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INTERACTION OF MEDIA AND DISEASE DYNAMICS AND ITS IMPACT ON EMERGING INFECTION MANAGEMENT

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Abstract. The 2002-2003 SARS outbreaks exhibited some distinct features such as rapid spatial spread, massive media reports, and fast self-control. These features were shared by the 2009 pandemic influenza and will be experienced by other emerging infectious diseases. We focus on the dynamic interaction of media reports, epidemic outbreak and behavior change in the population and formulate a compartmental model, that tracks the evolution of the human population. Such population is characterized by the disease progression (susceptible, infected, hospitalized, and recovered) and by the extent to which the media has impacted, so individuals have modified their behaviors to reduce their transmissibility and infectivity. The model also describes the dynamics of media reports by considering how media is influenced by the disease statistics (numbers of infected and hospitalized individuals, for example). We then conduct linear stability analysis and numerical simulations to study how interaction of media reports and disease progress affects the disease transmission dynamics, so as to shed light on what type of media will be the most effective for the control of an epidemic.

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1. **Introduction.** For infectious diseases, specially for emerging diseases, the interaction between awareness programs driven by media and infection control is complicated and highly nonlinear. On the one hand, the severity of the disease influences the number of awareness programs. On the other hand, the number of awareness programs influences the perception of disease [33], alerts the public, and affects individual behaviors, including contact patterns [4], which in turn impact the effectiveness of disease intervention measures [26, 24].

Extensive studies investigated the impact of media reports and behavior reaction on the propagation of emerging diseases [2, 22, 29]. Drache and Feldman [7] investigated the impact of media reports on 2003 SARS outbreak in Toronto and offered effective strategies for the crisis. Jones and Salathe [17] studied the relationship between emotional status and behavioral response of humans by performing an online survey of H1N1. Ferguson [8], Safi and Agusto [25], Agusto and Gumel [3], Gumel, McCluskey, and Watmough [11] presented several effective methods to prevent further spread of disease dissemination. Sun et al. [28] formulated an SIS model to investigate the impact of media reports on disease transmission and concluded that media coverage reduces the number of contacts and shortens the duration of disease outbreak. Moreover, Vaidya et al. [15] analyzed the interaction mechanism among Avian influenza, heterogeneous environment and behavior responses to prevent the spreading of disease.

Two fundamentally different approaches can be used to study the impact of media effect on disease spread. In the first classical approach, the scholars use different forms of incidence function to measure media effect. For example, Wang and Xiao [31] introduced a piecewise continuous transmission rate $\beta exp(-\alpha\epsilon I)SI$ to study the medial impact in disease spread, with $\epsilon = 1$ while $\sigma(S, I) = I - I_c > 0$ and $\epsilon = 0$ while $\sigma(S, I) = I - I_c < 0$, where I_c denotes certain threshold of infected individuals above which mass media starts to report the infectious disease. Cui,Tao, and Zhu [6] incorporated a standard incidence $\frac{\beta SI}{S+I}$ with classical SIS model to explore the influence of media coverage on the dissemination of infectious disease. Cui, Sun, and Zhu [5] applied an exponential incidence $\mu exp(-mI)$ and Liu et al.[20, 21] applied $\beta exp(-a_1E - a_2I - a_3H)$ to investigate the impact of media reports on disease spread. Li, Ma, and Cui [19] considered incidence rate $(\beta_1 - \frac{\beta_2 I}{m+I})\frac{SI}{N}(\beta_1 > \beta_2 > 0, m > 0)$ to reflect the effect of media reports on disease spread. Gao and Ruan [12], Hu, Ma, and Ruan [16] proposed a nonlinear incidence rate $\frac{\beta SI}{1+\alpha I}(\alpha \geq 0)$ as the incidence exhibits near-linear behavior when the infected individuals is low and approximates a constant when infected number is large. Xiao and Ruan [32] proposed a type of incidence function $\frac{kSI}{1+\alpha I^2}$,with kI measures the infection force and $\frac{1}{1+\alpha I^2}$ depicts the media effect to investigate the impact of media coverage on disease dissemination. Arino and McCluskey [1] assumed that a low population level leads to the appearance of mass action incidence, and a high population level induces the proportional incidence.

In the second approach, scholars incorporated the mass action incidence with an independent media effect function to study the influence of media reports on disease spread. For example, Misra et al. [23, 27] used the mass action incidence βXY and media impact term λXM with $X(t)$ denotes the susceptible population, $Y(t)$ denotes the infective population, and $M(t)$ describes the cumulative number of awareness programs and the growth rate of which is proportional to the number of infected population. Yuan, Xue, and Liu [34] combined the mass incidence $\lambda k S_k \Theta$ with the media effect independent function to explore the interaction between media

and disease spread on complex networks, where $\lambda = \frac{\sum_{j=1}^{n} jI_j}{\sum_{k=1}^{n} kN_k}$, N_k represents the number of nodes with degree k. Funk et al. [9, 10]. concluded that media awareness have impact on people's behavior either by reducing susceptibility or by promoting recovery rate, and assumed that the growth rate of aware population is proportional to the number of infected individuals and the proportional coefficient is determined by the rate of infected becoming aware.

