MATHEMATICAL BIOSCIENCES
AND ENGINEERING
Volume **11**, Number **6**, December **2014**

doi:10.3934/mbe.2014.11.1337

pp. **1337–1356**

MODELING THE IMPACT OF TWITTER ON INFLUENZA EPIDEMICS

Kasia A. Pawelek

Department of Mathematics and Computational Science
University of South Carolina Beaufort
Bluffton, SC 29909, USA

Anne Oeldorf-Hirsch

Department of Communication
University of Connecticut
Storrs, CT 06269, USA

Libin Rong*

Department of Mathematics and Statistics
Oakland University
Rochester, MI 48309, USA

(Communicated by Haiyan Wang)

Abstract. Influenza remains a serious public-health problem worldwide. The rising popularity and scale of social networking sites such as Twitter may play an important role in detecting, affecting, and predicting influenza epidemics. In this paper, we develop a simple mathematical model including the dynamics of "tweets" — short, 140-character Twitter messages that may enhance the awareness of disease, change individual's behavior, and reduce the transmission of disease among a population during an influenza season. We analyze the model by deriving the basic reproductive number and proving the stability of the steady states. A Hopf bifurcation occurs when a threshold curve is crossed, which suggests the possibility of multiple outbreaks of influenza. We also perform numerical simulations, conduct sensitivity test on a few parameters related to tweets, and compare modeling predictions with surveillance data of influenza-like illness reported cases and the percentage of tweets self-reporting flu during the 2009 H1N1 flu outbreak in England and Wales. These results show that social media programs like Twitter may serve as a good indicator of seasonal influenza epidemics and influence the emergence and spread of the disease.

1. **Introduction.** In the short time since its creation in 2006, Twitter has transformed from a status-update tool to a vast information network [51]. The site was initially used primarily as a personal communication tool, with most users engaging in "daily chatter" or routine personal updates prompted by the site's question "what are you doing?" [24]. Its function quickly shifted as users moved to sharing links to content relevant to their network contacts [37]. In years since, Twitter's users

2010 *Mathematics Subject Classification.* Primary: 92D25, 92D30; Secondary: 34D20, 34C23, 91C99.

Key words and phrases. Mathematical model, epidemiology, stability, Hopf bifurcation, influenza, social media, twitter, data fitting.

*Correspondence should be addressed to L. Rong (rong2@oakland.edu).

have shaped the site into a vast database of information. Over 600 million users as of January 2014, share information in 780 million "tweets" — short, 140-character Twitter messages — per day [1]. About 52% of online news consumers now get at least some of their news through Twitter or Facebook [36]. The demographics of Twitter users are growingly increasingly diverse in gender, ethnicity, and age [43]. With such rapid information dissemination, Twitter has become a viable option for spreading and tracking information.

Public-health organizations increasingly advocate for the use of social networking sites for their high-reach, low-cost information dissemination potential of infectious diseases [7,23,52]. While the effectiveness of such health campaigns may be hard to measure, they are found to be largely successful in changing behavior [53] of the users and the public. Several large health-related organizations including the Centers for Disease Control and Prevention (CDC), the Food and Drug Administration (FDA), and the American Red Cross have embraced this trend and used social media programs to spread important health information. For instance, the CDC used Twitter to post tips for preventing flu to help slow the spread of H1N1 influenza in 2009, growing from 2,500 followers to 370,000 followers during the 2009 outbreak [12]. Health organizations make use of Twitter, Facebook, and similar internet sites to mitigate the spread of contagious diseases like flu by teaching users the effectiveness of regular hand-washing, reminding them to stay at home when they are sick, and raising awareness about vaccines. Users of these sites will then further contribute by re-sharing this information and adding their own experience for others. Twitter users have indeed done so; many post publicly about their symptoms, primarily for the purposes of gathering information about a particular illness and sharing personal experience [13, 21, 41, 47]. Tweets about flu during a flu epidemic season may play an active role in reducing the transmission of the disease among a population.

Health strategies during an influenza epidemic or pandemic are influenced by previous disease outbreaks. In addition to archived surveillance data, mathematical models have been used to study disease dynamics, and test prevention and treatment strategies based on changes in mobility and contact patterns, viral evolution, and technological and medical advances [3, 5, 6, 14, 15, 19, 20, 27, 32, 35, 54]. Several mathematical models have also been developed to investigate the impact of behavior changes, partially due to social media programs, on the dynamics of infectious diseases [10, 17, 30, 31, 38, 39, 44–46] (also see review [16]). For example, Twitter data were used to examine the spread of Bieber Fever in a modeling paper by Tweedle and Smith? [46]. Liu et al. [30] showed that including the impact of media coverage on the number of reported and hospitalized individuals can generate multiple outbreaks of diseases or sustained periodic oscillations. The model was compared with the data of the 2003 severe acute respiratory syndrome (SARS) outbreak in the Great Toronto Area. Poletti et al. [39] described the circumstances for multiple epidemic outbreaks caused by the spontaneous alteration in behavior. The immediate disease condition, made available to the public via media, was shown to have a crucial role in reducing the spread of infectious diseases [33]. Media coverage may also cause vaccinating panic, over-certainty in vaccine effectiveness, and as a result increase the magnitude of the endemic steady state [45]. Social distancing and public behavior changes can provide more time for vaccine development [40].

Most of the above-mentioned models that considered the media effect assumed that the media-reported number of infected individuals reduces the transmission

rate of the disease. They did not investigated the dynamics of media coverage (e.g., the number or percentage of tweets that report the disease). Recent regression analyses suggested that there is a strong but nonsynchronous correlation between patterns in Twitter messages and national health statistics on flu [2, 48]. In this paper, we will develop a mathematical model that explicitly includes the dynamics of tweets about flu and the potential behavior change of the public to study the emergence and spread of influenza during an epidemic season. We will derive the basic reproductive number of the model and study the steady state stability and bifurcation. Sensitivity tests will be conducted on a few parameters that are related to tweets about flu. We will also compare model predictions with surveillance data of influenza-like illness reported cases and the percentage of tweets self-reporting flu during the 2009 H1N1 outbreak in the UK [48, 49].

2. **Model and preliminary results.** We develop the model on the basis of previous papers [10,30,31,44,45]. The population is divided into three groups: susceptible (S), exposed (E), and infected (I). All of them may tweet about influenza at the rates μ_1, μ_2, and μ_3, respectively, during an epidemic season. $T(t)$ represents the number of tweets that contain influenza at time t. We assume that the disease transmission rate, β, is reduced by a factor $e^{-\alpha T}$ due to the behavior change of the public after reading tweets about influenza, where α determines how effective the disease information can influence the transmission rate. This exponential term has been usually used to model the media effect. There are some other ways to describe this effect, which will be discussed later. We assume that individuals in the exposed class progress into the infectious stage after $1/\rho$ days, and that infectious individuals recover at the rate γ and gain permanent immunity to that strain of influenza. Tweets that appeared earlier are less visible and have less effect on the public. Thus, we assume that tweets become outdated at the rate τ. Because we are only interested in the disease outbreak during an epidemic season, we neglect the natural death and birth rates and assume that the number of susceptible people is relatively constant [30]. An additional equation describing the decline of susceptibles due to infection will be added to the model later. The model is described by the following system of differential equations. A schematic diagram of the model is shown in Figure 1.

$$\begin{aligned} \frac{d}{dt}E(t) &= \beta I S e^{-\alpha T} - \rho E, \\ \frac{d}{dt}I(t) &= \rho E - \gamma I, \\ \frac{d}{dt}T(t) &= \mu_1 S + \mu_2 E + \mu_3 I - \tau T. \end{aligned} \tag{1}$$

Using the next-generation method [50] or some other methods [29], we can obtain the basic reproductive number, $\Re_0$, which is the average number of new infected individuals caused by an infected person in a wholly susceptible population. We have the following matrix of new infection, F, and the matrix of transfer, V, both evaluated at the disease-free equilibrium $DFE = (0, 0, T_0)$, where $T_0 = \mu_1 S/\tau$.

$$F(DFE) = \begin{bmatrix} 0 & \beta S e^{-\alpha T_0} \\ 0 & 0 \end{bmatrix} \quad \text{and} \quad V(DFE) = \begin{bmatrix} \rho & 0 \\ -\rho & \gamma \end{bmatrix}.$$

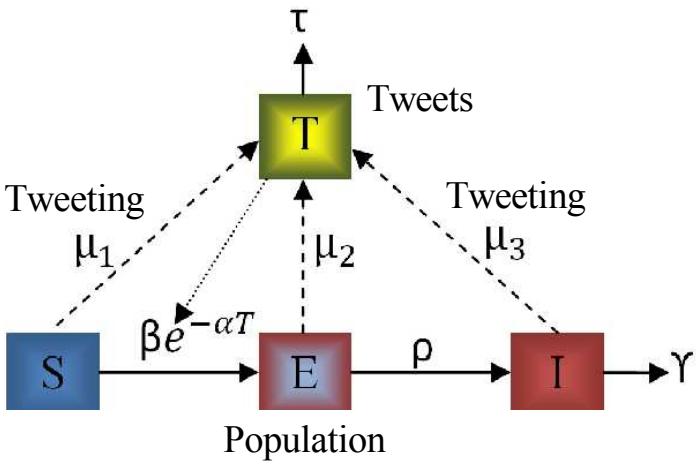

FIGURE 1. Schematic illustration of the model given by Eq. (1). Individuals from different groups can tweet about influenza at different rates (μ_1, μ_2 and μ_3). Tweets are assumed to reduce the disease transmission rate β by a factor $e^{-\alpha T}$.

