

The Impact of Media on the Control of Infectious Diseases

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We develop a three dimensional compartmental model to investigate the impact of media coverage to the spread and control of infectious diseases (such as SARS) in a given region/area. Stability analysis of the model shows that the disease-free equilibrium is globally-asymptotically stable if a certain threshold quantity, the basic reproduction number (\mathbb{R}_0), is less than unity. On the other hand, if $\mathbb{R}_0 > 1$, it is shown that a unique endemic equilibrium appears and a Hopf bifurcation can occur which causes oscillatory phenomena. The model may have up to three positive equilibria. Numerical simulations suggest that when $\mathbb{R}_0 > 1$ and the media impact is stronger enough, the model exhibits multiple positive equilibria which poses challenge to the prediction and control of the outbreaks of infectious diseases.

KEY WORDS: Infectious disease; SEI model; media impact; Hopf bifurcation; multiple outbreaks.

1. INTRODUCTION

In recent years, attempts have been made to develop realistic mathematical models for the transmission dynamics of infectious diseases. In modelling of communicable diseases, the incidence function has been considered to play a key role in ensuring that the models indeed give

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reasonable qualitative description of the transmission dynamics of the diseases [3,9]. Some factors, such as media coverage, density of population and life style, may affect the incidence rate directly or indirectly.

In the classical endemic models, the incidence rate is assumed to be mass action incidence with bilinear interactions given by βSI , where β is the probability of transmission per contact (a positive constant), and S and I represent the susceptible and infected populations, respectively. However, there are several reasons for using non-linear incidence rates such as saturating and nearly bilinear. For instance, Yorke and London [20] showed that the incidence rate $\beta(1-cI)IS$ with positive C and time dependent β is consistent with the results of the simulations for measles outbreaks. In order to avoid the unboundedness of the contact rate, Capasso and Serio [4] used a saturated incidence function of the form $\frac{\beta SI}{1+\beta\delta I}$, $\delta > 0$. To incorporate the effect of the behavioral changes of the susceptible individuals, Liu and coworkers [10,11] used a non-linear incidence rate given by $\frac{kI^l S}{1+\alpha I^h}$ with $k, l, \alpha, h > 0$. Ruan and Wang, [14] showed that endemic models with such non-linear incidence rates exhibit various bifurcations include Hopf, homoclinic, and even Bogdanov-Takens bifurcations. There have been many models using variety of different non-linear incidence functions to study the disease transmission, we refer the reader to Levin et al. [9] for a more detailed summarization.

The aim of this paper is to investigate the impact of media coverage to the spread and control of infectious diseases in a given region. In [12], the authors consider a model with the compartments of exposed (E), infectious (I) and hospitalized (H) individuals to explore the possible mechanism for multiple outbreaks of emerging infectious diseases due to the psychological impact of the reported numbers of infectious and hospitalized individuals. The model was simplified by assuming that the total population size remain a constant. In this paper, we extend the classical SEI model and the ideas in [12] to consider a new incidence functional which reflects the impact of the media coverage to the spreading and control of the disease.

This study was also originated from the observation of the spread of SARS coronavirus in Asia and some other regions of the world. SARS [7,15,19] as a new emerging infection disease, it was first appeared in Guangdong province, China in November, 2002. Then in the following year the SARS coronavirus spread rapidly throughout Asia and certain other part of the world [16,18]. For SARS in the cities of Beijing, Hongkong and Toronto, the spreading and outbreaks all experienced a typical process for people to see how the media coverage and the public alerting plays a role in the whole course of the spreading. For the case

in Beijing, it was not clear of the existence and type of such disease until April 21, 2003 [16]. During this period, more susceptible individuals might have been exposed to and infected with the disease unconsciously due to the lack of knowledge of the disease. This fact suggests us to consider the following question: *How does the media coverage affect the spreading and control of the infectious diseases like SARS?*

The media coverage is obviously not the most important factor responsible for the transmission of the infectious disease, but it is a very important issue which has to be taken care of seriously. In the case of a large number of infected cases, on one hand, the media coverage may cause the panic of the society, while on the other hand, it can certainly reduce the opportunity and probability of contact transmission among the alerted susceptible populations, which in turn helps to control and prevent the disease from further spreading.

In this paper, we use a compartmental model to address the impact of media coverage on the transmission of infectious diseases. In the SEI model, the incidence rate is assumed to be in the form μe^{-mI} . This paper is organized as follows. In Section 2 we develop a SEI model to incorporate the media impact to the spreading of the infectious diseases such as SARS. We calculate the reproduction number in Section 3 and prove the local and global stability of the disease free equilibrium. The model in general can have up to three positive equilibria, we shall restrict ourselves to the case when the media impact is small enough that there exists at most one endemic equilibrium. In Section 5 we shall study the local and global stability of the unique endemic equilibrium when the reproduction number $\mathbb{R}_0 > 1$ and m is small. We also study the Hopf bifurcation of the endemic equilibrium when the reproduction number is larger enough. The paper ends with a brief discussion of the results on the impact of media and related control and prediction issues.

2. A SEI MODEL WITH MEDIA IMPACT

Consider the transmission of certain infectious disease (such as SARS) in a given region/area. We classify the population into the following categories:

- $S(t)$, the number of susceptible individuals;
- $E(t)$, the number of individuals exposed to the infected but not infectious;
- $I(t)$, the infected who are infectious.

We assume that the infectious individuals I receive medical treatment in hospital settings as soon as they are identified from the category of

exposed. Once they are recovered, they no longer impose risk to the susceptible individuals. In most of the studies, the compartmental models were built by either assuming the total population to be a constant or satisfy exponential growth [1, 2, 5, 8]. It is more reasonable to assume that the population of a given region obey the Logistic growth. Then we have the model

$$\begin{cases} \frac{dS}{dt} = bS \left(1 - \frac{S}{K}\right) - \mu e^{-mI} SI, \\ \frac{dE}{dt} = \mu e^{-mI} SI - (c + d)E, \\ \frac{dI}{dt} = cE - \gamma I, \end{cases} \quad (2.1)$$

where all the parameters are positive, and

- b , the intrinsic growth rate of the human population, K is the carrying capacity for the human population of a given region/area.
- $\beta(I) = \mu e^{-mI}$ is the contact and transmission term, it measures the spreading of the virus from the infected to the susceptible individual. If $m = 0$, the transmission rate is a constant. Naturally the contact transmission rate is not only related to the spreading ability of the virus or disease, but also closely related to the alertness to the disease of each susceptible individual of the population. Here we use the parameter $m > 0$ to reflect the impact of media coverage to the contact transmission. Since the media coverage and alertness are not the intrinsic deterministic factor responsible for the transmission, hence it is reasonable to assume that $m > 0$ is a small parameter. Also for simplicity, the mass action law is assumed in the model [8]. As one can see that if $m > 0$ but comparatively small enough, this incidence term $\beta(I)$ is close to the constant μ . Also as $m > 0$ increases or the media coverage and the alertness to the public is comprehensive and in time, the general public will be more alert and aware of the virus/diseases. Hence the transmission rate will be decreasing as I increases.
- c is the rate per unit time (day) that infected individuals become infectious.
- d is natural death rate for the susceptible population.
- γ is the removed rate from the infected compartment, which include the recovery rate of the hospitalized infectious individuals and natural death. Hence we have $\gamma > d$.

Model (2.1) involves the interaction of both the population dynamics of logistic type and the transmission dynamics of disease epidemiology. Hence the dynamics of the system (2.1) can be very complicated. In this paper, we are going to study the impact of the media coverage/alert to the

spreading of the disease by assuming that $m > 0$ is small. We will show that if the media coverage fails to report the real situation of disease to alert and educate the public, then there will be an outbreak or even multiple outbreaks of such a disease.

