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Media/psychological impact on multiple outbreaks of emerging infectious diseases

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We use a compartmental model to illustrate a possible mechanism for multiple outbreaks or even sustained periodic oscillations of emerging infectious diseases due to the psychological impact of the reported numbers of infectious and hospitalized individuals. This impact leads to the change of avoidance and contact patterns at both individual and community levels, and incorporating this impact using a simple nonlinear incidence function into the model shows qualitative differences of the transmission dynamics.

Keywords: SARS; Media/psychological impact; Hopf bifurcation; Oscillatory behaviors; Endemic equilibrium

1. Introduction

SARS, the first severe infectious disease to have emerged in the twenty-first century, exhibits some distinct features such as rapid spatial spread and self-control. These features, associated with the increasing trend of globalization and the development of information technology, are expected to be shared by other emerging infectious diseases. It is therefore important to refine classical mathematical models to reflect these features by adding the new dimensions of massive news coverage and fast information flow that generate a profound psychological impact on the public and have great influence not only on the individual behaviors but also on the formation and implementation of public intervention and control policies.

There are now extensive research activities about the psychological impact of SARS outbreak on the general public and about the media impact on SARS control and prevention (see, for example, [1–3,9,11]), and there are several models proposed to describe the SARS transmission dynamics [5,6,10,20], but more comprehensive studies will require interdisciplinary research across traditional boundaries of social, natural, and medical sciences, and mathematics. In this paper, we use a compartmental model to illustrate the possibility of multiple outbreaks caused by the change of individual avoidance and contact patterns as a response to the reported information of infectious and hospitalized cases, while multiple outbreaks can hardly occur if the avoidance and contact patterns are adjusted according to the reported number of exposed individuals instead.

As shown in figure 1, the transmission of SARS in the Great Toronto Area experienced multiple outbreaks. We hope our work can provide some theoretical analysis for the

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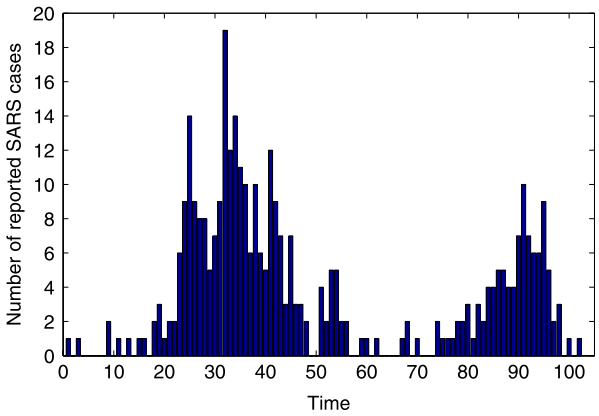

Figure 1. Number of reported SARS cases in Great Toronto area from February 23 to June 4, 2003. The data is taken from Ref. [7].

mechanisms of the phenomena of multiple outbreaks. In particular we hope to show that second or multiple outbreaks may be rooted in the change of individual behavior when the infectious process runs its own course. The media report and coverage, the information processing, the individual's alerted response to the information and media coverage, the official control and prevention measures and policies are so intertwined that they jointly make the undesirable multiple outbreaks feasible. We thus hope this work may be useful for consideration of what constitutes essential public information for disease control.

2. A model emphasizing the psychological impact

We start with a partition of the whole population into five distinct classes of susceptible, exposed, infectious, hospitalized and removed individuals in terms of certain specific infectious disease under consideration. We emphasize that this preliminary work will not consider the factors of mandatory quarantine and isolation, but concentrate only on the situation where effective contacts will be reduced when the infection level increases. We assume that the outbreak duration is sufficiently short and hence the total number of susceptible remains relatively unchanged and the demographic details (natural death and birth) can be ignored. It follows from Ref. [14], a standard formulation for the temporal evolution of the exposed (E), infectious (I) and hospitalized (H) sub-populations then takes the following format:

$$\begin{cases} \frac{\mathrm{d}E}{\mathrm{d}t} = \beta_0 IS - \alpha E, \\ \frac{\mathrm{d}I}{\mathrm{d}t} = \alpha E - dI - hI, \\ \frac{\mathrm{d}H}{\mathrm{d}t} = hI - d_h H - rH, \end{cases} \tag{2.1}$$

where $E = E(t)$ is the number of individuals who are exposed to the infected but not yet infectious, $I = I(t)$ is the number of infectious individuals, and $H = H(t)$ is the number of infectious individuals who are receiving medical treatment in hospital settings;

we also assume that the hospitalized individuals no longer impose risk to the susceptible individuals.

