A Dual SIS Epidemic Model for Virus Spread Analysis in Cluster-Based Wireless Sensor Networks

Shensheng Tang[1(✉)] and Chenghua Tang[2]

[1] Missouri Western State University, St. Joseph, MO 64506, USA
stang@missouriwestern.edu
[2] Guilin University of Electronic Technology, Guilin 541004, China
tch@guet.edu.cn

Abstract. In this paper, we propose a dual SIS epidemic model to study the dynamics of virus spread for a cluster-based wireless sensor network (WSN). The dual SIS model consists of two groups of general sensor nodes (SNs) and cluster heads (CHs) and describes the dynamics of virus spread through the interactions among the SNs and CHs. We transfer the proposed model to a nonlinear system of differential equations and perform detailed analysis about equilibrium points and stability. We develop the system stability conditions (i.e., R_0 and R_1) and draw the conclusions for the proposed system. Under specific conditions, the epidemic (virus spread) in both groups will either die out with any number of initial infectives or remain endemic and the number of infectives in each group will approach a nonzero constant positive level. We provide numerical results to validate our analysis. The proposed model and analysis is applicable to different types of networks with multiple groups of users.

Keywords: Wireless sensor network · SIS epidemic model · Susceptible node Infective node · Equilibrium point · Stability

1 Introduction

Recently, wireless sensor networks (WSNs) have received great attention due to their wide applications and the advances in micro-electro-mechanical systems (MEMS) technology. WSNs typically consist of a large number of sensor nodes (SNs) with limited signal transmission range. Cluster-based WSNs [1] can be managed locally by cluster heads (CHs). SNs in a cluster collect data and send them to its CH. An SN may exchange information with its neighbor SNs that are within its signal transmission range. Each CH manages the SNs in its cluster and relays the collected data to other CHs or the sink. A CH is located within the signal transmission range of all the sensors of its cluster and can communicate with its neighbor CHs at farther places. Thus in some applications, CHs may be more powerful than the SNs in terms of energy, bandwidth and memory [2] and provide inherent optimization and data aggregation/fusion.

Sensors are resource-restrained devices with low defense capabilities and become vulnerable to software attacks such as sensor worm [3] or virus attack. Thus, security is

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of great importance to WSNs. One promising method of analyzing virus spread in WSNs is to use the epidemiological models due to the similarity between software virus spread and epidemic disease transmission. In epidemic modeling, the total population is generally divided into three groups: susceptibles *S*, infectives *I*, and removed or immune *R*. Group *S* are the individuals that may be infected with a desease. Group *I* are the individuals that have been infected and can infect susceptibles. Group *R* are the individuals that have recovered from the desease and are immune to further infection. A model composed of the above three groups is referred to as a susceptible-infective-recovered (SIR) model. For some deseases, such as malaria, the recovered individuals are not immunized and can be infected again. These deseases are usually described by susceptible-infective-susceptible (SIS) models, where there are only two groups of population: *S* and *I*. Susceptibles become infectives due to infection, recovered after some infectious period due to medical or other factors, and become susceptibles again. Much research on epidemic modeling has been done for WSNs [4–9].

In [4], an SIR-M model was proposed to characterize the dynamics of virus spread process from a single node to the entire network. The proposed model can capture both the spatial and temporal dynamics of the virus spread process. In [5], a modified SIS epidemic model was proposed for virus spread analysis and an adjustable virus spread control scheme was developed to effectively restrain the virus outbreak. In [6], a susceptible-infected-quarantine-recovered-susceptible (SIQRS) model was proposed to describe the dynamics of worm propagation in WSNs. In [7], a hop-by-hop worm propagation model was proposed in mobile sensor networks and the worm infection capability was analyzed under a carryover epidemic model. In [8], a susceptible-infectious-quarantine-recovered (SIQR) model was proposed to describe dynamics of worms propagation with quarantine and to study the attacking behavior of possible worms in WSNs. In [9], an energy efficient susceptible-infected–terminally infected-recovered (SITR) model was formulated to analyze the attacking behaviour of worms in WSNs as well as the existence of equilibrium points and stability.

