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Analysis of a Chlamydia epidemic model with pulse vaccination strategy in a random environment

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Abstract. In this paper, we have considered a dynamical model of Chlamydia disease with varying total population size, bilinear incidence rate, and pulse vaccination strategy in a random environment. It has been shown that the Chlamydia epidemic model has global positive solutions and, under some conditions, it admits a unique positive periodic disease-free solution, which is globally exponentially stable in mean square. We have defined two positive numbers R_1 and R_2 ($< R_1$). It is proved that the susceptible population will be persistent in the mean and the disease will be going to extinct if $R_1 < 1$ and the susceptible population as well as the disease will be weakly persistent in the mean if $R_2 > 1$. Our analytical findings are explained through numerical simulation, which show the reliability of our model from the epidemiological point of view.

Keywords: Chlamydia trachomatis, pulse vaccination, white noise, persistent, extinction.

1 Introduction

Infectious diseases have tremendous influence on human life, and so the development of vaccines against infectious disease has been a boon to human being. Infectious diseases are usually caused by pathogenic microorganisms such as bacteria, viruses, parasites, or fungi. The diseases can be spread directly or indirectly. Controlling infectious diseases has been an increasingly complex and significant issue in recent years. Sexually transmitted infections (STIs) remain a major public health challenge globally and are among the most

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common infections in the United States. Chlamydia, caused by the bacterium Chlamydia trachomatis, is one of the most important sexually-transmitted infections spreading throughout the world.

Human immune system is an integrated collection of organs, special cells, and substances that help to protect from infections and some other diseases. Immune system cells and the substances they make travel through the body to protect it from pathogens (germs) causing infections. Pathogens are any disease-producing agent, especially a virus, bacterium, or other microorganism, which are also called foreign armies because they are not normally found in the body. They try to invade human body to use its resources to serve their own purposes, and so they can hurt the body in the process. The immune system is acting as body's defence force, which helps keep invading germs out or helps kill them if they do get into the body. It keeps track of all of the substances normally found in the body and any new substance in the body that the immune system does not recognize raises an alarm to attack it. Substances that cause an immune system response are called *antigens*. The immune response can lead to destruction of anything containing the antigen such as pathogens. Pathogens (viruses, bacteria, parasites) have substances on their outer surfaces, such as certain proteins, that are not normally found in the human body. The immune system recognizes these foreign substances as antigens [12].

Vaccine-induced immunity results after a vaccine is administered. The vaccine activates immune systems infection-attacking ability and memory without exposure to the actual disease-producing pathogens. A vaccine consists of a killed or weakened form or derivative of the infectious germ. When given to a healthy person, the vaccine activates an immune response and makes the body to think that it is being invaded by a specific organism. Then the immune system goes to work to kill the invader and prevent it from infection. If we are exposed to a disease for which we have been vaccinated, the invading germs are met by antibodies that will destroy them. The immunity developed through vaccination is similar to the immunity acquired from natural infection. Several doses of a vaccine may be needed for a full immune response. Some people may be unsuccessful to achieve full immunity to the first doses of a vaccine but respond to later doses. The immune response may decrease over time, one may require another dose of a vaccine (booster shot) to restore or increase immunity [12].

The pulse vaccination strategy (PVS) consists of repeated administration of vaccine at discrete time having equal interval in a population in contrast to the traditional constant vaccination [15]. Compared to the proportional vaccination models, the analysis of pulse vaccination models is in its infancy [15]. At each vaccination time, a constant fraction of the susceptible population is vaccinated successfully. Since 1993, attempts have been made to develop mathematical theory to control infectious diseases using pulse vaccination. Nokes and Swinton [9] analysed the control of childhood viral infections by implementing pulse vaccination strategy. Stone et al. [13] discussed a theoretical investigation of the pulse vaccination strategy in the SIR epidemic model, and d'Onofrio [3,4] analysed the use of pulse vaccination policy to eradicate infectious disease for SIR and SEIR epidemic models. Different types of vaccination policies and strategies combining pulse vaccination policy, treatment, pre-outbreak vaccination, or isolation have already been analysed by several researchers. Our real life is full of randomness, and so there has been

a growing interest in stochastic epidemic models. The introduction of a stochastic perturbation in epidemic models is justified by observation that the real life is full of social and environmental random variations. The presence of a stochastic noise in an epidemic model changes the behaviour of solution of correspondent deterministic model and modifies the thresholds of the system for an epidemic to occur, which can bring to light new insights.

In this work, we have used the Kermack–McKendrick compartmental modelling framework, which entails subdividing the entire high-risk human population into mutually-exclusive epidemiological compartments (based on disease status) to gain insights into the qualitative features of Chlamydia trachomatis in a human population (with the aim of finding effective ways to control its spread). The main feature of this paper is to introduce noise and valid pulse vaccination strategy, which greatly enriches biologic background. We have introduced two threshold values R_1 and R_2 ($< R_1$) and further obtained that the susceptible population will be persistent in the mean, and the disease will be going to extinct if $R_1 < 1$ and the susceptible as well as the disease will be weakly persistent in the mean if $R_2 > 1$. Here we have analysed whether PVS can also be administered to contrast Chlamydia diseases in a random environment, which might allow to apply PVS to fight in a realistic way. The important mathematical findings for the dynamical behaviour of the Chlamydia disease model are numerically verified using MATLAB, and also epidemiological implications of our analytical findings are addressed critically in Section 5. The aim of the analysis of this model is to trace the parameters of interest for further study with a view to informing and assisting policy-maker in targeting prevention and treatment resources for maximum effectiveness.

2 Model derivation and preliminaries

In the following, we consider a dynamical model of Chlamydia disease that spread by Chlamydia trachomatis (a type of bacteria) with pulse vaccination strategy (PVS) in a random environment. The aim of this work is to study whether PVS can also be administered to contrast Chlamydia disease in the random environment. Our Chlamydia epidemic model is based on the following assumptions:

The underlying high-risk human population is split up into four mutually-exclusive classes (compartments), namely, susceptible (S), infective (I), naturally recovered (infectious people who have cleared (or recovered from) Chlamydia infection naturally) (R), and vaccinated individuals (V). Here it is assumed that the recovered individuals acquire the permanent immunity but the vaccinated acquire temporary immunity. So, the natural immunity is permanent but the vaccine-induced immunity is temporary.

The susceptible population increases by the recruitment through new sexually-active individuals, migration, and the vaccinated individuals return to the susceptible class (due to immunity waning) and decreases due to direct contact with infected individuals, natural death, and pulse vaccination strategy.

Standard epidemiological models use a bilinear incidence rate βSI based on the law of mass action [1], and it is reasonable when the mixing of susceptible with infective is taken into account to be homogeneous.

The infected class is increased by infection of susceptible individuals. A fraction of infectious individuals recovers naturally and moves to the recovered compartment. The infected class is decreased through natural recovery from infection, by disease-related death and by natural death.

