Research Article

Modelling the Impact of Media in Controlling the Diseases with a Piecewise Transmission Rate

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An epidemic model with media is proposed to describe the spread of infectious diseases in a given region. A piecewise continuous transmission rate is introduced to describe that the media has its effect when the number of the infected exceeds a certain critical level. Furthermore, it is assumed that the impact of the media on the contact transmission is described by an exponential function. Stability analysis of the model shows that the disease-free equilibrium is globally asymptotically stable if the basic reproduction number is less than unity. On the other hand, when the basic reproduction number is greater than unity, a unique endemic equilibrium exists, which is also globally asymptotically stable. Our analysis implies that media coverage plays an important role in controlling the spread of the disease.

1. Introduction

When an infectious disease outbreaks, the spread and the control of the disease will be reported by the media including television programs, newspapers, and online social networks. Many examples are the massive reports and daily updates in the public media on the number of the infections and deaths, which had important impacts on the diseases control [1, 2]. It is shown that media coverage plays an important role in the spread and control of the infectious disease, such as the SARS in 2003 [3], the H1N1 in 2009 influenza epidemic [4, 5], and the Ebola in 2014 in Africa [6].

Recently, such impact on disease spreading and controlling has been investigated by mathematical modeling approach [5, 7–20]. In these research works some focused their attention on the incidence rate, and the recent survey identified three typical terms in [5]. Liu et al. [7] described the impact of media coverage using the transmission coefficient $\beta e^{-\alpha_1 E - \alpha_2 I - \alpha_3 H}$, and this impact leads to the change of avoidance and contact patterns at both individual and community levels. Cui et al. [8–11] developed a compartment model using incidence rate $\mu e^{-mI} SI$ with $m > 0$ to investigate the impact of media coverage on the transmission, and stability analysis of the models has shown that Hopf bifurcation can occur. Tchuente et al. [14] used an exponentially decreasing

function $e^{-M(t)}$ to reveal the force of media and showed the potential short-term beneficial effect of awareness programs.

More recently nonsmooth media functions [21–25] have been studied. Xiao et al. [21] introduced a segmented function to describe the media impact e^{-mI_c} , here m is also the strength of the media effect, and I_c is a threshold that people take the controlling measures or not. A Filippov epidemic model was proposed to describe the real characteristics of media impact in the spread of infectious diseases by incorporating a piecewise continuous transmission rate $\beta e^{(-\alpha_e I)} SI$ in [24], and mathematical analysis with regard to the local and global stability of equilibria and local sliding bifurcations are performed.

In fact, during infectious disease outbreaks, individuals may reduce their activities after receiving information about the risk of infection. For example, people will reduce the time that they go out, students will not attend school, and so on, and such information on the ongoing epidemics may impact the dynamics itself. In fact, at the initial stages of the prevalence of disease, most people and public mass media are unaware of the disease; thus, the individuals will not do any protective measures. Only when the number of infectious individuals reaches and exceeds a certain level, the individuals will take precautionary measures against

the diseases. Based on the above facts, in this paper we also focus on the incidence rate. Here we introduce $\sigma(I) = I - I_c$ to show this fact, where I_c is the threshold value. It is assumed that the impact of media is described by an exponential decreasing factor and the population obeys the logistic growth.

The rest of this paper is organized as follows: in the next section, a mathematical model is proposed in order to reveal the effect of media; then the existence of equilibria is given in Section 3. In Section 4 the local and global stability of the disease-free and the unique endemic equilibria are analyzed. Furthermore, in Section 5 some numerical simulations and discussions are given in the last section.

