A Stochastic Differential Equation SIRS Epidemic Model With Ratio-Dependent Incidence Rate

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Abstract

In this paper we study global dynamics of a general SIRS epidemic model with a ratio-dependent incidence rate and its corresponding stochastic differential equation version. For the deterministic model, we show that the disease reproduction number $R_0$ determines whether there is an endemic outbreak or not: the disease free dynamics occurs if $R_0 \leq 1$ while the unique endemic steady state is globally stable if $R_0 > 1$. For the stochastic model, we show that its related reproduction number $R_0^S$ can determine whether there is a unique disease-free stationary distribution or a unique endemic stationary distribution. In addition, we provide analytic results regarding the stochastic boundedness, permanence/extinction, asymptotic stability as well as its related ergodic properties. One of the most interesting findings is that random fluctuations introduced in our stochastic model can suppress disease outbreak, which can provide us some useful control strategies to regulate disease dynamics. Combining our analytical and numerical results, we conclude that different stochastic modeling approaches can have different dynamical outcomes. This provides us a caution when we derive the stochastic models from a given deterministic model.

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1. Introduction

According to the World Health Organization (WHO), infectious diseases are responsible for a quarter to a third of all deaths worldwide. As of 2008, four of the top ten causes of death were due to infectious diseases; and in low-income countries, five of the top killers were due to infectious diseases [1]. Mathematical models have been an important tool in analyzing the epidemiological characteristics of infectious diseases since the pioneer work of Kermack and McKendrick [2], which provide useful control measures [3, 4]. Many well-known classic models of infectious disease population dynamics have been deterministic [5]. For example, Kermack and McKendrick [2] developed a general SIR (susceptible, infective, recovered) differential equation model which provide useful insights regarding important factors affecting the rate of growth and the final size of an epidemic. When modeling epidemics, the most important consideration is to choose a model that best represents the demographics and epidemiology of the population being modeled. The nature of epidemic growth and spread is inherently random due to the unpredictability of person-to-person contacts [6] and population is subject to a continuous spectrum of disturbances [7, 8]. Because of the randomness in disease spread and the potential for a disease to rapidly spread throughout the world via air travel, emerging infectious diseases such as H5N1 avian influenza, Severe Acute Respiratory Syndrome (SARS) are a global problem from their inception. As in the early stages of an outbreak case, infectious number will be very small, random variations alone can cause an epidemic to die out, and it is particularly important to include this randomness in models for emerging infectious diseases [6].

Stochastic models could be a more appropriate way of modeling epidemics in many circumstances [9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30]. For example, stochastic models are able to take care of randomness of infectious contacts oc-
curring in the latent and infectious periods [31]. It also has been showed that some stochastic epidemic models can provide an additional degree of realism in comparison with their deterministic counterparts [32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49]. Especially, stochastic model should suit better for the question of disease extinction [8]. For instances, Herwaarden et al. [50] indicated that an endemic in a deterministic system can disappear in its corresponding stochastic system due to stochastic fluctuations. Mao et al. [14] showed that a sufficiently small noise can suppress explosions in population dynamics; Nåsell [35] developed stochastic models to show that some stochastic models are a better approach to describing epidemics for a large range of realistic parameter values in comparison with their deterministic counterparts.

Many realistic stochastic epidemic models can be derived based on their deterministic formulations. Excellent references for a variety of well-known stochastic models deriving from their deterministic counterparts include the books by Allen [8, 51]. Allen [8, 51] provided a great introduction to the methods of derivation for various types of stochastic models including stochastic differential equation (SDE) epidemic models. There are a fair amount of literature regarding SDE epidemic models. For example, Ball and Neal [42] studied a general stochastic SIR model among a closed finite population, and obtained a threshold parameter that governs whether or not global epidemics can occur; Tuckwell and Williams [5] investigated the properties of a simple discrete time stochastic SIR type epidemic model, especially focusing on the influence of individuals with small population numbers and fluctuations of environment. Britton [41] gave an excellent survey on SDE epidemic models which presented the exact and asymptotic properties of a simple stochastic epidemic model, and illustrated by studying effects of vaccination and inference procedures for important parameters such as the basic reproduction number and the critical vaccination coverage. Gray and coworkers [46] formulated a SDE SIS epidemic model, and proved that the model has a unique global positive solution and established conditions for extinction and persistence.
of infectious individuals. Yang et al. [52] investigated the ergodicity and extinction of SDE SIR and SEIR epidemic models with saturated incidence.

There are different possible approaches to including random effects in the model, both from a biological and mathematical perspective [53]. In this article, we adopt the approach by Beddington and May [7], which has been pursued in [17, 28, 37, 45, 54, 55, 56, 40, 57, 58, 52, 59, 49, 60, 48, 61, 62, 63]. Mathematically speaking, this approach is based on the assumption that the noise is uniform over the state space and over time [54]. Following their approach, we study a general SDE SIRS model with ratio-dependent nonlinear incidence rate where we assume that the environmental noise is proportional to the variables. The SIRS model is suitable for infectious diseases transmitted by a vector (often an invertebrate arthropod) conferring temporal acquired immunity. Since the first deterministic SIRS model with constant recruitment and disease-induced death developed by Anderson and May [64], there are many applications of such models. For instance, the host-vector SIRS models have been proposed to study spread of Japanese Encephalitis by Mukhopadhyay and Tapaswi [65] and malaria by Tumwiine et al. [66] in human populations. The main focus of this article is to investigate how environment fluctuations affect disease’s dynamics through studying the global dynamics of a general SIRS model with a ratio-dependent transmission rate in both deterministic and stochastic versions. In particular, we aim to answer the following questions through our analytic and numerical results of SIRS models:

1. What is disease dynamics of stochastic SIRS models with ratio-dependent incidence rate?
2. How may different types of environmental fluctuations produce different dynamical outcomes?
3. How may the dynamics of SIRS models with environmental fluctuations differ from the deterministic version?

The rest of this article is organized as follows: In Section 2, we derive a general SIRS
deterministic model and its stochastic version with necessary definitions that will be used in our analysis. In Section 3, we carry out the completed global analysis of the deterministic SIRS model \((8)\). In Section 4, we obtain the analytic results of dynamics of the SDE model \((9)\) and their related biological implications. In Section 5, we provide numerical simulations to support our answers to the research questions proposed in the above. In the last section, we provide a brief discussion and the summary of main results.

2. Model Derivation

Let \(S(t)\) be the number of susceptible individuals, \(I(t)\) the number of infective individuals, and \(R(t)\) the number of removed individuals at time \(t\). We assume that: i) infectious disease can cause additional mortality; ii) an infective individual can recover with a loss of immunity, then a general SIRS model can be described by the diagram \(1\) and be modeled by the following set of nonlinear differential equations \((1)\) deterministically.

\[
\begin{align*}
\frac{dS}{dt} &= b - dS - g(S, I) + \gamma R, \\
\frac{dI}{dt} &= g(S, I) - (d + \mu + \delta)I, \\
\frac{dR}{dt} &= \mu I - (d + \gamma)R,
\end{align*}
\]

where \(b\) is the recruitment rate of the population, \(d\) the natural death rate of the population, \(\mu\) the natural recovery rate of the infective individuals, \(\gamma\) the rate at which recovered individuals
lose immunity and return to the susceptible class, $\delta$ the disease inducing death rate, and $g(S, I)$ the transmission of the infection or called as incidence rate.

The transmission function $g(S, I)$ plays a key role in determining disease dynamics [67, 68, 69, 70]. Traditionally, the density-dependent transmission (or called bilinear incidence rate, $g(S, I) = kSI$, $k$ the proportionality constant) and the frequency-dependent transmission (or called standard incidence rate, $g(S, I) = \frac{kSI}{S+I}$) are two extreme forms of disease transmission, which have been frequently used in well-known epidemic models [71, 72]. There are several different nonlinear transmission functions proposed by researchers [73, 74, 75, 76, 68, 77, 78]. For example, Capasso and Serio [74] introduced a saturated transmission rate $g(S, I) = f(I)S$, where $f(I)$ tends to a saturation level as $I$ getting large, i.e.,

$$g(S, I) = f(I)S = \frac{kIS}{1 + \alpha I}. \tag{2}$$

The term $kI$ in (2) measures the infection force of the disease and $\frac{1}{1+\alpha I}$ measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals. Ruan and Wang [73] studied an epidemic model with a specific nonlinear incident rate (3)

$$g(S, I) = f(I)S = \frac{kI^2S}{1 + \alpha I^2}. \tag{3}$$

where they presented a detailed qualitative and bifurcation analysis of the model. Xiao and Ruan [75] explored an epidemic model with another specific nonlinear incident rate (4)

$$g(S, I) = f(I)S = \frac{kIS}{1 + \alpha I^2}, \tag{4}$$

where $1/(1 + \alpha I^2)$ describes the psychological or inhibitory effect from the behavioral change of the susceptible individuals when the number of infective individuals is very large. Especially, Liu et al. [76] proposed a general nonlinear transmission rate (5)

$$g(S, I) = f(I)S = \frac{kI^\gamma S}{1 + \alpha I^\gamma}, \tag{5}$$
where parameters $l$ and $h$ are positive constants, $\alpha$ a nonnegative constant measuring the psychological or inhibitory effect; the term $kI^l$ measures the infection force of the disease; and $\frac{1}{1 + \alpha I^h}$ measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals.

The infectious force $f(I)$ in (5) is a function of infective individuals which has been used in many classical disease models. The mechanisms regarding how a disease transmits may be different for different infections. The transmission of an infectious disease may involve susceptible $S$, infective $I$ and the total size of the population $N$ [68], or involve $S$ and $I$ only [77, 78], which is governed by the biological properties of the disease. Yuan and Li [77, 78] studied a ratio-dependent nonlinear incident rate which takes the following form (6):

$$g(S, I) = f \left( \frac{I}{S} \right) S = \frac{k (I/S)^l S}{1 + \alpha (I/S)^h} = \frac{k S^{h-l+1} I^l}{S^h + \alpha I^h},$$

(6)

where $\alpha$ is the parameter measures the psychological or inhibitory effect. In the case that $\alpha = 1$ and $h = l = 1$, (6) becomes the well-known frequency-dependent transmission rate $\frac{kSI}{S + I}$. See the graphic representation of $g(S, I)$ for varied values of $h$ and $l$ when $k = 0.5, \alpha = 0.5$ in Figs. 2 and 3.

In the case that $l = 1$, we obtain the ratio-dependent transmission rate $g(S, I)$ as the following form:

$$g(S, I) = f \left( \frac{I}{S} \right) S = \frac{kI}{1 + \alpha (I/S)^h} = \frac{k S^{h-1} I}{S^h + \alpha I^h},$$

(7)

which indicates that the transmission rate of disease is approximately governed by $kI$ when $\frac{I}{S}$ is small (e.g., at the beginning of disease’s spreading) and it is approximately governed by $\frac{k}{\alpha} \left( \frac{S}{I} \right)^{h-1} S$ while $\frac{I}{S}$ is large (e.g., in the endemic when almost everyone is infected). Therefore, the ratio-dependent transmission rate (7) indeed takes accounts for crowding effects and behavior changes during epidemics.
In this paper, we will focus on ratio-dependent incidence rate (7). Thus, the corresponding SIRS model (1) has the following form (8)
\[
\begin{align*}
\frac{dS}{dt} &= b - dS - \frac{kS^hI}{S^h + \alpha I^k} + \gamma R, \\
\frac{dI}{dt} &= \frac{kS^hI}{S^h + \alpha I^k} - (d + \mu + \delta)I, \\
\frac{dR}{dt} &= \mu I - (d + \gamma)R,
\end{align*}
\]
whose state space is the first quadrant \(\mathbb{R}_+^3 = \{(S, I, R) : S \geq 0, I \geq 0, R \geq 0, S + I + R > 0\}\). For convenience, we only consider the biologically meaningful initial conditions \(S(0) > 0, I(0) > 0, R(0) = 0\).

The initial fraction infective \(I(0) > 0\) is often assumed to be small and positive. The reason for assuming that \(R(0) = 0\) is that initially immune individuals play no part in the dynamics so, up to a normalizing constant, initially immune individuals may simply be ignored [6, 41].