In model (2.1), the parameters involved, which are positive, are

β_0: we assume that the exposed population is increased following infection acquired via contact between a susceptible and an infectious individual with a transmission coefficient;
β_0. This parameter measures the effect of both the infectiousness of the disease and the contact transmission rates;
S: as mentioned above, we assume that the total number of susceptible individuals remains unchanged, and thus S will be regarded as a parameter;
α: the transmission rate per unit of time (day, in case of SARS) that exposed individuals become infectious;
d: the disease induced death rate of infectious individuals before entering the health care settings;
h: the rate at which infectious individuals enter the health care settings seeking for treatment;
d_h: the disease-induced death rate of hospitalized individuals;
r: the recovery rate of the hospitalized individuals.

It is not difficult to observe that the system has only one equilibrium, the origin that corresponds to the disease free state. Simple calculation yields that the basic reproduction number (see [14,18]) is $\mathcal{R}_0 = (\beta_0 S)/(d + h)$, and hence the origin is asymptotically stable and the disease will die out exponentially if $\mathcal{R}_0 < 1$, while the origin is unstable and the disease will grow exponentially if $\mathcal{R}_0 > 1$.

The aforementioned model is clearly a crude reflection of the complicated nonlinear phenomena of the transmission dynamics, and it does not incorporate the self-control property due to the change of avoidance patterns of individuals at different stages of the infectious process. It is reported that the massive news coverage had significant impact on the avoidance behaviors at both individual and society levels, and the reported numbers of exposed, infectious and hospitalized individuals have clearly profound psychological impact on the social conduct that seems to reduce the effective contact of susceptible with infectious individuals. A detailed functional description for such a psychological impact is not available and would be extremely difficult to achieve; here we simply assume that this impact is described by an exponential decreasing factor, resulting the transmission coefficient as $\beta_0 = \beta e^{-a_1 E - a_2 I - a_3 H}$. Here β is the basic transmission rate if the impact of the reported numbers of exposed, infectious and hospitalized were ignored, and a_1, a_2, a_3 are non-negative parameters to measure the effect of psychological impact of media reported numbers of exposed, infectious and hospitalized individuals. The modified model of (2.1) then becomes

$$\begin{cases} \frac{dE}{dt} = \beta e^{-a_1 E - a_2 I - a_3 H} IS - \alpha E, \\ \frac{dI}{dt} = \alpha E - dI - hI, \\ \frac{dH}{dt} = hI - d_h H - rH. \end{cases} \tag{2.2}$$

In general, the first available information is the reported number of hospitalized patients when the infectious disease is at the emerging stage, hence we will focus more on the impact of the number of reported hospitalized cases.

3. Nonlinear dynamics and oscillatory behaviors

We study system (2.2) in the following biological feasible region $\mathcal{D} = \{(E, I, H) \in \mathbf{R}^3_+\}$. It can be verified that $\mathcal{D}$ is positively invariant for (2.2). One can verify that for any fixed a_1, a_2 and a_3, model (2.2) always has a disease free equilibrium (DFE) at the origin. We denote this DFE by $E_0 = (0,0,0)$. One can verify that the reproduction number $\mathcal{R}_0 = \beta S/(d + h)$ is the same as the reproduction number in (2.1). The following Lemma gives the global stability of the DFE.

Lemma 3.1. If the reproduction number $\mathcal{R}_0 < 1$, the DFE E_0 is globally asymptotically stable in $\mathcal{D}$.

