Journal of Biological Dynamics

ISSN: 1751-3758 (Print) 1751-3766 (Online) Journal homepage: https://www.tandfonline.com/loi/tjbd20

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To cite this article: Guihua Li & Yijing Dong (2019) Dynamic modelling of the impact of public health education on the control of emerging infectious disease, Journal of Biological Dynamics, 13:1, 502-517, DOI: 10.1080/17513758.2019.1639835

To link to this article: https://doi.org/10.1080/17513758.2019.1639835

Published online: 09 Jul 2019.

JOURNAL OF BIOLOGICAL DYNAMICS
2019, VOL. 13, NO. 1, 502–517
https://doi.org/10.1080/17513758.2019.1639835

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Dynamic modelling of the impact of public health education on the control of emerging infectious disease

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ABSTRACT

Public health education, including mass and interpersonal communication, has been recognized as an effective control of infectious disease. Compared to the well-studied mass communication by dynamic modelling, not much mathematical study has been done on the effect of interpersonal communication. Here, we build a model, to study the overall impact of mass communication and interpersonal communication on disease spread and disease control during the transmission process. By analysing the dynamic behaviour of our model, we find two threshold parameters on which the disease persistence and extinction condition depend. We further prove that the endemic equilibrium, whenever existing, is locally asymptotically stable. Its global stability is also verified. And the impact of public health education on the behaviours of the model is considered by numerical simulation. Our study confirms the value of various education activities and shows that public health education may affect the epidemic threshold.

ARTICLE HISTORY
Received 12 August 2018
Accepted 27 June 2019

KEYWORDS
Public health education; emerging infectious disease; stability; dynamic modelling

1. Introduction

Infectious diseases have always been a major public health threat to human life and health. We face the challenge of the resurgence of old infectious diseases, such as the cholera, malaria, diphtheria [2] and the emergence of increasingly new infectious diseases, such as AIDS, Ebola and SARS. Those have significant societal impacts not only through disease-induced morbidity and mortality, but also through their interference with socio-economic activities and population movement. Effective public health education is a cornerstone in the primary prevention and control of infectious disease and can reduce the social burden of the disease [11]. It is reasonable that at the beginning of the epidemic, a large number of news reports and rapid information flow to the public have a profound psychological impact, which greatly changed the behaviour of individuals, affecting the implementation of public intervention and control policies [13,20,24]. The more prophylactic knowledge the population has, the less possibility of epidemic disease spread [7]. The rational is that at the start of an epidemic, massive news coverage and fast information flow can generate profound psychological impacts on the public, and hence greatly alter individuals' behaviours and influence the implementation of public intervention and control policies [13,20,24].

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Public health education can be divided into two categories: *mass communication and interpersonal communication*. *Mass communication* is mainly done through channels, by applying audio-visual education measures, to educate the public on the various health problems, to encourage the public to take precautions and modify their unhealthy behaviours and lifestyle [6,7,10,15,22]. Television, radio and internet become the main forms of large-scale public health education because of wide coverage surface and fast transmission speed of information.

Interpersonal communication is more on interactions between individuals or specific population groups, and is more community or group focused. For example, counselling hotline, health counselling column and consultation clinic in hospital can provide face-to-face counselling service for general public [23]. According to the feedback information from targeted groups, communicators can adjust the content of public health education to make it easier to be accepted by the public promptly. This pattern of education is bidirectional transmission, which is suitable for public health education activities within a small area.

In recent years, with the impact of public health education on epidemic diseases, many mathematical models have been proposed, which focus on studying the impact of *mass communication*, by either stochastic differential equation model or a deterministic model. In these models, called compartmental models, *mass communication* is mainly reflected in two aspects: introducing some new population categories, and changing incidence function to reflect the 'psychological' effect. In the classical compartmental model, the population is divided into the susceptible, infective and recovered individuals. In references [11,15,16] and [13,20], the awareness population and the hospitalized population were introduced on the basis of earlier classification, respectively. In references [4,11,15,16,20,24,26], the cumulative density of awareness programs driven by the media was considered.

In the references on changing different incidence function to reflect the psychological effect, the main idea is that effective contacts between infective and susceptible individuals decrease at high infective levels due to the quarantine of infective individuals or the protection measures by the susceptible individuals after the information reported by the mass media. In references [3,5,6,13,14,18,19,25], the different incidence functions, generally decreasing functions as the current number of infected individuals, were chosen to describe the effect of media coverage. The main dynamical behaviour described in these literatures was given a threshold parameter for the prevalence of the disease.

Mass communication may not reach all population. For example, in remote mountainous areas, they have not been wildly introduced. Thus, *interpersonal communication* will be particularly important. *Interpersonal communication* can completely prevent the spread of the disease [8,9,12]. In references [1,8,9], every compartment is divided into unaware and aware populations. In these models, unaware susceptible population is transformed into an infectious after they contact with infected population. In fact, when unaware population is exposed to the infected population, they will become to aware one if they are not infected. Moreover, in a rapidly developing economy social,it is difficult to avoid the movement of populations. Here, we will be based on the model of literatures [1,8,9,17] and build a new compartment model that takes into account the effect of both *mass communication and interpersonal communication*, discuss how the combined communication can affect the spread of the epidemic disease.

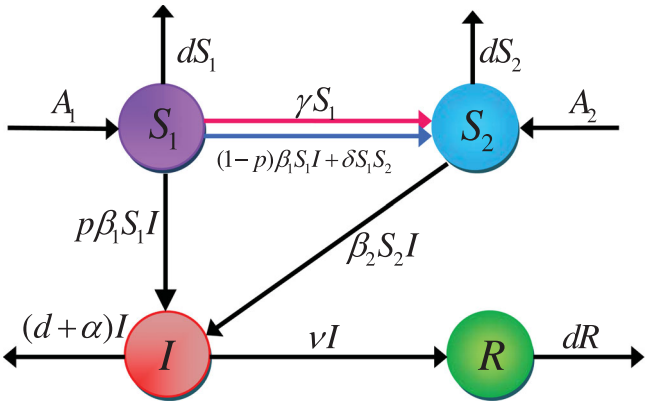

Figure 1. Schematic diagram of model.