2. Mathematical Model with Media

In this model the population is divided into three types: the susceptible, the infective and the recovered. Let $S(t)$, $I(t)$, and $R(t)$ denote the number of susceptible, infective, and recovered individuals at time t , respectively. It is assumed that the growth of the susceptible population obeys the logistic growth, the intrinsic growth rate of the population is b , and the carrying capacity for the population is K . The interactions between susceptible and infective individuals are assumed to be bilinear, and β is the contact rate of susceptible with infective individuals, γ is the recovery rate of the infective individuals, and d is the natural death rate of the population. Consider that the cumulative density of media about the disease is $M(t)$. The growth rate of cumulative density of media, σ , is assumed to be proportional to the number of infective individuals in the population, and ν is the depletion rate of cumulative density of media. Thus, we have the following model:

$$\begin{aligned} S'(t) &= bS \left(1 - \frac{S}{K}\right) - \beta SI, \\ I'(t) &= \beta SI - (\gamma + d)I, \\ R'(t) &= \gamma I - dR, \\ M'(t) &= \sigma I - \nu M. \end{aligned} \quad (1)$$

In this model we assume that the impact of media is described by an exponential decreasing factor as $\beta = \beta_0 e^{-\epsilon m M}$, where the parameter $m > 0$ is to reflect the impact of media. When $\epsilon = 0$, the transmission rate is the constant β_0 . When $\epsilon = 1$, as one can see if m is relatively small, the transmission rate β is close to the constant β_0 . Thus, the model can be rewritten as follows:

$$\begin{aligned} S'(t) &= bS \left(1 - \frac{S}{K}\right) - \beta_0 e^{-\epsilon m M} SI, \\ I'(t) &= \beta_0 e^{-\epsilon m M} SI - (\gamma + d)I, \\ R'(t) &= \gamma I - dR, \\ M'(t) &= \sigma I - \nu M, \end{aligned} \quad (2)$$

with

$$\epsilon = \begin{cases} 0, & \sigma(I) \leq 0, \\ 1, & \sigma(I) > 0, \end{cases} \quad (3)$$

where the function $\sigma(I) = I - I_c$ means that when infective individuals reach and exceed the certain level I_c , the media has its effects.

For the solutions of system (2) with (3), the region of attraction is given by the set:

$$\begin{aligned} \Omega &= \left\{ (S, I, R, M) \in \mathbb{R}_+^4 \mid 0 < S(t) + I(t) + R(t) \right. \\ &\quad \left. \leq K, 0 \leq M \leq \frac{\sigma K}{\nu} \right\}, \end{aligned} \quad (4)$$

and it attracts all solutions initiating in the interior of the positive orthant. The (S, I, R, M) phase space is split into two parts: $G_1 = \{(S, I, R, M) \in \mathbb{R}_+^4 \mid \sigma(I) \leq 0\}$ and $G_2 = \{(S, I, R, M) \in \mathbb{R}_+^4 \mid \sigma(I) > 0\}$. In region G_1 , there is no effect of the media and the transmission rate $\beta = \beta_0$, while in region G_2 , the transmission rate declines to $\beta_0 e^{-mM}$.

For convenience, let the vector $Z = (S, I, M)^T$, and denote

$$\begin{aligned} F_{G_1}(Z) &= \left(bS \left(1 - \frac{S}{K}\right) - \beta_0 SI, \beta_0 SI - (\gamma + d)I, \sigma I \right. \\ &\quad \left. - \nu M \right)^T, \\ F_{G_2}(Z) &= \left(bS \left(1 - \frac{S}{K}\right) - \beta_0 e^{-mM} SI, \beta_0 e^{-mM} SI \right. \\ &\quad \left. - (\gamma + d)I, \sigma I - \nu M \right)^T. \end{aligned} \quad (5)$$

Then system (2) with (3) can be rewritten as the following switching system:

$$Z' = \begin{cases} F_{G_1}(Z), & Z \in G_1, \\ F_{G_2}(Z), & Z \in G_2. \end{cases} \quad (6)$$

In the following, system (6) defined in region G_1 is the subsystem S_1 and that defined in region G_2 is the subsystem S_2 , and we consider the two subsystems in isolation firstly.

