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Dynamics of the impact of Twitter with time delay on the spread of infectious diseases

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In this paper, a mathematical model to study the impact of Twitter in controlling infectious disease is proposed. The model includes the dynamics of "tweets" which may enhance awareness of the disease and cause behavioral changes among the public, thus reducing the transmission of the disease. Furthermore, the model is improved by introducing a time delay between the outbreak of disease and the release of Twitter messages. The basic reproduction number and the conditions for the stability of the equilibria are derived. It is shown that the system undergoes Hopf bifurcation when time delay is increased. Finally, numerical simulations are given to verify the analytical results.

Keywords: Twitter; epidemic; Hopf bifurcation; time delay.

Mathematics Subject Classification 2010: 34C25, 92D30, 34K25

1. Introduction

Prevention and control of infectious disease have become more and more important. As we all know, many people will be infected by diseases because they lack the knowledge of diseases. In 2003, the outbreaks of SARS provided an example: media coverage will help to improve people's awareness of disease and control the spread of the disease.

In recent years, many mathematical models have been engaged in understanding the interplay between the dynamics of an epidemic and various interventional measures (e.g. behavioral changes, vaccination) [1–3]. In particular, Del Vale *et al.* [4] studied the effects of education, vaccination and treatment on HIV transmission,

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and they suggested that, due to unavailability of adequate vaccination and treatment for the disease, awareness programs could be an efficient option for reducing the spread of infection. Cui *et al.* [5] considered a new incidence function that reflects the impact of media coverage; and they suggested that media impact is strong enough to exert a positive effect when the threshold is larger than one. De la Sen *et al.* [6] established an SVEIRS model which involves a regular constant vaccination. Zhao *et al.* [7] proposed and analyzed an SIRS epidemic model incorporating media coverage with time delay, and that study showed the time delay affects the stability of the endemic equilibrium and produces limit cycle oscillations under certain conditions. Furthermore, De la Sen *et al.* [8] also discussed an SEIR propagation disease model subject to delays which potentially involve mixed regular and impulsive vaccination rules, where it is assumed that there is a finite number of time-varying distributed delays in the susceptible-infected coupling dynamics.

Twitter, as a microblog service, provides a new communication way for people to broadcast information. Given the popularity and prevalence of Twitter, its potential for serving as a new dissemination medium is growing [9]. Each day more than 500 million users share information by posting (tweeting or re-tweeting) Twitter messages [10, 11]. Individuals and public health organizations increasingly advocate the use of Twitter for its high-reach, low-cost information [12–14]. For instance, Center for Disease Control and Prevention used Twitter to post tips to help slow the spread of H1N1 influenza in 2009 [15]. Moreover, many infected individuals post about their illness publicly, and share their personal experiences with Twitter [16–18].

The goal of this paper is to study the impact of Twitter on the spread and control of infectious diseases. Twitter is regarded as a new medium of information sharing, and people acquire information not only directly from the original tweet, but also via re-tweets. The re-tweet mechanism has given everyone the power to spread information broadly. People who read these messages will become aware of the disease and form a new class, called the aware individuals. More specifically, the infectious and the aware post more Twitter messages because they are alert to the epidemic than those who are susceptible.

In this paper, we improve the model by introducing time delay to account for the time lag between the outbreak of disease and the release of Twitter messages. Our work is focused on a quantitative study of a mathematical model with time delay, in which the bifurcation appears. In fact, local bifurcations of continuous dynamical systems and the bifurcation caused by time delay are studied in some papers [19–22]. Shan *et al.* established an SIR model with a nonlinear recovery rate, and they presented the bifurcation diagram near the cusp type of the B–T bifurcation point [23]. Zhou *et al.* studied an SEIR epidemic model with a saturated recovery rate, and global dynamics are shown by compound matrices and geometric approaches [24].

The rest of this paper is organized as follows. In Sec. 2, a mathematical model with delay has been proposed to study the dynamics of effect of Titter. Then the conditions of the stability of equilibria and the existence of Hopf-bifurcation are analyzed in Sec. 3. At last, some numerical simulations to confirm the results are performed in Sec. 4.

2. The Model and Preliminary Results

Nowadays for people Internet has become a major tool to get all kinds of information. More than half of online news consumers get their news through Twitter or Facebook [25]. Here, we take Twitter as an example to discuss the impact of social media in the spread of epidemics. In this paper, the total population $N(t)$ at time t is divided into three classes: the susceptible $S(t)$, the infected $I(t)$ and the aware $S_T(t)$. As described in the introduction, the aware and infected individuals post Twitter messages with the rates μ_1 and μ_2, respectively. Here, we assume $\mu_2 > \mu_1$. Let $T(t)$ be the number of Twitter messages posted by S_T and I at time t, and μ be the depletion rate of these messages.

It is assumed that the disease spreads due to the direct contact between the susceptible and the infected only. Further, it is considered that due to the effect of Twitter, susceptible individuals avoid being in contact with the infected and form a different class, namely the aware susceptible. We assume that a proportion of infected individuals will recover with rate λ through treatment. After recovery, a fraction p of recovered people will become aware and join the aware susceptible class whereas the remaining fraction $1-p$ will become unaware susceptible. We also assume that the growth rate of the cumulative density of awareness programs is proportional to the disease induced mortality rate of the infected population. Figure 1 illustrates the disease spreading with the impact of Twitter in this model.

Using the fact that Twitter messages can influence the susceptible population to a limited extent, we have considered interaction between the susceptible and Twitter messages as Holling type-II functional response $h(T) = \frac{\alpha T}{k+T}$. The constant k limits the effect of Titter on the susceptible and is known as the half saturation point, and α represents the dissemination rate of Twitter among the susceptible

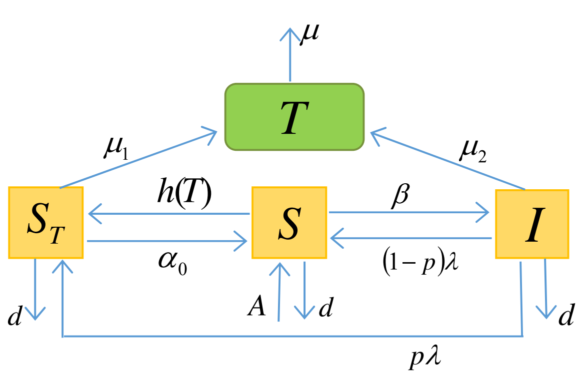

Fig. 1. Flow chart of the model.

due to which they form a different class. The interactions between susceptible and infective individuals are assumed to be bilinear.

In this model, the susceptible population immigrates with a constant rate A, β is the contact rate of the susceptible with the infected, and d is the natural death rate. α_0 is a constant which represents the rate of transfer of the aware to the susceptible due to fading of memory or certain social factors, etc. All the above constants are assumed to be positive.