Thus, a dynamical model of Chlamydia disease that spread by Chlamydia trachomatis with bilinear incidence and pulse vaccination strategy in a random environment under the following stochastic perturbation and the pulse vaccination scheme is formulated:

$$
\begin{aligned}
\frac{\mathrm{d}S(t)}{\mathrm{d}t} &= \Lambda(t) - \beta(t)S(t)I(t) - \mu(t)S(t) + \alpha(t)V(t) \\
&\quad + \sqrt{\alpha(t)}\big\{S(t) - V(t)\big\}\frac{\mathrm{d}W_1}{\mathrm{d}t}, \quad t \neq nT, \\
\frac{\mathrm{d}I(t)}{\mathrm{d}t} &= \beta(t)S(t)I(t) - r(t)I(t) - \big(\mu(t) + d(t)\big)I(t) \\
&\quad + \sigma_2(t)I(t)\frac{\mathrm{d}W_2}{\mathrm{d}t}, \quad t \neq nT, \\
\frac{\mathrm{d}V(t)}{\mathrm{d}t} &= -\big(\mu(t) + \alpha(t)\big)V(t) + \sigma_3(t)V(t)\frac{\mathrm{d}W_3}{\mathrm{d}t}, \quad t \neq nT, \\
\frac{\mathrm{d}R(t)}{\mathrm{d}t} &= r(t)I(t) - \mu(t)R(t) + \sigma_4(t)R(t)\frac{\mathrm{d}W_4}{\mathrm{d}t}, \quad t \neq nT, \\
S\big(t^+\big) &= (1-p)S(t), \quad t = nT, n = 1, 2, \ldots, \\
I\big(t^+\big) &= I(t), \quad t = nT,\ n = 1, 2, \ldots, \\
V\big(t^+\big) &= V(t) + pS(t), \quad t = nT,\ n = 1, 2, \ldots, \\
R\big(t^+\big) &= R(t), \quad t = nT,\ n = 1, 2, \ldots,
\end{aligned}
\tag{1}
$$

where $\Lambda(t)$, $\beta(t)$, $\mu(t)$, $\alpha(t)$, $r(t)$, $d(t)$, and $\sigma_i(t)$ $(i = 2, 3, 4)$ are all positive T-periodic continuous functions, T is a positive constant; T is the period of pulse vaccination. Here $S(t)$ denotes the number of susceptible, $I(t)$ denotes the number of infective, $R(t)$ denotes the number of recovered individuals, and $V(t)$ denotes the number of vaccinated individuals. The pulse vaccination does not give life-long immunity, there is an immunity waning for the vaccination with the per capita immunity waning rate $\alpha(t)$, and return to the susceptible class. The influx of susceptible comes from two sources: a time dependent recruitment $\Lambda(t)$ and vaccinated hosts $\alpha(t)V(t)$. The interpretation of the functions $\beta(t)$, $\mu(t)$, $d(t)$, $r(t)$, and the constant p are as follows:

$\beta(t)$: The coefficient of transmission (Chlamydia infection) rate from infective to susceptible humans and the rate of transmission of infection is of the form $\beta(t)S(t)I(t)$.

$\mu(t)$: The coefficient of natural death rate of all epidemiological human classes.

$d(t)$: The coefficient of additional disease-related death rate of infective class.

$r(t)$: The rate at which the infectious individuals eventually recover naturally and move to the class R.

p $(0 < p < 1)$: The constant fraction of susceptible who are vaccinated successfully at discrete time $t = T, 2T, 3T, \ldots$, which is called impulsive vaccination rate.

In system (1), $\eta_i = \mathrm{d}W_i/\mathrm{d}t$ $(i = 1, 2, 3, 4)$ are independent standard zero mean Gaussian white noises characterized by

$$\big\langle \eta_i(t) \big\rangle = 0, \quad \big\langle \eta_i(t_1)\eta_i(t_2) \big\rangle = \delta(t_1 - t_2), \quad \text{and} \quad \big\langle \eta_i(t_1)\eta_j(t_2) \big\rangle = 0 \quad (i \neq j),$$

where $\langle\cdot\rangle$ represents the average over the ensemble of the stochastic process, and $\delta(t)$ denotes the Dirac delta function.

It is well known that Gaussian white noise, which is a delta-correlated random process, is very irregular and as such it is to be treated with care. In spite of this, it is an immensely useful concept to model rapidly fluctuating phenomenon. Of course, true white noise does not occur in nature. However, as can be seen by studying their spectra, thermal noise in electrical resistance, the force acting on a Brownian particle and climate fluctuations, disregarding the periodicities of astronomical origin, etc. are white to a very good approximation. These examples support the usefulness of the white noise idealization in applications to natural systems. Furthermore, it can be proved that a solution of (1) is Markovian if and only if the external noises are white. These results explain the importance and appeal of the white noise idealization [5, 10].

Let us first consider the following stochastic SIVR Chlamydia epidemic model:

$$\begin{aligned}
\frac{\mathrm{d}\widehat{S}(t)}{\mathrm{d}t} &= \Lambda(t) - \beta(t)\widehat{S}(t)\widehat{I}(t) - \mu(t)\widehat{S}(t) + \alpha(t)\widehat{V}(t) \\
&\quad + \sqrt{\alpha(t)}\big\{\widehat{S}(t) - \widehat{V}(t)\big\}\frac{\mathrm{d}W_1}{\mathrm{d}t}, \\
\frac{\mathrm{d}\widehat{I}(t)}{\mathrm{d}t} &= \beta(t)\widehat{S}(t)\widehat{I}(t) - \mu_1(t)\widehat{I}(t) + \sigma_2(t)\widehat{I}(t)\frac{\mathrm{d}W_2}{\mathrm{d}t}, \\
\frac{\mathrm{d}\widehat{V}(t)}{\mathrm{d}t} &= -\mu_2(t)\widehat{V}(t) + \sigma_3(t)\widehat{V}(t)\frac{\mathrm{d}W_3}{\mathrm{d}t}, \\
\frac{\mathrm{d}\widehat{R}(t)}{\mathrm{d}t} &= r(t)\widehat{I}(t) - \mu(t)\widehat{R}(t) + \sigma_4(t)\widehat{R}(t)\frac{\mathrm{d}W_4}{\mathrm{d}t},
\end{aligned} \tag{2}$$

where $\mu_1(t) = r(t) + \mu(t) + d(t)$ and $\mu_2(t) = \mu(t) + \alpha(t)$. In general, if the coefficients of a stochastic differential equation satisfy the linear growth condition and local Lipschitz condition, then it has a global (i.e. no explosion in a finite time) solution for any given initial value [7,14]. However, the coefficients of system (2) do not satisfy the linear growth condition, though they are locally Lipschitz continuous, and so the solution of (2) may explode at a finite time [7,14]. In this section, we shall show that the solution of system (2) with positive initial value is positive and global by using the Lyapunov analysis method (mentioned in [7]).

In the rest of the article, we adopt the following definitions.

Definition 1. If $f(t)$ is an integrable function on $[0, \infty)$, then

$$\big\langle f(t) \big\rangle = \frac{1}{t}\int_0^t f(s)\,\mathrm{d}s, \quad t > 0.$$

Definition 2. If $f(t)$ is a bounded function on $[0,\infty)$, then

$$f^u = \sup_{t\in\mathbb{R}_+} f(t) \quad \text{and} \quad f^l = \inf_{t\in\mathbb{R}_+} f(t).$$

Theorem 1. *For any initial value $(\widehat{S}(0),\widehat{I}(0),\widehat{V}(0),\widehat{R}(0))\in\mathbb{R}^4_+$, system (2) has a unique global solution $(\widehat{S}(t),\widehat{I}(t),\widehat{V}(t),\widehat{R}(t))$, which remains in $\mathbb{R}^4_+$ with probability one for all $t\geqslant 0$.*

Proof. Since the coefficients of the system are locally Lipschitz continuous for any given initial value $(S(0),I(0),V(0),R(0))\in\mathbb{R}^4_+$, there is a unique local solution $(\widehat{S}(t),\widehat{I}(t),\widehat{V}(t),\widehat{R}(t))$ on $t\in[0,T_e)$, where T_e is the explosion time [8]. To show that this solution is global, we need to show that $T_e=\infty$ a.s. Let us first prove that $\widehat{S}(t),\widehat{I}(t)$, and $\widehat{V}(t)$ do not explode to infinity in a finite time. For that purpose set $k_0>0$ be sufficiently large for $S(0),I(0),V(0)\in[1/k_0,k_0]$. For each integer $k\geqslant k_0$, define the stopping time:

$$T_k=\inf\left\{t\in[0,T_e)\colon \widehat{S}(t)\notin\left(\frac{1}{k},k\right) \text{ or } \widehat{I}(t)\notin\left(\frac{1}{k},k\right), \text{ or } \widehat{V}(t)\notin\left(\frac{1}{k},k\right)\right\},$$

where throughout this article, it is set $\inf\phi=\infty$, where ϕ represents the empty set. It is evident that T_k is increasing as $k\to\infty$. Set $T_\infty=\lim_{k\to\infty}T_k$, and so $T_\infty\leqslant T_e$ a.s. If $T_\infty=\infty$ a.s. is true, then $T_e=\infty$ a.s. and $(\widehat{S}(t),\widehat{I}(t),\widehat{V}(t))\in\mathbb{R}^3_+$ for all $t\geqslant 0$ a.s. In other words, to complete the proof it is required to show that $T_\infty=\infty$ a.s. If this statement is false, then there exist a pair of constants $\tau>0$ and $\epsilon\in(0,1)$ such that $\mathbf{P}\{T_\infty\leqslant\tau\}>\epsilon$. Therefore, there exists an integer $k_1\geqslant k_0$ such that

$$\mathbf{P}\{T_k\leqslant\tau\}\geqslant\epsilon \quad \forall k\geqslant k_1. \tag{3}$$