In this paper, we focus on the dynamic interaction of media reports, disease outbreak, and behavior change in the population. We formulate a compartmental model that tracks the evolution of the population stratified by the disease progression (susceptible, infected, hospitalized, and recovered) and by the extent to which the media has impacted, so individuals have reduced their transmissibility and infectivity. We also model the dynamics of media reports by considering how media is influenced by the disease statistics (numbers of infected and hospitalized individuals, for example). The goal is to gain insights on how the nonlinear interaction of media reports and disease progress affects the daily incidence of infection, the accumulated cases over an epidemic, the number of hospitalized individuals, and the disease induced death rate. In such a way, we try to shed light on what type of media will be the most effective for the disease infection management, including both medical (such as vaccine) and non-medical interventions, so the optimal choice of focus could be determined and incorporated in the chosen goal(s) by the management.

2. **A disease spreading model with consideration of media reports.** Media reports and disease outbreaks are mutually dependent and interact with each other. It is therefore important to refine classical mathematical models to reflect this feature by adding the new dimension of massive news reports and fast information flow. Furthermore, media reports and fast information transmission generate a profound psychological impact on public and have great influence not only on individual behaviors but also on the formation and implementation of public intervention and control policies. Once a disease breaks out, media programs scramble to report the disease to alert the public.

We consider the interaction of disease outbreak and media impact into a susceptible-infected-hospitalized-recovered framework. We divide the individuals into six compartments: S_m, S_u, I_m, I_u, H, R, which represent the number of individuals that are susceptible aware, susceptible unaware, infected aware, infected unaware, hospitalized, and recovered, respectively. Let $M_e(t)$ denote the number of awareness programs driven by media reports at time t. For simplicity, in what follows, the symbol M_e is used instead. The graphical illustration of the disease spreading model with consideration of media reports (media impact model) is shown in Figure 1. The parameter list is described in Table 1.

Our model on the interaction of media and disease dynamics process is developed on the following simplified assumptions:

The growth rate of awareness programs increases in proportional to the total number of infected individuals $(I_u + I_m)$ at a rate ρ_i and the number of hospitalized individuals H at a rate ρ_h. Moreover, the number of awareness programs decays exponentially at a rate ρ. So ρ_i and ρ_h can be regarded as a kind of rates of infected and hospitalized becoming aware, and ρ is the decreasing rate of awareness due to ineffectiveness. We refer the reader to [32, 1, 23, 27, 34] for details.

Infected aware individuals in subclass I_m have the infectivity reduced by a factor $\delta_i (0 \leq \delta_i \leq 1)$ due to alertness or self-imposed quarantine. Similarly, susceptible

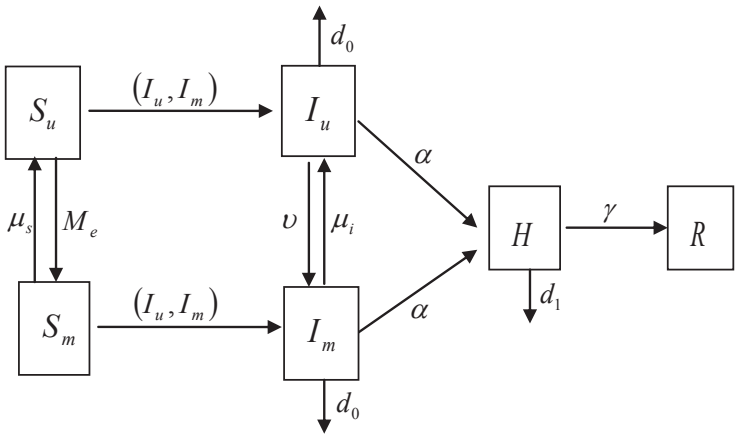


FIGURE 1. Flow chart for the media impact model.

TABLE 1. Meaning of parameters

d_1	disease-induced death rate of hospitalized individuals
d_0	disease-induced death rate of infected individuals
α	hospitalization rate of infected individuals
γ	recovery rate of hospitalized individuals
ν	self-awareness rate ν
β_m	the transmission rate of awareness
β_u	infection rate from subclass I_u to S_u
μ_s	awareness loss rate of susceptible individuals
μ_i	awareness loss rate of infected individuals
δ_s	reduced susceptibility factor
δ_i	reduced infectivity factor

aware individuals in S_m have the susceptibility reduced by a factor $\delta_s (0 \leq \delta_s \leq 1)$ as they lower contact with infected and hospitalized individuals. Specifically, symbol β_u represents infection rate from infected unaware to susceptible unaware. $\beta_u\delta_s$ denotes infection rate from infected unaware to susceptible aware. $\beta_u\delta_i$ is the infection rate from infected aware to susceptible unaware. $\beta_u\delta_s\delta_i$ describes the infection rate from infected aware to susceptible aware.

Individuals in the compartment S_u move to compartment S_m with the transmission rate β_m by receiving media reports. Individuals in compartment I_u move to compartment I_m at a self-awareness rate ν as when they are sick, they will be isolated and informed of the dangerous disease by hospital staffs.

Individuals in S_m move back to S_u at rate μ_s, and I_m move back to I_u at rate μ_i due to awareness loss after a certain period of time.

When susceptible unaware individuals S_u contact with infected ones including I_m and I_u, they may become infected unaware I_u. Similarly, when susceptible aware individuals S_m contact with infected ones, they may become infected aware I_m.