Proof. For $V = E + I$, we have

$$\dot{V} = (\beta S \mathrm{e}^{-a_1 E - a_2 I - a_3 H} - (d + h))I \leq (\beta S - (d + h))I.$$

This gives $\dot{V} \leq 0$ if $\mathcal{R}_0 < 1$. Furthermore, $\dot{V} = 0$ in $\mathcal{D}$ if and only if $I = 0$. Therefore, the largest compact invariant set in $\{(E, I, H) \in \mathcal{D}, \dot{V} = 0\}$ is the singleton E_0. The LaSalle's invariance principle [4] then implies that the DFE E_0 is globally asymptotically stable when $\mathcal{R}_0 < 1$. □

If $\mathcal{R}_0$ is increased through the critical value 1, a transcritical bifurcation occurs, the DFE $E_0 = (0,0,0)$ becomes unstable (hence the disease will be established in the community) and a positive equilibrium, the endemic equilibrium, appears in $\mathcal{D}$. By letting the right-hand sides of (2.2) equal zero, one can verify that if $\mathbb{R}_0 > 1$, this positive equilibrium $E^+ = (E^*, I^*, H^*)$ is given by

$$E^* = C(d_h + r)(d + h), \quad I^* = C(d_h + r)\alpha, \quad H^* = Ch\alpha, \tag{3.1}$$

where

$$C = \frac{\ln(\mathbb{R}_0)}{a_1(d + h)(r + d_h) + a_2\alpha(r + d_h) + a_3\alpha h}.$$

Note that if $a_i = 0$, $i = 1, 2, 3$ and $\mathcal{R}_0 = 1$, then all points $(((d + h)/\alpha), 1, (h/(d_h + r)))I$ with $I \geq 0$ are equilibria of model (2.2). From the expression of the endemic equilibrium E^+ in (3.1) we can see that when $\mathcal{R}_0 > 1$, $E^*, I^*, H^* \to \infty$ if $a_i \to 0^+$, $i = 1, 2, 3$. This means that if without media or psychological impact in the model, the number of exposed, infectious and hospitalized will grow exponentially which is consistent with the simple dynamics of the model (2.1).

It then becomes important to examine the behaviors of the system near the endemic equilibrium E^+. In what follows, we consider the case when the reproduction number $\mathcal{R}_0 > 1$ and study the stability of and possible bifurcations from the endemic equilibrium E^+. We will take contact transmission rate β and h as parameters. Note that $\mathbb{R}_0 > 1$ corresponds to $h > \beta S - d$. We denote the straight line defined by $\mathcal{R}_0 = 1$ by

$$\Gamma_0 : h = \beta S - d,$$

we restrict ourselves in the region defined by $0 < h < \beta S - d$ in the (h, β)-plane. We first note that Jacobian matrix of the model (2.2) at E^+ is

$$J(E^+) = \begin{pmatrix} -\beta a_1 D^* S I^* - \alpha & -\beta a_2 D^* S I^* + \beta D^* S & -\beta a_3 D^* S I^* \\ \alpha & -(d+h) & 0 \\ 0 & h & -(d_h + r) \end{pmatrix}, \tag{3.2}$$

where $D^* = \mathrm{e}^{-a_1 E^* - a_2 * I^* - a_3 * H^*}$.

3.1 Case $a_1 > 0, a_2 = 0, a_3 = 0$: the impact of the reported number of exposed individuals

We first explore the impact of the reported number of exposed individuals, hence $a_2 = a_3 = 0$ and $a_1 > 0$. In this case, the Jacobian of the endemic equilibrium J can be simplified to

$$J(E^+) = \begin{pmatrix} -\alpha(1 + \ln \mathcal{R}_0) & d + h & 0 \\ \alpha & -(d+h) & 0 \\ 0 & h & -(d_h + r) \end{pmatrix}.$$

The three eigenvalues are determined by

$$(\lambda + d_h + r)[\lambda^2 + (h + d + \alpha(1 + \mathcal{R}_0))\lambda + \alpha(d + h)\ln \mathcal{R}_0] = 0.$$

One can verify that all the three eigenvalues are negative. Therefore, the endemic equilibrium is a stable node and we have:

Theorem 3.2. For the model (2.2) with $a_2 = a_3 = 0$ and $a_1 > 0$, if $\mathcal{R}_0 > 1$, the unique endemic equilibrium is always a stable node.

The solution curves of the number of exposed, infectious and hospitalized individuals as functions of time are curves with at most one peak.

