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Stochastic SIRS models on networks: mean and variance of infection



Tingting Chen ^a, Guirong Liu ^{a, c, *}, Zhen Jin ^{b, c}

- ^a School of Mathematics and Statistics, Shanxi University, Taiyuan, Shanxi, 030006, China
- ^b Complex Systems Research Center, Shanxi University, Taiyuan, Shanxi, 030006, China
- ^c Key Laboratory of Complex Systems and Data Science of Ministry of Education, Shanxi University, Taiyuan, Shanxi, 030006, China

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ABSTRACT

Due to the heterogeneity of contact structure, it is more reasonable to model on networks for epidemics. Because of the stochastic nature of events and the discrete number of individuals, the spread of epidemics is more appropriately viewed as a Markov chain. Therefore, we establish stochastic SIRS models with vaccination on networks to study the mean and variance of the number of susceptible and infected individuals for large-scale populations. Using van Kampen's system-size expansion, we derive a high-dimensional deterministic system which describes the mean behaviour and a Fokker-Planck equation which characterizes the variance around deterministic trajectories. Utilizing the qualitative analysis technique and Lyapunov function, we demonstrate that the disease-free equilibrium of the deterministic system is globally asymptotically stable if the basic reproduction number $R_0 < 1$; and the endemic equilibrium is globally asymptotically stable if $R_0 > 1$. Through the analysis of the Fokker-Planck equation, we obtain the asymptotic expression for the variance of the number of susceptible and infected individuals around the endemic equilibrium, which can be approximated by the elements of principal diagonal of the solution of the corresponding Lyapunov equation. Here, the solution of Lyapunov equation is expressed by vectorization operator of matrices and Kronecker product, Finally, numerical simulations illustrate that vaccination can reduce infections and increase fluctuations of the number of infected individuals and show that individuals with greater degree are more easily infected.

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1. Introduction

Epidemics are harmful to people's health and cause huge economic losses. Therefore, it is necessary to take some measures to prevent and control epidemics. Epidemic models have been developed to understand the epidemiological dynamics. They provide the theoretical basis for forecasting transmission and assessing the effectiveness of control measures.

For various epidemics, vaccination has become a major intervention strategy. The implementation of vaccination may lead to changes in behavioral indicators and patterns. For instance, a nonlinear SIR model incorporating vaccination policy was analyzed and found that a saddle-node bifurcation for imperfect vaccination can cause the emergence of sustained epidemic

^{*} Corresponding author. School of Mathematics and Statistics, Shanxi University, Taiyuan, Shanxi, 030006, China. *E-mail address:* lgr5791@sxu.edu.cn (G. Liu).

equilibria (Kambali & Abbasi, 2023). Different vaccination strategies can also affect the basic reproduction number, as well as the maximum number of infections and the final size (Anita et al., 2021). So it is crucial to consider the impact of vaccination in epidemic models.

Additionally, the study of epidemics on networks can be seen as a powerful way to represent the complex structure of contacts that can spread disease in a population. Epidemic models on networks based on degree distributions have been a popular choice since they combine the ability to specify the degree distributions with analytical tractability. Their difference in node degrees affects the fundamental nature of the epidemic dynamics, for example, temporal evolution of infected individuals (Wang et al., 2018), as well as the final outbreak size (Cheng et al., 2023; Hébert-Dufresne et al., 2020). Currently, there are some studies to analyze the potential effect of vaccination measures on network models. Morita (2020) considered targeted immunization on networks, where a fraction of individuals with the highest connectivity are immunized. They derived a formula that can yield the immunization threshold by using the type reproduction number. Peng et al. (2013) studied an epidemic model including vaccinated compartments on different networks. The results show that vaccination can linearly reduce the epidemic prevalence in small-world networks and exponentially decrease for scale-free networks. These papers show that the study of infectious diseases with vaccination on networks can yield new discoveries. However, due to the high dimension and complexity of epidemic models on networks, the analysis is not easy such as stability of equilibrium.

Stochasticity is another factor that should be taken into account in models. This means that the system may not be completely predictable to an observer and probabilistic solutions are sought. However, stochastic models can capture the volatility and variability inherent in epidemiological models due to the chance nature of occurrence of events, which is more significant (Arino & Milliken, 2022; Maliyoni et al., 2019; Zhang et al., 2024). In stochastic epidemic models, although the probability of each event occurring within a short time period is derived from the rates in the deterministic model, stochastic models may predict different disease dynamics to the analogous deterministic models. A widely used approach employs Markov chains, which are particularly suitable for modeling epidemic spread and can reveal subtle but important details, such as quasi-stationary distribution, probability of epidemic fade-out and extinction time (Allen, 2010, 2017; Artaejo et al., 2013; Brittona & Traoré, 2017). These random transmission characteristics are generally studied using transition matrices, infinitesimal generating matrices, the generating function and Kolmogorov differential equations (Bharucha-Reid & Morse, 1960). There has also been some literature studying the stochastic epidemic models on networks. Stochastic SIR and SEIR epidemic models on networks with preventive rewiring were used to analyze the expected degree of the infectious nodes (Britton et al., 2016). Using the theory of large fluctuations, the extinction time in heterogeneous networks was studied (Hindes & Schwartz, 2016). By dividing a social contact network into households, the probability of a major outbreak was obtained in a stochastic SIR model with vaccination strategies (Ball & Sirl, 2018). The analysis of stochastic models on networks offers many interesting conclusions. Epidemic models are often analytically intractable due to nonlinearity inherent in the transmission rates. In this sense, the inclusion of networks and randomness makes epidemic models even more complicated.

In addition to the above studies, another important content of stochastic models is the mean and variance. The mean can reveal the long-term trend of the epidemic, and the variance quantifies the uncertainty and volatility of the development of the epidemic. They are the basis for forecasting and decision-making. The mean and variance can be derived from the probability distribution. The underlying probability distribution is typically defined as solutions of a specific master equation for a Markov chain. Nevertheless, explicit solutions are usually unavailable or intractable. It is common to take some other approaches for research. For example, for a continuous-time Markov chain model for Hepatitis C, numerical simulations were used to estimate the mean and variance of the number of susceptible, acute, chronic, isolated and recovered individuals (Imran et al., 2013). In stochastic SIR epidemic models, the algorithm for the recursive computation of the moments of the number of recovered individuals was obtained by using Laplace transform and the generating function (Artalejo et al., 2010). But these methods are time-consuming for large-scale populations. Voinson et al. (2018) used the generating function to calculate the mean and variance of the number of infected individuals. This approach is difficult to use in high-dimensional systems. Using a multi-type branching process approximation, a Markovian SIR epidemic model on configuration networks was explored. The results found closed-form analytic expressions for the mean and variance of the number of infectious individuals as a function of time and the degree of the initially infected individuals. They also confirmed that the mean prevalence of infection depends on the first two moments of the degree distribution and the variance in prevalence depends on the first three moments of the degree distribution (Ball & House, 2017). Notably, branching process approximation is suitable for capturing dynamics during the early stages of the disease. These methods mentioned above are not suitable for studying the mean and variance of the number of individuals in network-based epidemic models for large-scale populations.

Van Kampen's system-size expansion is often used to construct a continuous approximation for stochastic models of discrete states. This expansion provides an approximate method of converting the master equation into the more tractable Fokker-Planck equation, thereby simplifying the mathematical analysis. It also serves as a bridge between microscopic and macroscopic descriptions of systems. Utilizing this expansion, the time evolution of each class can be decomposed into deterministic and stochastic components. The deterministic components describe the mean behaviour and stochastic components describe the variance around deterministic trajectories. It not only demonstrates how a deterministic equation emerges from the stochastic description, but also provides a methodology for analyzing variance around deterministic attractors (Black & McKane, 2010; Fatehi et al., 2020; Kampen, 2010; Simões et al., 2008; Tao et al., 2005; Wang, Qian, & Qian, 2012; Wang et al., 2012). It is worth noting that this expansion primarily relies on the assumption of large scale populations.

Consequently, this method can only be used to study the mean and variance of the number of individuals in large scale populations.

Inspired by the above mentioned factors, we establish a continuous-time Markov chain SIRS model with vaccination measures on networks, and use van Kampen's system-size expansion method to investigate mean and asymptotic expressions for the variance of the number of susceptible and infected individuals for large-scale population around the endemic equilibrium. The outline of the paper is as follows. In Section 2, we describe the process of disease transmission on networks, which can be seen as a multi-dimensional Markov chain. Then we write the master equation that characterizes the dynamics of probability distribution. In Section 3, we use van Kampen's system-size expansion to analyze the master equation. In this way, we derive a high-dimensional deterministic system which describes the mean behaviour of system and a Fokker-Planck equation which characterizes the variance. Section 4 analyzes the basic reproduction number and stability of equilibrium of the deterministic system. In Section 5, the Fokker-Planck equation is studied. We get asymptotic expressions for variance of the number of susceptible and infected individuals around the endemic equilibrium. In Section 6, numerical simulations are carried out to illustrate the reasonableness of our results and effect of vaccines and networks on means and variances. Finally, a summary is presented in Section 7.

2. Model

In a population of large size N, without considering birth and death, individuals serve as nodes, and the contacts between individuals serve as edges. The degree of a node is the number of edges associated with this node. Denote K as the maximum degree in the network, and the group k is the set of all nodes with degree k. N_k denotes the total number of the nodes in the kth group and $N = \sum_{k=1}^K N_k$. Let $D_k = N_k/N$, k = 1, 2, ..., K. Thus $\sum_{k=1}^K D_k = 1$. Each node is in one of three states: susceptible (S), infected (I) or recovered (R). $S_k(t)$, $I_k(t)$ and $R_k(t)$ are the number of susceptible nodes, infected nodes and recovered nodes in the kth group at time t, where $S_k(t)$, $I_k(t)$, $R_k(t) \ge 0$, $S_k(t) + I_k(t) + R_k(t) = N_k$. Compared with the relatively long time period of being susceptible (S), infected (I) and recovered (R), the act of disease transmission is often very short, and can be considered as instantaneous. A susceptible node may be infected if it has an edge connected with an infected node. Infection is the Poisson process. Let λ be the rate of infection through an edge. Then, a susceptible node in the kth group is successfully infected at a rate $\lambda = \lambda k \left(\sum_{m=1}^K m I_m(t)\right) / \left(\sum_{m=1}^K m N_m\right)$.

Additionally, the infectious periods of all infected nodes follow an exponential distribution with a mean value of 1. Once the infectious period ends, infected nodes acquire immunity and become recovery nodes. But disease-acquired immunity is not permanent. Susceptible nodes are vaccinated at the rate α . Then susceptible nodes who have been vaccinated acquire temporary immunity and become recovery nodes. A recovered node can again become susceptible at a rate σ due to loss of disease-acquired immunity or waning of vaccine protection.