2.1. Stochastic differential equation SIRS model

There are many different approaches to deriving a stochastic version from the deterministic SIRS model with the ratio-dependent transmission function \(g(S, I) = \frac{kS^hI}{S^h + \alpha I^k}\). In this article, as discussed in Section 1, we follow the approach used by Beddington and May [7] and the related work pursued by other researchers on studying epidemic models [37, 45, 57, 58, 49, 60, 48, 62, 63]. Schreiber et al. [79] also used a similar approach to study the coexistence of species that can be modeled by SDE models. In their model, they assume that the environmental noise is generated by an \(m\)-dimensional standard Brownian motion and per-capita effect of environmental stochasticity on the growth of population for each species is given by a combination of population of all presented species. Thus, stochastic perturbation in our model is a white noise type that is directly proportional to \(S(t), I(t), R(t)\) and is influenced on the \(\frac{dS(t)}{dt}, \frac{dI(t)}{dt}, \text{ and } \frac{dR(t)}{dt}\), respectively. Following this approach, we
obtain the following SDE epidemic model (9) that is analog to its deterministic version (8) by introducing stochastic perturbation terms into the growth equations of susceptible, infective, recovered individuals to incorporate the effect of randomly fluctuating environments:

\[
\begin{align*}
    dS &= \left( b - dS - \frac{kS^h I}{S^h + \alpha I^h} + \gamma R \right) dt + \sigma_1 S dB_1(t), \\
    dI &= \left( \frac{kS^h I}{S^h + \alpha I^h} - (d + \mu + \delta) I \right) dt + \sigma_2 I dB_2(t), \\
    dR &= \left( \mu I - (d + \gamma) R \right) dt + \sigma_3 R dB_3(t),
\end{align*}
\]

(9)

where \( \sigma_i (i = 1, 2, 3) \) are real constants and known as the intensity of environmental fluctuations, \( B_i(t) (i = 1, 2, 3) \) independent standard Brownian motions.

We would like to point out that Cai et al [49] studied the dynamics of the special case of model (8) when \( h = 1 \) and \( \delta = 0 \):

\[
\begin{align*}
    \frac{dS}{dt} &= b - dS - \frac{kIS}{S + \alpha I} + \gamma R, \\
    \frac{dI}{dt} &= \frac{kIS}{S + \alpha I} - (d + \mu) I, \\
    \frac{dR}{dt} &= \mu I - (d + \gamma) R,
\end{align*}
\]

(10)

which can nondimensionalize to model (10) as follows:

\[
\begin{align*}
    \frac{dx}{dt} &= 1 - qx - y - \frac{axy}{x + py}, \\
    \frac{dy}{dt} &= \left( \frac{R_0 x}{x + py} - 1 \right) y.
\end{align*}
\]

(11)

The stochastic version of model (11) is the following model:

\[
\begin{align*}
    dx &= \left( 1 - qx - y - \frac{axy}{x + py} \right) dt + \sigma_1 x dB_1(t), \\
    dy &= \left( \frac{R_0 xy}{x + py} - y \right) dt + \sigma_2 y dB_2(t),
\end{align*}
\]

(12)

Cai et al [49] obtained some properties of model (12), such as the existence and uniqueness of the global positive solutions, stochastic boundedness and permanence. In this article, we have broaden the scope and the analysis of the dynamics of model (8) and its stochastic version.
2.2. Related definitions

We first define a bounded set $\Gamma$ as follows:

$$
\Gamma = \left\{ (S, I, R) \in \mathbb{R}^3_+ : 0 < S + I + R \leq \frac{b}{d} \right\} \subset \mathbb{R}^3_+. 
$$

Throughout this paper, let $(\Omega, \mathcal{F}, \mathcal{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t \in \mathbb{R}_+}$ satisfying the usual conditions (i.e., it is right continuous and increasing while $\mathcal{F}_0$ contains all $\mathcal{P}$-null sets). We denote

$$
x(t) = \left( S(t), I(t), R(t) \right) := (x_1(t), x_2(t), x_3(t)),
$$

and the norm $|x(t)| = \sqrt{S^2(t) + I^2(t) + R^2(t)}$. And denote $C^{2,1}(\mathbb{R}^3 \times (0, \infty); \mathbb{R}_+)$ as the family of all nonnegative functions $V(x, t)$ defined on $\mathbb{R}^3 \times (0, \infty)$ such that they are continuously twice differentiable in $x$ and once in $t$.

We define the differential operator $L$ associated with 3-dimensional stochastic differential equation:

$$
dx(t) = f(x, t)dt + \varphi(x, t)dB(t)
$$

as

$$
L = \frac{\partial}{\partial t} + \sum_{i=1}^{3} f_i(x, t) \frac{\partial}{\partial x_i} + \frac{1}{2} \sum_{i,j=1}^{3} [\varphi^T(x, t) \varphi(x, t)]_{ij} \frac{\partial^2}{\partial x_i \partial x_j}.
$$

If $L$ acts on a function $V \in C^{2,1}(\mathbb{R}^3 \times (0, \infty); \mathbb{R}_+)$, then we denote

$$
LV(x, t) = V_t(x, t) + V_x(x, t)f(x, t) + \frac{1}{2} \text{trace} [\varphi^T(x, t)V_{xx}(x, t)\varphi(x, t)],
$$

where "$T$" means transposition.

To further our study, we have the following definitions:

**Definition 2.1.** [Permanent [79]] We say the deterministic model (8) is permanent in its state space if there exists two positive constants $0 < B_1 < B_2$ such that for any initial condition taken from the state space, the following inequalities hold:

$$
B_1 \leq \liminf_{t \to \infty} \min \{S(t), I(t), R(t)\} \leq \limsup_{t \to \infty} \max \{S(t), I(t), R(t)\} \leq B_2.
$$
Definition 2.2. [Stochastically ultimately bounded [17]] The solution \( x(t) = (S(t), I(t), R(t)) \) of SDE model (9) is said to be *stochastically ultimately bounded*, if for any \( \varepsilon \in (0, 1) \), there is a positive constant \( \varrho = \varrho(\varepsilon) \), such that for any initial value \((S(0), I(0), R(0)) \in \Gamma \), the solution \( x(t) \) of model (9) has the property that
\[
\limsup_{t \to \infty} \mathcal{P}\{|x(t)| > \varrho\} < \varepsilon.
\]

Definition 2.3. [Stochastically permanent [80, 23]] The solution \( x(t) = (S(t), I(t), R(t)) \) of SDE model (9) is said to be *stochastically permanent* if for any \( \varepsilon \in (0, 1) \), there exists a pair of positive constants \( \varrho = \varrho(\varepsilon) \) and \( \chi = \chi(\varepsilon) \), such that for any initial value \((S(0), I(0), R(0)) \in \Gamma \), the solution \( x(t) \) of model (9) has the property that
\[
\liminf_{t \to \infty} \mathcal{P}\{|x(t)| \leq \varrho\} \geq 1 - \varepsilon, \quad \liminf_{t \to \infty} \mathcal{P}\{|x(t)| \geq \chi\} \geq 1 - \varepsilon.
\]

Remark 2.4. We would like to point out that the permanence of a deterministic model implies that population of each class/species in the system is bounded above zero and below certain number while the concept of *stochastically permanent* implies that the sum of all class/species population in the system is bounded above zero and below certain number with probability arbitrary close to 1.

Definition 2.5. [Stochastic extinction [46]] The infective \( I(t) \) is said to be *Stochastic extinct* if
\[
\mathcal{P}\{\lim_{t \to \infty} I(t) = 0\} = 1.
\]

Definition 2.6. [Stationary distribution [81]] Let \( P(t, x_0, \cdot) \) denote the probability measure induced by \( x(t) = (S(t), I(t), R(t)) \) with initial value \( x_0 = (S(0), I(0), R(0)) \); that is,
\[
P(t, x_0, B) = \mathcal{P}\{x(t) \in B : x(0) = x_0\}, \text{ for any Borel set } B \subset \Gamma.
\]

If there is a probability measure \( \pi(\cdot) \) on the measurable space \((\Gamma, \mathcal{B}(\Gamma))\) such that
\[
\lim_{t \to \infty} P(t, x_0, B) = \pi(B) \quad \text{for any } x_0 \in \Gamma,
\]
we then say that model (9) has a *stationary distribution* \( \pi(\cdot) \).
In the next two sections, we will derive analytic results of the deterministic SIRS model (8) and its corresponding stochastic differential model (9) with the assumption that environment noise is proportional to the variables.

3. Dynamics of the deterministic model (8)

A straightforward computation shows that model (8) is continuous and Lipschizian in $\mathbb{R}^3_+$. From the existence and uniqueness of the solution of the ordinary differential equation, the solution of model (8) with positive initial conditions exists and is unique.

The following result shows that the solutions of model (8) are bounded, and hence, lie in a compact set and are continuous for all positive time.

**Lemma 3.1.** Model (8) is positively invariant in $\Gamma$ which attracts every solution with initial conditions starting in its state space. Moreover, every trajectory of model (8) is eventually staying in a compact subset of $\Gamma$ excluding the origin $(0,0,0)$.

**Proof.** Summing up the three equations in (8) and denoting $N(t) = S(t) + I(t) + R(t)$, we have

$$b - (d + \delta)N \leq \frac{dN}{dt} = b - dN - \delta I \leq b - dN.$$  

Hence, by integration, we check

$$\frac{b}{d + \delta} + \left( N(0) - \frac{b}{d + \delta} \right) e^{-(d+\delta)t} \leq N(t) \leq \frac{b}{d} + \left( N(0) - \frac{b}{d} \right) e^{-dt}. $$

Hence,

$$\frac{b}{d + \delta} \leq \liminf_{t \to \infty} N(t) \leq \limsup_{t \to \infty} N(t) \leq \frac{b}{d},$$

which implies the conclusion. \qed

**Remark 3.2.** Lemma 3.1 indicates that we can study the dynamics of model (8) in the bounded set $\Gamma$ as defined in (13) in Section 2.
A main concern of deterministic epidemic models is to find conditions when a disease introduced into a community develops into a large outbreak, and if it does, the disease may become endemic. It’s worthy to note that, for stochastic models, all such questions are in terms of probabilities. A useful parameter in this regard for deterministic models, called the basic reproductive number, $R_0$, is defined as the expected number of secondary infective cases per primary case in a completely susceptible population [82, 83, 84]. For model (8), its basic reproduction number can be easily computed as

$$R_0 = \frac{k}{d + \mu + \delta},$$

$R_0$ acts as a sharp threshold between extinction and invasion of the disease.

And a simple calculation shows that model (8) has two equilibria points: the disease–free equilibrium $E_0 = (b/d, 0, 0)$ which exists for all parameter values and the endemic equilibrium state $E^* = (S^*, I^*, R^*)$ with

$$S^* = \frac{b(d + \gamma)}{d(d + \gamma) + \left(\frac{R_0 - 1}{\alpha}\right)^{1/h} (d + \delta)(d + \gamma) + d\mu},$$

$$I^* = \left(\frac{R_0 - 1}{\alpha}\right)^{1/h} S^*, \quad (15)$$

$$R^* = \frac{\mu}{d + \gamma} \left(\frac{R_0 - 1}{\alpha}\right)^{1/h} S^*,$$

which exists provided that $R_0 > 1$.

Now, we first consider the global stability of model (8) at the disease–free equilibrium $E_0$.

**Theorem 3.3.** The disease–free equilibrium $E_0$ is globally asymptotically stable if $R_0 \leq 1$ and unstable if $R_0 > 1$ in the set $\Gamma$.

**Proof.** We define the Lyapunov function by

$$V(S, I, R) = I.$$
Then the time derivative of $V$ along a solution of model (8) is

$$\frac{dV}{dt} = \frac{kS^h I}{S^h + \alpha I^h} - (d + \mu + \delta)I \leq (R_0 - 1)(d + \mu + \delta)I.$$ 

In view of $R_0 \leq 1$, we have $\frac{dV}{dt} \leq 0$.

