

GOPEN ACCESS

Citation: Zhou W, Xiao Y, Heffernan JM (2019) Optimal media reporting intensity on mitigating spread of an emerging infectious disease. PLoS ONE 14(3): e0213898. https://doi.org/10.1371/ journal.pone.0213898

Editor: Enrique Castro-Sánchez, Imperial College London, UNITED KINGDOM

Received: April 21, 2018

Accepted: March 4, 2019

Published: March 21, 2019

Copyright: © 2019 Zhou et al. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Data Availability Statement: All relevant data are within the paper and its Supporting Information files.

Funding: The authors were supported by the National Natural Science Foundation of China (NSFC, 11631012, 11571273), the Natural Science and Engineering Research Council of Canada (NSERC) and the York Research Chair. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript. **RESEARCH ARTICLE**

Optimal media reporting intensity on mitigating spread of an emerging infectious disease

Weike Zhou¹, Yanni Xiao¹*, Jane Marie Heffernan²

1 Department of Applied Mathematics, School of Mathematics and Statistics, Xi'an Jiaotong University, Xi'an 710049, PR China, 2 Department of Mathematics & Statistics, York University, Toronto, Canada

* yxiao@mail.xjtu.edu.cn

Abstract

Mass media reports can induce individual behaviour change during a disease outbreak. which has been found to be useful as it reduces the force of infection. We propose a compartmental model by including a new compartment of the intensity of the media reports, which extends existing models by considering a novel media function, which is dependent both on the number of infected individuals and on the intensity of mass media. The existence and stability of the equilibria are analyzed and an optimal control problem of minimizing the total number of cases and total cost is considered, using reduction or enhancement in the media reporting rate as the control. With the help of Pontryagin's Maximum Principle, we obtain the optimal media reporting intensity. Through parameterization of the model with the 2009 A/H1N1 influenza outbreak data in the 8th Hospital of Xi'an in Shaanxi Province of China, we obtain the basic reproduction number for the formulated model with two particular media functions. The optimal media reporting intensity obtained here indicates that during the early stage of an epidemic we should guickly enhance media reporting intensity, and keep it at a maximum level until it can finally weaken when epidemic cases have decreased significantly. Numerical simulations show that media impact reduces the number of cases during an epidemic, but that the number of cases is further mitigated under the optimal reporting intensity. Sensitivity analysis implies that the outbreak severity is more sensitive to the weight α_1 (weight of media effect sensitive to infected individuals) than weight α_2 (weight of media effect sensitive to media items).

Introduction

Emerging and reemerging infectious diseases including the 2003 severe acute respiratory syndrome (SARS) and the 2009 A/H1N1 influenza epidemic have become a major cause of mortality and morbidity in emergency situations. News reports have the potential to modify a community's knowledge of emerging infectious diseases, and affect people's attitudes and behaviours during infectious disease outbreaks [1, 2]. People informed by media reports can take precautions ranging from washing hands, wearing protective masks to avoiding social **Competing interests:** The authors have declared that no competing interests exist.

contact with infected individuals, to reduce their susceptibility. Informed infective individuals will also take measures to protect themselves from being exposed to others to reduce infectivity. It has been shown that behaviour change during infectious disease outbreaks can curb the effects of infectious diseases in populations [3].

In recent years, a growing number of studies have focused on understanding and quantifying the impact of such behaviour influencing factors on the spread of infectious diseases [4– 18]. A number of studies have employed mathematical models to assess the impact of media reports on emerging infectious disease prevention and control [4, 5, 19-30]. Recently, Greenhalgh et al. [19] presented a brief and nice commentary on the literature related to awareness and their effects on the dynamics of diseases. In summary, it has been found that there are three main methods being used to incorporate behaviour change in mathematical models due to awareness of disease. In the first method, the incidence rate of the disease is reduced by some factors that depend on the numbers of infected individuals, hospitalized individuals or exposed individuals, due to education about preventative knowledge of the disease through media coverage. The common choice of the reduction factors is a saturated [4, 6, 31, 32] or exponential [20, 24, 27] growth function. For example, Liu et al. [27] incorporated an exponential decreasing factor $\beta_0 = \beta e^{-a_1 E - a_2 I - a_3 H}$ into the transmission coefficient (with exposed (E), infectious (I), hospitalized (H)) to illustrate the possible mechanism for multiple outbreaks of SARS due to the psychological impact. Cui et al. [32] used the general nonlinear incidence function $\mu_1 - \mu_2 f(I)$ to represent the media and education impact on the spread of the infectious disease. In [5], the authors focused on simple endemic models by modelling the contact rate as a function of the available information on the present and the past disease prevalence.

In the second method, a separate compartment that effectively represents the level of awareness in the population is introduced, and individuals in the population can move from the unaware to aware compartments [7, 15, 16, 19, 21–23, 28]. For example, in [19], the authors proposed a mathematical model by inducing behavioural changes in the population through delineation of the susceptible class into unaware susceptible and aware susceptible subpopulations. [16] and [29] explicitly introduced distinct compartments for unaware and aware individuals in each of the disease states, and transitions between respective unaware and aware compartments took place at constant rates.

In the third method, a compartment representing the awareness program is incorporated [8, 18, 21, 25]. Yan and Tang et al. [25] described the effects of media reports on population infection by modifying the transmission rate β following an exponential function βe^{-pM} with M representing the level of media reports. Further, in [8], the media reporting is introduced as a separate compartment in a mathematical model and the susceptible population is divided into three awareness levels, each with a different infection rate. In [18], the authors considered the interaction of disease outbreak and media impact by formulating a susceptible-infected-hospitalized-recovered framework of population. By extension, susceptible and infected populations are subdivided into aware and unaware since individuals modified their behaviors to reduce their transmissibility and infectivity, and the dynamics of media reports was incorporated by considering how media was influenced by the numbers of infected and hospitalized individuals.

The majority of the mathematical modelling studies described above have incorporated media impact either in the disease transmission term or by dividing the susceptible population into subgroups with various awareness levels. However, the relationship between mass media and disease spread can be more complex than these models portray. On one hand, media reporting influences the public awareness of the disease and affects the effectiveness of prevention measures. On the other hand, the severity of the disease has an impact on the degree of mass media reporting. We've known that, in the first and third methods, media impact is

modelled through the inclusion of a "media function", which is proportional to the number of infected individuals and/or the level of media reports, to reduce the incidence rate through increased protective behaviour. However, it remains unclear as to whether awareness of the number of infections, or the awareness of media reports best modify individual behaviour during an infectious disease outbreak. This falls within the scope of this study.

