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RESEARCH PAPER

Modeling of Control Strategies for Cholera Epidemic

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ABSTRACT

The SIRB model for the dynamics of cholera epidemic introduced by Codeco is modified by incorporating some control strategies: vaccination is introduced to susceptible class, therapeutic treatment is applied to infected class, and water sanitation leads to the death of vibrios. The resulting system is solved numerically using the Runge-Kutta integration scheme with a modified version of Newton-Raphson shooting method with β, λ, σ, and υ as prescribed parameters. It is found out that with strong control measures, the concentration of toxigenic $V.cholerae$ in water, $B(t)$, would be zero so that the disease-free equilibrium is globally asymptotically stable. With weak controls, instead, a unique and globally stable endemic equilibrium would still occur, though at a lower infection level.

Keywords: Cholera, Equilibrium, Epidemic, Vaccination, Sanitation

INTRODUCTION

According to Sack et al. (2004), Cholera is an infection of the small intestine caused by the bacterium *Vibrio cholerae*. Intestinal infection with Vibrio cholerae results in the loss of large volumes of watery stool, leading to severe and rapidly progressing dehydration and shock. Without adequate and appropriate rehydration therapy, severe cholera kills about half of affected individuals. It is estimated that about one hundred million bacteria must typically be ingested in order to cause cholera in a normal healthy adult. The susceptibility is also higher in children, with two to four-year-olds having the highest rates of infection. Blood type is another factor that affects an individual's susceptibility to cholera, with those with type "O" blood being the most susceptible. Persons with lower immunity, such as persons with AIDS or children who are malnourished, are more likely to experience a severe case if they become infected. However, it should be noted that any individual, even a healthy adult in middle age, can experience a severe case, and each person's case should be measured by the loss of fluids, preferably in consultation with a doctor or some health worker. The severity of the diarrhea and vomiting can lead to rapid dehydration and electrolyte imbalance, and death in some cases.

About the earliest mathematical model of Cholera could be traced to Capasso and Paveri-Fontana (1979), which proposed a simple deterministic mathematical model that describes the dynamics of the 1973 cholera epidemic that occurred in the European Mediterranean region. This simple model consists of a system of two ordinary differential equations, that describe the dynamics of the infected individuals in a town community and of the free-living bacteria population in the sea. The model of Codeco (2001) includes an additional equation for the susceptible individuals in the population which, explicitly incorporated the environmental component, i.e., the *V. cholerae* concentration in the water supply (denoted by *B*), into a regular SIR system to form a combined human-environment (SIR-B) epidemiological model. Following this, Pascual *et al.* (2002) generalized the model of Codeco (2001) by including a fourth equation for the volume of water in which the formatives live. Thereafter, Hartley *et al.* (2006) introduced a more general five equations model which describe the dynamics of the susceptible, infectious, and removed human population and the dynamics of a hyper-infective state and the lower infective state of *v. cholerae* population.

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Pascual *et al.* (2008) examined the prediction ability of a semi-mechanistic time series model that incorporates the El Nino Southern Oscillation (ENSO) and the non-linear dynamics of the disease itself, through changes in the population levels of immunity.

This research is an attempt to investigate the effect of certain control strategies applied to the model of Codeco (2001), with a view to equipping public health practitioners with necessary and adequate information for the prevention and control of Cholera epidemic.

METHODOLOGY

Model Formulation

A cholera epidemic model which was first proposed by Codeco (2001) is considered. In this model, the concentration of cholera in water supply, denoted by $B(\tau)$, is incorporated into a regular SIR model to form a combined human environment (SIR-B) epidemiological model. The model is represented by (1).

$$\left.\begin{aligned} \frac{dS}{d\tau} &= nH - nS - \frac{aBS}{K+B} \\ \frac{dI}{d\tau} &= \frac{aBS}{K+B} - rI \\ \frac{dB}{d\tau} &= eI - mB \\ \frac{dR}{d\tau} &= rI - nR \end{aligned}\right\} \quad (1)$$

where S is the number of susceptibles, I is the number of infected, R is the number of recovered,

B is the concentration of toxigenic $V.cholerae$ in water, H is total human population, n is natural birth rate, r is the recovery rate, $m = mb - nb$ is the net death rate of $V.cholerae$, mb is the loss rate of $V.cholerae$ in the aquatic environment, nb is the growth rate of $V.cholerae$ in the aquatic environment, e is the contribution of each infected person to the population of $V.cholerae$ in the aquatic environment, $\frac{aB}{K+B}$ is the incidence, which determines the rate of new infection, a is contact rate with contaminated water, K is the half saturation rate.

