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随机多种群易感者、感染者和移出者传染病模型的阈值

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摘要: 本文建立了一类随机多种群易感者、感染者和移出者(susceptical infective and removal, SIR)传染病微分方程模型, 针对模型找到与随机因素相关的阈值用于判定疾病的消亡与否. 通过阈值里随机干扰的作用给出疾病防控的新方法—随机镇定. 与此同时, 本文探究无病平衡点的全局稳定性并通过数据仿真实例解释上述理论结果的正确性和可行性.

关键词: 随机多种群SIR传染病模型; 阈值; 渐进稳定性; 随机镇定

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Sharp threshold of a multi-group susceptical infective and removal model by stochastic perturbation

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Abstract: For a stochastic differential equation epidemic model of multi-group susceptical infective and removal (SIR) type, we define the basic reproduction number $R_0^{\rm S}$ and show that it is a sharp threshold for the dynamics of the stochastic multi-group SIR model which determines whether the epidemic occurs or not. Our analytic results of stochastic stabilization applies a new viable measure to disease control. Furthermore, we investigate the global asymptotic behaviour of the disease. Finally we give numerical simulation to illustrate our analytical results.

Key words: stochastic multi-group SIR model; sharp threshold; asymptotically stable; stochastic stabilization

1 Introduction

Epidemic models have been proposed for modeling the spread process of infectious diseases, they describe the transmission dynamics of an infectious disease in a host population. Multigroup model is a special type of epidemic model which describes infectious diseases transmission in heterogeneous host populations, such as meals, mumps, gonorrhea, HIV/AIDS, West-Nile virus and vector borne diseases such as Malaria. Groups can be separated by different factors: region graphical, gender, differences and so on. One of the earliest works on multi-group models is the seminal paper by Lajmanovich and Yorke[1] on a class of multi-group SIS model for the transmission dynamics of Gonorrhea, they established a complete analysis of the global dynamics. The global stability of the unique equilibrium is proved by using a complete analysis of the global Lyapunov function. Subsequently, much research has been done on multi-group models, such as[2], Beretta and Capasso developed a graph theoretic method to analysis the global stability of a multi-group SIR model. This effective tool has been used in many papers[3−8]. But how to establish the Lyapunov functions still be a challenging problem. Li et al.[9] derives the following multi-group SIR model:

$$\begin{cases} S_k' = A_k - d_k^s S_k - \sum\limits_{j=1}^{n} \beta_{kj} S_k I_j, \\ I_k' = \sum\limits_{j=1}^{n} \beta_{kj} S_k I_j - (d_k^{\rm I} + \epsilon_k + \gamma_k) I_k, \\ R_k' = \gamma_k I_k - d_k^{\rm R} R_k, \end{cases} \tag{1}$$

where $k = 1, 2, \cdots, n$, the model describes the spread of an infectious disease in a heterogeneous population, which is partitioned into n homogeneous group. Each group k is further compartmentalized into S_k, I_k and R_k, here S_k, I_k and R_k denote the susceptible, infective and recovered population at time t respectively. All parameters in the above model are summarized in the following list:

β_{ij}: transmission coefficient between compartments S_i and I_j,

$d_k^{\rm S}; d_k^{\rm I}; d_k^{\rm R}$: nature death rates of S, I, R compart-

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ments in the k-th group, respectively,

A_k: influx of individuals into the k-th group,

γ_i: recovery rate of infectious individuals in the i-th group,

ϵ_k: disease-caused death rate in the k-th group.

All parameter values are assumed to be nonnegative and $d_k^{\rm S}$, $d_k^{\rm I}$, $d_k^{\rm R}$, $A_k > 0$ for all k. Guo et al.[9] established the whole dynamic behaviour of the disease which determined by the basic reproduction number R_0.

In the real large-scale biological systems, there are always subject to environment noise. It is therefore necessary to develop stochastic models. To account for variability of the environment and stochasticity in the disease transmission process, as well as uncertainty in measurement of model parameters, There are generally two approaches to derive a SDE model[10−24]. To describe demographic stochasticity, Allen[10] discussed the approach of deriving stochastic differential equations from the forward Kolmogorov equation of continuous time Markov chain models. To incorporate stochasticity in measurement and estimation of model parameters, noise terms have been introduced into deterministic models as perturbations to model parameters. Tornatore et al.[16] studied a SDE model of SIR type by perturbing the transmission coefficient β. Lyapunov functions were used to prove that the disease-free equilibrium is asymptotically stable. This stability condition is only sufficient since simulations show that the disease-free equilibrium can be stable when this condition is not satisfied. Gray et al.[18] studied a stochastic SIS model and derived stability conditions by considering Lyapunov function $\log I(t)$. Note that the total population $N(t)$ remains a constant. Ji et al.[20] considered a multi-group stochastic SIR model by perturbing death rates. Lyapunov functions were used to investigate asymptotic behaviors near the disease-free equilibrium. Recently, Zhao et al.[25] investigated a stochastic SIS model and found out a threshold of the stochastic model in case the white noises are small. It remains an open question find an appropriate form of a sharp threshold parameter for the dynamics of SDE epidemic models that plays the role of R_0 for ODE models.

The above epidemic models are deterministic models based on the assumption that stochastic factors would be ignored. But the real biological systems are always perturbed by various types of environment noise. To be more accurate description of epidemic model, we establish a stochastic multi-group SIR model. We also find out a threshold $R_0^{\rm S}$ which is the expand of the deterministic threshold R_0.

Based on the fact that the transmission coefficient β is the most sensitive parameter[10], we perturb the transmission coefficient of the deterministic multi-group SIR model (1) by replacing β_{kk} to $\beta_{kk}+\sigma_k\dot{B}(t)$ where $B(t)$ is a standard brownian motion with $B(0)=0$. Then we formulate a stochastic multi-group SIR model as follows:

$$\begin{cases} {\rm d}S_k = s_k(t){\rm d}t - \sigma_k S_k I_k {\rm d}B(t), \\ {\rm d}I_k = i_1(t){\rm d}t + \sigma_k S_k I_k {\rm d}B(t), \\ {\rm d}R_k = (\gamma_k I_k - d_k^{\rm R} R_k){\rm d}t, \end{cases} \tag{2}$$

where

$$s_k(t) = A_k - d_k^s S_k - \sum_{j=1}^{n} \beta_{kj} S_k I_j,$$

$$i_k(t) = \sum_{j=1}^{n} \beta_{kj} S_k I_j - (d_k^{\rm I} + \epsilon_k + \gamma_k) I_k,$$

$$k = 1, 2, \cdots, n.$$

Adding the three equations in (2) gives

$$\begin{aligned} &{\rm d}(S_k + I_k + R_k) = \\ &(A_k - d_k^{\rm S} S_k - (d_k^{\rm I} + \epsilon_k) I_k - d_k^{\rm R} R_k){\rm d}t \leqslant \\ &(A_k - d_k^*(S_k + I_k + R_k)){\rm d}t, \end{aligned}$$