3.2 Case $a_1 = 0, a_2 > 0, a_3 = 0$ the impact of the reported number of infectious individuals

We now consider the impact of reported numbers of infectious cases, *i.e.* $a_1 = a_3 = 0$ and $a_2 > 0$. Note that the Jacobian of the endemic equilibrium J now can be simplified to

$$J(E^+) = \begin{pmatrix} -\alpha & (1 - \ln \mathcal{R}_0)(d + h) & 0 \\ \alpha & -(d+h) & 0 \\ 0 & h & -(d_h + r) \end{pmatrix}.$$

The corresponding characteristic equation becomes

$$(\lambda + d_h + r)[\lambda^2 + (h + d + \alpha)\lambda + \alpha(d + h)\ln \mathcal{R}_0] = 0.$$

One eigenvalue is $\lambda_1 = -(d_h + r) < 0$, the other two are determined by

$$\lambda^2 + (h + d + \alpha)\lambda + \alpha(d + h)\ln \mathcal{R}_0 = 0. \tag{3.3}$$

Denote the discriminant of (3.3) by $\Delta(h, \beta)$. Namely,

$$\Delta(h, \beta) = (\alpha + d + h)^2 - 4\alpha(d + h)\ln \mathcal{R}_0. \tag{3.4}$$

Note that both roots of (3.3) have negative real parts, and hence the endemic equilibrium is always locally asymptotically stable. However, as shown in figure 2, the sign of $\Delta(h, \beta)$ determines the type of this equilibrium. The endemic equilibrium is a focus if $\Delta(h, \beta) > 0$ and a node if $\Delta(h, \beta) < 0$. For the parameters in the region above the curve, multiple outbreaks with damped amplitudes may occur.

3.3 Case $a_1 = 0, a_2 = 0, a_3 > 0$: the impact of reported number of hospitalized individuals

Comparing to the previous two cases, media coverage on the number of hospitalized individuals has much stronger impact on the transmission patterns, and the endemic equilibrium can undergo a Hopf bifurcation. To show this, we first note that the Jacobian of the endemic equilibrium J can be simplified to

$$J(E^+) = \begin{pmatrix} -\alpha & d + h & -\frac{(d+h)(d_h+r)}{h}\ln \mathcal{R}_0 \\ \alpha & -(d + h) & 0 \\ 0 & h & -(d_h + r) \end{pmatrix}.$$

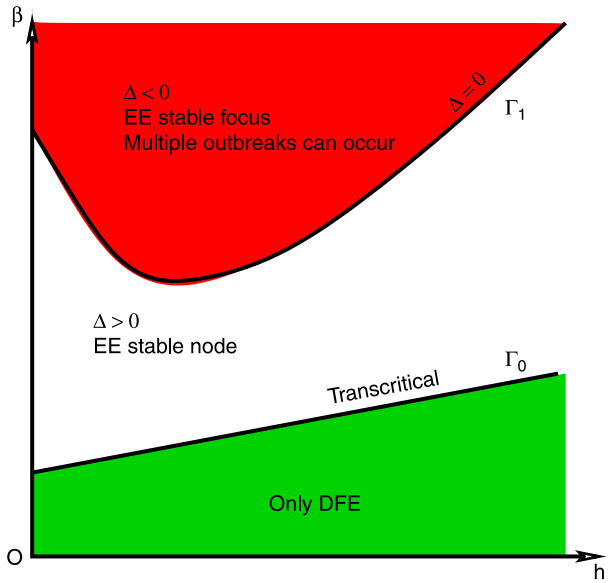

Figure 2. Bifurcation diagram for case $a_1 = a_3 = 0, a_2 > 0$. Γ_1 denotes the curve $\Delta(h,\beta) = 0$ defined in (3.4). Multiple outbreaks may occur for the parameters above the curve Γ_1.