Based on these assumptions, a continuous time 2K dimensional Markov chain

$$\{X(t) = ((S_1(t), I_1(t)), (S_2(t), I_2(t)), ..., (S_K(t), I_K(t))), t > 0\}$$

is defined on probability space $(\Gamma, \mathcal{F}, \mathbb{P})$, where Γ, \mathcal{F} and \mathbb{P} denote the sample spaces, the event space and probability measure, respectively. Let $\Omega_k = \{(a_1, a_2) | 0 \le a_1 \le N_k, \ 0 \le a_2 \le N_k - a_1\}$, then its state space is $\Omega = \Omega_1 \times \Omega_2 \times \cdots \times \Omega_K$. Set

$$\begin{split} & \mathbf{P}(\overrightarrow{\rho}\;;t) = \mathsf{Prob}\{X(t) = \overrightarrow{\rho} = (\overrightarrow{\rho_1},...\overrightarrow{\rho_k},...\overrightarrow{\rho_K})\}, \\ & \overrightarrow{e_k} = ((0,0),(0,0),...,(0,+1),...,(0,0)), \\ & \overrightarrow{v_k} = ((0,0),(0,0),...,(+1,0),...,(0,0)), \\ & \overrightarrow{\delta_k} = ((0,0),(0,0),...,(-1,+1),...,(0,0)), \\ & \overrightarrow{\delta_k} = ((0,0),(0,0),...,(-1,+1),...,(0,0)), \end{split}$$

Table 1 The transitions and corresponding probabilities for the Markov chain in a time interval Δt .

| Transitions | Probabilities |
|--|--|
| $\overrightarrow{ ho} ightarrow \overrightarrow{ ho} - \overrightarrow{e_k}$ | $J_{1-}^{k}(\overrightarrow{\rho}) = \rho_{k2}\Delta t + o(\Delta t)$ |
| $\overrightarrow{ ho} ightarrow \overrightarrow{ ho} + \overrightarrow{ u_k}$ | $J_{2+}^{k}(\overrightarrow{\rho}) = \sigma(N_k - \rho_{k1} - \rho_{k2})\Delta t + o(\Delta t)$ |
| $\overrightarrow{ ho} ightarrow \overrightarrow{ ho} - \overrightarrow{ u_k}$ | $J_{2-}^{k}(\overrightarrow{\rho}) = \alpha \rho_{k1} \Delta t + o(\Delta t)$ |
| $\overrightarrow{ ho} ightarrow \overrightarrow{ ho} + \overrightarrow{\delta_k}$ | $J_{3+}^{k}(\overrightarrow{\rho}) = \frac{\lambda k \rho_{k1} \sum_{m=1}^{K} m \rho_{m2}}{\sum_{m=1}^{K} m N_{m}} \Delta t + o(\Delta t)$ |

where $\overrightarrow{\rho_k} = (\rho_{k1}, \rho_{k2}) \in \Omega_k$ and $\overrightarrow{e_k}$, $\overrightarrow{v_k}$, $\overrightarrow{\delta_k}$ are 2K-dimensional vectors for k = 1, 2, ..., K. The transitions and corresponding probabilities of X(t) in a small time interval Δt are given in Table 1. There are 4K events that can occur.

According to Table 1, we can derive the following master equation to describe the temporal evolution of the probability distribution

$$\frac{\mathrm{d}P(\overrightarrow{\rho};t)}{\mathrm{d}t} = \sum_{k=1}^{K} J_{2+}^{k} (\overrightarrow{\rho} - \overrightarrow{v_{k}}) P(\overrightarrow{\rho} - \overrightarrow{v_{k}};t) + \sum_{k=1}^{K} J_{2-}^{k} (\overrightarrow{\rho} + \overrightarrow{v_{k}}) P(\overrightarrow{\rho} + \overrightarrow{v_{k}};t)
+ \sum_{k=1}^{K} J_{1-}^{k} (\overrightarrow{\rho} + \overrightarrow{e_{k}}) P(\overrightarrow{\rho} + \overrightarrow{e_{k}};t) + \sum_{k=1}^{K} J_{3+}^{k} (\overrightarrow{\rho} - \overrightarrow{\delta_{k}}) P(\overrightarrow{\rho} - \overrightarrow{\delta_{k}};t)
- \sum_{k=1}^{K} J_{2-}^{k} (\overrightarrow{\rho}) P(\overrightarrow{\rho};t) - \sum_{k=1}^{K} J_{2+}^{k} (\overrightarrow{\rho}) P(\overrightarrow{\rho};t) - \sum_{k=1}^{K} J_{1-}^{k} (\overrightarrow{\rho}) P(\overrightarrow{\rho};t) - \sum_{k=1}^{K} J_{3+}^{k} (\overrightarrow{\rho}) P(\overrightarrow{\rho};t).$$
(1)

Here, if $\overrightarrow{\rho} - \overrightarrow{v_k} \not\in \Omega$, then $J_{2+}^k(\overrightarrow{\rho} - \overrightarrow{v_k}) = 0$; if $\overrightarrow{\rho} + \overrightarrow{v_k} \not\in \Omega$, then $J_{2-}^k(\overrightarrow{\rho} + \overrightarrow{v_k}) = 0$; if $\overrightarrow{\rho} + \overrightarrow{e_k} \not\in \Omega$, then $J_{1-}^k(\overrightarrow{\rho} + \overrightarrow{e_k}) = 0$; if $\overrightarrow{\rho} - \overrightarrow{v_k} \not\in \Omega$, then $J_{3+}^k(\overrightarrow{\rho} - \overrightarrow{\delta_k}) = 0$.

Note that any state that satisfies $\sum_{m=1}^{K} I_m(t) = 0$ is absorbing.

3. Van Kampen's system-size expansion

Introducing the step operators

$$\begin{split} E_{S}^{k\pm}(f(\overrightarrow{\rho})) &= f(\overrightarrow{\rho} \pm \overrightarrow{\nu_{k}}), \\ E_{I}^{k\pm}(f(\overrightarrow{\rho})) &= f(\overrightarrow{\rho} \pm \overrightarrow{e_{k}}), \\ \mathbb{1}(f(\overrightarrow{\rho})) &= f(\overrightarrow{\rho}). \end{split}$$

Then Eq. (1) can be rewritten as

$$\frac{\mathrm{d}P(\overrightarrow{\rho};t)}{\mathrm{d}t} = \sum_{k=1}^{K} E_{S}^{k-} \left(J_{2+}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) \right) + \sum_{k=1}^{K} E_{S}^{k+} \left(J_{2-}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) \right) + \sum_{k=1}^{K} E_{I}^{k+} \left(J_{1-}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) \right) \\
+ \sum_{k=1}^{K} E_{S}^{k+} E_{I}^{k-} \left(J_{3+}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) \right) - \sum_{k=1}^{K} J_{2-}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) - \sum_{k=1}^{K} J_{2+}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) - \sum_{k=1}^{K} J_{1-}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) \\
- \sum_{k=1}^{K} J_{3+}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) = \sum_{k=1}^{K} \left(E_{S}^{k-} - 1 \right) \left(J_{2+}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) \right) + \sum_{k=1}^{K} \left(E_{S}^{k+} - 1 \right) \left(J_{2-}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) \right) \\
+ \sum_{k=1}^{K} \left(E_{I}^{k+} - 1 \right) \left(J_{1-}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) \right) + \sum_{k=1}^{K} \left(E_{S}^{k+} E_{I}^{k-} - 1 \right) \left(J_{3+}^{k}(\overrightarrow{\rho}) P(\overrightarrow{\rho};t) \right). \tag{2}$$

To analyze the mean and variance of the number of susceptible and infected individuals, we introduce the following variables. For k = 1, 2, ..., K, denote

$$\begin{split} y_k(t) &= \mathrm{E}\left(\frac{S_k(t)}{N}\right), p_k(t) = \mathrm{E}\left(\frac{I_k(t)}{N}\right), \\ \varphi_k(t) &= \frac{S_k(t) - Ny_k(t)}{\sqrt{N}}, \omega_k(t) = \frac{I_k(t) - Np_k(t)}{\sqrt{N}}, \end{split}$$

where $E(\cdot)$ denotes the expectation of random variable. Hence,

$$S_k(t) = Ny_k(t) + N^{\frac{1}{2}}\varphi_k(t),$$

$$I_k(t) = Np_k(t) + N^{\frac{1}{2}}\omega_k(t).$$

Further,

$$\begin{array}{ll} \mathsf{P}(\overrightarrow{\rho}\;;t) &= \mathsf{Prob}\; \{X(t) = ((\rho_{11},\rho_{12}),...,(\rho_{K1},\rho_{K2})\,)\,\} \\ &= \mathsf{Prob}\; \{S_1(t) = \rho_{11},I_1(t) = \rho_{12},...,S_K(t) = \rho_{K1},I_K(t) = \rho_{K2}\} \\ &= \mathsf{Prob}\; \{\varphi_1(t) = \xi_1(t),...,\varphi_K(t) = \xi_K(t),\omega_1(t) = \eta_1(t),...,\omega_K(t) = \eta_K(t)\} \\ &= \Pi\left(\overrightarrow{\xi},\overrightarrow{\eta},t\right), \end{array}$$

where $\overrightarrow{\xi} = (\xi_1, \xi_2, ..., \xi_K)$, $\overrightarrow{\eta} = (\eta_1, \eta_2, ..., \eta_K)$, $\xi_k(t) = N^{-1/2}(\rho_{k1} - Ny_k(t))$, $\eta_k(t) = N^{-1/2}(\rho_{k2} - Np_k(t))$, k = 1, 2, ..., K. The step operators $E_S^{k\pm}$ and $E_I^{k\pm}$ in the master equation (2) can be expressed as

$$E_{S}^{k\pm} = \mathbb{1}\pm N^{-\frac{1}{2}}\frac{\partial}{\partial \xi_{k}} + \frac{1}{2}N^{-1}\frac{\partial^{2}}{\partial \xi_{k}^{2}} + \dots,$$

$$E_{I}^{k\pm} = \mathbb{1}\pm N^{-\frac{1}{2}}\frac{\partial}{\partial \eta_{k}} + \frac{1}{2}N^{-1}\frac{\partial^{2}}{\partial \eta_{k}^{2}} + \dots$$

Further,

$$\frac{\mathrm{d}\xi_k}{\mathrm{d}t} = -N^{\frac{1}{2}}\frac{\mathrm{d}y_k}{\mathrm{d}t}, \quad \frac{\mathrm{d}\eta_k}{\mathrm{d}t} = -N^{\frac{1}{2}}\frac{\mathrm{d}p_k}{\mathrm{d}t}$$