In this paper, we propose a dual SIS epidemic model to study the dynamics of virus spread for a cluster-based WSN. The dual SIS model describes the behavior of individual SNs and CHs and the interactions among them as well as incorporates specific WSN parameters such as number of neighbor nodes of an SN/CH. Based on the proposed model, we answer two basic questions under the occurrence of some initial viruses in SNs and/or CHs: (1) Under what conditions will the viruses in both SNs and CHs die out? (2) Under what conditions will the viruses in both groups of SNs and CHs remain endemic and if so, will the number of infectives in each group approach a constant positive level?

The remainder of the paper is organized as follows. Section 2 develops the modeling of a clustered-based WSN by the dual SIS model. Section 3 presents the detailed analysis and discussion. Section 4 presents numerical results. Finally, the paper is concluded in Sect. 5.

2 System Description and Modeling

The cluster-based WSN consists of a constant number of N_1 SNs and N_2 CHs, which are divided into two groups: susceptibles and infectives of SNs; susceptibles and infectives of CHs. Let $S_1(t)$ and $I_1(t)$ denote the number of susceptible and infective SN nodes at time t; $S_2(t)$ and $I_2(t)$ denote the number of susceptible and infective CH nodes at time t. Then, $S_1(t) + I_1(t) = N_1$, $S_2(t) + I_2(t) = N_2$. The N_2 clusters are deployed identically with m SNs and one CH in each cluster, i.e., $N_1 = mN_2$. SNs and CHs are installed with anti-virus programs that check the nodes periodically and equipped with omnidirectional antennas that have limited signal transmission range. Figure 1 shows a model of a cluster and some of its neighbor clusters. The data sensed from individual SNs can be transmitted to their respective CHs. Each CH can communicate with its neighbor CHs and with all the SNs inside its cluster. An SN can also communicate with its neighbor SNs for necessary information exchange if the neighbor nodes are inside the signal transmission range of the SN.

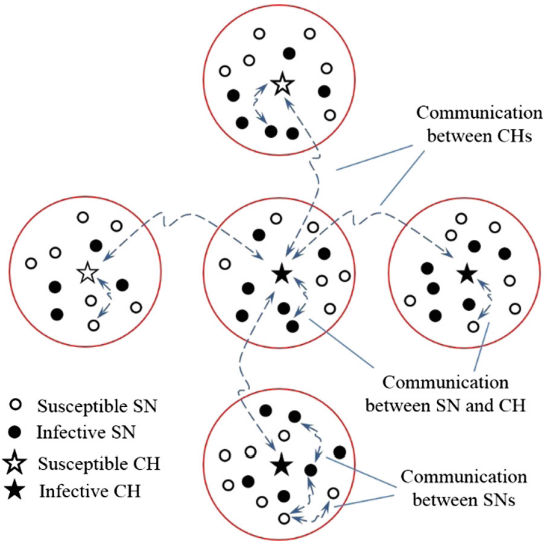

Fig. 1. A model of the cluster-based WSN.

In the dual SIS model without vital dynamics of population, i.e., no occurence of births and deaths of nodes, there are a constant number of susceptibles and infectives divided into two groups for the SNs and CHs respectively. The transition between different groups for a certain virus may be described as follows: a susceptible SN (in the first group, S_1) may become infected inside the same group (I_1) by contact with either an infective SN (in the first group, I_1) or an infective CH (in the second group, I_2), and after some infectious period, it is recovered by treatment and becomes a susceptible SN (S_1) again. Similarly, a susceptible CH (in the second group, S_2) may become infected inside its group (I_2) by contact with either an infective SN (I_1) or an

infective CH (I_2), and after some infectious period, it is recovered by treatment and becomes a susceptible CH (S_2) again.