We do not consider the demographic process such as natural death and birth rates because the time scale of the epidemic is usually much shorter than the timescale

of natural death and birth. Thus, the media impact model can be described by the following equations

$$\begin{cases} \dfrac{dS_m}{dt} = -\beta_u \delta_s S_m \left(I_u + \delta_i I_m\right) + \beta_m S_u M_e - \mu_s S_m, \\ \dfrac{dS_u}{dt} = -\beta_u S_u \left(I_u + \delta_i I_m\right) - \beta_m S_u M_e + \mu_s S_m, \\ \dfrac{dI_m}{dt} = \beta_u \delta_s S_m \left(I_u + \delta_i I_m\right) + \nu I_u - \left(d_0 + \alpha + \mu_i\right) I_m, \\ \dfrac{dI_u}{dt} = \beta_u S_u \left(I_u + \delta_i I_m\right) + \mu_i I_m - \left(d_0 + \alpha + \nu\right) I_u, \\ \dfrac{H}{dt} = \alpha \left(I_u + I_m\right) - \left(d_1 + \gamma\right) H, \\ \dfrac{dR}{dt} = \gamma H, \\ \dfrac{dM_e}{dt} = \rho_i \left(I_u + I_m\right) + \rho_h H - \rho M_e. \end{cases} \tag{1}$$

As the duration of the epidemic is short and the number of susceptible individuals is much higher than the total number of infected and hospitalized individuals, we simplify the assumptions that the total number of the susceptible is unchanged. The assumption is similar to those in [21, 9]. Then, we introduce $S = S_m + S_u$ and $I = I_m + I_u$ to denote the total numbers of susceptible and infected individuals, respectively. According to the assumption that $S = S_m + S_u$ is a constant, then S_u is equal to $S - S_m$. Thus, at the disease free equilibrium and the endemic equilibrium, we have the following equations

$$\begin{cases} \dot{S_m} = -\beta_u \delta_s S_m \left(I_u + \delta_i I_m\right) + \beta_m (S - S_m) M_e - \mu_s S_m, \\ \dot{I_m} = \beta_u \delta_s S_m \left(I_u + \delta_i I_m\right) + \nu I_u - \left(d_0 + \alpha + \mu_i\right) I_m, \\ \dot{I_u} = \beta_u (S - S_m) \left(I_u + \delta_i I_m\right) + \mu_i I_m - \left(d_0 + \alpha + \nu\right) I_u, \\ \dot{H} = \alpha \left(I_u + I_m\right) - \left(d_1 + \gamma\right) H, \\ \dot{M_e} = \rho_i \left(I_u + I_m\right) + \rho_h H - \rho M_e. \end{cases} \tag{2}$$

For the analysis of system (2), we define the invariant region which is given by the following set

$$\mathcal{D} = \{(S_m, I_m, I_u, H, M_e) | S_m, I_m, I_u, H, M_e \geq 0\}.$$

When the disease breaks out, authorities at all levels take precautions including both medical and non-medical measures to minimize the negative effects of disease. Some measures involved susceptible individuals such as education, vaccination and raising the alertness can prevent being infected. Other measures involved infected individuals such as isolation can avoid passing infection to susceptible individuals.

In fact, the media impact model is a more general one. A special case of $\delta_s = 0$ implies that susceptible aware individuals avoid being infected either by reducing contact with infected ones or by seeking for vaccine protection. The model studying the effects of awareness programs on infectious disease dissemination is a special case of our model if we simply combine the infected aware, infected unaware, and hospitalized individuals into one class [9]. Another special case of $\delta_i = 0$ has been studied by Misra et al., who modeled the disease transmission process by assuming

that infected aware individuals cannot pass infection to susceptible individuals as they have been isolated [23].

3. **The analysis of periodic oscillatory behaviors.** To illustrate the impact of media reports on people's behavior, we calculate the disease free equilibrium $E_0(0,0,0,0,0)$.

According to the definition of Van den Driessche and Watmough [30], we identify the infected compartments as I_m and I_u. We assume the rate of appearance on new infections is $\mathcal{W}$ and rate of transfer of individuals out of I_m and I_u is $\mathcal{V}$, thus

$$\mathcal{W} = \begin{bmatrix} \beta_u \delta_s S_m \left(I_u + \delta_i I_m\right) \\ \beta_u (S - S_m) \left(I_u + \delta_i I_m\right) \end{bmatrix}, \mathcal{V} = \begin{bmatrix} -\nu I_u + \left(d_0 + \alpha + \mu_i\right) I_m \\ -\mu_i I_m + \left(d_0 + \alpha + \nu\right) I_u \end{bmatrix}.$$

Then the partial derivatives of $\mathcal{W}$ and $\mathcal{V}$ with respect to I_m and I_u are respectively described as

$$W = \begin{bmatrix} \beta_u \delta_i \delta_s S_m & \beta_u \delta_s S_m \\ \beta_u (S - S_m) \delta_i & \beta_u (S - S_m) \end{bmatrix}, V = \begin{bmatrix} d_0 + \alpha + \mu_i & -\nu \\ -\mu_i & d_0 + \alpha + \nu \end{bmatrix}.$$

As the basic reproduction number R_0 equals the spectral radius of next generation matrix WV^{-1}, that is $R_0 = \rho(WV^{-1})$, so we have

$$R_0 = \frac{\beta_u S \left(d_0 + \alpha + \mu_i + \delta_i \nu\right)}{\left(d_0 + \alpha\right)\left(d_0 + \alpha + \mu_i + \nu\right)}.$$

Based on the analysis, we present a theorem to determine whether the infectious disease can break out or not.

