Global Stability of Cholera Epidemic with General Recovery Rate Involving External Source of Disease

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Abstract: In this article a mathematical model that describes the spread of infectious disease in a population is proposed and studied. This model describes the spread of cholera disease with external source of disease and nonlinear recovery function h(I), The local and global stability of the model is studied. Our results suggest that the basic reproduction number itself is not enough to describe whether cholera will prevail or not. Finally, the global dynamics of this model is studied numerically.

Keyword: Cholera disease, global stability, external source, recovery function.

1. Introduction:

Cholera is a dangerous disease caused by the bacterial Vibrio Cholera. It infects the small intestine. There are many types (strains) of the V. C. Bacterial. Some of them cause more serious illnesses than others. Because of this, some human who get cholera have no symptoms; others have symptoms that are not very bad, and others have very bad symptoms [1-4].

Cholera is a very old epidemic. It still affects many human throughout the world. Estimates from 2010 say that between 3 million and 5 million people get Cholera every year, and 58000-130000 people die from the disease every year. Today, Cholera is called a pandemic. However, it is most common in developing countries, especially in children [5-8]. Cholera is an acute intestinal infection caused by the bacterial V. C. Its dynamics are complicated by the multiple interactions between the human host, the pathogen and the environment which contribute to both direct human-to-human and indirect environment to-human transmission pathways [9].

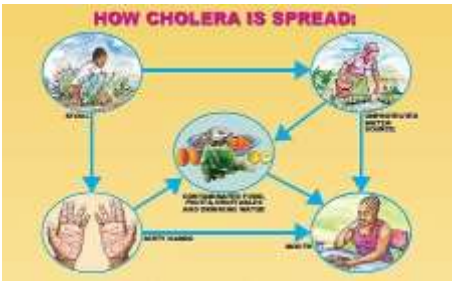


Figure 1: Simplified life cycle of cholera disease

Below, we briefly review some representative mathematical models proposed by various authors. In 2001, [10] extended the model of Capasso and Paveri-Fontana. He added an equation for the dynamics of the susceptible population. And he studied the role of the aquatic reservoir in the endemic and epidemic dynamics of Cholera.

In [11], Pascual et al., Generalized Codeco model by including a 4th equation for the volume of water in which the formative live following [10]. 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 [12]. Also, Ali and Zhou studied the model for the Cholera disease [13]. In this article is organized as follows. In Section 2, we introduce the generalized model and state the necessary assumptions. In Sections 3, we find the each equilibrium point in this model with derive the B. R. N. using the next-generation matrix approach. In Section 4 and 5, we show the local and global stability of the all equilibrium points. Finally, in order to confirm our obtained results and specify the effects of model's parameters on the dynamical behavior, numerical simulation of the cholera model is performed in Section 6.

2. The mathematical model:

In this articale, we suppose the epidemic model descript the cholera disease by the following equations:

$$\begin{aligned}
\dot{S} &= A - \frac{\beta_\circ S(t)B(t)}{K + B(t)} - \beta_1 S(t)I(t) - \beta_2 S(t) - \mu_1 S(t) \\
\dot{I} &= \frac{\beta_\circ S(t)B(t)}{K + B(t)} + \beta_1 S(t)I(t) + \beta_2 S(t) - (r + \mu_1 + \delta)I(t) - Ih(I) \\
\dot{B} &= \eta I(t) - \mu_1 B(t)
\end{aligned} \tag{1}$$

For all time *t*, the population are divided into three classes: a susceptible class *S(t)*, an infectious class *I(t)* and the virus class *B(t)*, that is to say *N=S(t)+I(t)*. All the parameters are positive constant, with descript in the following table:

Table 1: Description of parameters and frequently used symbols.

Parameters	description
A	The birth rate
$\beta_\circ$, β_1	The infection rate
β_2	The infection rate by external source $\beta_2 > 0$
μ_1, μ_2	Dead rate
r	The treatment rate
δ	The disease related death
$h(I) = m/v + wI$	The recovery function, with *m,v and w>0*
η	The new infected members from *I* class
K	The carrying capacity

Clearly, the equations of system (1) are continuously differentiable. In fact there is Lipschizan function on R_+^2. Therefore, the solution of system (1) with non-negative initial conditions is uniformly bounded as shown in the following theorem.