3. Existence of Equilibria

3.1. Equilibria of S_1 . Let $F_{G_1}(Z)$ be zero; one can verify that the origin $E_0^1 = (0, 0, 0)$ is a hyperbolic saddle point with eigenvalues $b, -(\gamma + d), -\nu$. The subsystem S_1 has one disease-free equilibrium $E_0^2 = (K, 0, 0)$, and the stability of E_0^2 can be obtained in the following section.

It follows from [26] that the reproduction number $R_0 = \beta_0 K / (\gamma + d)$ and we can verify that when $R_0 > 1$, S_1 has a unique endemic equilibrium $E_0^* = (S_0^*, I_0^*, M_0^*)$, where

$$\begin{aligned} S_0^* &= \frac{\gamma + d}{\beta_0}, \\ I_0^* &= \frac{b}{\beta_0} \left(1 - \frac{1}{R_0} \right), \\ M_0^* &= \frac{\sigma}{\nu} I_0^*. \end{aligned} \quad (7)$$

3.2. Equilibria of S_2 . The disease-free equilibria of the subsystem S_2 are same with the subsystem S_1 ; thus, we study the existence of the endemic equilibrium of S_2 .

Let $F_{G_2}(Z)$ be zero; one can see that the endemic equilibrium must satisfy

$$\begin{aligned} bS \left(1 - \frac{S}{K} \right) - \beta_0 e^{-mM} SI &= 0, \\ \beta_0 e^{-mM} S &= \gamma + d, \\ \sigma I - \nu M &= 0. \end{aligned} \quad (8)$$

If there exists a positive equilibrium, it is a positive solution of

$$\begin{aligned} S &= K \left(1 - \frac{\beta_0}{b} I e^{-(m\sigma/\nu)I} \right) := g(I), \\ S &= \frac{K}{R_0} e^{(m\sigma/\nu)I} := h(I). \end{aligned} \quad (9)$$

One can verify that $g(0) = K$, $h(0) = K/R_0$, and if $R_0 > 1$, $g(0) > h(0)$. Hence, the two curves $S = g(I)$ and $S = h(I)$ have at least one positive intersection. In order to determine the number of positive intersections we consider the tangency of two curves. If the two curves intersect, it must have $g(I) = h(I)$, $g'(I) = h'(I)$; that is,

$$\begin{aligned} 1 - \frac{\beta_0}{b} I e^{-(m\sigma/\nu)I} &= \frac{1}{R_0} e^{(m\sigma/\nu)I}, \\ \frac{\beta_0}{b} e^{-(m\sigma/\nu)I} \left(\frac{m\sigma}{\nu} I - 1 \right) &= \frac{m\sigma}{R_0 \nu} e^{(m\sigma/\nu)I}. \end{aligned} \quad (10)$$

From (10) I must satisfy the quadratic equation:

$$\left(2I - \frac{\nu}{m\sigma} \right)^2 = \frac{bR_0}{\beta_0} \left(I - \frac{\nu}{m\sigma} \right). \quad (11)$$

Let $\delta = bR_0/\beta_0$, $\alpha = \nu/m\sigma$; then we have

$$I = \frac{4\alpha + \delta \pm \sqrt{\delta(\delta - 8\alpha)}}{8}. \quad (12)$$

Let $m_0 = 8\beta_0\nu/b\sigma R_0$; if $0 < m < m_0$, (11) has no roots and the system has a unique endemic equilibrium. If $m = m_0$, (11) has one unique root and the system has one endemic equilibria of multiplicity at least two. If $m > m_0$, (11) has two roots and the system has three endemic equilibria. Thus, we have the following theorem.

Theorem 1. When $R_0 > 1$ and $0 < m < m_0$ subsystem S_2 has a unique endemic equilibrium $E_* = (S^*, I^*, M^*)$.