Herewithin, we establish a mathematical model incorporating media reports as a separate compartment by considering how media is influenced by disease statistics (number of newly observed individuals). Disease progression is characterized by an SEIR model of which the transmission rate is modified by a media function affected by the media reports and also the number of infected individuals. The model can be recognized as the combination of the first method and the third method of modelling. In our model, we formulate the novel media function $f(I, M, \alpha_1, \alpha_2)$ with α_i (i = 1, 2) denoting the weight of infected individuals and media reports, and examine their effects on disease spread. Further, we investigate an optimal control problem in order to seek the optimal reporting intensity of information to minimize the number of infected individuals (and costs). We parameterize the proposed model on the basis of the 2009 A/H1N1 data in Shaanxi province of China, and estimate the basic reproduction number and other unknown parameters. A sensitivity analysis is conducted to identify model parameters that most affect the peak magnitude of the epidemic, as well as the total number of infections over the entire epidemic.

Methods

Model

We are interested in studying the effects of *I* and *M* on the outcomes of an infectious disease outbreak/epidemic. We therefore consider a mass media compartment *M* and a media effect function that depends on both the number of infected *I* and media reports *M*. Consider an SEIR model that incorporates a compartment of media programs *M*, in which the media impact on the human behaviour is reflected in the contact rate.

$$\begin{cases} \frac{dS}{dt} = \Lambda - f(I, M, \alpha_1, \alpha_2)\beta SI - \mu S, \\ \frac{dE}{dt} = f(I, M, \alpha_1, \alpha_2)\beta SI - \sigma E - \mu E, \\ \frac{dI}{dt} = \sigma E - \gamma I - \mu I, \\ \frac{dR}{dt} = \gamma I - \mu R, \\ \frac{dM}{dt} = \rho \sigma E - \delta M. \end{cases}$$
(1)

where S(t), E(t), I(t), R(t) represent the susceptible, exposed, infective, and recovered populations, respectively, M(t) represents the number of news items. Here, Λ is the birth rate, μ is the natural death rate, σ is the progression rate from the exposed to infective classes, and γ is the recovery rate. The propagation of information depends on the number of newly observed individuals (σE), and ρ represents the reporting rate of the newly observed individuals. It is assumed that δ represents the spontaneous disappearance rate of media. The baseline transmission rate without media effect is represented by β and $f(M, I, \alpha_1, \alpha_2)$ is used to modify the transmission rate, which is induced by the media effect. Finally, α_1 , α_2 are the weights of media effect sensitive to infectives and media items, respectively. All the parameters are nonnegative.

It's obvious that the media impact on the behavior of humans increases as the number of infected individuals increases or the intensity of media reports increases. Thus the term of media impact factor reflecting the behavior change $f(I, M, \alpha_1, \alpha_2)$ is a decreasing function with

respect to the number of infected individuals *I* and the media intensity *M*, it should satisfy the following assumptions:

$$\frac{\partial f(I, M, \alpha_1, \alpha_2)}{\partial I} \le 0 \quad \text{for all} \quad I > 0, \quad \frac{\partial f(I, M, \alpha_1, \alpha_2)}{\partial M} \le 0 \quad \text{for all} \quad M > 0,$$

$$f(0, 0, \alpha_1, \alpha_2) = 1, \quad f(I, M, \alpha_1, \alpha_2) \to 0 \quad \text{as} \quad I \to \infty \quad \text{or} \quad M \to \infty.$$
(2)

Here, we choose two different media functions,

$$f_1(I, M, \alpha_1, \alpha_2) = e^{-\alpha_1 I - \alpha_2 M},$$

and

$$f_2(I,M,lpha_1,lpha_2)=rac{1}{1+lpha_1I+lpha_2M}$$

Optimal control

In system (1), the reporting rate is proportional to the newly reported number of infected individuals. It is natural to ask whether we should enhance the reporting rate to reduce the total infected number and minimize the cost of media reporting. Thus our main purpose is to minimize the total number of infective individuals as well as the cost required to reduce or enhance the media reporting intensity.

Consider the optimal control problem to minimize the objective functional

$$I(u) = \int_{t_0}^{T} [AI(t) + \frac{B}{2}u^2(t)]dt$$
(3)

subject to

$$\begin{cases} \frac{dS}{dt} = \Lambda - \beta f(I, M)SI - \mu S, \quad S(t_0) = S_0 > 0, \\ \frac{dE}{dt} = \beta f(I, M)SI - \sigma E - \mu E, \quad E(t_0) = E_0 \ge 0, \\ \frac{dI}{dt} = \sigma E - \gamma I - \mu I, \quad I(t_0) = I_0 \ge 0, \\ \frac{dR}{dt} = \gamma I - \mu R, \quad R(t_0) = R_0 \ge 0, \\ \frac{dM}{dt} = u(t)\rho\sigma E - \delta M, \quad M(t_0) = M_0 \ge 0. \end{cases}$$

$$(4)$$

where the coefficients *A* and *B*/2 are positive. Here we assume that A = 1, and that *B*/2 is the weight associated with the control u(t). Note that u(t) is a Lebesgue measurable function on a finite interval $[t_0, t_{end}]$, where $0 \le u(t) \le u_{max}$, $u_{max} > 1$, and $0 \le u(t) < 1$ represents reduction in reporting intensity, whereas $1 < u(t) \le u_{max}$ represents enhancement in reporting intensity.

Parameter values

We use data from the 8th hospital of Xi'an in Shaanxi province to study the effects of media reports. The data are fully available in <u>S2 File</u>. The data include information on the daily number of hospital notifications in the 8th hospital from September 3 to 30, 2009. Parameter values for Eq (1) are informed by the literature and are further estimated through model fits to the hospital notification data, using the Least Square Method.

Variables	Description		Resource			
		f_0	f_1	f_2		
S(t)	Susceptible population	28410	28410	28410	LS	
E(t)	Exposed population	59	59	61	LS	
I(t)	Infected population	4	4	4	data	
R(t)	Recovered population	0	0	0	[25]	
M(t)	Media items	8	8	8	[25]	
Parameters	Description		Value			
		f_0	f_1	f_2		
Λ	Birth rate of the population (per day)	0	0	0	-	
μ	Natural death rate of the population (per day)	0	0	0	-	
β	Contact transmission rate (per person per day)	0.0000154	0.0000158	0.0000158	LS	
α_1	Weight of media effect sensitive to infected individuals	0	0.00015	0.00015	LS	
α_2	Weight of media effect sensitive to media items	0	0.0138	0.0122	LS	
σ	Progression rate from exposed to infected (per day)	1/2.8	1/2.8	1/2.8	[34]	
γ	Recovery rate of infected population (per day)	1/4.16	1/4.16	1/4.16	[34]	
ρ	Media reporting rate (per day)	-	0.01	0.01	LS	
δ	Media waning rate (per day)	-	0.4940	0.2535	LS	

Table 1. Values of initial populations and parameters in the model (1).

https://doi.org/10.1371/journal.pone.0213898.t001

To ensure that our model estimates of the basic reproduction number, R_0 , are from the exponential growth phase of infection, we assume data from September 3-21 [33]. Also, so that we can compare the effects of *I* and *M* on the optimal control of media reporting, we assume a mass media compartment and a media function that depends both on *I* and *M*. Table 1 lists the best-fit parameters determined for model (1), without media impact $f(I, M, \alpha_1, \alpha_2) = f_0(I, M) = 1$ and with two different media functions $f_1 = e^{-\alpha_1 I - \alpha_2 M}$ and $f_2 = \frac{1}{1 + \alpha_1 I + \alpha_2 M}$, respectively.