Theorem 1: Each the solutions of system (1), which are initiate in R_+^2, are uniformly bounded.

proof: Let *(S(t),I(t))*, be any solutions of the system (1) with non-negative initial conditions *(S(0),I(0))*. Since *N=S(t)+I(t)*, then $\dot{N} = \dot{S} + \dot{I}$, This gives: $\dot{N} + \mu_1 N \le A$.

Now, by using Gronwall lemma [1], it obtains that:

$$N(t) \le \frac{A}{\mu_1}(1 - e^{-\mu_1 t}) + N(0)e^{-\mu_1 t}$$

Therefore, $N(t) \le A/\mu_1$, *as* $t \to \infty$, hence all the solutions of system (1) that in R_+^2, are confined in reign: $\Gamma_H = \{(S,I) \in R_+^2 : N \le A/\mu_1\}$. And the feasible region of pathogen population for system (1) is $\Gamma_Z = \{B : 0 \le B \le \eta A/\mu_1\mu_2\}$. Define $\Gamma = \Gamma_H \times \Gamma_Z$. Let *Int.*$\Gamma$, denote the interior of Γ. It is easy to verify that the region Γ is positively invariant region with respect to System (1), hence, system (1) will be considered mathematically and epidemiologically well posed in Γ.

4. Local and Global Stable Analysis of $E_\circ$

In this part, the stable analysis of D. F. point $E_\circ(A/\mu_1, 0, 0)$ of the system (1) as shown in the following theorems.

Let *Int.*Γ, denote the interior of Γ. It is easy to verify that t

3. Existence of Equilibrium Point

In system (1), there are always two biologically feasible points, namely the infection-free equilibrium point $E_\circ(S_\circ, 0, 0) = (A/\mu_1, 0, 0)$. This point exists when the basic reproduction number $R_\circ < 1$, where:

$$R_\circ = \frac{\beta_\circ \beta_1 \eta A}{K\mu_1\mu_2(r + \mu_1 + \delta + h(0))} \tag{2}$$

The positive equilibrium point $E_1(S_1, I_1, B_1)$ exists when where:

$$S_1 = \frac{A(K\mu_2 + \eta I_1)}{\eta\beta_\circ I + (K\mu_2 + \eta I_1)[\beta_1 + \beta_2 + \mu_1]} \tag{3}$$

$$B_1 = \frac{\eta I_1}{\mu_2} \tag{4}$$

And I_1 is the positive solution of the following equation:

$$D_1 I_1^4 + D_2 I_1^3 + D_3 I_1^2 + D_4 I_1 + D_5 = 0 \tag{5}$$

Here:

$$D_1 = \beta_1 A \eta^2 w > 0$$

$$\begin{aligned} D_2 = \eta\big[&A(\beta_\circ \eta w + \mu_2\beta_1 K w + \beta_1 \eta v + \beta_1 K \mu_2 w + \beta_2 \eta w) \\ &- \eta(m(\beta_\circ + \beta_1 + \beta_2 + \mu_1) + w(r + \mu_1 + \delta) \\ &(\beta_\circ + \beta_1 + \beta_2 + \mu_1)\big] \end{aligned}$$

$$\begin{aligned} D_3 = A\big[&\beta_1\mu_2^2 K^2 w + \eta(\beta_\circ \eta v + \beta_1\mu_2 v + \beta_1\mu_2 v K + 2\beta_2\mu_2 w K + \beta_2)\big] \\ &+ (\beta_1 + \beta_2 + \mu_1)[\eta v + \mu_2 K w + 2\mu_2 K m]\eta[\beta_\circ\mu_1 K m + (r + \mu_1 + \delta) \\ &\times(\beta_\circ \eta v + \beta_\circ \mu_2 w K + \mu_2 w K(\beta_1 + \beta_2 + \mu_1)] \end{aligned}$$

$$\begin{aligned} D_4 = \mu_2 K\big[&A(\beta_\circ \eta + \mu_2\beta_1 v + 2\beta_2 v\eta + \beta_2\mu_2 K w) \\ &- (r + \mu_1 + \delta)(\beta_\circ \eta v + (2\eta v + \mu_2 K w - \mu_2 K w)(\beta_1 + \beta_2 + \mu_1)\big] \end{aligned}$$

$$D_5 = \mu_2^2 K^2 v\big[A\beta_2 - (r + \mu_1 + \delta)(\beta_1 + \beta_2 + \mu_1)\big]$$

Clearly, equation (5) has unique positive root by I_1 if and only if $D_i < 0$, $i = 2,3,4,5$.

