On the Dynamical Behaviors of a Cholera Model with Holling Type II Functional Response

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Abstract

In this paper a mathematical model that describes the flow of Cholera disease in a population is proposed and studied. It is assumed that the disease divided the population into five classes: susceptible individuals (*S*), asymptomatic infectious individuals (I_A), symptomatic infectious individuals (I_S), removal individuals (*R*) and cholera population *(B)*. The existence, uniqueness and boundedness of the solution of the model are discussed. The local and global stability of the model is studied. Finally the global dynamics of the proposed model is studied numerically.

Keywords**:** Cholera model, Basic reproduction number, Holling Type II, Local and Global Stability.

1. Introduction

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 [1-8] 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 [9, 10]. 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 [11], introduced a simple deterministic model in 1979 to study a Cholera epidemic in the Mediterranean. In 2001, Codeco [12], extended the model of Capasso and Paveri-Fontana. He added an equation for the dynamics of the susceptible population. In [13], Pascual et al.

Generalized Codeco model by including a fourth equation for the volume of water in which the formative live following Codeco [12]. 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 hyperinfectious, 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 transmitted by contact by Holling Type II functional response. The local as well as global stability analysis of this model is investigated.

2. Mathematical Model

Let $S(t), I_A(t), I_B(t)$ and $R(t)$ be the number of the susceptible individuals, asymptomatic infectious individuals, symptomatic infectious individuals and removal individuals from infected classes at time *t* respectively. Let $B(t)$ be the cholera population at time *t* with grows logistically. The state equations, which cover this model, can be written as follows:

$$\left.\begin{aligned}
\dot{S} &= \theta - \frac{\beta_1 SB}{K_1 + B} - dS \\
\dot{I}_A &= \frac{\rho\beta_1 SB}{K_1 + B} - (\gamma_A + d)I_A \\
\dot{I}_S &= \frac{(1-\rho)\beta_1 SB}{K_1 + B} - (\gamma_S + d + \mu)I_S \\
\dot{R} &= \gamma_A I_A + \gamma_S I_S - dR \\
\dot{B} &= rB\left(1 - \frac{B}{K_2}\right) - \eta B + \zeta_1 I_A + \zeta_2 I_S
\end{aligned}\right\} \quad \text{..... (1)}$$

Note that all the parameters of system (1) are assumed to be positive constants and can describe as following: θ birth rate in susceptible class, assumed that the disease transmitted from class *S* to classes I_A and I_S by contact according to Holling types II interaction between *S* class and *B* class with infection rate constant β_1 with fraction ρ such that $(0 \le \rho \le 1)$, d is the natural death rate in each class while the μ, η are the disease related death from I_S and B respectively. γ_A, γ_S represents the recovery rate constant. r and K_2 are respectively, the intrinsic growth rate and carrying capacity of cholera population, finally, ζ_1 and ζ_2 are the new infected members arriving into the cholera population in unit time from I_A and I_S classes. Therefore, at any point of time *t* the total number of population becomes

$$N = S(t) + I_A(t) + I_S(t) + R(t) + B(t).$$

Obviously, due to the biological meaning of the variables $S(t), I_A(t), I_S(t), R(t)$ and $B(t)$, system (1) has the domain

$$\Re_+^5 = \{(S, I_A, I_S, R, B \in \Re_+^5, S \ge 0, I_A \ge 0,$$

$I_S \ge 0, R \ge 0, B \ge 0\}$, 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 $\Re_+^5$. Therefore the solution of system (1) exists and unique. Further, all solutions are uniformly bounded as shown in the following theorem:

Theorem (1):

All the solutions of system (1), which are initiate in $\Re_+^5$ if exists, are uniformly bounded.

Proof:

Let $(S(t), I_A(t), I_S(t), R(t), B(t))$ be any solution of system (1) with non-negative initial condition $(S(0), I_A(0), I_S(0), R(0), B(0))$, since $N(t) = S(t) + I_A(t) + I_S(t) + R(t) + B(t)$, then:

$$\dot{N} = \dot{S} + \dot{I}_A + \dot{I}_S + \dot{R} + \dot{B}$$

this gives

$$\dot{N} = \theta - dS - (d - \zeta_1)I_A - (d - \zeta_2)I_S - dR - (\eta - r)B - \mu I_S - \frac{rB^2}{K_2}$$

$$\dot{N} \le \theta - mN,$$

where $m = \min.\{d, (d - \zeta_1), (d - \zeta_2), (\eta - r)\}$

$$\dot{N} + mN \le \theta$$

Now, by using Gronwall lemma [16], it obtains that:

$$N(t) \le \frac{\theta}{\mu}\left(1 - e^{-\mu t}\right) + N(0)e^{-\mu t}$$

Therefore, $N(t) \le \frac{\theta}{\mu}$, as $t \to \infty$, hence all the solutions of system (1) that initiate in $\Re_+^5$ are confined in the region:

$$\tau = \left\{(S, I_A, I_S, R, B) \in \Re_+^5 : N \le \frac{\theta}{\mu}\right\}$$

Which is complete the proof. ■

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, $I_A = I_S = B = 0$),

$E_0 = (S_0, 0, 0, 0)$, where $S_0 = \frac{\theta}{d}$. Let

$X=(I_A,I_S,B,S)^T$. Then, we get:

$$\dot{X}=f(x)-v(x)$$

where

$$f(x)=\begin{pmatrix}\dfrac{\rho\beta_1 SB}{K_1+B}\\ \dfrac{(1-\rho)\beta_1 SB}{K_1+B}\\ rB(1-\dfrac{B}{K_2})\\ 0\end{pmatrix}$$

$$v(x)=\begin{pmatrix}(\gamma_A+d)I_A\\ (\gamma_S+d+\mu)I_S\\ \eta B-\zeta_1 I_A-\zeta_2 I_S\\ \dfrac{\beta_1 SB}{K_1+B}+dS-\theta\end{pmatrix}$$

We can obtain:

$$F=\begin{pmatrix}0 & 0 & \dfrac{\rho\beta_1 S_0}{K_1}\\ 0 & 0 & \dfrac{(1-\rho)\beta_1 S_0}{K_1}\\ 0 & 0 & r\end{pmatrix}$$

$$V=\begin{pmatrix}\gamma_A+d & 0 & 0\\ 0 & \gamma_S+d+\mu & 0\\ -\zeta_1 & -\zeta_2 & \eta\end{pmatrix},$$

giving

$$V^{-1}=\begin{pmatrix}\dfrac{1}{\gamma_A+d} & 0 & 0\\ 0 & \dfrac{1}{\gamma_S+d+\mu} & 0\\ \dfrac{\zeta_1}{\eta(\gamma_A+d)} & \dfrac{\zeta_2}{\eta(\gamma_S+d+\mu)} & \dfrac{1}{\eta}\end{pmatrix}$$

