GLOBAL DYNAMICS OF A GENERALIZED EPIDEMIOLOGICAL MODEL FOR DISTRIBUTED DENIAL OF SERVICE ATTACKS

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In this work, we propose and study a generalized epidemiological model for distributed denial of service (DDoS) attacks. This model incorporates a recognized epidemiological model for DDoS attacks with nonlinear incidence rates. By using a simple approach, which is based on global stabilizability of two cascade connected nonlinear systems, global asymptotic stability of disease-free and disease-endemic equilibrium points is analyzed rigorously. As an important consequence, global dynamics of the proposed model is fully determined. The obtained results not only improve upon some findings reported in previous studies, but also provide useful applications in both theory and practice. Lastly, the theoretical results are supported by a set of numerical examples.

Keywords: Epidemiological models, Nonlinear incidence, DDoS attacks, Global asymptotic stability, Cascade systems

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1. Introduction

In this work, we revisit a modified epidemiological model for distributed denial of service (DDoS) attacks on targeted resources in computer networks, which was proposed by Rao et al. in [19]. The mathematical model is given by

$$\begin{aligned} \dot{S}_t &= -\beta S_t I + \epsilon_t R_t, \\ \dot{I}_t &= \beta S_t I - \gamma I_t, \\ \dot{Q}_t &= \gamma I_t - \eta Q_t, \\ \dot{R}_t &= \eta Q_t - \epsilon_t R_t, \\ \dot{S} &= \mu - \beta S I - \mu S + \epsilon I, \\ \dot{I} &= \beta S I - (\mu + \epsilon) I. \end{aligned} \tag{1}$$

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In this model:

- the entire population of nodes is divided into attacking and targeted populations;
- the targeted population is divided into four compartments: susceptible compartment (S_t), infected compartment (I_t), quarantined compartment (Q_t) and recovered compartment (R_t);
- the attacking population is also divided in two compartments: susceptible (S) and infected (I) compartments;
- all the parameters are assumed to be positive due to epidemiological reasons.

We refer the readers to [19] for more details of the model (1) and its dynamical qualitative properties. Also, in a recent work [18], Pham and Hoang have analyzed global asymptotic stability (GAS) of the model (1) based on global stabilizability of two cascade connected nonlinear systems [20].

It is important to remark that the model (1) used the interaction term of the form $f(S, I) = \beta SI$, which was first introduced in a classical epidemic model proposed by Kermack and McKendrick in 1927 [10]. However, as pointed out by Capasso and Serio in [4] that this function may be quite unrealistic in many cases. For this reason, there have been many epidemic models that used nonlinear incidence rates of the form $f(S, I) = \beta g(I)S$, where $g(I)$ is a nonlinear function of I and satisfies (see, e.g., [4, 5, 7, 8, 9, 16, 15, 22, 23])
(P1): $g(0) \geq 0$ for $I \geq 0$ and $g(I) = 0$ if and only if $g(0) = 0$;
(**P2**): $g'(I) \geq 0$ for all $I \geq 0$;
and
(P3): $g''(I) \leq 0$ for all $I \geq 0$.
Note that two famous nonlinear incidence rates, namely the saturated incidence $f(S, I) = \beta SI/(1 + \gamma I)$ and the standard incidence $f(S, I) = \beta SI/(S + I)$, satisfy all the conditions (**P1**)-(**P3**).

In this work, we will study a generalized version of (1) by incorporating it with nonlinear incidence rates. More precisely, the generalized model under consideration is given by

$$\begin{aligned}
\dot{S}_t &= -\beta_1 S_t g_1(I) + \epsilon_t R_t, \\
\dot{I}_t &= \beta_1 S_t g_1(I) - \gamma I_t, \\
\dot{Q}_t &= \gamma I_t - \eta Q_t, \\
\dot{R}_t &= \eta Q_t - \epsilon_t R_t, \\
\dot{S} &= \mu - \beta_2 S g_2(I) - \mu S + \epsilon I, \\
\dot{I} &= \beta_2 S g_2(I) - (\mu + \epsilon) I,
\end{aligned} \tag{2}$$

where g_1 and g_2 are nonlinear functions satisfying the properties (**P1**)-(**P3**).

