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*Research article*

# Deterministic and stochastic model for the hepatitis C with different types of virus genome

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**Abstract:** In this paper, a deterministic and stochastic model for hepatitis C with different types of virus genomes is proposed and analyzed. Some sufficient conditions are obtained to ensure the stability of the deterministic equilibrium points. We perform a stochastic extension of the deterministic model to study the fluctuation between environmental factors. Firstly, the existence of a unique global positive solution for the stochastic model is investigated. Secondly, sufficient conditions for the extinction of the hepatitis C virus from the stochastic system are obtained. Theoretical and numerical results show that the smaller white noise can ensure the persistence of susceptible and infected populations while the larger white noise can lead to the extinction of disease. By introducing the basic reproduction number $R_0$ and the stochastic basic reproduction number $R_0^s$, the conditions that cause the disease to die out are indicated. The importance of environmental noise in the propagation of hepatitis C viruses is highlighted by these findings.

**Keywords:** hepatitis C; extinction; stochastic; stability; stationary distribution; Lyapunov function
**Mathematics Subject Classification:** 92D25, 92D30, 37C75, 37H30, 37L45

## 1. Introduction

Hepatitis C is a disease caused by a virus that infects the liver. The virus, called hepatitis C virus or HCV, is just one of the hepatitis viruses. Global statistics estimate that nearly 350 million people worldwide are infected with HCV, which significantly affects human health, especially the liver [16]. Mathematical models are currently a useful way to assess the transmissibility of many infectious diseases, predict future morbidity, and assess the efficacy of prevention and therapy [1, 11, 14, 17, 21, 34–36, 42, 46]. Many successful mathematical models investigating fundamental hepatitis C viral

dynamics have been produced in recent decades, including Feng et al. [12], Lestari et al. [23] and Li et al. [24] to cite only a few. Moneim and Mosa [31, 32], constructed a mathematical model to study the spread of HCV-subtype 4a amongst the Egyptian population. They divided the population into three groups, susceptible class, $S(t)$, the hepatitis C subtype 4a infective class, $I_1$ and the HCV from all other subtype classes, $I_2$. They assumed that all types of the class $I_2$ could mutate to $I_1$ at a constant rate $\mu > 0$. A few years after the appearance of [31, 32], effective treatment for the hepatitis C virus appeared. For this, we will add to the mathematical model another class ($R(t)$), which represents those who have been cured of this virus. Following [8,18,19,39,47], the saturated incidence rate will replace the bilinear incidence rate. The hepatitis C virus becomes as follows.

$$\begin{aligned}
\frac{dS}{dt} &= A - \frac{k_1 S I_1}{a+S} - \frac{k_2 S I_2}{a+S} - bS,\\
\frac{dI_1}{dt} &= \frac{k_1 S I_1}{a+S} + \mu I_1 I_2 - bI_1 - \gamma I_1,\\
\frac{dI_2}{dt} &= \frac{k_2 S I_2}{a+S} - \mu I_1 I_2 - bI_2 - \delta I_2,\\
\frac{dR}{dt} &= \gamma I_1 + \delta I_2 - bR,
\end{aligned} \tag{1.1}$$

where $A$ denotes the recruitment rate of susceptible individuals, $a$ expresses the half-saturation constant for susceptible individuals with HCV. $\gamma$ and $\delta$ are the recovery rates, $k_1$ is the transmission rate of virus C when susceptible individuals $S(t)$ contact with corresponding infected $I_1(t)$ individuals, and $k_2$ is the transmission rate when $S(t)$ contact with corresponding $I_2(t)$ individuals. Following [31, 32], we assume that the population is mixing in a homogenous manner, i.e., every person has the same chance of coming in contact with an infected person also we assume that the birth and death rates are equal and positive constant rate $b$ .

The deterministic HCV system (1.1) ignores the possible importance of environmental noise. In reality, stochastic effects can be important during the transmission of hepatitis C viruses, because various cells and infective virus particles react differently in the same environment. Consequently, the deterministic models do not provide an adequate understanding of viral dynamics due to the stochastic behavior of viruses and the complexity of the immune system. The primary purpose of this paper is to propose and analyze a deterministic and stochastic model of HCV. The paper is arranged as follows: In Section 2, the dynamics of the deterministic system are verified. The stochastic extension of the deterministic model is performed in Section 3, and the existence of a unique global positive solution for the stochastic model is investigated, and the sufficient conditions for the extinction of the hepatitis C virus from the stochastic system are obtained. In Section 4, some numerical simulations are presented to verify the obtained theoretical results. Finally, Section 5 contains the conclusion.