Assume that initially some SNs and/or CHs in the WSN become infected by viruses due to software attacks. The viruses can be spread together with normal data from the compromised node to its CH or its neighbor SNs through different communication protocols. As the virus spread process continues and the number of infected nodes increases, the virus spread might lead to endemic outbreak in a certain range, even the entire network failure due to insufficient workable nodes. On the other hand, anti-virus programs installed in SNs or CHs periodically check nodes and kill viruses for infective nodes. Thus, infective nodes (either SNs or CHs) can become susceptible (normal) from time to time and the viruses are possible to die out eventually in the network.

In order to formulate mathematical expressions, we make the following assumptions for the proposed model:

- The virus spread only happens through contact between a susceptible and an infective. Thus, contacting a neighbor does not necessarily lead to a new infective node. Only a susceptible neighbor of the infected node can become a new infective node. Contacting an infected neighbor by an infective obviously does not change the state of the system.
- The infection rate β_{ij} represents the average number of infections per unit time of an infective in the jth group with the susceptible nodes in the ith group. For example, β_{11} is the infection rate of an infectious SN with its susceptible neighbor SNs. Similarly, β_{12} is the infection rate of an infectious CH with its susceptible SNs. β_{21} is the infection rate of an infectious SN with its susceptible CH. β_{22} is the infection rate of an infectious CH with its susceptible neighbor CHs. Clearly, β_{ij} depends on the infectivity of a virus and the communication rate of a protocol since the virus spreads itself by piggybacking on normal data via regular communications. The larger the value of β_{ij}, the more susceptible nodes get infected every time.
- Infective nodes in group i (i = 1, 2) recover and are removed from the infective group at a constant rate γ_i (called recovery rate) proportional to the number of infectives in the group. γ_1 is the recovery rate of infective SNs; γ_2 is the recovery rate of infective CHs. The probability of nodes that is infected at time t_0 and still remains infective at time $t_0 + t$ is $\exp(-\gamma_i t)$, and the mean infectious period is $1/\gamma_i$.
- Each SN has m_1 neighbor SNs. Not all neighbors of an infective SN become infected every time. Let p_0 be the fraction of susceptible neighbor SNs infected by an infective SN every time; p_1 be the fraction of susceptible SNs infected by an infective CH every time.
- Similarly, each CH has m_2 neighbor CHs. Not all neighbors of an infective CH become infected every time. Let p_2 be the fraction of susceptible neighbor CHs infected by an infective CH every time.

For tractable analysis, we normalize the proper differential equations on $dI_1(t)/dt$ and $dI_2(t)/dt$ by dividing every $I_1(t)$ and $I_2(t)$ the population size N_1 and N_2 respectively, then the meanings of the variables $I_1(t)$ and $I_2(t)$ are changed to be the fractions of the total population in each group. Thus, the basic differential equations that describe the rate of change of the infective nodes in different groups are determined as:

$$I_1'(t) = \beta_{11} I_1 \frac{p_0 m_1}{m} S_1 + \beta_{12} I_2 \frac{p_1}{N_1} S_1 - \gamma_1 I_1, \tag{1}$$

$$I_2'(t) = \beta_{21} N_1 I_1 \frac{S_2}{N_2} + \beta_{22} I_2 \frac{p_2 m_2}{N_2} S_2 - \gamma_2 I_2, \tag{2}$$

$$S_1 + I_1 = 1, \quad S_2 + I_2 = 1, \tag{3}$$

and initial condition

$$I_1(0) = I_{10}, \quad I_2(0) = I_{20}. \tag{4}$$

Note that in the above differential equations, we omit the parts of $S_1'(t)$ and $S_2'(t)$ due to the relationship in (3).

3 Analysis

We rearrange the above equations via a series of mathematics and obtain the following nonlinear system of differential equations:

$$I_1'(t) = aI_1 + bI_2 - (a+\gamma_1)I_1^2 - bI_1 I_2, \tag{5}$$

$$I_2'(t) = cI_1 + dI_2 - cI_1 I_2 - (d+\gamma_2)I_2^2, \tag{6}$$

where $a = \beta_{11}\frac{p_0 m_1}{m} - \gamma_1, \quad b = \beta_{12} p_1 \frac{N_2}{N_1}, c = \beta_{21}\frac{N_1}{N_2}, \quad d = \beta_{22}\frac{p_2 m_2}{N_2} - \gamma_2$.

