ADDIS ABABA UNIVERSITY

COLLEGE OF NATURAL AND COMPUTATIONAL SCINCE

DEPARTMENT OF MATHEMATICS

M.sc THESIS ON MATHEMATICAL MODEL AND ANALYSIS OF THE IMPACTOF INFECTIVE IMMIGRANTS ON THE TRANSMISSION OFOF CHOLERA DISEASE WITH CONTROL STRATEGY.

By; Desta Worku

In Partial Fulfillment of the Requirement of Degree of Master of Science in Applied Mathematics.

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Approval sheet 1

This is to certify that the thesis titled" MATHEMATICAL MODEL AND ANALYSIS

OF THE IMPACT OF INFECTIVE IMMIGRANTS ON THE TRANSMISSION OF CHOLERA DISEASE WITH CONTROL STRATEGY" submitted in partial fulfillment of the requirement for the degree of Master of Science in Applied Mathematics to the Department of Mathematics Addis Ababa University, and is record of original research carried out by Desta Worku, ID.GSK/0602/09 under my supervision and no part of the thesis has been submitted for another degree. The assistance and the help received during the course of this investigation have been duly acknowledged. Therefore, I recommended that it may be accepted as fulfilling the thesis requirement.

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We, the undersigned, members of the Board of Examiners of the final open defense by **DESTA Worku** have read and evaluated his thesis entitled "**MATHEMATICAL MODEL ANDANALYSIS OF THE IMPACT OF INFECTIVE IMMIGRANTS ON THE TRANSMISSION OF CHOLERA DISEASE WITH CONTROL STRATEGY"** and Examined the candidate. This is therefore to certify that the thesis has been accepted in partial fulfillment of the requirement of the degree of Master of Science in Applied Mathematics.

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Declaration

I declare that this thesis is my original work and that all source materials used for this thesis have been properly cited and acknowledged. This thesis has been submitted in partial fulfillment of the requirements for M. Sc. degree in Applied Mathematics at Addis Ababa University. I earnestly declare that this thesis is not submitted to any other institution anywhere for the award of any academic degree.

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Abstract

Cholera is water born infectious disease caused by ingesting food or drinking water contaminated with a bacterium called vibrio cholera, characterized by extreme diarrhea and vomiting. In this thesis, we present mathematical model and analysis of the impact of infected immigrants on the transmission of cholera disease by adding some control strategy. In our work, we considered public health education, medical treatment and water treatment as the control strategy that limits the disease. The positivity and boundedness of the model system, and the existence and stability of the equilibrium points are discussed. Using next generation matrix, the reproduction number is calculated. Numerical simulation done using the MATLAB software code ode45 that shows when health education is effective or apply strongly, the number of infective are decrease faster, implying that health education and awareness are vital in controlling the spread of the cholera disease together with water treatment and medical treatment.

Keywords: mathematical model, cholera, control strategy, equilibrium point, numerical simulation and reproduction number.

List of Abbreviations

HIV - Human Immunodeficiency Virus.

AIDS - Acquired Immune Deficiency Syndrome.

SI -Susceptible, Infective.

SIB - Susceptible, Infective, Water born

SIBR - Susceptible, Infective, Waterborne, Recovered.

MATLAB -Matrix laboratory.

SIR- Susceptible, Infective, Recovered.

Definition of basic terms

Susceptible population(S) - The susceptible class has not yet infected by the disease, but they can infected by ingestion of water or food contamination and the interaction between infected human and susceptible human.

Infective population (I) - These populations are already infected by the disease and they can transfer the disease to the susceptible population.

Recovered populations(R) - These classes of human populations, who have been infected, and they are developing immunity and then removed from the disease either due to immunization or due to death.

Diarrhea - The passage of loose, fluid stools or an increase in stool frequency at least three times a day.

Vibrio cholera –species of gram-negative, comma shaped bacterium.

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CHAPTER ONE

1. INTRODUCTION

1.1. Background of the study

The outbreak of infectious disease causes death of millions of people as well as expenditure of enormous amount of money in health care and disease control [1,5]. It is important that adequate attention is paid to stopping the spread of such diseases by using effective control measures [5]. Many infectious diseases are as a direct result of poor hygienic conditions and contact between an infectious person and a susceptible person and immigration of infective individuals. Infectious diseases like smallpox, tuberculosis, cholera, polio, HIV/AIDS, influenza, malaria, dengue, Ebola are the second leading cause of death global, particularly in low and middle income countries [16].Cholera is a disease that is endemic in underdeveloped nations and is characterized by factors like inadequate access to clean water and sanitation, low employment rates, low levels of education, and inadequate schooling.

Vireo cholera transmission is encouraged by the use of high-risk foods, contaminated water, and inadequate sanitation, all of which are correlated with low socioeconomic position and poverty.

Humans are the only animal that affected by cholera disease [17].Cholera is an acute intestinal infectious disease caused by the bacterium vibrio cholera, it characterized by extreme diarrhea and vomiting. It is deadly water born disease, which usually results from poor hygienic conditions and sanitation and untreated water. The human body is dehydrated and prevented from absorbing liquids and toxins produced by the vibrio cholera. Individuals who are not treated may die from severe dehydration two or three days of the infection and this is due to the relatively short incubation period varying from about two hours to five days. The infected person shows the following symptoms after infection which includes watery diarrhea, frequent vomiting, loss of skin elasticity, muscle cramps, rapid heart rate, impatience, thirst, and low blood pressure [3,5,33].Increasing the protection against the disease can be gained by improving sanitation and hygiene. However, life threatening, controlling and prevention is achieved if proper sanitation practices are followed. Most cases of cholera currently occur in developing countries such as Peru, Bangladesh, India and some of the coastal region of South America Recent cholera outbreak in Haiti (2010-2011), Nigeria (2010), Kenya (2010),Vietn(2009), Zimbabwe (2008-2009), etc., continue leading to a large number of infections and receiving worldwide attention. Cholera has been a persistent epidemic and continues to be a global world health issue. Despite the studies of this disease for over one hundred years, it is estimated that approximately 120,000 people die from cholera annually [1].This work aims to present a more comprehensive understanding of the mechanism governing the transmission of cholera by mathematical modeling including four compartments of a nonlinear differential equation system.

Mathematical modeling that involving system of differential equation enables us to characterize the general and specific behavior of these systems analytically and to understand which aspects contribute the most to the observed dynamics as well as making policy decision for preventive measure and control strategy [5]. The application of system of differential equation in the transmission dynamics of infectious disease have been widely used in several papers. The analysis of these models predicted and suggested several control mechanisms or strategies for the control and eradication of the infections. It is important that adequate attention is paid to stopping the spread of the disease by using some control strategies and measures .Such as medical treatment, public health education and water treatment [5]. Key factor in the disease's ability to spread or transmit is a lack of knowledge about cholera.

Therefore, public health education is one of the basic mechanisms in control of cholera disease together with medical treatment and water treatment. Education provided by using different tools such as social media, radio, oral communication, etc. This creates awareness to the community in order to them improving sanitation, hygiene their selves and they identify the symptoms of the cholera disease and take medical treatment rapidly as the symptom happened.

1.2 Statement of the problem

Many infectious diseases are as a direct result of contaminated food and water, contact between an infectious person and a susceptible person, the role of infected person in adequate environment.

The bacterium vibrio cholera is the source of this severe intestinal infectious disease, which is characterized by severe diarrhea and vomiting. And it causes a worldwide mortality, especially in developing countries with limited health care resources. If one person is infected among the members of one family all other member can be infected through direct or indirect contamination and death can occurred if there is no treatment.
Most cases of cholera disease occur in remote areas of developing countries, and then the number of patients are uncertain because most cases go unreported. Individuals live in a place where insufficient water treatment, lack of public health education about transmission of cholera, poor sanitation and inadequate hygienic condition are a great risk for cholera. Therefore, cholera outbreak have a serious negative consequence on public health, psychologically, social and economic development.

Therefore, this study provides answers to the following research questions in order to manage the dynamics of cholera transmission.

- What is the role of public health education on the control of the transmission of cholera disease together with some control measure?
- How to investigate the cholera transmission mathematical model?
- How can the mathematical model of cholera transmission's basic reproduction number be found?
- What effect do infectious immigrants have on the updated model?

1.3 Objective of the study

This study has both general and specific objectives.

1.3.1 General objective

The general objective of this study is to formulate mathematical model and analysis of the impact of infective immigrants on the transmission of cholera disease with control strategy.

1.3.2 Specific objectives

The specific objectives of this study are:

- ✓ To formulate a modified mathematical model for cholera disease
- ✓ To ascertain equilibrium locations and the fundamental reproduction number R0
- ✓ To examines positivity and boundedness of solution of the modified model system.
- ✓ To analyzes the local and global stability of the equilibrium points.
- ✓ To demonstrate how various treatments such as medical care, public health awareness campaigns, and water treatment affect the altered mathematical model
- ✓ To use numerical simulation to determine the effects of various parameters.

1.4 Significance of the study

The study will be useful for the following purposes:

- ❖ To control cholera disease transmission using mathematical modeling.
- ❖ To suppose the controlling mechanism of cholera.
- ❖ To identify the impact of public health education, medical treatment and water treatment on the dynamics of cholera transmission.
- ❖ It gives background knowledge to researchers who wish to work on related subjects.

CHAPTER TWO

2. REVIEW OF RELATED LITERATURE

2.1 Mathematical model of infectious disease

This section examines the research on cholera transmission and the application of mathematical models to the dynamics of infectious diseases.

In order to develop a mathematical model of infectious disease, we have to identify the causative agents of the disease, because infectious disease may be caused by virus, bacteria, protozoa, etc. and they can be transfer from one host to another host through different mode of transmission and these disease comes to the host through direct contact, like touching, sexual intercourse for example HIV/AIDS, or indirect contamination of objects, food or water for example cholera. The main objective of mathematical modeling of infectious disease is to show the transmission way of the disease by dividing the population, consisting of N individuals into a group of compartments, like; Susceptible, Exposed, Infective and Recovery. Thus different researchers studied about infectious disease through mathematical modeling and using the concept of system of first order differential equation.

During the development of mathematical modeling in epidemiological aspects of infectious disease, then to study the transmission way of infectious disease, deterministic models played a central role. Various studies produced various mathematical models of the cholera disease's propagation and management plan.

Now we will see some of the literature that is done before this paper analyzes the cholera illness mathematical model. We use that literature as the guide for our work. A mathematical model is vital tool to control disease. The first mathematical model for the transmission dynamics of cholera was done by Capasso and PavarieFontana in Italy, 1973. They develop the model using system of differential equation to describe the transmission way of cholera disease [28].

Using the well-known cholera disease model created by Codeco, she extends and generalize the work of Capasso and Pavarie-Fontana by adding the susceptible compartment, and her work is more accessible and widely used for other researchers. Her work mainly focuses on the aquatic reservoir and in the population devoid of cholera every person was believed to be susceptible.And recruited by birth. Her model is simple and step forward to understand the concept of the outbreak and transmission of cholera disease [6].

Next to the Codeco cholera model, Hartley, Morris and Smith , proposed the cholera model by the inclusion of the hyper infection and the stated the about the vibrio bacteria using the novel laboratory. They proposed that hyper infection was found in the hallway of vibrio cholera through human host increases the infectivity of bacteria and after shedding the infectivity lived around five hours, and it returns to their usual state. They derived the reproduction number R_0 to examined the effect of hyper infection in the model [22].

The model was proposed by Bertuzo et al. to investigate the effectof river network, it is the hallway of bacteria to move between human population hosts to represent the river network.

Brtuzo defines environmental matrix which had information about the directed graph and each node represents the flow of the disease [23].

Miller-Neilanet, al. proposed the mathematical model to extend the work of Codeco and Hartley to develop the model of cholera by adding asymptotic case and hyper infection bacteria. Their goal was to identify the most effective plan to take for managing the disease, which included immunization, antibiotics, cleanliness, and rehydration [27].

Mukandavire ET, al., was proposed cholera outbreak model that include both direct and indirect transmission pathway in Zimbabwe.In order to minimize the disease, they calculated the basic reproduction number [24].

Mwasa and Tchuenche , propose the cholera mathematical model by adding or the inclusion of education as the control strategy. Their model considered a homogenous population and they include new compartments in order to vaccinated, educated, quarantined and treated populations. They assumed that educated person was very hygiene, and report their infection quickly as the infected. And also they assumed that initially human populations are susceptible. They examined the stability of disease free equilibrium point to show the effect of it on the basic reproduction number and they also examined the sensitivity analysis to show the consequence of every variable on the cholera disease transmission [25].

Andrew and Basu , proposed the cholera mathematical model by extending the Codeco's model of cholera disease to simulate the effect of clean water, vaccination on cholera cases in Haiti. They consider the population as a heterogeneously mixed [26].

Ochoche Jeffry M., proposed a simple SIB mathematical model by extending slightly the Codeco's mathematical model. He examined the control of movement of cholera transmission using water treatment, considering that cholera is spread by consumption ofcontaminated water and also In order check the stability of the endemic and disease-free equilibrium points, he evaluated the reproduction numberand point out the simulation to show the effect of water treatment within no control, weak control and strong control strategy [5].

One of the most important threshold parameters, or invariants, which characterize mathematical aspects associated with infectious disease models, is the basic reproduction number.
It is widely used in mathematical epidemiology models and it designed in a simple and cleared way by VandenDriessche and Watmongh (2002-2008) by using next generation matrix [8, 9 ,30].

In this study, we begin from the Ochoche J. M. mathematical model of cholera disease by considering the infective immigrant and Contact between people is the primary means of disease spread; medical treatment, water purification, and public health education are the methods of control.

