THE DYNAMICAL BEHAVIORS FOR AN EPIDEMIC DISEASE MODEL WITH GENERAL RECOVERY FUNCTION

Hassan F. Ridha[1] and Ahmed A. Muhseen[2,*]

[1]Department of Mathematics, College of Science, Baghdad University, Baghdad-Iraq, E-mail: mathhassanmath@yahoo.com

[2] Ministry of Education, Rusafa\1, Baghdad-Iraq,

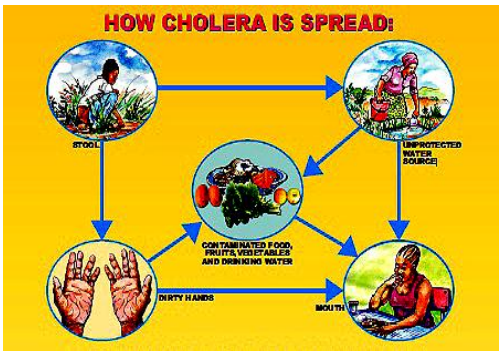
[*] Corresponding Author, E-mail: aamuhseen@gmail.com

ABSTRACT: *In this article, a mathematical model that describes the spread of infected disease in a population is studied. It is assumed that the disease divided the population into two classes: susceptible individuals (S), infectious individuals* (I) *. The existence, uniqueness and boundedness of the solution of the model are discussed. The local and global stability of the model is studied. The backward bifurcation is studied. Finally the global dynamics of the proposed model is studied numerically.*

Keywords: Infected diseases, Basic reproduction number, Bifurcation, Local and Global Stability, Recovery function.

1. INTRODUCTION

The establishment and spread of infectious diseases is a complex phenomenon with many interacting factors, e.g., the environment in which the pathogen and hosts are situated, the population(s) it is exposed to, and the intra- and inter-dynamics of the population it is exposed to. The role of mathematical epidemiology is to model the establishment and spread of pathogens. A predominant method of doing so is to use the notion of abstracting the population into compartments under certain assumptions, which represent their health status with respect to the pathogen in the system. One of the cornerstone works to achieve success in this method was done by Kermack and McKendrick in the early 1927[1]. These models are known as compartmental models in epidemiology or ecology, and serve as a base mathematical framework for understanding the complex dynamics of these systems, which hope to model the main characteristics of the system. Once one is able to model an infectious pathogen with compartmental models, one can predict the various properties of the pathogen spread, for example the prevalence (total number of infected from the epidemic) and the duration of the epidemic. Also, one can understand how different situations may affect the outcome of the epidemic, e.g., what the best technique is for issuing a limited number of vaccines in a given population. Here we show some of the diseases studied by epidemiological models:



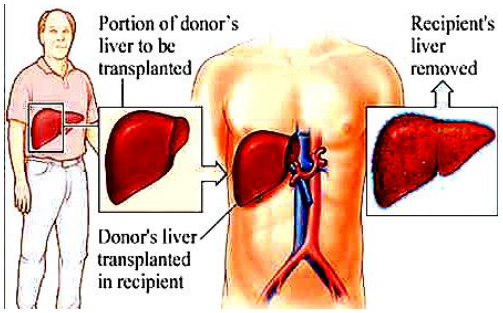


Figure 1: Spread the Cholera and Hepatitis disease

Cholera is an acute intestinal infectious disease caused by bacterium Vibrio Cholera. Recent Cholera outbreaks in Haiti (2010-2011), Nigeria (2010), Kenya (2010), Vietnam (2009), Zimbabwe (2008-2009), and Iraq (2007), etc. The container is leading to a large number of infections and receiving worldwide attention. Then, despite of many clinical and theoretical studies [2-9] and tremendous administrative efforts and interventions, Cholera remains a significant threat to public health in developing countries. In the year 2006 alone, about 240,000 Cholera cases were officially notified to the World Health Organization (WHO). A deep understanding of the disease dynamic would provide important guidelines to the effective prevention and control strategies [10, 11]. Mathematical modeling, simulation and analysis offer a promising way to look into the natural of Cholera dynamics, and many efforts have been devoted to this topic.