Theorem 3.1. *If $R_0 < 1$, the disease free equilibrium $E_0(0,0,0,0,0)$ is locally asymptotically stable in $\mathcal{D}$, where the biological region $\mathcal{D} = \{(S_m, I_m, I_u, H, M_e) \in \boldsymbol{R}_+^5\}$. If $R_0 > 1$, the disease free equilibrium $E_0(0,0,0,0,0)$ is unstable.*

Proof. Let I denote the identity matrix, then we have the characteristic equation

$$|\lambda I - J| = \begin{vmatrix} \lambda + d_0 + \alpha + \mu_i & -\nu & 0 & 0 & 0 \\ -\beta_u S \delta_i - \mu_i & \lambda + d_0 + \alpha + \nu - \beta_u S & 0 & 0 & 0 \\ 0 & 0 & \lambda + \mu_s & 0 & -\beta_m S \\ -\alpha & -\alpha & 0 & \lambda + d_1 + \gamma & 0 \\ -\rho_i & -\rho_i & 0 & -\rho_h & \lambda + \rho \end{vmatrix}$$
$$= (\lambda + d_1 + \gamma)(\lambda + \rho)(\lambda + \mu_s)[\lambda^2 + (2d_0 + 2\alpha + \mu_i + \nu - \beta_u S)\lambda + (d_0 + \alpha)(d_0 + \alpha + \mu_i + \nu)(1 - R_0)].$$

According to Vieta's formula we can see that all the five eigenvalues have negative real part if $R_0 < 1$. It is easy to conclude that the disease free equilibrium E_0 is locally asymptotically stable in $\mathcal{D}$ if $R_0 < 1$, which means that the disease may die out quickly around the disease free equilibrium. Otherwise, if $R_0 > 1$, the disease may break out and remain endemic. □

3.1. **Endemic equilibrium structure.** In this part, we analyze the endemic equilibrium structure in detail. For simplicity, we introduce three symbols p_1, p_2 and p_3, where

$$p_1 = \frac{\rho_i(\gamma + d_1) + \alpha\rho_h}{\rho(\gamma + d_1)},$$

$$
\begin{aligned}
p_2 &= \frac{(d_0+\alpha)\,(d_0+\alpha+\mu_i+\nu)}{d_0+\alpha+\mu_i+\delta_i\nu},\\
p_3 &= \frac{\delta_s(d_0+\alpha+\nu-\beta_u S)-\nu}{\delta_s(\beta_u S\delta_i+\mu_i)-(d_0+\alpha+\mu_i)}.
\end{aligned}
\tag{3}
$$

Then, we can calculate the endemic equilibrium $E_1(S_m^*, I_m^*, I_u^*, H^*, M_e^*)$.
(1): If $\delta_s \neq 0$, and let $\Psi_1 = \beta_m\beta_u S\delta_s$, then the endemic equilibrium can be written as the following form

$$
\begin{aligned}
I_u^* &= \frac{\mu_s[(d_0+\alpha+\mu_i)p_3-\nu]}{\Psi_1 p_1(1+p_3)(1+\delta_i p_3)-[(d_0+\alpha+\mu_i)p_3-\nu][\beta_m p_1(1+p_3)+\beta_u\delta_s(1+\delta_i p_3)]},\\
I_m^* &= p_3 I_u^*,\\
S_m^* &= \frac{(d_0+\alpha+\mu_i)p_3-\nu}{\beta_u\delta_s(1+\delta_i p_3)},\\
H^* &= \frac{\alpha(1+p_3)I_u^*}{\gamma+d_1},\\
M_e^* &= p_1(1+p_3)I_u^*.
\end{aligned}
\tag{4}
$$

(2): If $\delta_s = 0$, the endemic equilibrium can be written as

$$
\begin{aligned}
I_u^* &= \frac{\mu_s}{\beta_m p_1(1+p_3)}\left[\frac{S\beta_u(1+\delta_i p_3)}{d_0+\alpha+\nu-\mu_i p_3}-1\right],\\
I_m^* &= p_3 I_u^*,\\
S_m^* &= S-\frac{d_0+\alpha+\nu-\mu_i p_3}{\beta_u(1+\delta_i p_3)},\\
H^* &= \frac{\alpha(1+p_3)I_u^*}{d_1+\gamma},\\
M_e^* &= p_1(1+p_3)I_u^*.
\end{aligned}
\tag{5}
$$

Combining with the differential equations of the media impact model, it is easy to derive that if $R_0 > 1$, a unique equilibrium $E_1(S_m^*, I_m^*, I_u^*, H^*, M_e^*)$ exists in the biological region $\mathcal{D}$, meaning that if $R_0 > 1$, the disease breaks out and may remain endemic.

3.2. **Periodic oscillatory behaviors.** Two reasons result in oscillatory behaviors: Firstly, media report reduces the susceptibility and infectivity at rates δ_s and δ_i, respectively, which also changes the basic reproduction number. When the basic reproduction number $R_0 > 1$, oscillatory phenomena can occur. Secondly, it has been found that the psychological influence of the reported numbers of infected and hospitalized individuals result in the occurrence of Hopf bifurcation [5].

In the following, we will take the disease transmission coefficient β_u and the reduced infectivity δ_i as an example. Let Π_1 be the curve defined by $R_0 = 1$ in the (β_u, δ_i)-plane, where

$$
\Pi_1 : \delta_i^{(1)} = \delta_i(\beta_u) = \frac{(d_0+\alpha+\mu_i)(d_0+\alpha-\beta_u S)+\nu(d_0+\alpha)}{\beta_u S v}.
$$

In what follows, we consider the case $0 < \delta_i < \delta_i^{(1)}$ in the (β_u, δ_i)-plane.

