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# AN SEI INFECTION MODEL INCORPORATING MEDIA IMPACT

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ABSTRACT. To study the impact of media coverage on spread and control of infectious diseases, we use a susceptible-exposed-infective (SEI) model, including individuals' behavior changes in their contacts due to the influences of media coverage, and fully investigate the model dynamics. We define the basic reproductive number  $\Re_0$  for the model, and show that the modeled disease dies out regardless of initial infections when  $\Re_0 < 1$ , and becomes uniformly persistently endemic if  $\Re_0 > 1$ . When the disease is endemic and the influence of the media coverage is less than or equal to a critical number, there exists a unique endemic equilibrium which is asymptotical stable provided  $\Re_0$  is greater than and near one. However, if  $\Re_0$  is larger than a critical number, the model can undergo Hopf bifurcation such that multiple endemic equilibria are bifurcated from the unique endemic equilibrium as the influence of the media coverage is increased to a threshold value. Using numerical simulations we obtain results on the effects of media coverage on the endemic that the media coverage may decrease the peak value of the infectives or the average number of the infectives in different cases. We show, however, that given larger  $\Re_0$ , the influence of the media coverage may as well result in increasing the average number of the infectives, which brings challenges to the control and prevention of infectious diseases.

1. Introduction. Media coverage, awareness campaign or programs, and public education have played an important and significant role in the control and prevention of the spread of infectious diseases, such as influenza, AIDS/HIV, and SARS. It has been shown that media coverage may change individuals' behavior, while the epidemic is in progress, such as reducing the individuals' contacts or using more safer prevention strategies, to reduce the risk of getting infected [1–3, 19]. Indeed,

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people's response to the threat of disease is dependent on their perception of risk, which is influenced by public and private information disseminated widely by the media [33]. In a recent study for outbreaks of infectious diseases with high morbidity and mortality, Mummert and Weiss showed that individuals closely follow media reports of the outbreak, and that many will attempt to minimize contacts with other individuals in order to protect themselves from infection [30]. Therefore, understanding the effects of behavior changes due to influences of media coverage and awareness programs can help guide more effective media plans and strategies on the control and preventions of diseases.

Various mathematical models have been formulated for such purposes. The media coverage and awareness programs can be incorporated into disease transmission models in a more explicit way where an equation or equations for the media coverage or awareness programs are included into compartmental disease models [8, 27–29]. They can also be incorporated implicitly by being connected to individuals' behavior changes which are expressed in the contact rates or other model terms and therefore the infection or incidence rates [10,30,33,35–37]. In the latter case, several types of media influence-dependent incidence rates have been proposed to reflect the influences of media coverage. They include the reduction in contacts due to behavioral change when the number of infectious individuals increases, and have used such forms as  $e^{-a_1E-a_2I-a_2H}$ , where H denotes hospitalized individuals, I the infectives, E exposed individuals, with nonnegative constants  $m, a_i, i = 1, 2, 3, [23],$ 1/(1+mI) [26],  $\beta e^{-mI}$  [10,23,26], and  $c_1 - c_2 f(I)$ , where  $f(0) = 0, f'(I) \ge 0$ , and  $\lim_{I\to\infty} f(I) = 1, [11,32,33]$ . Piecewise smooth incidence rates to represent the reduction factor are also used in [36,37].

Employing distinct functions for media coverage in similar disease models can certainly exhibit different model dynamics. The following three distinct functions to present the effects of mass media are identified and compared from various perspectives in [9]

$$f(I, p_1) = e^{-p_1 \gamma I}, \quad f(I, p_2) = \frac{1}{1 + p_2 I^2}, \quad f(I, p_3) = 1 - \frac{1}{p_3 + I}.$$

It is demonstrated in [9] that, based on a same SEIR (susceptible-exposed-infectiousrecovered) compartmental model, the epidemic curves and key epidemic measurements vary depending on the media functions chosen. In particular, even the models with the distinct media functions have a very similar shape and the same basic reproductive number  $\Re_0$  at the beginning of the epidemic, the entire resulting epidemic curves can vary drastically.

We note that while the influence of media coverage is an important factor in the spread of diseases, it usually does not impact the disease transmissions so rapidly or sensitively as some other factors and thus the reactions to the media coverage from the public are normally slow and delayed. Susceptible individuals may lack the knowledge or information and tend to ignore the media coverage or awareness campaigns when a disease first spreads, especially for those less fatal diseases. As the disease spreads more widely or more severely, the public pays more attention to the media coverage, and individuals then more seriously change their behavior. As a result, the media impact gradually becomes more significant as the infections are clearly increased. Apparently, most of the media functions used in many studies mentioned above, such as  $e^{-mI}$  and 1/(1 + mI), do not characterize such special features of slow reactions of the public to the media impact appropriately because they both decrease rapidly as I increases. Nevertheless, while function f(I) =

 $1/(1+aI^2)$  is also a decreasing for  $I \ge 0$ , it has such a feature that

$$f''(I) \begin{cases} < 0, & \text{for } I < 1/\sqrt{a}, \\ > 0, & \text{for } I > 1/\sqrt{a}, \end{cases}$$

and may thus be used more appropriately to reflect the individuals slow reactions.

To explore the dynamic features of the disease models with this media function and investigate its possibly different impact on the disease transmissions, we consider an SEI model with media coverage or awareness programs in this paper. We assume that the media coverage or awareness programs directly affect individuals' contact rates. We first present the model system with its basic dynamical properties in Section 2. We then derive a formula for the reproductive number  $\Re_0$ , and show the global stability of the disease free equilibrium as  $\Re_0 < 1$  in Section 3. We prove the uniform persistence of the model for  $\Re_0 > 1$ , determine the existence of endemic equilibria and their stability, and verify the occurrence of Hopf-bifurcation with varying media coverage in section 4.1, to do that, we will develop the methods and techniques in [10, 16, 21, 22]. Numerical simulations are provided in Section 5.

2. The model. Considering the transmission of infectious diseases in some regions, we divide the population into the groups of susceptible individuals, denoted by S(t), individuals exposed to the infection but not yet infectious, denoted by E(t), and infected individuals who are infectious, denoted by I(t). We assume that after their recovery, the infective individuals no longer impose risk to the susceptible individuals. We further assume that the population follows the logistic growth in the absence of infection and then have the following baseline SEI model, in a more general setting, for the transmission dynamics:

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

$$\frac{dE}{dt} = \Lambda S - (c+d)E,$$

$$\frac{dI}{dt} = cE - \gamma I,$$
(1)

where b is the intrinsic growth rate of the human population, K is the carrying capacity for the human population of a given region, 1/c is the incubating period, d is the natural death rate,  $\gamma > d$  is the removal rate of the infectives which includes the natural death rate, and  $\Lambda$  is the infection rate.

