is a combination water sanitation and treatment. Furthermore, we have R_0^{VA}, which is a combination of vaccination and awareness, followed by R_0^{VS} which is a combination of vaccination and water sanitation.

From Figure 4.2, it can be further seen that most of the reproduction numbers are less than 0.20; this implies that there is a good control of the disease. Therefore, increasing the number of controls together with their associated parameters values yield a rapid decay of the reproduction number curves. This means that the disease is not endemic and it dies out. We can therefore conclude that combination of two control strategies is better than one control strategy as it yields better results in diminishing cholera from the community.

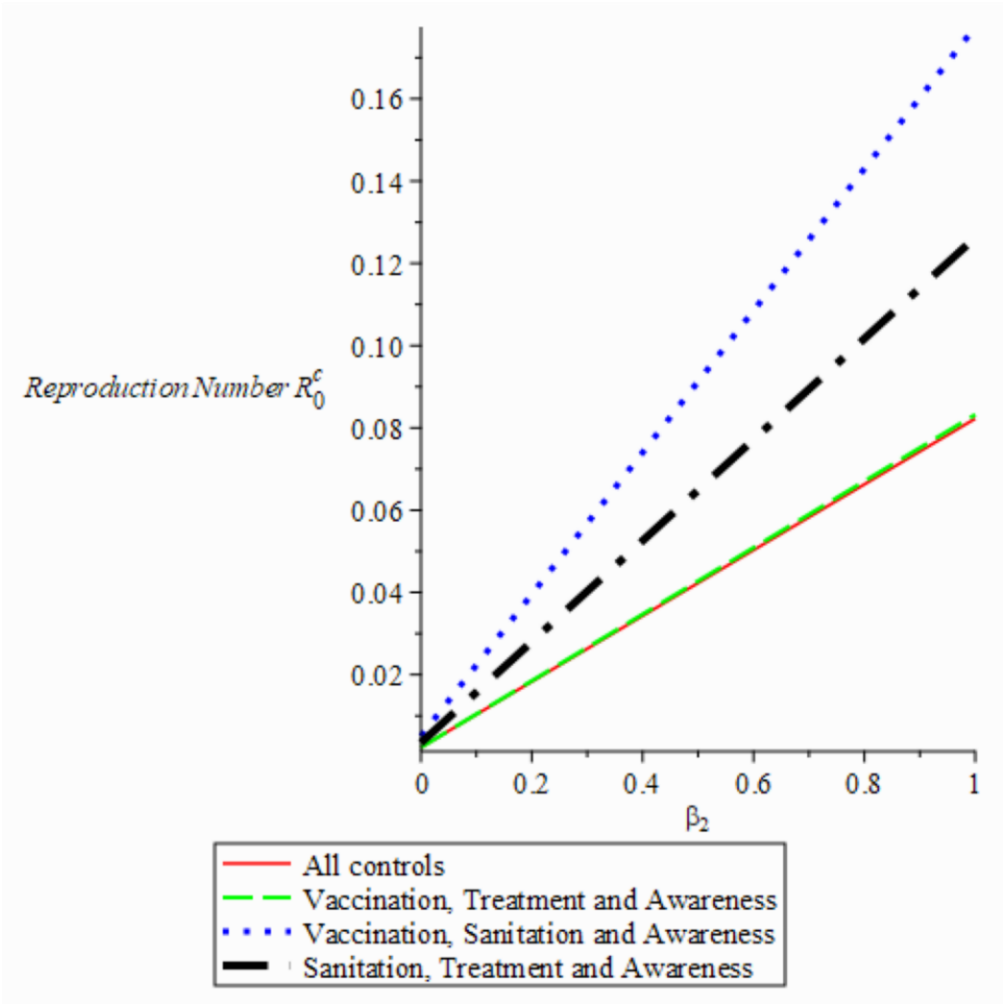

Figure 4.3: Variations of a tri-control reproduction numbers with respect to humanenvironment contact rate

Clearly, we observe in Figure 4.3 that there is a drastic reduction in disease as compared to both Figure 4.1 and Figure 4.2, where most of the reproduction numbers are far less than unity. This implies that there is a control of the disease. Three controls give results that are marked obtained with unique control and a combination of two interventions. Therefore, increasing the number of controls together with their associated parameters values yield a rapid decay of the reproduction number curves. This means that disease is not endemic and it dies out.

Our simulations with tri-controls lead to the inequality: $R_0^c R_0^{VTA} R_0^{STA} R_0^{VSA}$ as seen from Figure 4.3. It is obvious that R_0^{VSA} is worst case scenario, which is a combination of vaccination, sanitation and awareness as control strategies to limit the epidemic cholera. R_0^{VSA} is at the peak, this implies that there is a high increase in reproduction number with respect to respect to human-environment contact rate. Such an increase results in the eruption of cholera in the community.

With regard to the three controls, the best case scenario occurs at graph R_0^{VTA}, where three control strategies such as vaccination, treatment and awareness are incorporated. The next to best case scenario occurs at graph R_0^{STA}, in which the combination of sanitation, treatment and awareness were considered. Furthermore, we have R_0^{VSA}, which is a combination of vaccination, sanitation and awareness.

From Figure 4.3, it can be further seen that most of the reproduction numbers are less than 0.20, which implies that there is a good control of the disease. Therefore, increasing the number of controls together with their associated parameters values yield a rapid decay of the reproduction number curves. This means that the disease is not endemic and it dies out. It is obvious from Figure 4.3, reproduction number with all four controls, R_0^c is the least and of course the best strategy among all controls.

We can therefore conclude that the more you increase combination of control strategies the better you control cholera.

4.2.2 Sensitivity analysis graphs

Graphical representations showing how "sensitive" a model is to changes in the value of the parameters of the model and to changes in the structure of the model are provided in Figures

4.5 – 4.10.

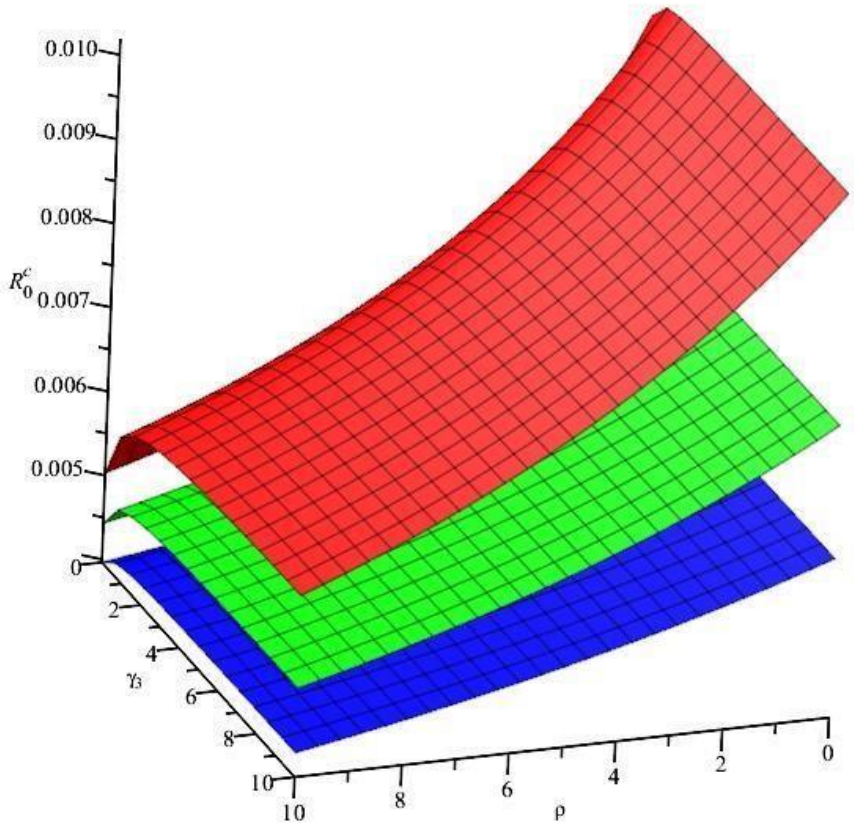

Figure 4.4: Sensitivity of the reproduction number R_0^c to the parameters β_2 and d at different values of β_2

Figure 4. γ_2,

4 showed that increase in the value of vaccination rate for unaware humans, resulted into a decrease in the reproduction number, which may reduce the persistence of cholera in the population. Also in Figure 4.4, it can be seen that an increase in the awareness rate, a will lead to decrease in the reproduction number and increase in the vaccination rate for aware human, γ_3 will lead to decrease in the reproduction number.

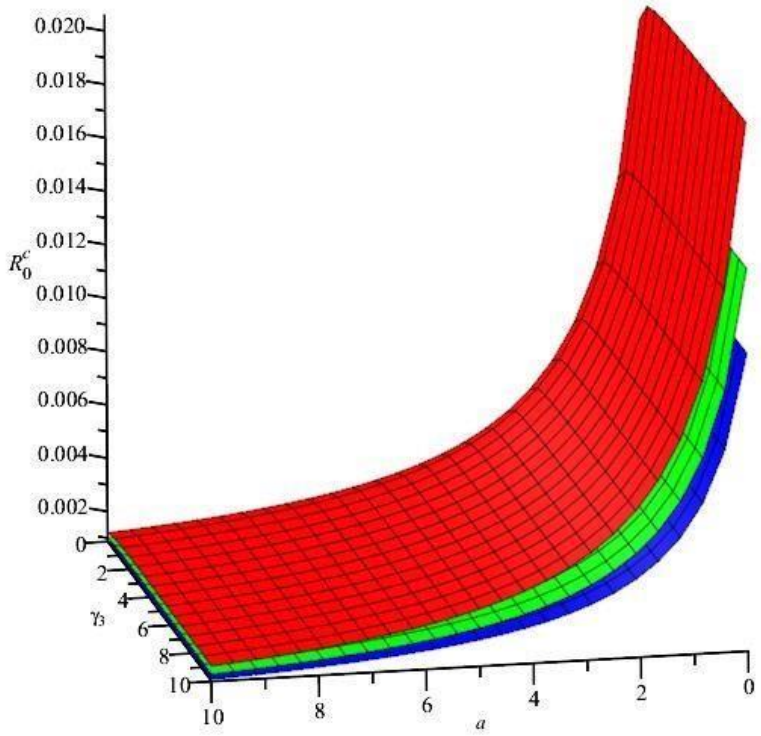

Figure 4. $\square_2$,

Figure 4.5: Sensitivity of the reproduction number R_0^c to the parameters $\square$ and a at different values of $\square_2$

5 showed that increase in the value of vaccination rate for unaware humans, resulted into a decrease in the reproduction number, which may reduce the persistence of cholera in the population. Also in Figure 4.5, it can be seen that an increase in the treatment rate, a , will lead to decrease in the reproduction number and increase in the vaccination rate for aware human, $\square_3$, will lead to decrease in the reproduction number.

Figure 4. $\square_2$,

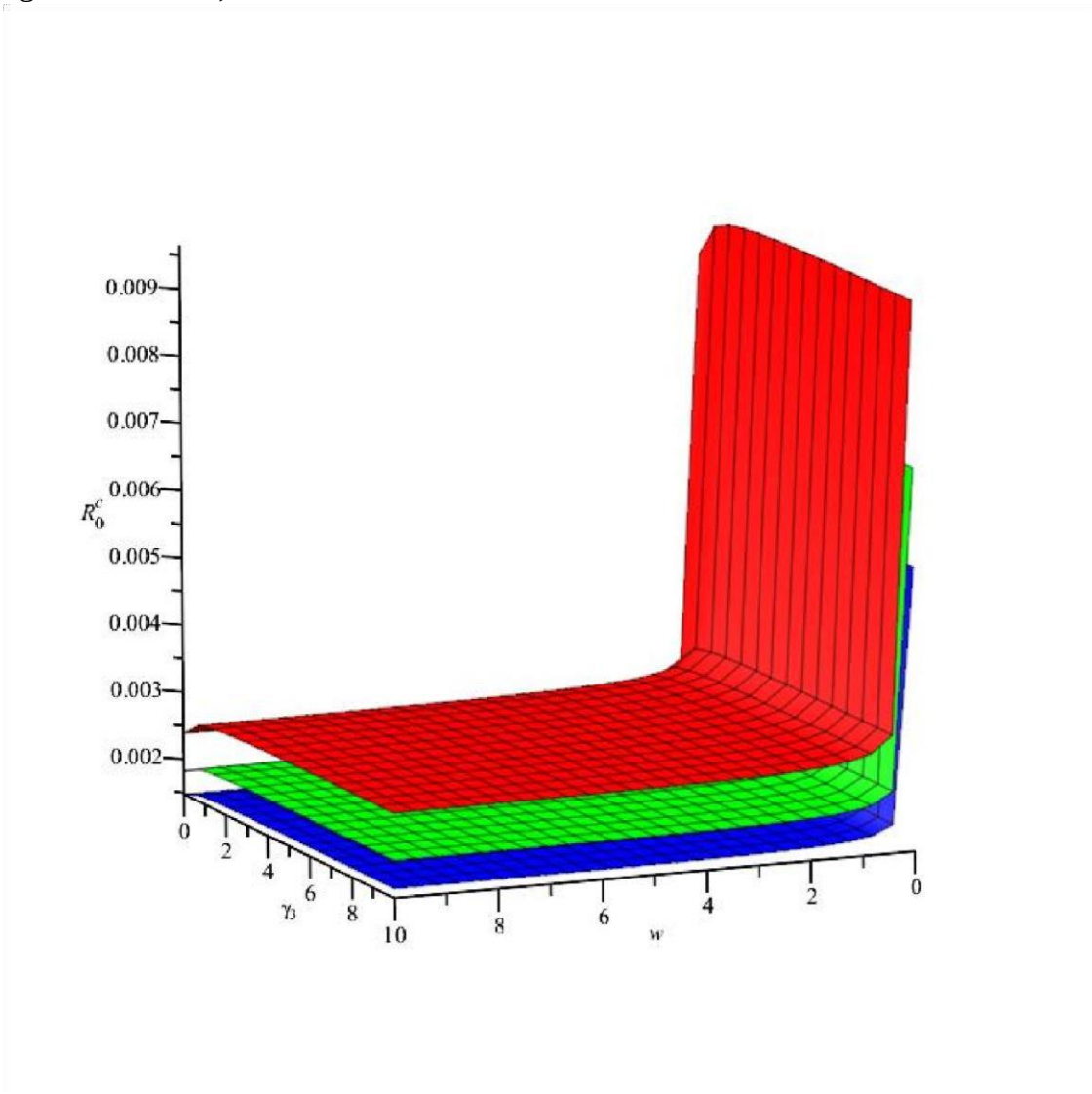

Figure 4.6: Sensitivity of the reproduction number R_0^c to the parameters $\square_3$ and w at different values of $\square_2$

6 showed that increase in the value of vaccination rate for unaware humans, resulted into a decrease in the reproduction number, which may reduce the persistence of cholera in the population. Also in Figure 4.6, it can be seen that an increase in the disinfection rate, w , will lead to decrease in the reproduction

$\square$

Figure 4. $\square_2$,
number and increase in the vaccination rate for aware human, $_3$, will lead to decrease in the reproduction number.

