Mathematical Modeling and Analysis of an Epidemic Model with Quarantine, Latent and Media Coverage

Bilal Boulfoul¹, Adlen Kerboua¹ and Xueyong Zhou^{2,†}

Abstract Epidemic models are very important in today's analysis of diseases. In this paper, we propose and analyze an epidemic model incorporating quarantine, latent, media coverage and time delay. We analyze the local stability of either the disease-free and endemic equilibrium in terms of the basic reproduction number \mathcal{R}_0 as a threshold parameter. We prove that if $\mathcal{R}_0 < 1$, the time delay in media coverage can not affect the stability of the diseasefree equilibrium and if $\mathcal{R}_0 > 1$, the model has at least one positive endemic equilibrium, the stability will be affected by the time delay and some conditions for Hopf bifurcation around infected equilibrium to occur are obtained by using the time delay as a bifurcation parameter. We illustrate our results by some numerical simulations such that we show that a proper application of quarantine plays a critical role in the clearance of the disease, and therefore a direct contact between people plays a critical role in the transmission of the disease.

Keywords Epidemic model, Stability, Hopf bifurcation, Delay.

MSC(2010) 92B05.

1. Introduction

For a long time, infectious and epidemics have been a great challenge to mankind, and people have tried every means to prevent and control them. Therefore, the analysis of the dynamics of diseases has always been a hot topic in people's research. At the same time, the frequent occurrence of HIV (see [34,38–41]), tuberculosis (TB) (see [19,20,29]), swine flu (see [26,47,49,56]), Avian flu (see [15,24,27,33]), Ebola (see [6,16,23,59]), human influenza (see [4,27,58]), Zika virus (see [2,3,10,11]), severe acute respiratory syndrome (SARS) (see [5,7,14]) and COVID-19 (see [12, 30–32, 50, 52]) in recent years has made people more aware of the importance of studying the prevention and transmission mechanism of diseases.

Many scholars rely on the transmission mechanism and impact of infectious and epidemics diseases factors from the perspective of reality, establishing a reasonable

[†]the corresponding author.

Email address: b.boulfoul@univ-skikda.dz; bilalboulfoul@yahoo.fr (B. Boulfoul), a.kerboua@univ-skikda.dz; kerboua_adlen@yahoo.fr (A. Kerboua), x-ueyongzhou@xynu.edu.cn (X. Zhou)

¹Department of Petrochemical and Process Engineering, Faculty of Technology, University of 20 August 1955-Skikda, B. P. 26, Skikda, 21000, Algeria

 $^{^2 {\}rm School}$ of Mathematics and Statistics, Xinyang Normal University, Xinyang, Henan 464000, China

infectious disease model, and through the specific analysis of each parameter in the model, the specific measures should be taken so as to theoretically provide a strong basis for the prevention and control of the occurrence and spread of disease. In addition, people's sense of self-protection has a positive effect on the prevention and control of diseases.

In many parts of the world, the mass media plays an irreplaceable role of changing public health-related behaviour. For example, in the early stage of a disease outbreak, the government and the Centers for Disease Control (CDC) can let people know the harm of the disease and related prevention measures through various media in a timely manner so as to minimize the chance of catching disease and achieve the purpose of curbing the epidemic. Therefore, it is particularly important to consider the infectious disease models incorporating media impact. Media reports can make the behavior of susceptible populations dependent on a continuous change in the number of cases, which is reflected by a continuous differentiable infection rate function. In [44], Liu et al. introduced an EIH epidemic model with media effects. The function of impact factor is $f(E, I, H) = e^{-a_1 E - a_1 I - a_3 H}$, where E, I, H are the exposed, infectious and hospitalized individuals, 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 rationality of the impact factor in the model was tested by simulating the outbreak of SARS in the greater Toronto area in 2003. In [43], Liu et al. constructed a novel saturation disease rate function $\beta(I) = \beta_1 - \frac{\beta_2 I}{m+I}$ to describe the intrinsic property of the continuous change in the number of cases reported by media in public behavior, which reduces the population exposure rate to a certain limited level, where β_1 is the contact rate before media alert, and β_2 is the maximal reduced effective contact rate due to mass media alert in the presence of infective individuals. The term $\frac{\beta_2 I}{m+I}$ measures the effect of reduction of the contact rate when infectious individuals are reported in the media (see [19, 42–46, 58, 61]). The function $g(I) = \frac{I}{m+I}$ is continuous bounded function which takes into account disease saturation or psychological effects (see [13,58,61]). Because the coverage report cannot prevent disease from spreading completely we have $\beta_1 \geq \beta_2 > 0$. The half saturation constant m > 0 reflects the impact of media coverage on the contact transmission ([19, 42–46, 58, 61]). They propose and analyze a mathematical model of tuberculosis (TB) transmission considering social awareness effects during an epidemic. Das et al. [19] proposed and analyzed a mathematical model of tuberculosis (TB) transmission considering social awareness effects during an epidemic. To quantify the effect of media awareness in disease transmission rate, they updated the transmission coefficient β_1 both in susceptible and exposed class by $\beta^a(I) = \beta_1 - \frac{\beta_2 I}{m+I}$ and $\beta^b(I) = \beta_1 - \frac{\beta_3 I}{m+I}$ respectively. Here, $\beta_1 > \beta_2, \beta_3$. In general, there are two approaches to account the effect of media awareness: (i) updating the disease transmission rate to accumulate the significant fall in transmission due to preventive measures (see [19, 43, 45, 46, 58]) and (ii) by incorporating a mass media compartment to represent the public interaction with mass media ([17, 36, 60]).

On the other hand, the delays are in the media coverage. Media coverage of an disease outbreak can be seen as following two major routes [5, 57, 61]. The first route is when the media report directly to the public about the facts, and the second has public health authorities that use the media or the Internet to inform about an outbreak. For the second route, the number of infections and the number of suspected infections reported by media today are often the statistical result of yesterday or the day before. Therefore, the effects of media coverage on the transmission dynamics can be modified as follows $\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}$.

Since no specific antiviral agent is available for treatment of the COVID-19 epidemic, and there is no vaccine. A metropolitan-wide quarantine of Wuhan and nearby cities was introduced on 23-24 January, 2020 [12]. Several airports and railway stations have started temperature screening to identify people with fevers [12]. All public transportation was suspended in Wuhan from 10 a.m., 23 January. After 23 January, strong government measures in all parts of China such as isolation, quarantine and public closings strongly undermined the transmission of new cases. In general, quarantine is one of the necessary measures taken by governments to affect the transmission of other diseases. We give some examples related to the COVID-19 pandemic as the model has the quarantine class (see [12,31,52]).

Motivated by the above factors, we propose an epidemic model incorporated with quarantine, latent and media coverage as follows:

$$\begin{cases} \frac{dS}{dt} = A - \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) SI - dS, \\ \frac{dE}{dt} = (1-c) \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) SI - \kappa \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) EI - dE, \\ \frac{dI}{dt} = c \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) SI + \kappa \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) EI \\ - (\varepsilon + \gamma_1 + \mu + d) I, \\ \frac{dQ}{dt} = \varepsilon I - \gamma_2 Q - dQ, \\ \frac{dR}{dt} = \gamma_1 I + \gamma_2 Q - dR, \end{cases}$$

$$(1.1)$$

where S(t), E(t), I(t), Q(t) and R(t) are the number of the susceptible, the exposed, the infected, the quarantined and the recovered at any time respectively. Thus, the total population size at time t is denoted by N(t) equals to S(t) + E(t) + I(t) + Q(t) + R(t).