Keeping the above facts in mind, the dynamics of this model are governed by the following system. This model is an extension of the traditional SIS model.

$$\begin{cases} \dfrac{dS(t)}{dt} = A - h(T)S(t) + \alpha_0 S_T(t) + (1-p)\lambda I(t) - \beta S(t)I(t) - dS(t), \\ \dfrac{dS_T(t)}{dt} = h(T)S(t) + p\lambda I(t) - \alpha_0 S_T(t) - dS_T(t), \\ \dfrac{dI(t)}{dt} = \beta S(t)I(t) - \lambda I(t) - dI(t), \\ \dfrac{dT(t)}{dt} = \mu_1 S_T(t) + \mu_2 I(t) - \mu T(t). \end{cases} \tag{1}$$

Here $S(0) = S_0 > 0$, $S_T(0) = S_{T0} \geq 0$, $I(0) = I_0 \geq 0$ and $T(0) = T_0 \geq 0$.

On Twitter, people acquire information not always directly from those they follow, but often via re-tweets. No matter how many followers a user has, the tweet is likely to reach a certain number of audience, once these tweets start spreading via re-tweets. This illustrates the power of re-tweeting. In [26], the author points out that re-tweeting occurs ranging from less than one day to more than one month. It is not hard to believe that it takes time for information to spread in social media. As a result, we introduce $\tau(\tau > 0)$ to reflect the fact that the number of tweets at time t will be in accordance with the original tweets (tweeting by S_T and I) at time $t - \tau$. Using the fact that $S + I + S_T = N$, system (1) is reduced to the following system:

$$\begin{cases} \dfrac{dS_T(t)}{dt} = \dfrac{\alpha T(t)}{k + T(t)}(N(t) - S_T(t) - I(t)) + p\lambda I(t) - (\alpha_0 + d)S_T(t), \\ \dfrac{dI(t)}{dt} = \beta(N(t) - S_T(t) - I(t))I(t) - (\lambda + d)I(t), \\ \dfrac{dN(t)}{dt} = A - dN(t), \\ \dfrac{dT(t)}{dt} = \mu_1 S_T(t-\tau) + \mu_2 I(t-\tau) - \mu T(t). \end{cases} \tag{2}$$

Here $S_T(\theta) = S_{T0} \geq 0$, $I(\theta) = I_0 \geq 0$ for $\theta \in [-\tau, 0]$ $N(0) = N_0 \geq 0$ and $T(0) = T_0 \geq 0$. Now it is sufficient to study system (2) in detail rather than system (1).

For the analysis of system (2), we need the region of attraction which is given by the set: $\Omega = \{(S_T, I, N, T) \in \Re_+^4 : 0 \le S_T, I \le N \le \frac{A}{d}, 0 \le T \le \frac{(\mu_1+\mu_2)A}{\mu d}\}$, and it attracts all solutions initiating in the interior of the positive orthant. The equilibria of system (2) are listed as follows:

(i) Disease-free equilibrium (DFE) $E_1^0(0, 0, \frac{A}{d}, 0)$, $E_2^0(\frac{\mu T^0}{\mu_1}, 0, \frac{A}{d}, T^0)$.
(ii) Endemic equilibrium (EE) $E_*(S_T^*, I^*, N^*, T^*)$.

Let us define the basic reproduction number $R_0 = \frac{\beta A}{d(\lambda+d)}$. Here, the existence of equilibrium E_1^0 is trivial. In the equilibrium $E_2^0(\frac{\mu T^0}{\mu_1}, 0, \frac{A}{d}, T^0)$, where $T^0 = \frac{\alpha\mu_1 A - \mu dk(\alpha_0+d)}{\mu d(\alpha_0+\alpha+d)}$, which can be obtained by solving the following equation:

$$\frac{\mu(\alpha_0+\alpha+d)}{\mu_1}T - \frac{\alpha\mu_1 A - \mu dk(\alpha_0+d)}{d\mu_1} = 0.$$

Then we can see that the equilibrium E_2^0 will exist only when $R_c = \frac{\mu_1}{\alpha_0+d}\frac{\alpha A}{kd}\frac{1}{\mu} > 1$. Here, R_c is the threshold of the spread of the information, and the first term of R_c is related to the number of tweets that S_T will post; the second term is proportional to the influenced population out of the susceptible group S and the third term indicates the effectiveness of the tweets posted during the course of the epidemics. If $R_c > 1$, then information will spread and the awareness will exist; if $R_c < 1$, it will not.

When $R_0 > 1$, the values of S_T^*, N^*, T^* in the equilibrium E_* are

$$S_T^* = \frac{A}{d} - \frac{\lambda+d}{\beta} - I^*, \quad N^* = \frac{A}{d},$$
$$T^* = \frac{\mu_1}{\mu}\left(\frac{A}{d} - \frac{\lambda+d}{\beta}\right) + \frac{\mu_2-\mu_1}{\mu}I^*,$$

with I^* satisfying the equation

$$F(I^*) = A_1 I^{*2} + B_1 I^* + C_1 = 0,$$

where

$$\begin{aligned}
A_1 &= \frac{\mu_2-\mu_1}{\mu}\beta(p\lambda+\alpha_0+d),\\
B_1 &= k\beta(p\lambda+\alpha_0+d) + \frac{\alpha(\lambda+d)(\mu_2-\mu_1)}{\mu}\\
&\quad + \frac{\beta}{\mu}\left(\frac{A}{d} - \frac{\lambda+d}{\beta}\right)[\mu_1(p\lambda+2\alpha_0+2d) - \mu_2(\alpha_0+d)],\\
C_1 &= \left(\frac{A}{d} - \frac{\lambda+d}{\beta}\right)\left[\frac{\mu_1\alpha(\lambda+d)}{\mu} - \frac{\mu_1\beta(\alpha_0+d)}{\mu}\left(\frac{A}{d} - \frac{\lambda+d}{\beta}\right) - k\beta(\alpha_0+d)\right]\\
&\quad < \beta(\alpha_0+d)\left(\frac{\lambda+d}{\beta} - \frac{A}{d}\right)\left[k - \frac{\mu_1}{\mu}\left(\frac{\lambda+d}{\beta} - \frac{A}{d}\right)\right].
\end{aligned}$$

Solving $F(I^*) = 0$, we get

$$I^*_{1,2} = \frac{-B_1 \pm \sqrt{B_1^2 - 4A_1C_1}}{2A_1}.$$

Clearly, $A_1 > 0$, and when $R_0 > 1$, we have $C_1 < 0$, then we get the positive solution $I^* = \frac{-B_1+\sqrt{B_1^2-4A_1C_1}}{2A_1}$.