4. Stability of Equilibria

In this section we present the locally and globally asymptotical stability of the equilibria of subsystem S_1 and subsystem S_2 .

Theorem 2. The disease-free equilibrium E_0^2 is locally asymptotically stable if $R_0 < 1$ and unstable when $R_0 > 1$.

Proof. The characteristic equation corresponding to the subsystem S_1 at E_0^2 is the form

$$(\lambda + b) [\lambda - \beta_0 K + (\gamma + d)] (\lambda + \nu) = 0, \quad (13)$$

where λ is the eigenvalue. We get

$$\begin{aligned} \lambda_1 &= -b, \\ \lambda_2 &= \beta_0 K - (\gamma + d) = (\gamma + d)(R_0 - 1), \\ \lambda_3 &= -\nu. \end{aligned} \quad (14)$$

Thus, E_0^2 is locally asymptotically stable if $R_0 < 1$ and unstable when $R_0 > 1$. The proof of the disease-free equilibrium of the subsystem S_2 is similar to S_1 ; then this proof is omitted. $\square$

Theorem 3. The disease-free equilibrium E_0^2 is globally asymptotically stable if $R_0 < 1$.

Proof. To establish the global stability of the disease-free equilibrium of subsystem S_2 , we consider the positive definite function $V(t) = I(t)$ by using Lyapunov's method. Now differentiating V with respect to t , we get

$$\begin{aligned} \frac{dV}{dt} &= [\beta e^{-mM} S - (\gamma + d)] I < [\beta S - (\gamma + d)] I \\ &= (R_0 - 1)(\gamma + d) I. \end{aligned} \quad (15)$$

If $R_0 < 1$, then $dV/dt \leq 0$. Thus, LaSalle's invariance principle implies that E_0^2 is globally asymptotically stable in Ω . We can use the same positive definite function when we prove the global stability of the disease-free equilibrium of subsystem S_1 . $\square$

In fact, when $R_0 < 1$ curves starting from G_2 will converge to $(K, 0, 0)$ that belongs to G_1 , and curves starting from G_1 also converge to $(K, 0, 0)$; therefore, $E_0^2 = (K, 0, 0)$ is globally asymptotically stable. In the following we consider the stability of the endemic equilibrium E_0^* of subsystem S_1 and E_* of subsystem S_2 . According to Hurwitz criterion, it is easy to get the following proposition.

Proposition 4. The unique endemic equilibrium E_0^* of subsystem S_1 is locally asymptotically stable if $R_0 > 1$. The unique endemic equilibrium E_* of subsystem S_2 is locally asymptotically stable if $R_0 > 1$.

FIGURE 1: The stability of the endemic equilibria E_0^* and E_* .

When the endemic equilibrium E_0^* belongs to G_1 , we get $1 < R_0 < b/(b - \beta_0 I_c)$ from the expression of I_0^* , so if $R_0 > b/(b - \beta_0 I_c)$, E_0^* belongs to G_2 , which lead to the existence of E_* that belongs to G_2 . Let $R_c = b/(b - \beta_0 I_c)$; then we have the following result.

Theorem 5. *When $1 < R_0 < R_c$ the endemic equilibrium E_0^* is globally asymptotically stable; when $R_0 > R_c$ the endemic equilibrium E_* is globally asymptotically stable.*

Proof. Let a Dulac function

$$D(S, I, M) = \frac{1}{SI}, \quad (16)$$

and we make $f_1 = b(1 - S/K)S - \beta_0 e^{-mM} SI$, $f_2 = \beta_0 e^{-mM} SI - (\gamma + d)I$, and $f_3 = \sigma I - \nu M$ for the subsystem S_2 . Then

$$\frac{\partial(Df_1)}{\partial S} + \frac{\partial(Df_2)}{\partial I} + \frac{\partial(Df_3)}{\partial M} = -\frac{b}{KI} - \frac{\nu}{SI} < 0. \quad (17)$$

Hence, we can exclude the existence of limit cycles in region G_2 . The same method can be applied in discussion for region G_1 .