Below, we briefly review some representative mathematical models proposed by various authors. For example, Capasso and Paveri-Fontana [12], introduced a simple deterministic model in 1979 to study a Cholera epidemic in the Mediterranean. In 2001, Codeco [13], extended the model of Capasso and Paveri-Fontana. He added an equation for the dynamics of the susceptible population. In 2009, Richard I. Joh et al. considered the dynamic of infectious disease for which the primary mode of transmission is indirect and mediated by contact with a contaminated reservoir [14]. In [15], Rachal L. Miller et al. formulated a mathematical model to include essential components such as a hyper infectious, a short-lived bacterial state, a separate class for mild human infections, and waning disease immunity. In this paper we proposed and studied a mathematical model of Cholera disease, in which it is assumed that the disease spread by directed contact by nonlinear functional response. We studied

stability analysis of this model is investigated. Also, the many types of bifurcations are discussed.

2. Mathematical Model

Let $S(t)$ and $I(t)$ be the number of the susceptible individuals and infectious individuals at time t respectively. The state equations, which cover this model, can be written as follows:

$$\left.\begin{aligned} \dot{S} &= (1-p)A - \frac{\beta SI}{K+I} - \mu S \\ \dot{I} &= pA + \frac{\beta SI}{K+I} - (\mu+\alpha)I - \frac{rI}{n+I} \end{aligned}\right\} \quad (1)$$

Note that all the parameters of system (1) are assumed to be positive constants and can describe as following: A birth rate in susceptible class with fraction p such that $(0 \le p < 1)$, assumed that the disease transmitted from class S to classes I by contact interaction with infection rate constant β, μ is the natural death rate in each class while the α are the disease related death from I. K the carrying capacity of disease, finally, c is the maximum recovery per unit of time t, and n is the infected size. Therefore, at any point of time t the total population becomes $N = S(t) + I(t)$. Obviously, due to the biological meaning of the variables $S(t)$ and $I(t)$, system (1) has the domain $\Re_+^2 = \left\{(S, I \in \Re_+^2, S \ge 0, I \ge 0\right\}$, which is positive invariant for system (1). Clearly, the interaction functions on the right hand side of system (1) are continuously differentiable. In fact they are Liptschizan function on $\Re_+^2$. Therefore the solution of system (1) exists and unique. Further, all solutions are uniformly bounded:

Theorem (1): Any solutions of system (1), which are initiate in $\Re_+^2$ if exists, are positive and bounded.

Proof: Let $\left(S(t), I(t)\right)$ be any solution of system (1) with non-negative initial condition $\left(S(0), I(0)\right)$, since $N(t) = S(t) + I(t)$, then:

$$\dot{N} = \dot{S} + \dot{I}$$

this gives

$$\dot{N} = A - \mu(S+I) - \alpha I - \frac{rI}{n+I}$$

$$\dot{N} \le A - \mu N$$

Now, by solving the above linear differential equation, we get that the total population is asymptotically constant by:

$$N(t) \le \frac{A}{\mu}$$

Hence all the solution of system (1) that initiate in $\Re_+^2$, are confined in the region:

$$\tau = \left\{(S, I) \in \Re_+^2 : N \le \frac{A}{\mu}\right\} \quad \blacksquare$$

3. The Basic Reproduction Number

For all infectious disease, the basic reproduction number, sometimes called basic reproductive ratio, is one of the most useful threshold parameters that characterizes mathematical problems concerning infectious disease. This metric is useful because it helps us to determine whether an infectious disease will spread through a population, we will calculate the basic reproduction number.

It easy to see that this system always has a disease free equilibrium point (the absence of infection, that is, $I = 0$). According to theorem 2 in [16], the basic reproduction number of system (1) is:

$$\Re_\circ = \frac{n\beta A}{\mu K[n(\mu+\alpha)+r]} \quad (2)$$

4. Equilibria Points

In this section, we shall discuss the existence of each possible equilibrium points of system (1).