The meaning of the remaining parameters used in the model is explained as follows:

- A is the recruitment rate of susceptible population;
- *d* is the natural mortality rate of the population;
- c is the portion developing active disease directly after the first infection;
- κ is the level of exogenous re-infection;
- μ is the constant death rate which is related to disease;
- ε is the quarantine rate for patients;
- γ_1 is the per capita recovery rate without quarantine;
- γ_2 represent the cure rate (the per capita recovery rate wit quarantine).

Since the first three equations of system (1.1) are independent of the last two equations of (1.1), the dynamics of system (1.1) is determined by the following subsystem:

$$\begin{cases} \frac{dS}{dt} = A - \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) SI - dS, \\ \frac{dE}{dt} = (1-c) \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) SI - \kappa \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) EI - dE, \\ \frac{dI}{dt} = c \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) SI + \kappa \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) EI \\ - (\varepsilon + \gamma_1 + \mu + d) I. \end{cases}$$
(1.2)

For $\tau > 0$, we denote by $X = C([-\tau, 0], \mathbb{R}^3)$ the Banach space of continuous functions from $[-\tau, 0]$ to \mathbb{R}^3 equipped with the supremum norm. The nonnegative cone of X is defined as $X^+ = \{\varphi \in X : \varphi(\theta) \ge 0, \ \theta \in [-\tau, 0]\}$. By the standard theory of functional differential equations (for example, see Hale and Verduyn Lunel [25], Kuang [35]). For any $\varphi = (\phi_1, \phi_2, \phi_3) \in X^+$, model (1.2) has a unique solution. From the view of mathematical biology, we consider (1.2) with the initial conditions in X_0^+ , where

$$X_0^+ = \{\varphi = (\phi_1, \phi_2, \phi_3) \in X^+ : \varphi(0) > 0\}.$$

That is,

$$S(\theta) = \phi_1(\theta), E(\theta) = \phi_2(\theta), I(\theta) = \phi_3(\theta),$$

$$\phi_i(\theta) \ge 0, \theta \in [-\tau, 0], \phi_i(0) > 0, i = 1, 2, 3.$$
(1.3)

Now, we prove positivity of solution of (1.2) with the initial condition (1.3).

Theorem 1.1. Let (S, E, I) be any solution of system (1.2) with the initial condition (1.3). Then, all S(t), E(t) and I(t) are non-negative for all t > 0, and they are ultimately bounded.

Proof. Let (S, E, I) be a solution of system (1.2) with initial condition (1.3). First, we prove that S(t) > 0, E(t) > 0 and I(t) > 0 for all t > 0. From the first equation of system (1.2), we have

$$\frac{dS(t)}{dt} \ge -\left(\left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right)I(t) + d\right)S(t).$$

Integrating both sides of the precedent inequality on (0, t), we see that

$$S(t) \ge S(0)e^{-\int_0^t \left(\left(\beta_1 - \beta_2 \frac{I(s-\tau)}{m+I(s-\tau)}\right)I(s) + d\right)ds}.$$

Then, from the initial condition (1.3), S(t) > 0 for all t > 0. From the third equation of system (1.2), we obtain

$$\frac{dI(t)}{dt} = \left(c\left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right)S(t) + \kappa\left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right)E(t) - (\varepsilon + \gamma_1 + \mu + d)\right)I(t),$$

which gives after integration on (0, t):

$$I(t) = I(0)e^{\int_0^t \left(c\left(\beta_1 - \beta_2 \frac{I(s-\tau)}{m+I(s-\tau)}\right)S(s) + \kappa\left(\beta_1 - \beta_2 \frac{I(s-\tau)}{m+I(s-\tau)}\right)E(s) - (\varepsilon+\gamma_1 + \mu + d)\right)ds}.$$

From the initial condition (1.3), we have I(t) > 0, for all t > 0. The second equation of (1.2) and since S(t) > 0 and I(t) > 0 for all t > 0 lead to

$$\frac{dE}{dt} \ge -\left(\kappa \left(\beta_1 - \beta_2 \frac{I(t-\tau)}{m+I(t-\tau)}\right) I(t) + d\right) E(t),$$

and we have E(0) > 0, which implies

$$E(t) \ge E(0)e^{-\int_0^t \left(\kappa \left(\beta_1 + \beta_2 \frac{I(s-\tau)}{m+I(s-\tau)}\right)I(s) - d\right)ds} > 0, \quad \forall t > 0.$$

Now, we show the boundedness of the solution of system (1.2). Denote M = S + E + I. Then, from system (1.2), we have

$$\frac{dM}{dt} = A - dS - dE - (\varepsilon + \gamma_1 + \mu + d) I$$
$$\leq A - dM.$$

By using the comparison principle, we obtain

$$\underset{t \to +\infty}{\lim \sup} M(t) \le \frac{A}{d}$$

Define

$$G = \left\{ (S, E, I) \in X^+ : S + E + I \le \frac{A}{d} \right\}.$$

By Theorem 1.1, we conclude that for all $(\phi_1, \phi_2, \phi_3) \in X^+$:

$$d(\Phi_t(\phi_1, \phi_2, \phi_3), G) \to 0, t \to +\infty,$$

where Φ_t is the semiflow of system (1.2). Then, the semiflow Φ_t is dissipative, and the solutions of system (1.2) are ultimately bounded.

The rest of the paper is organized as follows: In Section 2, we calculate the reproduction number and determine the number of possible endemic equilibrium by using the Descartes Rule of Signs. In Section 3, we prove that if the basic reproduction number R_0 is less than 1, the disease-free equilibrium is locally stable. We discuss the existence of Hopf bifurcation around the endemic equilibrium under certain conditions in terms of delay. In Section 4, we illustrate our results by some numerical simulations. The paper ends with a discussion.

2. Equilibria and basic reproduction number R_0

In this section, the number of equilibria of system (1.2) will be presented. To find equilibria of (1.2), we solve the algebraic system:

$$A - \left(\beta_1 - \beta_2 \frac{I}{m+I}\right) SI - dS = 0,$$

$$(1 - c) \left(\beta_1 - \beta_2 \frac{I}{m+I}\right) SI - \kappa \left(\beta_1 - \beta_2 \frac{I}{m+I}\right) EI - dE = 0,$$

$$\left(c \left(\beta_1 - \beta_2 \frac{I}{m+I}\right) S + \kappa \left(\beta_1 - \beta_2 \frac{I}{m+I}\right) E - (\varepsilon + \gamma_1 + \mu + d)\right) I = 0.$$
(2.1)

If I = 0, then from the first and the second equations of (2.1), we get $S = \frac{A}{d}$ and E = 0. System (1.2) admits the disease free equilibrium $P_0(S^0, 0, 0)$ with $S^0 = \frac{A}{d}$.

Now, we define the basic reproduction number \mathcal{R}_0 of model (1.2). The basic reproduction number R_0 is one of the most crucial quantities in infectious diseases, as \mathcal{R}_0 measures how contagious a disease is. System (1.2) always has a disease-free equilibrium $P_0(S^0, 0, 0)$. Inspired by the method in Driessche and Watmough [22], we define matrices F and V as:

$$F = \begin{pmatrix} 0 \ (1-c)\beta_1 S^0 \\ 0 \ c\beta_1 S^0 \end{pmatrix}, V = \begin{pmatrix} d & 0 \\ 0 \ \varepsilon + \gamma_1 + \mu + d \end{pmatrix}.$$

The basic reproduction number, \mathcal{R}_0 , is the spectral radius of the next generation matrix FV^{-1} , where

$$V^{-1} = \begin{pmatrix} \frac{1}{d} & 0\\ 0 & \frac{1}{\varepsilon + \gamma_1 + \mu + d} \end{pmatrix}, FV^{-1} = \begin{pmatrix} 0 & \frac{(1-\varepsilon)\beta_1 S^0}{\varepsilon + \gamma_1 + \mu + d}\\ 0 & \frac{c\beta_1 S^0}{\varepsilon + \gamma_1 + \mu + d} \end{pmatrix}.$$

Therefore,

$$\mathcal{R}_0 = \rho\left(FV^{-1}\right) = \frac{c\beta_1 S^0}{\varepsilon + \gamma_1 + \mu + d} = \frac{Ac\beta_1}{d(\varepsilon + \gamma_1 + \mu + d)},\tag{2.2}$$

where ρ represents the spectral radius.