We modify the model represented by adding the following three controls: vaccination, therapeutic treatment and water sanitation. Also, four assumptions are made thus.

i. Vaccination is introduced to susceptible at a rate of v so that vS individual per time are removed from the susceptible class and added to the recovered class

ii. Therapeutic treatment is applied to infected at a rate u so that uI individual per time are removed from the infected class and added to the recovered class.

iii. Water sanitation leads to the death of $V.cholerae$ at a rate of w.

iv. Another type of vaccination is applied to (some) newborns so that only a person $(0 < p \leq 1)$ for individuals entering the total populations are susceptible

Consequently, the model represented by (2) is obtained.

$$\left.\begin{aligned} \frac{dS}{d\tau} &= pnH - (n+v)S - \frac{aBS}{K+B} \\ \frac{dI}{d\tau} &= \frac{aBS}{K+B} - (r+u)I \\ \frac{dB}{d\tau} &= eI - (m+w)B \\ \frac{dR}{d\tau} &= (1-p)nH + (r-n+u)I - nR + vS \end{aligned}\right\} \quad 2)$$

Non-Dimensionalization

The model is non-dimensionalized using the parameter $t = (n+v)\tau$, $S' = \frac{n+v}{pnH}S$, $I' = \frac{n+v}{pnH}I$, $B' = \frac{n+v}{pnH}B$ and $R' = \frac{n+v}{pnH}R$. This results in

$$\left.\begin{aligned} \frac{dS}{dt} &= 1 - S - \frac{\beta BS}{\alpha+B} \\ \frac{dI}{dt} &= \frac{\beta BS}{\alpha+B} - \gamma I \\ \frac{dB}{dt} &= \sigma I - \upsilon B \\ \frac{dR}{dt} &= \lambda + \pi I - \eta R + \delta S \end{aligned}\right\} \quad (3)$$

In order to further reduce the parameters, we let $\gamma = \sigma = \pi$, to obtain the following equations

$$\left.\begin{aligned}\frac{dS}{dt} &= 1 - S - \frac{\beta BS}{\alpha + B}\\ \frac{dI}{dt} &= \frac{\beta BS}{\alpha + B} - \sigma I\\ \frac{dB}{dt} &= \sigma I - \upsilon B\\ \frac{dR}{dt} &= \lambda + \sigma I - \eta R + \delta S\end{aligned}\right\}, \qquad (4)$$

where, β is the contact rate with contaminated water parameter.

α is the water treatment parameter
σ is the infected rate parameter
υ is the vibrios death rate parameter
λ is the vaccine introduced to the new born parameter
η is the natural birth rate parameter
δ is the vaccine introduced to susceptible parameter

Equilibria

Considering the first three equations of system (3)

$$\left.\begin{aligned}\frac{dS}{dt} &= 1 - S - \frac{\beta BS}{\alpha + B}\\ \frac{dI}{dt} &= \frac{\beta BS}{\alpha + B} - \sigma I\\ \frac{dB}{dt} &= \sigma I - \upsilon B\end{aligned}\right\} \qquad (5)$$

We set

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dB}{dt} = 0 \qquad (6)$$

Then the steady state of the system (4) satisfy the following algebraic system

$$\left.\begin{aligned}1 - S - \frac{\beta BS}{\alpha + B} &= 0\\ \frac{\beta BS}{\alpha + B} - \sigma I &= 0\\ \sigma I - \upsilon B &= 0\end{aligned}\right\} \qquad (7)$$

There are two steady states for system (5): when $S(t) = 1$ and $I(t) = B(t) = 0$ for all t. The first corresponds to the situation with no infection present and the entire population is susceptible, that is, disease free equilibrium. Thus,

$$1 - S - \frac{\beta BS}{\alpha + B} = 0 \qquad (8)$$

If $I = 0, B = 0$ it implies $S = 1$. Therefore $P_1(S, I, B) = (1,0,0)$. The second corresponds to an endemic steady state (endemic equilibrium) with constant number in the population infected; this biologically reasonable only when $S(t) < 1$, that is when $R_0 > 1$, where R_0 is the basic reproduction rate of the infection:

$$P_2(S, I, B) = \frac{1 + \upsilon\alpha}{1 + \beta}, -\frac{\beta + \upsilon\alpha}{(1 + \beta)\sigma}, -\frac{\beta + \upsilon\alpha}{(1 + \beta)\upsilon} \qquad (9)$$

$$P_2(S, I, B) = (C_1, C_2, C_3) \qquad (10)$$

Where

$$C_1 = \frac{1+\upsilon\alpha}{1+\beta},\ C_2 = -\frac{\beta+\upsilon\alpha}{(1+\beta)\sigma}, C_3 = -\frac{\beta+\upsilon\alpha}{(1+\beta)\upsilon}$$