The original idea of our model was partially motivated by a study by Yu *et al.* [27] on competing products in a market. We denote by $S(t)$, $I(t)$, $R(t)$ and $N(t)$ the numbers of the susceptible, the infective and the recovered individuals and their sum at time t, respectively, so $N(t) = S(t) + I(t) + R(t)$. We suppose that the susceptibles are divided into two classes: unaware class S_1 and aware class S_2, and $S = S_1 + S_2$. Those in class S_1 have no idea of what the disease is and how it is spreading among the population, whereas those in class S_2 have a certain understanding of the disease (such as disease pathogenesis and the way of disease transmission). The individuals in class S_2 are assumed to take the corresponding prophylaxis measures to protect themselves against infection. Therefore, the population in class S_2 may be infected, but at a lower rate than those in class S_1.

Thus, our model is based on the schematic diagram shown in Figure 1, which describes how the population changes from one state to another, how new individuals are generated and how they are removed from the system. Using the figure, it is straightforward to obtain the following evolution equations for S_1, S_2, I, and R.

$$\begin{aligned}
\frac{\mathrm{d}S_1}{\mathrm{d}t} &= A_1 - \mathrm{d}S_1 - \gamma S_1 - \delta S_1 S_2 - \beta_1 S_1 I,\\
\frac{\mathrm{d}S_2}{\mathrm{d}t} &= A_2 - \mathrm{d}S_2 + \gamma S_1 + \delta S_1 S_2 + (1-p)\beta_1 S_1 I - \beta_2 S_2 I,\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= p\beta_1 S_1 I + \beta_2 S_2 I - dI - \alpha I - \nu I,\\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \nu I - dR,
\end{aligned} \tag{1}$$

where the meaning of the parameters is shown in Table 1. We denote awareness spread rate by *mass communication* with γ; The effect of *interpersonal communication*, is described as the individual in unaware susceptible class S_1 gaining the disease information through communicating with the aware susceptible individual S_2 or the infected I, with $\delta S_1 S_2$ and $(1-p)\beta_1 S_1 I$, respectively. According to the above description, the people in aware susceptible class S_2 may be infected at a lower rate than those in unaware susceptible class S_1, i.e. $p\beta_1 > \beta_2$. The transmission rate of information is faster than the infected rate of

Table 1. The meaning of parameters in system (1).

A_1, A_2	Rate of recruitment into unaware and aware susceptible populations, respectively
d	Rate of the natural death
γ	Rate of awareness spread by mass communication
δ	Effective contact rate from aware susceptible to unaware one
β_1	Effective contact rate from infected to unaware susceptible
$p\beta_1$	Probability of disease transmission per contact by an infective
$(1-p)\beta_1$	Probability of disease awareness transmission per contact by an infective
β_2	Effective contact rate from infected to aware susceptible
α	Death rate due to the disease
ν	Recovery rate of infected

susceptible individuals, i.e. $\delta > p\beta_1 > \beta_2$. Summing up the four equations of system (1), we obtain

$$\frac{\mathrm{d}N}{\mathrm{d}t} = A_1 + A_2 - \mathrm{d}N - \alpha I.$$

One can verify that the positive cone

$$D = \left\{(S_1, S_2, I, R) | S_1 \geq 0, S_2 \geq 0, I \geq 0, R \geq 0, S_1 + S_2 + I + R \leq \frac{A_1 + A_2}{d}\right\}$$

is positive invariant.

In this paper we shall show that *mass communication and interpersonal communication* have significant effects on the control of emerging infectious disease. Before going into any detail, we simplify the model. Since the first three equations are independent of the forth one, it suffices to consider the first three equations. Thus, we restrict our attention to the following reduced model:

$$\begin{aligned}
\frac{\mathrm{d}S_1}{\mathrm{d}t} &= A_1 - \mathrm{d}S_1 - \gamma S_1 - \delta S_1 S_2 - \beta_1 S_1 I,\\
\frac{\mathrm{d}S_2}{\mathrm{d}t} &= A_2 - \mathrm{d}S_2 + \gamma S_1 + \delta S_1 S_2 + (1-p)\beta_1 S_1 I - \beta_2 S_2 I,\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= p\beta_1 S_1 I + \beta_2 S_2 I - dI - \alpha I - \nu I.
\end{aligned} \tag{2}$$

The remaining of this paper is organized as follows. In Section 2, we discuss the number and type of equilibria. Section 3 describes local stability of the equilibria, and we will present a global analysis of the model in a special case in Section 4. We conclude our paper by a brief discussion in Section 5.