Our main objective is to conduct a rigorous mathematical analysis for global dynamics of the proposed model (2). After determining the basic reproduction number and the set of equilibrium points, we use the approach introduced by Pham and Hoang in [18] to study the GAS of the equilibrium points. Following this approach, the GAS analysis of the nonlinear system (2) is reduced to the GAS analysis of simple linear systems. As an important consequence, the GAS problem for (2) is resolved in a straightforward manner.

It is important to note that the model (1) can be considered as a generalization of a mathematical model for DDoS attacks, which was constructed by Haldar et al. in [6]. Other epidemiological models that are similar to (1) can be found in [12, 14, 24]. We can introduce the nonlinear incidence rates to the above-mentioned models. Then, the approach used in

this work can be applied to study the GAS of the resulted mathematical models with the nonlinear incidence rates.

With the incorporation of nonlinear incidence rates, the proposed model (2) becomes more flexible than the original model (2). As a result, it can capture a wider range of malware spreading and cyber-attack scenarios. This may have practical significance, particularly for studying parameter estimation problems with real data and for estimating the scale of cyber-attacks in reality.

On the other hand, from the GAS of the equilibrium points (Section **2**), we can propose measures and strategies to prevent malware spreading as well as cyber-attack based on controlling parameters so that the basic reproduction number $\mathcal{R}_0$ is greater than 1. This ensure that the disease-free equilibrium point is globally asymptotically stable; consequently, malware spreading and cyber-attacks are eventually eliminated.

The plan of this work is as follows: Global dynamics of (2) is studied in Section **2**. Some numerical examples supporting the theoretical assertions are reported in Section **3**. The last section presents some conclusions and open problems.

2. Dynamical analysis of the proposed model

In this section, we analyze dynamical qualitative properties of (2) on its positively invariant set defined by

$$\Omega = \{(S_t, I_t, Q_t, R_t, S, I) \in \mathbb{R}^6_+ | S_t + I_t + R_t + Q_t = S + I = 1\}. \tag{3}$$

We first determine the basic reproduction number of the attacking population by using the method developed in [25]. For this reason, consider the subsystem modeling the attacking population of (2):

$$\begin{aligned} \dot{S} &= \mu - \beta_2 S g_2(I) - \mu S + \epsilon I, \\ \dot{I} &= \beta_2 S g_2(I) - (\mu + \epsilon) I. \end{aligned}$$

Since $S + I = 1$, this system is reduced to

$$\dot{I} = \beta_2 (1 - I) g_2(I) - (\mu + \epsilon) I = \mathcal{F}(I) - \mathcal{V}(I),$$

where

$$\mathcal{F}(I) = \beta_2 g_2(I), \quad \mathcal{V}(I) = \beta_2 I g_2(I) + (\mu + \epsilon) I.$$

Consequently, at the disease-free equilibrium point $I_0 = 0$, we have

$$F := D\mathcal{F}(0) = \beta_2 g_2'(0), \quad V := D\mathcal{V}(0) = \mu + \epsilon.$$

By using the method developed in [25], the basic reproduction number is computed as the spectral radius of a matrix FV^{-1} and hence is given by

$$\mathcal{R}_0 = \frac{\beta_2 g_2'(0)}{\mu + \epsilon}.$$

As will be seen later, asymptotic stability of equilibrium points of (2) depends on the value of $\mathcal{R}_0$ relative to 1.

Next, we determine the set of equilibrium points of (2). It is easily seen that (2) always has a disease-free equilibrium (DFE) point, which is given by

$$E^0 = (S_t^0, I_t^0, Q_t^0, R_t^0, S^0, I^0) = (1, 0, 0, 0, 1, 0).$$

To determine the possible disease-endemic equilibrium points, we need the following auxiliary result.