Stability Analysis

The jacobian matrices of (5) is

$$Df(S, I, B) = \begin{pmatrix} -1 - \beta B & 0 & \frac{-\beta S\alpha}{(\alpha + \beta)^2} \\ \beta B & -\sigma & \frac{\beta S\alpha}{(\alpha + \beta)^2} \\ 0 & \sigma & -\upsilon \end{pmatrix} \qquad (11)$$

The linearization of (7) at $P_1$is

$$Df(100) = \begin{pmatrix} -1 & 0 & -k \\ 0 & -\sigma & k \\ 0 & \sigma & -\upsilon \end{pmatrix} \qquad (12)$$

where $K = \frac{-\beta}{\alpha}$, and the eigenvalue relation is computed to as

$$\left.\begin{aligned}\lambda_1 &= -1\\ \lambda_2 &= -(\sigma + \upsilon) + \frac{\sqrt{(\sigma + \upsilon)^2 - 4(\sigma\upsilon + \sigma k)}}{2}\\ \lambda_3 &= -(\sigma + \upsilon) - \frac{\sqrt{(\sigma + \upsilon)^2 - 4(\sigma\upsilon + \sigma k)}}{2}\end{aligned}\right\} \qquad (13)$$

1. If $(\sigma + \upsilon)^2 - 4(\sigma\upsilon + \sigma k) > 0$ the eigenvalues are real, unequal and negative. Hence, the critical point (1,0,0) is an asymptotically stable improper node of the system.
2. If $(\sigma + \upsilon)^2 - 4(\sigma\upsilon + \sigma k) = 0$ the eigenvalues are negative. Hence, the critical point (1,0,0) is asymptotically stable.
3. 3 If $(\sigma + \upsilon)^2 - 4\sigma(\upsilon + k) < 0$ we have one negative root and two complex root whose real part are equal and negative. Hence, the critical point (1,0,0) is globally asymptotically stable.

The linearization at P_2 is

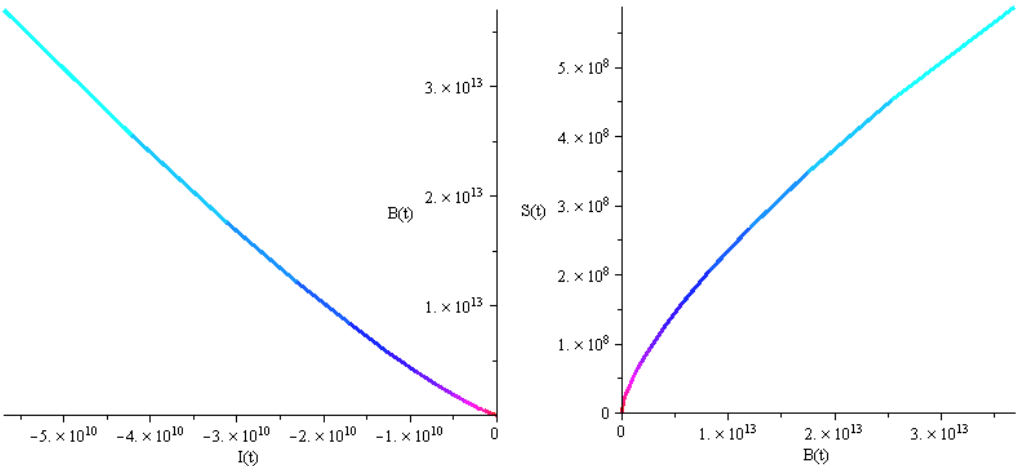
$$Df(C_1,C_2,C_3) = \begin{pmatrix} -1 - C_3\beta, & 0 & \frac{-\beta C_{1\alpha}}{(\alpha+\beta)^2} \\ C_3\beta & -\sigma & \frac{\beta C_{1\alpha}}{(\alpha+\beta)^2} \\ 0 & \sigma & -\upsilon \end{pmatrix} = \begin{pmatrix} q_1 & q_2 & -q_2 \\ q_3 & -\sigma & q_2 \\ 0 & \sigma & -\upsilon \end{pmatrix} \quad (14)$$

where $q_{1=} - 1 - C_3\beta$, $q_2 = \frac{\beta C_{1\alpha}}{(\alpha+\beta)^2}$ and $q_3 = C_3\beta$, with the following eigenvalue relations

