Three Dimensional Epidemic Model with Non-monotonic Incidence and Saturated Treatment: A Case Study of SARS Infection of Hong Kong 2003 Scenario

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Research Article

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Posted Date: March 18th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-310291/v1>

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Epidemic model with non-monotonic incidence and saturated treatment: A case study of SARS infection of Hong Kong 2003 scenario

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Received: date / Accepted: date

Abstract:

In this paper, we have proposed a mathematical compartmental model with non-monotonic incidence and saturated treatment and we have validated the model with SARS infection in Hong Kong, 2003. We have analysed the stability of disease free and endemic equilibria as well as different bifurcations. We have shown that the epidemic disappears if the cure rate of treatment crosses a threshold value. We have obtained a necessary and sufficient condition for backward bifurcation, which shows the basic reproduction number less than unity is not sufficient to eradicate the disease completely. Saddle-node and Hopf bifurcation with respect to awareness factor have been investigated, which shows that the awareness factor is effective to change the disease dynamics. The model has been fitted to SARS cases in Hong Kong. The most effective parameters for controlling infections have been identified through sensitivity analysis. Moreover, we have investigated how the number of infected cases reduces if there was some vaccination policies in SARS infection. Finally, the model has been also used as an optimal control problem as vaccination and treatment controls are time dependent functions.

Keywords: Non-monotonic incidence, Saturated treatment, SARS, MERS, COVID-19, Model validation, Optimal control.
AMS Subject Classification 2010: 37N25, 34C23, 49J15, 92D30.

1 Introduction

In mathematical epidemiology, the popular compartmental models were constructed by Kermack and McKendrick in 1927 [1] by which this subject was raised to a new level and many other biomathematicians have been developing it by introducing new ideas or concepts [2, 3, 4, 10]. In Kermack and McKendrick model, the total population is divided into three compartments, namely $S(t)$, $I(t)$ and $R(t)$, where they are, respectively, the abundance of susceptible, infected and recovered population at time t . The bilinear incidence rate βSI was considered in Kermack and McKendrick model, where the transmission rate of infection β is the product of contact rate c between susceptible and infected individuals and the probability p of transmission of infection through contacts i.e. $\beta = cp$. Here, the infection force $f(I) = \beta I$ is an increasing function of I and $f(0) = 0$. Also, $f(I) \rightarrow \infty$ as $I \rightarrow \infty$. But, the bilinear incidence rate does not include the crowding effects of infected individuals, protection measures and awareness or inhibitory factors, so it is not realistic for many diseases in large population [6, 9, 16]. Capasso and Serio [5] introduced a saturated incidence rate $\frac{\beta SI}{1+\alpha I}$, where β is the transmission rate of infection and α is the inhibitory co-efficient. Here, the infection

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Table 1: Model parameters and their descriptions

Parameters	Interpretations
β	Transmission rate of infection
A	Recruitment rate of the population
α	Parameter measuring inhibitory factors.
d	Natural death rate of the population.
μ	Natural recovery rate of the infected population.
δ	Disease induced death rate.
a	Cure rate of treatment.
b	Delayed parameter of treatment.
u_1	Vaccination control.
u_2	Treatment control.

force $f(I) = \frac{\beta I}{1+\alpha I}$ is also an increasing function of I . Also, $f(0) = 0$ and $f(I)$ tends to a saturation level $\frac{\beta}{\alpha}$ as I tends to infinity. Again, the force of infection decreases as the parameter α increases. In this saturated incidence rate, the parameter α represents the crowding effect of infected individuals or awareness or protection measures taken by susceptible individuals when the number of infected individuals increases. We have seen that the infection forces for both bilinear and saturated incidence rate are monotonic but infection force $f(I)$ may be non-monotonic for some diseases in some countries or communities. As for example, we mention the epidemic outbreak of Severe Acute Respiratory Syndrome (SARS) in 2002-2003 in different parts of World such as China, Hong Kong, Taiwan, Canada, etc. SARS is a contagious disease, it was initiated in China and spread quickly in different parts of Asia, North America and Europe through air travel [17]. It is an infectious disease, has high potential for transmission from infected to susceptible populations through contracts. The etiological agent for SARS is the coronavirus [18]. As the modern pharmaceutical was not able to prevent SARS infection, so it spread highly in the populations. For this disease, initially the incidence rate highly increases as the number of infected increases due to the poor knowledge about the new disease. But, when the disease becomes serious, the aggressive measures and policies are taken by the country to control the disease i.e. then the incidence rate decreases as number of infected individuals increases. To include this intervention policies taken by the countries when a serious disease emerges, Xiao and Ruan [19] proposed a non-monotonic incidence rate of the form $\frac{\beta SI}{1+\alpha I^2}$. Here, the infection force $f(I) = \frac{\beta I}{1+\alpha I^2}$ increases when $0 \leq I \leq \frac{1}{\sqrt{\alpha}}$ and decreases when $I > \frac{1}{\sqrt{\alpha}}$. Also, $f(0) = 0$ and $f(I)$ tends to zero as I tends to infinity. Again, this incidence rate decreases as the parameter α increases. Thus, this incidence rate also includes awareness or protection measures.

On the other hand, for the infectious diseases where the huge number of infections occurs in short span of time the treatment function of the form $T(I) = \frac{au_2 I}{1+bu_2 I}$ is considered, which characterizes the saturation phenomenon due to the limitation of medical resources [6, 7, 8]. In [22], Ghosh *et al* formulated a model by considering saturated incidence and saturated treatment. In this present work, we have reformulated a model just replacing the saturated incidence rate by a non-monotonic incidence rate and all other assumptions remain same as previous. Thus, the proposed model is as follows:

$$\begin{cases} \frac{dS}{dt} &= A - \frac{\beta SI}{1+\alpha I^2} - dS - u_1 S \\ \frac{dI}{dt} &= \frac{\beta SI}{1+\alpha I^2} - (d + \mu + \delta)I - \frac{au_2 I}{1+bu_2 I} \\ \frac{dR}{dt} &= \frac{au_2 I}{1+bu_2 I} + \mu I + u_1 S - dR, \end{cases} \quad (1)$$

where the model parameters have been interpreted in Table 1 and $S(0) > 0$, $I(0) > 0$, $R(0) \geq 0$.

This model is very effective for new emerging diseases like SARS, MERS (Middle East Respiratory Syndrome), COVID-19 (caused by novel coronavirus (SARS-CoV-2)) in some countries or communities. Here, we shall study the qualitative analysis of the proposed model and validate the proposed model with SARS infection cases in Hong Kong, 2003. We shall also deal the proposed model as an optimal control problem. The manuscript is organized in the following way: In Section 2, the positivity and boundedness of the solutions of the model (1) have been established. The stability and bifurcations at different equilibrium points have been analysed in Section 3. The Section 4 is devoted for parameter estimation and model validation. The sensitivity analysis is done in Section 5 and an optimal control problem has been formulated and solved in Section 6. Finally, some conclusions are given in Section 7.

2 Positivity and Boundedness of Solutions

In this section, by showing the positivity and boundedness of the solutions of the system (1) we shall establish the biological validity of the proposed model. More precisely, any solution of that system starting from the positive density level always stays at positive level.

Theorem 1 *All the solutions of the system (1) are feasible and bounded.*

Proof : From the 2nd equation of the system (1), we have

$$I(t) = I(0)e^{\int_0^t \left\{ \frac{\beta S}{1+\alpha I^2} - \frac{au_2}{1+bu_2 I} \right\} dt - (d+\mu+\delta)t}.$$

From other two equations, we have

$$S(t) \geq S(0)e^{-\int_0^t \frac{\beta I}{1+\alpha I^2} dt - (d+u_1)t} \quad \text{and} \quad R(t) \geq R(0)e^{-dt}.$$

Thus, by using initial conditions we can conclude that all the solutions of the system (1) are feasible.

Again, adding all the three equations of the system (1) and using the notation $N(t) = S(t) + I(t) + R(t)$, we get $\frac{dN}{dt} \leq A - dN$ which follows that $\limsup_{t \rightarrow \infty} N(t) \leq \frac{A}{d}$. This shows that all the solutions of the system (1) are ultimately bounded. Hence the theorem is proved.

In the next section, first we shall find the disease free equilibrium point and the basic reproduction number (R_0). Then by using Descartes' rule of signs, we shall investigate the endemic equilibrium points. Then, the characteristic of the equilibrium points for different values of R_0 and different bifurcation analysis will be studied.

3 Equilibria, Stability and Bifurcation Analysis

The system (1) always has the disease free equilibrium point (DFE) $E_0(\frac{A}{d+u_1}, 0, \frac{Au_1}{d(d+u_1)})$. Again, the basic reproduction number R_0 of a system gives a threshold that can be interpreted as the number of secondary infections that one infectious person will produce in a population consisting only of susceptible population. Here, we apply the next-generation approach [11] to determine R_0 . In model (1), the variable I is the only infected compartment and the system (1) has the DFE E_0 . The basic reproduction number R_0 is the spectral radius of the next-generation matrix FV^{-1} [11] with small domain, where $F = \left[\left(\frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} \right)_{1 \times 1} \right]_{at E_0} =$

$$\left(\frac{\beta A}{d+u_1} \right)_{1 \times 1} \quad \text{and} \quad V = \left[\left(d + \mu + \delta + \frac{au_2}{(1+bu_2 I)^2} \right)_{1 \times 1} \right]_{at E_0} = \left(d + \mu + \delta + au_2 \right)_{1 \times 1}.$$

Thus, $R_0 = \rho(FV^{-1}) = \frac{\beta A}{(d+u_1)(d+\mu+\delta+au_2)}$, where ρ is the spectral radius.