2. Existence of equilibria

To study the existence of equilibria, setting the right hand side of system (2) to zero yields:

$$\begin{aligned}
&A_1 - \mathrm{d}S_1 - \gamma S_1 - \delta S_1 S_2 - \beta_1 S_1 I = 0,\\
&A_2 - \mathrm{d}S_2 + \gamma S_1 + \delta S_1 S_2 + (1-p)\beta_1 S_1 I - \beta_2 S_2 I = 0,\\
&p\beta_1 S_1 I + \beta_2 S_2 I - dI - \alpha I - \nu I = 0.
\end{aligned} \tag{3}$$

Apparently, the disease-free equilibrium always exists, which is denoted by $E_0(S_1^0, S_2^0, 0)$. For the disease free equilibrium E_0, we have

$$\begin{aligned} A_1 - \mathrm{d}S_1^0 - \gamma S_1^0 - \delta S_1^0 S_2^0 = 0, \\ A_2 - \mathrm{d}S_2^0 + \gamma S_1^0 + \delta S_1^0 S_2^0 = 0. \end{aligned} \tag{4}$$

Adding the two equations of (4), we obtain

$$S_1^0 = \frac{A_1 + A_2}{d} - S_2^0. \tag{5}$$

Substituting (5) into the first equation of (4), we deduce that S_2^0 satisfies

$$d\delta (S_2^0)^2 - (\delta(A_1 + A_2) - d(d + \gamma))S_2^0 - \gamma(A_1 + A_2) - dA_2 = 0. \tag{6}$$

Solving the above quadratic equation, we have

$$\begin{aligned} S_2^{01} &= \frac{\delta(A_1 + A_2) - d(d + \gamma) + \sqrt{\Delta}}{2d\delta} > 0, \\ S_2^{02} &= \frac{\delta(A_1 + A_2) - d(d + \gamma) - \sqrt{\Delta}}{2d\delta} < 0, \end{aligned}$$

where

$$\Delta = [-\delta(A_1 + A_2) + d(d + \gamma)]^2 + 4d\delta[\gamma(A_1 + A_2) + dA_2].$$

Using Equation (5), we can get

$$\begin{aligned} S_1^0 &= \frac{\delta(A_1 + A_2) + d(d + \gamma) - \sqrt{\Delta}}{2d\delta}, \\ S_2^0 &= \frac{\delta(A_1 + A_2) - d(d + \gamma) + \sqrt{\Delta}}{2d\delta}. \end{aligned} \tag{7}$$

It is easily proved that

$$(\delta(A_1 + A_2) + d(d + \gamma))^2 - \Delta = 4d^2\delta A_1^2 > 0,$$

thus $S_1^0 > 0$. For $E_0(S_1^0, S_2^0, 0)$, by using the formula in [21], one can get the basic reproduction number

$$R_0 = \frac{p\beta_1 S_1^0}{d + \alpha + \nu} + \frac{\beta_2 S_2^0}{d + \alpha + \nu}. \tag{8}$$

If $S_1^0 = \frac{A_1 + A_2}{d}, S_2^0 = 0$, then the basic reproduction number of system (2) for susceptible who are totally unaware of the disease at the initial time is

$$R_1 \triangleq \frac{p\beta_1(A_1 + A_2)}{d(d + \alpha + \nu)}. \tag{9}$$

If $S_1^0 = 0, S_2^0 = \frac{A_1 + A_2}{d}$, then the basic reproduction number of system (2) that all susceptible people know about diseases. at the initial time becomes

$$R_2 \triangleq \frac{\beta_2(A_1 + A_2)}{d(d + \alpha + \nu)}. \tag{10}$$

From the above assumption $p\beta_1 > \beta_2$, we can get $R_1 > R_0 > R_2$. For the endemic equilibrium $E^*(S_1^*, S_2^*, I^*)$, it satisfies

$$S_1^* = \frac{\beta_2(d+\alpha+\nu)I^* + d(d+\alpha+\nu) - \beta_2(A_1+A_2)}{d(p\beta_1-\beta_2)},$$
$$S_2^* = \frac{-p\beta_1(d+\alpha+\nu)I^* - d(d+\alpha+\nu) + p\beta_1(A_1+A_2)}{d(p\beta_1-\beta_2)}, \tag{11}$$

and I^* should be a positive root of the quadratic equation

$$\mathcal{F}(I) \triangleq \mathcal{A}I^2 + \mathcal{B}I + \mathcal{C} = 0, \tag{12}$$

where

$$\begin{aligned}
\mathcal{A} &= \beta_1\beta_2(d+\alpha+\nu)[p\delta(d+\alpha+\nu) - d(p\beta_1-\beta_2)],\\
\mathcal{B} &= d(d+\alpha+\nu)[(p\beta_1-\beta_2)(dp\beta_1(R_2-1) - \beta_2(d+\gamma))\\
&\quad - \delta(d+\alpha+\nu)(p\beta_1(R_2-1) - \beta_2(R_1-1))],\\
\mathcal{C} &= d^2(d+\alpha+\nu)\left[\delta(d+\alpha+\nu)(R_2-1)(R_1-1)\right.\\
&\quad \left. + (p\beta_1-\beta_2)\left((\gamma+d)(R_2-1) + \frac{A_1(p\beta_1-\beta_2)}{d+\alpha+\nu}\right)\right]\\
&= \frac{d^2(d+\alpha+\nu)^2}{(A_1+A_2)^2}(R_1 - h_1(R_2))(R_1 - h_2(R_2)),\\
h_1(R_2) &= (1-a_1)R_2 + a_1,\\
h_2(R_2) &= (1-a_2)R_2 + a_2,\\
a_1 &= \frac{(A_1+A_2)(\delta(A_1+A_2) + d(d+\gamma) + \sqrt{\Delta})}{2A_1d^2},\\
a_2 &= \frac{(A_1+A_2)(\delta(A_1+A_2) + d(d+\gamma) - \sqrt{\Delta})}{2A_1d^2}.
\end{aligned} \tag{13}$$

As we study the existence of equilibria in $\mathbb{R}^{3+}$, the constraint that $S_1^* > 0, S_2^* > 0$, that is

$$I_0 < I^* < I_1 \quad (R_1 > R_2 \geq 1) \text{or} 0 < I^* < I_1 \quad (R_1 > 1 > R_2)$$

where

$$I_1 = \frac{p\beta_1(A_1+A_2) - d(d+\alpha+\nu)}{p\beta_1(d+\alpha+\nu)} = \frac{d}{p\beta_1}(R_1-1),$$
$$I_0 = \frac{\beta_2(A_1+A_2) - d(d+\alpha+\nu)}{\beta_2(d+\alpha+\nu)} = \frac{d}{\beta_2}(R_2-1).$$