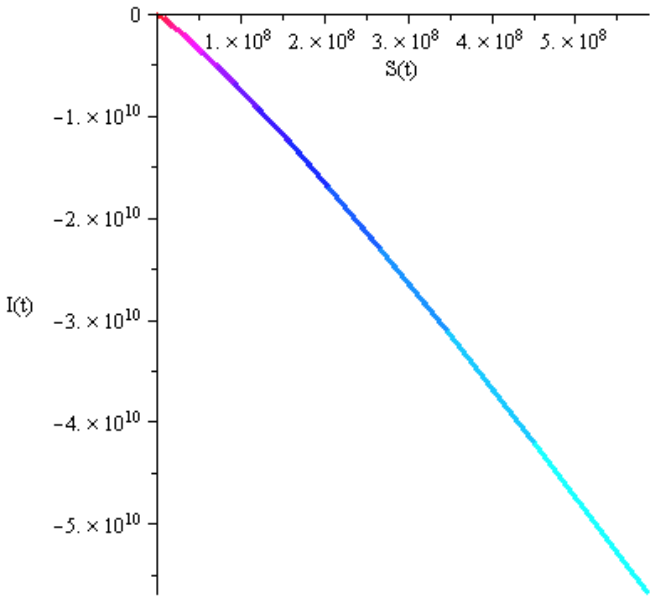
$$\left.\begin{aligned} \lambda_1 &= \frac{1}{6}(A+B)^{\frac{1}{3}} - \frac{6c}{(A+B)^{\frac{1}{3}}} + \frac{1}{3}a \\ \lambda_2 &= -\frac{1}{12}(A+B)^{\frac{1}{3}} + \frac{3c}{(A+B)^{\frac{1}{3}}} + \frac{1}{3}a + \frac{1}{2}i\sqrt{3}\left(\frac{1}{6}(A+B)^{\frac{1}{3}} + \frac{6c}{(A+B)^{\frac{1}{3}}}\right) \\ \lambda_3 &= -\frac{1}{12}(A+B)^{\frac{1}{3}} + \frac{3c}{(A+B)^{\frac{1}{3}}} + \frac{1}{3}a - \frac{1}{2}i\sqrt{3}\left(\frac{1}{6}(A+B)^{\frac{1}{3}} + \frac{6c}{(A+B)^{\frac{1}{3}}}\right) \end{aligned}\right\} \quad (15)$$

RESULTS AND DISCUSSION

We solve the differential system (4) numerically using the Runge-Kutta integration scheme with a modified version of Newton-Raphson shooting method. The results are presented in Figures 1 to 5.



(a) (b)



(c)

Figure 1 Phase portrait of system (6) when $\beta = 5, \alpha = 3, \sigma = 7, \upsilon = 9$