FV^{-1} is the next-generation matrix for model (3). It then follows that the spectral radius of matrix FV^{-1} is

$$\rho(FV^{-1})=\frac{r}{\eta}\left(\frac{\rho\zeta_1\beta_1 S_0}{\eta K_1(\gamma_A+d)}\right)\cdot\left(\frac{(1-\rho)\zeta_2\beta_1 S_0}{\eta K_1(\gamma_S+d+\mu)}\right)$$

According to theorem 2 in [17], the basic reproduction number of model (3) is:

$$\Re_\circ=\frac{r\theta\beta_1}{\eta^3 dK_1}\left(\frac{\rho\zeta_1}{\gamma_A+d}\right)\left(\frac{(1-\rho)\zeta_2}{\gamma_S+d+\mu}\right) \quad \text{..... (2)}$$

4. Existence of Equilibrium Points of System (1)

In this section, we shall discuss the existence of all possible equilibrium points of system (1). Now since recovery class R is related with infected classes I_A and I_S only, hence knowing the values of I_A and I_S leads directly to determine the value of R from solving the fifth equation in system (1). In fact, if the $I_i=0, i=A,S$, then R approaches to zero asymptotically. However, if $I_A=I_c$ and $I_S=I_k$ where I_c and I_k are positive constant, then R approaches to:

$$R=\frac{\gamma_A I_c+\gamma_S I_k}{d} \quad \text{..... (3)}$$

Consequently, system (1) can be written as below and then equation (3) can be used to give the value of R.

$$\left.\begin{aligned}\dot{S}&=\theta-\frac{\beta_1 SB}{K_1+B}-dS\\ \dot{I}_A&=\frac{\rho\beta_1 SB}{K_1+B}-(\gamma_A+d)I_A\\ \dot{I}_S&=\frac{(1-\rho)\beta_1 SB}{K_1+B}-(\gamma_S+d+\mu)I_S\\ \dot{B}&=rB\left(1-\frac{B}{K_2}\right)-\eta B+\zeta_1 I_A+\zeta_2 I_S\end{aligned}\right\} \quad \text{..... (4)}$$

Now, system (4) has at most two biologically feasible points, namely $E_i=(S_i,I_{Ai},I_{Si},B_i)$, $i=0,1$. The existence conditions for each of these equilibrium points are discussed in following:

1) If $I_A=0, I_S=0$, $B=0$ and $\Re_\circ<1$, then system (4) has an equilibrium point called disease free equilibrium point and denoted by $E_0=(S_0,0,0,0)$ where:

$$S_0=\frac{\theta}{d} \quad \text{..... (5)}$$

2) If $I_A\neq 0, I_S\neq 0, B\neq 0$, and $\Re_\circ>1$, then system (4) has an equilibrium point called endemic equilibrium point and denoted by

$E_1(S_1, I_{A1}, I_{S1}, B_1)$ where S_1, I_{A1}, I_{S1} and B_1 represent the positive solution of following set of equations:

$$\left.\begin{aligned} &\theta - \frac{\beta_1 SB}{K_1 + B} - dS = 0 \\ &\frac{\rho\beta_1 SB}{K_1 + B} - (\gamma_A + d)I_A = 0 \\ &\frac{(1-\rho)\beta_1 SB}{K_1 + B} - (\gamma_S + d + \mu)I_S = 0 \\ &B\left[r - \frac{Br}{K_2} - \eta\right] + \zeta_1 I_A + \zeta_2 I_S = 0 \end{aligned}\right\} \quad \text{..... (6)}$$

Obviously, from 1st, 2nd and 3rd equations of (6) we get:

$$S_1 = \frac{\theta(K_1 + B_1)}{G} \quad \text{.....(7a)}$$

$$I_{A1} = \frac{\rho\theta\beta_1(K_1 + B_1)B_1}{(K_1 + B_1)(\gamma_A + d)G} \quad \text{..... (7b)}$$

$$I_{S1} = \frac{(1-\rho)\beta_1\theta(K_1 + B_1)B_1}{(K_1 + B_1)(\gamma_S + d + \mu)G} \quad \text{.....(7c)}$$

Where $G = \beta_1 B_1 + d(K_1 + B_1)$

Substituting I_{A1} and I_{S1} in the 4th equation of (6) we get:

$$\Omega_1 B_1^4 + \Omega_2 B_1^3 + \Omega_3 B_1^2 + \Omega_4 B_1 = 0 \quad \text{..... (7d)}$$

Here:

$$\Omega_1 = -r(\beta_1 + d)(\gamma_A + d)(\gamma_S + d + \mu) < 0$$

$$\Omega_2 = (\gamma_A + d)(\gamma_S + d + \mu)\cdot \left[rK_2(\beta_1 + d) - (rK_1(\beta_1 + 2d) + \eta K_2(\beta_1 + d))\right]$$

$$\Omega_3 = \theta\beta_1 K_2\left[\rho\zeta_1(\gamma_S + d + \mu) + \zeta_2(1-\rho)(\gamma_A + d)\right] + rK_1K_2(\beta_1 + 2d)\cdot (\gamma_A + d)(\gamma_S + d + \mu) - \left[\eta K_1K_2 \cdot (\beta_1 + 2d)(\gamma_A + d)(\gamma_S + d + \mu) + rK_1^2 d(\gamma_A + \gamma_S + 2d + \mu)\right]$$

$$\Omega_4 = \beta_1\theta K_1K_2\left[\rho\zeta_1(\gamma_S + d + \mu) + \zeta_2(1-\rho)(\gamma_A + d)\right] + dK_1^2K_2(r - \eta)\cdot [\gamma_A + \gamma_S + 2d + \mu]$$

Clearly, equation (7d) by Descartes rule [18] has a unique positive root given by B_1 and then the equilibrium point (E_1) exists uniquely in Int. $\Re_+^4$ if and only if the $\Omega_4 > 0$ (positive) then we have the following three Cases:

Case (1):

If the following conditions are hold:

$$\left.\begin{aligned} &\Omega_2 > 0 \\ &\Omega_3 > 0 \end{aligned}\right\} \quad \text{.....(8a)}$$

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

$$\left.\begin{aligned} &\Omega_2 < 0 \\ &\Omega_3 < 0 \end{aligned}\right\} \quad \text{..... (8b)}$$

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

$$\left.\begin{aligned} &\Omega_2 < 0 \\ &\Omega_3 > 0 \end{aligned}\right\} \quad \text{.....(8c)}$$

5. Local Stability Analysis of System (4)

In this section, the local stability analysis of the each equilibrium points $E_i, i = 0,1$ of system (4) studied as shown in the following theorems.