where

$$d_k^* = \min\{d_k^{\rm S}, d_k^{\rm I} + \epsilon_k, d_k^{\rm R}\}.$$

Hence we obtain

$$\limsup_{t\to\infty}(S_k + I_k + R_k) \leqslant \frac{A_k}{d_k^*}.$$

Moreover, there is only one equilibrium: the disease-free equilibrium $P_0 = (S_1^0, 0, 0, \cdots, S_n^0, 0, 0)$, where $S_k^0 = \dfrac{A_k}{d_k^{\rm S}}$, $k = 1, 2, \cdots, n$. The main results of this paper is defined the sharp threshold $R_0^{\rm S}$ which determines the dynamic behaviour of our stochastic multi-group SIR model (2). Here $R_0^{\rm S} = \rho(M_0^{\rm S})$ denotes the spectral radius of the matrix

$$M_0^{\rm S} = \left(\frac{\beta_{kj} S_k^0}{d_i^{\rm I} + \epsilon_k + \gamma_k + \dfrac{(S_k^0 \sigma_k)^2}{2}} \right)_{1 \leqslant k,j \leqslant n}.$$

In Section 2, we prove the global existence of the positive solution, then we derive the stability of the disease-free equilibrium, a new threshold $R_0^{\rm S}$ different from the deterministic R_0 is found which determines the dynamics of the disease. The global stability of the disease-free equilibrium will be investigated in Section 4. At last we give some numerical examples, they will help us to illustrate our main results.

Throughout the article, unless otherwise specified, we will employ the following notions. Let $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geqslant 0}, \mathcal{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geqslant 0}$ satisfying the usual conditions (i.e., it is right continuous and $\mathcal{F}_0$ contains all P-null sets). We use $a \vee b$ to denote $\max(a, b)$, $a \wedge b$ to denote $\min(a, b)$ and a.s. to mean almost surely.

2 Sharp threshold theorem

As an epidemic model, we need to guarantee the existence and uniqueness of the positive global solution for the stochastic multigroup SIR epidemic model (2). The standard existence and uniqueness conditions is no longer viable as the functions of system (2) do not satisfy the linear growth condition, so the solution may explode at a finite time[23]. To solve this problem, we use the lyapunov analysis method to prove the global existence of the positive solution.

Theorem 1 If $B=(\beta_{ij})_{n\times n}$ is irreducible, then for any initial value of model (2), there exists a unique solution $x(t)=(S_1(t),I_1(t),R_1(t),\cdots,S_n(t),I_n(t),R_n(t))\in\mathbb{R}^{3n}_+$, and it satisfies

$$P\{x(t)|x(t)\in\mathbb{R}^{3n}_+\}=1.$$

Proof Since the coefficients of the equation are locally Lipschitz continuous, there is a unique local solution on $t\in[0,\tau_{\mathrm{e}})$, where τ_{e} is the explosion time (see Mao[23]). Considering the implicit solution of $S_k(t),I_k(t),R_k(t),\ k=1,2,\cdots,n$, the solutions are exponential functions, so we can conclude $S_k(t),I_k(t),R_k(t)$ are positive on $t\in[0,\tau_{\mathrm{e}})$. To prove the global existence of the solution we need to show that $\tau_{\mathrm{e}}=\infty$ almost surely. We choose a sufficiently large number m_0 such that $S(0),I(0),R(0)$ all lie in the interval $(0,m_0)$. For each integer $m>m_0$, we define the stopping time

$$\begin{aligned}\tau_m=\ &\inf\{t\in[0,\tau_{\mathrm{e}}):\\ &\max\{S_k(t)+I_k(t)+R_k(t)\}\geqslant m,\\ &k=1,2,\cdots,n\},\end{aligned}$$

where $\inf\varnothing=\infty$. Set $\tau_\infty=\lim\limits_{k\to\infty}\tau_k$ whence $\tau_\infty<\tau_{\mathrm{e}}$. If we can show that $\tau_\infty=\infty$ a.s., then we can conclude that $\tau_{\mathrm{e}}=\infty$ a.s. for all $t\geqslant 0$.

We use the method by contradiction, if this statement is false, then there are a pair of constants $T>0$ and $\varepsilon\in(0,1)$ such that $\mathrm{P}\{\tau_\infty\leqslant T\}>\varepsilon$, hence there is an integer $m_1\geqslant m_0$ such that $\mathrm{P}\{\tau_m\leqslant T\}\geqslant\varepsilon$ for all $m\geqslant m_1$.

Define a function $V(S_1,I_1,R_1,\cdots,S_n,I_n,R_n)=\sum\limits_{k=1}^{n}(S_k+I_k+R_k)$, using the Itô formula, for any $t\in[0,T]$ and $m\geqslant m_1$, we have

$$\begin{aligned}&\mathrm{E}V(S_1(t\wedge\tau_{\mathrm{m}}),I_1(t\wedge\tau_{\mathrm{m}}),R_1(t\wedge\tau_{\mathrm{m}}),\cdots,\\ &S_n(t\wedge\tau_{\mathrm{m}}),I_n(t\wedge\tau_{\mathrm{m}}),R_n(t\wedge\tau_{\mathrm{m}}))=\\ &V(S_1(0),I_1(0),R_1(0),\cdots,S_n(0),I_n(0),R_n(0))+\\ &\mathrm{E}\int_0^{t\wedge\tau_{\mathrm{m}}}\mathrm{L}V(S_1(s),I_1(s),R_1(s),\cdots,S_n(s),\\ &I_n(s),R_n(s))\mathrm{d}s,\end{aligned}$$

where the operator $\mathrm{L}V$ is defined by

$$\mathscr{L}V=\sum_{k=1}^{n}[A_k-d_k^{\mathrm{S}}S_k-(d_k^{\mathrm{I}}+\epsilon_k)I_k-d_k^{\mathrm{R}}R_k]\leqslant\sum_{k=1}^{n}A_k=A.$$

Therefore, if $t\leqslant T$, we have

$$\begin{aligned}&\mathrm{E}V(S_1(t\wedge\tau_{\mathrm{m}}),I_1(t\wedge\tau_{\mathrm{m}}),R_1(t\wedge\tau_{\mathrm{m}}),\cdots,\\ &S_n(t\wedge\tau_{\mathrm{m}}),I_n(t\wedge\tau_{\mathrm{m}}),R_n(t\wedge\tau_{\mathrm{m}}))=\\ &V(S_1(0),I_1(0),R_1(0),\cdots,\\ &S_n(0),I_n(0),R_n(0))+AT.\end{aligned}$$