Therefore, we have

$$F(DFE)V^{-1}(DFE) = \begin{bmatrix} \frac{\beta S e^{-\alpha T_0}}{\gamma} & \frac{\beta S e^{-\alpha T_0}}{\gamma} \\ 0 & 0 \end{bmatrix}.$$

The basic reproductive number of model (1) is given by the dominant eigenvalue of $F(DFE)V^{-1}(DFE)$, which is $\beta S e^{-\alpha T_0}/\gamma$. Thus, the basic reproductive number is

$$\Re_0 = \frac{\beta S e^{-\alpha T_0}}{\gamma}, \quad \text{where} \quad T_0 = \frac{\mu_1 S}{\tau}. \tag{2}$$

Model (1) has two possible equilibrium points: the disease-free equilibrium $DFE = (0, 0, T_0)$ and the endemic equilibrium $EE = (\bar{E}, \bar{I}, \bar{T})$, given by

$$\bar{E} = \frac{\gamma\Omega}{\alpha}, \quad \bar{I} = \frac{\rho\Omega}{\alpha}, \quad \bar{T} = \frac{\ln(\Re_0)}{\alpha} + T_0, \tag{3}$$

where

$$\Omega = \frac{\tau \ln(\Re_0)}{\rho\mu_3 + \mu_2\gamma} \quad \text{and} \quad T_0 = \frac{\mu_1 S}{\tau}. \tag{4}$$

It is clear that the endemic equilibrium point exists if and only if $\Re_0 > 1$.

3. **Model analysis.** Before we perform numerical simulations and compare modeling predictions with surveillance and Twitter data, we study the stability of the steady states of the model. We have the following results.

Theorem 3.1. *The DFE of model (1) is globally asymptotically stable when* $\Re_0 < 1$.

Proof. We first show that the DFE is locally asymptotically stable when $\Re_0 < 1$. We linearize the system, evaluate the Jacobian matrix at the DFE, and obtain the

characteristic equation

$$\det\left(\begin{bmatrix} -\rho-\zeta & \beta S e^{-\alpha T_0} & 0 \\ \rho & -\gamma-\zeta & 0 \\ \mu_2 & \mu_3 & -\tau-\zeta \end{bmatrix}\right) = 0,$$

where ζ is the eigenvalue.

One eigenvalue is $-\tau$ and others are determined by

$$(\zeta+\rho)(\zeta+\gamma) - \beta S\rho e^{-\alpha T_0} = 0.$$

From the expression of $\Re_0$ (Eq. 2), the above equation can be rewritten as

$$\zeta^2 + (\rho+\gamma)\zeta + \rho\gamma(1-\Re_0) = 0. \tag{5}$$

When $\Re_0 < 1$, the two roots of the above equation are negative. Thus, the DFE of model (1) is locally asymptotically stable.

To show the global asymptotic stability of the DFE, we define a Lyapunov function

$$M(t) = E(t) + I(t).$$

It is clear that $M(t) \geq 0$ and the equality holds if and only if $I(t) = E(t) = 0$.

From the T equation of model (1), we have $dT/dt \geq \mu_1 S - \tau T$. Using the result of differential inequalities [25], we have

$$\begin{aligned} T(t) &\geq T(0)e^{\int_0^t(-\tau)du} + \int_0^t \mu_1 S e^{\int_v^t(-\tau)du}dv \\ &= (T(0) - \frac{\mu_1 S}{\tau})e^{-\tau t} + \frac{\mu_1 S}{\tau}. \end{aligned}$$

Before the disease emerges, we assume that T is at the steady state level. Thus, $T(0)$ is $\frac{\mu_1 S}{\tau}$, which is equal to T_0 defined in Eq. (4). Therefore, $T(t) \geq \mu_1 S/\tau = T_0$, for all $t \geq 0$. Differentiating $M(t)$ and using $\beta S e^{-\alpha T_0} = \gamma\Re_0$, we obtain

$$\begin{aligned} \frac{dM}{dt} &= \frac{dE}{dt} + \frac{dI}{dt} = \beta I S e^{-\alpha T} - \rho E + \rho E - \gamma I \\ &= I(\beta S e^{-\alpha T} - \gamma) \leq I(\beta S e^{-\alpha T_0} - \gamma) \\ &= \gamma I(\Re_0 - 1) \leq 0 \end{aligned}$$

It follows that $M(t)$ is bounded and non-increasing. Therefore, $\lim_{t\to\infty} M(t)$ exists. Note that $\frac{\mathrm{d}M}{\mathrm{d}t} = 0$ if and only if that $E(t) = I(t) = 0$ and $T(t) = T_0$. By LaSalle's Invariance Principle [26], the DFE is globally attracting. Together with the local asymptotic stability, we show that the DFE is globally asymptotically stable when $\Re_0 < 1$. □

Theorem 3.2. *The endemic equilibrium (EE) of model (1) exists if and only if* $\beta > \beta_1$ *(i.e.,* $\Re_0 > 1$*), where* $\beta_1 = \dfrac{\gamma e^{\alpha\mu_1 S/\tau}}{S}$*. Moreover, the EE is locally asymptotically stable if (1)* $\mu_3 \leq \mu_3^*$ *or (2)* $\mu_3 > \mu_3^*$ *and* $\beta < \beta^*$*, where* $\mu_3^* = \dfrac{\mu_2(\rho+\tau)}{\rho}$ *and* β^* *is given by Eq. (9) below. When* $\beta > \beta^*$*, the EE is unstable. The results are illustrated in Figure 2.*

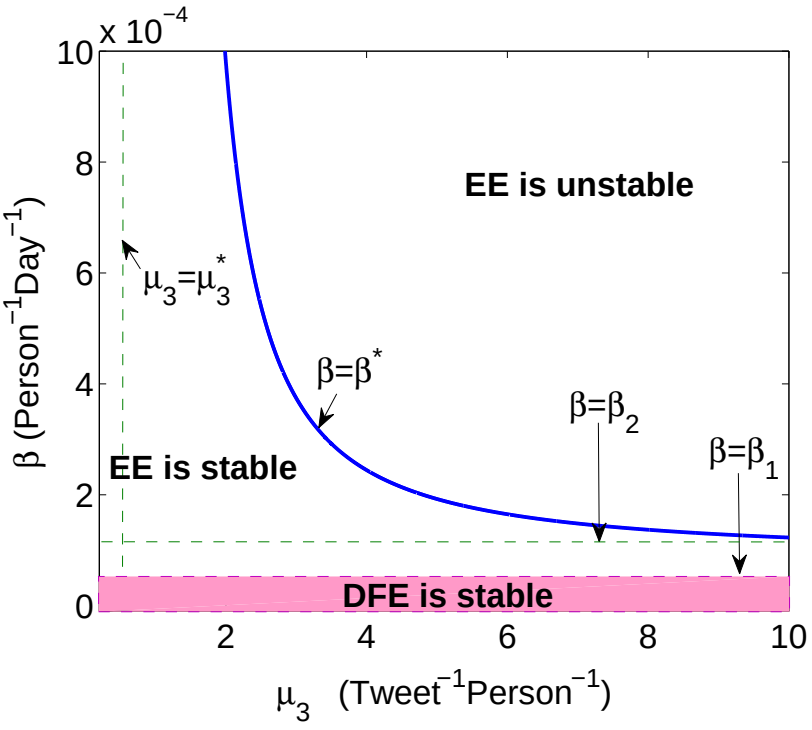

FIGURE 2. Schematic illustration of the stability of steady states of model (1). As $\mu_3 \to (\mu_3^*)^+$, $\beta^* \to +\infty$, where $\mu_3^* = \mu_2(\rho+\tau)/\rho$ and β^* is given in Eq. (9). As $\mu_3 \to +\infty$, $\beta^* \to \beta_2 > \beta_1$, where $\beta_1 = \gamma e^{\alpha\mu_1 S/\tau}/S$ and $\beta_2 = \gamma e^{(\rho+\gamma+\tau)(\rho+\gamma)/(\gamma\rho)} e^{\alpha\mu_1 S/\tau}/S$. Note that the endemic steady state exists if and only if $\beta > \beta_1$. Parameter values used in the simulation are $\rho = 1/6$ day^{-1}, $\gamma = 0.2$ day^{-1}, $\alpha = 1.0 \times 10^{-4}$ tweet^{-1}, $\mu_1 = 0$, and $\mu_2 = \mu_3 = \tau = 0.2$ day^{-1}.