Lemma 2.1. *If g is any function satisfying the properties (**P1**)-(**P3**), then $Ig_2'(I) \leq g_2(I)$ for all $I \geq 0$.*

Proof. Let us consider a function

$$z(I) = Ig_2'(I) - g_2(I), \quad I \geq 0.$$

Then, $z'(I) = Ig_2''(I) \leq 0$, which implies that $z(I) \leq z(0) = 0$ for $I \geq 0$. This is the desired conclusion. The proof is complete. □

Lemma 2.2. *The model* (2) *has a unique (positive) equilibrium point* $E^* = (S_t^*, I_t^*, Q_t^*, R_t^*, S^*, I^*)$ *if and only if* $\mathcal{R}_0 > 1$. *Furthermore, if* E^* *exists, it is defined by*

$$\begin{aligned} S^* &= 1 - I^*, \\ I_t^* &= \frac{\beta_1 g_1(I^*)}{\beta_1 g_1(I^*)\left(1 + \frac{\gamma}{\eta} + \frac{\gamma}{\epsilon_t}\right) + \gamma}, \\ Q_t^* &= \frac{\gamma}{\eta} I_t^*, \\ R_t^* &= \frac{\gamma}{\epsilon_t} I_t^*, \\ S_t^* &= 1 - I_t^* - Q_t^* - R_t^*, \end{aligned} \tag{4}$$

where I^* *is a unique positive solution of the equation*

$$\beta_2(1 - I)g_2(I) - (\mu + \epsilon)I = 0. \tag{5}$$

Proof. It follows from the two last equations of (2) that I^* must be a positive solution of the (5), or equivalently

$$F(I) = \frac{\beta_2(1 - I)g_2(I)}{I} - (\mu + \epsilon) = 0. \tag{6}$$

From the assumption $\mathcal{R}_0 > 1$ and Lemma **2**.1, we obtain

$$\begin{aligned} &\lim_{I \to 0} F(I) = \beta_2 g_2'(0) - (\mu + \epsilon) > 0, \\ &F'(I) = \frac{\beta_2(1 - I)(Ig_2'(I) - g_2(I)) - \beta_2 I g_2(I)}{I^2} \leq 0, \quad 0 < I \leq 1. \end{aligned} \tag{7}$$

On the other hand,

$$F(1) = -(\mu + \epsilon) < 0.$$

Therefore, (6) has a unique positive equation $I^* \in (0, 1)$. Then, (4) is obtained from the relation $S^* = 1 - I^*$ and from the first four equations of (2). The proof is complete. □

We now analyze the GAS of the DFE and disease-endemic equilibrium (DEE) points of (2). Since Ω defined in (3) is a positively invariant set, we only need to consider the following sub-system of (2):

$$\begin{aligned} \dot{I}_t &= \beta_1(1 - I_t - Q_t - R_t)g_1(I) - \gamma I_t, \\ \dot{Q}_t &= \gamma I_t - \eta Q_t, \\ \dot{R}_t &= \eta Q_t - \epsilon_t R_t, \\ \dot{I} &= \beta_2(1 - I)g_2(I) - (\mu + \epsilon)I \end{aligned} \tag{8}$$

on a feasible set defined by

$$\Omega^* = \{(I_t, Q_t, R_t, I) \in \mathbb{R}_+^4 | I_t + Q_t + R_t \leq 1, \ I \leq 1\}. \tag{9}$$

The DFE and DEE points of (8) are

$$e^0 = (I_t^0, Q_t^0, R_t^0, I^0) = (0, 0, 0, 0), \quad e^* = (I_t^*, Q_t^*, R_t^*, I^*),$$

respectively. Here, I_t^*, Q_t^*, R_t^* and I^* are given in (4).