Theorem (2):

The asymptomatic and symptomatic infectious free equilibrium point $E_0 = (S_0, 0, 0, 0)$ of System (4) is locally asymptotically stable when $\Re_\circ < 1$ and then the following conditions are satisfied, but E_0 unstable when $\Re_\circ > 1$:

$$d > \max.\{\zeta_1 - \gamma_A, \zeta_2 - (\gamma_S + \mu)\} \quad \text{.....(9a)}$$

$$\eta K_1 > 2\beta_1 K_1 S_0 + rK_1 \quad \text{..... (9b)}$$

Proof:

The Jacobian matrix of system (4) at (E_0) that denoted by $J(E_0)$ and can be written:

$$J(E_0) = [a_{ij}]_{4\times 4},$$

where:

$$a_{11} = -d \;;\quad a_{14} = -\frac{\beta_1 S_0}{K_1};\quad a_{22} = -(\gamma_A + d);$$

$$a_{24} = \frac{\rho\beta_1 S_0}{K_1};\qquad a_{33} = -(\gamma_S + d + \mu);$$

$$a_{34} = \frac{(1-\rho)\beta_1 S_0}{K_1};\qquad a_{42} = \zeta_1;\qquad a_{43} = \zeta_2;$$

$a_{44} = r - \eta$ and zero otherwise.

Now, according to Gershgorin theorem [19] if the following condition hold:

$$|a_{ii}| > \sum_{\substack{i=1 \\ i\neq j}}^{4} |a_{ij}|$$

Then all eigenvalues of $J(E_0)$ exists in the region:

$$\Lambda = \bigcup \left\{ U^* \in C : |U^* - a_{ii}| < \sum_{\substack{i=j \\ i\neq j}}^{4} |a_{ij}| \right\}$$

Therefore, according to the given conditions (9a), (9b) all the eigenvalues of $J(E_0)$ exists in the left half plane and hence, (E_0) is locally asymptotically stable. ■

Theorem (3):

The endemic equilibrium point $E_1 = (S_1, I_{A1}, I_{S1}, B_1)$ of System (4) is locally asymptotic stable when $\Re_\circ > 1$ and then the following conditions are satisfied:

$$\left[\frac{\beta_1 K_1 S_1}{(K_1+B_1)^2}\right]^2 < \frac{4}{9}\left[\frac{\beta_1 B_1}{K_1+B_1}+d\right] \cdot \left[\frac{2rB_1}{(K_1+B_1)^2}+\eta-r\right] \quad \text{.....(10a)}$$

$$\left[\frac{\rho\beta_1 B_1}{K_1+B_1}\right]^2 < \frac{2}{3}\left[\frac{\beta_1 B_1}{K_1+B_1}+d\right]\cdot [\gamma_A+d] \quad \text{..... (10b)}$$

$$\left[\frac{(1-\rho)\beta_1 B_1}{K_1+B_1}\right]^2 < \frac{2}{3}\left[\frac{\beta_1 B_1}{K_1+B_1}+d\right]\cdot [\gamma_S+d+\mu] \quad \text{.....(10c)}$$

$$\left[\frac{\rho\beta_1 K_1 S_1}{(K_1+B_1)^2}+\zeta_1\right]^2 < \frac{2}{3}[\gamma_A+d]\cdot \left[\frac{2rB_1}{K_2}+\eta-r\right] \quad \text{..... (10d)}$$

$$\left[\frac{(1-\rho)\beta_1 K_1 S_1}{(K_1+B_1)^2}+\zeta_2\right]^2 < \frac{2}{3}[\gamma_S+d+\mu] \cdot \left[\frac{2rB_1}{K_2}+\eta-r\right] \quad \text{..(10e)}$$

$$r < \frac{2rB_1}{K_2}+\eta \quad \text{.....(10f)}$$

Proof:

The Jacobian matrix of System (4) at $E_1 = (S_1, I_{A1}, I_{S1}, B_1)$ written by:

$J(E_1) = [z_{ij}]_{4\times 4}$, where:

$$z_{11} = -\left(\frac{\beta_1 B_1}{K_1+B_1}+d\right); \qquad z_{14} = -\frac{\beta_1 K_1 S_1}{(K_1+B_1)^2};$$

$$z_{21} = \frac{\rho\beta_1 B_1}{K_1+B_1};\ z_{22} = -(\gamma_A+d)$$

$$z_{24} = \frac{\rho\beta_1 K_1 S_1}{(K_1+B_1)^2}; \qquad z_{31} = \frac{(1-\rho)\beta_1 B_1}{K_1+B_1};$$

$$z_{33} = -(\gamma_S+d+\mu);\ z_{34} = \frac{(1-\rho)\beta_1 K_1 S_1}{(K_1+B_1)^2}$$

$$z_{42} = \zeta_1; z_{43} = \zeta_2; \quad z_{44} = r - \left(\frac{2rB_1}{K_2}+\eta\right)$$ and zero otherwise.

It is easy to verify that the linearization system of system (4) can be written as:

$$\dot{N} = \dot{X} = J(E_1)\cdot X$$

Here, $N = (S, I_A, I_S, B)^T$ and

$X = (x_1, x_2, x_3, x_4)^T$, where:

$x_1 = S - S_1; x_2 = I_A - I_{A1};\ x_3 = I_S - I_{S1};$

$x_4 = B - B_1.$

Now, consider the following positive definite function:

$$V = \frac{x_1^2}{2}+\frac{x_2^2}{2}+\frac{x_3^2}{2}+\frac{x_4^2}{2}$$

It is clearly that $V: R_+^4 \to R$ and a continuously differentiable function so that $V(S_1, I_{A1}, I_{S1}, B_1) = 0$ and $V(S, I_A, I_S, B) > 0$ otherwise. So by differentiating V with respect to time t, gives:

$$\dot{V} = x_1\cdot\dot{X}_1 + x_2\cdot\dot{X}_2 + x_3\cdot\dot{X}_3 + x_4\cdot\dot{X}_4$$

Substituting the values of $\dot{X}_1, \dot{X}_2, \dot{X}_3$ and $\dot{X}_4$ in the above equation, and after doing some algebraic manipulation; we get that:

$$\dot{V} = -\frac{1}{3}\left[\frac{\beta_1 B_1}{K_1+B_1}+d\right]x_1^2 - \left[\frac{\beta_1 K_1 S_1}{(K_1+B_1)^2}x_1x_4\right]$$
$$-\frac{1}{3}\left[\frac{2rB_1}{K_2}+\eta-r\right]x_4^2 - \frac{1}{3}\left[\frac{\beta_1 B_1}{K_1+B_1}+d\right]x_1^2$$
$$+\left[\frac{\rho\beta_1 B_1}{K_1+B_1}\right]x_1x_2 - \frac{1}{2}[\gamma_A+d]x_2^2$$
$$-\frac{1}{3}\left[\frac{\beta_1 B_1}{K_1+B_1}+d\right]x_1^2 + \left[\frac{(1-\rho)\beta_1 B_1}{K_1+B_1}\right]x_1x_3$$
$$-\frac{1}{2}[\gamma_S+d+\mu]x_3^2 - \frac{1}{2}[\gamma_A+d]x_2^2$$
$$+\left[\frac{\rho\beta_1 K_1 S_1}{(K_1+B_1)^2}+\zeta_1\right]x_2x_4 - \frac{1}{3}\left[\frac{2rB_1}{K_2}+\eta-r\right]x_4^2$$
$$-\frac{1}{2}[\gamma_S+d+\mu]x_3^2 + \left[\frac{(1-\rho)\beta_1 K_1 S_1}{(K_1+B_1)^2}+\zeta_2\right]x_3x_4$$
$$-\frac{1}{3}\left[\frac{2rB_1}{K_2}+\eta-r\right]x_4^2$$