We can write the above Eqs. (5) and (6) in a vector form:

$$\vec{I}'(t) = A\vec{I}(t) + \vec{G}(t), \tag{7}$$

where $\vec{I}'(t) = \begin{bmatrix} I_1' \\ I_2' \end{bmatrix}$, $A = \begin{bmatrix} a & b \\ c & d \end{bmatrix}$, $\vec{I}(t) = \begin{bmatrix} I_1 \\ I_2 \end{bmatrix}$, $\vec{G}(t) = \begin{bmatrix} G_1 \\ G_2 \end{bmatrix}$, $G_1 = -(a+\gamma_1)I_1^2 - bI_1 I_2$, and $G_2 = -cI_1 I_2 - (d+\gamma_2)I_2^2$.

Therefore, the analysis of the virus spread in the proposed dual SIS model has been transferred to the analysis of a nonlinear system of differential equations represented by (7). In general, it may not be possible to find solutions for such a nonlinear system in terms of elementary functions. However, we can analyze some interesting questions without finding an explicit solution for the system. For example, what is the equilibrium point (or equilibrium solution) of the system? Is the equilibrium point stable? Under what condition does the equilibrium point converge to the origin or at a constant positive level on the phase plane?

3.1 Origin and Stability

An equilibrium solution of the nonlinear system (7) is a point (I_1^*, I_2^*) on the phase plane (i.e., I_1I_2 plane) that makes $I_1'(t) = 0$ and $I_2'(t) = 0$, which is also called critical point,

stationary point or rest point. By observation, it is easily determined that the point (I_1^*, I_2^*) = (0, 0) is an equilibrium solution. This point denotes that no virus of SNs and CHs exists eventually (the viruses die out) and thus is referred to as a virus-free equilibrium. The proposed nonlinear system may have several equilibrium points. However, it is difficult to find them in terms of elementary functions. All the equilibrium points should be in the rectangular region D bounded by the I_1 and I_2 axes:

$$D = \{I_1, I_2 | 0 \le I_1, I_2 \le 1, I_1 + S_1 = 1, I_2 + S_2 = 1\}. \tag{8}$$

In order to find the local behavior of the proposed nonlinear system and determine the stability property of equilibrium points, one of the most useful methods is to approximate the nonlinear system with a linear system around the equilibrium points, which is referred to as linearization of the nonlinear system. We observe that the proposed system is almost linear system [10] since the vector $\vec{I}'(t)$ is a continuously differentiable function and the Jacobian matrix of the system at this equilibrium point is invertible (i.e., its determinant is not equal to zero). The Jacobian matrix is calculated by respectively differentiating (5) and (6) with respect to I_1 and I_2 [11]:

$$J\big|_{(I_1,I_2)=(0,0)} = \begin{bmatrix} \partial F_1/\partial I_1 & \partial F_1/\partial I_2 \\ \partial F_2/\partial I_1 & \partial F_2/\partial I_2 \end{bmatrix} = \begin{bmatrix} a & b \\ c & d \end{bmatrix}. \tag{9}$$

Equation (9) also verifies that J = A in the almost linear system [10]. The characteristic equation associated with (9) is

$$\lambda^2 - (a+d)\lambda + ad - bc = 0$$

with the characteristic roots are given by

$$\lambda_{1,2} = [(a+d) \pm \sqrt{(a-d)^2 + 4bc}]/2. \tag{10}$$

From the stability properties of differential equations [10, 11], if the characteristic roots are distinct and both are negative, then the equilibrium point is asymptotically stable. That is, if $\lambda_2 < \lambda_1 < 0$, ($I_1$, I_2) approaches the equilibrium point (0, 0) as t approaches infinity. The condition can be easily transferred to the following condition:

$$a + d < 0 \text{ and } ad - bc \ge 0. \tag{11}$$

From the above condition and substituting in (11) by the specific arguments of a, b, c and d, we derive the following theorem.