Figure 4. $\square_2$,

$\square$

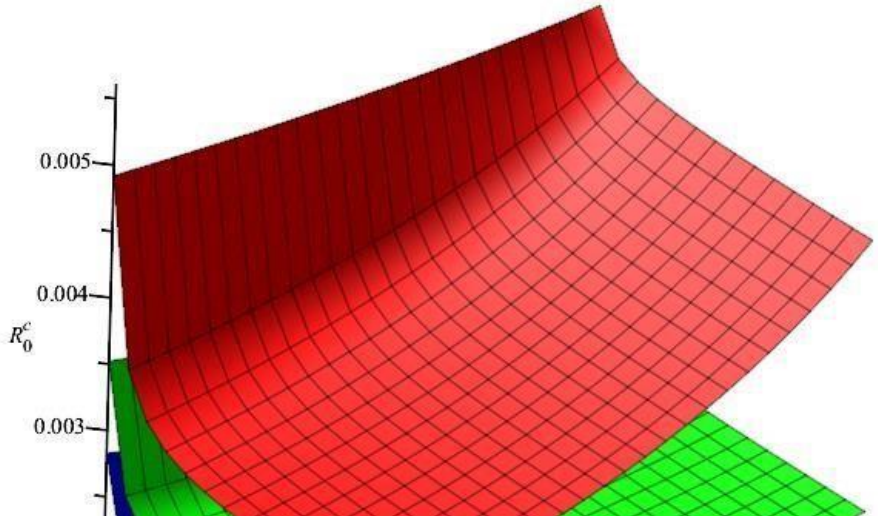

Figure 4. $\square_2$,

Figure 4. hygiene conscious rate for unaware humans,

θ_1

7 showed that increase in the value of

, resulted into a decrease in the reproduction number, which may reduce the persistence of cholera in the population. Also in Figure 4.7, it can be seen that an increase in the awareness rate, , will lead to decrease in the reproduction number and increase in the hygiene conscious rate for aware human, $_2$, will lead to decrease in the reproduction number.

Figure 4. hygiene conscious rate for unaware humans,

$\square_1$

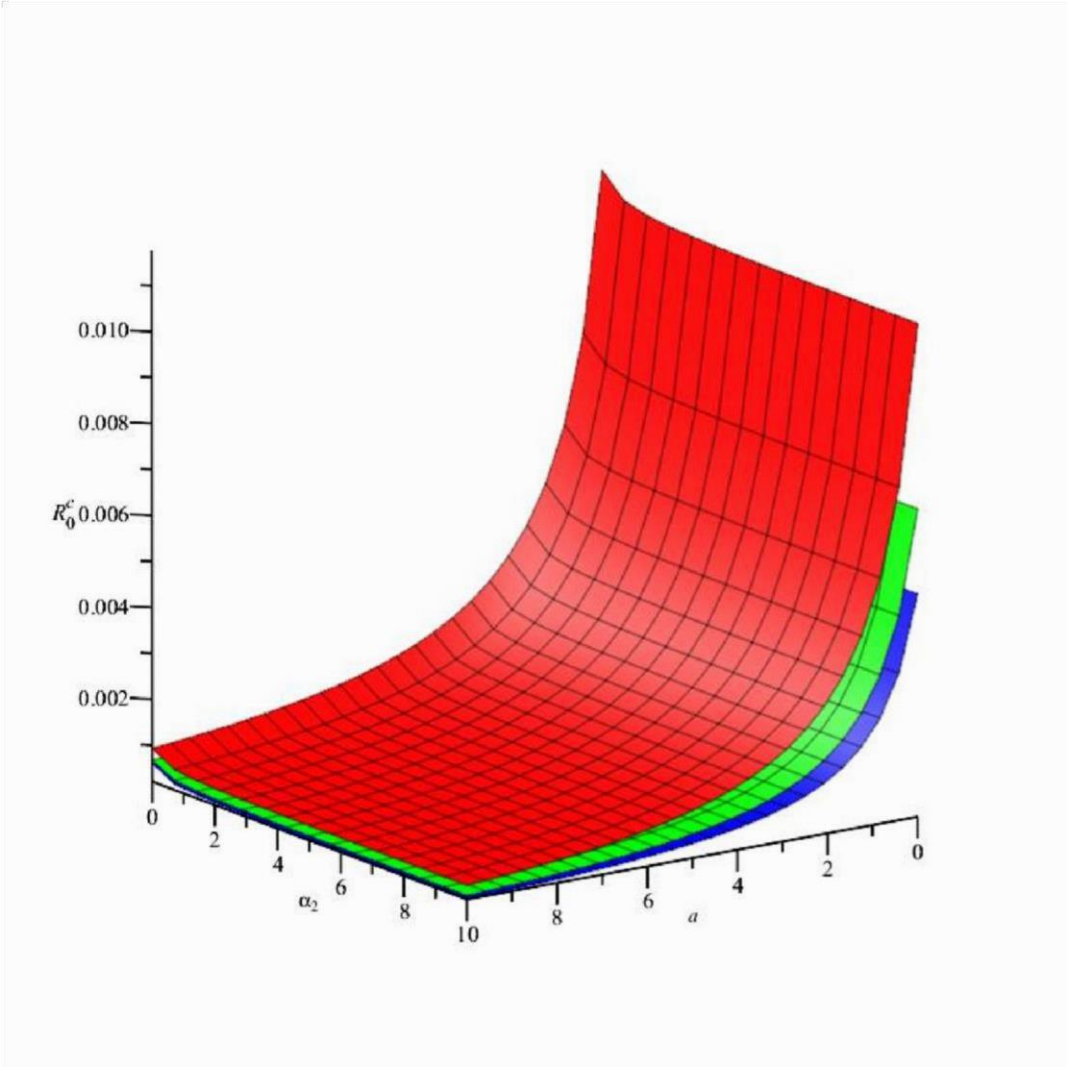

Figure 4.8: Sensitivity of the reproduction number R_0^c to the parameters $\square_2$ and a at different values of $\square_1$

8 showed that increase in the value of

, resulted into a decrease in the reproduction number, which may reduce the persistence of cholera in the population. Also in Figure 4.8, it can be seen that an increase in the treatment

Figure 4. hygiene conscious rate for unaware humans,

$\square_1$

rate, a , will lead to decrease in the reproduction number and increase in the hygiene conscious rate for aware human, $\square_2$, will lead to decrease in the reproduction number.

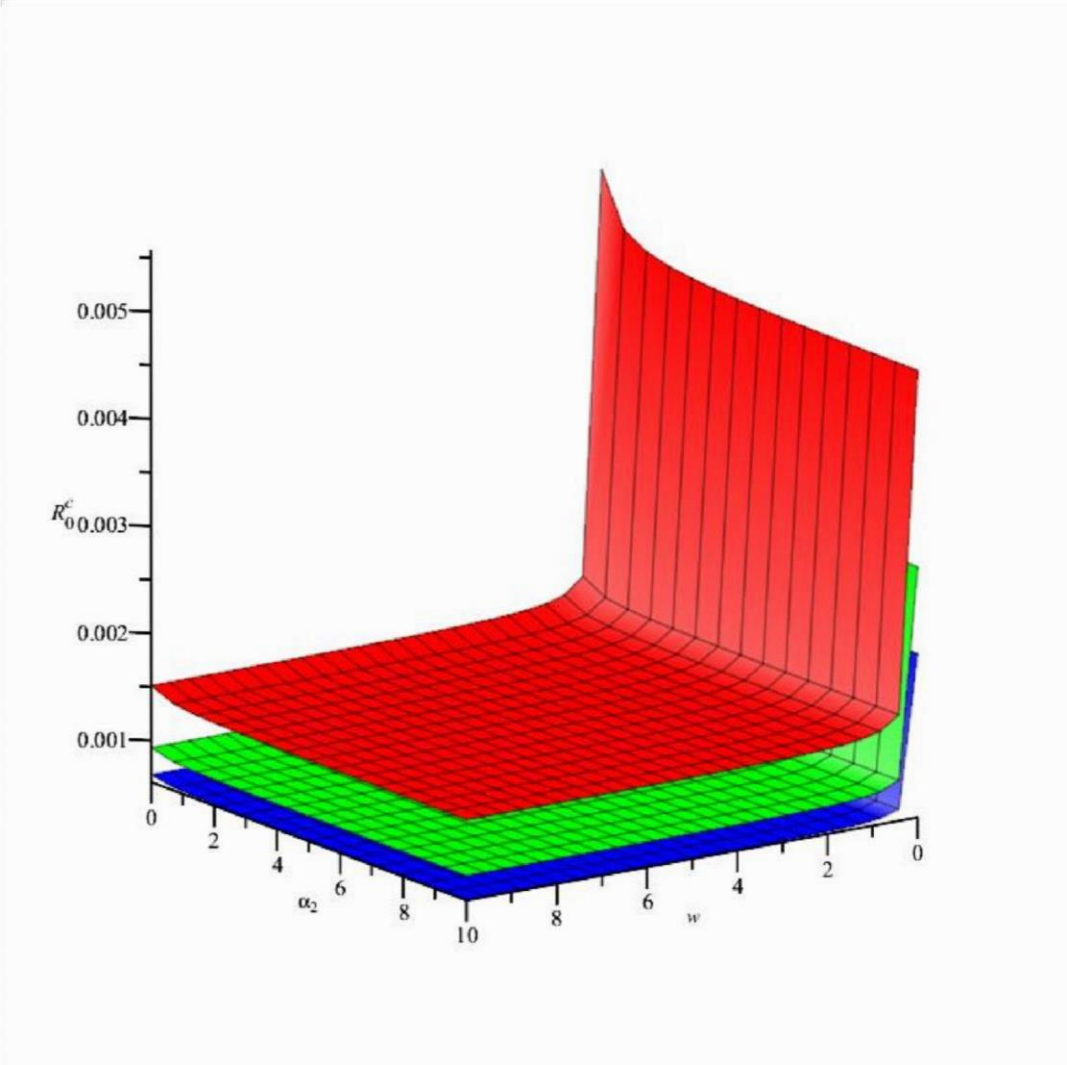

Figure 4.9: Sensitivity of the reproduction number R_0^c to the parameters $\square_2$ and w at different values of $\square_1$

Figure 4. hygiene conscious rate for unaware humans,

$\square_1$

9 showed that increase in the value of , resulted into a decrease in the reproduction number, which may reduce the persistence of cholera in the population. Also in Figure 4.9, it can be seen that an increase in the disinfection rate, w , will lead to decrease in the reproduction number and increase in the hygiene conscious rate for aware human, $_2$, will lead to decrease in the reproduction number.

4.2.3 HPM Simulation graphs

Graphical representations showing the variations in human population and bacteria concentration with respect to time are provided in Figures 4.10 – 4.18. Graphical representations showing the variations in human population with respect to time are provided in Figures 4.10 – 4.16. Figure 4.17 showed a change in the concentration of the bacteria in the environment while Figure 4.18 is the graph of number of the awareness program driven by disease prevalence and media coverage.

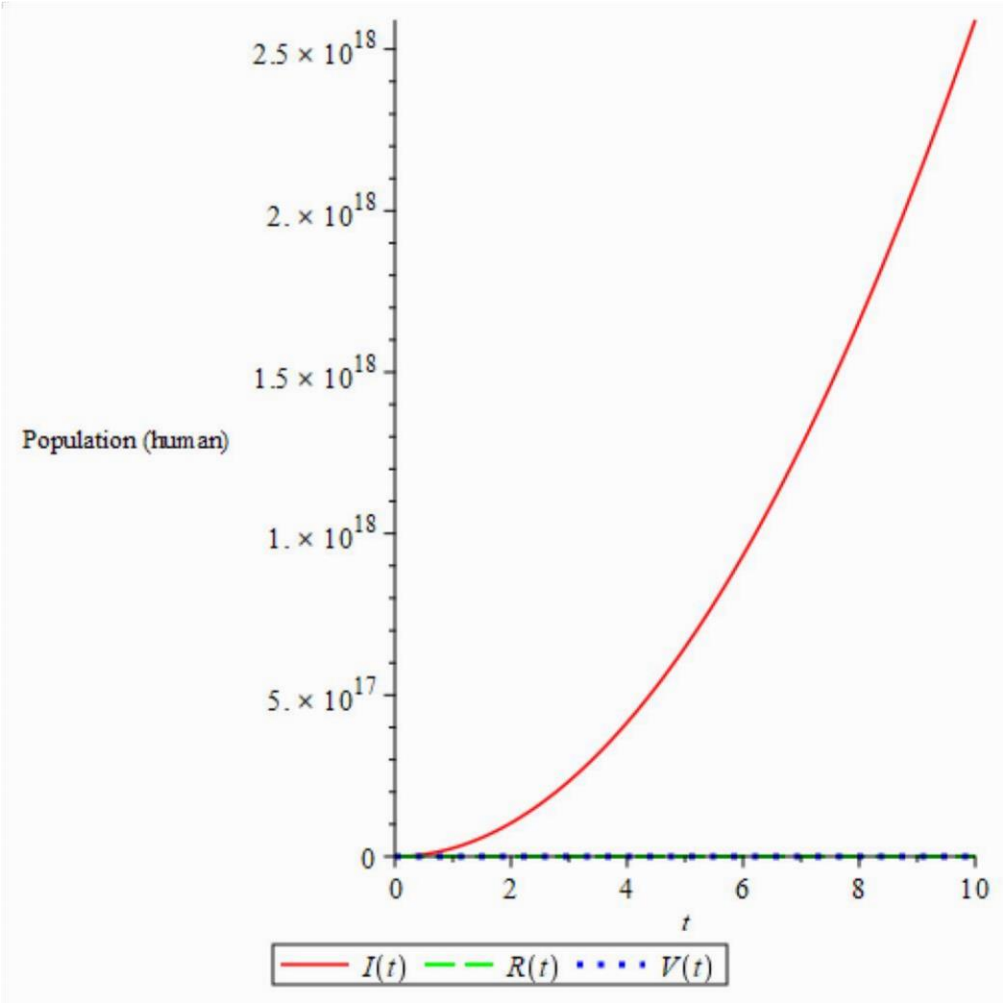

Figure 4.10: A graph showing the population of human

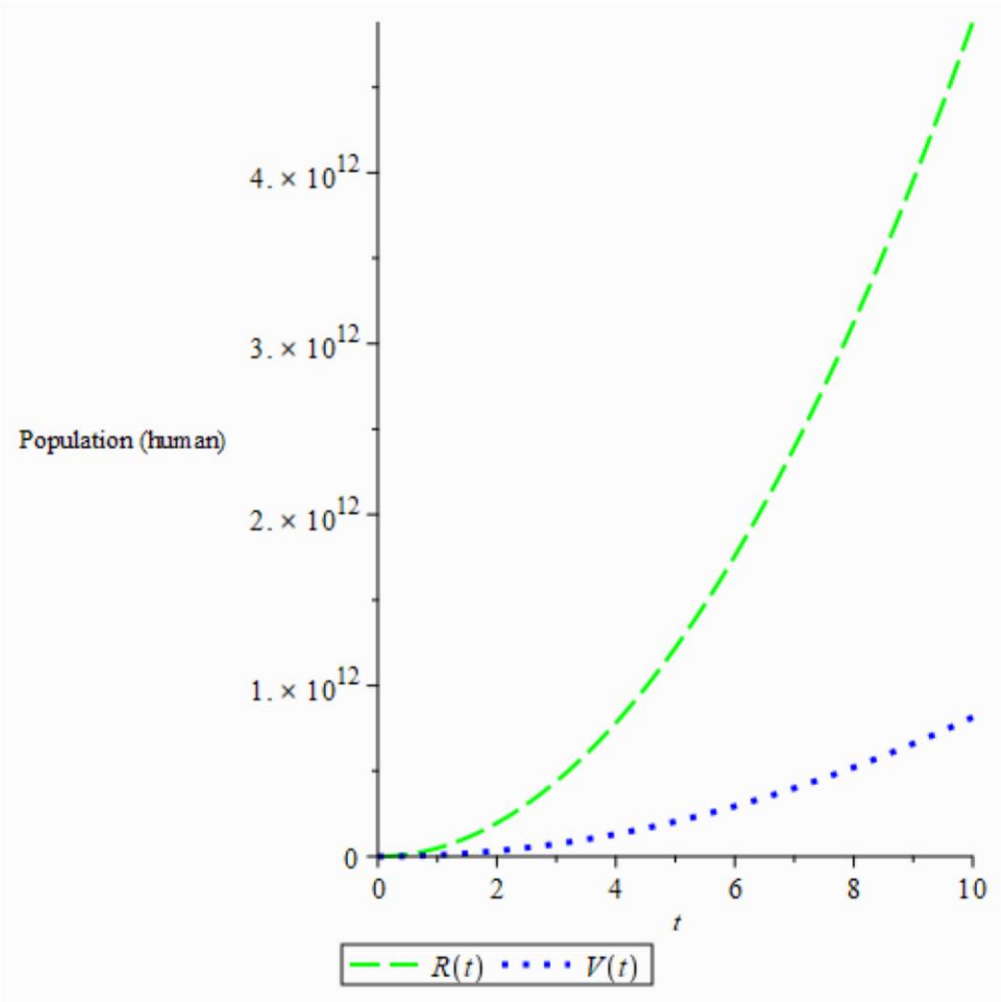

Figure 4.11: A graph showing the population of human

Figure 4.10 and Figure 4.11 show the population of Infected human, Recovered human and Vaccinated human. They are all increase with time.