Furthermore,

$$\begin{aligned}
\mathcal{F}(I_0) &= d^2(p\beta_1-\beta_2)^2A_1 > 0,\\
\mathcal{F}(I_1) &= \frac{d^2(p\beta_1-\beta_2)^2}{p^2\beta_1}h(p),\\
h(p) &= A_1\beta_1p^2 - [\beta_1(A_1+A_2) + (d+\gamma)(d+\alpha+\nu)]p + d(d+\alpha+\nu),
\end{aligned}$$

Denote

$$p_1 = \frac{d(d+\alpha+\nu)}{\beta_1(A_1+A_2)}.$$

Theorem 2.1: *For the system (2),*

(1) *The disease free equilibrium E_0 always exists*;
(2) *If $R_0 < 1$, there is no endemic equilibrium*;
(3) *If $R_0 > 1$, there are the following several cases*:
   (i) *If $1 < R_2 < R_1$, there exists a unique endemic equilibrium $E^*(S_1^*, S_2^*, I^*)$*;
   (ii) *If $R_1 > 1 > R_2$, then there exists an endemic equilibrium $E^*(S_1^*, S_2^*, I^*)$ when $R_1 > h_1(R_2)$; there is no endemic equilibrium when $R_1 \le h_1(R_2)$.*

Proof: If $R_0 < 1$, which means that $R_2 < R_1 \le 1$, then $I_2, I_1 \le 0$. Thus, system (2) has no endemic equilibrium. If $R_0 > 1$, the existence of positive equilibrium needs to be discussed in different situations. When $R_1 > 1$, it means that $I_1 > 0$ and $p_1 < p < 1$. It is easy to verify

$$h(p_1) = -\frac{d(d+\alpha+\nu)^2[\gamma(A_1+A_2)+dA_2]}{\beta_1(A_1+A_2)^2} < 0,$$
$$h(1) = -\beta_1 A_2 - \gamma(d+\alpha+\nu) < 0.$$

Thus, $\mathcal{F}(I_1) < 0$, when $p_1 < p < 1$. Then, we analyse the existence of endemic equilibria from the following two cases, $R_1 > R_2 \ge 1, R_1 > 1 > R_2$.

Case 1: $R_1 > R_2 \ge 1$.

$R_2 \ge 1$ means $\beta_2 \ge \frac{d(d+\alpha+\nu)}{A_1+A_2}$. Thus $\beta_2/\beta_1 \ge p_1$. Naturally, $\mathcal{F}(I_1) < 0$ in $\beta_2/\beta_1 < p < 1$. It is easily proved that system (2) has a unique endemic equilibrium.

Case 2: $R_1 > 1 > R_2$.

In this case, p should be in $(p_1, 1)$. Thus $\mathcal{F}(I_1) < 0$. Furthermore, the existence of endemic equilibria will be determined by the sign of $\mathcal{F}(0) = \mathcal{C}$. But $\mathcal{F}(I_0)$ is always more than zero. Thus, no matter $\mathcal{A}$ is greater than zero or less than zero, system (2) has an endemic equilibrium if and only if $\mathcal{C} > 0$. The sign of $\mathcal{C}$ is determined by the sign of $(R_1 - h_1(R_2))(R_1 - h_2(R_2))$ from (13). The two straight lines $R_1 = h_1(R_2)$ and $R_1 = h_2(R_2)$ cross the point $(1, 1)$ in the plane (R_2, R_1), because $h_1(1) = h_2(1) = 1$. Furthermore, by means of the parameter expression of S_1^0 and S_2^0 in (7), $a_1 > 1, a_2 < 1$ can be verified. Naturally, from the graphics of $R_1 = h_1(R_2)$ and $R_1 = h_2(R_2)$, $h_1(R_2) > 1$ and $h_2(R_2) < 1$ is obtained when $R_2 < 1$. According to $R_1 > 1$, we have that $R_1 - h_2(R_2) > 0$ is always satisfied. Thus, the sign of $\mathcal{C}$ is determined by the sign of $R_1 - h_1(R_2)$, that is, if $R_1 > h_1(R_2)$, $\mathcal{C} > 0$, and the system (2) has a unique endemic equilibrium. Otherwise, there is no endemic equilibrium. ■

Remark 2.1: By geometric method we will illustrate the existence areas of endemic equilibria of system (2) in the (R_2, R_1) parametric plane. In Figure 2, the (R_2, R_1) parametric plane is divided into several different regions by L_1, L_2, L_3 and L_4, where $L_1 : R_1 = h_1(R_2)$, i.e. $\mathcal{C} = 0$, $L_2 : R_1 = R_2$, $L_3 : R_1 = 1$ and $L_4 : R_2 = 1$. Here, we only consider the regions with different colours above L_2, because $R_1 > R_2$ always holds throughout this paper. The