Now it is easy to verify that the above set of conditions (10a)-(10e) guarantees the quadratic terms given below:

$$\dot{V} \le -\left[\sqrt{\frac{1}{3}\left(\frac{\beta_1 B_1}{K_1+B_1}+d\right)}x_1 + \sqrt{\frac{1}{3}\left(\frac{2rB_1}{K_2}+\eta-r\right)}x_4\right]^2$$
$$-\left[\sqrt{\frac{1}{3}\left(\frac{\beta_1 B_1}{K_1+B_1}+d\right)}x_1 + \sqrt{\frac{1}{2}(\gamma_A+d)}x_2\right]^2$$
$$-\left[\sqrt{\frac{1}{3}\left(\frac{\beta_1 B_1}{K_1+B_1}+d\right)}x_1 + \sqrt{\frac{1}{2}(\gamma_S+d+\mu)}x_3\right]^2$$
$$-\left[\sqrt{\frac{1}{2}(\gamma_A+d)}x_2 + \sqrt{\frac{1}{3}\left(\frac{2rB_1}{K_2}+\eta-r\right)}x_4\right]^2$$
$$-\left[\sqrt{\frac{1}{2}(\gamma_S+d+\mu)}x_3 + \sqrt{\frac{1}{3}\left(\frac{2rB_1}{K_2}+\eta-r\right)}x_4\right]^2$$

So, $\dot{V}$ is a negative definite, and hence $\dot{V}$ is a Lyapunov function. Thus, (E_1) is a local asymptotically stable and the proof is complete.

6. Global Stability Analysis of System (4)

In this section, the global stability analysis of the all equilibrium points $E_i, i = 0,1$ of system (4) studied as shown in the following theorems.

Theorem (4):

Assume that, the disease free equilibrium point E_0 of System (4) is locally asymptotically stable. Then the basin of attraction of (E_0), say $Q(E_0) \subset R_+^4$, it is globally asymptotically stable if satisfy the following condition:

$$d > \max.\{\zeta_1+\gamma_A, \zeta_2+\gamma_S+\mu\} \quad \text{.....(11a)}$$
$$\frac{\eta K_2 + rB}{K_2} > \frac{r(K_1+B)+B_1 S}{K_1+B} \quad \text{..... (11b)}$$

Proof: Consider the following positive definite function:

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

Clearly, $V_1 : R_+^4 \to R$ is a continuously differentiable function such that $V_1(S_0,0,0,0) = 0$, and $V_1(S, I_A, I_S, B) > 0, \forall (S, I_A, I_S.B) \neq (S_0,0,0,0)$. Further we have:

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

By simplifying this equation we get:

$$\dot{V}_1 = -\frac{d}{S}(S-S_0)^2 - \frac{\beta_1 B}{K_1+B}(S-S_0)$$
$$+[\zeta_1 - (\gamma_A+d)]I_A + [\zeta_2 - (\gamma_S+d+\mu)]I_S$$
$$+B\left[(r+\frac{\beta_1 S}{K_1+B}) - (\eta+\frac{rB}{K_2})\right]$$

Obviously, $\dot{V}_1 < 0$, for every initial points and then V_1 is a Lyapunov function provided that conditions (11a)-(11b) hold. Thus E_0 is globally asymptotically stable in the interior of $Q(E_0)$, which means that $Q(E_0)$ is the basin of attraction and that complete the proof. ■

Theorem (5):

Let the endemic equilibrium point E_1 of System (4) is locally asymptotically stable. Then it is globally asymptotically stable provided that:

$$rK_2 < r(B+B_1) + \eta K_2 \quad \text{.....(12a)}$$

$$\left[\frac{\rho\beta_1 B_1}{K_1+B_1}\right]^2 < \frac{2}{3}\left[\frac{G}{K_1+B_1}\right]\cdot[\gamma_A+d] \quad \text{..... (12b)}$$

$$\left[\frac{(1-\rho)\beta_1 B_1}{K_1+B_1}\right]^2 < \frac{2}{3}\left[\frac{G}{K_1+B_1}\right]\cdot[\gamma_S+d+\mu] \quad \text{.....(12c)}$$

$$\left[\frac{\beta_1 K_1 S_1}{(K_1+B)(K_1+B_1)}\right]^2 < \frac{4}{9}\left[\frac{G}{K_1+B_1}\right]\cdot\left[\frac{G'}{K_2}\right] \quad \text{..... (12d)}$$

$$\left[\frac{G''}{(K_1+B)(K_1+B_1)}\right]^2 < \frac{2}{3}[\gamma_A+d]\cdot\left[\frac{G'}{K_2}\right] \quad \text{.....(12e)}$$

$$\left[\frac{G'''}{(K_1+B)(K_1+B_1)}\right]^2 < \frac{2}{3}[\gamma_S+d+\mu]\cdot\left[\frac{G'}{K_2}\right] \quad \text{.....(12f)}$$

Where:

$G=\beta_1 B_1+d(K_1+B_1)$

$G'=r(B+B_1)+(\eta-r)K_2$

$G''=\rho\beta_1 K_1 S_1+\zeta_1(K_1+B)(K_1+B_1)$

$G'''=(1-\rho)\beta_1 K_1 S_1+\zeta_2(K_1+B)(K_1+B_1)$

Proof:

Consider the following positive definite function:

$$V_2=\frac{(S-S_1)^2}{2}+\frac{(I_A-I_{A1})^2}{2}+\frac{(I_S-I_{S1})^2}{2}+\frac{(B-B_1)^2}{2}$$

Clearly, $V_2: R_+^4 \to R$ is a continuously differentiable function such that $V_2(S_1, I_{A1}, I_{S1}, B_1)=0$ and $V_2(S, I_A, I_S, B)>0,\ \forall (S, I_A, I_S, B)\neq(S_1, I_{A1}, I_{S1}, B_1)$

Further, we have:

$$\dot{V}_2=(S-S_1)\dot{S}+(I_A-I_{A1})\dot{I}_A+(I_S-I_{S1})\dot{I}_S+(B-B_1)\dot{B}$$