Now, this system has two biologically feasible points, denoted by $E_i = (S_i, I_i)$, $i = 0,1$, are discussed in following:

1) When $I = 0$ and $\Re_\circ < 1$, then system (1) has a disease free equilibrium point and denoted by $E_0 = (S_0, 0)$ where:

$$S_0 = \frac{A}{\mu} \quad (3)$$

2) When $I \ne 0$, and $\Re_\circ > 1$, then system (1) has endemic equilibrium point and denoted by $E_1(S_1, I_1)$ where S_1 and I_1 represent the positive solution of following set of equations:

$$\left.\begin{aligned} (1-p)A - \frac{\beta SI}{K+I} - \mu S &= 0 \\ pA + \frac{\beta SI}{K+I} - (\mu+\alpha)I - \frac{rI}{n+I} &= 0 \end{aligned}\right\} \quad (4)$$

Obviously, from 1st equation of (4) we get:

$$S_1 = \frac{(1-p)(K+I)A}{\beta I + \mu(K+I)} \quad (5)$$

Substituting S_1 in the 2nd equation of (4) we get:

$$\Omega_1 I_1^4 + \Omega_2 I_1^3 + \Omega_3 I_1^2 + \Omega_4 I_1 + \Omega_5 = 0 \quad (6)$$

Here:

$$\Omega_1 = -\left[\beta + \mu(\mu+\alpha)\right] < 0$$

$$\Omega_2 = pA(\beta+\mu) + \beta A(1-p) - (\mu+\alpha)(\beta K + \beta n + 2\mu K + \mu n) - r(\beta+\mu)$$

$$\Omega_3 = p\beta A(K+n) + pA\mu(2K+n) + \beta A(1-p)(K+n) - K(\mu+\alpha)(\beta n + \mu K + 2\mu n) - rK(\beta+2\mu)$$

$$\Omega_4 = pAK(\beta n + \mu K + 2\mu n + n(1-p) - \mu K^2\left(n(\mu+\alpha)+r\right)$$

$$\Omega_5 = pA\mu n K^2 > 0$$

Clearly, equation (6) by Descartes rule [17] has one positive root given by I_1 and then the equilibrium point (E_1) exists uniquely in Int. $\Re_+^2$ if and only if we have one from the following four Cases:

Case (1): If the following conditions are hold:

$$\Omega_2, \Omega_3 \text{ and } \Omega_4 > 0 \quad (7a)$$

Case (2): If the following conditions are hold:

$$\Omega_2, \Omega_3 \text{ and } \Omega_4 < 0 \quad (7b)$$

Case (3): If the following conditions are hold:

$$\Omega_2, \Omega_3 < 0 \text{ and } \Omega_4 > 0 \quad (7c)$$

Case (4): If the following conditions are hold:

$$\Omega_2 < 0, \text{ and } \Omega_3, \Omega_4 > 0 \quad (7d)$$

5. Local Settled Analysis

In this part, the local stability analysis of the each equilibria $E_i, i = 0,1$ of system (1) studied.

Theorem (2): The asymptomatic and symptomatic infectious free equilibrium point $E_0 = (S_0 = \frac{A}{\mu}, 0)$ of System (1) is locally settled when $\Re_\circ < 1$ and then the following conditions are satisfied, but E_0 unsettled when $\Re_\circ > 1$:

$$\beta n S_0 < K[n(\mu+\alpha)+r] \quad (8)$$

Proof: The Jacobian matrix of system (1) at (E_0) that denoted by $J(E_0)$ and can be written:

$$J(E_0) = [a_{ij}]_{2\times 2},$$

where:

$$a_{11} = -\mu \; ; a_{12} = -\frac{\beta S_0}{K} ; a_{21} = 0 ;$$

$$a_{22} = \frac{\beta S_0}{K} - (\mu+\alpha) - \frac{r}{n}$$

Then the characteristic equation of the Jacobian matrix $J(E_0)$ is given by :

$$\lambda^2 + H\lambda + B = 0 \quad (9)$$

Where:

$$\left.\begin{aligned} H &= -\left[\frac{\beta S_0}{K} - \mu - (\mu+\alpha) - \frac{r}{n}\right] > 0 \\ B &= -\mu\left[\frac{\beta S_0}{K} - (\mu+\alpha) - \frac{r}{n}\right] > 0 \end{aligned}\right\} \quad (10)$$

Consequently equation (9) have the following roots (eigenvalues) of $J(E_0)$:

$$\lambda_{S,I} = \frac{-H}{2} \pm \frac{1}{2}\left(\frac{\beta S_0}{K} - \left(\frac{r}{n} + \alpha\right)\right) < 0$$

(11)

Where λ_S and λ_I describe the dynamics in the *S* and *I* direction respectively. Clearly λ_S and λ_I are negative depending on condition (8).