Remark 2.1. System (2) without Twitter and time delay becomes

$$\begin{cases} \dfrac{dS(t)}{dt} = A - \beta S(t)I(t) + \lambda I(t) - dS(t) \\ \dfrac{dI(t)}{dt} = \beta S(t)I(t) - \lambda I(t) - dI(t) \end{cases} \tag{3}$$

where S, I and other parameters are the same as defined in system (2). There are two non-negative equilibria in system (3): The DFE $\bar{E}_0(\frac{A}{d}, 0)$; The EE $\bar{E}_*(\frac{\lambda+d}{\beta}, \frac{A}{d} - \frac{\lambda+d}{\beta})$. The basic reproduction number for system (3) is $\bar{R}_0 = \frac{\beta A}{d(\lambda+d)}$, which is the same as R_0. In addition, $\bar{I} = \frac{A}{d} - \frac{\lambda+d}{\beta} > I^*$ holds, so we can find that Twitter cannot eradicate the infection whenever $R_0 > 1$, but it can reduce the equilibrium number of infected individuals. Further, we note that the existence of DFE E_2^0 of system (2) is caused by the existence of newly formed awareness susceptible S_T.

Remark 2.2. It is easy to note that $\frac{\partial I^*}{\partial \alpha} < 0$ if $\frac{B_{1\alpha}I^* + C_{1\alpha}}{2A_1I^* + B_1} > 0$, $\frac{\partial I^*}{\partial \mu_1} < 0$ if $\frac{A_{1\mu_1}I^{*2} + B_{1\mu_1}I^* + C_1}{2A_1I^* + B_1} > 0$, and $\frac{\partial I^*}{\partial \mu_2} < 0$ if $\frac{A_{1\mu_2}I^{*2} + B_{1\mu_2}I^*}{2A_1I^* + B_1} > 0$, which shows that the equilibrium number of infected individuals decreases as the implementation rate of Twitter increases and the efficacy of Twitter increases. Here, $i_{1j} (i = A, B, C)$ denotes the partial differentiation of i_1 with respect to the parameter j.

3. Stability Analysis

In this section, we present the local stability of E_1^0, E_2^0 and E_*. We also explore the conditions of Hopf-bifurcation by taking delay τ as a bifurcation parameter.

3.1. *Stability of equilibria without delay* ($\tau = 0$)

The stability conditions of equilibria E_1^0, E_2^0 and E_* with $\tau = 0$ are stated in the following theorems.

Theorem 3.1. *When $R_0 < 1$ and $R_c < 1$, the* DFE *E_1^0 is locally asymptotically stable* (LAS).

Proof. The characteristic equation at E_1^0 is

$$(\eta + d)\left[\eta - \beta\left(\frac{A}{d} - \frac{\lambda + d}{\beta}\right)\right] g_1(\eta) = 0,$$

where η is the eigenvalue and $g_1(\eta) = \eta^2 + (\alpha_0 + \mu + d)\eta + \mu(\alpha_0 + d) - \frac{\mu_1 \alpha A}{dk}$.

When $R_0 < 1$, it is clear that

$$\eta_1 = -d < 0, \quad \eta_2 = \beta\left(\frac{A}{d} - \frac{\lambda + d}{\beta}\right) < 0.$$

For $g_1(\eta)$, $\alpha_0 + \mu + d > 0$ holds and when $R_c < 1$ we have $\mu(\alpha_0 + d) - \frac{\alpha\mu_1 A}{dk} > 0$, then we get $\eta_3 < 0$, $\eta_4 < 0$, thus E_1^0 is LAS if $R_0 < 1$ and $R_c < 1$. □

Theorem 3.2. *When* $R_0 < 1$ *and* $R_c > 1$, *the* DFE E_2^0 *is* LAS.

Proof. The characteristic equation at E_2^0 is

$$(\eta + d)\left(\eta - \frac{\beta A}{d} + \lambda + d + \frac{\mu\beta T^0}{\mu_1}\right) g_2(\eta) = 0,$$

where η is the eigenvalue and

$$g_2(\eta) = \eta^2 + \left(\mu + \alpha_0 + d + \frac{\alpha T^0}{k + T^0}\right)\eta + k_1.$$

When $R_0 < 1$ and $R_c > 1$, it is clear that

$$\eta_1^0 = -d < 0, \quad \eta_2^0 = \frac{\beta A}{d} - (\lambda + d) - \frac{\mu\beta T^0}{\mu_1} < 0.$$

For $g_2(\eta)$, we have $\mu + \alpha_0 + d + \frac{\alpha T^0}{k+T^0} > 0$ and

$$\begin{aligned} k_1 &= \mu\left(\alpha_0 + d + \frac{\alpha T^0}{k + T^0}\right) - \frac{\alpha\mu_1 k}{(k + T^0)^2} = \frac{\alpha\mu_1 A}{\beta(k + T^0)} - \frac{\alpha\mu_1 k}{(k + T^0)^2} \\ &= \frac{\alpha\mu_1}{k + T^0}\left(\frac{A}{\beta} - \frac{k}{k + T^0}\right) = \frac{\alpha\mu_1}{k + T^0}\left(\frac{A}{\beta} - 1\right) > 0. \end{aligned}$$

Then we get $\eta_3^0 < 0$, $\eta_4^0 < 0$, thus E_2^0 is LAS when $R_0 < 1$ and $R_c > 1$. □

Theorem 3.3. *The* EE E_* *is* LAS *if* $R_0 > 1$.

Proof. The variational matrix of system (2) at E_* is obtained as follows:

$$\begin{bmatrix} -\Pi_{11} & \Pi_{12} & \dfrac{\alpha T^*}{k + T^*} & \Pi_{14} \\ -\beta I^* & -\beta I^* & \beta I^* & 0 \\ 0 & 0 & -d & 0 \\ \mu_1 & \mu_2 & 0 & -\mu \end{bmatrix}.$$

