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AN SIS INFECTION MODEL INCORPORATING MEDIA COVERAGE

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ABSTRACT. We develop a model to explore the impact of media coverage on the control of spreading of emerging or reemerging infectious diseases in a given population. The model can have up to two equilibria: a disease free equilibrium and a unique endemic equilibrium. Stability analysis of the model shows that the disease free equilibrium is globally asymptotically stable if the reproduction number $\mathbf{R}_0$ is less than unity, and the endemic equilibrium is globally asymptotically stable when it exists. Though the media coverage itself is not a determined fact to eradicate the infection of the diseases, the analysis of model indicates that, to a certain extent, the more media coverage in a given population, the less number of individuals will be infected. Therefore, media coverage is critical for educating people in understanding the possibility of being infected by the disease.

1. Introduction. When an infectious disease appears and spreads in a region, the departments for disease control and prevention will do everything possible to prevent the disease from spreading. One of the immediate measures to take is to educate people about preventative knowledge of the disease through media coverage. It is common sense that the more preventative knowledge the population has, the better the possibility of preventing the spread of the disease. According to a recent statistical analysis on acquired immunodeficiency syndrome (AIDS), see [**11**], media and education play a tremendous role in mounting AIDS awareness among the residents. Another study showed that public awareness can play a dominating role in preventing the AIDS epidemic [**7**]. According to [**11**], the odds of awareness among higher educated women and men were 4.67 and 77.73 times that of

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uneducated women and men, respectively. In addition, among both women and men, those who regularly watch TV were 8.6 times more likely to be aware about AIDS compared to those who never watch TV [**11**]. These results inspired us to study how the disease spreading dynamics changes in response to media coverage and education.

Media coverage and education may have reduced the contact rate of human beings as we observed during the spread of severe acute respiratory syndrome (SARS) during 2002 and 2004. SARS [**5, 9, 12, 15–18**] as a new emerging infectious disease first appeared in Guangdong province, China, in November, 2002. Then in the following year it spread rapidly throughout Asia and certain other regions of the world [**13, 15**]. For SARS in Beijing and Hong Kong, China, and Toronto, in Canada, the spread and outbreak showed a typical process for people to see how media coverage and alerting the public plays a role in the whole course of the spread. The first SARS cases in Beijing were reported in early March, 2003 [**15**]. Before media coverage and education, the residents of the city did not know what SARS was and how it was spreading among the population. Soon after the official media report and education, people knew that SARS spread from one infected individual to another by close contact and people started to reduce their contact with others by staying home or reducing social activities. All these measures, including the later quarantine strategy greatly reduced the contact transmission of the disease among the population.

There have been mathematical modeling studies to investigate the impact of media coverage and psychology on the spread and control of infectious diseases in a given population or region. In [**9**], the authors proposed a model with the compartments of exposed (E), infectious (I) and hospitalized (H) individuals to explore possible mechanisms for multiple outbreaks of emerging infectious diseases due to the psychological impact of the reported numbers of infectious and hospitalized individuals. The model was simplified by assuming that the total population size remains constant during the course of the spread of the disease. In [**2**], the authors extend the classical SEI model by considering a new incidence functional which reflects the impact of media coverage on the spread and control of the disease.

In this paper we propose a model with general nonlinear incidence function to reflect some intrinsic characters of media coverage and

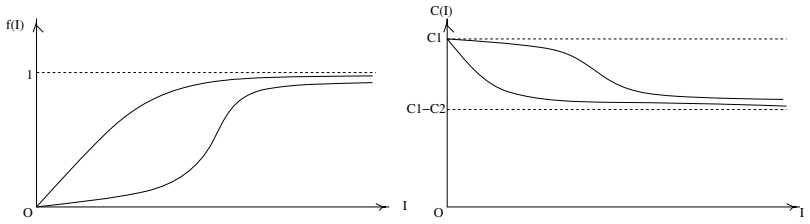

FIGURE 1. Limited reduction of the contact rate due to infected individuals.

education. The paper is organized as follows. In Section 2, we propose an SIS infection model to incorporate the media and education impact on the spread of the infectious disease in a given population. In Section 3, we calculate the basic reproduction number $\mathbf{R}_0$ and discuss the existence and stability of the disease free equilibrium as well as the endemic equilibrium when it exists. We end the paper with simulations and a brief discussion.