Note that, for completeness, we consider the extended dataset from September 3-30 in a sensitivity analysis. Also, we have provided model fits and parameter values when the mass media compartment M is not included in the model in <u>S1 File</u> Appendix C. However, as we are interested in comparing the effects of I and M in the current study, we consider the full model with the M compartment in the optimal control and sensitivity analysis.

Results

Equilibrium

The basic reproduction number for system (1) can be calculated as

$$\mathcal{R}_0 = \frac{\beta \sigma \Lambda}{\mu (\gamma + \mu) (\sigma + \mu)} \tag{5}$$

easily using the next generation method [35] or the survival function method (see [36] for a review of this method and other methods that are commonly used). Note that the basic reproduction number is independent of the mass media compartment. Also note that it is not affected by the media function f(I, M). Therefore, it is the same as it would be in a model without media impact, which means that media coverage does not play a role in affecting this epidemic threshold. This has also been observed in previous studies [4, 6, 11].

Omitting the equation for *R*, the system (1) can be rewritten as a four dimensional model. Here, the disease free equilibrium is $\mathbb{E}_0(\frac{\Lambda}{n}, 0, 0, 0)$, and the endemic equilibrium

 $\mathbb{E}^*(S^*, E^*, I^*, M^*)$ should satisfy

$$\begin{cases} \Lambda - f(I^*, M^*)\beta S^*I^* - \mu S^* = 0, \\ f(I^*, M^*)\beta S^*I^* - \sigma E^* - \mu E^* = 0, \\ \sigma E^* - \gamma I^* - \mu I^* = 0, \\ \rho \sigma E^* - \delta M^* = 0, \end{cases}$$
(6)

Simplifying gives

 $S^* = \frac{\Lambda}{\mu} - \frac{\delta(\sigma + \mu)}{\rho \sigma \mu} M^*, \quad E^* = \frac{\delta}{\rho \sigma} M^*, \quad I^* = \frac{\delta}{\rho(\gamma + \mu)} M^*, \tag{7}$

and

$$f(I^*, M^*)\left(\Lambda - \frac{\delta(\sigma + \mu)}{\rho\sigma}M^*\right) = \frac{\mu(\sigma + \mu)(\gamma + \mu)}{\beta\sigma}.$$
(8)

Since I^* can be expressed by M^* , we can rewrite $f(I^*, M^*)$ as $h(M^*) = f(\frac{\delta}{\rho(\gamma+\mu)}M^*, M^*)$. Denote $h(M) = f(\frac{\delta}{\rho(\gamma+\mu)}M, M)$, then h(M) is a decreasing function with respect to M. Let

$$g(M) = h(M) \left(\Lambda - \frac{\delta(\sigma + \mu)}{\rho \sigma} M \right) - \frac{\mu(\sigma + \mu)(\gamma + \mu)}{\beta \sigma}.$$
 (9)

Then we have $g(0) = \Lambda - \frac{\mu(\sigma+\mu)(\gamma+\mu)}{\beta\sigma} > 0$ when $\mathcal{R}_0 > 1$, $g(\frac{\rho\sigma\Lambda}{\delta(\sigma+\mu)}) = -\frac{\mu(\sigma+\mu)(\gamma+\mu)}{\beta\sigma} < 0$, and $g'(M) = h'(M)(\Lambda - \frac{\delta(\sigma+\mu)}{\rho\sigma}M) - \frac{\delta(\sigma+\mu)}{\rho\sigma}h(M) < 0$ holds true for any M satisfying $\Lambda - \frac{\delta(\sigma+\mu)}{\rho\sigma}M > 0$. Thus there must exist one and only one positive root $M^* < \frac{\rho\sigma\Lambda}{\delta(\sigma+\mu)}$ that satisfies $g(M^*) = 0$ if $\mathcal{R}_0 > 1$. Particularly, if we choose

$$f_1(I,M) = e^{-\alpha_1 I - \alpha_2 M}$$

and

$$f_2(I,M) = \frac{1}{1 + \alpha_1 I + \alpha_2 M},$$

we have

$$M^{*} = \frac{\rho \sigma \Lambda}{\delta(\sigma + \mu)} - \frac{\rho(\gamma + \mu)}{\alpha_{1}\delta + \alpha_{2}\rho(\gamma + \mu)} LambertW(\frac{\mu(\alpha_{1}\delta + \alpha_{2}\rho(\gamma + \mu))}{\beta\delta}e^{\frac{(\alpha_{1}\delta + \alpha_{2}\rho(\gamma + \mu))\sigma\Lambda}{\delta(\sigma + \mu)(\gamma + \mu)}}),$$

and

$$M^* = rac{
ho \mu(\gamma+\mu)(\mathcal{R}_0-1)}{\mu(lpha_1\delta+lpha_2
ho(\gamma+\mu))+eta\delta},$$

respectively. (Please find detailed definition of Lambert W function in paper [10, 37]).

Note that the disease free equilibrium $\mathbb{E}_0(\frac{\Lambda}{\mu}, 0, 0, 0)$ is globally asymptotically stable if $\mathcal{R}_0 \leq 1$, and unstable if $\mathcal{R}_0 > 1$. Meanwhile, the unique endemic equilibrium $\mathbb{E}^*(S^*, E^*, I^*, M^*)$ exists if and only if $\mathcal{R}_0 > 1$ and it is locally asymptotically stable if it is feasible. For more information about the stability of the disease free equilibrium and the endemic equilibrium, see S1 File Appendix A.

Existence of optimal control

Denote the control set

 $U = \{u(t) : 0 \le u(t) \le u_{max}, t_0 \le t \le t_{end}, u(t) \text{ is Lebesgue measurable}\}$. The existence of optimal control can be shown using the results from Theorem 4.1 in [38]. We can easily verify the following properties:

- 1. The set of control and corresponding state variable is non-empty, which can be shown by the boundedness of solutions of system (4) using the results from Theorem 9.2 in [39].
- 2. The control set \mathcal{U} is closed and convex by definition.
- 3. The right-hand side of the state system is bounded above by a linear function in the state and control, since the solutions are bounded, which determines the compactness needed for the existence of the optimal control.
- 4. The integrand of the objective functional is convex on the control u(t), and there exists $q_1 > 0$, $q_2 > 1$ such that $AI(t) + B/2u(t)^2 \ge q_1|u(t)|^{q_2} q_3$, where we can choose $q_1 = B/2$ and $q_2 = 2$.

Then we have that, for the control problem (3) and (4), there exists an optimal control $u^* \in \mathcal{U}$ such that min $J(u) = J(u^*)$ on the interval $[t_0, t_{end}]$.