Observe first $\lim_{t \to \infty} I(t) = 0$ if $R_0 < 1$. If $R_0 = 1$, we have $I = V(I) = V(0) = 0$. Hence, if $R_0 \leq 1$, we then obtain

$$\lim_{t \to \infty} I(t) = \lim_{t \to \infty} R(t) = 0.$$ 

Therefore, the set

$$\bar{\Gamma} = \left\{ (S, I, R) \in \Gamma : \frac{dV}{dt} = 0 \right\}$$

is the same as the set $\{(S, I, R) \in \Gamma : I = R = 0\}$. It is not difficult to see that on the set $\bar{\Gamma}$ we have $\lim_{t \to \infty} S(t) = \frac{b}{d}$. This implies that the largest compact invariant set of model (8) on the $\bar{\Gamma}$ is the singleton $E_0$. So LaSalle’s Invariance Principle [85, 86] shows that $E_0$ is globally asymptotically stable on the set $\Gamma$ if $R_0 \leq 1$.

When $R_0 > 1$, the Jacobian matrix of model (8) evaluated at $E_0$ is

$$J(E_0) = \begin{pmatrix}
-d & -k & \gamma \\
0 & k - d - \mu - \delta & 0 \\
0 & \mu & -d - \gamma
\end{pmatrix},$$

which has eigenvalues $-d < 0$, $k - d - \mu - \delta = (R_0 - 1)(d + \mu + \delta) > 0$, $-d - \gamma < 0$. Thus the disease–free equilibrium $E_0$ is unstable whenever $R_0 > 1$. The proof is complete.

For the stability of endemic equilibrium $E^*$ of model (8), we have the following theorem:

**Theorem 3.4.** If $R_0 > 1$, model (8) admits a unique endemic equilibrium $E^*$ which is globally asymptotically stable.
Proof. The Jacobian matrix of model (8) evaluated at \( E^* \) is

\[
J(E^*) = \begin{pmatrix}
-d - \frac{\alpha h k}{R_0^2} \left( \frac{R_0 - 1}{\alpha} \right)^{1+1/h} & \frac{k(h-1)(R_0-1)-k}{R_0^2} & \frac{\gamma}{\alpha} \\
\frac{\alpha h k}{R_0^2} \left( \frac{R_0 - 1}{\alpha} \right)^{1+1/h} & -\frac{h k (R_0 - 1)}{R_0^2} & 0 \\
0 & 0 & -d - \gamma
\end{pmatrix}
\]

The characteristic polynomial of \( J(E^*) \) is

\[
\lambda^3 + c_1 \lambda^2 + c_2 \lambda + c_3 = 0,
\]

where

\[
c_1 = -J_{11} - J_{22} + d + \gamma,
\]

\[
c_2 = -(d + \gamma)(J_{11} + J_{22}) + J_{11}J_{22} - J_{12}J_{21},
\]

\[
c_3 = (d + \gamma)(J_{11}J_{22} - J_{12}J_{21}) - J_{21} \gamma \mu.
\]

It is easy to verify that \( c_1, c_2, c_3 \) are positive, and \( c_1 c_2 - c_3 > 0 \) if \( R_0 > 1 \). Thus, it follows from the Routh–Hurwitz criterion that \( E^* \) is locally asymptotically stable.

By summing up all the equations of model (8) we find that the total population \( N = S + I + R \) satisfies the following equation:

\[
\frac{dN}{dt} = b - dN - \delta I.
\]

Thus, model (8) is equivalent to the following model:

\[
\begin{cases}
\frac{dN}{dt} = b - dN - \delta I, \\
\frac{dI}{dt} = \frac{k(N - I - R)^h I}{(N - I - R)^h + \alpha I^h} - (d + \mu + \delta) I, \\
\frac{dR}{dt} = \mu I - (d + \gamma) R.
\end{cases}
\]
For convenience, we change the variables by setting $x = N - N^*, y = I - I^*, z = R - R^*$, where $N^* = S^* + I^* + R^*$, then model (17) becomes

$$
\begin{align*}
\frac{dx}{dt} &= -dx - \delta y, \\
\frac{dy}{dt} &= \frac{hk(R_0 - 1)I^*}{R_0^2 S^*} (x - (1 + S^* I^*)^{-1}y - z), \\
\frac{dz}{dt} &= \mu y - (d + \gamma)z,
\end{align*}
$$

(18)

Now define the following function

$$
V(x, y, z) = \frac{1}{2}(k_1 x^2 + y^2 + k_2 z^2),
$$

where $k_1$ and $k_2$ are positive constants which will be determined later. Then the derivative of $V$ along the solution of model (18) is given by

$$
\frac{dV}{dt} = k_1 x \frac{dx}{dt} + y \frac{dy}{dt} + k_2 z \frac{dz}{dt}
$$

$$
= k_1 x(-dx - \delta y) + \frac{hk(R_0 - 1)I^*}{R_0^2 S^*} y (x - (1 + S^* I^*)^{-1}y - z) + k_2 z(\mu y - (d + \gamma)z)
$$

$$
= -dk_1 x^2 - \frac{hk(R_0 - 1)I^*}{R_0^2 S^*} (1 + S^* I^*)^{-1}y^2 - k_2(d + \gamma)z^2 + \left(\frac{hk(R_0 - 1)I^*}{R_0^2 S^*} - k_1 \delta\right) xy
$$

$$
+ \left(k_2 \mu - \frac{hk(R_0 - 1)I^*}{R_0^2 S^*}\right) yz.
$$

Let us choose $k_1$ and $k_2$ such that

$$
\frac{hk(R_0 - 1)I^*}{R_0^2 S^*} - k_1 \delta = 0,
$$

$$
k_2 \mu - \frac{hk(R_0 - 1)I^*}{R_0^2 S^*} = 0,
$$

then $k_1 = \frac{hk(R_0 - 1)I^*}{\delta R_0^2 S^*}$ and $k_2 = \frac{hk(R_0 - 1)I^*}{\mu R_0^2 S^*}$. Thus, we have

$$
\frac{dV}{dt} = -dk_1 x^2 - \frac{hk(R_0 - 1)I^*}{R_0^2 S^*} (1 + S^* I^*)^{-1}y^2 - k_2(d + \gamma)z^2 \leq 0.
$$

By applying the Lyapunov–LaSalle asymptotic stability theorem \[85, 86\], we can obtain that the endemic equilibrium $E^*$ of model (8) is globally asymptotically stable. \[\square\]
Remark 3.5. Theorem 3.3 and 3.4 indicate that the basic reproductive number $R_0$ is a sharp parameter determining when there is an endemic for model (8). In addition, Theorem 3.4 implies that model (8) is permanent with simple dynamics whenever $R_0 > 1$. However, this is not the case for the stochastic model (9) as we show in the following section.

4. Dynamics of the SDE model (9)

In this section, we first show that the existence of the unique positive global solution of SDE model (9).

**Theorem 4.1.** Consider model (9), for any given initial value $\left(S(0), I(0), R(0)\right) \in \Gamma$, there is a unique solution $\left(S(t), I(t), R(t)\right)$ on $t \geq 0$ and will remain in $\Gamma$ with probability one.

**Proof.** Since the coefficients of model (9) satisfy the local Lipschitz condition, there is a unique local solution on $[0, \tau_n]$, where $\tau_n$ is the explosion time. Therefore, the unique local solution to model (9) is positive by Itô’s formula. Now, let us show that this solution is global, i.e., $\tau_\infty = \infty$ a.s.

Let $n_0 > 0$ be sufficiently large for $S(0), I(0)$ and $R(0)$ lying within the interval $\left[\frac{1}{n_0}, n_0\right]$. For each integer $n > n_0$, define the stop-times

$$
\tau_n = \inf \left\{ t \in [0, \tau_e] : \min\{S(t), I(t), R(t)\} \leq \frac{1}{n} \text{ or } \max\{S(t), I(t), R(t)\} \geq n \right\}.
$$

Set $\inf \emptyset = \infty$ ($\emptyset$ represents the empty set). $\tau_n$ is increasing as $n \to \infty$. Let $\tau_\infty = \lim_{n \to \infty} \tau_n$, then $\tau_\infty \leq \tau_e$ a.s.

In the following, we need to show $\tau_\infty = \infty$ a.s. If this statement is violated, there exists a constant $T > 0$ and for any $\varepsilon \in (0, 1)$ such that $P\{\tau_\infty \leq T\} > \varepsilon$. As a consequence, there is an integer $n_1 \geq n_0$ such that

$$
P\{\tau_n \leq T\} \geq \varepsilon, \ n \geq n_1. \tag{19}
$$

Define a $C^3$–function $V : \mathbb{R}_+^3 \to \mathbb{R}_+$ by

$$
V(S, I, R) = \left(S - 1 - \ln S\right) + \left(I - 1 - \ln I\right) + \left(R - 1 - \ln R\right).
$$
which is a non-negativity function.

If \((S(t), I(t), R(t)) \in \mathbb{R}^3_+\), by the Itô formula, we compute

\[
dV = \left(1 - \frac{1}{S}\right) dS + \frac{1}{2S^2} (dS)^2 + \left(1 - \frac{1}{I}\right) dI + \frac{1}{2I^2} (dI)^2 + \left(1 - \frac{1}{R}\right) dR + \frac{1}{2R^2} (dR)^2
\]

\[
\triangleq LV dt + \sigma_1 (S - 1) dB_1(t) + \sigma_2 (I - 1) dB_2(t) + \sigma_3 (R - 1) dB_3(t),
\]

(20)

where

\[
LV = \left(1 - \frac{1}{S}\right) \left( b - dS - \frac{kS^h I}{S^h + \alpha I^h} + \gamma R \right) + \left(1 - \frac{1}{I}\right) \left( \frac{kS^h I}{S^h + \alpha I^h} - (d + \mu + \delta) I \right) + \left(1 - \frac{1}{R}\right) (\mu I - (d + \gamma) R) + \frac{\sigma_1^2 + \sigma_2^2 + \sigma_3^2}{2} + \frac{kS^{h-1} I}{S^h + \alpha I^h} - \frac{kS^h}{S^h + \alpha I^h} - d(S + I + R)
\]

\[
- \delta I - \frac{b}{S} - \frac{\gamma R}{S} - \frac{\mu I}{R}
\]

\[
< b + 3d + \mu + \delta + \gamma + \frac{\sigma_1^2 + \sigma_2^2 + \sigma_3^2}{2} + \frac{kS^{h-1} I}{S^h + \alpha I^h}.
\]

Now if \(S \leq I\), one has

\[
\frac{kS^{h-1} I}{S^h + \alpha I^h} = \frac{k}{\left(\frac{I}{S}\right)^{-1} + \alpha \left(\frac{I}{S}\right)^{h-1}} \leq \frac{k}{\alpha}.
\]

If \(S > I\), one has

\[
\frac{kS^{h-1} I}{S^h + \alpha I^h} = \frac{I}{S} \cdot \frac{kS^h}{S^h + \alpha I^h} < k.
\]

Hence there exists a suitable constant \(M > 0\) independent of \(S, I, R\) and \(t\) such that \(LV \leq M\).

Substituting this inequality into (20), we see that

\[
dV(S, I, R) \leq MdI + \sigma_1 (S - 1) dB_1(t) + \sigma_2 (I - 1) dB_2(t) + \sigma_3 (R - 1) dB_3(t),
\]

which implies

\[
\int_0^{\tau_{n,T}} dV(S(r), I(r), R(r)) \leq \int_0^{\tau_{n,T}} MdI + \int_0^{\tau_{n,T}} \sigma_1 (S(r) - 1) dB_1(r)
\]

\[
+ \int_0^{\tau_{n,T}} \sigma_2 (I(r) - 1) dB_2(r) + \int_0^{\tau_{n,T}} \sigma_3 (R(r) - 1) dB_3(r),
\]

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where $\tau_n \wedge T = \min\{\tau_n, T\}$. Taking the expectations of the above inequality leads to

$$
\mathbb{E}V(S(\tau_n \wedge T), I(\tau_n \wedge T), R(\tau_n \wedge T)) \leq V(S(0), I(0), R(0)) + MT. \quad (21)
$$

Set $\Omega_n = \{\tau_n \leq T\}$ for $n \geq n_1$ and from (19), we have $\mathcal{P}(\Omega_n) \geq \varepsilon$. Note that for every $\omega \in \Omega_n$, there is at least one of $S(\tau_n, \omega) I(\tau_n, \omega)$ and $R(\tau_n, \omega)$ equaling either $n$ or $1/n$, hence

$$
V(S(\tau_n, \omega), I(\tau_n, \omega), R(\tau_n, \omega)) \geq (n - 1 - \ln n) \wedge \left(\frac{1}{n} - 1 - \ln \frac{1}{n}\right).
$$

It then follows from (21) that

$$
V(S(0), I(0), R(0)) + MT \geq \mathbb{E} \left[ I_{\Omega_n(\omega)} V(S(\tau_n), I(\tau_n), R(\tau_n)) \right]
$$

$$
\geq \varepsilon \left( (n - 1 - \ln n) \wedge \left(\frac{1}{n} - 1 - \ln \frac{1}{n}\right) \right),
$$

where $I_{\Omega_n}$ is the indicator function of $\Omega_n$. Letting $n \to \infty$, we have that

$$
\infty > V(S(0), I(0), R(0)) + MT = \infty \text{ a.s.},
$$

is a contradiction, then we must have $\tau_\infty = \infty$. Therefore, the solution of model (9) will not explode at a finite time with probability one. This completes the proof.

\[\square\]

4.1. Stochastic ultimate boundedness and permanence

Theorem 4.1 shows that the solutions to model (9) will remain in $\Gamma$. Generally speaking, the existence and uniqueness of the solution are not enough but the property of boundedness and permanence are more desirable since they mean the long time survival in the population dynamics. Thus, we show the following theorem:

**Theorem 4.2.** The solutions of model (9) are stochastically ultimately bounded for any initial value $(S(0), I(0), R(0)) \in \Gamma$.

