Mathematical Theory and Modeling
ISSN 2224-5804 (Paper) ISSN 2225-0522 (Online)
Vol.10, No.7, 2020

GLOBAL STABILITY OF AN EPIDEMIC MODEL WITH TWO INFECTED STAGES AND MASS-ACTION INCIDENCE

Assefa Erba [1*], Girma Gadisa [2*]

[1]Assefa Erba, College of Natural and Computational Science, Department of Mathematics,Wolaita Sodo University, Sodo ,Ethiopia

2*Girma Gadisa, Adama Science and Technology University, School of Applied Natural Science, Department of Mathematics

Email: asebikila@gmail.com

Abstract

In this research work, we study the global stability of the SIR model which describes the dynamics of infectious disease with two classes of infected stages and varying total population size. The incidence used in the mathematical modeling was the mass-action incidence. The basic reproduction number R_0 is computed. If the basic reproduction number is less than one, then the disease-free equilibrium point is locally and globally asymptotically stable. Existence and uniqueness of the endemic equilibrium is established when the basic reproduction number is greater than one and locally stable. We prove that global stability of the disease free equilibrium point using Lyapunov function. Numerical simulations have been carried out applying mat lab. Our result show that if the basic reproduction number R_0 is below one the disease free equilibrium point is locally and globally stable in the feasible region, so that the disease dies out. If the basic reproduction number R_0 is greater than one a unique endemic equilibrium point is locally asymptotically stable and the disease free equilibrium point is unstable in the interior of the feasible region and the disease will persist at the endemic equilibrium point if it is initially present.

Keywords: *Equilibrium Stability, SIR, Basic Reproduction Number, Local and Global Stability*

INTRODUCTION

Back ground of the study

In the mid-fourteenth century, the Black Death, a plague epidemic, killed roughly one-third of Europe's population. More recently, in 1918, an outbreak of the flu killed an estimated 20 million people, more people than died in all of World War I. In our own times, the acquired immune deficiency syndrome (AIDS) pandemic has brought untold personal suffering and social losses. The Centers for Disease Control (CDC) estimates that, from 1981 to 2001, approximately 21 million people died from AIDS worldwide. Millions of people all over the world are currently infected with the human immunodeficiency syndrome virus (HIV), about 95% of them in developing countries [6].

Although political, social, and economic factors play a large role in setting public health policies, understanding the dynamics of contagion is an important step. The worldwide eradication of smallpox, through a carefully developed vaccination campaign initiated by the World Health Organization in 1967, is a remarkable example of what can be achieved with a well-designed plan.

Mathematical models have become important tools in analyzing the spread and control of infectious diseases. The model formulation process clarifies assumptions, variables, and parameters; moreover, models provide conceptual results such as thresholds, basic reproduction numbers, contact numbers, and replacement numbers. Understanding the transmission characteristics of infectious diseases in communities, regions, and countries can lead to better approaches to decreasing the transmission of these diseases. Mathematical models are used in comparing, planning, implementing, evaluating, and optimizing various detection, prevention, therapy, and control programs. The mathematical modeling of the dynamics of infectious disease, though often inexact, has enormous potential to help improve human lives. Biologists use mathematical models to understand the dynamics of interaction between populations. We further develop the idea of mathematical model to explore the dynamics of infectious disease [8].

Transmission of a disease is carried out by different agents. A disease transmitted by a virus, such as HIV, influenza, measles, chickenpox, mumps, or polio, generally confers immunity against re-infection while diseases caused by bacteria, such as tuberculosis, typhoid or gonorrhea, offer no immunity. Another form of transmission is due to vectors, which are agents infected by humans which then transfer the disease to another human. A common

example of a vector is the mosquito, which spreads malaria and filariasis. Other disease carrying agents include protozoa, helminthes (worms), and the recently discovered prions, which are thought to cause infections such as mad cow disease.

Epidemiology is the branch of science which essentially deals with the mathematical modeling of spread of diseases. The interest here lies in formulating a mathematical model which will explain the population dynamics of disease causing agents, and can then be analyzed with a view to controlling or eradicating the spread of those agents. The formulation of a model is a process which includes statement of the relevant assumptions, relationship among variables, and parameters and relations governing their behaviors. Of course, the choice of these factors is critical to the model and depends largely on the particular disease to be modeled and the intended purpose of that model. Simple models, by its nature, simplifies the situation by making many assumptions but may still describe qualitative behavior to a reasonable extent, while a more detailed model may provide quantitative predictions, but are usually impossible to solve analytically. The transmission dynamics of a disease could be studied from different perspectives, such as at various levels of a spatial, temporal, or organizational scale. One of the important aspects of the modeling process is how much organizational detail like population structure, immunity, and genetic variability will be included in the model. Then the model builder decides strategy to model these details to effectively describe the disease spread [5]

Mathematical modeling of the epidemic dynamics is an important method of studying the spread of infectious disease qualitatively and quantitatively. It is based on the specific property of population growth, the spread rules of infectious diseases, and the related social factors, etc. To construct a mathematical model which reflects the dynamical properties of infectious diseases and to analyze the dynamical behavior of the disease it is of paramount important to understand the biology of the infectious disease [4].When it is realized that an epidemic has begun, individuals are likely to modify their behavior by avoiding crowds to reduce their contacts and by being more careful about hygiene to reduce the risk that a contact will produce infection.

We formulate our descriptions as compartmental models, with the population under study being divided into compartments and with assumptions about the nature and time rate of transfer from one compartment to another. In most cases the population is divided into three classes denotes as S, I, and R. where:

- S (t) denotes the number of individuals who are susceptible to the disease at any time t ,
- I (t) denotes the number of infected individuals, assumed infectious and able to spread the disease by contact with susceptible,
- R (t) denotes the number of individuals who have been infected and then removed from the possibility of being infected again or of spreading infection. Removal is carried out either through isolation from the rest of the population, or through recovery from the disease with full immunity against re infection.

The rates of transfer between compartments are expressed mathematically using differential equations. Ross (1910) devised ordinary differential equations (ODEs) models to understand the mechanisms of how disease spread. For instance Ross developed transmission models for malaria and derived the first threshold theorem that identified a critical mosquito density required for malaria epidemics. He also introduced the mass action idea in continuous time in his study of the transmission of malaria [2].

The Kermack-McKendrick model is a compartmental model based on relatively simple assumptions on the rates of flow between different classes of members of the population. In order to model an epidemic disease; the population is divided into various classes. In some cases the population is divided into three senior classes: the class of the susceptible individuals, denoted by S, and the class of the infected individuals, denoted by I. Sometimes, the class of the infected can be split into several classes which allow highlighting the state of the disease, and the classes of individuals recovered from the class I and have permanent immunity denoted by R. In our case, the infected are divided into two categories, denoted I_1 and I_2, with I_1 the first stage of the disease and I_2 the worsened case.

DESIGN AND METHODOLOGY

This section includes design of the study instruments and data analysis. We use system nonlinear ordinary differential equations to describe the dynamics of infectious disease with two stages of infected I_1 and I_2. The analysis of the mathematical model which describes the dynamics of infectious disease transmission will be done. The next generation matrix was used to find the basic reproduction number. Jacobean matrix was used to show local stability of the equilibrium points of the model equation. The Lyapunov functions were used to show

global stability of the equilibrium points. The analytical solution of the model equations was supplemented by the numerical simulations using MATLAB and Mathematica.