Here,

$$\Pi_{11} = \frac{\alpha T^*}{k + T^*} + \alpha_0 + d > 0,$$

$$\Pi_{12} = -\frac{\alpha T^*}{k + T^*} + p\lambda,$$

$$\Pi_{14} = \left(N^* - S_T^* - I^*\right)\frac{\alpha k}{(k + T^*)^2} > 0.$$

The characteristic equation at E_* is

$$(\eta + d)\big(\eta^3 + a_1\eta^2 + a_2\eta + a_3\big) = (\eta + d)g_3(\eta) = 0,$$

where η is the eigenvalue and

$$\begin{aligned}
a_1 &= \mu + \beta I^* + \Pi_{11} > 0,\\
a_2 &= (\Pi_{11} + \Pi_{12})\beta I^* + \mu\big(\Pi_{11} + \beta I^*\big) - \mu\Pi_{14}\\
&= (\alpha_0 + d + p\lambda + \mu)\beta I^* + \mu\left(\frac{\alpha T^*}{k + T^*}\alpha_0 + d\right) + \frac{\mu_1 k}{T^*(k + T^*)}\big(p\lambda I^* - (\alpha_0 + d)S_T^*\big)\\
&= (p\lambda + \mu)\beta I^* + \frac{\mu\alpha T^*}{k + T^*} + \frac{\mu_1 k}{T^*(k + T^*)} + (\alpha_0 + d)\left(\mu + \beta I^* - \frac{\mu_1 k S_T^*}{T^*(k + T^*)}\right)\\
&= (p\lambda + \mu)\beta I^* + \frac{\mu\alpha T^*}{k + T^*} + \frac{\mu_1 k}{T^*(k + T^*)}\\
&\quad + (\alpha_0 + d)\left(\beta I^* + \frac{\mu_2 k I^*}{T^*(k + T^*)} + \mu\left(1 - \frac{k}{k + T^*}\right)\right) > 0,\\
a_3 &= [\mu(\Pi_{11} + \Pi_{12}) + \Pi_{14}(\mu_2 - \mu_1)]\beta I^*\\
&= [\mu(\alpha_0 + d + p\lambda) + \Pi_{14}(\mu_2 - \mu_1)]\beta I^* > 0.
\end{aligned}$$

Furthermore,

$$\begin{aligned}
&a_1 a_2 - a_3\\
&\quad = \big(\mu + \beta I^* + \Pi_{11}\big)\big[\beta I^*(\Pi_{11} + \Pi_{12}) + \mu\big(\Pi_{11} + \beta I^*\big) - \mu\Pi_{14}\big]\\
&\qquad - \beta I^*[\mu(\Pi_{11} + \Pi_{12}) + \Pi_{14}(\mu_2 - \mu_1)]\\
&\quad > \big(\Pi_{11} + \beta I^*\big)\big(\beta I^*(\mu + \Pi_{11} + \Pi_{12}) + \mu(\mu + \Pi_{11})\big) + \frac{\alpha k(\mu_2 N^* - \mu_1(p\lambda + d))}{(k + T^*)^2}\\
&\quad > \beta I^*\big(\Pi_{11} + \beta I^*\big)(\mu + \alpha_0 + d + p\lambda) + \frac{\alpha k\mu_2}{(k + T^*)^2}(N^* - (p\lambda + d))\\
&\quad > \mu\beta I^*(\Pi_{11} + \beta I^*) + \frac{\alpha k\mu_2}{(k + T^*)^2}\left(N^* - \frac{p\lambda + d}{\beta}\right)\\
&\quad > \mu\beta I^*\big(\Pi_{11} + \beta I^*\big) + \frac{\alpha k\mu_2}{(k + T^*)^2}\left(N^* - \frac{\lambda + d}{\beta}\right)\\
&\quad = \mu\beta I^*\big(\Pi_{11} + \beta I^*\big) + \frac{\alpha\beta k\mu_2}{(\lambda + d)(k + T^*)^2}(R_0 - 1).
\end{aligned}$$

It is easy to see that $a_1 a_2 - a_3 > 0$ if $R_0 > 1$. For the above characteristic polynomial, we can claim that all the values of η will be either negative or have negative real part according to the Routh–Hurwitz conditions, thus E_* is LAS when $R_0 > 1$. $\square$

3.2. *Stability of equilibria with delay ($\tau > 0$)*

The stability of equilibria E_1^0 when $\tau > 0$ is similar to Theorem 3.1. Here, we derive the stability conditions for DFE E_2^0 and EE E_*. When $\tau > 0$, the variational matrix of system (2) at E_2^0 is obtained as follows:

$$\begin{bmatrix} -a_{11} & -\dfrac{\alpha T^0}{k+T^0} + p\lambda & \dfrac{\alpha T^0}{k+T^0} & a_{14} \\ 0 & a_{22} & 0 & 0 \\ 0 & 0 & -d & 0 \\ \mu_1 e^{-\eta\tau} & \mu_2 e^{-\eta\tau} & 0 & -\mu \end{bmatrix},$$

where

$$a_{11} = -\left(d + \alpha_0 + \frac{\alpha T^0}{k+T^0}\right) = -\frac{\alpha\mu_1 A}{\mu\beta(k+T^0)} < 0,$$

$$a_{14} = \frac{\alpha k}{(k+T^0)^2} > 0, \quad a_{22} = \frac{\beta A}{d} - (\lambda + d) - \frac{\mu\beta}{\mu_1}T^0.$$

The character equation at E_2^0 is

$$(\eta + d)(\eta - a_{22})f_1(\eta) = 0, \tag{4}$$

where η is the eigenvalue and $f_1(\eta) = \eta^2 + b_1\eta + b_2 + b_3 e^{-\eta\tau}$,

$$b_1 = \mu - a_{11} = \mu + \frac{\alpha\mu_1 A}{\mu\beta(k+T^0)} > 0,$$

$$b_2 = -\mu a_{11} = \frac{\alpha\mu_1 A}{\beta(k+T^0)} > 0,$$

$$b_3 = -\mu_1 a_{14} = -\frac{\alpha\mu_1 k}{(k+T^0)^2} < 0.$$

When $R_0 < 1$ and $R_c > 1$, Eq. (4) has the following two negative roots:

$$\eta_1 = -d < 0, \quad \eta_2 = \frac{\beta A}{d} - (\lambda + d) - \frac{\mu\beta}{\mu_1}T^0 < 0.$$

For $f_1(\eta)$, we first prove that $f_1(\eta) = 0$ does not have non-negative real roots. Without loss of generality, and assuming that $\eta_0 \geq 0$ is a root of $f_1(\eta) = 0$, we have

$$b_2 + b_3 e^{-\eta_0\tau} = -\eta_0^2 - b_1\eta_0 < 0,$$

$$b_2 + b_3 e^{-\eta_0\tau} \geq b_2 + b_3 = \frac{\alpha\mu_1}{k+T^0}\left(\frac{A}{\beta} - \frac{k}{k+T^0}\right) = \frac{\alpha\mu_1}{k+T^0}\left(\frac{A}{\beta} - 1\right) > 0.$$

Thus, $f_1(\eta) = 0$ does not have non-negative real roots.