Theorem 1. For the dual SIS model in (7), if the two thresholds $R_0 < 1$ and $R_1 \le 1$, then the epidemic (virus) will die out in both groups for any number of initial infectives (i.e., the origin is asymptotically stable in the rectangular region D in (8)). The thresholds are defined as follows.

$$R_0 = \frac{\beta_{11}\frac{p_0 m_1}{m} + \beta_{22}\frac{p_2 m_2}{N_2}}{\gamma_1 + \gamma_2}, \tag{12}$$

$$R_1 = \frac{\beta_{11}\beta_{22}p_1}{(\beta_{11}\frac{p_0 m_1}{m} - \gamma_1)(\beta_{22}\frac{p_2 m_2}{N_2} - \gamma_2)}. \tag{13}$$

3.2 Endemic Equilibrium Point and Stability

If $ad - bc < 0$, we can check from (10) that the characteristic roots λ_1 and λ_2 will have one positive and one negative. Note that this result is obtained regardless of the sign of $(a + d)$. From the stability properties of differential equations [10, 11], if the characteristic roots have different signs, then the equilibrium system state will get away from the origin. In this case, the point (0, 0) is called a saddle point and is obviously unstable. Then another question arises, under this condition (i.e., $ad - bc < 0$), is there any other equilibrium point at some positive level in the region D for the proposed system (7)?

We observe that in the system, the number of infectives in each group at a positive equilibrium point is impossible to be 1 (for example, if $I_1 = 1$ in (5), then $I_1'(t) < 0$). The only equilibrium point where the stable value is zero is the origin (for example, for $I_1'(t) = 0$ with $I_1 = 0$, we have $I_2 = 0$). Thus, we limit the analysis of equilibrium points to the region:

$$D_0 = \{I_1, I_2 | 0 < I_1, I_2 < 1, I_1 + S_1 = 1, I_2 + S_2 = 1\}. \tag{14}$$

Consider our analysis in the I_1I_2-plane. For $0 < I_2 < 1$, applying $I_1 = 0$ to (7), we have $I_1'(t)|_{I_1=0} = bI_2 > 0$; applying $I_1 = 1$ to (7), we have $I_1'(t)|_{I_1=1} = -\gamma_1 < 0$. Therefore, there exists a value $I_1^* \in (0, 1)$ such that $I_1'(t)\Big|_{I_1=I_1^*} = 0$ and the value is unique. The following gives the proof of uniqueness.

Assume that there is another nonzero equilibrium solution $K_1 \in (0, 1)$ in (7) that is not equal to I_1^*. Without loss of generality, we let $I_1^* < K_1$, then we have

$$0 = aI_1^* + bI_2 - (a + \gamma_1)(I_1^*)^2 - bI_1^*I_2 = aK_1 + bI_2 - (a + \gamma_1)K_1^2 - bK_1I_2. \tag{15}$$

Multiplying K_1/I_1^* on both sides of the first equation and noting that a, b, and γ_1 are all positive, we have

$$0 = aK_1 + bI_2\frac{K_1}{I_1^*} - (a + \gamma_1)K_1^2\frac{I_1^*}{K_1} - bK_1I_2 > aK_1 + bI_2 - (a + \gamma_1)K_1^2 - bK_1I_2. \tag{16}$$

There is a contradiction for (16) and (15), so there is only one equilibrium solution of $I_1(t)$ in (7) in D_0.

Similar result can be shown that there is a unique equilibrium solution $I_2^* \in (0,\ 1)$ of $I_2(t)$ in (7) in D_0. The earlier condition $ad - bc < 0$ can be converted as $R_1 > 1$. The expression of R_1 is referred to as (13). We give the following theorem to summarize the above analysis.

Theorem 2. For the dual SIS model in (7), if the threshold $R_1 > 1$, then the epidemic (virus) will remain endemic in both groups for any number of initial infectives and the number of infectives in each group will approach a nonzero constant positive level (i.e., a unique equilibrium point exists inside the region D_0 in (14)).