Model Formulation and Analysis

The model Formulation

The SIR models are well known in the dynamic of infectious population. In this section, we present the SIR model used in research work. We consider two stages of infected population. The population of size N is divided into subclasses of individuals, who are susceptible, infected into the first stage of the disease and infected into the second stage, and recover with sizes denoted by S, I_1 and $I_2$and R.

To build our model we make the following assumptions:

- In the models there are births and deaths, so that the total population size is not constant,
- Transmission of the disease occurs following adequate contacts between a susceptible individual and infectious in respectively the compartments $I_1$and I_2. The standard mass balance incidence expressions $\beta_1 I_1 \frac{S}{N}$ and$\beta_2 I_1 \frac{S}{N}$are used to indicate successful transmission of disease, with β_1 and $\beta_2$denote the per capita contact rate of the infectious in the respective compartments I_1 and I_2.Thus, the new infection is given by$(\beta_1 I_1 + \beta_2 I_2) \frac{S}{N}$.
- Natural death rate, μ, is constant across all the classes;
- The transition rate (denoted γ) from the first stage of infection I_1 to the second stage I_2 is different from the rate of disease-induced death (denoted d).

So, the rate of change of infected population in the first stage is given by:

$\frac{dI_1}{dt} = (\beta_1 I_1 + \beta_2 I_2)\frac{S}{N} - \gamma I_1 - \mu I_1 - \alpha I_1$, and,

The rate of change of infected population in the second stage is given by

$\frac{dI_2}{dt} = \gamma I_1 - \mu I_2 - dI_2 - \alpha I_2$

Hence, the system of differential equation which describes the dynamics of the transmission of the infection is in SI_1I_2R model with disease-induced and natural death is

$$\begin{cases} \frac{dS}{dt} = bN - (\beta_1 I_1 + \beta_2 I_2)\frac{S}{N} - \mu S \\ \frac{dI_1}{dt} = (\beta_1 I_1 + \beta_2 I_2)\frac{S}{N} - \gamma I_1 - \mu I_1 - \alpha I_1 \\ \frac{dI_2}{dt} = \gamma I_1 - \mu I_2 - dI_2 - \alpha I_2 \\ \frac{dR}{dt} = \alpha I_1 + \alpha I_2 - \mu R \end{cases} \quad (1)$$

where $N = S + I_1 + I_2 + R$ is the total population size, b represent the per capita birth rate, μ is the per capita natural death rate of the population, β_1 and β_2 are respectively the per capita transmission rate of the compartments I_1 and I_2, γ is the per capita rate of transfer of infected individuals from the infected stage 1 to stage 2, d is the disease induced death rate, and α is the recovery rate of infected individuals.

Properties of the model

Since system of equation (1) describes the evolution of a human population, it is important to prove the individual's number in each compartment should remain non negative and bounded. So, we establish, in this section, the invariant region of solutions of model and the positivity of non-dimensionalized of system of equation (1).

Invariant Region

The feasible solution shows the region in which the solutions of the equations of the system are biologically meaningful and the solution of the system is non-negative in this region.

Let the total population size N satisfies the equation:

$$\frac{dN}{dt} = (b - \mu)N - dI_2$$

Let $bN = A$, and constant, then;

$$\frac{dN}{dt} = A - \mu N - dI_2$$

$$\leq A - \mu N$$

Integrating and rearranging we have $N \leq \frac{A}{\mu}$

Hence, the feasible region for the system of equations (1) is given as:

$$\Gamma = \left\{(S, I_1, I_2, R) \in \mathbb{R}^4 : S + I_1 + I_2 + R \leq \frac{A}{\mu}\right\}$$

Using proportions: $s = \frac{S}{N}, i_1 = \frac{I_1}{N}, i_2 = \frac{I_2}{N}$, and $w = \frac{R}{N}$we can non-dimensionalized the system of equations (1) as follows:

$$\frac{ds}{dt} = b - bs - (\beta_1 i_1 + \beta_2 i_2)s + dsi_2$$

In the same way: $i_1 = \frac{I_1}{N}$

$$\frac{di_1}{dt} = (\beta_1 i_1 + \beta_2 i_2)s - (b + \gamma + \alpha)i_1 + di_1 i_2$$

Similarly for i_2; we have $i_2 = \frac{I_2}{N}$

$$\frac{di_2}{dt} = \gamma i_1 - (b + d + \alpha)i_2 + di_2{}^2, \text{ and;}$$

$$\frac{dw}{dt} = (i_1 + i_2)\alpha - wb - dwi_2$$

Thus, the non-dimensionalized system of differential equation is

$$\begin{cases} \frac{ds}{dt} = b(1 - s) - (\beta_1 i_1 + \beta_2 i_2)s + dsi_2 \\ \frac{di_1}{dt} = (\beta_1 i_1 + \beta_2 i_2)s - (b + \gamma + \alpha)i_1 + di_1 i_2 \\ \frac{di_2}{dt} = \gamma i_1 - (b + d + \alpha)i_2 + di_2{}^2 \\ \frac{dw}{dt} = (i_1 + i_2)\alpha - wb - dwi_2 \end{cases} \quad (2)$$

2. Positivity of Solutions

We proved that all the variables in the model equations are non-negative.

Lemma: If the initial data set $be(s, i_1, i_2, w)\ (0) \geq 0 \in \Omega$, then the solution set $(s, i_1, i_2, w)\ (t)$ of the equations in system (2) is positive for all $t > 0$.

Proof: From equation (1) if it is assumed that:

$$\frac{ds}{dt} \leq b - bs$$

Integrating and rearranging we have: $s(t) \leq b^2 + ce^{-bt}$

Applying initial conditions when t=0, s (t) =s (0) we get

$$s(0) = b^2 + c \Rightarrow c = s(0) - b^2$$

$$s(t) \leq b^2 + (s(0) - b^2)e^{-bt}, \text{ but } b^2 + (s(0) - b^2)e^{-bt} > 0$$

It then follows that;

$$s(t) \geq 0, \forall t \geq 0$$

From equation (2) of the system of equation (2)

$$\frac{di_1}{dt} = (\beta_1 i_1 + \beta_2 i_2)s + di_1 i_2 - (b + \gamma + \alpha)i_1$$

If it is assumed that: $\frac{di_1}{dt} > -(b + \gamma + \alpha)i_1$

Integrating and rearranging we have: $i_1(t) > e^{-(b+\gamma+\alpha))t+c}$

Applying initial conditions that when t=0,$i_1(t) = i_1(0)$ we get

$i_1(t) > i_1(0)e^{-(b+\gamma+\alpha))t}$, hence $i_1(0)e^{-(b+\gamma+\alpha))t} \geq 0$

It follows that;