To show the Hopf-bifurcation, we need to show that Eq. (4) has a pair of purely imaginary roots, which equivalent to show $f_1(\eta) = 0$ has a pair of purely imaginary roots. For this assuming that $\eta = i\theta (\theta > 0)$ is a root of $f_1(\eta) = 0$ without loss of generality. That is the case if and only if θ satisfies the equation

$$-\theta^2 + ib_1\theta + b_2 = -b_3(\cos\theta\tau - i\sin\theta\tau). \tag{5}$$

Separating the real and imaginary parts, we have the following system:

$$\theta^2 - b_2 = b_3\cos\theta\tau, \quad b_1\theta = b_3\sin\theta\tau.$$

Squaring both sides of above each equation to eliminate the trigonometric functions and adding the squared above equations to obtain the following equation with substituting $\theta^2 = \xi$, we have

$$f_2(\xi) = \xi^2 + l_1\xi + l_2 = 0, \tag{6}$$

where

$$\begin{aligned} l_1 &= b_1^2 - 2b_2 = \mu^2 + a_{11}^2 > 0, \\ l_2 &= b_2^2 - b_3^2 = (b_2 - b_3)\left(\frac{\alpha\mu_1 A}{\beta(k+T^0)} - \frac{\alpha\mu_1 k}{(k+T^0)^2}\right) \\ &= \frac{(b_2-b_3)\alpha\mu_1}{k+T^0}\left(\frac{A}{\beta} - \frac{k}{k+T^0}\right) > \frac{(b_2-b_3)\alpha\mu_1}{k+T^0}\left(\frac{A}{\beta} - 1\right) > 0. \end{aligned}$$

We can see that Eq. (6) has no positive real roots for $l_i > 0$ $(i = 1, 2)$. That is to say, we cannot get any positive θ which satisfies Eq. (4). So all the eigenvalues have negative real parts for any $\tau > 0$. Thus, E_2^0 is locally asymptotically stable. The results can be summarized in the following theorem.

Theorem 3.4. *If $R_0 < 1$ and $R_c < 1$, the* DFE E_1^0 *is LAS for all delay $\tau > 0$. If $R_0 < 1$ and $R_c > 1$, the* DFE E_2^0 *is* LAS *for all delay $\tau > 0$.*

In the following, we consider the stability of E_*. Linearizing system (2) at E_* and get

$$\frac{du}{dt} = \begin{bmatrix} -\Pi_{11} & \Pi_{12} & \dfrac{\alpha T^*}{k+T^*} & \Pi_{14} \\ -\beta I^* & -\beta I^* & \beta I^* & 0 \\ 0 & 0 & -d & 0 \\ 0 & 0 & 0 & -\mu \end{bmatrix} u(t) + \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & -d & 0 \\ \mu_1 & \mu_2 & 0 & 0 \end{bmatrix} u(t-\tau),$$

where $u(t) = [x(t) \quad y(t) \quad z(t) \quad m(t)]^T$, x, y, z and m are small perturbations around E_*. The characteristic equation at E_* is

$$P(\eta) = \eta^4 + c_1\eta^3 + c_2\eta^2 + c_3\eta + c_4 + (c_5\eta^2 + c_6\eta + c_7)e^{-\eta\tau} = 0, \tag{7}$$

where η is the eigenvalue and

$$\begin{aligned} c_1 &= d + a_1, \\ c_2 &= da_1 + \beta I^*(\mu + \Pi_{11} + \Pi_{12}) + \mu\Pi_{11}, \end{aligned}$$

$$c_3 = d\beta I^*(\mu + \Pi_{11} + \Pi_{12}) + d\mu\Pi_{11} + \mu\beta I^*(\Pi_{11} + \Pi_{12}),$$

$$c_4 = d\mu\beta I^*(\Pi_{11} + \Pi_{12}), \quad c_5 = -\mu_1\Pi_{14},$$

$$c_6 = \mu_2\Pi_{14}\beta I^* - \mu_1\Pi_{14}(d + \beta I^*),$$

$$c_7 = (\mu_2 - \mu_1)d\Pi_{14}\beta I^*.$$

To show the Hopf-bifurcation, we need to show that Eq. (7) has a pair of purely imaginary roots. For this, substituting $\eta = i\omega(\omega > 0)$ into Eq. (7) and separating real and imaginary parts, we get the following transcendental equations:

$$\omega^4 - c_2\omega^2 + c_4 = -c_6\omega\sin(\omega\tau) + (c_5\omega^2 - c_7)\cos(\omega\tau),$$

$$b_1\omega^3 - c_3\omega = c_6\omega\cos(\omega\tau) + (c_5\omega^2 - c_7)\sin(\omega\tau).$$

Squaring and adding above equations and substituting $\varphi = \omega^2$, we get

$$f_3(\varphi) = \varphi^4 + p_1\varphi^3 + p_2\varphi^2 + p_3\varphi + p_4 = 0, \tag{8}$$

where $p_1 = c_1^2 - 2c_2$, $p_2 = c_2^2 - c_5^2 + 2c_4 - 2c_1c_3$, $p_3 = c_3^2 - c_6^2 + 2c_5c_7 - 2c_2c_4$, $p_4 = c_4^2 - c_7^2$.

If the coefficients of $f_3(\varphi)$ satisfy the conditions of the Routh–Hurwitz criterion, then E_* is locally asymptotically stable for all delay $\tau > 0$, provided it is stable in the absence of delay. On the other hand, we consider that the values of $p_i(i = 1, 2, 3, 4)$ do not satisfy the Routh–Hurwitz criterion.

Lemma 3.1. *If* $\frac{\mu_1(\alpha_0+d+p\lambda)+\alpha k(\mu_2-\mu_1)}{\mu_1(\mu_2-\mu_1)} < \frac{\alpha kA}{d(k+T^*)^2}$, *Eq.* (8) *has at least one positive root* φ_0.

Proof. For $f_3(\varphi)$, we have

$$\begin{aligned} c_4 - c_7 &= d\beta I^*(\mu(\Pi_{11} + \Pi_{12}) - (\mu_2 - \mu_1)\Pi_{14}) \\ &= d\beta I^*\left(\mu(\alpha_0 + d + p\lambda) + \frac{\alpha k(\mu_2 - \mu_1)(S_T^* + I^*)}{(k + T^*)^2} - \frac{\alpha k(\mu_2 - \mu_1)N^*}{(k + T^*)^2}\right) \\ &< d\mu\beta I^*\left(\alpha_0 + d + p\lambda + \frac{\alpha k(\mu_2 - \mu_1)}{\mu_1} - \frac{\alpha kA(\mu_2 - \mu_1)}{d(k + T^*)^2}\right). \end{aligned}$$

It is obvious that $p_4 = (c_4 + c_7)(c_4 - c_7) < 0$ if $\frac{\mu_1(\alpha_0+d+p\lambda)+\alpha k(\mu_2-\mu_1)}{\mu_1(\mu_2-\mu_1)} < \frac{\alpha kA}{d(k+T^*)^2}$, then we have $f_3(0) < 0$ and $f_3(+\infty) \to +\infty$, thus Eq. (8) has at least one positive root φ_0. Denote $\omega_0 = \sqrt{\varphi_0}$, then Eq. (8) has a pair of purely imaginary roots $(\pm i\omega_0)$. $\square$

Now we turn to the bifurcation analysis. To establish the Hopf bifurcation at $\tau = \tau_0$, we need to show that $\Re\frac{d\eta(\tau)}{d\tau}|_{\tau=\tau_0} > 0$.

