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SICR rumor spreading model in complex networks: Counterattack and self-resistance

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HIGHLIGHTS

- We build two new rumor spreading models which consider the counterattack mechanism.
- We study the self-resistance feature of networks and its influence on rumor spreading.
- There do not exist rumor thresholds in the two new models.
- The final state of the rumor spreading process increases as self-resistance increases.
- The peak value of infective density decreases as self-resistance increases.

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ABSTRACT

Rumor is an important form of social interaction. However, spreading of harmful rumors could have a significant negative impact on the well-being of the society. In this paper, considering the counterattack mechanism of the rumor spreading, we introduce two new models: Susceptible–Infective–Counterattack–Refractory (SICR) model and adjusted-SICR model. We then derive mean-field equations to describe their dynamics in homogeneous networks and conduct the steady-state analysis. We also introduce the self-resistance parameter τ, and study the influence of this parameter on rumor spreading. Numerical simulations are performed to compare the SICR model with the SIR model and the adjusted-SICR model, respectively, and we investigate the spreading peak of the rumor and the final size of the rumor with various parameters. Simulation results are congruent exactly with the theoretical analysis. The experiment reveals some interesting patterns of rumor spreading involved with counterattack force.

1. Introduction

Rumor is usually defined as the unconfirmed elaboration or annotation of the public interesting things, events or issues that spread through various channels, in itself neither true nor false [1,2]. Rumor is an important form of social interaction, and its spreading has a great impact on human lives. Sometimes the rumor spreading may play a positive role, for example, we can utilize the rapid and efficient characteristic of rumor spreading to wake general public awareness and lead them to

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take pertinent precaution measures [3]; however, most rumors induce panic psychology or potential loss in the accompanying unexpected events [4]. So how to control and prevent the spreading of harmful rumors is a valuable research topic.

To understand the mechanism of the rumor propagation, it is helpful to study rumor spreading models in complex networks and to explore the pattern and structure of such a model. Rumor models can be used to elaborate many phenomena, including the dissemination of information, viral marketing, panics caused by epidemics or emergencies [2,5]. The classical model for the spread of rumor is the Daley–Kendal (DK) model, which has been used extensively for quantitative studies of rumor spreading [6–8], but major shortcomings of these models are that they did not take into account the topological characteristics of social networks and were not suitable for describing the rumor spreading mechanism on large-scale social networks. Sudbury [9] studied dynamic mechanisms of information transmission on social networks and insisted that the dynamic behavior of rumor spreading matched the SIR (susceptible–infective–refractory) model for epidemiology. Zanette [10,11] established a rumor spreading model on small-world networks and provided a threshold of rumor spreading. Moreno [12] studied the stochastic DK model on scale-free networks and claimed that the uniformity of networks had a significant impact on the dynamic mechanism of rumor spreading. Isham [13] studied the final size distribution of rumors on general networks. Liu et al. [14] and Zhou et al. [15] revealed that the final percentage of population who heard the rumor decreases with a network structure parameter p. Zhao et al. [16] considered a forgetting mechanism and researched rumor spreading on Live Journal. And based on the SIR model, Zhao et al. [17] presented a SIHR rumor spreading model by adding a new group (Hibernators).

However, most of the previous models did not consider that a susceptible individual may not agree with the rumor when he/she hears it. In social networks, different people may have different views to a rumor, so when a person hears a rumor which is in serious conflict with his/her belief, he/she may counterattack the rumor, and even do the best to prevent the rumor propagation. In fact, this is the self-resistance feature of networks to rumor spreading, which is different from immunization strategies in many previous studies. The expression of the self-resistance of networks and the study of its effect are the main subjects of this paper. Based on the discussion above, we add a new counterattack group (C) to the classical SIR model and build a new rumor spreading model, referred to as the SICR (susceptible–infective–counterattack–refractory) rumor spreading model. Moreover, in the real life, sometimes we encounter a special psychological phenomenon in the rumor spreading: certain spreader (such as nosy Parker) enjoys spreading rumors and does it cheerfully. When such an infective node i contacts another infective node j, then instead of becoming refractory, the node i may get the impression that many more people out there are still interested in the rumor, and so the node i decides to remain as an infective node and continues to spread the rumor. In order to study this phenomenon, we propose the adjusted-SICR model, which is a special case of the SICR model.

Within the mechanism of the SICR model, when a susceptible node contacts an infective node, it may become a counterattack node with a certain probability. A counterattack individual will persuade the infective neighbors to not continue spreading the rumor and become refractory nodes with probability η. An infective node may become a refractory individual with probability γ when it contacts another infective node or a refractory node. In general, we assume that the two probabilities, η and γ, are different. Singh et al. [18] and Gu et al. [19] also included the reject state into the rumor spreading model, but in their models, the probabilities γ and η were considered the same, and so did not further emphasize the opposition and restraint functions of the counterattack group. In fact, their models are two special cases of the models discussed in this paper.

The paper is organized as follows. In Section 2, we present the propagation mechanism of the SICR model and the adjusted-SICR model in complex networks, and derive their mean-field equations to describe the dynamics in homogeneous networks, respectively. In Section 3, we provide analytical results for the steady-state of both SICR model and adjusted-SICR model. In Section 4, a numerical simulation in the Strogatz–Watts (SW) network is performed to test and verify the theoretical results. Finally, conclusions are given in Section 5.

2. SICR rumor propagation model

As mentioned earlier, we build a susceptible–infective–counterattack–refractory (SICR) rumor spreading model by including an additional group in which the individuals refute the rumor. This group, referred to as counterattack (C), comes from the susceptible nodes with a certain probability. We assume that the rumor is disseminated by direct contacts of infective nodes with others, and the population is divided into four groups: susceptible (S), infective (I), counterattack (C), refractory (R), where S, I, C, R represent, respectively, the people who never heard the rumor (Susceptible), those who are spreading the rumor (Infective), those who do not agree but refute the rumor, and persuade neighbors not to believe and spread it (Counterattack), and the ones who heard the rumor but have lost interest in diffusing it (Refractory). From now on, we refer to the SICR rumor spreading model as the SICR model, in short.

As shown in Fig. 1, the rules of the SICR model and their expressions can be summarized as follows.