$i_1(t) \geq 0 , \forall t \geq 0$

From equation (3) of the system of equations (2)

$$\frac{di_2}{dt} = \gamma i_1 + d{i_2}^2 - (b + d + \alpha)i_2$$

If it is assumed that: $\frac{di_2}{dt} > -(b + d + \alpha)i_2$

Integrating and rearranging we have: $i_2(t)) > e^{-(b+d+\alpha)t+c}$

Applying initial conditions that when t=0,$i_2(t) = i_2(0)$ we get

$i_2(t)) > i_2(0)e^{-(b+d+\alpha)t}$, but $i_2(0)e^{-(b+d+\alpha)t} > 0.$

Hence $i_2(t) \geq 0 , \forall t \geq 0$

From equation (4) of the system of equations (2) we have

$$\frac{dw}{dt} = (i_1 + i_2)\alpha - w(b + di_2)$$

If it is assumed that; $\frac{dw}{dt} \geq -wb$, then

Integrating and rearranging we have:$w(t) \geq e^{-bt+c}$

Applying initial conditions that when t=0, $w(t) = w(0)$ we get

$w(t) \geq w(0)e^{-bt}$, but $w(0)e^{-bt} \geq 0, \forall t \geq 0$

$w(t) \geq 0 , \forall t \geq 0$

Therefore it is true that

$s(t) \geq 0 , i_1(t) \geq 0, i_2(t) \geq 0$ and $w(t) \geq 0,$ for all $t \geq 0.$

Equilibrium Points

Equilibrium points are found by setting the right hand sides of system (2) equal to zero. This gives two equilibrium points in the feasible region, the disease-free equilibrium point E_1 = (1, 0, 0, 0) and the unique endemic equilibrium point

E_2= (s*, i_1*, i_2*, w*). The unique endemic equilibrium point $E_2$for the system (2) assuming that b≥ d, where b and d represent the birth and the disease induced rate respectively.

Proposition If R_0> 1 the endemic equilibrium point exists and is unique.

Proof: From the third equation of system (2) we have:

$$i_1^* = \frac{b+d+\alpha}{\gamma} i_2^* - \frac{d}{\gamma} i_2^{*2} \qquad (3)$$

Substituting i_1 in the second equation of system (2) we obtain i_1^*

$$\left(\beta_1(b+d+\alpha) - \beta_1 d i_2^* + \beta_2\gamma\right)s^* - (b+\gamma+\alpha)(b+d+\alpha) +$$

$$d(b+d+\alpha)i_2^* + d(b+d+\alpha)i_2^* - d^2 i_2^{*2} = 0 \qquad (4)$$

Also, in (4) we replace s∗ by its expression given by:

$$S^* = 1 - i_1^* - i_2^*$$

Then, $s^* = 1 - \frac{b+d+\alpha}{\gamma} i_2^* - \frac{d}{\gamma} i_2^{*2} - i_2^*$

Solution of the polynomial:

$$P(i_2^*) = A(i_2^*)^3 + B(i_2^*)^2 + C(i_2^*) + D = 0$$

where $A = -\beta_1 \frac{d^2}{\gamma}$, $B = 2\beta_1 d \frac{b+d+\alpha}{\gamma} + \beta_1 d + \beta_2 d - d^2$

$$C = -\beta_1 d \frac{(b+d+\alpha)^2}{\gamma} - \beta_1(b+d+\alpha) - \beta_1 d - \beta_2(b+d+\alpha) - \beta_2\gamma + d(b+\gamma+\alpha)$$

$$+ d(b+d+\alpha)$$

$$= -R_0(b+d+\alpha)(b+\gamma+\alpha)\left(1 + \frac{b+d+\alpha}{\gamma}\right)4 - \beta_1 d + d(2(b+\alpha)+d+\gamma)$$

$$D = \beta_1(b+d+\alpha) + \beta_2\gamma - (b+d+\alpha)(b+\gamma+\alpha)$$

$$= (b+d+\alpha)(b+\gamma+\alpha)(R_0 - 1).$$

Using the fact that R_0> 1, A < 0, B > 0, C < 0, and D > 0.

We have P (i_2^*) = 0 ⇔ Q (i_2^*) = R_0, where Q is the polynomial given by

$$Q(i_2^*) = -\frac{A}{k}(i_2^*)^3 - \frac{B}{k}(i_2^*)^2 - \frac{C}{k} i_2^* + 1, \text{and } k = (b+d+\alpha)(b+\gamma+\alpha)$$

We have: $Q(0) = 1$

$$Q(1) = \frac{\beta_1(b^2+b\gamma+d\gamma)+\gamma[b(b-d+\gamma)+\beta_2(b+\gamma)]}{k}$$

Also

$$Q(1) - R_0 = b\frac{\beta_1 b+\beta_2\gamma+b\gamma+\gamma^2-d\gamma}{k\gamma},$$

This is positive if and only if

$$\beta_1 b + \beta_2\gamma + b\gamma + \gamma^2 > d\gamma \ . \qquad (5)$$

The relation (5) is satisfied using to the assumption b ≥ d. Thus,

1 = Q (0) < R_0< Q (1). Let us localize exactly the domain of i_2^*. We have

$$i_1^* + i_2^* < 1 \qquad (6)$$

And since, by the relation (3), $i_1^* = \frac{b+d+\alpha}{\gamma} i_2^* - \frac{d}{\gamma} i_2^{*2}$ we deduce that i_2^* must verify the following inequality:

$$R(i_2^*) = -d i_2^{*2} + (b + d + \gamma) i_2^* - \gamma < 0.$$

The discriminate of the polynomial R is

$$\Delta_R = (b + d + \gamma)^2 - 4d\gamma = b^2 + 2b(d + \gamma) + (d - \gamma)^2 > 0 \ .$$

The roots of R are: $r_1 = \frac{(b+d+\gamma-\sqrt{\Delta_R})}{2d}$ and $r_2 = \frac{(b+d+\gamma+\sqrt{\Delta_R})}{2d}$.

We have: $r_1 < \frac{\gamma}{2d} < r_2$, and with the assumption b ≥ d we have r_2> 1; i_2^* mustsatisfy

0 < i_2^* < min {r_1, 1} ≤ min $\{\frac{\gamma}{2d}, 1\}$, that is i_2^* must belong to the interval

I = (0, min {r_1, 1}) ⊂ (0, min $\{\frac{\gamma}{2d}, 1\}$).

On the other hand, we have

$$Q(r) - R_0 = \left(\frac{1}{2k}\right)[b(b + d + \gamma + \sqrt{\Delta_R}] > 0.$$

Since Q (0) = 1 < R_0, Q (r_1) > R_0, and Q (1) > R_0, the graph of Q intersects the horizontal line y = R_0 at least one time in I.

Now let us show that there is exactly one intersection in I. The derivative of Q is:

$$\frac{dQ}{di_2^*} = -\left(\frac{1}{k}\right)(3Ai_2^{*2} + 2Bi_2^* + C).$$

Note that by Descartes rules of signs there is no negative root; on the other hand, the discriminate of $\frac{dQ}{di_2^*}$ is $\Delta = B^2 - 3AC$, we then have two cases:

- If $\Delta \leq 0$, $\frac{dQ}{di_2^*}$ is positive on R(the set of real numbers)
- If $\Delta > 0$, we have two roots x_1 and x_2, and $x_1 + x_2 = -(\frac{2B}{3A})$. However

$$-\frac{2B}{3A} = \frac{4}{3}\frac{b+d}{d} + \frac{2}{3}\{\frac{\gamma}{d} + \frac{\beta_2\gamma}{\beta_1 d} - \frac{\gamma}{\beta_1}\}$$

$$= \frac{2}{3}\frac{b+d}{d} + \frac{2}{3}\{\frac{b+d}{d} + \frac{\gamma}{d} + \frac{\beta_2\gamma}{\beta_1 d} - \frac{\gamma}{\beta_1}\}$$

$$= \frac{2}{3}\frac{b+d}{d} + \frac{2}{3\beta_1 d}\{\beta_1(b + d) + \beta_2\gamma + \beta_1\gamma - d\gamma\}$$

$$= \frac{2}{3}\frac{b+d}{d} + \frac{2}{3\beta_1 d}\{(b+d)(b+\gamma)R_0 + \beta_1\gamma - d\gamma\}$$

Thus

$$-\frac{2B}{3A} = \frac{2}{3}\frac{b+d}{d} + \frac{2}{3\beta_1 d}\{b(b+\gamma)R_0 + bdR_0 + \beta_1\gamma + d\gamma(R_0 - 1)\}$$

We know that$b(b+\gamma)R_0 = \beta_1 b + \frac{\beta_2 b\gamma}{b+d}$.Since $b \geq d$ we have $-\frac{2B}{3A} = 2$.