## 2. Dynamics of deterministic system

In the following, two critical parameters $R_{01}$, and $R_{02}$, can be used to classify the dynamics of the hepatitis C virus model (1.1). The threshold parameter $R_{01}$ defined by $R_{01} = \frac{Ak_1}{\rho(A+ab)}$, $\rho = b + \gamma$. While the threshold parameter $R_{02}$ defined by $R_{02} = \frac{Ak_2}{\theta(A+ab)}$, $\theta = b + \delta$. Using the next generation method, one can obtain the basic reproduction number $R_0$ defined by $R_0 = \max\{R_{01}, R_{02}\}$.

The hepatitis C virus model (1.1) has the following four equilibrium points:

(1) The disease-free equilibrium point $E_0 = \left(\frac{A}{b}, 0, 0, 0\right)$, which always exists. At this point, all individuals are susceptible, and there is no infection present in the population.

(2) The equilibrium point $E_1 = (S_1, I_{11}, 0, R_1)$, where

$$S_1 = \frac{a\rho}{k_1 - \rho},\ I_{11} = \frac{(a + S_1)(A + ab)}{ak_1}(R_{01} - 1),\ R_1 = \frac{\gamma(a + S_1)(A + ab)}{abk_1}(R_{01} - 1).$$

$E_1$ exists if $k_1 > \rho$ and $R_{01} > 1$.

(3) The equilibrium point $E_2 = (S_2, 0, I_{22}, R_2)$, where

$$S_2 = \frac{a\theta}{k_2 - \theta},\ I_{22} = \frac{(a + S_2)(A + ab)}{ak_2}(R_{02} - 1),\ R_2 = \frac{\delta(a + S_2)(A + ab)}{abk_2}(R_{02} - 1).$$

$E_2$ exists if $k_2 > \theta$ and $R_{02} > 1$.

(4) The coexistence equilibrium point $E_3 = (S_3, I_{13}, I_{23}, R_3)$, where

$$S_3 = \frac{-\beta + \sqrt{\beta^2 + 4abA}}{2b},\ I_{13} = \frac{1}{\mu}\left(\frac{k_2 S_3}{a + S_3} - \theta\right),\ I_{23} = \frac{1}{\mu}\left(\rho - \frac{k_1 S_3}{a + S_3}\right),\ R_3 = \frac{1}{\mu}(\gamma I_{13} + \delta I_{23}),$$

$\beta = ab - A + \frac{\rho k_2}{\mu} - \frac{\theta k_1}{\mu}$. The coexistence equilibrium point $E_3$ exists if $\frac{k_2 S_3}{\theta(a+S_3)} > 1$ and $\frac{k_1 S_3}{\rho(a+S_3)} < 1$.

The boundedness of the solutions of model (1.1) is given as follows. Let $(S(t), I_1(t), I_2(t), R(t))$ be any solution of system (1.1) with non-negative initial conditions and assume that the total population size $N(t) = S(t) + I_1(t) + I_2(t) + R(t)$, then $\frac{dN}{dt} + bN = A$, consequentially $N(t) = \frac{A}{b} + N(0)e^{-bt}$, thus it follows that $0 \le N(t) \le \frac{A}{b}$, as $t \to \infty$.

The locally asymptotically stable equilibrium points of system (1.1) are now investigated. The Jacobian matrix is given as follows:

$$J = \begin{pmatrix} -\frac{k_1 aI_1}{(a+S)^2} - \frac{k_2 aI_2}{(a+S)^2} - b & -\frac{k_1 S}{a+S} & -\frac{k_2 S}{a+S} & 0 \\ \frac{k_1 aI_1}{(a+S)^2} & \frac{k_1 S}{a+S} + \mu I_2 - \rho & \mu I_1 & 0 \\ \frac{k_2 aI_2}{(a+S)^2} & -\mu I_2 & \frac{k_2 S}{a+S} + \mu I_1 - \theta & 0 \\ 0 & \gamma & \delta & -b \end{pmatrix}.$$

The eigenvalues of $J(E_0)$ are $-b$, $-b$, $\rho(R_{01} - 1)$, and $\theta(R_{02} - 1)$. Thus, $E_0$ is locally asymptotically stable if $R_0 < 1$. The eigenvalues of $J(E_1)$ are $\lambda_1 = -b$ and $\lambda_2 = \frac{k_2 S_1}{a+S_1} - \mu I_{11} - \theta$. The other roots are determined by

$$\lambda^2 + (\frac{k_1 aI_{11}}{(a + S_1)^2} + b)\lambda + \frac{k_1^2 aS_1 I_{11}}{(a + S_1)^3} = 0.$$