Again, the existence of the endemic equilibrium point(s) can be determined by the relation:

$$S = \frac{(1+\alpha I^2)}{\beta} \left\{ (d+\mu+\delta) + \frac{au_2}{1+bu_2 I} \right\} = \frac{A}{\left(\frac{\beta I}{1+\alpha I^2} + d+u_1 \right)}.$$

Thus, the component I of the endemic equilibrium point (S, I, R) must satisfy the following relation:

$$(1+\alpha I^2)(d+\mu+\delta + \frac{au_2}{1+bu_2 I}) \left(\frac{\beta I}{1+\alpha I^2} + d+u_1 \right) - A\beta = 0.$$

After simplification, we can conclude that the endemic equilibrium point(s) $E^*(S^*, I^*, R^*)$ exist(s) if and only if the Eq.(2) has positive root(s).

$$k_0 I^3 + k_1 I^2 + k_2 I + k_3 = 0, \tag{2}$$

where $k_0 = bu_2 \alpha (d+\mu+\delta)(d+u_1)$,

$k_1 = (d+\mu+\delta+au_2)(d+u_1) \left\{ \alpha + \frac{bu_2 R_0 (d+\mu+\delta)}{A} \right\}$,

$k_2 = bu_2 (d+\mu+\delta)(d+u_1) + (d+\mu+\delta+au_2)(d+u_1) R_0 \left\{ \frac{d+\mu+\delta+au_2}{A} - bu_2 \right\}$,

$k_3 = (d+\mu+\delta+au_2)(d+u_1)(1-R_0)$.

Now, by using Descartes' rule of signs we can make following conclusions:

(i) For $R_0 > 1$, the system (1) has unique endemic equilibrium point.

(ii) For $R_0 < 1$, the system (1) has either no endemic equilibrium point or two endemic equilibrium points.

To investigate the stability analysis of equilibrium points, the variational matrix J plays a crucial role. For

the system (1), the variational matrix at the equilibrium point (S, I, R) is given below:

$$J(S, I, R) = \begin{pmatrix} -\frac{\beta I}{1+\alpha I^2} - (d + u_1) & -\frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} & 0 \\ \frac{\beta I}{1+\alpha I^2} & \frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} - (d + \mu + \delta) - \frac{au_2}{(1+bu_2 I)^2} & 0 \\ u_1 & \frac{au_2}{(1+bu_2 I)^2} + \mu & -d \end{pmatrix}. \quad (3)$$

Theorem 2 *The DFE E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof : The eigenvalues of the variational matrix J at DFE E_0 are $-d$, $-(d+u_1)$ and $(d+\mu+\delta+au_2)(R_0-1)$. Thus, E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. Hence the theorem is proved.

Theorem 3 *Suppose that $R_0 < 1$. The DFE E_0 is globally asymptotically stable if the condition $\frac{\beta A}{d} < d + \mu + \delta$ holds.*

Proof : Analysing the variational matrix J of the system (1) at DFE E_0 , we have already established in previous theorem that E_0 is locally asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$. To prove the global stability when $R_0 < 1$, we consider the Liapunov function $L = I$.

$$\begin{aligned} \text{Then } \frac{dL}{dt} &= \left\{ \frac{\beta S}{1+\alpha I^2} - (d + \mu + \delta) - \frac{au_2}{1+bu_2 I} \right\} I \\ &\leq \{ \beta S - (d + \mu + \delta) \} I \\ &\leq \left\{ \frac{\beta A}{d} - (d + \mu + \delta) \right\} I \quad (\text{as } \limsup_{t \rightarrow \infty} N(t) \leq \frac{A}{d}) \end{aligned}$$

$$\leq 0, \text{ if } \frac{\beta A}{d} < d + \mu + \delta.$$

Thus, for a positive definite function L the derivative function $\frac{dL}{dt}$ is negative semi definite if $\frac{\beta A}{d} < d + \mu + \delta$. Now, we consider a subset Ω of $\mathbb{R}_+^3$ where $\frac{dL}{dt} = 0$. Thus, $\Omega = \{(S, I, R) \in \mathbb{R}_+^3 : I = 0\}$. Let Φ be the largest invariant set in Ω . Then in Ω , we have

$$\begin{cases} \frac{dS}{dt} = A - (d + u_1)S, \\ \frac{dR}{dt} = u_1 S - dR. \end{cases} \quad (4)$$

From first equation of Eq.(4), we have $S \rightarrow \frac{A}{d+u_1}$ as $t \rightarrow \infty$. So, from second equation of Eq.(4), we also get $R \rightarrow \frac{Au_1}{d(d+u_1)}$ as $t \rightarrow \infty$. Hence, Φ is singleton $\left\{ \left(\frac{A}{d+u_1}, 0, \frac{Au_1}{d(d+u_1)} \right) \right\}$. Then, by applying LaSalle-Liapunov theory [23], we can conclude that E_0 is globally asymptotically stable if the condition $\frac{\beta A}{d} < d + \mu + \delta$ holds. Hence the theorem is proved.

Since for $R_0 = 1$ i.e. $a = \frac{\beta A}{u_2(d+u_1)} - \frac{d+\mu+\delta}{u_2} \equiv a^{[TR]}$, one eigenvalue of the variational matrix at DFE is zero and other two are negative real, hence the eigen analysis method fails. In the next theorem, we shall establish the existence of transcritical bifurcation when the cure rate of treatment crosses the critical value $a = a^{[TR]}$, which means that there is an exchange of stability that takes place at the disease free equilibrium point of the system (1) at the bifurcation value $a = a^{[TR]}$.

Theorem 4 *The system (1) experiences the transcritical bifurcation at the DFE E_0 with respect to the cure rate of treatment a if the condition $\beta A > (d + u_1)(d + \mu + \delta)$ holds.*

$$\text{Proof : Let } f(S, I, R; a) = \begin{pmatrix} A - \frac{\beta SI}{1+\alpha I^2} - dS - u_1 S \\ \frac{\beta SI}{1+\alpha I^2} - (d + \mu + \delta)I - \frac{au_2 I}{1+bu_2 I} \\ \frac{au_2 I}{1+bu_2 I} + \mu I + u_1 S - dR \end{pmatrix}.$$

The bifurcation value $a^{[TR]}$ is positive by the given condition, otherwise it has no biological sense.

$$\text{Thus, } Df(E_0, a^{[TR]}) = \begin{pmatrix} -(d + u_1) & -\frac{\beta A}{d+u_1} & 0 \\ 0 & 0 & 0 \\ u_1 & \frac{\beta A}{d+u_1} - (d + \delta) & -d \end{pmatrix} \text{ and } f(E_0, a^{[TR]}) = 0. \text{ Obviously, } Df(E_0, a^{[TR]})$$

has a simple eigenvalue $\lambda = 0$. Therefore, we can apply Sotomayor theorem [12] to determine the direction of

the bifurcation at DFE E_0 . Now, $V = \begin{pmatrix} \beta A \\ -(d + u_1)^2 \\ \frac{(d+\delta)(d+u_1)^2 + \beta Au_1 - \beta A(d+u_1)}{d} \end{pmatrix}$ and $W = \begin{pmatrix} 0 \\ 1 \\ 0 \end{pmatrix}$ are, respectively,

the eigenvector of $Df(E_0, a^{[TR]})$ and $(Df(E_0, a^{[TR]}))^T$ corresponding to the eigenvalue $\lambda = 0$. Let, f_a

denote the vector of partial derivatives of the components of f with respect of a . Thus, $f_a = \begin{pmatrix} 0 \\ -\frac{u_2 I}{(1+bu_2 I)} \\ \frac{u_2 I}{(1+bu_2 I)} \end{pmatrix}$

and so $f_a(E_0, a^{[TR]}) = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}$.

Therefore, $W^T f_a(E_0, a^{[TR]}) = \mathbf{0}$,

$W^T (Df_a(E_0, a^{[TR]})V) = (u_2(d + u_1)^2) \neq \mathbf{0}$, and

$W^T (D^2 f(E_0, a^{[TR]})(V, V)) = -2(d + u_1)^2 (\beta^2 A + bu_2(d + \mu + \delta)(d + u_1)^2 - bu_2\beta A(d + u_1)) \neq \mathbf{0}$.
($\mathbf{0}$ denotes the null matrix).

Therefore, all the conditions for transcritical bifurcation in Sotomayor theorem have been satisfied. Hence, the system (1) exhibits the transcritical bifurcation at the equilibrium point E_0 when the cure rate of treatment a crosses the bifurcation value $a = a^{[TR]}$. Hence the theorem is proved.

The unstable DFE becomes stable if the cure rate a in the treatment function crosses the threshold value $a = a^{[TR]}$. Epidemiologically, the cure rate of treatment plays key role to control the epidemic.

Fig. 1: Backward bifurcation diagram for the parametric values $\beta = 0.15, \alpha = 0.7, d = 0.02, \mu = 0.01, \delta = 0.01, a = 4, b = 1.8, u_1 = 0.5, u_2 = 0.7$. The red and blue lines, respectively, represent the lines of unstable and stable equilibrium points.