Thus there is at least one root of $\frac{dQ}{di_2^*}$ larger than one.

All these observations show that the graph of Q intersects the line y = R_0 only once.

i_1^* is deduced by $i_1^* = \frac{b+d+\alpha}{\gamma} i_2^* - \frac{d}{\gamma} i_2^{*2}$, $s^* = 1 - \frac{b+d+\gamma}{\gamma} i_2^* - \frac{d}{\gamma} i_2^{*2}$, and

$$w^* = \frac{\left[(b+d+\alpha)i_2^* - di_2^{*2} + \gamma i_2^*\right]\alpha}{\gamma(b+di_2^*)}$$

Then, the endemic equilibrium exists and is unique.

The basic reproduction number (R_0)

Basic reproduction number, denoted by R_0, represents the average number of secondary infectious infected by an individual of infective during whose whole course of disease in the case that all the members of the population are susceptible. According to this meaning, it is easy to understand that if R_0< 1 then the infective will decrease so that the disease will go to extinction; if R_0>1 then the infective will increase so that the disease cannot be eliminated and usually develop into an endemic. From the mathematical point of view, usually when R_0< 1, the model has only disease free equilibrium E_1(1, 0, 0) and $E_1$is globally asymptotically stable; when

R_0> 1, the equilibrium becomes unstable and usually a positive equilibrium point

E_2= (s^*, i_1^*, i_2^*, w^*) appears.$E_2$is called an endemic equilibrium and in this case it is stable. Hence, if all the members of a population are susceptible in the beginning, then Ro = 1 is usually a threshold whether the disease go to extinction or go to an endemic.

We determine the basic reproduction number (R_0) using next generation matrix by linearization about the disease-free equilibrium$E_1(1,0,0,0)$.

Let $\boldsymbol{\mathcal{F}}_j(s, i_1, i_2)$ the rate of appearance of new infections in compartment j, and by $V_j(s, i_1, i_2)$the rate of transfer of individuals in and out the compartment j by all other means. The difference between $\mathcal{F}_j(s, i_1, i_2)$ and $V_j(s, i_1, i_2)$gives the rate changes of compartment j. The crucial point which we have to notice here is that $\boldsymbol{\mathcal{F}}_i$ should include only

infections that are newly arising, but does not include terms which describe the transfer of infectious individuals from one infected compartment to another; i.e.

$$\mathcal{F} = \begin{bmatrix} 0 \\ (\beta_1 i_1 + \beta_2 i_2)s \\ 0 \\ 0 \end{bmatrix}, \text{and} V = \begin{bmatrix} b(1-s) - (\beta_1 i_1 + \beta_2 i_2)s + dsi_2 \\ -(b+\gamma+\alpha)i_1 + di_1 i_2 \\ \gamma i_1 - (b+d+\alpha)i_2 + di_2{}^2 \\ (i_1+i_2)\alpha - wb - dwi_2 \end{bmatrix}$$

$$F = \mathcal{D}\mathcal{F}(E_1) = \begin{pmatrix} 0 & 0 & 0 \\ 0 & \beta_1 & \beta_2 \\ 0 & 0 & 0 \end{pmatrix} \text{ and}$$

$$V = \mathcal{D}V(E_1) = \begin{pmatrix} -b & -\beta_1 & -\beta_2 + d \\ 0 & -(b+\gamma+\alpha) & 0 \\ 0 & \gamma & -(b+d+\alpha) \end{pmatrix}$$

$$|V| = \begin{vmatrix} -b & -\beta_1 & -\beta_2 + d \\ 0 & -(b+\gamma+\alpha) & 0 \\ 0 & \gamma & -(b+d+\alpha) \end{vmatrix} = -b(b+\gamma+\alpha)(b+d+\alpha) \text{ , and}$$

$$\text{adj}V =$$

$$\begin{pmatrix} (b+\gamma+\alpha)(b+d+\alpha) & -\beta_1(b+d+\alpha) + (-\beta_2+d)\gamma & (-\beta_2+d)(b+\gamma+\alpha) \\ 0 & b(b+d+\alpha) & 0 \\ 0 & b\gamma & b(b+\gamma+\alpha) \end{pmatrix}$$

Then

$$V^{-1} = \frac{1}{|V|}\text{adj}V = \begin{pmatrix} \frac{-1}{b} & \frac{-\beta_1(b+d+\alpha)+(-\beta_2+d)\gamma}{b(b+\gamma+\alpha)(b+d+\alpha)} & \frac{-\beta_2+d}{b(b+d+\alpha)} \\ 0 & \frac{-1}{(b+\gamma+\alpha)} & 0 \\ 0 & \frac{-r}{(b+\gamma+\alpha)(b+d+\alpha)} & \frac{-1}{b+d+\alpha} \end{pmatrix}$$

The next generation matrix is given as $\mathcal{F}V^{-1}$

$$\mathcal{F}V^{-1} = \begin{pmatrix} 0 & 0 & 0 \\ 0 & \frac{-\beta_1}{b+\gamma+\alpha} - \frac{\beta_2\gamma}{(b+\gamma+\alpha)(b+d+\alpha)} & \frac{-\beta_2}{b+d+\alpha} \\ 0 & 0 & 0 \end{pmatrix}$$

The Basic Reproduction Number is the Eigen values of largest magnitude, or spectral radius of the next generation matrix, that is, the number of all new infectious host types in the next generation.

$$R_0 = \rho(-\mathcal{F}V^{-1})$$

Thus, $R_0 = \frac{\beta_1}{b+\gamma+\alpha} + \frac{\beta_2\gamma}{(b+\gamma+\alpha)(b+d+\alpha)}$

Stability Analysis

Local Stability at Equilibrium Point

Local Stability at Disease Free Equilibrium Point

Theorem If $R_0 < 1$, then the disease-free equilibrium point is locally asymptotically stable.