Then Eq. (2) can be expressed as

$$\frac{\partial \Pi}{\partial t} - N^{\frac{1}{2}} \sum_{k=1}^{K} \frac{\partial \Pi}{\partial \xi_{k}} \frac{dy_{k}}{dt} - N^{\frac{1}{2}} \sum_{k=1}^{K} \frac{\partial \Pi}{\partial \eta_{k}} \frac{dp_{k}}{dt} \\
= \sum_{k=1}^{K} \left(-N^{-\frac{1}{2}} \frac{\partial}{\partial \xi_{k}} + \frac{1}{2} N^{-1} \frac{\partial^{2}}{\partial \xi_{k}^{2}} \right) \left[\sigma \left(ND_{k} - Ny_{k} - N^{\frac{1}{2}} \xi_{k} - Np_{k} - N^{\frac{1}{2}} \eta_{k} \right) \Pi \right] \\
+ \sum_{k=1}^{K} \left(N^{-\frac{1}{2}} \frac{\partial}{\partial \xi_{k}} + \frac{1}{2} N^{-1} \frac{\partial^{2}}{\partial \xi_{k}^{2}} \right) \left[\alpha \left(Ny_{k} + N^{\frac{1}{2}} \xi_{k} \right) \Pi \right] + \sum_{k=1}^{K} \left(N^{-\frac{1}{2}} \frac{\partial}{\partial \eta_{k}} + \frac{1}{2} N^{-1} \frac{\partial^{2}}{\partial \eta_{k}^{2}} \right) \left[\left(Np_{k} + N^{\frac{1}{2}} \eta_{k} \right) \Pi \right] \\
+ \sum_{k=1}^{K} \left(N^{-\frac{1}{2}} \frac{\partial}{\partial \xi_{k}} + \frac{1}{2} N^{-1} \frac{\partial^{2}}{\partial \xi_{k}^{2}} - N^{-\frac{1}{2}} \frac{\partial}{\partial \eta_{k}} + \frac{1}{2} N^{-1} \frac{\partial^{2}}{\partial \eta_{k}^{2}} - N^{-1} \frac{\partial^{2}}{\partial \xi_{k} \partial \eta_{k}} \right) \left[\frac{\lambda k \left(Ny_{k} + N^{\frac{1}{2}} \xi_{k} \right) \sum_{m=1}^{K} m \left(p_{m} + N^{-\frac{1}{2}} \eta_{m} \right)}{\sum_{m=1}^{K} m D_{m}} \Pi \right]. \tag{3}$$

Let $\langle k \rangle = \sum_{m=1}^{K} m D_m$. Collecting terms of order $N^{1/2}$ at the left and right sides of Eq. (3), we get

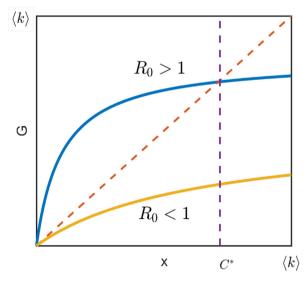


Fig. 1. The functions G with two different values of R_0 .

$$\begin{cases}
\frac{\mathrm{d}y_k}{\mathrm{d}t} = \sigma(D_k - y_k - p_k) - \alpha y_k - \frac{\lambda k y_k \sum_{m=1}^K m p_m}{\langle k \rangle}, \\
\frac{\mathrm{d}p_k}{\mathrm{d}t} = -p_k + \frac{\lambda k y_k \sum_{m=1}^K m p_m}{\langle k \rangle},
\end{cases} (4)$$

where k = 1, 2, ..., K. Similarly, collecting terms of order N^0 at the left and right sides of Eq. (3), we get

$$\begin{split} \frac{\partial \Pi\left(\overrightarrow{\xi},\overrightarrow{\eta},t\right)}{\partial t} &= \sum_{k=1}^{K} \left\{ \sigma \frac{\partial}{\partial \xi_{k}} (\xi_{k}\Pi) + \sigma \frac{\partial}{\partial \xi_{k}} (\eta_{k}\Pi) + \alpha \frac{\partial}{\partial \xi_{k}} (\xi_{k}\Pi) + \frac{\lambda k y_{k}}{\langle k \rangle} \frac{\partial}{\partial \xi_{k}} \left(\sum_{m=1}^{K} m \eta_{m} \Pi \right) \right. \\ &+ \frac{\lambda k \sum_{m=1}^{K} m p_{m}}{\langle k \rangle} \frac{\partial}{\partial \xi_{k}} (\xi_{k}\Pi) + \frac{\partial}{\partial \eta_{k}} (\eta_{k}\Pi) - \frac{\lambda k y_{k}}{\langle k \rangle} \frac{\partial}{\partial \eta_{k}} \left(\sum_{m=1}^{K} m \eta_{m} \Pi \right) - \frac{\lambda k \sum_{m=1}^{K} m p_{m}}{\langle k \rangle} \frac{\partial}{\partial \eta_{k}} (\xi_{k}\Pi) \right\} \\ &+ \frac{1}{2} \sum_{k=1}^{K} \left\{ \left[\sigma(D_{k} - y_{k} - p_{k}) + \alpha y_{k} + \frac{\lambda k y_{k} \sum_{m=1}^{K} m p_{m}}{\langle k \rangle} \right] \frac{\partial^{2}}{\partial \xi_{k}^{2}} \Pi \right. \\ &+ \left. \left(p_{k} + \frac{\lambda k y_{k} \sum_{m=1}^{K} m p_{m}}{\langle k \rangle} \right) \frac{\partial^{2}}{\partial \eta_{k}^{2}} \Pi - \frac{2\lambda k y_{k} \sum_{m=1}^{K} m p_{m}}{\langle k \rangle} \frac{\partial^{2}}{\partial \xi_{k}^{2}} \frac{\partial}{\partial \xi_{k}^{2}} \Pi \right\}. \end{split}$$

4. Mean

In fact, $E(S_k(t)) = Ny_k(t)$ and $E(I_k(t)) = Np_k(t)$. Hence, we just need to analyze $y_k(t)$ and $p_k(t)$ for the study of the mean behavior of $S_k(t)$ and $I_k(t)$.

4.1. Basic reproduction number

Let $\overrightarrow{y}=(y_1,y_2,...,y_K)$, $\overrightarrow{p}=(p_1,p_2,...,p_K)$. It is easy to see that $\Lambda=\left\{\left(\overrightarrow{y},\overrightarrow{p}\right)\left|y_i\geq 0,p_i\geq 0,y_i+p_i\leq D_i,i=1,2,...,K\right.\right\}$ is positively invariant for system (4).

To obtain the equilibrium state of system (4), consider the following algebraic equations

$$\begin{cases} y_k = \frac{\sigma(D_k - p_k)}{(\alpha + \sigma) + \frac{\lambda k \sum_{m=1}^K m p_m}{\langle k \rangle}}, \\ p_k = \frac{\sigma D_k \lambda k \sum_{m=1}^K m p_m}{(\alpha + \sigma) \langle k \rangle + \lambda k \sum_{m=1}^K m p_m + \sigma \lambda k \sum_{m=1}^K m p_m}, \end{cases}$$

where k = 1, 2, ..., K. Let $C = \sum_{m=1}^{K} m p_m$. Then

$$p_k = \frac{\sigma D_k \lambda k C}{(\alpha + \sigma) \langle k \rangle + \lambda k C + \sigma \lambda k C}.$$

Let

$$G(x) = \sum_{k=1}^{K} \frac{\sigma D_k \lambda k^2 x}{(\alpha + \sigma)\langle k \rangle + \lambda k x + \sigma \lambda k x},$$

where $x \in [0, \langle k \rangle]$. Next we need to find solutions that satisfies G(x) = x. Clearly, G(0) = 0, G'(x) > 0, G''(x) < 0 and

$$G(\langle k \rangle) = \sum_{k=1}^{K} k D_k \frac{\sigma \lambda k \langle k \rangle}{(\alpha + \sigma) \langle k \rangle + \lambda k \langle k \rangle + \sigma \lambda k \langle k \rangle} < \sum_{k=1}^{K} k D_k = \langle k \rangle.$$

In conclusion, we see that function G is monotonically increasing and concave in the region $[0, \langle k \rangle]$ and

$$G'(0) = \sum_{k=1}^{K} \frac{\sigma D_k \lambda k^2 (\alpha + \sigma) \langle k \rangle}{[(\alpha + \sigma) \langle k \rangle]^2} = \frac{\lambda \sigma \sum_{k=1}^{K} k^2 D_k}{(\alpha + \sigma) \langle k \rangle} = \frac{\lambda \sigma \langle k^2 \rangle}{(\alpha + \sigma) \langle k \rangle},$$

where $\sum_{k=1}^{K} k^2 D_k = \langle k^2 \rangle$. Let

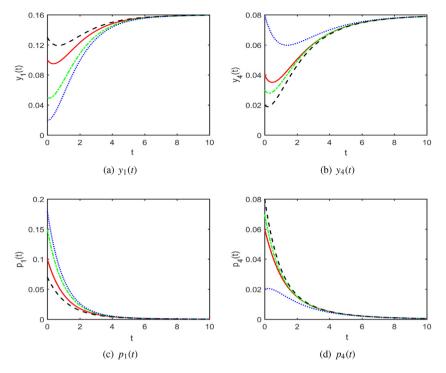
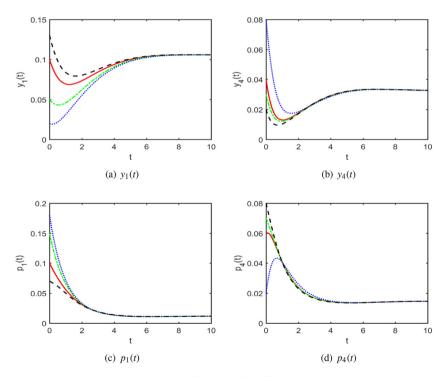


Fig. 2. The solutions of system (4) for different initial values.



 $\textbf{Fig. 3.} \ \ \textbf{The solutions of system (4) for different initial values}.$

$$R_0 = \frac{\lambda \sigma \langle k^2 \rangle}{(\alpha + \sigma) \langle k \rangle}.$$
 (6)

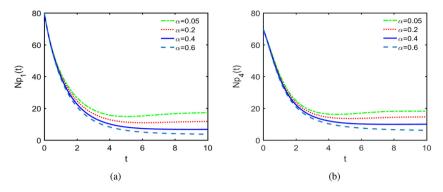


Fig. 4. (a) $Np_1(t)$ for different α when $\lambda=0.8$ and $\sigma=0.4$. (b) $Np_4(t)$ for different α when $\lambda=0.8$ and $\sigma=0.4$.