Lemma 3.2. *The following transversality condition holds*

$$\Re \frac{d\eta(\tau)}{d\tau}\bigg|_{\tau=\tau_0} > 0.$$

Proof. Differentiating Eq. (7) with respect to τ, we get

$$\left(\frac{d\eta}{d\tau}\right)^{-1} = \frac{4\eta^3 + 3c_1\eta^2 + 2c_2\eta + c_3 + (2c_5\eta + c_6)e^{-\eta\tau}}{\eta\big(c_5\eta^2 + c_6\eta + c_7\big)e^{-\eta\tau}} - \frac{\tau}{\eta}.$$

So

$$\begin{aligned}
\mathrm{Sign}\left(\Re\frac{d\eta}{d\tau}\right)_{\tau=\tau_0} &= \mathrm{Sign}\left(\Re\frac{d\eta}{d\tau}\right)^{-1}_{\eta=i\omega_0} \\
&= \mathrm{Sign}\left(\Re\left[\frac{4\eta^3 + 3c_1\eta^2 + 2c_2\eta + c_3}{-\eta\big(\eta^4 + c_1\eta^3 + c_2\eta^2 + c_3\eta + c_4\big)}\right.\right. \\
&\qquad \left.\left. + \frac{2c_5\eta + c_6}{\eta\big(c_5\eta^2 + c_6\eta + c_7\big)}\right]\right)_{\eta=i\omega_0} \\
&= \mathrm{Sign}\left(\Re\left[\frac{q_1 + q_2 i}{q_3 + q_4 i} + \frac{c_6 + q_5 i}{q_6 + q_7 i}\right]\right) \\
&= \mathrm{Sign}\left(\Re\left[\frac{\Phi_1(\omega_0)}{q_3^2 + q_4^2} + \frac{\Phi_2(\omega_0)}{q_6^2 + q_7^2}\right]\right),
\end{aligned}$$

where

$$\begin{aligned}
\Phi_1(\omega_0) &= \omega_0^2\big(4\omega_0^6 + 3p_1\omega_0^4 + 2\big(p_2 + c_5^2\big)\omega_0^2 + c_3^2 - 2c_2c_4\big), \\
\Phi_2(\omega_0) &= \omega_0^2\big(2c_5^2\omega_0^2 + 2c_5c_7 - c_6^2\big),
\end{aligned}$$

and

$$\begin{aligned}
&q_1 = -3c_1\omega_0^2 + c_3, \quad q_2 = -4\omega_0^3 + 2c_2\omega_0, \\
&q_3 = c_3\omega_0^2 - c_1\omega_0^4, \quad q_4 = -\omega_0^5 + c_2\omega_0^3 - c_4\omega_0, \\
&q_5 = 2c_5\omega_0, \quad q_6 = -c_6\omega_0^2, \quad q_7 = c_7\omega_0 - c_5\omega_0^3.
\end{aligned}$$

□

If $\Phi_1(\omega_0) > 0, \Phi_2(\omega_0) > 0$, then $\mathrm{Sign}(\Re\frac{d\eta}{d\tau})_{\tau=\tau_0} > 0$, this proves the Lemma. In this case, the results can be summarized in the following theorem:

Theorem 3.5. *The endemic equilibrium E_* of the system is* LAS *when $\tau < \tau_0$ and becomes unstable for $\tau > \tau_0$ provided*

$$\frac{\mu_1(\alpha_0 + d + p\lambda) + \alpha k(\mu_2 - \mu_1)}{\mu_1(\mu_2 - \mu_1)} < \frac{\alpha k A}{d(k + T^*)^2}.$$

When $\tau = \tau_0$ and $\Phi_1(\omega_0) > 0, \Phi_2(\omega_0) > 0$ holds, a Hopf-bifurcation occurs, leading a family of periodic solutions bifurcating from E_* as τ passes through the critical value τ_0, where

$$\tau_0 = \frac{1}{\omega_0} \arccos \frac{c_5\omega_0^6 + (c_1c_6 - c_2c_5 - c_5)\omega_0^4 + (c_2c_7 - c_4c_5 - c_3c_6)\omega_0^2 - c_4c_7}{c_5^2\omega_0^4 + \left(c_6^2 - 2c_5c_7\right)\omega_0^2 + c_7^2}.$$

4. Numerical Simulations

In order to confirm the obtained results, the numerical simulations are carried out by using Matlab. The parameters are given as follows:

The values of E_* for these data are obtained as: $S_T^* = 1.165\times 10^5$, $I^* = 265.1035$, $N^* = 1.25 \times 10^5$, $T^* = 81.2471$. The basic reproduction number R_0 is 24.9980. The value of τ_0 is 12.

As is shown above, for $\tau < \tau_0$, there is a unique LAS endemic equilibrium which is plotted in Fig. 2. The system undergoes Hopf bifurcation when τ increases through τ_0 and the limit cycle oscillations are shown in Fig. 3. In Fig. 4, the bifurcation diagram is given to show the swaps in the stability of the positive equilibrium, and here the delay τ is bifurcation parameter, thus it is known that τ plays a key role in the prevention and control of the diseases.

In the following, we choose μ_1, μ_2 and α changing from 0.0001 to 0.9 to study the variations of $\frac{\partial I^*}{\partial \mu_1}$, $\frac{\partial I^*}{\partial \mu_2}$, $\frac{\partial I^*}{\partial \alpha}$ and plot them in Fig. 5, respectively. It is clear that if one of parameters μ_1, μ_2 and α increases, the number of infected individuals decreases, which confirms the results given in Remark 2.2.

A comparison between the oscillations in I and T is also shown in Fig. 5. From the last figure in Fig. 5, it can be seen that the peak of infected individuals appears slightly earlier than the peak of Twitter messages. It is reasonable because Twitter messages are generated by infectious individuals and awareness individuals, and time delay occurs when people spend time in collecting and processing data.

Table 1. Parameters for the simulation.