3. DISEASE FREE EQUILIBRIUM (DFE), STABILITY AND REPRODUCTION NUMBER

Let the right hand side of (2.1) be zero, one can verify that the origin $E_0 = (0, 0, 0)$ is an equilibrium with eigenvalues $b, -(c+d), -\gamma$. Hence E_0 is a hyperbolic saddle point.

The model (2.1) has one disease free equilibrium (DFE) at $E_{10} = (K, 0, 0)$. The local stability of E_{10} can be obtained through a straightforward calculation for the eigenvalues.

It follows from [17] that for the compartmental models, the local stability of the disease free equilibrium is governed by the reproduction number of the model. Using the notations in [17], we have two vectors \mathbb{F} and \mathbb{V} to represent the new infection term and remaining transfer terms, respectively:

$$\mathbb{F} = \begin{pmatrix} \mu e^{-mI} SI \\ 0 \\ 0 \end{pmatrix}, \quad \mathbb{V} = \begin{pmatrix} (c+d)E \\ -cE + \gamma I \\ -bS(1 - \frac{S}{K}) + \mu e^{-mI} SI \end{pmatrix}. \quad (3.1)$$

The infected compartments are E and I , hence a straightforward calculation gives

$$F = \begin{pmatrix} 0 & \mu K \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} (c+d) & 0 \\ -c & \gamma \end{pmatrix}, \quad (3.2)$$

where F is non-negative and V is a non-singular M-matrix, therefore FV^{-1} is non-negative, and

$$FV^{-1} = \frac{1}{\gamma(c+d)} \begin{pmatrix} \mu K c & \mu K(c+d) \\ 0 & 0 \end{pmatrix}. \quad (3.3)$$

Hence the reproduction number is given by $\rho(FV^{-1})$, and

$$\mathbb{R}_0 = \frac{\mu c K}{\gamma(c+d)}. \quad (3.4)$$

It follows from [17] that we have

Proposition 3.1. *For the model (2.1), the disease free equilibrium E_{10} is locally asymptotically stable if $\mathbb{R}_0 < 1$, and unstable if $\mathbb{R}_0 > 1$.*

Note that for the characteristic equation of (2.1) at E_{10}

$$(\lambda + b)[\lambda^2 + (c + d + \gamma)\lambda + \gamma(c + d) - \mu c K] = 0, \quad (3.5)$$

it follows from the Routh-Hurwitz criteria [13] that all the eigenvalues have negative real parts if and only if $\mathbb{R}_0 < 1$.

Theorem 3.2. *For the model (2.1), the disease free equilibrium E_{10} is globally asymptotically stable whenever $\mathbb{R}_0 < 1$.*

Proof. From (2.1) we have $\frac{dS}{dt} \leq bS(1 - \frac{S}{K})$. For $S = K$ is the globally asymptotically stable equilibrium of $\frac{dS}{dt} = bS(1 - \frac{S}{K})$, so for any $\varepsilon > 0$, when $t \rightarrow +\infty$ we have

$$S(t) \leq K + \varepsilon. \quad (3.6)$$

Then we have

$$\begin{cases} \frac{dE}{dt} \leq \mu e^{-mI}(K + \varepsilon)I - (c + d)E, \\ \frac{dI}{dt} = cE - \gamma I. \end{cases} \quad (3.7)$$

Now we consider

$$\begin{cases} \frac{dE}{dt} = \mu e^{-mI}(K + \varepsilon)I - (c + d)E = P(E, I), \\ \frac{dI}{dt} = cE - \gamma I = Q(E, I). \end{cases} \quad (3.8)$$

System (3.8) has a unique equilibrium $(0, 0)$ and the corresponding eigenvalues are determined by

$$\lambda^2 + (c + d + \gamma)\lambda + \gamma(c + d) - \mu c(K + \varepsilon) = 0. \quad (3.9)$$

For $\varepsilon > 0$ sufficiently small, since $\mathbb{R}_0 < 1$, hence $\gamma(c + d) - \mu c(K + \varepsilon) > 0$. Thus all the eigenvalues of (3.9) have negative real parts. Hence $(0, 0)$ is locally asymptotically stable. Since $\frac{\partial P(E, I)}{\partial E} + \frac{\partial Q(E, I)}{\partial I} = -(c + d + \gamma) < 0$, system (3.8) has no close orbit. Let

$$D = \left\{ (S, E, I) \mid S, E, I \geq 0, S + E + I \leq \bar{K}, \bar{K} = \frac{bK}{l}, l = \min\{b, d, \gamma\} \right\}.$$

We first prove that D is positively invariant. By (2.1), for $(S, E, I) \in D$ we have $\frac{dS}{dt} \Big|_{S=0} = 0$, $\frac{dE}{dt} \Big|_{E=0} = \mu e^{-mI}SI \geq 0$, $\frac{dI}{dt} \Big|_{I=0} \geq 0$, and $\frac{dS}{dt} \leq bS(1 - \frac{S}{K})$.

For $\frac{dS}{dt} = bS(1 - \frac{S}{K})$ with $S < \bar{K}$, we have $\lim_{t \rightarrow \infty} S(t) = K$, and $S(t) \leq K$. Note that if we let $N(t) = S(t) + E(t) + I(t)$, then

$$\begin{aligned} \frac{dN}{dt} |_{N=\bar{K}, (S,E,I) \in D} &= [bS(1 - \frac{S}{K}) - dE - \gamma I]_{S+E+I=\bar{K}} \\ &\leq [bK - bS - dE - \gamma I]_{S+E+I=\bar{K}} \\ &\leq bK - lN |_{N=\bar{K}} = bK - l\bar{K} = 0. \end{aligned}$$

Hence D is positively invariant. Therefore, $(0, 0)$ is globally asymptotically stable for (3.8). Consequently, for system (3.7) there holds

$$\lim_{t \rightarrow \infty} E(t) = 0, \quad \lim_{t \rightarrow \infty} I(t) = 0.$$

Then for the above $\varepsilon > 0$, there exists $T > 0$ such that for all $t > T$, $I(t) < \varepsilon$. By (2.1), we have

$$\frac{dS}{dt} > bS \left(1 - \frac{S}{K} \right) - \mu \varepsilon S.$$

Note that for $\varepsilon > 0$ sufficiently small, $S = K(1 - \frac{\mu \varepsilon}{b})$ is a globally asymptotically stable equilibrium of

$$\frac{dS}{dt} = bS \left(1 - \frac{\mu \varepsilon}{b} - \frac{S}{K} \right),$$

thus we have

$$S(t) \geq K - \varepsilon. (t \rightarrow \infty). \tag{3.10}$$

It follows from (3.6) and (3.10) that we have

$$\lim_{t \rightarrow \infty} S(t) = K.$$

Hence E_{10} is the globally asymptotically stable equilibrium of (2.1). \square

4. EXISTENCE OF THE ENDEMIC EQUILIBRIUM (EE)

First note that if $m = 0$, i.e., if the media impact is not considered, one can verify that when $\mathbb{R}_0 > 1$, system (2.1) has a unique endemic equilibrium (EE) (S_0^*, E_0^*, I_0^*) where

$$S_0^* = \frac{\gamma(c+d)}{\mu c} = \frac{K}{\mathbb{R}_0}, \quad E_0^* = \frac{b\gamma^2(c+d)}{\mu^2 c^2 K} (\mathbb{R}_0 - 1), \quad I_0^* = \frac{b\gamma(c+d)}{\mu^2 c K} (\mathbb{R}_0 - 1). \tag{4.1}$$

But if the media and psychological impact are incorporated, system (2.1) can have up to three equilibria.