We first note that the Jacobian matrix of Eq. (2) at the endemic equilibrium E_1 can be written as

$$J(E_1) = \begin{bmatrix} J_1 & \beta_u\delta_s S_m^* + \nu & \beta_u\delta_s\left(I_u^* + \delta_i I_m^*\right) & 0 & 0 \\ J_4 & J_2 & -\beta_u(I_u^* + \delta_i I_m^*) & 0 & 0 \\ -\beta_u\delta_i\delta_s S_m^* & -\beta_u\delta_s S_m^* & J_3 & 0 & J_5 \\ \alpha & \alpha & 0 & -(d_1+\gamma) & 0 \\ \rho_i & \rho_i & 0 & \rho_h & -\rho \end{bmatrix},$$

where

$$\begin{aligned} J_1 &= \beta_u\delta_i\delta_s S_m^* - (d_0 + \alpha + \mu_i), \\ J_2 &= \beta_u(S - S_m^*) - (d_0 + \alpha + \nu), \\ J_3 &= -\beta_u\delta_s\left(I_u^* + \delta_i I_m^*\right) - \mu_s - \beta_m M_e^*, \\ J_4 &= -\beta_u\delta_s\left(I_u^* + \delta_i I_m^*\right) - \mu_s - \beta_m M_e^*, \\ J_5 &= \delta_i\beta_u(S - S_m^*) + \mu_i. \end{aligned}$$

Let I represent the identity matrix, we can obtain

$$\begin{aligned} |\lambda I - J| &= \begin{vmatrix} \lambda - J_1 & -\beta_u\delta_s S_m^* - \nu & -\beta_u\delta_s\left(I_u^* + \delta_i I_m^*\right) & 0 & 0 \\ -J_4 & \lambda - J_2 & \beta_u(I_u^* + \delta_i I_m^*) & 0 & 0 \\ \beta_u\delta_i\delta_s S_m^* & \beta_u\delta_s S_m^* & \lambda - J_3 & 0 & -J_5 \\ -\alpha & -\alpha & 0 & \lambda + d_1 + \gamma & 0 \\ -\rho_i & -\rho_i & 0 & -\rho_h & \lambda + \rho \end{vmatrix} \\ &= \lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5. \end{aligned} \tag{6}$$

Substituting the endemic equilibrium $E_1(S_m^*, I_m^*, I_u^*, H^*, M_e^*)$ into the characteristic equation, we can calculate the coefficients of the characteristic equation. Actually, we find that the coefficients are determined by those parameters listed in Table 1 by using a mathematical software called Maple. For convenience, we do not present the explicit formula of these coefficients as the formulas are very complex.

Let λ_i $(i = 1, 2, ..., 5)$ be the eigenvalues of the matrix J. If one of the eigenvalues has a positive real part, the equilibrium is unstable. If all the eigenvalues have negative real parts, the equilibrium is stable. To investigate periodic oscillations of the media impact model, we consider the case of the characteristic equation with a pair of purely imaginary roots, which implies that the parameters satisfy the following discriminant

$$\Delta(\beta_u, \delta_i) = a_4(a_1a_2 - a_3)^2 - a_2(a_1a_4 - a_5)(a_1a_2 - a_3) + (a_1a_4 - a_5)^2 = 0. \tag{7}$$

Let Π_2 denote the curve satisfying $\Delta(\beta_u, \delta_i) = 0$ in the (β_u, δ_i)-plane

$$\Pi_2 : \beta_u = \beta_u(\delta_i).$$

It is easy to conclude that when the point (β_u, δ_i) is below the curve Π_1, there exists only one locally asymptotically stable disease free equilibrium E_0. According to Routh-Hurwitz criteria, when (β_u, δ_i) is between the curve Π_1 and Π_2, the endemic equilibrium E_1 is locally asymptotically stable. Further, when Π_2 is crossed, a Hopf bifurcation can occur.

Now, we will derive the transversality condition. Taking the parameter of δ_i as an example, we finally obtain a theorem which can determine whether the periodic oscillations exist or not.

Theorem 3.2. *The periodic oscillations can occur when δ_i satisfies*

$$(5\omega_0^4 - 3a_2\omega_0^2 + a_4)(\frac{da_1}{d\delta_i}\omega_0^4 - \frac{da_3}{d\delta_i}\omega_0^2 + \frac{da_5}{d\delta_i}) - (2a_3\omega_0 - 4a_1\omega_0^3)(\frac{da_4}{d\delta_i}\omega_0 - \frac{da_2}{d\delta_i}\omega_0^3) < 0. \tag{8}$$

Proof. Assume there exists a parameter $\delta_i^{(2)}$, such that $\lambda = \pm i\omega_0$ ($\omega_0 \in \mathcal{R}$) is a pair of purely imaginary roots. Differentiating Eq. (6) with respect to δ_i, we are led to

$$(5\lambda^4 + 4a_1\lambda^3 + 3a_2\lambda^2 + 2a_3\lambda + a_4)\frac{d\lambda}{d\delta_i} + \frac{da_1}{d\delta_i}\lambda^4 + \frac{da_2}{d\delta_i}\lambda^3 + \frac{da_3}{d\delta_i}\lambda^2 + \frac{da_4}{d\delta_i}\lambda + \frac{da_5}{d\delta_i} = 0.$$

Then we can have

$$(\frac{d\lambda}{d\delta_i})^{-1} = -\frac{5\lambda^4 + 4a_1\lambda^3 + 3a_2\lambda^2 + 2a_3\lambda + a_4}{\frac{da_1}{d\delta_i}\lambda^4 + \frac{da_2}{d\delta_i}\lambda^3 + \frac{da_3}{d\delta_i}\lambda^2 + \frac{da_4}{d\delta_i}\lambda + \frac{da_5}{d\delta_i}},$$