2.2 Life history of vibrio cholera

Historically, one of the most ancient epidemic involving diarrheas that attacks various regions of the world is cholera.Humans are the only animal who affected by the cholera disease. The word cholera derived from the Greek word chole, meaning"flow of bile". First it was discovered in the subpart of India by Thomas Sydenham in 1817 [19]. Flippo pacini discover the curved shap**e** bacteria in the feces of Italian cholera victims

That cause for cholera disease in 1854, and he gave the name vibrio cholera when he was published his observational work [20]. Today the term cholera called constantly suspected infection caused by vibrio cholera, sometimes called Asiatic cholera; rice water etc. John snow was the first man who hypothesizes the modern study of cholera. As his hypothesis cholera is transmitted by the pass way of contaminated water in 1849 [19].

The disease reaches out Asian continent in 1991, however it is endemic all parts of the world especially in Africa and Asia. 177,963 cholera cases and 4031 death cases were reported by world health organization in 53 countries from these 67% cases occurred in African countries including Ethiopia and Somalia [20].

In general from analysis of vibrio cholera, cholera affects mainly African countries which have densely populated area. It has been lived different geographical regions with different climatic conditions; mostly it lived in unhygienic climatic conditions. Social disruption area, poverty, poor sanitation, hygienic conditions are the prevalence and mortality rates in Africa.

Epidemiologicallythe ability of cholera to remain endemic in all affected regions and its capacity to generate epidemics with pandemic potential are its distinctive features, maybe the most common among many nations.

The new observation towards the life cycles of vibrio cholera has allowed a better understanding of cholera transmission. Vibrio cholera lives in aquatic environment which its natural reservoirs. From its aquatic environment vibrio cholera is induced in to human population through different transmission pathway, such as contamination of food and water sources.When contaminated humans transfer the bacteria into the environment and pollute food and water sources, the transmission pathway cycle is closed. After human infection, a significant attack rate may occur, particularly in populations that had not previously been exposed. [21].

2.3 Cholera disease and microbiology

Cholera defined in the sense of microbiology as, vibrio cholera is a. gram-negative, curved bacillus with dimensions ranging from 0.5 to 0.8 m in diameter and 1 to 3 m in length.

The bacteria's single polar flagellum gives them a unique movement that may be viewed using a microscope.

Figure 1: image of vireo cholera

CHAPTER THREE

3. MATHEMATICAL METHODOLOGIES

3.1 Mathematical epidemiology

The subject that deals with the transfer of infectious disease among people is called epidemiology.Mathematical epidemiology concerns about obtaining, understanding, putting assumptions according to epidemiological behaviors within mathematical language and finding the solution of mathematical problems by relating the solution with the real life epidemiological aspects. It also concerns with quantitative aspects of the subject and usually consists of: model construction, parameter estimation, finding numerical solution, and Sensitivity evaluation of the model to different parameter changes**.**

We have two mathematical epidemiological models for infectious disease. These are:

1. Stochastic model: studies small population size in the community, which is a chance of variation in risk of exposure, disease and other factors.

2. Deterministic model: known as compartmental model that describes what is likely to occur on average at a scale population and matches with a big population.

The model classifies individuals into different groups or compartments using the concept of systems of first order differential equation. These compartments are SI, SIS, SIR, SIRS, SEIR etc. These basic models are formulated with demography or none demography.

3.2 Method of the study

This section aims to introduce a number of mathematical ideas and methods that will be applied throughout the thesis in order to do a qualitative analysis of the models that are discussed in the following chapters.

3.3 Equilibrium points

Definition3.3: Equilibrium point is a critical or fixed point, which makes the given system of equation is zero. Let $y^0(t) = f(y)$, where $f : R^n \rightarrow R^n$ be a map and y is a function of t, be a certain system of differential equation, then y_e is said to be the equilibrium point of the given system if $f(y_e) = 0$, for all time $t \in [t_0, \infty)$ where t_0 is the initial time of the system[31].

3.4 Equilibrium point stability

Definition3.4. the phrase "equilibrium point stability" describes the behavior of the solutions of the model equation with initial conditions near the equilibrium points, as time tends to infinity ($t \to \infty$) [31]. The behavior of stability is corresponds to the perturbation of the equilibrium point. i.e. the equilibrium point y_e is said to be stable, if given$e > 0$, there is $\delta > 0$, such that, for any y_0,$|y_e - y_0| < \delta$, the solution y(t) through y_0 which satisfy$|y(t) - y_e| < e, \forall t \geq t_0$. y_e is,if it is stable, asymptotically stable and the solutions are exactly converges to the equilibrium point. i.e. there exist $\delta > 0$, such that $|y_e - y_0| \to y_e$, for all $t \to \infty$ [31].

3.4.1 Routh-Hurwitz criteria

Criterion of Routh-Hurwitz Stability is used to determine asymptotic stable of an equilibrium point for a nonlinear system of differential equation. At any steady state solution E we will have the Jacobean matrix of the system of differential equation given by $det(J_E - \lambda I) = 0$ and the characteristics equation is: $P_n(\lambda) = a_n\lambda^n + a_1\lambda^{(n-1)} + \ldots + a_{(n-1)}\lambda + a_0 = 0$. Solving this equation might be difficult, but for the stability, we need to determine the sign of the eigen values. We know that the equilibrium point is stable if $Re\ (\lambda) < 0$ for all λ the stability conditions of determined by the Routh-Hurwitz criteria as follow using the above characteristics equation where$a_i$are all constants using the coefficients $a_i$of the,The characteristics equation of the nth Hurwitz matrices is defined by i = 1,2,...,n., for $n = 1,2,3,\ldots,k$ then the Routh Hurwitz matrix becomes:$H_1 = (a_1)$, $H_2 = \begin{pmatrix} a_1 & 1 \\ 0 & a_2 \end{pmatrix}$ $H_3 = \begin{pmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_1 \end{pmatrix}$

And in general,$H_n = \begin{pmatrix} a_1 & 1 & 0 & 0 & \cdots & 0 \\ a_3 & a_2 & a_1 & 1 & \cdots & 0 \\ a_5 & a_4 & a_3 & a_2 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 & 0 & \cdots & a_k \end{pmatrix}$

.Where if i > k, $a_i = 0$. If and only if the determinants of the Routh Hurwitz matrices are positive, then all of the polynomial's roots have negative real parts.$det(H_i) > 0$ for $i = 1,2,3,\ldots,n$ for a polynomial of degree $n = 2,3and4$, the Routh-Hurwitz criteria are summarized as follows. n = 2, $a_1 > 0$ and $a_1a_2 > 0$, n = 3, $a_1 > 0$,$a_3 > 0$,and $a_1a_2 > a_3$, n = 4, $a_1 > 0$,$a_3 > 0$,$a_4 > 0$ and $a_1a_2a_3 > (a_3)^2 + (a_1)^2a_4$.

3.4.2 The Basic Reproduction number

One of the most important threshold parameters, or invariants, that characterize mathematical aspects related to infectious disease models is the basic reproduction number.

It is commonly used in models of mathematical epidemiology. The analysis of the model includes finding equilibrium points the model,calculating the fundamental reproduction number R0 and examining the stability of the equilibrium points according to endemic and disease-free conditions [8, 9, 31].

3.4.3 Next generation matrix

The compartmental infectious disease model with ordinary differential equations can effectively determine the biologically acceptable reproduction number using the Jacobian matrix.

But, the model is complex to.Use the Routh-Hurwitz criteria to determine the Jacobian matrix's stability.It is designed by in a simple and cleared way by VandenDriessche and Watmongh (2002-2008) by using next generation matrix [8, 30].

Let X = $(x_1, x_2, \ldots, x_n)^t$ represent the number of people in each compartment, with $x_i > 0$ and $\forall$ i = 1,2,3,...,n. and the compartmental model contains infected individuals and x_0 be the disease free equilibrium point which is stable in the absence of disease and $x_1, x_2, \ldots, x_m$ be the linearized equation at x_0.

Thus $\frac{dx_i}{dt} = f_i(x) - v_i(x)$), for I = 1, 2, 3... M, where $v_i(x) = (v_i^-(x)) - (v_i^+(x))$, and

. f_i Is the rate at which new infections emerge in the compartment i , which only comprise recently emerging illnesses, but it does not include the terms which are the transfer of infectious person from one sick room to another.

.The rates of other transmission into and out of the compartment are $v_i^+(x)$ and $v_i^-(x)$, respectively.

Thus F= $\frac{\partial f_i}{\partial x_j}(x_0)$V= $\frac{\partial V_i}{\partial x_j}(x_0$ and, for j ≤ *i*, *j*≤ *m* and *F* has no negative entries and *V* is

a Metzler matrix (or the off diagonal elements of F are non-negative), then there exist non negative entries of V^{-1} (inverse matrix of *V*) [8, 30, 31]. Thus we have the matrix FV^{-1} ofa non-negative entrance vector that predicts the quantity of new infections and it has (*I*, *j*) entries where *i* = *j* and is equals to the rate at which the expected number of secondary infections in compartment *i* produced by an infection introduced in compartment j. And the (*j*,*k*) entry of V^{-1} is the average time an infected individual spend in compartment j during its life time. Thus, FV^{-1} has an entry of (*i*,*k*), where *j* = *k*, which is the expected number of new infection in compartment i produced by the infected individual originally introduced into compartment j . Thus, by considering the infected human and vibrio cholera, reproduction number is computed by using the next generation matrix FV^{-1}, approach. Since ρ is the spectral radius (dominant eigenvalue of the matrix $FV^{-1} - \lambda I$), the reproduction number R0 = ρ (FV^{-1}) I is the identity matrix of order equal to order of FV^{-1}. These functions *F* and *V* supposed to be at least twice continuously differentiable. Also it satisfy the condition that $x_e = \{x_i \geq 0 : x_i = 0; i = 1,2,\ldots,m\}$, here x_e represents the set of all disease free states. We assume that these functions satisfy the following hypothesis [8, 30].

H_1: If $x_i \geq 0$, then $v_i(x) \geq 0$, $v_i^-(x) \geq 0$, for *i* = 1,2,...,*n*.

H_2: $x_i = 0$, then $v_i^-(x) = 0$ and in particular $v_i^+(x) = 0$ if $X \in X_e$ for *i*= 1,2,...,*m*.

H_3: $f_i = 0$, i > *m*, the fact that the rate at which infection occurs in an uninfected compartment is zero.

H_4: $f_i = 0$ and $v_i^+(x) = 0$ if $X_e \in$, i = 1, 2... m. This condition is to protect

Against the disease-free subspace being altered and this assumption H_4 implies that if a population is free of disease then it remains free with no possibility for immigration of infective into the compartment.H_5: All of Df(x0)'s eigenvalues have negative real parts if F(x) = 0

3.4.4 Descartes' rule of signs

We can use Descartes' rule of sign to find a polynomial function's real zero value.

Definition3.2: For a non-negative integer n, $f(x) = a_n x^n + a_{(n-1)}x^{(n-1)} + ... + a_1 x + a_0$ be a polynomial function, where $a_n, a_{(n-1)}, ..., a_1, a_0$ are real coefficients and $a_n \neq 0$. Then:

Until 1 or 0 is reached, the number of positive real zeros of f is either less than or equal to the number of sign changes of the subsequent terms of f(x).
The number of negative real zeros in f is either fewer than that amount by an even integer (up to 1 or 0 is obtained) or equal to the number of sign changes of the subsequent terms in f(−x).

CHAPTER FOUR

4. THE MATHEMATICAL MODEL FORMULATION

4.1 The Existing Model:

The following is the existing mathematical model for the transmission of cholera.

$\frac{ds}{dt}$= bH-$\frac{\alpha B}{K+B}$S-μS …… (1)

$\frac{dI}{dt}$=$\frac{\alpha B}{K+B}$S-(΄Y+μ)I ……(2)

$\frac{dB}{dt}$=eI − (w-p)B,……(3)S (0) = H > 0, I (0) ≥ 0, B (0) ≥ 0.

The first equation explains the susceptible dynamics of the constant size H. Susceptible people may become infected at a rate$\frac{\alpha B}{K+B}$where α is the rate of contact with untreated water and $\frac{B}{K+B}$ is the probability of such a person to contract cholera. The natural death rate of μ also results in a decrease in the susceptible population.

.The second equation describes the dynamics of infected people in the community. Infected population increases due to the incidence rate of$\frac{\alpha B}{K+B}$and they decrease because of a natural death mortality because of cholera the rate of μ and ΄Y respectively.

The third equation describes the dynamics of the pathogenic vibrio cholera in the aquatic reservoir, in this case, the untreated water consumed by the population. The pathogenic vibrio cholera increases due to the contribution of infected population at the rate of e and the decreases due to water treating rate w. The pathogenic vibrio cholera incidence rate rises as a result of p. The formula for this model's reproduction number is‖ R_O=eαbH/(Kμ(w-p)(΄Y+μ)).

*Asumption*s of existing model are All parameters used in model are positive existing model lacks some control strategy that limits spread of cholera disease. Like public health education, water treatment and medical treatment .Infected individuals become recovered.

4.2 The Modified model

4.2.1 Model formulation

Mathematical model helps to understand the transmission and control of cholera disease. To make the mathematical model that describe the transmission dynamics of cholera disease we use the concept of system of differential equation, where human population and vibrio cholera population interact through contaminated environment, food or water. We investigate the relationship between the susceptible, infected, recuperating, and vibrio cholera in the modified model in order to examine the dynamics of cholera disease transmission.

We begin the model which done by Ochoche J.M. in 2013, by introducing public health education parameter, medical treatment to the infective individual and these individuals are joined to the recovered individual through either by death or by developing immunity. Public health education is the control strategy together with medical treatment and water treatment. In order to simplify our study analysis we divide the total population N (t) in to two groups. These are human population and bacteria population at time t.

Susceptible humans:

These human populations are not yet infected by the disease; However, they can spread through contaminated food or water consumption, as well as through contact between an infected person and a susceptible person.