Let

$$g(I) = K \left(1 - \frac{\mu}{b} I e^{-mI} \right). \quad (4.2)$$

Then the model (2.1) becomes

$$\begin{cases} \frac{dS}{dt} = \frac{b}{K} S(g(I) - S), \\ \frac{dE}{dt} = \frac{b}{K} (K - g(I)) S - (c + d) E, \\ \frac{dI}{dt} = cE - \gamma I. \end{cases} \quad (4.3)$$

Let the right hand side of (4.3) be zero. If a positive equilibrium exists, it is a positive solution of

$$\begin{cases} S = g(I), \\ S = \frac{K\gamma(c+d)}{bc} \frac{I}{K-g(I)} = h(I), \\ cE - \gamma I = 0, \end{cases} \quad (4.4)$$

where using the expression of $g(I)$ in (4.2) was used, $h(I)$ can be simplified to

$$h(I) = \frac{\gamma(c+d)}{\mu c} e^{mI}. \quad (4.5)$$

Then if a positive equilibrium, an endemic equilibrium exists, its (S, I) coordinates must satisfy

$$S = g(I), \quad S = h(I), \quad (4.6)$$

and the E coordinate is given by $E = \frac{\gamma}{c} I$.

One can verify that if $\mathbb{R}_0 > 1$, then $g(0) > h(0)$. Note $\lim_{I \rightarrow \infty} g(I) = K$ and $\lim_{I \rightarrow \infty} h(I) = \infty$. Hence if $\mathbb{R}_0 > 1$, the two curves $S = g(I)$ and $S = h(I)$ have at least one positive intersection which gives at least one endemic equilibrium. As shown in Fig. 1(a)–(c), the two planar curves $S = g(I)$ and $S = h(I)$ can have up to three intersections in the SI -plane.

Now we develop conditions to decide the tangency of the two curves in order to determine the number of positive equilibria. If the two curves

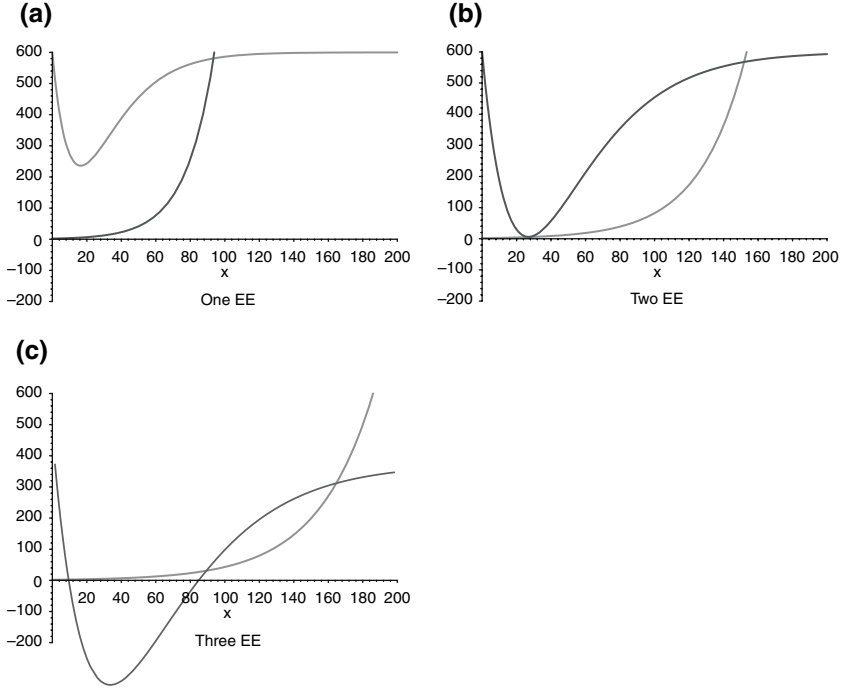


Figure 1. Three possible cases of the intersection of the curves $S = g(I)$ and $S = h(I)$ indicating the existence of up to three positive equilibria. The curves were plotted using Maple.

$S = g(I)$ and $S = h(I)$ are tangent at some positive points, we must have $S = g(I) = h(I)$, $g'(I) = h'(I)$. Or equivalently,

$$\begin{cases} K \left(1 - \frac{\mu}{b} I e^{-mI}\right) = \frac{\gamma(c+d)}{\mu c} e^{mI}, \\ -\frac{K\mu}{b} (1 - mI) e^{-mI} = \frac{\gamma(c+d)m}{\mu c} e^{mI}. \end{cases} \quad (4.7)$$

Eliminating the exponential terms in (4.7), if the two curves are tangent, the I coordinate must satisfy the quadratic equation

$$\frac{b}{\mu} \mathbb{R}_0 m (mI - 1) = (2mI - 1)^2. \quad (4.8)$$

It follows from (4.7) and (4.8) that if the tangency occurs at some point, its I coordinate must satisfy $mI > 1$.

Let

$$\delta := \frac{\mu}{b}, \quad m_0 := \frac{8\mu}{b\mathbb{R}_0} = \frac{8\delta}{\mathbb{R}_0}. \quad (4.9)$$

A straightforward calculation can verify that (4.8) has two distinct positive roots satisfying $mI > 1$ if and only if $\mathbb{R}_0 > 1$ and $m > m_0$. For $m > m_0$, solving (4.8) in terms of I , we have

$$I = \frac{m\mathbb{R}_0 + 4\delta \pm \sqrt{\Delta}}{8m\delta}, \quad (4.10)$$

where $\Delta = m\mathbb{R}_0(m\mathbb{R}_0 - 8\delta)$.

In summary, regarding the existence and the number of the endemic equilibria, we have:

Proposition 4.1. *Consider the model (2.1) with all parameters positive. Let m_0 be defined in (4.9). If $\mathbb{R}_0 > 1$, then model (2.1) has at least one and at most three positive equilibrium (endemic equilibria). Furthermore,*

- if $0 < m < m_0$, the model has a unique endemic equilibrium;
- if $m > m_0$, the model has three endemic equilibria;
- if $m = m_0$, the model has one endemic equilibria of multiplicity at least two.

By the above proposition, if $m = m_0$, the model can have a more degenerate endemic equilibrium with multiplicity three (both the eigenvalues are zero), and model can have a Bogdanov-Takens bifurcation of codimension two, even codimension three [6,21]. The study of the Bogdanov-Takens bifurcations is certainly out of the scope of this paper.

5. STABILITY AND HOPF BIFURCATION OF THE ENDEMIC EQUILIBRIUM (EE)

In this section, we shall study the stability and Hopf bifurcation of the endemic equilibria and determine how the media impact can influence the periods of the oscillations of virus/disease transmission.

5.1. $m = 0$

When $m = 0$, model (2.1) has a unique endemic equilibrium (S_0^*, E_0^*, I_0^*) . A straightforward calculation yields the associate characteristic equation:

$$\lambda^3 + \left(c + d + \gamma + \frac{b}{\mathbb{R}_0}\right)\lambda^2 + \frac{b}{\mathbb{R}_0}(c + d + \gamma)\lambda + b\gamma(c + d)\left(1 - \frac{1}{\mathbb{R}_0}\right) = 0. \quad (5.1)$$

Let

$$\mathbb{R}_{H_0} = \frac{1}{2} \left[1 + \frac{(c+d+\gamma)^2}{\gamma(c+d)} + \sqrt{1 + \frac{2(c+d+\gamma)(2b+c+d+\gamma)}{\gamma(c+d)} + \frac{(c+d+\gamma)^4}{\gamma^2(c+d)^2}} \right]. \quad (5.2)$$

Obviously, for any positive parameters we have $\mathbb{R}_{H_0} > 1$. Next proposition is about the local stability of the equilibrium (S_0^*, E_0^*, I_0^*) .