Theorem 1 There exists an optimal control u^* that minimizes J(u) over U. Moreover, there exists adjoint functions

$$\begin{cases} \lambda_{s}'(t) = \beta f(I, M) I(\lambda_{s} - \lambda_{E}) + \mu \lambda_{E}, \\ \lambda_{E}'(t) = \sigma(\lambda_{E} - \lambda_{I} - \rho u(t) \lambda_{M}) + \mu \lambda_{E}, \\ \lambda_{I}'(t) = -A + \beta f(I, M) S(\lambda_{s} - \lambda_{E}) + \beta SI \frac{\partial f(I,M)}{\partial I} (\lambda_{s} - \lambda_{E}) + \gamma(\lambda_{I} - \lambda_{R}) + \mu \lambda_{I}, \\ \lambda_{R}'(t) = \mu \lambda_{R}, \\ \lambda_{M}'(t) = \beta SI \frac{\partial f(I,M)}{\partial M} (\lambda_{s} - \lambda_{E}) + \delta \lambda_{M}, \end{cases}$$
(10)

with the transversality conditions

$$\lambda_{S}(t_{end}) = 0, \lambda_{E}(t_{end}) = 0, \lambda_{I}(t_{end}) = 0, \lambda_{R}(t_{end}) = 0, \lambda_{M}(t_{end}) = 0.$$
(11)

The optimal control u^{*} *is given by*

$$u^*(t) = \max\{\min\{\frac{-\lambda_M \rho \sigma E}{B}, u_{max}\}, 0\}.$$
(12)

For detailed derivation of the optimal control for the control problem (3) and (4), see <u>S1</u> File Appendix B.

Numerical simulation

During the 2009 A/H1N1 influenza pandemic, media coverage was used to spread precaution information about the disease, which influenced human behaviours [11, 25]. Using data extracted from the initial laboratory-confirmed cases of 2009 A/H1N1 that were admitted to the 8th hospital of Xi'an for the period 3rd September to 21st September, and the Least Square Method, we estimated the parameters shown in Table 1 and Fig 1, with R-square value being 0.9588, 0.9577, 0.9583, using three different media functions f_0 , f_1 , f_2 . Note that the goodness of fit is significant, but similar for each case. This is not unexpected as the data spans 19 days only. Also, however, as shown in our discussion of R_0 , during the early stages of an epidemic

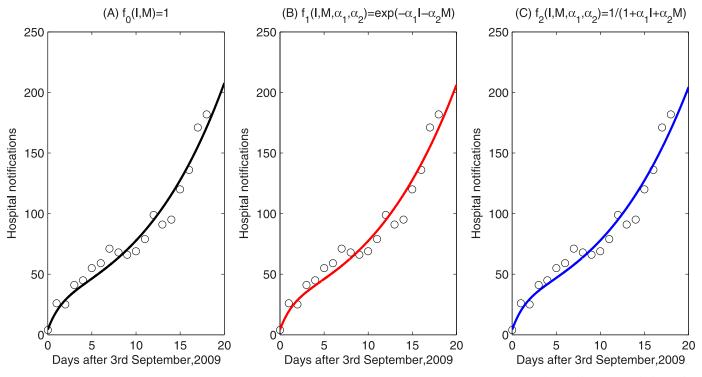


Fig 1. Data fitting for the daily number of hospital notifications from September 3rd to 21st, 2009 in the 8th hospital of Xi'an. Circles represent the hospital notifications in the 8th hospital of Xi'an, black curve, red curve and blue curve represent the estimating solutions for the number of infected individuals in system (1) without media impact (i.e. $f_0 = 1$), and by using media function $f_1 = e^{-\alpha_1 l - \alpha_2 M}$, $f_2 = \frac{1}{1 + \alpha_1 l + \alpha_2 M}$, respectively.

there is little effect due to mass media reports. Therefore it is reasonable that the model fits and parameter values are similar comparing the model with $(f_1, \text{ or } f_2)$ and without (f_0) media.

Using the Akaike Information Criterion (*AIC*) for Least-Squares case, $AIC = n \log \left(\frac{RSS}{n}\right) + 2k$, where *n* is the sample size, *k* is the number of parameters, and *RSS* denotes residual sum of squares of fitted model, we obtain an *AIC* of 90.8918 for the model without media impact (i.e. the model with f_0), 99.3676 for the model with f_1 , and 99.0981 for the model with f_2 . We note that the model without media impact has the lowest *AIC* and models that do not consider the mass media compartment *M* also fit the data well (see S1 File Appendix C). However, as we are interested in understanding the effects of mass media reports *M* and known infectives *I* to an individual in the population, we continue our study considering the model with the *M* compartment, and media functions f_1 and f_2 . Considering the R-square and *AIC*, we conclude that model (1) with f_2 fits the observed data better than the model with media function f_1 .

To show the sensitivity of the basic reproduction number R_0 with respect to the time interval considered, we also estimated key epidemic parameters and initial conditions considering the time periods between September 3rd and 23rd, 25th, 28th, and 30th, respectively. Results are presented in Table 2 and shown in Fig 2. When different periods are considered, the reproduction number varies from 1.8715 to 2.0463. The results indicate that, for each period we consider, the model with both f_1 and f_2 can fit the observed data well. Note that we have chosen to consider the model fits using data from the 3rd to 21st of September in the analysis below. This is done to ensure that we estimate R_0 during the exponential growth phase of the outbreak [33].

Parameter	Sep. 3-21	Sep. 3-21		Sep. 3-23		Sep. 3-25		Sep. 3-28		Sep. 3-30	
	f_1	f_2	f_1	f_2	f_1	f_2	f_1	f_2	f_1	f_2	
S(0)	28410	28410	28970	29000	29512	29510	28483	28558	29352	29351	
E(0)	59	61	58	57	77	79	60	62	66	66	
в	1.58e-5	1.58e-5	1.583e-5	1.58e-5	1.6622e-5	1.6571e-5	1.6401e-5	1.5947e-5	1.68e-5	1.672e-5	
α_1	1.5e-4	1.5e-4	1.5e-4	1.5e-4	1.9691e-4	1.7433e-4	4.9951e-4	3.6903e-4	0.0011	0.0013	
α2	0.0138	0.0122	0.01	0.01	0.1124	0.1782	0.0166	0.0146	0.0598	0.0563	
0	0.01	0.01	0.0352	0.01	0.0032	0.0024	0.01	0.01	0.01	0.01	
8	0.4940	0.2535	0.5	0.2940	0.2616	0.3086	0.2914	0.2577	0.4469	0.4481	
R _o	1.8715	1.8716	1.9118	1.9101	2.0463	2.0401	1.9477	1.8989	2.0563	2.0589	
R-square	0.9577	0.9583	0.9663	0.9687	0.9796	0.9787	0.9803	0.9797	0.9602	0.9583	
AIC	99.3676	99.0981	106.6231	105.1710	107.4610	107.3906	117.3241	117.9277	140.9177	141.999	

Using data from September 3-21, the basic reproduction number is estimated as $R_0 =$ 1.8248 without media impact ($f(I, M, \alpha_1, \alpha_2) = f_0 = 1$), which is lower than the estimates for $R_0 =$ 1.8715 for media function $f(I, M, \alpha_1, \alpha_2) = f_1$ and 1.8716 for media function $f(I, M, \alpha_1, \alpha_2) = f_2$. These three values of R_0 are all in agreement with the result in [25], in which the mean value of the basic reproduction number was estimated as 1.794 with 95% confidence interval [1.3858, 1.9091]. Again, we note that the similar R_0 values reflect the fact that there is little impact of media reports in the early days of the epidemic.