Proof. Let $\beta_1 = \dfrac{\gamma e^{\alpha\mu_1 S/\tau}}{S}$. $\Re_0 > 1$ is equivalent to $\beta > \beta_1$. Thus, the endemic steady state exists if and only if $\beta > \beta_1$. To study the local stability of the EE, we linearize the system and evaluate the characteristic equation at the EE, and get

$$\det\left(\begin{bmatrix} -\rho-\zeta & \beta S e^{-\alpha\bar{T}} & -\alpha\beta\bar{I}Se^{-\alpha\bar{T}} \\ \rho & -\gamma-\zeta & 0 \\ \mu_2 & \mu_3 & -\tau-\zeta \end{bmatrix}\right) = 0.$$

Using $\Re_0 = \dfrac{\beta S e^{-\alpha T_0}}{\gamma}$ and $\bar{T} = \dfrac{\ln(\Re_0)}{\alpha} + T_0$, we have

$$\beta S e^{-\alpha\bar{T}} = \Re_0 \gamma e^{\alpha(T_0-\bar{T})} = \Re_0 \gamma e^{-\ln(\Re_0)} = \gamma.$$

Thus, the characteristic equation becomes

$$\det\left(\begin{bmatrix} -\rho-\zeta & \gamma & -\alpha\gamma\bar{I} \\ \rho & -\gamma-\zeta & 0 \\ \mu_2 & \mu_3 & -\tau-\zeta \end{bmatrix}\right) = 0.$$

Expanding the determinant, the characteristic equation can be rewritten as

$$P(\zeta) = \zeta^3 + a_1\zeta^2 + a_2\zeta + a_3, \tag{6}$$

where

$$\begin{aligned} a_1 &= \rho + \gamma + \tau, \\ a_2 &= \tau(\rho+\gamma) + \mu_2\alpha\gamma\bar{I} = \tau\left[\rho + \gamma + \frac{\mu_2\gamma\rho\ln(\Re_0)}{\rho\mu_3 + \gamma\mu_2}\right], \\ a_3 &= \gamma\alpha\bar{I}(\rho\mu_3 + \gamma\mu_2) = \gamma\rho\tau\ln(\Re_0). \end{aligned} \tag{7}$$

It is clear that $a_i > 0$, $i = 1, 2, 3$, when $\Re_0 > 1$. From the Routh–Hurwitz criteria, a_1a_2 needs to be greater than a_3 for all the roots of the polynomial $P(\zeta)$ to be negative or have negative real parts. Let $\Lambda = a_1a_2 - a_3$. Then

$$\begin{aligned} \Lambda &= \tau(\rho+\gamma+\tau)\left[\rho + \gamma + \frac{\mu_2\gamma\rho\ln(\Re_0)}{\rho\mu_3 + \gamma\mu_2}\right] - \gamma\rho\tau\ln(\Re_0) \\ &= \frac{\tau}{\rho\mu_3 + \gamma\mu_2}\Big[(\rho+\gamma+\tau)(\rho+\gamma)(\rho\mu_3+\gamma\mu_2) + (\rho+\gamma+\tau)\mu_2\gamma\rho\ln(\Re_0) \\ &\quad -\gamma\rho\ln(\Re_0)(\rho\mu_3+\gamma\mu_2)\Big] \\ &= \frac{\tau}{\rho\mu_3 + \gamma\mu_2}\Big[(\rho+\gamma+\tau)(\rho+\gamma)(\rho\mu_3+\gamma\mu_2) + \gamma\rho\ln(\Re_0)[\mu_2(\rho+\tau) - \rho\mu_3]\Big]. \end{aligned}$$

For $\Lambda > 0$ we need

$$H = (\rho+\gamma+\tau)(\rho+\gamma)(\rho\mu_3+\gamma\mu_2) + \gamma\rho\ln(\Re_0)[\mu_2(\rho+\tau) - \rho\mu_3] > 0.$$

Hence, the EE is locally asymptotically stable if and only if $H > 0$.

Let $\mu_3^* = \dfrac{\mu_2(\rho+\tau)}{\rho}$. When $\mu_2(\rho+\tau) - \rho\mu_3 \geq 0$ (i.e. $\mu_3 \leq \mu_3^*$), we have $H > 0$. When $\mu_3 > \mu_3^*$, for $H > 0$ the following inequality needs to be satisfied

$$\gamma\rho\ln(\Re_0) < \frac{(\rho+\gamma+\tau)(\rho+\gamma)(\rho\mu_3+\gamma\mu_2)}{\rho\mu_3 - \mu_2(\rho+\tau)}. \tag{8}$$

Using $\Re_0 = \dfrac{\beta S e^{-\alpha T_0}}{\gamma}$, we solve β from (8) and obtain $\beta < \beta^*$, where

$$\beta^* = \frac{\gamma}{S}e^{(\rho+\gamma+\tau)(\rho+\gamma)(\rho\mu_3+\gamma\mu_2)/[\gamma\rho(\rho\mu_3-\mu_2(\rho+\tau))]}e^{\alpha\mu_1 S/\tau}. \tag{9}$$

We consider β^* as a function of μ_3. It is easy to show that $\beta^* \to +\infty$ as $\mu_3 \to (\mu_3^*)^+$, where $\mu_3^* = \dfrac{\mu_2(\rho+\tau)}{\rho}$. As $\mu_3 \to +\infty$, $\beta^* \to \beta_2 > \beta_1$, where

$$\beta_2 = \frac{\gamma}{S}e^{(\rho+\gamma+\tau)(\rho+\gamma)/(\gamma\rho)}e^{\alpha\mu_1 S/\tau} \quad \text{and} \quad \beta_1 = \frac{\gamma}{S}e^{\alpha\mu_1 S/\tau}.$$

Therefore, we show that the EE is locally asymptotically stable if (1) $\mu_3 \leq \mu_3^*$ or (2) $\mu_3 > \mu_3^*$ and $\beta < \beta^*$. When $\beta > \beta^*$, the EE is unstable. These results are illustrated in Figure 2. This finishes the proof of Theorem 3.2. □

The function $\beta = \beta^*(\mu_3)$ defines a curve in the (β, μ_3) plane (Figure 2). Below the curve, the endemic steady state is locally asymptotically stable when it exists. Above the curve, the endemic steady state is unstable. The following theorem shows that a Hopf bifurcation occurs when β increases and the curve $\beta^*(\mu_3)$ is crossed.

Theorem 3.3. *A Hopf bifurcation occurs when β increases and the curve β^* is crossed, where β^* is defined in Eq. (9).*

Proof. All the coefficients of the third degree characteristic polynomial (6) are positive. Therefore, there are no positive roots of the polynomial and there exist at least one negative root. Suppose $P(\zeta)$ has a real root x and a pair of complex roots $a \pm bi$, where $x < 0$, and $a, b \in \mathbb{R}$. We have

$$\begin{aligned} P(\zeta) &= (\zeta - x)[\zeta - (a + bi)][\zeta - (a - bi)] \\ &= \zeta^3 - (2a + x)\zeta^2 + (a^2 + b^2 + 2ax)\zeta - x(a^2 + b^2). \end{aligned} \tag{10}$$

Comparing with Eq. (6), we get

$$a_1 = -(2a + x),\ a_2 = a^2 + b^2 + 2ax,\ a_3 = -x(a^2 + b^2),$$

where a_i, $i = 1, 2, 3$, are given in Eq. (7).

We are interested in possible sustained periodic oscillations. Thus, we explore the case when $P(\zeta) = 0$ has a pair of purely imaginary roots; i.e., $a = 0$. In this case, we have

$$a_1 = -x,\ a_2 = b^2,\ a_3 = -xb^2.$$

Thus, $a_1 a_2 = a_3$, which leads to $\beta = \beta^*$, as shown in the proof of Theorem 3.2. Therefore, the occurrence of a pair of purely imaginary roots corresponds to the threshold curve $\beta = \beta^*$.

To see how the real part of the eigenvalues $a \pm bi$ changes its sign, we check the transversality condition of the Hopf bifurcation. Substituting $a + bi$ into the characteristic equation (6), we have

$$P(a + bi) = 0.$$

Thus, $Re[P(a + bi)] = 0$, where Re denotes the real part of a complex number. Calculating $Re[P(a + bi)]$, we have

$$\begin{aligned} R &= Re[(a + bi)^3 + a_1(a + bi)^2 + a_2(a + bi) + a_3] \\ &= a^3 - 3ab^2 + a_1 a^2 - a_1 b^2 + a_2 a + a_3 \\ &= 0. \end{aligned} \tag{11}$$

From (7), we know that both a_2 and a_3 depend on β because $\Re_0$ contains β. Thus, R is a function of a and β. Therefore, $R(a, \beta) = 0$ defines an implicit function $a(\beta)$ with the independent variable β.