Define a C^3-function $G:\mathbb{R}^3_+\to\mathbb{R}_+$ as follows:

$$G(\widehat{S},\widehat{I},\widehat{V})=\left(\widehat{S}-a-a\ln\frac{\widehat{S}}{a}\right)+(\widehat{I}-1-\ln\widehat{I})+(\widehat{V}-1-\ln V),$$

where a is a positive constant to be defined later. The nonnegativity of G follows from the following inequality:

$$u-1-\ln u\geqslant 0 \quad \forall u>0.$$

Let $k\geqslant k_0$ and $\tau>0$ be arbitrary. Applying Itô's formula, we have

$$\begin{aligned}
\mathrm{d}G(\widehat{S},\widehat{I},\widehat{V}) &= \left\{\left(1-\frac{a}{\widehat{S}}\right)(\Lambda-\beta\widehat{S}\widehat{I}-\mu\widehat{S}+\alpha\widehat{V})\right\}\mathrm{d}t\\
&\quad+\left\{\left(1-\frac{1}{\widehat{I}}\right)(\beta\widehat{S}\widehat{I}-\mu_1\widehat{I})\right\}\mathrm{d}t\\
&\quad+\left\{-\left(1-\frac{1}{\widehat{V}}\right)\mu_2\widehat{V}+0.5\left(a\alpha\frac{(\widehat{S}-\widehat{V})^2}{\widehat{S}^2}+\sigma_2^2+\sigma_3^2\right)\right\}\mathrm{d}t\\
&\quad+\sqrt{\alpha}(\widehat{S}-a)\left(1-\frac{\widehat{V}}{\widehat{S}}\right)\mathrm{d}W_1+\sigma_2(\widehat{I}-1)\,\mathrm{d}W_2+\sigma_3(\widehat{V}-1)\,\mathrm{d}W_3
\end{aligned}$$

$$= LG(\widehat{S},\widehat{I},\widehat{V})\,\mathrm{d}t + \sqrt{\alpha}(\widehat{S}-a)\biggl(1-\frac{\widehat{V}}{\widehat{S}}\biggr)\,\mathrm{d}W_1$$
$$+ \sigma_2(\widehat{I}-1)\,\mathrm{d}W_2 + \sigma_3(\widehat{V}-1)\,\mathrm{d}W_3,$$

where $LG:\mathbb{R}^3_+\to\mathbb{R}_+$ is given by

$$LG(\widehat{S},\widehat{I},\widehat{V}) = \biggl\{(\Lambda + a\mu + \mu_1 + \mu_2) + 0.5\biggl(a\alpha\frac{(\widehat{S}-\widehat{V})^2}{\widehat{S}^2} + \sigma_2^2 + \sigma_3^2\biggr)\biggr\}$$
$$+ \bigl\{a\beta - (r+\mu+d)\bigr\}\widehat{I} - (\mu+\beta)\widehat{S} - \Lambda\frac{a}{\widehat{S}} - a\alpha\frac{\widehat{V}}{\widehat{S}}.$$

Let us choose $a = (r^l+\mu^l+d^l)/\beta^u$, which implies $a\beta(t)-(r(t)+\mu(t)+d(t))\leqslant 0$ for all $t\geqslant 0$. Then

$$LG(\widehat{S},\widehat{I},\widehat{V}) \leqslant \Lambda^u + a\mu^u + \mu_1^u + \mu_2^u + 0.5\bigl\{a\alpha^u + \bigl(\sigma_2^2\bigr)^u + \bigl(\sigma_3^2\bigr)^u\bigr\} = K \quad \text{(say)}.$$

Hence,

$$\mathrm{d}G(\widehat{S},\widehat{I},\widehat{V}) \leqslant K\,\mathrm{d}t + \sqrt{\alpha}(\widehat{S}-a)\biggl(1-\frac{\widehat{V}}{\widehat{S}}\biggr)\,\mathrm{d}W_1 + \sigma_2(\widehat{I}-1)\,\mathrm{d}W_2$$
$$+ \sigma_3(\widehat{V}-1)\,\mathrm{d}W_3. \tag{4}$$

Integrating both sides of (4) from 0 to $T_k\wedge\tau$ (where $T_k\wedge\tau = \min\{T_k,\tau\}$) and then taking the expectations, we get

$$\mathbf{E}\bigl\{G\bigl(\widehat{S}(T_k\wedge\tau),\widehat{I}(T_k\wedge\tau),\widehat{V}(T_k\wedge\tau)\bigr)\bigr\} \leqslant G\bigl(S(0),I(0),V(0)\bigr) + K\mathbf{E}(T_k\wedge\tau)$$
$$\Longrightarrow\quad \mathbf{E}\bigl\{G\bigl(\widehat{S}(T_k\wedge\tau),\widehat{I}(T_k\wedge\tau),\widehat{V}(T_k\wedge\tau)\bigr)\bigr\}$$
$$\leqslant G\bigl(S(0),I(0),V(0)\bigr) + KT. \tag{5}$$

Set $\Omega_k = \{T_k\leqslant\tau\}$ for all $k\geqslant k_1$, and so, by (3), $\mathbf{P}(\Omega_k)\geqslant\epsilon$. It is evident that for every $\omega\in\Omega_k$, there exists $\widehat{S}(T_k\wedge\tau,\omega)$ or $\widehat{I}(T_k\wedge\tau,\omega)$, or $\widehat{V}(T_k\wedge\tau,\omega)$ equals either k or $1/k$, and hence $G(\widehat{S}(T_k\wedge\tau),\widehat{I}(T_k\wedge\tau),\widehat{V}(T_k\wedge\tau))$ is no less than either $k-1-\ln k$ or $1/k-1-\ln(1/k) = 1/k-1+\ln k$. Consequently,

$$G\bigl(\widehat{S}(T_k\wedge\tau),\widehat{I}(T_k\wedge\tau),\widehat{V}(T_k\wedge\tau)\bigr) \geqslant (k-1-\ln k)\wedge\biggl(\frac{1}{k}-1+\ln k\biggr). \tag{6}$$

It then follows from (5) and (6) that

$$G\bigl(S(0),I(0),V(0)\bigr) \geqslant \mathbf{E}\bigl\{1_{\Omega_k}(\omega)G\bigl(\widehat{S}(T_k\wedge\tau),\widehat{I}(T_k\wedge\tau),\widehat{V}(T_k\wedge\tau)\bigr)\bigr\}$$
$$\geqslant \epsilon(k-1-\ln k)\wedge\biggl(\frac{1}{k}-1+\ln k\biggr), \tag{7}$$

where 1_{Ω_k} is the indicator function of Ω_k. Letting $k\to\infty$ and using (5)–(7), we get

$$+\infty > G\bigl(S(0),I(0),V(0)\bigr) + KT = \infty \quad \text{(a contradiction)}$$
$$\Longrightarrow\quad T_\infty = \infty \quad \text{a.s.} \tag{8}$$

Therefore, it implies that $\widehat{S}(t)$, $\widehat{I}(t)$, and $\widehat{V}(t)$ will not explode in a finite time with probability one.