The infection rate is given by

$$\Lambda=\beta\xi\frac{I}{N}$$

where  $\beta$  is the transmission probability,  $\xi$  is the number of contacts per individual per unit of time, and N = S + E + I. We assume random mixing between individuals such that  $\xi = rN$ , and the contact factor r characterizes the behavior change of individuals according to the infection level in the region. We then incorporate the influence of media coverage into the model by assuming the contact factor to be a function of the number of infectives with the form of

$$r(I) = \frac{m}{1 + aI^2},$$

where m is the maximum effectiveness of the media coverage, and  $a \ge 0$  measures the sensitivity of individuals to the infection level in the region. Substituting the infection rate into (1), we arrive at the following model system

$$\frac{dS}{dt} = bS\left(1 - \frac{S}{K}\right) - \frac{\mu I}{1 + aI^2}S,$$

$$\frac{dE}{dt} = \frac{\mu I}{1 + aI^2}S - (c + d)E,$$

$$\frac{dI}{dt} = cE - \gamma I,$$
(2)

where we write  $\mu := \beta m$  for convenience.

Define region

$$D := \left\{ (S, E, I) \in \mathbb{R}^3 : S, E, I \ge 0, S + E + I \le \frac{bK}{l} \right\},\$$

where  $l := \min\{b, d, \gamma\}$ . Then D is positively invariant for system (2), and for any given initial condition  $(S(0), E(0), I(0)) \in D$  with S(0) > 0, there exists a unique solution of system (2) with S(t) > 0, for all  $t \ge 0$ .

3. Basic reproduction number and system permanence. We first derive a formula for the basic reproduction number,  $\Re_0$ , for system (2). Following the procedure introduced in [34],  $\Re_0 = \rho(FV^{-1})$ , where  $\rho$  represents the spectral radius of a matrix, and we have two vectors  $\mathbb{F}$  and  $\mathbb{V}$  to represent the new infection term and remaining transfer terms, respectively:

$$\mathbb{F} = \begin{pmatrix} \frac{\mu SI}{1+aI^2} \\ 0 \\ 0 \end{pmatrix}, \mathbb{V} = \begin{pmatrix} (c+d)E \\ -cE + \gamma I \\ -bS(1-\frac{S}{K}) + \frac{\mu SI}{1+aI^2} \end{pmatrix}.$$

The infected compartments are E and I. A straightforward calculation yields .

$$F = \begin{pmatrix} 0 & \mu K \\ 0 & 0 \end{pmatrix}, V = \begin{pmatrix} c+d & 0 \\ -c & \gamma \end{pmatrix},$$
$$FV^{-1} = \frac{1}{\gamma(c+d)} \cdot \begin{pmatrix} \mu Kc & \mu K(c+d) \\ 0 & 0 \end{pmatrix}.$$

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Hence the basic reproduction number for system (2) is

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$$\Re_0 = \rho(FV^{-1}) = \frac{\mu cK}{\gamma(c+d)}$$

and then from [34], the disease-free equilibrium  $E_1 := (K, 0, 0)$  of system (2) is locally asymptotically stable if  $\Re_0 < 1$  and unstable if  $\Re_0 > 1$ .

We next show that  $E_1$  is globally asymptotically stable as well if  $\Re_0 < 1$ .

**Theorem 3.1.** For model (2), if  $\Re_0 < 1$ , the disease-free equilibrium  $E_1$  is globally asymptotically stable.

*Proof.* We define a Lyapunov functional for system (2) as

$$U(t) := S(t) - K \ln \frac{S(t)}{K} - K + E(t) + \frac{c+d}{c}I(t).$$

Thus we have  $U(t) \ge 0$  for  $t \ge 0$ , and U(t) = 0 if and only if S(t) = K, E(t) = 0, I(t) = 0. Differentiating U(t) along the solutions of system (2), we obtain

$$\begin{aligned} \frac{\mathrm{d}U}{\mathrm{d}t}\Big|_{(2)} &= \frac{S-K}{S} \cdot \left(bS(1-\frac{S}{K}) - \frac{\mu SI}{1+aI^2}\right) + \frac{\mu SI}{1+aI^2} - (c+d)E \\ &+ \frac{c+d}{c} \cdot (cE - \gamma I) \\ &= -\frac{b}{K} \cdot (S-K)^2 + \frac{\mu KI}{1+aI^2} - \frac{\gamma(c+d)}{c} \cdot I \\ &= -\frac{b}{K} \cdot (S-K)^2 + \frac{\gamma(c+d)}{c} \cdot \left(\frac{\Re_0}{1+aI^2} - 1\right) \cdot I \le 0. \end{aligned}$$

Set

$$\mathcal{A}_0 = \{ (S, E, I) \in D | U' = 0 \} \,.$$

Then  $U^{'} = 0$  if and only if

$$S(t) = K, \quad I(t) = 0.$$
 (3)

Substituting (3) into the second equation of system (2) then yields E(t) = 0. By the LaSalle-Lyapunov theorem ([18], Theorem 3.4.7), the largest compact invariant set of  $\mathcal{A}_0$  is the singleton point  $E_1$ . Thus we conclude that  $E_1$  is globally attractive in D. Since  $E_1$  is locally asymptotically stable,  $E_1$  is globally asymptotically stable in D as  $\Re_0 < 1$ .

We further show that system (2) is permanence if the basic reproduction number  $\Re_0 > 1$ .

**Theorem 3.2.** Assume  $\Re_0 > 1$ . Then there is a positive constant  $\epsilon$  such that every solution (S(t), E(t), I(t)) of (2) in D satisfies

$$\lim_{t \to \infty} \inf(S(t), E(t), I(t)) \ge \eta := (\epsilon, \epsilon, \epsilon),$$

and thus system (2) is permanence.

*Proof.* Define sets

$$X := \{ (S, E, I) : S \ge 0, E \ge 0, I \ge 0 \},\$$
  
$$X_0 := \{ (S, E, I) \in X : S > 0, E > 0, I > 0 \},\$$

and

$$\partial X_0 := X \setminus X_0.$$

Let  $\phi(t) : X \to X$  be the solution semiflow of system (2). As shown above, the solution semiflow  $\phi(t)$  of (2) has a global attractor on X. Since  $X_0$  is relatively closed in X and system (2) is positively invariant and point dissipative in  $D, X_0$  is positively invariant for  $\phi$ .

Define

$$\Omega_{\partial} := \{ \phi(t) \in \partial X_0, \forall t \ge 0 \}.$$

We now claim that

$$\Omega_{\partial} = \{ \phi(t) \in \partial X_0 : E(t) = 0 \text{ and } I(t) = 0, \forall t \ge 0 \}.$$

$$(4)$$

Let  $\phi(t) \in \Omega_{\partial}$ . Without loss of generality, we only need to prove that  $E(t) = 0, \forall t \ge 0$ .