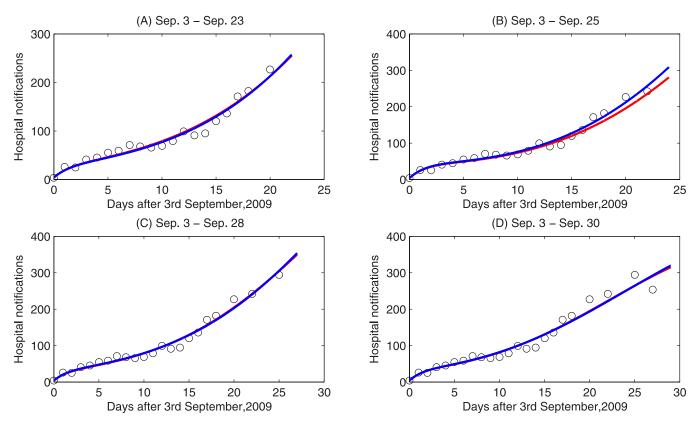


Fig 2. Data fitting for four time intervals. (A) September 3-23; (B) September 3-25; (C) September 3-28; (D) September 3-30, 2009. Circles represent the hospital notifications in the 8th hospital of Xi'an, red and blue curves are the fitting curves for the model with f_1 and f_2 , respectively.

https://doi.org/10.1371/journal.pone.0213898.g002

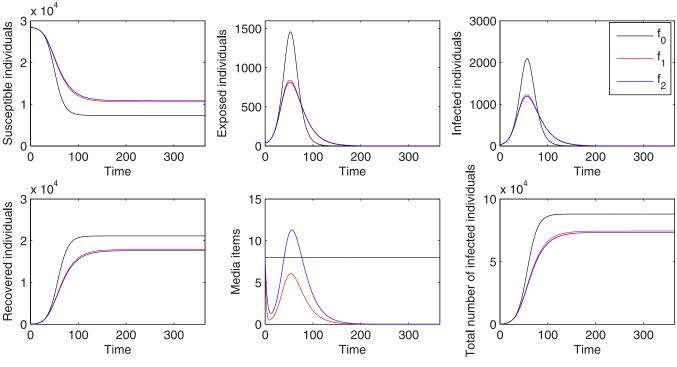


Fig 3. Comparison of solutions for system (1) with and without media effect. Black, red, blue curves represent the result of without media effect ($f_0 = 1$), with media function $f_1 = e^{-\alpha_1 I - \alpha_2 M}$, and with media function $f_2 = \frac{1}{1 + \alpha_1 I + \alpha_2 M}$, respectively.

We plot the epidemic curves of system (1) without media function ($f_0 = 1$) and with media function f_1 , f_2 , in Fig 3, using the parameter values listed in Table 1. This figure shows that the number of infected individuals is greatly reduced when media impact is considered. For example, the peak magnitude is reduced from 2091 to 1225 (1189) when we use the media function f_1 (f_2), giving a reduction of 41.4% (43.1%). The total number of infected individuals over one year is also reduced, from 87974 to 74391 (73370). It also follows from Fig 3 that there is no obvious difference on the epidemic prevalence for system (1) with media function f_1 or function f_2 , though the number of media items looks very different for the different media functions, which may be attributed to the small numbers of media items and the small value of the weight of media effects sensitive to the media reports. This is also confirmed in Fig 4(A) and 4(B) that media functions f_1 and f_2 are almost the same under their estimated parameters.

The contour plots of Fig 5 show the dependence of the peak magnitude and the total number of infections on the weight of media effects sensitive to infected individuals α_1 and the weight of media effects sensitive to the media reports α_2 , using the two different media functions. With increases in α_1 or α_2 , both the peak magnitude of the number of infected individuals and the total number of infections over a year decrease greatly, indicating that the media effect reduces outbreak severity. To identify key parameters that influence the disease infection dynamics, we use Latin Hypercube Sampling (LHS) and partial rank correlation coefficients (PRCCs) to examine the dependence of the peak magnitude and total number of infections on corresponding model parameters. It follows from Figs 6 and 7 that, despite the fact that the baseline value of the transmission rate β and the recovery rate γ are the most sensitive parameters to the peak magnitude and the total number of infections, parameters related to the media coverage $\alpha_1, \alpha_2, \rho, \delta$ can also significantly affect the results. In particular, increases in the weight of infective cases α_1 and media reports α_2 in the media function will significantly

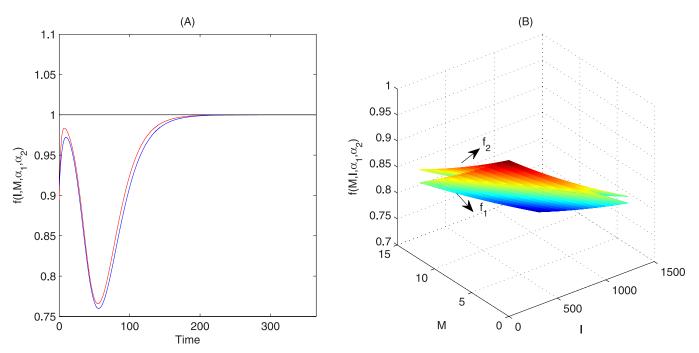


Fig 4. Comparison of media functions. (A) Comparison of media functions in system (1) varying with time, black curve, red curve and blue curve represent $f_0 = 1, f_1 = e^{-\alpha_1 I - \alpha_2 M}, f_2 = \frac{1}{1 + \alpha_1 I + \alpha_2 M}$, respectively. (B) Comparison of f_1 and f_2 when parameters α_1 and α_2 are fixed as values estimated by using Least Square Method and *I*, *M* vary.