Therefore, E_0 is asymptotically settled equilibrium point provided that condition (8). ■

Theorem (3): The endemic equilibrium point $E_1 = (S_1, I_1)$ of System (1) is local asymptotic settled when $\Re_\circ > 1$ and then the following conditions are satisfied:

$$(K+I_1)^2[\beta I_1(n+I_1)^2 + (2\mu+\alpha)(K+I_1)(n+I_1)^2 + rn(K+I_1)] > \beta S_1 K(K+I_1)(n+I_1)^2 \quad (12)$$

$$\beta I_1(K+I_1)[(\mu+\alpha)(n+I_1)^2 + rn] + \mu(n+I_1)^2[(\mu+\alpha)(K+I_1)^2 + rn] > \mu\beta S_1 K(n+I_1)^2 \quad \text{..... (13)}$$

Proof: The Jacobian matrix of System (1) at $E_1 = (S_1, I_1)$ written by:

$J(E_1) = [b_{ij}]_{2\times 2}$, where:

$$b_{11} = -\left(\frac{\beta I_1}{K+I_1} + \mu\right) ; b_{12} = -\frac{\beta K S_1}{(K+I_1)^2} ; \quad b_{21} = \frac{\beta I_1}{K+I_1} ;$$

$$b_{22} = \frac{\beta K S_1}{(K+I_1)^2} - (\mu+\alpha) - \frac{rn}{(n+I_1)^2}$$

Then the characteristic equation of the Jacobian matrix $J(E_1)$ is given by :

$$\lambda^2 + H_1\lambda + B_1 = 0 \quad (14)$$

Where:

$$\left.\begin{aligned} H_1 &= -\left[\frac{\beta S_1 K}{(K+I_1)^2} - \frac{\beta I_1(n+I_1)^2 + \tilde{B} + rn(K+I_1)}{(K+I_1)(n+I_1)^2}\right] > 0 \\ B_1 &= \left[\frac{\beta I_1(K+I_1)[(\mu+\alpha)(n+I_1)^2 + rn] + \mu(n+I_1)^2[B+rn] - Q}{(K+I_1)^2(n+I_1)^2}\right] > 0 \end{aligned}\right\} \quad \text{.....(15)}$$

Her $\tilde{B} = (2\mu+\alpha)(K+I_1)(n+I_1)^2$

$$B = (\mu+\alpha)(K+I_1)^2 \; ; \; Q = \mu\beta S_1 K(n+I_1)^2$$

Consequently equation (14) have the following roots (eigenvalues) of $J(E_1)$:

$$\lambda_{S,I} = \frac{-H_1}{2} \pm \frac{1}{2}\sqrt{H_1^2 - 4B_1} < 0 \quad (16)$$

Where λ_S and λ_I describe the dynamics in the *S* and *I* direction respectively. Clearly λ_S and λ_I are negative depending on conditions (12-13). Therefore, E_1 is asymptotically settled equilibrium point provided that condition (12-13) hence the proof is complete. ■

6. Global Settled Analysis

In this part, the global analysis of the all points $E_i, i = 0,1$ of system (1) studied.

Theorem (4): If the disease free equilibrium point E_0 of System (1) is local settled. Then it is global settled if satisfy the following condition:

$$S_0(n+I)[pA(K+I) + \beta SI] < S(K+I)[(\mu+\alpha)(n+I)+r]I$$

..... (17)

Proof: Consider the following positive definite function:

$$V_1 = \left(S - S_0 - S_0 \ln \frac{S}{S_0} \right) + I$$

Clearly, $V_1 : R_+^2 \to R$ is a continuo differentiable function such that $V_1(S_0, 0) = 0$, and $V_1(S, I) > 0, \forall (S, I) \neq (S_0, 0)$. Further we have:

$$\dot{V}_1 = \left(\frac{S - S_0}{S} \right) \dot{S} + \dot{I}$$

By simplifying this equation we get:

$$\dot{V}_1 = \frac{S_0[pA(K+I) + \beta SI]}{S(K+I)} - \frac{[(\mu+\alpha)(n+I) + r]I}{(n+I)}$$

Obviously, $\dot{V}_1 < 0$, for any initial points and then V_1 is a Lyapunov function provided that condition (17) hold. Thus E_0 is global settled. ■