The population members who recover at the rate of q increased the susceptible human populations. and the recruitment of people (either by birth or immigration) into the population, all recruited individuals are assumed to be susceptible at a rate of Λ. This class of population decreases by natural death rate μ, by the contact rate of susceptible population and infected population at the rate $(1-\beta_e)\boldsymbol{\psi}$ where $0 < \beta_e < 1$ is public health education, and they infect at the probability rate of

$(1-\beta_e)\frac{\alpha B_v}{K+B_v}$where α is the rate of contact with bacteria and also they infected with the probability of $\frac{B_v}{K+B_v}$, where K is the concentration of vibrio cholera in the aquatic environment that yields 50% chance of catching cholera , this means that the probability of infection given contact saturated as the concentration of bacteria in the environment becomes very large .

Infected Humans:

These populations are already infected by the disease and they can transfer the disease to the susceptible population. They increase because of the rate of infected immigrant they go to joined the infective compartment at the rate of δ, contact rate between susceptible and infective groups at the rate $(1-\beta_e)\boldsymbol{\psi}$, and contaminated environment at the probability rate of $(1-\beta_e)\frac{\alpha B_v}{K+B_v}$ and they decreases due to natural death by the rate μ, death due to disease by the rate γ and they joined to the recovered individual due to medical treatment by the rate σ and due to developing immunity at therate θ this is dependent on their age, nutrition, and immunity. And also they joined the vibrio cholera byeach affected person's contribution to the environment at the rate of

$(1-\beta_e)$ e.

Recovered populations:

These classes of human populations, who have been infected, and they are developing immunity eliminated from this disease as a result of and afterwards vaccination or mortality. These populations are increased through the infected individuals are get medical treatment at the rate σ and developing immunity at the rate of θ. They will decrease through natural death rate of μ and they join to the susceptible group by immunity loose rate of ρ.

Vibrio cholera (B_v) **population:**

These classes of the bacteria population are infected through contaminated environment and they leave out from the population as a result of water treatment at the rate of ω, and natural death rate of μ_b.Additionally, they rise as a result of the growth rate of the bacteria, which is measured by φ, and the contribution of infected persons, which is measured by $(1-\beta e)\varepsilon$.

4.2.2 The Mathematical Model Assumptions:

We make the following assumptions for the model:

- Recovery occurs either by immunity or successful treatment after infective.
- Susceptible human population recruited by birth or immigration.
- The populations in the model are homogeneous
- Medical treatment applied on the infected human population.
- Cholera occurs in a relatively short period of time and it does not confer a lifelong immunity.
- Public health education is applied on human population.
- Every single parameter in the model is positive

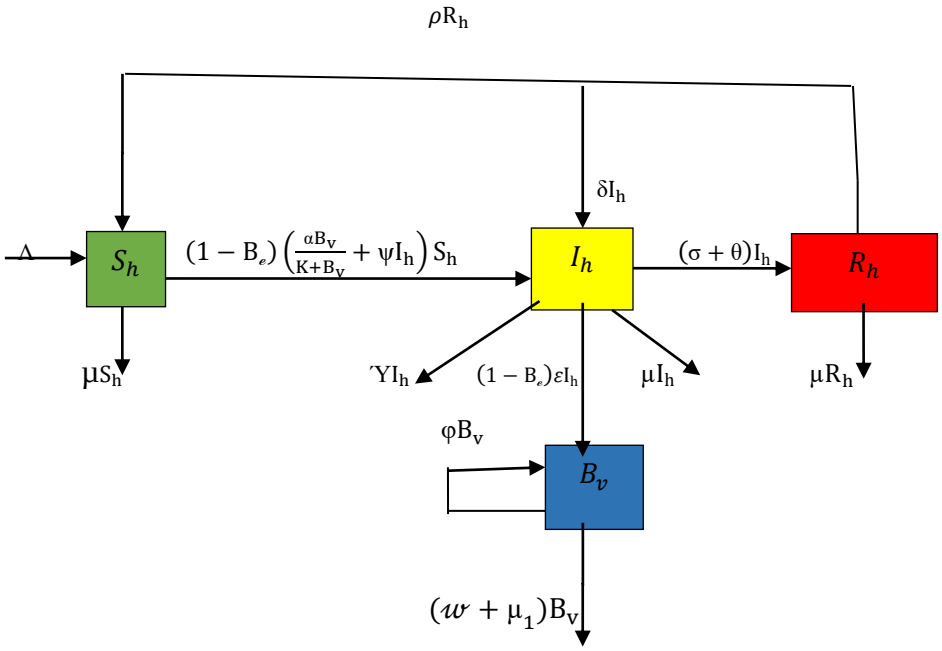

Figure 2: flow chart of the model system

The following table provides an explanation of the state variables and parameters:

Description of state variables

State variables	Description
$S_h(t)$	The number of susceptible human at a time t
$I_{h(t)}$	The number of infective human at a time t
$R_{h(t)}$	The number of recovered human at a time t
$B_{v(t)}$	The concentration of vireo cholera at a time t
$N(t)$	Population total at time t

Table 1: Description for state variables

Description of parameters

Parameters	Description
Λ	Recruitment rate of the susceptible human population
α	Rate of exposure to contaminated food and water
μ	Natural death rate of human population
κ	Concentration of vibrio cholera in water
$\boldsymbol{w}$	Death rate of vibrio cholera as a result of water treatment (water treatment)
β_e	The Efficiency rate of public health education such that $0 < \beta_e < 1$
ε	.Each sick person's contribution to the vibrio cholera population in the aquatic environment
σ	Efficiency Medical Treatment rate of contagious humans
θ	Immunity develop rate of percentage of contagious humans
ρ	Theresistance loose rate of recovered individuals
Υ	The loose rate of infective due to disease
φ	The growth rate of vibrio cholera
μ_1	Natural vibrio cholera mortality rate
δ	The rate of infected immigrants
$\boldsymbol{\psi}$	The interaction rate of individual population

Table 2: an explanation of the parameters

The following system of differential equations is obtained from the model in figure 2.

$$\frac{dS_h}{dt} = \Lambda - \left(1 - \beta_e\right)\left(\frac{\alpha B_v}{K+B_v} + \psi I_h\right) S_h - \mu S_h + \rho R_h \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (4)$$

$$\frac{dI_h}{dt} = (1 - B_e)\left(\frac{\alpha B_v}{K+B_v} + \psi I_h\right) S_h - (\sigma + \theta + \Upsilon + \mu - \delta) I_h \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (5)$$

$\frac{dB_v}{dt}=(1-B_e)\varepsilon I_h-(\omega+\mu_1-\varphi)B_v$…….. (6)

$\frac{dR_h}{dt}=(\sigma+\theta)I_h-(\rho+\mu)R_h$, …… (7) $S_h>0$, $R_h\geq 0, I_h\geq 0$, $B_v\geq 0$

Here, $\frac{\alpha B_v}{K+B_v}B_v$is the incidence or probability rate which determines the rate of new infection.

4.3 Basic properties of the model

4.3.1 Positive solution of state variables

We must demonstrate that the solutions to the system of model equations (4) through (7) are positive for all values of time, as the model examines the population of individuals.
For example, if a population is negative it is unfeasible. This fact is stated in the following theorem and proved as follow:

Theorem 3.1 (positive solution of state variables)

Let $\Omega\{(S_{h(t)}, I_{h(t)}, R_h(t), B_{v(t)}) \in R_+^4 : \ S_h>0\ R_h\geq 0\ I_h\geq 0\ B_v\geq 0\}$, then

the solutions of($S_{h(t)}, I_{h(t)}, R_h(t), B_{v(t)}$) of the system equation (4) − (7) are positive for all $t \geq 0$ [16].

Proof: We have to prove that for all $t\geq 0(S_{h(t)}, I_{h(t)}, R_h(t), B_{v(t)})$ is positive inR_+^4. We know that all the parameters used in the system are positive.

If we consider the first differential equation (4) of the system (4) - (7), we have:

$\frac{dS_h}{dt}=\Lambda-(1-\beta_e)\left(\frac{\alpha B_v}{K+B_v}+\psi I_h\right)S_h-\mu S_h+R_h$. By omitting the expression $-(1-\beta_e)\left(\frac{\alpha B_v}{K+B_v}+\psi I_h\right)S_h+\rho R_h$due to absence of disease we have $I_{h(t)}, R_h(t), B_{v(t)} equallsto\ zero$

Then this can be expressed as:$\frac{dS_h}{dt}\geq \Lambda-\mu S_h$.

$$\Rightarrow \frac{dS_h}{dt}+\mu S_h\geq \Lambda$$

$\frac{dS_h}{dt}+\mu S_h\geq \Lambda$ …….. (8)

This is first order non-homogeneous ordinary differential inequality. The integrating factor of inequality (8) is given by $I(t)=exp^{-\int \mu dt}(exp^{\int \mu dt}\Lambda dt+c)$hence the analytic solution of the inequality (8) becomes:

$$S_h(t)\geq exp^{-\int \mu dt}(exp^{\int \mu dt}\Lambda dt+c\ ),$$

$$\Rightarrow S_h(t)\geq exp^{-\mu t}(exp^{\mu t}\Lambda dt+c),$$

$\Rightarrow S_h(t) \geq exp^{-\mu t}\left(\frac{exp^{\mu t}\Lambda}{\mu} + c\right)$,

$\Rightarrow S_h(t) \geq cexp^{-\mu t} + \left(\frac{\Lambda}{\mu}\right)$,

This together with the initial condition $S_h(0) = S_{h0}$, we get $S_h(t) \geq exp^{-\mu t}\left(S_{h0} - \frac{\mu}{\Lambda}\right) \geq 0$

Now as $t \to \infty$, the solution of $S_h \to \frac{\mu}{\Lambda}$.Therefore,$S_h(t) \geq 0$ for all t.

Therefore the susceptible state variable is always positive for all forward time t.

Again if we take the second differential equation (5) of the system (4) - (7) we have

$\frac{dI_h}{dt} = (1 - B_e)\left(\frac{\alpha B_v}{K + B_v} + \psi I_h\right) S_h - (\sigma + \theta + \acute{\Upsilon} + \mu - \delta) I_h$. And this implies,

$\frac{dI_h}{dt} \geq -(\sigma + \theta + \acute{\Upsilon} + \mu - \delta) I_h$ …… (9)

Integrating equation (9) above using separation of variable method and the analytic solution obtained as:

$$\int \frac{dI_h}{I_h} \geq \int -(\sigma + \theta + \acute{\Upsilon} + \mu - \delta)dt$$

$\Rightarrow \ln I_h \geq -(\sigma + \theta + \acute{\Upsilon} + \mu - \delta)t + c_2$,

Wherec_2 is constant of integration.

$\Rightarrow I_h \geq exp^{-(\sigma+\theta+\acute{\Upsilon}+\mu-\delta)t + c_2}$,

$\Rightarrow I_h \geq exp^{c_2} exp^{-(\sigma+\theta+\acute{\Upsilon}+\mu-\delta)t}$$I_h(0) = I_{h0}$, then we have$exp^{c_2} = I_{h0}$, $c_2 = \ln I_{h0}$.

Therefore,$I_h(t) \geq I_h(0) exp^{-(\sigma+\theta+\acute{\Upsilon}+\mu-\delta)t}$ where $I_h(0)$is constant and it represents initial population of infective state variable and it is positive. Now as $t \to \infty$, the solution of

$I_h(t) \geq 0$. Therefore the infective state variable$I_h(t)$is always positive for all $t \geq 0$.

From the third differential equation (6) of the system (4) - (7) we have:

$\frac{dB_v}{dt} = (1 - B_e)\varepsilon I_h - (\omega + \mu_b - \varphi) B_v$.And this implies

$\frac{dB_v}{dt} \geq -(\omega + \mu_1) B_v$ …… (10)

Similarly integrate equation (10) above using separation of variable and the analytic solution obtained as:$\Rightarrow \int \frac{dB_v}{B_v} \geq \int -(\omega + \mu_1)dt$

$\Rightarrow \ln B_v \geq -(\omega + \mu_1)t + c_3$, where c_3 is constant of integration.

$\Rightarrow B_v \geq exp^{-(}(\omega + \mu_1)^t + c_3$, from initial conditions$B_v(0) = B_{v0}$, then we have

$exp^{c3} = B_{v0}$, and this implies $c_3 = \ln B_{v0}$

Therefore,$B_v(t) \geq B_v(0) exp^{-(\omega+\mu_1)t}$where$B_v(0)$is constant and it represents initial population of vibrio cholera state variable and it is positive. Now as $t \to \infty$, the solution of

$B_v(t) \geq 0$. Therefore the vibrio cholera state variable is always positive for all $t \geq 0$.

And also from the fourth differential equation (7) of the system (4) - (7) we have:

$\frac{dR_h}{dt} = (\sigma + \theta)I_h - (\rho + \mu)R_h$.Since$I_h$is positive we get:

$\frac{dR_h}{dt} \geq -(\rho + \mu)R_h$ …… (11)

Integrate equation (11) using separation of variable and the analytic solution obtained as:

$\int \frac{dR_h}{R_h} \geq \int -(\rho + \mu)dt, \Rightarrow \ln R_h \geq -(\rho + \mu)t + c_4$, where c_4 is constant of integration.

$\Rightarrow R_h \geq exp^{-(\rho+\mu)t+c4} \Rightarrow R_h \geq exp^{c4} exp^{-(\rho+\mu)t}$,

From initial conditions $R_h(0) = R_{h0}$, then we have $\exp^{c4} = R_{h0}$, and this gives, $c_4 = \ln R_{h0}$.

Thus, $\Rightarrow$, $R_h(t) \geq R_h(0)exp^{-(q+\mu)t}$.Where $R_h(0)$ is constant and it represents initial population of recovered state variable and it is positive. Now as $t \to \infty$, the solution of $R_h \geq 0$. Therefore, the recovered state variable$R_h(t)$ is always positive for all $t \geq 0$.

As a result, all of the state variables S_h(t), I_h(t) ,R_h (t) and B_v(t)are positive for all values of t and are also mathematically and biologicall y relevant.