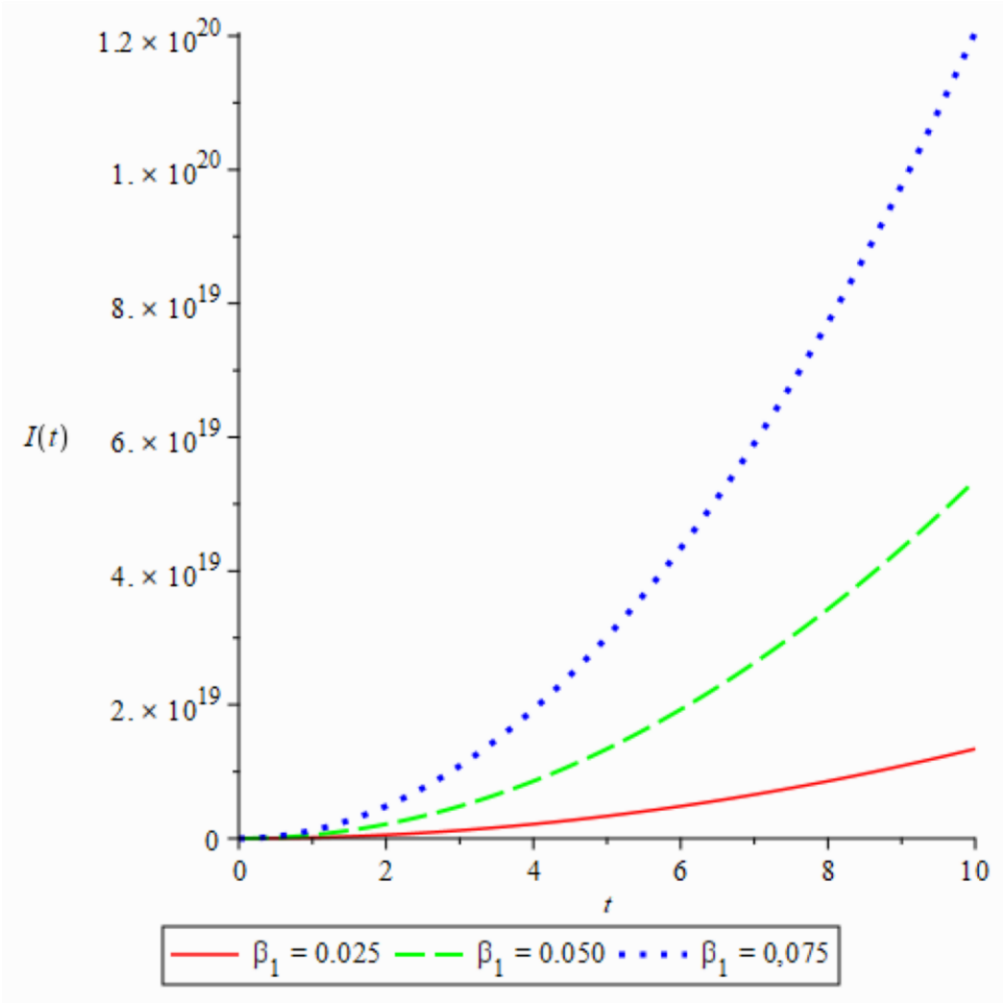

Figure 4.12: A graph showing the effects of the human to human contact rate on Infectious population

Figure 4.12 shows effects of the human to human contact rate in disease transmission. An increase in human to human interactions increase the infectious population and thereby contribute significantly to the spread of the cholera infections in the population.

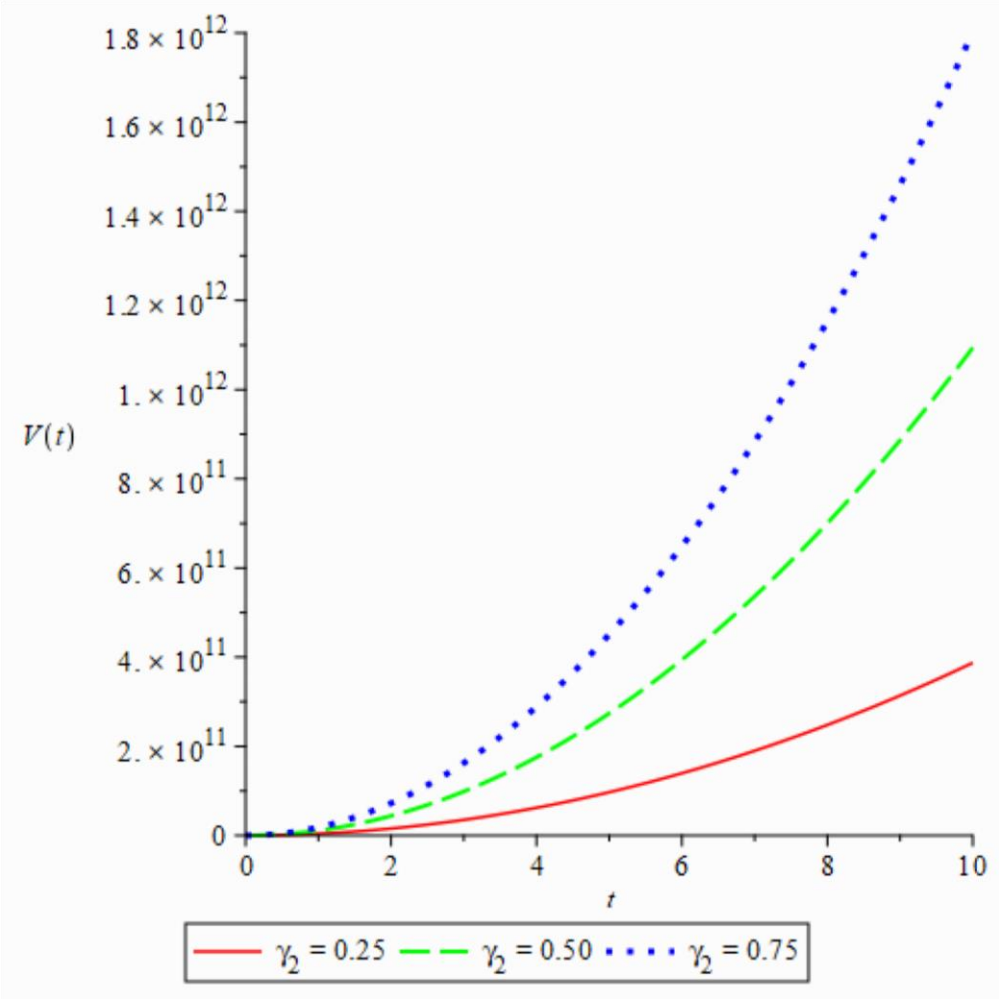

Figure 4.13: A graph showing the effects of the vaccination rate for unaware humans on vaccinated humans population

Figure 4.13 shows effects of the vaccination rate for unaware humans in disease transmission. An increase in vaccination rate for unaware humans increase the vaccinated humans population.

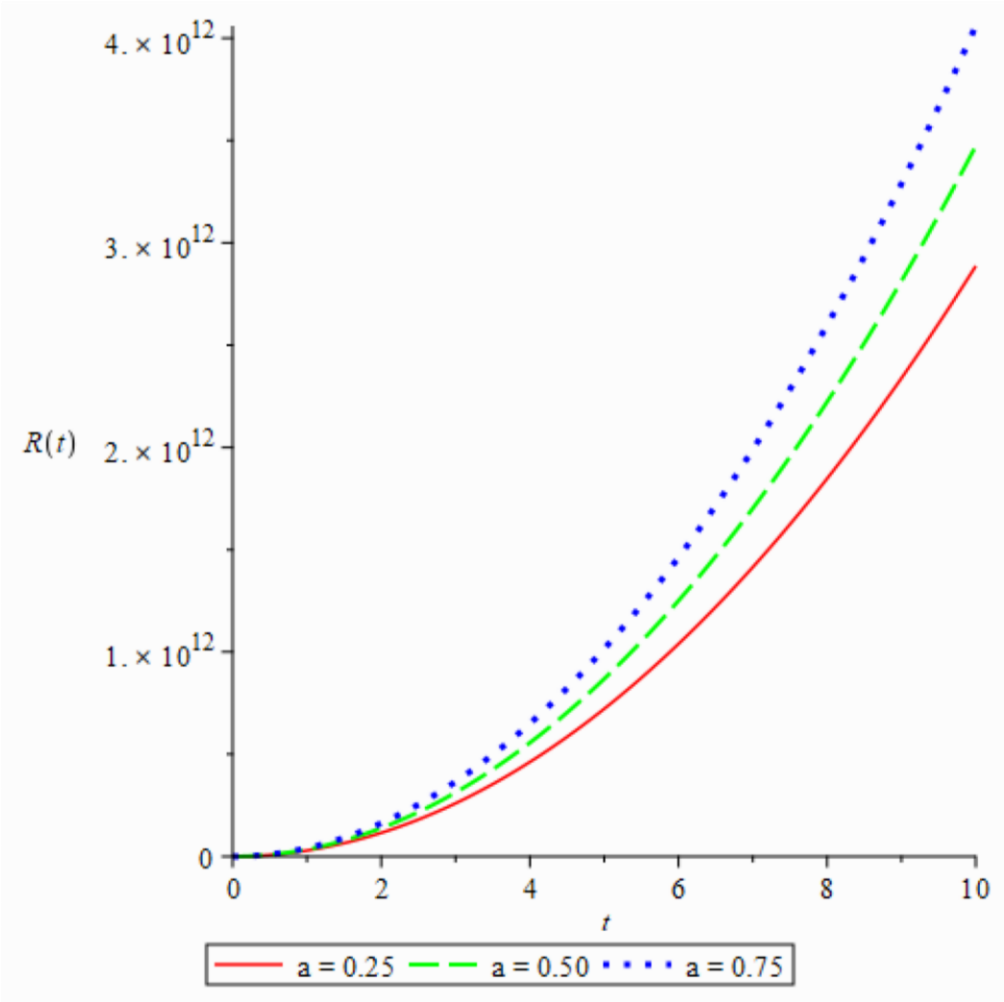

Figure 4.14: A graph showing the effects of the treatment rate on recovered humans population

Figure 4.14 shows effects of the treatment rate in disease transmission. An increase in treatment rate lead to an increase in the recovered humans population.

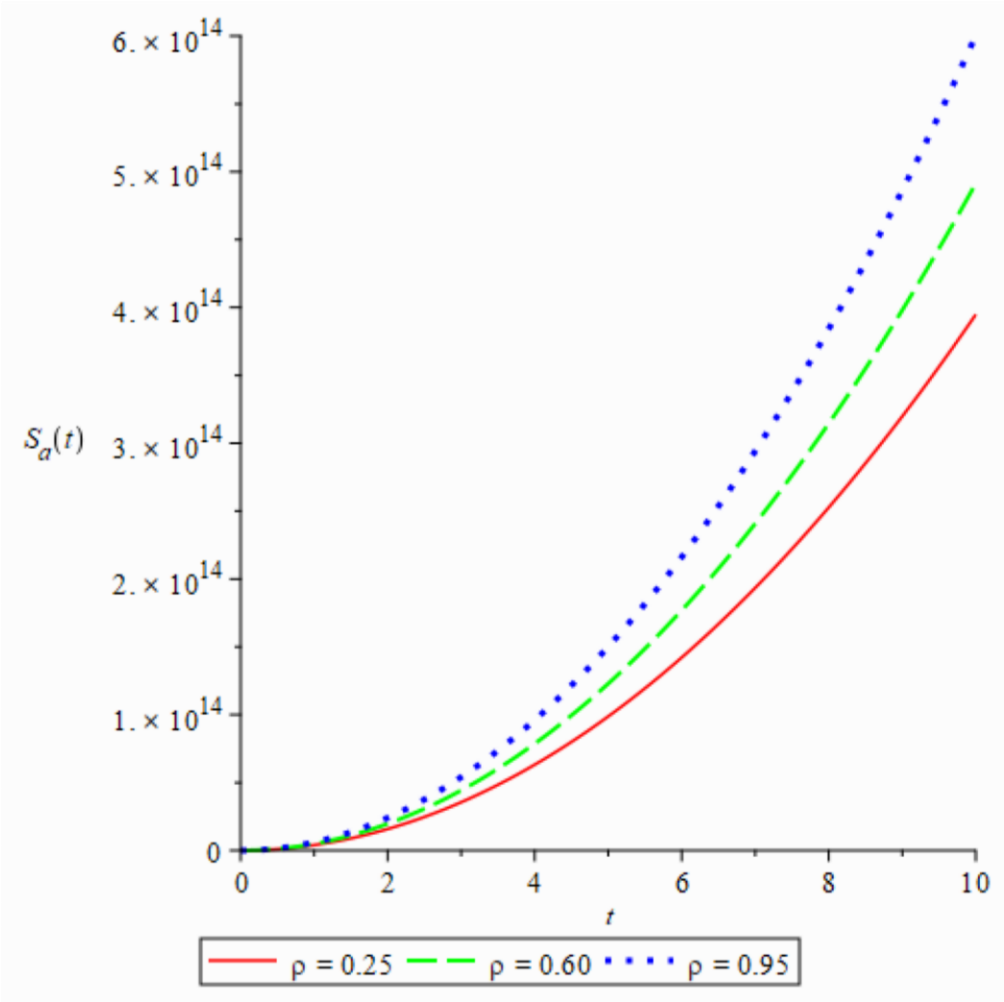

Figure 4.15: A graph showing the effects of the awareness rate on susceptible aware humans population

Figure 4.15 shows effects of the awareness rate in disease transmission. An increase in awareness rate lead to an increase in the susceptible aware humans population.