Differentiating R with respect to β, we have

$$\frac{\partial R}{\partial \beta} = 0,$$

which leads to

$$\frac{\partial R}{\partial a}\frac{\partial a}{\partial \beta} + \frac{\partial R}{\partial \beta} = 0.$$

Thus,

$$\frac{\partial a}{\partial \beta} = -\frac{\partial R}{\partial \beta} \Big/ \frac{\partial R}{\partial a}.$$

Next, we determine the sign of $\dfrac{\partial a}{\partial \beta}$ along the curve $\beta = \beta^*$. Noticing that in Eq. (11) only a_2 and a_3 depend on β, and that $a = 0$ and $a_2 = b^2$ on the curve $\beta = \beta^*$,

we have

$$\left.\frac{\partial R}{\partial \beta}\right|_{\beta=\beta^*} = \left.\left(a\frac{\partial a_2}{\partial \beta} + \frac{\partial a_3}{\partial \beta}\right)\right|_{\beta=\beta^*} = \left.\frac{\partial a_3}{\partial \beta}\right|_{\beta=\beta^*} = \frac{\gamma\rho\tau}{\beta^*} > 0$$

and

$$\left.\frac{\partial R}{\partial a}\right|_{\beta=\beta^*} = 3a^2 - 3b^2 + 2a_1 a + a_2\big|_{\beta=\beta^*} = -2b^2 < 0.$$

Thus, we have

$$\left.\frac{\partial a}{\partial \beta}\right|_{\beta=\beta^*} = -\left.\frac{\partial R}{\partial \beta}\right/\frac{\partial R}{\partial a}\right|_{\beta=\beta^*} > 0.$$

This shows that when β increases and crosses the curve $\beta = \beta^*$, a Hopf bifurcation occurs. This completes the proof of Theorem 3.3. □

The results in Theorems 3.2 and 3.3 are summarized in Figure 2. The biological explanation of these results is as follows. When the transmission rate (β) is small, the infection cannot be established and the disease-free steady state is stable. For a large β, when the rate of tweeting (μ_3) by infectious people is small, the endemic steady state is stable whenever it exists. This is expected because a small tweeting rate generates a small number of tweets, which have less media effect on reducing the transmission rate. Thus, the infection can be established and the endemic steady state is stable. As the tweeting rate μ_3 increases, the influence of media also increases, which reduces the disease transmission and undermines the stability of the endemic steady state. When the curve $\beta = \beta^*(\mu_3)$ is crossed, a threshold condition between the transmission rate and the media influence is reached. In this scenario, increasing I generates more tweets, which reduce E and thus reduce I. On the contrary, reducing I generates few tweets, which increase E and thus increase I. This leads to a periodic system.

In the above model and analysis, we assume that the number of susceptibles remains at a constant level. The number of susceptibles may be significantly reduced due to infection. If we ignore the natural birth and death of susceptibles during an epidemic, the dynamics of $S(t)$ can be described by the following equation

$$\frac{d}{dt}S(t) = -\beta I S e^{-\alpha T}. \tag{12}$$

The remaining equations are the same as Eq. (1). The new model has two possible steady states: one is trivial (all the variables are 0) and the other is the disease-free equilibrium $(S_0, 0, 0, \mu_1 S_0/\tau)$, where S_0 is the initial value of susceptibles. The analysis of the second steady state is almost the same as our above analysis for the disease-free equilibrium.

4. Numerical results.

We choose some parameter values on the basis of available data. The incubation time for the 2009 H1N1 influenza pandemic was reported to be between 2 and 10 days with a mean of 6 days [8, 34]. Thus, we assume that people in the exposed group move to the infectious class at a rate $\rho = 1/6$ day^{-1}. The infectious period was estimated to be between 4 and 7 days with a mean of 5 days [28, 34]. Thus, we choose the recovery rate to be $\gamma = 0.2$ day^{-1}. The susceptible population size S is set to 1 million and initially 10 people get exposed to the disease [34]. The other parameters are chosen to illustrate the theoretical results. In the next section, we will fit model predictions to some surveillance and Twitter data and estimate parameter values based on the fitting.

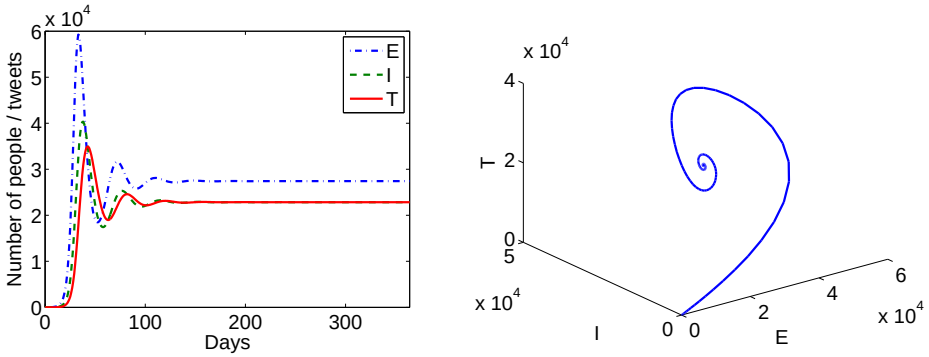

FIGURE 3. The endemic steady state of model (1) is stable for parameter values satisfying the conditions in Theorem 3.2. The parameter values are $\beta = 1.6 \times 10^{-6}$ person^{-1}day^{-1}, $\mu_1 = \mu_2 = 0$, $\mu_3 = 0.2$ day^{-1}, $\tau = 0.2$ day^{-1}, $\alpha = 9.1 \times 10^{-5}$ tweet^{-1}, $\rho = 1/6$ day^{-1}, and $\gamma = 0.2$ day^{-1}.

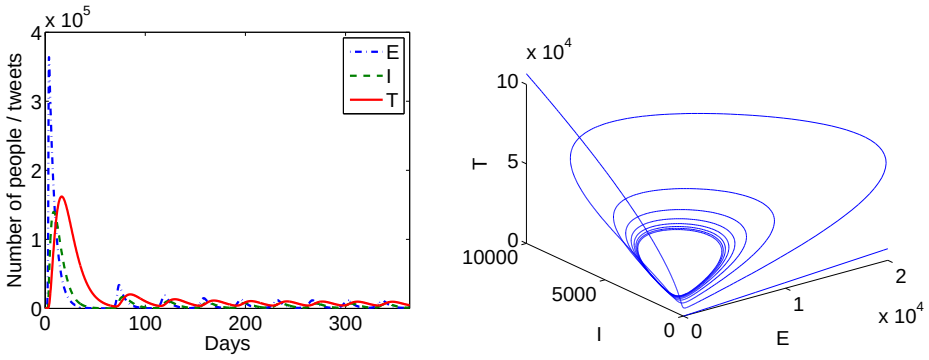

FIGURE 4. The system (Eq. 1) converges to a sustained periodic solution for parameter values satisfying the conditions in Theorem 3.3. The parameter values are $\beta = 8.1 \times 10^{-5}$ person^{-1}day^{-1}, $\mu_1 = \mu_2 = 0$, $\mu_3 = 0.2$ day^{-1}, $\tau = 0.1$ day^{-1}, $\alpha = 0.001$ tweet^{-1}, $\rho = 1/6$ day^{-1}, and $\gamma = 0.2$ day^{-1}.