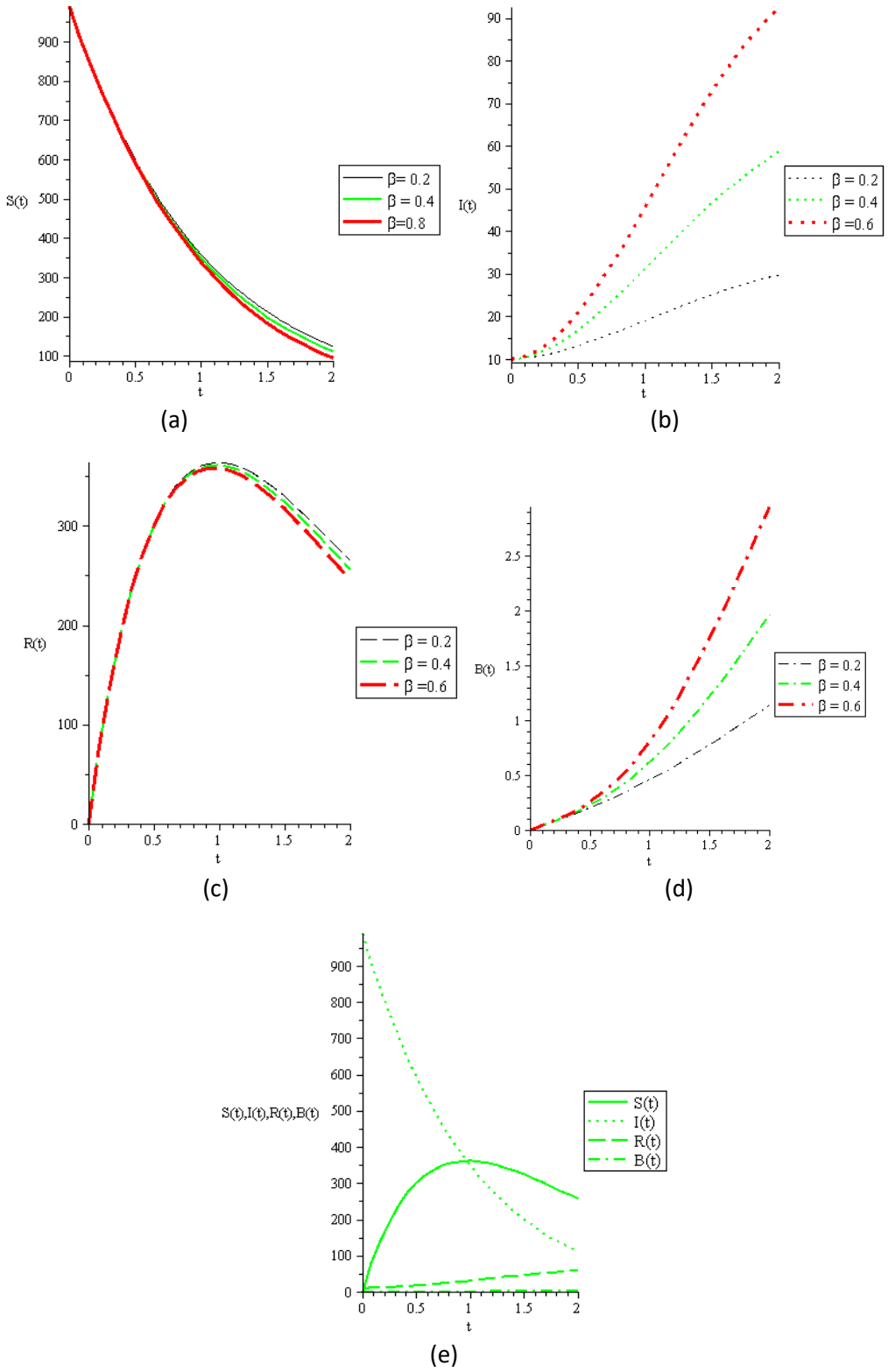


Figure 2 Plots of $S(t),\ I(t), R(t), B(t)$ against t for different values of β when $\upsilon = 0.4,\ \lambda = 2, \sigma = 0.04, \eta = 1, \alpha = 2, \delta = 1$