Theorem 5 *The system (1) undergoes the backward bifurcation at $R_0 = 1$ if and only if $(d + \mu + \delta + au_2)^2 < Aabu_2^2$.*

Proof : The infected component I of the endemic equilibrium point (S, I, R) satisfies the Eq.(2). If we consider I as a function of R_0 , then the slope of the curve $I = I(R_0)$ must be negative at the point $(R_0, I) = (1, 0)$ to occur backward bifurcation. Now, differentiating Eq.(2) implicitly with respect to R_0 , we obtain

$$\left[\frac{\partial I}{\partial R_0} \right]_{R_0=1, I=0} = \frac{A(d + \mu + \delta + au_2)}{(d + \mu + \delta + au_2)^2 - Aabu_2^2}.$$

Therefore, the system (1) experiences a backward bifurcation at $R_0 = 1$ if and only if the value of the slope $\left[\frac{\partial I}{\partial R_0} \right]_{R_0=1, I=0}$ of the curve $I = I(R_0)$ is less than zero.

Hence the theorem is proved.

From the above theorem it is clear that if $b = 0$, then there can not be backward bifurcation. Epidemiologically, the phenomenon of backward bifurcation occurs due to saturated treatment. So, there is a positive number $R_0^* < 1$ for which two endemic equilibria exist for $R_0^* < R_0 < 1$ and these two endemic equilibria coincide for $R_0 = R_0^*$, which has been illustrated by the backward bifurcation diagram (Fig.1). Now, we shall focus on the stability analysis of the endemic equilibria for different values of R_0 and we shall prove in the following theorem that the bi-stability occurs for $R_0^* < R_0 < 1$.

Theorem 6 If $R_0 > 1$, then the system (1) has unique endemic equilibrium point $E_1^*(S_1^*, I_1^*, R_1^*)$ which is locally asymptotically stable if $\beta \geq \max\{abu_2^2, \frac{a\alpha}{b}\}$. If $R_0 < 1$ and $(d + \mu + \delta + au_2)^2 > Aabu_2^2$, then the system (1) has no endemic equilibrium point. If $R_0^* < R_0 < 1$ and $(d + \mu + \delta + au_2)^2 < Aabu_2^2$, then the system (1) has two endemic equilibria $E_2^*(S_2^*, I_2^*, R_2^*)$ and $E_3^*(S_3^*, I_3^*, R_3^*)$. The equilibrium point with the smaller number of infective, I_2^* , is unstable, while other equilibrium point, with higher number of infective, I_3^* , is locally asymptotically stable if $\beta \geq \max\{abu_2^2, \frac{a\alpha}{b}\}$.

Proof : The characteristic equation of the variational matrix (3) at any endemic equilibrium point (S, I, R) is

$$\begin{vmatrix} -\frac{\beta I}{1+\alpha I^2} - (d + u_1 + \lambda) & -\frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} & 0 \\ \frac{\beta I}{1+\alpha I^2} & \frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} - (d + \mu + \delta) - \frac{au_2}{(1+bu_2 I)^2} - \lambda & 0 \\ u_1 & \frac{au_2}{(1+bu_2 I)^2} + \mu & -(d + \lambda) \end{vmatrix} = 0,$$

or equivalently,

$$(d + \lambda) \begin{vmatrix} -\frac{\beta I}{1+\alpha I^2} - (d + u_1 + \lambda) & -\frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} \\ \frac{\beta I}{1+\alpha I^2} & \frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} - (d + \mu + \delta) - \frac{au_2}{(1+bu_2 I)^2} - \lambda \end{vmatrix} = 0.$$

Thus, $-d$ is one of the three eigenvalues of the variational matrix and the remaining eigenvalues are the solutions of the equation $G(\lambda) = 0$,

where

$$G(\lambda) \equiv \begin{vmatrix} -\frac{\beta I}{1+\alpha I^2} - (d + u_1 + \lambda) & -\frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} \\ \frac{\beta I}{1+\alpha I^2} & \frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} - (d + \mu + \delta) - \frac{au_2}{(1+bu_2 I)^2} - \lambda \end{vmatrix}.$$

Then,

$$G(0) = \begin{vmatrix} -\frac{\beta I}{1+\alpha I^2} - (d + u_1) & -\frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} \\ \frac{\beta I}{1+\alpha I^2} & \frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} - (d + \mu + \delta) - \frac{au_2}{(1+bu_2 I)^2} \end{vmatrix}.$$

The existence of the endemic equilibrium point(s) can be determined by the relation

$$S = \frac{(1 + \alpha I^2)}{\beta} \left\{ (d + \mu + \delta) + \frac{au_2}{1 + bu_2 I} \right\} = \frac{A}{\left(\frac{\beta I}{1+\alpha I^2} + d + u_1 \right)}.$$

Thus, the component I of the endemic equilibrium point(s) is the solution of the equation $H(I) \equiv \frac{A}{\beta I + (d + u_1)(1 + \alpha I^2)} - \frac{au_2}{\beta(1 + bu_2 I)} - \frac{d + \mu + \delta}{\beta} = 0$. Since $H(0) = \left(\frac{d + \mu + \delta + au_2}{\beta} \right)(R_0 - 1)$, hence $R_0 > 1$ if and only if $H(0) > 0$ and $R_0 < 1$ if and only if $H(0) < 0$. Now, we have to establish a relation between $G(0)$ and $H'(I)$ to make conclusions.

Now, $H'(I) \equiv \frac{abu_2^2}{\beta(1 + bu_2 I)^2} - \frac{A\{\beta + 2\alpha(d + u_1)I\}}{\{\beta I + (1 + \alpha I^2)(d + u_1)\}^2}$ and so

$$\begin{aligned} G(0) &= \begin{vmatrix} -\frac{\beta I}{1+\alpha I^2} - (d + u_1) & -\frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} \\ \frac{\beta I}{1+\alpha I^2} & \frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} - (d + \mu + \delta) - \frac{au_2}{(1+bu_2 I)^2} \end{vmatrix} \quad (\text{By using } d + \mu + \delta = \frac{\beta S}{1+\alpha I^2} - \frac{au_2}{1+bu_2 I}) \\ &= \begin{vmatrix} -\frac{\beta I}{1+\alpha I^2} - (d + u_1) & -\frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} \\ -(d + u_1) & \frac{abu_2^2 I}{(1+bu_2 I)^2} - \frac{\beta S}{1+\alpha I^2} \end{vmatrix} \quad (\text{By applying the row operation } R_2' = R_2 + R_1) \\ &= \left(-\frac{A}{S}\right) \begin{vmatrix} 1 & \frac{\beta A(1-\alpha I^2)}{\{\beta I + (d + u_1)(1 + \alpha I^2)\}^2} \\ -(d + u_1) & \frac{abu_2^2 I}{(1+bu_2 I)^2} - \frac{\beta S}{1+\alpha I^2} \end{vmatrix} \quad (\text{By using } \frac{A}{S} = \frac{\beta I}{1+\alpha I^2} + d + u_1, S = \frac{A(1+\alpha I^2)}{\beta I + (d + u_1)(1 + \alpha I^2)}) \\ &= \left(-\frac{A}{S}\right) \begin{vmatrix} 1 & \frac{\beta A(1-\alpha I^2)}{\{\beta I + (d + u_1)(1 + \alpha I^2)\}^2} \\ -(d + u_1) & \frac{abu_2^2 I}{(1+bu_2 I)^2} - \frac{\beta A}{\beta I + (d + u_1)(1 + \alpha I^2)} \end{vmatrix} = \left(-\frac{\beta A I}{S}\right) H'(I). \end{aligned}$$

Thus, we have

$G(0) > 0$ if and only if $H'(I) < 0$,

$G(0) = 0$ if and only if $H'(I) = 0$, and

$G(0) < 0$ if and only if $H'(I) > 0$.

Here, we assume that all the roots of $H(I) = 0$ are simple. Because for some root with higher multiplicity, $H'(I)$ must be zero and so $G(0) = 0$, which implies that one eigenvalue of the variational matrix is zero and so the stability or instability of that non-hyperbolic equilibrium point can not be determined easily.

Fig. 2: The function $H(I)$ for : (a) $R_0 > 1$ (Case I), (b) $R_0^* < R_0 < 1, (d + \mu + \delta + au_2)^2 < Aabu_2^2$ (Subcase II of Case II).

Now, two cases shall be discussed below.

Case I: In this case we suppose that $R_0 > 1$. At the beginning of Section 3, it has been already established by applying Descartes' rule of signs that the system (1) has only one endemic equilibrium point $E_1^*(S_1^*, I_1^*, R_1^*)$ for $R_0 > 1$. Here, we investigate the stability analysis of that unique endemic equilibrium point E_1^* . Since $R_0 > 1$, hence $H(0) > 0$. Thus, $H(I)$ should decrease to cross the x-axis (see Fig.2(a)). Thus, $H'(I_1^*) < 0$ and so $G(0) > 0$ for E_1^* . Again, we know that one of the eigenvalues of the variational matrix is $-d$ and the remaining eigenvalues are the solutions of the equation $G(\lambda) = 0$ i.e. the equation $\lambda^2 + C_1\lambda + C_2 = 0$, where $C_1 = 2d + \mu + \delta + u_1 + \frac{au_2}{(1+bu_2I)^2} + \frac{\beta I}{1+\alpha I^2} - \frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2}$ and $C_2 = G(0) > 0$. After simplification C_1 can be written as

$$C_1 = d + u_1 + \frac{2\alpha\beta SI^2}{(1+\alpha I^2)^2} + \frac{I}{(1+\alpha I^2)(1+bu_2I)^2} \{(\beta - abu_2^2) + 2\beta bu_2I + (\beta b^2u_2^2 - \alpha abu_2^2)I^2\}.$$

Thus, $C_1 > 0$ if the condition $\beta \geq \max\{abu_2^2, \frac{\alpha\alpha}{b}\}$ holds. Hence by applying Routh-Hurwitz criterion on algebraic equation, we can conclude that all the eigenvalues of the variational matrix have negative real parts. Therefore, the unique endemic equilibrium point $E_1^*(S_1^*, I_1^*, R_1^*)$ is asymptotically stable if $\beta \geq \max\{abu_2^2, \frac{\alpha\alpha}{b}\}$.