Assume otherwise. Then there exists nonnegative constant  $t_0$  such that  $E(t_0) > 0$ . Following the definition of  $\Omega_{\partial}$ , one must have  $I(t_0) = 0$ . Notice by system (2)

that X is invariant for  $\phi$ . Thus by the second equation in (2), we have  $\frac{dE(t)}{dt} \ge -(c+d)E(t), \forall t \ge t_0$ , showing that  $E(t) > 0, \forall t \ge t_0$ .

 $-(c+d)E(t), \forall t \geq \iota_0$ , showing that  $E(v_t \geq v_0)$ , we have  $\left. \frac{dI(t)}{dt} \right|_{t=t_0} = cE(t_0) > 0$ . On the other hand, by the last equation in (2), we have  $\left. \frac{dI(t)}{dt} \right|_{t=t_0} = cE(t_0) > 0$ . Thus, we get I(t) > 0 for any  $t > t_0$  and  $t - t_0$  sufficiently small. Similarly to the arguments above, we also have I(t) > 0,  $\forall t > t_0$ . Thus we get I(t) > 0, E(t) > 0,  $\forall t > t_0$ , which contradicts  $\phi(t) \in \partial X_0, \forall t \geq 0$ .

We now let

$$\Psi_0 := \bigcap_{x \in Z_0} w(x)$$

Here  $Z_0$  is the global attractor of  $\phi(t)$  restricted to  $\partial X_0$ . We claim that  $\Psi_0 = \{E_0\} \bigcup \{E_1\}$ . In fact,  $\Psi_0 \subseteq \Omega_\partial = \{(S(t), 0, 0)\}$ . From system (2), we obtain S = 0 or S = K. Thus  $E_0, E_1 \in \Psi_0$ .

Since  $\{E_0\}, \{E_1\}$  are two isolated invariant sets of  $\phi(t)$  in  $\Omega_\partial$ , using the similar arguments from Theorem 3.1 and  $\Re_0 > 1$ , we can prove that  $E_1$  is asymptotically stable in  $\Omega_\partial$ , defined in (4). Hence  $\Psi_0$  has an acyclic covering.

Next, we prove that  $W^s((0,0,0)) \cap X_0 = \phi$ . Suppose that it is not true. Then for any  $\epsilon_1 > 0$ , there exists  $T_0 > 0$  such that  $(S(t), E(t), I(t)) < \xi_1 := (\epsilon_1, \epsilon_1, \epsilon_1)$ , as  $t > T_0$ .

By the first equation of (2), we have

$$\frac{\dot{S}}{S} = b\left(1 - \frac{S}{K}\right) - \frac{\mu I}{1 + aI^2} > b\left(1 - \frac{\epsilon_1}{K}\right) - \mu\epsilon_1 > 0, \ t > T_0,$$

where we let a = 0 and  $\epsilon_1$  small enough. Thus  $S(t) \to \infty$  as  $t \to \infty$ , which leads to a contradiction.

Next, we prove  $W^s((K, 0, 0)) \cap X_0 = \phi$ . Suppose that it is not true. For any  $\epsilon_2 > 0$ , there exists  $T_1 > 0$  such that  $(|S(t) - K|, E(t), I(t)) < \xi_2 := (\epsilon_2, \epsilon_2, \epsilon_2)$ , as  $t > T_1$ .

By the second equation and the third equation in (2) and a = 0, we have

$$\begin{pmatrix} \dot{E} \\ \dot{I} \end{pmatrix} = \begin{pmatrix} \frac{\mu SI}{1+aI^2} - (c+d)E \\ cE - \gamma I \end{pmatrix} \ge \begin{pmatrix} -(c+d) & \mu(K-\epsilon_2) \\ c & -\gamma \end{pmatrix} \cdot \begin{pmatrix} E \\ I \end{pmatrix}.$$

Let

$$A = \begin{pmatrix} -(c+d) & \mu(K-\epsilon_2) \\ c & -\gamma \end{pmatrix}.$$

The characteristic polynomial of A takes the form

$$\frac{\lambda + (c+d)}{-c} \frac{-\mu(K-\epsilon_2)}{\lambda + \gamma} = \lambda^2 + (c+d+\gamma)\lambda + (c+d)\gamma - c\mu(K-\epsilon_2).$$

As  $\epsilon_2$  small enough and  $\Re_0 = \frac{\mu cK}{\gamma(c+d)} > 1$ , we have  $(c+d)\gamma - c\mu(K-\epsilon_2) < 0$ . Thus A has a simple positive eigenvalue. Using the comparison theorem, we have either  $E(t) \to \infty$  or  $I(t) \to \infty$ , as  $t \to \infty$ . By [39, Theorem 1.3.2], we conclude that there exists  $\xi > 0$  such that  $\lim_{t\to\infty} \inf(S(t), E(t), I(t)) \ge \xi$ . This shows the uniform persistence of solutions of system (2).

4. Equilibria and media impact. Letting the right hand side of (2) equal zero, we find that the origin  $E_0 = (0, 0, 0)$  is an equilibrium with eigenvalues  $b, -(c + d), -\gamma$ , and model (2) has one disease free equilibrium at  $E_1 = (K, 0, 0)$ . Clearly,  $E_0$  is a hyperbolic saddle point.

From model (2), an endemic equilibrium satisfies the following equations:

$$bS\left(1-\frac{S}{K}\right) = \frac{\mu SI}{1+aI^2} = \frac{(c+d)\gamma I}{c},\tag{5}$$

which leads to

$$\mu S - \alpha (1 + aI^2) = 0, \tag{6}$$

$$bS(K-S) - \alpha KI = 0, \tag{7}$$

where we write  $\alpha := \frac{(c+d)\gamma}{c}$ . Solving (6) for *I* yields

$$I = f_1(S) = \sqrt{\frac{\mu}{a\alpha} \left(S - \frac{\alpha}{\mu}\right)},\tag{8}$$

which is a parabola for  $S \geq \frac{\alpha}{\mu}$ . Define

$$I = f_2(S) = \sqrt{\frac{b}{K\alpha}(K - S)},\tag{9}$$

for  $0 \leq S \leq K$ .

Clearly, the curves of functions  $f_1$  and  $f_2$  intersect once if  $\frac{K}{2} < \frac{\alpha}{\mu} < K$ , that is  $1 < \Re_0 \leq 2$ . The situation for  $\frac{\alpha}{\mu} < \frac{K}{2}$  could be complex. it follows from (6) and (7) that

$$F(I) = I^4 + \frac{1}{a}(2 - \Re_0)I^2 + \frac{\mu \Re_0}{a^2 b}I + \frac{1}{a^2}(1 - \Re_0) = 0.$$
(10)

Notice that as  $\Re_0 > 1$ , equation (10) has at least one positive root and has at most three positive roots. More specifically, equation (10) has one positive root as  $1 < \Re_0 \leq 2$ . To investigate the existence of multiple endemic equilibria, we only consider the Case  $\Re_0 > 2$ .

We first give a simple lemma for convenience.

