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Research article

Dynamics of an epidemic model with general incidence rate dependent on a class of disease-related contact functions

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Abstract: Starting from the idea of constructing the standard incidence rate, we take the effective contact times of individuals in the population per unit time as a contact function, $T(\cdot)$, which depends on the population size. Considering the influence of disease on the contact function, the influence intensity factor of the disease affected by the infected person is integrated into the nonlinear incidence rate. We propose an epidemic model with a class of disease-related contact functions. Then, we analyze the well-posedness of the solutions of the model. By using the next generation matrix method, we get the basic reproduction number $\mathcal{R}_0$. We find that the existence and stability of the equilibria are not only related to $\mathcal{R}_0$, but also to the intensity of the disease affected for the infected person, η, and the contact function, $T(\cdot)$. We obtain some stability results under different assumptions about the contact function. Finally, we use MATLAB to simulate the system for several different contact functions. The numerical simulation results agree with our qualitative study. At the same time, we also prove that the system may have a Hopf bifurcation when the contact function $T(\cdot)$ satisfies some corresponding conditions.

Keywords: SI model; the effective contact function; stability; Hopf bifurcation

1. Introduction

During disease transmission, most pathogens are transmitted by person-to-person contact. Therefore, establishing mathematical models to study infectious diseases, especially infection links, is a very important research goal. The rate at which new compartments of infected persons are added to a model is usually referred to as the incidence of disease. Different infectious diseases have different transmission routes, so the selection of disease incidence rate in the infectious disease model cannot be studied in the same form. Many infectious diseases break out repeatedly in large numbers of people. Mechanisms that can explain this phenomenon include seasonal change [1, 2], transmission rate, infection or the time delay in the process of recovery [3] and heterostructure of host

population [4], etc. As research continues, many scholars have focused on no periodicity of bilinear transmission research [5–10]. Through the study of different nonlinear incidence, more abundant dynamics are obtained, which can be used to describe and explain some periodic epidemic phenomena in reality.

The contact form within the population has an important influence on the disease transmission process [11]. In [12], a mixed proportion of infection incidence was proposed and the number of effective contacts per population is considered to be a fixed constant number. However, with the change of population within the population and the development of society, the contact pattern between populations will also change. It is not very effective to take the number as a constant. We consider it as a function dependent on population size. In a population, it is assumed that the members are evenly distributed and the number of contacts between each member in unit time is the same. Because the population is homogeneous, we take the contact function as $T(N)$, where N is the total number of the population.

For the effective contact function $T(N)$, we propose assumptions (A1)–(A3) described below. Since the function represents the number of effective contacts per unit time of each member of the population, it is non-negative. Thus, the first assumption is as follows:

(**A1**) $\forall N \in [0, +\infty), 0 = T(0) \leq T(N) < +\infty$.

Considering that the effective contact times of each member of the crowd in unit time will not decrease with the increase of the population, $T(N)$ is a monotone undecreasing function. Thus, we give the second assumption:

(**A2**) $\forall N \in [0, +\infty), T'(0) > 0, T'(N) \geq 0$.

We make the third assumption of $T(N)$ regarding the growth rate N as follows:

(**A3**) $\forall N \in [0, +\infty), T'(N)N - T(N) \leq 0$.

Table 1 shows two special cases of the effective contact function $T(N)$ that satisfy assumptions (A1)–(A3). These two types of contact functions correspond exactly to the standard incidence rate and the bilinear incidence rate.

Table 1. The effective contact function $T(N)$.

Function $T(N)$	The type of disease incidence rate	The previous achievements
$T(N) = c$	Standard incidence rate	[12]
$T(N) = cN$	Bilinear incidence rate	[13]

The disease we are concerned with may affect the contact times of the infected person (measures taken by the infected person or the disease itself will cause a behavior change of the infected person). At the same time, we regard susceptible persons S as a reference group, and record the ratio of the number of contacts of infected person I to S as η, $\eta \in [0, \infty)$. Also, we say that η is an intensity factor of disease affecting the behavior of the infected person.

Considering η as the observed variable, we have the following description

1. $\eta = 0$**.** This can be regarded as the disease can not be transmitted or infected through regular contact. Infected persons are completely isolated;

2. $\eta \in (0, 1)$**.** It can be seen that when the susceptible person gets sick, the disease will reduce (or the infected person has taken some measures) the number of contacts of the infected person compared to the susceptible person;

3. $\eta = 1$**.** The disease has no effect on the effective contact function $T(N)$;

4. $\eta \in (1, +\infty)$**.** It can be seen that when the susceptible person gets sick, the disease will increase the number of contacts of the infected person compared to the susceptible person.