Proposition 5.1. *For the model (2.1) with $m=0$, the endemic equilibrium (S_0^*, E_0^*, I_0^*) is locally asymptotically stable if $1 < \mathbb{R}_0 < \mathbb{R}_{H_0}$.*

Proof. To prove, we only need to show that all roots of the characteristic equation (5.1) have negative real parts.

Since $\mathbb{R}_0 > 1$, all the coefficients of the cubic polynomial (5.1) are positive.

If we also have $\mathbb{R}_0 < \mathbb{R}_{H_0}$, then we have $\mathbb{R}_0^2\gamma(c+d) - \mathbb{R}_0[\gamma(c+d) + (c+d+\gamma)^2] - b(c+d+\gamma) < 0$. This is equivalent to $\frac{b}{\mathbb{R}_0}(c+d+\gamma)(c+d+\gamma + \frac{b}{\mathbb{R}_0}) - b\gamma(c+d)(1 - \frac{1}{\mathbb{R}_0}) > 0$.

So if $1 < \mathbb{R}_0 < \mathbb{R}_{H_0}$, we have $c+d+\gamma + \frac{b}{\mathbb{R}_0} > 0$, $b\gamma(c+d)(1 - \mathbb{R}_0) > 0$, $\frac{b}{\mathbb{R}_0}(c+d+\gamma)(c+d+\gamma + \frac{b}{\mathbb{R}_0}) - b\gamma(c+d)(1 - \frac{1}{\mathbb{R}_0}) > 0$.

It follows from the Routh-Hurwitz criteria [13] that all eigenvalues of (5.1) have negative real parts, hence (S_0^*, E_0^*, I_0^*) is locally asymptotically stable if $1 < \mathbb{R}_0 < \mathbb{R}_{H_0}$. \square

From (3.4) one can see that the reproduction number is linearly dependent on the parameter μ , hence solving $\mathbb{R}_0 = \mathbb{R}_{H_0}$ in terms of μ , one gets a threshold condition on the parameter μ for the endemic equilibrium to be locally asymptotically stable:

$$\mu_{H_0} = \frac{\gamma(c+d)}{cK} \mathbb{R}_{H_0}. \quad (5.3)$$

Hence it follows from Proposition 5.1 that if $\mu < \mu_{H_0}$, then the endemic equilibrium is locally asymptotically stable.

Theorem 5.2. *For the model (2.1) with $m=0$, when $\mathbb{R}_0 = \mathbb{R}_{H_0}$ or equivalently when $\mu = \mu_{H_0}$, (S_0^*, E_0^*, I_0^*) becomes unstable and model (2.1) undergoes a Hopf bifurcation.*

Proof. First note that if $\mathbb{R}_0 = \mathbb{R}_{H_0}$ or $\mu = \mu_{H_0}$, we have

$$\frac{b}{\mathbb{R}_0}(c+d+\gamma)(c+d+\gamma+\frac{b}{\mathbb{R}_0})=b\gamma(c+d)(1-\frac{1}{\mathbb{R}_0}),$$

then one can verify that equation (5.1) has a negative root and a pair of purely imaginary roots $\lambda = \pm\omega_0 i$, where

$$\omega_0^2 = \frac{b(c+d+\gamma)}{\mathbb{R}_0}. \quad (5.4)$$

For the the characteristic equation (5.1), we consider the characteristic root λ as a function of \mathbb{R}_0 or a function of μ . Differentiating equation (5.1) with respect to μ , we get

$$\begin{aligned} & \left[3\lambda^2 + 2\left(c+d+\gamma+\frac{b}{\mathbb{R}_0}\right)\lambda + \frac{b}{\mathbb{R}_0}(c+d+\gamma) \right] \frac{d\lambda}{d\mu} \\ & = \left[\frac{b}{\mathbb{R}_0^2}\lambda^2 + \frac{b}{\mathbb{R}_0^2}(c+d+\gamma)\lambda - \frac{b}{\mathbb{R}_0^2}\gamma(c+d) \right] \frac{d\mathbb{R}_0}{d\mu}. \end{aligned}$$

This gives

$$\left(\frac{d\lambda}{d\mu}\right)^{-1} = \frac{3\lambda^2 + 2(c+d+\gamma+\frac{b}{\mathbb{R}_0})\lambda + \frac{b}{\mathbb{R}_0}(c+d+\gamma)}{\frac{b}{\mathbb{R}_0^2}[\lambda^2 + (c+d+\gamma)\lambda - \gamma(c+d)]} \cdot \frac{cK}{\gamma(c+d)}.$$

Thus

$$\begin{aligned} & \text{sign} \left\{ \frac{d(\text{Re}\lambda)}{d\mu} \right\} \Big|_{\lambda=i\omega_0} = \text{sign} \left\{ \text{Re} \left(\frac{d\lambda}{d\mu} \right)^{-1} \right\} \Big|_{\lambda=i\omega_0} \\ & = \text{sign} \left\{ \text{Re} \left(\frac{3\lambda^2 + \frac{b}{\mathbb{R}_0}(c+d+\gamma) + 2(c+d+\gamma+\frac{b}{\mathbb{R}_0})\lambda}{\lambda^2 - \gamma(c+d) + (c+d+\gamma)\lambda} \right) \right\} \Big|_{\lambda=i\omega_0} \\ & = \text{sign} \left\{ \text{Re} \left(\frac{-3\omega_0^2 + \frac{b}{\mathbb{R}_0}(c+d+\gamma) + 2i(c+d+\gamma+\frac{b}{\mathbb{R}_0})\omega_0}{-\omega_0^2 - \gamma(c+d) + i(c+d+\gamma)\omega_0} \right) \right\} \\ & = \text{sign} \left\{ \frac{2b(c+d+\gamma)}{\mathbb{R}_0} \left[\frac{b}{\mathbb{R}_0}(c+d+\gamma) + \gamma(c+d) \right] \right. \\ & \quad \left. + \frac{2b}{\mathbb{R}_0}(c+d+\gamma)^2 \left(c+d+\gamma + \frac{b}{\mathbb{R}_0} \right) \right\} \\ & > 0. \end{aligned}$$

Therefore, as $\mu > 0$ increases, the real part of a pair of characteristic roots changes from negative to positive through zero, the transversality condition holds. Hence, the model with $m = 0$ undergoes an Hopf bifurcation when $\mathbb{R}_0 = \mathbb{R}_{H_0}$. This completes the proof. \square

5.2. m is Sufficiently Small

When $\mathbb{R}_0 > 1$ and $0 \leq m < m_0$, the model (2.1) has a unique endemic equilibrium (S^*, E^*, I^*) . Evaluating the Jacobian of (2.1) at the equilibrium gives

$$J(S^*, E^*, I^*) = \begin{pmatrix} -\frac{b}{K}S^* & 0 & -\frac{\gamma(c+d)}{c} + mbS^* \left(1 - \frac{S^*}{K}\right) \\ b \left(1 - \frac{S^*}{K}\right) & -(c+d) & \frac{\gamma(c+d)}{c} - mbS^* \left(1 - \frac{S^*}{K}\right) \\ 0 & c & -\gamma \end{pmatrix}.$$

The characteristic equation about (S^*, E^*, I^*) is given by

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0, \tag{5.5}$$

where

$$\begin{aligned} a_2 &= c + d + \gamma + \frac{b}{K}S^* > 0, \\ a_1 &= cmbS^* \left(1 - \frac{S^*}{K}\right) + \frac{bS^*}{K}(c + d + \gamma), \\ a_0 &= b \left(1 - \frac{S^*}{K}\right) \left[\gamma(c + d) + cmbS^* \left(\frac{2S^*}{K} - 1\right) \right]. \end{aligned} \tag{5.6}$$

Since we do not have a closed form for the endemic equilibrium, it is not easy to study the bifurcations analytically for the general case of m . We are going to use the fact that $m > 0$ is small to study the Hopf bifurcation of the endemic equilibrium.