Lemma 4.1. The cubic equation

$$H(x) := Ax^3 - Bx + C = 0,$$

with A, B, and C all positive, has no, one, or two positive solutions if and only if

$$27AC^2 > 4B^3$$
,  $27AC^2 = 4B^3$ ,  $27AC^2 < 4B^3$ ,

respectively.

*Proof.* Function H has a positive critical point  $\bar{x} = \sqrt{\frac{B}{3A}}$ , and then has no, one, or two positive solutions if and only if  $H(\bar{x}) > 0$ ,  $H(\bar{x}) = 0$ , or  $H(\bar{x}) < 0$ , respectively. Substituting  $\bar{x}$  into  $H(\bar{x})$  leads to

$$H(\bar{x}) = \frac{1}{3}\bar{x}(3A\bar{x}^2 - B) + \frac{1}{3}B\bar{x} - B\bar{x} + C = C - \frac{2}{3}B\bar{x}.$$

Then the conclusion follows directly by substituting  $\bar{x}$  into  $H(\bar{x})$  again.

Apply Lemma 4.1 to

$$F'(I) = 4I^3 + \frac{2}{a}(2 - \Re_0)I + \frac{\mu \Re_0}{a^2 b} = 0$$
(11)

and define

$$a_h := \frac{27\mu^2 \Re_0^2}{8b^2 (\Re_0 - 2)^3}.$$
(12)

Then F'(I) has no critical points, and thus F(I) has a unique positive solution if  $a \leq a_h$ .

Assume  $a > a_h$ . Then F'(I) has two critical points  $0 < I_1 < I_2$ ; that is, F(I) has two local extreme values at  $I_k$ , k = 1, 2.

We consider the case where F(I) = 0 has two positive solutions. To this end, it is necessary to have a unique positive solution to both F(I) = 0 and F'(I) = 0. Substituting (11) into (10) yields

$$F(I) = I\left(I^3 + \frac{1}{2a}(2 - \Re_0)I + \frac{\mu\Re_0}{4a^2b}\right) + \frac{1}{2a}(2 - \Re_0)I^2 + \frac{3\mu\Re_0}{4a^2b}I + \frac{1}{a^2}(1 - \Re_0)$$
$$= \frac{1}{2a}(2 - \Re_0)I^2 + \frac{3\mu\Re_0}{4a^2b}I + \frac{1}{a^2}(1 - \Re_0) := G(I).$$

It follows from  $\Re_0 > 2$  that the quadratic equation G(I) = 0 has a unique positive solution if and only if

$$\left(\frac{3\mu\Re_0}{4a^2b}\right)^2 = 4\left(\frac{1}{2a}(2-\Re_0)\frac{1}{a^2}(1-\Re_0)\right),$$

that is

$$a = a_c := \frac{9\mu^2 \Re_0^2}{32b^2(2 - \Re_0)(1 - \Re_0)}.$$
(13)

Under condition (13), the unique positive solution to G(I) = 0 is

$$I = \frac{3\mu\Re_0}{4a^2b}\frac{2a}{2(\Re_0 - 2)} = \frac{3\mu\Re_0}{4ab(\Re_0 - 2)}.$$
 (14)

Substituting (14) into F'(I) = 0 leads to

$$\left(\frac{3\mu\Re_0}{4ab(\Re_0-2)}\right)^3 + \frac{2-\Re_0}{2a}\frac{3\mu\Re_0}{4ab(\Re_0-2)} + \frac{\mu\Re_0}{4a^2b} = \left(\frac{3\mu\Re_0}{2ab(\Re_0-2)}\right)^3 - \frac{\mu\Re_0}{2a^2b} = 0,$$

or equivalently,

$$a = \frac{27\mu^2 \Re_0^2}{8b^2(\Re_0 - 2)^3} = a_h$$

That is, there exists a positive solution to both F(I) = 0 and F'(I) = 0 if only if  $a = a_c = a_h$ . However, as shown above, F(I) has a unique positive solution if  $a = a_h$ . Therefore, it is impossible to have two positive solutions to F(I) = 0.

Under the assumption of  $a > a_h$ , there exist two critical points  $I_1 < I_2$  to F(I). If  $F(I_1)F(I_2) < 0$ , function F(I) has three positive solutions. In summary, we have

**Theorem 4.2.** Assume  $R_0 > 2$ . Equation (10) has a unique positive solution if  $a \leq a_h$  where  $a_h$  is given in (12). It is impossible to have two positive solutions to F(I) = 0 in any case. If  $a_c > a_h$ , function F(I) has two critical points  $0 < I_1 < I_2$ , and if  $F(I_1)F(I_2) < 0$ , equation (10) has three positive solutions.

**Proposition 1.** Consider model (2) with all parameters positive. Clearly we could always find parameter  $a_0 = \frac{\mu cbS^*}{\Re_0} + bc(\frac{2S^*}{K} - 1)(\frac{\mu K}{\Re_0} - \frac{2\mu K^2}{\Re_0^2 S^*})$ . If  $\Re_0 > 1$ , then model (2) has one or three positive equilibrium. Furthermore,

- if  $0 < a \leq a_0$ , the model has a unique endemic equilibrium;
- if  $a > a_0$ , the model has three endemic equilibria;

4.1. Endemic equilibrium for sufficiently small media impact *a*. In this section, we study the stability and Hopf bifurcation of the endemic equilibria and determine how the media impact can influence the periods of the oscillations of disease transmissions.

The case a = 0 is same as [10], and from [10] we have the following conclusions:

• When a = 0, model (2) has a unique endemic equilibrium  $(S_0^*, E_0^*, I_0^*)$ , and the associate characteristic equation of model (2) is

$$\lambda^3 + (c+d+\gamma + \frac{b}{\Re_0})\lambda^2 + \frac{b}{\Re_0}(c+d+\gamma)\lambda + b\gamma(c+d)(1-\frac{1}{\Re_0}) = 0.$$

Let

$$R_{H_0} = \frac{1}{2} \left[ 1 + \frac{(c+d+\gamma)^2}{\gamma(c+d)} + \sqrt{1 + \frac{2(c+d+\gamma)(2b+c+d+\gamma)}{\gamma(c+d)} + \frac{(c+d+\gamma)^4}{\gamma^2(c+d)^2}} \right].$$
 (15)

Obviously, for any positive parameters we have  $R_{H_0} > 1$ .

• For model (2) with a = 0, the endemic equilibrium  $(S_0^*, E_0^*, I_0^*)$  is locally asymptotically stable if  $1 < \Re_0 < R_{H_0}$ .

• For model (2) with a = 0, when  $\Re_0 = R_{H_0}$ ,  $(S_0^*, E_0^*, I_0^*)$  becomes unstable and model (2) undergoes a Hopf bifurcation.