4.3.2 **Boundedness of solutions of model**

In order to make the model equations are biologically valid, we must show that solutions of the system (or all the state variables) of model equations (4) - (7) are bounded for all values of independent time t. In order to show the boundedness of all the state variables it is enough to show that the total human population $N_h(t)$ and bacteria population$B_v(t)$ are bounded, where $N_h(t)=S_h(t) + I_h(t) + R_h(t)$. All the parameters used on the model are positive for all values of t. The following theorem generalizes this fact, and it is demonstrated as follows:

Theorem3.2 (boundedness)

With a positive initial condition, for every $t \geq 0$.
$(S_h(0) > 0 \ , I_h(0) \geq 0, R_h(0) \geq 0, \ B_v(0) \geq 0)$,The region contains all of the solutions to the model system equations (4) to (7).

$$\Omega\left\{(S_h, I_h, R_h, B_v) \in R_+^4 \ : \ N_h = \ S_h + R_h + \ I_h \leq \left(\frac{\Lambda}{\mu}\right), \qquad B_v \leq \frac{(1-B_e)\varepsilon\Lambda}{(\omega+\mu_1)\mu}\right\}.$$

Proof:To clarify the model equation's solution (4) - (7) of each compartment is bounded it is enough to show that the total population size of the human population$N_h(t)$ and the concentration of bacteria population$B_v(t)$ are bounded for all $t \geq 0$.

For human population: Let (S_h, R_h, I_h) be solution of system of equation (4) - (7) with non-negative initial conditions $(S_h(0), I_h(0), R_h(0))$ and the total human population $N_h(t)$ of the system is defined by$N_h = (\ S_h + R_h + \ I_h)$. The model equation's solution represents the derivative of N_h (t) with respect to time t.

(4) - (7) is defined as: $\frac{dN_h}{dt} = \frac{d}{dt}(S_h + R_h + \ I_h) = \frac{dS_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt}$

$=\Lambda - \mu(S_h + R_h + I_h)$

This can be written as, $\frac{dN_h}{dt} \leq \Lambda - \mu N_h$.

$$\Rightarrow \frac{dN_h}{dt} + \mu N_h \leq \Lambda.$$

This is first order non-homogenous linear ordinary differential inequality. Next, we apply the integrating factor approach to solve this, and as a result

$N_h \leq exp^{-\int \mu dt}(exp^{\int \mu dt}\Lambda dt + c)$, where c is the integration constant

$\Rightarrow N_h \leq exp^{-\mu t}\left(\frac{exp^{\mu t}\Lambda}{\mu} + c\right)$ Apply the first condition that we find

$N_h \leq \frac{\Lambda}{\mu}(1\text{-}exp^{-\mu t}) + N_h(0)exp^{-\mu t}$,, therefore, $N_h(t) \leq \frac{\Lambda}{\mu}$, as $t \to \infty$.

Therefore, as $t \to \infty N_h(t) \to \frac{\Lambda}{\mu}$. Hence, from the above two cases, all the solutions of the system of equation (4) - (7) that are confined in the set The solution of the entire human population is N_h bounded and $\Omega\_h = \{(, (S_h, R_h I_h,) \in R_+^{\,3}; 0 < N_h \leq \frac{\Lambda}{\mu}\}$

For concentration of vibrio cholera: if we take the differential equation (6) from the system of equation (4) - (7) and we have $\frac{dB_v}{dt} = (1 - B_e)\varepsilon I_h - (\omega + \mu_1 - \varphi)B_v$. From the human population we know that $I_h \leq N_h \leq \frac{\Lambda}{\mu}$. Using this fact, $\frac{dB_v}{dt} = (1 - B_e)\varepsilon I_h - (\omega + \mu_1 - \varphi)B_v$. , $\frac{dB_v}{dt} + (\omega + \mu_1)B_v = (1 - B_e)\varepsilon I_h + \varphi B_v \leq (1 - B_e)\varepsilon I_h$ in another form,

$\frac{dB_v}{dt} + (\omega + \mu_1)B_v \leq \frac{(1-B_e)\varepsilon I_h}{\mu}$ and this is first order non-homogenous ordinary differentia Equation. Then the solution of this inequality is obtained by using method of integrating factor as follow

$B_v \leq exp^{-\int(\omega+\mu_1)dt}\left(\int exp^{\int(\omega+\mu_1)dt}\frac{(1-B_e)\varepsilon\Lambda dt}{\mu} + c\right)$, where c is the integration constant.

$B_v \leq exp^{-(\omega+\mu_1)t}(\frac{(1-B_e)\varepsilon\Lambda exp^{(\omega+\mu_1)t}}{\mu} + c)$ Applying the initial condition,

$B_v(0) = B_{v0}, B_{v(t)} \leq \frac{(1-B_e)\varepsilon\Lambda}{(\omega+\mu_1)\mu}(1\text{-}exp^{-(\omega+\mu_1)t} + B_{v0}exp^{-(\omega+\mu_1)t})$

Therefore $B_{v(t)} \leq \frac{(1-B_e)\varepsilon\Lambda}{(\omega+\mu_1)\mu}$, as $t \to \infty$ and hence the solution of the concentration of vibrio

Cholera population is confined in the region $\Omega_B = \left\{B_v \in R_+ : 0 \leq B_{v(t)} \leq \frac{(1-B_e)\varepsilon\Lambda}{(\omega+\mu_1)\mu}\right\}$. Further

More the state variable B_V is also bounded for all $t \geq 0$.

Thus, the feasible region for the model equation (4) - (7) is given by $\Omega = \Omega_B\Omega_h$. Hence, $\Omega =$

$\left\{(S_h, I_h, R_h, B_v) \in R_+^4 \ : \ S_h, R_h, I_h, B_v \geq 0 \ , \ 0 \leq N_h \leq \left(\frac{\Lambda}{\mu}\right) \ , 0 \leq B_v \leq \frac{(1-B_e)\varepsilon\Lambda}{(\omega+\mu_1)\mu} \ , \ \right\}$, which is positive

Invariant set and since the model is first order and continuous at $(S_h(0), 0,0,0)$ then the system is biologically feasible or valid and mathematically well posed in the region .Thus all the solutions of the state variables are bounded.

CHAPTER FIVE

5 QUALITATIVE ANALYSIS OF THE MODEL

In this chapter, we determine the equilibrium points, the basic reproduction number, and conduct a stability study of equilibrium points.

5.1 The Equilibrium points:

An equilibrium point is a point where the whole model equations are equal to zero. System at equilibrium point does not change at a time. This is a

Point that makes $\frac{dS_h}{dt} = 0$, $\frac{dI_h}{dt} = 0$ $\frac{dB_v}{dt} = 0$ and $\frac{dR_h}{dt} = 0$ in another form, if$(S_h^* , I_h^* , B_h^* , R_h^*)$

Is an equilibrium point of the model system (4)-(7), then it satisfies the equation

$\Lambda - (1 - \beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \boldsymbol{\psi I_h^*}\right)S_h^* - \mu S_h^* + \rho R_h^* = 0$ ..... (12)

$(1 - \beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \boldsymbol{\psi I_h^*}\right)S_h^* - (\sigma+\theta+\Upsilon+\mu-\delta)I_h^* = 0$ .....(13)

$(1-\beta_e)\,\varepsilon I_h^* - (\omega + \mu_1 - \varphi)B_v^* = 0$ ..... (14)

$(\sigma+\theta)I_h^* - (\rho + \mu)R_h^* = 0$ .....(15)

There are two equilibrium points: disease-free E_0 and endemic E∗.

5.1.1 Disease free equilibrium point E_0

A disease-free equilibrium point is one in which the disease does not exist in the population. The infective compartments are zero. Let E_0be the point of equilibrium free from disease.

$$I_h = R_h = B_v = 0$$

$$\text{Let } \frac{dS_h}{dt} = \Lambda - (1 - \beta_e)\left(\frac{\alpha B_v}{K+B_v} + \psi I_h\right)S_h - \mu S_h + \rho R_h$$

$= \Lambda - (1 - \beta_e)\left(\frac{\alpha.0}{K+0} + \psi.0\right)S_h - \mu S_h + \rho.$

$= \Lambda - \mu S_h$

$E_0 = \frac{\Lambda}{\mu}$

Thus, the disease free equilibrium point $E_0 = (S_0, I_0, R_0, B_0)$

$= (\frac{\Lambda}{\mu}, 0, 0, 0)$

5.2 Basic reproduction number (R0)

The basic reproduction number is symbolized by R_0, is the average number of secondary infections caused when a single infectious individual is introduced into a purely susceptible population [8, 9, 30]. If $R_0 < 1$, then on average an infected individual produces less than one new infected individual over the course of infectious period, and hence the disease cannot invade the population means that the infection cannot grow. If $R_0 > 1$, then each infected individual produce more than one new infection, and hence the disease can invade the population. We determine the reproduction number R_0, using the next generation matrix approach [29]. The basic reproduction number is the spectral radius given by $R_0 = \rho (FV^{-1})$ and the matrix is given by:

$$FV^{-1} = \left[\frac{\partial F_i(E_0)}{\partial x_j}\right]\left[\frac{\partial V_i(E_0)}{\partial x_j}\right]^{-1}$$

The rate at which new infections arise in the compartment of i is denoted by f_i.
AndThe movement of people into and out of compartment i is denoted byv_i. And E_0 is the disease free equilibrium point.

Consequently, using next generation matrix method and taking the infected compartments$I_h$and $B_v$from the model equation (4)-(7) we have:

$$\frac{dI_h}{dt} = (1-\beta_e)\left(\frac{\alpha B_v}{k+B_v} + \psi I_h\right)S_h - (\sigma+\theta+\gamma+\mu-\delta)I_h$$

$$\frac{dB_v}{dt} = (1-\beta_e)\,\varepsilon I_h - (\omega + \mu_1 - \varphi)B_v$$

Thus, we can define $f_i$and $v_i$as follow:

$$F_i = \begin{pmatrix} f_1 \\ f_2 \end{pmatrix} = \begin{pmatrix} (1-\beta_e)\left(\frac{\alpha B_v}{k+B_v} + \psi I_h\right)S_h \\ 0 \end{pmatrix}$$

$$V_i = \begin{pmatrix} v_1 \\ v_2 \end{pmatrix} = \begin{pmatrix} (\sigma+\theta+\gamma+\mu-\delta)I_h \\ (\omega+\mu_1-\varphi)B_v - (1-\beta_e)\,\varepsilon I_h \end{pmatrix}$$

To get the matrices F and V, we find the partial derivative of $F_i$and V_i with respect to I_h and $B_v$as follow:
$\frac{\partial f_1}{\partial I_h} = (1-\beta_e)\psi S_h$, $\frac{\partial f_1}{\partial B_v} = \frac{(1-\beta_e)\alpha S_h K}{(k+B_v)^2}$, $\frac{\partial f_2}{\partial I_h} = 0$, $\frac{\partial f_2}{\partial B_v} = 0$ and

$\frac{\partial v_1}{\partial I_h} = \sigma+\theta+\gamma+\mu-\delta$, $\frac{\partial v_1}{\partial B_v} = 0$, $\frac{\partial v_2}{\partial I_h} = -(1-\beta_e)\,\varepsilon$, $\frac{\partial v_2}{\partial B_v} = \omega+\mu_1-\varphi$

Hence $F = \begin{pmatrix} \frac{\partial f_1}{\partial I_h} & \frac{\partial f_1}{\partial B_v} \\ \frac{\partial f_2}{\partial I_h} & \frac{\partial f_2}{\partial B_v} \end{pmatrix} = \begin{pmatrix} (1-\beta_e)\psi S_h & \frac{(1-\beta_e)\alpha S_h K}{(k+B_v)^2} \\ 0 & 0 \end{pmatrix}$

$V = \begin{pmatrix} \frac{\partial v_1}{\partial I_h} & \frac{\partial v_1}{\partial B_v} \\ \frac{\partial v_2}{\partial I_h} & \frac{\partial v_2}{\partial B_v} \end{pmatrix} = \begin{pmatrix} \sigma+\theta+\gamma+\mu-\delta & 0 \\ -(1-\beta_e)\,\varepsilon & \omega+\mu_1-\varphi \end{pmatrix}$, Computing$FV^{-1}$as follows

$$FV^{-1} = \begin{pmatrix} (1-\beta_e)\psi S_h & \frac{(1-\beta_e)\alpha S_h K}{(k+B_v)^2} \\ 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{\sigma+\theta+\gamma+\mu-\delta} & 0 \\ \frac{(1-\beta_e)\,\varepsilon}{(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi\ )} & \frac{1}{\omega+\mu_1-\varphi} \end{pmatrix}$$

$$= \begin{pmatrix} \frac{(1-\beta_e)\psi S_h}{\sigma+\theta+\gamma+\mu-\delta} + \frac{(1-\beta_e)^2\alpha S_h K\,\varepsilon}{(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi\ )(k+B_v)^2} & \frac{(1-\beta_e)\alpha S_h K}{(\omega+\mu_1-\varphi)(k+B_v)^2} \\ 0 & 0 \end{pmatrix}$$

And

$$FV^{-1}(E_0) = \begin{pmatrix} \frac{(1-\beta_e)\psi\Lambda(\omega+\mu_1-\varphi)+(1-\beta_e)\alpha\varepsilon\Lambda}{\mu K(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi\ )} & \frac{(1-\beta_e)\alpha\Lambda}{(\omega+\mu_1-\varphi)K\mu} \\ 0 & 0 \end{pmatrix}$$

.