By simplifying this equation we get:

$$\begin{aligned}\dot{V}_1=&-\frac{p_{11}}{3}(S-S_1)^2+p_{12}(S-S_1)(I_A-I_{A1})\\&-\frac{p_{22}}{2}(I_A-I_{A1})^2-\frac{p_{11}}{3}(S-S_1)^2\\&+p_{13}(S-S_1)(I_S-I_{S1})-\frac{p_{33}}{2}(I_S-I_{S1})^2\\&-\frac{p_{11}}{3}(S-S_1)^2+p_{14}(S-S_1)(B-B_1)\\&-\frac{p_{44}}{3}(B-B_1)^2-\frac{p_{22}}{2}(I_A-I_{A1})^2\\&+p_{24}(I_A-I_{A1})(B-B_1)-\frac{p_{44}}{3}(B-B_1)^2\\&-\frac{p_{33}}{2}(I_S-I_{S1})^2+p_{34}(I_S-I_{S1})(B-B_1)\\&-\frac{p_{44}}{3}(B-B_1)^2\end{aligned}$$

With:

$$p_{11}=\frac{G}{K_1+B_1};\ p_{12}=\frac{\rho\beta_1 B_1}{K_1+B_1};\ p_{22}=\gamma_A+d;$$

$$p_{13}=\frac{(1-\rho)\beta_1 B_1}{K_1+B_1};\ p_{33}=\gamma_S+d+\mu;$$

$$p_{14}=\frac{\beta_1 S_1 K_1}{(K_1+B)(K_1+B_1)};\ p_{44}=\frac{G'}{K_2};$$

$$p_{24}=\frac{G''}{(K_1+B)(K_1+B_1)};\ p_{34}=\frac{G'''}{(K_1+B)(K_1+B_1)}$$

Therefore, according to the conditions (12a)-(12f) we obtain that:

$$\begin{aligned}\dot{V}_2\le&-\left[\sqrt{\frac{p_{11}}{3}}(S-S_1)-\sqrt{\frac{p_{22}}{2}}(I_A-I_{A1})\right]^2\\&-\left[\sqrt{\frac{p_{11}}{3}}(S-S_1)+\sqrt{\frac{p_{33}}{2}}(I_S-I_{S1})\right]^2\\&-\left[\sqrt{\frac{p_{11}}{3}}(S-S_1)-\sqrt{\frac{p_{44}}{3}}(B-B_1)\right]^2\\&-\left[\sqrt{\frac{p_{22}}{2}}(I_A-I_{A1})+\sqrt{\frac{p_{44}}{3}}(B-B_1)\right]^2\\&-\left[\sqrt{\frac{p_{33}}{2}}(I_S-I_{S1})+\sqrt{\frac{p_{44}}{3}}(B-B_1)\right]^2\end{aligned}$$

Clearly, $\dot{V}_2<0$, and then V_2 is a Lyapunov function provided that the given conditions(12a)-(12f) hold. Therefore, (E_1) is globally asymptotically stable.

7. Numerical Simulation of System (1)

In this section, system (1) is solved numerically for different sets of hypothesis data and different sets of initial conditions, and then the time series for the trajectories of system (1) are confirm our obtained analytical results. By using (0.5, 0.7, 0.3, 0.6, 0.9) and (50, 40, 25, 30, 0.1) as initial points and the numerical simulations are carried out in the following cases:

Case I:

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

$$\theta = 50\ ;\ \beta_1 = 0.0000001;\ K_1 = 0.5\ ;\ d = 0.3\ ;\ \rho = 0\ ;\ \gamma_A = 0.4\ ;\ \gamma_S = 0.1\ ;\ \mu = 0.01\ ;\ r = 0.1\ ;\ K_2 = 0.001\ ;\ \eta = 0.3\ ;\ \zeta_1 = 0.4\ ;\ \zeta_2 = 0.1\ ;\ \Re_\circ = 0 < 1 \quad \text{..... (13)}$$

Therefore, the disease free equilibrium point E_0 of system (1) is globally asymptotically stable and is identically to (167, 0, 0, 0, 0) for any time. See Fig.(1).

Case II:

For the endemic equilibrium point E_1, we choose the following data:

$$\theta = 50\ ;\ \beta_1 = 0.01;\ K_1 = 0.5\ ;\ d = 0.1\ ;\ \rho = 0.1\ ;\ \gamma_A = 0.2\ ;\ \gamma_S = 0.001\ ;\ \mu = 0.01\ ;\ r = 6\ ;\ K_2 = 50\ ;\ \eta = 7\ ;\ \zeta_1 = 4\ ;\ \zeta_2 = 8\ ;\ \Re_\circ = 15.128831 > 1 \quad \text{.... (14)}$$

Therefore, the endemic equilibrium point E_1 of system (1) is globally asymptotically stable and is identically to (255, 148, 36, 297, 23) for any time. See Fig.(2).

Case III:

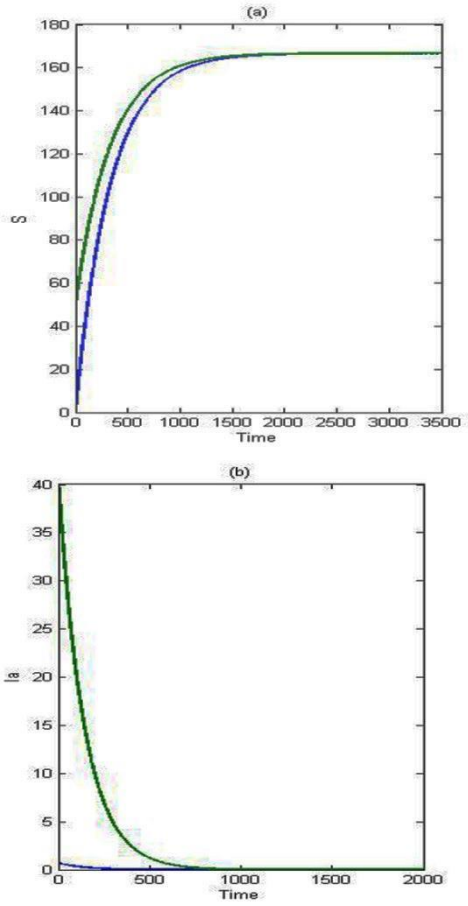
We fixed all parameters in equation (14) but we change infection rate value $\beta_1 = 0.01, 0.05, 0.1, 0.2, 0.5$ respectively, we get the trajectories of system (1) still approaches to endemic equilibrium point but the number of asymptomatic infectious individuals decrease while the number of the symptomatic infectious and cholera population increases. See Fig.(3a-3c).

Case IV:

We choose fraction rate $\rho = 0, 0.2, 0.5, 0.8, 1$ respectively, keeping other parameters fixed as given in equation (14), we get the trajectories of system (1) still approaches to endemic equilibrium point but the number of symptomatic infectious individuals decrease while the number of the asymptomatic infectious and cholera population increases. See Fig.(4a-4c).

Case V:

Now we choose intrinsic growth rate $r = 1, 6, 15, 40$ respectively, keeping other parameters fixed as given in equation (14), we get the trajectories of system (1) still approaches to endemic equilibrium point but the number of asymptomatic infectious individuals and the number of the symptomatic infectious are smoothly decreases while the cholera population is decreases too. See Fig.(5a-5c), and we get inverse above results from increases of carrying capacity rate (K_2).