When $1 < R_0 < R_c$, it is easy to get the endemic equilibrium E_0^* that belongs to G_1 and all curves starting from G_1 will verge to E_0^* . We know that all curves starting from G_2 will converge to E_* , but E_* also belongs to G_1 if $1 < R_0 < R_c$, and there is a unique endemic equilibrium and no limit cycles in region G_1 ; hence, curves starting from G_2 will enter G_1 and converge to E_0^* . Thus, when $1 < R_0 < R_c$ the endemic equilibrium E_0^* is globally asymptotically stable. Similarly when $R_0 > R_c$ the endemic equilibrium E_0^* belongs to G_2 ; hence, curves starting from G_1 will enter G_2 and converge to the unique endemic equilibrium E_* in region G_2 . So when $R_0 > R_c$ the endemic equilibrium E_* is globally asymptotically stable. $\square$

5. Numerical Simulations and Results

To check the analysis in Section 4 about E_0^* and E_* , we do some numerical simulations in this section.

When $1 < R_0 < R_c$, we set the following values of parameters: $b = 0.00095$, $K = 500000$, $\beta_0 = 0.0000001$, $m = 0.05$, $\gamma = 0.003$, $d = 0.0002$, and $I_c = 9000$, and we get $R_0 = 8.8$, $R_c = 12.3$, and $1 < R_0 < R_c$. Next we consider another case $R_0 > R_c$ by taking $b = 0.001$, $\beta_0 = 0.00000009$, and $I_c = 10000$, and get $R_0 = 13.2$, $R_c = 10$. We, respectively, choose two sets of initial values that belong to G_1 ($I_0 < I_c$) and two sets of initial values that belong to G_2 ($I_0 > I_c$). Thus, we get $I_0^* = 8771 < 9000$, $I_* = 10680 > 10000$; see Figure 1. As shown in Figure 1(a) the trajectories all approach towards E_0^* no matter whether $I_0 \in G_1$ or G_2 . Hence, E_0^* is globally asymptotically stable when $1 < R_0 < R_c$. Whereas when $R_0 > R_c$ the trajectories approach towards E_* what Figure 1(b) responses. In conclusion, E_0^* and E_* are globally asymptotically stable if $R_0 > 1$.

In the following we consider the impact of media coverage on the spread of diseases by drawing the curves between $I(t)$, $S(t)$, and m , respectively, as shown in Figure 2. It is shown that the number of $I(t)$ reduces with the increase of m , which is opposite to $S(t)$. This is common sense that media coverage influences people's behavior and reminds them to prevent themselves from the diseases through staying at home, reducing travel or quarantine, and so on. So it is clear that media coverage plays a key role in the preventing and controlling the diseases.

6. Discussion

In this paper, a nonlinear mathematical model has been proposed and analyzed to describe the impact of media coverage on the transmission dynamics of infectious diseases. Considering the fact that public mass media usually do not work when the number of the infected individuals is generally small at the initial stages of prevalence of the disease, and by incorporating a piecewise continuous transmission rate to represent that media coverage has its effects only when

FIGURE 2: The curves of $I(t)$ and $S(t)$ with different m .

the number of the infected individuals exceeds a certain level. The disease-free equilibrium has been shown to be globally asymptotically stable when the basic reproduction number $R_0 < 1$. When $R_0 > 1$ and $0 < m < m_0$ sufficiently small, it leads to the global stability of endemic equilibrium. But when $1 < R_0 < R_c$ the trajectories all approach towards E_0^* whenever initial starts $\in G_1$ or G_2 , and the trajectories approach towards E_* if $R_0 > R_c$. Numerical simulations prove the stability of equilibria and the impact of media coverage in the spread of diseases, which suggest that if we want to reduce the number of the infected individuals, we should increase media coverage.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

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