In Figures 3 and 4, we perform numerical simulations to illustrate the results showed in Theorems 3.2 and 3.3. For simplicity, we assume $\mu_1 = \mu_2 = 0$ and $\mu_3 > 0$; i.e., only infectious individuals report or tweet about influenza as they are more likely to share the disease status with their followers on Twitter. We demonstrate that the endemic steady state of the model is stable (Figure 3) with the parameter values satisfying the assumption in Theorem 3.2. The numbers of exposed and infectious individuals and the number of tweets converge to their steady states after damped oscillations. In Figure 4, we show that the system converges to a sustained periodic solution. In both simulations, the peak of infectious individuals appears earlier than the peak of tweets. This is not surprising because tweets self-reporting disease are assumed to be generated by infectious people. In reality, disease outbreaks according to the influenza-like illness data are usually 1 to 2 weeks later than the real-time assessment provided by Twitter [48]. This time delay may

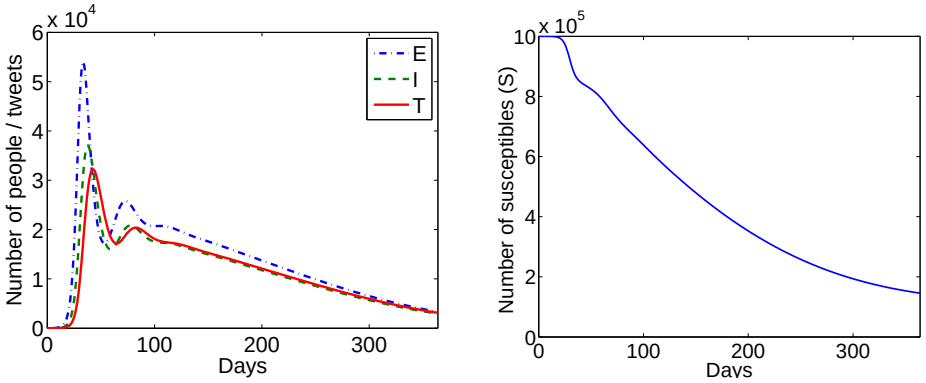

FIGURE 5. Simulation of the model that includes time-varying susceptibles (Eq. 12). The parameter values are the same as those in Figure 3.

be due to the time needed for data collection and processing. We will account for this time delay when fitting model predictions to surveillance and Twitter data in the next section.

To demonstrate the effect of time-varying susceptibles on the disease dynamics, we perform a simulation of the model that includes the dynamics of susceptible (Eq. 12) and plot the dynamics in Figure 5. We choose the initial value of susceptibles to be 1 million, which is the same as the number of susceptibles fixed in model (1). The other parameter values and initial conditions are the same as those in Figure 3. As expected, the number of susceptibles is reduced substantially during the simulation over 1 year (Figure 5). Exposed individuals, infectious individuals and tweets in the early stage have similar dynamics to those predicted in Figure 3 but have a lower level because of a reduced number of susceptibles (Figure 5). Eventually the disease is predicted to die out. This is in agreement with the theoretical result.

We also evaluate the influence of Twitter on the dynamics of the disease. We compare the dynamics of infectious individuals when exposed individuals (Figure 6A) or infectious individuals (Figure 6B) post on average 0.5, 1, or 2 tweets about the disease per day. The simulation shows that the more tweets posted per day, the lower infectious cases. Changing the number of tweets posted per day does not affect the time when the epidemic reaches the peak. In Figure 6C, we compare the dynamics with different rates at which tweets reduce the disease transmission rate. Increasing this rate significantly reduces the spread of the disease. In Figure 6D, we study the dynamics assuming tweets become outdated after 10, 5, and 2 days ($\tau = 0.1, 0.2,$ and 0.5 per day, respectively). A longer effect of tweets significantly reduces the number of infectious cases.

In Figure 7, we compare the dynamics of infectious individuals between three cases: all susceptible (dotted line) or exposed (dashed line) or infectious (solid line) people post 1 tweet about the disease per day. We found that the epidemic is significantly suppressed if only susceptible people tweet about the disease. This is not surprising because the fixed number of susceptible people is much larger than the other population groups considered in this model. It also shows that the disease cannot be well controlled if only the infectious people with symptoms tweet about flu on the social networking site.

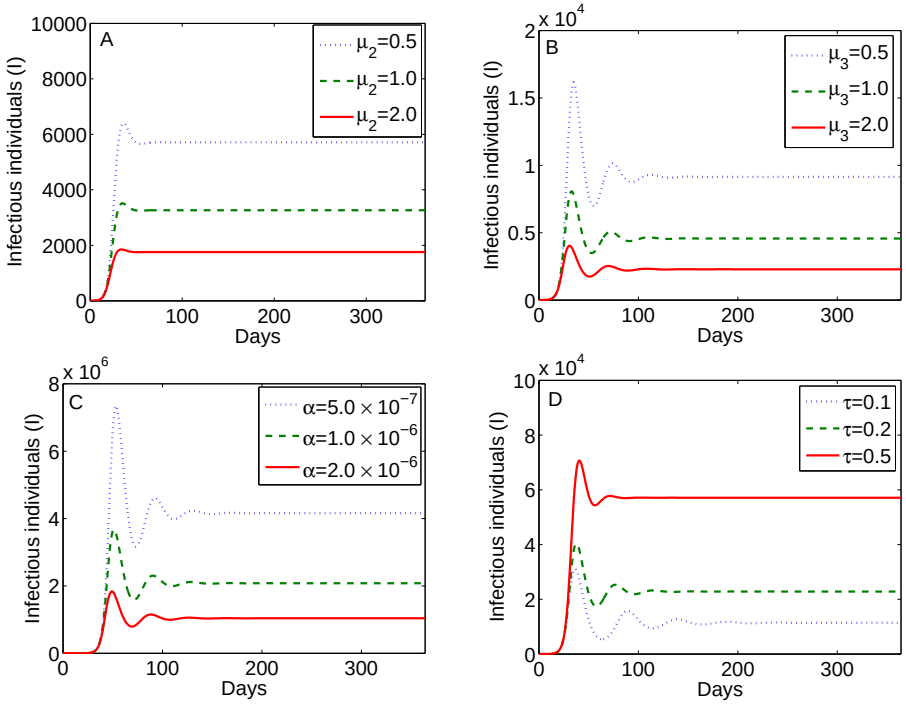

FIGURE 6. The effect of tweet-related parameters on the dynamics of infectious individuals. The parameter that varies is indicated in each figure and the remaining parameter values are the same as those in Figure 3.

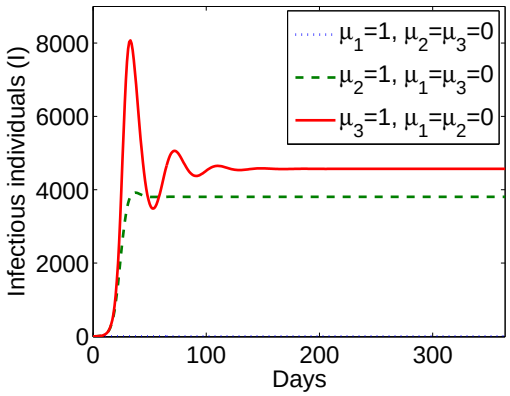

FIGURE 7. The effect of tweets posted by different population groups on the disease dynamics. The other parameter values are the same as those in Figure 3.

5. **Comparison with surveillance and Twitter data.** The surveillance data of flu were retrieved from the UK Health Protection Agency, which provides weekly reports on the influenza-like illness (ILI) consultation rate for England and Wales,

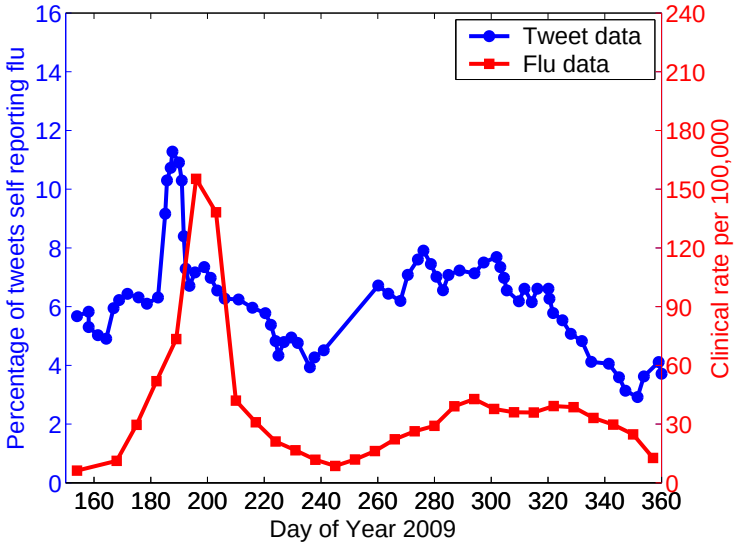

FIGURE 8. The flu and tweet data collected by Szomszor et al. [48]. The red square points represent the influenza-like illness consultation rate and the blue circle points represent the percentage of tweets that are self-reporting flu between May and December 2009.

Scotland, and Northern Ireland. Szomszor et al. [48] collected about 3 million tweets between May and December 2009 from the same area. They filtered each tweet and searched for phrases that indicate the user may have the flu. These phrases include "have flu", "have the flu", "have swine flu", and "have the swine flu" in present and past tenses. With this information, they calculated the percentage of tweets that were self-reporting flu each day in the investigation period. In Figure 8, we showed the data of the ILI consultation rate in England and Wales, as well as the the percentage of tweets self-reporting flu in 2009. Although some users who posted flu tweets may not have flu, the cross-correlation between tweets and the surveillance data suggested a strong correlation between them [48]. We will compare our model predictions with both the tweet and surveillance data showed in Figure 8.