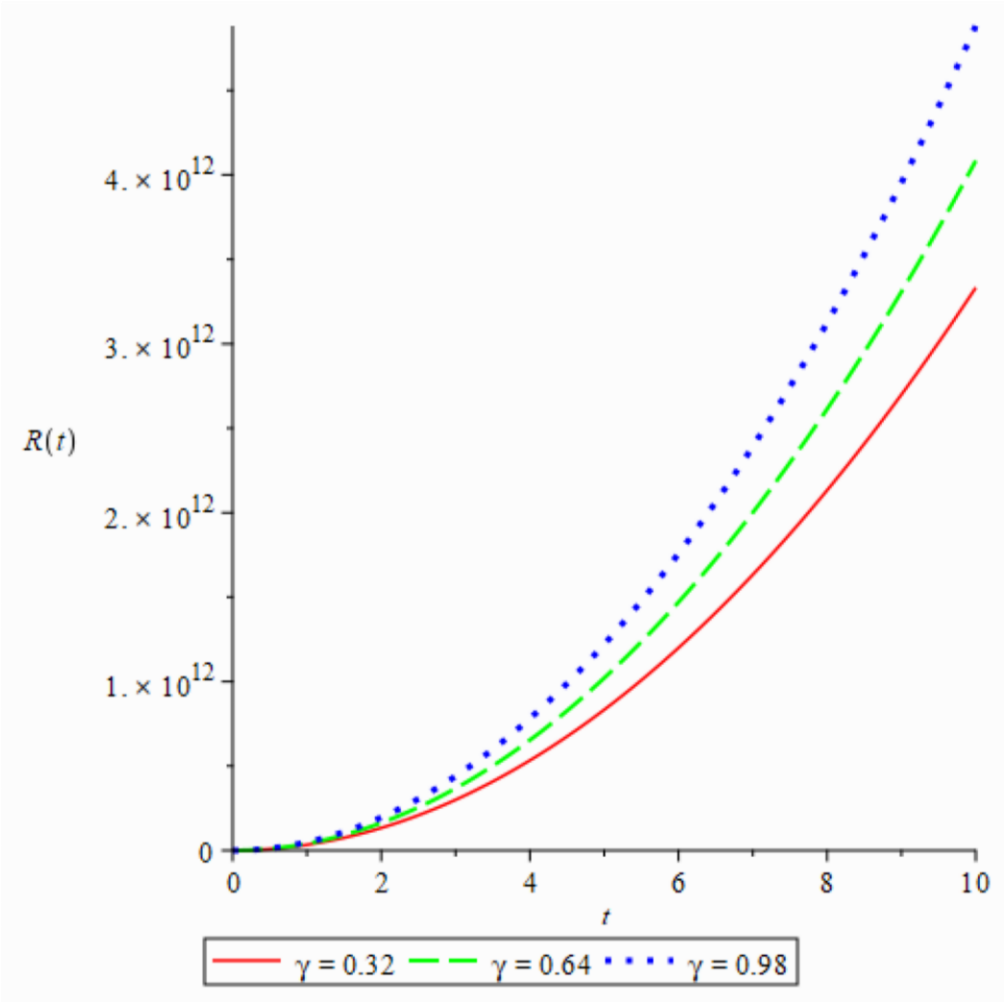

Figure 4.16: A graph showing the effects of the recovery rate on recovered humans population

Figure 4.16 shows effects of the recovery rate in disease transmission. An increase in recovery rate lead to an increase in the recovered humans population.

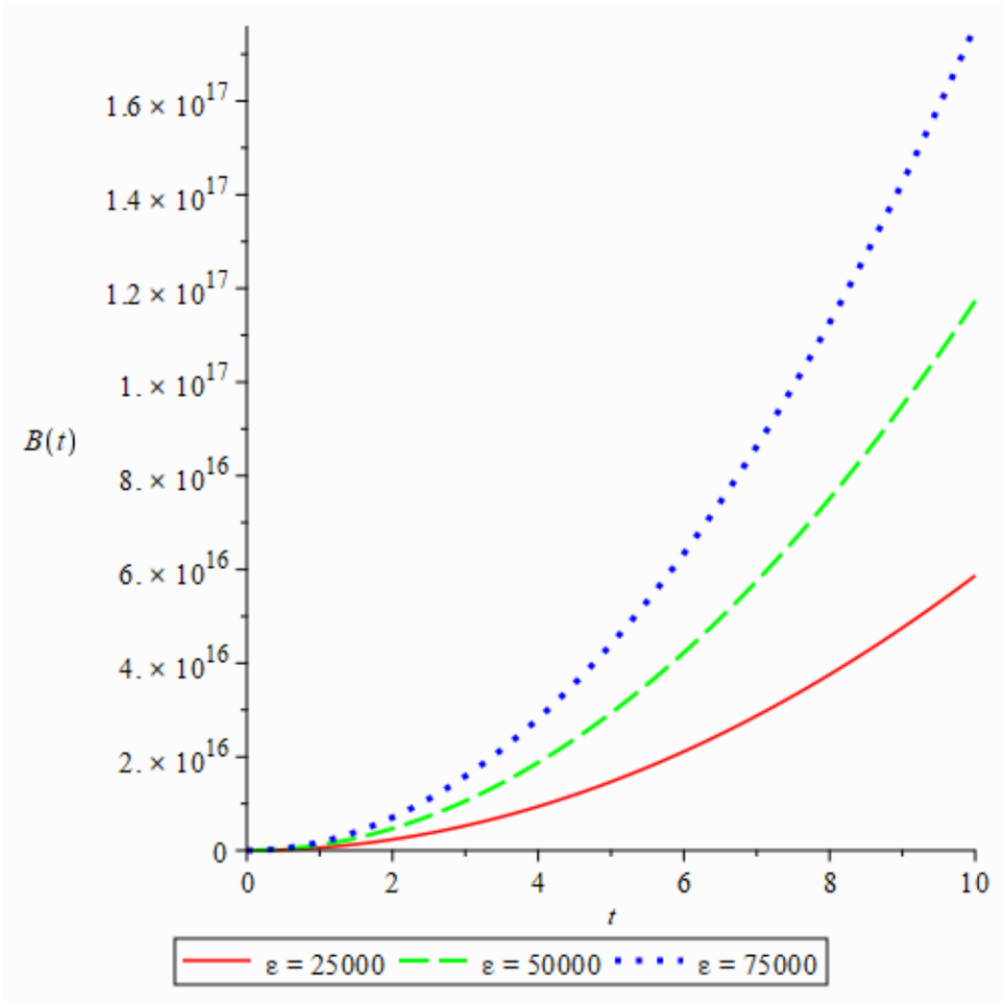

Figure 4.17: A graph showing the effects of the infectious contact with environment on Bacteria concentration

In Figure 4.17, as infectious humans interact with the environment by activities that contaminate the environment, the bacteria responsible for the cholera infections increases in the environment. This could be attributed to the infectious humans' contributions to the pollution of the environment.

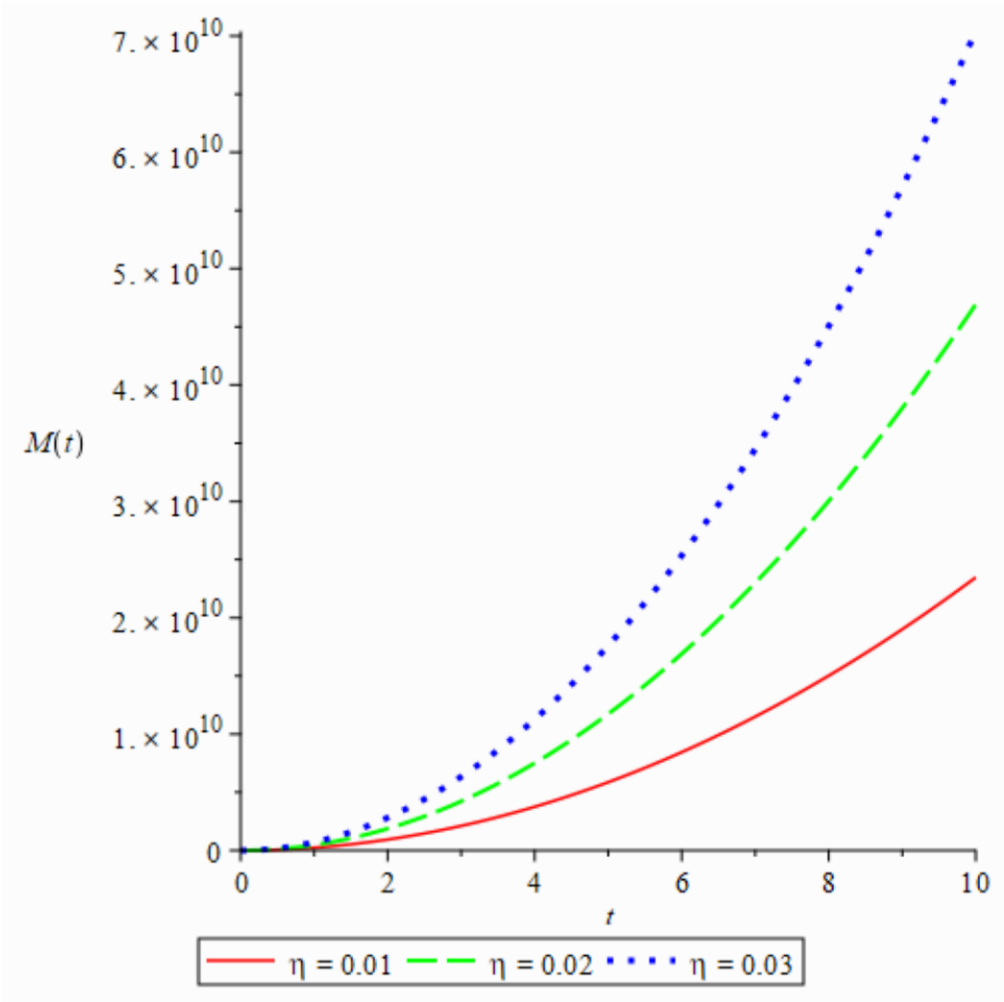

Figure 4.18: A graph showing the effects of the awareness stimulated rate on number of the awareness programmes

Figure 4.18 shows effects of the awareness stimulated rate in disease transmission. An increase in awareness stimulated rate lead to an increase in the number of the awareness programmes.

4.3 Optimal Control Simulations

Maple 17 version was used to simulate the optimal control model using the set of parameters obtained from the datasets. Some of these parameters are estimated for the sake of

illustrations. Table 3.5 represents the values of the model parameters used for the simulations.

The following initial conditions were also considered:

$S_u(0) = S_{u0} = 100423617$, $S_a(0) = S_{a0} = 43038693$,

$I(0) = I_0 = 42466$, $V(0) = V_0 = 5244305$, R

275000, $M(0) = M_0 = 100$

$H(0) = H_0 = 49596906$,

$(0) = R_0 = 41636$, $B(0) = B_0$

We described the controls using the following strategies (I, II, III, IV and V). However, Figures 4.19, 4.22 – 4.24 represent the control profiles while the rest of the plots are the graphs of infectious human population and bacteria population plotted against time. They represent the effect of the optimal controls u_1,u_2,u_3 and u_4 in reducing the number of infected individuals as well as the bacteria in the environment.

4.3.1 Strategy I: Control with awareness campaign, vaccination of susceptible individual, treatment of infected individual and treatment of water bodies

The strategy applied is to obtain the optimal control simulations that describes the effectiveness of the four control measures, that is, $u_1 = 0, u_2 = 0, u_3 = 0$ and $u_4 = 0$, when applied on the infectious class. Figure 4.19 shows the control profile when all four controls are incorporated and when there is no control.

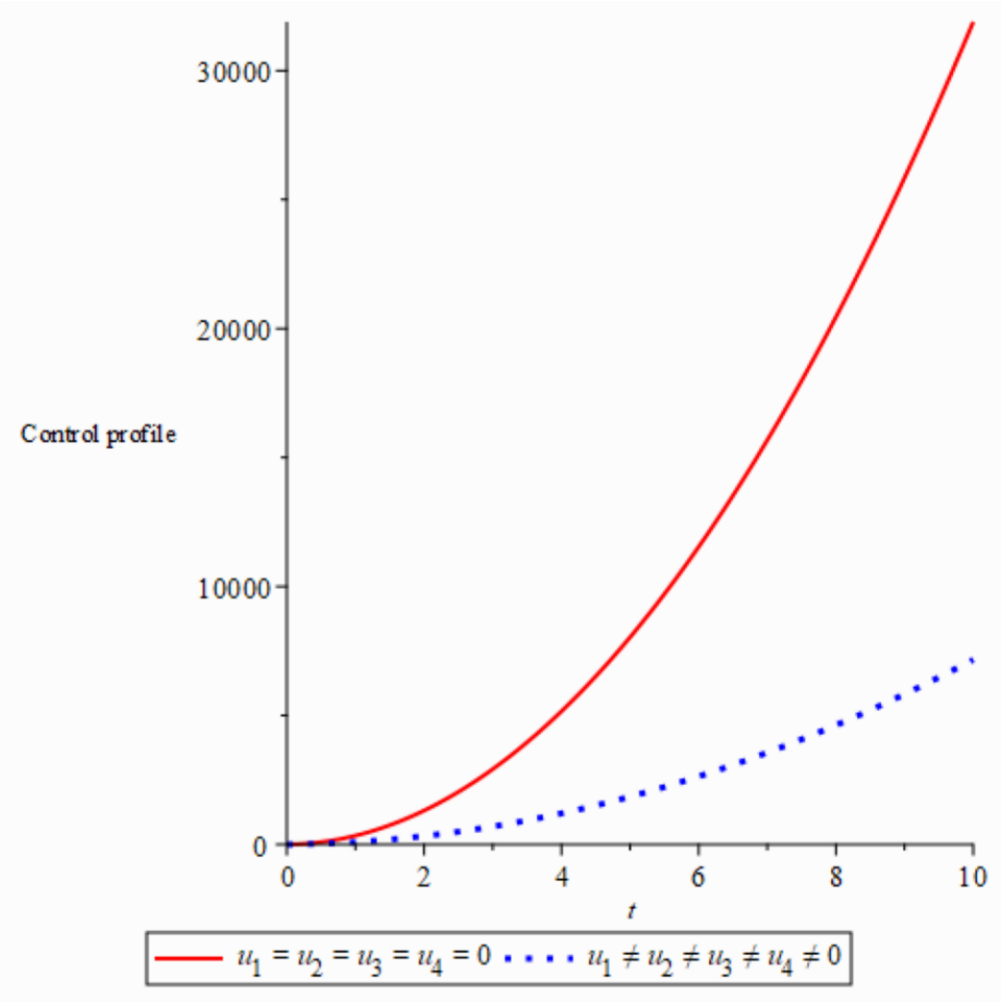

Figure 4.19: Control profile when $u_1 \neq 0$, $u_2 \neq 0$, $u_3 \neq 0$ **and** $u_4 \neq 0$

Figure 4.19 is the control profile when all four controls were considered. Clearly, we observed in Figure 4.19 that there is a drastic reduction in disease when all controls are incorporated as compare to when there is no control.