Proof: The Jacobean matrix of the linearized system of equations (2) at the disease free equilibrium point $(1, 0, 0, 0)$ is:

$$J(1,0,0,0) = \begin{pmatrix} -b & -\beta_1 - \beta_2 + d & 0 \\ 0 & \beta_1 - (b+\gamma+\alpha)\beta_2 & 0 \\ 0 & \gamma - (b+d+\alpha) & 0 \\ 0 & & \alpha\alpha - b \end{pmatrix}$$

The determinant of J (E_1) is given by:

$$\det(J(E_2)) = \begin{vmatrix} -b & -\beta_1 - \beta_2 + d & 0 \\ 0 & \beta_1 - (b+\gamma+\alpha)\beta_2 & 0 \\ 0 & \gamma - (b+d+\alpha) & 0 \\ 0 & & \alpha\alpha - b \end{vmatrix}$$

$$= -b \begin{vmatrix} \beta_1 - (b+\gamma+\alpha) & \beta_2 & 0 \\ \gamma & -(b+d+\alpha) & 0 \\ \alpha & \alpha & -b \end{vmatrix}$$

$$= -b\left[\left(\beta_1 - (b+\gamma+\ \alpha)\right)(b+d+\alpha)b + \ \beta_2\gamma\, b\right]$$

$$= -b^2\left[\beta_1(b+d+\alpha) + \beta_2\gamma\, b - (b+\gamma+\alpha)(b+d+\alpha)\right]$$

$$= -b^2[(b+\gamma+\alpha)(b+d+\alpha)[R_0 - 1],$$

$\det(J(E_2)) > 0$, since $R_0 < 1$

The determinant is positive furthermore the trace is negative, because it is givenby:

$$\mathrm{tr}J(E_2) = -(2b - \beta_1) - (b+\gamma+\alpha) - (b+d+\alpha) < 0$$

Thus, the disease-free equilibrium is locally stable using Routh-Hurwitz conditions.

Local Stability of the Endemic Equilibrium point

With the assumption $b \geq d$ we have the following result:

Theorem The endemic equilibrium point is locally asymptotically stable if $R_0 > 1$.

Proof: Since $s + i_1 + i_2 + w = 1$, we can eliminate s in system of equations (2). Therefore, we get the following system:

$$\begin{cases} \frac{di_1}{dt} = (\beta_1 i_1 + \beta_2 i_2)(1 - i_1 - i_2 + w) - (b+\gamma+\alpha)i_1 + di_1 i_2 \\ \frac{di_2}{dt} = \gamma i_1 - (b+d+\alpha)i_2 + d{i_2}^2 \end{cases} \quad (7)$$

The Jacobean of system of equations (7) at the endemic equilibrium $E_2(i_1^*, i_2^*)$ is:

$$J(E_2) = \begin{pmatrix} \beta_1 + \beta_1 w^* - 2\beta_1 i_1^* - \beta_1 i_2^* - \beta_2 i_2^* - (b+\gamma+\alpha) + d i_2^* & \beta_2 - 2\beta_2 i_2^* - \beta_2 i_1^* - \beta_1 i_1^* - \beta_2 w^* + d i_1^* \\ \gamma & -(b+d+\alpha) + 2 d i_2^* \end{pmatrix}$$

At the endemic equilibrium point we have:

$$\beta_1 - 2\beta_1 i_1^* - \beta_1 i_2^* - \beta_2 i_2^* - (b+\gamma+\alpha) + d i_2^* + \beta_1 w^* = -\beta_2 i_2^* \frac{1 - i_2^* - w^*}{i_1^*} - \beta_1 i_1^*$$

The determinant of J (E_2) is given by:

$$\det(J(E_2)) = \beta_2 (b+d+\alpha) i_2^* \frac{1 - i_2^* - w^*}{i_1^*} + \beta_1 (b+d+\alpha) i_1^* - 2\beta_2 d {i_2^*}^2 \frac{1 - i_2^* - w^*}{i_1^*}$$
$$- 2\beta_1 d i_1^* i_2^* - \beta_2 \gamma + 2\beta_2 \gamma i_2^* + \beta_2 \gamma i_1^* + \beta_1 \gamma i_1^* - d\gamma i_1^* + \beta_2 w^* \gamma$$
$$= \beta_2 (b+d+\alpha) i_2^* \frac{1 - i_2^* - w^*}{i_1^*} + (\beta_1 (b+d+\alpha) + \beta_2 \gamma) i_1^*$$
$$+ 2\beta_2 i_2^* \left(\gamma - d i_2^* \frac{1 - i_2^* - w^*}{i_1^*} \right) - 2\beta_1 d i_1^* i_2^* - \beta_2 \gamma + \beta_1 \gamma i_1^* - d\gamma i_1^* + \beta_2 w^* \gamma$$

In the first term of the determinant, we replace $(b+d+\alpha) i_2^*$ by $\gamma i_1^* + d {i_2^*}^2$ and we get:

$$\det(J(E_2)) = \beta_2 \left(\gamma i_1^* + d {i_2^*}^2 \right) \frac{1 - i_2^* - w^*}{i_1^*} + (b+d+\alpha)(b+\gamma+\alpha) R_0 i_1^* +$$
$$2\beta_2 \frac{i_2^*}{i_1^*} \left(\gamma i_1^* - d i_2^* + d {i_2^*}^2 + d i_2 w^* \right) - 2\beta_1 d i_1^* i_2^* - \beta_2 \gamma + \beta_1 \gamma i_1^* - d\gamma i_1^* + \beta_2 w^* \gamma$$

We replace again $\gamma i_1^* - d i_2^* + d {i_2^*}^2$ by $(b+\alpha) i_2^*$ and bydeveloping the first term of the determinant, we get:

$$\det(J(E_2)) = \beta_2 \gamma - \beta_2 \gamma i_2^* - \beta_2 \gamma w^* + \beta_2 d {i_2^*}^2 \frac{1 - i_2^*}{i_1^*} + (b+d+\alpha)(b+\gamma+\alpha) R_0 i_1^* +$$
$$2\beta_2 (b+\alpha) \frac{{i_2^*}^2}{i_1^*} + \beta_2 d \frac{{i_2^*}^2 w^*}{i_1^*} - 2\beta_1 d i_1^* i_2^* - \beta_2 \gamma + \beta_1 \gamma i_1^* - d\gamma i_1^* + \beta_2 w^* \gamma$$
$$\det(J(E_2)) = \beta_2 i_2^* \left(-\gamma + d i_2^* \frac{1 - i_2^*}{i_1^*} + (b+\alpha) \frac{i_2^*}{i_1^*} \right) + (b+d+\alpha)(b+\gamma+\alpha) R_0 i_1^*$$
$$+ \beta_2 (b+\alpha) \frac{{i_2^*}^2}{i_1^*} + \beta_2 d \frac{{i_2^*}^2 w^*}{i_1^*} - 2\beta_1 d i_1^* i_2^* + \beta_1 \gamma i_1^* - d\gamma i_1^*$$
$$\det(J(E_2)) = \beta_2 \frac{i_2^*}{i_1^*} \left(-\gamma i_1^* + (b+d+\alpha) i_2^* - d {i_2^*}^2 \right) + [(b+d+\alpha)(b+\gamma+\alpha) R_0 - d\gamma] i_1^*$$
$$+ \beta_2 (b+\alpha+d w^*) \frac{{i_2^*}^2}{i_1^*} - 2\beta_1 d i_1^* i_2^* + \beta_1 \gamma i_1^*$$

Thus

$$\det(J(E_2)) = [(b+d+\alpha)(b+\gamma+\alpha)R_0 - d\gamma]i_1^* + \beta_2 \frac{i_2^*}{i_1^*}[2(b+\alpha)i_2^* - \gamma i_1^* + (1 - i_2^* + w^*)di_2^*] + \beta_1 i_1^*(\gamma - 2di_2^*)$$

The determinant is positive because $i_2^* \in (0, \frac{\gamma}{2d})$furthermore the trace is negative, because it is givenby:

$$trJ(E_2) = -\beta_2 i_2^* \frac{1 - i_2^* - w^*}{i_1^*} - \beta_1 i_1^* - (b+d+\alpha) + 2di_2^*$$

Then the endemic equilibrium point is asymptotically stable using Routh-Hurwitz conditions.