2. Model. The simplest structure for a deterministic compartmental model of an infectious disease divides the population into two compartments, susceptible individuals (S) and infected individuals (I). With some infections, for example the common cold, the infected individuals do not confer any long-lasting immunity; such infections do not have a recovered state and individuals become susceptible again after recovering from the infection. It follows from the book of Brauer and Castillo-Chavez [**1**] that a classical SIS model with standard incidence takes the following form

$$\begin{cases} \dot{S} = A - dS - \beta SI/(S+I) + \gamma I, \\ \dot{I} = \beta SI/(S+I) - (d+\nu+\gamma)I, \end{cases} \tag{2.1}$$

where all the parameters are positive constants and A is the recruitment rate of the population, d is the natural death rate of the population, γ is the recovery rate of infective individuals, ν is the disease induced death rate, β is the contact transmission coefficient with $\beta = pc$, in which p is the transmission probability per contact per unit of time and c is the contact rate.

The model in the form of (2.1), or similar models, has been used to study the transmission dynamics of infectious diseases since the

pioneering work of Kermack and Mckendrick [**6**]. In general, the contact transmission coefficient β depends at least on the number of susceptible and infected individuals, as well as on the pattern of contact among the susceptible and infected individuals. In many of the compartmental models the contact transmission coefficient β was treated as a constant for the sake of simplicity. Indeed, the contact rate c is a constant in this model. But actual media coverage and a reported number of infected individuals lead to a lower contact rate as we observed in the course of the SARS outbreak in 2003 [**9**]. This fact suggests a more reasonable contact rate to reflect such effects of media coverage on the spread of the disease and control. Hence, we modify the contact rate c as a function of the number of infected individuals:

$$c(I) = c_1 - c_2 f(I)$$

where c_1 and c_2 are positive constants. Here c_1 is the usual contact rate without considering the infective individuals and c_2 is the maximum reduced contact rate due to the presence of the infected individuals. Obviously, we must have $f(0) = 0$. When infective individuals appear in a region, people reduce their contact with others to avoid being infected when they are aware of the potential danger of being infected, and the more infective individuals being reported, the less contact the susceptible will make with others. Therefore, it is reasonable to assume that $f'(I) \geq 0$. But we know that everyone cannot avoid contact with others in every case so we assume that $c_2 < c_1$. The limited power of the infection due to contact is reflected by the saturating function $\lim_{I\to\infty} f(I) = 1$. In summary, the functional $f(I)$ satisfies

$$f(0) = 0, f'(I) \geq 0, \quad \lim_{I\to\infty} f(I) = 1. \tag{2.2}$$

Let $\mu_1 = pc_1$ and $\mu_2 = pc_2$. Then an SIS infection model with media coverage takes the following form:

$$\begin{cases} \dot{S} = A - dS - (\mu_1 - \mu_2 f(I))SI/(S+I) + \gamma I, \\ \dot{I} = (\mu_1 - \mu_2 f(I))SI/(S+I) - (d+\nu+\gamma)I. \end{cases} \tag{2.3}$$

Note that when $c_2 = 0$, or $\mu_2 = 0$, model (2.3) becomes the classical model (2.1) studied in [**1, 3**]. In [**8**], Liu and Cui have suggested a special expression for the function $f(I)$ to study the dynamics of an SIR system with media coverage.

Let

$$\Omega = \left\{ (S, I) \mid S, I \geq 0, S + I \leq \frac{A}{d} \right\}.$$

We first prove that Ω is a positive invariant for system (2.3). In fact, for $(S, I) \in \Omega$ we have $dS/dt|_{S=0} > 0$, $dI/dt|_{I=0} = 0$ and $(d(S + I))/(dt) \leq A - d(S + I)$. Hence, $(d(S + I))/(dt)|_{S+I=(A/d),(S,I)\in\Omega} \leq 0$, and Ω is a positive invariant. Hence, we shall focus only in the region Ω.

3. Equilibria and the basic reproduction number. Though we do not use an explicit expression for the function $f(I)$, model (2.3) can have up to two biologically meaningful equilibria. One can verify that the model (2.3) always has one disease-free equilibrium at $E_0 = ((A/d), 0)$.