4.2. Stability for  $\Re_0$  slightly larger than 1. When  $\Re_0 > 1$  and  $0 \le a \le a_0$ , model (2) has a unique endemic equilibrium  $E_2 = (S^*, E^*, I^*)$ . The Jacobian matrix evaluated at  $E_2$  is

$$J_{E_2} = \begin{pmatrix} -\frac{bS^*}{K} & 0 & \frac{\mu K}{\Re_0} - \frac{2\mu K^2}{\Re_0^2 S^*} \\ b(1 - \frac{S^*}{K}) & -(c+d) & -\frac{\mu K}{\Re_0} + \frac{2\mu K^2}{\Re_0^2 S^*} \\ 0 & c & -\gamma \end{pmatrix},$$

and the characteristic equation of  $J_{E_2}$  is given by

$$\lambda^3 + a_2 \lambda^2 + a_1 \lambda + a_0 = 0, \tag{16}$$

where

$$a_{2} = c + d + \gamma + \frac{bS^{*}}{K}, a_{1} = \frac{bS^{*}}{K}(c + d + \gamma) + \frac{2\mu cK}{\Re_{0}} - \frac{2\mu cK^{2}}{\Re_{0}^{2}S^{*}}.$$
(17)

The coordinates of the endemic equilibrium  $(S^*, E^*, I^*)$  are smooth functions of a. When  $0 < a \leq a_0$  is sufficiently small, we can expand the coordinates for  $S^*$  as

$$S^* = S_0^* + aS_1^* + O(a^2) \tag{18}$$

where particularly, by (5) we have

$$S_0^* = \frac{K}{\Re_0}, \ S_1^* = S_0^* \frac{b}{\mu} (1 - \frac{1}{\Re_0}).$$
(19)

Next, we study the impact of the media coverage on the dynamics of the disease transmissions, and consider the case of  $0 < a \leq a_0$  and sufficiently small.

It is easy to verify that (16) has a pair of purely imaginary roots if and only if  $a_1a_2 = a_0$ . Let

$$\Delta = a_1 a_2 - a_0. \tag{20}$$

If  $\Delta = 0$ , the endemic equilibrium has a pair of purely imaginary roots. Now we calculate equation (20) as follows:

$$\begin{split} \Delta &= a_1 a_2 - a_0 \\ &= (c + d + \gamma + \frac{bS^*}{K}) \left[ \frac{bS^*}{K} (c + d + \gamma) + \frac{2\mu cK}{\Re_0} - \frac{2\mu cK^2}{\Re_0^2 S^*} \right] \\ &\quad - \frac{\mu cbS^*}{\Re_0} - bc (\frac{2S^*}{K} - 1) (\frac{\mu K}{\Re_0} - \frac{2\mu K^2}{\Re_0^2 S^*}) \\ &= \frac{1}{\Re_0^2 S^*} \left[ \frac{b\Re_0^2 S^{*2}}{K} (c + d + \gamma)^2 + 2\mu cK \Re_0 S^* (c + d + \gamma) \right. \\ &\quad - 2\mu cK^2 (c + d + \gamma) + \frac{b^2 \Re_0^2 S^{*3}}{K^2} (c + d + \gamma) + b\mu c(K \Re_0 S^* \\ &\quad + 2KS^* - \Re_0 S^{*2} - 2K^2) \right]. \end{split}$$

By (17), (18) and (19), we have

$$\Delta = \frac{1}{\Re_0^5 S^*} \cdot (\widetilde{\Delta}(a, \Re_0) + O(a^2)), \tag{22}$$

where

$$\begin{split} \widetilde{\Delta}(a, \Re_{0}) &= \frac{b\Re_{0}^{5}(c+d+\gamma)^{2}}{K} \left[ \frac{K^{2}}{\Re_{0}^{2}} + \frac{2abK^{2}}{\mu\Re_{0}^{2}} (1-\frac{1}{\Re_{0}}) \right] \\ &+ 2\mu c K \Re_{0}^{4}(c+d+\gamma) \left[ \frac{K}{\Re_{0}} + \frac{abK}{\mu\Re_{0}} (1-\frac{1}{\Re_{0}}) \right] \\ &- 2\mu c K^{2} \Re_{0}^{3}(c+d+\gamma) + \frac{b^{2} \Re_{0}^{5}(c+d+\gamma)}{K^{2}} \left[ \frac{K^{3}}{\Re_{0}^{3}} + \frac{3abK^{3}}{\mu\Re_{0}^{3}} (1-\frac{1}{\Re_{0}}) \right] \\ &- \frac{1}{\Re_{0}} \right] + bc \mu \Re_{0}^{3} \left[ (2K+K\Re_{0}) \left[ \frac{K}{\Re_{0}} + \frac{abK}{\mu\Re_{0}} (1-\frac{1}{\Re_{0}}) \right] \right] \\ &- 2K^{2} - \Re_{0}^{1} \left[ \frac{K^{2}}{\Re_{0}^{2}} + \frac{2abK^{2}}{\mu\Re_{0}^{2}} (1-\frac{1}{\Re_{0}}) \right] \right] \\ &= \Re_{0}^{3} \left[ bK(c+d+\gamma)^{2} (1+\frac{2ab}{\mu}) + 2cK^{2}ab(c+d+\gamma) + bc \mu K^{2} (\frac{ab}{\mu}-1) \right] \\ &+ \Re_{0}^{2} \left[ -\frac{2ab^{2}K(c+d+\gamma)^{2}}{\mu} + b^{2}K(c+d+\gamma)(1+\frac{3ab}{\mu}) \right] \\ &- 2abcK^{2}(c+d+\gamma) - ab^{2}cK^{2} + b\mu cK^{2} \right] \\ &+ \Re_{0} \left[ -\frac{3ab^{3}K(c+d+\gamma)}{\mu} \right]. \end{split}$$

It is not difficult to verify that  $\Delta = 0$  is equivalent to  $\widetilde{\Delta}(a, \Re_0) = 0$ . Note that when  $\widetilde{\Delta}(a, \Re_0) = 0$ , the endemic equilibrium has a pair of purely imaginary roots  $\lambda = \pm \omega i$ , where

$$\omega^{2} = \frac{bS^{*}}{K}(c+d+\gamma) + \frac{2\mu cK}{\Re_{0}} - \frac{2\mu cK^{2}}{\Re_{0}^{2}S^{*}}.$$
(24)

If the parameters a and  $\Re_0$  satisfy  $\widetilde{\Delta}(a, \Re_0) = 0$ , a Hopf bifurcation could occur. Next we investigate the function determined by  $\widetilde{\Delta}(a, \Re_0) = 0$ .