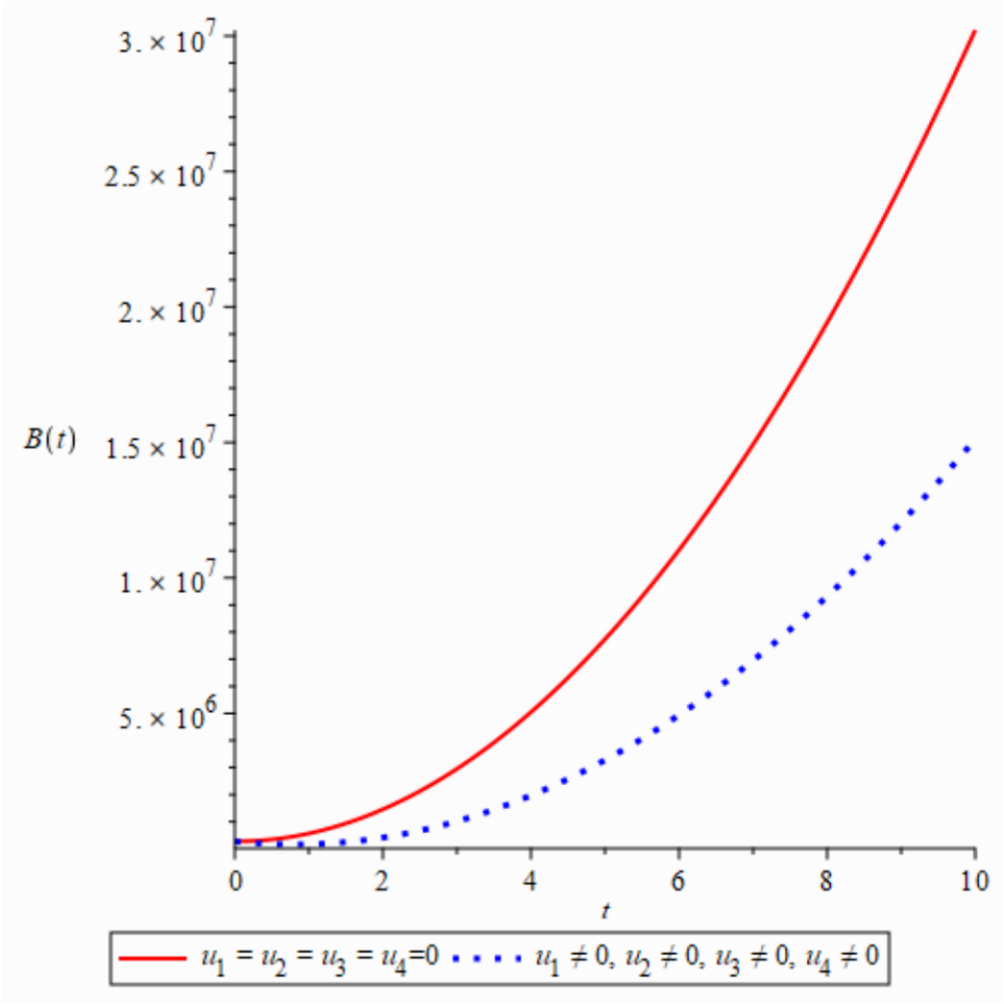

Figure 4.20: A graph showing the effectiveness of the four control measures u_1, u_2, u_3 and u_4 in the bacteria population to check its effect on the spread of cholera

(1) *Bacteria Population*. When no control measures are applied, that is, $u_1 = u_2 = u_3 = u_4 = 0$, the bacteria population is observed to increase. Access to safe drinking water and sanitation is very critical when it comes to the transmission of cholera. Therefore, when no control measure is being applied, the disease becomes persistent in the population. In the presence of control measures; that is, $u_1 \neq 0$, $u_2 \neq 0$, $u_3 \neq 0$ and $u_4 \neq 0$, the bacteria population in the environment decreases. Because treatment of water bodies minimizes the concentration of

the bacteria in the population, individuals thus get access to safe drinking water. Also awarene ss campaign creates the awareness for infected individuals more especially not to spread the disease since the purpose of awareness campaign strategy is to explore the awareness of the disease, mode of transmission, and prevention. This therefore reduces the bacteria population in the environment as shown in Figure 4.20.

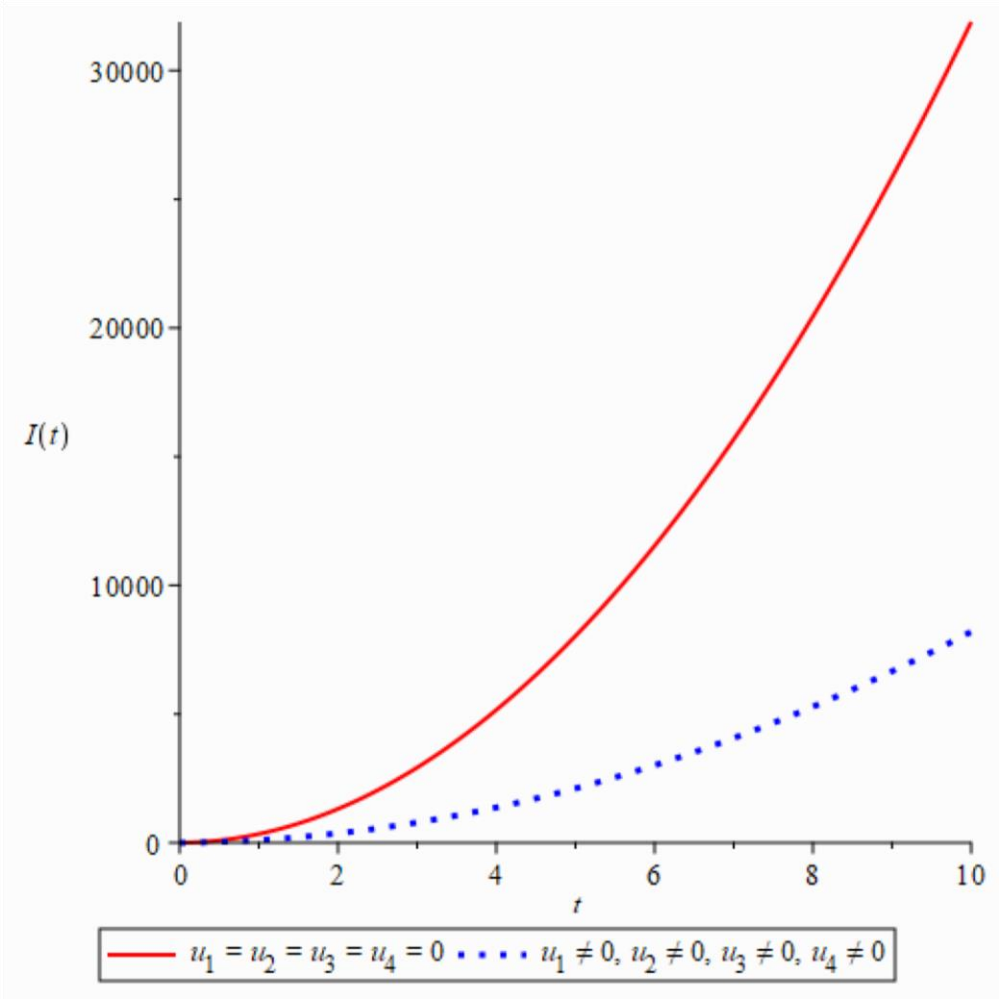

Figure 4.21: A graph showing the effectiveness of the four control measures u_1, u_2, u_3 and u_4 in the infected population to check its effect on the spread of cholera

(2) *Infected Individuals*. When no control measure is applied, the number of infected individuals will increase in the population. On the other hand, when control measures are applied such as awareness campaign, vaccination of susceptible individuals and treatment of infected individuals, infected individuals would know the causes, transmission, and effects of cholera. This would reduce their actions which target environmental conditions that spread cholera. These activities may include disruption of water bodies by defecating near water banks, individuals littering around causing bad sanitation systems, individuals crowding up in camps.

Hence, the number of infected individuals in the population would decrease as shown in Figure 4.21. On the other hand, when water bodies are treated, the rate of being reinfected decreases.

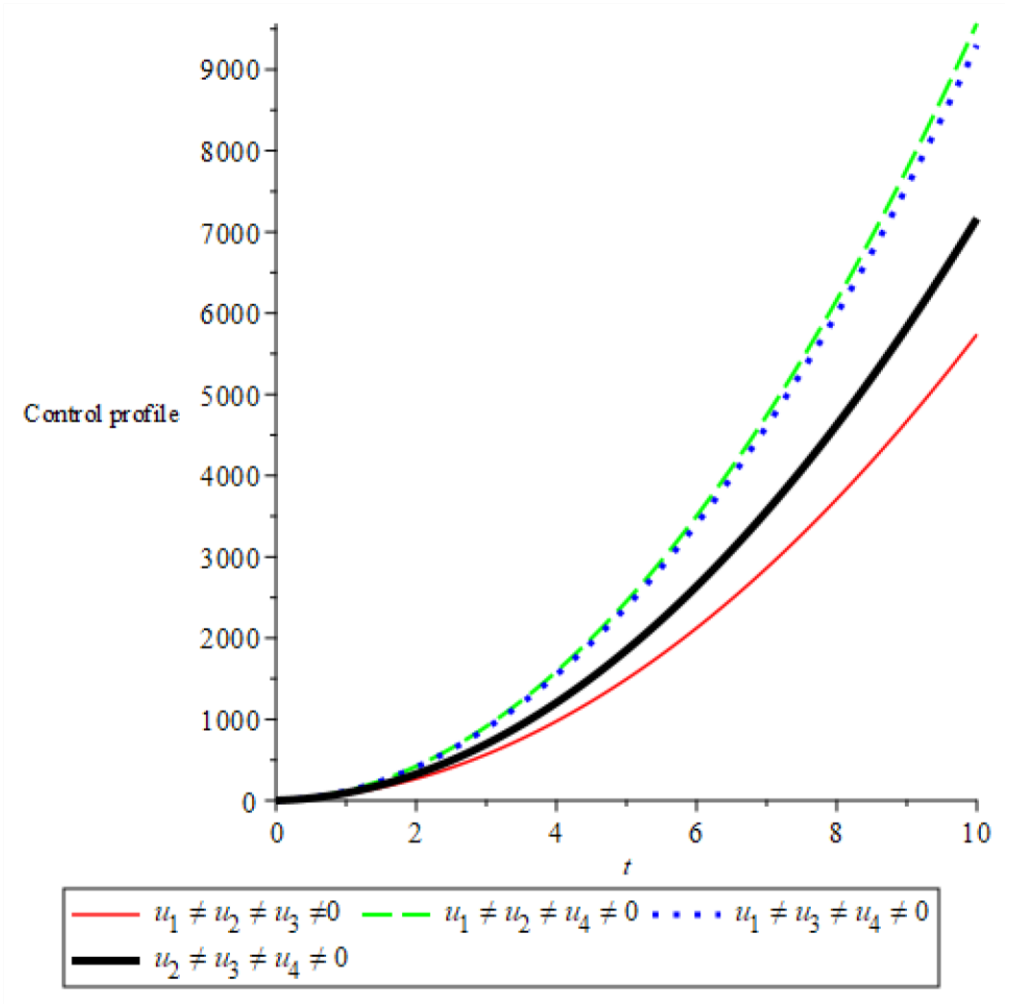

Figure 4.22: Control profile when $u_1 \neq u_2 \neq u_3 \neq 0$, $u_1 \neq u_2 \neq u_4 \neq 0$, $u_1 \neq u_3 \neq u_4 \neq 0$ **and** $u_2 \neq u_3 \neq u_4 \neq 0$

Figure 4.22 is a control profile when only three different controls are being applied. This shows that a combination of awareness campaign with vaccination of susceptible individuals and treatment of infected individuals gives a better result in reducing the number of infected individuals in the population and this is follow by a combination of water treatment with vaccination of susceptible individuals and treatment of infected individuals.

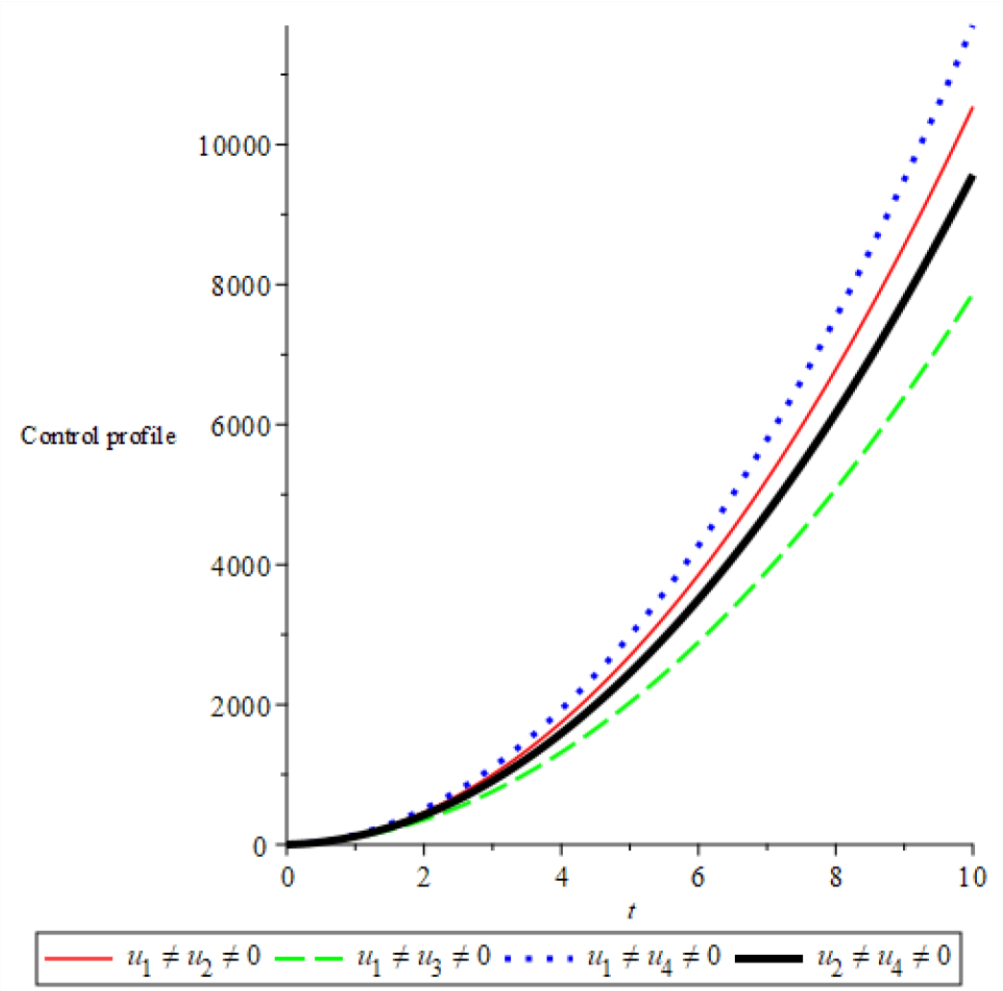

Figure 4.23: Control profile when $u_1 \neq u_2 \neq 0$, $u_1 \neq u_3 \neq 0$, $u_1 \neq u_3 \neq 0$ **and** $u_1 \neq u_4 \neq 0$

Figure 4.23 is a control profile when only two different controls are being applied. This shows that a combination of awareness campaign with treatment of infected individuals gives a better result in reducing the number of infected individuals in the population and this is follow by a combination of water treatment with vaccination of susceptible individuals and then by a combination of awareness campaign with vaccination of susceptible individuals.