For the intensity factor, we propose assumptions (A4)–(A5) described below.

(**A4**) $\eta \in (0, 1]$.

(**A5**) $\eta \in \left(0, 1 + \dfrac{\mu}{d}\right]$.

Based on the above discussion, we have established the following system (1.1).

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = \Lambda - dS - \dfrac{\eta\beta T(S + \eta I)S I}{S + \eta I} + \gamma I, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = \dfrac{\eta\beta T(S + \eta I)S I}{S + \eta I} - (d + \mu + \gamma)I, \end{cases} \tag{1.1}$$

where $S(t)$ is the number of susceptible persons in the population. $I(t)$ is the number of infected persons in the population. Λ is the number of constant increase (birth and immigration) of the population in unit time. d is migration (death and emigration) rate of population. β is the probability that a susceptible person will get sick when an effective contact occurs between a susceptible person and an infected person. μ is the case fatality rate. γ is the recovery rate of the disease. η is the intensity factor.

By using $N(t) = S(t) + I(t)$, we can easily obtain a system equivalent to system (1.1).

$$\begin{cases} \dfrac{\mathrm{d}N}{\mathrm{d}t} = \Lambda - dN - \mu I & \triangleq: P(N, I), \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = \dfrac{\eta\beta T(N + \eta I - I)(N - I)I}{N + \eta I - I} - (d + \mu + \gamma)I & \triangleq: Q(N, I). \end{cases} \tag{1.2}$$

where $N(t)$ is the total number of the population. The other parameters are the same as the system (1.1).

Let

$$\begin{aligned} \mathcal{X}_0 &:= \{(\phi_1, \phi_2) | \phi_1 \geq 0,\ \phi_2 \geq 0,\ \phi_1 + \phi_2 > 0\}, \\ \mathcal{Y}_0 &:= \{(\phi_1 + \phi_2, \phi_2) | (\phi_1, \phi_2) \in \mathcal{X}_0\}. \end{aligned}$$

From a biological perspective, it is clear that the dynamics of system (1.1) is a dynamical system in $\mathcal{X}_0$. We obtain the dynamics of (1.1) by studying the dynamics of the equivalent system (1.2) in $\mathcal{Y}_0$.

In Section 2, we analyze the dynamics of system (1.1). First, we calculate equilibra and the basic reproduction number. Then, we study the asymptotic stability of equilibria. We show that there exists a limit cycle. In Section 3, we provide some numerical simulation results which show the existence of a Hopf bifucation. In Section 4, we give some conclusions, discuss the effective contact function and propose future research directions.

2. Dynamics of the model

In this section, we study the dynamics of the model (1.1).

2.1. Equilibra and basic reproduction number

Simple calculations quickly show that system (1.2) admits a disease free equilibrium (DFE) $E_0 = (\Lambda/d, 0)$.

For system (1.2), we use the next generation matrix method [14] to calculate the threshold $\mathcal{R}_0$. Let

$$\begin{cases} F = (\eta\beta T(\Lambda/d)), \\ V = (d + \mu + \gamma). \end{cases}$$

By calculating, we have

$$FV^{-1} = \left(\frac{\eta\beta T(\Lambda/d)}{d + \mu + \gamma}\right).$$

So, we define the threshold of system (1.2) as

$$\mathcal{R}_0 = \rho(FV^{-1}) = \frac{\eta\beta T\,(\Lambda/d)}{d + \mu + \gamma}. \tag{2.1}$$

It is simple to get that E_0 also is the DFE of the system (1.1), and $\mathcal{R}_0$ also is the threshold of (1.1).