Because the tweet data are given as the percentage of tweets self-reporting flu, we let $\mu_1 = \mu_2 = 0$ and $\mu_3 > 0$; i.e., only infectious individuals are tweeting reporting their disease. In addition, only the percentage of tweets reporting flu and the clinical rate per 100,000 are available. Thus, we used two positive constants, c_1 and c_2, to rescale T and I in our model to the percentage of tweets and clinical rate, respectively. Further, there is usually a 1–2 week delay between the time when a patient is infected and the moment when the data become available in ILI reports. Therefore, we compared $c_1T(t)$ with the tweet data and $c_2I(t-\sigma)$ with the surveillance data at time t, where σ represents the 1–2 week delay mentioned above. Lastly, because of the relatively large magnitude of the flu data (clinical rate per 100,000; see Figure 8) compared with the percentage of tweets self-reporting flu, we divided the flu data values by 10 and rescaled them to clinical cases per 10,000. After this rescaling, all the flu data values are between 0 and 16, comparable to the tweet data. Therefore, equal weights for both tweet and flu data were employed in

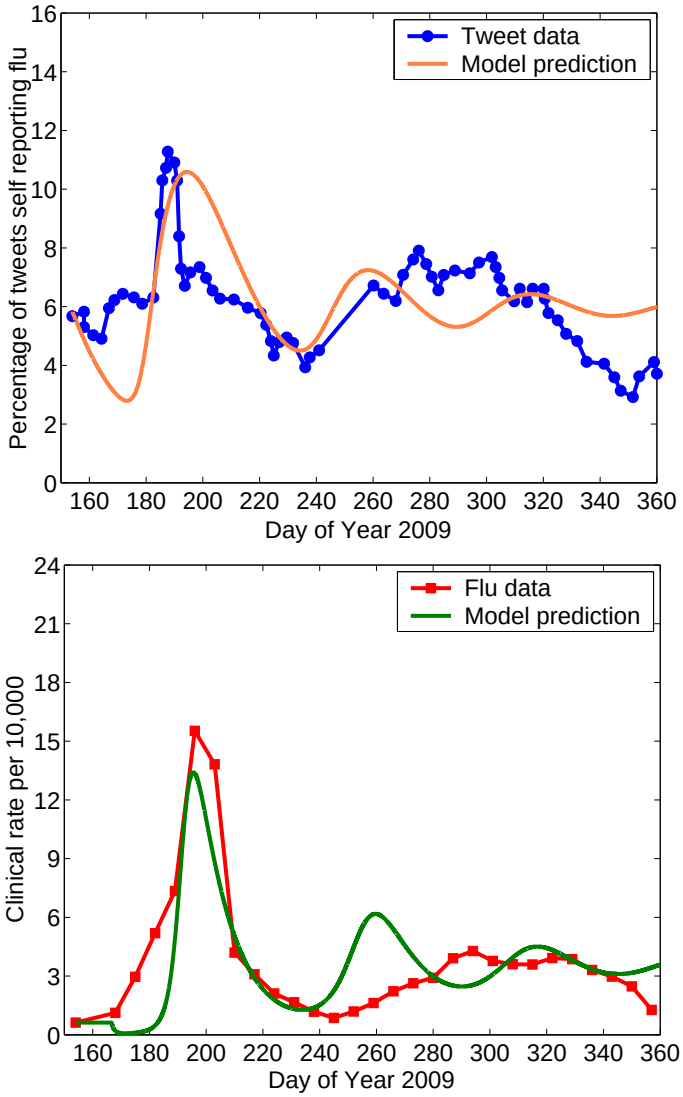

FIGURE 9. Data fits of model (1) to the Twitter data (the upper panel) and the flu data (the lower panel). Parameter values on the basis of the fits are $\beta = 3.7\times10^{-5}$ person^{-1} day^{-1}, $\mu_3 = 0.8$ day^{-1}, $\tau = 0.05$ day^{-1}, $\gamma = 0.9$ day^{-1}, $\rho = 0.09$ day^{-1}, $\alpha = 4.5 \times 10^{-6}$ tweet^{-1}, $c_1 = 7.3 \times 10^{-6}$, $c_2 = 7.2 \times 10^{-5}$, and the delay σ is 12.7 days.

data fitting. Using different weights generates a similar fit, although the estimates of parameter values are slightly different.

Using model (1), we fit $c_1T(t)$ to the tweet data and $c_2I(t-\sigma)$ to the surveillance data simultaneously. For the ease of illustration, we presented the fits in two panels in Figure 9. The upper panel shows the fit to the tweet data and the lower panel shows the fit to the surveillance data. The model can capture the peaks of both

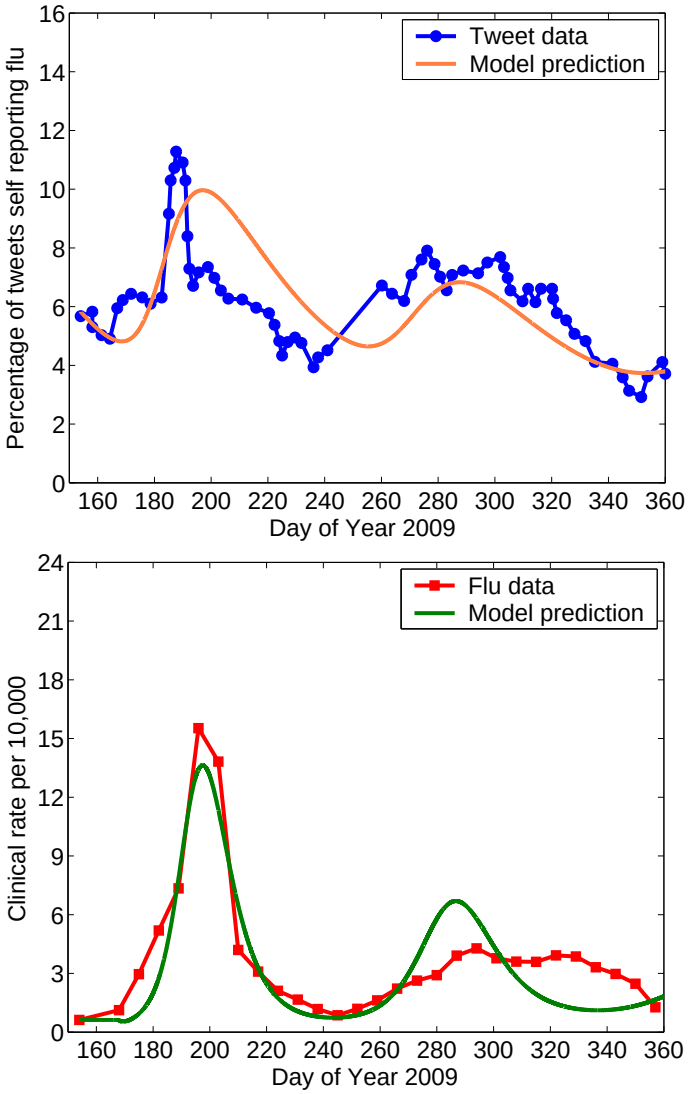

FIGURE 10. Data fits of the model including time-varying susceptibles (Eq. 12) to the Twitter data (the upper panel) and the flu data (the lower panel). Parameter values on the basis of the fits are $\beta = 2.6 \times 10^{-6}$ person^{-1} day^{-1}, $\mu_3 = 0.2$ day^{-1}, $\tau = 0.02$ day^{-1}, $\gamma = 0.18$ day^{-1}, $\rho = 0.38$ day^{-1}, $\alpha = 1.1 \times 10^{-5}$ tweet^{-1}, $c_1 = 3.3 \times 10^{-5}$, $c_2 = 1.8 \times 10^{-4}$, and the delay σ is 13.8 days.

infectious cases and tweets self-reporting flu, although it predicts more oscillations than the observation. Parameter values on the basis of the fit are given in the caption of Figure 9. The time delay between the disease outbreak and the ILI report, σ, is estimated to be 12.7 days, which agrees with the empirical estimate. The initial flat phase in the prediction of flu in the lower panel is due to this time delay. The initial decrease in the number of tweets predicted in the upper panel is

because of the low level of infectious cases. Many tweets become outdated before infectious people post messages about their disease on Twitter.

We also fit the model that includes the dynamics of susceptibles (Eq. 12) to the same tweet and surveillance data (Figure 10). The fits are similar to those in Figure 9 but has less oscillations. The parameter estimates based on the fitting are different. We cannot determine which fit is better from the statistical viewpoint.

6. **Conclusion and discussion.** Although the ILI data are useful in studying influenza epidemics, they are associated with a high cost and a slow reporting time. Tracking and predicting the outbreak of infectious diseases has recently become possible on the basis of internet activities. Ginsberg et al. [18] showed that the level of influenza activity in a region can be estimated using the proportion of flu-related queries submitted to the Google search engine over the same time period. Social networking sites like Twitter have also served as excellent platforms for providing timely and more descriptive information about the disease. Symptoms and disease status reported by Twitter users could be useful in detecting and even predicting the outbreak of the disease [11].