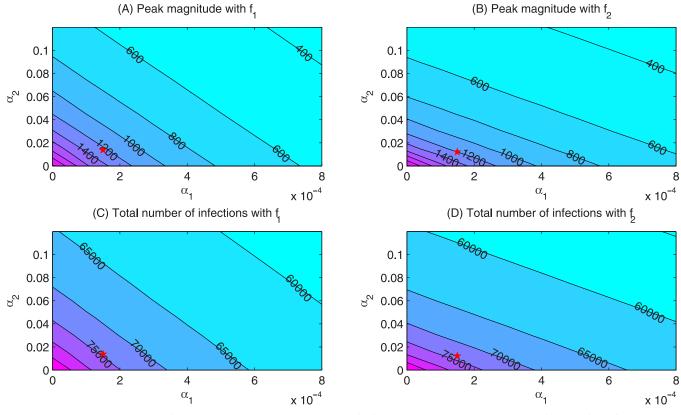
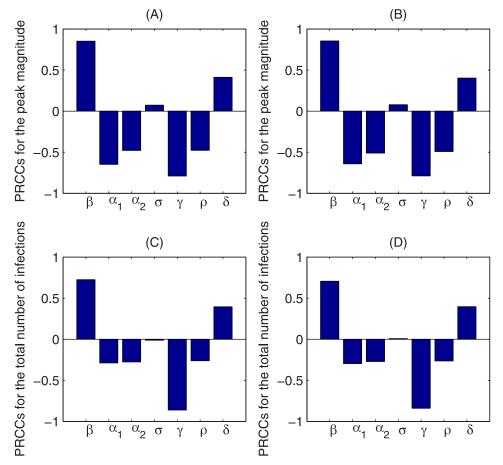
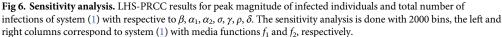


Fig 5. Contour plots. Contour plots of the peak magnitude and the total number of infections versus α_1 and α_2 by using media functions f_1 and f_2 , respectively. The red star represents the α_1 and α_2 we have parameterized by using the real data.

https://doi.org/10.1371/journal.pone.0213898.g005

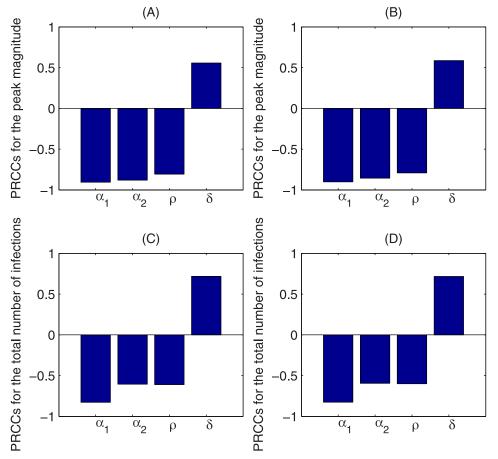


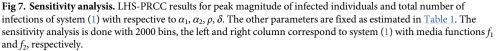


reduce the peak magnitude and total number infections. Also, decreases in the media reporting rate ρ , and increases in the media waning rate will lead to more severe outbreaks.

To access the effectiveness of enhancing the media reporting on the epidemic outbreak, we plot the variation in peak magnitude and total infections with the corresponding parameter ρ , as shown in Fig 8(A) and 8(B). It shows that increasing ρ by 4 times from baseline value (while keeping other parameters fixed) can reduce the peak magnitude from 1225 to 779 (decreased by 36.4%) for the media function f_1 or 1189 to 687 (decrease by 42.2%) for the media function f_2 , and also can reduce the number of total infections from 74391 to 66783 for f_1 or 73370 to 64102 for f_2 .

To further investigate what pattern of media report is optimal in minimizing the number of infected individuals and costs, we simulate the optimal control system (4) and obtain the optimal control. Here, we fix the parameter values as listed in Table 1 and employ the Forward-Backward Sweep method. It follows from Fig 9(A) that the optimal control is to continuously strengthen/increase the media reports at the beginning of an epidemic, when the disease starts to spread. A maximum level should then be maintained in times surrounding the peak number of infections, and then it should be slowly weakened as the infection reduces in the population. It is clear that the optimal control is similar for the two different media functions f_1 and f_2 ,





however, the optimal media reporting intensity for system (4) with media function f_2 is stronger than that for system (4) with media function f_1 (i.e., $u_2^* > u_1^*$).

Figs 9(B) and 10 show the optimal epidemic curves under the optimal reporting intensity. These figures indicate that the optimal control significantly reduces the peak magnitude and total number of infected individuals. They also show that the peak magnitude appears earlier in time for both media functions f_1 and f_2 . Comparing Fig 3 to Fig 9(B) and Fig 10(A), we see that, while simply having media reports during an epidemic can greatly reduce the severity of an epidemic, it is further mitigated under the optimal reporting intensity. Moreover, in such a scenario, a stronger media report intensity u_2^* for media function f_2 gives rise to a greater reduction in peak magnitude and the total number of infected individuals than the media report intensity u_1^* .

Discussion

It is known that media reports can play an important role in generating public awareness and promoting disease mitigation measures. Quantifying and evaluating the media impact on the control of emerging infectious diseases is quite challenging. Our study here included the intensity of media reports as a separate compartment, and a modified transmission rate that is

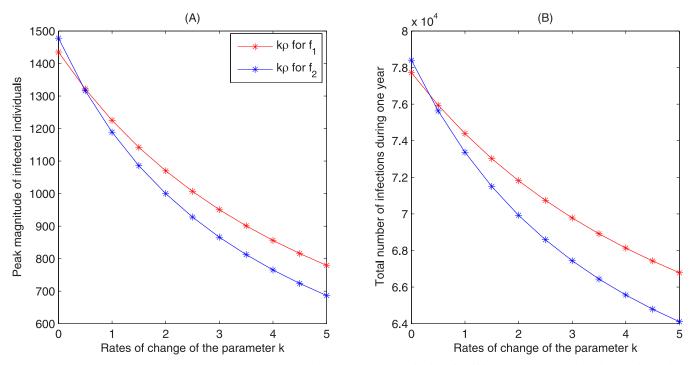


Fig 8. Plots of the peak magnitude and total infections by varying k (the rates of change of ρ). The red and blue curves with asterisk markers are the results by using function f_1 and f_2 , respectively. Each column of markers denotes that k increases 50% per time. All the other parameters are fixed as shown in Table 1.

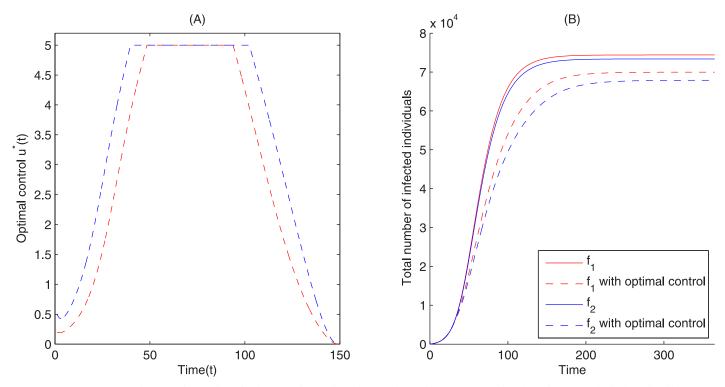
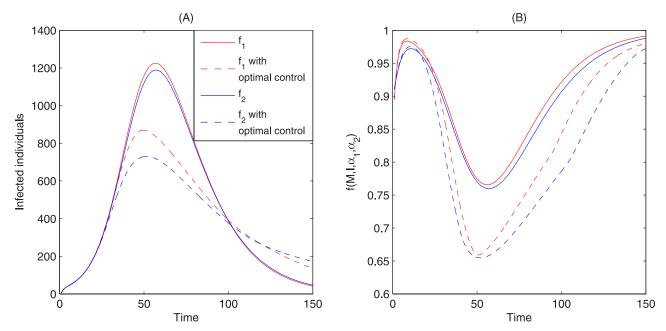
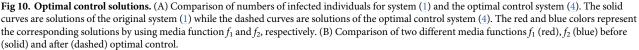


Fig 9. Optimal control. (A) The optimal control u(t) for the optimal control problem (4) obtained by using Forward-Backward sweep method. The red and blue dashed curves represent the optimal control for system (4) with different media functions f_1 and f_2 , respectively. (B) Comparison of total infections for system (1) and the optimal control system (4). A = 1, B = 5, $u_{max} = 5$, $t_0 = 0$, $t_{end} = 150$, all other parameters are shown in Table 1.

https://doi.org/10.1371/journal.pone.0213898.g009





reduced using a media function that is bounded between 0 and 1. We also considered a novel media function which depends both on the number of infected individuals *I* and the intensity of mass media *M* (where mass media reports were assumed to depend on the reporting of newly infected individuals, $\rho\sigma E$) [8].