Remark 2.1. *β is the probability that a susceptible person will get sick during one active contact with an infected person. $\eta T(N_0)$ is the number of effective contacts with susceptible persons in unit time when an infected person is introduced into a DFE state. $1/(d + \mu + \gamma)$ is the average duration of illness for an infected person. Therefore, the threshold $\mathcal{R}_0$ is the basic reproduction number of systems* (1.1) *and* (1.2)*.*

Theorem 2.1. *Assume* (*A1*) *and* (*A2*) *hold. Then we have the following two conclusions:*

(I) *The DFE E_0 always exists for system* (1.1)*. Furthermore, if $\mathcal{R}_0 \leq 1$, assume* (*A3*) *or* (*A5*) *holds, then the DFE E_0 is the unique equilibrium for system* (1.1)*.*

(II) *If $\mathcal{R}_0 > 1$, then system* (1.1) *has at least one endemic equilibrium (EE). Furthermore, if $\mathcal{R}_0 > 1$, assume* (*A3*) *or* (*A5*) *holds, then system* (1.1) *has a unique EE, E^*.*

Proof. Obviously, the DFE E_0 always exists. We let the endemic equilibria of (1.1), $E^* = (S^*, I^*)$, where $S^* = \Lambda/d - (1 + \mu/d)I^*$, and I^* satisfies the following equation

$$\frac{\eta\beta T(\Lambda/d + (\eta - 1 - \mu/d)I^*)(\Lambda/d - (1 + \mu/d)I^*)}{\Lambda/d + (\eta - 1 - \mu/d)I^*} - (d + \mu + \gamma) = 0.$$

Let $I_m = \Lambda/(d + \mu)$. For $S^* > 0$, we have $I^* \in [0, I_m)$. In $[0, I_m]$, we let

$$H(I) := \frac{\eta\beta T(\Lambda/d + (\eta - 1 - \mu/d)I)(\Lambda/d - (1 + \mu/d)I)}{\Lambda/d + (\eta - 1 - \mu/d)I} - (d + \mu + \gamma).$$

About the continuous function $H(I)$, we have the following conclusions:

$$\begin{aligned} H(0) = &\ \eta\beta T(\Lambda/d) - (d+\mu+\gamma) = (d+\mu+\gamma)(\mathcal{R}_0 - 1), \\ H(I_m) = &\ -(d+\mu+\gamma) < 0, \\ \frac{\mathrm{d}H}{\mathrm{d}I} = &\ (\eta - 1 - \mu/d)\,\frac{\eta\beta T'\left(N_\eta\right)(I_m - I)}{dN_\eta} - \frac{\eta^2\beta T\left(N_\eta\right)N_0}{N_\eta^2} \\ = &\ -\frac{(d+\mu)^2\,\eta\beta T'\left(N_\eta\right)(I_m - I)}{d^2 N_\eta} - \frac{\eta^2\beta(d+\mu)IT'\left(N_\eta\right)}{dN_\eta} \\ &\ -\frac{\eta^2\beta N_0\left(T\left(N_\eta\right) - N_\eta T'\left(N_\eta\right)\right)}{N_\eta^2}, \end{aligned}$$

where $N_\eta = \Lambda/d + (\eta - 1 - \mu/d)I > 0,\ I \in (0, I_m)$.

If $\mathcal{R}_0 > 1$, we have $H(0) > 0$. If $\mathcal{R}_0 \le 1$, we have $H(0) \le 0$. Assume (A3) or (A5) holds. Then, $\mathrm{d}H/\mathrm{d}I < 0$.

By the properties of the continuous function $H(I)$, we have the following conclusions:

(a) If $\mathcal{R}_0 > 1$, then $H(I) = 0$ has at least one root in $(0, I_m)$.

(b) $\mathcal{R}_0 \le 1$. If assume (A3) or (A5) holds, then $H(I) = 0$ has no roots in $(0, I_m)$.

(c) $\mathcal{R}_0 > 1$. If assume (A3) or (A5) holds, then $H(I) = 0$ has a unique root, I^*, in $(0, I_m)$.

Therefore, (I) and (II) are proved. □

It is simple to get that if $E^* = (S^*, I^*)$ is the EE of system (1.1), then $\bar{E}^* = (S^* + I^*, I^*)$ is the EE of system (1.2).

2.2. Local stability of equilibria

We give the local stability of all equilibria of (1.1).