Next, using the fourth equation of (2), we get

$$\widehat{R}(t) = \left[R(0) + \int_0^t r(s)\widehat{I}(s)\exp\left\{\int_0^s \big(\mu(\theta) + 0.5\sigma_4^2(\theta)\big)\,\mathrm{d}\theta - \int_0^s \sigma_4(\theta)\,\mathrm{d}W_4(\theta)\right\}\mathrm{d}s\right] \times \exp\left\{-\int_0^s \big(\mu(\theta) + 0.5\sigma_4^2(\theta)\big)\,\mathrm{d}\theta + \int_0^s \sigma_4(\theta)\,\mathrm{d}W_4(\theta)\right\},$$

where the integrations are taken in Stratonovich's sense.

Since $\widehat{S}(t)$, $\widehat{I}(t)$, and $\widehat{V}(t)$ have been proved to be global and positive, $\widehat{R}(t)$ is also global and positive. Hence the proof is completed. □

Theorem 2. *For any initial value $X_0 = (S(0), I(0), V(0), R(0)) \in \mathbb{R}^4_+$, system* (1) *has a unique global solution $X(t) = (S(t), I(t), V(t), R(t))$, which remains in $\mathbb{R}^4_+$ with probability one for all $t \geqslant 0$.*

Proof. By Theorem 1, for $t \in [0, T]$ and for any initial condition $X_0 \equiv (S(0), I(0), V(0), R(0)) \in \mathbb{R}^4_+$, system (2) has a unique global solution $\widehat{X}(t; 0, X_0) \in \mathbb{R}^4_+$ that is defined and continuous on interval $[0, T]$. Hence system (1) also has a unique global solution $X(t; 0, X_0) = \widehat{X}(t; 0, X_0) \in \mathbb{R}^4_+$ on interval $[0, T]$. At $t = T$, there is an impulse, which transfers solution $X(T) = \widehat{X}(T; 0, X_0) = (\widehat{S}(T), \widehat{I}(T), \widehat{V}(T), \widehat{R}(T)) \in \mathbb{R}^4_+$ into $X(T^+) = ((1-p)\widehat{S}(T), \widehat{I}(T), \widehat{V}(T) + p\widehat{S}(T), \widehat{R}(T)) \in \mathbb{R}^4_+$, where $0 < p < 1$. Proceeding as in Theorem 1, it can be shown that there is a unique global solution $X(t, T, X(T^+)) = \widehat{X}(t; T, X(T^+))$ that is defined on $[T^+, 2T]$, and $X(2T^+) = ((1-p)\widehat{S}(2T), \widehat{I}(2T), \widehat{V}(2T) + p\widehat{S}(2T), \widehat{R}(2T)) \in \mathbb{R}^4_+$. It is evident that the above deduction can go on infinitely. Hence the proof is completed. □

3 Disease-free periodic solution

Note that the variable $R(t)$ does not appear in the first three equations of (1), and so, in the rest of this article, we only consider the following subsystem of (1):

$$\begin{aligned}
\frac{\mathrm{d}S(t)}{\mathrm{d}t} &= \Lambda(t) - \beta(t)S(t)I(t) - \mu(t)S(t) + \alpha(t)V(t) \\
&\quad + \sqrt{\alpha(t)}\big\{S(t) - V(t)\big\}\frac{\mathrm{d}W_1}{\mathrm{d}t}, \quad t \neq nT, \\
\frac{\mathrm{d}I(t)}{\mathrm{d}t} &= \beta(t)S(t)I(t) - \mu_1(t)I(t) + \sigma_2(t)I(t)\frac{\mathrm{d}W_2}{\mathrm{d}t}, \quad t \neq nT, \\
\frac{\mathrm{d}V(t)}{\mathrm{d}t} &= -\mu_2(t)V(t) + \sigma_3(t)V(t)\frac{\mathrm{d}W_3}{\mathrm{d}t}, \quad t \neq nT,
\end{aligned} \tag{9a}$$

$$\begin{aligned} S\big(t^+\big) &= (1-p)S(t), \quad t = nT,\ n = 1,2,\ldots, \\ I\big(t^+\big) &= I(t), \quad t = nT,\ n = 1,2,\ldots, \\ V\big(t^+\big) &= V(t) + pS(t), \quad t = nT,\ n = 1,2,\ldots. \end{aligned} \tag{9b}$$

In this section, we discuss the existence of the disease-free periodic solution of system (9) in which infectious individuals are completely absent, that is, $I(t) = 0$ for all $t \geqslant 0$. Under this circumstances, system (9) reduces to the following stochastic impulsive system:

$$\begin{aligned} \frac{\mathrm{d}S(t)}{\mathrm{d}t} &= \Lambda(t) - \mu(t)S(t) + \alpha(t)V(t) \\ &\quad + \sqrt{\alpha(t)}\big\{S(t) - V(t)\big\}\frac{\mathrm{d}W_1}{\mathrm{d}t}, \quad t \neq nT, \\ \frac{\mathrm{d}V(t)}{\mathrm{d}t} &= -\mu_2(t)V(t) + \sigma_3(t)V(t)\frac{\mathrm{d}W_3}{\mathrm{d}t}, \quad t \neq nT, \\ S\big(t^+\big) &= (1-p)S(t), \quad t = nT,\ n = 1,2,\ldots, \\ V\big(t^+\big) &= V(t) + pS(t), \quad t = nT,\ n = 1,2,\ldots. \end{aligned} \tag{10}$$

Theorem 3. *If $2\mu^l > \alpha^u$, then system* (10) *has a unique positive T-periodic solution $(S(t), V(t))$, which is globally exponentially stable in mean square.*

Proof. Consider a multidimensional stochastic differential:

$$\mathrm{d}\mathbf{X}(t,\omega) = \mathbf{f}(t,\omega)\,\mathrm{d}t + G(t,\omega)\,\mathrm{d}\mathbf{W}(t,\omega),$$

where

$$\begin{aligned} \mathbf{X}(t,\omega) &= \big[X_1(t,\omega), X_2(t,\omega), \ldots, X_n(t,\omega)\big]^{\mathrm{T}}, \\ \mathbf{f}(t,\omega) &= \big[f_1(t,\omega), f_2(t,\omega), \ldots, f_n(t,\omega)\big]^{\mathrm{T}}, \\ \mathbf{W}(t,\omega) &= \big[W_1(t,\omega), W_2(t,\omega), \ldots, W_m(t,\omega)\big]^{\mathrm{T}}, \end{aligned}$$

and

$$\big(G(t,\omega)\big)_{ij} = g_{ij}(t,\omega), \quad \text{where } G(t,\omega) \text{ is an } n \times m \text{ matrix.}$$

Here $\mathbf{W}(t)$ is an m-dimensional Wiener process having independent elements $W_i(t)$ and $W_j(t)$ for $i \neq j$. For a smooth function $F(t,\mathbf{X})$ with respect to t and $\mathbf{X}$, Itô's formula yields the following stochastic differential for F:

$$\begin{aligned} \mathrm{d}F(t,\mathbf{X}) &= \left(\frac{\partial F}{\partial t} + \sum_{i=1}^{n}\frac{\partial F}{\partial x_i}f_i + \sum_{i=1}^{n}\sum_{j=1}^{n}\sum_{k=1}^{m}\frac{1}{2}\frac{\partial^2 F}{\partial x_i \partial x_j}g_{ik}g_{jk}\right)\mathrm{d}t \\ &\quad + \sum_{i=1}^{n}\sum_{j=1}^{m}\frac{\partial F}{\partial x_i}g_{ij}\,\mathrm{d}W_j(t). \end{aligned}$$