(1) When a susceptible node contacts an infective node, the susceptible node may have three outcomes: (i) it becomes an infective node with probability α, namely spreading rate; (ii) it becomes a refractory node with probability β, namely ignoring rate; (iii) it becomes a counterattack node with probability θ, namely refuting rate. Here we assume that the state C is a constant state, i.e., once a node becomes state C, it would keep the state C until the end, which is similar to the state R:

$$S_i + I_j \xrightarrow{\alpha} I_i + I_j,$$

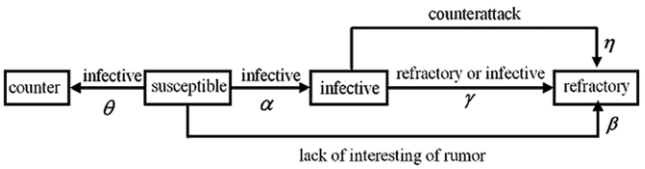

Fig. 1. Structure of the rumor spreading process.

$$S_i + I_j \xrightarrow{\beta} R_i + I_j, \tag{1}$$
$$S_i + I_j \xrightarrow{\theta} C_i + I_j.$$

(2) When an infective node contacts another infective or a refractory node, it becomes a refractory node with probability γ, namely stifling rate:

$$I_i + I_j \xrightarrow{\gamma} R_i + I_j, \tag{2}$$
$$I_i + R_j \xrightarrow{\gamma} R_i + R_j. \tag{3}$$

(3) If a counterattack node has infective neighbor nodes, then the infective neighbors become refractory nodes with probability η, namely persuading rate. Here we consider that the counterattack nodes do not persuade the susceptible neighbors, because the susceptible neighbors do not know the rumor and thus do not spread it:

$$I_i + C_j \xrightarrow{\eta} R_i + C_j. \tag{4}$$

Consider a closed and homogeneously mixed population consisting of N individuals as a social network, where individuals are nodes and contacts between individuals are edges. Under a homogeneously mixing assumption, the following parameters are discussed in the homogeneous network: $S(t)$, $I(t)$, $C(t)$, and $R(t)$ denote the density of population that are susceptible nodes, infective nodes, counterattack nodes, and refractory nodes at time t, respectively, where $S(t) + I(t) + C(t) + R(t) = 1$.

Considering the rumor spreading mechanism, the mean-field equations of the SICR model can be described as follows:

$$\frac{dS(t)}{dt} = -(\alpha + \beta + \theta)\langle k\rangle S(t)I(t), \tag{5}$$
$$\frac{dI(t)}{dt} = \alpha\langle k\rangle S(t)I(t) - \eta\langle k\rangle I(t)C(t) - \gamma\langle k\rangle I(t)(R(t) + I(t)), \tag{6}$$
$$\frac{dC(t)}{dt} = \theta\langle k\rangle S(t)I(t), \tag{7}$$
$$\frac{dR(t)}{dt} = \beta\langle k\rangle S(t)I(t) + \eta\langle k\rangle I(t)C(t) + \gamma\langle k\rangle I(t)(R(t) + I(t)), \tag{8}$$

where $\langle k\rangle$ denotes the average degree of the network.

As mentioned earlier, in an adjusted-SICR model, an infective individual may become refractory at stifling rate γ only when he/she contacts a refractory neighbor. And considering the rumor spreading rules (1), (3), (4), the mean-field equations of an adjusted-SICR model can be described as follows:

$$\frac{dS(t)}{dt} = -(\alpha + \beta + \theta)\langle k\rangle S(t)I(t),$$
$$\frac{dI(t)}{dt} = \alpha\langle k\rangle S(t)I(t) - \eta\langle k\rangle I(t)C(t) - \gamma\langle k\rangle I(t)R(t), \tag{9}$$
$$\frac{dC(t)}{dt} = \theta\langle k\rangle S(t)I(t),$$
$$\frac{dR(t)}{dt} = \beta\langle k\rangle S(t)I(t) + \gamma\langle k\rangle I(t)R(t) + \eta\langle k\rangle I(t)C(t). \tag{10}$$

We assume that there is only one spreader at the beginning of the rumor spreading. When $t = 0$, the initial condition of rumor spreading is given as follows:

$$S(0) = \frac{N-1}{N} \approx 1(N \to \infty), \qquad I(0) = \frac{1}{N} \approx 0, \qquad C(0) = 0, \qquad R(0) = 0. \tag{11}$$

Note that for a susceptible individual, there are three possible outcomes once he/she has heard of the rumor, so we have $\alpha+\beta+\theta \leqslant 1$, and $(1-\alpha-\beta-\theta)$ is the probability that no one tells him/her the rumor. Note that the classical SIR rumor spreading model, Singh's model [18] and Gu's model [19] are special cases of the new models in this paper, as illustrated below:

Case (i): When $\theta = 0$, no matter η equals 0 or not, the SICR model becomes the classical SIR model, because if $\theta = 0$, then $C(t) = 0$, and the counterattack group would be ineffective in the spreading process.

Case (ii): When $\gamma = \eta$, the SICR model is equivalent to Singh's model, and the adjusted-SICR model is equivalent to Gu's model.

3. Steady-state analysis

In the process of rumor spreading, the number of infective nodes at first increases, then decreases and reaches zero when the rumor dies out. At that time, the system reaches an equilibrium state and has only susceptible, counterattack and refractory nodes. Denote the final state of the susceptible group by S, where $S = S(\infty) = \lim_{t\to\infty} S(t)$. Similarly, denote the final state of the infective, counterattack and refractory groups by I, C, R, respectively. Note that $S(t)+I(t)+C(t)+R(t) = 1$ for any given time t and $I = 0, S + C + R = 1$. For the convenience of discussion, we often use the indicator $C + R = 1 - S$, which measures the level of rumor influence. Taking $S = 0.15$ as an example, it means that 85% of individuals, who are in the counterattack or refractory state, have heard of the rumor at the end. Another indicator that can be used to measure the maximum rumor influence is the peak value of the infective density, denoted by $I_{\max}$, which represents the highest density of people who spread the rumor. So, to some extent, $I_{\max}$ is the larger impact of rumor spreading.

This paper also investigates the self-resistance of networks to the rumor, which is mainly reflected in the reduction of infective nodes. According to Eq. (6), there are two conditions which may influence an infective individual to change his/her state: first, when meeting a counterattack neighbor, he/she may become refractory with probability η; and second, when contacting another infective or refractory neighbor, he/she may become refractory with probability γ. According to Eq. (7), the probability that he/she has a counterattack neighbor is proportional to θ, and from the sum of Eqs. (6) and (8), the probability that he/she has an infective or refractory neighbor is proportional to $\alpha+\beta = 1-\theta$. Therefore, $\theta\eta$ reflects the reduction rate of infective nodes due to the first condition, and $(1-\theta)\gamma$ reflects the reduction rate of infective nodes due to the second condition. Now, we combine these two reduction rates and introduce a new parameter $\tau = \theta\eta + (1-\theta)\gamma$, which represents the self-resistance of networks to the rumor, and reflects the obstructive factor of networks themselves.