The characteristic equation is

$$\begin{aligned} f(\lambda) =& \lambda^3 + (\alpha + h + d_h + r + d)\lambda^2 + (d_h + r)(h + \alpha + d)\lambda \\ &+ \alpha(d + h)(d_h + r)\ln \mathcal{R}_0 = 0. \end{aligned} \tag{3.5}$$

Let λ_i ($i = 1, 2, 3$) be the three eigenvalues, then we have

$$\begin{cases} \lambda_1 + \lambda_2 + \lambda_3 = -(\alpha + h + d_h + r + d), \\ \lambda_1\lambda_2 + \lambda_1\lambda_3 + \lambda_2\lambda_3 = (d_h + r)(h + \alpha + d), \\ \lambda_1\lambda_2\lambda_3 = -\alpha(d + h)(d_h + r)\ln \mathcal{R}_0. \end{cases} \tag{3.6}$$

We are interested in possible sustained periodic oscillations. Thus we consider the case when $f(\lambda) = 0$ has a pair of purely imaginary roots. For $f(\lambda) = 0$ to have a pair of purely imaginary roots, we must have

$$G(h, \beta) = (h + \alpha + d)(\alpha + h + d_h + r + d) - \alpha(d + h)\ln \mathcal{R}_0 = 0. \tag{3.7}$$

Therefore, if h and β satisfy $G(h, \beta) = 0$, in addition to one eigenvalue $\lambda_1 = -(\alpha + h + d_h + r + d) < 0$, the endemic equilibrium has a pair of imaginary eigenvalues:

$$\lambda_{2,3} = \pm i\sqrt{\frac{\alpha \ln \mathcal{R}_0(d + h)(d_h + r)}{\alpha + h + d_h + r + d}} = \pm i\sqrt{(h + \alpha + d)(d_h + r)}.$$

Let Γ_2 be the curve defined by $G(h, \beta) = 0$ in the (h, β)-plane:

$$\Gamma_2 : \beta = \beta(h) = \frac{d + h}{S} \mathrm{e}^{\frac{(h+\alpha+d)(h+\alpha+d_h+r+d)}{\alpha(d+h)}}. \tag{3.8}$$

As shown in figure 3 Γ_2 has a local minimum at $(\hat{h}_H, \hat{\beta}_H)$ with $\hat{h}_H = -(1/2)\alpha - d + (1/2)\sqrt{5\alpha^2 + 4\alpha d_h + 4\alpha r}$ and $\hat{\beta}_H = \beta(\hat{h}_H)$. When Γ_2 is crossed from below, a Hopf bifurcation occurs. The stability of the possible Hopf bifurcations from the endemic equilibrium is described in next theorem.

Theorem 3.3. Consider the model (2.2) with $a_1 = a_2 = 0$ and $a_3 > 0$. For (h, β) in the region above Γ_0 of the first quadrant of the (h, β)-plane, we have, as shown in figure 3, the following conclusions:

- if $((h + d)/S) < \beta < ((d + h)/S)\mathrm{e}^{(((h+\alpha+d)(h+\alpha+d_h+r+d))/(\alpha(d+h)))}$, the endemic equilibrium is locally asymptotically stable; if $\beta > ((d + h)/S)\mathrm{e}^{(((h+\alpha+d)(h+\alpha+d_h+r+d))/(\alpha(d+h)))}$, the endemic equilibrium is unstable.
- as β increases and Γ_2 is crossed, a Hopf bifurcation occurs. System (2.2) has a stable periodic solution.

Proof. Using the Routh–Hurwitz criteria, we get that if (h, β) is between Γ_2 and Γ_0, then all eigenvalues of the endemic equilibrium have negative real parts, hence the endemic equilibrium E^+ is locally asymptotically stable.