Before investigating the stability dynamics of the system (1.2), it is instructive to determine the number of equilibrium points of the system (2.1). To do so, let $P^*(S^*, E^*, I^*)$ be any arbitrary equilibrium of (1.2). To find conditions for the existence of an equilibrium for which the infection is endemic in the population (i.e., at least one of E^*, I^* is positive).

Now, assume that $I^* \neq 0$, from the first equation of (2.1), we have

$$S^* = \frac{A}{\left(\beta_1 - \beta_2 \frac{I^*}{m + I^*}\right)I^* + d}.$$
 (2.3)

From the third equation of (2.1) and (2.3), we get

$$E^* = \frac{\varepsilon + \gamma_1 + \mu + d}{\kappa \left(\beta_1 - \beta_2 \frac{I^*}{m + I^*}\right)} - \frac{Ac}{\kappa \left(\left(\beta_1 - \beta_2 \frac{I^*}{m + I^*}\right)I^* + d\right)}.$$
 (2.4)

By replacing (2.3) and (2.4) in the second equation of (2.1), after some straightforward calculation, we show that

$$C_0 I^{*4} + C_1 I^{*3} + C_2 I^{*2} + C_3 I^* + C_4 = 0, (2.5)$$

where

$$\begin{split} C_0 &= \kappa \left(\varepsilon + \gamma_1 + \mu + d \right) \left(\beta_1 - \beta_2 \right)^2, \\ C_1 &= \left(\beta_1 - \beta_2 \right) \left[\left(\varepsilon + \gamma_1 + \mu + d \right) \left(2\beta_1 m \kappa + \kappa d + d \right) - A \kappa \left(\beta_1 - \beta_2 \right) \right], \\ C_2 &= \left(\varepsilon + \gamma_1 + \mu + d \right) \left[\beta_1^2 m^2 \kappa + \kappa d \left(2\beta_1 m - \beta_2 m \right) + d \left(2\beta_1 m - \beta_2 m + d \right) \right] \end{split}$$

Cases	C_0	C_1	C_2	C_3	C_4	\mathcal{R}_0	Number of sign	Number of possible
							changes	positive real roots
1	+	+	+	+	+	$\mathcal{R}_0 < 1$	0	0
	+	+	+	+	_	$\mathcal{R}_0 > 1$	1	1
2	+	_	_	_	+	$\mathcal{R}_0 < 1$	2	0, 2
	+	_	_	_	_	$\mathcal{R}_0 > 1$	1	1
3	+	+	_	_	+	$\mathcal{R}_0 < 1$	2	0, 2
	+	+	_	_	_	$\mathcal{R}_0 > 1$	1	1
4	+	_	+	_	+	$\mathcal{R}_0 < 1$	4	0, 2, 4
	+	_	+	_	_	$\mathcal{R}_0 > 1$	3	1, 3
5	+	_	_	+	+	$\mathcal{R}_0 < 1$	2	0, 2
_	+	_	_	+	_	$\mathcal{R}_0 > 1$	3	1, 3
6	+	+	+	_	+	$\mathcal{R}_0 < 1$	2	0, 2
	+	+	+	_	_	$\mathcal{R}_0 > 1$	1	1
7	+	+	_	+	+	$\mathcal{R}_0 < 1$	2	0, 2
	+	+	_	+	_	$\mathcal{R}_0 > 1$	3	1, 3
8	+	_	+	+	+	$\mathcal{R}_0 < 1$	2	0, 2
	+	_	+	+	_	$\mathcal{R}_0 > 1$	3	1, 3

Table 1. Number of possible positive real roots of $g(I^*)$ for $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$

$$-A (\beta_1 - \beta_2) (2\kappa\beta_1 m + cd), \qquad (2.6)$$

$$C_3 = (\varepsilon + \gamma_1 + \mu + d) [\beta_1 m^2 \kappa d + d (\beta_1 m^2 + 2dm)]$$

$$-A\kappa\beta_1^2 m^2 - Acd (2\beta_1 m - \beta_2 m),$$

$$C_4 = -d^2 m^2 (\varepsilon + \gamma_1 + \mu + d) (\mathcal{R}_0 - 1).$$

From (2.6), it is easy to see that $C_0 > 0$ (since all the model parameters are non-negative). Further, $C_4 < 0$, if $\mathcal{R}_0 > 1$ ($C_4 > 0$, if $\mathcal{R}_0 < 1$). Thus, the number of possible positive real roots the polynomial (2.5) depends on the signs of C_1 , C_2 and C_3 . We can use the Descartes Rule of Signs on the quartic $g(I^*) =$ $C_0I^{*4} + C_1I^{*3} + C_2I^{*2} + C_3I^* + C_4$ given in (2.5) to find out how many positive real roots. The various possibilities for the roots of $g(I^*)$ are tabulated in Table 1.

The following results (Theorem 2.1 and Lemma 2.1) follow from the various possibilities enumerated in Table 1.

Theorem 2.1. The system (1.2):

(i) has a unique endemic equilibrium, if $\mathcal{R}_0 > 1$, and whenever Cases 1, 2, 3 and 6 are satisfied;

- (ii) could have more than one endemic equilibria, if $\mathcal{R}_0 > 1$, and Cases 4, 5, 7 and 8 are satisfied;
- (iii) could have two or more endemic equilibria, if $\mathcal{R}_0 < 1$, and Cases 2-8 are satisfied.

Lemma 2.1. The system (1.2) has at least one endemic equilibrium whenever $\mathcal{R}_0 > 1$, and could have zero, two, or four endemic equilibria whenever $\mathcal{R}_0 < 1$.

3. Local stability of equilibria and Hopf bifurcation analysis

The characteristic equation of system (1.2) at disease-free equilibrium P_0 is given by

$$\begin{pmatrix} \lambda + d & 0 & \beta_1 S_0 \\ 0 & \lambda + d & -(1-c)\beta_1 S_0 \\ 0 & 0 & \lambda + (\varepsilon + \gamma_1 + \mu + d) - c\beta_1 S_0 \end{pmatrix} = 0.$$
 (3.1)

Theorem 3.1. (i) If $\mathcal{R}_0 < 1$, the disease-free equilibrium P_0 of the system (1.2) is asymptotically stable for all $\tau \ge 0$.

(ii) If $\mathcal{R}_0 > 1$, the disease-free equilibrium P_0 of the system (1.2) is unstable.

Proof. At the disease-free equilibrium $P_0(S_0, 0, 0)$, the characteristic equation (3.1) takes the form:

$$(\lambda+d)\left(\lambda+d\right)\left(\lambda+\left(\varepsilon+\gamma_1+\mu+d\right)\left(1-\frac{c\beta_1S_0}{\varepsilon+\gamma_1+\mu+d}\right)\right)=0.$$

Then, $\lambda_1 = \lambda_2 = -d < 0$ and since $\mathcal{R}_0 = \frac{c\beta_1 S_0}{\varepsilon + \gamma_1 + \mu + d} < 1$, $\lambda_3 = (\varepsilon + \gamma_1 + \mu + d)$ $(\mathcal{R}_0 - 1) < 0$. Thus, P_0 is locally asymptotically stable, when $\mathcal{R}_0 < 1$ for all $\tau \ge 0$. On the other hand, $\lambda_3 > 0$, when $\mathcal{R}_0 > 1$. Hence, P_0 is unstable, when $\mathcal{R}_0 > 1$.