Global Stability of the Disease Free Equilibrium Point

Theorem3.3. If $R_0 < 1$, the disease free equilibrium point is globally asymptotically stable.

We consider the following Lyapunov function

$$V = s - \ln s + i_1 + \left(\frac{b+\gamma+\alpha}{\gamma} - \frac{\beta_1}{\gamma}\right) i_2.$$

We obtain

$$\frac{\partial V}{\partial t} = \frac{\partial V}{\partial s}\frac{ds}{dt} + \frac{\partial V}{\partial i_1}\frac{di_1}{dt} + \frac{\partial V}{\partial i_2}\frac{di_2}{dt}$$

$$= \left(1 - \frac{1}{s}\right)\frac{ds}{dt} + \frac{di_1}{dt} + \left(\frac{b+\gamma+\alpha}{\gamma} - \frac{\beta_1}{\gamma}\right)\frac{di_2}{dt}$$

$$= \left(1 - \frac{1}{s}\right)(b - bs - (\beta_1 i_1 + \beta_2 i_2)s + dsi_2) + \left((\beta_1 i_1 + \beta_2 i_2)s - (b+\gamma+\alpha)i_1 + di_1 i_2\right) + \left(\frac{b+\gamma+\alpha}{\gamma} - \frac{\beta_1}{\gamma}\right)\left(\gamma i_1 - (b+d+\alpha)i_2 + di_2^{\,2}\right)$$

$$= (b+bs)\left(1 - \frac{1}{s}\right) - (\beta_1 i_1 + \beta_2 i_2)s + (\beta_1 i_1 + \beta_2 i_2) + dsi_2 - di_2 + (\beta_1 i_1 + \beta_2 i_2)s - (b+\gamma+\alpha)i_1 + di_1 i_2 + (b+\gamma+\alpha)i_1 - \frac{(b+\gamma+\alpha)(b+d+\alpha)}{\gamma} i_2 + \frac{d(b+\gamma+\alpha)}{\gamma} i_2^2 - \beta_1 i_1 + \beta_1 \frac{b+d+\alpha}{\gamma} i_2 - \beta_1 \frac{d}{\gamma} i_2^2$$

We get

$$\frac{\partial V}{\partial t} = -\frac{b}{s}(1-s)^2 + \beta_2 i_2 + di_2(s + i_1 - 1) - \frac{(b+\gamma+\alpha)(b+d+\alpha)}{\gamma} i_2 + \frac{bd}{\gamma} i_2^2 + di_2^2 + \beta_1 \frac{b+d+\alpha}{\gamma} i_2 - \beta_1 \frac{d}{\gamma} i_2^2$$

We have the followings equalities:

$$\frac{b}{s}(1-s)^2 = \frac{b}{s}(i_1 + i_2 + w)^2 \text{ and } di_2(s + i_1 - 1) = -di_2\,(i_2 + w)$$

Then V becomes:

$$\frac{\partial V}{\partial t} = -\frac{b}{s}(i_1 + i_2 + w)^2 + \beta_2 i_2 - \frac{(b+\gamma+\alpha)(b+d+\alpha)}{\gamma} i_2 + \frac{bd}{\gamma} i_2^2 + \beta_1 \frac{b+d+\alpha}{\gamma} i_2 - \beta_1 \frac{d}{\gamma} i_2^2 - d i_2 w$$

$$= -\frac{b}{s}(i_1 + i_2 + w)^2 + \frac{(b+\gamma+\alpha)(b+d+\alpha)}{\gamma}(R_0 - 1) i_2 + \frac{bd}{\gamma} i_2^2 - \beta_1 \frac{d}{\gamma} i_2^2 - d i_2 w$$

$$= -\frac{b}{s}(i_1 + i_2 + w)^2 - \frac{(b+\gamma+\alpha)(b+d+\alpha)}{\gamma}(1 - R_0) i_2 - \beta_1 \frac{d}{\gamma} i_2^2 + \frac{bd}{\gamma} i_2^2 - d i_2 w$$

As $\frac{1}{s} \geq 1$ and $i_2 \geq i_2^2$ since $s < 1$, $i_2 < 1$, & $-(i_1 + i_2)^2 \geq -(i_1 + i_2 + w)^2$ we have

$$\frac{\partial V}{\partial t} \leq -b(i_1 + i_2)^2 - \frac{(b+\gamma+\alpha)(b+d+\alpha)}{\gamma}(1 - R_0) i_2^2 - \beta_1 \frac{d}{\gamma} i_2^2 + \frac{bd}{\gamma} i_2^2 - d i_2 w$$

$$\leq -b i_1^2 - 2b i_1 i_2 - b i_2^2 - d i_2 w - \frac{(b+\gamma+\alpha)(b+d+\alpha)}{\gamma}(1 - R_0) i_2^2 - \beta_1 \frac{d}{\gamma} i_2^2 + \frac{bd}{\gamma} i_2^2$$

$$\leq -b i_1^2 - 2b i_1 i_2 - d i_2 w - \frac{i_2^2}{\gamma}[b\gamma + (b+\gamma+\alpha)(b+d+\alpha)(1 - R_0) + \beta_1 d - bd]$$

Let $D = b\gamma + (b+\gamma+\alpha)(b+d+\alpha)(1 - R_0) + \beta_1 d - bd$, then

$$\frac{\partial V}{\partial t} \leq -b i_1^2 - 2b i_1 i_2 - d i_2 w - D \frac{i_2^2}{\gamma}$$

Therefore, $\frac{\partial V}{\partial t} \leq 0$ if $D \geq 0$.

We rewrite D in the following form:

$$D = b\gamma + (b+\gamma+\alpha)(b+d+\alpha) - \beta_1(b+d+\alpha) - \beta_2\gamma + \beta_1 d + \beta_1\alpha - bd$$

$$= b\gamma + b^2 + bd + b\gamma + d\gamma + b\alpha + \gamma\alpha + b\alpha - \beta_1 b - \beta_1 d - \beta_1\alpha - \beta_2\gamma + \beta_1 d - bd$$

$$= b^2 + 2b\gamma + d\gamma + 2b\alpha + \gamma\alpha - \beta_1 b - \beta_2\gamma$$

$$= b(b + 2\alpha - \beta_1) + \gamma(2b + d + \alpha - \beta_2)$$

Since $R_0 < 1$ then $R_0 = \frac{\beta_1}{b+\gamma+\alpha} + \frac{\beta_1(b+\gamma+\alpha) + \beta_2\gamma}{(b+\gamma+\alpha)(b+d+\alpha)} < 1$,

Thus, $\beta_1 b + \beta_2\gamma < b^2 + 2\alpha b + 2b\gamma + \gamma d + \gamma\alpha$, and, $b > \beta_1$ we get $D \geq 0$. Therefore we conclude that $V \leq 0$. So the disease free equilibrium is globally asymptotically stable.