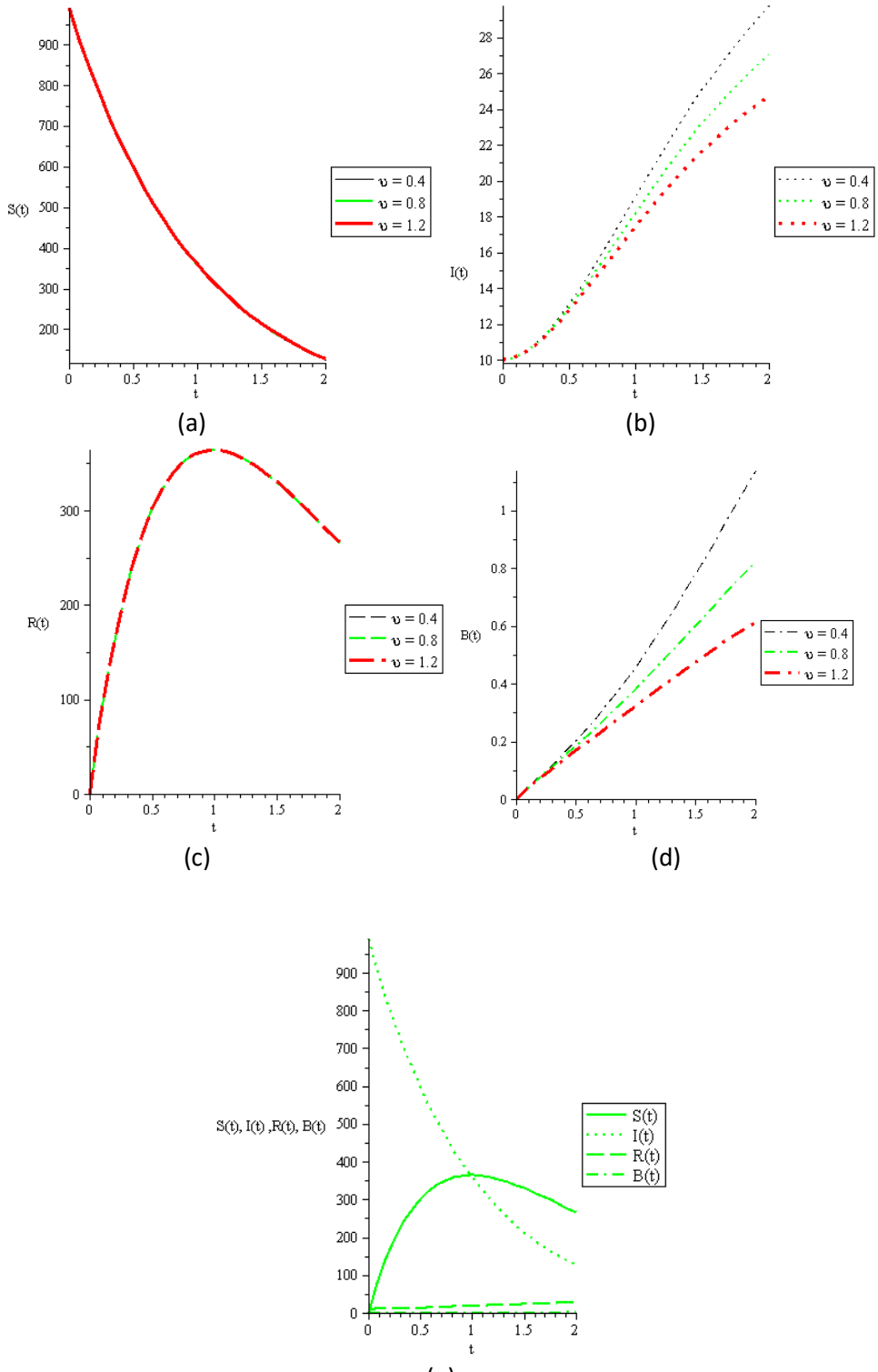


Figure 3 Plots of $S(t),\ I(t), R(t), B(t)$ against t for different values of υ when $\beta = 0.2,\ \lambda = 2, \sigma = 0.04, \eta = 1, \alpha = 2, \delta = 1$