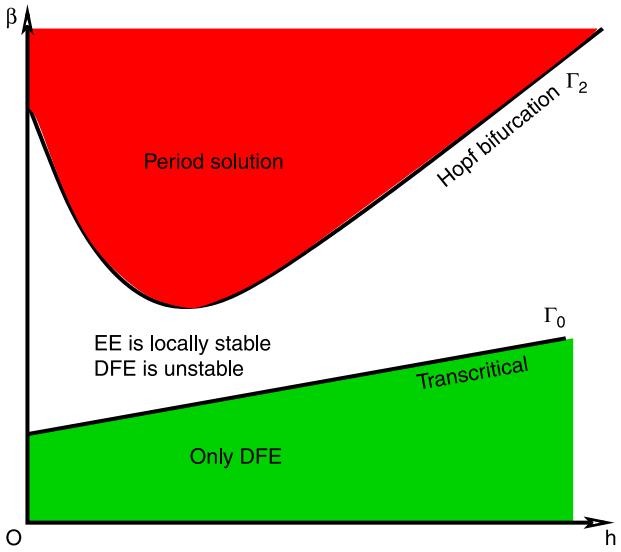

Figure 3. Bifurcation diagram for case $a_1 = a_2 = 0$ and $a_3 > 0$. Γ_2 is the Hopf bifurcation curve defined by $G(h,\beta) = 0$ in (3.7). When Γ_2 is crossed from below, a Hopf bifurcation occurs which results in the existence of sustained periodic solutions (periodic outbreaks).

By the definition of Γ_2 in (3.8) we know that a Hopf bifurcation may occur when Γ_2 is crossed [19]. Note that one eigenvalue is always negative. To see how the real parts of the other two eigenvalues change their signs, we check the transversality condition of the Hopf bifurcation. Let $\lambda = a \pm bi$ with $a, b \in \mathbf{R}$ be the pair of complex eigenvalues. Substituting $\lambda = a + bi$ into the characteristic equation, the real part of $f(\lambda)$ then becomes

$$R(a,b,h) = \alpha(d+h)(d_h+r)\ln\mathcal{R}_0 + (d_h + r + \alpha + d + h)a^2 + (-d_h - 3a - d - r - h - \alpha)b^2 + (\alpha + d + h)(d_h + r)a + a^3. \tag{3.9}$$

Note that if $G(h, \beta) = 0$, we have $a = 0$. Using implicit differentiation of (3.9), we have

$$\begin{aligned}\left.\frac{\partial a}{\partial h}\right|_{a=0,G=0} &= \left.\frac{-(\partial R/\partial\beta)}{\partial R/\partial a}\right|_{a=0,G=0} \\ &= -\left.\frac{\alpha(d+h)(d_h+r)}{\beta(3a^2 - 3b^2 + 2a(\alpha+h+d_h+r+d) + (d_h+r)(\alpha+d+h))}\right|_{a=0,G=0} \\ &= \frac{\alpha(d+h)}{2\beta(\alpha+d+h)} > 0,\end{aligned}$$

where if $a = 0$ and $G = 0$, $b^2 = -((\alpha(d+h)(d_h+r)\ln\mathcal{R}_0)/(\alpha+h+d_h+r+d)) = (\alpha+d+h)(d_h+r)$. It follows from [15] (Chapter 5) and [12] that a Hopf bifurcation occurs when the curve Γ_2 is crossed.

In order to decide the stability of the Hopf bifurcation, we have to compute the first Lyapunov coefficient of the endemic equilibrium. By using the formula in Ref. [15] (p. 175–178) and a short straightforward calculation, the first Lyapunov coefficient is negative.

Therefore, the Hopf bifurcation is supercritical, and the periodic solution bifurcated from E^+ is orbitally asymptotically stable. □

Recall that h is the reciprocal of the median time for an infectious individual to be admitted to a hospital for medical treatment and for isolation. Therefore, the above results show that sustained periodic oscillations can take place by either increasing this median time from relatively small to a moderate level, or by decreasing this median time from relatively large to a moderate level. This is because the individual's behavior and contact patterns are changed as a function of the number of hospitalized individuals and this represents a delayed response to the true level of infection (reflected more actually by the numbers of exposed and infectious individuals).

4. Simulations and discussions

The qualitative behaviors described in the previous section are independent of the specific disease under consideration. To explain the impact of media/psychology on possible multiple outbreaks of the transmission of infectious disease, we take the SARS outbreaks in Great Toronto Area in 2003 as an example. We now discuss the parameter values to be used in our simulations.

During the SARS outbreaks in Great Toronto Area, the media mainly focused on the number of hospitalized SARS patients, and also the only available and reliable data was the number of hospitalized cases, shown in figure 1. Hence in model (2.2), we take $a_1 = a_2 = 0$ and consider the model in the case discussed in section 3.3. For the parameter S, the total population in the Toronto area, based on the 1996 census adjusted by 1999 intercensus, we assume that $S = 5{,}446{,}104$ for the year 2003.