Theorem (5): If the endemic point E_1 of System (1) is local settled. Then it is global settled provided that:

$$\beta SK(n+I)(n+I_1) < [(\mu+\alpha)(n+I)(n+I_1) + rn](K+I)(K+I_1) \quad (18a)$$

$$\left[\frac{-\beta}{K+I_1} \left(\frac{SK}{K+I} - I_1 \right) \right]^2 < 4 \left[\mu + \frac{\beta I_1}{K+I_1} \right] \cdot \left[(\mu+\alpha) + \frac{rn}{(n+I)(n+I_1)} - \frac{\beta SK}{(K+I)(K+I_1)} \right] \quad \text{…….. (18b)}$$

Proof: Consider the following positive definite function:

$$V_2 = \frac{(S - S_1)^2}{2} + \frac{(I - I_1)^2}{2}$$

Clearly, $V_2 : R_+^2 \to R$ is a continuo differentiable function whrer $V_2(S_1, I_1) = 0$ and $V_2(S, I) > 0$, $\forall (S, I) \neq (S_1, I_1)$. Further, we have:

$$\dot{V}_2 = (S - S_1)\dot{S} + (I - I_1)\dot{I}$$

Now, simplifying this equation we get:

$$\dot{V}_2 = -p_{11}(S - S_1)^2 + p_{12}(S - S_1)(I - I_1) - p_{22}(I - I_1)^2$$

With:

$$p_{11} = \mu + \frac{\beta I_1}{K + I_1} \; ; \; p_{12} = \frac{-\beta I_1}{K_1 + I_1} \left(\frac{SK}{K+I} - I \right);$$

$$p_{22} = (\mu+\alpha) + \frac{rn}{(n+I)(n+I_1)} - \frac{\beta SK}{(K+I)(K+I_1)} ;$$

Therefore, according to the conditions (18a)-(18b) we obtain that:

$$\dot{V}_2 \leq -\left[\sqrt{\frac{p_{11}}{3}} (S - S_1) - \sqrt{\frac{p_{22}}{2}} (I_A - I_{A1}) \right]^2$$

Clearly, $\dot{V}_2 < 0$, and then V_2 is a Lyapunov function provided that the given conditions(18a)-(18b) hold. Therefore, (E_1) is globally asymptotically settled. ■

7. The Hpof bifurcation analysis

In this part, the periodic dynamic due to changing the value of one parameter is studied in the following theorem.

Theorem (6): The system (1) has a Hopf-bifurcation around the endemic equilibrium point E_1 satisfy the following condition:

$$\mu^* = \left[\frac{\beta S_1 K - (K + I_1)[\beta I_1 + \alpha(K + I_1) + rn(K + I_1)]}{2(K + I_1)^2} \right] \quad (19)$$

Clearly μ^* is positive provided that:

$$\beta S_1 K > (K + I_1)[\beta I_1 + \alpha(K + I_1) + rn(K + I_1)] \quad (20)$$

Proof: Consider the Jacobian matrix of system (1) at E_1 with the characteristic equation can be written in the following form:

$$J(E_1) = \begin{bmatrix} -\left(\frac{\beta I_1}{K + I_1} + \mu \right) & -\frac{\beta K S_1}{(K + I_1)^2} \\ \frac{\beta I_1}{K + I_1} & \frac{\beta K S_1}{(K + I_1)^2} - (\mu + \alpha) - \frac{rn}{(n + I_1)^2} \end{bmatrix}$$

Then

$$\lambda^2 + T\lambda + D = 0 \quad (21)$$

Clearly, the eigenvalues of above equation can be written:

$$\lambda = \frac{1}{2} \left[-T \pm \sqrt{T^2 - 4D} \right]$$

here:

$$T(trace) = \frac{\beta S_1 K}{(K + I_1)^2} - \frac{\beta I_1 (n + I_1)^2 + H + rn(K + I_1)}{(K + I_1)(n + I_1)^2}$$

$$D(\det) = \frac{\tilde{H} + \mu(n + I_1)^2[(\mu + \alpha)(K + I_1)^2 + rn] - \mu\beta S_1 K(n + I_1)^2}{(K + I_1)^2 (n + I_1)^2}$$

Her $H = (2\mu + \alpha)(K + I_1)(n + I_1)^2$

$$\tilde{H} = \beta I_1 (K + I_1)[(\mu + \alpha)(n + I_1)^2 + rn]$$

Clearly, system (1) dose not a Hopf-bifurcation around the endemic equilibrium point E_1 if trace of eigenvalues $T \neq 0$.