Case II: In this case we suppose that $R_0 < 1$. There are two subcases.

Subcase I: Suppose $R_0 < 1$ and $(d + \mu + \delta + au_2)^2 > Aabu_2^2$. It follows from backward bifurcation theorem (Theorem 5) that the system (1) has no endemic equilibrium point.

Subcase II: Suppose $R_0^* < R_0 < 1$ and $(d + \mu + \delta + au_2)^2 < Aabu_2^2$. Then, the backward bifurcation theorem also implies that the system (1) has two endemic equilibria $E_2^*(S_2^*, I_2^*, R_2^*)$ and $E_3^*(S_3^*, I_3^*, R_3^*)$ with $I_2^* < I_3^*$. Since $R_0^* < R_0 < 1$, hence $H(0) < 0$. So, the function $H(I)$ must increase to cross the x-axis and then decrease to cross it again (see Fig.2(b)). Therefore, $H'(I_2^*) > 0$ and $H'(I_3^*) < 0$. In this subcase, we have reached following two conclusions.

(1) For the endemic equilibrium point $E_2^*(S_2^*, I_2^*, R_2^*)$, we have $H'(I_2^*) > 0$. So, for E_2^* $G(0) < 0$. Again, $\lim_{\lambda \rightarrow \infty} G(\lambda) = \infty$ as $\lambda \rightarrow \infty$. Since $G(\lambda)$ is a continuous function of λ , hence it follows from Bolzano theorem on continuous function that $G(\lambda_i) = 0$ for some $\lambda_i > 0$. So, at least one eigenvalue of the variational matrix is positive. Therefore, $E_2^*(S_2^*, I_2^*, R_2^*)$ is unstable.

(2) For the endemic equilibrium point $E_3^*(S_3^*, I_3^*, R_3^*)$, we have $H'(I_3^*) < 0$ and so for E_3^* $G(0) > 0$. Thus, we proceed same as Case I and derive that $E_3^*(S_3^*, I_3^*, R_3^*)$ is asymptotically stable if $\beta \geq \max\{abu_2^2, \frac{\alpha\alpha}{b}\}$. Hence the theorem is proved.

It is clear from the bifurcation diagram (Fig.1) that when $R_0 < 1$ then the disease free equilibrium point is stable and as R_0 crosses the critical value $R_0 = 1$ then it becomes unstable. The phenomenon of backward bifurcation is epidemiologically important because in this case for $R_0 < 1$ there exists at least one stable endemic equilibrium point. Thus, eradication of the disease is not only dependent on the condition $R_0 < 1$ but also on the initial number of infected populations.

We know that the system (1) has two endemic equilibrium points for $R_0^* < R_0 < 1$ (see Fig.1), which means that the Eq.(2) has two positive roots for $R_0^* < R_0 < 1$. Suppose that two positive roots of the Eq.(2) coincide at $\alpha = \alpha^{[SN]}$ and the corresponding coincident equilibrium point is $E^*(S^*, I^*, R^*)$.

Theorem 7 The system (1) undergoes the saddle-node bifurcation at coincident endemic equilibrium point $E^*(S^*, I^*, R^*)$ as the inhibitory factor α passes through the bifurcation value $\alpha = \alpha^{[SN]}$.

Proof : Let $f(S, I, R; \alpha) = \begin{pmatrix} A - \frac{\beta SI}{1+\alpha I^2} - dS - u_1 S \\ \frac{\beta SI}{1+\alpha I^2} - (d + \mu + \delta)I - \frac{au_2 I}{1+bu_2 I} \\ \frac{au_2 I}{1+bu_2 I} + \mu I + u_1 S - dR \end{pmatrix}$.

From (3) we have one eigenvalue of the variational matrix at any equilibrium point (S, I, R) is $-d$ and other eigenvalues are the roots of the following equation:

$$\lambda^2 - \text{Trace}(M)\lambda + \text{Det}(M) = 0,$$

where $M = \begin{pmatrix} -\frac{\beta I}{1+\alpha I^2} - (d + u_1) & -\frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} \\ \frac{\beta I}{1+\alpha I^2} & \frac{\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} - (d + \mu + \delta) - \frac{au_2}{(1+bu_2 I)^2} \end{pmatrix}$.

We choose the critical value $\alpha^{[SN]}$ of the inhibitory factor α for which two roots of the Eq.(2) coincide and $\text{Det}(M) = 0$ and $\text{Trace}(M) \neq 0$ for $\alpha = \alpha^{[SN]}$ and for the coincident endemic equilibrium point $E^*(S^*, I^*, R^*)$.

Clearly, $f(E^*, \alpha^{[SN]}) = 0$ and $Df(E^*, \alpha^{[SN]})$ has a simple zero eigenvalue. So, we can apply Sotomayor the-

orem [12] to check the nature of solutions near the equilibrium point E^* . Let, $V = \begin{pmatrix} v_1 \\ v_2 \\ v_3 \end{pmatrix}$ and $W = \begin{pmatrix} w_1 \\ w_2 \\ w_3 \end{pmatrix}$

be, respectively, the eigenvector of $Df(E^*, \alpha^{[SN]})$ and $(Df(E^*, \alpha^{[SN]}))^T$ corresponding to the eigenvalue $\lambda = 0$, where $v_1 = 1$,

$$v_2 = -\frac{(1+\alpha^{[SN]}I^{*2})^2}{\beta S^*(1-\alpha^{[SN]}I^{*2})} \left\{ \frac{\beta I^*}{1+\alpha^{[SN]}I^{*2}} + (d+u_1) \right\}, v_3 = \frac{1}{d} \left[u_1 - \frac{(1+\alpha^{[SN]}I^{*2})^2}{\beta S^*(1-\alpha^{[SN]}I^{*2})} \left\{ \frac{\beta I^*}{1+\alpha^{[SN]}I^{*2}} + (d+u_1) \right\} \left\{ \frac{au_2}{(1+bu_2 I^*)^2} + \mu \right\} \right], w_1 = 1, w_2 = \frac{(1+\alpha^{[SN]}I^{*2})^2}{\beta I^*} \left\{ \frac{\beta I^*}{1+\alpha^{[SN]}I^{*2}} + (d+u_1) \right\} \text{ and } w_3 = 0.$$

Let, f_α denote the vector of partial derivatives of the components of f with respect to α . Thus $f_\alpha = \begin{pmatrix} \frac{\beta SI^3}{(1+\alpha I^2)^2} \\ -\frac{\beta SI^3}{(1+\alpha I^2)^2} \\ 0 \end{pmatrix}$ and so $f_\alpha(E^*, \alpha^{[SN]}) =$

$$\frac{\beta S^* I^{*3}}{(1+\alpha^{[SN]}I^{*2})^2} \begin{pmatrix} 1 \\ -1 \\ 0 \end{pmatrix}.$$

Then, $W^T f_\alpha(E^*, \alpha^{[SN]}) = \left(\frac{(1-w_2)\beta S^* I^{*3}}{(1+\alpha^{[SN]}I^{*2})^2} \right) \neq 0$ and

$$W^T (D^2 f(E^*, \alpha^{[SN]})(V, V)) = \begin{pmatrix} \frac{2\alpha^{[SN]}\beta S^* I^*(3-\alpha^{[SN]}I^{*2})v_2^2}{(1+\alpha^{[SN]}I^{*2})^3} - \frac{2\beta(1-\alpha^{[SN]}I^{*2})v_2}{(1+\alpha^{[SN]}I^{*2})^2} + \frac{2\beta(1-\alpha^{[SN]}I^{*2})w_2 v_2}{(1+\alpha^{[SN]}I^{*2})^2} \\ + \frac{2abu_2^2 v_2^2 w_2}{(1+bu_2 I^*)^3} - \frac{2\alpha^{[SN]}\beta S^* I^*(3-\alpha^{[SN]}I^{*2})v_2^2 w_2}{(1+\alpha^{[SN]}I^{*2})^3} \end{pmatrix} \neq 0$$

Therefore, by Sotomayor theorem [12] we can say that the system (1) experiences a saddle-node bifurcation at the endemic equilibrium point $E^*(S^*, I^*, R^*)$ when the parameter α passes through $\alpha = \alpha^{[SN]}$.

Hence the theorem is proved.

From Figure 3, we see that when the inhibitory or awareness factor α touches a threshold value $\alpha^{[SN]}$ then two endemic equilibrium points coincide and when α crosses $\alpha^{[SN]}$ then they disappear which shows that the awareness factor plays very important role to control the disease.

Note: An equilibrium point of the system (1) is called degenerate equilibrium point of the system (1) if at least one eigenvalue of the variational matrix J corresponding to the system (1) at that equilibrium point is zero [12]. From above definition of degenerate equilibrium point, we see that the model (1) has a degenerate equilibrium point at $R_0 = R_0^* < 1$. It has been established that the system (1) exhibits the backward and saddle-node bifurcation around the degenerate equilibrium point. One can check whether the system undergoes Bogdanov-Takens bifurcation [24, 25] around the degenerate equilibrium point or not.