The eigenvalues $\lambda_3$ and $\lambda_4$ have negative real parts. Thus, if $\frac{k_2 S_1}{(\mu I_{11} + \theta)(a+S_1)} < 1$ then the equilibrium point $E_1$ is locally asymptotically stable. The eigenvalues of $J(E_2)$ are $\lambda_1 = -b$ and $\lambda_2 = \frac{k_1 S_2}{(a+S_2)} + \mu I_{22} - \rho$. The other roots are determined by

$$\lambda^2 + (\frac{k_2 aI_{22}}{(a + S_2)^2} + b)\lambda + \frac{k_2^2 aS_2 I_{22}}{(a + S_2)^3} = 0.$$

The eigenvalues $\lambda_3$ and $\lambda_4$ have negative real parts. Thus, if $\frac{k_1 S_2}{(a+S_2)} + \mu I_{22} < \rho$ then the equilibrium point $E_2$ is locally asymptotically stable. The stability of the coexistence equilibrium point $E_3 = (S_3, I_{13}, I_{23}, R_3)$ is investigated as follows

The first eigenvalues of $J(E_3)$ is $\lambda_1 = -b$. The other roots are determined by

$$\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0, \tag{2.1}$$

where

$$\begin{aligned}
c_1 &= \frac{a\,(ab + k_1 I_{13} + k_2 I_{23}) + 2abS_3 + bS_3^2}{(a+S_3)^2},\\
c_2 &= \frac{I_{23}\left(ak_2^2 S_3 + \mu^2 I_{13}\,(a+S_3)^3\right) + ak_1^2 S_3 I_{13}}{(a+S_3)^3},\\
c_3 &= \frac{\mu^2 I_{13} I_{23}\left(a\,(ab + k_1 I_{13} + k_2 I_{23}) + 2abS_3 + bS_3^2\right)}{(a+S_3)^2}.
\end{aligned}$$

$$c_1 c_2 - c_3 = \frac{aS_3\left(k_1^2 I_{13} + k_2^2 I_{23}\right)\left(a\,(ab + k_1 I_{13} + k_2 I_{23}) + 2abS_3 + bS_3^2\right)}{(a+S_3)^5}.$$

It is clear that $c_1, c_2, c_3 > 0$ and $c_1 c_2 - c_3 > 0$. It follows from the Routh-Hurwitz criterion that all roots of (2.1) have negative real parts. Thus, the equilibrium point $E_3$ is locally asymptotically stable.

## 3. Dynamics of stochastic model

In this section, we will perform a stochastic extension of model (1.1). The hepatitis virus C deterministic model (1.1) will be extended to include the environmental noise as follows.

$$\begin{aligned}
dS &= \left[A - \frac{k_1 S I_1}{a+S} - \frac{k_2 S I_2}{a+S} - bS\right]dt + \sigma_1 S\, dW_1,\\
dI_1 &= \left[\frac{k_1 S I_1}{a+S} + \mu I_1 I_2 - bI_1 - \gamma I_1\right]dt + \sigma_2 I_1\, dW_2,\\
dI_2 &= \left[\frac{k_2 S I_2}{a+S} - \mu I_1 I_2 - bI_2 - \delta I_2\right]dt + \sigma_3 I_2\, dW_3,\\
dR &= [\gamma I_1 + \delta I_2 - bR]\, dt + \sigma_4 R\, dW_4,
\end{aligned} \tag{3.1}$$

where $W = \{W_1, W_2, W_3, W_4, t \geq 0\}$ represents the four-dimensional standard Brownian motions with $W_i(0) = 0$ and $\sigma_i^2 (i = 1, 2, 3, 4)$ denote the intensities of the white noise. The white noise is defined in a complete probability space $(\Omega, \mathcal{F}_{t\geq 0}, \mathbb{P})$ with a filtration $\mathcal{F}_{t\geq 0}$ satisfying the usual conditions. In many applications, the solution of the Itô stochastic differential equation must preserve the positivity of the solutions [9, 10, 37]. According to Theorem 2.2 and Corollary 1 in [9], the solutions of (3.1) emanating from nonnegative initial data (almost surely) remain nonnegative as long as they exist. The next theorem gives another approach according to [30] to prove the existence and uniqueness of a positive global solution of the system (3.1). This approach has recently been used in many papers, and one can highlight [4–7, 13, 17, 20, 22, 27, 28, 33, 38, 40–45, 48].