The coordinates of the endemic equilibrium (S^*, E^*, I^*) are smooth functions of m . When $m > 0$ is sufficiently small, or if $0 < m < m_0$, we can expand the coordinates for the unique endemic equilibrium (S^*, E^*, I^*) as

$$\begin{cases} S^* = S_0^* + mS_1^* + O(m^2), \\ E^* = E_0^* + mE_1^* + O(m^2), \\ I^* = I_0^* + mI_1^* + O(m^2), \end{cases} \tag{5.7}$$

where particularly, by (4.4) we have

$$S_0^* = \frac{K}{\mathbb{R}_0}, \quad S_1^* = S_0^* \frac{b}{\mu} \left(1 - \frac{1}{\mathbb{R}_0}\right). \tag{5.8}$$

Note that the cubic polynomial (5.5) reduces to (5.1) when $m = 0$. Similar to the case of $m = 0$ in the above subsection, we will now study how the media coverage has an impact on the dynamics of the disease transmission by the method of perturbation.

It is not difficult to verify that (5.5) has a pair of purely imaginary roots if and only if $a_1 a_2 = a_0$. Let

$$H = a_1 a_2 - a_0. \quad (5.9)$$

If $H = 0$, the endemic equilibrium has a pair of purely imaginary roots. Using the expressions in (5.6), (5.7) and (5.8), one can verify that $H = 0$ is equivalent to $H(m, \mathbb{R}_0) = 0$, where

$$\begin{aligned} H(m, \mathbb{R}_0) = & \mathbb{R}_0^3 \left[\gamma(c+d) - \frac{m}{\mu} \gamma(c+d)(b+c+d+\gamma) \right] \\ & + \mathbb{R}_0^2 \left[\left(\frac{bm}{\mu} - 1 \right) \gamma(c+d) - \left(1 + \frac{bm}{\mu} \right) (c+d+\gamma)^2 \right. \\ & \quad \left. + \frac{m}{\mu} \gamma(c+d)(c+d+\gamma) \right] \\ & + \mathbb{R}_0 \left[-b(c+d+\gamma) - \frac{2b^2m}{\mu} (c+d+\gamma) + \frac{bm}{\mu} (c+d+\gamma)^2 \right] \\ & + \frac{2b^2m}{\mu} (c+d+\gamma). \end{aligned} \quad (5.10)$$

Note that when $H(m, \mathbb{R}_0) = 0$, the endemic equilibrium has a pair of purely imaginary eigenvalues $\lambda = \pm \omega i$, where

$$\omega^2 = cmbS^* \left(1 - \frac{S^*}{K} \right) + \frac{bS^*}{K} (c+d+\gamma). \quad (5.11)$$

Hence if the parameters m and \mathbb{R}_0 satisfy $H(m, \mathbb{R}_0) = 0$, an Hopf bifurcation may occur. Now we develop the function determined by $H(m, \mathbb{R}_0) = 0$.

Proposition 5.3. *Consider $H(m, \mathbb{R}_0) = 0$ for $0 \leq m < m_0$ and $\mathbb{R}_0 > 1$. In the neighborhood of $(0, \mathbb{R}_{H_0})$, there exists a unique smooth function $\mathbb{R}_0 = \mathbb{R}_0(m)$ such that $H(m, \mathbb{R}_0(m)) = 0$ for $0 \leq m < m_0$ sufficiently small. Furthermore, we have*

$$\mathbb{R}_0(m) = \mathbb{R}_{H_0} + m\mathbb{R}_{H_1} + O(m^2), \quad (5.12)$$

where \mathbb{R}_{H_0} is defined as in (5.2) and

$$\mathbb{R}_{H_1} = \frac{(c+d+\gamma)^2(2b+c+d+\gamma)\mathbb{R}_{H_0}^2 + b^2(c+d+\gamma)(3\mathbb{R}_{H_0}-2)}{\mu[\gamma(c+d)\mathbb{R}_{H_0}^2 + b(c+d+\gamma)]}. \quad (5.13)$$

Proof. Note that $H(0, \mathbb{R}_{H_0}) = 0$ and in the neighborhood of $(0, \mathbb{R}_{H_0})$, we have

$$\begin{aligned} \frac{\partial H}{\partial \mathbb{R}_0} \Big|_{m=0, \mathbb{R}_0=\mathbb{R}_{H_0}} &= 3\mathbb{R}_{H_0}^2 \gamma(c+d) - 2\mathbb{R}_{H_0}[\gamma(c+d) + (c+d+\gamma)^2] \\ &\quad - b(c+d+\gamma) = \mathbb{R}_{H_0}^2 \gamma(c+d) + b(c+d+\gamma) \neq 0, \end{aligned}$$

then by the Implicit Function Theorem, there exists a unique function $\mathbb{R}_0 = \mathbb{R}_0(m)$ such that $H(m, \mathbb{R}_0(m)) = 0$ for $m \geq 0$ sufficiently small.

If we write the Taylor expansion for $\mathbb{R}_0(m)$ in terms of m as in (5.12) and plug it into (5.10), we have

$$\begin{aligned} H(m, \mathbb{R}_0(m)) &= H(m, \mathbb{R}_{H_0} + m\mathbb{R}_{H_1} + O(m^2)) \\ &= (\mathbb{R}_{H_0}^3 + 3\mathbb{R}_{H_0}^2 \mathbb{R}_{H_1} m) \left[\gamma(c+d) - m \frac{\gamma(c+d)}{\mu} (b+c+d+\gamma) \right] \\ &\quad + (\mathbb{R}_{H_0}^2 + 2\mathbb{R}_{H_0} \mathbb{R}_{H_1} m) \left\{ -\gamma(c+d) - (c+d+\gamma)^2 \right. \\ &\quad \left. + m \left[\frac{\gamma(c+d)}{\mu} (b+c+d+\gamma) - \frac{b}{\mu} (c+d+\gamma)^2 \right] \right\} \\ &\quad + (\mathbb{R}_{H_0} + m\mathbb{R}_{H_1}) \left\{ -b(c+d+\gamma) \right. \\ &\quad \left. + m \left[\frac{b}{\mu} (c+d+\gamma)^2 - \frac{2b^2}{\mu} (c+d+\gamma) \right] \right\} + \frac{2b^2}{\mu} (c+d+\gamma)m \\ &= 0. \end{aligned} \tag{5.14}$$