**Lemma 4.3.** Consider  $\widetilde{\Delta}(a, \Re_0) = 0$  for  $\Re_0 > 1$  and  $0 < a \leq a_0$ . In the neighborhood of  $(0, R_{H_0})$ , there exists a unique smooth function  $\Re_0 = \Re_0(a)$  such that

 $\widetilde{\Delta}(a, \Re_0(a)) = 0$  for  $0 < a \le a_0$  sufficiently small. Furthermore, we have

$$\Re_0(a) = R_{H_0} + aR_{H_1} + O(a^2), \tag{25}$$

where  $R_{H_0}$  is defined as in (15) and satisfy

$$R_{H_0}^2 \gamma(c+d) - R_{H_0}[(c+d+\gamma)^2 + \gamma(c+d)] - b(c+d+\gamma) = 0$$
(26)

and

$$R_{H_{1}} = \frac{-R_{H_{0}}^{2} \left[\frac{2b^{2}K(c+d+\gamma)^{2}}{\mu} + 2bcK^{2}(c+d+\gamma) + b^{2}cK^{2}\right]}{3R_{H_{0}}[bK(c+d+\gamma)^{2} - b\mu cK^{2}] + 2[b^{2}K(c+d+\gamma) + b\mu cK^{2}]} \\ + \frac{R_{H_{0}} \left[\frac{2b^{2}K(c+d+\gamma)^{2}}{\mu} - \frac{3b^{3}K(c+d+\gamma)}{\mu} + 2bcK^{2}(c+d+\gamma) + b^{2}cK^{2}\right]}{3R_{H_{0}}[bK(c+d+\gamma)^{2} - b\mu cK^{2}] + 2[b^{2}K(c+d+\gamma) + b\mu cK^{2}]} \\ + \frac{\frac{3b^{3}K(c+d+\gamma)}{\mu}}{3R_{H_{0}}[bK(c+d+\gamma)^{2} - b\mu cK^{2}] + 2[b^{2}K(c+d+\gamma) + b\mu cK^{2}]}.$$
(27)

Proof. Note that

 $\widetilde{\Delta}(0, R_{H_0}) = R_{H_0}^3 \left[ bK(c+d+\gamma)^2 - bcK^2\mu \right] + R_{H_0}^2 \left[ b^2K(c+d+\gamma) + bcK^2\mu \right] = 0$ where in the neighborhood of  $(0, R_T)$ , we have

where in the neighborhood of 
$$(0, R_{H_0})$$
, we have

$$\frac{\partial \widetilde{\Delta}}{\partial \Re_{0}}|_{a=0,\Re_{0}=R_{H_{0}}} = 3R_{H_{0}}^{2} \left[ bK(c+d+\gamma)^{2} - bcK^{2}\mu \right] 
+2R_{H_{0}} \left[ b^{2}K(c+d+\gamma) + bcK^{2}\mu \right] 
= -R_{H_{0}} \left[ b^{2}K(c+d+\gamma) + bcK^{2}\mu \right] \neq 0.$$
(28)

then by the Implicit Function Theorem, there exists a unique smooth function  $\Re_0 = \Re_0(a)$  such that  $\widetilde{\Delta}(a, \Re_0(a)) = 0$  for a > 0 sufficiently small. If we write the Taylor expansion for  $\Re_0(a)$  in terms of a in (25) and bring it into (23), we have

$$\begin{aligned} \Delta(a.\Re_0(a)) &= \widetilde{\Delta}(a, R_{H_0} + aR_{H_1} + O(a^2)) \\ &= (R_{H_0}^3 + 3aR_{H_0}^2R_{H_1})[bK(c+d+\gamma)^2(1+\frac{2ab}{\mu}) + 2cK^2ab(c+d+\gamma) \\ &+ bc\mu K^2(\frac{ab}{\mu} - 1)] + (R_{H_0}^2 + 2aR_{H_0}R_{H_1})[-\frac{2ab^2K(c+d+\gamma)^2}{\mu} \\ &+ b^2K(c+d+\gamma)(1+\frac{3ab}{\mu}) - 2abcK^2(c+d+\gamma) - ab^2cK^2 \\ &+ b\mu cK^2] + (R_{H_0} + aR_{H_1})[-\frac{3ab^3K(c+d+\gamma)}{\mu}] = 0 \end{aligned}$$
(29)

Equalizing the same power terms of parameter a on both sides in (29), at the same time taking into (26), we have

$$\begin{aligned} &3R_{H_0}^2 R_{H_1} [bK(c+d+\gamma)^2 - b\mu cK^2] \\ &+ R_{H_0}^3 [\frac{2b^2 K(c+d+\gamma)^2}{\mu} + 2bcK^2(c+d+\gamma) + b^2 cK^2] \\ &+ 2R_{H_0} R_{H_1} [b^2 K(c+d+\gamma) + b\mu cK^2] \end{aligned}$$

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$$+R_{H_{0}}^{2}\left[-\frac{2b^{2}K(c+d+\gamma)^{2}}{\mu}+\frac{3b^{3}K(c+d+\gamma)}{\mu}-2bcK^{2}(c+d+\gamma)-b^{2}cK^{2}\right]$$
$$+R_{H_{0}}\left[-\frac{3b^{3}K(c+d+\gamma)}{\mu}\right]$$
$$=0$$
(30)  
we solve  $R_{H_{1}}$ .

thus we solve  $R_{H_1}$ .

**Theorem 4.4.** Consider model (2). If  $1 < \Re_0 < \Re_0(a)$ , where  $\Re_0(a)$  is defined in (25), then  $E_2 = (S^*, E^*, I^*)$  is locally asymptotically stable.

*Proof.* When  $R_0 > 1$ , if  $a_2 > 0$ ,  $a_0 > 0$  and  $a_2a_1 - a_0 > 0$ , then all eigenvalues of (16) have negative real parts by Routh-Hurwitz criteria [31], we can prove this conclusion. Obviously,  $a_2 > 0$ . In the following, we need to prove  $a_0 > 0$  and  $a_2a_1 - a_0 > 0$ . By (18) and (19), we have for a > 0 small that

$$a_{0} = \frac{\mu cbS^{*}}{\Re_{0}} + bc(\frac{2S^{*}}{K} - 1) \cdot (\frac{\mu K}{\Re_{0}} - \frac{2\mu K^{2}}{\Re_{0}^{2}S^{*}})$$

$$= \frac{\mu cb}{\Re_{0}S^{*}} \cdot (3S^{*2} - KS^{*} - \frac{4KS^{*}}{\Re_{0}} + \frac{2K^{2}}{\Re_{0}})$$

$$= \frac{\mu cbK^{2}}{\Re_{0}^{2}S^{*}} \cdot \left[ (1 - \frac{1}{\Re_{0}})(1 + \frac{2ab}{\Re_{0}\mu} - \frac{ab}{\mu}) + O(a^{2}) \right].$$
(31)

if  $1 < \Re_0 \le 2$ , obviously  $a_0 > 0$ ; if  $2 < \Re_0$ , from relationship of root and coefficient in (10), we can obtain  $a < \frac{\mu \Re_0}{b(\Re_0 - 2)}$ , the same as  $a_0 > 0$ .