If $R_0 < 1$, G(x) = x has a unique solution 0 on $[0, \langle k \rangle]$. Otherwise, there exists $x_1 \neq 0$ satisfying $G(x_1) = x_1$ on $(0, \langle k \rangle]$. Hence, there exists $\epsilon \in (0, x_1)$ such that $G'(\epsilon) = 1$. Note that $G'(0) = R_0 < 1$ and G''(x) < 0. Hence, $G'(\epsilon) < 1$. This is a contradiction. Further, if $R_0 < 1$, system (4) exists a unique equilibrium

$$E_0 = \left(\frac{\sigma D_1}{\alpha + \sigma}, ..., \frac{\sigma D_K}{\alpha + \sigma}, 0, ..., 0\right).$$

In the following, we shall prove that G(x) = x has two solutions 0 and $C^* > 0$ on $[0, \langle k \rangle]$ if $R_0 > 1$. First, it is shown that G(x) = x has a solution C^* on $(0, \langle k \rangle)$. Let h(x) = G(x) - x. Note that h(0) = 0, h'(0) = G'(0) - 1 > 0 and h(x) is continuous. There exists $0 < \epsilon < \langle k \rangle$ such that $h(\epsilon) > 0$. Since $h(\langle k \rangle) = G(\langle k \rangle) - \langle k \rangle < 0$, there exists $C^* \in (\epsilon, \langle k \rangle)$ such that $h(C^*) = 0$. Hence, $G(C^*) = C^*$. Next, it is shown that C^* is the unique solution on $(0, \langle k \rangle)$. Suppose there exists another solution $x_2 \in (0, \langle k \rangle)$. Note that $h(C^*) = 0$, $h(x_2) = 0$ and h(0) = 0. There exist u_1 and u_2 such that $0 < u_1 < x_2 < u_2 < C^* < \langle k \rangle$ if $x_2 \in (0, C^*)$ or $0 < u_1 < C^* < u_2 < x_2$ if $x_2 \in (C^*, \langle k \rangle)$ such that $h'(u_1) = h'(u_2) = 0$. Then $G'(u_1) = 1 = G'(u_2)$. This is a contradiction because G'(x) is strictly decreasing on $(0, \langle k \rangle)$. Thus, G(x) = x has two solutions 0 and $C^* > 0$ on $[0, \langle k \rangle]$ if $R_0 > 1$. Furthermore, if $R_0 > 1$, system (4) exists a disease-free equilibrium E_0 and an endemic equilibrium $E_1 = (y_1^*, \dots, y_K^*, p_1^*, \dots, p_K^*)$ in Λ , where

$$\begin{cases} y_k^* = \frac{\sigma D_k \langle k \rangle}{(\alpha + \sigma) \langle k \rangle + \lambda k C^* + \sigma \lambda k C^*}, \\ p_k^* = \frac{\sigma D_k \lambda k C^*}{(\alpha + \sigma) \langle k \rangle + \lambda k C^* + \sigma \lambda k C^*}. \end{cases}$$
(7)

Two scenarios are shown in Fig. 1.

Here, R_0 is the basic reproduction number of system (4). According to Eq. (6), it is easy to see that vaccination can lower the value of R_0 .

4.2. Stability analysis of equilibrium

Theorem 1. If $R_0 < 1$, then the disease-free equilibrium E_0 of system (4) is globally asymptotically stable in Λ ; if $R_0 > 1$, E_0 is unstable.

Proof. The coefficient matrix of the linearized system of system (4) at its equilibrium $(y_1^{**}, ..., y_K^{**}, p_1^{**}, ..., p_K^{**})$ is

$$A = \begin{bmatrix} a_{11} & 0 & \cdots & 0 & b_{11} & b_{12} & \cdots & b_{1K} \\ 0 & a_{22} & \cdots & 0 & b_{21} & b_{22} & \cdots & b_{2K} \\ \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & a_{KK} & b_{K1} & b_{K2} & \cdots & b_{KK} \\ c_{11} & 0 & \cdots & 0 & d_{11} & d_{12} & \cdots & d_{1K} \\ 0 & c_{22} & \cdots & 0 & d_{21} & d_{22} & \cdots & d_{2K} \\ \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & c_{KK} & d_{K1} & d_{K2} & \cdots & d_{KK} \end{bmatrix},$$

where

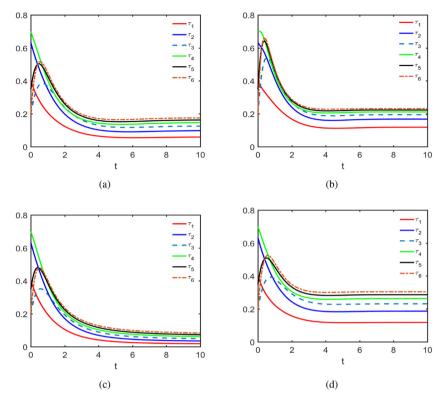


Fig. 5. τ_k as functions of t for different degree k. (a) The parameters are chosen as $\lambda = 0.8$, $\alpha = 0.2$ and $\sigma = 0.4$. (b) The parameters are chosen as $\lambda = 1.5$, $\alpha = 0.2$ and $\sigma = 0.4$. (c) The parameters are chosen as $\lambda = 0.8$, $\alpha = 0.6$ and $\sigma = 0.4$. (d) The parameters are chosen as $\lambda = 0.8$, $\alpha = 0.2$ and $\sigma = 0.8$.

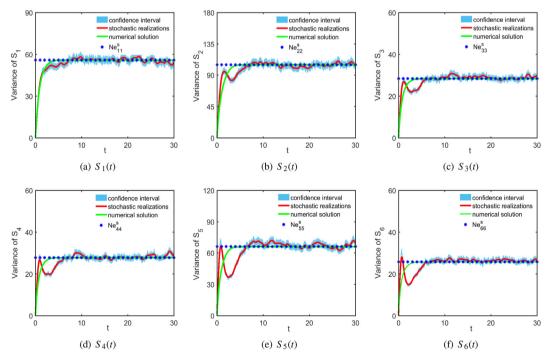


Fig. 6. Time evolution of variance of the number of susceptible nodes.

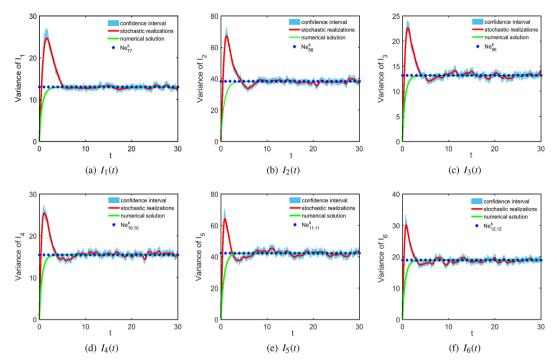


Fig. 7. ime evolution of variance of the number of infected nodes.

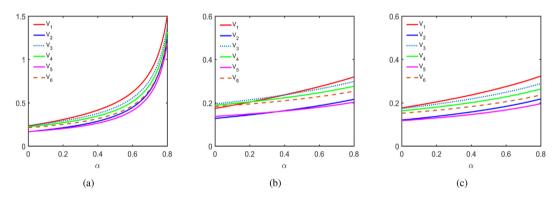


Fig. 8. V_k as a function of vaccination rate α . (a) The parameters are chosen as $\lambda = 0.8$ and $\sigma = 0.4$. (b) The parameters are chosen as $\lambda = 1.5$ and $\sigma = 0.4$. (c) The parameters are chosen as $\lambda = 0.8$ and $\sigma = 0.8$.

$$\begin{split} a_{ii} &= -(\alpha + \sigma) - \frac{i\lambda \sum_{k=1}^{K} k p_k^{**}}{\langle k \rangle}, \ b_{ii} = -\sigma - \frac{i^2 \lambda y_i^{**}}{\langle k \rangle}, \\ c_{ii} &= \frac{i\lambda \sum_{k=1}^{K} k p_k^{**}}{\langle k \rangle}, \ d_{ii} = -1 + \frac{i^2 \lambda y_i^{**}}{\langle k \rangle}, \\ b_{ij} &= -\frac{ij\lambda y_i^{**}}{\langle k \rangle}, \ d_{ij} = \frac{ij\lambda y_i^{**}}{\langle k \rangle}, \end{split}$$

for $i \neq j$, i, j = 1, 2, ..., K.

When $(y_1^{**}, ..., y_K^{**}, p_1^{**}, ..., p_K^{**}) = E_0$, all the eigenvalues of matrix A are $\mu_1 = ... = \mu_K = -(\alpha + \sigma)$, $\mu_{k+1} = ... = \mu_{2K-1} = -1$, $\mu_{2K} = \lambda \sigma \langle k^2 \rangle / ((\alpha + \sigma) \langle k \rangle) - 1$. From $R_0 < 1$, $\mu_{2K} < 0$. Hence, the disease-free equilibrium E_0 of system (4) is locally asymptotically stable. If $R_0 > 1$, we have $\mu_{2K} > 0$. Then E_0 is unstable.

Next we just need to prove the global attractiveness of E_0 . From system (4), we have

$$\frac{\mathrm{d}y_k}{\mathrm{d}t} \leq \sigma D_k - (\sigma + \alpha)y_k.$$

Further, for $t \ge 0, k \in \{1, 2, ..., K\}$,

$$y_k(t) \le y_k(0)e^{-(\sigma+\alpha)t} + \frac{\sigma D_k}{\sigma+\alpha} \Big[1 - e^{-(\sigma+\alpha)t}\Big],$$

which yields

$$\limsup_{t \to \infty} y_k(t) \le \frac{\sigma D_k}{\sigma + \alpha}.$$
 (8)

It follows from $R_0 < 1$ that exists $\varepsilon_1 > 0$ such as

$$\theta_1 := -1 + \frac{\lambda \sigma \langle k^2 \rangle}{(\sigma + \alpha) \langle k \rangle} + \frac{\lambda}{\langle k \rangle} \left(\sum_{k=1}^K k^2 \right) \epsilon_1 < 0.$$

From (8), there exists $T_1 > 0$ such that

$$y_k(t) \le \frac{\sigma D_k}{\sigma + \alpha} + \epsilon_1, \ t \ge T_1, \ k = 1, 2, ..., K,$$

which together with system (4), implies

$$\begin{split} \frac{\mathrm{d}\left(\sum_{k=1}^{K}kp_{k}\right)}{\mathrm{d}t} &= -\sum_{k=1}^{K}kp_{k} + \sum_{m=1}^{K}mp_{m} \cdot \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{K}k^{2}y_{k} \\ &\leq \left(\sum_{k=1}^{K}kp_{k}\right) \left[-1 + \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{K}k^{2}\left(\frac{\sigma D_{k}}{\sigma + \alpha} + \epsilon_{1}\right)\right] \\ &= \theta_{1}\left(\sum_{k=1}^{K}kp_{k}\right). \end{split}$$

Hence, for $t \geq T_1$,

$$\sum_{k=1}^{K} k p_k(t) \le \sum_{k=1}^{K} k p_k(T_1) e^{\theta_1(t-T_1)}.$$

Further,

$$\lim_{t\to\infty}\sum_{k=1}^K kp_k(t)=0.$$

That is.

$$\lim_{t\to\infty}p_k(t)=0,\tag{9}$$

where k = 1, 2, ..., K. Hence, for any $\varepsilon > 0$, there exists T > 0 such that $0 \le p_k(t) < \varepsilon$ and $\sum_{k=1}^K k p_k(t) < \epsilon$ when t > T and k = 1, 2, ..., K. From system (4), we have

$$\begin{split} \frac{\mathrm{d}y_k}{\mathrm{d}t} &= \sigma(D_k - y_k - p_k) - \alpha y_k - \frac{\lambda k y_k \sum_{m=1}^K m p_m}{\langle k \rangle} \\ &> \sigma D_k - (\sigma + \alpha) y_k - \epsilon \sigma - \frac{\lambda k \epsilon y_k}{\langle k \rangle} \\ &= \sigma(D_k - \epsilon) - \left(\sigma + \alpha + \frac{\lambda k \epsilon}{\langle k \rangle}\right) y_k. \end{split}$$

This yields

$$y_k(t) > y_k(T) e^{\left(\sigma + \alpha + \frac{\lambda k \epsilon}{\langle k \rangle}\right)(T - t)} + \frac{\sigma(D_k - \epsilon)}{\sigma + \alpha + \frac{\lambda k \epsilon}{\langle t \rangle}} \left[1 - e^{\left(\sigma + \alpha + \frac{\lambda k \epsilon}{\langle k \rangle}\right)(T - t)}\right],$$

where t > T and k = 1, 2, ..., K. Further,

$$\liminf_{t \to \infty} y_k(t) \ge \frac{\sigma(D_k - \epsilon)}{\sigma + \alpha + \frac{\lambda k \epsilon}{\langle k \rangle}}.$$

From the arbitrariness of ε , we have

$$\liminf_{t\to\infty} y_k(t) \ge \frac{\sigma D_k}{\sigma + \alpha},$$

which together with (8), yields

$$\lim_{t \to \infty} y_k(t) = \frac{\sigma D_k}{\sigma + \alpha}.$$
 (10)

From (9) and (10), E_0 is globally asymptotically stable in Λ . Theorem 1 holds.