The eigenvalues of FV^{-1} are found by solving the characteristics equation,

$$|FV^{-1}(E_0) - \lambda I_2| = 0$$

Where $I_2$is a 2x2 square identity matrix and λ is the eigenvalue of the given matrix, then this gives as:

$$\left| \begin{matrix} \frac{(1-\beta_e)\psi\Lambda(\omega+\mu_1-\varphi)+(1-\beta_e)\alpha\,\varepsilon\Lambda}{\mu k(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi\ )} - \lambda & \frac{(1-\beta_e)\alpha\Lambda}{(\omega+\mu_1-\varphi)K\mu)} \\ 0 & -\lambda \end{matrix} \right| = 0.$$

$$-\lambda\left(\frac{(1-\beta_e)\psi\Lambda(\omega+\mu_1-\varphi)+(1-\beta_e)\alpha\,\varepsilon\Lambda}{\mu K(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi\ )} - \lambda\right) = 0$$

$$\lambda^2 - \frac{(1-\beta_e)\psi\Lambda(\omega+\mu_1-\varphi)+(1-\beta_e)\alpha\,\varepsilon\Lambda}{\mu K(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi\ )}\lambda = 0$$

$$\lambda = 0 \ \text{ and } \ \lambda = \frac{(1-\beta_e)\psi\Lambda(\omega+\mu_1-\varphi)+(1-\beta_e)\alpha\,\varepsilon\Lambda}{\mu K(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi\ )}$$

Thus, the eigenvalues are,$\lambda_1 = 0 \ \text{ and } \ \lambda_2 = \frac{(1-\beta_e)\psi\Lambda(\omega+\mu_1-\varphi)+(1-\beta_e)\alpha\,\varepsilon\Lambda}{\mu K(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi\ )}$

Therefore $\rho F V^{-}$ is the fundamental reproduction number R0.

$$R_o = \frac{(1-\beta_e)\psi\Lambda(\omega + \mu_1 - \varphi) + (1-\beta_e)\alpha\,\varepsilon\Lambda}{\mu K(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi\ )}$$

. So $R_o = \frac{(1-\beta_e)\psi\Lambda(\omega+\mu_1-\varphi)+(1-\beta_e)\alpha\,\varepsilon\Lambda}{\mu K(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi\ )}$ and it is positive when $\sigma + \theta + \gamma + \mu > \delta$ and $w + \mu_1 > \varphi$

5.2.1 Local stability of disease free equilibrium point

The model equations (4) to (7) are linear zed at the equilibrium point E_0 to calculate the local stability of the disease-free equilibrium point. Using the following Jacoben matrix:

$f_1(S_h, I_h, B_v, R_h) = \Lambda - (1-\beta_e)(\frac{\alpha B_v}{k+B_v} + \psi I_h)S_h - \mu S_h + \rho R_h,$

$$f_2(S_h, I_h, B_v, R_h) = (1-\beta_e)(\frac{\alpha B_v}{k+B_v} + \psi I_h)S_h - (\sigma+\theta+\gamma+\mu-\delta)I_h$$

$f_3(S_h, I_h, B_v, R_h) = (1-\beta_e)\varepsilon I_h - (\omega+\mu_1-\varphi)B_v$, and

$$f_4(S_h, I_h, B_v, R_h) = (\sigma+\theta)I_h - (\rho+\mu)R_h$$

Then, differentiating functions f_1, f_2, f_3, f_4 with respect to the state variables S_h, I_h, B_v and R_h like

$\frac{\partial f_1}{\partial S_h} = -(1-\beta_e)\frac{\alpha B_v}{k+B_v} - (1-\beta_e)\psi I_h - \mu$, $\frac{\partial f_1}{\partial I_h} = -(1-\beta_e)\psi S_h,$

$\frac{\partial f_1}{\partial B_v} = -(1-\beta_e)\frac{\alpha S_h K}{(k+B_v)^2}$, $\frac{\partial f_1}{\partial R_h} = \rho$

$\frac{\partial f_2}{\partial S_h} = (1-\beta_e)\frac{\alpha B_v}{k+B_v} + (1-\beta_e)\psi I_h$, $\frac{\partial f_2}{\partial I_h} = (1-\beta_e)\psi S_h + \delta - (\sigma+\theta+\gamma+\mu)$

$\frac{\partial f_2}{\partial B_v} = (1-\beta_e)\frac{\alpha K B_v S_h}{(k+B_v)^2}$, $\frac{\partial f_2}{\partial R_h} = 0$

$\frac{\partial f_3}{\partial S_h} = 0$, $\frac{\partial f_3}{\partial I_h} = (1-\beta_e)\varepsilon$, $\frac{\partial f_3}{\partial B_v} = -(\omega+\mu_1-\varphi)$ $\frac{\partial f_3}{\partial R_h} = 0$ and

$\frac{\partial f_4}{\partial S_h} = 0$, $\frac{\partial f_4}{\partial I_h} = (\sigma+\theta)$, $\frac{\partial f_4}{\partial B_v} = 0$, $\frac{\partial f_4}{\partial R_h} = -(\rho+\mu)$

The Jacobean Matrix is given as follows.

$$J(S_h, I_h, R_h, B_v) = \begin{pmatrix} \frac{\partial f_1}{\partial S_h} & \frac{\partial f_1}{\partial I_h} & \frac{\partial f_1}{\partial B_V} & \frac{\partial f_1}{\partial R_h} \\ \frac{\partial f_2}{\partial S_h} & \frac{\partial f_2}{\partial I_h} & \frac{\partial f_2}{\partial B_V} & \frac{\partial f_2}{\partial R_h} \\ \frac{\partial f_3}{\partial S_h} & \frac{\partial f_3}{\partial I_h} & \frac{\partial f_3}{\partial B_V} & \frac{\partial f_3}{\partial R_h} \\ \frac{\partial f_4}{\partial S_h} & \frac{\partial f_4}{\partial I_h} & \frac{\partial f_4}{\partial B_V} & \frac{\partial f_4}{\partial R_h} \end{pmatrix}$$

$$= \begin{pmatrix} -(1-\beta_e)\frac{\alpha B_v}{k+B_v} - (1-\beta_e)\psi I_h - \mu & -(1-\beta_e)\psi S_h & \frac{-(1-\beta_e)\alpha S_h K}{(k+B_v)^2} & \rho \\ (1-\beta_e)\frac{\alpha B_v}{k+B_v} + (1-\beta_e)\psi I_h & (1-\beta_e)\psi S_h + \delta - (\sigma+\theta+\gamma+\mu) & (1-\beta_e)\frac{\alpha K B_v S_h}{(k+B_v)^2} & 0 \\ 0 & (1-\beta_e)\varepsilon & -(\omega+\mu_1-\varphi) & 0 \\ 0 & (\sigma+\theta) & 0 & -(\rho+\mu) \end{pmatrix}$$

Hence,

$$J(E_0) = \begin{pmatrix} -\mu & \frac{-(1-\beta_e)\psi\Lambda}{\mu} & \frac{-(1-\beta_e)\psi\Lambda}{K\mu} & \rho \\ 0 & \frac{(1-\beta_e)\psi S_h}{\mu} + \delta - (\sigma+\theta+\gamma+\mu) & \frac{-(1-\beta_e)\psi\Lambda}{K\mu} & 0 \\ 0 & (1-\beta_e)\varepsilon & -(\omega+\mu_1-\varphi) & 0 \\ 0 & (\sigma+\theta) & 0 & -(\rho+\mu) \end{pmatrix}$$

Thus, the characteristic polynomial equation of the Jacobean matrix at E_0 becomes

$|J(E_0) - \lambda I_4| = 0$, Where I_4 is a 4x4 identity matrix and λ is the eigenvalue of the matrix.

$$\begin{vmatrix} -\mu-\lambda & \frac{-(1-\beta_e)\psi\Lambda}{\mu} & -b & q \\ 0 & h-\lambda & b & 0 \\ 0 & (1-\beta_e)\varepsilon & d-\lambda & 0 \\ 0 & c & 0 & -(\rho+\mu)-\lambda \end{vmatrix} = 0$$

Where, $h = \frac{(1-\beta_e)\psi\Lambda}{\mu} + \delta - (\sigma+\theta+\gamma+\mu)$, $b = \frac{(1-\beta_e)\alpha\Lambda}{K\mu}$

$$d = -(\omega+\mu_1-\varphi), \text{ and } c = \sigma+\theta$$

$$(-\mu-\lambda)[(h-\lambda)(d-\lambda)(-(\mu+\rho)-\lambda) - ((1-\beta_e)\varepsilon b(-(\mu+\rho)-\lambda))] = 0,$$

$$\Rightarrow (-\mu-\lambda)[(h-\lambda)(d-\lambda)(-((\mu+\rho)+\lambda)) + ((1-\beta_e)\varepsilon b((\mu+\rho)+\lambda))] = 0,$$

$$\Rightarrow (-\mu-\lambda)[(-((h-\lambda))(d-\lambda)((\mu+\rho)+\lambda)) + ((1-\beta_e)\varepsilon\, b((\mu+\rho)+\lambda))] = 0$$

take $((\mu+\rho)+\lambda)$, as common we have,

$$\Rightarrow (\mu+\lambda)[((\mu+\rho)+\lambda)((h-\lambda)(d-\lambda) - (1-\beta_e)\varepsilon b)] = 0,$$

$$\Rightarrow (\mu+\lambda)((\mu+\rho)+\lambda)((h-\lambda)(d-\lambda) - (1-\beta_e)\varepsilon b) = 0,$$

Clearly $\lambda_1 = -\mu < 0$, $\lambda_2 = -(\mu+\rho) < 0$ and the other eigenvalue can be calculated as follow:

$$(h-\lambda)(d-\lambda) - (1-\beta_b)\varepsilon b = 0$$

$$hd - h\lambda - d\lambda + \lambda^2 - (1-\beta_e)\varepsilon b = 0,$$

$$\lambda^2 - h\lambda - hd - (1-\beta_e)\varepsilon b = 0$$

$$\lambda^2 - (h+d)\lambda + hd - (1-\beta_e)\varepsilon b = 0 \quad \text{..... (16)}$$

The block of trace matrix is represented by h + d, $\begin{pmatrix} h-\lambda & b \\ (1-\beta_e)\varepsilon & d \end{pmatrix}$

And $hd - (1 - \beta_e)\varepsilon b$ is its determinant. Thus, when $h + d < 0$ and $hd - (1 - \beta_e)\varepsilon b > 0$, the disease free equilibrium point E_0 is locally asymptotically stable.

Thus, $h + d = (1 - \beta_e)\frac{\varphi\Lambda}{\mu} - (\sigma + \theta + \gamma + \mu - \delta) - (\omega + \mu_1 - \varphi)$, if we take $\sigma + \theta + \gamma + \mu - \delta$ to express in another expression ,

i.e., $h + d = (1 - \beta_e)\frac{\varphi\Lambda}{\mu} - \left[(1 - \beta_e)\frac{\Lambda}{\mu R_0}\left(\omega + \frac{(1-\beta_e)\alpha\varepsilon}{K(\omega+\mu_1-\varphi)}\right)\right] - (\omega + \mu_1 - \varphi)$

If the following criteria are met, this is negative:

$$R_0 < 1,$$
$$(\omega + \mu_1 - \varphi) > 0$$

Therefore, $tr(J(E_0)) < 0$. And again, $detJ(E_o) > 0$, this implies $hd - (1 - \beta_b)\varepsilon b > 0$.

$$hd - (1 - \beta_b)\varepsilon b > 0,$$

$$\Rightarrow -(1 - \beta_e)\frac{\varphi\Lambda}{\mu}(\omega + \mu_1 - \varphi) + (\sigma + \theta + \gamma + \mu - \delta)(\omega + \mu_1 - \varphi) - (1 - \beta_e)\frac{\varphi\Lambda}{\mu} > 0$$

$$\Rightarrow (1 - \beta_e)\frac{\varphi\Lambda}{\mu}(\omega + \mu_1 - \varphi) + (1 - \beta_e)\frac{\varepsilon\alpha\Lambda}{\mu K} < (\sigma + \theta + \gamma + \mu - \delta)(\omega + \mu_1 - \varphi) \ldots\ldots\ldots\ldots\ldots\ldots(17)$$

Divide equation (17) to both sides by $(\sigma + \theta + \gamma + \mu - \delta)(\omega + \mu_1 - \varphi)$

Gives $\frac{(1-\beta_e)\Lambda(\varphi K(\omega+\mu_1-\varphi)+(1-\beta_e)\varepsilon\alpha)}{(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi)\mu K} < 1$ and $R_o = \frac{(1-\beta_e)\Lambda(\varphi K(\omega+\mu_1-\varphi)+(1-\beta_e)\varepsilon\alpha)}{(\sigma+\theta+\gamma+\mu-\delta)(\omega+\mu_1-\varphi)\mu K}$

From this we arrive at $R_o < 1$. Therefore, we have the following theorem.

Theorem3.3: The disease free equilibrium point $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$ is locally asymptotically stable if $R_o < 1$ and $\omega + \mu_1 - \varphi > 0$.

5.2.2 Global stability of disease free equilibrium point

In this section, we examine the global stability of the disease free equilibrium point. In order to show the global stability of disease free equilibrium point, we use the following Castillo-chavez comparison theorem [29]. Thus, the global asymptotic stability of the disease free equilibrium point of the mathematical model equation (4) - (7) is determined by using Castillo-Chavez et al. [29]. Next, the model can be expressed as follows:

Let $\frac{dX}{dt} = P(X, I)\frac{dI}{dt} = G(X, I), G(X, 0) = 0$

Where $X = S_h$ and $I = (I_h, B_v)$.

Here, the compartment of $X \in R$ denotes the susceptible individual and the component of $I \in R^2$, denotes the infected individual and the disease free equilibrium point $E_0 = (X, 0), X = \frac{\Lambda}{\mu}$. In order to ensure global asymptotic stability, the following two requirements need to be satisfied.

(H_1), For $\frac{dX}{dt} = P(X, 0), E_0$ is globally asymptotically stable.

$(H_2), G(X, I) = AI - G(X, I), G(X, I) \geq 0.$

For $(X, I) \in \Omega$ and $A = D_1 G(X, 0)$ is a Metzler-matrix (or the off diagonal elements of A are non-negative), and Ω is the region where the model makes biological sense or meaningful.