Theorem 8 The system (1) experiences the Hopf bifurcation leading to a family of periodic solutions that bifurcates from the endemic equilibrium point $E^*(S^*, I^*, R^*)$ for suitable value of inhibitory factor α in a neighbourhood of the critical value $\alpha^{[HB]}$. In addition, if $\Gamma < 0$ then it is supercritical and if $\Gamma > 0$ then it is subcritical, where Γ is defined in the text.

Fig. 3: Saddle-node bifurcation diagram with respect to α for the parametric values $A = 8.039, \beta = 0.15, d = 0.02, \mu = 0.01, \delta = 0.01, a = 4, b = 1.8, u_1 = 0.5, u_2 = 0.7$. The red and blue lines, respectively, denote the lines of unstable and stable equilibrium points.

Proof : We know that the characteristic equation of the variational matrix of the system (1) at any equilibrium point (S, I, R) is $(d + \lambda)Det(M - \lambda I_2) = 0$, where the matrix M has been defined in previous theorem. Thus, one of the three eigenvalues of the variational matrix at that equilibrium point is $-d$ and other two are the roots of the equation $\lambda^2 - Trace(M)\lambda + Det(M) = 0$. Since $Trace(M)$ and $Det(M)$ are dependent on α , hence we may choose the critical value $\alpha^{[HB]}$ of α in such a manner that $Trace(M) = 0$ and $Det(M) > 0$ at some endemic equilibrium point $E^*(S^*, I^*, R^*)$. Then two eigenvalues of $M(S^*, I^*, R^*)$ are $\lambda = \pm \sqrt{Det(M(S^*, I^*, R^*))}i \equiv \pm \sqrt{D}i$. Now, for $\alpha \in (\alpha^{[HB]} - \varepsilon, \alpha^{[HB]} + \varepsilon)$, the roots are, in general, of the form $\chi_{1,2}(\alpha) = \eta(\alpha) \pm i\kappa(\alpha)$, where η and κ are real. To apply the Hopf bifurcation theorem [12], we need to verify the transversality condition $Re[\frac{d\chi_i}{d\alpha}]_{\alpha=\alpha^{[HB]}} \neq 0, i = 1, 2$.

Now, we translate the equilibrium point $E^*(S^*, I^*, R^*)$ to the origin. So, we put $S' = S - S^*, I' = I - I^*$ and $R' = R - R^*$ in (1) and we get (omitting the dash sign)

$$\frac{dX}{dt} = EX + F(X), \quad (5)$$

$$E = \begin{pmatrix} a_{11} & a_{12} & 0 \\ a_{21} & a_{22} & 0 \\ a_{31} & a_{32} & a_{33} \end{pmatrix}, X = \begin{pmatrix} S \\ I \\ R \end{pmatrix}, F(X) = \begin{pmatrix} a_{13}SI + a_{14}I^2 \\ a_{23}SI + a_{24}I^2 \\ a_{34}I^2 \end{pmatrix}, \text{ where}$$

$$a_{11} = -\frac{\beta I^*}{\alpha I^{*2}+1} - d - u_1, a_{12} = \frac{2\alpha\beta S^* I^{*2}}{\alpha I^{*2}+1} - \beta S^*, a_{13} = \frac{2\alpha\beta I^* (\alpha I^{*3} + I^*)}{(\alpha I^{*2}+1)^2} - \beta, a_{14} = \frac{2\alpha\beta I^* S^* (1 - \alpha I^{*2})}{(\alpha I^{*2}+1)^2} + \frac{\beta\alpha S^* I^*}{\alpha I^{*2}+1}, a_{21} = \frac{\beta I^*}{\alpha I^{*2}+1}, a_{22} = \frac{\beta S^* - 2\alpha\beta S^* I^{*2}}{\alpha I^{*2}+1} - d - \mu - \delta - \frac{au_2 - \frac{abu_2^2 I^*}{1+bu_2 I^*}}{1+bu_2 I^*}, a_{23} = \frac{\beta - 2\alpha\beta I^* (\alpha I^{*3} + I^*)}{(\alpha I^{*2}+1)^2}, a_{24} = -\frac{2\alpha\beta I^* S^* (1 - \alpha I^{*2})}{(\alpha I^{*2}+1)^2} + \frac{\beta\alpha S^* I^*}{\alpha I^{*2}+1} + \frac{abu_2^2}{(1+bu_2 I^*)^3}, a_{31} = u_1, a_{32} = \mu + \frac{au_2 - \frac{abu_2^2 I^*}{1+bu_2 I^*}}{1+bu_2 I^*}, a_{33} = -d, a_{34} = -\frac{abu_2^2}{(1+bu_2 I^*)^3}.$$

We know that the eigenvalues of the matrix E are $\lambda = \pm \sqrt{D}i, -d$ at $\alpha = \alpha^{[HB]}$. An eigenvector of E corresponding to $\lambda = \sqrt{D}i$ is $\begin{pmatrix} \alpha_1 \\ \alpha_2 \\ \alpha_3 \end{pmatrix} + i \begin{pmatrix} \beta_1 \\ \beta_2 \\ \beta_3 \end{pmatrix}$ and corresponding to the eigenvalue $\lambda = -d$ is $\begin{pmatrix} \gamma_1 \\ \gamma_2 \\ \gamma_3 \end{pmatrix}$, where $\alpha_1 = 1, \alpha_2 = -\frac{a_{11}}{a_{12}}, \alpha_3 = \frac{Da_{32}+a_{33}(a_{32}a_{11}-a_{31}a_{12})}{a_{12}(D+a_{33}^2)}, \beta_1 = 0, \beta_2 = \frac{\sqrt{D}}{a_{12}}, \beta_3 = \frac{\sqrt{D}(a_{32}a_{11}-a_{31}a_{12}-a_{33}a_{32})}{a_{12}(D+a_{33}^2)}, \gamma_1 = 1, \gamma_2 = -\frac{a_{11}+d}{a_{12}}, \gamma_3 = \frac{(a_{11}+d)a_{32}-a_{31}a_{12}}{a_{12}(d+a_{33})}$.

Now, by the transformation $X = PY$, where $P = \begin{pmatrix} \beta_1 & \alpha_1 & \gamma_1 \\ \beta_2 & \alpha_2 & \gamma_2 \\ \beta_3 & \alpha_3 & \gamma_3 \end{pmatrix}, Y = \begin{pmatrix} y_1 \\ y_2 \\ y_3 \end{pmatrix}$, the system (5) is transformed

to

$$\begin{pmatrix} \dot{y}_1 \\ \dot{y}_2 \\ \dot{y}_3 \end{pmatrix} = \begin{pmatrix} 0 & -\sqrt{D} & 0 \\ \sqrt{D} & 0 & 0 \\ 0 & 0 & -d \end{pmatrix} \begin{pmatrix} y_1 \\ y_2 \\ y_3 \end{pmatrix} + \begin{pmatrix} f^1 \\ f^2 \\ f^3 \end{pmatrix}. \quad (6)$$

Here, $f^i(y_1, y_2, y_3) \equiv p_{i1}\{a_{13}(\beta_1 y_1 + \alpha_1 y_2 + \gamma_1 y_3)(\beta_2 y_1 + \alpha_2 y_2 + \gamma_2 y_3) + a_{14}(\beta_2 y_1 + \alpha_2 y_2 + \gamma_2 y_3)^2\} + p_{i2}\{a_{23}(\beta_1 y_1 + \alpha_1 y_2 + \gamma_1 y_3)(\beta_2 y_1 + \alpha_2 y_2 + \gamma_2 y_3) + a_{24}(\beta_2 y_1 + \alpha_2 y_2 + \gamma_2 y_3)^2\} + p_{i3}a_{34}(\beta_2 y_1 + \alpha_2 y_2 + \gamma_2 y_3)^2$ ($i = 1, 2, 3$), where $P^{-1} = (p_{ij})_{3 \times 3}$.

By the Centre Manifold Theory there exists a centre manifold of (6), which can be expressed by $W^c(0) = \{(y_1, y_2, y_3) : y_3 = h(y_1, y_2), |y_1| < \delta, |y_2| < \delta, \delta > 0\}$. Thus, $y_3 = h(y_1, y_2) = s_1 y_1^2 + s_2 y_1 y_2 + s_3 y_2^2 + \dots$, where the constants $s_i (i = 1, 2, 3, \dots)$ can be easily determined from the following identity:

$$(2s_1 y_1 + s_2 y_2 + \dots)\{-\sqrt{D} y_2 + f^1(y_1, y_2, s_1 y_1^2 + s_2 y_1 y_2 + s_3 y_2^2 + \dots)\} + (s_2 y_1 + 2s_3 y_2 + \dots)\{\sqrt{D} y_1 + f^2(y_1, y_2, s_1 y_1^2 + s_2 y_1 y_2 + s_3 y_2^2 + \dots)\} + d \cdot (s_1 y_1^2 + s_2 y_1 y_2 + s_3 y_2^2 + \dots) - f^3(y_1, y_2, s_1 y_1^2 + s_2 y_1 y_2 + s_3 y_2^2 + \dots) = 0.$$

The expressions of the constants $s_i (i = 1, 2, 3)$ have been given in Appendix II. Thus, the flow on the center manifold is of the following form

$$\begin{pmatrix} \dot{y}_1 \\ \dot{y}_2 \end{pmatrix} = \begin{pmatrix} 0 & -\sqrt{D} \\ \sqrt{D} & 0 \end{pmatrix} \begin{pmatrix} y_1 \\ y_2 \end{pmatrix} + \begin{pmatrix} g^1 \\ g^2 \end{pmatrix}, \quad (7)$$

where $g^1(y_1, y_2) \equiv f^1(y_1, y_2, h(y_1, y_2))$, $g^2(y_1, y_2) \equiv f^2(y_1, y_2, h(y_1, y_2))$.