Performing some operations we can derive that

$$\begin{aligned}
&\frac{d(Re\lambda)}{d\delta_i}|_{\lambda=i\omega_0,\Delta=0} \\
&= Re(\frac{d\lambda}{d\delta_i})^{-1}|_{\lambda=i\omega_0,\Delta=0} \\
&= Re\left[-\frac{5\lambda^4 + 4a_1\lambda^3 + 3a_2\lambda^2 + 2a_3\lambda + a_4}{\frac{da_1}{d\delta_i}\lambda^4 + \frac{da_2}{d\delta_i}\lambda^3 + \frac{da_3}{d\delta_i}\lambda^2 + \frac{da_4}{d\delta_i}\lambda + \frac{da_5}{d\delta_i}}\right]|_{\lambda=i\omega_0,\Delta=0} \\
&= -\left[\Psi_2(\frac{da_1}{d\delta_i}\omega_0^4 - \frac{da_3}{d\delta_i}\omega_0^2 + \frac{da_5}{d\delta_i}) - \Psi_3(\frac{da_4}{d\delta_i}\omega_0 - \frac{da_2}{d\delta_i}\omega_0^3)\right].
\end{aligned}$$

where $\Psi_2 = 5\omega_0^4 - 3a_2\omega_0^2 + a_4, \Psi_3 = 2a_3\omega_0 - 4a_1\omega_0^3$.

Incorporating the above result with the definition of periodic oscillation

$$\frac{d(Re\lambda)}{d\delta_i}|_{\delta_i=\delta_i^{(2)},\omega=\omega_0} > 0, \tag{9}$$

we then finally prove the theorem. □

According to the Vieta's formula we can prove that the characteristic equation always has one eigenvalue whose real part is negative. Based on the above analysis, we can conclude that if (β_u, δ_i) is below the curve Π_1, then the system only has one disease free equilibrium. If (β_u, δ_i) falls between Π_1 and Π_2, then the endemic equilibrium is locally asymptotically stable. As (β_u, δ_i) increases through Π_2, we can obtain a critical value $\delta_i^{(2)}$ such that the characteristic equation has a pair of purely imaginary roots satisfying the condition equation (7) and inequality (9). That is to say, for (β_u, δ_i) that above Π_2 and in the first quadrant region, the periodic oscillations can occur. Similar analysis can be conducted for other parameters. To show the case of periodic oscillations, we carry out simulations in the next section.

4. **A case study.** The interaction of media impact and disease spreading is very complex. When the disease is serious, government would take measures to prevent the disease from further dissemination. In this case, people's behavior, the susceptibility and infectivity of the disease maybe influenced greatly. Furthermore, at an early stage, mass media is greatly influenced by the reported number of infected individuals, but as time goes on, media reports are largely affected by the number of hospitalized individuals. To explore the interaction of media reports and disease dynamics, we carry out a case study of SARS outbreak in Great Toronto Area (GTA)

TABLE 2. Parameters for the simulation of SARS in GTA

Symbol	d_1	d_0	α	γ	δ_s	δ_i	ν	ρ_h	ρ_i	ρ	μ_s	μ_i
Value	0.01	0.00001	0.33	0.05	0.50	0.50	0.26897	0.08	0.01	0.06	0.01	0.001

The value of parameters $d_1, d_0, \alpha, \gamma, \rho_h$ come from [21], value of parameters δ_s, δ_i, ρ come from [23], others are estimated according to experience.

in this section. Definitely, the effect of uncertainties in the parameter estimates does exist because uncertainty is the only certainty there is. But a detailed functional description for such uncertainty that follows certain distribution [18, 13, 14] is not available and would be very hard to reach as the model is already very complex. Thus, uncertainty is not included in this manuscript. Related parameter values are listed in Table 2.

We know that SARS attacked GTA on February 23, 2003. Up to May 14, 2003 on which World Health Organization removed GTA from the list of SARS emergency, the disease made 257 people ill and stay in hospitals. Unfortunately, the second outbreak swept GTA due to the infection between doctors and patients, which led to the tighten control within the hospitals and health care facilities.

According to 1996 census, the susceptible individuals of GTA was 5,446,104 in 2003. We take the value of the hospitalization rate from infected compartment to hospitalized compartment as $\alpha = 0.33$. The disease induced death rate of infected individuals and hospitalized individuals are $d_0 = 0.00001$ and $d_1 = 0.01$ in GTA, respectively. The hospital stay of patients is about 20 days, so we set the value of the recovery rate of hospitalized individuals as $\gamma = 0.05$ [21]. Due to the presence of SARS, avian influenza, and H1N1, many scholars try to explore the impact of media reports on disease outbreak. Based on previous studies [29, 21], we take the transmission rate of awareness as $\beta_m = 9.1809 \times 10^{-8}$ and take the infection rate as $\beta_u = 8.63 \times 10^{-8}$ to investigate the effect of media reports on SARS epidemic. We fix the rates of infected and hospitalized individuals becoming aware at $\rho_i = 0.01$ and $\rho_h = 0.08$. The decreasing rate of awareness is set to $\rho = 0.06$. The values of reduced susceptibility and infectivity are $\delta_s = 0.50$ and $\delta_i = 0.50$, respectively [23].