A combination of awareness campaign with water treatment is worst case scenario.

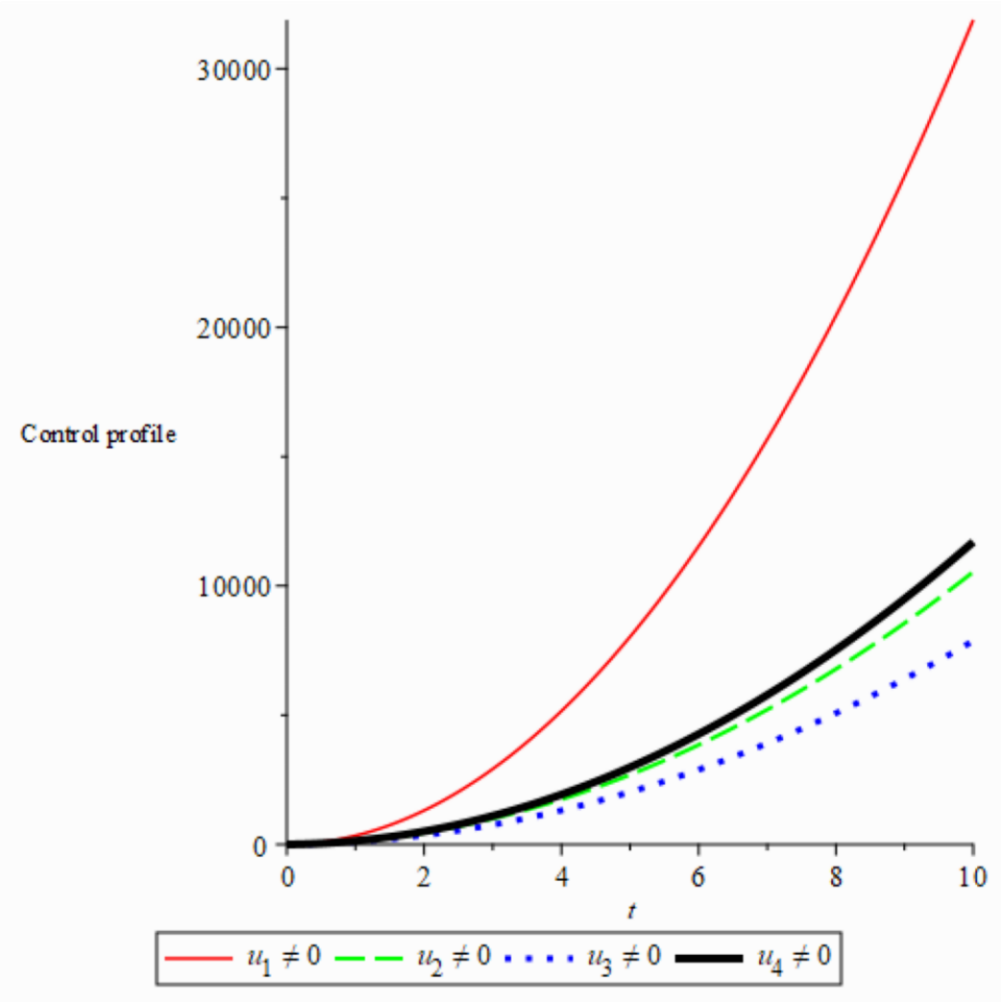

Figure 4.24: Control profile when $u_1 \neq 0$, $u_2 \neq 0$, $u_3 \neq 0$ and $u_4 \neq 0$

Figure 4.24 is a control profile when only one control is being applied. This shows that treatment of infected individuals gives a better result in reducing the number of infected individuals in the population and this is follow by vaccination of susceptible individuals, then by water treatment and lastly by awareness campaign.

4.3.2 Strategy II: Control with awareness campaign only

The strategy applied is to obtain the optimal control simulations that describes the effectiveness of one control measure (that is, awareness campaign) when applied. This strategy is show in shown in Figure 4.25.

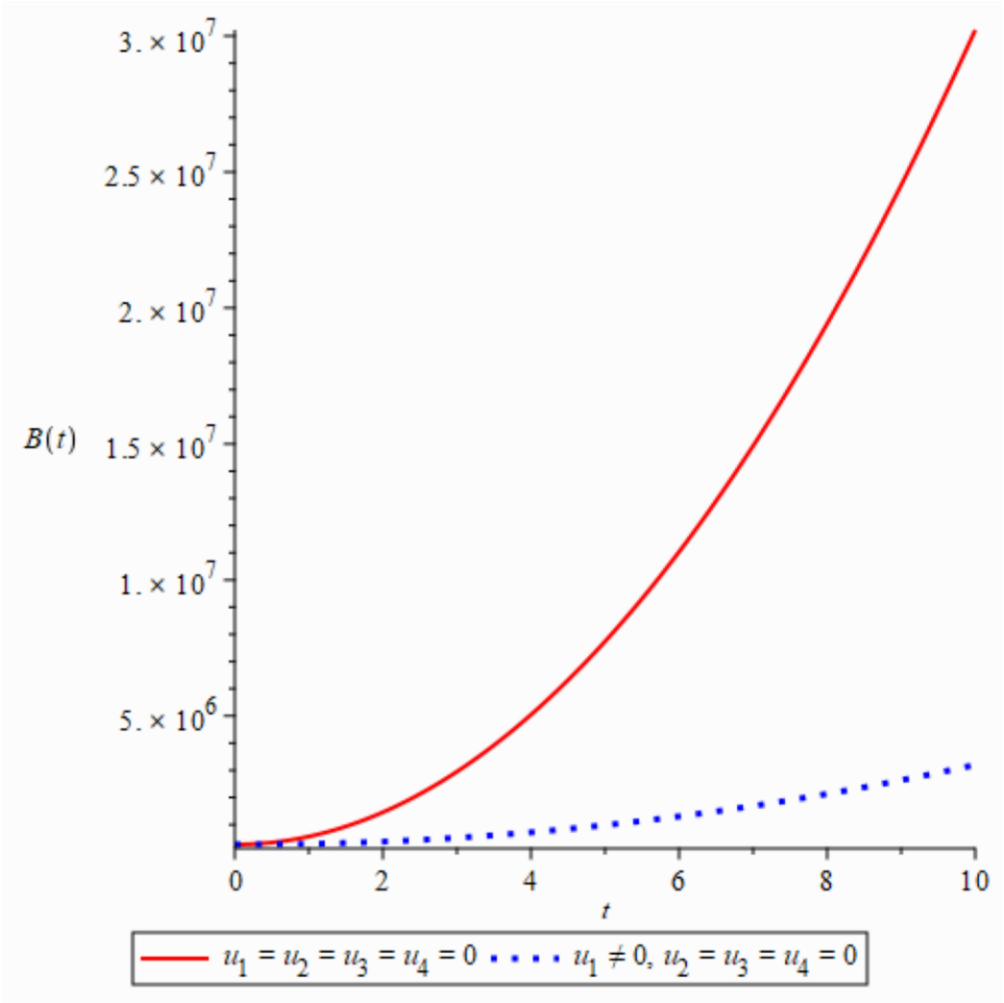

Figure 4.25: A graph showing the effectiveness of awareness campaign as the only control measure to check its effect on the spread of cholera

Bacteria Population. When there are no control measures being applied, the bacteria population increases in the environment. Community engagement helps in the control of cholera. When individuals are educated by practically showing individuals how and the need to wash one's hands with soap and running water after visiting the toilet, it will reduce the

bacteria they pick up from such areas. This helps reduce the bacteria population in the environment as shown in Figure 4.25.

Thus, applying just awareness campaign as a control measure is effective in controlling the spread of cholera.

4.3.3 Strategy III: Control with vaccination of susceptible individual only

The strategy applied is to obtain the optimal control simulations that describes the effectiveness of one control measure (that is, vaccination of susceptible individuals) when applied. This strategy is show in shown in Figure 4.26.

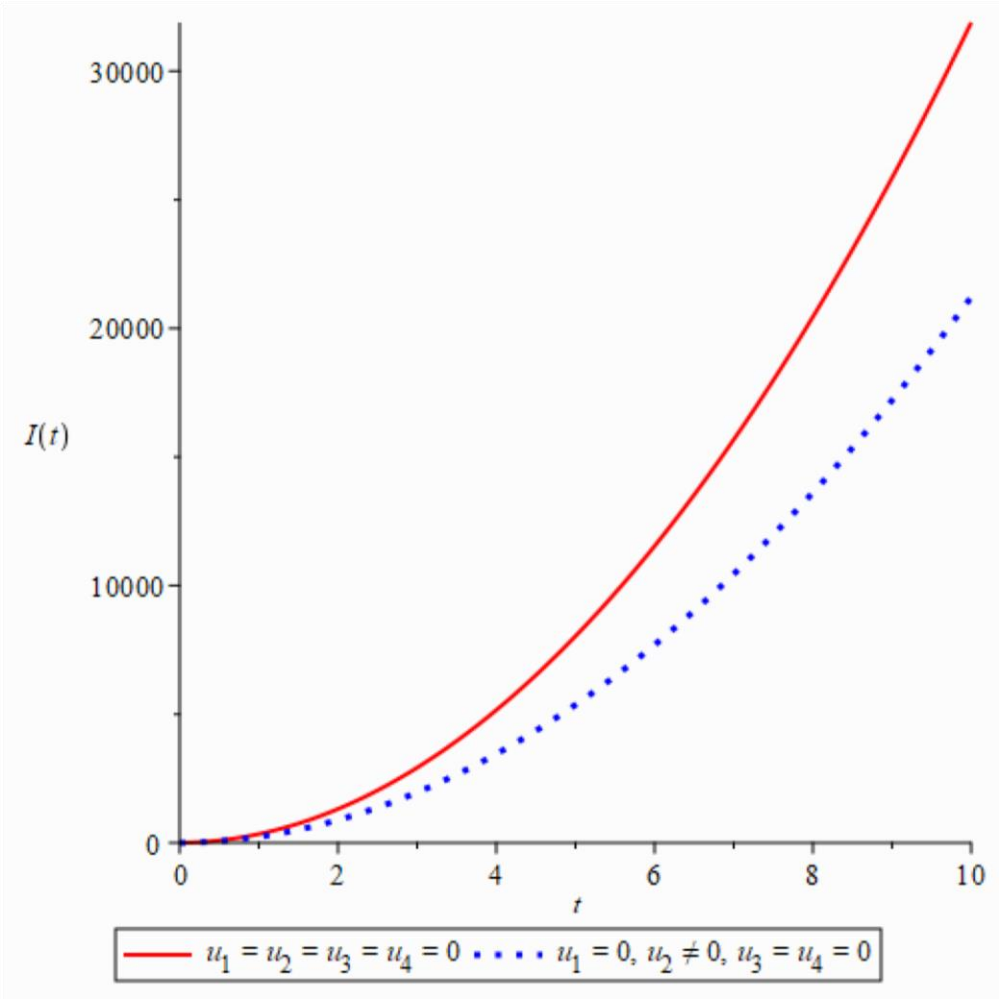

Figure 4.26: A graph showing the effectiveness of vaccination as the only control measure in the infected population

Infected Individuals. Infected individuals increase when there are no control measures being applied. Meanwhile, when susceptible individuals are vaccinated, their immunity will be boosted against cholera. This help reduces the number of infected individuals in the population as shown in Figure 4.26.

4.3.4 Strategy IV: Control with treatment of infected individual only

The strategy applied is to obtain the optimal control simulations that describes the effectiveness of one control measure (that is, treatment of infected individual) when applied.

This strategy is show in shown in Figure 4.27 and Figure 4.28.

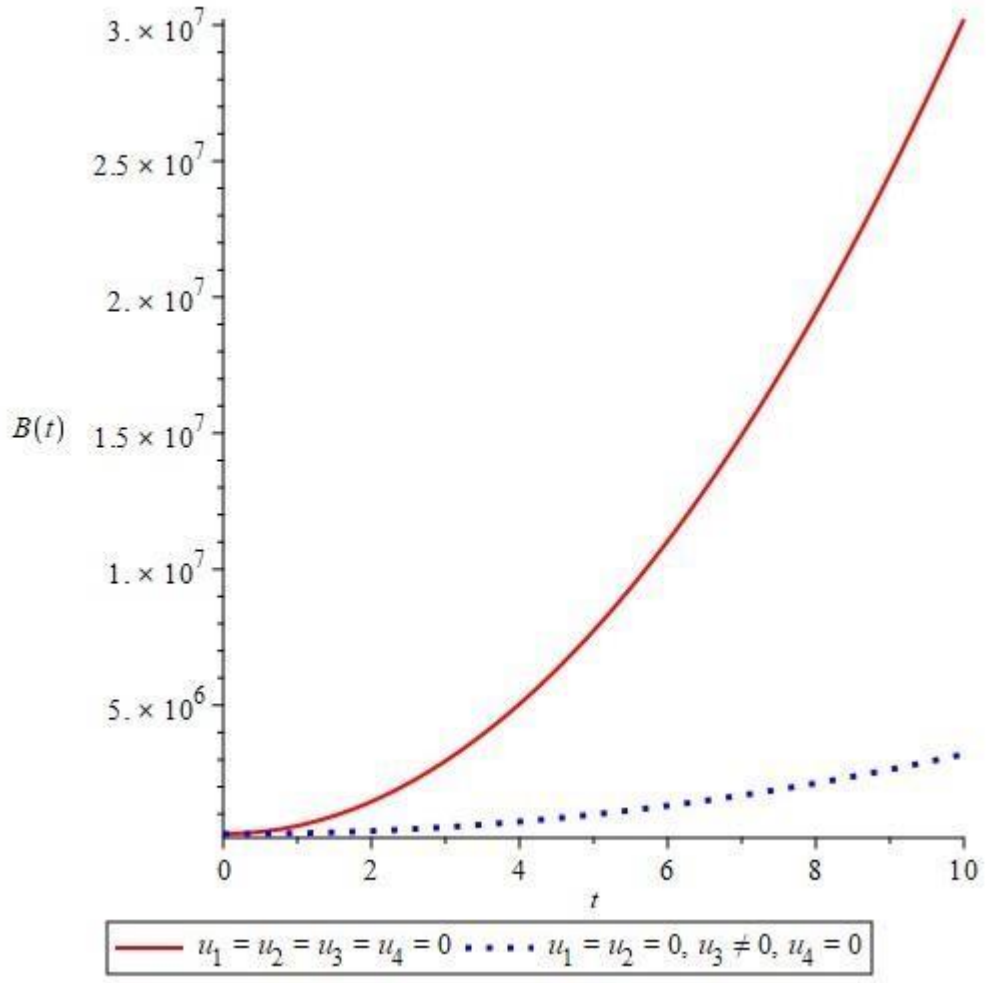

Figure 4.27: A graph showing the effectiveness of treatment as the only control measure in the bacteria population

(1) *Bacteria Population*. The bacteria population in the environment increases when there are no control measures applied. However, when number of infected individuals as a major contributors of the bacteria in the environment are reduced through therapeutic treatment, the spread of the bacteria in the environment would reduce as shown in Figure 4.27.