Equalizing the terms of same power of m on both sides of the above equation, from the constant term, we have

$$\mathbb{R}_{H_0}^3 \gamma(c+d) - \mathbb{R}_{H_0}^2 [\gamma(c+d) + (c+d+\gamma)^2] - \mathbb{R}_{H_0} b(c+d+\gamma) = 0,$$

which is the same as the equation to define \mathbb{R}_{H_0} . For the coefficients for the first term, we have

$$\begin{aligned} 3\mathbb{R}_{H_0}^2 \mathbb{R}_{H_1} \gamma(c+d) - \mathbb{R}_{H_0}^3 \frac{\gamma(c+d)}{\mu} (b+c+d+\gamma) \\ + \mathbb{R}_{H_0}^2 \left[\frac{\gamma(c+d)}{\mu} (b+c+d+\gamma) - \frac{b}{\mu} (c+d+\gamma)^2 \right] \\ + 2\mathbb{R}_{H_0} \mathbb{R}_{H_1} [-\gamma(c+d) - (c+d+\gamma)^2] \\ + \mathbb{R}_{H_0} \left[\frac{b}{\mu} (c+d+\gamma)^2 \right. \\ \left. - \frac{2b^2}{\mu} (c+d+\gamma) \right] - b\mathbb{R}_{H_1} (c+d+\gamma) + \frac{2b^2}{\mu} (c+d+\gamma) \\ = 0. \end{aligned} \tag{5.15}$$

Solving equation (5.15) in terms of \mathbb{R}_{H_1} we obtain (5.13). \square

Theorem 5.4. *If $1 < \mathbb{R}_0 < \mathbb{R}_0(m)$, where $\mathbb{R}_0(m)$ is defined in (5.12) for $m \geq 0$ sufficiently small, then the endemic equilibrium (S^*, E^*, I^*) is locally-asymptotically stable.*

Proof. When $\mathbb{R}_0 > 1$, consider the characteristic equation for the equilibrium (S^*, E^*, I^*) in (5.5). Obviously, $a_2 > 0$. We need to prove $a_0 > 0$ and $a_2 a_1 - a_0 > 0$ in order to use Routh-Hurwitz criteria [13] to conclude.

By (5.8), we have for $m > 0$ small that

$$\begin{aligned} a_0 &= \gamma(c+d) + mbcS^* \left(\frac{2S^*}{K} - 1 \right) \\ &= \gamma(c+d) + mbc \frac{K}{\mathbb{R}_0} \left[\frac{2}{K} \frac{K}{\mathbb{R}_0} - 1 \right] + O(m^2). \end{aligned} \quad (5.16)$$

Since $m < m_0 = \frac{8\gamma(c+d)}{bcK}$, we have $\gamma(c+d) + \frac{mbcK}{\mathbb{R}_0} \left(\frac{2}{\mathbb{R}_0} - 1 \right) > 0$, hence $a_0 > 0$.

Next we prove $a_2 a_1 - a_0 > 0$. By (5.8) and $\mathbb{R}_0 \gamma(c+d) = \mu c K$, a straightforward calculation gives

$$\begin{aligned} a_2 a_1 - a_0 &= (c+d+\gamma + \frac{b}{K} S^*) \left[mbcS^* \left(1 - \frac{S^*}{K} \right) + \frac{bS^*}{K} (c+d+\gamma) \right] \\ &\quad - b \left(1 - \frac{S^*}{K} \right) \left[\gamma(c+d) + mbcS^* \left(\frac{2S^*}{K} - 1 \right) \right] \\ &= \frac{b}{\mathbb{R}_0^3} \left\{ \mathbb{R}_0^2 \left[\left(1 + \frac{bm}{\mu} \right) (c+d+\gamma)^2 + (b+c+d+\gamma)cKm - \mu c K \right] \right. \\ &\quad \left. + \mathbb{R}_0 \left[\mu c K + b(c+d+\gamma) + \frac{2b^2 m}{\mu} (c+d+\gamma) \right. \right. \\ &\quad \quad \left. \left. - \frac{bm}{\mu} (c+d+\gamma)^2 - (b+c+d+\gamma)cKm \right] \right. \\ &\quad \left. - \frac{2b^2 m}{\mu} (c+d+\gamma) \right\} + O(m^2) > 0. \end{aligned} \quad (5.17)$$

Then it follows from Routh-Hurwitz criteria [13] that all eigenvalues of (5.5) have negative real parts. Hence E_2 is locally-asymptotically stable when $1 < \mathbb{R}_0 < \mathbb{R}_0(m)$ and $m > 0$ is sufficiently small. \square

Theorem 5.5. *When $0 < m < m_0$ and $\mathbb{R}_0 = \mathbb{R}_0(m)$, the system undergoes a Hopf bifurcation.*

Proof. It follows from Proposition 5.3 and Theorem 5.4, we only need to prove the transversality to conclude the existence of the bifurcation.

Differentiating Eq.(5.5) with respect μ , we get

$$\frac{d\lambda}{d\mu} = \frac{dS^*}{d\mu} \cdot \frac{B_1}{B_2}, \tag{5.18}$$

where

$$\begin{aligned} B_1 &= -\frac{b}{K}\lambda^2 - \left[cmb \left(1 - \frac{2S^*}{K} \right) + \frac{b}{K}(c+d+\gamma) \right] \lambda \\ &\quad + \frac{b}{K} \left[\gamma(c+d) + cmbS^* \left(\frac{2S^*}{K} - 1 \right) \right] - \left(1 - \frac{S^*}{K} \right) cmb^2 \left(\frac{4S^*}{K} - 1 \right), \\ B_2 &= 3\lambda^2 + 2 \left(c+d+\gamma + \frac{bS^*}{K} \right) \lambda + \left[cmbS^* \left(1 - \frac{S^*}{K} \right) + \frac{b}{K}S^*(c+d+\gamma) \right]. \end{aligned} \tag{5.19}$$

Recall that when $\mathbb{R}_0 = \mathbb{R}_0(m)$, or equivalently when

$$\begin{aligned} &\left(c+d+\gamma + \frac{bS^*}{K} \right) \left[cmbS^* \left(1 - \frac{S^*}{K} \right) + \frac{bS^*}{K}(c+d+\gamma) \right] \\ &= b \left(1 - \frac{S^*}{K} \right) \left[\gamma(c+d) + cmbS^* \left(\frac{2S^*}{K} - 1 \right) \right], \end{aligned} \tag{5.20}$$

Equation (5.5) has a pair of purely imaginary roots $\lambda = \pm\omega i$ with

$$\begin{aligned} \omega^2 &= cmbS^* \left(1 - \frac{S^*}{K} \right) + \frac{bS^*}{K}(c+d+\gamma) \\ &= \frac{b(c+d+\gamma)}{\mathbb{R}_0} + m \left[\frac{bck}{\mathbb{R}_0} \left(1 - \frac{1}{\mathbb{R}_0} \right) + \frac{b^2(c+d+\gamma)}{\mu\mathbb{R}_0} \left(1 - \frac{1}{\mathbb{R}_0} \right) \right] + O(m^2). \end{aligned}$$

Note that

$$S^* = \frac{K}{\mathbb{R}_0} e^{\frac{mc}{\gamma} E^*} = \frac{\gamma(c+d)}{\mu c} e^{\frac{cmb}{\gamma(c+d)} S^* \left(1 - \frac{S^*}{K} \right)},$$

so we get

$$\frac{dS^*}{d\mu} = \frac{\gamma(c+d)}{\mu} \cdot \frac{S^*}{cmbS^* \left(1 - \frac{2S^*}{K} \right) - \gamma(c+d)}.$$

By (5.20), we have $\gamma(c+d) + cmbS^* \left(\frac{2S^*}{K} - 1 \right) > 0$. Hence we always have $\frac{dS^*}{d\mu} < 0$.