The detailed parameter values are listed in Table 2. Based on the above analysis, we can get the basic reproduction number $R_0 = 1.105$, and the endemic equilibrium

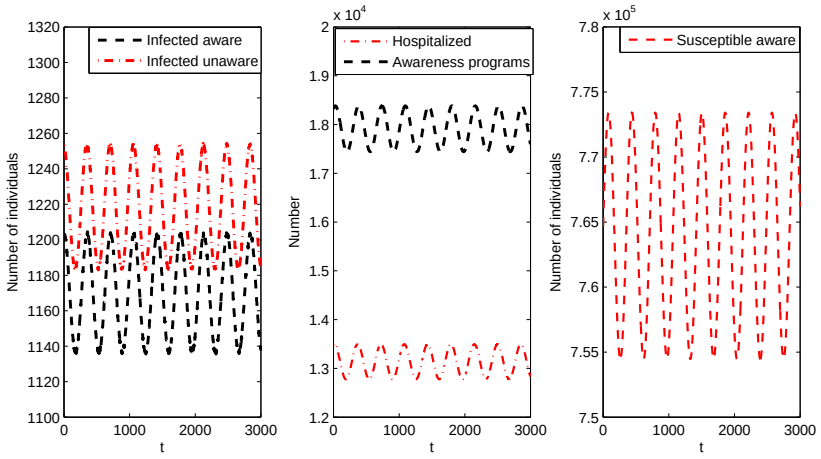
$$E_1 = (763920, 1169.6, 1218.4, 13134, 17909).$$

In this scenario, we obtain five eigenvalues as follows

$$\lambda_{1,2} = \pm 0.0261i, \lambda_3 = -0.0746, \lambda_4 = -0.3374, \lambda_5 = -0.2985.$$

Using the Runge-Kutta method, Figure 2(a) shows the variation of the number of infected-aware and infected-unaware individuals with respect to time step. It is apparent that as time goes on, the numbers of infected-aware and infected unaware individuals experience several obvious fluctuations and behave in a timely periodically oscillatory fashion. From Figure 2(b), we observe that the curves denoting the numbers of hospitalized individuals and awareness programs also show sustained periodic oscillatory behavior. However, the peak of hospitalized individuals is much bigger than infected individuals as most sick people stay in hospitals seeking for medical treatment. Figure 2(c) shows the developing trend of the number of susceptible aware individuals versus time. The number of susceptible aware individuals is greatly influenced by media reports as when mass media struggles to alert the

public to take precautions to protect themselves, the number of susceptible aware individuals reaches a peak point.



(a) (b) (c)

FIGURE 2. Variation of different numbers versus time.

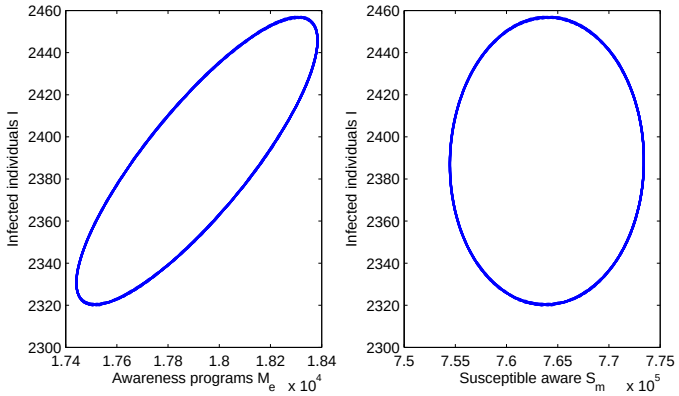


FIGURE 3. Phase portrait of the system.

To show the stability of (M_e^*, I^*) in $(M_e - I)$ plane and (S_m^*, I^*) in $(S_m - I)$ plane we present Figure 3, which illustrates the relationship between the number of infected individuals versus awareness programs and susceptible aware individuals. From the two limit circles, we can see that with the intervention of media reports, both trajectories in $(M_e - I)$ and $(S_m - I)$ planes behave periodic oscillations.

Furthermore, the analysis of the impact of related parameters on the SARS progression is fairly important. Figure 4 shows the variation of the number of infected individuals versus time under different transmission rates of awareness, infection rates from susceptible unaware to infected unaware, susceptibility and infectivity coefficients. As can be seen from Figure 4 (a), the number of infected individuals decreases sharply as transmission rate of awareness increases. Stronger awareness lessens the number of infected individuals although the amplitude of

oscillations is amplified and the infected individuals still experience several sustained periodic oscillations. Figure 4 (b) reveals that infection rate β_u greatly affects the infected numbers. The periodic oscillations occur for $\beta_u = \frac{0.47}{5446104}$. As β_u decreases to $\beta_u = \frac{0.45}{5446104}$, the endemic equilibrium is locally asymptotically stable. In other words, higher infection rate β_u is detrimental to the control of infectious disease spreading as the higher the infection rate becomes, the more frequent and powerful oscillations occur, which implies the harder to eradicate the infectious disease. From Figures 4 (c) and 4 (d), it can be seen that reducing infectivity is favorable to suppress further deterioration of the situation. But only reducing the susceptibility without limiting the infectivity is still not an effective way to control the disease propagation.