3.1. Steady-state analysis of SICR model

We now proceed to the steady-state analysis of the SICR model.[1] Based on Eqs. (5)–(8) and (11) we see that

$$C(t) = \frac{\theta}{\alpha+\beta+\theta}(1-S(t)), \tag{12}$$

$$R(t)+I(t) = \left(1-\frac{\theta}{\alpha+\beta+\theta}\right)(1-S(t)), \tag{13}$$

$$R(t) = \frac{1}{\alpha+\beta+\theta}\left(\gamma-\beta+\frac{\eta\theta-\gamma\theta}{\alpha+\beta+\theta}\right)(S(t)-1) - \frac{1}{\alpha+\beta+\theta}\left(\gamma+\frac{\eta\theta-\gamma\theta}{\alpha+\beta+\theta}\right)\ln S(t). \tag{14}$$

Notice that $R = 1-S-C$, so we have

$$S = \mathrm{e}^{-\varepsilon(1-S)}, \tag{15}$$

$$R + C = 1 - \mathrm{e}^{-\varepsilon(R+C)}, \tag{16}$$

where

$$\varepsilon = 1 + \frac{(\alpha+\beta+\theta)\alpha}{(\alpha+\beta)\gamma+\theta\eta} \geq 1.$$

Obviously, Eq. (16) is the same as the transcendental equation of the SIR rumor spreading model [12].

Note that the SICR model pays more attention to the influence of the infective group and the counterattack group in the rumor spreading process, so we may assume that $\alpha \neq 0$, $\theta \neq 0$, and thus $\varepsilon > 1$.

Theorem 1. *For $\varepsilon > 1$, the equation $x = \mathrm{e}^{-\varepsilon(1-x)}$ has two solutions: $x = 1$ and a nontrivial solution x_1, where $0 < x_1 < 1 - \frac{\ln\varepsilon}{\varepsilon}$.*

[1] The detailed derivation process and the proofs of theorems are given in the Appendix.

According to Theorem 1, Eq. (15) always admits the trivial solution $S = 1$. In the mean time, since $\varepsilon > 1$, all nontrivial values of the parameters α, β, γ, η and θ can be realized and Eq. (15) has another solution S_1, where $0 < S_1 < 1 - \frac{\ln \varepsilon}{\varepsilon}$. That is, there does not exist a rumor threshold in the SICR model. Moreover, if $\alpha = 0$, then $\varepsilon = 1$, and the equation $S = e^{-(1-S)}$ has only the trivial solution $S = 1$. For this case, Eq. (15) is rational, because $\alpha = 0$ means that there is no rumor spreading, and all people in the network never heard the rumor.

Another indicator which measures the maximum rumor influence is the peak value of the infective density $I_{\max}$. Without loss of generality, we assume $\alpha + \beta + \theta = 1$. From Eqs. (13) and (14), we have

$$I_{\max} = \alpha + \tau \ln \tfrac{\tau}{\tau+\alpha}\,. \tag{17}$$

We next present the influence relations among S_1, $I_{\max}$ and the parameters in the next two theorems.

Theorem 2. *Let $\alpha + \beta + \theta = 1$. If the parameters α, θ, τ in the SICR model are nonzero, then we have the following:*

(1) *For fixed β, γ, η, the final state S_1 decreases as α increases, and increases as θ increases;*
(2) *For fixed α, β, θ, the final state S_1 increases as τ increases;*
(3) *For fixed α, β, θ, the final state S_1 increases as γ increases.*

Theorem 3. *Let $\alpha + \beta + \theta = 1$. If the parameters α, θ, τ in the SICR model are nonzero, then we have the following:*

(1) *For fixed β, γ, η, the peak value of the infective density $I_{\max}$ increases as α increases, and decreases as θ increases;*
(2) *For fixed α, β, θ, the peak value of the infective density $I_{\max}$ decreases as τ increases;*
(3) *For fixed α, β, θ, the peak value of the infective density $I_{\max}$ decreases as γ increases, and decreases as η increases.*

3.2. Steady-state analysis of adjusted-SICR model

According to the spreading mechanism of the adjusted-SICR model, when an infective node i contacts another infective node j, instead of becoming refractory with a probability, the node i may remain as an infective node. Assuming $\alpha+\beta+\theta = 1$ for simplicity, and $\alpha \neq 0$, $\theta \neq 0$, we do the steady-state analysis on the adjusted-SICR model as follows.

From Eqs. (5), (10), and (11), we have

$$\gamma(\beta - \eta\theta)S(t)^{\gamma+1} + (\gamma+1)(\gamma R(t) + \eta\theta)S(t)^{\gamma} = \beta\gamma + \eta\theta.$$

Note that $R = 1 - S - C = (1-\theta)(1-S)$, so the relational equation between S and R in the adjusted-SICR model becomes

$$-(\tau+\alpha)\gamma S^{\gamma+1} + (\gamma+1)\tau S^{\gamma} = \beta\gamma + \eta\theta. \tag{18}$$

We notice that the transcendental equation of the adjusted-SICR model is very different from the transcendental equations of the SIR model [12] and the SICR model.

Theorem 4. *If $x \geq 0$, then the equation*

$$-(\tau+\alpha)\gamma x^{\gamma+1} + (\gamma+1)\tau x^{\gamma} = \beta\gamma + \eta\theta$$

has two positive solutions, $x = 1$ and a nontrivial solution x_2, where $0 < x_2 < \frac{\tau}{\tau+\alpha}$.

According to Theorem 4, Eq. (18) always admits the trivial solution $S = 1$. And for all nontrivial values of the parameters α, β, γ, η and θ, Eq. (18) also has another solution S_2, where $0 < S_2 < \frac{\tau}{\tau+\alpha}$, which implies that there does not exist a rumor threshold in the adjusted-SICR model.

4. Numerical simulation

To support our analysis of the SICR model in Section 3, we perform numerical simulations in the homogeneous networks. Without loss of generality, we consider the Watts–Strogatz (WS) small-world network with size $N = 10^4$ and the average degree $\langle k \rangle = 6$, and the initial condition has only one infective individual in the network, thus $S(0) = \frac{10^4-1}{10^4}$, $I(0) = \frac{1}{10^4}$, $C(0) = 0$, $R(0) = 0$.

Fig. 2 shows the general trends of the four types of groups in the SICR model. From the simulation we see that there is a sharp increase in the density of infective people as they begin to propagate a rumor. With further spreading of the rumor, the density of infective people reaches a peak and thereafter declines. Finally, the density of infective people is zero and this leads to the termination of rumor spreading. In this process, the density of susceptible people always reduces while the density of refractory people always increases until they reach the stability, respectively. The variation trend of the density of counterattack people is similar to that of the refractory, which increases until it reaches the balance. But the trend of increasing process of the counterattack group is much more moderate than the refractory, and the numerical changes of the counterattack mainly depend on the refuting rate θ.