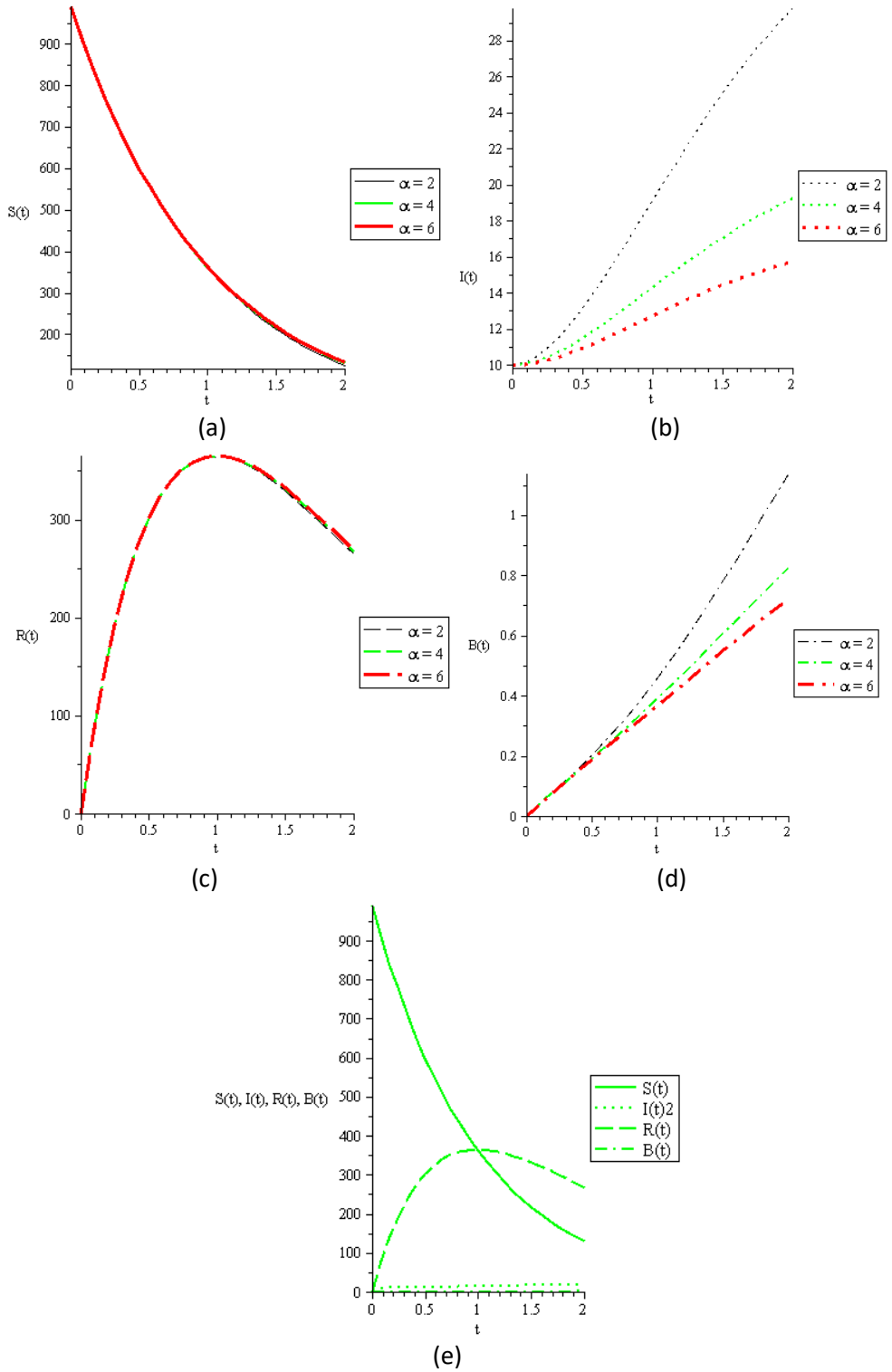


Figure 4 Plots of $S(t)$, $I(t), R(t), B(t)$ against t for different values of α when $\beta = 0.2$, $\lambda = 2, \sigma = 0.04, \eta = 1, \upsilon = 2, \delta = 1$