The Castillo-Chavez et al. comparison theorem's requirements are fulfilled.

Theorem3.4: The disease free equilibrium point$E_0 = \left(\frac{\Lambda}{\mu}, 0,0,0\right)$ is globally stable if$R_0 < 1$, and

Proof: By applying the Castillo-Chavez et al. comparison theorem[29]., the differential equation of the state variables representing the infected compartments of the model equations (4) - (7) can be expressed as in the form:$\frac{dX}{dt} = P(X, I)$

$\frac{dI}{dt} = G(X, I), G(X, 0) = 0$, where $X = S_h$ and $I = (I_h, B_v)$.

Here, the compartment of $X \in R$ denotes the susceptible individual and the component of

$I \in R$, denotes the infected individual and the disease free equilibrium point $E_0 = (X, 0), X = \frac{\Lambda}{\mu}$

Then to guarantee globally asymptotically stable, the following two conditions must be satisfied.

(H_1), For $\frac{dX}{dt} = P(X, 0), E_0$ is globally asymptotically stable.

$(H_2), G(X, I) = [F - V]\begin{pmatrix} I_h \\ B_v \end{pmatrix} - \widehat{G}(X, I), \widehat{G}(X, I) \geq 0$ (The component of X and I are positive).

Where, $A = [F - V]$ and $I = \begin{pmatrix} I_h \\ B_v \end{pmatrix}$

For $(X, I) \in \Omega$ and $A = D_1 G(X, 0)$ is a Metzler-matrix (or the off diagonal elements of A are non-negative), and Ω is the region where the model makes biological sense or meaningful. Thus$\frac{dX}{dt} = P(X, 0)$ which implies$\frac{dS_h}{dt} = \Lambda - \mu S_h$, this is first order linear ordinarydifferential equation and has a solution:

$S_h(t) = \frac{\Lambda}{\mu}(1 - \exp^{\mu t} + S_h(0)\exp^{-\mu t})$Where$S_h(0)$ is the initial condition. the solution $S_h(t) \rightarrow \frac{\Lambda}{\mu}$ as $t \rightarrow \infty$and since E_0 is locally asymptotically stable, this follows that it is globally asymptotically stable for$\frac{dX}{dt} = P(X, 0)$. Thus the first condition (H_1) is satisfied.

Next, after computing $[F - V]$ and substitute in to the second condition, then we perform:

$$G(X, I) = [F - V]\begin{pmatrix} I_h \\ B_v \end{pmatrix} - \widehat{G}(X, I)$$

$$\Rightarrow \begin{pmatrix} \frac{dI_h}{dt} \\ \frac{dB_v}{dt} \end{pmatrix} = [F - V]\begin{pmatrix} I_h \\ B_v \end{pmatrix} - \widehat{G}(X, I)$$

$$\Rightarrow \begin{pmatrix} \left(\left(1 - \beta_e\right)\frac{\alpha B_v}{K + B_v} + \varphi I_h\right)S_h - (\sigma + \theta + \gamma + \mu - \delta)I_h \\ \left(1 - \beta_e\right)\varepsilon I_h - \left(\omega + \mu_1 - \varphi\right)B_v \end{pmatrix}$$

$$= \begin{pmatrix} (1-\beta_e)\varphi S_h & (1-\beta_e)\frac{\alpha S_h}{K} \\ (1-\beta_e)\varepsilon & -(w+\mu_1-\varphi) \end{pmatrix}\begin{pmatrix} I_h \\ B_v \end{pmatrix} - \begin{pmatrix} (1-\beta_e)\frac{\alpha S_h {B_v}^2}{K(K+B_v)} \\ 0 \end{pmatrix}$$

.

Since $0 < S_h \leq N_h = \frac{\Lambda}{\mu}$ when $t \in [0,\infty)$ and it is clear that

$$\widehat{G}(X,I) = \begin{pmatrix} (1-\beta_e)\frac{\alpha S_h {B_v}^2}{K(K+B_v)} \\ 0 \end{pmatrix}$$

Here the components of the matrix $G(X,I)$ are positive, and then the second condition(H_2) is satisfied. And this follows that the equilibrium point E_0 is globally asymptotically stable.

5.2.3 Existence of endemic equilibrium point

An endemic equilibrium point is a stable state in which the cholera epidemic is prevalent among the individuals. This means at the endemic equilibrium point there are infective individuals. $E^* = (S_h^*, I_h^*, R_h^*, B_v^*)$, its coordinates will satisfy the conditions ;$E^* = (S_h^*, I_h^*, R_h^*, B_v^*) \neq 0$, where$S_h^* > 0$, $I_h^* > 0, R_h^* > 0, B_v^* > 0$. The endemic equilibrium point$E^*$is calculated by setting the right side of model equation (4) - (7) to zero and calculating state variables i.e.$\frac{dS_h}{dt} = 0, \frac{dI_h}{dt} = 0, \frac{dR_h}{dt} = 0, \frac{dB_v}{dt} = 0$, thus we have,

$$\begin{cases} \Lambda - (1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v} + \psi I_h^*\right) S_h^* - \mu S_h^* + \rho R_h^* \\ (1-B_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right) S_h^* - (\sigma+\theta+\acute{Y}+\mu-\delta) I_h^* \\ (1-B_e)\varepsilon I_h^* - (w+\mu_1-\varphi) B_v^* \\ (\sigma+\theta) I_h^* - (\rho+\mu) R_h^* \end{cases} \quad \text{.............................. (18)}$$

Based the third equation of the system equation (18), gives,$(1-B_e)\varepsilon I_h^* = (w+\mu_1-\varphi) B_v^*$.

$(1-B_e)\varepsilon I_h^* = (w+\mu_1-\varphi) B_v^*$,

$$\Rightarrow B_v^* = \frac{(1-B_e)\varepsilon I_h^*}{(w+\mu_1-\varphi)} \quad \text{... (19)}$$

Based the second equation of the system equation (18), gives

$$(1-B_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right) S_h^* = (\sigma+\theta+\acute{Y}+\mu-\delta) I_h^*. \text{................................. (20)}$$

Based the first equation of the system equation (18), gives

$$\Lambda - (1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v} + \psi I_h^*\right) S_h^* - \mu S_h^* + \rho R_h^* = 0 \text{.................................. (21)}$$

Based the fourth equation of the system equation (18), gives

$$R_h^* = \frac{(\sigma+\theta) I_h^*}{\rho+\mu} \text{... (22)}$$

Substitute (19) into (20), gives

$$B_v^* = \frac{(1-B_e)\varepsilon I_h^*}{(w+\mu_1-\varphi)}$$

$$(1-B_e)\left(\frac{\alpha B_v^*}{K+B_v^*}+\psi I_h^*\right)S_h^* = (\sigma+\theta+\acute{Y}+\mu-\delta)I_h^*.$$

$$\Lambda-(1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*}+\psi I_h^*\right)\frac{K+B_v^*}{\alpha B_v^*+\psi I_h^*(K+B_v^*)}(\sigma+\theta+\acute{Y}+\mu-\delta)I_h^* - \frac{\mu(K+B_v^*)}{(1-\beta_e)\alpha B_v^*+(1-\beta_e)\psi I_h^*(K+B_v^*)}(\sigma+\theta+\acute{Y}+\mu-\delta)I_h^*+\rho R_h^*=0$$

After rearrangement it becomes

$$\Rightarrow \Lambda-(\sigma+\theta+\acute{Y}+\mu-\delta)I_h^*\left(1-\frac{\mu(K+B_v^*)}{(1-\beta_e)\alpha B_v^*+(1-\beta_e)\psi I_h^*(K+B_v^*)}\right)+\rho R_h^*=0 \;\ldots\ldots\ldots\ldots\ldots\ldots\; (23)$$

Substitute equations (18) and (21) into equation (22) we get,

$$\Lambda-hI_h^*\left(\frac{(1-\beta_e)\alpha\varepsilon I_h^*+(1-\beta_e)\psi K d I_h^*+(1-\beta_e)^2\psi\varepsilon I_h^{*2}+\mu K d-(1-\beta_e)\mu\varepsilon I_h^*}{(1-\beta_e)^2\varepsilon\alpha I_h^*+(1-\beta_e)\psi\, K d I_h^*+(1-\beta_e)^2\psi\varepsilon I_h^{*2}}\right)+\frac{\rho c}{m}I_h^*=0$$

$$\Rightarrow \Lambda(1-\beta_e)^2\alpha\varepsilon I_h^*+\Lambda(1-\beta_e)\psi\, K d I_h^*+\Lambda(1-\beta_e)^2\psi\varepsilon I_h^{*2}-(1-\beta_e)\alpha\varepsilon h I_h^{*2} - (1-\beta_e)\psi\, K d h I_h^{*2}-(1-\beta_e)^2\psi\varepsilon h I_h^{*3}-\mu K d h I_h^*+(1-\beta_e)\mu\varepsilon h I_h^{*2} + \frac{\rho c}{m}(1-\beta_e)^2\alpha\varepsilon I_h^{*2}+\frac{\rho c}{m}(1-\beta_e)\psi\, K d I_h^{*2}+\frac{\rho c}{m}(1-\beta_e)^2\psi e\varepsilon I_h^{*3}=0$$

From this we have, $\Rightarrow I_h^*\left(\left(\frac{\rho c}{m}(1-\beta_e)^2\psi\varepsilon-(1-\beta_e)^2\psi\varepsilon h\right)I_h^{*2}+\Lambda\left((1-\beta_e)^2\psi\varepsilon+\frac{\rho c}{m}(1-\beta_e)^2\alpha\varepsilon+\frac{\rho c}{m}(1-\beta_e)\psi\, K d-(1-\beta_e)\alpha\varepsilon h-(1-\beta_e)\psi\, K d h\right)I_h^*+\left(\Lambda(1-\beta_e)^2\alpha\varepsilon+\Lambda(1-\beta_e)\psi\, K d-\mu K d h\right)\right)=0$

Where, $h=\sigma+\theta+\gamma+\mu-\delta, d=w+\mu_1-\varphi, c=\sigma+\theta, m=\rho+\mu$

And this implies that, $I_h^*=0$, or $PI_h^{*2}+QI_h^*+T=0$ ……………………………………………(24)

Where,

$$P=\frac{\rho c}{m}-h(1-\beta_e)^2\psi\varepsilon,$$

$$Q=\Lambda(1-\beta_e)^2\psi\varepsilon+\frac{\rho c}{m}(1-\beta_e)^2\alpha\varepsilon+\frac{\rho c}{m}(1-\beta_e)\psi\, K d-(1-\beta_e)\alpha\varepsilon h-(1-\beta_e)\psi\, K d h,$$

$$T=\Lambda(1-\beta_e)^2\alpha\varepsilon+\Lambda(1-\beta_e)\psi\, K d-\mu K d h = R_0-1$$

Assume $h=\sigma+\theta+\gamma+\mu-\delta>0, d=w+\mu_1-\varphi>0$

We use the Descartes rule of signs to determine the solution of equation (24). By dropping $I_h^* = 0$ or the zero solution, the positive endemic equilibrium point of the system equation (4) - (7) exists if the roots of equation (24) are real and positive. Whatever the sign of P, Q ispositive if

$\Lambda(1-\beta_e)^2\psi\varepsilon + \frac{\rho c}{m}(1-\beta_e)^2\alpha\varepsilon + \frac{\rho c}{m}(1-\beta_e)\psi Kd > (1-\beta_e)\alpha\varepsilon h + (1-\beta_e)\psi Kdh$ AndT is positive if $R_0 > 1$. To satisfy the Descartes rule to determine the sign of the coefficient of equation (24), there exists at least one positive real root for$I_h^*$and the other equilibrium point's S_h^*, B_v^* and $R_h^*$are determined and hence this justifies the endemic equilibrium point $E^* = (S_h^*, I_h^*, B_v^*, R_h^*)$ exists.

Where,$S_h^* = \frac{1}{\frac{\mu}{\Lambda}R_0 + (1-\beta_e)\varepsilon I_h^*} + \frac{\varepsilon I_h^*}{K\psi(w+\mu_1-\varphi)+(1-\beta_e)\alpha\varepsilon+(1-\beta_e)\varepsilon \quad eI_h^*}$, $B_v^* = \frac{(1-B_e)\varepsilon I_h^*}{(w+\mu_1-\varphi)}$ and $R_h^* = \frac{(\sigma+\theta)I_h^*}{\rho+\mu}$

5.2.4 Local stability of endemic equilibrium point

The local stability of endemic equilibrium point$E^* = (S_h^*, I_h^*, B_v^*, R_h^*)$ where$S_h^* > 0$, $I_h^* > 0$, $B_v^* > 0$ *and* $R_h^* > 0$is determined by linearizing the model equation (4) - (7) at the equilibrium pointE^*. This is done by using the Jacobian matrix of the system, the Jacobian matrix is determined by differentiating the model equation (4) - (7) with respect to every state variable. Thus to analyze the stability analysis of endemic equilibrium point E^*we use the following theorem [5 , 13,14]

Theorem 3.3: Endemic equilibrium point $E^* = (S_h^*, I_h^*, B_v^*, R_h^*)$ of the model equation (4) - (7) Is locally asymptotically stable if $R_0 > 1$ and unstable otherwise.

Proof: To determine the local stability analysis of the endemic equilibrium point, we determine the Jacobian matrix J of equations (4) - (7) at the equilibrium point E^*. Thus, the

Jacobian matrix at$E^* = (S_h^*, I_h^*, B_v^*, R_h^*)$given above becomes:

Where, $J(E^*) = \begin{pmatrix} -u-\mu & -(1-\beta_e)\psi S_h^* & -v & \rho \\ u & -h & v & 0 \\ 0 & (1-\beta_e)\varepsilon & -d & 0 \\ 0 & c & 0 & -m \end{pmatrix}$

$u = (1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right)$, $v = \frac{(1-\beta_e)\alpha S_h^* K}{(K+B_v^*)^2}$, $h = \sigma + \theta + \gamma + \mu - \delta$, $c = \sigma + \theta$, $d = w + \mu 1 - \varphi$, $m = \mu + \rho$. The characteristic polynomial equation is given by $\det(J - \lambda I_4) = 0$, where I_4 is a 4x4 identity matrix and λ is the eigenvalue of the matrix J.