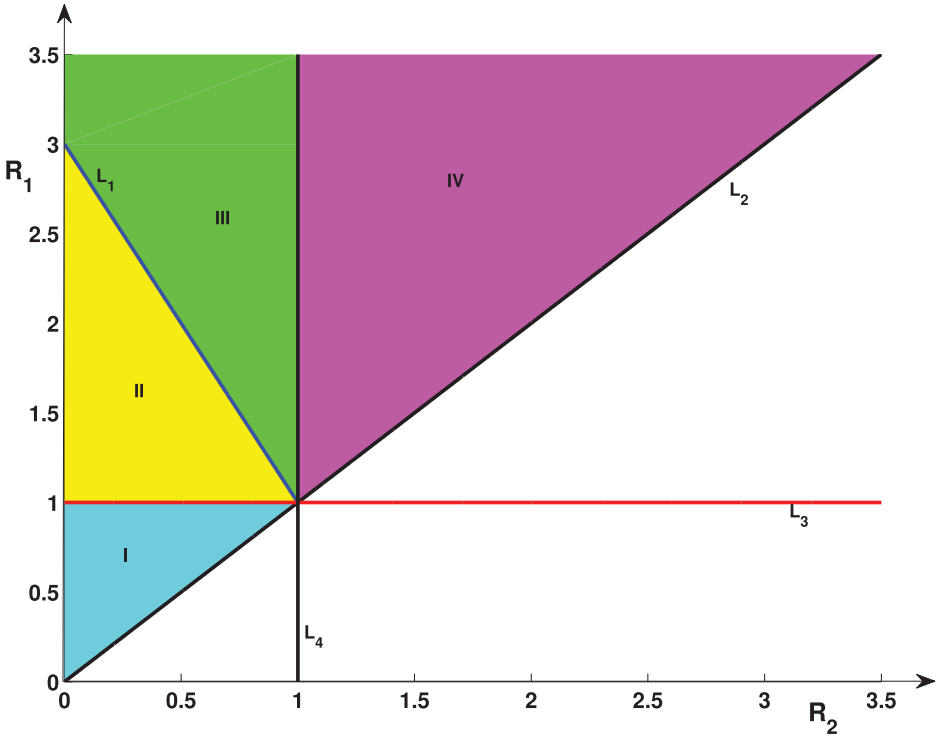

Figure 2. The distribution diagram of positive equilibria in the (R_2, R_1) parametric plane.

region I represents that $R_2 < R_1 < 1$, where system (2) has no endemic equilibrium. The region II and III denote that $R_2 < 1 < R_1$, where the region located at the lower-left side of L_1 implies $R_1 < h_1(R_2)$, that is the region II, so that there is no positive equilibrium, while the area III between the upper-right side of L_1 and L_4 has a unique endemic equilibrium. Moreover, there is a unique endemic equilibrium in the region IV, representing $1 < R_2 < R_1$.

3. Stability analysis

In this section, the local stability of the disease-free equilibrium and positive equilibrium will be studied. Firstly, computing the Jacobian matrix of system (2) at the disease-free equilibrium E_0, we obtain

$$J_0 = \begin{pmatrix} -\dfrac{A_1}{S_1^0} & -\delta S_1^0 & -\beta S_1^0 \\ \delta S_2^0 + \gamma & -\dfrac{\gamma S_1^0 + A_2}{S_2^0} & -\beta_2 S_2^0 \\ 0 & 0 & p\beta_1 S_1^0 + \beta_2 S_2^0 - (d + \alpha + \nu) \end{pmatrix}. \tag{14}$$

The eigenvalues of (14) are

$$\lambda_1 = p\beta_1 S_1^0 + \beta_2 S_2^0 - (d + \alpha + \nu) = (d + \alpha + \nu)(R_0 - 1), \tag{15}$$

and the roots of the quadratic polynomial

$$\lambda^2 + (\frac{A_1}{S_1^0} + \frac{\gamma S_1^0 + A_2}{S_2^0})\lambda + \frac{A_1(\gamma S_1^0 + A_2)}{S_1^0 S_2^0} + \delta S_1^0(\delta S_2^0 + \gamma) = 0.$$

The roots of the quadratic polynomial have negative real parts. Therefore, the disease-free equilibrium E_0 is locally asymptotically stable if and if only $R_0 < 1$.

Theorem 3.1: *For the system (2),*

(1) *The disease-free equilibrium E_0 is stable if $R_0 < 1$;*
(2) *E_0 is unstable if $R_0 > 1$.*

Remark 3.1: In order to compare the condition of the stability of the disease-free equilibrium point with the existence condition of the positive equilibrium, we analyse the relationship between the three thresholds. From (15) and $p\beta_1 > \beta_2$, one gets

$$\beta_2(S_1^0 + S_2^0) - (d + \alpha + \nu) < \lambda_1 < p\beta_1(S_1^0 + S_2^0) - (d + \alpha + \nu).$$

Furthermore, using (5) and (9), we have

$$(d + \alpha + \nu)(R_2 - 1) < \lambda_1 < (d + \alpha + \nu)(R_1 - 1).$$

It is not difficult to find that $\lambda_1 < 0$ if $R_2 < R_1 < 1$, and $\lambda_1 > 0$ if $1 < R_2 < R_1$. When $R_1 > 1 > R_2$, substituting with S_1^0, S_2^0 of (5) in (15) yields

$$\lambda_1 = \frac{b_2 A_1 d^2(d + \alpha + \nu)}{\delta(A_1 + A_2)^2}(R_1 - h_1(R_2)).$$

It is obvious that $\lambda_1 < 0$ implies $R_1 < h_1(R_2)$. It is found that *the existence conditions of positive equilibrium is equivalent to the stability conditions of disease-free equilibrium for system* (2).

Theorem 3.2: *The endemic equilibrium E^* of system (2) is locally asymptotically stable as long as it exists.*

Proof: Evaluating the jacobian matrix of (2) at E^* gives

$$J_{E^*} = \begin{pmatrix} -\frac{A_1}{S_1^*} & -\delta S_1^* & -\beta_1 S_1^* \\ \delta S_2^* + \gamma + (1-p)\beta_1 I^* & -\frac{\gamma S_1^* + A_2 + (1-p)\beta_1 S_1^* I^*}{S_2^*} & (1-p)\beta_1 S_1^* - \beta_2 S_2^* \\ p\beta_1 I^* & \beta_2 I^* & 0 \end{pmatrix}. \quad (16)$$