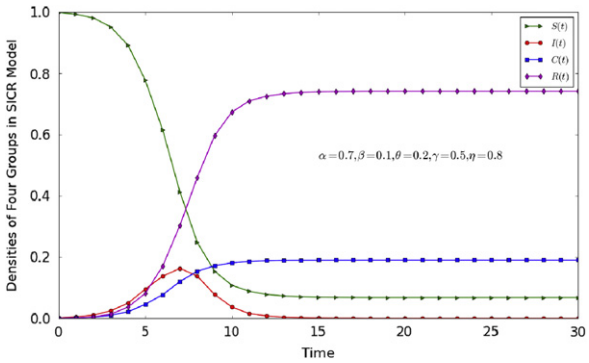

Fig. 2. Densities of susceptible (S), infective (I), counterattack (C), and refractory (R) over time with $\alpha = 0.7$, $\beta = 0.1$, $\theta = 0.2$, $\gamma = 0.5$, $\eta = 0.8$.

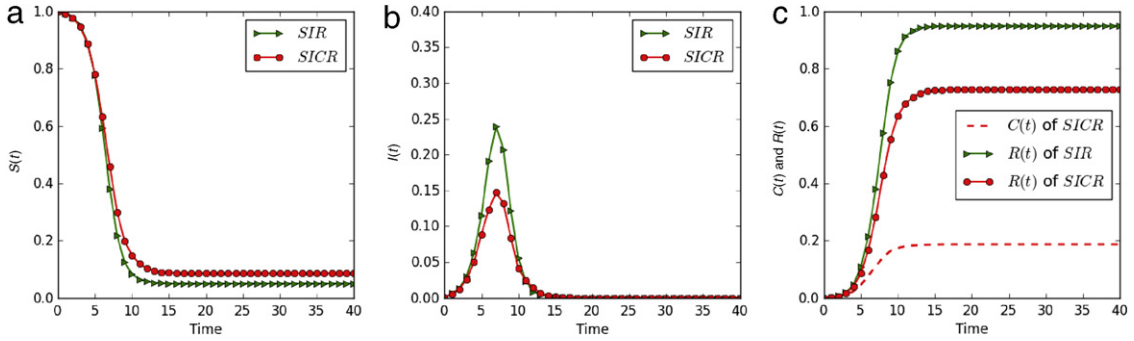

Fig. 3. Compare the density of each group over time in the SICR model with that in the SIR model, where $\alpha = 0.7$, $\beta = 0.1$, $\theta = 0.2$, $\gamma = 0.5$, and $\eta = 0.8$ in the SICR model.

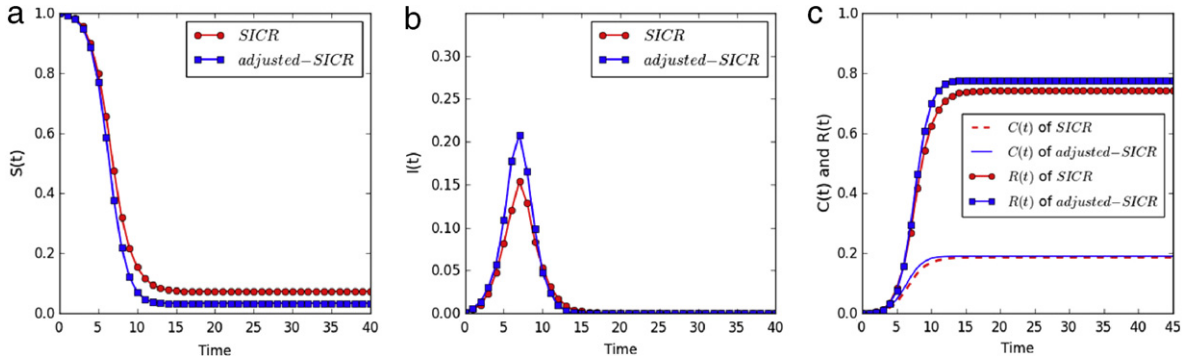

Fig. 4. Compare the density of each group over time in the SICR model with that in the adjusted-SICR model, where $\alpha = 0.7$, $\beta = 0.1$, $\theta = 0.2$, $\gamma = 0.5$, $\eta = 0.8$.

As mentioned in Section 2, when $\theta = 0$, the SICR model becomes the classical SIR model. The reason is that, if $\theta = 0$, then $C(t) = 0$ and the counterattack group would be ineffective in the spreading process. In order to demonstrate the effect of the new counterattack group in the rumor spreading process, we perform a simulation to compare the process of the SICR model with the traditional SIR model under the same parameters (see Fig. 3), where $\alpha = 0.7$, $\beta = 0.1$, $\theta = 0.2$, $\gamma = 0.5$ in both SICR model simulation and SIR simulation. Note that the parameter η does not exist in the SIR model, and we set $\eta = 0.8$ in the SICR simulation. From Fig. 3(a) we see that the final susceptible scale of the SICR model is higher than that of the SIR model, which means that more individuals would not be affected by the rumor spreading if including the counterattack group in the rumor spreading model. Fig. 3(b) shows that the value $I_{\max}$ of the SICR model is smaller than that of the SIR model, which means that the existence of the counterattack group can prevent the speed of rumor spreading and reduce the highest density of people who are spreading the rumor. Fig. 3(c) indicates the range of rumor influence at the end.

In this paper, an adjusted-SICR rumor model is also proposed. The main difference between the SICR model and the adjusted-SICR model is that we use both of rules (2) and (3) in the former and only rule (3) in the latter. We also simulate their spreading processes with the same parameters $\alpha = 0.7$, $\beta = 0.1$, $\theta = 0.2$, $\gamma = 0.5$, $\eta = 0.8$ (see Fig. 4). From Fig. 4(a)

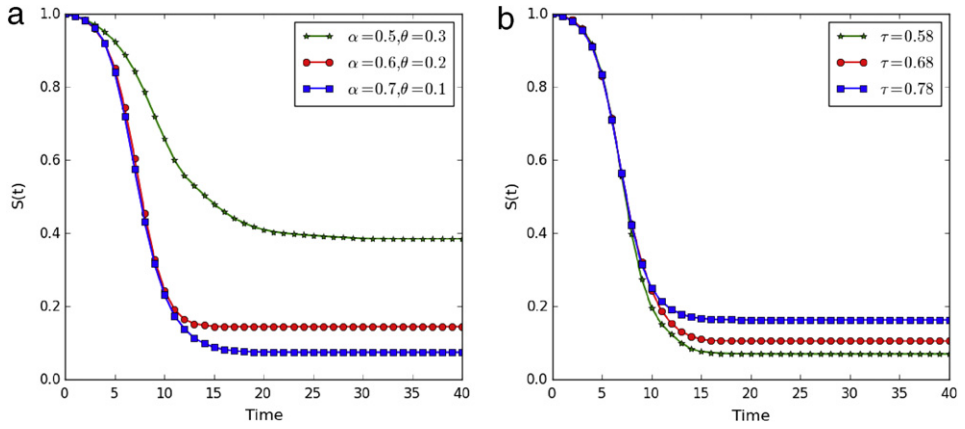

Fig. 5. Density of the susceptible group over time under different parameters in the SICR model. Suppose $\alpha+\beta+\theta=1$, (a) for $\beta=0.2$, $\gamma=0.5$, $\eta=0.6$, the densities of the susceptible group over time under different α or θ; (b) for $\alpha=0.7$, $\beta=0.1$, $\theta=0.2$, the densities of the susceptible group over time under different τ. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