Let $(S(t,\zeta),V(t,\zeta'))$ be an arbitrary solution of the system of equations (10) and define $S(T,\zeta)=\xi$ and $V(T,\zeta')=\xi'$. Then $(S(t,\xi),V(t,\xi'))$ is also a solution of the system of equations (10). Define $\widehat{G}(t)=[S(t,\zeta)-S(t,\xi)]^2$ and $\widehat{G}(0)=(\zeta-\xi)^2$. Now, $\widehat{G}(t)$ is continuous and positive on $t\geqslant 0$. Applying Itô's formula, we have

$$\begin{aligned}\mathrm{d}\widehat{G}(t)={}&\big[-2\mu(t)\big(S(t,\zeta)-S(t,\xi)\big)^2\\&+2\alpha(t)\big(S(t,\zeta)-S(t,\xi)\big)\big(V(t,\zeta')-V(t,\xi')\big)\\&+\alpha(t)\big(S(t,\zeta)-S(t,\xi)\big)^2+\alpha(t)\big(V(t,\zeta')-V(t,\xi')\big)^2\\&-2\alpha(t)\big(S(t,\zeta)-S(t,\xi)\big)\big(V(t,\zeta')-V(t,\xi')\big)\big]\,\mathrm{d}t\\&+2\big(S(t,\zeta)-S(t,\xi)\big)\sqrt{\alpha(t)}\big(S(t,\zeta)-V(t,\zeta')\big)\,\mathrm{d}W_1\\&-2\big(S(t,\zeta)-S(t,\xi)\big)\sqrt{\alpha(t)}\big(S(t,\xi)-V(t,\xi')\big)\,\mathrm{d}W_1.\end{aligned}$$

Therefore,

$$\begin{aligned}\mathrm{d}\widehat{G}(t)={}&\big\{\alpha(t)-2\mu_2(t)\big\}\widehat{G}(t)\,\mathrm{d}t+\alpha(t)\big(V(t,\zeta')-V(t,\xi')\big)^2\,\mathrm{d}t\\&+2\big(S(t,\zeta)-S(t,\xi)\big)\sqrt{\alpha(t)}\big\{\big(S(t,\zeta)-S(t,\xi)\big)\\&-\big(V(t,\zeta')-V(t,\xi')\big)\big\}\,\mathrm{d}W_1.\end{aligned}\tag{11}$$

Integrating (11) from 0 to t ($t\geqslant 0$),

$$\begin{aligned}\widehat{G}(t)={}&\widehat{G}(0)+\int_0^t\big\{\alpha(s)-2\mu(s)\big\}\widehat{G}(s)\,\mathrm{d}s+\int_0^t\alpha(s)\big(V(s,\zeta')-V(s,\xi')\big)^2\,\mathrm{d}s\\&+2\int_0^t\sqrt{\alpha(s)}\big\{\widehat{G}(s)-\big(S(s,\zeta)-S(s,\xi)\big)\big(V(s,\zeta')-V(s,\xi')\big)\big\}\,\mathrm{d}W_1(s)\\\Longrightarrow\quad\mathbf{E}\widehat{G}(t)={}&\mathbf{E}\widehat{G}(0)+\int_0^t\big\{\alpha(s)-2\mu(s)\big\}\mathbf{E}\widehat{G}(s)\,\mathrm{d}s\\&+\int_0^t\alpha(s)\mathbf{E}\big(V(s,\zeta')-V(s,\xi')\big)^2\,\mathrm{d}s\\\Longrightarrow\quad\frac{\mathrm{d}\mathbf{E}\widehat{G}(t)}{\mathrm{d}t}={}&\big\{\alpha(t)-2\mu(t)\big\}\mathbf{E}\widehat{G}(t)+\alpha(t)\mathbf{E}\big(V(t,\zeta')-V(t,\xi')\big)^2\\\leqslant{}&\big\{\alpha^u-2\mu^l\big\}\mathbf{E}\widehat{G}(t),\quad t\neq nT.\end{aligned}\tag{12}$$

When $t=nT$, $\mathbf{E}\widehat{G}(nT^+)=(1-p)^2\mathbf{E}\widehat{G}(nT)$ holds good. By (12) and using [6, Lemma 2.1] and [2, Thm. 3.1], we get

$$\mathbf{E}\widehat{G}(t)\leqslant\gamma\mathbf{E}\widehat{G}(0)\mathrm{e}^{-\rho t}\quad\text{for } t\geqslant 0,\tag{13}$$

where $\rho=2\mu^l-\alpha^u-2\ln(1-p)/T>0$ and $\gamma>1$ are two positive constants.

Using (13) and the integral property of measurable functions, we have

$$\big(S(t,\zeta)-S(t,\xi)\big)^2 \leqslant \gamma|\zeta-\xi|^2\mathrm{e}^{-\rho t} \quad \text{a.e. for } t \geqslant 0. \tag{14}$$

Then from (14) it follows that, for any given $t \geqslant 0$,

$$\begin{aligned}
&\sum_{m=1}^{\infty}\big\{S(t+mT,\zeta)-S\big(t+(m-1)T,\zeta\big)\big\}\\
&\quad=\lim_{k\to\infty}\sum_{m=1}^{k}\big\{S(t+mT,\zeta)-S\big(t+(m-1)T,\zeta\big)\big\}\\
&\quad\leqslant\sqrt{\gamma}|\zeta-\xi|\lim_{k\to\infty}\sum_{m=1}^{k}\mathrm{e}^{-\rho(t+(m-1)T)/2}\\
&\quad\leqslant\sqrt{\gamma}|\zeta-\xi|\mathrm{e}^{-\rho t/2}\sum_{m=1}^{\infty}\mathrm{e}^{-\rho(m-1)T/2}<\infty\\
&\quad\Longrightarrow\quad \lim_{k\to\infty}S(t+kT,\zeta)\quad\text{exists a.e.}
\end{aligned}$$

Set $S_p^*(t,\eta)=\lim_{k\to\infty}S(t+kT,\zeta)$, then it is evident that $S_p^*(t,\eta)$ is a periodic solution (with period T) of $S(t)$ for system (10). If possible, let us assume that there is another periodic solution $\widehat{S_p}(t,\eta^*)$ (with period T) of $S(t)$ for system (10). Then it can be easily obtained that for $k\in Z_+$ and $t\geqslant 0$,

$$\begin{aligned}
\big(S_p^\star(t,\eta)-\widehat{S_p}(t,\eta^*)\big)^2&=\big(S_p^*(t+kT,\,\eta)-\widehat{S_p}(t+kT,\,\eta^*)\big)^2\\
&\leqslant\gamma|\zeta-\xi|^2\mathrm{e}^{-\rho(t+kT)}\quad\text{a.e.}
\end{aligned}$$

It is evident that for $t\geqslant 0$, $S_p^*(t,\eta)=\widehat{S_p}(t,\eta^*)$ as $k\to\infty$ a.e. It follows that for system (10), $S(t)$ has a unique positive T-periodic solution $S_p^*(t,\eta)$, and all solutions converge exponentially to it as $t\to\infty$. This completes the proof. □

Remark 1. If $2\mu^l>\alpha^u$, then from the second and fourth equations of system (10), it is evident that $V(t)$ has a unique positive T-periodic solution $V_p^*(t)$, which is globally exponentially stable in mean square.