We now focus on the last equation of (8):

$$\dot{I} = G(I) := \beta_2(1 - I)g_2(I) - (\mu + \epsilon)I. \tag{10}$$

Before analyzing the GAS of (8), we need the following auxiliary result.

Theorem 2.1. *(i) If $\mathcal{R}_0 < 1$, then the trivial equilibrium point $f^0 = 0$ of (10) is globally asymptotically stable with respect to the set $\{I \in \mathbb{R} | I \geq 0\}$.*
(ii) Assume that $\mathcal{R}_0 > 1$. Then, (10) has a unique positive equilibrium point $f^* = I^*$. Moreover, f^* is globally asymptotically stable with respect to the set $\{I \in \mathbb{R} | I > 0\}$.

Proof. **Proof of Part (i).** First, we show that f^0 is locally asymptotically stable. Indeed, the Jacobian matrix of (10) evaluated at $f^0 = 0$ is given by

$$J(f^0) = \beta_2 g_2'(0) - (\mu + \epsilon) < 0.$$

So, $J(f^0)$ is locally asymptotically stable ([11, 21]).

On the other hand, it follows from the properties **(P1)**-**(P3)** and $\mathcal{R}_0 < 1$ that the derivative of G satisfies

$$\begin{aligned} G'(I) &= \beta_2 g_2'(I) - \beta_2 I g_2'(I) - \beta_2 g_2(I) - (\mu + \epsilon) \\ &\leq \beta_2 g_2'(I) - (\mu + \epsilon) \leq \beta_2 g_2'(0) - (\mu + \epsilon) < 0, \end{aligned}$$

which implies that $\dot{I} = G(I) \leq G(0) = 0$ for $I \geq 0$. Since $I(t)$ is decreasing and $0 \leq I(t) \leq 1$ for $t \geq 0$, its limit exists and equals f^0, that is $\lim_{t\to\infty} I(t) = f^0$. Hence, the GAS of f^0 is proved.

Proof of Part (ii). First, it is easy to verify that (10) admits the set $\Omega_1 := \{I | I > 0\}$ as a positively invariant set and has a unique positive equilibrium point $f^* = I^*$. We now rewrite (10) in the form

$$\dot{I} = IF(I) = I(F(I) - F(I^*)), \tag{11}$$

where F is defined in (6). Using Taylor's expansion theorem gives

$$F(I) - F(I^*) = F'(\xi_I)(I - I^*), \tag{12}$$

where ξ_I is between I and I^*. Consider a Lyapunov function candidate (see [13, 26])

$$V_2(I) = I - I^* \ln \frac{I}{I^*} - I^*.$$

From (11) and (12), we have that

$$\frac{dV_2}{dt} = \frac{dV_2}{dI}\frac{dI}{dt} = \frac{I - I^*}{I} I F'(\xi_I)(I - I^*) = F'(\xi_I)(I - I^*)^2.$$

On the other hand, we deduce from the last estimate of (7) that $F'(I) < 0$ for all $0 < I \leq 1$. As a consequence, $dV_2/dt \leq 0$ for all $I > 0$ and $dV_2/dt = 0$ if and only if $I = I^*$. Hence, the Lyapunov function V_2 satisfies Barbashin-Krasovskii-LaSalle theorem (see [11, Theorem 4.2] and [17]). Hence, we conclude that f^* is globally asymptotically stable. The proof is complete. □

The following theorem is the main result of this section.

Theorem 2.2 (GAS analysis)**.** *(i) If $\mathcal{R}_0 < 1$, then the DFE point e^0 of (8) is globally asymptotically stable.*
(ii) Suppose that $\mathcal{R}_0 > 1$. Then, the DEE point e^* of (8) is globally asymptotically stable if $I(0) > 0$.