4 Numerical Results

In this section, we present numerical results to validate our analytic results for the dual SIS model. We study the phase portraits in the I_1I_2-plane to visualize how the trajectories traced by the solutions of the proposed system would behave in the long run as well as the number of infective SNs and CHs $I_1(t)$ and $I_2(t)$ with respect to time t. The evaluation is performed under a WSN of m = 40 identical clusters with 25 SNs and one CH in each cluster. The values of other parameters are shown in individual figures. Note that all parameters are given in dimensionless units, which can be mapped to specific units of measurement.

Figure 2 shows the direction field for our system of differential equations along with two trajectories on the phase plane. Two starting points $(I_{10}, I_{20}) = (0.55, 0.25)$ and (0.25, 0.15) are evaluated respectively. It is clearly observed that the equilibrium point (0, 0) is asymptotically stable regardless of any starting points. The trajectories of two different starting points eventually converge to the origin. This verifies the result of Theorem 1. The thresholds under the given parameter configuration are obtained as $R_0 = 0.0145 < 1$; $R_1 = 0.1238 < 1$. Note that the arrows from the top left of the trajectories go down towards to the origin, while the arrows from the bottom right of the

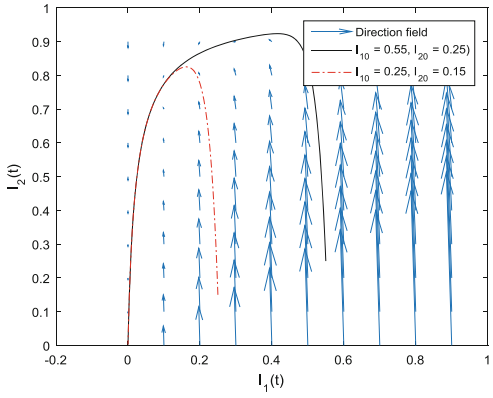

Fig. 2. A direction field and some trajectories for the dual SIS system with origin equilibrium point (Parameter values: $p_0 = 0.2$; $p_1 = 0.3$; $p_2 = 0.1$; $m_1 = 5$; $m_2 = 4$; $\beta_{11} = 0.3$; $\beta_{12} = 0.5$; $\beta_{21} = 0.7$; $\beta_{22} = 0.4$; $\gamma_1 = 0.5$; $\gamma_2 = 0.6$).

trajectories go up along with the trajectories to the origin. The arrows in the direction field [11] are tangents to the actual solutions to the differential equations, in which we can learn the solution property of the nonlinear system. The direction field can also be used to find information on the long term behavior of the solution.

Figure 3 shows the numerical simulations of the virus spread dynamics of $I_1(t)$ and $I_2(t)$ with respect to time for the dual SIS system with origin equilibrium point. We observe that under the current system configuration, $I_1(t)$ decreases with respect to time; while $I_2(t)$ first increases with respect to time and when it goes to a certain infection level, it begins to decrease. Both $I_1(t)$ and $I_2(t)$ eventually approach to the origin (0, 0), which means the infectives eventually die out regardless of their initial conditions.

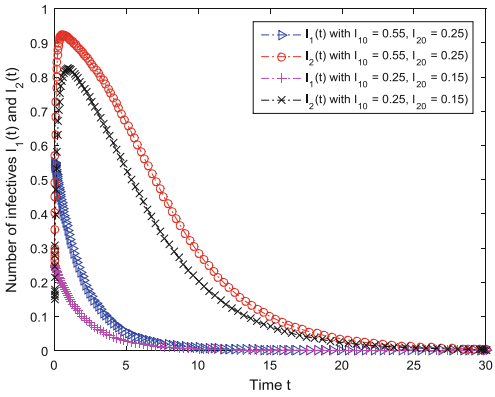

Fig. 3. The dynamics of $I_1(t)$ and $I_2(t)$ for the dual SIS system with origin equilibrium point (See Fig. 2 for the parameter values).