Parameters	Value	The source of data
A	$5\,\text{day}^{-1}$	[28]
β	$0.0001\,\text{day}^{-1}$	Estimation
α	$0.002\,\text{day}^{-1}$	Estimation
α_0	$0.001\,\text{day}^{-1}$	Estimation
λ	$0.5\,\text{day}^{-1}$	[27]
d	$0.00004\,\text{day}^{-1}$	[28]
μ	$0.09\,\text{day}^{-1}$	[29]
μ_1	$0.00005\,\text{day}^{-1}$	Estimation
μ_2	$0.005\,\text{day}^{-1}$	Estimation
k	$150\,\text{day}^{-1}$	Estimation
p	$0.6\,\text{day}^{-1}$	[29]

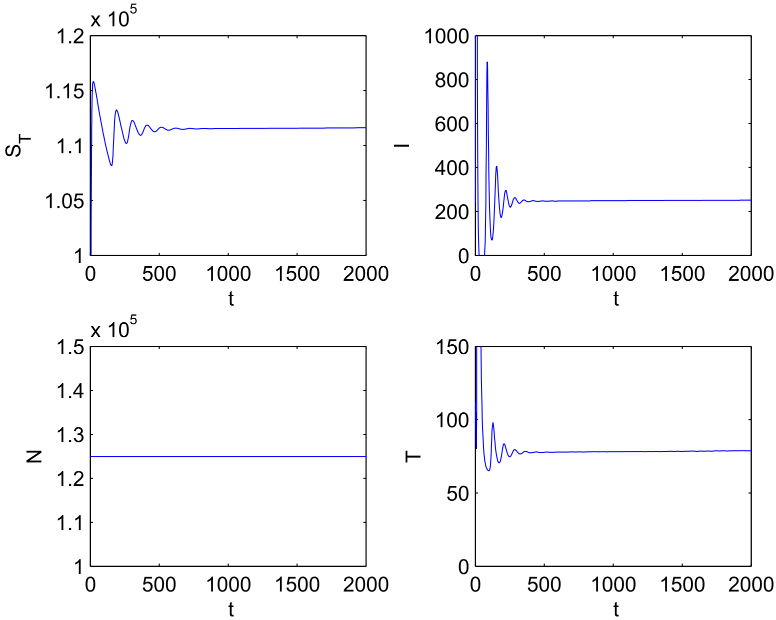

Fig. 2. Stability of system (2) when $\tau = 8$.

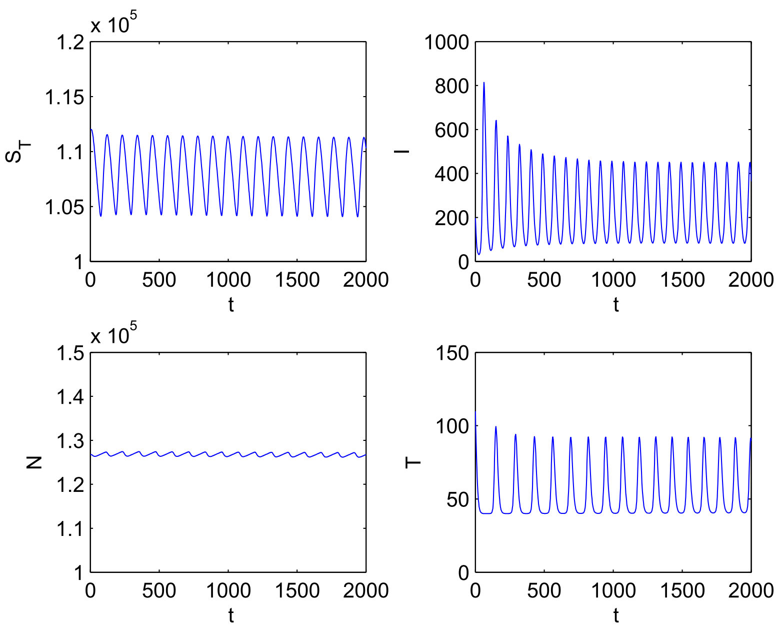

Fig. 3. Hopf bifurcation of system (2) when $\tau = 20$.

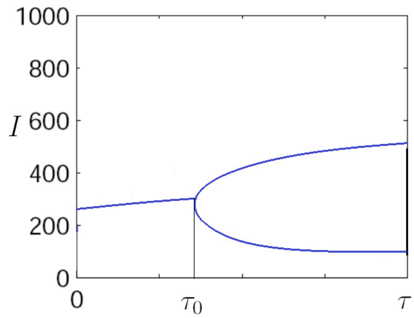

Fig. 4. Bifurcation diagram of system (2) with τ.

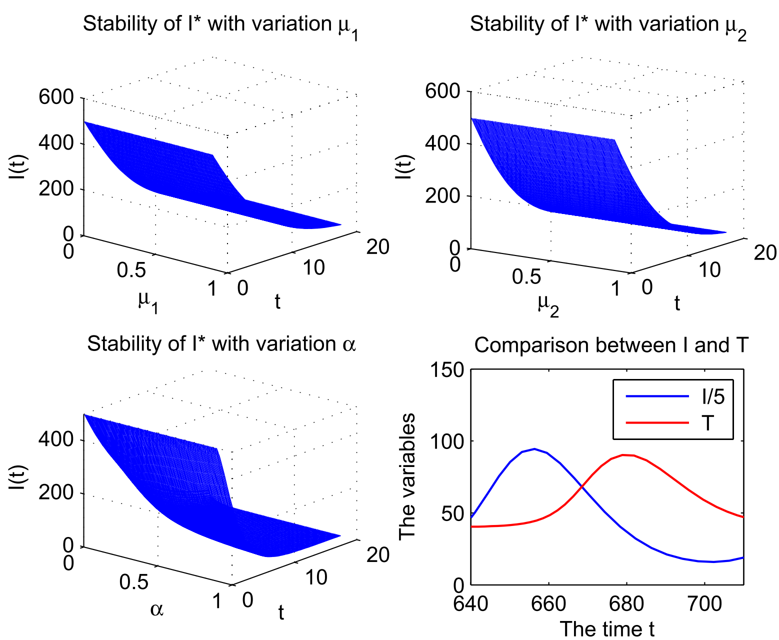

Fig. 5. The variations and comparison.

5. Conclusion and Discussion

The aim of this paper is to study the impact of Twitter on the spread and control of infectious diseases. A mathematical model with delay and the dynamics of tweets is developed, and the saturated incident rate of the disease is given because the behaviors will change between individuals when they received tweets. The model exhibits three equilibria, specifically, E_1^0 is stable when $R_0 < 1$ and the threshold value $R_c < 1$, while E_2^0 is stable when $R_0 < 1$ and $R_c > 1$. Here we note that the threshold value R_c is caused by the existence of awareness susceptible S_T. The stability of equilibria E_1^0 and E_2^0 will not change when the delay exists.