Proof. **Proof of Part (i).** From the conclusion of Part (i) of Theorem **2**.1 and [20, Corollary 4.3], it is enough to study the GAS of the following system, which is obtained from (8) by substituting $I = 0$ into the first three equations of (8):

$$\dot{I}_t = -\gamma I_t, \quad \dot{Q}_t = \gamma I_t - \eta Q_t, \quad \dot{R}_t = \eta Q_t - \epsilon_t R_t. \tag{13}$$

The system (13) has a unique equilibrium point $\widetilde{e}^0 = (0, 0, 0)$. We will show that $\widetilde{e}^0$ is globally asymptotically stable. Indeed, (13) can be rewritten in the matrix form $\dot{v} = Av$, where A and v are given by in (14)

$$v = \begin{bmatrix} I_t & Q_t & R_t \end{bmatrix}^T, \quad A = \begin{bmatrix} -\gamma & 0 & 0 \\ \gamma & -\eta & 0 \\ 0 & \eta & -\epsilon_t \end{bmatrix}. \tag{14}$$

Three eigenvalues of A are $-\gamma, -\eta$ and $-\epsilon_t$, which are all negative. So, from the stability theory of systems of linear ODEs (see [3, 11, 21]), we conclude that the origin is a globally asymptotically stable equilibrium point of (13). Then, the GAS of f^0 is obtained by using [20, Corollary 4.3].

Proof of Part (ii). From the conclusion of Part (ii) of Theorem **2**.1, we have I^* is the globally asymptotically stable equilibrium point of the fourth equation of (8). Hence, by applying [20, Corollary 4.3] to the cascade system (8), it is enough to study the GAS of the following system, which is obtained from (8) by substituting $I = I^*$ into the first three equations of (8):

$$\begin{aligned} \dot{I}_t &= \beta_1 g_1(I^*)(1 - I_t - Q_t - R_t) - \gamma I_t, \\ \dot{Q}_t &= \gamma I_t - \eta Q_t, \\ \dot{R}_t &= \eta Q_t - \epsilon_t R_t. \end{aligned} \tag{15}$$

Since (I_t^*, Q_t^*, R_t^*) is a unique equilibrium point of (15), we can write (15) in the matrix form $\dot{u} = Bu$, where u and B are given by in (16)

$$u = \begin{bmatrix} I_t - I^* & Q_t - Q_t^* & R_t - R_t^* \end{bmatrix}^T, \ B = \begin{bmatrix} -\beta_1 g_1(I^*) - \gamma & -\beta_1 g_1(I^*) & -\beta_1 g_1(I^*) \\ \gamma & -\eta & 0 \\ 0 & \eta & -\epsilon_t \end{bmatrix}. \tag{16}$$

We will show that any eigenvalue λ of the coefficient matrix B satisfies $Re(\lambda) < 0$. Indeed, the characteristic polynomial of B is

$$P_B(x) = x^3 + a_1 x^2 + a_2 x + a_3,$$

where

$$\begin{aligned} a_1 &= \epsilon_t + \eta + \gamma + \beta_1 \big(g_1(I^*)\big)^2, \\ a_2 &= \epsilon_t(\eta + \gamma + \beta_1 g_1(I^*)) + \eta(\gamma + \beta_1 g_1(I^*)) + \beta_1 g_1(I^*)\gamma, \\ a_3 &= \epsilon_t[\eta(\gamma + \beta_1 g_1(I^*)) + \beta_1 g_1(I^*)\gamma] + \beta_1 \eta g_1(I^*)\gamma. \end{aligned}$$

It is clear that a_1, a_2 and a_3 are positive. On the other hand,

$$\begin{aligned} a_1 a_2 - a_3 = {} & (\beta_1^2 \epsilon_t + \beta_1^2 \eta + \beta_1^2 \gamma) g_1(I^*)^2 \\ & + (\beta_1 \epsilon_t^2 + 2\beta_1 \epsilon_t \eta + 2\beta_1 \epsilon_t \gamma + \beta_1 \eta^2 + 2\beta_1 \eta\gamma + \beta_1 \gamma^2) g_1(I^*) \\ & + \epsilon_t^2 \eta + \epsilon_t^2 \gamma + \epsilon_t \eta^2 + 2\epsilon_t \eta\gamma + \epsilon_t \gamma^2 + \eta^2 \gamma + \eta\gamma^2 > 0. \end{aligned}$$

Hence, the Routh-Hurwitz criteria [1] implies that the real parts of all the roots of $P_B(x)$ are negative. By using the stability theory of systems of linear ODEs ([3, 11, 21]), we conclude that (I_t^*, Q_t^*, R_t^*) is a globally asymptotically stable equilibrium point of (15).