We calculated the basic reproduction number of system (1) which is the same as that for the classical SEIR model. This result illustrates that the mass media has no effect on the basic reproduction number, which agrees with the previous studies [11, 24, 27]. We then investigated the threshold dynamics of the proposed model with a general media function effect. We theoretically investigated the optimal control problem by seeking an optimal media reporting intensity to minimize the total infected individuals and the cost of media reporting. The optimal media reporting intensity obtained here indicated that during the early stage of epidemic we should quickly enhance the media reporting intensity, keep it at the maximum level during the time period around the peak of the epidemic, and then decrease the intensity after the epidemic vanishes significantly.

By fitting our proposed model to laboratory-confirmed case data from the 8th Hospital of Xi'an over the first 19 days of the 2009 H1N1 influenza pandemic (to ensure that the data lie in the exponential growth phase of the epidemic), we estimated the unknown model parameters and the basic reproduction numbers without media impact and with two special media functions. We found that the basic reproduction number may be underestimated if the media impact is not considered. We also illustrated that the peak magnitude of the endemic would greatly decrease when the mass media function is considered, which has also been demonstrated in [26]. Sensitivity analysis indicated that the severity of the disease outbreak is sensitive to the parameters associated with the media impact (the reporting rate ρ , media waning rate δ , weight of media effect sensitive to infected individuals α_1 , and weight of the media effect sensitive to media items α_2) besides the epidemiological parameters (like transmission rate β and

the recovery rate γ). In particular, the outbreak severity is more sensitive to the weight α_1 than weight α_2 (shown in Figs 5–7), indicating that more response to the number of infected individuals will lead to greater reductions in the peak magnitude and the total number of infections.

For the particular media functions f_1 and f_2 , we observed no obvious differences in the epidemic curve (shown in Fig 3) when the optimal reporting rate was not considered, though from the point of R-square or *AIC* for the Least-Square case, the model with f_2 can fit the observed data better than the model with f_1 . However, with the optimal media reporting rate the optimal epidemic curves are quite different. In particular, the shape of the optimal control u_1^* and u_2^* are similar, but u_2^* is greater than u_1^* , causing the optimal solution of system (4) with f_2 to be less than the optimal solution of system (4) with f_1 . This means that there is optimal media reporting function such that the number of infected individuals and costs reach the minimum, which helps design an optimal news releasing patterns that mostly affect individuals' behaviour changes, and hence result in the infection significantly decline.

In previous studies, media campaigns have been characterized as a dynamic variable [21]. Media data can be collected to inform the mass media compartment [8, 25]. In our current study, we include a separate compartment for mass media reports. We will consider the incorporation of mass media data in future work.

In summary, we extend the classical SEIR model by incorporating the media as a separate compartment and through the modification of the transmission rate by a media factor associated with not only the number of infected individuals *I* but also the media items *M*. Through the inclusion of the media compartment and the modified transmission rate $\beta f(I, M)$ we can use model (1) to study the effects of *I* and *M* separately. In this study, we focus our work on understanding the media impact on the transmission of 2009 H1N1 in Shaanxi, China, and explore the efficiency of optimal control on the media reporting rate. Ultimately, we find that response to the number of infected individuals *I* will lead to greater reductions in the peak magnitude and the total number of infections. We also find that the optimal media reporting intensity should be enhanced early in the outbreak, be kept at the maximum level during the time period around the peak of the epidemic, and then be decreased after the epidemic reaches a low level to ensure a minimal level of infection in a population.

Supporting information

S1 File. Stability of equilibria, calculation of the optimal control, model fits without M. (PDF)

S2 File. Data from the 8th hospital of Xi'an. (XLSX)

S1 Fig. Data fitting for four candidate models. (EPS)

Acknowledgments

The authors were supported by the National Natural Science Foundation of China(NSFC, 11631012, 11571273), the Natural Science and Engineering Research Council of Canada (NSERC) and the York Research Chair.

Author Contributions

Funding acquisition: Yanni Xiao, Jane Marie Heffernan.

Methodology: Weike Zhou.

Supervision: Yanni Xiao, Jane Marie Heffernan.

Writing – original draft: Weike Zhou.

Writing – review & editing: Yanni Xiao, Jane Marie Heffernan.