Several studies have investigated the relationship between Twitter messages and the ILI data. A good example is the 2009 H1N1 influenza outbreak. Approximately 3 million tweets containing the term "flu" between May and December 2009 were collected to determine the spread of the infection [48]. A simple comparison of tweets with the weekly rate of ILI consultation measured by the UK Health Protection Agency revealed a strong correlation between the number of tweets and flu cases. Similarly, Signorini et al. used flu-related tweets to predict the ILI cases reported by the CDC [42]. They analyzed over 4 million tweets containing various flu-related terms from October to December in 2009 and found a similarity between the tweet pattern and CDC reports. In another study [9], over 2 million tweets containing the terms "swine flu", "swineflu", or "H1N1" were archived between May and December in 2009. Topic-related tweets increased from 8.8% to 40.5% in the meantime. Content analysis indicated that tweet data correlated well with the H1N1 incidence data reported by the World Health Organization. There is some other related work in the literature [2, 4, 11, 22]. These results highlight Twitter's role as an informational tool that can be used for real-time content analysis and knowledge translation research.

The aim of this study is to assess how the messages posted on Twitter can influence public risk perception and influenza epidemics. We developed a simple mathematical model which explicitly includes the dynamics of tweets. We assumed that the transmission rate of flu between individuals is reduced because of the behavior change caused by reading tweets about flu. We defined the basic reproductive ratio $\Re_0$ and showed that it plays an important role in determining the stability of the steady states of the model. Specifically, the disease-free steady state of the model is globally asymptotically stable when $\Re_0 < 1$. The endemic steady state is locally asymptotically stable when the transmission rate is less than a threshold value. When the threshold is crossed, a Hopf bifurcation occurs (see Figure 2) and sustained periodic solutions appear. Numerical simulations of the model illustrated our theoretical results. Sensitivity tests on a few parameters related to tweets, such as the impact of tweets on reducing the infection force and the rates at which exposed and infected individuals tweet about influenza, show that Twitter can greatly influence the emergence and spread of the disease. Before the solution

of the model converges to the endemic steady state, there exhibit damped (or even periodic) oscillations. This suggests the possibility of multiple outbreaks of flu during an epidemic season. The periodicity of the disease may also be caused by cycles of Twitter like other news media. If the decline of susceptibles due to infection is taken into account, then the disease is predicted to die out after a few damped oscillations (Figure 5).

We also compared the modeling predictions with both the tweet and surveillance data during the 2009 H1N1 outbreak [48]. We found that the model can capture the peaks of both the percentage of tweets self-reporting flu and the surveillance data given by the ILI consultation rate. Because we only have data on tweets that self report flu, we assume only infectious people post their flu status on Twitter in data fitting. With this assumption, we find the peak of tweets emerges later than the peak of infectious people. This is expected because tweets are assumed to be generated by these infected people. In the simulation, even when we choose a large rate μ_1 (the rate at which susceptible people tweet about flu), we still predict that the two peaks appear at approximately the same time. Because the peak of flu-related tweets is usually one to two weeks earlier than the outbreak of influenza reported by the national surveillance data, it was thought that Twitter might be used as an early warning detection system [48]. Our results suggest that this may not be true. The time delay between Twitter messages and national ILI data is because of the time needed for data collection and processing. Thus, Twitter can serve as a good real-time assessment of the current epidemic condition rather than an early warning detection system. However, tweet data may be used to compensate for the lack of surveillance data and also help predict flu trends ahead of the report by public-health authorities.

It should be noted that Twitter users are only a subset of susceptibles. However, the model is still valid because the transmission rate (β) and the generation rate (μ_1) of tweets by susceptibles can be assumed to have already included the ratio of susceptibles who use Twitter to all the susceptibles. In the model, we also ignored the natural death and birth rate of susceptibles. This assumption may not be valid if the duration of an epidemic is long [30]. We have included a case of declining susceptibles due to infection in Eq. (12). The model can be extended to include a more complicated equation for susceptible people [10, 44]. Whether the modeling predictions are the same remains to be investigated. In addition, we used an exponential term to describe the effect of Twitter messages on reducing the transmission rate. This has been usually used to model the impact of media on the dynamics of infectious diseases. Some other ways were also used to describe this effect. For example, a linear combination of $I(t)$ (infectious people) and its derivative was used in the exponential term in that either the number of cases or a significant change in the number of cases may affect the media coverage and change the transmission rate [44]. In another study [45], a saturation function (proportional to $I(t)/[C + I(t)]$ where C is the half-saturation constant) was subtracted from the transmission rate to take into account the saturation effect of media coverage on disease spread. Lastly, we did not consider the possible differences in Twitter users from different age groups in the model. An age-based flu prediction analysis indicates that, for most regions, Twitter data best fit the age groups of 5–24 and 25–49 years, correlating well with the fact that they are likely the most active user age groups on Twitter [2]. Incorporating different age groups and geographical factors

into the model may provide more insights into the effect of Twitter on influenza epidemics.

In summary, we show that social networking tools such as Twitter may have substantial influence on the dynamics of influenza virus infection during an epidemic season. It is crucial to educate the public through social media about non-pharmaceutical interventions that can reduce the probability of becoming infected. Although Twitter may not serve as an early warning system, it can provide a good real-time assessment of the current disease condition ahead of public-health authorities. This will provide more time for various interventions to contain the epidemic or pandemic.

Acknowledgments. The research of Pawelek was supported by the ASPIRE and RISE grants from the Office of the Vice President for Research at the University of South Carolina, University of South Carolina Magellan Scholar Program, and SC EPSCoR/IDeA Scientific Advocate Network. The research of Rong was supported by the NSF grant DMS-1122290 and Oakland University Research Excellence Fund. We thank two reviewers whose comments improved this manuscript.

REFERENCES

[1] http://www.statisticbrain.com/twitter-statistics.
[2] H. Achrekar, A. Gandhe, R. Lazarus, S. Yu and B. Liu, Twitter improves seasonal influenza prediction, *HEALTHINF, SciTePress*, (2012), 61–70.
[3] M. Ajelli, S. Merler, A. Pugliese and C. Rizzo, Model predictions and evaluation of possible control strategies for the 2009 A/H1N1v influenza pandemic in Italy, *Epidemiol. Infect.*, **139** (2011), 68–79.
[4] E. Aramaki, S. Maskawa and M. Morita, Twitter catches the flu: Detecting influenza epidemics using Twitter, *Proceedings of the 2011 Conference on Empirical Methods in Natural Language Processing*, (2011), 1568–1576.
[5] M. Baguelin, A. J. Hoek, M. Jit, S. Flasche, P. J. White and W. J. Edmunds, Vaccination against pandemic influenza A/H1N1v in England: A real-time economic evaluation, *Vaccine*, **28** (2010), 2370–2384.
[6] D. Balcan, H. Hu, B. Goncalves, P. Bajardi, C. Poletto, J. J. Ramasco, D. Paolotti, N. Perra, M. Tizzoni, W. V. Broeck, V. Colizza and A. Vespignani, Seasonal transmission potential and activity peaks of the new influenza A(H1N1): A Monte Carlo likelihood analysis based on human mobility, *BMC Medicine*, **7** (2009), 45.
[7] J. Cain, F. Romanelli and B. Fox, Pharmacy, social media, and health: Opportunity for impact, *J. Am. Pharm. Assoc.*, **50** (2010), 745–751.
[8] Centers for Disease Control and Prevention (CDC), Interim guidance for clinicians on identifying and caring for patients with swine-origin influenza A (H1N1) virus infection, (2009).
[9] C. M. Chew and G. Eysenbach, Pandemics in the age of Twitter: Content analysis of tweets during the 2009 H1N1 outbreak, *PLoS One*, **5** (2010), e14118.
[10] J. Cui, Y. Sun and H. Zhu, The impact of media on the control of infectious diseases, *J. Dyn. Differ. Equ.*, **20** (2008), 31–53.
[11] A. Culotta, Towards detecting influenza epidemics by analyzing Twitter messages, *Proceedings of the First Workshop on Social Media Analytics*, (2012), 115–122.
[12] D. Currie, Public health leaders using social media to convey emergencies: New tools a boon, *The Nation's Health*, **39** (2009), 1–10.
[13] L. Donelle and R. G. Booth, Health tweets: an exploration of health promotion on twitter, *Online J. Issues Nurs.*, **17** (2012), manuscript 4.
[14] N. M. Ferguson, D. A. Cummings, S. Cauchemez, C. Fraser, S. Riley, A. Meeyai, S. Iamsirithaworn and D. S. Burke, Strategies for containing an emerging influenza pandemic in Southeast Asia, *Nature*, **437** (2005), 209–214.
[15] N. M. Ferguson, D. A. Cummings, C. Fraser, J. C. Cajka, P. C. Cooley and D. S. Burke, Strategies for mitigating an influenza pandemic, *Nature*, **442** (2006), 448–452.