Now, by applying [20, Corollary 4.3], we obtain the GAS of e^* of (8). The proof is completed. □

Remark 2.1. *From the conclusion of Theorem* **2***.2, the GAS of the DFE and DEE points of the full models* (2) *is shown.*

3. Numerical experiments

In this section, two numerical examples are reported to support the theoretical findings, in which the classical four stages Runge-Kutta method (see [2, 21]) using a small step size, namely $h = 10^{-4}$, is implemented to numerically solve the model (2) over the time interval $0 \leq t \leq 100$. In all the examples, we will use saturated incidence rates of the form

$$\beta_i g_i(I)S = \beta_i \frac{SI}{1+\alpha_i I}, \quad i = 1, 2.$$

Consequently, the basic reproduction number is given by

$$\mathcal{R}_0 = \frac{\beta_2}{\mu + \epsilon}.$$

Now, consider the model (2) with the parameters and initial data given in Table 1 and Table 2, respectively.

Table 1. Parameters used in the numerical examples.

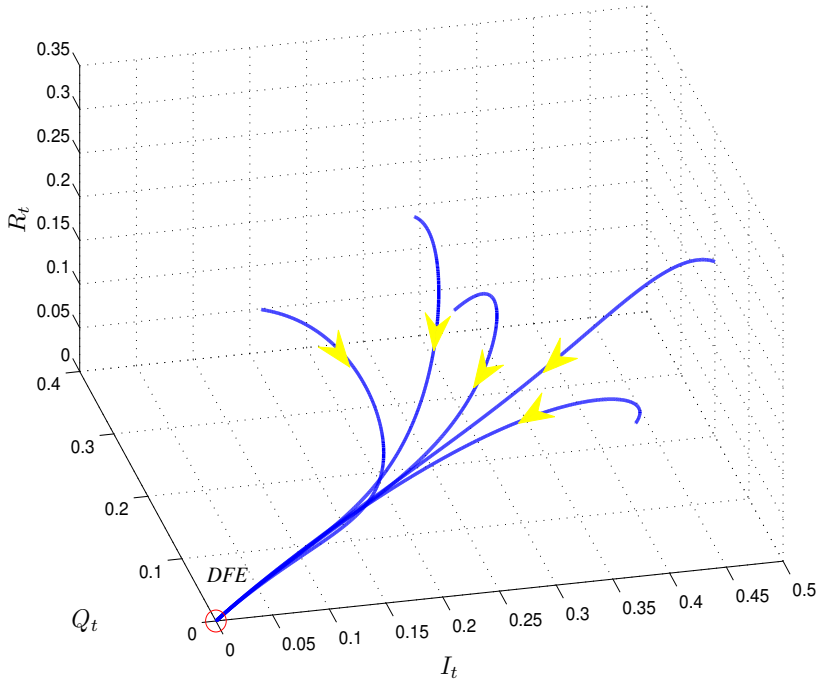
Case	β_1	β_2	α_1	α_2	γ	η	ϵ	ϵ_t	μ	$\mathcal{R}_0$	GAS Equilibrium point
1	0.25	0.2	0.1	0.15	0.1	0.5	0.15	0.25	0.2	0.5714	(1, 0, 0, 0, 0, 1, 0)
2	0.4	0.5	0.02	0.05	0.1	0.1	0.05	0.025	0.2	2	(0.0794, 0.1534, 0.1534, 0.6137, 0.5122, 0.4878)

Table 2. Initial data used in the numerical examples.