Next, we turn our attention to the endemic equilibrium $P^*(S^*, E^*, I^*)$. The characteristic equation at this equilibrium has the form:

$$\begin{pmatrix} \lambda - a_1 & 0 & -a_2 - a_9 e^{-\lambda\tau} \\ -a_3 & \lambda - a_4 & -a_5 - a_{10} e^{-\lambda\tau} \\ -a_6 & -a_7 & \lambda - a_8 - a_{11} e^{-\lambda\tau} \end{pmatrix} = 0,$$
(3.2)

where

$$a_{1} = -d - \eta_{1}I^{*},$$

$$a_{2} = -\eta_{1}S^{*},$$

$$a_{3} = (1 - c)\eta_{1}I^{*},$$

$$a_{4} = -d - \kappa\eta_{1}I^{*},$$

$$a_{5} = (1 - c)\eta_{1}S^{*} - \kappa\eta_{1}E^{*},$$

$$a_{6} = c\eta_{1}I^{*},$$

$$a_{7} = \kappa\eta_{1}I^{*},$$

$$a_{8} = -(\varepsilon + \gamma_{1} + \mu + d) + c\eta_{1}S^{*} + \kappa\eta_{1}E^{*},$$

$$a_{9} = \eta_{2}S^{*},$$

$$a_{10} = -(1 - c)\eta_{2}S^{*} + \kappa\eta_{2}E^{*},$$

$$a_{11} = -c\eta_{2}S^{*} - \kappa\eta_{2}E^{*},$$

$$\eta_{1} = \beta_{1} - \frac{\beta_{2}I^{*}}{m + I^{*}},$$

$$\eta_{2} = \frac{\beta_{2}mI^{*}}{(m + I^{*})^{2}}.$$

After some calculations, the characteristic equation (3.2) takes the following form:

$$\lambda^{3} + b_{1}\lambda^{2} + b_{2}\lambda + b_{3} + (b_{4}\lambda^{2} + b_{5}\lambda + b_{6})e^{-\lambda\tau} = 0, \qquad (3.3)$$

where

$$\begin{split} b_1 &= -\left(a_1 + a_4 + a_8\right), \\ b_2 &= a_1 a_4 + a_8 (a_1 + a_4) - a_2 a_6 - a_5 a_7, \\ b_3 &= a_1 a_5 a_7 + a_2 a_4 a_6 - a_1 a_4 a_8 - a_2 a_3 a_7, \\ b_4 &= -a_{11}, \\ b_5 &= a_{11} (a_1 + a_4) - a_7 a_{10} - a_6 a_9, \\ b_6 &= a_1 a_7 a_{10} + a_4 a_6 a_9 - a_1 a_4 a_{11} - a_3 a_7 a_9. \end{split}$$

When $\tau = 0$, the characteristic equation (3.3) at the endemic equilibrium P^* takes the following form:

$$\lambda^{3} + \hat{b}_{1}\lambda^{2} + \hat{b}_{2}\lambda + \hat{b}_{3} = 0, \qquad (3.4)$$

where

$$\hat{b}_1 = b_1 + b_4,$$

 $\hat{b}_2 = b_2 + b_5,$
 $\hat{b}_3 = b_3 + b_6.$

It is hard to verify the signs of the coefficients of (3.4). Routh-Hurwitz criterion (see [9]) implies that when $\tau = 0$, all roots of (3.4) have negative real parts, if and only if the following conditions are satisfied:

$$\begin{split} \Delta_1 &= \hat{b}_1 > 0, \\ \Delta_2 &= \hat{b}_1 \hat{b}_2 - \hat{b}_3 > 0, \\ \Delta_3 &= \hat{b}_3 \Delta_2 > 0. \end{split} \tag{H}_1$$

Theorem 3.2. Assume $\mathcal{R}_0 > 1$. When $\tau = 0$, the endemic equilibrium P^* is locally asymptotically stable, if and only if (H_1) holds.

Condition (H₁) will be used to determine the stability of P^* at $\tau = 0$. In the following parts, we will let τ be bifurcation parameter and investigate Hopf bifurcation for system (3.3) and the stability of P^* by adapting the method in [51, 61]. For P^* to become unstable, characteristic roots have to cross the imaginary axis to

the right when τ increases. Let $\lambda = i\omega$ ($\omega > 0$) be a purely imaginary root of (3.3). Substituting it into (3.3) and separating the real and imaginary parts, we obtain

$$b_1\omega^2 - b_3 = (b_6 - b_4\omega^2)\cos(\omega\tau) + b_5\omega\sin(\omega\tau), -\omega^3 + b_2\omega = (b_6 - b_4\omega^2)\sin(\omega\tau) - b_5\omega\cos(\omega\tau).$$
(3.5)

Squaring and adding both equations of (3.5), it follows that

$$\omega^6 + p\omega^4 + q\omega^2 + r = 0, (3.6)$$

where

$$p = b_1^2 - 2b_2 - b_4^2,$$

$$q = b_2^2 + 2b_4b_6 - 2b_1b_3 - b_5^2,$$

$$r = b_3^2 - b_6^2.$$

Let $z = \omega^2$, and then (3.6) becomes

$$F(z) := z^3 + pz^2 + qz + r = 0.$$
(3.7)

We give some hypotheses on the parameters, under which (3.7) has at least one positive root in the following lemma. To proceed, we further denote

$$\Delta = p^2 - 3q, \, z_1^* = \frac{-p + \sqrt{\Delta}}{3}, \, z_2^* = \frac{-p - \sqrt{\Delta}}{3}.$$
(3.8)

Lemma 3.1. For the polynomial equation (3.7), we have the following results.

- (i) If r < 0, then (3.7) has at least one positive root.
- (ii) If $r \ge 0$, we have the following two cases:
 - 1) when $\Delta < 0$ or $\Delta = 0$, (3.7) has no positive root.
 - 2) when $\Delta > 0$, (3.7) has positive roots, if and only if $z_1^* > 0$, and $F(z_1^*) \leq 0$.

Proof. (i) If r < 0, it is clear that F(0) = r < 0 and $\lim_{z \to +\infty} F(z) = +\infty$. (3.7) has at least one positive root by the intermediate value theorem.

(ii) Assume that $r \ge 0$, and we have from (3.7):

$$F'(z) = 3z^2 + 2pz + q,$$

for which the discriminant is Δ given by (3.8). Then, we have the following two cases:

1) If $\Delta < 0$ or $\Delta = 0$, then the function F(z) is increasing on $[0, +\infty)$ and since $F(0) = r \ge 0$. Thus, if $r \ge 0$ and $\Delta \le 0$, (3.7) has no positive real root.

2) When $\Delta > 0$, F'(z) has two real roots z_1^* and z_2^* given by (3.8). On the other hand, we know that $F''(z_1^*) = 2\sqrt{\Delta} > 0$ and $F''(z_2^*) = -2\sqrt{\Delta} < 0$. That is, z_1^* and z_2^* are the local minimum and the local maximum of F(z) respectively. Therefore, if $r \ge 0$ and $\Delta > 0$, (3.7) has positive real roots, if and only if $z_1^* > 0$, and $F(z_1^*) \le 0$.