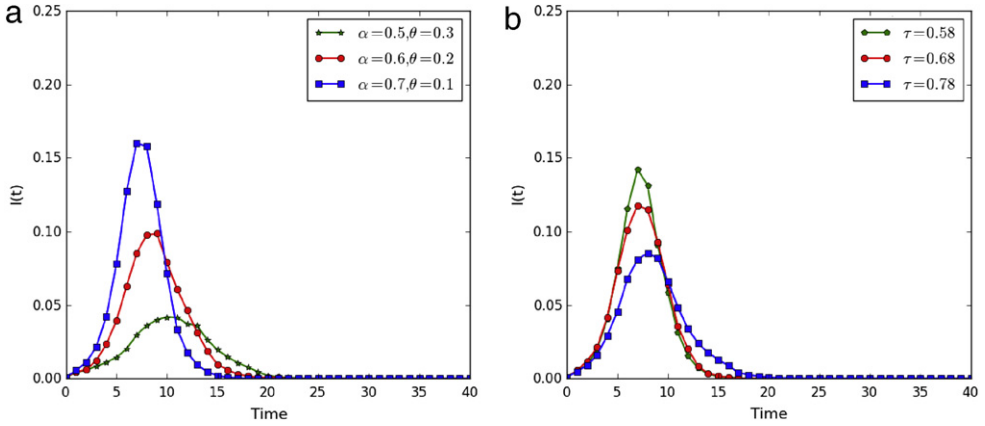

Fig. 6. Densities of the infective group over time under different parameters in the SICR model. Suppose $\alpha+\beta+\theta=1$, (a) for $\beta=0.2$, $\gamma=0.4$, $\eta=0.5$, the densities of the infective group over time under different α (or θ); (b) for $\alpha=0.7$, $\beta=0.1$, $\theta=0.2$, the densities of the infective group over time under different τ. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

we observe that the final susceptible scale of the adjusted-SICR model is below that of the SICR model. Fig. 4(b) shows that the $I_{\max}$ of the adjusted-SICR model is higher than that of the SICR model. Fig. 4(a) and (b) tell us that according to the spreading rules of the adjusted-SICR model, the rumor would spread faster than that of the SICR model, and it would affect more individuals at the end. Fig. 4(c) indicates the range of rumor influence and the final state of the influenced individuals at the end.

Fig. 5 displays the densities of the susceptible group over time under different parameters in the SICR model. Suppose $\alpha+\beta+\theta=1$. In Fig. 5(a), let $\beta=0.2$, $\gamma=0.5$, $\eta=0.6$, and the green star marker line, red circle marker line and blue square marker line represent the different value pairs of (α,θ) : $(0.5,0.3)$, $(0.6,0.2)$ and $(0.7,0.1)$, respectively. From Fig. 5(a), we see that keeping the values of β,γ,η unchanged, the final state of the susceptible group decreases as α increases, and increases as θ increases. In Fig. 5(b), let $\alpha=0.7$, $\beta=0.1$, $\theta=0.2$, and the green star marker line, red circle marker line and blue square marker line represent three different value pairs of (γ,η) : $(0.5,0.6)$, $(0.6,0.7)$, $(0.7,0.8)$, which correspond to $\tau=0.58$, $\tau=0.68$, $\tau=0.78$, respectively. From Fig. 5(b), we see that fixing the values of α,β,θ, the final state of the susceptible group increases as τ increases. Obviously, the simulation demonstrates the same result as Theorem 2 in Section 3.

Fig. 6 displays the densities of the infective group over time under different parameters in the SICR model. Suppose $\alpha+\beta+\theta=1$. In Fig. 6(a), for $\beta=0.2$, $\gamma=0.4$, $\eta=0.5$, let the green star marker line, red circle marker line and blue square marker line represent the different value pairs of (α,θ): $(0.5,0.3)$, $(0.6,0.2)$ and $(0.7,0.1)$, respectively. From Fig. 6(a), we see that when the values of β,γ,η are unchanged, $I_{\max}$ (the peak value of the infective density) increases as α increases, and decreases as θ increases. In Fig. 6(b), for $\alpha=0.7$, $\beta=0.1$, $\theta=0.2$, let the green star marker line, red circle marker line and blue square marker line represent the different value pairs of (γ,η) : $(0.5,0.6)$, $(0.6,0.7)$, $(0.7,0.8)$, which correspond to $\tau=0.58$, $\tau=0.68$, $\tau=0.78$, respectively. From Fig. 6(b), we see that fixing the values of α,β,θ, $I_{\max}$ decreases as τ increases. Obviously, the simulation results are congruent exactly to Theorem 3 in Section 3.

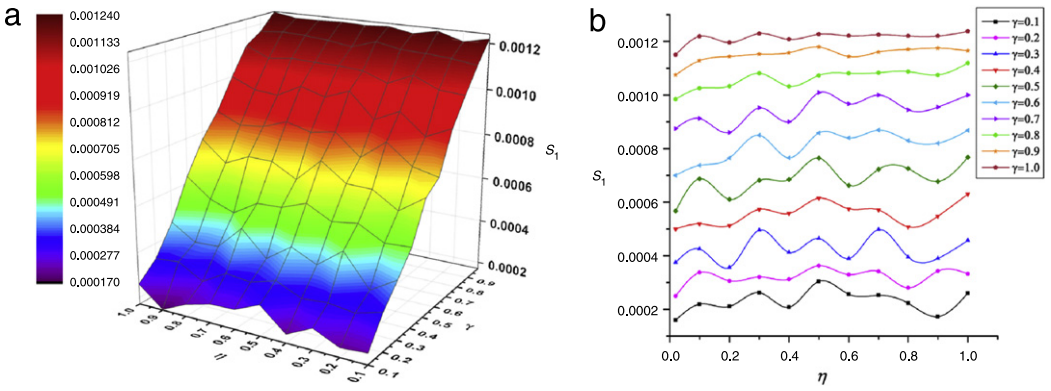

Fig. 7. For $\alpha = 0.5$, $\beta = 0.1$, $\theta = 0.4$, the final susceptible density S_1 under different γ and η in the SICR model.