Theorem 2. If $R_0 > 1$, then the endemic equilibrium E_1 of system (4) is globally asymptotically stable in $\Lambda \setminus \{E_0\}$.

Proof. Let $r_k = D_k - y_k - p_k$. Constructing Lyapunov function

$$V(y_1,...,y_K,p_1,...,p_K) = \frac{1}{2} \sum_{k=1}^K \left[v_1(k) (y_k - y_k^*)^2 + v_2(k) (r_k - r_k^*)^2 \right] + \theta - \theta^* - \theta^* \ln \frac{\theta}{\theta^*},$$

where

$$\theta = \frac{\sum_{m=1}^{K} mp_m}{\langle k \rangle}, \ \theta^* = \frac{\sum_{m=1}^{K} mp_m^*}{\langle k \rangle}, v_1(k) = \frac{k}{\langle k \rangle y_k^*},$$

$$v_2(k) = \begin{cases} \frac{(\alpha(2+\sigma)+\sigma)v_1(k)}{(\alpha-1)^2}, \text{else} \\ v_1(k)\sigma, & \alpha = 0 \\ \frac{v_1(k)\sigma}{4\alpha}. & \alpha = 1 \end{cases}$$

Obviously, we have V=0 if $(\overrightarrow{y},\overrightarrow{p})=E_1$ and V>0 if $(\overrightarrow{y},\overrightarrow{p})\neq E_1$ in $\Lambda\smallsetminus\{E_0\}$. That is, V is positive definite. Moreover, the solution of system (4) is bounded.

Case 1. $\alpha \neq 0$ and $\alpha \neq 1$. Calculating the full derivative of *V* along system (4) yields

$$\begin{split} \frac{\mathrm{d}V}{\mathrm{d}t}\Big|_{(4)} &= \sum_{k=1}^{K} \left[v_{1}(k)(y_{k} - y_{k}^{*}) \frac{\mathrm{d}y_{k}}{\mathrm{d}t} + v_{2}(k)(r_{k} - r_{k}^{*}) \frac{\mathrm{d}r_{k}}{\mathrm{d}t} \right] + \left(1 - \frac{\theta^{*}}{\theta} \right) \frac{\mathrm{d}\theta}{\mathrm{d}t} \\ &= -\sum_{k=1}^{K} v_{1}(k)(\lambda k\theta + \alpha)y_{k}(y_{k} - y_{k}^{*}) + \sum_{k=1}^{K} v_{1}(k)\delta r_{k}(y_{k} - y_{k}^{*}) + \sum_{k=1}^{K} v_{2}(k)(\alpha - 1)y_{k}(r_{k} - r_{k}^{*}) \\ &+ \sum_{k=1}^{K} v_{2}(k)(D_{k} - (1 + \delta)r_{k})(r_{k} - r_{k}^{*}) + \sum_{k=1}^{K} \frac{\lambda k^{2}y_{k}}{\langle k \rangle}(\theta - \theta^{*}) - (\theta - \theta^{*}) \\ &= -\sum_{k=1}^{K} v_{1}(k)(\lambda k\theta + \alpha)(y_{k} - y_{k}^{*})^{2} - \sum_{k=1}^{K} v_{2}(k)(1 + \delta)(r_{k} - r_{k}^{*})^{2} \\ &- \sum_{k=1}^{K} v_{1}(k)\lambda ky_{k}^{*}(y_{k} - y_{k}^{*})(\theta - \theta^{*}) - \sum_{k=1}^{K} v_{1}(k)\lambda ky_{k}^{*}\theta^{*}(y_{k} - y_{k}^{*}) - \sum_{k=1}^{K} v_{1}(k)\alpha y_{k}^{*}(y_{k} - y_{k}^{*}) \\ &+ \sum_{k=1}^{K} v_{1}(k)\delta r_{k}(y_{k} - y_{k}^{*}) + \sum_{k=1}^{K} v_{2}(k)(\alpha - 1)y_{k}(r_{k} - r_{k}^{*}) - \sum_{k=1}^{K} v_{2}(k)(1 + \delta)r_{k}^{*}(r_{k} - r_{k}^{*}) \\ &+ \sum_{k=1}^{K} v_{2}(k)D_{k}(r_{k} - r_{k}^{*}) + \sum_{k=1}^{K} \frac{\lambda k^{2}}{\langle k \rangle}(y_{k} - y_{k}^{*})(\theta - \theta^{*}) + \left(\sum_{k=1}^{K} \frac{\lambda k^{2}y_{k}^{*}}{\langle k \rangle} - 1 \right)(\theta - \theta^{*}) \\ &= -\sum_{k=1}^{K} v_{1}(k)(\lambda k\theta + \alpha)(y_{k} - y_{k}^{*})^{2} - \sum_{k=1}^{K} v_{2}(k)(1 + \delta)(r_{k} - r_{k}^{*})^{2} - \sum_{k=1}^{K} \lambda k \left(v_{1}(k)y_{k}^{*} - \frac{k}{\langle k \rangle} \right)(y_{k} - y_{k}^{*})(\theta - \theta^{*}) \\ &- \sum_{k=1}^{K} v_{1}(k)(\lambda k\theta + \alpha)(y_{k} - y_{k}^{*})^{2} - \sum_{k=1}^{K} v_{2}(k)(1 + \delta)(r_{k} - r_{k}^{*})^{2} - \sum_{k=1}^{K} \left[v_{1}(k)\lambda ky_{k}^{*} - \frac{k}{\langle k \rangle} \right)(y_{k} - y_{k}^{*})(\theta - \theta^{*}) \\ &- \sum_{k=1}^{K} v_{1}(k)(\lambda k\theta + \alpha)(y_{k} - y_{k}^{*})^{2} - \sum_{k=1}^{K} v_{2}(k)(1 + \delta)(r_{k} - r_{k}^{*})^{2} - \sum_{k=1}^{K} \left[v_{1}(k)\lambda ky_{k}^{*} - \frac{k}{\langle k \rangle} \right)(y_{k} - y_{k}^{*})(\theta - \theta^{*}) \\ &+ \sum_{k=1}^{K} v_{1}(k)(\lambda k\theta + \alpha)(y_{k} - y_{k}^{*})^{2} - \sum_{k=1}^{K} v_{2}(k)(1 + \delta)(r_{k} - r_{k}^{*})^{2} - \sum_{k=1}^{K} \left[v_{1}(k)\lambda ky_{k}^{*} - \frac{k}{\langle k \rangle} \right)(y_{k} - y_{k}^{*})(\theta - \theta^{*}) \\ &+ \sum_{k=1}^{K} v_{1}(k)(\lambda k\theta + \alpha)(y_{k} - y_{k}^{*})^{2} - \sum_{k=1}^{K} v_{2}(k)(1 + \delta)(r_{k} - r_{k}^{*})^{2} + \sum_{k=1}^{K} \left[v_{1}(k)\lambda ky_{k}$$

Let
$$x_k = y_k - y_k^*$$
, $z_k = r_k - r_k^*$. So

$$\begin{split} \frac{\mathrm{d}V}{\mathrm{d}t}\bigg|_{(4)} &= -\sum_{k=1}^K v_1(k)(\lambda k\theta + \alpha) x_k^2 - \sum_{k=1}^K v_2(k)(1+\sigma) z_k^2 + \sum_{k=1}^K [v_1(k)\sigma + v_2(k)(\alpha-1)] x_k z_k \\ &< -\sum_{k=1}^K v_1(k)\alpha x_k^2 - \sum_{k=1}^K v_2(k)(1+\sigma) z_k^2 + \sum_{k=1}^K [v_1(k)\sigma + v_2(k)(\alpha-1)] x_k z_k \\ &= \sum_{k=1}^K (x_k, z_k) D(k)(x_k, z_k)^T, \end{split}$$

where

$$D(k) = \begin{pmatrix} -\alpha \nu_1(k) & \frac{1}{2} [\sigma \nu_1(k) + \nu_2(k)(\alpha - 1)] \\ \\ \frac{1}{2} [\sigma \nu_1(k) + \nu_2(k)(\alpha - 1)] & -(\sigma + 1)\nu_2(k) \end{pmatrix}.$$

Then $-\alpha v_1(k) < 0$ and

$$\det(D(k)) = \alpha v_1(k)(\sigma + 1)v_2(k) - \frac{1}{4}[v_1(k)\sigma + v_2(k)(\alpha - 1)]^2 > 0,$$

where det(•) denotes the determinant of a matrix.

Case 2. $\alpha = 0$ or $\alpha = 1$. Similarly, it can be proved that $\det(D(k)) > 0$. Thus $(x_k, z_k)D(k)(x_k, z_k)^T$ is negative definite for $k = 1, 2, \dots, K$.

So $(dV/dt)|_{(4)}$ is negative definite. According to Corollary 4.2 (Lv & Lu, 2019), the endemic equilibrium E_1 of system (4) is globally asymptotically stable when $R_0 > 1$. Theorem 2 holds.