Therefore, we can easily compute the index Γ , which is as follows:

$$\begin{aligned} \Gamma &= \frac{1}{16} (g_{y_1 y_1 y_1}^1 + g_{y_1 y_2 y_2}^1 + g_{y_1 y_1 y_2}^2 + g_{y_2 y_2 y_2}^2) \\ &\quad + \frac{1}{16\sqrt{D}} \{g_{y_1 y_2}^1 (g_{y_1 y_1}^1 + g_{y_2 y_2}^1) - g_{y_1 y_2}^2 (g_{y_1 y_1}^2 + g_{y_2 y_2}^2) - g_{y_1 y_1}^1 g_{y_1 y_1}^2 + g_{y_2 y_2}^1 g_{y_2 y_2}^2\} \\ &= \frac{1}{8} A_{12} + \frac{3}{8} A_{30} + \frac{3}{8} B_{03} + \frac{1}{8} B_{21} + \frac{1}{16\sqrt{D}} \{A_{11}(2A_{02} + 2A_{20}) - B_{11}(2B_{02} + 2B_{20}) - 4A_{20}B_{20} + 4B_{02}A_{02}\}. \end{aligned}$$

The expressions of $A_{20}, A_{11}, A_{02}, A_{30}, A_{12}, B_{20}, B_{11}, B_{02}, B_{21}, B_{03}$ have been given in Appendix II. By using Hopf bifurcation theorem, we obtain that Hopf bifurcation is supercritical if $\Gamma < 0$ and subcritical if $\Gamma > 0$. Hence the theorem is proved.

To verify the Hopf bifurcation theorem, numerically, we consider $\beta = 0.35, u_1 = 0.25, d = 0.1, \delta = 0.2, b = 5.0, \mu = 0.6, u_2 = 0.37, a = 6.5, A = 3.25$ and vary α . For $\alpha = 0.25$ one characteristic root of the variational matrix at the endemic equilibrium point is negative and other two have negative real parts and so the equilibrium point is stable focus (see Fig.4(a)). On the other hand if we choose $\alpha = 0.12$ then one characteristic root at the endemic equilibrium is negative real and other two have positive real parts, hence the solution is unstable spiral in the neighbourhood of that equilibrium point and a stable limit cycle arises here (see Fig.4(b - c)). Thus the system exhibits subcritical Hopf bifurcation with respect to the awareness parameter for considered parametric values.

4 Parameter Estimation and Model Validation

In previous section, we discussed the qualitative analysis of the considered model (1). Now, we shall validate the model numerically with the real data. For this purpose, we have considered the reported SARS infection data for 88 days in Hong Kong from 11th March to 6th June, 2003 [20]. After the fitting model with data, we have compared the cumulative number of real infected cases with the predicted cumulative cases. To calculate the expected number of infected cases from the model we have used the formula $C(t) = \int_0^t \frac{\beta SI}{1 + \alpha I^2} dt$ [20]. The best fitted model parameters are obtained when the sum of squares error (SSE) in the considered time span is minimum. We have fitted the model (1) with the SARS infection data in Hong Kong by using MATLAB minimization software package fmincon, which has been shown by Figure 5 and Figure 6. In order to fit the model (1) with the data, we have considered that the initial number of susceptible, infected and recovered population are 6800000, 2, 0, respectively. Among the eight model parameters, we have estimated five parameters. The estimated parametric values with other values have been summarized in Table 2.

Since to estimate the model parameters we have considered $u_1 = 0$ as there was no proper vaccination at the time of SARS infection in Hong Kong in 2003. But if there was vaccination strategies then the disease could be very easy to control which has been justified by Figure 7. In Figure 7 the dots denote the number of real infected cases in Hong Kong and the red, black, cyan and magenta coloured lines, respectively,

Fig. 4: Phase portrait for (a) $\alpha = 0.25$, where the endemic equilibrium point is stable focus, (b) $\alpha = 0.12$, where the endemic equilibrium point is unstable focus & a stable limit cycle arises and (c) limit cycle in Fig (b), where other parametric values are $\beta = 0.35, u_1 = 0.25, d = 0.1, \delta = 0.2, b = 5.0, \mu = 0.6, u_2 = 0.37, a = 6.5, A = 3.25$.

Table 2: Values of the model parameters

Parameter	Value	Source
A	0.8264162687	Estimated
β	$1.246876836 \times 10^{-7}$	Estimated
α	$7.03479319 \times 10^{-6}$	Estimated
d	0.045	Assume
μ	0.06	Assume
δ	0.00002	Assume
a	8.5027018775	Estimated
b	4.0897875	Estimated
u_1	0	Assume
u_2	0.17	Assume

represent the predicted number infected populations in the considered time domain for $u_1 = 0, 0.005, 0.01$ and 0.015 . It is clear from the figure that the vaccination strategy is the key policy to reduce the infections significantly.

5 Sensitivity Analysis

In this section, we shall perform the sensitivity analysis to determine the parameters that have high or low impact of influence on basic reproduction number R_0 . Again, the basic reproduction number R_0 of the model plays very important role to eradicate the disease. For the large value of R_0 , the controlling of spread of disease is very difficult whereas low value of R_0 ($R_0 < 1$) indicates the eradication of infection of

Fig. 5: Bar diagram for SARS cases, Hong Kong, March-June, 2003 and the predictions for the model.

Fig. 6: (a) The reported SARS cases (the blue dots) and the best-fitted curve (solid red curve), (b) the cumulative number of SARS cases (the blue dots) and the best fitted solution curve (solid red curve), (c) the residuals are randomly distributed.

Fig. 7: Time series of infected population for different values of u_1 . Red, black, cyan and magenta colour curves have been generated for $u_1 = 0$, $u_1 = 0.005$, $u_1 = 0.01$, $u_1 = 0.015$, respectively.

the disease. Thus, the main objective of performing sensitivity analysis is to identify the model parameters that have high or low influence on spread of disease and therefore it helps the public health authorities for preventing or controlling the spread of disease. This type of information are also important for experimental design and data assimilation. For this purpose we shall use normalized forward sensitivity index for the basic reproduction number R_0 with respect to the model parameters. The normalized forward sensitivity index ($\gamma_r^{R_0}$) with respect to the characteristic model parameter (r) is defined as $\gamma_r^{R_0} = \frac{r}{R_0} \frac{\partial R_0}{\partial r}$ [21]. A little variation in the highly sensitive parameters will highly effect on the basic reproduction number and consequently effect the disease dynamics. In Figure 8, we have presented the sensitivity indices of R_0 with respect to the model parameters. It is clear from the Figure 8 that β , A , d , a and u_2 are the high sensitive parameters whereas μ , δ are low sensitive parameters. It is obvious from the figure that as the parameters β , A increase the basic reproduction number also increases and hence the possibility of stability of DFE decreases. On the other hand with the increase of d , a and u_2 the basic reproduction number decreases and consequently the possibility of stability of DFE increases. Thus to reduce the epidemic the cure rate of treatment, treatment control parameter and the natural death rate play important role whereas to enhance infections the transmission rate of infection and the growth rate of population plays key role.

To realize the effects of sensitive model parameters, numerically, we plot the time series of the infected populations for different values of the sensitive parameters with base values of the parameters given in Table 2 with the initial populations $S(0) = 6800000$, $I(0) = 2$, $R(0) = 0$ (see Figure 9). It is clear from the figure that with the increase of the transmission rate of infection (β) the number of infected populations increases. On the other hand the parameters d , a and u_2 have opposite effects i.e. with decrease of these parameters the infected populations increase.

6 Optimal Control Problem

Now, we shall deal with the model (1) as an optimal control problem by considering vaccination control u_1 and treatment control u_2 as the function of time t . Previously, we considered fixed value of control parameter u_1 and u_2 for analysing qualitative behaviour of the system (1), but in reality, they are time dependent as they are applied according to the necessity. Our goal is to minimize the total loss that occurs due to infection and the cost of vaccination and treatment. Here, we construct the objective functional to be minimized as follows:

$$J(u_1, u_2) = \int_0^T (A_1 I + B_1 u_1^2 + B_2 u_2^2) dt,$$

Fig. 8: Sensitivity indices of R_0 corresponding to the model parameters for the parametric values given in Table 2.

Fig. 9: The effects of the model parameters: (a) β , (b) a , (c) u_2 and (d) d .

Fig. 10: Time series of: (a) susceptible individuals, (b) infected individuals, (c) recovered individuals with control ($u_1 \neq 0, u_2 \neq 0$) and without control ($u_1 = 0, u_2 = 0$).

where the constant A_1 represents the per capita loss due to presence of infected population at any time instant t . Also, the constants B_1 and B_2 are, respectively, the costs associated with vaccination of susceptible individuals and treatment of infected individuals. We also assume that the time interval is $[0, T]$, where T is fixed. The problem is to find optimal functions $u_1^*(t)$ and $u_2^*(t)$ satisfying $J(u_1^*, u_2^*) = \min\{J(u_1, u_2), (u_1, u_2) \in U\}$, where the control set $U = \{(u_1, u_2)/u_i(t) \text{ is Lebesgue measurable on } [0, 1], 0 \leq u_1(t), u_2(t) \leq 1 \text{ for } t \in [0, T]\}$.