Without the loss of generality, we assume that (3.7) has three positive roots z_k (k = 1, 2, 3) with $z_k > 0$. Then, by the relation of $z = \omega^2$, (3.6) has three positive roots:

$$\omega_1 = \sqrt{z_1}, \ \omega_2 = \sqrt{z_2}, \ \omega_3 = \sqrt{z_3}.$$
 (3.9)

From the equalities in (3.5), we obtain

$$\begin{aligned}
\cos(\omega\tau) &= \frac{b_5\omega(\omega^3 - b_2\omega) - (b_1\omega^2 - b_3)(b_4\omega^2 - b_6)}{(b_4\omega^2 - b_6)^2 + b_5^2\omega^2} = G_c(\omega), \\
\sin(\omega\tau) &= \frac{b_5\omega(b_1\omega^2 - b_3) + (\omega^3 - b_2\omega)(b_4\omega^2 - b_6)}{(b_4\omega^2 - b_6)^2 + b_5^2\omega^2} = G_s(\omega).
\end{aligned}$$
(3.10)

Then, for the imaginary root $\lambda = i\omega$ of (3.3), we have from (3.10) the following expression for delay τ :

$$\tau_k^{(j)} = \begin{cases} \frac{\arccos(G_c(\omega_k)) + 2\pi j}{\omega_k}, & G_s(\omega) \ge 0, \\ \frac{2\pi - \arccos(G_c(\omega_k)) + 2\pi j}{\omega_k}, & G_s(\omega) < 0, \end{cases}$$
(3.11)

where j = 0, 1, 2, ... and k = 1, 2, 3. Thus, $\pm i\omega_k$ is a pair of purely imaginary roots of (3.3) with $\tau = \tau_k^{(j)}$. Assuming

$$\tau_0 = \tau_{k_0}^{(0)} = \min_{k \in \{1,2,3\}} \left\{ \tau_k^{(0)} \right\} \text{ and } \omega_0 = \omega_{k_0}, \tag{3.12}$$

i.e., τ_0 is the minimum value associated with the imaginary solution $i\omega_0$ of the characteristic equation (3.3).

From Theorem 3.2, if $\mathcal{R}_0 > 1$, the endemic equilibrium P^* is locally asymptotically stable, when $\tau = 0$. Now, we now consider the following exponential polynomial:

$$\begin{split} P(\lambda, e^{-\lambda\tau_1}, \dots e^{-\lambda\tau_m}) = &\lambda^n + p_1^{(0)}\lambda^{n-1} + \dots + p_{n-1}^{(0)}\lambda + p_n^{(0)} \\ &+ \left(p_1^{(1)}\lambda^{n-1} + \dots + p_{n-1}^{(1)}\lambda + p_n^{(1)}\right)e^{-\lambda\tau_1} \\ &+ \dots + \left(p_1^{(m)}\lambda^{n-1} + \dots + p_{n-1}^{(m)}\lambda + p_n^{(m)}\right)e^{-\lambda\tau_m}, \end{split}$$

where $\tau_i \ge 0$ (i = 1, 2, ..., m) and $p_j^{(i)}$ (i = 0, 1, ..., m; j = 1, 2, ..., n) are constants. We need the following result in Ruan and Wei [51] to analyze (3.3).

Lemma 3.2 (Corollary 2.4, [51]). As $(\tau_1, \tau_2, ..., \tau_m)$ vary, the sum of the order of the zeros of $P(\lambda, e^{-\lambda\tau_1}, ..., e^{-\lambda\tau_m})$ in the open right half plane can change only if a zero appears on or crosses the imaginary axis.

By Lemmas 3.1 and 3.2, we obtain the following proposition.

Proposition 3.1. For the third degree transcendental equation (3.3), we have:

- (i) If $r \ge 0$ and either $\Delta < 0$ or $\Delta = 0$, then all roots of (3.3) have negative real parts for all $\tau \ge 0$.
- (ii) If either r < 0 or $r \ge 0$, $\Delta > 0$, $z_1^* > 0$ and $F(z_1^*) \le 0$, then all roots of (3.3) have negative real parts for $\tau \in [0, \tau_0)$.

Let

$$\lambda(\tau) = \alpha(\tau) + i\omega(\tau)$$

be the root of (3.3) near $\tau = \tau_k^{(j)}$ satisfying $\alpha(\tau_k^{(j)}) = 0$ and $\omega(\tau_k^{(j)}) = \omega_k$. We have the following transversality condition.

Proposition 3.2. Assume (H_1) holds and $F'(\omega_k^2) \neq 0$, when $\tau_k^{(j)} > 0$ (correspondingly ω_k for some k = 1, 2, 3). Then, the characteristic equation (3.3) admits a pair of simple conjugate pure imaginary roots $\lambda = i\omega_k$ and $\lambda = -i\omega_k$, which crosses the imaginary axis from left to right if $\delta > 0$ and crosses the imaginary axis from right to left if $\delta < 0$, where

$$\delta = sign\left\{ \left. \frac{d(Re\lambda)}{d\tau} \right|_{\tau = \tau_k^{(j)}} \right\} = sign\left\{ F'(\omega_k^2) \right\}.$$

Proof. First, by differentiating the equation (3.3) with respect to delay τ , we get

$$\left[3\lambda^2 + 2b_1\lambda + b_2 + (2b_4\lambda + b_5)e^{-\lambda\tau} - \tau (b_4\lambda^2 + b_5\lambda + b_6)e^{-\lambda\tau} \right] \frac{d\lambda}{d\tau}$$

= $\lambda (b_4\lambda^2 + b_5\lambda + b_6)e^{-\lambda\tau},$

which gives

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{3\lambda^2 + 2b_1\lambda + b_2}{\lambda(b_4\lambda^2 + b_5\lambda + b_6)e^{-\lambda\tau}} + \frac{2b_4\lambda + b_5}{\lambda(b_4\lambda^2 + b_5\lambda + b_6)} - \frac{\tau}{\lambda}.$$
 (3.13)

From (3.3), we have

$$e^{-\lambda\tau} = -\frac{\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3}{b_4\lambda^2 + b_5\lambda + b_6}.$$
 (3.14)

By replacing (3.14) in the equation (3.13), we have

$$\left(\frac{d\lambda}{d\tau}\right)^{-1} = \frac{3\lambda^2 + 2b_1\lambda + b_2}{-\lambda(\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3)} + \frac{2b_4\lambda + b_5}{\lambda(b_4\lambda^2 + b_5\lambda + b_6)} - \frac{\tau}{\lambda}$$

Evaluating $\left(\frac{d\lambda}{d\tau}\right)^{-1}$ at $\tau = \tau_k^{(j)}$ (i.e., $\lambda(\tau_k^{(j)}) = i\omega_k$) and taking the real part, we obtain

$$\begin{aligned} Re\left(\frac{d\lambda}{d\tau}\right)^{-1} \bigg|_{\tau=\tau_{k}^{(j)}} = & Re\left[\frac{3\lambda^{2}+2b_{1}\lambda+b_{2}}{-\lambda(\lambda^{3}+b_{1}\lambda^{2}+b_{2}\lambda+b_{3})} + \frac{2b_{4}\lambda+b_{5}}{\lambda(b_{4}\lambda^{2}+b_{5}\lambda+b_{6})} - \frac{\tau}{\lambda}\right]_{\lambda=i\omega_{k}} \\ = & Re\left[\frac{2b_{1}\omega_{k}^{2}+i\left(3\omega_{k}^{3}-b_{2}\omega_{k}\right)}{\omega_{k}^{2}(b_{1}\omega_{k}^{2}-b_{3}+i\left(\omega_{k}^{3}-b_{2}\omega_{k}\right))} + \frac{2b_{4}\omega_{k}^{2}-ib_{5}\omega_{k}}{\omega_{k}^{2}(b_{6}-b_{4}\omega_{k}^{2}+ib_{5}\omega_{k})}\right] \\ = & \frac{2b_{1}^{2}\omega_{k}^{4}-2b_{1}b_{3}\omega_{k}^{2}+3\omega_{k}^{6}-3b_{2}\omega_{k}^{4}-b_{2}\omega_{k}^{4}+b_{2}^{2}\omega_{k}^{2}}{\omega_{k}^{2}((b_{1}\omega_{k}^{2}-b_{3})^{2}+(\omega_{k}^{3}-b_{2}\omega_{k})^{2})} \\ + & \frac{2b_{4}b_{6}\omega_{k}^{2}-2b_{4}^{2}\omega_{k}^{4}-b_{5}^{2}\omega_{k}^{2}}{\omega_{k}^{2}((b_{6}-b_{4}\omega_{k}^{2})^{2}+b_{5}^{2}\omega_{k}^{2})}. \end{aligned}$$