Theorem 2: The disease-free equilibrium point of $E_\circ(A/\mu_1, 0, 0)$ the system (1) is local asymptotically stable provided that:

$$\beta_1 S_\circ < r + \mu_1 + \delta + h(0) \tag{6}$$

$$\eta\beta_\circ S_\circ < \min.\{K(\beta_2+\mu_1)^2,\ K\beta_1 S_\circ(\beta_2+\mu_1)(\beta_1 S_\circ - (r+\mu_1+\delta+h(0)),\ K\mu_2(\beta_1 S_\circ-(r+\mu_1+\delta+h(0))\} \quad (7)$$

Proof: The Jacobian matrix of system (1) at $E_\circ$ that denoted by $J(E_\circ)$ and we can be written as:

$$J(E_\circ)=\begin{pmatrix} -(\beta_2+\mu_1) & -\beta_1 S_\circ & -\beta_\circ S_\circ / K \\ \beta_2 & \beta_1 S_\circ-(r+\mu_1+\delta+h(0)) & \beta_\circ S_\circ / K \\ 0 & \eta & -\mu_2 \end{pmatrix}$$

Clearly, the characteristic equation of the Jacobian matrix $J(E_\circ)$ of the system (1) at the disease-free equilibrium point $E_\circ$ is given by $\lambda^3+A_1\lambda^2+A_2\lambda+A_3=0$

Here:

$A_1=-[a_{11}+a_{22}+a_{33}]$

$A_2=[a_{11}a_{22}-a_{12}a_{21}+a_{22}a_{33}-a_{23}a_{32}+a_{11}a_{33}]$

$A_3=-[a_{33}a_{12}a_{21}+a_{11}a_{23}a_{32}-a_{11}a_{22}a_{33}-a_{21}a_{32}a_{13}]$

Further:

$\Delta=A_1A_2-A_3$

Now according to (Routh-Hurwitz) criterion $E_\circ(A/\mu_1,0,0)$ will be local stable provided that $A_i>0, i=1,3$ and $\Delta=A_1A_2-A_3>0$. Clearly: $A_i>0, i=1,3$ with $\Delta=A_1A_2-A_3$ provided that condition (6-7) holds. Hence the proof is complete.

Theorem 3: Let the disease-free equilibrium point $E_\circ$ of System (1) is local stable. Then the basin of attraction of $E_\circ$, say $B(E_\circ)\subset R_+^3$, it is global stable provided the condition is satisfied:

$$\left(\beta_\circ B/K+B+\beta_1 I+\beta_2\right)S_\circ+\eta I<\left(\mu/S\,(S-S)^2+(r+\mu_1+\delta+h(0))I+\mu_2 B\right) \quad (8)$$

Proof: Consider the following positive definite function:

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

Clearly, $V_1:R_+^3\to R$ is a continuously differentiable function such that $V_1(S_\circ,0,0)=0$, and $V_1(S,I,B)>0, \forall (S,I,B)\neq(S_\circ,0,0)$. Further we have:

$$\dot V_1=\left(\frac{S-S_\circ}{S}\right)\dot S+\dot I+\dot B$$

By simplifying this equation we get:

$$\dot V_1=-\frac{\mu}{S}(S-S_\circ)^2-(r+\mu_1+\delta+h(0))-\mu_2 B+\left(\frac{\beta_\circ B}{K+B}+\beta_1 I+\beta_2\right)S_\circ+\eta I$$

Obviously, $\dot V_1<0$ for each initial point and then V_1 is a Lyap. function provided that condition (8) hold. Thus $E_\circ$ is global stable in $B(E_\circ)$, and that complete the proof.

5. Local with Global Stability Analysis of Positive Point E_1

In this part, the local and global dynamics of system (1) is studied by use the Ruth-Hurwitz and Lyap. function as shown in the theorems.