Its characteristic equation is

$$\lambda^3 + m_1\lambda^2 + m_2\lambda + m_3 = 0,$$

where

$$m_1 = \frac{A_1}{S_1^*} + \frac{\gamma S_1^* + A_2 + (1-p)\beta_1 S_1^* I^*}{S_2^*} > 0,$$

$$m_2 = \beta_2 I^*(\beta_2 S_2^* - (1-p)\beta_1 S_1^*) + \delta S_1^*(\delta S_2^* + \gamma + (1-p)\beta_1 I^*) + p\beta_1^2 S_1^* I^* + \frac{A_1(\gamma S_1^* + A_2 + (1-p)\beta_1 S_1^* I^*)}{S_1^* S_2^*},$$

$$m_3 = \gamma\beta_1\beta_2 S_1^* I^* + (1-p)\beta_1 S_1^* I^*(\delta p\beta_1 S_1^* + \beta_1\beta_2 I^* + \delta\beta_2 S_2^*) + \frac{p\beta_1^2 S_1^* I^*(\gamma S_1^* + A_2 + (1-p)\beta_1 S_1^* I^*)}{S_2^*} + \frac{A_1\beta_2 I^*}{S_1^*}(\beta_2 S_2^* - (1-p)\beta_1 S_1^*).$$

Substituting (11) into m_3, one can verify that the sign of m_3 depends on the derivative of $F'(I)$ at $I = I^*$:

$$m_3 = -I^* F'(I^*).$$

Then, from (12), it's easy to find that $m_3 > 0$. Define $H = \left|\begin{smallmatrix} m_1 & 1 \\ m_3 & m_2 \end{smallmatrix}\right| = m_1 m_2 - m_3$. Substituting

$$\frac{A_1}{S_1^*} = d + \gamma + \delta S_2^* + \beta_1 I^*$$

into H to obtain that

$$H = p\beta_1^2 S_1^* I^*(d + \delta S_2^* + \beta_1 I^*) + \delta S_1^*(\gamma + \delta S_2^*)\left(d + \gamma + \delta S_2^* + \beta_1 I^* + \frac{\gamma S_1^* + A_2}{S_2^*}\right) + \gamma\beta_1 S_1^* I^*(p\beta_1 - \beta_2) + \left[(\delta - \beta_2)\left(\delta S_2^* + \beta_1 I^* + \frac{\gamma S_1^* + A_2 + (1-p)\beta_1 S_1^* I^*}{S_2^*}\right) + \delta S_1^*(\delta - p\beta_1) + \delta(d+\gamma) + \frac{\gamma\delta S_1^*}{S_2^*}\right](1-p)\beta_1 S_1^* I^* + \frac{\gamma S_1^* + A_2 + (1-p)\beta_1 S_1^* I^*}{S_2^*} \times \left[\beta_2^2 S_2^* I^* + \frac{A_1}{S_1^*}\left(\frac{A_1}{S_1^*} + \frac{\gamma S_1^* + A_2 + (1-p)\beta_1 S_1^* I^*}{S_2^*}\right)\right].$$

Considering the biological implications of parameters, we can obtain that the relationship of parameter values is $\delta > p\beta_1 > \beta_2$, then $H > 0$. Therefore, we can conclude that the real part of all eigenvalues of J_{E^*} are all negative followed by Hurwitz theorem, i.e. the endemic equilibrium E^* is locally asymptotically stable. ■

4. Global stability

In this section, we consider the global stability of equilibrium of system (2). Assume that the following assumptions hold for system (2).

(H) For $0 \le S_i \le S_i^0$, $i=1,2$, $I>0$,

$$\begin{aligned}
&\big(S_1 - S_1^*\big)\big(\frac{S_1^*}{S_1} - \frac{S_2^*}{S_2}\big) \le 0, && \big(S_1S_2 - S_1^*S_2^*\big)\big(\frac{S_1^*}{S_1} - \frac{S_2^*}{S_2}\big) \le 0,\\
&\big(S_1I - S_1^*I^*\big)\big(\frac{S_1^*}{S_1} - \frac{S_2^*}{S_2}\big) \le 0, && \big(S_1I - S_1^*I^*\big)\big(\frac{S_2^*}{S_2} - \frac{I^*}{I}\big) \le 0,\\
&\big(\frac{S_2^*}{S_2} - \frac{I^*}{I}\big)\big(\frac{S_1^*}{S_1} - \frac{S_2^*}{S_2}\big) \le 0.
\end{aligned}$$

Theorem 4.1: *Suppose that assumption (H) holds. Then, as long as the positive equilibrium exists, it must be globally asymptotically stable.*

***Proof*:** For system (2), we consider the following Lyapunov function:

$$L = S_1 - S_1^* - S_1^* \ln \frac{S_1}{S_1^*} + S_2 - S_2^* - S_2^* \ln \frac{S_2}{S_2^*} + I - I^* - I^* \ln \frac{I}{I^*}. \tag{17}$$

Differentiating L along solutions of (2) and using equilibrium equations

$$\begin{aligned}
&p\beta_1 S_1^* = (d+\alpha+\nu) - \beta_2 S_2^*,\\
&A_1 = \mathrm{d}S_1^* + \gamma S_1^* + \delta S_1^* S_2^* + \beta_1 S_1^* I^*,\\
&A_2 = \mathrm{d}S_2^* - \gamma S_1^* - \delta S_1^* S_2^* - \beta_1 S_1^* I^* + (d+\alpha+\nu) I^*,\\
&A_1 + A_2 = \mathrm{d}S_1^* + \mathrm{d}S_2^* + (d+\alpha+\nu) I^*.
\end{aligned}$$