Set $\Omega_{\mathrm{m}}=\{\tau_{\mathrm{m}}\leqslant T\}$, for $m\geqslant m_1$, then we know $P(\Omega_{\mathrm{m}})>0$. For every $\omega\in\Omega_{\mathrm{m}}$, $\max\{S_k(t)+I_k(t)+R_k(t)\geqslant m,\ k=1,2,\cdots,n\}$, hence

$$\begin{aligned}&V(S_1(0),I_1(0),R_1(0),\cdots,\\ &S_n(0),I_n(0),R_n(0))+AT\geqslant\\ &\mathrm{E}[\mathrm{I}_{\Omega_{\mathrm{m}}}(\omega)V(S_1(t\wedge\tau_{\mathrm{m}}),I_1(t\wedge\tau_{\mathrm{m}}),R_1(t\wedge\tau_{\mathrm{m}}),\cdots,\\ &S_n(t\wedge\tau_{\mathrm{m}}),I_n(t\wedge\tau_{\mathrm{m}}),R_n(t\wedge\tau_{\mathrm{m}}))]\geqslant\\ &\varepsilon\max\{S_k(t)+I_k(t)+R_k(t),k=1,2,\cdots,n\}\geqslant\\ &\varepsilon m,\end{aligned}$$

Letting $m\to\infty$ leads to the contradiction $\infty>V(S(0),I(0),R(0))+AT\geqslant\infty$, so we have $\tau_\infty=\infty$ a.s., whence the proof is completed.

Next, we will show the sharp threshold theorem for the multi-group SIR model (2). The main idea of finding the sharp threshold comes from the linearized system around the disease-free equilibrium.

Theorem 2 Assume that $B=(\beta_{ij})_{n\times n}$ is irreducible, $\sigma_kS_k^0=\sigma,\ k=1,2,\cdots,n$.

1) If $R_0^{\mathrm{S}}<1$, the disease-free equilibrium P_0 is almost sure asymptotically stable, which means the disease will die out almost surely.

2) If $R_0^{\mathrm{S}}>1$, the disease-free equilibrium P_0 is unstable.

Proof Considering the following linearized system of model (2) at the disease-free equilibrium:

$$\begin{cases}\mathrm{d}u_k=U_k(t)\mathrm{d}t-\sigma_kS_k^0v_k\mathrm{d}B(t),\\ \mathrm{d}v_k=V_k(t)\mathrm{d}t+\sigma_kS_k^0v_k\mathrm{d}B(t),\\ \mathrm{d}w_k=(\gamma_kv_k-d_k^{\mathrm{R}}w_k)\mathrm{d}t,\end{cases}\tag{3}$$

where

$$U_k(t)=-d_k^{\mathrm{S}}u_k-\sum_{j=1}^{n}\beta_{kj}S_k^0v_j,$$

$$v_k(t)=\sum_{j=1}^{n}\beta_{kj}S_k^0v_j-(d_k^{\mathrm{I}}+\epsilon_k+\gamma_k)v_k.$$

Let $V(t)=(v_1(t),v_2(t),\cdots,v_n(t))$, we rewrite the second equation of model (3) as

$$\mathrm{d}V(t)=FV(t)\mathrm{d}t)+GV(t)\mathrm{d}B(t),\tag{4}$$

where

$$F=\begin{bmatrix}F_{11}&\cdots&\beta_{1n}S_1^0\\ \vdots&\ddots&\vdots\\ \beta_{nn}S_n^0&\cdots&F_{nn}\end{bmatrix},$$

$$G=\begin{bmatrix}\sigma_1 S_1^0 & & & \\ & \sigma_2 S_2^0 & & \\ & & \ddots & \\ & & & \sigma_n S_n^0\end{bmatrix},$$

where

$$F_{11}=\beta_{11}S_1^0-d_1^{\mathrm{I}}-\epsilon_1-\gamma_1,$$
$$F_{nn}=\beta_{n1}S_n^0-d_n^{\mathrm{I}}-\epsilon_n-\gamma_n.$$

Since $\sigma_k S_k^0=\sigma$, the matrices F and G commute, then the explicit solution of the linearized system (4) can be computed as

$$V(t)=V(0)\exp[(F-\frac{1}{2}G^2)t+GB(t)], \quad (5)$$

where

$$F-\frac{1}{2}G^2=\begin{bmatrix}Q_{11} & \cdots & Q_{1n}\\ \vdots & \ddots & \vdots \\ Q_{n1} & \cdots & Q_{nn}\end{bmatrix},$$
$$Q_{11}=\beta_{11}S_1^0-d_1^{\mathrm{I}}-\epsilon_1-\gamma_1-\frac{(S_1^0\sigma_1)^2}{2},$$
$$Q_{1n}=\beta_{1n}S_1^0,$$
$$Q_{n1}=\beta_{n1}S_n^0,$$
$$Q_{nn}=\beta_{nn}S_n^0-d_n^{\mathrm{I}}-\epsilon_n-\gamma_n-\frac{(S_n^0\sigma_n)^2}{2}.$$

According to the definition of R_0^{S}, if $R_0^{\mathrm{S}}<1$, all the eigenvalues of $F-\frac{1}{2}G^2$ have negative real parts. Then there are a pair of positive constants C and λ such that

$$\|\exp[(F-\frac{1}{2}G^2)t]\|\leqslant C\mathrm{e}^{-\lambda t}.$$

It then follows from (5) that

$$|V(t)|\leqslant C|V(0)|\exp\left[-\lambda t+\|G\||B(t)|\right]. \quad (6)$$

Using the strong law of large numbers states that $\lim\limits_{t\to\infty}\dfrac{B(t)}{t}=0$ a.s. we obtain

$$\limsup_{t\to\infty}\frac{1}{t}\log|V(t)|\leqslant-\lambda \quad \text{a.s..}$$

In other words, the trivial solution of equation (4) is almost surely exponentially stable. Next, we give estimate for $u_k(t)$ and $w_k(t)$. Using the Itô formula, we derive that

$$w_k(t)=\mathrm{e}^{-d_k^{\mathrm{R}}t}\big[R_k(0)+\int_0^t\gamma_k v_k\mathrm{e}^{d_k^{\mathrm{R}}u}\mathrm{d}u\big]. \quad (7)$$

Substituting (6) into (7) we get

$$w_k(t)\leqslant$$
$$\mathrm{e}^{-d_k^{\mathrm{R}}t}[R_k(0)+\int_0^t\gamma_k\mathrm{e}^{(d_k^{\mathrm{R}}-\lambda)u}\mathrm{d}u]=$$
$$R_k(0)\mathrm{e}^{-d_k^{\mathrm{R}}t}+\frac{\gamma_k}{d_k^R+-\lambda}\mathrm{e}^{-\lambda t}-\frac{\gamma_k}{d_k^R+-\lambda}\mathrm{e}^{-d_k^{\mathrm{R}}t},$$

therefore

$$\limsup_{t\to\infty}\frac{1}{t}\log|w_k(t)|=-d_k^{\mathrm{R}}\vee-\lambda<0.$$

Similarly we gain the assertion for $u_k(t)$ that

$$\limsup_{t\to\infty}\frac{1}{t}\log|u_k(t)|=-\lambda\vee-d_k^{\mathrm{R}}\vee-d_k^{\mathrm{S}}<0.$$

In this way we proved that (3) is exponentially stable. According to the Oseledec multiplicative ergodic theorem[11], the necessary and sufficient condition for the almost sure asymptotic stability of the trivial solution is the largest lyapunov exponent of the linearized system is negative.