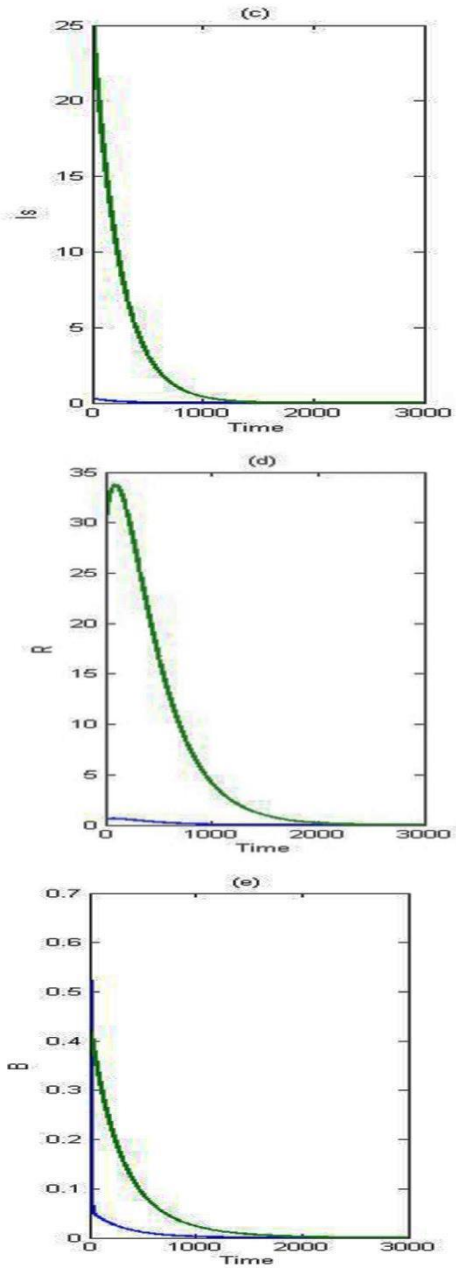
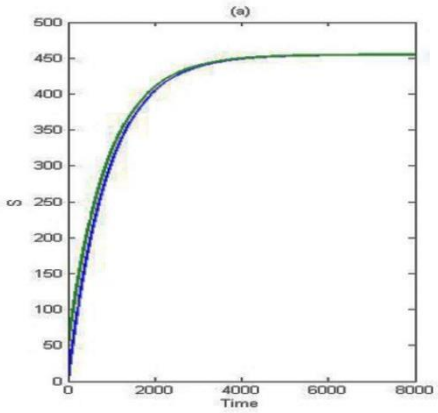


Fig.(1): Time series of the trajectories of system (1) from different initial points for data given in Eq. (13) which show that E_0 is globally asymptotically stable. (a) For S, (b) For I_A, (c) For I_S, (d) For R, (e) For B.



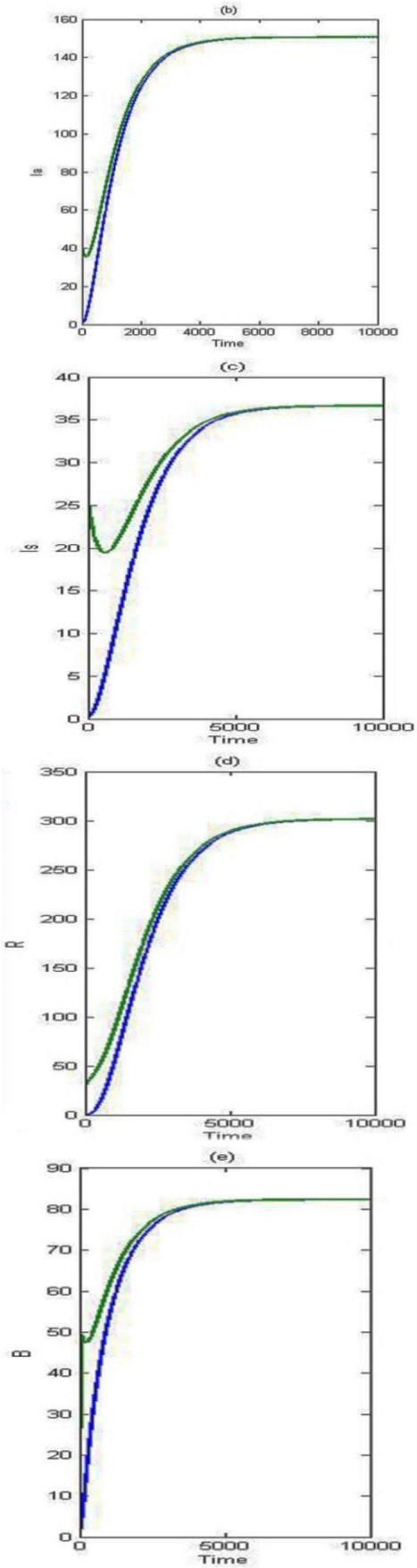


Fig.(2): Time series of the trajectories of system (1) from different initial points for data given in Eq. (14) which show that E_1 is globally asymptotically stable. (a) For S, (b) For I_A, (c) For I_S, (d) For R, (e) For B.