4 Extinction and persistent of the disease

In this section, we wish to discuss the extinction and permanence of the disease of system (1). The word persistent means the long-term survival (i.e., will not extinct as time goes) of the infectious population ($I(t)$) of system (1). It demonstrates how the disease will be permanent (i.e., will not vanish as time goes) under some conditions. Here we always assume that $2\mu^l>\alpha^u$, and so system (10) admits the unique positive periodic

solution $S_p^*(t)$ for $S(t)$. Let us define the following two positive numbers:

$$R_1 = \frac{\beta^u (S_p^*)^u}{\mu_1^l + 0.5(\sigma_2^2)^l}, \qquad R_2 = \frac{\beta^l (S_p^*)^l}{\mu_1^u + 0.5(\sigma_2^2)^u},$$

obviously,

$$R_2 < R_1. \tag{15}$$

Theorem 4. *If $2\mu^l > \alpha^u$ and $R_1 < 1$, then the susceptible population $S(t)$ is persistent in the mean, and the endemic population $I(t)$ is going to extinct as $t \to \infty$.*

Proof. Let $(S(t), I(t), V(t))$ be the solution of system (9) with initial values $S(0) > 0$, $I(0) > 0$, $V(0) > 0$, and $\overline{S}(t) = \overline{S}(t, S(0))$ be the solution of $S(t)$ for system (10) with initial value $S(0)$. Using comparison theorem for SDEs, we have [14]

$$S(t) \leqslant \overline{S}(t) \quad \Longrightarrow \quad \limsup_{t\to\infty} \big\langle S(t) \big\rangle \leqslant \limsup_{t\to\infty} \big\langle \overline{S}(t) \big\rangle \leqslant \big(S_p^*\big)^u. \tag{16}$$

Also,

$$R_1 < 1 \quad \Longrightarrow \quad \beta^u (S_p^*)^u - \mu_1^l - 0.5\big(\sigma_2^2\big)^l < 0. \tag{17}$$

Using Itô's formula to $\ln(I(t))$, we have

$$\begin{aligned} \frac{1}{t} \ln \frac{I(t)}{I(0)} &= \big\langle \beta(t) S(t) \big\rangle - \big\langle \mu_1(t) + 0.5\sigma_2^2(t) \big\rangle + \frac{1}{t} \int_0^t \sigma_2(s)\, \mathrm{d}W_2(s) \\ &\leqslant \beta^u \big\langle \overline{S}(t) \big\rangle - \mu_1^l - 0.5\big(\sigma_2^2\big)^l + \frac{1}{t} \int_0^t \sigma_2(s)\, \mathrm{d}W_2(s). \end{aligned} \tag{18}$$

Now, $M(t) = (1/t) \int_0^t \sigma_2(s) \mathrm{d}W_2(s)$ is a local martingale with quadratic variation $\langle M(t) M(t) \rangle = \int_0^t \sigma_2^2(s)\, \mathrm{d}s \leqslant (\sigma_2^2)^u t$. Applying the strong law of large numbers for local martingales, we get

$$\lim_{t\to\infty} \frac{M(t)}{t} = \lim_{t\to\infty} \frac{1}{t} \int_0^t \sigma_2(s) \mathrm{d}W_2(s) = 0 \quad \text{a.s.} \tag{19}$$

From (16)–(19) it follows that

$$\begin{aligned} &\limsup_{t\to\infty} \frac{1}{t} \ln \frac{I(t)}{I(0)} \leqslant \beta^u (S_p^*)^u - \mu_1^l - 0.5\big(\sigma_2^2\big)^l < 0 \\ &\quad \Longrightarrow \quad \lim_{t\to\infty} I(t) = 0 \quad \text{a.s.} \end{aligned}$$

Hence, given $\epsilon > 0$, no matter however small, there exists $t_0 > 0$ and a set Ω_ϵ such that $\mathbf{P}(\Omega_\epsilon) \geqslant 1 - \epsilon$ and $I(t) \leqslant \epsilon$ for all $t \geqslant t_0$ and $\omega \in \Omega_\epsilon$. Therefore, from the first

equation of system (9) we get, for $t \geqslant t_0$ and $\omega \in \Omega_\epsilon$,

$$\begin{aligned} \frac{\mathrm{d}S(t)}{\mathrm{d}t} &\geqslant \Lambda(t) - \big\{\mu(t) + \epsilon\beta(t)\big\}S(t) + \alpha(t)V(t) \\ &\quad + \sqrt{\alpha(t)}\big\{S(t) - V(t)\big\}\frac{\mathrm{d}W_1}{\mathrm{d}t}, \quad t \neq nT, \\ S\big(t^+\big) &= (1-p)S(t), \quad t = nT,\ n = 1, 2, \ldots . \end{aligned} \tag{20}$$

Now, $2\mu^l > \alpha^u \implies 2(\mu^l + \epsilon\beta^l) > \alpha^u$ for a sufficiently small $\epsilon > 0$. By Theorem 3, the system

$$\begin{aligned} \frac{\mathrm{d}S(t)}{\mathrm{d}t} &= \Lambda(t) - \big\{\mu(t) + \epsilon\beta(t)\big\}S(t) + \alpha(t)V(t) \\ &\quad + \sqrt{\alpha(t)}\big\{S(t) - V(t)\big\}\frac{\mathrm{d}W_1}{\mathrm{d}t}, \quad t \neq nT, \\ S\big(t^+\big) &= (1-p)S(t), \quad t = nT,\ n = 1, 2, \ldots, \end{aligned} \tag{21}$$

has a unique positive T-periodic solution $\widehat{S}_\epsilon(t)$ for $S(t)$, which is globally exponentially stable in mean square. From (20) and (21) and by the comparison theorem for stochastic differential equations we get $\liminf_{t\to\infty}\langle S(t)\rangle \geqslant \widehat{S}_\epsilon^l$. This completes the proof. □

Remark 2. In a deterministic environment the following result holds [11]: If $R_0 < 1$, then the disease-free periodic solution $(\widetilde{S}_e(t), 0, \widetilde{V}_e(t), 0)$ of system (1) is globally asymptotically stable, where

$$\begin{aligned} R_0 &= \frac{\beta\Lambda}{\mu^2} + \frac{\beta}{\mu T}\left\{\left(S^* - \frac{\Lambda}{\mu}\right)\frac{1}{\mu+\alpha}\big(1 - \mathrm{e}^{-(\mu+\alpha)T}\big)\right\}, \\ \widetilde{S}_e(t) &= \frac{\Lambda}{\mu} + \left(S^* - \frac{\Lambda}{\mu}\right)\mathrm{e}^{-(\mu+\alpha)(t-nT)}, \quad nT < t \leqslant (n+1)T, \\ \widetilde{V}_e(t) &= \frac{\Lambda}{\mu} - \widetilde{S}_e(t), \quad \text{and} \quad S^* = \frac{\Lambda(1-p)(1-\mathrm{e}^{-(\mu+\alpha)T})}{\mu\{1-(1-p)\mathrm{e}^{-(\mu+\alpha)T}\}}. \end{aligned}$$

Theorem 5. *If $2\mu^l > \alpha^u$ and $R_2 > 1$, then the susceptible population $S(t)$ and the endemic population $I(t)$ is weakly persistent in the mean.*

Proof. Here we need to show that there exists a constant $\kappa > 0$ such that for any solution $(S(t), I(t), V(t))$ of system (9) with initial values $S(0) > 0$, $I(0) > 0$, $V(0) > 0$, we have $\limsup_{t\to\infty}\langle I(t)\rangle \geqslant \kappa$ a.s. Otherwise, given $\epsilon > 0$, no matter however small, there exists a solution $(\widehat{S}(t), \widehat{I}(t), \widehat{V}(t))$ with positive initial values $(\widehat{S}(0), \widehat{I}(0), \widehat{V}(0))$ such that $\mathbf{P}\{\limsup_{t\to\infty}\langle\widehat{I}(t)\rangle < \epsilon\} > 0$. Let $\overline{S}(t) = \overline{S}(t, \widehat{S}(0))$ be the solution of $S(t)$ for system (10) with initial value $\widehat{S}(0)$. Using comparison theorem for stochastic differential equations, we have [14]

$$\widehat{S}(t) \leqslant \overline{S}(t) \quad \implies \quad \limsup_{t\to\infty}\big\langle\widehat{S}(t)\big\rangle \leqslant \limsup_{t\to\infty}\big\langle\overline{S}(t)\big\rangle \leqslant (S_p^*)^u = h_1 \quad \text{(say)}.$$

Also,

$$R_2 > 1 \quad \Longrightarrow \quad h_2 - \mu_1^u - 0.5\big(\sigma_2^2\big)^u - \frac{h_1^2\beta^u\big(\beta^u+1\big)\epsilon}{\Lambda^l + \alpha^l (V_p^*)^l} > 0, \tag{22}$$

where $h_2 = \beta^l (S_p^*)^l$.