**Proof.** Define

$$
V(S, I, R) = S^\theta + I^\theta + R^\theta,
$$

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Note that this implies \( \lim_{\varepsilon \to 0} \frac{1}{\varepsilon} \int_0^1 \sigma^2 \, dW_s \to 0 \) almost surely as \( \varepsilon \to 0 \).

By the Itô formula, we have
\[
dV = LV \, dt + \theta(\sigma_1 S^\theta \, dB_1(t) + \sigma_2 I^\theta \, dB_2(t) + \sigma_3 R^\theta \, dB_3(t)),
\]
where
\[
LV = \theta S^{\theta-1} \left( b - dS - \frac{kS^h I}{S^h + \alpha I^h} + \gamma R \right) + \theta I^{\theta-1} \left( \frac{kS^h I}{S^h + \alpha I^h} - (d + \mu + \delta) I \right)
\]
\[+ \theta R^{\theta-1}(\mu I - (d + \gamma)R) + \frac{\theta(\theta - 1)}{2} \left( \sigma_1^2 S^{2\theta} + \sigma_2^2 I^{2\theta} + \sigma_3^2 R^{2\theta} \right).\]

Now, by the Itô formula, we have
\[
d[e^t V(S, I, R)] = e^t (V + LV) \, dt + e^t \theta(\sigma_1 S^\theta dB_1(t) + \sigma_2 I^\theta dB_2(t) + \sigma_3 R^\theta dB_3(t))
\]
\[
\quad \leq M_1 e^t dt + e^t \theta(\sigma_1 S^\theta dB_1(t) + \sigma_2 I^\theta dB_2(t) + \sigma_3 R^\theta dB_3(t)),
\]
and \( M_1 > 0 \) is a suitable constant.

Let \( k_0 > 0 \) be sufficiently large for every component of \((S(0), I(0), R(0))\) lying within the interval \([1/k_0, k_0]\). For each integer \( k \geq k_0 \), define the stopping time
\[
\tau_k = \inf \{ t \geq 0 : S(t), I(t), R(t) \notin [1/k, k] \}.
\]
Clearly \( \tau_k \to \infty \) almost surely as \( k \to \infty \). It then follows from (22) that
\[
\mathbb{E}[e^{t \wedge \tau_k} V(S(t \wedge \tau_k), I(t \wedge \tau_k), R(t \wedge \tau_k)] \leq V(S(0), I(0), R(0)) + M_1 \mathbb{E} \int_0^{t \wedge \tau_k} e^s ds.
\]
Letting \( k \to \infty \) yields
\[
e^t \mathbb{E} V(S(t), I(t), R(t)) \leq V(S(0), I(0), R(0)) + M_1 (e^t - 1).
\]
This implies
\[
\mathbb{E} V(S(t), I(t), R(t)) \leq e^{-t} V(S(0), I(0), R(0)) + M_1.
\]

Note that
\[
|x(t)|^\theta = (S^2(t) + I^2(t) + R^2(t))^{\frac{\theta}{2}} \leq 3^{\frac{\theta}{2}} \max \{ S^\theta(t), I^\theta(t), R^\theta(t) \} \leq 3^{\frac{\theta}{2}} V(S(t), I(t), R(t)).
\]
So we get
\[
\mathbb{E}|x(t)|^\theta \leq 3^\frac{\theta}{2}(e^{-t}V(S(0), I(0), R(0)) + M_1).
\]

This implies
\[
\limsup_{t \to \infty} \mathbb{E}|x(t)|^\theta \leq 3^\frac{\theta}{2} M_1 < +\infty.
\]

As a result, there exists a positive constant \( \varphi_1 \) such that
\[
\limsup_{t \to \infty} \mathbb{E}(\sqrt{|x(t)|}) < \varphi_1.
\]

Now, for any \( \varepsilon > 0 \), let \( \varrho = \frac{\varphi_1^2}{\varepsilon^2} \), then by Chebyshev’s inequality,
\[
\mathcal{P}\{|x(t)| > \varrho\} \leq \frac{\mathbb{E}(\sqrt{|x(t)|})}{\sqrt{\varrho}}.
\]
Hence,
\[
\limsup_{t \to \infty} \mathcal{P}\{|x(t)| > \varrho\} \leq \frac{\varphi_1}{\sqrt{\varrho}} = \varepsilon,
\]
which yields the required assertion. \( \square \)

We are now in the position to show the stochastic permanence. Let us present some hypothesis and a useful lemma first.

**Lemma 4.3.** Assume \( d + \delta < b \). For any initial value \((S(0), I(0), R(0)) \in \Gamma\), the solution \((S(t), I(t), R(t))\) satisfies that
\[
\limsup_{t \to \infty} \mathbb{E}\left( \frac{1}{|x(t)|^\rho} \right) \leq H,
\]
where \( \rho \) is an arbitrary positive constant satisfying
\[
d + \delta + \frac{\rho + 1}{2} \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\} < b,
\]
and
\[
H = \frac{3^\rho(C_2 + 4\eta C_1)}{4\eta C_1} \max\left\{ 1, \left( \frac{2C_1 + C_2 + \sqrt{C_2^2 + 4C_1^2} C_2}{2C_1} \right)^{\rho - 2} \right\}, \tag{24}
\]
in which \( \eta \) is an arbitrary positive constant satisfying

\[
\rho(d + \delta) + \eta + \frac{\rho(\rho + 1)}{2} \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\} < \rho b,
\]

and

\[
C_1 = \rho(b - d - \delta) - \frac{\rho(\rho + 1)}{2} \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\} - \eta,
\]

\[
C_2 = \rho(d + \delta) + \frac{\rho(\rho + 1)}{2} \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\} + 2\eta.
\]

**Proof.** Set the function \( U(S, I, R) = \frac{1}{S + I + R} \) for \((S(t), I(t), R(t)) \in \Gamma\), by the Itô formula, we have

\[
dU = -U^2 (b - d(S + I + R) - \delta I) dt + U^3 (\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2) dt
\]

\[
- U^2 (\sigma_1 S dB_1(t) + \sigma_2 I dB_2(t) + \sigma_3 R dB_3(t))
\]

\[
= LU dt - U^2 (\sigma_1 S dB_1(t) + \sigma_2 I dB_2(t) + \sigma_3 R dB_3(t)),
\]

where

\[
LU = -U^2 (b - d(S + I + R) - \delta I) + U^3 (\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2).
\]

Then we choose a positive constant \( \rho \) such that it satisfies (23). Applying the Itô formula again, we can get

\[
L[(1 + U)^\rho] = \rho(1 + U)^{\rho - 1} LU + \frac{\rho(\rho - 1)}{2} U^4 (1 + U)^{\rho - 2} (\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2)
\]

\[
= \rho(1 + U)^{\rho - 2} \Phi,
\]

where

\[
\Phi = -U^3 \left( b - d \frac{U}{U} - \delta I \right) - U^2 \left( b - d \frac{U}{U} - \delta I \right) + U^3 (\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2)
\]

\[
+ \frac{\rho + 1}{2} U^4 (\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2)
\]

\[
\leq -(b - d - \delta) U^2 + (d + \delta) U + U^3 (\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2)
\]

\[
+ \frac{\rho + 1}{2} U^4 (\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2).
\]
Using the facts that
\[ U^3(\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2) < \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\} U, \]
and
\[ U^4(\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2) < (\max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\}) U^2, \]
hence,
\[ \Phi \leq -\left(b - d - \frac{\rho + 1}{2} \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\}\right) U^2 + (d + \delta + (\max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\}) U. \]

Now, let \( \eta > 0 \) be sufficiently small such that it satisfies (25), by the Itô formula, then
\[
L[e^{kt}(1 + U)^\rho] = \eta e^{nt}(1 + U)^\rho + e^{kt} L(1 + U)^\rho \\
= e^{nt}(1 + U)^{\rho-2} (\eta(1 + U)^2 + \rho \Phi) \\
\leq e^{nt}(1 + U)^{\rho-2} (-C_1 U^2 + C_2 u + \eta) \\
\leq H_1 e^{nt},
\]
where \( H_1 = \frac{C_2 + 4\eta C_1}{4C_1} \max\left\{1, \left(\frac{2C_1 + C_2 + \sqrt{C_2^2 + 4C_1 C_2}}{2C_1}\right)^{\rho-2}\right\} \) and \( C_1, C_2 \) have been defined in the statement of the theorem. Thus
\[
\mathbb{E}[e^{nt}(1 + U)^\rho] \leq (1 + U(0))^\rho + \frac{H_1}{\eta} e^{nt}.
\]
So we can have
\[
\limsup_{t \to \infty} \mathbb{E}[U(t)^\rho] \leq \limsup_{t \to \infty} \mathbb{E}(1 + U)^\rho \leq \frac{H_1}{\eta}.
\]
For \((S, I, R) \in \mathbb{R}^3_+\), noting that \((S+I+R)^\rho \leq 3^\rho(S^2+I^2+R^2)^{\rho/2} \leq 3^\rho|\rho(x(t)|^\rho, \) consequently, one has
\[
\limsup_{t \to \infty} \mathbb{E} \left[ \frac{1}{|x(t)|^\rho} \right] \leq 3^\rho \limsup_{t \to \infty} \mathbb{E}[U(t)^\rho] \leq \frac{3^\rho H_1}{\eta} = H,
\]
which completes the proof.
Applying Chebyshev inequality, Theorem 4.2 and Lemma 4.3 together, we immediately obtain the following result.

**Theorem 4.4.** Assume \( d + \delta + \frac{1}{2} \max\{\sigma^2_1, \sigma^2_2, \sigma^2_3\} < b \), then the solutions of model (9) is stochastically permanent.

**Proof.** By Theorem 4.2, we have \( \mathcal{P}\{|x(t)| > \varrho\} \leq \varepsilon \). This implies
\[
\mathcal{P}\{|x(t)| \leq \varrho\} \geq 1 - \varepsilon,
\]
which follows that
\[
\liminf_{t \to \infty} \mathcal{P}\{|x(t)| \leq \varrho\} \geq 1 - \varepsilon.
\]

By Lemma 4.3, we know
\[
\limsup_{t \to \infty} \mathbb{E}\left( \frac{1}{|x(t)|^\rho} \right) \leq H.
\]

Now, for any \( \varepsilon > 0 \), let \( \chi = \frac{\varepsilon^\rho}{H^\rho} \). Then
\[
\mathcal{P}\{|x(t)| < \chi\} = \mathcal{P}\{1/|x(t)| > 1/\chi\} \leq \frac{\mathbb{E}(1/|x(t)|^\rho)}{\chi^{1/\rho}} = \chi^{1/\rho} \mathbb{E}(1/|x(t)|^\rho).
\]
Hence,
\[
\limsup_{t \to \infty} \mathcal{P}\{|x(t)| < \chi\} \leq \chi^{1/\rho} H = \varepsilon,
\]
which follows that
\[
\liminf_{t \to \infty} \mathcal{P}\{|x(t)| \geq \chi\} \geq 1 - \varepsilon.
\]
The proof is complete. \( \square \)

From Theorem 4.2 and Theorem 4.4, we can conclude the following corollary.