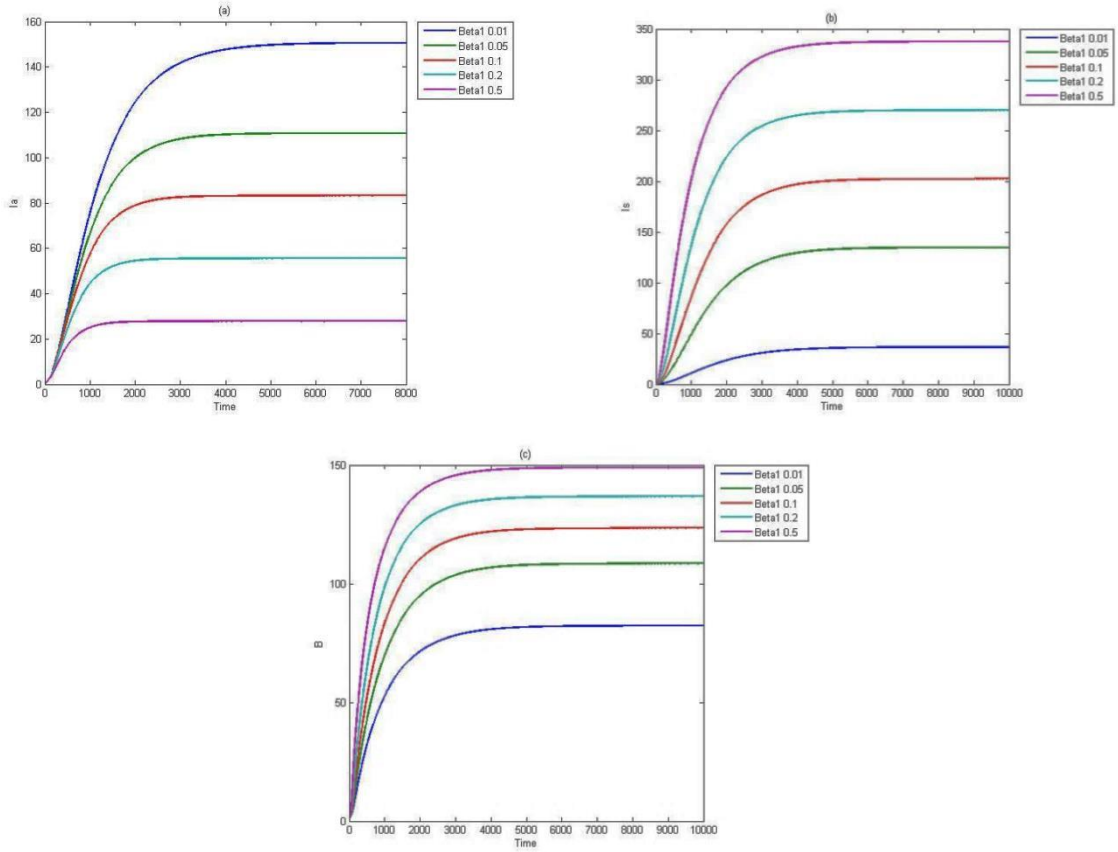


Fig.(3): Time series of the trajectories of system (1). (a) For I_A, (b) For I_S, (c) For B.

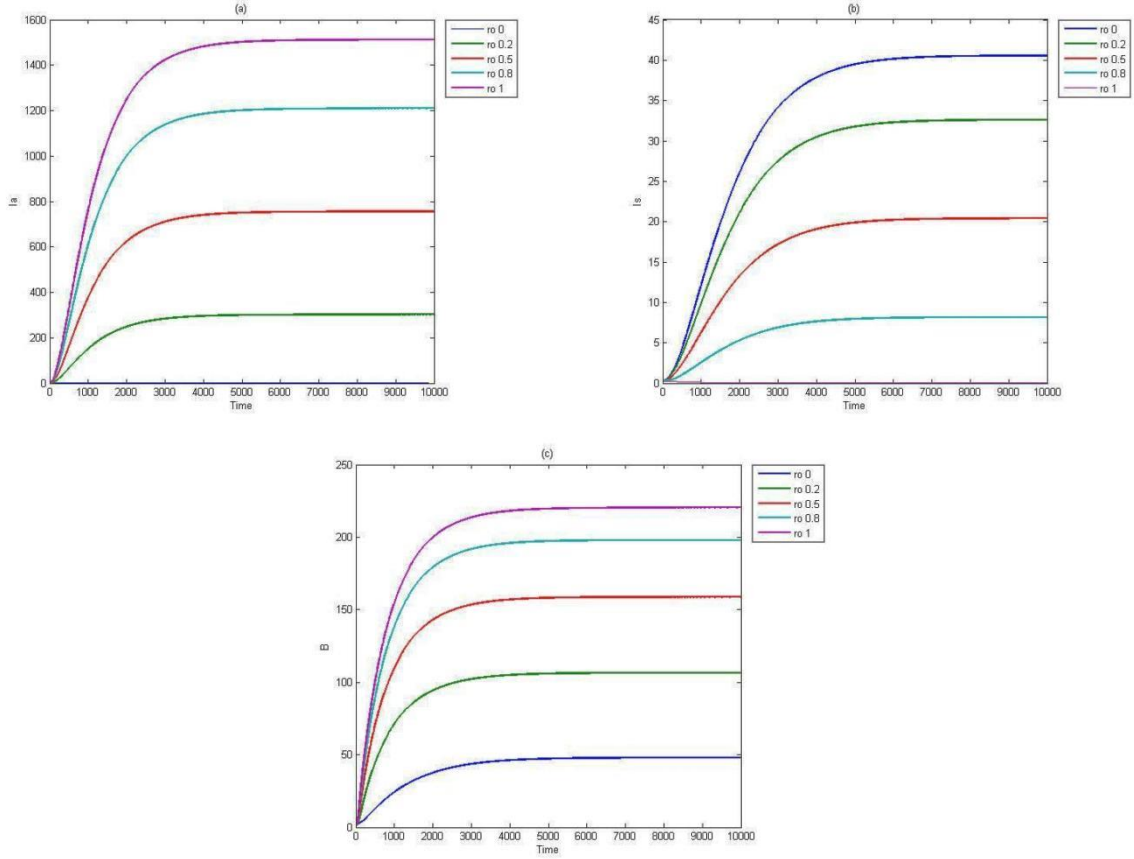


Fig.(4): Time series of the trajectories of system (1). (a) For I_A, (b) For I_S, (c) For B.

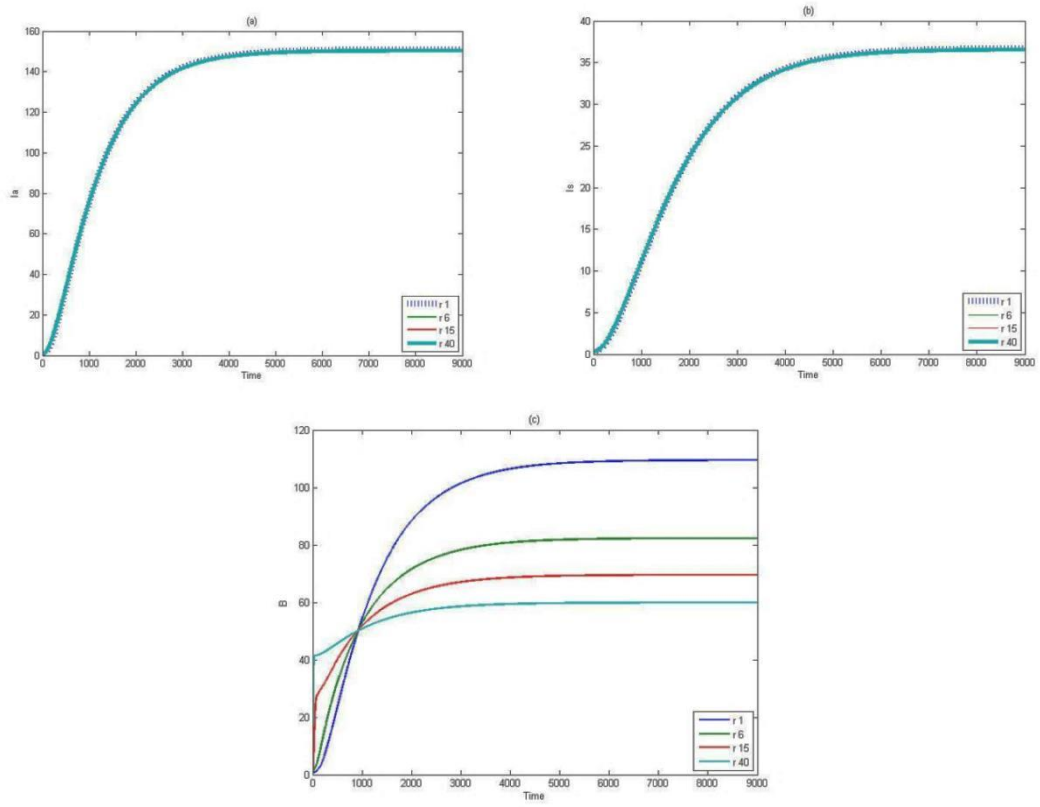


Fig. (5): Time series of the trajectories of system (1). (a) For I_A, (b) For I_S, (c) For B.