Theorem 4: The positive point E_1 of the system (1) is local stable if:

$$\beta_1 S_1<r+\mu_1+\delta+h(I_1)+\frac{dh(I_1)I_1}{dI_1} \quad (9)$$

$$\eta\beta_\circ S_1 K<\min.\{(K+B_1)^2L^2,\ \beta_1 S_1(K+B_1)^2L\times(\beta_1 S_1-(r+\mu_1+\delta+h(I_1)+\frac{dh(I_1)I_1}{dI_1})),\ \mu_2(K+B_1)^2(\beta_1 S_1-(r+\mu_1+\delta+h(I_1)+\frac{dh(I_1)I_1}{dI_1})\} \quad \ldots\ldots\ldots\ldots (10)$$

Where: $L=\frac{\beta_\circ B_1}{K+B_1}+\beta_1 I_1+\beta_2+\mu_1$

Proof: The Jacobian matrix of system (1) at E_1 that denoted by $J(E_1)$ and can be written as:

$$J(E_\circ)=\begin{pmatrix} -(\beta_\circ B_1/K+B_1+\beta_1 I_1+\beta_2+\mu_1) & -\beta_1 S_\circ & -\beta_\circ S_1 K/(K+B_1)^2 \\ (\beta_\circ B_1/K+B_1+\beta_1 I_1+\beta_2) & \beta_1 S_1-(r+\mu_1+\delta+h(I_1)+dh(I_1)I_1/dI_1) & \beta_\circ S_1 K/(K+B_1)^2 \\ 0 & \eta & -\mu_2 \end{pmatrix}$$

Clearly, the characteristic equation of the Jacobian matrix $J(E_1)$ of the system (1) at the positive point E_1 is given by $\lambda^3+B_1\lambda^2+B_2\lambda+B_3=0$

Here:

$$B_1 = -[b_{11} + b_{22} + b_{33}]$$

$$B_2 = [b_{11}b_{22} - b_{12}b_{21} + b_{22}b_{33} - b_{23}b_{32} + b_{11}b_{33}]$$

$$B_3 = -[b_{33}b_{12}b_{21} + b_{11}b_{23}b_{32} - b_{11}b_{22}b_{33} - b_{21}b_{32}b_{13}]$$

Further:

$$\Delta = B_1B_2 - B_3$$

Now according to (Routh-Hurwitz) criterion E_1 will be local stable provided that $B_1 > 0$; $B_3 > 0$ and $\Delta = B_1B_2 - B_3 > 0$. Clearly, $B_i > 0, i = 1,3$ and $\Delta = B_1B_2 - B_3 > 0$, provided that condition (9-10) holds. Hence the proof is complete.

Theorem 5: If the positive point E_1 of System (1) is local stable. Then it is global stable if satisfy the following conditions:

$$\beta_1 S_1 < r + \mu_1 + \delta + X \quad (11)$$

$$\left(\beta_\circ B_1 / K + B_1 + \beta_1 I_1 + \beta_2 - \beta_1 S_1\right)^2 < L(r + \mu_1 + \delta + X - \beta_1 S_1) \quad \text{…….. (12)}$$

$$\left(\frac{\beta_\circ SK}{(K+B)(K+B_1)}\right)^2 < \mu_2 L \quad (13)$$

$$\left(\frac{\beta_\circ SK}{(K+B)(K+B_1)} + \eta\right)^2 < \mu_2(r + \mu_1 + \delta + L - \beta_1 S_1) \quad \text{…….. (14)}$$

Where: $X = \dfrac{mv}{(v+mI)(v+wI_1)}$

Proof: Consider the following positive definite function:

$$V_2 = \frac{(S-S_1)^2}{2} + \frac{(I-I_1)^2}{2} + \frac{(B-B_1)^2}{2}$$

Clearly, $V_2 : R_+^3 \to R$ is a continuously differentiable function such that $V_2(S_1, I_1, B_1) = 0$ and $V_2(S, I, B) > 0, \forall (S_1, I_1, B_1) \neq (S, I, B)$. Further, we have:

$$\dot{V}_2 = (S-S_1)\dot{S} + (I-I_1)\dot{I} + (B-B_1)\dot{B}$$

By simplifying this equation we get:

$$\dot{V}_2 = \frac{q_{11}}{2}(S-S_1)^2 - q_{12}(S-S_1)(I-I_1) - \frac{q_{22}}{2}(I-I_1)^2 - \frac{q_{11}}{2}(S-S_1)^2 + q_{13}(S-S_1)(B-B_1) - \frac{q_{33}}{2}(B-B_1)^2 - \frac{q_{22}}{2}(I-I_1)^2 + q_{23}(I-I_1)(B-B_1) - \frac{q_{33}}{2}(B-B_1)^2$$