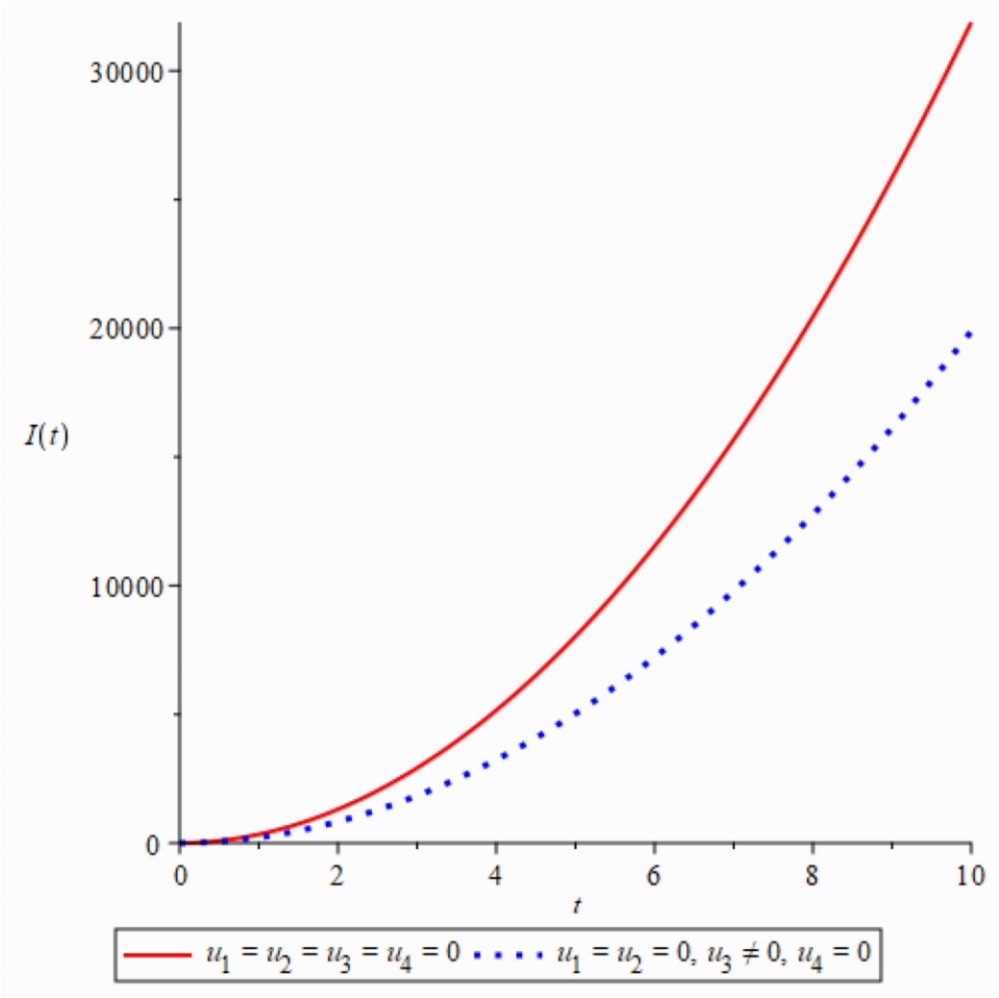

Figure 4.28: A graph showing the effectiveness of treatment as the only control measure in the infected population

(2) *Infected Individuals*. Infected individuals increase when there are no control measures being applied. Meanwhile, when infected individuals are treated and recovered, the number of infected individuals in the population will be reduced as shown in Figure 4.28.

4.3.5 Strategy V: Control with treatment of water bodies only

Here, the strategy applied is to obtain the optimal control simulations that describe the effectiveness of treatment of water bodies as the only control measure being applied. This strategy is show in shown in Figure 4.29 and Figure 4.30.

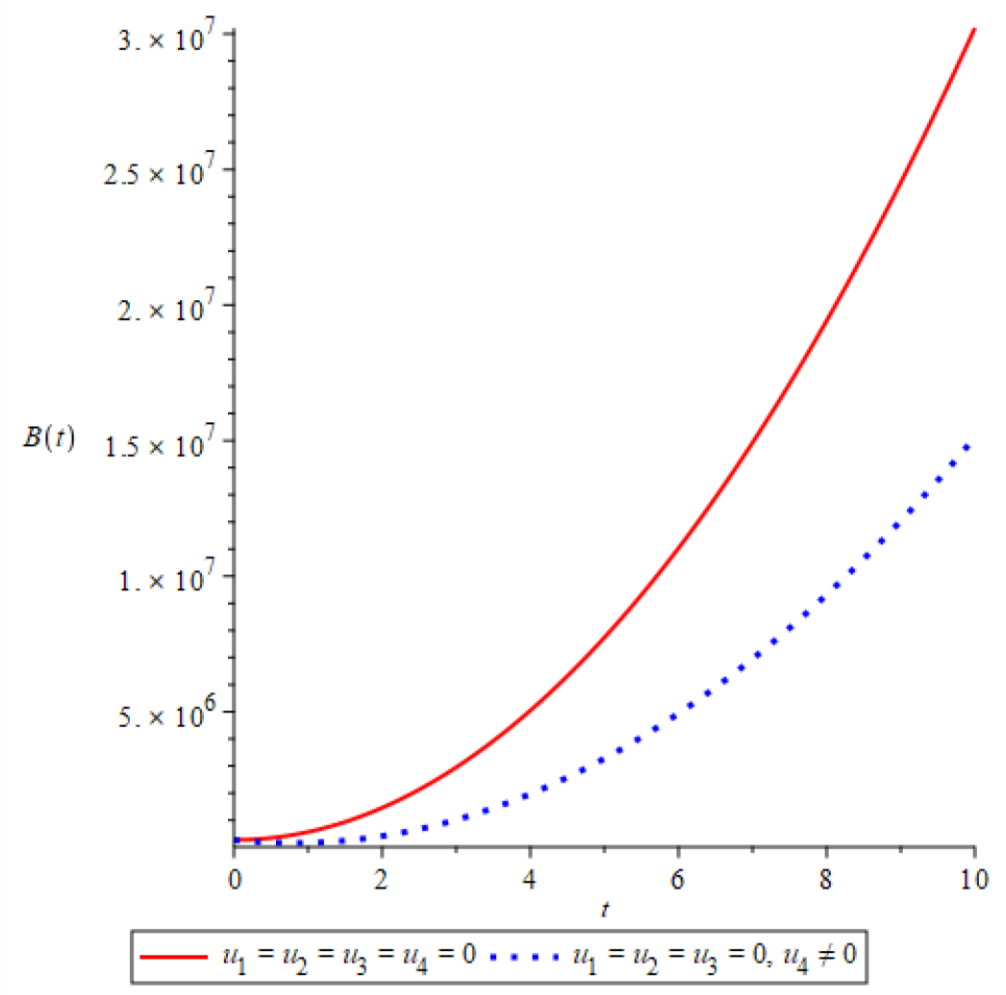

Figure 4.29: A graph showing the effectiveness of treatment of water bodies as the only control measure in the bacteria population

(1) *Bacteria Population*. The bacteria population in the environment increases when there are no control measures applied. However, when infected water bodies are treated, the

bacteria population in the water body decreases due to the reduction in the concentration of the growth of the bacteria as shown in Figure 4.29.

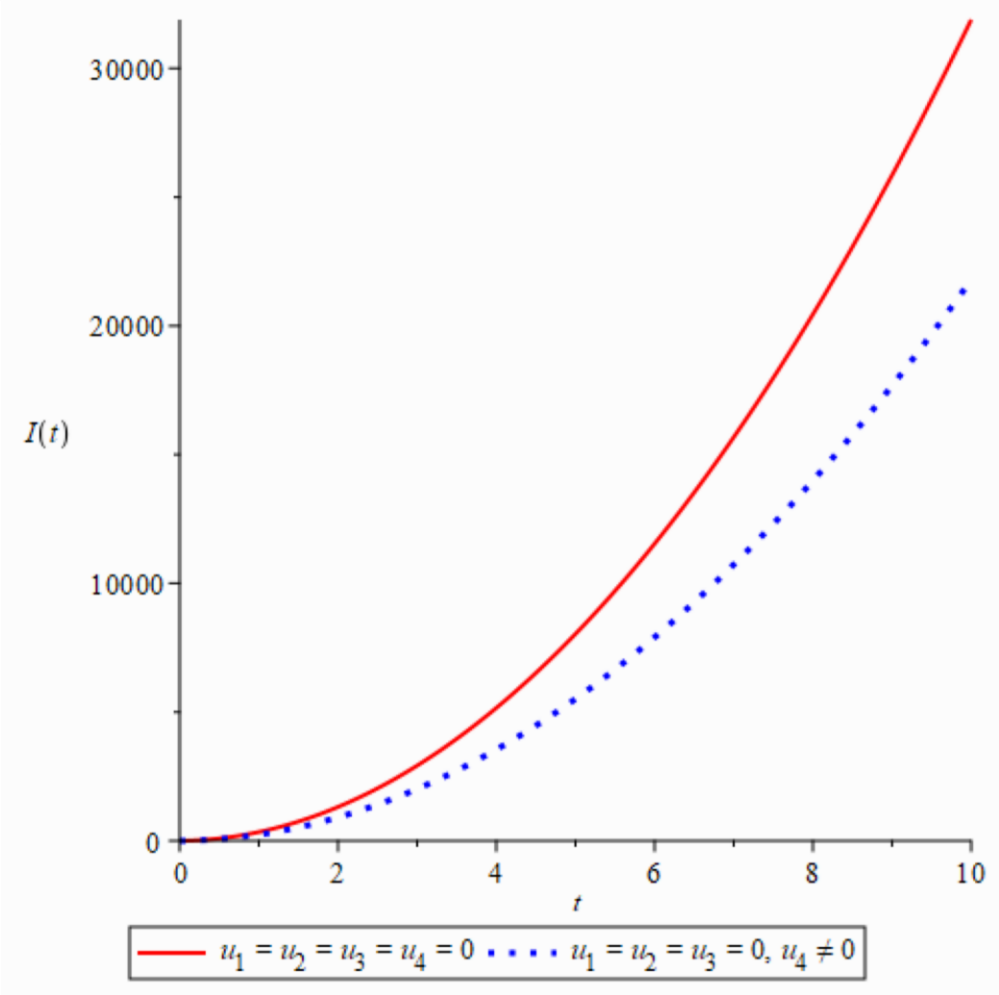

Figure 4.30: A graph showing the effectiveness of treatment of water bodies as the only control measure in the infected population

(2) *Infected Individuals*. Infected individuals increase when there are no control measures being applied. Meanwhile, when infected water bodies are treated, individuals will have access to good drinking water and this will help in reducing the number of infected population.

CHAPTER FIVE

5.0 CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

In this thesis, we have formulated a deterministic mathematical model for transmission dynamics of cholera that incorporates four control strategies namely awareness campaign, vaccination, treatment, and sanitation. From the model we have derived the effective reproduction number from which we have deduced the basic reproduction number, and the reproduction numbers with combination of two, and three control strategies. The effective reproduction number computed has been used to measure the relative impact for individual or combined intervention for effective disease control. We have derived both the Disease Free Equilibrium (DFE) and the Endemic Equilibrium points (EE) and proved that both the DFE and EE are locally and globally asymptotically stable when R_0^c 1.

Moreover, we have performed sensitivity analysis on the basic reproduction with all control strategies, from which we have noted that the most sensitive parameters are the vaccination loss rate, vaccination rate for aware humans, awareness loss rate, effective contact between the susceptible and infected individuals, effective contact between the susceptible and environment, bacteria shed rate into the water supply by infected human, and recruitment rate. These strategies need high attention when at all we need to control cholera outbreak wherever it occurs.

Numerical simulations of the model have shown that, whenever the control strategies are carried out solely then treatment is best alternative to cholera, but when there are two combinations strategies then the best combination is treatment and awareness.

On the other hand, when a combination of three control strategies is implemented then the best combination is the one with vaccination, treatment and awareness. It has been noted that the best combination is the one that incorporated all four control strategies. From this study we conclude that the more one increases combination of control strategies then cholera can be eradicated from the community.

5.2 Recommendations

Based on the findings from this research, we recommend as follows:

(i) Proper awareness campaign and sensitization be given to the public by relevant authorities and NGO's of the dangers of open defecation and urinating in source of drinking water. This will reduce the contribution of each infected person to the aquatic reservoir (parameter).

(ii) Government should provide portable water to the populace in order to discourage drinking of untreated water. This will reduce the rate of exposure to contaminated water (parameter $_2$).The same was recommended by Ochoche (2013).

(iii) People suffering from cholera should be immediately quarantined so as to reduce the contact rate between the infected and the susceptible humans (parameter $_2$).

Early treatment of all cholera patients is highly recommended to save the life of the sick humans since cholera kill in very short time (parameter).

(iv) People should be restricted to enter in places where cholera outbreak occurs, this will help to limit the spread of the disease (parameter). In case the economy allows, vaccination strategy should be established to areas where cholera is chronic.

5.3 Contributions to Knowledge

1. Our study provides a modeling framework to investigate more difficult situation on cholera dynamics under the impact of awareness programmes and humans' hygiene consciousness, and the findings from the model confirm the positive effect of awareness programmes and humans' hygiene consciousness in lowering the infection risk and reducing the disease prevalence.

2. The thesis established that the best strategy for the controlling of cholera is the application of all the control measures, that is, applying both awareness campaigns, vaccination of susceptible individual, treatment of infected individual and treatment of water bodies with

c_0 0.07471714947 1 attained.

3. The thesis established that the best alternative strategy

(i) when three control measures are to be applied are the combination of awareness campaigns, vaccination of susceptible individual and treatment of infected individuals with $\mathcal{R}_{c_0} = 0.213353986 < 1$ attained.

(ii) when two control measures are to be applied are the combination of awareness campaigns and treatment of infected individuals with $\mathcal{R}_{c_0} = 0.539801403 < 1$ attained.

(iii) when one control measures is to be applied is the treatment of infected individuals with $\mathcal{R}_{c_0} = 0.250439559 < 1$ attained.

4. The thesis also affirmed that the worst case scenario occurs when there is no control strategy for the epidemic.

REFERENCES

Adewale, S. O., Adeniran, G. A., Olopade, I. A. & Mohammed, I. T. (2015). Mathematical analysis of the effect of growth rate of vibrio-cholera in the dynamical spread of cholera. *Research Journal of Mathematics*, 2(3), 1-18.

Andrews, J. R. & Basu, S. (2011). Transmission dynamics and control of cholera in Haiti: An epidemic model. *Lancet*, 377, 1248–55.