Next, we prove  $a_1a_2 - a_0 > 0$ . By (18) and (19), we straightforward calculate

$$a_{2}a_{1} - a_{0} = \left(c + d + \gamma + \frac{bS^{*}}{K}\right) \cdot \left[\frac{bS^{*}}{K}(c + d + \gamma) + \frac{2\mu cK}{\Re_{0}} - \frac{2\mu cK^{2}}{\Re_{0}^{2}S^{*}}\right] \\ - \left[\frac{\mu cbS^{*}}{\Re_{0}} + bc(\frac{2S^{*}}{K} - 1) \cdot \left(\frac{\mu K}{\Re_{0}} - \frac{2\mu K^{2}}{\Re_{0}^{2}S^{*}}\right)\right] \\ = \frac{bK}{\Re_{0}^{5}S^{*}} \cdot \left[\Re_{0}^{3}(\triangle^{2} - \mu cK + T_{1}a) + \Re_{0}^{2}(b\triangle + \mu cK - T_{1}a) + T_{2}a) - \Re_{0}T_{2}a + T_{2}a + O(a^{2})\right] \\ = \frac{bK}{\Re_{0}^{5}S^{*}} \cdot \left[\Re_{0}^{3}(\triangle^{2} - \mu cK) + \Re_{0}^{2}(b\triangle + \mu cK) + \Re_{0}^{2}T_{1}a(\Re_{0} - 1) + \Re_{0}T_{2}a(\Re_{0} - 1)\right] > 0$$

$$(32)$$

where  $\triangle = c + d + \gamma$ ,  $T_1 = \frac{2b(c+d+\gamma)^2}{\mu} + 2cK(c+d+\gamma) + bcK$  and  $T_2 = \frac{3b^2(c+d+\gamma)}{\mu}$ . Hence according to Routh-Hurwitz criteria [31],  $E_1 = (S^*, E^*, I^*)$  is locally asymptotically asymptotic definition. totically stable when  $1 < \Re_0 < \Re_0(a)$  and a > 0 is sufficiently small. 

### 4.3. Hopf bifurcation for $\Re_0$ larger than some critical value.

**Theorem 4.5.** Consider model (2). If  $\Re_0 = \Re_0(a)$ ,  $0 < a \leq a_0$ , then system undergoes a Hopf bifurcation.

*Proof.* Differentiating (16) with respect  $\mu$ , we get

$$\frac{\mathrm{d}\lambda}{\mathrm{d}\mu} = -\frac{\mathrm{d}S^*}{\mathrm{d}\mu} \cdot \frac{B_1}{B_2},\tag{33}$$

where

$$B_{1} = \frac{b}{K}\lambda^{2} + \left[\frac{b}{K}(c+d+\gamma) + \frac{2Kr(c+d)}{\Re_{0}S^{*2}}\right]\lambda + b\gamma(c+d)\left(\frac{3}{K} - \frac{2K}{\Re_{0}S^{*2}}\right), \\ B_{2} = 3\lambda^{2} + 2(c+d+\gamma + \frac{bS^{*}}{K})\cdot\lambda + \frac{bS^{*}}{K}(c+d+\gamma) + \frac{2\mu cK^{2}}{\Re_{0}} - \frac{2\mu cK^{2}}{\Re_{0}^{2}S^{*}}.$$
(34)

When  $\Re_0 = \Re_0(a)$ , equation (16) has a pair of purely imaginary roots  $\lambda = \pm \omega i$  with  $\omega^2 = \frac{bS^*}{K}(c+d+r) + \frac{2\mu cK}{\Re_0} - \frac{2\mu cK^2}{\Re_0^2 S^*}$ . Note that

$$S^* = \frac{\gamma(c+d)}{\mu c} + \frac{ab^2 c(K-S^*)^2 S^{*2}}{\mu K^2 \gamma(c+d)},$$

so

$$\frac{\mathrm{dS}^{*}}{\mathrm{d}\mu} = -\frac{S^{*}}{\mu} + \frac{2ab^{2}c(K-S^{*})(K-2S^{*})S^{*}}{\mu K^{2}\gamma(c+d)} \cdot \frac{\mathrm{dS}^{*}}{\mathrm{d}\mu}$$
$$\frac{\mathrm{dS}^{*}}{\mathrm{d}\mu} = -\frac{bcS(K-S)}{\Re_{0}a_{0}}.$$

Hence we have  $\frac{dS^*}{d\mu} < 0$ . Following from (33) and  $\frac{dS^*}{d\mu} < 0$ , we get

$$\begin{aligned} & sign\left\{\frac{\mathrm{d}(\mathrm{Re}\lambda)}{\mathrm{d}\mu}\right\}_{\lambda=i\omega} \\ &= sign\left\{Re\left(\frac{\mathrm{d}\lambda}{\mathrm{d}\mu}\right)\right\}_{\lambda=i\omega} \\ &= sign\left\{Re\left(\frac{\frac{b}{K}\omega^2 - \left[\frac{b}{K}(c+d+\gamma) + \frac{2K\gamma(c+d)}{\Re_0S^{*2}}\right] \cdot i\omega - b\gamma(c+d)\left(\frac{3}{K} - \frac{2K}{\Re_0S^{*2}}\right)\right)\right\} \\ &= sign\left\{Re\left(\frac{b}{K}\omega^2 - \left[\frac{b}{K}(c+d+\gamma) + \frac{bS^*}{K}\right] \cdot i\omega + \frac{bS^*}{K}(c+d+\gamma) + \frac{2\mu cK^2}{\Re_0} - \frac{2\mu cK^2}{\Re_0^2S^*}\right)\right\} \\ &= sign\left\{Re\left(\frac{b}{K}\omega^2 - \left[\frac{b}{K}(c+d+\gamma) + \frac{2K\gamma(c+d)}{\Re_0S^{*2}}\right] \cdot i\omega - b\gamma(c+d)\left(\frac{3}{K} - \frac{2K}{\Re_0S^{*2}}\right)\right) \right. \\ &\times (-2\omega^2 - 2(c+d+\gamma + \frac{bS^*}{K}) \cdot i\omega)\right\} \\ &= sign\left\{\frac{2b}{K}\omega^4 + 2\omega^2\left[\frac{b}{K}(\gamma - \frac{c+d}{2})^2 + \frac{3(c+d)^2}{4} + cd\right] + (c+d+\gamma)\frac{b^2S^*}{K^2} + (c+d+\gamma+b+\frac{bS^*}{K})\frac{2K\gamma(c+d)}{\Re_0S^{*2}}\right]\right\} > 0. \end{aligned}$$

$$(35)$$

Therefore, the system undergoes a Hopf bifurcation when  $\Re_0 = \Re_0(a)$  and a is small.

5. Numerical simulations. To analyze our results, we provide numerical examples in this section.

We choose parameter a from Table 1. The other parameters K is 5000000 people, b is selected as 0.001  $day^{-1}$ , c is chosen as 0.1  $day^{-1}$ , d is 0.001  $day^{-1}$  and  $\mu$  is selected as  $1.2 \times 10^{-8} day^{-1}$ . We let  $\gamma$  and a vary.