Theorem 2.2. *Assume (A1) and (A2) hold. The following statements are valid for system* (1.1).
(I) *If $\mathcal{R}_0 < 1$, then E_0 is local asymptotically stable. If $\mathcal{R}_0 > 1$, then E_0 is unstable (Saddle).*
(II) *If $\mathcal{R}_0 > 1$, assume (A3) or (A4) holds, then E^* is local asymptotically stable.*

Proof. In order to analyze the local stability of the DFE $E_0 = (\Lambda/d, 0)$ of (1.1), by using Eq (2.1), we linearize system (1.2) at the DFE E_0,

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = -dS + (\gamma - \eta\beta T(\Lambda/d))I, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = (d+\mu+\gamma)(\mathcal{R}_0 - 1)I. \end{cases} \tag{2.2}$$

The Jacobian matrix at E_0 is

$$J(E_0) = \begin{pmatrix} -d & \gamma - \eta\beta T(\Lambda/d), \\ 0 & (d+\mu+\gamma)(\mathcal{R}_0 - 1) \end{pmatrix}.$$

It is easy get that $\lambda_1 = -d$ and $\lambda_2 = (d + \mu + \gamma)(\mathcal{R}_0 - 1)$ are the characteristic roots of $J(E_0)$. It follows that all eigenvalues of $J(E_0)$ have negative real parts if and only if $\mathcal{R}_0 < 1$.

Therefore, if $\mathcal{R}_0 < 1$, E_0 is local asymptotically stable; if $\mathcal{R}_0 > 1$, then E_0 is unstable. The proof of (I) is completed.

The $E^* = (S^*, I^*)$ satisfies the following equation

$$\frac{\eta\beta T(S^* + \eta I^*)S^*}{S^* + \eta I^*} = d + \mu + \gamma. \tag{2.3}$$

We take $T^* = T(S^* + \eta I^*)$, $T^{'*} = T'(S^* + \eta I^*)$. In order to analyze the local stability of E^* of (1.1), by using E^* and (2.3), we linearize system (1.1) at E^*,

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = a_{11}S + a_{12}I, \\ \dfrac{\mathrm{d}I}{\mathrm{d}t} = a_{21}S + a_{22}I, \end{cases} \tag{2.4}$$

where

$$\begin{cases} a_{11} = -\dfrac{\eta\beta T^{'*}S^*I^*}{S^* + \eta I^*} - \dfrac{\eta^2\beta(I^*)^2T^*}{(S^* + \eta I^*)^2} - d, \\ a_{12} = -\dfrac{\eta^2\beta T^{'*}S^*I^*}{S^* + \eta I^*} - \dfrac{\eta\beta(S^*)^2T^*}{(S^* + \eta I^*)^2} + \gamma, \\ a_{21} = \dfrac{\eta\beta T^{'*}S^*I^*}{S^* + \eta I^*} + \dfrac{\eta^2\beta(I^*)^2T^*}{(S^* + \eta I^*)^2}, \\ a_{22} = \dfrac{\eta^2\beta T^{'*}S^*I^*}{S^* + \eta I^*} + \dfrac{\eta\beta(S^*)^2T^*}{(S^* + \eta I^*)^2} - (d + \mu + \gamma). \end{cases}$$

We obtain that the Jacobian matrix at E^* is

$$J(E^*) = \begin{pmatrix} a_{11} & a_{12}, \\ a_{21} & a_{22} \end{pmatrix}.$$

Thus,

$$\begin{aligned} \mathrm{tr}(J(E^*)) &= a_{11} + a_{22} \\ &= -d - a_{21} + a_{22} \\ &= -d - (1 - \eta)\frac{\eta\beta T^{'*}S^*I^*}{S^* + \eta I^*} - \frac{\eta^2\beta T^*I^*(S^* + I^*)}{(S^* + \eta I^*)^2}, \end{aligned} \tag{2.5}$$

$$\begin{aligned} \det(J(E^*)) &= a_{11}a_{22} - a_{21}a_{12} \\ &= -da_{22} + (d + \mu)a_{21} \\ &= (\mu + d - \eta d)\frac{\eta\beta T^{'*}S^*I^*}{S^* + \eta I^*} + \frac{\eta^2\beta T^*I^*(dS^* + (\mu + d)I^*)}{(S^* + \eta I^*)^2}. \end{aligned} \tag{2.6}$$

As $T(N)$ required in (A1)–(A3), we may take

$$T^* > 0,\ T^{'*} > 0,\ (S^* + \eta I^*)T^{'*} - T^* \le 0.$$

Therefore, we have $a_{21} > 0$ and

$$a_{22} = \frac{\eta^2\beta S^* I^* \left((S^* + \eta I^*)T'^* - T^*\right)}{(S^* + \eta I^*)^2} \leq 0.$$

.

If (A1)–(A3) hold, then we have $\mathrm{tr}(J(E^*)) < 0$ and $\det(J(E^*)) > 0$. According to the Routh-Hurwitz criteria, we get that, if the conditions (A1)–(A3) hold, E^* is local asymptotically stable.