**Corollary 4.5.** The set \( \Gamma \) is almost surely positively invariant of model (9), that is, if \( (S(0), I(0), R(0)) \in \Gamma \), then \( \mathcal{P}\{(S(t), I(t), R(t)) \in \Gamma\} = 1 \) for all \( t \geq 0 \).
Remark 4.6. Theorem 4.4 only tells us the behaviour of the solution \( x(t) = (S(t), I(t), R(t)) \) of model (9). In other words, it says that, if \( d + \delta + \frac{1}{2} \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\} < b \) holds, then one can get
\[
P\{\sqrt{S^2(t) + I^2(t) + R^2(t)} > 0\} = 1.
\]
It doesn’t give us any information whether \( S(t)I(t)R(t) > 0 \) or not. One can easily give an example that the model (9) is stochastically permanent while the infective \( I(t) \) dies out.

4.2. Disease-free dynamics

One of the main concerns in epidemiology is how we can regulate the disease dynamics so that the disease will be eradicated in a long term. Theorem 3.3 indicates that if \( R_0 < 1 \) then disease-free equilibrium \( E_0 = (b/d, 0, 0) \) of the deterministic model (8) is globally asymptotically stable. However, the condition of \( R_0 < 1 \) does not imply the extinction of disease for the stochastic model (9). In this subsection, we provide a sharp result of the extinction of disease for the stochastic model (9) and the related disease-free stationary distribution.

Theorem 4.7. If
\[
R^*_0 := R_0 - \frac{\sigma^2}{2(d + \mu + \delta)} = R_0 \left(1 - \frac{\sigma^2}{2k}\right) < 1, \tag{26}
\]
then for any given initial value \((S(0), I(0), R(0)) = (S_0, I_0, R_0) \in \Gamma, I(t) \) almost surely tends to zero exponentially.

Proof. By the Itô formula, we have
\[
d \ln I(t) = \left(\frac{kS^h(t)}{S^h(t) + \alpha I^h(t)} - (d + \mu + \delta) - \frac{\sigma_2^2}{2}\right) dt + \sigma_2 dB_2(t). \tag{27}
\]
Hence,

$$
\ln I(t) = \ln I_0 + \int_0^t \left( \frac{kS(s) h}{S(h(s) + \alpha I^h(s))} - (d + \mu + \delta) - \frac{\sigma_2^2}{2} \right) ds + \int_0^t \sigma_2 dB_2(s)
\leq \ln I_0 + \int_0^t \left( k - (d + \mu + \delta) - \frac{\sigma_2^2}{2} \right) ds + \int_0^t \sigma_2 dB_2(s)
= \ln I_0 + \left( k - (d + \mu + \delta) - \frac{\sigma_2^2}{2} \right) t + G(t),
$$

(28)

where $G(t)$ is a martingale defined by

$$
G(t) = \int_0^t \sigma_2 dB_2(s).
$$

This implies

$$
\langle G, G \rangle_t = \int_0^t \sigma_2^2 ds = \sigma_2^2 t.
$$

By the strong law of large numbers for martingales [11], we have

$$
\limsup_{t \to \infty} \frac{G(t)}{t} = 0 \ a.s.
$$

It finally follows from (28) by dividing $t$ on the both sides and then letting $t \to \infty$ that

$$
\limsup_{t \to \infty} \frac{\ln I(t)}{t} \leq k - (d + \mu + \delta) - \frac{\sigma_2^2}{2} < 0 \ a.s.
$$

(29)

which is the required assertion.

\[ \square \]

**Remark 4.8.** Theorem 4.7 implies that $I(t)$ goes extinct exponentially a.s. whenever $R_0^S < 1$ with the consequence that $R$-class also goes to extinction a.s. In summary, Theorem 4.7 gives a sharp condition when solutions to model (9) are converging to the disease-free dynamics a.s.

**Remark 4.9.** It’s worthy to note that, the infectives $I(t)$ of the deterministic model (8) die out whenever $R_0 \leq 1$ (c.f., Theorem 3.3) while the disease $I(t)$ is persistent whenever $R_0 > 1$ (c.f., Theorem 3.4). However, this result does not apply to the stochastic model (9) according to Theorem 4.7. We can easily find examples when $R_0 > 1$ but $R_0^S < 1$ such that
The disease goes to extinct exponentially a.s since \( R_0^* = R_0 - \frac{\sigma_0^2}{2(d + \mu + \delta)} \). This implies that large environment fluctuations in \( I \)-class can suppress the outbreak of disease, which partially answer our proposed questions in the introduction.

Next, we will focus on the stochastic dynamics of \( S(t) \) when \( R_0^* < 1 \).

**Theorem 4.10.** If \( R_0^* < 1 \), the distribution of the process \( \psi(t) = \ln S(t) \) converges weakly to the measure which has the density

\[
f_*(\psi) = K_1 \exp \left\{ -\frac{1}{\sigma_1^2} (2b \exp\{-\psi\} + 2d\psi + \sigma_1\psi) \right\},
\]

where \( K_1 = \left( \int_{-\infty}^{\infty} \exp \left\{ -\frac{1}{\sigma_1^2} (2b \exp\{-\psi\} + 2d\psi + \sigma_1\psi) \right\} d\psi \right)^{-1} \).

**Proof.** According to Theorem 4.7, if \( R_0^* < 1 \), we get \( \lim_{t \to \infty} I(t) = 0 \), a.s. . Otherwise, from the last equation of model (9), we easily get \( \lim_{t \to \infty} R(t) = 0 \). That is to say, for \( \forall 0 < \varepsilon \ll 1 \), there exists a constant \( T_1 = T_1(\omega) \) and a set \( \Omega_\varepsilon \) such that \( P(\Omega_\varepsilon) > 1 - \varepsilon \), \( 0 \leq I(t) \leq \frac{\varepsilon}{k} \) and \( 0 \leq R(t) \leq \frac{\varepsilon}{\gamma} \) for \( t > T_1 \) and \( \omega \in \Omega_\varepsilon \). Then

\[
b - dS - \varepsilon + \sigma_1 S dB_1(t) \leq dS(t) \leq b - dS + \varepsilon + \sigma_1 S dB_1(t).
\]

For the arbitrary of \( \varepsilon \), we get

\[
dS(t) = b - dS + \sigma_1 S dB_1(t).
\]

By putting \( S(t) = \exp\{\psi(t)\} \), we have

\[
d\psi(t) = \left( b \exp\{-\psi(t)\} - d - \frac{\sigma_1^2}{2} \right) dt + \sigma_1 dB_1(t).
\]

Then the above equation has a unique stationary distribution which has a density \( f_*(\psi) \) satisfying the Fokker–Planck equation

\[
\frac{1}{2}\sigma_1^2 \frac{d^2 f_*(\psi)}{d\psi^2} - \frac{d}{d\psi} \left( \left( b \exp\{-\psi\} - d - \frac{\sigma_1^2}{2} \right) f_*(\psi) \right) = 0.
\]
The general solution to the equation (32) is

$$f_*(\psi) = \exp \left\{ -\frac{1}{\sigma_1^2} (2b \exp\{\psi\} + 2d\psi + \sigma_1\psi) \right\} \left( K_1 - K_2 \int_0^\infty \exp \left\{ \frac{1}{\sigma_1^2} (2b \exp\{\psi\} + 2d\psi + \sigma_1\psi) \right\} \, dr \right),$$

where $K_1, K_2$ are two constants. It is easy to follow from the conditions

$$f_*(\psi) \geq 0, \quad \int_{-\infty}^{\infty} f_*(\psi) d\psi = 1,$$

that $K_2 = 0$ and

$$K_1 = \left( \int_{-\infty}^{\infty} \exp \left\{ -\frac{1}{\sigma_1^2} (2b \exp\{-\psi\} + 2d\psi + \sigma_1\psi) \right\} \, d\psi \right)^{-1}. $$

Therefore,

$$f_*(\psi) = K_1 \exp \left\{ -\frac{1}{\sigma_1^2} (2b \exp\{-\psi\} + 2d\psi + \sigma_1\psi) \right\}. $$

By the existence of a stationary distribution [87], $\psi(t) = \ln S(t)$ converges to the measure with the density $f_*(\psi)$ as $t \to \infty$.

**Remark 4.11.** Theorem 4.10 indicates that if $R_0^* < 1$, then we have $\lim_{t \to \infty} I(t) = 0$ and $\lim_{t \to \infty} R(t) = 0$ a.s. This leads to the situation when the susceptible $S(t)$ has a unique stationary distribution and $\ln S(t)$ converges weakly to the measure with the density distribution function (30).

### 4.3. Endemic dynamics

The deterministic SIRS model (8) is globally stable at its endemic equilibrium $E^*$ whenever $R_0 = \frac{k}{d + \mu + \delta} > 1$. We will first study the asymptotic behavior of the solutions to the SDE model (9) around $E^*$ of model (8).

#### 4.3.1. Stochastic asymptotic stability

**Theorem 4.12.** Assume $d + \delta + \frac{1}{2} \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\} < b$. If the following two conditions are satisfied,

(i) $R_0^S > 1$;
(ii) \( \sigma_1^2 < d \left( 1 + \frac{2d}{\gamma} \right), \quad \sigma_2^2 < d + \delta + \frac{2d}{\gamma}(d + \mu + \delta), \quad \sigma_3^2 < d + \frac{\delta}{\mu}(d + \gamma), \)
then for any initial value \((S(0), I(0), R(0)) \in \Gamma,\) the solution \((S(t), I(t), R(t))\) of model \((9)\) has the property

\[
\limsup_{t \to \infty} \frac{1}{t} \mathbb{E} \int_0^t \left[ (S(\tau) - \theta_1 S^*)^2 + (I(\tau) - \theta_2 I^*)^2 + (R(\tau) - \theta_3 R^*)^2 \right] d\tau \leq \frac{\Psi}{\Theta},
\]

where

\[
\begin{align*}
\theta_1 &= \frac{d(\gamma + 2d)}{d(\gamma + 2d) - \gamma \sigma_1^2}, \\
\theta_2 &= \frac{\gamma(d + \delta) + 2d(d + \mu + \delta)}{\gamma(d + \delta) + 2d(d + \mu + \delta) - \gamma \sigma_2^2}, \\
\theta_3 &= \frac{\mu d + \delta(d + \gamma)}{\mu d + \delta(d + \gamma) - \mu \sigma_3^2},
\end{align*}
\]

and

\[
\begin{align*}
\Psi &= \frac{d^2(\gamma + 2d)}{d(\gamma + 2d) - \gamma \sigma_1^2} S^* + \frac{\sigma_2^2[\gamma(d + \delta) + 2d(d + \mu + \delta)]}{\gamma(d + \delta) + 2d(d + \mu + \delta) - \gamma \sigma_2^2} I^* + \frac{\sigma_3^2 d(\mu d + \delta(d + \gamma))}{\mu d + \delta(d + \gamma) - \mu \sigma_3^2} R^* \\
&\quad + \frac{\sigma_2^2 d^{h-1}(S^* + \alpha I^*)}{2k h \beta^{h-1}} I^*.
\end{align*}
\]

**Proof.** Since \( R_0^S > 1, \) hence \( R_0 = R_0^S + \frac{\sigma_2^2}{2(d + \mu + \delta)} > 1, \) there is an endemic equilibrium \( E^* = (S^*, I^*, R^*) \) of model \((8)\). Then we have

\[
b = d S^* + \frac{k S^* I^*}{S^* + \alpha I^*} - \gamma R^*, \quad \frac{k S^* I^*}{S^* + \alpha I^*} = (d + \mu + \delta) I^*, \quad \mu I^* = (d + \gamma) R^*. \tag{33}
\]