Remark 1 It is useful to observe that either for the classical deterministic model or the stochastic model, there is a threshold which reflects the prevalent or extinction of the epidemic, but the thresholds are different between them, the stochastic threshold R_0^{S} is smaller then the deterministic one. In other words, the conditions for $I(t)$ to become extinct in the SDE epidemic model are weaker than those in the classical deterministic epidemic model, which gives a new method on disease control: stochastic stabilization.

Actually, stochastic epidemic models have been studied by many authors[9−32]. Most of the results concentrate on the sufficient conditions of the stability using Lyapunov function method for the stochastic models, and they establish the results based on the corresponding deterministic model's R_0. For stochastic multigroup epidemic model, Yang et al.[23] considered a multi-group SEIR epidemic model, they studied the extinction and recurrence of the model based on the related deterministic model's reproduction number R_0. Ji et al.[29−30] discussed stochastic multigroup SIR models, they investigated the model's asymptotic behavior by using Lyapunov function. Their results also based on the related deterministic model's reproduction number R_0. There are few references investigate the stochastic model's own threshold R_0^{S}. Gray et al.[18] established a stochastic SIS model and found out the sufficient and necessary condition for the disease-free equilibrium and the condition of the persistence of the disease. Jiang et al.[15,20] studied a stochastic SIS model and a stochastic SIR model, they found out the threshold of the model under small stochastic perturbation. Zhang et al.[24] studied a stochastic predator-prey model, they discussed the asymptotical behaviour of the model by using Lyapunov analysis models. For our results, we give a method to calculate stochastic epidemic model's sharp threshold(the basic reproduction number).

3 Stability of equilibria and global dynamics

Using the linearized system (3), we have found out the sharp threshold of model (2), and have given the local stability results for the disease-free equilibrium. Next, we will investigate the global dynamics of our stochastic model (2).

Theorem 3 Assume $B=(\beta_{ij})$ is irreducible, and $\sigma_k^2\leqslant\dfrac{d_k^{\mathrm{S}}\beta_{kk}}{A_k}$. If $R_0^{\mathrm{S}}<1$, then the disease-free

equilibrium $P_0 = (S_1^0, 0, 0, \cdots, S_n^0, 0, 0)$ is asymptotic stable in the large.

Proof We assume $S = (S_1, S_2, \cdots, S_n)$, $S^0 = (S_1^0, S_2^0, \cdots, S_n^0)$ and

$$M(S) = \left(\frac{\beta_{kj} S}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S)^2}{2}} \right)_{1 \leqslant k,\, j \leqslant n},$$

then $R_0^{\mathrm{S}} = \rho(M(S^0))$.

Let $(\omega_1, \omega_2, \cdots, \omega_n)$ is the eigenvector of $M(S^0)$ corresponding to $\rho(M(S^0))$, then

$$(\omega_1, \omega_2, \cdots, \omega_n)\rho(M(S^0)) = (\omega_1, \omega_2, \cdots, \omega_n)M(S^0),$$

where $\omega_k > 0$, $k = 1, 2, \cdots, n$. Since $B = (\beta_{ij})$ is irreducible，then we have $M(S)$ and M_0 are also irreducible. Considering the Lyapunov function

$$V(I_1, I_2, \cdots, I_n) = \sum_{k=1}^{n} \frac{\omega_k}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \frac{1}{2}(\sigma_k S_k^0)^2} I_k^a,$$

where $a < 1$ is a constant which will be determined later. Using Itô formula we obtain

$$\begin{aligned}
&\mathscr{L}V = \\
&\sum_{k=1}^{n} \frac{\omega_k}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2}} a (I_k)^{a-1} \\
&[\sum_{j=1}^{n} \beta_{kj} S_k I_j - (d_k^{\mathrm{I}} + \epsilon_k + \gamma_k) I_k] + \\
&\sum_{k=1}^{n} \frac{\omega_k}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2}} \frac{1}{2} a(a-1) I_k^{a-2} \sigma_k^2 S_k^2 I_k^2 = \\
&\sum_{k=1}^{n} \frac{a I_k^{a-1} \omega_k}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2}} \times \\
&[\sum_{j=1}^{n} \beta_{kj} S_k I_j - (d_k^{\mathrm{I}} + \epsilon_k + \gamma_k) I_k + \frac{1}{2}(a-1)\sigma_k^2 S_k^2 I_k] = \\
&\sum_{k=1}^{n} \frac{a I_k^{a-1} \omega_k}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2}} \times \\
&[\sum_{j=1}^{n} \beta_{kj} S_k I_j - (d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \\
&\frac{1}{2}\sigma_k^2 S_k^2) I_k + \frac{1}{2} a \sigma_k^2 S_k^2 I_k].
\end{aligned}$$

As $S_k + I_k + R_k \leqslant \dfrac{A_k}{d_k}$ and $S_k, I_k, R_k > 0$ for $k = 1, 2, \cdots, n$, we have $I_k \leqslant \dfrac{A_k}{d_k}$. Let $\dfrac{A}{d} = \max\{\dfrac{A_k}{d_k}, k = 1, 2, \cdots, n\}$, then for every $k = 1, 2, \cdots, n$, we have $I_k \leqslant \dfrac{A}{d}$. The above infinitesimal operator can be rewritten as

$$\begin{aligned}
&\mathscr{L}V \leqslant \\
&\frac{a A^{a-1}}{d^{a-1}} \sum_{k=1}^{n} \frac{\omega_k}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2}} [\sum_{j=1}^{n} \beta_{kj} S_k I_j - \\
&(d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \frac{1}{2}\sigma_k^2 S_k^2) I_k] + \\
&\frac{a A^{a-1}}{d^{a-1}} \sum_{k=1}^{n} \frac{\omega_k}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2}} \frac{1}{2} a \sigma_k^2 S_k^2 I_k.
\end{aligned}$$