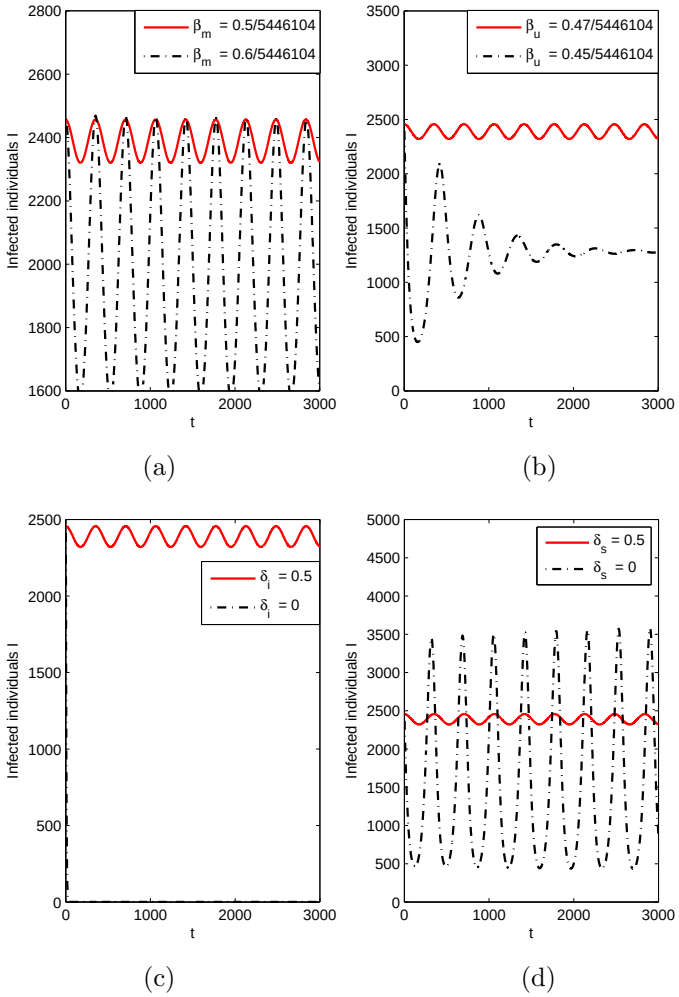
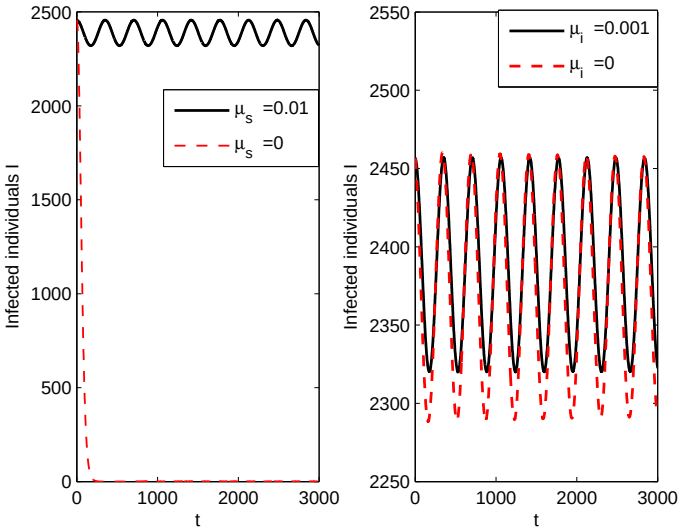


FIGURE 4. Variation of the number of infected individuals versus time under different impact factors.

Figure 5 (a) reveals that the awareness of susceptible individuals highly affects the progression of infectious disease. Reminding susceptible individuals to stay alert to



(a) (b)

FIGURE 5. Variation of the number of infected individuals versus time under different awareness loss rates.

the potential infection danger and avoid going to public places which can reduce the contact rate with infected individuals can prevent themselves from further infection. Figure 5 (b) shows that only raising the awareness of infected individuals without alerting susceptible individuals is not an effective way to alleviate the infection although all infected individuals are familiar with the potential infection possibility. This may suggest related authorities that keeping releasing true information to alert the susceptible individuals is necessary.

Last point that should be mentioned is that the rates of infected and hospitalized becoming aware ρ_i, ρ_h and the decreasing rate of awareness ρ significantly influence the number of awareness programs and people's behavior, which in turn affect the disease progression. Applying official media with higher credibility and audience rating is beneficial to alleviate further infection. Not the more the number of awareness programs, the better the disease can be controlled. Too many media reports which go against the stability condition may cause a storm of serious panic and irrational behaviors.

5. **Conclusions and remarks.** In this paper, a media impact model has been proposed to study the interaction of media impact and disease dynamics. Key implications are listed as follows.

1) We have formulated a media impact model incorporating a susceptible-infected-hospitalized-recovered compartment model with awareness programs driven by media reports. Both susceptible and infected classes are subdivided into 2 types, including aware and unaware subclasses, according to whether they keep alertness to infectious disease. We have assumed that the growth rate of awareness programs is proportional to the number of both infected and hospitalized individuals. Moreover, disease spread leads to the increase of awareness, which in turn lower susceptibility

and infectivity as susceptibles reduce contacts with infected ones and infected ones take positive cure measures to fight off the disease.

2) To analyze the periodic oscillation behavior of the model, we carry out stability analysis of disease free equilibrium and endemic equilibrium by using Routh-Hurriwitz criteria. Moreover, the condition determining the occurrence of sustained periodic oscillations is investigated to show how media reports impact the disease dynamics.

3) We carry out a case study of SARS outbreaks in GTA to illustrate the interaction of media reports and disease dynamics. Results show that the system is highly complex as lots of factors affect the SARS progression, such as the number of awareness programs,transmission rate of awareness, infection rate of unaware individuals, reduced susceptibility and infectivity, awareness loss rate, rates of infected and hospitalized individuals becoming aware, and decreasing rate of awareness loss due to ineffectiveness. We should deal with the epidemic carefully, otherwise, it can result in potential disastrous consequences.

Our results could offer some useful suggestions for authorities and share implications for crisis management. Authorities at all levels need to justify whether media reports positively influence the situation or negatively throw the public into a panic. Current work is in no way to illustrate the interaction of media impact and disease dynamics exhaustively and can be expanded from many aspects, for example, incorporating complex network with this model, investigating a nonlinear contact incidence rate which better describes the actual disease dynamics and considering a partially effective media report which is much more complicated but worth trying.

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