to simplify, we obtain

$$\begin{aligned}
L'|_{(2)} &= S_1' - \frac{S_1^*}{S_1} S_1' + S_2' - \frac{S_2^*}{S_2} S_2' + I' - \frac{I^*}{I} I'\\
&= 2\mathrm{d}S_1^* - \mathrm{d}S_1 - d\frac{S_1^{*2}}{S_1} + 2\mathrm{d}S_2^* - \mathrm{d}S_2 - d\frac{S_2^{*2}}{S_2} + 2(d+\alpha+\nu)I^* - (d+\alpha+\nu)I\\
&\quad - (d+\alpha+\nu)I^*\frac{S_2^*}{S_2} - \gamma\frac{S_1^{*2}}{S_1} + \gamma S_1^*\frac{S_2^*}{S_2} + \gamma S_1^* - \gamma S_1\frac{S_2^*}{S_2} - \delta S_2^*\frac{S_1^{*2}}{S_1} + \delta S_1^*\frac{S_2^{*2}}{S_2}\\
&\quad + \delta S_1^* S_2 - \delta S_1 S_2^* - \beta_1 I^*\frac{S_1^{*2}}{S_1} + \beta_1 S_1^* I^*\frac{S_2^*}{S_2} + \beta_1 S_1^* I - (1-p)\beta_1 S_1 I\frac{S_2^*}{S_2}\\
&\quad - p\beta_1 S_1 I^* + \beta_2 S_2^* I - \beta_2 S_2 I^*\\
&= \mathrm{d}S_1^*\left(2 - \frac{S_1}{S_1^*} - \frac{S_1^*}{S_1}\right) + \mathrm{d}S_2^*\left(2 - \frac{S_2}{S_2^*} - \frac{S_2^*}{S_2}\right) + (d+\alpha+\nu)I^*\left(2 - \frac{I}{I^*} - \frac{I^*}{I}\right)\\
&\quad + (d+\alpha+\nu)\frac{1}{S_1^*}(S_1I - S_1^*I^*)\left(\frac{S_2^*}{S_2} - \frac{I^*}{I}\right) + \delta(S_1S_2 - S_1^*S_2^*)\left(\frac{S_1^*}{S_1} - \frac{S_2^*}{S_2}\right)\\
&\quad + \gamma(S_1 - S_1^*)\left(\frac{S_1^*}{S_1} - \frac{S_2^*}{S_2}\right) + \beta_1(S_1I - S_1^*I^*)\left(\frac{S_1^*}{S_1} - \frac{S_2^*}{S_2}\right)\\
&\quad + \beta_2 I\frac{S_1}{S_1^*}\frac{S_2^2}{S_2^*}\left(\frac{S_2^*}{S_2} - \frac{I^*}{I}\right)\left(\frac{S_1^*}{S_1} - \frac{S_2^*}{S_2}\right).
\end{aligned} \tag{18}$$

By assumption (H) and

$$2-\frac{S_1}{S_1^*}-\frac{S_1^*}{S_1}\leq 0,$$
$$2-\frac{S_2}{S_2^*}-\frac{S_2^*}{S_2}\leq 0,$$
$$2-\frac{I}{I^*}-\frac{I^*}{I}\leq 0,$$

with equality holding if and only if $S_1=S_1^*, S_2=S_2^*, I=I^*$, we obtain $L'\leq 0$. Therefore, the only compact invariant subset in the set $\{L'=0\}$ is the singleton $\{E^*\}$. By LaSalle's invariance principle, E^* is globally asymptotocally stable in feasible region D if and only if E^* exists. ■

5. Numerical simulation and discussion

In system (1), the basic reproduction number

$$R_0=\frac{p\beta_1 S_1^0}{d+\alpha+\nu}+\frac{\beta_2 S_2^0}{d+\alpha+\nu}.$$

If $p\beta_1=\beta_2$, the basic reproduction number becomes

$$R_0=R_1=R_2=\frac{\beta_2(A_1+A_2)}{d(d+\alpha+\nu)},$$

In other words, when the incidence rate of aware and unaware individuals is the same value, the basic reproduction number will not be affected by information during an endemic. In fact, the incidence rate of aware and unaware individuals should be different and the latter is greater than the former.

If $p\beta_1\neq\beta_2$, from Equation (6) and the basic reproduction number R_0, we get R_0 is a implicit function on parameters (γ,δ),

$$\begin{aligned}G(R_0,\gamma,\delta)&=d^2\delta(d+\alpha+\nu)^2R_0^2-d(d+\alpha+\nu)[(A_1+A_2)(p\beta_1+\beta_2)\delta\\&\quad+d(p\beta_1-\beta_2)(d+\gamma)]R_0+p\beta_1\beta_2\delta(A_1+A_2)^2\\&\quad+d(p\beta_1-\beta_2)[d(p\beta_1A_1+\beta_2A_2)+\beta_2\gamma(A_1+A_2)]\\&=0.\end{aligned}$$

To study the relation of parameters δ, γ and the basic reproduction number R_0, we take parameters $A_1=10$, $A_2=2$, $d=0.6$, $\beta_1=0.75$, $p=0.4$, $\beta_2=0.5$, $\alpha=0.2$, $\nu=0.8$ and plot the surface $G(R_0,\delta,\gamma)=0$. From Figure 3, we can observe that R_0 will reduce with parameter γ increasing, which means that *mass communication* can reduce the threshold for disease invasion; But R_0 will firstly increase then decrease with parameter δ, which means *interpersonal communication* can raise the threshold for disease invasion. This explains that information can be distorted and can cause panic when transmitted between individuals in the early stages of disease spread. When people really understand the disease,