5. Variance

Next we analyze the variance behavior of $S_k(t)$ and $I_k(t)$. Since $y_k(t)$ and $p_k(t)$ are time-varying, it is difficult to directly analyze Eq. (5). From Theorem 2, we know that the endemic equilibrium E_1 of system (4) is globally asymptotically stable if $R_0 > 1$. In this section, we just consider the case of $R_0 > 1$. So we can study the variance of the susceptible and infected individuals around the endemic equilibrium E_1 . When t is sufficiently large, $y_k(t) \approx y_k^*$, $p_k(t) \approx p_k^*$, where y_k^* and p_k^* satisfy Eq. (7). Let $\overrightarrow{\beta} = (\beta_1, ..., \beta_K, \beta_{K+1}, ..., \beta_{2K})$, where $\beta_k = \xi_k$, $\beta_{K+k} = \eta_k$, k = 1, 2, ..., K. Replacing $y_k(t)$ with y_k^* and $p_k(t)$ with p_k^* in Eq. (5) yields

$$\begin{split} \frac{\partial \Pi\left(\overrightarrow{\beta},t\right)}{\partial t} &= \sum_{k=1}^{K} \left[\sigma \frac{\partial (\beta_{k}\Pi)}{\partial \beta_{k}} + \sigma \frac{\partial (\beta_{K+k}\Pi)}{\partial \beta_{k}} + \alpha \frac{\partial (\beta_{k}\Pi)}{\partial \beta_{k}} + \frac{\lambda k y_{k}^{*}}{\langle k \rangle} \frac{\partial}{\partial \beta_{k}} \left(\sum_{i=1}^{K} i \beta_{K+i} \Pi \right) + \frac{\lambda k \sum_{i=1}^{K} i p_{i}^{*}}{\langle k \rangle} \frac{\partial (\beta_{k}\Pi)}{\partial \beta_{k}} \right. \\ &\quad + \frac{\partial (\beta_{K+k}\Pi)}{\partial \beta_{K+k}} - \frac{\lambda k y_{k}^{*}}{\langle k \rangle} \frac{\partial}{\partial \beta_{K+k}} \left(\sum_{i=1}^{K} i \beta_{K+i} \Pi \right) - \frac{\lambda k \sum_{i=1}^{K} i p_{i}^{*}}{\langle k \rangle} \frac{\partial (\beta_{k}\Pi)}{\partial \beta_{K+k}} \right] \\ &\quad + \frac{1}{2} \sum_{k=1}^{K} \left\{ \left[\sigma(D_{k} - y_{k}^{*} - p_{k}^{*}) + \alpha y_{k}^{*} + \frac{\lambda k y_{k}^{*} \sum_{i=1}^{K} i p_{i}^{*}}{\langle k \rangle} \frac{\partial^{2}\Pi}{\partial \beta_{k}^{2}} \right. \\ &\quad + \left. \left(p_{k}^{*} + \frac{\lambda k y_{k}^{*} \sum_{i=1}^{K} i p_{i}^{*}}{\langle k \rangle} \right) \frac{\partial^{2}\Pi}{\partial \beta_{K+k}^{2}} - \frac{2\lambda k y_{k}^{*} \sum_{i=1}^{K} i p_{i}^{*}}{\langle k \rangle} \frac{\partial^{2}\Pi}{\partial \beta_{k}^{2}} \right. \\ &\quad = \sum_{k=1}^{K} \left[\sigma \frac{\partial (\beta_{k}\Pi)}{\partial \beta_{k}} + \sigma \frac{\partial (\beta_{K+k}\Pi)}{\partial \beta_{k}} + \alpha \frac{\partial (\beta_{k}\Pi)}{\partial \beta_{k}} + \frac{\lambda k y_{k}^{*}}{\langle k \rangle} \frac{\partial}{\partial \beta_{k}} \left(\sum_{i=1}^{K} i \beta_{K+i} \Pi \right) + \frac{\lambda k \sum_{i=1}^{K} i p_{i}^{*}}{\langle k \rangle} \frac{\partial (\beta_{k}\Pi)}{\partial \beta_{k}} \right. \\ &\quad + \frac{\partial (\beta_{K+k}\Pi)}{\partial \beta_{K+k}} - \frac{\lambda k y_{k}^{*}}{\langle k \rangle} \frac{\partial}{\partial \beta_{K+k}} \left(\sum_{i=1}^{K} i \beta_{K+i} \Pi \right) - \frac{\lambda k \sum_{i=1}^{K} i p_{i}^{*}}{\langle k \rangle} \frac{\partial (\beta_{k}\Pi)}{\partial \beta_{K+k}} \right] \\ &\quad + \frac{1}{2} \sum_{k=1}^{K} \left[2\sigma(D_{k} - y_{k}^{*} - p_{k}^{*}) \frac{\partial^{2}\Pi}{\partial \beta_{k}^{2}} + (2p_{k}^{*}) \frac{\partial^{2}\Pi}{\partial \beta_{K+k}^{2}} - (2p_{k}^{*}) \frac{\partial^{2}\Pi}{\partial \beta_{k}^{2} \partial \beta_{K+k}} \right]. \end{aligned}$$

Further, the above equation can be expressed as

$$\frac{\partial \Pi(\overrightarrow{\beta},t)}{\partial t} = -\sum_{k=1}^{2K} a_{kj} \frac{\partial (\beta_j \Pi)}{\partial \beta_k} + \frac{1}{2} \sum_{k=1}^{2K} b_{kj} \frac{\partial^2 \Pi}{\partial \beta_k \partial \beta_j},\tag{11}$$

where a_{kj} is the element in the k-th row and j-th column of the matrix A at the endemic equilibrium E_1 and b_{kj} is the element in the k-th row and j-th column of matrix

$$B = \begin{bmatrix} e_{11} & 0 & \cdots & 0 & m_{11} & 0 & \cdots & 0 \\ 0 & e_{22} & \cdots & 0 & 0 & m_{22} & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & e_{KK} & 0 & 0 & \cdots & m_{KK} \\ m_{11} & 0 & \cdots & 0 & t_{11} & 0 & \cdots & 0 \\ 0 & m_{22} & \cdots & 0 & 0 & t_{22} & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & m_{KK} & 0 & 0 & \cdots & t_{KK} \end{bmatrix}$$

with $e_{ii} = 2(D_i - y_i^* - p_i^*)\sigma$, $m_{ii} = -p_i^*$, $t_{ii} = 2p_i^*$, i = 1, ..., K. Let $\overrightarrow{\chi} = (\chi_1, ..., \chi_K, \chi_{K+1}, ..., \chi_{2K})^T$, where $\chi_k(t) = \varphi_k(t)$, $\chi_{K+k}(t) = \omega_k(t)$, k = 1, 2, ..., K. The stochastic process $\overrightarrow{\chi}$ which takes the solution of the Fokker-Plank equation (11) as its probability density function satisfies the following linear stochastic differential equation:

$$d\overrightarrow{\chi} = A\overrightarrow{\chi}(t)dt + \Gamma dW(t)$$

with the initial condition $\overrightarrow{\chi}(0) = 0$, where Γ is a $2K \times 2K$ constant matrix and $\Gamma\Gamma^T = B$, dW(t) are 2K-dimensional column vectors. W(t) contains 2K independent standard Brownian motions. Then the solution to Eq. (11) is a multivariate Gaussian distribution

$$\Pi\left(\overrightarrow{\beta},t\right) = \frac{1}{\left(\sqrt{2\pi}\right)^{2K}|\psi(t)|^{\frac{1}{2}}} \exp\left\{-\frac{1}{2}\left(\overrightarrow{\beta} - \overrightarrow{\mu}(t)\right)^{T}\psi(t)^{-1}\left(\overrightarrow{\beta} - \overrightarrow{\mu}(t)\right)\right\},\,$$

where $\overrightarrow{\mu}(t) = \mathbf{E}(\overrightarrow{\chi}(t))$ and

$$\psi(t) = \mathbf{E}\Big((\overrightarrow{\chi}(t) - \overrightarrow{\mu}(t))(\overrightarrow{\chi}(t) - \overrightarrow{\mu}(t))^T\Big) = (e_{ij}(t))_{2K \times 2K}$$

satisfy

$$\begin{cases}
\frac{d\overrightarrow{\mu}(t)}{dt} = A\overrightarrow{\mu}(t), \\
\overrightarrow{\mu}(0) = 0,
\end{cases}$$
(12)

and

$$\begin{cases} \frac{\mathrm{d}\psi(t)}{\mathrm{d}t} = A\psi + \psi A^T + B, \\ \psi(0) = 0. \end{cases}$$
 (13)

Hence, $\overrightarrow{\mu}(t) \equiv 0$. Next we shall analyze $\psi(t)$.

Lemma 1. If $(y_1^{**},...,y_K^{**},p_1^{**},...,p_K^{**})=E_1$, all eigenvalues of A have negative real parts.

Proof. The characteristic equation of A at the endemic equilibrium E_1 is

$$|\mu I - A| = \begin{vmatrix} D & F \\ G & H \end{vmatrix} = 0,\tag{14}$$

where *I* is an identity matrix and $D = (d_{ii})_{K \times K}$, $F = (f_{ii})_{K \times K}$, $G = (g_{ii})_{K \times K}$, $H = (h_{ii})_{K \times K}$ with

$$d_{ii} = \mu + \alpha + \sigma$$
, $d_{ij} = 0$, $f_{ii} = \mu + \sigma + 1$, $f_{ij} = 0$,

$$g_{ii} = -\frac{i\lambda \sum_{k=1}^{K} k p_k^*}{\langle k \rangle}, g_{ij} = 0,$$

$$h_{ii} = \mu + 1 - \frac{i^2 \lambda y_i^*}{\langle k \rangle}, \ h_{ij} = -\frac{ij \lambda y_i^*}{\langle k \rangle}$$

for $i \neq j$, i, j = 1, 2, ..., K.

We assume that μ_1 is any eigenvalue of A. Let $Re(\mu_1)$ and $Im(\mu_1)$ denote the real and imaginary parts of μ_1 . Next, we prove that $Re(\mu_1) < 0$.

Case 1. $\alpha = 1$. (i) We first consider

$$\sigma \neq \frac{i\lambda \sum_{k=1}^{K} k p_k^*}{\langle k \rangle}.$$

Since

$$\left| \left(-1 - \frac{i\lambda \sum_{k=1}^{K} kp_{k}^{*}}{\langle k \rangle} \right) I - A \right| = -\left(\sigma - \frac{i\lambda \sum_{k=1}^{K} kp_{k}^{*}}{\langle k \rangle} \right)^{K} \frac{i^{2}\lambda y_{i}^{*} \left(\lambda \sum_{k=1}^{K} kp_{k}^{*} \right)^{K-1}}{\langle k \rangle^{K}} \prod_{j=1}^{K} (j-i) \neq 0,$$

we have $\mu_1 \neq -1 - i\lambda \sum_{k=1}^K k p_k^* / \langle k \rangle$. According to (14), we have

$$|\mu_1 I - A| = g_1(\mu_1)h_1(\mu_1) = 0,$$

where $g_1(\mu_1) = (\mu_1 + \sigma + 1)^K$ and

$$h_1(\mu_1) = \left[1 - \sum_{k=1}^K \frac{k^2 \lambda y_k^*}{\langle k \rangle \left(\mu_1 + 1 + \frac{k \lambda \sum_{m=1}^K m p_m^*}{\langle k \rangle}\right)}\right] \prod_{i=1}^K \left(\mu_1 + 1 + \frac{i \lambda \sum_{k=1}^K k p_k^*}{\langle k \rangle}\right).$$

That is, $\mu_1 = -(\sigma + 1)$ or μ_1 satisfies

$$\sum_{k=1}^{K} \frac{k^2 \lambda y_k^*}{\langle k \rangle \left(\mu_1 + 1 + \frac{k \lambda \sum_{m=1}^{K} m p_m^*}{\langle k \rangle} \right)} = 1. \tag{15}$$