Thus, $\det(J - \lambda I_4) = 0$,

$$\det(J-\lambda I_4) = \begin{vmatrix} -u-\mu-\lambda & -(1-\beta_e)\psi S_h^* & -v & \rho \\ u & -h-\lambda & v & 0 \\ 0 & (1-\beta_e)\varepsilon & -d-\lambda & 0 \\ 0 & c & 0 & -m-\lambda \end{vmatrix} = 0$$

$$\Rightarrow -\big((u+\mu)-\lambda\big)\begin{vmatrix} -h-\lambda & v & o \\ (1-\beta_e)\varepsilon & -d-\lambda & 0 \\ c & 0 & -\lambda \end{vmatrix} + v\begin{vmatrix} -(1-\beta_e)\psi S_h^* & -v & \rho \\ (1-\beta_e)\varepsilon & -d-\lambda & 0 \\ c & 0 & -m-\lambda \end{vmatrix} = 0$$

$$\Rightarrow -\big((u+\mu)+\lambda\big)\Big[-(h+\lambda)(d+\lambda)(m+\lambda) - \Big(-(1-\beta_e)\varepsilon\, v(m+\lambda)\Big)\Big] - v\big[-(-c\,\rho(d+\lambda) + (1-\beta_e)\varepsilon(m+\lambda)\big] = 0$$

After simplification we have:

$$\lambda^4 + ((\mu+u) + hm + dm + hd + m)\lambda^3 + ((\mu+u)h + (\mu+u)d + (\mu+u)mhd + md)\lambda^2 + ((\mu + u)hd + (\mu + u)mh + (\mu + u)md + mhd - (1 - \beta_e)\varepsilon uc\rho + (1 - \beta_e)\varepsilon u)\lambda + ((\mu + u)mhd - (1 - \beta_e)m\varepsilon v - uc\rho d + (1 - \beta_e)m\varepsilon uv) = 0$$

Thus, using Routh Hurwitz criteria for stability [9, 15], polynomial of degree four has the form

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0$$

where, $a_1 = (\mu+u) + hm + dm + hd + m$, $a_2 = (\mu+u)h + (\mu+u)d + (\mu+u)mhd + md$,

$$a_3 = (\mu + u)hd + (\mu + u)mh + (\mu + u)md + mhd - (1 - \beta_e)\varepsilon uc\,\rho + (1 - \beta_e)\varepsilon u),$$

$$a_4 = (\mu + u)mhd - (1 - \beta_e)m\varepsilon v - uc\,\rho d + (1 - \beta_e)m\varepsilon\, uv$$

Therefore, by Routh Hurwitz criteria the roots of the characteristic polynomial has negative real part if $a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0$ and $a_1a_2a_3 > {a_3}^2 + {a_2}^2a_4$ and this condition holds true if the following conditions are satisfied.

$$a_1 > 0 \Rightarrow (\mu+u) + hm + dm + hd + m > 0$$

$$a_3 > 0 \Rightarrow (\mu + u)(hd + mh + md) + mhd > \big(1 - \beta_e\big)\varepsilon + uc\rho - (1 - \beta_e)\varepsilon u)$$

$$a_4 > 0 \Rightarrow (\mu + u)mhd - \big(1 - \beta_e\big)m\varepsilon v > uc\rho d - (1 - \beta_e)m\varepsilon uv$$

$a_1a_2a_3 > {a_3}^2 + {a_2}^2a_4$

$$\Rightarrow ((\mu + u) + hm + dm + hd + m)((\mu + u)h + (\mu + u)d + (\mu + u)m + hd + md)((\mu + u)hd + (\mu + u)mh + (\mu + u)md + mhd - (1 - \beta_e)\varepsilon - uc\,\rho + (1 - \beta_e)\varepsilon\, u)$$

$$> ((\mu+u)hd + (\mu+u)mh + (\mu+u)md + mhd - (1-\beta_e)\varepsilon - uc\rho + (1-\beta_e)\varepsilon u)^2 + ((\mu+u)h + (\mu + u)d + (\mu + u)m + hd + md)^2((\mu + u)mhd - (1 - \beta_e)m\varepsilon\, v - uc\rho d + (1 - \beta_e)m\varepsilon\, uv).$$

Therefore, if the above four conditions are satisfied, then the endemic equilibrium point $E^* = (S_h^*, I_h^*, B_v^*, R_h^*)$ is locally asymptotically stable.

5.2.5 Global stability of endemic equilibrium point

Theorem: For $R_0 > 1$, the endemic equilibrium point is globally asymptotically stable. **Proof**: Using the constructed lyapunov function by (Cai, L. and Li, Z., 2010), the global stability of the endemic equilibrium is proved. By defining the lyapunov function as follows:

$$V = \left(S_h - S_h^* - S_h^* \ln\frac{S_h^*}{S_h}\right) + \left(I_h - I_h^* - I_h^* \ln\frac{I_h^*}{I_h}\right) + \left(B_v - B_v^* - B_v^* \ln\frac{B_v^*}{B_v}\right) + \left(R_h - R_h^* - R_h^* \ln\frac{R_h^*}{R_h}\right)$$

By direct calculation the derivative of V along the solution of the system equation (4) - (7) with respect to time we have:

$$\frac{dV}{dt} = \left(\frac{S_h - S_h^*}{S_h}\right)\frac{dS_h}{dt} + \left(\frac{I_h - I_h^*}{I_h}\right)\frac{dI_h}{dt} + \left(\frac{B_v - B_v^*}{B_v}\right)\frac{dB_v}{dt} + \left(\frac{R_h - R_h^*}{R_h}\right)\frac{dR_h}{dt}$$

$$\frac{dV}{dt} = \left(\frac{S_h - S_h^*}{S_h}\right)\left(\Lambda - (1-\beta_e)\left(\frac{\alpha B_v^*}{K + B_v^*} + \psi I_h\right)S_h - \mu S_h + \rho R_h\right)$$

$$+\left(\frac{I_h - I_h^*}{I_h}\right)\left((1-\beta_e)\left(\frac{\alpha B_v}{K+B_v} + \psi I_h\right)S_h - (\sigma + \theta + \gamma + \mu - \delta)I_h\right) + \left(\frac{B_v - B_v^*}{B_v}\right)\left((1-\beta_e)I_h - (\omega + \mu 1 - \varphi)B_v\right) + \left(\frac{R_h - R_h^*}{R_h}\right)\left((\sigma + \theta)I_h - (\rho - \mu)R_h\right) \quad \ldots\ldots (25)$$

But, the model equation (4) - (7) satisfies the following relation at the non-trivial equilibrium Point $E^* = (S_h^*, I_h^*, B_v^*, R_h^*)$.i.e.

$$\Lambda = (1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right)S_h^* - \mu S_h^* + \rho R_h^* \quad \ldots\ldots (26)$$

Then substitute (26) into (25) we get:

$$\frac{dV}{dt} = \left(\frac{S_h - S_h^*}{S_h}\right)\left((1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right)S_h^* - \mu S_h^* + \rho R_h^*\right) - (1-\beta_e)\left(\frac{\alpha B_v}{K+B_v} + \psi I_h\right)S_h - \mu S_h + \rho R_h + \left(\frac{I_h - I_h^*}{I_h}\right)\left((1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h\right)S_h - \frac{(1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right)S_h^*}{I_h^*}I_h\right) + \left(\frac{B_v - B_v^*}{B_v}\right)\left((1-\beta_e)\varepsilon I_h - \frac{(1-\beta_e)\varepsilon I_h^*}{B_v^*}B_v\right) + \left(\frac{R_h - R_h^*}{R_h}\right)\left((\sigma + \theta)I_h - (\rho - \mu)R_h\right) = (S_h - S_h^*)^2(1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right)S_h^* - (\mu S_h^* + \rho R_h^*)(S_h - S_h^*)^2 - (1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right)(S_h - S_h^*)^2 - \mu S_h + \rho R_h^*(S_h - S_h^*)^2(1-\beta_e)\left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right)S_h^*(I_h - I_h^*)^2 - (\sigma + \theta + \gamma + \mu - \delta)(I_h - I_h^*)^2(1-\beta_e)\varepsilon I_h^*(B_v - B_v^*)^2 - (\omega + \mu 1 - \varphi)B_v^*(B_v - B_v^*)^2 + (\sigma + \theta)I_h^*(R_h - R_h^*)^2 - (\rho + \mu)R_h^*(R_h - R_h^*)^2$$

After rearranging the positive and negative terms we obtain the following term:

$$= \left((1-\beta_e)\left(\frac{\alpha B_v^*}{K + B_v^*} + \psi I_h^*\right)S_h^*(S_h - S_h^*)^2 + \rho R_h^*(S_h - S_h^*)^2 + (1-\beta_e)\left(\frac{\alpha B_v^*}{K + B_v^*} + \psi I_h^*\right)S_h^*\right)(I_h - I_h^*)^2 + (\sigma + \theta)I_h^*(R_h - R_h^*)^2 - (\mu S_h^* + \rho R_h^*)(S_h - S_h^*)^2 + (1-\beta_e)\left(\frac{\alpha B_v^*}{K + B_v^*} + \psi I_h^*\right)(S_h - S_h^*)^2 + \mu + (\sigma + \theta + \gamma + \mu - \delta)(I_h - I_h^*)^2 + (\omega + \mu 1 - \varphi)B_v^*(B_v - B_v^*)^2 + (\rho + \mu)R_h^*(R_h - R_h^*)^2$$

From this we have$\frac{dV}{dt} = X - Y$where$X = \left(\left(1 - \beta_e\right) \left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right) S_h^* (S_h - S_h^*)^2 + \rho R_h^* (S_h - S_h^*)^2 + \left(1 - \beta_e\right) \left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right) S_h^* \right) (I_h - I_h^*)^2 + (\sigma + \theta) I_h^* (R_h - R_h^*)^2andY = (\mu S_h^* + \rho R_h^*)(S_h - S_h^*)^2 + \left(1 - \beta_e\right) \left(\frac{\alpha B_v^*}{K+B_v^*} + \psi I_h^*\right) (S_h - S_h^*)^2 + \mu + (\sigma + \theta + \gamma + \mu - \delta)(I_h - I_h^*)^2 + (\omega + \mu 1 - \varphi) B_v^* (B_v - B_v^*)^2 + (\rho + \mu) R_h^* (R_h - R_h^*)^2$

Hence, if $X < Y$ then we obtain $\frac{dV}{dt} < 0$, whenever the following two conditions are satisfied; $1\ \sigma + \theta + \gamma + \mu > \delta \quad 2\ \omega + \mu b > \varphi$ and, if $S_h = S_h^*, I_h = I_h^*, B_h = B_v^*, R_h = R_h^*$then$\frac{dV}{dt} =$Ttherefore, the largest invariant set$(S_h^*, I_h^*, B_v^*, R_h^*) \in \Omega$: $\frac{dV}{dt} = 0$is the singleton setE^*, where $E^*$is endemic equilibrium point. Hence by the LaSalle's invariant principle [32], the endemic equilibrium point$E^*$is globally asymptotically stable in the setΩ if $X < Y$[32]. .

CHAPTER SIX

6. **NUMERICAL SIMULATION**

In this chapter, we perform numerical simulation for the model equation (4) - (7) in order to show the impact of all used parameters and separated parameters in the model we used. The numerical simulation is carried out using Mat Lab software ode45 of order four. The main emphasis of the simulation is to analyze the impact of infected immigrant, infected human to susceptible human contact rate, impact of public health education followed by some control measure such as medical treatment and water treatment. The parameter used in the simulation is based on some published literature. And reasonable assumption that is fit within our model system and their values are listed in the following table.

Parameters	Unit	Value	Source
Λ	per day	10	Kadaleka (2011) and Musekwa (2011)
μ	per day	0.022	J. Wang and C. Modenak (2011)
α	per day	0.8	codeco (2011) and Ochoche,J.M (2013)
κ	cell/ml	10^6	Ochoche, J.M (2013)
ω	Dimensionless	0.5	Ochoche,J.M (2013)
β_e	Dimensionless	0.98	Assumed
ε	cell/ml $day^{-1}person^{-1}$	10	Ochoche J.M (2013)
σ	per day	0.79	Kadaleka (2011)
θ	per day	0.31	Assumed
ρ	per day	0.012	Assumed
γ	per day	0.015	Kadaleka (2011)
φ	per day	0.02	S.Edward and N.Nyerere (2015)
μ_1	per day	0.04	Assumed

δ	per day	0.5	Assumed
ψ	per day	0.021	Assumed

Table 3: Model parameter values with their unit and source

And the following initial conditions have been used in our simulation;

$S_h(0)$ = 400, $I_h(0)$ = 150, $B_V(0)$ = 120, and $R_h(0)$ = 200 at time t_0 = 0 and t_f = 100.

Let us discuss the impact of the new added parameters on the new model as follow:

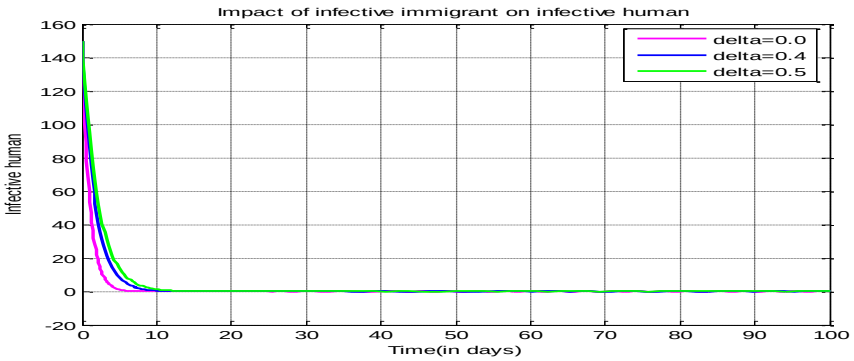

Figure 3: Numerical simulation on the infected human that shows the impact of infected immigrant.