Of course, if we replace condition (A3) with (A4), we have

$$T^* > 0,\ T'^* > 0,\ 1 - \eta > 0,\ \mu + d - \eta d > 0.$$

Then we easily get $\mathrm{tr}(J(E^*)) < 0$ and $\det(J(E^*)) > 0$. Thus, E^* is local asymptotically stable. The proof of (II) is completed. □

2.3. Global stability of equilibria

Lemma 2.3. *Assume (A1), (A2) hold. If $\mathcal{R}_0 > 1$, assume (A3) or (A4) holds, then $\bar{E}^*$ is globally asymptotically stable for system* (1.2) *in $\mathcal{Y}_0\backslash N$-axis.*

Proof. For system (1.2), we take the Dulac function $B(N, I)$ as

$$B(N, I) = I^{-1},\ (N, I) \in \mathcal{Y}_0\backslash N\text{-axis}.$$

$$\begin{aligned}
&\frac{\partial(BP)}{\partial N} + \frac{\partial(BP)}{\partial I} \\
=\ & -\frac{d}{I} - (1-\eta)\frac{\beta\eta T'(N + \eta I - I)(N - I)}{N + \eta I - I} - \frac{\beta\eta^2 T(N + \eta I - I)N}{(N + \eta I - I)^2} \\
=\ & -\frac{d}{I} - \frac{\beta\eta T'(N + \eta I - I)(N - I)}{N + \eta I - I} - \frac{\beta\eta^2 I T'(N + \eta I - I)}{(N + \eta I - I)} \\
& -\frac{\beta\eta^2 N}{(N + \eta I - I)^2}\left(T(N + \eta I - I) - (N + \eta I - I)T'(N + \eta I - I)\right).
\end{aligned}$$

Assume (A1), (A2) hold, we have

$$T(N + \eta I - I) > 0,\ T'(N + \eta I - I) > 0,\ (N, I) \in \mathcal{Y}_0.$$

If $\mathcal{R}_0 > 1$, assume (A3) or (A4) holds, then $\bar{E}^*$ is the unique EE and local asymptotically stable. Also, we obtain that

$$\frac{\partial(BP)}{\partial N} + \frac{\partial(BP)}{\partial I} < 0,\ \ (N, I) \in \mathcal{Y}_0\backslash N\text{-axis}.$$

By Theorems 2.1 and 2.2, system (1.2) has only one equilibrium, E_0, in $\mathcal{Y}_0 \cap N$-axis. We have E_0 is unstable. According to the Bendixson-Dulac criteria, the conclusion in the theorem holds. □

So, we have the condition for the stability of equilibria of (1.1).

Theorem 2.4. *Assume (A1), (A2) hold. The undermentioned statements are valid for system* (1.1).

(I) The DFE E_0 always exists.

(II) If $\mathcal{R}_0 < 1$, then the DFE E_0 is locally asymptotically stable.

(III) If $\mathcal{R}_0 < 1$, assume (A3) or (A5) holds, then the DFE E_0 is the unique equilibrium and globally asymptotically stable in $\mathcal{X}_0$.

(IV) If $\mathcal{R}_0 > 1$, then the DFE E_0 is unstable.

(V) If $\mathcal{R}_0 > 1$, then system (1.1) has at least one EE.

(VI) If $\mathcal{R}_0 > 1$, assume (A3) holds, then the EE E^* is the unique EE and globally asymptotically stable in $\mathcal{X}_0 \backslash S$-axis.

(VII) If $\mathcal{R}_0 > 1$, assume (A5) holds, then the EE E^* is the unique EE. Furthermore, assume (A4) holds, then E^* is globally asymptotically stable in $\mathcal{X}_0 \backslash S$-axis.

2.4. Hopf bifurcation

From Theorem 2.4, we can find that complex dynamics in system (1.1) are only possible when the assumption (A3) of the contact function is not valid. We construct a series of simple polynomial functions to satisfy assumptions (A1) and (A2), but assumption (A3) does not hold. Then, we conduct numerical simulations to see if periodic solutions can be found. In the next section, we provide a contact function and corresponding parameter values that enable system (1.1) to discover periodic solutions. In order to analyze how parameter affects the direction of Hopf bifurcation, we may choose η as a bifurcation parameter.