Since $s + i_1 + i_2 + w = 1$, we can reduce the system of equations (2) toa planar system and investigate the global attraction of the endemic equilibrium point when

$R_0 > 1$. To this end, let us consider the following system:

$$\begin{cases} \frac{ds}{dt} = b(1-s) - (\beta_1 i_1 + \beta_2(1 - i_1 - s))s + d s(1 - i_1 - s) \\ \frac{di_1}{dt} = (\beta_1 i_1 + \beta_2(1 - i_1 - s))s - (b+\gamma) i_1 + d i_1(1 - i_1 - s) \end{cases} \quad (8)$$

Defined on the set $\Omega = \{0 \leq s \leq 1, 0 \leq i_1 \leq 1, w \leq 1, s + i_1 \leq 1\}$. We establish by the Jacobian matrix if $R_0 > 1$, disease free equilibrium is unstable.

Proposition When the endemic equilibrium E_2exists and is globally asymptotically stable on Ω (i.e., if $R_0 > 1$), the infection-free equilibrium E_1 is an unstable.

Proof: If R_0> 1, the Jacobean matrix of system of (8) at the point (1, 0) given as;

$$J(1,0) = \begin{pmatrix} -b + \beta_2 - d & -\beta_1 + \beta_2 - d \\ -\beta_2 & \beta_1 - \beta_2 - (b + \gamma + \alpha) \end{pmatrix} = A$$

The eigenvalues of the Jacobean matrix at the disease free equilibrium point are the solution of the characteristic equation

$$\lambda^2 - (\beta_1 - 2b - \gamma - d)\lambda + (b + d + \alpha)(b + \gamma + \alpha)\left[1 - \frac{\beta_1}{b + \gamma + \alpha} - \frac{\beta_2 \gamma}{(b + d + \alpha)(b + \gamma + \alpha)}\right]$$

$$= \lambda^2 - (\beta_1 - 2b - \gamma - d)\lambda + (b + d + \alpha)(b + \gamma + \alpha)[1 - R_0] = 0.$$

Thus

$$\lambda_{1,2} = (\beta_1 - 2b - \gamma - d) \pm \sqrt{(\beta_1 - 2b - \gamma - d)^2 - 4(b + d + \alpha)(b + \gamma + \alpha)(1 - R_0)}$$

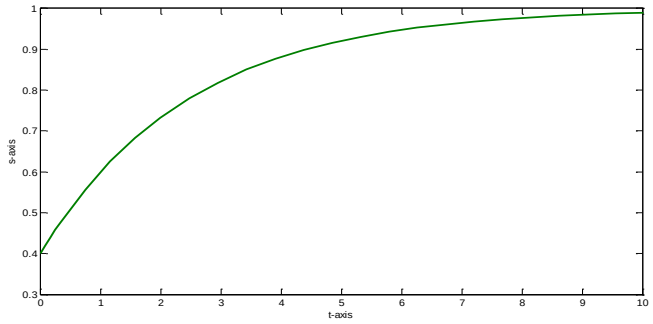
One of the two eigenvalues is positive, which gives that the disease free equilibrium is not stable. The ω−limit set of the system (8) on $\Omega - \Gamma$ is reduced to the endemic equilibrium point. Because of the local stability of the endemic equilibrium point for R_0>1, the endemic equilibrium point is globally asymptotically stable.

Numerical Simulations

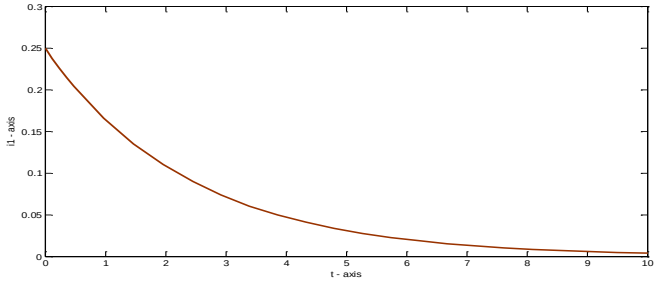
Numerical Simulations of Model

In this section, we present numerical simulations to illustrate the various theoretical results previously obtained. Thus, we draw first the curves of system (2) for parameters verifying R_0less than 1, and we shall do the same for parameters verifying R_0 upper to 1.

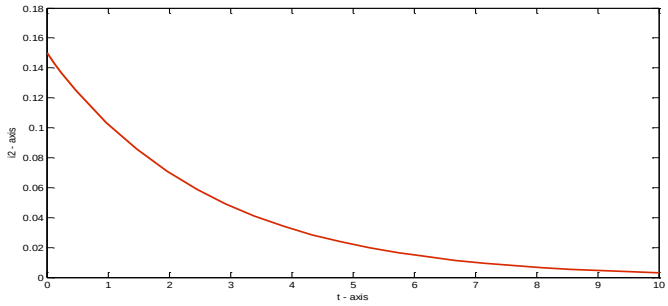
We take the value of the parameters as: $\beta_1 = 0.0001$, $\beta_2 = 0.0015$, $\gamma = 0.02$, b = 0.4, d = 0.015and α=0.0000003 which corresponds to R_0 = 0.0004102. We have theoretically proved that, in this case, R_0< 1, the disease free equilibrium is globally asymptotically stable. From figure (1), we see that the curves of the infected i_1 and i_2 towards zero. Thus the disease disappears in the host population.



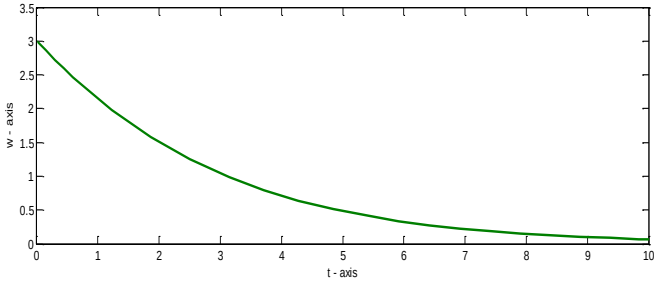
a) Curve of s



b) Curve of i_1



c) Curve of i_2



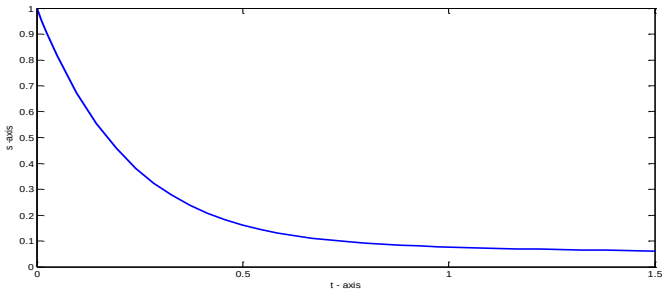
d) Curve of w

Figure 1: The curves of the system (2) for different initial conditions when $R_0<1$

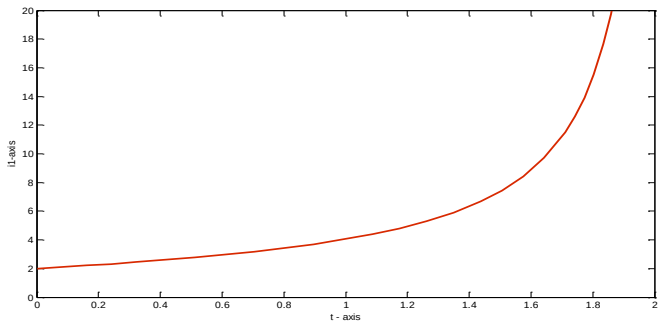
In the second case, we take the value of the parameters as:$\beta_1 = 0.3$, $\beta_2 = 0.8$,

$\gamma = 0.5$, b = 0.4, d = 0.1 and $\alpha = 0.00003$ which corresponds to $R_0 = 1.222$.