According to the condition $\sigma_k^2 \leqslant \dfrac{d_k^{\mathrm{S}} \beta_{kk}}{A_k}$, we obtain

$$\begin{aligned}
&\sum_{j=1}^{n} \beta_{kj} S_k I_j - \frac{1}{2}\sigma_k^2 S_k^2 I_k + \frac{1}{2} a \sigma_k^2 S_k^2 I_k \leqslant \\
&\sum_{j=1}^{n} \beta_{kj} S_k^0 I_j - \frac{1}{2}\sigma_k^2 (S_k^0)^2 I_k + \frac{1}{2} a \sigma_k^2 (S_k^0)^2 I_k,
\end{aligned}$$

it then leads to

$$\begin{aligned}
&\mathscr{L}V \leqslant \\
&\frac{a A^{a-1}}{d^{a-1}} \sum_{k=1}^{n} \frac{\omega_k}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2}} \times \\
&[\sum_{j=1}^{n} \beta_{kj} S_k^0 I_j - (d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \frac{1}{2}\sigma_k^2 (S_k^0)^2) I_k] + \\
&\frac{a A^{a-1}}{d^{a-1}} \sum_{k=1}^{n} \frac{\omega_k}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2}} \frac{1}{2} a \sigma_k^2 (S_k^0)^2 I_k = \\
&\frac{a A^{a-1}}{d^{a-1}} \sum_{k=1}^{n} \omega_k [\sum_{j=1}^{n} \frac{\beta_{kj} S_k^0 I_j}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2}} - I_k] + \\
&\frac{a A^{a-1}}{d^{a-1}} \sum_{k=1}^{n} \frac{\omega_k}{2(d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2})} a \sigma_k^2 (S_k^0)^2 I_k.
\end{aligned}$$

Let $I = (I_1, I_2, \cdots, I_n)$, then

$$\begin{aligned}
&\mathscr{L}V \leqslant \\
&\frac{a A^{a-1}}{d^{a-1}} \sum_{k=1}^{n} \omega_k \cdot \\
&[\sum_{j=1}^{n} \frac{\beta_{kj} S_k^0 I_j}{d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2}} - I_k] + \\
&\frac{a A^{a-1}}{d^{a-1}} \sum_{k=1}^{n} \frac{\omega_k}{2(d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2})} a \sigma_k^2 (S_k^0)^2 I_k = \\
&\frac{a A^{a-1}}{d^{a-1}} (\omega_1, \omega_2, \cdots, \omega_n)[M(S^0) I - I] + \\
&\frac{a A^{a-1}}{d^{a-1}} \sum_{k=1}^{n} \frac{\omega_k}{2(d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2})} a \sigma_k^2 (S_k^0)^2 I_k = \\
&[\rho(M(S^0)) - 1](\omega_1, \omega_2, \cdots, \omega_n) I + \\
&\frac{a A^{a-1}}{d^{a-1}} \sum_{k=1}^{n} \frac{\omega_k}{2(d_k^{\mathrm{I}} + \epsilon_k + \gamma_k + \dfrac{(\sigma_k S_k^0)^2}{2})} a \sigma_k^2 (S_k^0)^2 I_k.
\end{aligned}$$

Let a is a small enough positive constant, then we finally obtain

$$\mathscr{L}V \leqslant [\rho(M(S^0)) - 1](\omega_1, \omega_2, \cdots, \omega_n)I,$$

which means

$$\lim_{t\to\infty} I(t) = 0 \text{ a.s..}$$

So $I_k(t)$ tends to zero in the large for $k = 1, 2, \cdots, n$. Next we need to prove $\lim\limits_{t\to\infty} S_k(t) = 0$ and $\lim\limits_{t\to\infty} R_k(t) = 0$. As $\lim\limits_{t\to\infty} I_k(t) = 0$ a.s., so for every $\varepsilon > 0$, there exists $T > 0$, such that $t > T$, then

$$I_k(t) \leqslant \varepsilon.$$

From the third equation in model (2), the solution of $R_k(t)$ can be solved as

$$R_k(t) = \mathrm{e}^{-d_k^R t}[R_k(0) + \int_0^t \mathrm{e}^{d_k^R s}\gamma_k I_k(s)\mathrm{d}s].$$

By applying the estimation for $I(t)$,

$$\begin{aligned}
&R_k(t) = \\
&\mathrm{e}^{-d_k^R t}[R_k(0) + \int_0^t \mathrm{e}^{d_k^R s}\gamma_k I_k(s)\mathrm{d}s] = \\
&\mathrm{e}^{-d_k^R t}[R_k(0) + \int_0^T \mathrm{e}^{d_k^R s}\gamma_k I_k(s)\mathrm{d}s + \\
&\int_T^t \mathrm{e}^{d_k^R s}\gamma_k I_k(s)\mathrm{d}s] \leqslant \\
&\mathrm{e}^{-d_k^R t}[R_k(0) + \int_0^T \mathrm{e}^{d_k^R s}\gamma_k I_k(s)\mathrm{d}s] + \\
&\gamma_k\varepsilon\mathrm{e}^{-d_k^R t}\int_T^t \mathrm{e}^{d_k^R s}\mathrm{d}s = \\
&\mathrm{e}^{-d_k^R t}[R_k(0) + \int_0^T \mathrm{e}^{d_k^R s}\gamma_k I_k(s)\mathrm{d}s] + \\
&\frac{\gamma_k\varepsilon}{d_k^R}(1 - \mathrm{e}^{d_k^R T - d_k^R t}),
\end{aligned}$$

so we can conclude that $\lim\limits_{t\to\infty} R_k(t) = 0$. Using the same method we can obtain $\lim\limits_{t\to\infty} S_k(t) = 0$. In this way, we proved that $P_0 = (S_1^0, 0, 0, \cdots, S_n^0, 0, 0)$ is asymptotic stable in the large.

Remark 2 In Theorem 3.2, we give the global stability of the disease-free equilibrium. It is worth mentioning that based on a group-theoretic approach to the method of global Lyapunov function, we separate the interaction terms in the stochastic model (2). Then the multigroup model's disease terms $I_i(t)$ can be considered separately. Using the classical methods by calculating the lyapunov exponents like references[25,27,30] we obtain the global stability of the disease-free equilibrium.

4 Numerical simulation

Consider the system (2), when $k = 2$, it becomes

$$\begin{cases}
\mathrm{d}S_1 = f_{11}(t)\mathrm{d}t - \sigma_1 S_1 I_1 \mathrm{d}B(t), \\
\mathrm{d}I_1 = f_{12}(t)\mathrm{d}t + \sigma_1 S_1 I_1 \mathrm{d}B(t), \\
\mathrm{d}R_1 = f_{13}(t)\mathrm{d}t, \\
\mathrm{d}S_2 = f_{21}(t)\mathrm{d}t - \sigma_2 S_2 I_2 \mathrm{d}B(t), \\
\mathrm{d}I_2 = f_{22}(t)\mathrm{d}t + \sigma_2 S_2 I_2 \mathrm{d}B(t), \\
\mathrm{d}R_2 = f_{23}(t)\mathrm{d}t,
\end{cases} \tag{8}$$

where

$$\begin{aligned}
f_{11}(t) &= A_1 - d_1^{\mathrm{S}} S_1 - \beta_{11} S_1 I_1 - \beta_{12} S_1 I_2, \\
f_{12}(t) &= \beta_{11} S_1 I_1 + \beta_{12} S_1 I_2 - (d_1^{\mathrm{I}} + \epsilon_1 + \gamma_1) I_1, \\
f_{13}(t) &= \gamma_1 I_1 - d_1^{\mathrm{R}} R_1, \\
f_{21}(t) &= A_2 - d_2^{\mathrm{S}} S_1 - \beta_{21} S_2 I_1 - \beta_{22} S_2 I_2, \\
f_{22}(t) &= \beta_{21} S_2 I_1 + \beta_{22} S_2 I_2 - (\epsilon_2 + d_2^{\mathrm{I}} + \gamma_2) I_2, \\
f_{23}(t) &= \gamma_2 I_2 - d_2^{\mathrm{R}} R_2.
\end{aligned}$$