Therefore, it follows from (5.18) that we have

$$\begin{aligned}
& \text{sign} \left\{ \frac{d(\text{Re}\lambda)}{d\mu} \right\}_{\lambda=i\omega} \\
&= \text{sign} \left\{ \text{Re} \left(\frac{d\lambda}{d\mu} \right) \right\}_{\lambda=i\omega} \\
&= \text{sign} \left\{ -\text{Re} \left(\frac{\frac{b}{K}[\omega^2 + \gamma(c+d) + cmbS^*(\frac{2S^*}{K}-1)] - (1-\frac{S^*}{K})cmb^2(\frac{4S^*}{K}-1)}{-3\omega^2 + cmbS^*(1-\frac{S^*}{K}) + \frac{b}{K}S^*(c+d+\gamma) + 2i(c+d+\gamma + \frac{b}{K}S^*)\omega} \right. \right. \\
&\quad \left. \left. - \frac{i[cmb(1-\frac{2S^*}{K}) + \frac{b}{K}(c+d+\gamma)]\omega}{-3\omega^2 + cmbS^*(1-\frac{S^*}{K}) + \frac{b}{K}S^*(c+d+\gamma) + 2i(c+d+\gamma + \frac{b}{K}S^*)\omega} \right) \right\} \\
&= \text{sign} \left\{ cmb^2(1-\frac{S^*}{K})(1-\frac{3S^*}{K}) + cmb(1-\frac{2S^*}{K})(c+d+\gamma) \right. \\
&\quad \left. + \frac{b}{K}\gamma(c+d) + \frac{b}{K}(c+d+\gamma)^2 + \frac{2b^2}{K^2}S^*(c+d+\gamma) \right\} \\
&> 0. \tag{5.21}
\end{aligned}$$

Therefore, the transversality condition holds and hence a Hopf bifurcation occurs when $\mathbb{R}_0 = \mathbb{R}_0(m)$ and m is small. \square

5.3. Numerical Simulations

For the purpose of simulations, here we fix some of the parameters in Table I and shall consider the cases when γ and m are varied.

First we consider the case when the disease transmission is mild with a lower reproduction number. In the case when $\gamma = 0.05$ and all other

Table I. Part of the parameters for the simulations

Parameters	Value
Carrying capacity K	5,000,000
Intrinsic growth rate of the population b	0.001
Contact transmission rate μ	1.2×10^{-8}
Time that an exposed becomes infected $\frac{1}{c}$	10
Natural death rate of the population d	0.001

Table II. Endemic equilibrium (S^*, E^*, I^*) when $m > 0$ is varied. In the table, except for the parameters given in Table I, here we have $\gamma = 0.05$. In this case, $\mathbb{R}_0 = 1.188$ and $\mathbb{R}_{H_0} = 5.52$

Parameter m	S^*	E^*	I^*
$m = 0$	4208333	6597	13194
$m = 1 \times 10^{-6}$	4261135	6235	12468
$m = 6 \times 10^{-6}$	4457313	4790	9580

parameters as in Table I, we have $\mathbb{R}_0 = 1.188$. As shown in Fig. 2(a), (b), the transmission of the disease experiences multiple peaks without the media alert, the thin curve represents the case when $m = 0$, the application of media was not considered. The other two thicker curves represent the cases when $m = 0.000001$ and $m = 0.000006$, respectively. As shown in Table II, if $\gamma = 0.05$, we have $\mathbb{R}_0 = 1.188$ and $\mathbb{R}_{H_0} = 5.52$. For all the cases, the endemic equilibrium is a spiral sink which is local asymptotically stable. The population in each compartment approaches its equilibrium value. From the simulation results in Fig. 2, one can see that the effective media coverage (larger values of m) stabilizes the oscillation, and less number of the individuals become infected in the course of transmission.

The media impact to the transmission is also simulated in Fig. 3(a), (b) where γ is reduced to 0.02, with all other parameters are given in Table I.

6. DISCUSSION

6.1. Multiple Peaks of the Transmission and the Media Impact

We knew that when $m = 0$, the Hopf bifurcation occurs and a periodic solution appears. When the media impact is not considered, if $\mathbb{R}_0 > 1$ and close to \mathbb{R}_{H_0} , the disease will be endemic with multiple peaks. The time between between the two peaks can be approximated by

$$T_0 = \frac{2\pi}{\omega} = \frac{2\pi}{\sqrt{\frac{b}{\mathbb{R}_0}(c + d + \gamma)}}.$$

But when the media coverage/alert is introduced, or when $0 < m < m_0$ is sufficiently small, if there are multiple peaks, the time between each of

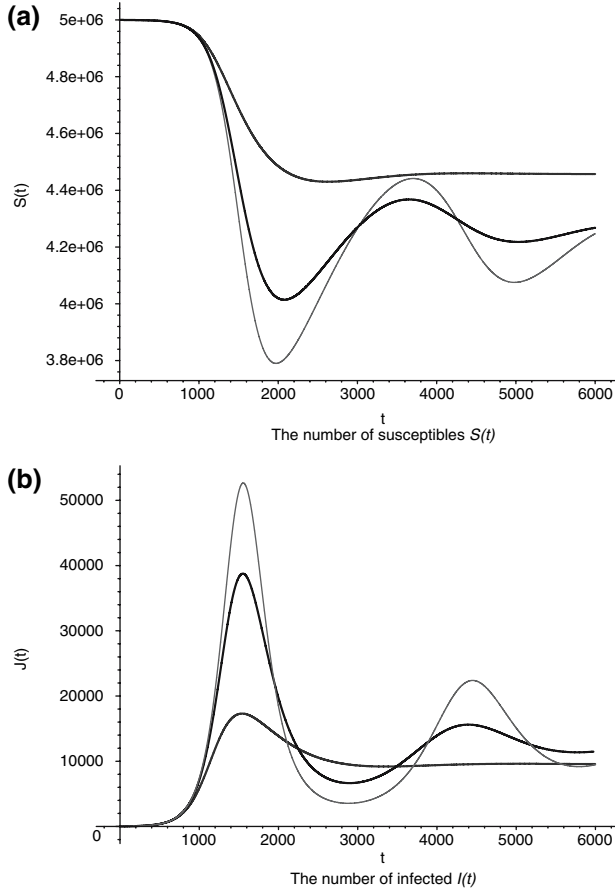


Figure 2. Simulations for the case when $\gamma = 0.05$. Here $\mathbb{R}_0 = 1.188$ and $\mathbb{R}_{H_0} = 5.52$. The thickness of the curves increases when the parameter $m=0$ changes from 0, 10^{-6} to 6×10^{-6} .

the two peaks can be approximated by

$$T_m = \frac{2\pi}{\sqrt{\frac{b(c+d+\gamma)}{\mathbb{R}_0} + m\left[\frac{bck}{\mathbb{R}_0}\left(1 - \frac{1}{\mathbb{R}_0}\right) + \frac{b^2(c+d+\gamma)}{\mu\mathbb{R}_0}\left(1 - \frac{1}{\mathbb{R}_0}\right)\right] + O(m^2)}}.$$

This shows that the media alert shortens the time of the secondary peak of the disease transmission. This effect is also verified by the simulations in Fig. 3 (a), (b).