With:

$$q_{11} = L\ ;\ q_{12} = \frac{\beta_\circ B_1}{K+B_1} + \beta_1 I_1 + \beta_2 - \beta_1 S_1$$

$$q_{22} = r + \mu_1 + \delta + X - \beta_1 S_1\ ;\ q_{13} = \beta_\circ SK(K+B)(K+B_1)$$

$$q_{33} = \mu_2\ ;\ q_{23} = \frac{\beta_\circ SK}{(K+B)(K+B_1)} + \eta$$

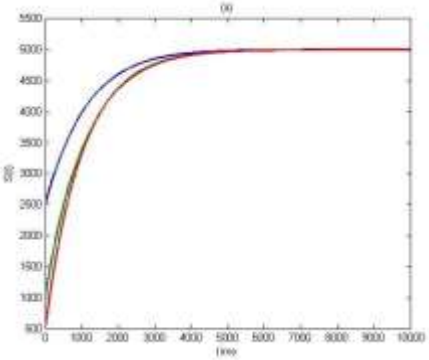
Therefore, according to the conditions (11-14) we obtain that:

$$\dot{V}_2 \le -\left[\sqrt{\frac{q_{11}}{2}}(S-S_1) - \sqrt{\frac{q_{22}}{2}}(I-I_1)\right]^2 - \left[\sqrt{\frac{q_{11}}{2}}(S-S_1) - \sqrt{\frac{q_{33}}{2}}(B-B_1)\right]^2 - \left[\sqrt{\frac{q_{22}}{2}}(I-I_1) + \sqrt{\frac{q_{33}}{2}}(B-B_1)\right]^2$$

Clearly, $\dot{V}_2 < 0$ and then V_2 is a Lyap. function provided that the given conditions (11-14) hold. Therefore, E_1 is global stable.

6. Numerical Simulation of system (1)

In this part, the dynamical behavior of system (1) is studied numerically. The objectives of this study are confirming our obtained analytical results and understand the effects of some parameters on the dynamics of system (1). Consequently, system (1) is solved numerically for different sets of initial conditions and for different sets of parameters. It is observed that, for the following set of hypothetical parameters that satisfies stability conditions of all equilibrium points $(E_i, i = \circ, 1)$ system (1) has a globally asymptotically stable disease-free equilibrium point as shown in following figures.



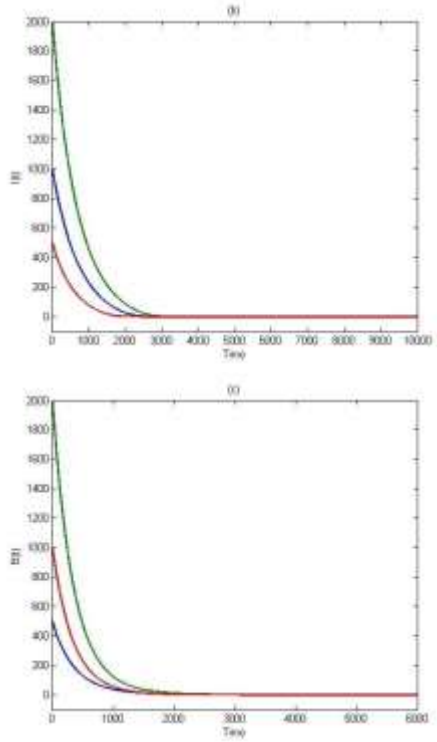
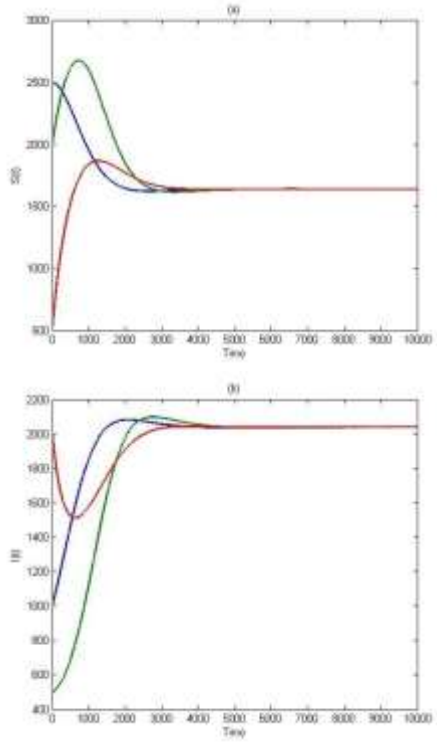


Figure 2: The unique point of system (1) is global stable. In this case, $A=500$, $\beta_{\circ}=0.00001$, $\beta_1=0.00001$, $\beta_2=0$, $\eta=0.01$, $\mu_1=0.1$, $\mu_2=0.3$, $r=0.05$, $m=1$, $v=2$, $w=0.1$, $K=5$, $\delta=0.01$. And the trajectories of system (1) approaches to $E_{\circ}=(5000,0,0)$, from three initial conditions are (2500,1000,500), (1000,2000,2000) and (500,500,1000).