Now, the necessary and sufficient conditions for a Hopf-bifurcation to occur we need to find a parameter $(say\ \mu^*)$ satisfy that:

$$T(\mu^*) = 0 \quad (22a)$$

$$\left. \frac{dT}{d\mu} \right|_{\mu = \mu^*} \neq 0 \quad (22b)$$

Then the system (1) has two complex conjugate eigenvalues. Clearly, the 1st condition (22a) for the Hopf-bifurcation is satisfied at $\mu = \mu^*$ if and only if provided the conditions (19-20).

Let as now check the 2nd condition (22b) in the following:

$$\left. \frac{dT}{d\mu} \right|_{\mu = \mu^*} = -2 \neq 0$$

Hence, the system (1) has a Hopf-bifurcation [18], a round the endemic equilibrium point E_1 at the parameter $\mu = \mu^*$. ■

8. Backward Bifurcation

In this part, the bifurcation analysis of system (1) is studied. In fact, across $\Re_\circ = 1$, the disease free equilibrium point changes its stability. In the following, we consider system (1) and investigate the nature of the bifurcation involving the disease free equilibrium (E_0) for $\Re_\circ = 1$. Now, we look for conditions on the parameter values that cause a forward or a backward bifurcation to occur. In order to do that, we will make use of the result summarized below, which has been obtained in Castillo-Chavez and Song [19].

Theorem 7: We show that system (1) may exhibit a backward or forward bifurcation when $\Re_\circ = 1$. We consider the disease free equilibrium point (E_0) and observe that condition $\Re_\circ = 1$ can be seen, in terms of the parameter $\beta^* = \frac{K}{S_0}(\mu+\alpha+\frac{r}{n})$. Now we can, introducing $S = x_1$, $I = x_2$ then system (1) becomes:

$$\left.\begin{aligned} \dot{x}_1 &= (1-p)A - \frac{\beta x_1 x_2}{K+x_2} - \mu x_1 \\ \dot{x}_2 &= pA + \frac{\beta x_1 x_2}{K+x_2} - (\mu+\alpha)x_2 - \frac{r x_2}{n+x_2} \end{aligned}\right\} \quad (23)$$

Then, from the jacobian matrix:

$$J(E_0) = \begin{bmatrix} -\mu & -\frac{\beta^* S_0}{K} \\ 0 & \frac{\beta^* S_0}{K} - (\mu+\alpha) - \frac{r}{n} \end{bmatrix},$$

We get one of eigenvalues of matrix $J(E_0, \beta^*)$ is zero and the other are real and negative. Therefore, we can use the center manifold theory. Hence, when $\beta = \beta^*$ (or equivalent, where $\Re_\circ = 1$), the disease free point E_0 is non-hyperbolic point: the assumption (A_1) of Castillo-Chavez and Song [19], is then verified.

Now, we denoted by $V = [v_1, v_2]^T$, a right eigenvector associated with the zero eigenvalue. It is found by:

$$\begin{bmatrix} -\mu & -\frac{\beta S_0}{K} \\ 0 & \frac{\beta S_0}{K} - (\mu+\alpha+\frac{r}{n}) \end{bmatrix} \cdot \begin{bmatrix} v_1 \\ v_2 \end{bmatrix} = 0$$

Thus, we can get:

$$-\mu v_1 + \frac{\beta S_0}{K} v_2 = 0$$

$$\left[\frac{\beta S_0}{K} - (\mu+\alpha+\frac{r}{n})\right] v_2 = 0$$

Therefore, the right eigenvector is $V = \left[\frac{\beta S_0}{\mu K}, 1\right]^T$.

Now, we find the left eigenvector $W = [w_1, w_2]^T$ satisfying V.W=1 is given by:

$$W = \left[1, \frac{\beta S_0}{(\beta S_0 - K(\mu+\alpha+\frac{r}{n}))}\right]^T$$

Evaluating the partial derivatives at E_0, we obtain:

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_2} = \frac{-\beta K}{(K+x_2)^2} \; ; \; \frac{\partial^2 f_1}{\partial x_2^2} = \frac{2\beta K x_1}{(K+x_2)^3} \; ;$$