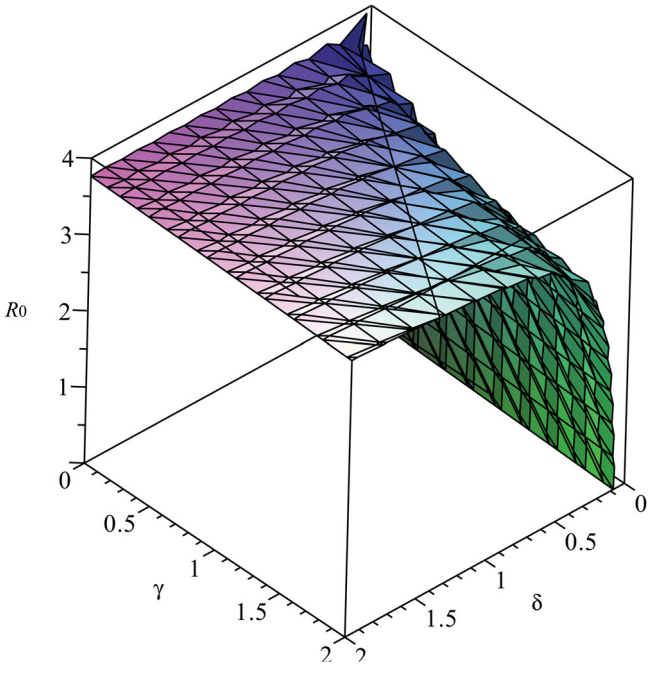

Figure 3. Graph of the surface $G(R_0, \delta, \gamma) = 0$. Here, $A_1 = 10, A_2 = 2, d = 0.6, \beta_1 = 0.75, p = 0.4, \beta_2 = 0.5, \alpha = 0.2, \nu = 0.8$.

interpersonal communication will contribute to decrease the threshold for disease invasion. This result is consistent with the conclusions in the literature [9].

To summarize our results, we have discovered local and global stability results which are similar to those of the classic model. But we achieved two distinct threshold parameters here, R_1 and R_2. $R_1 > R_2$. An equilibrium exists where the disease persists if $R_2 > 1$. The disease will die out if $R_1 < 1$. On the other hand if $R_1 > 1 > R_2$ then the fraction of infected individuals tends to be a non-zero constant value. R_1 can be considered as the expected number of secondary cases produced by a single infectious individual entering a susceptible population with none disease awareness at equilibrium. R_2 has a similar interpretation.

Although $R_1 > 1$, if awareness of the disease of susceptible individuals reaches a certain level, the disease can be extinct. Whether the disease persists will be determined by the sign of $R_1 - h_1(R_2)$ when $R_1 > 1 > R_2$. $R_2 = \frac{\beta_2(A_1+A_2)}{d(d+\alpha+\nu)}$ is proportional to β_2 (the infective rate of S_2). Increasing the intensity of public health education results in a decline of the infective rate β_2. Furthermore, $h_1(R_2) = a_1R_2 + b_1$, b_1 is a increasing function with respect to δ, γ, respectively. If we take δ, γ as the different values and other parameters are fixed, we find that when the value of δ or γ becomes larger, the existence region of endemic equilibrium becomes smaller from Figure 4.

We can conclude that *mass communication* and *interpersonal communication* have an positive influence on the control of emerging infectious disease. The effect of *mass communication* corresponds to the parameters γ and β_2 in the model (1). The effect of *interpersonal communication* is characterized by the parameter δ in our model. From

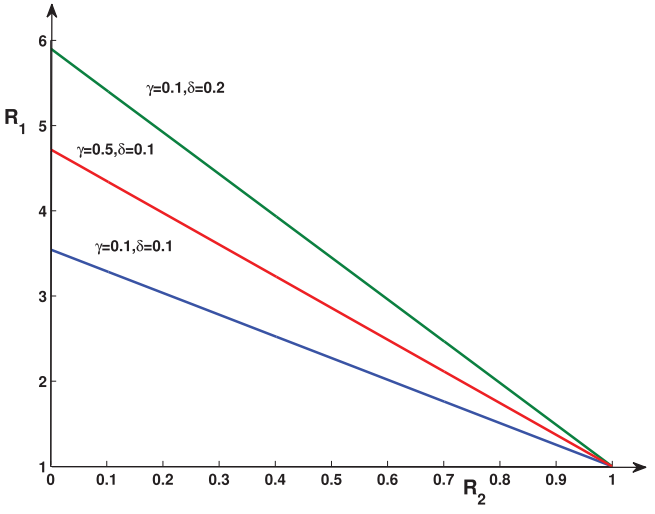

Figure 4. The effect of public health education parameter by $\mathcal{C} = 0$, δ, γ, on the existence of equilibrium, with the following parameter values: $A_1 = 4, A_2 = 2, p = 0.4, d = 0.6, \alpha = 0.2, \nu = 0.8$. The values of δ and γ are different for each line.

these analysis, it can be shown that when an emerging or chronic infectious diseases is onset, *mass communication* and *interpersonal communication* will be very effective for the controlling of infectious diseases. Thus it is crucial to increase in disease awareness and communication between individuals.

In addition, in remote mountainous areas, TV, Internet and other technology products and services have been limited. This will lead to the limitations of dissemination of information. Coupling with many unwholesome living habits, many infectious diseases are likely to spread rapidly whenever appearing. Therefore it is important to promote different types of education activities on the basis of geographical conditions. For example, distributing the free cell phones to local residents to make sure that every user can receive relevant information through text messages. Combining with the local indigenous culture to write jingles, organizing the general public to attend the lectures in their villages and to watch healthy programs at regular basis. Meanwhile, issuing leaflets, disease protection manuals and basic protective articles like gloves and respirators to each household to combine the public health education with people's daily life directly or indirectly.

Acknowledgments

Thank the anonymous reviewers for their valuable comments.

Disclosure statement

No potential conflict of interest was reported by the authors.

Funding

This work is supported by the National Science Foundation of China [11801340], Research Project Supported by the Scientific Activities of Selected Returned Overseas Professionals in Shanxi

Province, and is completed while the author is visiting Rutgers University under the funding of a Shanxi Bairen Project whose PI is Prof. Zheng-Chao Han.

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