Figure 4 shows the direction field for the nonlinear system along with two trajectories on the phase plane. Two starting points (I_{10}, I_{20}) = (0.55, 0.25) and (0.10, 0.10) are evaluated respectively. It is observed that the system state approaches to a constant positive equilibrium point. Each group of SNs and CHs has a different constant value. The equilibrium point is asymptotically stable regardless of any starting point. This verifies the result of Theorem 2. The threshold condition in this case is obtained as $R_1 = 4.9205 > 1$.

Figure 5 shows the numerical simulations of the virus spread dynamics of $I_1(t)$ and $I_2(t)$ with respect to time for the dual SIS system with a positive equilibrium point. We observe that under the current system configuration, when the starting point is (I_{10}, I_{20}) = (0.55, 0.25), $I_1(t)$ decreases from its initial value to a constant positive value (endemic of SNs); $I_2(t)$ first increases from its initial value and when it goes to a certain infection level, it begins to decrease to another constant positive level (endemic of CHs). When the starting point is (I_{10}, I_{20}) = (0.10, 0.10), $I_1(t)$ increases from its initial value to a constant positive value (endemic of SNs); $I_2(t)$ also increases from its initial value to another constant positive level (endemic of CHs).

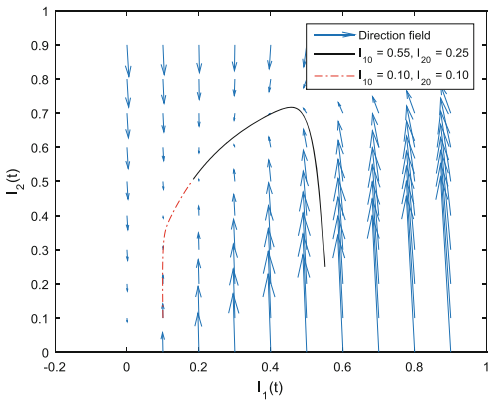

Fig. 4. A direction field and some trajectories for the dual SIS system with endemic equilibrium point (Parameter values: $p_0 = 0.8$; $p_1 = 0.8$; $p_2 = 0.1$; $m_1 = 5$; $m_2 = 2$; $\beta_{11} = 0.7$; $\beta_{12} = 0.8$; $\beta_{21} = 0.1$; $\beta_{22} = 0.15$; $\gamma_1 = 0.15$; $\gamma_2 = 0.45$).

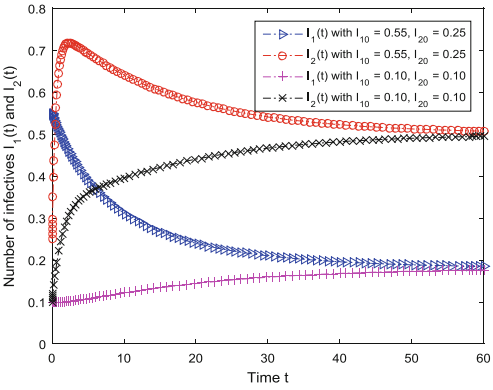

Fig. 5. The dynamics of $I_1(t)$ and $I_2(t)$ for the dual SIS system with endemic equilibrium point (See Fig. 4 for the parameter values).

5 Conclusions

We proposed a dual SIS epidemic model to study the dynamics of virus spread for a cluster-based WSN. The dual SIS model consists of two groups of SNs and CHs and describes the dynamics of virus spread through the interactions between SNs and CHs. We performed detailed analysis about equilibrium points and stability and developed the system stability conditions. Finally, we drew the conclusion for the proposed system: if the two thresholds $R_0 < 1$ and $R_1 \leq 1$, then the epidemic (virus) will die out in both groups for any number of initial infectives; if the threshold $R_1 > 1$, then the epidemic (virus) will remain endemic in both groups for any number of initial infectives and the number of infectives in each group will approach a nonzero constant

positive level. We provided numerical results to validate our analysis. The proposed model and analysis is applicable to different types of networks with multiple groups of users.

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