Set

\[
\tilde{V}(S, I, R) = \frac{1}{2}(S - S^* + I - I^* + R - R^*)^2 + \frac{\lambda_1}{2}(S - S^* + I - I^*)^2 \\
+ \lambda_2 \left( I - I^* - I^* \ln \frac{I}{I^*} \right) + \frac{\lambda_3}{2}(R - R^*)^2
\]

\[
:= V_1 + \lambda_1 V_2 + \lambda_2 V_3 + \lambda_3 V_4
\]
where $\lambda_1, \lambda_2, \lambda_3$ are positive constants to be determined later. $\hat{V}$ is positive definite. By the Itô formula, we have

$$dV_1 = \left[(S - S^* + I - I^* + R - R^*)(b - dS - (d + \delta)I - dR) + \frac{1}{2}(\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2)\right] dt$$

$$+ (S - S^* + I - I^* + R - R^*)(\sigma_1 S dB_1(t) + \sigma_2 I dB_2(t) + \sigma_3 R dB_3(t))$$

$$:= LV_1 dt + W_1,$$

$$dV_2 = \left[(S - S^* + I - I^*)(b - dS - (d + \mu + \delta)I + \gamma R) + \frac{1}{2}(\sigma_1^2 S^2 + \sigma_2^2 I^2)\right] dt$$

$$+ (S - S^* + I - I^*)(\sigma_1 S dB_1(t) + \sigma_2 I dB_2(t))$$

$$:= LV_2 dt + W_2,$$

$$dV_3 = \left[(I - I^*) \left(\frac{k S^h}{S^h + \alpha} - (d + \mu + \delta)\right) + \frac{1}{2} I^* \sigma_2^2\right] dt + (I - I^*) \sigma_2 dB_2(t)$$

$$:= LV_3 dt + W_3,$$

$$dV_4 = \left[(R - R^*)(\mu I - (d + \gamma)R) + \frac{1}{2} \sigma_3^2 R^2\right] dt + (R - R^*) \sigma_3 dB_3(t)$$

$$:= LV_4 dt + W_4.$$

It follows from (33), we compute in detail

$$LV_1 = (S - S^* + I - I^* + R - R^*)(-d(S - S^*) - (d + \delta)(I - I^*) - d(R - R^*))$$

$$+ \frac{1}{2}(\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2)$$

$$= -d(S - S^*)^2 - (d + \delta)(I - I^*)^2 - d(R - R^*)^2 - (2d + \delta)(S - S^*)(I - I^*)$$

$$- 2d(S - S^*)(R - R^*) - (2d + \delta)(I - I^*)(R - R^*) + \frac{1}{2}(\sigma_1^2 S^2 + \sigma_2^2 I^2 + \sigma_3^2 R^2)$$

30
\( LV_2 = (S - S^* + I - I^*)(-d(S - S^*) - (d + \mu + \delta)(I - I^*) + d(R - R^*)) \\
+ \frac{1}{2}(\sigma_1^2 I^2 + \sigma_2^2 I^2) \\
= -d(S - S^*)^2 - (d + \mu + \delta)(I - I^*)^2 - (2d + \mu + \delta)(S - S^*)(I - I^*) \\
+ \gamma(S - S^*)(R - R^*) + \gamma(I - I^*)(R - R^*) + \frac{1}{2}(\sigma_1^2 I^2 + \sigma_2^2 I^2), \)

\( LV_3 = k(I - I^*) \left( \frac{S^h}{S^h + \alpha I^h} - \frac{S^{*h}}{S^{*h} + \alpha I^{*h}} \right) + \frac{1}{2}I^* \sigma_2^2 \)

\[ \begin{align*}
&= \frac{k S^h (I - I^*) [S^{*h} - S^h + \alpha (I^{*h} - I^h)]}{(S^h + \alpha I^h)(S^{*h} + \alpha I^{*h})} + \frac{k (S^h - S^{*h})(I - I^*)}{S^{*h} + \alpha I^{*h}} + \frac{1}{2}I^* \sigma_2^2 \\
&\leq \frac{k S^h A(S^* - S)(I^* - I)}{(S^h + \alpha I^h)(S^{*h} + \alpha I^{*h})} + \frac{k A(S - S^*)(I - I^*)}{S^{*h} + \alpha I^{*h}} + \frac{1}{2}I^* \sigma_2^2, 
\end{align*} \]

where

\[ A = S^{h-1} + S^{h-2}S^* + S^{h-3}S^{*2} + \cdots + SS^{*h-2} + S^{*h-1} \leq \frac{hh^{h-1}}{d^{h-1}}, \]

hence,

\[ LV_3 \leq \frac{kh^{h-1}}{d^{h-1}(S^{*h} + \alpha I^{*h})} (S - S^*)(I - I^*) \text{sgn}(S - S^*)(I - I^*), \]

also,

\[ LV_4 = (R - R^*)[(\mu(I - I^*) - (d + \gamma)(R - R^*)) + \frac{1}{2}\sigma_3^2 R^2] \]

\[ = \mu(R - R^*)(I - I^*) - (d + \gamma)(R - R^*)^2 + \frac{1}{2}\sigma_3^2 R^2. \]

Choose

\[ \lambda_1 = \frac{2d}{\gamma}, \lambda_2 = \frac{d^{h-1}(S^{*h} + \alpha I^{*h})[2d + \delta + \lambda_1(2d + \mu + \delta)]}{kh^{h-1}}, \lambda_3 = \frac{\delta}{\mu}. \]
through dividing both sides of (35) by $t$ and letting $t \to \infty$, we obtain

$$
\limsup_{t \to \infty} \frac{1}{t} \mathbb{E} \int_0^t \left[ \left( S(\tau) - \frac{d (1 + \lambda_1)}{d (1 + \lambda_1) - \sigma_1^2 S^*} \right)^2 + \left( I(\tau) - \frac{d + \delta + \lambda_1(d + \mu + \delta)}{d + \delta + \lambda_1(d + \mu + \delta) - \sigma_2^2} \times \frac{I^*}{I^*} \right) \right] d\tau

+ \limsup_{t \to \infty} \frac{1}{t} \mathbb{E} \int_0^t \left( R(\tau) - \frac{d + \lambda_3(d + \gamma)}{d + \lambda_3(d + \gamma) - \sigma_3^2} \times \frac{R^*}{R^*} \right)^2 d\tau \leq \frac{B}{\Theta}.
$$

The proof is complete. \hfill \Box
Remark 4.13. Here we briefly discuss our analytic results: Theorem 4.4 shows that the SDE model (9) is stochastically permanent whenever the equality $d + \delta + \frac{1}{2} \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\} < b$ holds; Theorem 4.12 provides us sharper conditions on the behavior of the stochastic persistence, i.e., that whether the solution of the stochastic system (9) is oscillating around the endemic $E^*$ with the amplitudes of vibration depends on the values of $\sigma_i (i = 1, 2, 3)$. In addition, Theorem 4.12 suggests the possibility of endemic stationary distribution under different conditions where we have further analytic study in the next subsection and numerical simulations in Section 5.

4.3.2. Ergodic property

As we discuss in Remark 4.13, Theorem 4.12 suggests that the SDE model (9) may have the endemic stationary distribution when the conditions of Theorem 4.12 are satisfied. In order to rigorously show this result, we study the ergodic property of model (9) in this subsubsection.

Before giving the main theorem about the endemic stationary distribution, we start with the following Lemmas by Khasminskii [88].

Let $X(t)$ be a regular temporally homogeneous Markov process in $\Omega \subset \mathbb{R}_+^3$ described by the stochastic differential equation

$$dX(t) = f(X,t)dt + \sum_{r=1}^{3} \sigma_r(X)dB_r(t)$$

and the diffusion matrix is defined as follows

$$A(x) = ((a_{ij}(x))), \quad a_{ij}(x) = \sum_{r=1}^{3} \sigma_r^i(x)\sigma_r^j(x).$$

For model (9), the diffusion matrix is

$$A(x) = \text{diag}\left(\sigma_1^2I^2, \sigma_2^2I^2, \sigma_3^2I^2\right).$$

Lemma 4.14. [88] We assume that there exists a bounded domain $U \subset \Omega$ with regular boundary, which has the following properties:
(i) In the domain $U$ and some neighborhood thereof, the smallest eigenvalue of the diffusion matrix $A(x)$ is bounded away from zero;

(ii) If $x \in \Omega \setminus U$, the mean time $\tau$ at which a path issuing from $x$ reaches the set $U$ is finite, and $\sup_{x \in K} \mathbb{E}_x \tau < \infty$ for every compact subset $K \subset \Omega$.

**Lemma 4.15.** [88] Suppose that Lemma 4.14 holds. Then the Markov process $X(t)$ has a unique stationary distribution $\pi(\cdot)$. Moreover, if $f(X,t)$ is a function integrable with respect to the measure $\pi$, then

$$P_x \left\{ \lim_{T \to \infty} \frac{1}{T} \int_0^T f(X(t)) dt = \int_{\Omega} f(t) \pi(dx) \right\} = 1,$$

for all $x \in \Omega$.

We here omit the proofs of the above Lemmas, and the proofs can be found in [88].

Applying these Lemmas, we get the following result.

**Theorem 4.16.** Consider the stochastic model (9) with initial condition $(S(0), I(0), R(0)) \in \Gamma$. Suppose that the assumptions in Theorem 4.12 and

$$0 < \Psi < \min \left\{ m_1 \theta_1^2 S^2, m_2 \theta_2^2 I^2, m_3 \theta_3^2 R^2, \frac{m_1 m_2 m_3 (\theta_1 S^* + \theta_2 I^* + \theta_3 R^* - \frac{b}{d})^2}{m_2 m_3 + m_1 m_3 + m_1 m_2} \right\}$$

holds, where

$$m_1 = \frac{1}{\gamma}(d(\gamma + 2d) - \gamma \sigma_1^2),$$

$$m_2 = \frac{1}{\gamma}(\gamma(d + \delta) + 2d(d + \mu + \delta) - \gamma \sigma_2^2),$$

$$m_3 = \frac{1}{\mu}(\mu d + \delta(d + \gamma) - \mu \sigma_3^2),$$

and $\theta_i (i = 1, 2, 3)$, $\Psi$ has the same definitions as it does in Theorem 4.12. Then there exists a unique stationary distribution $\pi(\cdot)$, and the solution $x(t) = (S(t), I(t), R(t))$ to model (9) is ergodic.
Proof. To verify (i) of Lemma (4.14), with reference to Zhu and Yin [89], it is sufficient to show that there exists some neighborhood \( U \) and a nonnegative \( C^2 \)-function \( V(x) \) such that, for some constants \( C > 0 \),

\[
LV(x) < -C \quad \text{for any} \quad x \in \Omega \setminus U.
\]

To this end, we use the nonnegative \( C^2 \)-function \( \tilde{V}(S, I, R) \) as Theorem (4.12). Hence, it follows from Theorem (4.12) that

\[
L\tilde{V} \leq -m_1 (S - \theta_1 S^*)^2 - m_2 (I - \theta_2 I^*)^2 - m_3 (R - \theta_3 R^*)^2 + \Psi.
\]

Now if \( \Psi \) satisfies the following conditions,

\[
0 < \Psi < \min \left\{ m_1 \theta_1^2 S^*^2, m_2 \theta_2^2 I^*^2, m_3 \theta_3^2 R^*^2 \right\}
\]

and

\[
\frac{1}{m_1} + \frac{1}{m_2} + \frac{1}{m_3} < \left( \frac{\theta_1 S^* + \theta_2 I^* + \theta_3 R^* - b_i}{\Psi} \right)^2
\]

then the ellipsoid

\[
m_1 (S - \theta_1 S^*)^2 + m_2 (I - \theta_2 I^*)^2 + m_3 (R - \theta_3 R^*)^2 = \Psi
\]

lies entirely in \( \Gamma \). One can then take \( U \) as any neighborhood of the ellipsoid such that \( \bar{U} \subset \Gamma \), where \( \bar{U} \) is the closure of \( U \). Thus, we have \( LV(S, I, R) < 0 \) for \( (S, I, R) \in \Gamma \setminus U \), which implies that condition (ii) in Lemma (4.14) is satisfied.