From figure 3, we notice that initially infected immigrant increases the infected human also increases, but after the time increases around from 5 days to 12 days then the infected immigrant decreases and the infected human population also decreases because of the control method we used such as medical treatment, water treatment and public health education. Additionally, we see that, when the infected immigrant increases, then the infected human population also increases proportionally through the time increases. Thus, to control the disease which is caused by the infected immigrant human population in the community, we have to separate those who are infected and give them medical treatment and give awareness about the transmission ways of the cholera disease through public health education. Otherwise the disease spreads out through the community.

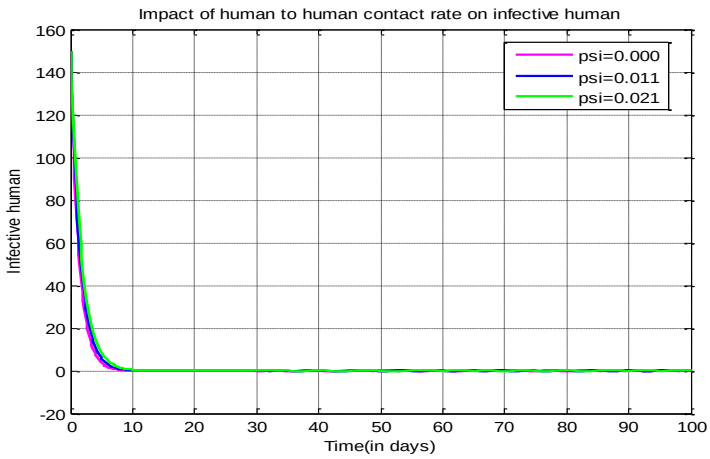

***Figure 4: Numerical simulation on the infected person that illustrates the impact of contact between infective person and susceptible person*.** From figure 4, we have seen that, the impact the contact between infected individual and susceptible individual is directly proportional with the infected human population. i.e., the contact between infected human and susceptible human increases, then the infected human increases proportionally, and it decreases then the infected human population also decreases proportionally. Thus, to minimize the disease caused by such type of transmission way, In addition to providing medical care and clean water, we must educate the public about health issues and teach them how to prevent cholera by washing their hands with soap after using the toilet. If not, the disease getting worse.

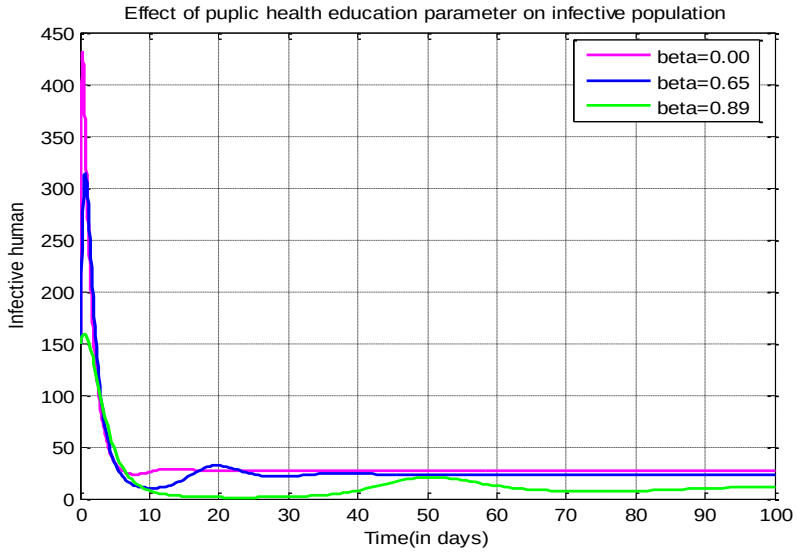

Figure 5: Numerical simulation on the infected human that shows the impact of public health education.

.Figure 5 shows that when there is a disease outbreak, the number of infectious individuals initially rises gradually because many people are not yet educated, but then falls as a result of medical care and water purification. If the health education given in some extent about an outbreak of the disease increased in some extent and it decreases because of the control method applied to the outbreak of the disease. When a high level of health education is provided, the number of infectious cases drops significantly because more people become aware of the outbreak and implement other control measures like applying water and medical treatment.

Consequently, health education is very important to reduce the outbreak of the cholera disease in the society followed by some control measure such as medical treatment and water treatment.

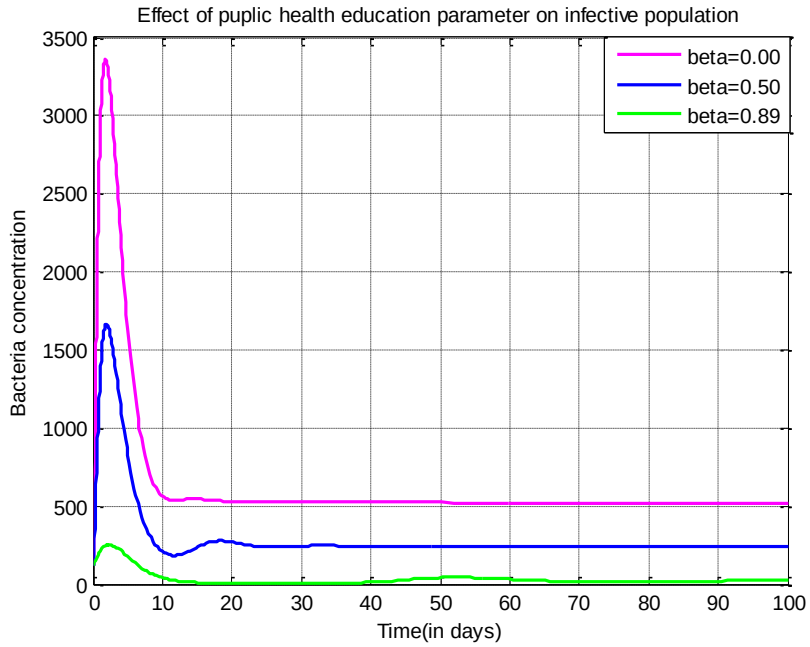

Figure 6: Numerical simulation on the bacteria concentration that shows the impact of public health education. From figure 6, we conclude that, without health education for the human population the contribution of infective population with contaminated aquatic reservoir increase initially and it decreases because of additional control methods are applied to decrease the contribution of infective population with contaminated aquatic reservoir. But, if health education is applied in medium level for the human population about the spread of bacteria population, then the contribution of infective population with contaminated aquatic reservoir increases in some extent and then it decreases. And also, health education is applying strongly, then the contribution of infective population with contaminated aquatic reservoir decreases to zero through the time increases, and as a result we can minimize the concentration of bacteria because of educated human population apply different techniques to reduce the growth rate of the concentration bacteria. Thus, health education is vital method to minimize the concentration of bacteria followed by some control measures such as medical treatment and water treatment.

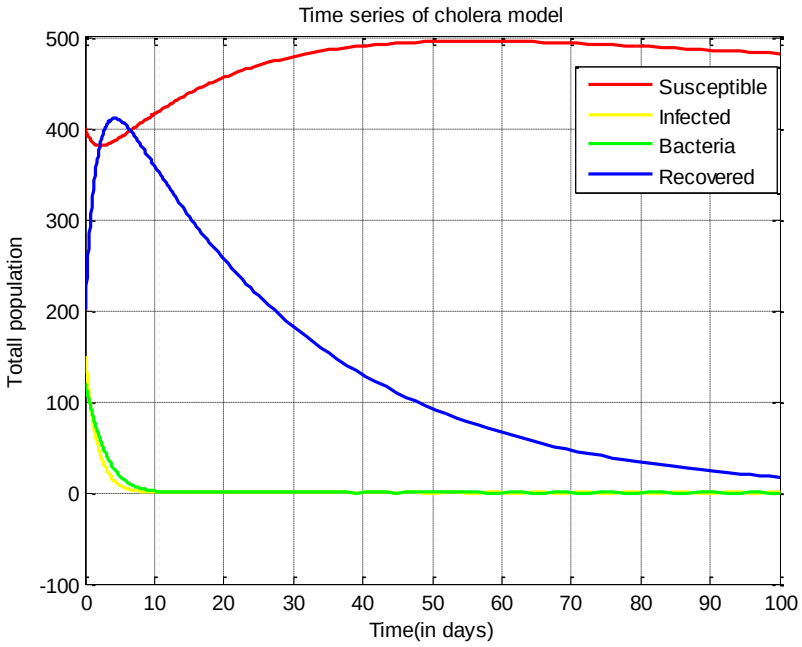

Figure 7: Numerical simulation of the modified model that shows the effect of all used parameters when R0 = 0.2841 < 1, δ = 0.4 and β_e ***= 0.98.*** From figure 7, we conclude that, for the basic reproduction number $R_0 < 1$, disease dies out and the susceptible individuals are increase but the recovered individuals are decreases and also all the solution of the state variables are tends to their equilibrium point. This supports our analytic solution of the model system.

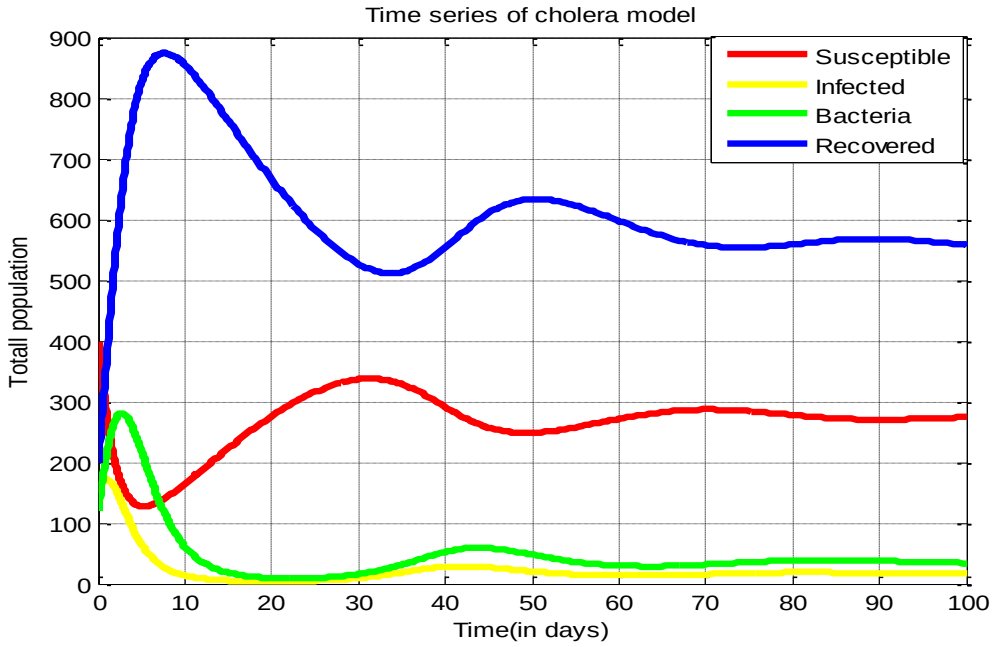

Figure 8: Numerical simulation of the modified model that shows the effect of all used parameters when R0 = 1.5628 > 1, $\delta = 0.5$ and $\beta_e = 0.89$.

From figure 8, we conclude that, when the concentration of bacteria population and infective individuals increases, but the susceptible individuals are decrease due to disease. From the figure we see that for the basic reproduction number $R_0 > 1$ and the disease persist and all the solution of the state variables are tends to their equilibrium point. Thus, the parameter of public health education and infective immigrant have their own effect or impact to increase or decrease the cholera disease followed by the medical treatment and water treatment. i.e., if$\beta_e = 0.89$ and $\delta = 0.5$, then $R_0 > 1$ and the disease persists which means.The disease does spread among the people. But, if $\beta_e = 0.98$ and $\delta = 0.4$, then $R_0 < 1$, then the disease disappears, it cannot spread among individuals.

CHAPTER SEVEN

7. CONCULUSION AND RECOMENDATION

7.1 Conclusion

In this work, we adjusted Ochoche J.M. (2013)'s mathematical model for cholera disease [5] to include medical treatment and public health education as control options in addition to water treatment.

From the model we have derived the basic reproduction number and this determines whether the disease is persistent or dies out from the community. The disease eventually goes extinct if $R_0 < 1$, while it continues to affect the community if $R_0 > 1$. The existence of disease free and endemic equilibrium points are discussed. The local and global stability of both disease free and endemic equilibrium points are proved by using different methods such as standard comparison theorem, Routh-Hurwitz criteria for stability and constructed Lyapunov function.
We have established the boundedness and positivity solution of the equation (4)to(7).

The numerical simulation is formulated to show the impact of public health education followed by some control strategy such as water treatment, medical treatment. Here, health education is apply strongly it is very important method to control the disease together with medical treatment and water treatment. And also, from the simulation we show that the impact of infected immigrant and the contact between infected human with susceptible human for the transmission of the cholera disease. This leads to the spread of the disease, which diminishes when we apply health education in addition to medical care and water treatment to reduce the contact rate between infected individuals and susceptible individuals as well as the rate of infected immigrants.

7.2 Recommendation

From my study, I would like to recommend health organization, governmental and nongovernmental organizations and other community to take responsible in order to protect the outbreak of vibrio cholera disease. Governmental and health organization have a great duty or responsible to create an awareness about the transmission way of vibrio cholera for the community to stop the spread of the cholera disease. And also we recommend for health organizations to apply medical treatment and water treatment together with giving health education for the community and creating awareness about the disease.

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