From (3.5), we have

$$(b_1\omega_k^2 - b_3)^2 + (\omega_k^3 - b_2\omega_k)^2 = (b_6 - b_4\omega_k^2)^2 + b_5^2\omega_k^2.$$

Thus,

$$Re\left(\frac{d\lambda}{d\tau}\right)^{-1}\Big|_{\tau=\tau_k^{(j)}} = \frac{\omega_k^2 \left[3\omega_k^4 + 2\left(b_1^2 - 2b_2 - b_4^2\right)\omega_k^2 + \left(b_2^2 + 2b_4b_6 - 2b_1b_3 - b_5^2\right)\right]}{\omega_k^2((b_6 - b_4\omega_k^2)^2 + b_5^2\omega_k^2)}$$
$$= \frac{3\omega_k^4 + 2\left(b_1^2 - 2b_2 - b_4^2\right)\omega_k^2 + \left(b_2^2 + 2b_4b_6 - 2b_1b_3 - b_5^2\right)}{\left(b_6 - b_4\omega_k^2\right)^2 + b_5^2\omega_k^2}.$$

Since $z_k = \omega_k^2$ and $F'(z_k) \neq 0$, then

$$Re\left.\left(\frac{d\lambda}{d\tau}\right)^{-1}\right|_{\tau=\tau_{k}^{(j)}} = \frac{F'(\omega_{k}^{2})}{\left(b_{6} - b_{4}\omega_{k}^{2}\right)^{2} + b_{5}^{2}\omega_{k}^{2}} \neq 0.$$

Meanwhile, noticing the fact

$$\operatorname{sign}\left\{ \left. \frac{d(Re\lambda)}{d\tau} \right|_{\tau=\tau_k^{(j)}} \right\} = \operatorname{sign}\left\{ Re\left(\frac{d\lambda(\tau_k^{(j)})}{d\tau} \right)^{-1} \right\}.$$

Therefore,

$$\operatorname{sign}\left\{ \left. \frac{d(Re\lambda)}{d\tau} \right|_{\tau=\tau_k^{(j)}} \right\} = \operatorname{sign}\left\{ \frac{F'(\omega_k^2)}{\left(b_6 - b_4 \omega_k^2\right)^2 + b_5^2 \omega_k^2} \right\} \\ = \operatorname{sign}\left\{ F'(\omega_k^2) \right\}.$$

From Proposition 3.1 and 3.2, we have the following theorem.

Theorem 3.3. Assume that $\mathcal{R}_0 > 1$, (H_1) holds, and $\tau_k^{(j)}$, ω_0 , and τ_0 defined by (3.11) and (3.12), respectively. Then,

- (i) if $r \ge 0$ and either $\Delta < 0$ or $\Delta = 0$, the endemic equilibrium P^* of system (1.2) is locally asymptotically stable for all $\tau \ge 0$.
- (ii) if either r < 0 or $r \ge 0$, $\Delta > 0$, $z_1^* > 0$ and $F(z_1^*) \le 0$, the endemic equilibrium P^* of system (1.2) is locally asymptotically stable for $\tau \in [0, \tau_0)$.
- (iii) if the conditions of (ii) are satisfied and $F'(z_k) \neq 0$, system (1.2) exhibits Hopf bifurcation at the endemic equilibrium P^* , when τ pass through $\tau = \tau_k^{(j)}$.

4. Numerical simulation

In this section, several illustrative numerical examples are presented to confirm the theoretical results and to examine the dynamical behavior of system (1.2). Graphs have been plotted for S, E and I for various values of τ . The parameters are given in Table 2. Then, we associate systems (1.2) with the following initial conditions:

 $S(0) = 385, \quad E(0) = 15 \quad \text{and} \quad I(0) = 0.6$

In Figure 1, we have plotted the contour of the basic reproduction number \mathcal{R}_0 in two parameter planes, Figure 1(a) illustrates the contour plot of the basic

reproduction number \mathcal{R}_0 with respect to the disease transmission rate β_1 varying from 0 to 0.01 and the quarantine rate ε varying from 0 to 1. Figure 1(b) illustrates the contour plot of the basic reproduction number \mathcal{R}_0 with respect to β_1 varying from 0 to 0.02 and the fraction of susceptible population acquiring direct route to active disease c varying from 0 to 1. We can see that from Figure 1(a), the decrease of ε and increase β_1 can significantly increase the value of \mathcal{R}_0 , which verifies that proper application of quarantine plays a critical role in the clearance of the disease. Whilst, from Figure 1(b), the increase of β_1 and c can significantly increase the value of \mathcal{R}_0 , which confirms that direct contact between people plays a critical role in the transmission of the disease. We can see also that in the blue region (or equivalently $\mathcal{R}_0 < 1$), and Theorem 3.1 implies that the disease-free equilibrium $P_0(333.3333333, 0, 0)$ is locally asymptotically stable. Hence, the infection is cleared and in the region when $\mathcal{R}_0 > 1$, and by Theorem 3.3, the disease equilibrium P^* is locally stable for some conditions on parameters of model (1.2).

Parameter	Meaning	Value						
A	Recruitment rate of susceptible	20						
β_1	Disease transmission rate in the absence of media alerts	0.022						
β_2	Maximal reduction rate in effective contact (transmission)	0.021						
m	Half saturation constant	0.9						
d	Natural death rate of the population	0.06						
с	Fraction of susceptible population acquiring direct route	0.45						
	to active disease							
κ	Exogenous re-infection level	0.3						
ε	Quarantine rate for patients	0.8						
γ_1	Per capita recovery rate without quarantine	0.05						
μ	Disease induced death rate	0.03						

 Table 2. Parameters and values used for numerical simulation of model (1.2)

In Figure 2, we have plotted the basic reproduction number, \mathcal{R}_0 , in (a) as a function of the parameters $c \in [0,1]$, $\beta_1 \in [0,0.025]$, and (b) as a function of $\varepsilon \in [0,1]$, $\beta_1 \in [0,0.025]$. Figure 2(a) confirms the results of Figure 1(a), and Figure 2(b) confirms the results of Figure 1(b).

For the model without delay, as predicted by Theorem 3.2 plots of Figure 3 show that the endemic equilibrium $P^*(265.2204889, 34.78227204, 2.127483342)$ is locally asymptotically stable, when $\mathcal{R}_0 > 1$. That is, if $\tau = 0, S, E$ and I converge to their equilibrium, when $\mathcal{R}_0 = 3.510638298 > 1$.

Next, we use a same set of parameter values as those in Table 2, but we vary the value of τ , so that the conditions (ii) or (iii) of Theorem 3.3 are satisfied. Figure 4 shows that the endemic equilibrium P^* is stable for $\tau = 0.8$, when $\mathcal{R}_0 =$ 3.510638298 > 1. Figure 5 shows, as predicted by Theorem 3.3 (iii), that if $\tau = 0.95$, the endemic equilibrium E^* is unstable and the system (1.2) has a periodic orbit, when $\mathcal{R}_0 = 3.510638298 > 1$. Plots of Figure 5 are the oscillations of S, E and I. From Figures 3-5, we can find that for $\mathcal{R}_0 < 1$, the disease is expected to stop spreading; the disease can spread and become epidemic if \mathcal{R}_0 is greater than 1 [28]. From Figure 6, we can find that the role of media impact is positive, and it can reduce the number of infectious individuals.