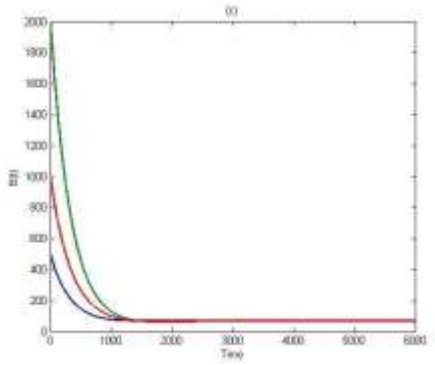
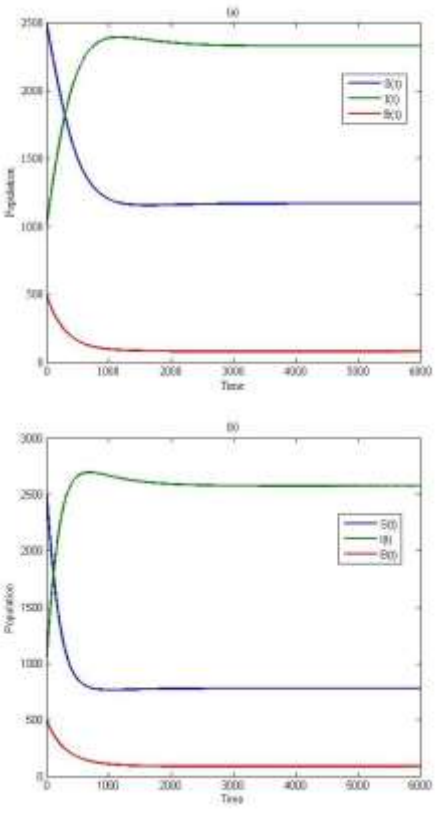


Figure 3: The positive point of system (1) is global stable. In this case, $A=500$, $\beta_{\circ}=0.001$, $\beta_1=0.0001$, $\beta_2=0.0001$, $\eta=0.01$, $\mu_1=0.1$, $\mu_2=0.3$, $r=0.05$, $m=1$, $v=2$, $w=0.1$, $K=5$, $\delta=0.01$. And the trajectories of system (1) approaches to $E_1=(1600,2075,95)$, from three initial conditions are (2500,1000,500), (2000,500,2000) and (500,2000,1000).

Now, we choose the set of hypothetical parameters $A=500$, $\beta_1=0.0001$, $\beta_2=0.0001$, $\eta=0.01$, $\mu_1=0.1$, $\mu_2=0.3$, $r=0.05$, $m=1$, $v=2$, $w=0.1$, $K=5$, $\delta=0.01$. but we change the infection rate value ($\beta_{\circ}=0.1,0.3,0.5$) respectively, we get the trajectories of system (1) still approaches to positive point but the number of *S(t)* decrease while the numbers of the *I(t)* and virus class increases.



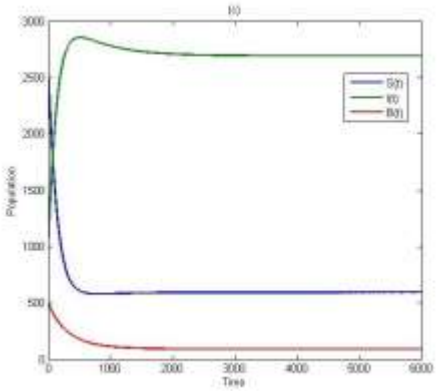
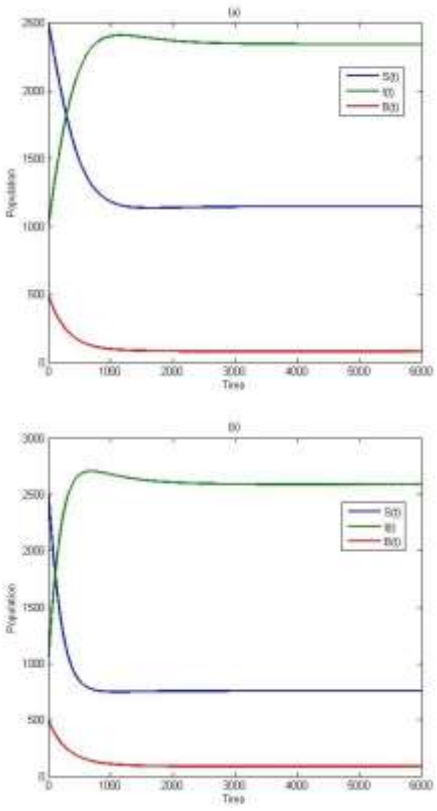