we use the parameter values as follows:

$$\begin{aligned}
&A_1 = 100,\ A_2 = 300,\ d_1^{\mathrm{S}} = 2,\ d_2^{\mathrm{S}} = 3, \\
&d_1^{\mathrm{I}} = 2,\ d_2^{\mathrm{I}} = 5,\ d_1^{\mathrm{R}} = 3,\ d_2^{\mathrm{R}} = 5, \\
&\beta_{11} = 0.1,\ \beta_{12} = 0.2,\ \beta_{21} = 0.1,\ \beta_{22} = 0.1, \\
&\epsilon_1 = 1,\ \epsilon_2 = 1,\ \gamma_1 = 1,\ \gamma_2 = 1.
\end{aligned}$$

Firstly, the matrix B is irreducible. For the deterministic model (1), It's easy to calculate

$$\begin{aligned}
&R_0 = \rho(M_0) = \\
&\rho(\begin{bmatrix} 1.25 & 2.5 \\ 1.4286 & 0.4762 \end{bmatrix}) = 2.7921 > 1,
\end{aligned}$$

which means that the disease will persistent. For our stochastic model (2), our main interest will be focused on the extinction of the disease: $I_t(1), I_2(t)$. Let $\sigma_1 = \sigma_2 = 0.1$, we have

$$\begin{aligned}
&R_0^{\mathrm{S}} = \rho(M_0) = \\
&\rho(\begin{bmatrix} 0.3030 & 0.6061 \\ 0.1754 & 0.0585 \end{bmatrix}) = 0.529 < 1.
\end{aligned}$$

According to Theorem 2, the disease-free equilibrium is stable. We try the initial value $I_1(0) = 1, I_2(0) = 1$ which is near the disease-free equilibrium. By our condition, the sample pathes of $I_1(t), I_2(t)$ for the stochastic model (8) go to zero while the corresponding deterministic model's $I_1(t), I_2(t)$ go to the endemic equilibrium. The numerical simulations in Fig.1 supports these results clearly.

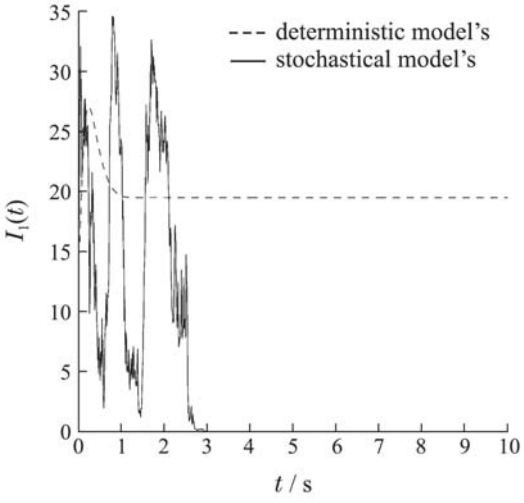


Fig. 1 Computer simulation of paths $I_1(t)$ for the system (8) and its corresponding deterministic model

The condition $\sigma_1^2 = 0.01 < S_1^0\beta_{11} = 5$ is satisfied, so the disease will extinction globally by Theorem 2.3. We keep the parameter value and start our computer simulation at the initial value $I_1(0) = 10, I_2(0) = 20$, Fig.2 shows that $I_1(t), I_2(t)$ also tend to zero.

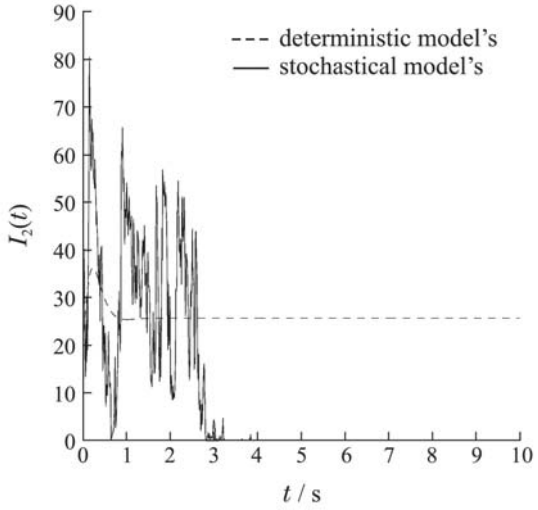


Fig. 2 Computer simulation of paths $I_1(t)$ for the system (8) and its corresponding deterministic model

Fig.1 and Fig.2 just give the sample path of $I_1(t)$, $I_2(t)$ separately. The sample path will change through every simulation as our stochastic model's solution is a stochastic process. To illustrate the extinction of the disease clearly, we try the statistic data for $I_1(t)$ and $I_2(t)$ which would be considered as the distribution of the solution. In Fig.3, we start our simulation at $I_1(0) = 10, I_2(0) = 20$ and run the simulation 10000 times to get the value of $I_1(5), I_2(5)$, $I_1(10), I_2(10)$ and $I_1(20), I_2(20)$.

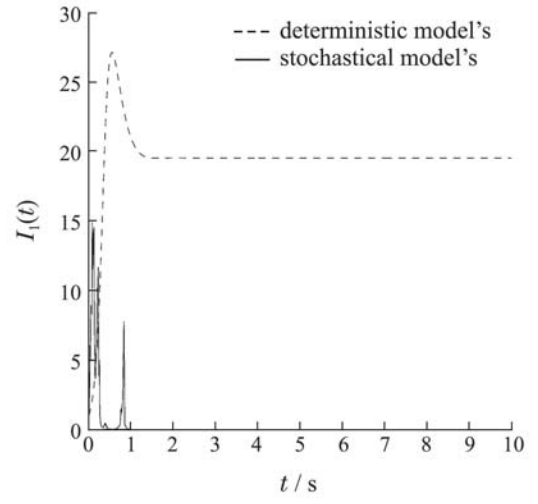


Fig. 3 Computer simulation of paths $I_1(t)$ for the system (8) and its corresponding deterministic model

Moreover, we give the related point set of $(I_1(20), I_2(20))$ in Fig.4. It is clearly that all most every goes to zero.