Figure 1. Contour plot of R_0 in $\beta_1 - \varepsilon$ and $\beta_1 - c$ parameter plane



Figure 2. Surface plot of the basic reproduction number, R_0 , in (a) for combination of the parameters $\varepsilon \in [0, 1]$, $\beta_1 \in [0, 0.025]$ and (b) for combination of the parameters $c \in [0, 1]$, $\beta_1 \in [0, 0.025]$



Figure 3. When $\tau = 0$, the endemic equilibrium E^* is locally stable.



Figure 4. When $\tau = 2$, the endemic equilibrium E^* is locally stable.



Figure 5. When $\tau = 3$, the endemic equilibrium E^* is unstable and periodic solutions exist.



Figure 6. When $\tau = 2$, the endemic equilibrium E^* is stable for $\beta_2 = 0.017$, $\beta_2 = 0.019$, $\beta_2 = 0.021$.

5. Discussion

In this paper, we investigate a differential equation model of disease transmission including quarantine susceptible, latent and media coverage with time delay. In this analysis, the basic reproduction number \mathcal{R}_0 is identified and established as

a threshold parameter. Numerical simulations show that the decrease of ε and increase β_1 can significantly increase the value of \mathcal{R}_0 , which verifies that proper application of quarantine plays a critical role in the clearance of the disease. Also, we have proved that \mathcal{R}_0 is a increasing function of β_1 and c which means that a direct contact between people plays a critical role in the transmission of the disease. Stability analysis shows that if the basic reproduction number $\mathcal{R}_0 < 1$, then the disease free equilibrium P_0 is locally asymptotically stable for all $\tau \geq 0$. That is to say, the time delay in media coverage cannot affect the stability of the disease free equilibrium. This means that we can ignore the effect of time delay for $\mathcal{R}_0 < 1$. If $\mathcal{R}_0 > 1$, system (1.2) has at least one positive endemic equilibrium P^* and we obtain the conditions for the Hopf bifurcation exists such that the time delay is chosen as the bifurcation parameter, which can destabilize the positive equilibrium when it increases.

References

- G. O. Agaba, Y. N. Kyrychko and K. B. Blyuss, *Time-delayed SIS epidemic model with population awareness*, Ecological Complexity, 2017, 31, 50–56.
- [2] F. B. Agusto, S. Bewick and W. F. Fagan, Mathematical model for Zika virus dynamics with Sexual transmission route, Ecological Complexity, 2017, 29, 61– 81.
- [3] F. B. Agusto, S. Bewick and W. F. Fagan, *Mathematical model of Zika virus with vertical transmission*, Infectious Disease Modelling, 2017, 2(2), 244–267.
- [4] M. E. Alexander, C. Bowman, S. M. Moghadas, R. Summers, A. B. Gumel and B. M. Sahai, A vaccination Model for Transmission Dynamics of Influenza, SIAM Journal on Applied Dynamical Systems, 2004, 3(4), 503–524.
- [5] P. M. Arguin, A. W. Navin, S. F. Steele, L. H. Weld, and P. E. Kozarsky, *Health communication during SARS*, Emerging Infectious Diseases, 2004, 10(2), 377–380.
- [6] S. Baize, D. Pannetier, L. Oestereich, T. Rieger, L. Koivogui, N. Magassouba, et al., *Emergence of Zaire Ebola virus disease in Guinea*, The New England Journal of Medicine, 2014, 371(15), 1418–1425.
- [7] C. T. Bauch, J. O. Lloyd-Smith, M. P. Coffee and A. P. Galvani, Dynamically modeling SARS and other newly emerging respiratory illnesses: past, present, and future, Epidemiology, 2005, 16(6), 791–801.
- [8] E. Beretta and Y. Kuang, Geometric stability switch criteria in delay differential systems with delay dependent parameters, SIAM Journal on Mathematical Analysis, 2002, 33(5), 1144–1165.
- [9] A. Berman and R. Plemmons, *Non-Negative Matrices in the Mathematical Sciences*, Society for Industrial and Applied Mathematics, Philadelphia, 1979.
- [10] K. Best and A. S. Perelson, Mathematical modeling of within-host Zika virus dynamics, Immunological Reviews, 2018, 285, 81–96.
- [11] S. K. Biswas, U. Ghosh and S. Sarkar, Mathematical model of zika virus dynamics with vector control and sensitivity analysis, Infectious Disease Modelling, 2020, 5, 23–41.

- [12] P. Boldog, T. Tekeli, Z. Vizi, A. Dénes, F. A. Bartha and G. Röst, Risk assessment of novel coronavirus COVID-19 outbreaks outside China, Journal of Clinical Medicine, 2020, 9(2), 571, 12 pages. DOI: 10.3390/jcm9020571
- [13] V. Capasso and G. Serio, A generalization of the Kermack-McKendrick deterministic epidemic model, Mathematical Biosciences, 1978, 42(1-2), 43–62.
- [14] J. Chen, F. Yang, S. Zhan, et al., Processing on the parameters and initial values of SARS simulation model for Beijing, Journal of System Simulation, 2003, 15(7), 995–998.
- [15] N. S. Chong, J. M. Tchuenche and R. J. Smith, A mathematical model of avian influenza with half-saturated incidence, Theory In Biosciences, 2014, 133, 23– 38.
- [16] J. P. Chretien, S. Riley and D. B. George, Mathematical modeling of the West Africa Ebola epidemic, Elife, 2015, 4, e09186, 21 pages. DOI: 10.7554/eLife.09186
- S. Collinson, K. Khan and J. M. Heffernan, The effects of media reports on disease spread and important public health measurements, PLoS ONE, 2015, 10(11), e0141423, 21 pages.
 DOI: 10.1371/journal.pone.0141423
- [18] W. A. Coppel, Stability and Asymptotic Behaviour of Differential Equations, Heat, Boston, 1965.
- [19] D. K. Das, S. Khajanchi and T. K. Kar, The impact of the media awareness and optimal strategy on the prevalence of tuberculosis, Applied Mathematics and Computation, 2020, 366, Article ID 124732, 23 pages. DOI: 10.1016/j.amc.2019.124732
- [20] D. K. Das, S. Khajanchi and T. K. Kar, Transmission dynamics of tuberculosis with multiple re-infections, Chaos, Solitons & Fractals, 2020, 130, Article ID 109450, 13 pages. DOI: 10.1016/j.chaos.2019.109450
- [21] O. Diekmann, J. A. P. Heesterbeek and J. Metz, On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations, Journal of Mathematical Biology, 1990, 29(4), 365–382.
- [22] P. van den Driessche and J. Watmough, Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission, Mathematical Biosciences, 2002, 180(1-2), 29–48.
- [23] D. Gatherer, The 2014 Ebola virus disease outbreak in West Africa, Journal of General Virology, 2014, 95(8), 1619–1624.
- [24] A. B. Gumel, Global dynamics of a two-strain avian influenza model, International Journal of Computer Mathematics, 2009, 86(1), 85–108.
- [25] J. K. Hale and S. M. Verduyn Lunel, Introduction to functional differential equations, Springer-Verlag, New York, 1993.
- [26] T. Hussain, M. Ozair, K. O. Okosun, M. Ishfaq, A. U. Awan and A. Aslam, Dynamics of swine influenza model with optimal control, Advances in Difference

Equations, 2019, 508, 22 pages. DOI:10.1186/s13662-019-2434-4

- [27] S. Iwami, Y. Takeuchi and X. Liu, Avian-human influenza epidemic model, Mathematical Biosciences, 2007, 207(1), 1–25.
- [28] S. Khajanchi, S. Bera and T. K. Roy, Mathematical analysis of the global dynamics of a HTLV-I infection model, considering the role of cytotoxic Tlymphocytes, Mathematics and Computers in Simulation, 2021, 180, 354–378.
- [29] S. Khajanchi, D. K. Das and T. K. Kar, Dynamics of tuberculosis transmission with exogenous reinfections and endogenous reactivation, Physica A, 2018, 497, 52–71.
- [30] S. Khajanchi and K. Sarkar, Forecasting the daily and cumulative number of cases for the COVID-19 pandemic in India, Chaos, 2020, 30, Article ID 071101, 17 pages.