When $R_0 > 1$, both E_1^0 and E_2^0 become unstable and the endemic equilibrium E_* exists. Stability analysis demonstrates that in absence of the delay, the endemic equilibrium E_* is locally asymptotically stable when $\tau < \tau_0$, while a Hopf bifurcation will occur when $\tau > \tau_0$. When the threshold is crossed, a Hopf bifurcation occurs and sustained periodic solutions appear. That is to say, Twitter plays a key role in controlling the disease. In order to control the prevalence of infectious diseases more effectively, tweets should be posted in time. In addition, we believe that the model can be extended to other models describing the dynamics with noise and delay [30, 31], nonlinear integro differential equations with time delay [32, 33].

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Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

References

[1] B. Buonomo, A. d'Onofrio and D. Lacitignola, Global stability of an SIR epidemic model with information dependent vaccination, *Math. Biosci.* **216**(1) (2008) 9–16.

[2] A. d'Onofrio, P. Manfredi and E. Salinelli, Vaccinating behaviour, information, and the dynamics of SIR vaccine preventable diseases, *Theoret. Popul. Biol.* **71**(3) (2007) 301–317.

[3] P. Poletti, B. Caprile, M. Ajelli *et al.*, Spontaneous behavioural changes in response to epidemics, *J. Theoret. Biol.* **260**(1) (2009) 31–40.

[4] S. Del Valle, A. M. Evangelista, M. C. Velasco *et al.*, Effects of education, vaccination and treatment on HIV transmission in homosexuals with genetic heterogeneity, *Math. Biosci.* **187**(2) (2004) 111–133.

[5] J. Cui, Y. Sun and H. Zhu, The impact of media on the control of infectious diseases, *J. Dyn. Diff. Equ.* **20**(1) (2008) 31–53.

[6] M. De La Sen, R. P. Agarwal, A. Ibeas *et al.*, On the existence of equilibrium points, boundedness, oscillating behavior and positivity of a SVEIRS epidemic model under constant and impulsive vaccination, *Adv. Diff. Equ.* **2011**(1) (2011) 1–32.

[7] H. Zhao, Y. Lin and Y. Dai, An SIRS epidemic model incorporating media coverage with time delay, *Comput. Math. Methods Med.* **2014** (2014).

[8] M. De la Sen, R. P. Agarwal, A. Ibeas *et al.*, On a generalized time-varying SEIR epidemic model with mixed point and distributed time-varying delays and combined regular and impulsive vaccination controls, *Adv. Diff. Equ.* **2010**(1) (2010) 1–42.

[9] J. V. Dijck Tracing Twitter: The rise of a microblogging platform, *Int. J. Media Cultural Pol.* **7**(3) (2011) 333–348.

[10] D. Brown, 52 cool facts and stats about social media, http://dannybrown.me/2012/06/06/52-cool-facts-social-media-2012.

[11] I. Lunden, Twitter may have 500M+ users but only 170M are active, 75 percent on Twitter's own clients, TechCrunch, 2012, July, 31.

[12] J. Cain, F. Romanelli and B. Fox, Pharmacy, social media, and health: Opportunity for impact, *J. Amer. Pharm. Assoc.* **50**(6) (2009) 745–751.

[13] C. Holt, Emerging technologies: Web 2.0, *Health Inf. Manag. J.* **40**(1) (2011) 33.

[14] K. Vance, W. Howe and R. P. Dellavalle, Social internet sites as a source of public health information, *Dermatologic Clinics* **27**(2) (2009) 133–136.

[15] D. Currie, Public health leaders using social media to convey emergencies: New tools a boon, *The Nation's Health* **39**(6) (2009) 1–10.

[16] L. Donelle and R. G. Booth, Health tweets: An exploration of health promotion on twitter, *Online J. Issues Nurs.* **17**(3) (2012).

[17] N. Heaivilin, B. Gerbert, J. E. Page *et al.*, Public health surveillance of dental pain via Twitter, *J. Dental Res.* **90**(9) (2011) 1047–1051.

[18] D. Scanfeld, V. Scanfeld and E. L. Larson, Dissemination of health information through social networks: Twitter and antibiotics, *Amer. J. Infection Control* **38**(3) (2010) 182–188.

[19] F. Balibrea, A. Martinez and J. C. Valverde, Local bifurcations of continuous dynamical systems under higher order conditions, *Appl. Math. Lett.* **23**(3) (2010) 230–234.

[20] J. Guckenheimer and P. Holmes, *Nonlinear Oscillations, Dynamical Systems, and Bifurcations of Vector Fields* (Springer Science and Business Media, 2013).

[21] S. Wiggins, *Introduction to Applied Nonlinear Dynamical Systems and Chaos* (Springer Science and Business Media, 2003).

[22] Y. A. Kuznetsov, *Elements of Applied Bifurcation Theory* (Springer Science and Business Media, 2013).

[23] C. Shan and H. Zhu, Bifurcations and complex dynamics of an SIR model with the impact of the number of hospital beds, *J. Diff. Equ.* **257**(5) (2014) 1662–1688.

[24] X. Zhou and J. Cui, Analysis of stability and bifurcation for an SEIR epidemic model with saturated recovery rate, *Commun. Nonlinear Sci. Numer. Simul.* **16**(11) (2011) 4438–4450.

[25] A. Mitchell, T. Rosenstiel and L. Christian, What Facebook and Twitter mean for news, Pew Research Center, The State of the News Media, 2012.

[26] H. Kwak, C. Lee, H. Park *et al.*, What is Twitter, a social network or a news media? *Proc. 19th Int. Conf. World Wide Web* (ACM, 2010), pp. 591–600.

[27] K. Pawelek, A. Oeldorf-Hirsch and L. Rong, Modeling the impact of twitter on influenza epidemics, *Math. Biosci. Eng.* **11** (2014) 1337–1356.

[28] A. K. Misra, A. Sharma and V. Singh, Effect of awareness programs in controlling the prevalence of an epidemic with time delay, *J. Biolog. Syst.* **19**(02) (2011) 389–402.

[29] D. Greenhalgh, S. Rana, S. Samanta *et al.*, Awareness programs control infectious disease-Multiple delay induced mathematical model, *Appl. Math. Comput.* **251** (2015) 539–563.

[30] R. Erban, J. Haskovec and Y. A. Sun, Cucker–Smale model with noise and delay, *SIAM J. Appl. Math.* **76**(4) (2016) 1535–1557.

[31] Y. Sun, W. Lin and R. Erban, Time delay can facilitate coherence in self-driven interacting particle systems, *Phys. Rev. E* **90** (2014) 062708.

[32] L. Li, F. Meng and P. Ju, Some new integral inequalities and their applications in studying the stability of nonlinear integro differential equations with time delay, *J. Math. Anal. Appl.* **377** (2011) 853–862.

[33] Q. Feng, F. Meng and B. Zheng, Gronwall-Bellman type nonlinear delay integral inequalities on times scales, *J. Math. Anal. Appl.* **382** (2011) 772–784.