References

- Kristiansen IS, Halvorsen PA, Gyrd-Hansen D. Influenza pandemic: perception of risk and individual precautions in a general population. Cross sectional study. BMC Public Health. 2007; 7:48. PMID: 17407563
- De Silva UC, Warachit J, Waicharoen S, Chittaganpich M. A preliminary analysis of the epidemiology of influenza A(H1N1) virus infection in Thailand from early outbreak data, June-July 2009. Euresurveilance. 2009; 14:1–3.
- **3.** Roth DZ, Henry B. Social Distancing as a Pandemic Influenza Prevention Measure. National Collaborating Centre for Infectious Diseases; 2011.
- 4. Tchuenche JM, Dube N, Bhunu CP, Bauch CT. The impact of media coverage on the transmission dynamics of human influenza. BMC Public Health. 2011; 11(1):S5. PMID: 21356134
- D'Onofrio A, Manfredi P. Information-related changes in contact patterns may trigger oscillations in the endemic prevalence of infectious diseases. Jour. Theor. Biol. 2009; 256(3):473–478. https://doi.org/10. 1016/j.jtbi.2008.10.005
- Sun C, Yang W, Arino J, Khan K. Effect of media-induced social distancing on disease transmission in a two patch setting. Math. Biosci. 2011; 230:87–95. https://doi.org/10.1016/j.mbs.2011.01.005 PMID: 21296092
- Kassa S, Ouhinou A. The impact of self-protective measures in the optimal interventions for controlling infectious diseases of human population. J. Math. Biol. 2015; 70(12):213–236. https://doi.org/10.1007/ s00285-014-0761-3 PMID: 24526258
- Collinson S, Khan K, Heffernan JM. The effects of media reports on disease spread and important public health measures. Plos ONE. 2015; 10(11):e0141423. https://doi.org/10.1371/journal.pone.0141423 PMID: 26528909
- 9. Wang AL, Xiao YN. A Filippov system describing media effects on the spread of infectious diseases. Nonlinear Anal. Hybrid Syst. 2014; 11:84–97. https://doi.org/10.1016/j.nahs.2013.06.005
- Xiao YN, Zhao TT, Tang SY. Dynamics of an infectious disease with media/psychology induced nonsmooth incidence. Math. Biosci. Eng. 2013; 10:445–461. PMID: 23458308
- Xiao YN, Tang SY, Wu JH. Media impact switching surface during an infectious disease outbreak. Sci. Rep. 2015; 5:7838. https://doi.org/10.1038/srep07838 PMID: 25592757
- Funk S, Gilad E, Watkins C, Jansen VAA. Modelling the influence of human behaviour on the spread of infectious diseases: A review. J. R. Soc. Interface. 2010; 7:1247–1256. <u>https://doi.org/10.1098/rsif.</u> 2010.0142 PMID: 20504800
- Misra AK, Sharma AA. A mathematical model for control of vector borne diseases through media compaigns. Discrete cont. Dyn-B. 2013; 18(7):1909–1927.
- Hou J, Teng ZD. Continuous and impulsive vaccination of SEIR epidemic models with saturation incidence rates. Math. Comput. Simulat. 2009; 10:3038–3054. https://doi.org/10.1016/j.matcom.2009.02. 001
- Funk S, Gilad E, Watkins C, Jansen V. The spread of awareness and its impact on epidemic outbreaks. Proc. Natl. Acad. Sci. 2009; 106(16):6872–6877. https://doi.org/10.1073/pnas.0810762106 PMID: 19332788
- Funk S, Gilad E, Jansen V. Endemic disease, awareness, and local behavioural response. J. Theor. Biol. 2010; 264(2):501–509. https://doi.org/10.1016/j.jtbi.2010.02.032 PMID: 20184901
- Kiss IZ, Cassell J, Recker M, Simon PL. The impact of information transmission on epidemic outbreaks. Math. Biosci. 2010; 225:1–10. https://doi.org/10.1016/j.mbs.2009.11.009
- Wang Q, Zhao LJ, Huang RB, Wu JH. Interaction of media and disease dynamics and its impact on emerging infection management. Discrete cont. Dyn-B. 2015; 20:215–230.
- Greenhalgh D, Rana S, Samanta S, Sardar T, Bhattacharya S, Chattopadhyay J. Awareness programs control infectious disease-Multiple delay induced mathematical model. Appl. Math. Comput. 2015; 251:539–563.

- Tchuenche JM, Bauch CT. Dynamics of an infectious disease where media coverage influences transmission. ISRN Biomath. 2012; 2012:581274. https://doi.org/10.5402/2012/581274
- Misra AK, Sharma A, Shukla JB. Modeling and analysis of effects of awareness programs by media on the spread of infectious diseases. Math. Comput. Model. 2011; 53:1221–1228. https://doi.org/10.1016/ j.mcm.2010.12.005
- Misra AK, Sharma A, Singh V. Effect of awareness programs in controlling the prevalence of an epidemic with time delay. J. Biol. Sys. 2011; 19:389–402. https://doi.org/10.1142/S0218339011004020
- Misra AK, Sharma A, Shukla JB. Stability analysis and optimal control of an epidemic model with awareness programs by media. Biosyst. 2015; 138:53–62. https://doi.org/10.1016/j.biosystems.2015.11.002
- 24. Cui JA, Sun YH, Zhu HP. The impact of media on the control of infectious diseases. J. Dynam. Diff. Eqns. 2008; 20:31–53. https://doi.org/10.1007/s10884-007-9075-0
- Yan QL, Tang SY, Gabriele S, Wu JH. Media coverage and hospital notifications: Correlation analysis and optimal media impact duration to manage a pandemic. J. Theor. Biol. 2016; 390:1–13. https://doi. org/10.1016/j.jtbi.2015.11.002
- Collinson S, Heffernan JM. Modelling the effects of media during an influenza epidemic. BMC Public Health. 2014; 14(1):376. https://doi.org/10.1186/1471-2458-14-376 PMID: 24742139
- Liu RS, Wu JH, Zhu HP. Media/psychological impact on multiple outbreaks of emerging infectious diseases. Comput. Math. Methods Med. 2007; 8:153–164. https://doi.org/10.1080/17486700701425870
- Samanta S, Rana S, Sharma A, Misra AK, Chattopadhyay J. Effect of awareness programs by media on the epidemic outbreaks: a mathematical model. Appl. Math. Comput. 2013; 219:6965–6977.
- Agaba GO, Kyrychko YN, Blyuss KB. Mathematical model for the impact of awareness on the dynamics of infectious disease. Math. Biosci. 2017; 286:22–30. https://doi.org/10.1016/j.mbs.2017.01.009 PMID: 28161305
- Song PF, Xiao YN. Global hopf bifurcation of a delayed equation describing the lag effect of media impact on the spread of infectious disease. J. Math. Biol. 2018; 76:1249–1267. <u>https://doi.org/10.1007/s00285-017-1173-y PMID: 28852830</u>
- Li Y, Cui J. The effect of constant and pulse vaccination on SIS epidemic models incorporating media coverage. Commun. Nonlin. Sci Numer. Simulat. 2009; 14:2353–2365. <u>https://doi.org/10.1016/j.cnsns.</u> 2008.06.024
- Cui JA, Tao X, Zhu HP. An SIS infection model incorporating media coverage. Rocky Mt. J. Math. 2008; 38:1323–1334. https://doi.org/10.1216/RMJ-2008-38-5-1323
- Tang SY, Xiao YN, Yuan L, Cheke RA, Wu JH. Campus quarantine (Fengxiao) for curbing emergent infectious diseases: Lessons from mitigating A/H1N1 in Xi'an, China. J. Theor. Biol. 2012; 295:47–58. https://doi.org/10.1016/j.jtbi.2011.10.035 PMID: 22079943
- Tuite AR, Greer AL, Whelan M, et al. Estimated epidemiologic parameters and morbidity associated with pandemic H1N1 influenza. Canadian Medical Association Journal. 2010; 182(2):131–136. https:// doi.org/10.1503/cmaj.091807 PMID: 19959592
- Driessche P, Watmough J. Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission. Math. Biosci. 2002; 180:29–48. https://doi.org/10.1016/S0025-5564(02)00108-6 PMID: 12387915
- Heffernan JM, Smith RJ, Wahl LM. Perspectives on the basic reproductive ratio. J. R. Soc. Interface. 2005; 2:281–293. https://doi.org/10.1098/rsif.2005.0042 PMID: 16849186
- Corless RM, Gonnet GH, et al. On the Lambert W function. Adv. Comput. Math. 1996; 5:329–359. https://doi.org/10.1007/BF02124750
- Fleming WH, Rishel RW. Deterministic and Stochastic Optimal Control. New York: Springer-Verlag.; 1975.
- Lukes DL. Differential Equations: Classical to Controlled, Mathematics inScience and Engineering. New York: Academic Press; 1982.