From Theorem 2.2, we obtain that the Jacobian matrix at E^* is

$$J(E^*) = \begin{pmatrix} a_{11} & a_{12}, \\ a_{21} & a_{22} \end{pmatrix}.$$

The characteristic equation is

$$\lambda^2 - \mathrm{tr}(J(E^*))\lambda + \det(J(E^*)) = 0,$$

where $\mathrm{tr}(J(E^*))$ is as shown in (2.5), $\det(J(E^*))$ is as shown in (2.6). If there is an E^* that makes $\mathrm{tr}(J(E^*)) = 0$, $\det(J(E^*)) > 0$, then there exists a periodic solution around the EE E^* for $\Gamma_1 > 0$.

$$\Gamma_1 = \frac{\mathrm{d}\mathrm{tr}(J(E^*)(\eta))}{\mathrm{d}\eta}. \tag{2.7}$$

For η, the Hopf bifurcation point is a solution that holds at the following conditions

$$\begin{aligned} H(I^*) &= 0, \\ \mathrm{tr}(J(E^*)) &= 0, \\ \det(J(E^*)) &> 0, \\ \Gamma_1 &> 0. \end{aligned} \tag{2.8}$$

The Lyapunov number can be computed by [15]

$$\Gamma_2 = \frac{1}{\sqrt{\det(J(E^*))}} \left[\left(\frac{\partial^2 f(S,I)}{\partial S^2} \right)^2 - \left(\frac{\partial^2 f(S,I)}{\partial I^2} \right)^2 \right] \Bigg|_{(S^*,I^*)}, \tag{2.9}$$

where

$$f(S,I) = \frac{\eta\beta T(S + \eta I)SI}{S + \eta I}.$$

Now, we are able to state the results for the Hopf bifurcation.

Theorem 2.5. *If* $\mathcal{R}_0 > 1$, $\mathrm{tr}(J(E^*)) = 0$, $\det(J(E^*)) > 0$, *then system* (1.1) *has a subcritical (supercritical) Hopf bifurcation at* E^* *if* $\Gamma_2 > 0$ *(*$\Gamma_2 < 0$*).*

3. Numeric simulations

In this section, the influence of stability of equilibria of the model on the basic reproduction number $\mathcal{R}_0$, the contact function $T(N)$ and the intensity factor η is investigated. The numerical simulations are performed using MATLAB (R2018a) software. The set of parameter values given in Table 2 and the initial conditions $(S(0), I(0)) = (1, 0.5)$ are used for the simulations.

Table 2. Parameter values used in numerical simulations, the units are dimensionless.

Parameter	Description	Value
Λ	The number of constant increase (birth and immigration) of the population in unit time	2
d	The migration (death and emigration) rate of population	1
μ	The case fatality rate	1
γ	The recovery rate of the disease	1
β	The probability that a susceptible person will get sick when an effective contact occurs between a susceptible person and an infected person	0.8

From Theorems 2.1, 2.2 and 2.4, we find that the relationship between the threshold $\mathcal{R}_0$ and 1 determines the equilibria of the system, but the stability of the equilibriia is also influenced by the selection of contact functions and parameter η. About contact function $T(N)$, we choose $T(N)$ as

$$T(N) = \frac{N^4 - 8N^3 + 18N^2}{9.6}, \tag{3.1}$$

where this $T(N)$ satisfies assumptions (A1) and (A2), and does not satisfy assumption (A3). Because these functions are too broad, the contact function (3.1) is just an example of the complex dynamic behavior where the system (1.1) will exhibit periodic solutions when the conditions in Theorem 2.4 are not met.

Figures 1 and 2 show the simulation of susceptible person $S(t)$ for the connect function $T(N) = 2$, $T(N) = 2N$, with η being a variable, the given initial conditions and other parameter values in Table 2. From the figure, when $\mathcal{R}_0 < 1$, susceptible individuals ultimately reach their maximum scale. When $\mathcal{R}_0 > 1$, susceptible individuals ultimately reach a stable value. As $\mathcal{R}_0$ increases ($\mathcal{R}_0 > 1$), the final size of susceptible individuals also gradually decreases. These results are consistent with the conclusions in Theorem 2.4.