7. Conclusion and Discussion

In this paper, we proposed and analyzed an epidemiological model that described the dynamical behavior of an epidemic model, where the infectious disease transmitted directly from contact between them by Holling type II. The model included five non-linear autonomous differential equations that describe the dynamics of five different populations, namely susceptible individuals (S), asymptomatic infectious individuals (I_A), symptomatic infectious individuals (I_S), removal individuals from infected classes (R) and B is cholera population. The boundedness of system (1) has been discussed. The conditions for existence, stability for each equilibrium points are obtained. Further, it is observed that the disease free equilibrium point (E_0) exists when $I_A = I_S = B = 0$ and it locally stable if the conditions (9a-9b) are hold, and then it is globally stable if and only if the conditions (11a-11b) are hold. The endemic equilibrium point (E_1) exists if $\Omega_4 > 0$ and one of three conditions is hold (7a or 7b or 7c) and locally stable if the conditions (10a-10f) are hold more than it is globally stable if and only if the conditions (12a-12f) hold. Finally, to understand the effect of varying each parameter on the global system (1) and confirm our above analytical results, system (1) has been solved numerically for different sets of initial points and different sets of parameters given by equation (14), and the following observations are made:

1) System (1) do not has periodic dynamic, instead it they approach either to the all equilibrium point.
2) As the incidence rate of disease (contact incidence rate (β_1)) increase, the asymptotic behavior of systems (1) approaching to endemic equilibrium point. In fact are (β_1) increase it is observed that the number of (I_A) decrease and the number of $(I_S \text{ and } B)$ increase.
3) As the fraction rate $(0 \le \rho \le 1)$ increase, the asymptotic behavior of systems (1) approaching to endemic equilibrium point. In fact as (ρ) increase it is observed that the number of (I_S) decrease and the number of $(I_A \text{ and } B)$ increase.
4) As the intrinsic growth rate or carrying capacity of cholera population rate, are increases (r, K_2) respectively the

asymptotic behavior of systems (1) approaching to endemic equilibrium point with increase it is observed that the numbers of (I_A, I_S, B) are increase.

5) As the recovery rate $(\gamma_A \text{ or } \gamma_S)$ increases, then increase it is observed that the numbers of (I_A, I_S, B) are decrease.

References

[1] Alam, LaRocque R.C., Harris J.B., Vanderspurt C., Ryan E.T., Qadri F., and Calderwood S.B., Hyperinfectivity of human-passaged Vibrio cholerae can be modeled by growth in the infant mouse, Infect. Immun. 73, 6674–6679, 2005.

[2] Hendrix T.R., The pathophysiology of cholera, Bull. NY Acad. Med. 47, 1169–1180, 1971.

[3] Kaper J.B., Morris J.G., and Levine M.M., Cholera, Clin. Microbiol. Rev. 8, 48–86, 1995.

[4] King A.A., Lonides E.L., Pascual M., and Bouma M.J., Inapparent infections and Cholera dynamics, Nature 454, 877–881, 2008.

[5] Merrell D.S., Butler S.M., and Qadri et al. F., Host-induced epidemic spread of the cholera bacterium, Nature 417, 642–645, 2002.

[6] Nelson E.J., Harris J.B., Morris J.G., Calderwood S.B., and Camilli A., Cholera transmission: the host, pathogen and bacteriophage dynamics, Nat. Rev.: Microbiology 7, 693–702, 2009.

[7] Pascual M., Bouma M., and Dobson A., Cholera and climate: revisiting the quantitative evidence, Microbes Infections 4, 237–245, 2002.

[8] Tudor V., and Strati I., Smallpox, Cholera, Abacus Press, Tunbridge Wells, 1977.

[9] Dietz K., The estimation of the basic reproduction number for infections diseases, Stat. Methods Med. Res. 2, 23–41, 1993.

[10] Hethcote H.W., The mathematics of infectious diseases, SIAM Rev. 42, 599–653, 2000.

[11] Capasso V., and Paveri-Fontana S.L., A mathematical model for the cholera epidemic in the European Mediterranean region, Rev. dépidémoligié et de santé Publiqué 27, 121–132, 1979.

[12] Codeco C.T., Endemic and epidemic dynamics of cholera: the role of the aquatic reservoir, BMC Infectious Diseases 1:1, 2001.

[13] Pascual M., Bouma M.J., Dobson A.P., Cholera and climate: revisiting the quantitative evidence, Microbes and Infection 4 (2), 237–245, 2002.

[14] Joh R.I., Wang H., Weiss H., Weitz J.S., Dynamics of indirectly transmitted infectious diseases with immunological threshold, Bulletin of Mathematical Biology 71, 845–862, 2009.

[15] Miller Neilan R.L., Schaefer E., Gaff H., Fister K.R., Lenhart S., Modeling optimal intervention strategies for cholera, Bulletin of Mathematical Biology 72 (8), 2004–2018, 2010.

[16] Hirsch M. W., and Smale, S., Differential Equation, Dynamical System, and Linear Algebra. *Academic Press, Inc.*, New York. 169-170, 1974.

[17] Van den Driessche P., Watmough J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical* Biosciences, 180:29–48. DOI: 10.1016/S0025-5564(02)00108-6 2002.

[18] Lial M. L., precalculus et. al., Adison-wesly, New York, 2001.

[19] Horn R. A., Johanson C. R., matrix analysis, Cambridge University press, 1985.

الخلاصة

في هذا البحث تم عرض ودراسة نموذج رياضي يصف انتشار مرض الكوليرا في المجتمع السكاني, افترضنا ان المرض يقسم المجتمع السكاني الى خمسة اقسام هي افراد معرضين للاصابة وافراد مصابين بالمرض اصابة غير خطره وافراد مصابين بالمرض اصابة خطره وافراد معافين من المرض والقسم الاخير الافراد الحاملين لفيروس مرض الكوليرا. تمت مناقشة وجود و وحدانية وقيود الحل للنموذج المقترح. قمنا بدراسة السلوك المحلي والشامل له. واخيرا من اجل تأكيد نتائجنا وتحديد تأثير معلمات النموذج الوبائي المقترح على السلوك الديناميكي له اجرينا محاكاة عددية له.