Since the average time for an infectious individual to be admitted to a hospital was estimated to be 3 days [5], we take $h = 0.33\ \text{day}^{-1}$. $1/d$ is the average period before an infectious individual dies without being admitted to a hospital. This number is small, reflecting a relatively low death rate. The 21 day survival in a Toronto cohort of 144 hospitalized cases was 93.5%, with negative outcomes most often associated with diabetes or other comorbid conditions [5]. Estimates for case fatality rates are also associated with age, with a fatality rate of 13.2% for those under 60, rising to 43.3% for those ($\geq$) 60 reported in Hong Kong [16]. Therefore we use $d = 0.00001$. Similarly, we use $d_h = 0.01$ if we think of death rate as 0.1 and the average hospitalization day as 10 days for those SARS patients who eventually die of the disease. In reality, both d_h and d are small, and the small perturbation of these parameters will not affect significantly the numerical simulations during a short period of time.

In Ref. [20], the authors study the critical roles of the nosocomial transmission of SARS, and the study yields an average infectious induced secondary infection 1.6 for the general community, and an average hospitalized patient induced secondary infection 4.5 for the health care setting, in comparison with the basic reproduction number around 3 estimated in Refs. [8,17] when the health care setting is not isolated from the general community. Note that those values of the reproduction number are the average value during the whole period of the SARS outbreak. While in model (2.2), we assume the contact rate is reduced as an exponential function of the number of hospitalized. So it is reasonable to take the basic reproduction number as 6.6, which is about twice as great as the estimated average reproduction number during the whole period of the SARS outbreak. We now use the reproduction number $\mathcal{R}_0 = (\beta S/(d + h))$ to estimate the contact rate β. Using the values of $\mathcal{R}_0$, d and h, we obtain that $\beta = 0.0000004$.

The value of a_3 is one of the main factors which decide the value of "controlled" average contact rate and the "controlled" average reproduction number. Here, "controlled" average contact rate β_c is defined as $\int_0^T \beta e^{-a_3 H(t)dt}/T$ where T is the length of the outbreak of the disease. "Controlled" average reproduction number $\mathcal{R}_c$ is given by $\beta_c/(d+h)$. Using the real hospitalized data, shown in figure 1, and the basic reproduction number around 3 estimated in Refs. [8,17] when the health care setting is not isolated from the general community, we can estimate $a_3 = 0.08$. Since the median time from self-reported earliest known exposure to symptoms onset is 5–6 days [5], we take $1/\alpha = 5$ days, *i.e.* $\alpha = 0.2$ day^{-1}. Though the median hospital stay was 10 days [5], the hospital stay for the infectious individuals in the early stage was much larger, hence $0 < r < 0.1$ and we take $r = 0.05$ day^{-1}.

We summarize all the parameters in table 1. We refer to [20] for more detailed discussions of the identification on the parameters.

The data shown in figure 1, of reported hospitalized SARS cases is taken from Ref. [7] which begins at February 23, 2003 and ends at July 4, 2003. In the simulations, the time unit is day, and $t = 0$ corresponds to February 23, 2003. The data is represented in figure 4 by blue bar. The smooth curve in figure 4 is the simulation prediction from the model. Note that there exists a second outbreak in the prediction curve, due to the media coverage of hospitalized individuals.

The individual's contact rate was reduced due to the media coverage of the number of hospitalized cases. Figure 5 shows how the contact transmission rate changes over the time. In this case the "controlled" average contact rate is $\beta_c = 0.00000009$, which yields the "controlled" average reproduction number 1.55.

System (2.2) seems to be the first model that incorporates the media or psychological impact based on a simple EIH model. Our analysis and simulations based on model (2.2) suggested a possible mechanism for multiple outbreaks of highly infectious diseases: the impact of the emphasis of media coverage for the hospitalized individuals. These results show that caution must be exercised when the number of hospitalized individuals decreases, and premature relaxing of control measures may lead to increase in the contact rate which can result in a possible secondary outbreak.