It is well known that the basic reproduction number, a threshold representing how many secondary infections result from the introduction of one infected individual into a population of susceptibles [**4**], is denoted by $\mathbf{R}_0$. This quantity is not only important in describing the infectious power of the disease, but also can supply information for controlling the spread of the disease. A general formula was developed in [**14**] to calculate the reproduction number associated with the disease-free equilibrium. Using the formula, one can calculate the eigenvalues of the second generation matrix, and the maximum eigenvalue gives the basic reproduction number

$$\mathbf{R}_0 = \frac{\mu_1}{d + \nu + \gamma}. \tag{3.1}$$

An easy calculation yields the characteristic equation of (2.3) at E_0:

$$(\lambda + d)(\lambda + (1 - \mathbf{R}_0)(d + \nu + \gamma)) = 0. \tag{3.2}$$

It follows from the Routh-Hurwitz criterion that the two eigenvalues have negative real parts if and only if $\mathbf{R}_0 < 1$. Hence, we have

Theorem 3.1. *For the model* (2.3)*, the disease-free equilibrium* E_0 *is locally asymptotically stable if* $\mathbf{R}_0 < 1$ *and unstable if* $\mathbf{R}_0 > 1$.

We have the following lemma regarding the existence of positive endemic equilibrium.

Lemma 3.2. *For any choice of function of $f(I)$ which satisfies (2.2), the model (2.3) has no endemic equilibria if $\mathbf{R}_0 \leq 1$, and there exists a unique endemic equilibrium if $\mathbf{R}_0 > 1$.*

Proof. An endemic equilibrium (S, I) satisfies $S > 0$, $I > 0$ and

$$\begin{aligned} A - dS - (\mu_1 - \mu_2 f(I))\frac{SI}{S+I} + \gamma I = 0, \\ (\mu_1 - \mu_2 f(I))\frac{S}{S+I} - (d + \nu + \gamma) = 0. \end{aligned} \tag{3.3}$$

Eliminating the nonlinear terms from the two equations of (3.3), we have

$$S = \frac{A - (d+\nu)I}{d}. \tag{3.4}$$

Substituting (3.4) into the second equation of (3.3), we get

$$\phi(I) := \frac{dI}{A - \nu I} - \left[1 - \frac{d + \nu + \gamma}{\mu_1 - \mu_2 f(I)}\right] = 0. \tag{3.5}$$

Hence, if an endemic equilibrium exists, its I coordinate must be a root of $\phi(I) = 0$ in the interval $I \in (0, (A/d + \nu))$.

Note that

$$\phi'(I) = \frac{Ad}{(A - \nu I)^2} + \frac{\mu_2(d + \nu + \gamma) f'(I)}{(\mu_1 - \mu_2 f(I))^2}, \tag{3.6}$$

and $f'(I) \geq 0$. Hence, $\phi(I)$ is monotonically increasing for $I > 0$.

On the other hand,

$$\phi(0) = -1 + \frac{1}{\mathbf{R}_0},$$

and

$$\phi\left(\frac{A}{d+\nu}\right) = \frac{d + \nu + \gamma}{\mu_1 - \mu_2 f((A/d + \nu))} > 0.$$

So we have $\phi(0) < 0$ provided $\mathbf{R}_0 > 1$. Therefore, $\mathbf{R}_0 > 1$ and $f(I)$ have unique positive roots in the interval $(0, (A/d + \nu))$. Denote the unique positive root as I^* and the associated S value given by (3.4) by S^*. Therefore, we have proved that for any function $f(I)$ satisfying (2.2), the model (2.3) has a unique endemic equilibrium $E(S^*, I^*)$ when $\mathbf{R}_0 > 1$. Otherwise, there is no endemic equilibrium when $\mathbf{R}_0 \leq 1$. □

Now we consider the global stability of the equilibria. First we consider the disease-free equilibrium.

Theorem 3.3. *For the model* (2.3)*, the disease-free equilibrium* E_0 *is globally asymptotically stable whenever* $\mathbf{R}_0 < 1$.