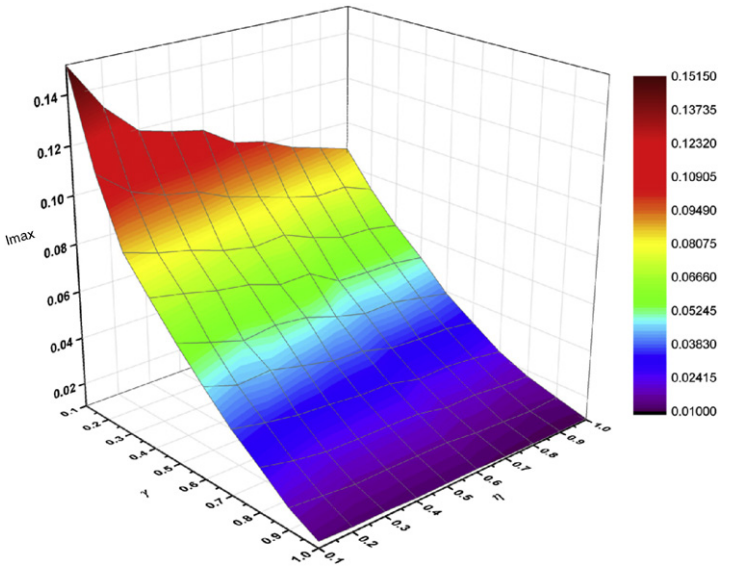

Fig. 8. For $\alpha = 0.5$, $\beta = 0.1$, $\theta = 0.4$, the peak values $I_{\max}$ of the infective density under different γ and η in the SICR model.

Fig. 7 shows the final susceptible density S_1 under different γ and η in the SICR model. Suppose $\alpha+\beta+\theta = 1$. In Fig. 7(a), let $\alpha = 0.5$, $\beta = 0.1$, $\theta = 0.4$; we see that the final state S_1 significantly increases as γ increases, which is consistent with the conclusion (3) of Theorem 2 in Section 3. But with the increase of η, we do not see the same increase trend of S_1. And from Fig. 7(b), for each fixed γ, we see that the value of S_1 fluctuates with the increase of η. Fig. 8 shows the peak value $I_{\max}$ of the infective density under different γ and η in the SICR model. From Fig. 8, we note that the peak value $I_{\max}$ of the infective density decreases as γ and η increase, respectively. The simulation is consistent with the conclusion (3) of Theorem 3 in Section 3. Furthermore, we observe that with the increase of γ, the decrease trend of $I_{\max}$ is more dramatic, while with the increase of η, the decrease trend of $I_{\max}$ is more gentle.

5. Conclusions

In this paper, considering the counterattack mechanism of the rumors' spreading, we add a new group, counterattack individuals (C), to the classical SIR model, and introduce the SICR model and the adjusted-SICR model. We then conduct the steady-state analysis, and obtain the corresponding transcendental equations of the two models, respectively. The transcendental equation, Eq. (16), of the SICR model is similar to that of the SIR model, while the transcendental equation, Eq. (18), of the adjusted-SICR model is quite different, which is a non-integral power equation of S. Theorems 1–4 reveal that there is no threshold in the two models. Regarding the self-resistance force of rumor spreading, we introduce the parameter τ to express such a self-resistance, and illustrate the influences to the final size S_1 and the spreading peak $I_{\max}$ of a rumor under various parameters in simulations.

The refuting–persuading mechanism in the SICR model reflects the self-resistance characteristic of networks to a rumor, and our experiment reveals some interesting patterns of rumor spreading involved with counterattack force. In the future,

we will extend the research to heterogeneous networks, such as the scale-free networks. Further investigations about spreading mechanisms and models in complex networks will open a new door to understanding the secrets of rumor spreading.

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Appendix A. The detailed derivation of steady-state analysis

Considering the rumor spreading mechanism, the mean-field equations of the SICR model can be described as follows:

$$\frac{dS(t)}{dt} = -(\alpha+\beta+\theta)\langle k\rangle S(t)I(t), \tag{A.1}$$

$$\frac{dI(t)}{dt} = \alpha\langle k\rangle S(t)I(t) - \eta\langle k\rangle I(t)C(t) - \gamma\langle k\rangle I(t)(R(t)+I(t)), \tag{A.2}$$

$$\frac{dC(t)}{dt} = \theta\langle k\rangle S(t)I(t), \tag{A.3}$$

$$\frac{dR(t)}{dt} = \beta\langle k\rangle S(t)I(t) + \eta\langle k\rangle I(t)C(t) + \gamma\langle k\rangle I(t)(R(t)+I(t)). \tag{A.4}$$

And the mean-field equations of the adjusted-SICR model are described as follows:

$$\frac{dS(t)}{dt} = -(\alpha+\beta+\theta)\langle k\rangle S(t)I(t),$$

$$\frac{dI(t)}{dt} = \alpha\langle k\rangle S(t)I(t) - \eta\langle k\rangle I(t)C(t) - \gamma\langle k\rangle I(t)R(t), \tag{A.5}$$

$$\frac{dC(t)}{dt} = \theta\langle k\rangle S(t)I(t),$$

$$\frac{dR(t)}{dt} = \beta\langle k\rangle S(t)I(t) + \gamma\langle k\rangle I(t)R(t) + \eta\langle k\rangle I(t)C(t). \tag{A.6}$$

Dividing Eq. (A.2) by (A.4), we have

$$\frac{dS(t)}{dC(t)} = -\frac{\alpha+\beta+\theta}{\theta}.$$

With the initial conditions $S(0) = \frac{N-1}{N} \approx 1 (N \to \infty)$, $C(0) = 0$, we can derive the next relational expression by separation of variable,

$$C(t) = \frac{\theta}{\alpha+\beta+\theta}(1 - S(t)). \tag{A.7}$$

Furthermore, we see that

$$R(t) + I(t) = 1 - S(t) - C(t) = \left(1 - \frac{\theta}{\alpha+\beta+\theta}\right)(1 - S(t)). \tag{A.8}$$

Substituting Eqs. (A.7) and (A.8) into (A.4), it becomes

$$\frac{dR(t)}{dt} = \left(\beta - \gamma - \frac{\eta\theta - \gamma\theta}{\alpha+\beta+\theta}\right)\langle k\rangle S(t)I(t) + \left(\gamma + \frac{\eta\theta - \gamma\theta}{\alpha+\beta+\theta}\right)\langle k\rangle I(t). \tag{A.9}$$

Dividing Eq. (A.2) by (A.9), we obtain

$$\frac{dR(t)}{dS(t)} = \frac{1}{\alpha+\beta+\theta}\left(\gamma - \beta + \frac{\eta\theta - \gamma\theta}{\alpha+\beta+\theta}\right) - \frac{1}{\alpha+\beta+\theta}\left(\gamma + \frac{\eta\theta - \gamma\theta}{\alpha+\beta+\theta}\right)\frac{1}{S(t)}. \tag{A.10}$$

With the initial conditions $S(0) = \frac{N-1}{N} \approx 1 (N \to \infty)$, $R(0) = 0$, solving the differential equations above by the method of separation of variables, we have

$$R(t) = \frac{1}{\alpha+\beta+\theta}\left(\gamma - \beta + \frac{\eta\theta - \gamma\theta}{\alpha+\beta+\theta}\right)(S(t) - 1) - \frac{1}{\alpha+\beta+\theta}\left(\gamma + \frac{\eta\theta - \gamma\theta}{\alpha+\beta+\theta}\right)\ln^{S(t)}. \tag{A.11}$$

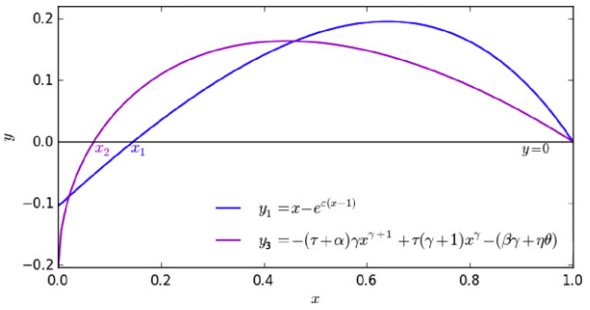

Fig. B.1. The graph of functions y_1 and y_3.