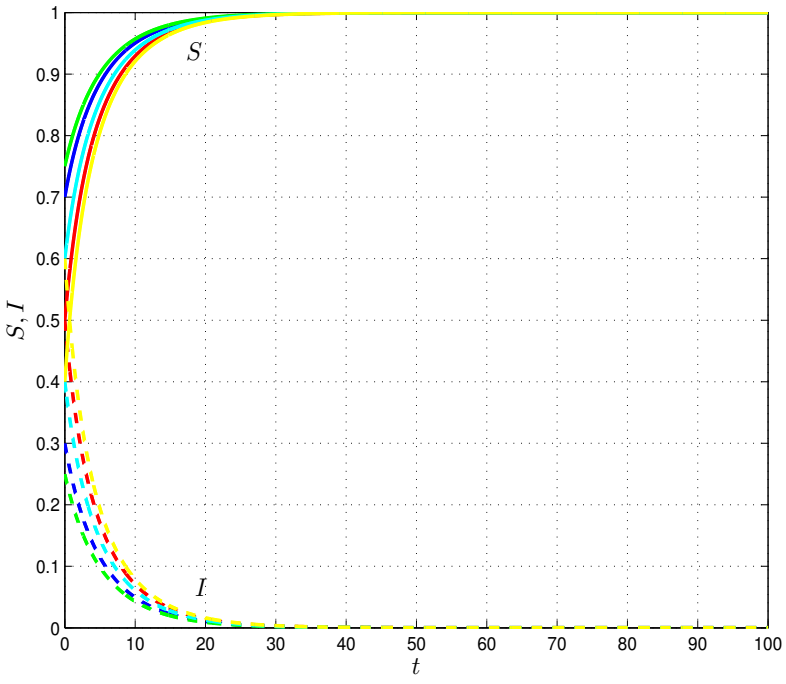
Set	$S_t(0)$	$I_t(0)$	$Q_t(0)$	$R_t(0)$	$S(0)$	$I(0)$
1	0.5	0.1	0.2	0.2	0.7	0.3
2	0.4	0.4	0.1	0.1	0.5	0.5
3	0.25	0.25	0.25	0.25	0.75	0.25
4	0.3	0.3	0.3	0.1	0.6	0.4
5	0.1	0.5	0.2	0.2	0.4	0.6

The solutions of the generalized system (2), which are associated with the five sets of initial data given in Table 2, are depicted in Figures 1-2. In Figures 1(A) and 2(A), each blue curve represents a trajectories of the targeted population that starts from one initial condition in Table 2, and the yellow arrows describe the evolution of the model. Meanwhile, each solid-dashed pair of curves of the same color in Figures 1(B) and 2(B) represents a solution of the attacking population that starts from one initial condition in Table 2.

It is clear that all the solutions are stable and converge to the corresponding GAS equilibrium points. This is evidence supporting the theoretical assertions presented in Section **2**.