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \frac{\beta K}{(K+x_2)^2} \; ; \; \frac{\partial^2 f_2}{\partial x_2^2} = 2\left[\frac{rn}{n+x_2} - \frac{\beta K x_1}{(K+x_2)^3}\right]$$

$$\frac{\partial^2 f_1}{\partial x_1 \partial \beta} = \frac{-x_2}{K+x_2} \; ; \; \frac{\partial^2 f_1}{\partial x_2 \partial \beta} = \frac{-\beta x_1}{(K+x_2)^2} \; ;$$

$$\frac{\partial^2 f_2}{\partial x_1 \partial \beta} = \frac{x_2}{K+x_2} \; ; \; \frac{\partial^2 f_2}{\partial x_2 \partial \beta} = \frac{K x_1}{(K+x_2)^2}$$

And all other partial derivatives are equal to zero. Clearly, we can find calculate the coefficients a and b defined in Castillo-Chavez and Song [19].

$$a = \sum_{k,i,j=1}^{2} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(P_0, \beta^*) \; ; \quad b = \sum_{k,i=1}^{2} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(P_0, \beta^*)$$

Taking into account the system (1) and considering in a and b only the nonzero derivatives for the terms $\frac{\partial^2 f_k}{\partial x_i \partial x_j}(P_0, \beta^*)$ and $\frac{\partial^2 f_k}{\partial x_i \partial \beta}(P_0, \beta^*)$, it follows that:

$$a = \frac{2\beta S_0}{\mu K}\left(\frac{-\beta K}{(K+x_2)^2}\right)\left(\frac{\beta S_0}{\beta S_0 - K(\mu+\alpha+\frac{r}{n})}\right)$$

$$+ \frac{\beta S_0}{\mu K}\left(\frac{2\beta K x_1}{(K+x_2)^3}\right)\left(\frac{\beta S_0}{\beta S_0 - K(\mu+\alpha+\frac{r}{n})}\right)^2$$

$$+ \frac{2\beta K}{(K+x_2)^2}\left(\frac{\beta S_0}{\beta S_0 - K(\mu+\alpha+\frac{r}{n})}\right)$$

$$+ 2\left(\frac{\beta S_0}{\beta S_0 - K(\mu+\alpha+\frac{r}{n})}\right)^2\left(\frac{rn}{n+x_2} - \frac{\beta K x_1}{(K+x_2)^3}\right)$$

$$b = \frac{\beta S_0}{\mu K}\left(\frac{-x_2}{K+x_2}\right) + \frac{\beta S_0}{\mu K}\left(\frac{-x_1 K}{(K+x_2)^2}\right)\left(\frac{\beta S_0}{\beta S_0 - K(\mu+\alpha+\frac{r}{n})}\right) + \frac{x_2}{K+x_2} + \frac{K x_1}{(K+x_2)^2}\left(\frac{\beta S_0}{\beta S_0 - K(\mu+\alpha+\frac{r}{n})}\right)$$

Clearly, we need the coefficient b is always positive so that, according to theorem in [19], it clearly, when $a<0$, such a bifurcation is forward, but if $a>0$ the bifurcation is backward. ■

9. Numerical Simulation

In this part, system (1) is solved numerically for different sets of data and different sets of initial conditions, and then the solutions of system (1) are confirm our obtained analytical results. By using (150, 550) and (300, 100) as initial points and the numerical simulations are carried out in the following:

For the disease free E_0, we choose the following data:

$$A=100\ ;\ p=0;\beta=0.001;\ K=2;\quad r=0.5;n=2;\alpha=0.2;\ \Re_\circ=0.9 \qquad (24)$$

Therefore, the disease free E_0 of system (1) is global settled and is identically to (1000, 0) for any time.

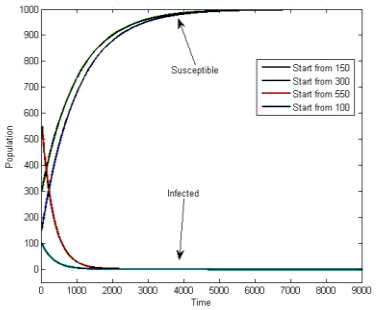


Figure 2: The solutions of system (1) from different initial points for data given in equation (24) which show that E_0 is globally asymptotically settled.

Now, for the data given equation (24) with *p=0.1*. The solutions of system (1) starting from different sets of initial data (150, 550) and (300, 100) are drawn in Figure (3). Then, the endemic equilibrium point E_1 of system (1) is globally asymptotically settled and is identically to (891, 34) for any time.