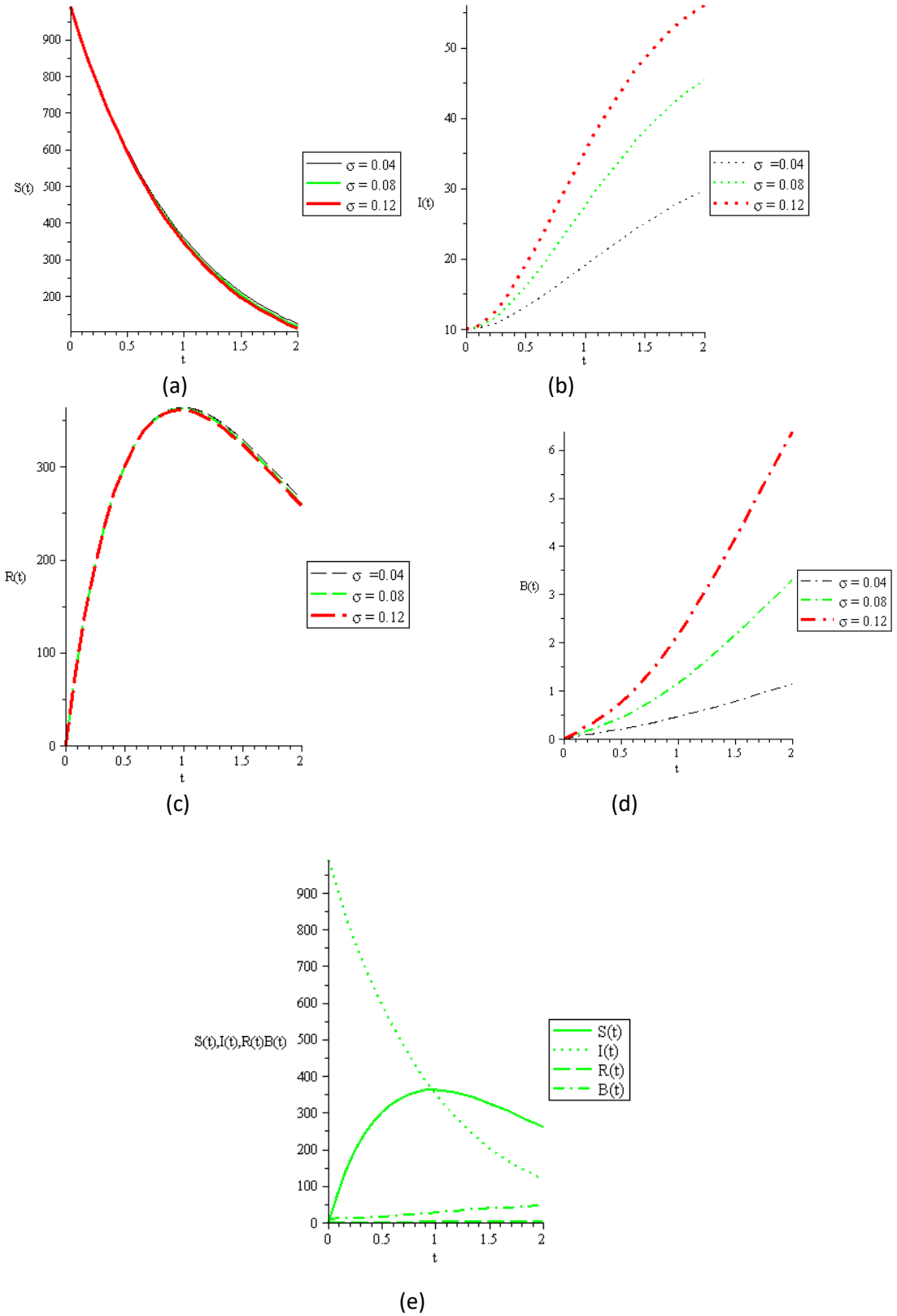


Figure 4 Plots of $S(t),\ I(t), R(t), B(t)$ against t for different values of σ when $\beta = 0.2,\ \lambda = 2, \alpha = 2,\ \eta = 1, \upsilon = 2, \delta = 1$

The phase portrait of the system (6) presented in Figure 1 (a – c) display the trajectory of the system, which give a very good understanding of the qualitative properties of the solutions even without solving. In Figure 2 (a) the number of susceptible human decreases as contact rate with contaminated water parameter β increases. Figure 2 (b) showed that the number of infected human increases as contact rate with contaminated water parameter β increases. While Figure 2 (c) indicates the number of recovered human increases and later decreases as contact rate with contaminated water parameter β increases, Figure 2 (d) showed that the concentration of toxigenic $V.cholerae$ in water increases as contact rate with contaminated water parameter β increases. In Figure 2 (e), we see the interaction between the Susceptible, Infected and Recovered human and the concentration of toxigenic $V.cholerae$ in water against t. Figures 3 (a) – (e) is interpreted thus: the number of susceptible human does not change much as $vibrios$ death rate parameter υ increases, the number of infected human decreases as $V.cholerae$ death rate parameter increases, the number of recovered human increases as $vibrios$ death rate parameter υ increases, the concentration of toxigenic $V.cholerae$ in water decreases as *vibrios* death rate parameter υ increases, the interaction between the susceptible, infected and recovered human and the concentration of toxigenic $V.cholerae$ in water against t, respectively. Similarly, the interpretation of Figures 4 (a) – (e) is made as follow: the number of susceptible human does not change much as the water treatment parameter α increases, the number of infected human decreases as the water treatment parameter α increases, the number of recovered human increases as the water treatment parameter α increases, the concentration of toxigenic $V.cholerae$ in water decreases as the water treatment parameter α increases, the interaction between the Susceptible, Infected and Recovered human and the concentration of toxigenic $V.cholerae$ in water against t, respectively.

And lastly, Figures 5 (a) – (e) showed that: the number of susceptible human decreases as the infected rate parameter σ increases, the number of infected human increases as the infected rate parameter σ increases, the number of recovered human increases as the infected rate parameter σ increases, the concentration of toxigenic $V.cholerae$ in water Increases as the infected rate parameter σ increases, the interaction between the susceptible, infected and recovered human and the concentration of toxigenic $V.cholerae$ in water against $t, respectively.$

CONCLUSION

For the relationship between Susceptible, $S(t)$), Infected, $I(t)$, recovered, $R(t)$ and the concentration of toxigenic $V.cholerae$ in water, $B(t)$, a linear stability analysis was presented. The governing parameters of the problem are the contact rate with contaminated water parameter, β, the water treatment parameter, α, the infected rate parameter, σ, the $vibrios$ death rate υ. The computations were done numerically using the classical Runge Kutta scheme with a modified version of the Newton Raphson shooting method. The result obtained showed that the increment in contact rate with contaminated water parameter β and increment in the infected rate parameter σ increases the rate of infection and the concentration of toxigenic $V.cholerae$ in water, while its increment decreases the susceptible and recovered rate. Similarly, as the water treatment parameter α and the $vibrios$ death rate υ increases, it increases the recovered and does not have much effect on the susceptible human, while it decreases the infected human and the concentration of toxigenic $V.cholerae$ in water.

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