SARS was first recognized in Toronto on February 23, 2003. Transmission to other persons resulted subsequently in an outbreak among 257 persons in several Greater Toronto Area hospitals. After implementation of province wide public health measures that included strict infection-control practices, the number of recognized cases of SARS declined substantially, and no new cases were detected and reported after April 20. On May 14, 2003, WHO [13] removed Toronto from the list of areas with recent local SARS transmission because 20 days (*i.e.* twice the maximum incubation period) had elapsed since the most recent case of locally acquired SARS was isolated or a SARS patient had died, suggesting that the chain of transmission had terminated. Unfortunately, a secondary outbreak occurred after WHO's removal of the travel advisory against Toronto. Whether the relaxation of the control measures contributed to the

Table 1. Parameters for the simulation of the SARS outbreak in GTA in 2003.

Parameters	Value	The source of data
β	0.0000004	Estimation
h	0.33 day^{-1}	[20]
a_3	0.08	Estimation
α	0.2 day^{-1}	[20]
d	0.000001 day^{-1}	Estimation
d_h	0.01 day^{-1}	Estimation
r	0.05 day^{-1}	[20]

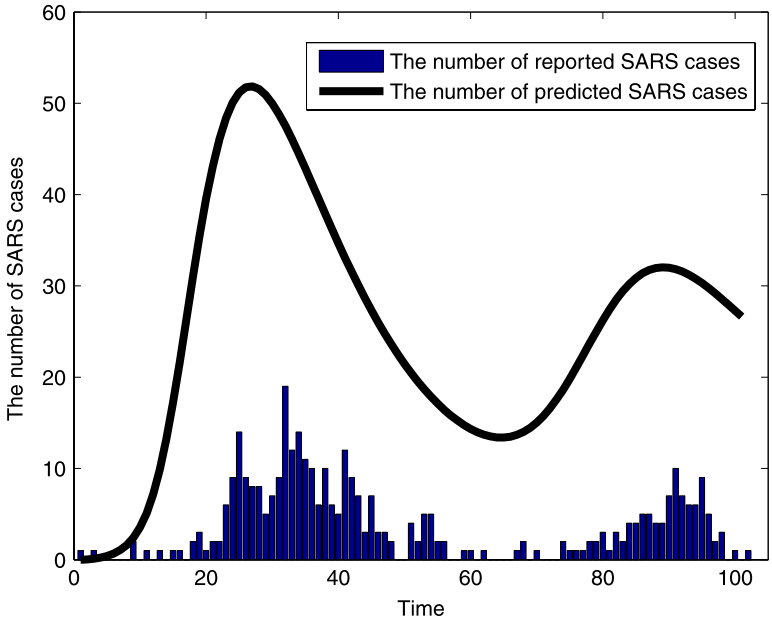

Figure 4. The comparison of the real data and the model simulation for the case of 2003 SARS outbreak in GTA.

secondary outbreak remains debatable; our study confirms that when the total population size remains a constant and other factors are ignored, the media coverage/impact is not the intrinsic factor that decides if the disease will outbreak, but has great impact on the pattern and scale of the transmission. Therefore, it is critical to maintain a high level of alertness especially after a decline of the hospitalized cases is reported [7].

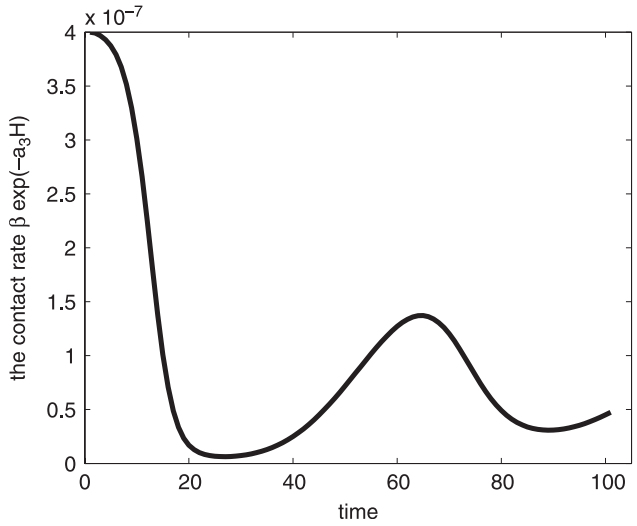

Figure 5. The time dependent contact rate changes with respect to time.

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