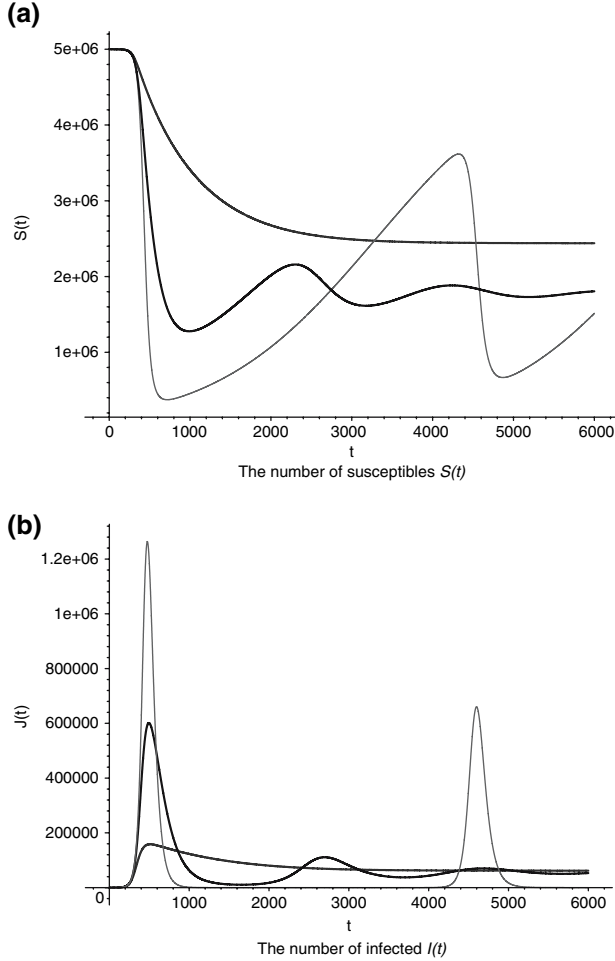


Figure 3. Simulations for the case when $\gamma = 0.02$. Here $\mathbb{R}_0 = 2.97$ and $\mathbb{R}_{H_0} = 8.26$. The thickness of the curves increases when the parameter $m = 0$ changes from $0, 10^{-6}$ to 6×10^{-6} .

6.2. The Media Coverage/Alert and the Endemic State

Note that whenever $\mathbb{R}_0 > 1$, an endemic equilibrium appears and its coordinates (S^*, E^*, I^*) are given by

$$bS^* \left(1 - \frac{S^*}{K}\right) - (c + d)E^* = 0, \quad S^* = \frac{K}{R_0} e^{\frac{mc}{\gamma} E^*}, \quad I^* = \frac{c}{\gamma} E^*.$$

If we consider S^* , E^* and I^* as functions of $m > 0$, then we have

$$\begin{cases} b \left(1 - \frac{2S^*}{K}\right) \frac{dS^*}{dm} - (c+d) \frac{dE^*}{dm} = 0, \\ \frac{dS^*}{dm} = \frac{cK}{R_0\gamma} e^{\frac{mc}{\gamma} E^*} + \frac{cmK}{R_0\gamma} e^{\frac{mc}{\gamma} E^*} \frac{dE^*}{dm}. \end{cases} \quad (6.1)$$

Thus, we get

$$\begin{cases} \frac{dS^*}{dm} = \frac{cb(S^*)^2 \left(1 - \frac{S^*}{K}\right)}{\gamma(c+d) - mcbS^* \left(1 - \frac{2S^*}{K}\right)}, \\ \frac{dE^*}{dm} = \frac{1}{c+d} \cdot \frac{cb(S^*)^2 \left(1 - \frac{S^*}{K}\right) \left(1 - \frac{2S^*}{K}\right)}{\gamma(c+d) - mcbS^* \left(1 - \frac{2S^*}{K}\right)}, \\ \frac{dI^*}{dm} = \frac{c}{\gamma} \frac{dE^*}{dm}. \end{cases}$$

Since the endemic equilibrium is locally-asymptotically stable, we have $\gamma(c+d) - mcbS^* \left(1 - \frac{2S^*}{K}\right) > 0$. Hence $\frac{dS^*}{dm} > 0$, thus S^* is always an increasing function of m , and if $1 < \mathbb{R}_0 < 2$ one can also verify that $\frac{dE^*}{dm} < 0$ and $\frac{dI^*}{dm} < 0$, therefore, E^* and I^* are decreasing functions of m . This is verified by the numerical simulations in Table II and Fig. 3 (a), (b).

6.3. Other Comments and Further Improvement

In this paper, we are trying to explore the impact of media coverage to the transmission of infection diseases. The model (2.1) is a toy model for the purpose of analyzing the impact of media on the spreading of the disease. In the model, we used a contact transmission rate $\beta(I) = \mu e^{-mI}$. For further study, it would be ideal to consider more realistic contact transmission rates to reflect the impact of media coverage and alertness. Yet, the analysis of such a new model can be mathematically more challenge due to the high dimension of the models and nonlinearity of the incidence function.

REFERENCES

1. Brauer, F., and Castillo-Chavez, C.(2000). *Mathematical Models in Population Biology and Epidemics*. Springer-Verlag, New York.
2. Busenberg, S., and Cooke, K.(1993). *Vertically Transmitted Diseases*. Springer-Verlag, New York.
3. Capasso, V.(1993). *Mathematical Structure of Epidemic System, Lecture Note in Biomathematics*, Vol. 97. Springer, Berlin.
4. Capasso, V., and Serio, G.(1978). A generalization of the Kermack-McKendrick deterministic epidemic model. *Math. Biosci.* **42**, 43.
5. Diekmann, O., and Heesterbeek, J. A. P.(2000). *Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation*. Wiley, New York.
6. Dumortier, F., Roussarie, R., and Sotomayor, J.(1987). Generic 3-parameter families of vector fields on the plane, unfolding a singularity with nilpotent linear part. The cusp case of codimension 3. *Ergodic Theory Dynamical Systems* **7**(3), 375–413.
7. Health Canada: <http://www.hc-sc.gc.ca/pphb-dgspssp/sars-sras/prof-e.html>
8. Hethcote, H. W.(2000). The mathematics of infectious diseases. *SIAM Revi.* **42**, 599–653.
9. Levin, S. A., Hallam, T. G., and Gross, L. J. (1989). *Applied Mathematical Ecology*. Springer, New York.
10. Liu, W. M., Hethcote, H. W., Levin, S. A.(1987). Dynamical behavior of epidemiological models with nonlinear incidence rates. *J. Math. Biol.* **25**, 359.
11. Liu, W. M., Levin, S. A., and Iwasa, Y.(1986). Influence of nonlinear incidence rates upon the behavior of SIRS epidemiological models. *J. Math. Biol.* **23**, 187.
12. Liu, R., Wu, J., and Zhu, H.(2005). *Medial Psychological Impact on Multiple Outbreaks of Emerging Infectious Diseases*, preprint.
13. Murray, J. D.(1998). *Mathematical Biology*, Springer-Verlag, Berlin.
14. Ruan, S., and Wang, W.(2003). Dynamical behavior of an epidemic model with a nonlinear incidence rate. *J. Diff. Equs.* **188**, 135.
15. SARS EXPRESS: <http://www.syhao.com/sars/20030623.htm>
16. Shen, Z. et al.(2004). Superspreading SARS events, Beijing, 2003. *Emerg. Infect. Dis.* **10**(2), 256–260.
17. van den Driessche, P., and Watmough, J.(2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math. Biosci.* **180**, 29–48.
18. Wang, W., and Ruan, S.(2004). Simulating SARS outbreak in Beijing with limit data. *J. Theor. Biol.* **227**, 369.
19. WHO. Epidemic curves: Serve Acute Respiratory Syndrome (SARS) <http://www.who.int/csr/sars/epicurve/epiindex/en/print.html>
20. Yorke, J. A., and London, W. P.(1973). Recurrent outbreaks of measles, chickenpox and mumps II. *Am. J. Epidemiol.* **98**, 469.
21. Zhu, H., Campbell, S. A., and Wolkowicz, G. S.(2002). Bifurcation analysis of a predator-prey system with nonmonotonic function response. *SIAM J. Appl. Math.* **63**, (2), 636–682.