On the other hand, there is \( M = \min \{ \sigma_1^2 S^2, \sigma_2^2 I^2, \sigma_3^2 R^2 \} > 0 \) such that

\[
\text{diag} \left( \sigma_1^2 S^2, \sigma_2^2 I^2, \sigma_3^2 R^2 \right) \xi_i \xi_j = \sigma_1^2 S^2 \xi_1^2 + \sigma_2^2 I^2 \xi_2^2 + \sigma_3^2 R^2 \xi_3^2 \geq M |\xi|^2,
\]

for all \( (S, I, R) \in \bar{U}, \xi \in \mathbb{R}^3 \). Thus, by Rayleigh’s principle (see [90], p.342), condition (i) in Lemma (4.14) is verified for model (8). As a consequence, the stochastic model (8) has a stationary distribution \( \pi(\cdot) \) and it is ergodic. \( \square \)
Remark 4.17. Theorem 4.16 gives the possibility that an asymptotically stationary distribution exists for the solution of model (9) which in turn implies the stability in a stochastic sense. Furthermore, Theorem 4.16 suggests that if the condition of the theorem is satisfied, then the stochastic model (9) oscillates around the endemic equilibrium $E^*$ of the deterministic model (8), and model (9) has the ergodic property where the positive solution converges to the unique stationary distribution. This reveals the persistence of the disease a.s. under certain conditions. We provide numerical simulations to explore the endemic stationary distribution in Section 5 (see Figure 6 for details).

5. Numerical simulations and dynamics comparison

In this section, we give some numerical simulations to show the effect of noise on the dynamics of the SIRS models by using the Milstein method mentioned in Higham [91]. In this way, the SDE model (9) can be rewritten as the following discretization equations:

\[
\begin{align*}
S_{k+1} &= S_k + \left( b - dS - \frac{kS^h I}{S^h + \alpha I^h} + \gamma R \right) \Delta t + \sigma_1 S_k \sqrt{\Delta t} \xi_k + \frac{\sigma_1^2}{2} S_k (\xi_k^2 - 1) \Delta t, \\
I_{k+1} &= I_k + \left( \frac{kS^h I}{S^h + \alpha I^h} - (d + \mu + \delta)I \right) \Delta t + \sigma_2 I_k \sqrt{\Delta t} \eta_k + \frac{\sigma_2^2}{2} I_k (\eta_k^2 - 1) \Delta t, \\
R_{k+1} &= R_k + \left( \mu I - (d + \gamma)R \right) \Delta t + \sigma_3 R_k \sqrt{\Delta t} \zeta_k + \frac{\sigma_3^2}{2} R_k (\zeta_k^2 - 1) \Delta t,
\end{align*}
\]

where $\xi_k$, $\eta_k$ and $\zeta_k$, $k = 1, 2, \ldots, n$, are the Gaussian random variables $N(0, 1)$. The main goal of this section is to further investigate the answers to the following three questions as we proposed in the introduction:

1. Compare different dynamic outcomes of the deterministic model (8) v.s. its stochastic version (9).
2. Two different stationary distribution of the solutions to the stochastic model (9) under different conditions.
3. Different dynamical outcomes are produced by different types of environmental noises.
5.1. Stochasticity suppresses the disease outbreak

In this subsection, we give an example to show different dynamic outcomes of the deterministic model (8) v.s. its stochastic version (9) with the same set of parameter values.

Example 5.1. For the deterministic model (8) and its stochastic model (9), the parameters are taken as follows

\[ b = 1, \, d = 0.2, \, k = 1, \, \alpha = 0.5, \gamma = 0.25, \, \mu = 0.3, \, \delta = 0.1, \, h = 2. \]  \hspace{1cm} (36)

1. For the deterministic model (8), \( R_0 = \frac{k}{d + \mu + \delta} = 1.667 > 1 \), thus it admits a unique endemic equilibrium \( E^* = (1.428, 1.649, 1.099) \) which is globally stable for any initial values \( (S(0), I(0), R(0)) \in \Gamma \) according to Theorem 3.4 (see, Fig. 4(a)).

2. For the corresponding stochastic model (9), we choose \( \sigma_1 = 0.2, \sigma_2 = 0.9, \sigma_3 = 0.1 \), then we have

\[ \frac{b}{d + \mu + \delta} + \frac{\sigma_2^2}{2(d + \mu + \delta)} = \frac{1}{0.6} - \frac{0.81}{1.2} = 0.9917 < 1. \]

Thus according to Theorem 4.7, we can conclude that for any initial value \( (S(0), I(0), R(0)) \in \Gamma \), \( I(t) \) obeys

\[ \limsup_{t \to \infty} \frac{\ln I(t)}{t} \leq -0.005 \quad \text{a.s.} \]

That is, \( I(t) \) will tend to zero exponentially with probability one (see Fig. 4(b)).

To see the disease dynamics of (9) when \( R_0^s > 1 \), we decrease the noise intensity \( \sigma_2 \) of infective \( I \) to be 0.6, i.e., \( \sigma_2 = 0.6 \), and keep the other parameters unchanged. Then we have

\[ R_0^s = \frac{k}{d + \mu + \delta} + \frac{\sigma_2^2}{2(d + \mu + \delta)} = \frac{1}{0.6} - \frac{0.36}{1.2} = 1.3667 > 1. \]

Therefore, the condition of Theorem 4.7 is not satisfied. In this case, our simulations suggest that \( I(t) \) is stochastically persistent (see Fig. 4(c)).

Brief summary: Theorem 3.4 and 4.7 as well as numerical simulations (see examples illustrated in Fig. 4) indicate that environmental fluctuations can suppress the disease outbreak.
The deterministic model (8) admits a globally stable endemic equilibrium $E^*$ while introducing large noise intensity $\sigma_2$ in $I$ of the stochastic model (9) can eradicate the disease a.s. As we discuss in Remark 4.6, Theorem 4.4 indicates that the norm of $(S(t), I(t), R(t))$ of model (9) is bounded away from extinction almost surely whenever the condition $d + \delta + \frac{1}{2} \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\} < b$ holds. The parameters of the above two cases of Model (9) (i.e., Figures 4(b) and (c)) satisfy the conditions, thus they both are stochastically permanent. Even though Theorem 4.4 doesn’t give us any information whether $S(t)I(t)R(t) > 0$ or not, simulations (e.g., Figures 4) suggest that the persistence of infective $I$ can be governed by the value of $R_0^S$: if $R_0^S < 1$ (e.g., Fig.4(b)), while $I$ is stochastically persistent if $R_0^S > 1$ (e.g., Fig.4(c)). We have invested more on the case of $R_0^S > 1$ in the next subsection.

5.2. Stationary distribution

In the above subsection, we give an example to show $R_0^s$ may play a sharp role in the SDE model (9) as $R_0$ does for the deterministic model (8): If $R_0^s < 1$, the infective $I$ goes extinct a.s.; while $R_0^s > 1$ the infective $I$ is stochastically persistent. In this subsection, we further investigate two stationary distributions of the SDE model (9) governed by the value $R_0^S$: the SDE model (9) has disease-free stationary distribution when $R_0^S < 1$; while it has an endemic stationary distribution when $R_0^S > 1$.

1. The unique disease-free stationary distribution occurs when $R_0^S < 1$ according to Theorem 4.7 where both $I(t)$ and $R(t)$ goes extinct exponentially a.s. This leads to the situation that $S(t)$ has a unique stationary distribution according to Theorem 4.10, thus $\ln S(t)$ converges the measure with the density (30). To illustrate the disease-free stationary distribution, we provide numerical results in Fig.5 whose parameters are the same as those in Fig.4(b).

**Example 5.2.** We chose the parameters taken as (36) and choose $\sigma_1 = 0.2, \sigma_2 =$
0.9, \sigma_3 = 0.1. In this case,

\[ R_0^s = \frac{k}{d + \mu + \delta} - \frac{\sigma_2^2}{2(d + \mu + \delta)} = \frac{1}{0.6} - \frac{0.81}{1.2} = 0.9917 < 1. \]

And we present the frequency histograms based on 10000 stochastic simulations for \( S(t), I(t) \) and \( R(t) \) at time \( t = 100 \) by using the Statistical Software R in Fig.5. One can see that \( I(t) \) and \( R(t) \) go extinct exponentially a.s., and \( S(t) \) has a unique stationary distribution.

2. The endemic stationary distribution occurs when \( R_0^s > 1 \) suggested by Theorem 4.16.

**Example 5.3.** To investigate the endemic stationary distribution, we present the frequency histograms based on 10000 stochastic simulations for \( S(t), I(t) \) and \( R(t) \) at time \( t = 100 \) by using the Statistical Software R, where the parameters are taken as \((36)\) and \( \sigma_1 = 0.01, \sigma_2 = 0.02, \sigma_3 = 0.01 \) (see Fig.6). In this case, we have

\[
\Psi = \frac{\sigma_1^2(d(\gamma + 2d))}{d(\gamma + 2d) - \gamma \sigma_1^2 S^*} + \frac{\sigma_2^2[\gamma(d + \delta) + 2d(d + \mu + \delta)]}{\gamma(d + \delta) + 2d(d + \mu + \delta) - \gamma \sigma_2^2 I^*} + \frac{\sigma_3^2d[\mu d + \delta(d + \gamma)]}{\mu d + \delta(d + \gamma) - \mu \sigma_3^2 R^*}
\]

\[
+ \frac{\sigma_2^2d^{\alpha - 1}(S^* + \alpha I^*)}{2h^{\alpha - 1}} \left( 2d + \delta + \frac{2d}{\gamma}(2d + \mu + \delta) \right) I^* = 0.001199,
\]

\[
m_1\theta_1 S^* = \frac{1}{\gamma}(d(\gamma + 2d) - \gamma \sigma_1^2) \left( \frac{d(\gamma + 2d)}{d(\gamma + 2d) - \gamma \sigma_1^2 S^*} \right)^2 = 1.060307,
\]

\[
m_2\theta_2 I^* = \frac{1}{\gamma}(\gamma(d + \delta) + 2d(d + \mu + \delta) - \gamma \sigma_2^2) \left( \frac{\gamma(d + \delta) + 2d(d + \mu + \delta)}{\gamma(d + \delta) + 2d(d + \mu + \delta) - \gamma \sigma_2^2 I^*} \right)^2 = 2.569526,
\]

\[
m_3\theta_3 R^* = \frac{1}{\mu}(\mu d + \delta(d + \gamma) - \mu \sigma_3^2) \left( \frac{\mu d + \delta(d + \gamma)}{\mu d + \delta(d + \gamma) - \mu \sigma_3^2} R^* \right)^2 = 0.422954,
\]

\[
\frac{m_1m_2m_3(\theta_1 S^* + \theta_2 I^* + \theta_3 R^* - \frac{1}{2})^2}{m_2m_3 + m_1m_3 + m_1m_2} = 0.195565,
\]

and

\[ 0 < \Psi = 0.001199 < \min\{1.060307, 2.569526, 0.422954, 0.195565\}. \]

Thus according to Theorem 4.16, we can conclude that model \((9)\) has a unique endemic stationary distribution.
If $R_0^S > 1$, the SDE model (9) has a unique endemic stationary distribution as the solution to stochastic model (9) around endemic $E^*$ of model (8). Figure 6 and 7(a) as examples illustrate the existence and uniqueness of the stationary distribution of $S(t), I(t)$ and $R(t)$. To further explore the effect of the intensity of noises, we choose the main parameters the same as those in Fig. 6 but with different intensity of noise. In this case, we still have $R_0 = 1.667$, thus the deterministic model (8) admits a unique endemic equilibrium $E^* = (S^*, I^*, R^*) = (1.428, 1.649, 1.099)$ which is globally stable according to Theorem 3.4. Let $\sigma_1 = 0.01, \sigma_2 = 0.02, \sigma_3 = 0.01$, then we have

$$R_0^s = \frac{k}{d + \mu + \delta} - \frac{\sigma_2^2}{2(d + \mu + \delta)} = 1.666 > 1,$$

$$\sigma_1^2 = 0.01^2 < \frac{d(\gamma + 2 d)}{\gamma} = 0.52,$$

$$\sigma_2^2 = 0.02^2 < \frac{\gamma (d + \delta) + 2 d (d + \mu + \delta)}{\gamma} = 1.26,$$

$$\sigma_3^2 = 0.01^2 < \frac{d\mu + \delta (d + \gamma)}{\mu} = 0.35,$$

$$b + \delta + \frac{1}{2} \max\{\sigma_1^2, \sigma_2^2, \sigma_3^2\} = 0.3002 < b = 1.$$ 

If we keep all the parameters in (36) unchanged but increase $(\sigma_1, \sigma_2, \sigma_3)$ to $(0.1, 0.2, 0.1)$, then we have $R_0^s = 1.633 > 1$, $\sigma_1^2 = 0.01 < 0.52, \sigma_2^2 = 0.04 < 1.26, \sigma_3^2 = 0.01 < 0.35$. According to Theorem 4.12, we can conclude that for any initial value $(S(0), I(0), R(0)) \in \Gamma$, the solutions of the stochastic model (9) are oscillating around the endemic equilibrium $E^* = (1.428, 1.649, 1.099)$ of model (8) for both cases $(\sigma_1, \sigma_2, \sigma_3) = (0.01, 0.02, 0.01)$ (see Figure 6) and $(\sigma_1, \sigma_2, \sigma_3) = (0.1, 0.2, 0.1)$ (see Figure 7(a)). Our Theorem 4.12 indicates that amplitude of oscillations increases with the intensity of the noise level. This is supported by our computer simulations shown in Figure 6 and 7(a).