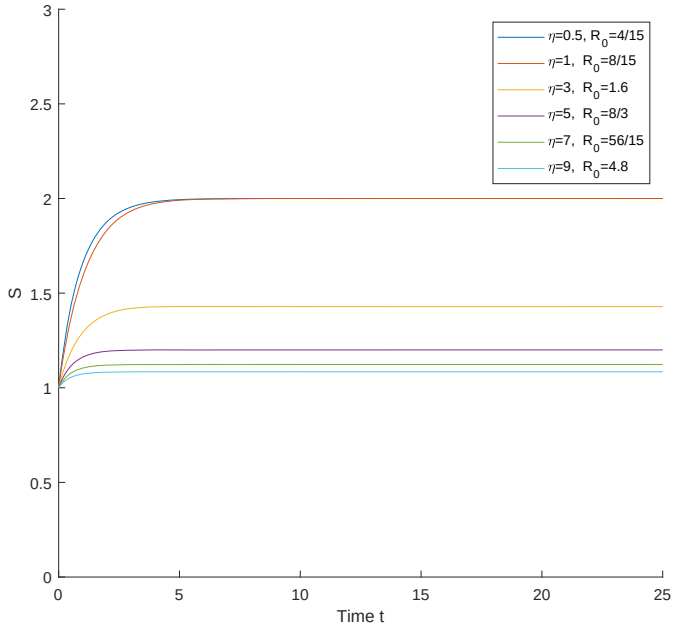

Figure 1. Simulation results illustrating the number of susceptible persons for the connect function $T(N) = 2$, η as variable, the given initial conditions and other parameter values in Table 2.

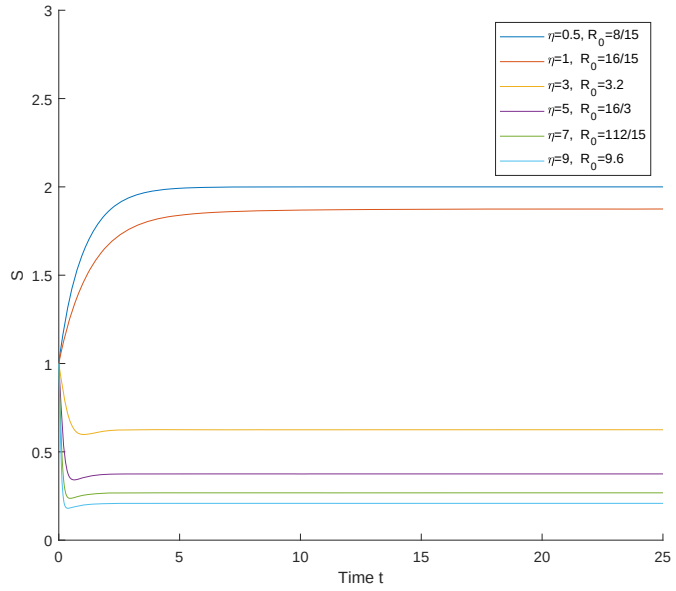

Figure 2. Simulation results illustrating the number of susceptible persons for the connect function $T(N) = 2N$, η as variable, the given initial conditions and other parameter values in Table 2.

Figure 3 shows the simulation of susceptible person $S(t)$ for the connect function $T(N)$ in (3.1), with η being a variable, the given initial conditions and other parameter values in Table 2. From the figure, it can be seen that as η increases, the basic regeneration number $\mathcal{R}_0$ linearly increases. and the disease progresses from extinction to epidemic, while there may be periodic epidemics. And as η increases, the final size of susceptible individuals continues to decline, and the scale of disease outbreaks is increasing.

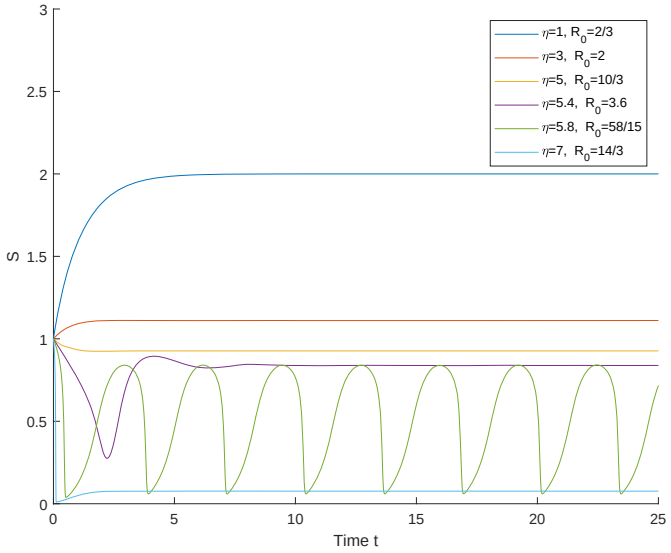

Figure 3. Simulation results illustrating the number of susceptible persons for the connect function $T(N)$ using (3.1), η as variable, the given initial conditions and other parameter values in Table 2.