Now, we claim that $Re(\mu_1) < 0$. Otherwise, we can assume $Re(\mu_1) \ge 0$. In this case, we have

$$\begin{split} \left| \sum_{k=1}^{K} \frac{k^2 \lambda y_k^*}{\langle k \rangle \left(\mu_1 + 1 + \frac{k \lambda \sum_{m=1}^{K} m p_m^*}{\langle k \rangle} \right)} \right| &\leq \sum_{k=1}^{K} \left| \frac{k^2 \lambda y_k^*}{\langle k \rangle \left(\mu_1 + 1 + \frac{k \lambda \sum_{m=1}^{K} m p_m^*}{\langle k \rangle} \right)} \right| \\ &\leq \sum_{k=1}^{K} \frac{k^2 \lambda y_k^*}{\langle k \rangle \left(\operatorname{Re}(\mu_1) + 1 + \frac{k \lambda \sum_{m=1}^{K} m p_m^*}{\langle k \rangle} \right)} \\ &< \sum_{k=1}^{K} \frac{k^2 \lambda y_k^*}{\langle k \rangle} \\ &= 1. \end{split}$$

This is a contradiction with (15). Hence, $Re(\mu_1) < 0$. (ii) If

$$\sigma = \frac{i\lambda \sum_{k=1}^{K} k p_k^*}{\langle k \rangle},$$

we can obtain that

$$\mu_1 = -1 - \frac{i\lambda \sum_{k=1}^{K} k p_k^*}{\langle k \rangle}$$

for some $i \in \{1, 2, ..., K\}$, or μ_1 satisfies (15). Similarly, we can prove $Re(\mu_1) < 0$.

Case 2. $\alpha \neq 1$. Since

$$|-(\alpha+\sigma)I-A|=(1-\alpha)^K\prod_{i=1}^K\frac{i\lambda\sum_{m=1}^Kmp_m^*}{\langle k\rangle}\neq 0,$$

we know $\mu_1 \neq -(\alpha + \sigma)$.

(i) If
$$\mu_1 = -1 - \alpha$$
, Re(μ_1) < 0.

(ii) If $\mu_1 \neq -1 - \alpha$, then

$$\left| \left(-1 - \frac{(\mu_1 + \alpha + 1)i\lambda \sum_{k=1}^{K} k p_k^*}{(\mu_1 + \alpha + \sigma)\langle k \rangle} \right) I - A \right| = (\mu_1 + \alpha + \sigma)^K \left[\frac{(\mu_1 + \alpha + 1)\lambda \sum_{k=1}^{K} k p_k^*}{(\mu_1 + \alpha + \sigma)\langle k \rangle} \right]^{K-1} \left(-\frac{i^2 \lambda y_i^*}{\langle k \rangle} \right) \prod_{\substack{j=1 \ j \neq i}}^{K} (j-i) \neq 0.$$

$$(16)$$

From (16), we have

$$\mu_1 \neq -1 - \frac{(\mu_1 + \alpha + 1)i\lambda \sum_{k=1}^K k p_k^*}{(\mu_1 + \alpha + \sigma)\langle k \rangle}.$$

According to (14),

$$|\mu_1 I - A|$$

$$= (\mu_1 + \alpha + \sigma)^K \prod_{i=1}^K \left[\mu_1 + 1 + \frac{(\mu_1 + \sigma + 1)i\lambda\sum_{k=1}^K kp_k^*}{(\mu_1 + \alpha + \sigma)\langle k \rangle} \right] \left[1 - \sum_{k=1}^K \frac{k^2\lambda y_k^*}{\langle k \rangle \left(\mu_1 + 1 + \frac{(\mu_1 + \sigma + 1)k\lambda\sum_{m=1}^K mp_m^*}{(\mu_1 + \alpha + \sigma)\langle k \rangle} \right)} \right]$$

= 0.

Hence, μ_1 satisfies

$$\sum_{k=1}^{K} \frac{k^2 \lambda y_k^*}{\langle k \rangle \left(\mu_1 + 1 + \frac{(\mu_1 + \sigma + 1)k \lambda \sum_{m=1}^{K} m p_m^*}{(\mu_1 + \alpha + \sigma) \langle k \rangle} \right)} = 1.$$
 (17)

Now, we claim that $Re(\mu_1) < 0$. Otherwise, we can assume $\mu_1 = a + bi$, where $a \ge 0$. In this case, we have

$$\begin{split} &\left|\mu_{1}+1+\frac{(\mu_{1}+\sigma+1)k\lambda\sum_{m=1}^{K}mp_{m}^{*}}{(\mu_{1}+\alpha+\sigma)\langle k\rangle}\right| \\ &=\left(\left[a+1+\frac{k\lambda\sum_{m=1}^{K}mp_{m}^{*}}{\langle k\rangle}\left(\frac{(a+\sigma+1)(a+\sigma+\alpha)+b^{2}}{(a+\sigma+\alpha)^{2}+b^{2}}\right)\right]^{2}+\left[(\alpha-1)b\frac{k\lambda\sum_{m=1}^{K}mp_{m}^{*}}{\langle k\rangle}+b\right]^{2}\right)^{\frac{1}{2}} \\ &>1. \end{split}$$

Then.

$$\left| \sum_{k=1}^K \frac{k^2 \lambda y_k^*}{\langle k \rangle \left(\mu_1 + 1 + \frac{(\mu_1 + \sigma + 1)k\lambda \sum_{m=1}^K mp_m^*}{(\mu_1 + \alpha + \sigma)\langle k \rangle} \right)} \right| \leq \sum_{k=1}^K \frac{k^2 \lambda y_k^*}{\langle k \rangle \left| \mu_1 + 1 + \frac{(\mu_1 + \sigma + 1)k\lambda \sum_{m=1}^K mp_m^*}{(\mu_1 + \alpha + \sigma)\langle k \rangle} \right|} < \sum_{k=1}^K \frac{k^2 \lambda y_k^*}{\langle k \rangle} = 1.$$

This is a contradiction with (17). Then $Re(\mu_1) < 0$. Hence, all eigenvalues of A have negative real parts. Lemma 1 holds.

From Lemma 1 and Theorem 4.3 in Qian (2001), we have $\psi(t) \to \psi^s$ when $t \to \infty$, where $\psi^s = \left(e^s_{ij}\right)_{2K \times 2K}$ satisfies the following Lyapunov equation

$$A\psi^S + \psi^S A^T = -B. \tag{18}$$

In fact, the elements of principal diagonal of $\psi(t)$ are

$$e_{11}(t) = \operatorname{Var}\left(\frac{S_1(t) - Ny_1^*}{\sqrt{N}}\right) = \operatorname{Var}\left(\frac{S_1(t)}{\sqrt{N}}\right),$$

:

$$e_{KK}(t) = Var\left(\frac{S_K(t) - Ny_K^*}{\sqrt{N}}\right) = Var\left(\frac{S_K(t)}{\sqrt{N}}\right),$$

$$e_{K+1,K+1}(t) = \operatorname{Var}\left(\frac{I_1(t) - Np_1^*}{\sqrt{N}}\right) = \operatorname{Var}\left(\frac{I_1(t)}{\sqrt{N}}\right),$$

:

$$e_{2K,2K}(t) = \operatorname{Var}\left(\frac{I_K(t) - Np_K^*}{\sqrt{N}}\right) = \operatorname{Var}\left(\frac{I_K(t)}{\sqrt{N}}\right),$$

where $Var(\cdot)$ denotes the variance of random variable. Further, we can obtain $Var(S_k(t))$ and $Var(I_k(t))$ around endemic equilibrium E_1 . That is, the asymptotic expressions for the variance of $S_k(t)$ and $I_k(t)$ (k = 1, 2, ..., K) are

$$Var(S_1(t)) \rightarrow Ne_{11}^s, ..., Var(S_K(t)) \rightarrow Ne_{KK}^s$$

$$Var(I_1(t)) \rightarrow Ne_{K+1,K+1}^{s}, ..., Var(I_K(t)) \rightarrow Ne_{2K,2K}^{s}$$

when $t \to \infty$. From Eq. (18), we can know

$$\operatorname{vec}(A\psi^{S} + \psi^{S}A^{T}) = (I \otimes A + A \otimes I)\operatorname{vec}(\psi^{S}) = \operatorname{vec}(-B).$$

where $\mathbf{vec}(\cdot)$ is vectorization operator of matrices, and \otimes is Kronecker product. Further,

$$\mathbf{vec}(\psi^{S}) = (I \otimes A + A \otimes I)^{-1} \mathbf{vec}(-B) \triangleq \overrightarrow{u}, \tag{19}$$

where \overrightarrow{u} is a $4K^2 \times 1$ vector and e_{kk}^s equals the (2K(k-1)+k)-th component of \overrightarrow{u} .

It can be seen that the analytical expression of e_{kk}^s is difficult to be obtained directly. So we can use numerical methods to get e_{kk}^s .

In addition, let $C_k(t)$ and $V_k(t)$ be the coefficients of variation of $S_k(t)$ and $I_k(t)$, respectively. Hence,

$$C_k(t) = \frac{\sqrt{\text{Var}(S_k(t))}}{\text{E}(S_k(t))} \rightarrow C_k = \frac{\sqrt{Ne_{kk}^S}}{Ny_k^*}$$

and

$$V_k(t) = \frac{\sqrt{\operatorname{Var}(I_k(t))}}{\operatorname{E}(I_k(t))} \to V_k = \frac{\sqrt{Ne_{K+k,K+k}^s}}{Np_k^s}$$

when $t \to \infty$, k = 1, 2, ..., K.

6. Numerical simulations

Numerical simulations and Monte Carlo simulations are carried out in this section. Let K = 6, N = 1000, $D_1 = 0.2$, $D_2 = 0.3$, $D_3 = 0.1$, $D_4 = 0.1$, $D_5 = 0.2$, $D_6 = 0.1$, $\langle k \rangle = 3.1$, $\langle k^2 \rangle = 12.5$.

Example 1. Let the infection rate $\lambda = 0.2$, the vaccination rate $\alpha = 0.2$, the rate of loss of immunity $\sigma = 0.8$. The calculation yields $R_0 \approx 0.645 < 1$, so system (4) only has disease-free equilibrium $E_0 = (0.1600, 0.2400, 0.0800, 0.0800, 0.0800, 0.0800, 0.090, 0.$

Example 2. Let the infection rate $\lambda = 0.8$, the vaccination rate $\alpha = 0.2$, the rate of loss of immunity $\sigma = 0.4$. The calculation yields $R_0 \approx 2.15 > 1$, so system (4) has an endemic equilibrium $E_1 = (0.1058, 0.1315, 0.0374, 0.0326, 0.0579, 0.0260, 0.0118, 0.0294, 0.0125, 0.0146, 0.0323, 0.0174). From Theorem 2, the endemic equilibrium <math>E_1$ is globally asymptotically stable in $\Lambda \setminus \{E_0\}$. Fig. 3 only presents time evolution curves of $y_1(t)$, $y_4(t)$, $p_1(t)$, $p_4(t)$ for different initial values. Numerical simulation illustrates this fact.