Proof. Let $V = I$ as a Lyapunov function in Ω. Calculating the derivative along the solutions of system (2.3), we have

$$\begin{aligned} \dot{I} &= (\mu_1 - \mu_2 f(I))\frac{SI}{S+I} - (d+\nu+\gamma)I \\ &< (\mu_1 - \mu_2 f(0))I - (d+\nu+\gamma)I \\ &= (d+\nu+\gamma)I(R_0 - 1). \end{aligned} \tag{3.7}$$

Hence, $\dot{I} < 0$ when $\mathbf{R}_0 < 1$ and the disease-free equilibrium E_0 is globally asymptotically stable. □

For the endemic equilibrium, when it exists ($\mathbf{R}_0 > 1$), it is not only locally stable, but also globally asymptotically stable.

Theorem 3.4. *For the model* (2.3)*, the endemic equilibrium* $E(S^*, I^*)$ *is globally asymptotically stable when* $\mathbf{R}_0 > 1$.

Proof. Evaluating the Jacobian of (2.3) at the endemic equilibrium $E(S^*, I^*)$ gives

$$J = \begin{pmatrix} -d - (\mu_1 - \mu_2 f(I^*))(\frac{I^*}{S^*+I^*})^2 & \\ \qquad\qquad \mu_2 f'(I^*)\frac{S^*I^*}{S^*+I^*} - (\mu_1 - \mu_2 f(I^*))(\frac{S^*}{S^*+I^*})^2 + \gamma & \\ (\mu_1 - \mu_2 f(I^*))(\frac{I^*}{S^*+I^*})^2 & \\ \qquad -\mu_2 f'(I^*)\frac{S^*I^*}{S^*+I^*} + (\mu_1 - \mu_2 f(I^*))(\frac{S^*}{S^*+I^*})^2 - (d+\nu+\gamma) & \end{pmatrix}. \tag{3.8}$$

Because

$$(\mu_1 - \mu_2 f(I^*))\frac{S^*}{S^* + I^*} = d + \nu + \gamma,$$

then

$$\begin{aligned}(\mu_1 - \mu_2 f(I^*))\left(\frac{S^*}{S^* + I^*}\right)^2 &= (d + \nu + \gamma)\frac{S^*}{S^* + I^*} \\ &= (d + \nu + \gamma) - (d + \nu + \gamma)\frac{I^*}{S^* + I^*}.\end{aligned}$$

Substituting into (3.8), we have

$$J = \begin{pmatrix} -d - B & C - (d + \nu) \\ B & -C \end{pmatrix}, \tag{3.9}$$

where

$$\begin{aligned} B &= (\mu_1 - \mu_2 f(I^*))\left(\frac{I^*}{S^* + I^*}\right)^2 > 0, \\ C &= (\mu_2 f^{'}(I^*)S^* + d + \nu + \gamma)\frac{I^*}{S^* + I^*} > 0. \end{aligned} \tag{3.10}$$

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

$$\lambda^2 + (d + B + C)\lambda + dC + dB + \nu B = 0. \tag{3.11}$$

It follows from the Routh-Hurwitz criterion that all the eigenvalues have negative real parts. Hence, E is locally asymptotically stable when it exists. Next we prove the global stability of E.

Choose a Dulac function

$$D = \frac{S + I}{SI}.$$

Denote

$$\begin{aligned} F &= A - dS - (\mu_1 - \mu_2 f(I))\frac{SI}{S + I} + \gamma I, \\ G &= (\mu_1 - \mu_2 f(I))\frac{SI}{S + I} - (d + \nu + \gamma)I. \end{aligned} \tag{3.12}$$

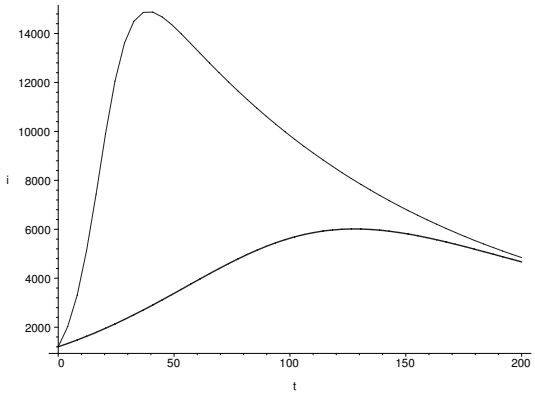

FIGURE 2. The thicker infection curve represents the case when $\mu_2 = 0.12$, the thin infection curve represents the case when $\mu_2 = 0$.