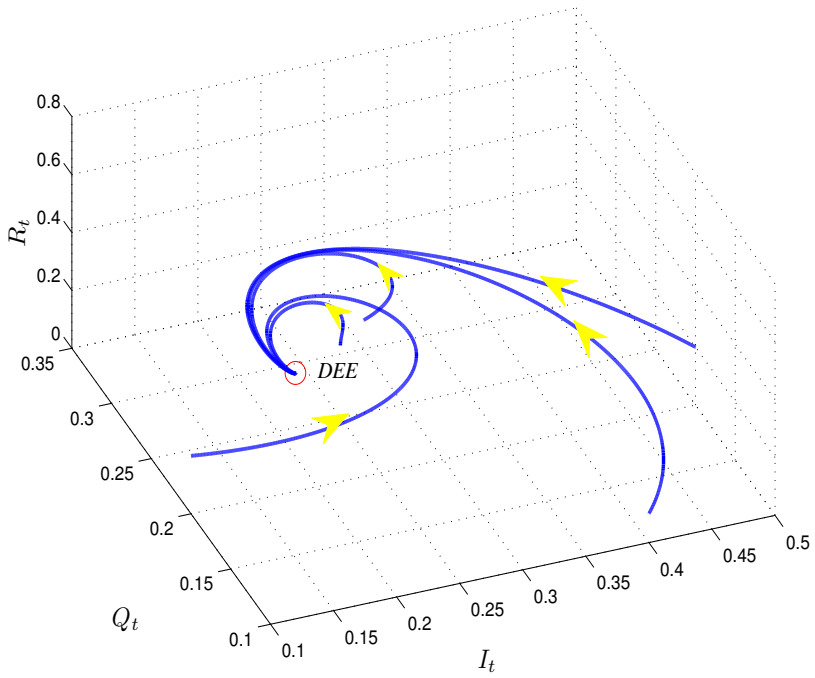


(A) Dynamics of I_t, Q_t and R_t for Case 1 parameters in Table 1

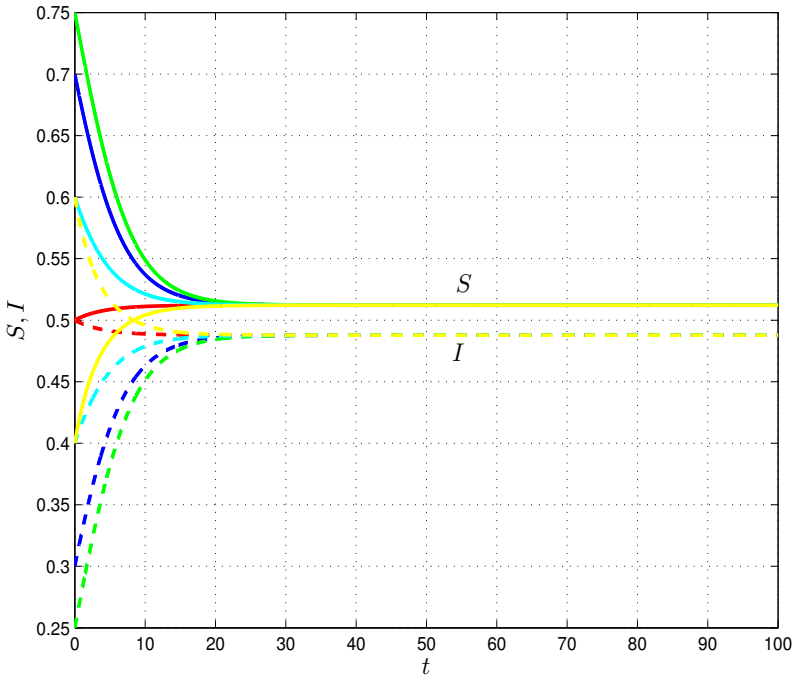


(B) Dynamics of S and I for Case 1 parameters in Table 1

FIGURE 1. Dynamics of the model (2) corresponding to the parameters in Case 1 of Table 1.



(A) Dynamics of I_t, Q_t and R_t for Case 2 parameters in Table 1



(B) Dynamics of S and I for Case 2 parameters in Table 1

FIGURE 2. Dynamics of the model (2) corresponding to the parameters in Case 1 of Table 1.

4. Conclusions and discussions

As the main conclusion of this work, we have proposed and studied a generalized epidemiological model for DDoS attacks, which combines a well-known mathematical model for DDoS attacks with a general family of nonlinear incidence rates. Global dynamics of the proposed model has been analyzed rigorously and supported by a set of numerical examples. The obtained results not only improve upon some findings in previous studies, but also provide useful applications in both theory and practice. In the near future, we will further develop the approach and extend the obtained results in this work to study mathematical and computational modeling of DDoS attacks with applications in science and engineering.

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