Applying Itô's formula to $\ln(\widehat{I}(t))$, we have

$$\begin{aligned}
\frac{1}{t}\ln\frac{\widehat{I}(t)}{\widehat{I}(0)} &= \big\langle \beta(t)\widehat{S}(t)\big\rangle - \big\langle \mu_1(t) + 0.5\sigma_2^2(t)\big\rangle + \frac{1}{t}\int_0^t \sigma_2(s)\,\mathrm{d}W_2(s) \\
&\geqslant \beta^l\big\langle S_p^*(t)\big\rangle - \mu_1^u - 0.5\big(\sigma_2^2\big)^u - \beta^u\big\langle |S_p^*(t) - \widehat{S}(t)|\big\rangle + \frac{1}{t}\int_0^t \sigma_2(s)\,\mathrm{d}W_2(s) \\
&\geqslant h_2 - \mu_1^u - 0.5\big(\sigma_2^2\big)^u - \beta^u\big\langle \big|S_p^*(t) - \widehat{S}(t)\big|\big\rangle + \frac{1}{t}\int_0^t \sigma_2(s)\,\mathrm{d}W_2(s). \qquad (23)
\end{aligned}$$

Next, let us take $G_1(t) = |\ln S_p^*(t) - \ln \widehat{S}(t)|$ as a Lyapunov function. It is noted that at $t = nT$, $G_1(t^+) = |\ln S_p^*(t^+) - \ln \widehat{S}(t^+)| = |\ln S_p^*(t) - \ln \widehat{S}(t)| = G_1(t)$ holds good. A direct calculation of the right differential $d^+G_1(t)$ yields the following result:

$$\begin{aligned}
d^+G_1(t) &\leqslant \left[-\big\{\Lambda(t) + \alpha^l (V_p^*)^l\big\}\frac{|S_p^*(t) - \widehat{S}(t)|}{S_p^*(t)\widehat{S}(t)} + \beta(t)\widehat{I}(t)\right]\mathrm{d}t \\
&\leqslant \left[-\big\{\Lambda(t) + \alpha^l (V_p^*)^l\big\}\frac{|S_p^*(t) - \widehat{S}(t)|}{S_p^*(t)\widehat{S}(t)} + \epsilon\beta^u\right]\mathrm{d}t \\
\Longrightarrow \quad & \big(\Lambda^l + \alpha^l (V_p^*)^l\big)\left\langle \frac{|S_p^*(t) - \widehat{S}(t)|}{S_p^*(t)\widehat{S}(t)}\right\rangle \leqslant \epsilon\beta^u + \frac{G_1(0)}{t} \\
\Longrightarrow \quad & \left\langle \frac{|S_p^*(t) - \widehat{S}(t)|}{S_p^*(t)\widehat{S}(t)}\right\rangle \leqslant \frac{\epsilon(\beta^u+1)}{\Lambda^l + \alpha^l (V_p^*)^l} \quad \text{for a sufficiently large } t \\
\Longrightarrow \quad & \big\langle \big|S_p^*(t) - \widehat{S}(t)\big|\big\rangle \leqslant \frac{h_1^2(\beta^u+1)\epsilon}{\Lambda^l + \alpha^l (V_p^*)^l}. \qquad (24)
\end{aligned}$$

From (22)–(24) it follows that

$$\begin{aligned}
&\limsup_{t\to\infty}\frac{1}{t}\ln\frac{\widehat{I}(t)}{\widehat{I}(0)} \geqslant h_2 - \mu_1^u - 0.5\big(\sigma_2^2\big)^u - \frac{h_1^2\beta^u(\beta^u+1)\epsilon}{\Lambda^l + \alpha^l (V_p^*)^l} > 0 \\
&\quad \Longrightarrow \quad \mathbf{P}\left\{\limsup_{t\to\infty}\frac{\ln\widehat{I}(t)}{t} > 0\right\} > 0, \quad \text{a contradiction.}
\end{aligned}$$

This completes the proof. □

5 Numerical simulation

Beside analytical findings, numerical simulations are also important; because simulation can be used to validate the analytical findings. For various choices of the parameters of the model, we have performed the simulations using MATLAB. It is observed that they are in good agreement with our analytical findings.

Now we will study the illustrative examples by means of some constant values of the parameters to demonstrate the effectiveness of our results. First we take $\Lambda = 0.2$, $\beta = 0.5$, $\mu = 0.08$, $\alpha = 0.5$, $\mu_1 = r + \mu + d = 0.5$, $\sigma_2 = 0.8$, $\mu_2 = \mu + \alpha = 0.58$, $\sigma_3 = 0.8$, $r = 0.2$, $\sigma_4 = 0.3$ in model (1). Then by the Theorem 2 we may obtain a unique positive periodic solution X_t, which is asymptotically stable in mean square for some given initial value $X_0(S(0) = 0.4, I(0) = 0.5, V(0) = 0.4, R(0) = 0.8)$. In Fig. 1(a), the orbit of $X(t)$ has been depicted for the impulsive vaccination rate $p = 0.4$.

By Theorem 3 and Remark 1 we obtain that if $2\mu^l > \alpha^u$, then for system (10), $S(t)$ and $V(t)$ have unique positive T-periodic solution, which is globally exponentially stable. If we consider $\Lambda = 0.2$, $\beta = 0.5$, $\mu^l = \mu = 0.3$, $\alpha^u = \alpha = 0.5$, $\mu_1 = 0.5$, $\sigma_2 = 0.8$, $\mu_2 = 0.8$, $\sigma_3 = 0.8$, $r = 0.2$, $\sigma_4 = 0.3$ satisfying the condition of Theorem 3, we may obtain a globally exponentially stable solution for $S(t)$ and $V(t)$. For $p = 0.6$ and three different choice of initial values, we get a unique stable T-periodic orbit in finite time (Fig. 1(b)).

In Section 4, the criteria for extinction and permanence of the disease of system (1) has been discussed by Theorems 4 and 5. If $2\mu^l > \alpha^u$ and $R_1 < 1$, then the susceptible population $S(t)$ will be persistent in the mean, and the endemic population $I(t)$ will be going to extinct as time goes. On the other hand, if $R_2 > 1$ along with $2\mu^l > \alpha^u$, then both these population will be weakly persistent in the mean. For $\Lambda = 0.2$, $\beta = 0.5$, $\mu = 0.3$, $\alpha = 0.5$, $\mu_1 = 0.5$, $\sigma_2 = 0.8$, $\mu_2 = \mu + \alpha = 0.8$, $\sigma_3 = 0.8$, $r = 0.2$, $\sigma_4 = 0.3$, the computed value of R_1 is $0.4968 < 1$. Hence, all the conditions of Theorem 4 are satisfied,

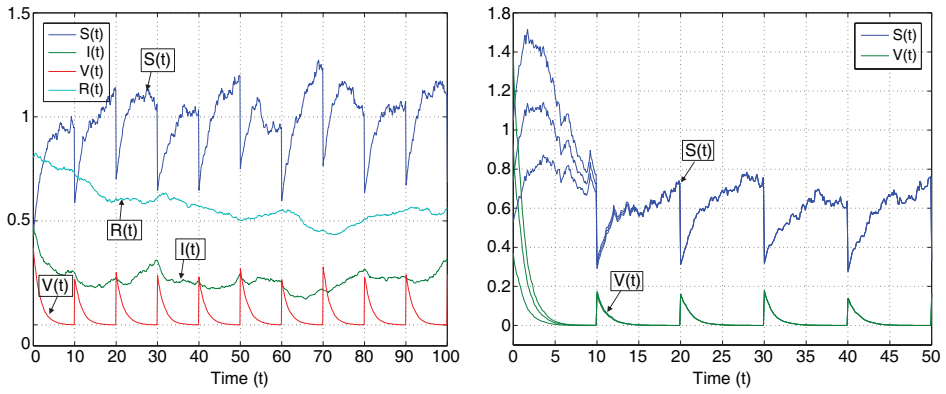

Figure 1. Stable time series.