Theorem 9 *The control functions u_1^* and u_2^* exist for which $J(u_1^*, u_2^*) = \min\{J(u_1, u_2) : (u_1, u_2) \in U\}$.*

Proof : Since the constants B_1 and B_2 are positive, hence the integrand of the objective functional $J(u_1, u_2)$ is a convex function of u_1 and u_2 . Also, from Theorem 1 we can conclude that the system (1) satisfies the Lipschitz property with respect to the variables S, I and R . Therefore, there are optimal functions u_1^* and u_2^* for the system (1). Hence the theorem is proved.

Theorem 10 *The optimal controls u_1^* and u_2^* which minimize the objective functional J over the region U are given by $u_1^* = \max\{0, \min\{\frac{(\lambda_1^* - \lambda_3^*)S^*}{2B_1}, 1\}\}$ and $u_2^* = \max\{0, \min\{\bar{u}_2, 1\}\}$, where $\bar{u}_2$ is the non-negative root of the equation $2B_2b^2I^{*2}u_2^3 + 4B_2bI^*u_2^2 + 2B_2u_2 + aI^*(\lambda_3^* - \lambda_2^*) = 0$. Here, S^*, I^*, R^* are, respectively, the optimum values of S, I, R and $(\lambda_1^*, \lambda_2^*, \lambda_3^*)$ is the solution of the system (8) with the conditions (9).*

Proof : Here, the Lagrangian of the problem is $L = A_1I + B_1u_1^2 + B_2u_2^2$.

Now, we define the Hamiltonian H for the problem which is given by

$$H(S, I, R, u_1, u_2, \lambda_1, \lambda_2, \lambda_3) = A_1I + B_1u_1^2 + B_2u_2^2 + \lambda_1(t)\{A - \frac{\beta SI}{1+\alpha I^2} - dS - u_1S\} + \lambda_2(t)\{\frac{\beta SI}{1+\alpha I^2} - (d + \mu + \delta)I - \frac{au_2I}{1+bu_2I}\} + \lambda_3(t)\{\frac{au_2I}{1+bu_2I} + \mu I + u_1S - dR\}.$$

In order to determine the adjoint equations and transversality conditions, we apply Pontryagin's Maximum Principle [15, 13, 22, 14] which gives $\frac{d\lambda_1(t)}{dt} = -\frac{\partial H}{\partial S}$, $\frac{d\lambda_2(t)}{dt} = -\frac{\partial H}{\partial I}$, $\frac{d\lambda_3(t)}{dt} = -\frac{\partial H}{\partial R}$, with the transversality conditions $\lambda_i(T) = 0, i = 1, 2, 3$. Thus, the adjoint variables λ_1, λ_2 and λ_3 can be obtained from the following:

$$\begin{cases} \frac{d\lambda_1}{dt} &= \frac{(\lambda_1 - \lambda_2)\beta I}{1+\alpha I^2} + d\lambda_1 + u_1(\lambda_1 - \lambda_3) \\ \frac{d\lambda_2}{dt} &= -A_1 + \frac{(\lambda_1 - \lambda_2)\beta S(1-\alpha I^2)}{(1+\alpha I^2)^2} + \frac{(\lambda_2 - \lambda_3)au_2}{(1+bu_2I)^2} + (d + \delta)\lambda_2 + \mu(\lambda_2 - \lambda_3) \\ \frac{d\lambda_3}{dt} &= d\lambda_3 \end{cases} \quad (8)$$

and

$$\lambda_1(T) = 0, \lambda_2(T) = 0, \lambda_3(T) = 0. \quad (9)$$

In order to determine optimal control functions, we use the optimality conditions $\frac{\partial H}{\partial u_1} = 0$ and $\frac{\partial H}{\partial u_2} = 0$ and we get $u_1 = \frac{(\lambda_1 - \lambda_3)S}{2B_1}$ and $2B_2b^2I^2u_2^3 + 4B_2bIu_2^2 + 2B_2u_2 + aI(\lambda_3 - \lambda_2) = 0$. Thus, the optimal control functions are $u_1^* = \max\{0, \min\{\frac{(\lambda_1^* - \lambda_3^*)S^*}{2B_1}, 1\}\}$ and $u_2^* = \max\{0, \min\{\bar{u}_2, 1\}\}$, where $\bar{u}_2$ is the non-negative root of the equation $2B_2b^2I^{*2}u_2^3 + 4B_2bI^*u_2^2 + 2B_2u_2 + aI^*(\lambda_3^* - \lambda_2^*) = 0$ and (S^*, I^*, R^*) are the optimum value of (S, I, R) . Also, $(\lambda_1^*, \lambda_2^*, \lambda_3^*)$ is the solution of the system (8) satisfying the transversality conditions (9). Hence the theorem is proved.

Fig. 11: Time series of the control function: (a) $u_1(t)$, (b) $u_2(t)$.

Fig. 12: Time series of infected individuals in presence of both vaccination & treatment controls (i.e. $u_1 \neq 0, u_2 \neq 0$, blue line) and only for treatment control ($u_1 = 0, u_2 \neq 0$, red line).

Now, we have used forward-backward sweep method to solve the optimal control problem (1) numerically. We have performed the simulation with a set of parametric values given in Table 2. For this simulation, we assume that $A_1 = 0.025$, $B_1 = 0.001$, $B_2 = 0.01$ and the initial conditions $S(0) = 6800000$, $I(0) = 2$, $R(0) = 0$. Here, we also assume that $T = 1$. So, the controls vaccination and treatment have been applied for 1 unit of time and after that time both the controls are stopped. For these parametric values and initial conditions, the corresponding optimal control functions are given in Figure 11. From Figure 10, it is clear that the vaccination and treatment play crucial role to control disease. Again, Figure 12 represents the comparative study between the effect of both vaccination & treatment and the effect of only treatment.

7 Conclusions

This manuscript deals with a SIR epidemic model with a non-monotonic incidence and saturated treatment that characterizes the awareness factors, crowding effects, limited medical facilities, and intervention policies when a new threatening disease emerges in some countries or communities. Here, we have analysed the local as well as the global stability of the disease free equilibrium point. The DFE is locally asymptotically stable if the basic reproduction number R_0 is less than unity and if the transmission rate of infection is less than some quantity then the DFE is globally asymptotically stable for $R_0 < 1$. The existence and stability of endemic equilibria have been investigated for each values of R_0 . The system undergoes a transcritical bifurcation with respect to the cure rate of treatment, which shows the effects of cure rate for controlling the epidemic. A necessary and sufficient condition for backward bifurcation has been obtained, which proves

that the phenomenon of backward bifurcation has been occurred due to saturated treatment. When $R_0 < 1$ then the bistability exists if the condition for backward bifurcation holds. Epidemiologically, making R_0 less than unity is not enough to eradicate the disease completely. The system also experiences saddle-node and Hopf bifurcation with respect to the awareness or inhibitory factor, that shows what role does the awareness play in changing the dynamics the disease.

The model has also been fitted with the SARS infection cases in Hong Kong, 2003 to study the SARS dynamics in the country and to validate our proposed model with real data. For the best-fitted parametric values, the necessary and sufficient condition for backward bifurcation does not hold, thus we can conclude that for SARS infection in Hong Kong, 2003 the phenomenon of backward bifurcation did not occur. We have predicted the number of infected populations if there was some vaccine policies for SARS. It has been identified from sensitivity analysis that the transmission rate, recruitment rate, natural death rate, cure rate and treatment control parameter are the most influential parameters to control the disease.

The proposed model has also been used as an optimal control problem as the vaccination and treatment controls are the functions of time. The optimal vaccine and treatment to minimize the infection as well as the costs for implementing the controls have been obtained both analytically and numerically. This optimal control modelling is a theoretical modelling and it can be further applied for controlling some infectious diseases in some countries or communities.

Conflict of interest:

The authors declare that they have no conflict of interest.

Appendix I

Theorem (Sotomayor) [12]. Let, the solution set of the dynamical system $\frac{dx}{dt} = f(x, \mu)$, depending on a parameter $\mu \in \mathbb{R}$, change as the parameter μ varies through a bifurcation value μ_0 . Suppose that $f(x_0, \mu_0) = 0$ and the matrix $Df(x_0, \mu_0)$ has a simple eigenvalue $\lambda = 0$ with eigenvector V and that the matrix $(Df(x_0, \mu_0))^T$ has an eigenvector W corresponding to the eigenvalue $\lambda = 0$.

The dynamical system experiences a saddle-node bifurcation at the equilibrium point x_0 as the parameter μ passes through the bifurcation value $\mu = \mu_0$ if the conditions $W^T f_\mu(x_0, \mu_0) \neq 0$ and $W^T (D^2 f(x_0, \mu_0)(V, V)) \neq 0$ are satisfied.

Again, the system experiences a transcritical bifurcation at the equilibrium point x_0 as the parameter μ passes through the bifurcation value $\mu = \mu_0$ if the conditions $W^T f_\mu(x_0, \mu_0) = 0$, $W^T (Df_\mu(x_0, \mu_0)V) \neq 0$, and $W^T (D^2 f(x_0, \mu_0)(V, V)) \neq 0$ are satisfied. Here, f_μ denotes the vector of partial derivatives of the components of f with respect to μ .