Note that $R = 1 - S - C = (1 - \frac{\theta}{\alpha+\beta+\theta})(1 - S)$; we have

$$S = e^{-\varepsilon(1-S)}, \tag{A.12}$$

$$R + C = 1 - e^{-\varepsilon(R+C)}, \tag{A.13}$$

where

$$\varepsilon = 1 + \frac{(\alpha+\beta+\theta)\alpha}{(\alpha+\beta)\gamma+\theta\eta} \geq 1.$$

Based on Eqs. (A.8) and (A.2), we have

$$I(t) = (\tau+\alpha)(1 - S(t)) + \tau \ln^{S(t)}, \tag{A.14}$$

where $\tau = \theta\eta + (1-\theta)\gamma$. In order to get $I_{\max}$, we take the derivative of $I(t)$ with respect to t,

$$\frac{dI(t)}{dt} = \left(\frac{\tau}{S(t)} - (\tau+\alpha)\right)\frac{dS(t)}{dt}.$$

From Eq. (A.2), it is easy to see that $\frac{dS(t)}{dt} < 0$ in the process of rumor spreading. Let $\frac{dI(t)}{dt} = 0$, we obtain $S(t) = \frac{\tau}{\tau+\alpha}$. It is not hard to see that

$$I_{\max} = \alpha + \tau \ln^{\frac{\tau}{\tau+\alpha}}. \tag{A.15}$$

Appendix B. The proofs of theorems

Theorem 1. For $\varepsilon > 1$, the equation $x = e^{-\varepsilon(1-x)}$ has two solutions, $x = 1$ and a nontrivial solution x_1, where $0 < x_1 < 1 - \frac{\ln\varepsilon}{\varepsilon}$.

Proof. Obviously, $x = 1$ is a solution of $x = e^{-\varepsilon(1-x)}$.

Let $y_1 = x - e^{-\epsilon(1-x)}$, and take the derivative of y_1 with respect to x:

$$y_1'(x) = 1 - \epsilon(e^{-\epsilon(1-x)}).$$

Let $y_1'(x) = 0$, we obtain the unique extreme point $\widehat{x} = 1 - \frac{\ln\epsilon}{\epsilon}$. In addition, $y_1'(0) = 1 > 0$, $y_1'(1) = 1 - \epsilon < 0$, so $\widehat{x}$ is a maximum point.

For $\varepsilon > 1$, it is easy to see that $0 < \frac{\ln\varepsilon}{\varepsilon} \leq \frac{1}{e}$, and

$$0 < 1 - \frac{1}{e} \leq \widehat{x} < 1.$$

For $\varepsilon > 1$, $y_1(\widehat{x}) = 1 - \frac{\ln x+1}{\varepsilon} > 0$, $y_1(0) = -e^{-\varepsilon} < 0$, so the function y_1 is a convex function, and according to the Mean Value Theorem, y_1 has a nontrivial solution x_1, where $0 < x_1 < 1 - \frac{\ln\epsilon}{\epsilon}$. The graph of y_1 is shown in Fig. B.1. □

Theorem 2. Let $\alpha + \beta + \theta = 1$. If the parameters α, θ, τ in the SICR model are nonzero, then we have the following:

(1) For fixed β, γ, η, the final state S_1 decreases as α increases, and increases as θ increases;
(2) For fixed α, β, θ, the final state S_1 increases as τ increases;
(3) For fixed α, β, θ, the final state S_1 increases as γ increases.

Proof. Because the parameters are nonzero, and $\alpha+\beta+\theta=1$, we have $\varepsilon=1+\frac{\alpha}{(1-\theta)\gamma+\theta\eta}=1+\frac{\alpha}{\tau}>1$. As shown in the above, the equation

$$S=\mathrm{e}^{-\varepsilon(1-S)}$$

has two solutions, 1 and a nontrivial solution $0<S_1<1-\frac{\ln\varepsilon}{\varepsilon}$. For fixed parameters except χ, where χ represents one of the parameters α, γ and τ, S_1 and ε are functions of χ. Taking the derivative of S_1 and ε with respect to χ, we have

$$\frac{\mathrm{d}S_1}{\mathrm{d}\chi}=\mathrm{e}^{-\varepsilon(1-S_1)}\left(\frac{\mathrm{d}\varepsilon}{\mathrm{d}\chi}(S_1-1)+\varepsilon\frac{\mathrm{d}S_1}{\mathrm{d}\chi}\right),$$

which implies

$$(1-\varepsilon\mathrm{e}^{-\varepsilon(1-S_1)})\frac{\mathrm{d}S_1}{\mathrm{d}\chi}=\mathrm{e}^{-\varepsilon(1-S_1)}(S_1-1)\frac{\mathrm{d}\varepsilon}{\mathrm{d}\chi}.$$

Obviously, $\mathrm{e}^{-\varepsilon(1-S_1)}>0$. Besides, since $0<S_1<1-\frac{\ln\varepsilon}{\varepsilon}$ and $\varepsilon>1$, it is easy to see that $(1-\varepsilon\mathrm{e}^{-\varepsilon(1-S_1)})>0$. So, if we know the plus–minus sign of $\frac{\mathrm{d}\varepsilon}{\mathrm{d}\chi}$, then the plus–minus sign of $\frac{\mathrm{d}S_1}{\mathrm{d}\chi}$ can be determined, and furthermore, the monotonicity between S_1 and parameters χ can be proved.

(1) Since $\alpha+\beta+\theta=1$, given fixed $\beta,\theta=1-\beta-\alpha$ would decrease as α increases, so we need only to show that S_1 decreases as α increases. In fact, we can see that $\varepsilon=1+\frac{\alpha}{(\alpha+\beta)(\gamma-\eta)+\eta}>1$ and

$$\frac{\mathrm{d}\varepsilon}{\mathrm{d}\alpha}=\frac{\beta\gamma+(1-\beta)\eta}{((\alpha+\beta)(\gamma-\eta)+\eta)^2}>0,$$

so $\frac{\mathrm{d}S_1}{\mathrm{d}\alpha}<0$, that is, S_1 decreases as α increases, and also implies that S_1 increases as θ increases.