Figure 4 is a bifurcation diagram drawn using Matcont, where $\eta = 5.5273$ and $\eta = 6.0396$ are Hopf bifurcation points, and their first Lyapunov exponents are 22.6683 and -23.1353 respectively. It can be seen that when system (1.1) is at $\eta \in (5.5273, 6.0396)$, other parameter values in Table 2 and the connect function $T(N)$ using (3.1), the equilibrium of endemic disease disease is unstable, and there is a stable limit cycle, that is, the disease will eventually circulate periodically.

4. Conclusions and discussion

In this paper, we have studied an SI model that has the general contact function and the impact of diseases on infected persons. We have provided some sufficient conditions for the ultimate extinction or epidemic of the disease. This model admits two types of equilibria and the global dynamics are determined by the basic reproduction number $\mathcal{R}_0$ and the intensity factor η. It must be pointed out that the results obtained in this paper only apply to simple cases where the contact function $T(N)$ satisfies the assumptions (A1) and (A2), as well as partially satisfies (A3).

From a biological perspective, assumptions (A1) and (A2) are usually valid. From Theorems 2.1, 2.2 and 2.4, if assumption (A3) holds, system (1.1) will only have two types of the equilibria, E_0 and E^*, and their global dynamics are determined by the basic reproduction number $\mathcal{R}_0$. If assumption

(A3) does not hold, assumption (A5) and the relationship between the threshold $\mathcal{R}_0$ and 1 are sufficient conditions for Theorem 2.1 (Theorems 2.2 and 2.4) to hold. The necessary and sufficient conditions for the theorems to hold true regarding the range of η are work that needs to be further studied.

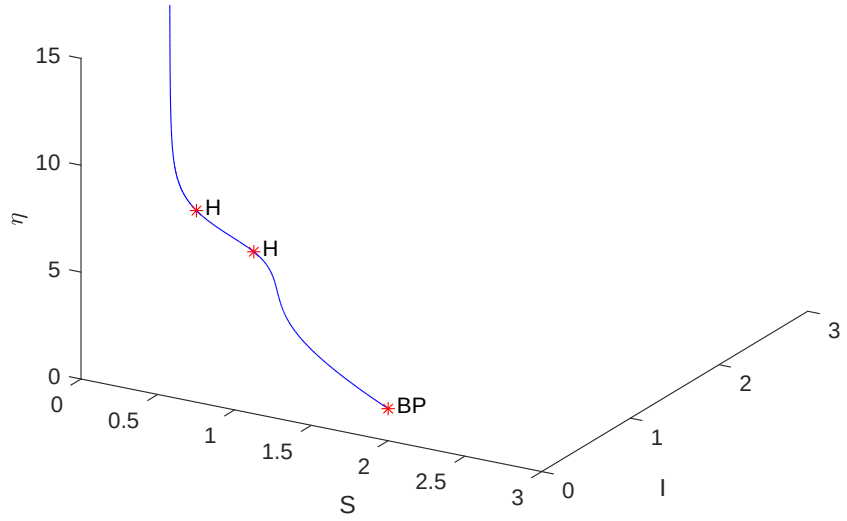

Figure 4. Bifurcation diagram of system (1.1) regarding parameters η, where other parameter values are in Table 2, and the connect function $T(N)$ uses (3.1).

Numerical simulations were conducted to show that if the contact function $T(N)$ satisfies assumptions (A1) and (A2), and does not satisfy assumption (A3), system (1.1) may have a Hopf bifurcation, resulting in a stable periodic solution. Whether there will be other bifurcations is still a question that needs to be studied. This reveals that the epidemic of the disease cycle may be related to the mode of disease transmission and the impact of the disease on patient behavior patterns. When a disease is prevalent, behavioral constraints on potential carriers may be more efficient public health policies.

$T(N)$ describes the effective number of contacts between individuals within a population, and the population activity habits at different times and regions are usually not consistent. Therefore, when studying practical problems, it is necessary to analyze them in a specific manner and not use the same contact function $T(N)$ without thinking. The dynamic behaviors of models where the contact functions do not satisfy assumption (A3) require further study. Our main research in the future is to find a type of contact function that makes Hopf branches widely exist.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declared that they have no conflicts of interest to this work.

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