Appendix II

$$\begin{aligned}
s_1 &= (2Da_{13}\alpha_1\alpha_2p_{31} + 2Da_{13}\beta_1\beta_2p_{31} + 2Da_{14}\alpha_2^2p_{31} + 2Da_{14}\beta_2^2p_{31} + 2Da_{23}\alpha_1\alpha_2p_{32} + 2Da_{23}\beta_1\beta_2p_{32} + \\
&2Da_{24}\alpha_2^2p_{32} + 2Da_{24}\beta_2^2p_{32} + 2Da_{34}\alpha_2^2p_{33} + 2Da_{34}\beta_2^2p_{33} - \sqrt{D}a_{13}\alpha_1\beta_2dp_{31} - \sqrt{D}a_{13}\alpha_2\beta_1dp_{31} - 2\sqrt{D}a_{14}\alpha_2\beta_2dp_{31} - \\
&\sqrt{D}a_{23}\alpha_1\beta_2dp_{32} - \sqrt{D}a_{23}\alpha_2\beta_1dp_{32} - 2\sqrt{D}a_{24}\alpha_2\beta_2dp_{32} - 2\sqrt{D}a_{34}\alpha_2\beta_2dp_{33} + a_{13}\beta_1\beta_2d^2p_{31} + a_{14}\beta_2^2d^2p_{31} + \\
&a_{23}\beta_1\beta_2d^2p_{32} + a_{24}\beta_2^2d^2p_{32} + a_{34}\beta_2^2d^2p_{33})/((d^2 + 4D)d), \\
s_2 &= -(2\sqrt{D}a_{13}\alpha_1\alpha_2p_{31} - 2\sqrt{D}a_{13}\beta_1\beta_2p_{31} + 2\sqrt{D}a_{14}\alpha_2^2p_{31} - 2\sqrt{D}a_{14}\beta_2^2p_{31} + 2\sqrt{D}a_{23}\alpha_1\alpha_2p_{32} - 2\sqrt{D}a_{23}\beta_1\beta_2p_{32} + \\
&2\sqrt{D}a_{24}\alpha_2^2p_{32} - 2\sqrt{D}a_{24}\beta_2^2p_{32} + 2\sqrt{D}a_{34}\alpha_2^2p_{33} - 2\sqrt{D}a_{34}\beta_2^2p_{33} - da_{13}\alpha_1\beta_2p_{31} - da_{13}\alpha_2\beta_1p_{31} - 2da_{14}\alpha_2\beta_2p_{31} - \\
&da_{23}\alpha_1\beta_2p_{32} - da_{23}\alpha_2\beta_1p_{32} - 2da_{24}\alpha_2\beta_2p_{32} - 2dp_{33}a_{34}\alpha_2\beta_2)/(d^2 + 4D), \\
s_3 &= 2Da_{13}\alpha_1\alpha_2p_{31} + 2Da_{13}\beta_1\beta_2p_{31} + 2Da_{14}\alpha_2^2p_{31} + 2Da_{14}\beta_2^2p_{31} + 2Da_{23}\alpha_1\alpha_2p_{32} + 2Da_{23}\beta_1\beta_2p_{32} + \\
&2Da_{24}\alpha_2^2p_{32} + 2Da_{24}\beta_2^2p_{32} + 2Da_{34}\alpha_2^2p_{33} + 2Da_{34}\beta_2^2p_{33} + \sqrt{D}a_{13}\alpha_1\beta_2dp_{31} + \sqrt{D}a_{13}\alpha_2\beta_1dp_{31} + 2\sqrt{D}a_{14}\alpha_2\beta_2dp_{31} + \\
&\sqrt{D}a_{23}\alpha_1\beta_2dp_{32} + \sqrt{D}a_{23}\alpha_2\beta_1dp_{32} + 2\sqrt{D}a_{24}\alpha_2\beta_2dp_{32} + 2\sqrt{D}a_{34}\alpha_2\beta_2dp_{33} + a_{13}\alpha_1\alpha_2d^2p_{31} + a_{14}\alpha_2^2d^2p_{31} + \\
&a_{23}\alpha_1\alpha_2d^2p_{32} + a_{24}\alpha_2^2d^2p_{32} + a_{34}\alpha_2^2d^2p_{33})/((d^2 + 4D)d). \\
A_{20} &= p_{11}(a_{13}\beta_1\beta_2 + a_{14}\beta_2^2) + p_{12}(a_{23}\beta_1\beta_2 + a_{24}\beta_2^2) + p_{13}a_{34}\beta_2^2, \\
A_{11} &= p_{11}(a_{13}\alpha_1\beta_2 + a_{13}\alpha_2\beta_1 + 2a_{14}\alpha_2\beta_2) + p_{12}(a_{23}\alpha_1\beta_2 + a_{23}\alpha_2\beta_1 + 2a_{24}\alpha_2\beta_2) + 2p_{13}a_{34}\alpha_2\beta_2, \\
A_{02} &= p_{11}(a_{13}\alpha_1\alpha_2 + a_{14}\alpha_2^2) + p_{12}(a_{23}\alpha_1\alpha_2 + a_{24}\alpha_2^2) + p_{13}a_{34}\alpha_2^2, \\
A_{30} &= p_{11}(a_{13}\beta_1\gamma_2s_1 + a_{13}\beta_2\gamma_1s_1 + 2a_{14}\beta_2\gamma_2s_1) + p_{12}(a_{23}\beta_1\gamma_2s_1 + a_{23}\beta_2\gamma_1s_1 + 2a_{24}\beta_2\gamma_2s_1) + 2p_{13}a_{34}\beta_2\gamma_2s_1, \\
A_{12} &= p_{11}(a_{13}\alpha_1\gamma_2s_2 + a_{13}\alpha_2\gamma_1s_2 + a_{13}\beta_1\gamma_2s_3 + a_{13}\beta_2\gamma_1s_3 + 2a_{14}\alpha_2\gamma_2s_2 + 2a_{14}\beta_2\gamma_2s_3) + p_{12}(a_{23}\alpha_1\gamma_2s_2 + \\
&a_{23}\alpha_2\gamma_1s_2 + a_{23}\beta_1\gamma_2s_3 + a_{23}\beta_2\gamma_1s_3 + 2a_{24}\alpha_2\gamma_2s_2 + 2a_{24}\beta_2\gamma_2s_3) + 2p_{13}a_{34}\alpha_2\gamma_2s_2 + 2p_{13}a_{34}\beta_2\gamma_2s_3, \\
B_{20} &= p_{21}(a_{13}\beta_1\beta_2 + a_{14}\beta_2^2) + p_{22}(a_{23}\beta_1\beta_2 + a_{24}\beta_2^2) + p_{23}a_{34}\beta_2^2, \\
B_{11} &= p_{21}(a_{13}\alpha_1\beta_2 + a_{13}\alpha_2\beta_1 + 2a_{14}\alpha_2\beta_2) + p_{22}(a_{23}\alpha_1\beta_2 + a_{23}\alpha_2\beta_1 + 2a_{24}\alpha_2\beta_2) + 2p_{23}a_{34}\alpha_2\beta_2, \\
B_{02} &= p_{21}(a_{13}\alpha_1\alpha_2 + a_{14}\alpha_2^2) + p_{22}(a_{23}\alpha_1\alpha_2 + a_{24}\alpha_2^2) + p_{23}a_{34}\alpha_2^2,
\end{aligned}$$

$$\begin{aligned}
B_{21} &= p_{21}(a_{13}\alpha_1\gamma_2s_1 + a_{13}\alpha_2\gamma_1s_1 + a_{13}\beta_1\gamma_2s_2 + a_{13}\beta_2\gamma_1s_2 + 2a_{14}\alpha_2\gamma_2s_1 + 2a_{14}\beta_2\gamma_2s_2) + p_{22}(a_{23}\alpha_1\gamma_2s_1 + \\
&a_{23}\alpha_2\gamma_1s_1 + a_{23}\beta_1\gamma_2s_2 + a_{23}\beta_2\gamma_1s_2 + 2a_{24}\alpha_2\gamma_2s_1 + 2a_{24}\beta_2\gamma_2s_2) + 2p_{23}a_{34}\alpha_2\gamma_2s_1 + 2p_{23}a_{34}\beta_2\gamma_2s_2, \\
B_{03} &= p_{21}(a_{13}\alpha_1\gamma_2s_3 + a_{13}\alpha_2\gamma_1s_3 + 2a_{14}\alpha_2\gamma_2s_3) + p_{22}(a_{23}\alpha_1\gamma_2s_3 + a_{23}\alpha_2\gamma_1s_3 + 2a_{24}\alpha_2\gamma_2s_3) + 2p_{23}a_{34}\alpha_2\gamma_2s_3.
\end{aligned}$$

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Figures

Figure 1

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(a)

(b)

Figure 2

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Figure 3

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Figure 4

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Figure 5

Bar diagram for SARS cases, Hong Kong, March-June, 2003 and the predictions for the model.

Figure 6

(a) The reported SARS cases (the blue dots) and the best-fitted curve (solid red curve), (b) the cumulative number of SARS cases (the blue dots) and the best fitted solution curve (solid red curve), (c) the residuals are randomly distributed.

Figure 7

Time series of infected population for different values of u_1 . Red, black, cyan and magenta colour curves have been generated for $u_1 = 0$, $u_1 = 0.005$, $u_1 = 0.01$, $u_1 = 0.015$, respectively.

Figure 8

Sensitivity indices of R_0 corresponding to the model parameters for the parametric values given in Table 2.

Figure 9

The effects of the model parameters: (a) β , (b) a , (c) u_2 and (d) d .

Figure 10

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Figure 11

Time series of the control function: (a) $u_1(t)$, (b) $u_2(t)$.

Figure 12

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