DOI: 10.1063/5.0016240

- [31] S. Khajanchi, K. Sarkar and J. Mondal, Dynamics of the COVID-19 pandemic in India, 2021. arXiv: 2005.06286v2
- [32] M. A. Khan and A. Atangana, Modeling the dynamics of novel coronavirus (2019-nCov) with fractional derivative, Alexandria Engineering Journal, 2020, 59(4), 2379–2389.
- [33] A. R. Kimbir, T. Aboiyar and P. N. Okolo, Numerical simulation for the transmission dynamics of avian influenza, Mathematical Theory and Modeling, 2014, 4(14), 79–93.
- [34] M. Kouche, B. Boulfoul and B. Ainseba, Mathematical analysis of an HIV infection model including quiescent cells and periodic antiviral therapy, International Journal of Biomathematics, 2017, 10(5), Article ID 1750065, 32 pages. DOI: 10.1142/S1793524517500656
- [35] Y. Kuang, Delay Differential Equations with Applications in Population Dynamics, Academic Press, San Diego, 1993.
- [36] A. Kumar, P. K. Srivastava and Y. Takeuchi, Modeling the role of information and limited optimal treatment on disease prevalences, Journal of Theoretical Biology, 2017, 414, 103–119.
- [37] J. P. LaSalle, The Stability of Dynamical Systems, SIAM, Philadelphia, 1976.
- [38] P. De Leenheer and S. Pilyugin, Virus dynamics: a global analysis, SIAM Journal on Applied Mathematics, 2003, 63, 1313–1327.
- [39] B. Li, Y. Chen, X. Lu and S. Liu, A delayed HIV-1 model with virus waning term, Mathematical Biosciences and Engineering, 2016, 13, 135–157.
- [40] F. Li and J. Wang, Analysis of an HIV infection model with logistic targetcell growth and cell-to-cell transmission, Chaos, Solitons & Fractals, 2015, 81, 136–145.
- [41] M. Li and H. Shu, Joint effects of mitosis and intracellular delay on viral dynamics: two-parameter bifurcation analysis, Bulletin of Mathematical Biology, 2012, 64, 1005–1020.

- [42] W. Liu, A SIRS epidemic model incorporating media coverage with random perturbation, Abstract and Applied Analysis, 2013, Article ID 792308, 10 pages. DOI: 10.1155/2013/792308
- [43] Y. Liu and J. Cui, The impact of media coverage on the dynamics of infectious disease, International Journal of Biomathematics, 2008, 1(1), 65–74.
- [44] R. Liu, J. Wu and H. Zhu, Media/psychological impact on multiple outbreaks of emerging infectious diseases, Computational and Mathematical Methods in Medicine, 2007, 8(3), 153–164.
- [45] W. Liu and Q. Zheng, A stochastic SIS model incorporating media coverage in a two patch setting, Applied Mathematics and Computation, 2015, 262, 160–168.
- [46] L. Mitchell and J. V. Ross, A data-driven model for influenza transmission incorporating media effects, Royal Society Open Science, 2016, 3, Article ID 160481, 10 pages. DOI: 10.1098/rsos.160481
- [47] N. Nirwani and V. H. Badshah, Mathematical Analysis of a Swine Flu Model with Mixed Transmission, Journal of Advances in Mathematics and Computer Science, 2016, 14(5), 1–8.
- [48] M. A. Nowak and R. M. May, Mathematical Principles of Immunology and Virology, Virus Dynamics, Oxford University Press, Oxford, 2000.
- [49] J. J. H. Reynolds, M. Torremorell and M. E. Craft, Mathematical Modeling of Influenza A Virus Dynamics within Swine Farms and the Effects of Vaccination, PLoS ONE, 2014, 9(8), e106177. DOI: 10.1371/journal.pone.0106177
- [50] X. Rong, L. Yang, H. Chu and M. Fan, Effect of delay in diagnosis on transmission of COVID-19, Mathematical Biosciences and Engineering, 2020, 17(3), 2725–2740.
- [51] S. Ruan and J. Wei, On the zeros of transcendental functions with applications to stability of delay differential equations with two delays, Dynamics of Continuous, Discrete & Impulsive Systems Series A, 2003, 10(6), 863–874.
- [52] K. Sarkar, S. Khajanchi and J. J. Nieto, Modeling and forecasting the COVID-19 pandemic in India, Chaos, Solitons & Fractals, 2020, 139, Article ID 110049, 54 pages.
 DOI: 10.1016/j.chaos.2020.110049
- [53] O. Sharomi and A. B. Gumel, Re-infection-induced backward bifurcation in the transmission dynamics of Chlamydia trachomatis, Journal of Mathematical Analysis and Applications, 2009, 356(1), 96–118.
- [54] H. L. Smith, Monotone dynamical systems, an introduction to the theory of competitive and cooperative systems, American Mathematical Society, Providence, 1995.
- [55] Y. Song and J. Wei, Bifurcation analysis for Chen's system with delayed feedback and its application to control of chaos, Chaos, Solitons & Fractals, 2004, 22(1), 75–91.
- [56] A. K. Srivastav and M. Ghosh, Modeling and analysis of the symptomatic and asymptomatic infections of swine flu with optimal control, Modeling Earth

Systems and Environment, 2016, 2, 177, 9 pages. DOI: 10.1007/s40808-016-0222-7

- [57] C. Sun, W. Yang, J. Arino and K. Khan, Effect of media-induced social distancing on disease transmission in a two patch setting, Mathematical Biosciences, 2011, 230(2), 87–95.
- [58] J. M. Tchuenche, N. Dube, C. P. Bhunu, R. J. Smith and C. T. Bauch, The impact of media coverage on the transmission dynamics of human influenza, BMC Public Health, 2011, 11(1), S5, 16 pages. DOI: 10.1186/1471-2458-11-S1-S5
- [59] G. Webb, C. Browne, X. Huo, O. Seydi, M. Seydi and P. Magal, A model of the 2014 ebola epidemic in west Africa with contact tracing, PLoS Currents, 2015, 7.
 POOL 10 1271 / currents outbrooks 846b2s21s627018b7d1126s0s8sdf22s

 $\label{eq:DOI: 10.1371/currents.outbreaks. 846b2a31ef37018b7d1126a9c8adf22a} DOI: 10.1371/currents.outbreaks. 846b2a31ef37018b7d1126a9c8adf22a$

- [60] Q. Yan, S. Tang, S. Gabriele and J. Wu, Media coverage and hospital notification: correlation analysis and optimal media impact duration to manage pandemic, Journal of Theoretical Biology, 2016, 390, 1–13.
- [61] H. Zhao, Y. Lin and Y. Dai, An SIRS epidemic model incorporating media coverage with time delay, Computational and Mathematical Methods in Medicine, 2014, Article ID 680743, 10 pages. DOI: 10.1155/2014/680743
- [62] X. Zhao, Dynamical systems in population biology, Springer, Berlin, 2003.