Ayoade, A. A., Ibrahim, M. O., Peter, O. J. & Oguntolu, F. A. (2018). On the global stability of cholera model with prevention and control. *Malaysian Journal of Computing*, 3(1), 28–36.

Bertuzzo, E. Casagrandi, R. Gatto, M. Rodriguez-Iturbe, I. & Rinaldo, A. (2010). On spatially explicit models of cholera epidemics. *Journal of the Royal Society Interface*, 7(43), 321–333.

Bertuzzo, E., Azaele, S., Maritan, A., Gatto, M., Rodriguez-Iturbe, I. & Rinaldo, A. (2008). On the space-time evolution of a cholera epidemic. *Water Resources Research*, 44(1), 123 - 132.

Bolarin G. & Bamigbola O. M. (2014): Pulse Vaccination Strategy in a SVEIRS epidemic Model with Two-Time Delay and Saturated Incidence. *Universal Journal of Applied Mathematics*, 2(5), 222 – 232.

Bolarin G. & Abdullahi U. A. (2016): A Model of Measles Dynamics in the Presence of Weak and Strong Vitamine A. *Journal of the Nigeria Association of Mathematical Physics (JNAMP)*, 38, 219 – 230.

Brachman P. S. & Abrutyn E. (2009). *Bacterial infections of humans: epidemiology and control*. New York: Springer.

CDC (2004). *Cholera–vibrio cholerae infection*. Antlanta: Centers for Disease Control and prevention.

CDC&P (2014). *Cholera: General information*. Available at www.cdc.gov/cholera/general / accessed on 30/09/2061.

Centers for Disease Control & Prevention (CDC&P) (2013). *Cholera Vibrio Cholera Infection*. July 30, 2013. http://www.cdc.gov/cholera/general/index.html Accessed December 5. 2014

Chaignat C. L. (2014). *10 facts on cholera*. Geneva: World Health Organization.

CNN Library (2014). *Cholera Fast Facts*. http://www.cnn.com/2013/10/16/health/cholera - fast - facts/ Accessed December 8, 2014.

Codeco, C. T. (2001). Endemic and epidemic dynamics of cholera: the role of the aquatic reservoir. *BMC Infectious Diseases*, 1(1), 1 - 20.

Davis, C. P. (2014). *Cholera: what is the history of cholera*. http://www.medicinenet.eom/cholera/ Accessed December 8, 2014.

Diekmann, O., Heesterbeek, J. A. P. & Metz, J. A. J. (1990). On the Definition and the Computation of the Basic Reproduction Ratio R_0 in Models for Infectious Diseases in Heterogeneous Populations. .*Journal of Mathematical Biology*, 28(4), 365–382.

Edward, S. & Nyerere, N. (2015). A Mathematical Model for the Dynamics of Cholera with Control Measures, *Applied and Computational Mathematics*, 4(2), 53 – 63.

Eustace, K. A., Osman, S. & Wainaina, M. (2018). Mathematical modelling and analysis of the dynamics of cholera. *Global Journal of Pure and Applied Mathematics*, 14(9), 1259–1275.

Finkelstein, R. A. (2014). *Cholera, Vibrio Cholera Oland 0139, and Other Pathogenic*

Vibrios. Medical Microbiology 4th Edition. 1996. http://www.ncbi.nlm.nih.gov/books/NBK8407/ Accessed December 5, 2014

Hartley, D. M., Morris, J. G. & Smith. D. L (2006). Hyperinfectivity: A critical element in the ability of V. cholerae to cause epidemics. *PLoS Medicine,* 3, 0063–0069.

He, J. H. (2006). Some asymptotic methods for strongly non-linear equations. *International Journal of Modern Physics B,* **20**(10), 1141-1199.

Hethcote, H. W. (2000). The mathematics of infectious diseases. *International Journal of Science and Technology,* 2 (11), SIAM Review 42.

Jensen M. A., Faruque S. M., Mekalanos J. J. & Levin B. R. (2006). Modeling the role of bacteriophage in the control of cholera outbreaks. *Proc Natl Acad Sci USA*, 103(12), 4652–7.

Joh, R. I., Wang, H. Weiss, H. & Weitz, J. S. (2009). Dynamics of indirectly transmitted infectious diseases with immunological threshold. *Bull. Math. Biol.* 71, 845–862.

Kumar, A., Kumar, M. & Nilam, A. (2020). A study on the stability behavior of an epidemic model with ratio-dependent incidence and saturated treatment. *Theory in Biosciences*, 139 (2), 225–234.

Kwasi-Do, N., Ohene Opoku & Afriyiel, C. (2020). The role of control measures and the environment in the transmission dynamics of cholera. *Abstract and Applied Analysis*, 2020, Article ID 2485979, 16 pages.

LaSalle, J. P. (1979). The stability of dynamical systems. *Society for Industrial and Applied mathematics*, 21(3), 418–420.

Lewnard, J. A., Ndeffo Mbah, M. L. & Alfaro-Murillo, J. A. (2014). Dynamics and control of Ebola virus transmission in Montserrado, Liberia: a mathematical modelling analysis. *The Lancet Infectious Diseases*, 14(12), 1189–1195.

Lemos-Paiao, A. P., Silva, C. J. and Torres, D. F. M. (2016). An epidemic model for cholera with optimal control treatment. *Journal of Computational and Applied Mathematics*, 11(002): 1 – 20. ISSN 0377-0427.

Liao, S. & Wang, J. (2011). Stability analysis and application of a mathematical cholera model. *Math. Bioscience. Engineering*. 8 (2011), 733–752.

Lilje, J., Mosler, H.-J. & Kessely, H. (2015). Factors determining water treatment behavior for the prevention of cholera in Chad. *The American Journal of Tropical Medicine and Hygiene,* 93(1), 57–65.

Madubueze, C. E., Madubueze, S. C. & Ajama, S. (2015). Bifurcation and Stability Analysis of the Dynamics of cholera model with controls. *International Journal of*

Mathematical, Computational. Physical, Electrical and Computer Engineering, 9(11), 11-21.

Mari, L., Bertuzzo, E. & Righetto L. (2012). Modelling cholera epidemics: the role of waterways, human mobility and sanitation. *Journal of the Royal Society Interface*, 9(67), 376–388.

Mayo Clinic Staff. (2014). *Cholera: Symptoms*. http://www.mavoclinic.org/diseases—conditions/cholera/basics/simptoms/ April 5, 2014. con- —20031469 Accessed December 6, 2014.

Merrell, D. S., Butler, S. M. & Qadri, F. (2002). Host-induced epidemic spread of the cholera bacterium. *Nature*, 417 (2002), 642–645.

Mukandavire, Z., Liao, S., Wang, J., Gaff, H., Smith, D. L. & Morris, J. G. Jr. (2011). Estimating the reproductive numbers for the 2008-2009 cholera outbreaks in Zimbabwe. *Proc. Nat. Acad. Sci.*, 108 (2011), 8767–8772.

Mushanyu, J., Nyabadza, F., Muchatibaya, G., Mafuta, P. & Nhawu, G. (2018). Assessing the potential impact of limited public health resources on the spread and control of typhoid. *J Math Biol*, 77, 647. https://doi.org/10.1007/s0028 5-018-1219-9.

NCDC (2019). Nigeria Centre for Disease Control and Prevention. https://ncdc.gov.ng/diseases/info/C

Neilan, R. L. M., Schaefer, E., Gaff, H., Fister, K. R. & Lenhart, S. (2010). Modeling Optimal Intervention Strategies for Cholera, *Bull. Math. Biol.* 72 (2010), 2004 - 2018.

Nisbet, R. M. & Gurney, W. S. C. (1982). *Modeling Fluctuating Populations*, John Wiley & Sons, New York, 1982.

Nyabadza, F., Aduamah, J. M. & Mushanyu, J. (2019). Modelling cholera transmission dynamics in the presence of limited resources. *BMC Research Notes*, 12(1), 475.

Nyerere, N., Luboobi, L. S. & Nkansah-Gyekye, Y. (2014a). Modelling the Effect of Screening and Treatment on the Transmission of Tuberculosis Infections. *Mathematical Theory and Modelling*, 4(7), 51-62.

Nyerere, N., Luboobi, L. S. & Nkansah-Gyekye, Y. (2014b). Bifurcation and Stability analysis of the dynamics of Tuberculosis model incorporating, vaccination, Screening and treatment. *Communications in Mathematical biology and Neuroscience*, 2014 (2014), Article ID 3.

Ochoche, J. M. (2013). A Mathematical Model for the Transmission Dynamics of Cholera with Control Strategy. *International Journal of Science and Technology 2*(11), 797803

Osman, S., Makinde, O. D. & Theuri, D. M. (2018). Stability analysis and modelling of listeriosis dynamics in human and animal populations. *Global Journal of Pure and Applied Mathematics*, 14 (1), 115–137.

Panja, P. (2019). Optimal control analysis of a cholera epidemic model. *Biophysical Reviews and Letters*, 14 (1), 27–48.

Prasenjit, D. & Debasis, M. (2012). *Biomathematics*, 2012(2012). Article ID 621939, 13 pages. http://dx.doi.orp/l 0.5402/2012621939.

Ratchford, C. & Wang, J. (2019). Modelling cholera dynamics at multiple scales: environmental evolution, between-host transmission, and within-host interaction. *Mathematical Biosciences and Engineering*, 16 (2), 782–812.

Sani, A. F., Ibrahim, M. O. & Danbaba, A. (2013). Deterministic Mathematics model of cholera predicting chances of its outbreak. *International Journal of Scientific and Technology Research*, 2 (4), 1-4.

Stephen, E., Dmitry, K. & Silas, M. (2014a). Modelling and Stability Analysis for a Varicella Zoster Virus Model with Vaccination. *Applied and Computational Mathematics*, 3 (4), 150-162. doi:10.11648/j.acm.20140304.16.

Stephen, E., Dmitry, K., & Silas, M. (2014b). Modelling the Impact of Immunization on the epidemiology of Varicella-Zoster Virus. *Mathematical theory and Modeling*, 4 (8), 46-56.

Sun G., Xie J., Huang S, Zhen J., Ming-Tao, L. & Liqun, L. (2017). Transmission dynamics of cholera: Mathematical modeling and control strategies, *Commun Nonlinear Sci Numer Simulat*, 45, 235–244.

Tian, J. & Wang, J. (2011). Global stability for cholera epidemic models, *Math. Biosci*. 232, 31–41.

Tien, J. H . & Earn, D. J. D. (2010). Multiple transmission pathways and disease dynamics in a Water borne pathogen model, Bull. *Mathematical. Biology*, 72, 1506-1533.

Wang, J. & Modnak, C. (2011). Modeling cholera dynamics with controls. *Canadian applied mathematics quarterly*, 19 (3), 255–273.

WHO (2019). Cholera—World Health Organization, 2019, https://www.who.int/newsroom/fact-sheets/detail/cholera.

WHO (2018a). World Health Organization. Cholera-Zimbabwe. Disease outbreak news. 2018. http://www.who.int/csr/don/20-septe mber-2018-chole ra-zimbabwe/en/.

WHO (2018b). Global health observatory data repository: number of reported cases data by country. 2018, http://apps.who.int/gho/data/node.main.175.

WHO (2018c). World Health Organization, *Vaccine-Preventable Diseases Surveillance Standards*, World Health Organization, Geneva, Switzerland, 2018, https://www.who.int/immunization/monitoring_surveillance/burden/vpd/WHO_SurveillanceVaccinePreventable_02_Cholera_R2.pdf.

World Health Organization (WHO) (2014). Global Alert and Response (GAR): Cholera. Continually updated, http://www.who.int/csr/don/archive/disease/cholera/en/ Accessed December 8, 2014

Yang C. (2017). *Impact of Awareness Progams on Cholera Dynamics: Two Modeling Approaches*, M.Sc. Thesis University of Tennessee at Chattanooga, Tennessee.

APPENDIX

Estimation of Variables and Parameter Values

We estimated the parameter values based on the available data from the Nigeria Centre for Disease Control (NCDC), Worldometers and Macro Trends and reliable related literature. The estimates are clearly explained in the following sub-sections.

E1: The Total Population, N

According to Nigeria Centre for Disease Control (NCDC), Worldometers and Macro Trends, the Nigeria total population at 2018, is 198,387,623

The total population N 198,387,623

E2: Birth Rate

The number birth per 1,000 is 38.25

$$\text{Birth rate} = \frac{38.25}{1,000} = 0.03825$$

E3: Constant Recruitment

The number of new birth in 2018 = Recruitment Rate × Birth Rate × Total Population

$$0.03825 \times 198,387,623 = 7,588,327$$

E4: Number of Symptomatic Infected I

The NCDC estimate that, there are 42,466 cases of cholera in Nigeria in 2018, resulting in 830 deaths. We assume that the number of infected persons to be the same as the Total Confirmed Cases

$$I = 42,466$$

E5: Number of Recovered R

From E4 the number of cases is 42,466 and the number of death is 830. Recovered R = 42,466 - 830 = 41,636.

$$R = 41,636$$

E6: Number of Vaccinated V

According to Global Task Force on Cholera Control, the number of vaccinated individual in 2018 is 5,244,305 and the percentage of vaccinated is 101%. Therefore,

Vaccinated V = 5,244,305

E7: Number of Hygiene conscious Individual H

We assume that the 25% of the total population are hygiene conscious.

$$H = 25\% \text{ of } 198,387,623 = 49,596,906$$

E8: Number of Susceptible *S*

$$S = N - V - H - I - R$$

$$S = 198,387,623 - 5,244,305 - 49,596,906 - 42,466 - 41,636$$

$$S =$$

198,387,623 54,925,313 143,462,310

E9: Number of Susceptible aware S_a

We assume that the number of Susceptible aware S_a are less than the number of Susceptible unaware S_u. Also, that the 30% of the number of Susceptible **E8** are aware of cholera.

S_a = 30% *of* 143,462,310 = 43,038,693

E10: Number of Susceptible unaware S_u

Number of Susceptible unaware

S_u = *Numberof Susceptible* − *Numberof Susceptible aware*

= 143,462,310 − 43,038,693 = 100,423,617

E11: Natural Death Rate

The number death per 1,000 is 18

$$\text{Natural Death rate} = \frac{18}{1000} = 0.018$$

E12: Disease-induced death Rate α_1

$$\alpha_1 = \frac{\text{Total Death of Cholera}}{\text{Total Confirmed cases}} = \frac{830}{42466} = 0.0195$$

E13: Recovery Rate

From E4 and E5

$$= \frac{\text{Recovered}}{\text{Number of Cases}} = \frac{41634}{42466} = 0.98$$

E14: Vaccination rates for susceptible unaware humans α_2

We assume that the rate at which susceptible unaware humans are vaccinated α_2 is less than the rate at which susceptible aware humans are vaccinated α_3. According to Global Task Force on Cholera Control, the percentage of vaccinated is 101%. Therefore,

$$\frac{1}{0.3366} x\ 1.01$$

E15: Vaccination rates for susceptible aware humans α_3

We assume that the rate at which susceptible unaware humans are vaccinated α_2 is less than the rate at which susceptible aware humans are vaccinated α_3. According to Global Task Force on Cholera Control, the percentage of vaccinated is 101%. Therefore,

$$\alpha_3 = \frac{2}{3}\ x\ 1.01 = 0.6733$$