Figure 4: The trajectories of system (1): (a) $\beta_{\circ}$ =0.1, (b) $\beta_{\circ}$ =0.3, (c) $\beta_{\circ}$ =0.5.

Now the effect of external sources in the environment on the dynamics of system (1) is studied by solving the system numerically for the parameters values β_2 =0.1, 0.3, 0.5 respectively, in following figure:



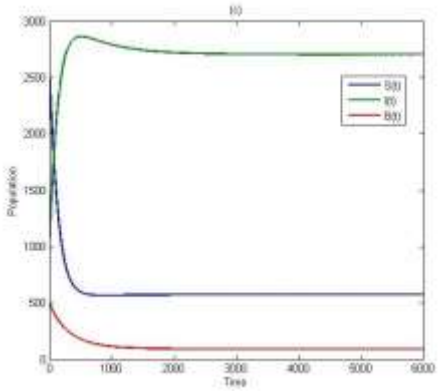
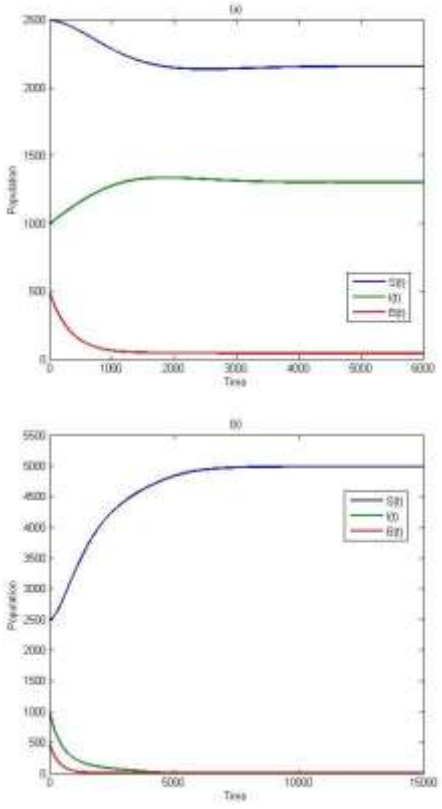


Figure 5: The trajectories of system (1), we use, *A=500*, $\beta_{\circ}$ =0.001, β_1 =0.0001, η =0.01, μ_1 =0.1, μ_2 =0.3, *r*=0.05, *m*=1, *v*=2, *w*=0.1, *K*=5, δ =0.01, with (a) β_2 =0.1, (b) β_2 =0.3, (c) β_2 =0.5.

According to Figure (5), as the spread of disease by increases the external sources parameter, the trajectory of system (1) approaches to the positive point. In fact as β_2 increases it is observed that the number of *S(t)* individuals decrease and the number of *I(t)* and virus individuals increases.

Clearly, we present the effect of treatment rate that is by change value for *r*=0.1, 0.3, 0.5 respectively, we get the trajectories of system (1) still approaches to positive point but the number of *I(t)* and virus individuals decreases while the *S(t)* individuals is increases.



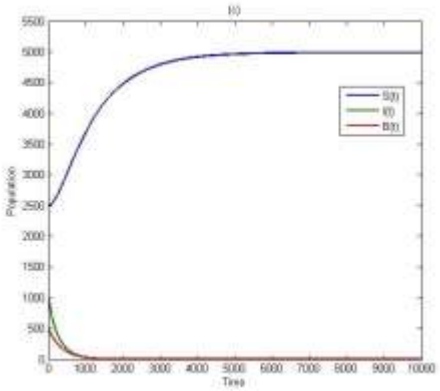
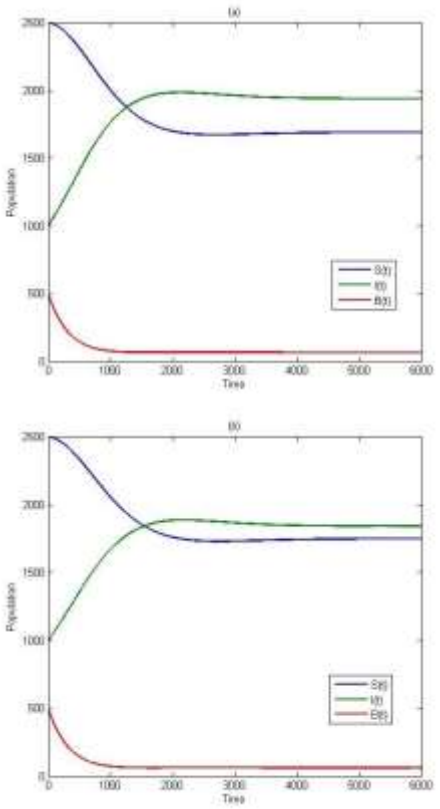