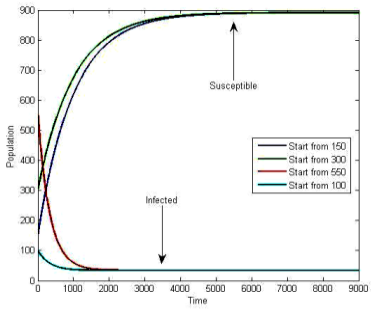


Figure 3: The solutions of system (1) from different initial points for data in equation (24) with p=0.1, which show that E_1 is globally asymptotically settled.

Now, we choose values for the fraction rate $p=0.1, 0.3, 0.5$ respectively, with parameters fixed as given in (24), we get the solutions of system (1) still approaches to endemic equilibria point but the number of symptomatic infectious individuals increase while the number of the asymptomatic susceptible individuals decreases.

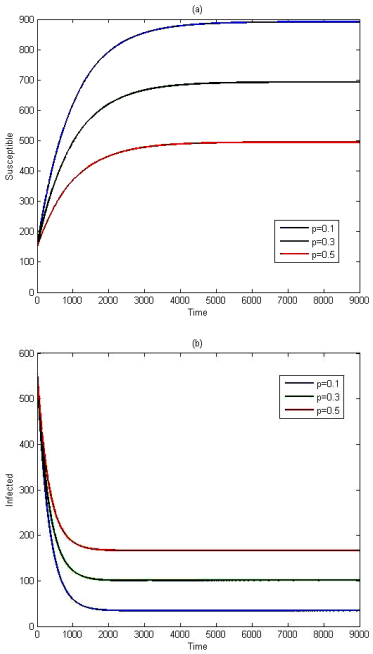


Figure 4: The solutions of system (1). (a) For S , (b) For I .

We fixed all parameters in equation (24) but we change infection rate value $\beta=0.01, 0.2, 0.5$ respectively, we get the solutions of system (1) still approaches to endemic point but the number of susceptible individuals decrease while the number of the symptomatic infectious increases.

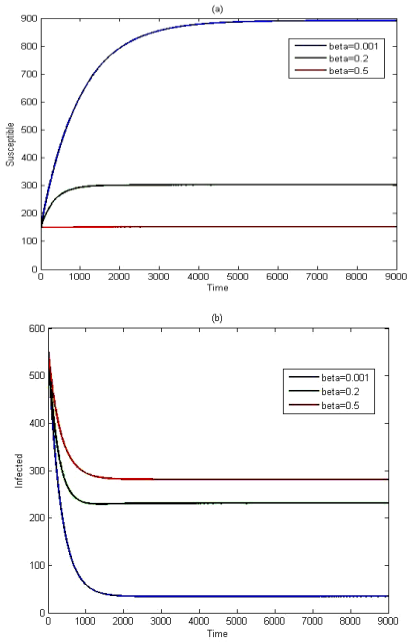


Figure 5: The solutions of system (1). (a) For S , (b) For I .

Clearly, we present the effect of recovery rate that is by change value for $r = 0.5,1,5$ respectively, with parameters fixed as given in (24), we get the solutions of system (1) still approaches to endemic point but the number of asymptomatic infected individuals decreases while the susceptible individuals is smoothly increases.

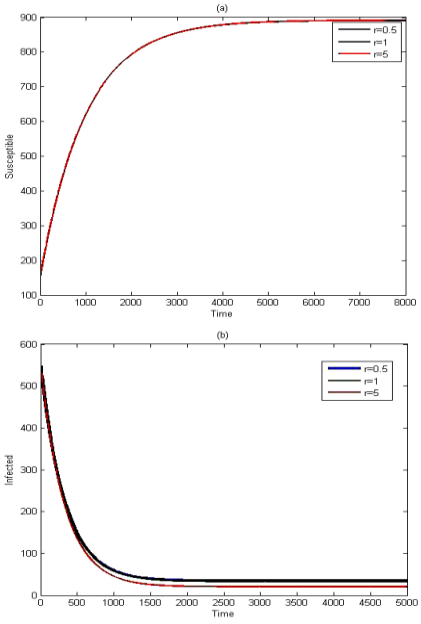


Figure 6: The solutions of system (1). (a) For S , (b) For I .

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