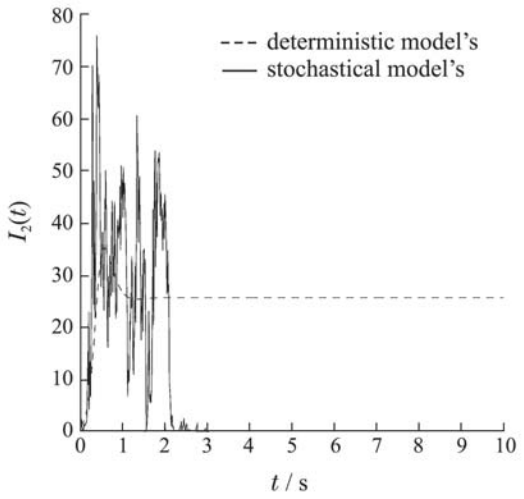


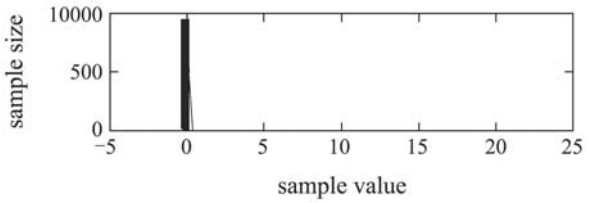
Fig. 4 Computer simulation of paths $I_2(t)$ for the system (8) and its corresponding deterministic model

From Fig.3 and Fig.4 we can conclude that the disease will extinct through the perturb intensity $\sigma_1 = 0.1$, $\sigma_2 = 0.1$, even though the corresponding deterministic model tends to be persistent. Adding stochastic perturbation can be considered as a method of stabilization on disease control.

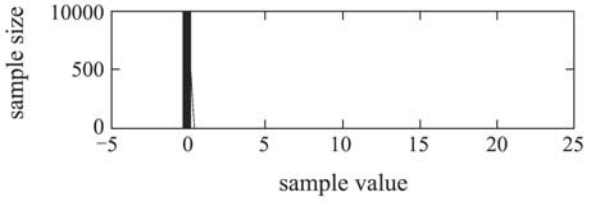
Next, we choose $\sigma_1 = 0.2, \sigma_2 = 0$, then R_0 keeps the same, but

$$R_0^{\mathrm{S}} = \rho(M_0) = \rho(\begin{bmatrix} 0.3030 & 0.6061 \\ 1.4286 & 0.4762 \end{bmatrix}) = 1.3241 > 1.$$

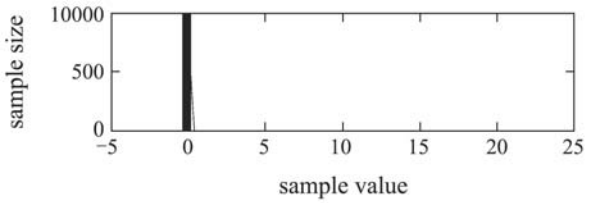
From Theorem 2, the disease will not extinct. Fig.5 illustrates this phenomenon clearly.



(a) The density function $I_1(5)$

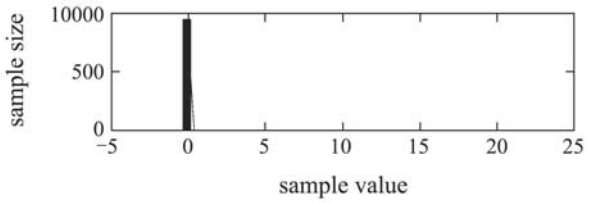


(b) The density function $I_1(10)$

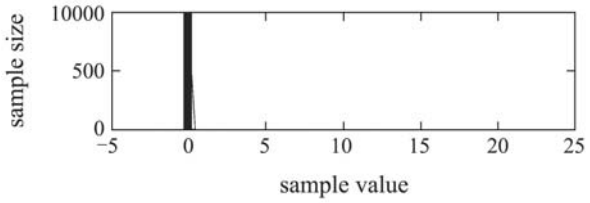


(c) The density function $I_1(20)$

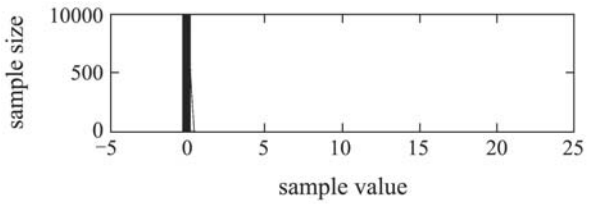
Fig. 5 The density function of $I_1(t)$



(a) The density function $I_1(5)$



(b) The density function $I_1(10)$



(c) The density function $I_1(20)$

Fig. 6 The density function of $I_2(t)$

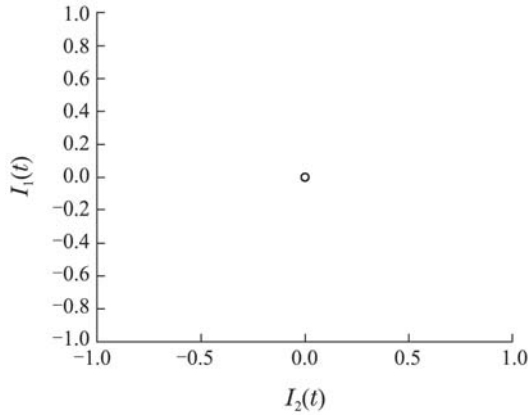


Fig. 7 The point set of $I_1(t)$ and $I_2(t)$ at $t = 20$

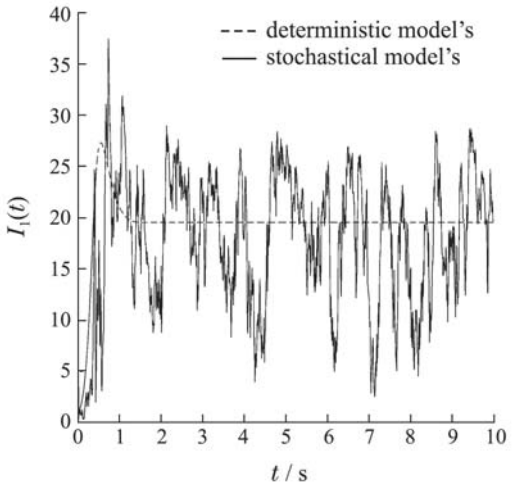


Fig. 8 computer simulation of paths $I_1(t)$ for the system (8)

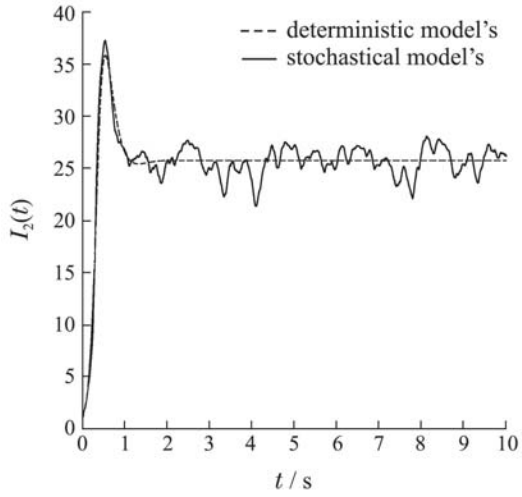


Fig. 9 computer simulation of paths $I_2(t)$ for the system (8)

5 Conclusions

In this paper, we established a new type stochastic multi-group SIR model driving by Ito process. Different from the traditional method, we define the basic reproduction number $R_0^{\rm S}$ which determines the extinction of the disease. We need to point out that the basic reproduction number relates to the stochastic magnitude σ. Moreover, we generalize the graph-theoretic method to obtain the global stability of the disease-free equilibria. Of cause, our theoretical results can be realize in numerical simulation.