Note

$$\frac{\partial(DF)}{\partial S} + \frac{\partial(DG)}{\partial I} = -d\frac{S+I}{SI} - \mu_2 f'(I) - \frac{d+\nu+\gamma}{S} - \frac{A - dS + \gamma I}{S^2} < 0$$

in the interior of the positive invariant set Ω. The Dulac criterion holds ([**10**]) and there are no close orbits in Ω. This proves that the endemic equilibrium E is globally asymptotically stable. □

4. Simulations, discussion and conclusion. At the endemic equilibrium point $E(S^*, I^*)$, we know that the infected population I^* is the unique positive solution of (3.5) and it is a function of μ_2. Calculating the derivative of I^* about μ_2 from (3.5), we get $(dI^*/d\mu_2) > 0$. Hence, the infected size can be reduced because of media alerts and education.

Choose $f(I) = I/(b+I)$, $b > 0$; for example, we fix some of the parameter values: $A = 25$, $b = 10$, $d = 0.007$, $\mu_1 = 0.15$, $\gamma = 0.002$ and $\nu = 0.0015$. We have $\mathbf{R}_0 = 14.286$. As shown in Figure 2, the transmission of the disease experiences a single peak; the thin curve represents the case when $\mu_2 = 0$, the application of media was not considered. The thicker curve represents the case when $\mu_2 = 0.12$. Figure 2 shows that effective media coverage postpones the arrival of the infection peak, and a fewer number of individuals become infected in the course of transmission.

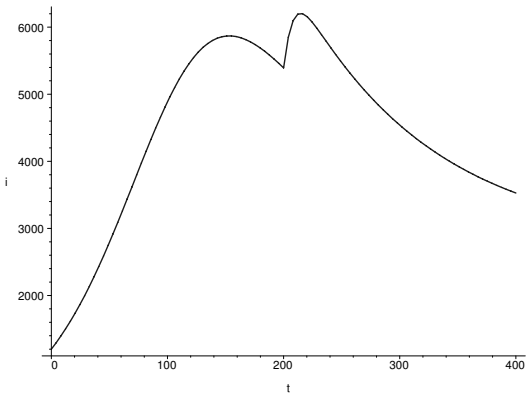

FIGURE 3. The infection curve in [0,200] represents the case when $\mu_2 = 0.12$, and the other infection curve in [200,400] represents the case when $\mu_2 = 0$.

In the absence of media coverage and education in the sense that $\mu_2 = 0$, model (2.3) is just the classical SIS model (2.1). One can find some known results on this model, for example, in [**1, 3**]. System (2.1) admits two steady states $((A/d), 0)$ and $(\overline{S}^*, \bar{I}^*)$, in which

$$(4.1)\quad \overline{S}^* = \frac{A(d+\nu+\gamma)}{d\beta + \nu[\beta - (d+\nu+\gamma)]}, \quad \bar{I}^* = \frac{A[\beta - (d+\nu+\gamma)]}{d\beta + \nu[\beta - (d+\nu+\gamma)]}.$$

The disease-free steady state $((A/d), 0)$ exists for all parameter values and is globally stable provided $\mathbf{R}_0 < 1$. The endemic steady state $(\overline{S}^*, \bar{I}^*)$ exists provided $\mathbf{R}_0 > 1$ and it is globally stable if it exists. Comparing these results between $\mu_2 = 0$ and $\mu_2 > 0$, we find that media coverage does not induce any complicated dynamical behaviors under the assumptions of this paper.

The results of the present paper imply that media coverage is an important factor regarding the transmission of the infectious disease and it is therefore important for the control of the spreading of the disease. The total number of infected population can be reduced if appropriate media coverage is applied. But if we relax or stop the media alert before the disease disappears, the transmission of the disease may experience another infection peak as shown in Figure 3, which is exactly the case of the SARS outbreak in the greater Toronto area in 2003 [**5, 9, 16**].

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