In Fig. 4, we show the effect of vaccination rate α on the mean $Np_k(t)$ of $I_k(t)$. Here we just present $Np_1(t)$ and $Np_4(t)$. The other $Np_k(t)$ is similarly. It can be seen that Np_k^* decreases as α increases when $R_0 > 1$ and the other parameters are fixed. This finding indicates that vaccination can reduce infection. Consequently, increasing the rate and coverage of vaccination is a necessary and effective means for disease prevention and control.

Let

$$\tau_k(t) = \frac{\mathrm{E}(I_k(t))}{N_k} = \frac{p_k(t)}{D_k}, k = 1, 2, ..., K.$$

 $\tau_k(t)$ can be used to evaluate the effect of networks on mean of the number of infected individual. In Fig. 5, we find that τ_k increases as k increases when t is sufficiently large. This indicates individuals with greater degree are more easily infected. The contact network implies that reducing crowd gatherings can decrease the number of edges of a individual, thereby lowering the risk of infection. In addition, from Fig. 5(a) and (b), we find that τ_k is larger with a larger λ when t is sufficiently large. In Fig. 5(a) and (c), it can be seen that τ_k is smaller for a larger α when t is sufficiently large. By comparing Fig. 5(a) and (d), it can be observed that τ_k is larger with a larger σ when t is sufficiently large. These results emphasize the significance of wearing masks and extending the duration of vaccine efficacy for disease control. Additionally, the t and t an

$$A = \begin{pmatrix} A_{11} & A_{12} \\ A_{21} & A_{22} \end{pmatrix},$$

where

 $A_{11} = -\text{diag}(0.7116, 0.8233, 0.9349, 1.0466, 1.1582, 1.2699),$

 $A_{21} = diag(0.1116, 0.2233, 0.3349, 0.4466, 0.5582, 0.6699),$

$$A_{12} = - \begin{pmatrix} 0.4273 & 0.0546 & 0.0819 & 0.1092 & 0.1365 & 0.1638 \\ 0.0679 & 0.5357 & 0.2036 & 0.2715 & 0.3393 & 0.4072 \\ 0.0290 & 0.0579 & 0.4869 & 0.1159 & 0.1449 & 0.1738 \\ 0.0337 & 0.0674 & 0.1011 & 0.5348 & 0.1685 & 0.2022 \\ 0.0747 & 0.1494 & 0.2242 & 0.2989 & 0.7736 & 0.4483 \\ 0.0403 & 0.0806 & 0.1208 & 0.1611 & 0.2014 & 0.6417 \end{pmatrix}$$

and

$$A_{22} = \begin{pmatrix} -0.9727 & 0.0546 & 0.0819 & 0.1092 & 0.1365 & 0.1638 \\ 0.0679 & -0.8643 & 0.2036 & 0.2715 & 0.3393 & 0.4072 \\ 0.0290 & 0.0579 & -0.9131 & 0.1159 & 0.1449 & 0.1738 \\ 0.0337 & 0.0674 & 0.1011 & -0.8652 & 0.1685 & 0.2022 \\ 0.0747 & 0.1494 & 0.2242 & 0.2989 & -0.6264 & 0.4483 \\ 0.0403 & 0.0806 & 0.1208 & 0.1611 & 0.2014 & -0.7583 \end{pmatrix}$$

Further,

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{12} & B_{22} \end{pmatrix},$$

where

 $B_{11} = \text{diag}(0.0659, 0.1113, 0.0400, 0.0422, 0.0878, 0.0453),$

$$B_{12} = -\text{diag}(0.0118, 0.0294, 0.0125, 0.0146, 0.0323, 0.0174)$$

and

 $B_{22} = diag(0.0236, 0.0587, 0.0251, 0.0292, 0.0647, 0.0348).$

According to Eq. (19), we obtain

$$\psi^{s} = \begin{pmatrix} T_1 & T_2 \\ T_2 T & T_3 \end{pmatrix},$$

where

$$T_1 = \begin{pmatrix} 0.0558 & 0.0135 & 0.0053 & 0.0057 & 0.0117 & 0.0059 \\ 0.0135 & 0.1050 & 0.0123 & 0.0132 & 0.0273 & 0.0138 \\ 0.0053 & 0.0123 & 0.0283 & 0.0053 & 0.0109 & 0.0055 \\ 0.0057 & 0.0132 & 0.0053 & 0.0277 & 0.0119 & 0.0061 \\ 0.0117 & 0.0273 & 0.0109 & 0.0119 & 0.0660 & 0.0127 \\ 0.0059 & 0.0138 & 0.0055 & 0.0061 & 0.0127 & 0.0257 \end{pmatrix}$$

$$T_2 = - \begin{pmatrix} 0.0093 & 0.0073 & 0.0030 & 0.0034 & 0.0076 & 0.0041 \\ 0.0074 & 0.0304 & 0.0073 & 0.0083 & 0.0183 & 0.0099 \\ 0.0030 & 0.0071 & 0.0077 & 0.0034 & 0.0075 & 0.0041 \\ 0.0033 & 0.0079 & 0.0033 & 0.0086 & 0.0085 & 0.0046 \\ 0.0069 & 0.0167 & 0.0070 & 0.0081 & 0.0274 & 0.0098 \\ 0.0035 & 0.0086 & 0.0036 & 0.0042 & 0.0093 & 0.0096 \end{pmatrix}$$

and

$$T_3 = \begin{pmatrix} 0.0130 & 0.0047 & 0.0020 & 0.0024 & 0.0053 & 0.0029 \\ 0.0047 & 0.0382 & 0.0050 & 0.0059 & 0.0133 & 0.0073 \\ 0.0020 & 0.0050 & 0.0131 & 0.0025 & 0.0057 & 0.0031 \\ 0.0024 & 0.0059 & 0.0025 & 0.0154 & 0.0067 & 0.0037 \\ 0.0053 & 0.0133 & 0.0057 & 0.0067 & 0.0422 & 0.0082 \\ 0.0029 & 0.0073 & 0.0031 & 0.0037 & 0.0082 & 0.0189 \end{pmatrix}$$

Further, when t is sufficiently large,

$$Var(S_1(t)) \rightarrow 55.8$$
, $Var(S_2(t)) \rightarrow 105.0$, $Var(S_3(t)) \rightarrow 28.3$,

$$Var(S_4(t)) \rightarrow 27.7$$
, $Var(S_5(t)) \rightarrow 66.0$, $Var(S_6(t)) \rightarrow 25.7$,

$$Var(I_1(t)) \rightarrow 13.0$$
, $Var(I_2(t)) \rightarrow 38.2$, $Var(I_3(t)) \rightarrow 13.1$,

$$Var(I_4(t)) \rightarrow 15.4$$
, $Var(I_5(t)) \rightarrow 42.2$, $Var(I_6(t)) \rightarrow 18.9$.

Figs. 6 and 7 plot three lines. The red lines represent the variance of $S_k(t)$ and $I_k(t)$ with 95 % confidence interval based on 2000 Monte Carlo simulations when X(0) = ((175, 25), (260, 40), (90, 10), (82, 18), (170, 30), (85, 15)). The green lines represent numerical solutions of the variance which are the elements of principal diagonal of $N\psi(t)$ obtained by solving Eq. (13). And the blue dots represent Ne_{kk}^s which are obtained by solving Eq. (19). From these figures, it can be seen that the green lines and blue dots are very close to the red lines when t is sufficiently large. Therefore, it is valid to measure the variance of the number of

infected individuals by using $N\psi^s$ when t is sufficiently large. However, we can find that red lines and green lines are not very close in the first half of the curve in Figs. 6 and 7. Because the results are only valid around the endemic equilibrium.

To better understand the effect of vaccination on infectious diseases, we analyze the change of coefficients of variation V_k with respect to vaccination rate α . In Fig. 8, V_k (k=1,2,...,6) increases as α increases. That is, vaccination can increase the magnitude of fluctuations of the number of infected individuals. Hence, vaccination is more beneficial to extinction of infectious disease. Additionally, we notice that V_k has no monotonic relationship with degree k from Fig. 8. By comparing Fig. 8(a), (b) and 8(c), it can be observed that the change of α has a greater effect on V_k when λ and σ are smaller. These observations offer a useful strategy: during periods of low infection rates, it is advisable to intensify vaccination efforts to amplify fluctuations around the endemic equilibrium and facilitate disease extinction.

7. Conclusion

Considering the heterogeneity of the contact structure and the implementation of vaccination, this paper analyzes stochastic SIRS models with vaccination on networks for large-scale populations. We employ Van Kampen's system-size expansion to derive a deterministic system for the mean behavior and a Fokker-Planck equation that characterizes the variance around the endemic equilibrium. Our results suggest that the variance of the number of susceptible and infected individuals around the endemic equilibrium can be approximated by the elements of principal diagonal of the solution ψ^S of Lyapunov equation when t is sufficiently large. The solution of Lyapunov equation can be given by applying the vectorization operator of matrices and Kronecker product. By theoretical results and numerical simulations, we illustrate that vaccination can reduce infection and is more beneficial to elimination of infectious disease and show that individuals with greater degree are more easily infected. Therefore, accelerating vaccination and reducing contact between individuals contribute to the control of infectious diseases.

On a technical level, this paper also overcomes some challenges. For instance, the covariance matrix converges to ψ^S provided that all eigenvalues of the coefficient matrix A of the linearized system at the endemic equilibrium have negative real parts. However, due to the high dimensionality of epidemic models on networks, it's complex to prove directly that this condition holds. In this paper, we employ proof by contradiction and mathematical analysis techniques to prove that this condition holds. Consequently, the asymptotic expression for the variance can be obtained.

Additionally, besides Van Kampen's system-size expansion, the theory of density dependent jump Markov processes can also be used to investigate the mean and variance. Its core content is the law of large numbers and the central limit theorem. The theory shows that a normed jump Markov process almost sure converges to a deterministic process and a centered and scaled process converges weakly to a Gaussian process when population size is sufficiently large. By employing this theory, similar results regarding the mean and variance of the number of susceptible and infected individuals can also be obtained as those presented in this paper. However, two methods are valid for large-scale populations.

For epidemic models on network with small-scale populations, the mean and variance can be analyzed by developing a recursive algorithm. The recursive algorithm is based on first-step analysis, the generating functions and infinitesimal generator matrices. It is exact, numerically stable and computationally efficient for numerical implementation when population sizes are small. But the recursive algorithm is not suitable for large-scale populations, as it becomes very time-consuming.

CRediT authorship contribution statement

Tingting Chen: Writing — original draft, Validation, Methodology, Formal analysis, Conceptualization. **Guirong Liu:** Writing — review & editing, Validation, Supervision, Methodology, Investigation, Funding acquisition, Conceptualization. **Zhen Jin:** Writing — review & editing, Supervision, Methodology, Funding acquisition.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Prof. Yanni Xiao is an editorial board member but she did not involved in the editorial process of this paper.

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