References:

[1] LAVISH A, YORKORK J A. A deterministic model for gonorrhea in a nonhomogeneous population [J]. *Mathematical Biosciences*, 1976, 28(3/4): 221 – 236.

[2] BERETTA E, CAPASSO V. *Global Stability Results for a Multigroup SIR Epidemic Model* [M]. Singapore: World Scientific, 1986: 317 – 342.

[3] KERMACK W, MCKENDRICK A G. Contributions to the mathmatical theory of epidemics (part 1) [J]. *Bulletin of Mathematical Biology*, 1991, 53(1/2): 33 – 55.

[4] SONG B, CASTILLO C C, APARICIO J P. Tuberculosis models with fast and slow dynamics: the role of close and casual contacts [J]. *Mathematical Biosciences*, 2002, 180(1/2): 187 – 205.

[5] WEN L, YANG X. Global stability of a delayed SIRS model with temporary immunity [J]. *Chaos, Solitons & Fractals*, 2008, 38(1): 221 – 226.

[6] HSU S B, ROEGER L W. The final size of a SARS epidemic model without quarantine [J]. *Journal of Mathematical Analysis and Applications*, 2007, 333(2): 557 – 566.

[7] SUN R Y. Global stability of the endemic equilibrium of multigroup SIR models with nonlinear incidence [J]. *Computers & Mathematics with Applications*, 2010, 60(8): 2286 – 2291.

[8] LAUKO I G. Stability of disease free sets in epidemic models [J]. *Mathematical and Computer Modeling*, 2006, 43(11/12): 1357 – 1366.

[9] LI M Y, SHUAI Z S. Global-stability problem for coupled systems of differential equations on networks [J]. *Journal of Differential Equations*, 2010, 248(1): 1 – 20.

[10] ALLEN E. *Modeling with Itô Stochastic Differential Equations* [M]. Springer, 2005.

[11] OSELEDEC V I. A multiplication ergodic theorem. Lyapunov characteristic numbers for dynamicc system [J]. *Transaction of the Moscow Mathematical Society*, 1968, 19(2): 197 – 231.

[12] LIU J, ZHOU Y. Global stability of an SIRS epidemic model with transport-related infection [J]. *Chaos Solitons & Fractals*, 2009, 40(1): 145 – 158.

[13] MCCLUSKEY C C. Complete global stability for an SIR epidemic model with delay - distributed or discrete [J]. *Nonlinear Analysis: Real World Applications*, 2010, 11(1): 55 – 59.

[14] JIANG D Q, SHI N Z, LI X Y. Global stability and stochastic permanence of a non-autonomous logistic equation with random perturbation [J]. *Journal of Mathematical Analysis and Applications*, 2008, 303(1): 164 – 172.

[15] ZHAO Y N, JIANG D Q, DONAL O R. The extinction and persistence of the stochastic SIS epidemic model with vaccination [J]. *Physica A*, 2013, 392(20): 4916 – 4927.

[16] TORNATORRE E, BUCCELLATO S M, VETRO P. Stability of a stochastic SIR system [J]. *Physica A*, 2005, 354(1): 111 – 126.

[17] CARLETTI M. On the stability properties of a stochastic model for phage-bacteria interaction in open matine environment [J]. *Mathematical Bioscience*, 2002, 175(2): 117 – 131.

[18] GRAY A, GREENHALGH D, HU L, et al. A stochastic differential equation SIS epidemic model [J]. *SIAM Journal of Applied Mathematics*, 2011, 71(3): 876 – 902.

[19] DALAL N, GREENHALGH D, MAO X R. A stochastic model of AIDS and condom use [J]. *Journal of Mathematical Analysis and Applications*, 2007, 325(1): 36 – 53.

[20] JI C Y, JIANG D Q, SHI N Z. The behavior of an SIR epidemic model with stochastic perturbation [J]. *Stochastic Analysis and Application*, 2012, 30(5): 755 – 773.

[21] YU J J, JIANG D Q, SHI N Z. Global stability of two-group SIR model with random perturbation [J]. *Journal of Mathematical Analysis and Applications*, 2009, 360(2): 235 – 244.

[22] LIU H, YANG Q S, JIANG D Q. The asymptotic behavior of stochastically perturbed DI SIR epidemic models with saturated incidences [J]. *Automatica*, 2012, 48(5): 820 – 825.

[23] YANG Q S, MAO X R. Extinction and recurrence of multi-group SEIR epidemic models with stochastioc perturbations [J]. *Nonlinear Anylysis: Real World Applications*, 2013, 14(3): 1434 – 1456.

[24] ZHANG Q, JIANG D Q, LIU Z W, et al. The long time behavior of a predator-prey model with disease in the prey by stochastic perturbation [J]. *Applied Mathematical Letteres*, 2014, 245(15): 305 – 320.

[25] ZHAO Y N, JIANG D Q. The threshold of a stochastic SIS epidemic model with vaccination [J]. *Applied Mathematics and Computation*, 2014, 243(1): 718 – 727.

[26] LIU Q, CHEN Q M. Analysis of the deterministic and stochastic SIRS epidemic models with nonlinear incidence [J]. *Physica A*, 2015, 428(1): 140 – 153.

[27] LIN Y G, JIANG D Q, LIU T H. Nontrivial periodic solution of a stochastic epidemic model with seasonal variation [J]. *Applied Mathematics Letters*, 2015, 45(1): 103 – 107.

[28] JI C Y, JIANG D Q. Threshold behaviour of a stochastic SIR model [J]. *Applied Mathematical Modeling*, 2014, 38(21/22): 5067 – 5079.

[29] JI C L, JIANG D Q, YANG Q S, et al. Dynamics of a multigroup SIR epidemic model with stochastic perturbation [J]. *Automatica*, 2012, 48(1): 121 – 131.

[30] JI C L, JIANG D Q, YANG Q S, et al. Multigroup SIR epidemic model with stochastic perturbation [J]. *Physica A*, 2011, 390(10): 1747 – 1762.

[31] ZHU C, YIN G. Asymptotic properties of hybrid diffusion systems [J]. *SIAM Journal of Control Optimations*, 2007, 49(4): 1155 – 1179.

[32] LIU M, WANG K, WU Q. Survival analysis of stochastic competitive models in a polluted environment and stochastic competitive exclusion principle [J]. *Bulletin of Mathematical Biology*, 2011, 73(1): 1969 – 2012.

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