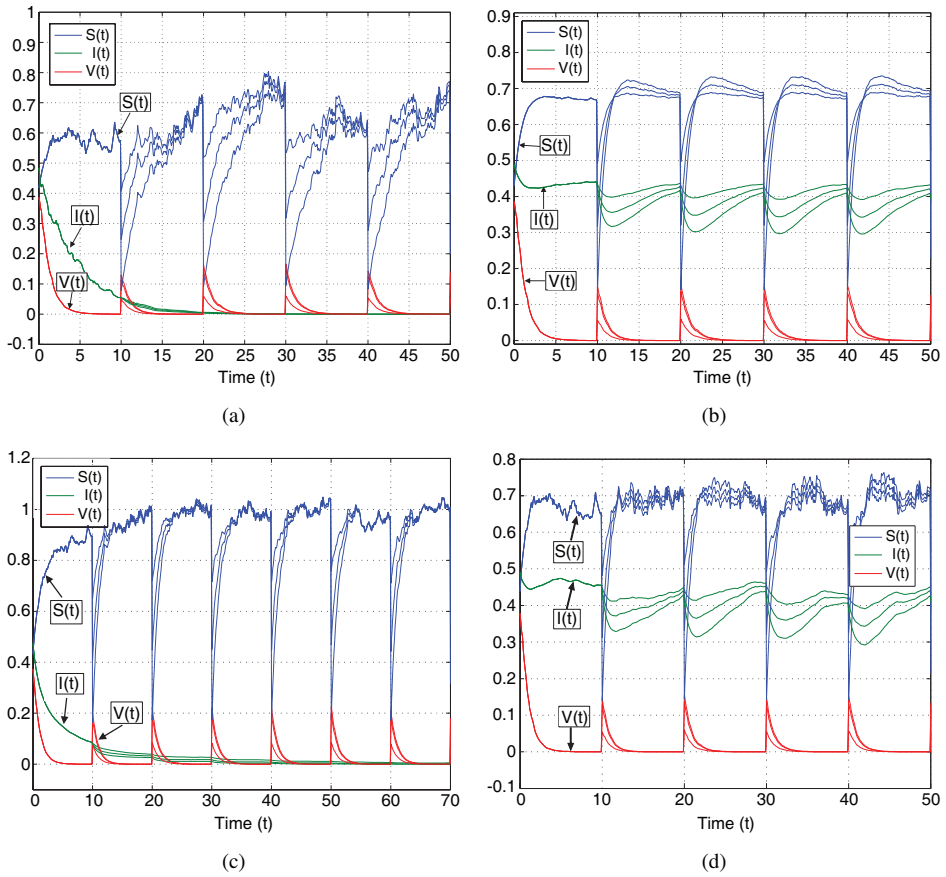

Figure 2. T-periodic stable orbits.

and we obtain the T-periodic stable orbits with $S(0) = 0.4$, $I(0) = 0.5$, $V(0) = 0.4$ for three different choice of impulsive vaccination rate: $p = 0.3$, $p = 0.6$, and $p = 0.9$. It is observed that the infected population $I(t)$ extinct in finite time (Fig. 2(a)) irrespective of the values of p.

Changing the parameters a little bit as $\Lambda = 0.8$, $\beta = 0.9$, $\mu = 0.8$, $\alpha = 0.002$, $\mu_1 = 0.6$, $\sigma_2 = 0.03$, $\mu_2 = \mu + \alpha = 0.802$, $\sigma_3 = 0.8$, $r = 0.05$, $\sigma_4 = 0.3$ we obtain $R_2 = 1.006 > 1$. In Fig. 2(b), we obtain T-periodic stable orbits showing that the infected population persists for different choice of impulsive vaccination rate ($p = 0.3$, 0.6, 0.9). This result is in good agreement with Theorem 5.

We also consider the case when $R_1 = 1.0543 > 1$ and $R_2 = 0.8961 < 1$ with $\Lambda = 0.8$, $\beta = 0.9$, $\mu = 0.8$, $\alpha = 0.1$, $\mu_1 = 0.9$, $\sigma_2 = 0.03$, $\mu_2 = \mu + \alpha = 0.9$, $\sigma_3 = 0.4$, $r = 0.05$, $\sigma_4 = 0.3$ for $p = 0.3$, 0.6, and 0.9. Using these parameter values, the movement paths of $S(t)$, $I(t)$, and $V(t)$ are presented in Fig. 2(c). This figure shows that the disease dies out.

For $R_1 = 1.1136 > 1$ and $R_2 = 0.9513 < 1$ with $\Lambda = 0.8$, $\beta = 0.9$, $\mu = 0.8$, $\alpha = 0.2$, $\mu_1 = 0.6$, $\sigma_2 = 0.03$, $\mu_2 = \mu + \alpha = 1$, $\sigma_3 = 0.8$, $r = 0.05$, $\sigma_4 = 0.3$ for $p = 0.3$, 0.6, and 0.9. Using these parameter values, the movement paths of $S(t)$, $I(t)$, and $V(t)$ are presented in Fig. 2(d). This figure shows that the disease is still permanent.

Remark 3. From (15) it is noticed that $R_2 < R_1$. When $R_2 \leqslant 1$ and $R_1 \geqslant 1$, the dynamical behaviour of the epidemic model (1) has not been clear (see also Figs. 2(c), 2(d).

6 Conclusions

In this paper, we have considered a dynamical model of Chlamydia diseases with bilinear incidence rate under stochastic perturbation and the pulse vaccination scheme. Noise and pulse are introduced into Chlamydia epidemic model, which greatly enriches biologic background. It is justified by observation that the real life is full of social and environmental random variations. The entire high-risk human population is split up into four mutually-exclusive epidemiological compartments (based on disease status), namely, susceptible (S), infective class (I), naturally recovered individuals from Chlamydia infection (R), and vaccinated individuals (V). It is assumed that the recovered individuals acquire the permanent immunity but the vaccinated acquire temporary immunity. So, the natural immunity is permanent but the vaccine-induced immunity is temporary. The susceptible population increases by the recruitment through new sexually-active individuals, migration, and vaccinated hosts and decreases due to direct contact with infected individuals, natural death, and pulse vaccination strategy. The infected class is increased by infection of susceptible. A fraction of the infectious individuals recovers naturally. The infected class is decreased through natural recovery from infection, by disease-related death and by natural death. The most basic and important questions to ask for the systems in the theory of mathematical epidemiology are the persistence, extinctions, the existence of periodic solutions, global stability, etc. It is seen that our epidemic model has global positive solutions, and under some conditions, it admits a unique positive periodic disease-free solution, which is globally exponentially stable in mean square. Here we have established some sufficient conditions on the persistent and extinction of the disease by introducing two threshold values R_1 and R_2 ($< R_1$), and further we obtained that the disease will be going to extinct when $R_1 < 1$ and the susceptible as well as the disease will be weakly persistent in the mean when $R_2 > 1$. It can be shown that if impulsive vaccination rate p is larger than some critical value p_0 such that $R_1 < 1$, then it is possible to prevent the Chlamydia disease from generating endemic. Thus, the Chlamydia disease can be eradicated from the entire population in the random environment in a stable way by PVS. The important mathematical findings for the dynamical behaviour of the Chlamydia disease model are also numerically verified using MATLAB. It is observed that when $R_2 \leqslant 1$ and $R_1 \geqslant 1$, the dynamical behaviour is not clear.

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