5.3. The effects of different environmental noises

There can be many different approaches on deriving SDE model from the deterministic model (8), which may lead to different dynamical outcomes. In this subsection, we compare
the dynamics of our SDE model (9) to the SDE model (37) by adopting the approach of Schreiber et al. [79]. The SDE model (37) is represented as follows:

\[
\begin{align*}
    dS &= b - dS - \frac{k S^h I}{S^h + \alpha I^h} + \gamma R + \sigma_{11} SdB_{11}(t) + \sigma_{12} IdB_{12}(t) + \sigma_{13} RdB_{13}(t), \\
    dI &= \frac{k S^h I}{S^h + \alpha I^h} - (d + \mu + \delta)I + \sigma_{21} SdB_{21}(t) + \sigma_{22} IdB_{22}(t) + \sigma_{23} RdB_{23}(t), \\
    dR &= \mu I - (d + \gamma)R + \sigma_{31} SdB_{31}(t) + \sigma_{32} IdB_{32}(t) + \sigma_{33} RdB_{33}(t),
\end{align*}
\]  

(37)

where \(\sigma_{ij}, i, j = 1, 2, 3\) are real constants and known as the intensity of environmental fluctuations, \(B_{ij}(t), i, j = 1, 2, 3\) independent standard Brownian motions. Notice that the stochastic model (9) is a special case of (37). However, we may expect different dynamic outcomes for (37) due to the extra terms. For simplicity, we can take the values of \(\sigma_{ij}, i = 1, 2, 3\) of (37) to be the same as \(\sigma_j\) of (9), i.e., \(\sigma_{ij} = \sigma_j, i = 1, 2, 3\). Thus, we have the following model:

\[
\begin{align*}
    dS &= b - dS - \frac{k S^h I}{S^h + \alpha I^h} + \gamma R + \sigma_{11} SdB_{11}(t) + \sigma_{12} IdB_{12}(t) + \sigma_{13} RdB_{13}(t), \\
    dI &= \frac{k S^h I}{S^h + \alpha I^h} - (d + \mu + \delta)I + \sigma_{11} SdB_{21}(t) + \sigma_{12} IdB_{22}(t) + \sigma_{13} RdB_{23}(t), \\
    dR &= \mu I - (d + \gamma)R + \sigma_{11} SdB_{31}(t) + \sigma_{12} IdB_{32}(t) + \sigma_{13} RdB_{33}(t),
\end{align*}
\]  

(38)

We illustrate the different dynamical outcomes of two SDE models based on simulation observations and the theorems obtained from previous sections. See examples as follows:

**Example 5.4.** In Model (38), we take the parameters as \(b = 1, d = 0.2, k = 1, \alpha = 0.5, \gamma = 0.25, \mu = 0.3, \delta = 0.1, h = 2\) and \(\sigma_1 = 0.01, \sigma_2 = 0.02, \sigma_3 = 0.01\), the same as those in Fig. 6 and Fig. 7(a). Figure 8 illustrates the dynamical properties for these given parameters’ values of Model (38), i.e., Figure 8 provides the frequency histograms based on 10000 stochastic simulations for \(S(t), I(t)\) and \(R(t)\) at time \(t = 100\) by using the Statistical Software R. We observe that the stochastic dynamics of model (38) are dramatically different from Model (9) in the following sense: For the given values of parameters, our SDE model (9) has an endemic stationary distribution (see Fig. 6) while the new model (38) is disease-free, i.e., \(I(t)\) dies out
with probability one. We have performed a large number of numerical simulations for the new model (38): We observe that the new model (38) has only one stationary distribution which is disease-free, i.e., \( I(t) \) dies out with probability one. This is different from our SDE model (9) since model (9) may have a disease-free distribution (see Fig. 5) or an endemic stationary distribution (see Fig. 6) depending on the values of \( R^S_0 \). In addition, the new stochastic model (38) can have negative values in \( I \) and \( R \) due to the fact that they are not positively invariant: for example, if \( I(t) = 0 \), the extra term \( \sigma_{21} S dB_{21}(t) \) can lead the dynamics of \( I \) to be negative; similar to the case of \( R(t) \). This indicates that different stochastic models derived from the same deterministic model can have dramatic different dynamical outcomes, thus we should be very careful in deriving the proper SDE models for a given deterministic model.

6. Discussions

Fluctuations in the natural environment introduce variability into the biological systems that exist within them [15]. In many instances, environmental variations have a critical influence on the development of an epidemic [9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30]. For example, many species of fungi have threshold temperatures and humidity levels for germination to occur [15, 20]. Variability of temperature and rainfall may induce pronounced fluctuations in the dynamics of pathogenic fungi [20]. For human disease, the nature of epidemic growth and spread is inherently random due to the unpredictability of person-to-person contacts [6] and population is subject to a continuous spectrum of disturbances [7, 8]. Hence the variability and randomness of the environment is fed through to the state of the epidemic [15]. In this article, we focus on a stochastic differential equation SIRS model with ratio-dependent incidence rate.

Among the various ways to construct a stochastic model systems for a given deterministic system, in the present paper, we propose a stochastic version of the SIRS epidemic model with nonlinear incident rate. In our model, we suppose that stochastic environmental factor
acts simultaneously on each individual in the population, and assume that the stochastic perturbation is a white noise type that is directly proportional to $S(t)$, $I(t)$, and $R(t)$ and is influenced on the $\frac{dS(t)}{dt}$, $\frac{dI(t)}{dt}$, and $\frac{dR(t)}{dt}$, respectively. This is a well-established way of introducing stochastic environmental noise into biologically realistic population dynamic models that have been used in [7, 37, 45, 57, 58, 52, 49, 60, 48, 62, 63].

The value of our study lies in two aspects: Mathematically, we show that the global dynamics of the deterministic model (8) can be governed by its reproduction number $R_0$ while the dynamics of its stochastic version (9) seem to be governed by $R_0^S = R_0 \left(1 - \frac{\sigma^2}{2\pi}\right)$. In addition, we have provided the analytic results on the existence of global positive solution, stochastic boundedness, permanence/extinction, asymptotic stability and ergodic property of the solution to the SDE model (9). Epidemiologically, we partially provide answers to the three questions proposed in the introduction: What is the disease dynamics of stochastic SIRS model with ratio-dependent incidence rate? How may different types of environmental fluctuations produce different dynamical outcomes? How may the dynamics of SIRS models with environmental fluctuations differ from the deterministic version? We summarize our main findings as follows:

1. **Noise can suppress the disease outbreak**: Theorem 4.7 indicates that the extinction of disease in the stochastic model (9) occurs if the basic reproduction number $R_0^S = R_0 - \frac{\sigma^2}{2(d+\mu+\delta)} < 1$. Theorem 3.4 shows that the deterministic model (8) admits a unique endemic equilibrium $E^*$ which is globally asymptotically stable if its basic reproduction number $R_0 > 1$. Notice that $R_0^S < R_0$, thus it is possible that $R_0^S < 1 < R_0$. This is the case when the deterministic model (8) has endemic (see figure 4(a)) while the stochastic model (9) has disease extinction with probability one (see figure 4(b)).

2. **Two stationary distribution governed by $R_0^S$**: As suggested by Theorems 4.10, 4.12 and simulations (figures 5 and 6), model (9) has two kinds of stationary distributions depending on the value of $R_0^S$: If $R_0^S < 1$, it has disease–free stationary distribution
(see Fig.5) which means that the disease will die out with probability one; while it has endemic stationary distribution (c.f., Fig.6) if $R_0^S > 1$. The latter leads to the stochastically persistence of the disease. The numerical results in Fig.6 suggest the existence and uniqueness of the endemic stationary distribution of $S(t), I(t)$ and $R(t)$.

3. **The effects of the intensity of noise levels**: From Theorems 4.10, 4.12 and numerical results (e.g., Figs.4(a) and 7(a)), we can conclude that, when the intensity of noise is small, the stochastic model preserves the property of the global stability. In this case, we can ignore noise and use the deterministic model to approximate the population dynamics. However, the large intensity of noise can force the solutions of model (9) to oscillate strongly around the disease-free (c.f., Theorem 4.10) or endemic points (c.f., Theorem 4.12 and Fig.7(b)), or to extinct (c.f., Theorem 4.7 and Fig.4(c)). In these cases, we cannot ignore the effect of noise, therefore, we cannot use deterministic model but stochastic model to describe the population dynamics.

4. **The effects of different types of noise**: Our numerical study on a new SDE model (38) indicates that different stochastic modeling approaches can have different dynamical outcomes, e.g., by introducing additional terms in the noises, we find that the new SDE model (38) has only one stationary distribution which is disease-free (figure 8). However, our SDE model can have disease-free stationary distribution and endemic stationary distribution based on the values of $R_0^S$. In addition, we notice that $h$ is independent on the existence of the endemic point $E^*$ of model (8) but dependent on the value of $E^*$ (see the equilibrium expression (15)). We would like to point out that all theorems related to the deterministic model (8) and the stochastic model (9) (except Theorem 4.12 and Theorem 4.16), are not depend on the parameter $h$. The reason is that Theorem 4.12 and Theorem 4.16 are related to $E^* = (S^*, I^*, R^*)$. While others are independent on $h$ during the proving process since we amplify the values too large to contain $h$ by using the method of amplification.
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Figure 2: The graphic representation of $g(S, I)$ for varied values of $h$ and $l$ when $k = 0.5, \alpha = 0.5$. 

(a) $g(S, I) = \frac{kIS}{1 + \alpha I}$ 

(b) $g(S, I) = \frac{kIS}{1 + \alpha I^2}$ 

(c) $g(S, I) = \frac{kSI}{S + \alpha I}$ 

(d) $g(S, I) = \frac{kS^2I}{S^2 + \alpha I^2}$
Figure 3: Section drawing of $g(S,I)$ with respect to $S$ (the first row) and $I$ (the second row) for varied values of $h$ and $l$ when $k = 0.5, \alpha = 0.5$. 
Figure 4: The path $S(t)$, $I(t)$ and $R(t)$ for the stochastic model (9) with initial values $(S(0), I(0), R(0)) = (0.9, 0.1, 0)$. The parameters are taken as $b = 1$, $d = 0.2$, $k = 1$, $\alpha = 0.5$, $\gamma = 0.25$, $\mu = 0.3$, $\delta = 0.1$. 

(a) $\sigma_1 = 0, \sigma_2 = 0, \sigma_3 = 0$

(b) $\sigma_1 = 0.2, \sigma_2 = 0.9, \sigma_3 = 0.1$

(c) $\sigma_1 = 0.2, \sigma_2 = 0.6, \sigma_3 = 0.1$
Figure 5: Frequency histograms based on 10000 stochastic simulations for each population at time $t = 100$. The parameter values are the same as them in Fig. 4(b).
Figure 6: Frequency histograms based on 10000 stochastic simulations for each population at time $t = 100$.

The parameter values in (a), (b) and (c) are the same as them in Fig. 7(a).
Figure 7: The asymptotic behavior of the solutions to the stochastic model (9) with parameters (36) around the endemic equilibrium $E^* = (1.428, 1.649, 1.099)$ of model (8) with initial values $S(0) = 0.9, I(0) = 0.1, R(0) = 0$. 

(a) $\sigma_1 = 0.01, \sigma_2 = 0.02, \sigma_3 = 0.01$

(b) $\sigma_1 = 0.1, \sigma_2 = 0.2, \sigma_3 = 0.1$
Figure 8: Frequency histograms based on 10000 stochastic simulations for each population at time $t = 100$.

The parameter values are the same as them in Fig. 6.