[16] S. Funk, M. Salathe and V. A. Jansen, Modelling the influence of human behaviour on the spread of infectious diseases: A review, *J. R. Soc. Interface*, **7** (2010), 1247–1256.
[17] S. Funk, E. Gilad, C. Watkins and V. A. Jansen, The spread of awareness and its impact on epidemic outbreaks, *PNAS*, **106** (2009), 6872–6877.
[18] J. Ginsberg, M. H. Mohebbi, R. S. Patel, L. Brammer, M. S. Smolinski and L. Brilliant, Detecting influenza epidemics using search engine query data, *Nature*, **457** (2009), 1012–1014.
[19] M. Z. Gojovic, B. Sander, D. Fisman, M. D. Krahn and C. T. Bauch, Modelling mitigation strategies for pandemic (H1N1) 2009, *CMAJ*, **181** (2009), 673–680.
[20] M. E. Halloran, N. M. Ferguson, S. Eubank, I. M. Longini, Jr., D. A. Cummings, B. Lewis, S. Xu, C. Fraser, A. Vullikanti, T. C. Germann, D. Wagener, R. Beckman, K. Kadau, C. Barrett, C. A. Macken, D. S. Burke and P. Cooley, Modeling targeted layered containment of an influenza pandemic in the United States, *PNAS*, **105** (2008), 4639–4644.
[21] N. Heaivilin, B. Gerbert, J. E. Page and J. L. Gibbs, Public health surveillance of dental pain via Twitter, *J. Dent. Res.*, **90**(9) (2011), 1047–1051.
[22] H. Hirose and L. Wang Prediction of infectious disease spread using Twitter: A case of influenza, *The Fifth International Symposium on Parallel Architectures, Algorithms and Programming*, (2012), 100–105.
[23] C. Holt, Emerging technologies: Web 2.0, *Health Information Management Journal*, **40** (2011), 33–35.
[24] A. Java, X. Song, T. Finin and B. Tseng, Why we Twitter: Understanding Microblogging Usage and Communities, in *Proceedings of the 9th WebKDD and 1st SNA-KDD 2007 Workshop on Web Mining and Social Network Analysis*, New York, ACM Press, 2007, 56–65.
[25] V. Lakshmikantham, S. Leela and A. A. Martynyuk, *Stability Analysis of Nonlinear Systems*, Marcel Dekker, Inc., New York and Basel, 1989.
[26] J. P. LaSalle, *The Stability of Dynamical Systems*, Regional Conference Series in Applied Mathematics, SIAM, Philadelphia, 1976.
[27] B. Y. Lee, S. T. Brown, G. W. Korch, P. C. Cooley, R. K. Zimmerman, W. D. Wheaton, S. M. Zimmer, J. J. Grefenstette, R. R. Bailey, T. M. Assi and D. S. Burke, A computer simulation of vaccine prioritization, allocation, and rationing during the 2009 H1N1 influenza pandemic, *Vaccine*, **28** (2010), 4875–4879.
[28] S. Leekha, N. L. Zitterkopf, M. J. Espy, T. F. Smith, R. L. Thompson and P. Sampathkumar, Duration of influenza A virus shedding in hospitalized patients and implications for infection control, *Infect. Cont. Hosp. Ep.*, **28** (2007), 1071–1076.
[29] J. Li, D. Blakeley and R. J. Smith?, The failure of R_0, Computational and Mathematical Methods in Medicine, (2011), Art. ID 527610, 17 pp.
[30] R. Liu, J. Wu and H. Zhu, Media/psychological impact on multiple outbreaks of emerging infectious diseases, *Comput. Math. Methods Med.*, **8** (2007), 153–164.
[31] Y. Liu and J. Cui, The impact of media coverage on the dynamics of infectious disease, *Int. J. Biomath.*, **1** (2008), 65–74.
[32] I. M. Longini, Jr., M. E. Halloran, A. Nizam and Y. Yang, Containing pandemic influenza with antiviral agents, *Am. J. of Epidemiol.*, **159** (2004), 623–633.
[33] P. Manfredi, P. D. Posta, A. d'Onofrio, E. Salinelli, F. Centrone, C. Meo and P. Poletti, Optimal vaccination choice, vaccination games, and rational exemption: An appraisal, *Vaccine*, **28** (2009), 98–109.
[34] S. M. Tracht, S. Y. Del Valle and J. M. Hyman, Mathematical modeling of the effectiveness of facemasks in reducing the spread of novel influenza A (H1N1), *PLoS One*, **5** (2011), e9018.
[35] S. Merler, M. Ajelli and C. Rizzo, Age-prioritized use of antivirals during an influenza pandemic, *BMC Infect. Dis.*, **9** (2009), 117.
[36] A. Mitchell, T. Rosenstiel and L. Christian, What Facebook and Twitter mean for news, Pew Research Center's Project for Excellence in Journalism, (2012). Available from: http://stateofthemedia.org/2012/mobile-devices-and-news-consumption-some-good-signs-for-journalism/what-facebook-and-twitter-mean-for-news/.
[37] L. Odden, Top 10 uses of Twitter, Online Marketing Blog, (2008). Available from: http://www.toprankblog.com/2008/05/top-10-twitter-uses/.
[38] P. Poletti, M. Ajelli and S. Merler, Risk perception and effectiveness of uncoordinated behavioral responses in an emerging epidemic, *Math. Biosci.*, **238** (2012), 80–89.

[39] P. Poletti, B. Caprile, M. Ajelli, A. Pugliese and S. Merler, Spontaneous behavioural changes in response to epidemics, *J. Theoret. Biol.*, **260** (2009), 31–40.
[40] T. C. Reluga, Game theory of social distancing in response to an epidemic, *PLoS Comput. Biol.*, **6** (2010), e1000793.
[41] D. Scanfeld, V. Scanfeld and E. L. Larson, Dissemination of health information through social networks: Twitter and antibiotics, *Am. J. Infect. Control*, **38** (2010), 182–188.
[42] A. Signorini, A. M. Segre and P. M. Polgreen, The use of Twitter to track levels of disease activity and public concern in the U.S. during the influenza A H1N1 pandemic, *PLoS One*, **6** (2011), e19467.
[43] A. Smith and J. Brenner, Twitter use 2012, *Pew Internet and American Life Project*, (2012). Available from: `http://www.pewinternet.com/Reports/2012/Twitter-Use-2012`.
[44] J. M. Tchuenche, and C. T. Bauch, Dynamics of an infectious disease where media coverage influences transmission, *ISRN Biomathematics*, **2012** (2012), 1–10.
[45] 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*, **11** (2011).
[46] V. Tweedle and R. J. Smith, A mathematical model of Bieber Fever: The most infectious disease of our time?, in *Understanding the Dynamics of Emerging and Re-Emerging Infectious Diseases Using Mathematical Models* (eds. S. Mushayabasa and C. P. Bhunu), 2012, 157–177.
[47] S. J. Sullivan, A. G. Schneiders, C. W. Cheang, E. Kitto, H. Lee, J. Redhead, S. Ward, O. H. Ahmed and P. R. McCrory, 'What's happening?' A content analysis of concussion-related traffic on Twitter, *Brit. J. Sports Med.*, **46** (2012), 258–263.
[48] M. Szomszor, P. Kostkova and E. de Quincey, Swineflu: Twitter predicts Swine Flu outbreak in 2009, in *Electronic Healthcare*, Lecture Notes of the Institute for Computer Sciences, Social Informatics and Telecommunications Engineering, 69, Springer Berlin Heidelberg, 2010, 18–26.
[49] UK Health Protection Agency (HPA), `https://www.hpa.org.uk/`, 2010.
[50] P. van den Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.*, **180** (2002), 29–48.
[51] J. van Dijck, Tracing Twitter: The rise of a microblogging platform, *Int. J. of Media Cultural Polit.*, **7** (2012), 333–348.
[52] K. Vance, W. Howe and R. P. Dellavalle, Social Internet sites as a source of public health information, *Dermatol. Clin.*, **27** (2009), 133–136.
[53] M. Wazny, Using viral marketing in campaigns supporting health promotion, *Proceedings of the 13th World Congress on Public Health*, (2012).
[54] J. T. Wu, S. Riley, C. Fraser and G. M. Leung, Reducing the impact of the next influenza pandemic using household-based public health interventions, *PLoS Med.*, **3** (2006), e361.

Received December 16, 2013; Accepted June 18, 2014.

E-mail address: `kpawelek@uscb.edu`
E-mail address: `anneo@uconn.edu`
E-mail address: `rong2@oakland.edu`