Figure 6: The trajectories of system (1), we use, $A=500$, $\beta_{\circ}=0.001$, $\beta_1=0.0001$, $\beta_2=0.0001$, $\eta=0.01$, $\mu_1=0.1$, $\mu_2=0.3$, m=1, $v=2$, $w=0.1$, $K=5$, $\delta=0.01$, with (a) $r=0.1$, (b) $r=0.3$, (c) $r=0.5$.

Similar results are obtained, as those shown in case of increasing r, in case of increasing the recovery rate, that means increasing m as shown in the following figures:



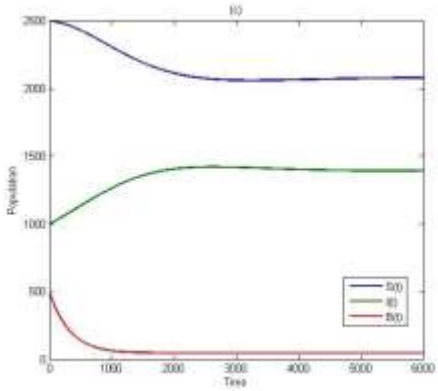


Figure 7: The trajectories of system (1), we use, $A=500$, $\beta_{\circ}=0.001$, $\beta_1=0.0001$, $\beta_2=0.0001$, $\eta=0.01$, $\mu_1=0.1$, $\mu_2=0.3$, $r=0.05$, $v=2$, $w=0.1$, $K=5$, $\delta=0.01$, with (a) $m=2$, (b) $m=3$, (c) $m=7$.

References

[1] Barbalat, I., System d' equation differentially d' Oscillation nonlinear. Rev. Roum. Math. Pure. Appl. 4, 267-270, 1959.

[2] Finkelstein, Richard A., "Chapter 24: Cholera, Vibrio cholera and Other Pathogenic Vibrios." In Medical Microbiology. Galveston, Texas University of Texas Medical Branch at Galveston, 1996.

[3] Tudor V., and Strati I., Smallpox, Cholera, Abacus Press, Tunbridge Wells, 2012.

[4] Lozano R, Naghavi M, et al. "Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: A systematic analysis for the Global Burden of Disease Study 2010". Lancet 380 (9859), 2095-128, 2012.

[5] Reidl J and Klose KE. "Vibrio cholera and cholera: Out of the water and into the host". FEMS Microbiology Reviews 26 (2): 125-39, 2002.

[6] Capasso, V., Paveri-Fontana, S.L., A mathematical model for the 1973 cholera epidemic in the European Mediterranean region. Rev. Epidemiology. Sante 27, 121-132, 1979.

[7] WHO. International travel and Health, 2010. Geneva: World Health Organization, 2010, [Available from: http://www.who.int/ith/chapters/ith2011chap6.pdf].

[8] WHO. Guidelines for cholera control. Geneva: World Health Organization; 1993 [Available from:

http://whqlibdoc.who.int/publications/1993/924154449X.pdf].

[9] E.J. Nelson, J.B. Harris, J.G. Morris, S.B. Calder wood, and A. Camilli, Cholera transmission: the host, pathogen and bacteriophage dynamics, Nat. Rev.: Microbiology 7, pp. 693-702, 2009.

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

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

[12] 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.

[13] Ahmed A. and Xueyong Zhou, On the Dynamical Behaviors of a Cholera Model with Holling Type II Functional Response, Journal of Al-Nahrain University, March, 2016.

[14] Castillo-Chavez C, Song BJ. Dynamical models of tuberculosis and their applications. Mathematical Biosciences and Engineering, 1,361-404, 2004.