(2) Given fixed α,β,θ, we have

$$\frac{\mathrm{d}\varepsilon}{\mathrm{d}\tau}=-\frac{\alpha}{\tau^2}<0,$$

and so $\frac{\mathrm{d}S_1}{\mathrm{d}\tau}>0$, which proves (2).

(3) Given fixed α,β,θ, we have

$$\frac{\mathrm{d}\varepsilon}{\mathrm{d}\gamma}=\frac{\mathrm{d}\varepsilon}{\mathrm{d}\tau}\frac{\mathrm{d}\tau}{\mathrm{d}\gamma}=-\frac{\alpha(1-\theta)}{\tau^2}<0,$$

and so $\frac{\mathrm{d}S_1}{\mathrm{d}\gamma}>0$, which proves (3). □

Theorem 3. Let $\alpha+\beta+\theta=1$. If the parameters α,θ,τ in the SICR model are nonzero, then we have the following:

(1) For fixed β,γ,η, the peak value of the infective density $I_{\max}$ increases as α increases, and decreases as θ increases;
(2) For fixed α,β,θ, the peak value of the infective density $I_{\max}$ decreases as τ increases;
(3) For fixed α,β,θ, the peak value of the infective density $I_{\max}$ decreases as γ increases, and also decreases as η increases.

Proof. (1) Since $\alpha+\beta+\theta=1$, given fixed $\beta,\theta=1-\beta-\alpha$ would decrease as α increases, so we need only to show that $I_{\max}$ increases as α increases. For fixed β,γ,η, note that $\tau=\theta\eta+(1-\theta)\gamma=(1-\alpha-\beta)\eta+(\alpha+\beta)\gamma$ and $\frac{\mathrm{d}\tau}{\mathrm{d}\alpha}=(\gamma-\eta)$, we have

$$\frac{\mathrm{d}I_{\max}}{\mathrm{d}\alpha}=\frac{\alpha}{\tau+\alpha}+\left(\frac{\alpha}{\tau+\alpha}+\ln^{\frac{\tau}{\tau+\alpha}}\right)(\gamma-\eta).$$

Let $y_2(x)=1-x+(1-x+\ln^x)(\gamma-\eta)(0<x<1)$, so $\frac{\mathrm{d}I_{\max}}{\mathrm{d}\alpha}=y_2(\frac{\tau}{\tau+\alpha})$. Obviously, $y_2(1)=0$. Taking the derivative of y_2 with respect to x, we have

$$y_2'(x)=-1+(\gamma-\eta)\left(\frac{1}{x}-1\right)\quad(0<x<1).$$

If $\gamma-\eta\le0$, then $y_2'(x)<0$, $(0<x<1)$, so $y_2(x)>y_2(1)=0$ and $\frac{\mathrm{d}I_{\max}}{\mathrm{d}\alpha}=y_2(\frac{\tau}{\tau+\alpha})>0$.

If $\gamma-\eta<0$, let $y_2'(x)=0$, we get $0<\tilde{x}=\frac{\gamma-\eta}{\gamma-\eta+1}<1$. Since

$$\frac{\tau}{\tau+\alpha}-\tilde{x}=\frac{\tau}{\tau+\alpha}-\frac{\gamma-\eta}{\gamma-\eta+1}=\frac{\beta\gamma+(\alpha+\theta)\eta}{(\tau+\alpha)(\gamma-\eta+1)}>0,$$

and $y_2'(1)=-1<0$, so we have $\tilde{x}<\frac{\tau}{\tau+\alpha}<1$ and $\frac{\mathrm{d}I_{\max}}{\mathrm{d}\alpha}=y_2(\frac{\tau}{\tau+\alpha})>y_2(1)=0$.

Therefore, $\frac{\mathrm{d}I_{\max}}{\mathrm{d}\alpha}>0$, which implies that $I_{\max}$ increases as α increases.

(2) For fixed α, β, θ, we have

$$\frac{dI_{max}}{d\tau} = \frac{\alpha}{\tau+\alpha} + \ln\frac{\tau}{\tau+\alpha}.$$

Note that $0 < \frac{\tau}{\tau+\alpha} < 1$, and function $f(x) = \ln x + 1 - x$, $(0 < x < 1)$ is strictly decreasing, it is easy to see that $\frac{dI_{max}}{d\tau} < 0$, which implies that the peak value of the infective density I_{max} decreases as τ increases.

(3) For fixed α, β, θ, we have

$$\frac{dI_{max}}{d\gamma} = \frac{dI_{max}}{d\tau}\frac{\tau}{d\gamma}, \qquad \frac{dI_{max}}{d\eta} = \frac{dI_{max}}{d\tau}\frac{\tau}{d\eta}.$$

From the proof of (2) above, we see that $\frac{dI_{max}}{d\tau} < 0$, and note that $\frac{\tau}{d\gamma} = (1-\theta) > 0$, $\frac{\tau}{d\eta} = \theta > 0$, so $\frac{dI_{max}}{d\gamma} < 0$ and $\frac{dI_{max}}{d\eta} < 0$, i.e., the peak value of the infective density I_{max} decreases as γ increases, and decreases as η increases. □

Theorem 4. If $x \geq 0$, then the equation

$$-(\tau+\alpha)\gamma x^{\gamma+1} + (\gamma+1)\tau x^{\gamma} = \beta\gamma + \eta\theta$$

has two positive solutions, $x = 1$ and a nontrivial solution x_2, where $0 < x_2 < \frac{\tau}{\tau+\alpha}$.

Proof. Let

$$y_3 = -(\tau+\alpha)\gamma x^{\gamma+1} + (\gamma+1)\tau x^{\gamma} - (\beta\gamma + \eta\theta).$$

Clearly, $y_3(1) = 0$, and $x = 1$ is a positive solution of the equation.

Taking the derivative of y_3 with respect to x, we have

$$y_3'(x) = -(\tau+\alpha)\gamma(\gamma+1)x^{\gamma} + \tau(\gamma+1)\gamma x^{\gamma-1}.$$

Let $y_3'(x) = 0$, we obtain the unique extreme point in the interval $(0, +\infty)$:

$$\widehat{x_2} = \frac{\tau}{\tau+\alpha}.$$

Clearly, $0 < \widehat{x_2} < 1$.

Besides, it is easy to see that $y_3'(x) > 0$ $(0 < x < \widehat{x_2})$, and $y_3'(1) = -\alpha\gamma(\gamma+1) < 0$. So $\widehat{x_2}$ is a maximum point. In addition, $y_3(\widehat{x_2}) > 0$ and $y_3(0) = -(\beta\gamma + \eta\theta) < 0$, according to the Mean Value Theorem, y_3 has a nontrivial solution x_2, where $0 < x_2 < \widehat{x_2}$. The graph of y_3 is shown in Fig. B.1. □

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