First, we consider the case where the removal rate from the infected compartment is relatively higher, that is  $\gamma = 0.05 \ day^{-1}$ . We then  $\Re_0 = 1.1765$  and  $R_{H_0} = 5.5206$ . As shown in Fig. 1 (a), (b), the green thin curve represents the case when a = 0, where the application of media was not consider; the blue thick curve and the red thicker curve represent the cases when  $a = 1 \times 10^{-11}$  and  $a = 1 \times 10^{-10}$ , respectively. In Table 1, if  $\gamma = 0.05 \ day^{-1}$ ,  $\Re_0 = 1.1765$  and  $R_{H_0} = 5.5206$ , we solve for the solutions of system (2) as a increases from 0 to  $1 \times 10^{-11}$ , to  $1 \times 10^{-10}$ , and we found that  $S^*$  becomes gradually larger and  $E^*$ ,  $I^*$  become smaller when a increases. From Fig. 1 and Table 1, it is not difficult to find that the endemic equilibrium is local

TABLE 1. Endemic equilibrium  $(S^*, E^*, I^*)$  when a > 0 is varied. In the table, expect for the parameters given in Table 2, here we have  $\gamma = 0.05$ . In this case,  $\Re_0 = 1.1765$  and  $R_{H_0} = 5.5206$ 

Parameter a	$S^*$	$E^*$	$I^*$
a = 0 $a = 1 \times 10^{-11}$ $a = 1 \times 10^{-10}$	$\begin{array}{c} 4208333\\ 4215551\\ 4272153\end{array}$	$\begin{array}{c} 6597 \\ 6548 \\ 6157 \end{array}$	$     13194 \\     13096 \\     12314 $

asymptotically stable, and the effective media coverage (larger values of a) not only stabilizes the oscillation but also reduces the number of the infected individuals in the course of transmission.

Fig. 2 (a), (b) simulate the media impact to the transmission when  $\gamma = 0.02$   $day^{-1}$ . As shown in Fig. 2 (a), (b), the trough of the number of susceptible individuals reduce and come sooner, but the peak rise and retard as the parameters  $\gamma$  decreases, simultaneously the trough of the infected individuals also reduce and come sooner, nevertheless the peak of the infected individuals rise and come sooner. The number of the peak and the trough of the susceptible individuals or the infected individuals all improve when the media impact parameter a increases, and when a increases to a specific value, the number of the susceptible individuals and the infected individuals tend to stable, but is less than  $\gamma = 0.05$ .

Fig. 3 describes the change of the maximum infected individuals when a increases from 0 to  $1 \times 10^{-8}$ . It is obvious that the maximum value of infected individuals is from falling sharply to steady reduction as the media impact parameter a increases.

6. Discussion. In this paper, we explore the impact of media coverage a to the transmission of infection diseases. In [10], Cui et al. used a contact transmission rate  $\beta(I) = \mu e^{-mI}$ . However, the contact transmission rate in [10] fails to satisfy that it is unsensitive to the increase of infectious diseases, at least in the early outbreak stage. For further study, we consider the more realistic media function  $\beta(I) = \frac{\mu SI}{1+aI^2}$  to reflect the impact of media coverage and alertness. Comparing to contact transmission rate in model [10], our media function is weaken in the beginning period of the outbreak for some infectious diseases, and once the media impact reaches to a certain degree, the declining on the infectives for the contact transmission rate tends to gentle.

We derive formulas for the basic reproductive number of infection  $\Re_0$ . If  $\Re_0 < 1$  (Theorem 3.1), the disease free equilibrium  $E_1$  is globally asymptotically stable. If  $\Re_0 > 1$  (Theorem 3.2), system (2) is uniformly persistent with endemic equilibrium. If  $0 \le a \le a_0$ , the model is shown to have only a unique endemic equilibrium, and system (2) is uniformly persistent with the unique endemic equilibrium. If  $a > a_0$ , it is shown that the model may have three endemic equilibria and it is uniformly persistent with the maximum endemic equilibrium. The case of a = 0 has been studied by Cui et al. in [10], so we only investigate the case of  $0 < a \le a_0$  with a sufficiently small. When  $0 < a \le a_0$  and  $1 < \Re_0 < \Re_0(a)$ , where  $\Re_0(a)$  is defined in 25, then  $E_2 = (S^*, E^*, I^*)$  is locally asymptotically stable (Theorem 4.4). If  $\Re_0 = \Re_0(a), 0 < a \le a_0$ , then system (2) undergoes a Hopf bifurcation (Theorem 4.5).



(b) The number of the infective I(t)

FIGURE 1. Effects of media impact *a* on the value of S(t), I(t) under different media impacts. Here,  $\gamma = 0.05$  and the initial point are all  $(5 \times 10^6, 1, 1)$ ;  $\Re_0 = 1.1765$  and  $R_{H_0} = 5.5206$ .

From the numerical simulations, we obtain that the media coverage a impacts multiple peaks and troughs. In fact, if  $\Re_0 > 1$  and close to  $R_{H_0}$ , the disease will be endemic with multiple peaks and troughs when a = 0. The time between the two peaks or two troughs can be approximated by

$$T_0 = \frac{2\pi}{\omega} = \frac{2\pi}{\sqrt{\frac{b}{\Re_0}(c+d+\gamma)}}.$$



(a) The number of susceptible S(t) under different media impacts a



(b) The number of the infective I(t) under different a

FIGURE 2. Effects of media impact *a* on the value of S(t), I(t) under different media impacts. Here  $\gamma = 0.02$ , the initial point is  $(5 \times 10^6, 1, 1)$ ;  $\Re_0 = 3$  and  $R_{H_0} = 8.2523$ .

But when the media impact parameter a is introduced, or when  $0 < a \leq a_0$  is sufficiently small, if there are multiple peaks and multiple troughs, the time between each of the two peaks or the two troughs can be approximated by

$$T_a = \frac{2\pi}{\sqrt{\frac{b(c+d+\gamma)}{\Re_0} + a\frac{b^2(c+d+\gamma)}{\mu\Re_0}}(1-\frac{1}{\Re_0}) + \frac{2\mu cK}{\Re_0}(1-\frac{1}{1+a\frac{b}{\mu}(1-\frac{1}{\Re_0})})}$$

This shows that the media alert shortens the time of the secondary peak and trough of the disease transmission. This effect is also verified by the simulations in Fig. 2 (a), (b).



FIGURE 3. The peak value of the infective number  $I_{max}$  when a from 0 to  $a = 1 \times 10^{-8}$ .

For the case where  $\Re_0 > 2$ , because of the complexity of the contact transmission rate, we are unable to calculate the endemic equilibrium of system (2). The stability analysis for each of the solutions seems analytically untractable when system (2) has three endemic equilibria, and further investigations of the impact of parameter a on the model dynamics become more challenging, that we leave in our future work.

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