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SPIR: The potential spreaders involved SIR model for information diffusion in social networks

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HIGHLIGHTS

- A novel SPIR information diffusion model with the potential spreader set is established. The potential spreader set makes the state-changing mechanism more reasonable and accurate for the diffusion process.
- The proposed model analyses the information diffusion process based on the discrete time and solves the repeated calculation problem of infective nodes existing in traditional SIR-based models.
- The proposed model depicts the information diffusion process with high accuracy under various circumstances.

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ABSTRACT

The Susceptible–Infective–Removed (SIR) model is one of the most widely used models for the information diffusion research in social networks. Many researchers have devoted themselves to improving the classic SIR model in different aspects. However, on the one hand, the equations of these improved models are regarded as continuous functions, while the corresponding simulation experiments use discrete time, leading to the mismatch between numerical solutions got from mathematical method and experimental results obtained by simulating the spreading behaviour of each node. On the other hand, if the equations of these improved models are solved discretely, susceptible nodes will be calculated repeatedly, resulting in a big deviation from the actual value. In order to solve the above problem, this paper proposes a Susceptible–Potential–Infective–Removed (SPIR) model that analyses the diffusion process based on the discrete time according to simulation. Besides, this model also introduces a potential spreader set which solve the problem of repeated calculation effectively. To test the SPIR model, various experiments have been carried out from different angles on both artificial networks and real world networks. The Pearson correlation coefficient between numerical solutions of our SPIR equations and corresponding simulation results is mostly bigger than 0.95, which reveals that the proposed SPIR model is able to depict the information diffusion process with high accuracy.

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1. Introduction

In recent years, with the rapid growth of social network services (SNS) such as Weibo, Twitter and Facebook, social networks have become the most popular and convenient way to acquire and disseminate information in human society. Thus, revealing the mechanisms of information diffusion in social networks and even predicting the diffusion process are

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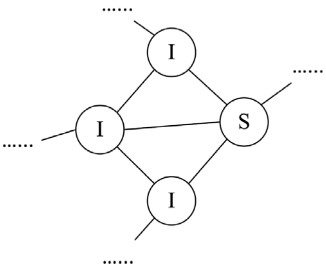

Fig. 1. A specific case where several infective nodes will affect the same susceptible node.

undoubtedly of great significance both theoretically and practically. Many kinds of information diffusion models [1] have been proposed, which mainly include epidemic models [2–5], topological graph based models [6,7], statistical inference based models [8,9], and some other kind of models such as opinion model [10]. Since the pattern of information diffusion in social networks is very similar to the spreading of infectious diseases [11,12] among the real world, epidemic models are more widely used [13–15] compared with others. The typical and popular epidemic model is SIR.

The SIR (Susceptible–Infective–Removed) model divides the nodes in social networks into three categories according to their states: susceptible (S), infective (I), and removed (R) [16]. State S means the node has not known the information yet and is infectible; State I means the node has already known the information and is able to spread it; State R means the node loses interest in the known information, thus does not have the ability to spread it. The infective individuals contact and affect its S-state neighbours (turn them into the I-state) at an average rate β per unit time, and recover and acquire immunity (become the R-state) at an average rate γ per unit time. If we denote the number of nodes for the above three states by $S(t)$, $I(t)$ and $R(t)$, the average degree by $\langle k\rangle$ and the total number of nodes by N, the classic SIR model can be governed by the following coupled non-linear differential equations based on the mean-field theory [17,18]:

$$\frac{dS(t)}{dt} = -\beta\langle k\rangle I(t)S(t)/N, \tag{1}$$

$$\frac{dI(t)}{dt} = \beta\langle k\rangle I(t)S(t)/N - \gamma I(t), \tag{2}$$

$$\frac{dR(t)}{dt} = \gamma I(t). \tag{3}$$

As the classic SIR model is relatively concise, many researchers have proposed various improved models based on the classic one. Their improvements cover various aspects such as presenting new categories of nodes [19–22], improving spreading and immune parameter setting [23–25], or introducing other mechanisms to optimize the traditional ones [26–28]. They all made contributions to improving the classic model and helping people understand the diffusion process better. However, they solve the differential equations of their models as continuous functions while their simulation experiments are performed based on the discrete time. According to research [29], comparison of numerical solutions and simulation results has revealed that there is a big time deviation between two outcomes, which mainly due to the fact that the model will accelerate the diffusion process when calculated in continuous time. In other words, it is inevitable that the time cannot match when equations are solved as continuous functions. Besides, if those equations are solved discretely in accordance with the simulation time, the problem of repeated calculation will be caused (as described below).

If we denote the incremental part of $I(t)$ per unit time as ΔI_+ (corresponding to $\beta\langle k\rangle I(t)S(t)/N$ in Eq. (2)), then $\Delta I_+ = -dS(t)/dt$ will be established in most improved models (referred to as traditional SIR-based models in the following) mentioned above. The problem is that although their ΔI_+ can represent the number of susceptible nodes which each infective node may affect per unit time correctly, it does not take into account the case where more than one infective node may affect the same susceptible node. Consequently, it is likely that one susceptible node will turn into several infective nodes according to their equations. To be precise, $\Delta I_+ = \beta\langle k\rangle I(t)S(t)/N$ can be interpreted as follows: per unit time, each infective node ($I(t)$ in total) will affect its neighbours ($\langle k\rangle$ for average) with average rate β, and the percentage of S-state nodes among those neighbours is $S(t)/N$. Therefore, as shown in Fig. 1, the single S-state node will be calculated three times and may turn into three infective nodes.

The main reason is that, when solved discretely, their ΔI_+ views each infective node as an independent individual and does not consider the actual configuration of the whole network. Consequently, when calculating the total number of all the susceptible nodes which infective nodes have affected in a dense network, the result will far exceed the actual value. As a result, in models where $\Delta I_+ = \beta\langle k\rangle I(t)S(t)/N$ (or similar constructed), the number of infective nodes will grow rapidly and even break the upper limit in the early stage, which is obviously not in agreement with reality. Moreover, even in a relatively sparser network where the number of infective nodes would not grow that fast, the numerical solutions of these

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