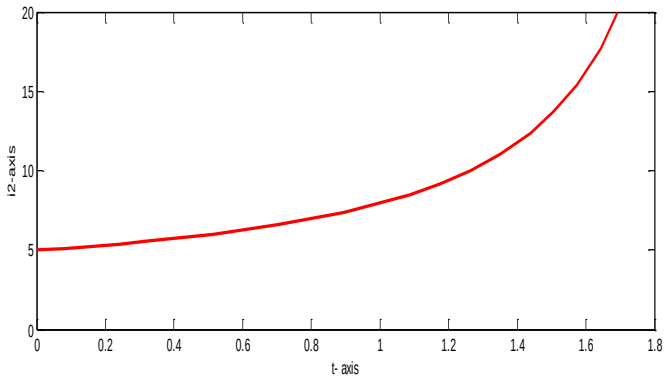
We have theoretically proved that, in this case, $R_0 > 1$ the endemic equilibrium is locally asymptotically stable. From this figure (2), we see that the curves of the infected i_1 and i_2 converge to positive and finite limit, which is the endemic equilibrium. Therefore, the disease will persist in the host population irrespective of the initial conditions. It is thus important to reduce the reproduction number to below unit in order to control the epidemic.



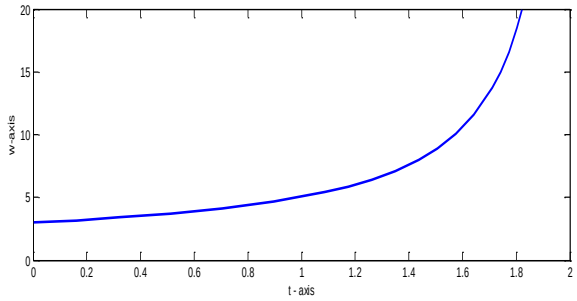
a) Curve of s



b) Curve of i_1^*



c) Curve of i_2^*



d) Curve of w^*

Figure 2: The curves of the system (2) for different initial conditions when $R_0>1$

DISCUSSION AND CONCLUSION

DISCUSSION

Mathematical modeling of the epidemic dynamics is an important method of studying the spread of infectious disease qualitatively and quantitatively. It is based on the specific property of population growth, the spread rules of infectious diseases, and the related social factors, etc. To construct a mathematical model which reflects the dynamical properties of infectious diseases and to analyze the dynamical behavior of the disease it is of paramount important to understand the biology of the infectious disease.

The main study of this thesis is to develop a mathematical model which describes the dynamics of the transmission of infectious disease.

The SIR models are well known in the dynamic of infectious population. We consider two stages of infected population. The population of size N is divided into subclasses of individuals, who are susceptible, infected into the first stage of the disease and infected into the second stage, and recover with sizes denoted by S, I_1, I_2 and R. In the models there are births and deaths, so that the total population size is not constant.

There are two equilibrium points are exist in the feasible region, the disease-free equilibrium point and the unique endemic equilibrium point.

The basic reproduction number depends on the rate of contact between individuals, the probability of transmission given contact, and the time for which an infected remains able to transmit the infection. These components are all the subject of disease control methods: isolating those with the infection from the rest of the community, for example in hospital or at home, reduces their rate of contact with others; hygiene measures reduce either the contact rate or the probability of transmission given contact.

Our result show that if the basic reproduction number R_0 is below one the disease free equilibrium point is locally and globally stable in the feasible region, so that the disease dies out. If the basic reproduction number R_0 is greater than one a unique endemic equilibrium point is locally asymptotically stable and the disease free equilibrium point is unstable in the interior of the feasible region and the disease will persist at the endemic equilibrium point if it is initially present.

We applied numerical simulations to illustrate the various theoretical results obtained. Thus, from graph of system (2) for parameters verifying R_0 less than 1 and we do the same for parameters verifying R_0 upper to 1.

We have theoretically proved that, in the case, $R_0< 1$, the disease free equilibrium is globally asymptotically stable. From figure1, we see that the curves of the infected i_1 and i_2 towards zero. Thus the disease disappears in the host population.

In the case $R_0>1$ the endemic equilibrium is locally asymptotically stable. From figure2, we see that the curves of the infected i_1 and i_2 converge to positive and finite limit, which is the endemic equilibrium. Therefore, the disease will persist in the host population irrespective of the initial conditions. It is thus important to reduce the reproduction number to below unit in order to control the epidemic.

CONCLUSION

The model SIR is one of the most important epidemiological models. This study work gives a qualitative analysis of the stability of the model with a non-linear incidence. The model allows for two classes of infected stages and with varying total population size. This model was studied theoretically, and it was found that the dynamic behavior of the model can be determined by its basic reproduction number R_0.When the basic reproduction number R_0 is less than one, then the disease free equilibrium is locally and globally asymptotically stable in the feasible region. Using a Lyapunov function, we have proved that global stability of the disease free equilibrium point. The global stability of the disease free equilibrium state implies that for any initial condition, the disease will eventually dies out. If the basic reproduction number R_0 is greater than one a unique endemic equilibrium point is locally asymptotically stable and the disease free equilibrium point is unstable in the interior of the feasible region and the disease will persist at the endemic equilibrium point if it is initially present.

It is thus important to reduce the reproduction number below 1 in order to control the epidemic. Numerical simulations were carried out using theoretical set of parameter to illustrate the analytical results. It would be interesting to generalize the work to study the system with arbitrary n infected stages.

REFERENCES

[1]Fred Brauer 2012, Deterministic Compartmental disease Transmission Models 2:43

[2] Howie Weiss 2010, Mathematical Introduction to Population Dynamics under Construction, 4: 76 - 77

[3]D.Y.Melesse and A. B. Gumel, (2010), Global asymptotic properties of an SEIRS model with multiple infectious stages. Math.Anal.And Appl., 366:202–217.

[4] Zhien Ma, Yicang Zhou, Jianhong Wu., (2009), Modeling and Dynamics of Infectious diseases' 1-3

[5]Neeta J Singh 2006,Epidemiological Models for Mutating Pathogens with Temporary Immunity 1:12-15

[6] Elizabeths. Allman, John A. Rhodes, (2004), Mathematical models in Biology, An Introduction.7: 279- 281.

[7] C. C. McCluskey.A, (2003), model of HIV/AIDS with staged progression and Amelioration. Mathematical Biosciences, 181:1–16.

[8]H. W. Heathcoat, (2000), The Mathematics of infectious diseases SIAM, 42:599–653.

[9]R. M. Anderson and R. M(1991), Infection diseases of Humans. Oxford University Press, London.

[10]C. P. Simon and J. A. Jacquez, (1990), Reproduction Numbers and the stability of equilibrium of SI models for hetere-geneous population. SIAM, 52(2):541–576

[11] M. Y. Li,J. R. Graef, L Wang and J Karsai. Global dynamics of a SEIR with varying total population size, Math. Biosciences 160 (1999) 191-213.