**Theorem 1.** *For any given initial value* $(S(0), I_1(0), I_2(0), R(0)) \in \mathbb{R}^4_+$*, there exists a unique solution* $(S(t), I_1(t), I_2(t), R(t))$ *of system* (3.1) *for* $t \geq 0$ *and the global positive solution remains in* $\mathbb{R}^4_+$ *with probability one.*

*Proof.* Firstly, one can consider the local solution $(S(t), I_1(t), I_2(t), R(t))$ of system (3.1) for $t \in [0, \tau_e)$, where $\tau_e$ is the explosion time [30]. By making the transformation of variables

$$x(t) = \ln S(t),\ \ y(t) = \ln I_1(t),\ \ z(t) = \ln I_2(t),\ \ w = \ln R(t).$$

Using Itô formula, one can change system (3.1) as follows

$$\begin{aligned} d\,x(t) &= \left[\frac{A}{e^x} - \frac{k_1 e^y}{a+e^x} - \frac{k_2 e^z}{a+e^x} - b - \frac{\sigma_1^2}{2}\right]dt + \sigma_1\, dW_1, \\ d\,y(t) &= \left[\frac{k_1 e^x}{a+e^x} + \mu e^z - \rho - \frac{\sigma_2^2}{2}\right]dt + \sigma_2\, dW_2, \\ d\,z(t) &= \left[\frac{k_2 e^x}{a+e^x} - \mu e^y - \theta - \frac{\sigma_3^2}{2}\right]dt + \sigma_3\, dW_3, \\ d\,w(t) &= \left[\gamma e^{y-w} - \delta e^{z-w} - b - \frac{\sigma_4^2}{2}\right]dt + \sigma_4\, dW_4. \end{aligned} \tag{3.2}$$

For $X_0 = (x_0, y_0, z_0, w_0) \in \mathbb{R}^4_+$, the coefficients of system (3.2) satisfy the local Lipschitz conditions, consequently, there exists a unique local solution $(S(t), I_1(t), I_2(t), R(t)) = (e^{x(t)}, e^{y(t)}, e^{z(t)}, e^{w(t)})$ on $[0, \tau_e)$. To ensure that this solution is global, one needs to prove that $\tau_e = \infty$ a.s. Let $s_0 > 0$ be sufficiently large for every coordinate $(S(0), I_1(0), I_2(0), R(0))$ in the interval $[\frac{1}{s_0}, s_0]$. For each integer $s > s_0$, we define the stopping time

$$\tau_s = \inf\left\{t \in [0, \tau_e) : \min\{S(t), I_1(t), I_2(t), R(t)\} \notin (\frac{1}{s}, s) \text{ or } \max\{S(t), I_1(t), I_2(t), R(t)\} \notin (\frac{1}{s}, s)\right\}. \tag{3.3}$$

From (3.3), one can note that $\tau_s$ is increasing as $s \to \infty$. Assume $\tau_\infty = \lim_{s\to\infty} \tau_s$, then $\tau_\infty \leq \tau_e$ almost sure. In the next, one needs to verify that $\tau_\infty = \infty$. If this is not true, then there exists a constant $T > 0$ and $\epsilon \in (0, 1)$ such that $\mathbb{P}(\tau_\infty \leq T) \geq \epsilon$. As a result, there exists an integer $s_1 \geq s_0$ such that $\mathbb{P}(\tau_s \leq T) \geq \epsilon,\ \ s \geq s_1$. Define the following $C^2$ positive definite function $V_1(S, I_1, I_2, R)$ as

$$V_1(S, I_1, I_2, R) = (S + 1 - \ln S) + (I_1 + 1 - \ln I_1) + (I_2 + 1 - \ln I_2) + (R + 1 - \ln R).$$

Using Itô's formula, one obtains

$$\begin{aligned} dV_1 =& \left[(S-1)\left(\frac{A}{S} - \frac{k_1 I_1}{a+S} - \frac{k_2 I_2}{a+S} - b\right) + (I_1 - 1)\left(\frac{k_1 S}{a+S} + \mu I_2 - \rho\right)\right. \\ &\left. + (I_2 - 1)\left(\frac{k_2 S}{a+S} - \mu I_1 - \theta\right) + (1 - \frac{1}{R})(\gamma I_1 + \delta I_2 - bR) + \frac{1}{2}\sum_{i=1}^{4}\sigma_i^2\right]dt \\ &+ \sigma_1(S-1)dW_1 + \sigma_2(I_1 - 1)dW_2 + \sigma_3(I_2 - 1)dW_3 + \sigma_4(R-1)dW_4 \\ \leq& \left[A + 2b + \rho + \theta + \frac{1}{2}\sum_{i=1}^{4}\sigma_i^2 + \left(\frac{k_1}{a} + \mu + \gamma\right)I_1 + (\frac{k_2}{a} + \delta)I_2\right]dt \\ &+ \sigma_1(S-1)dW_1 + \sigma_2(I_1 - 1)dW_2 + \sigma_3(I_2 - 1)dW_3 + \sigma_4(R-1)dW_4. \end{aligned}$$

Using the inequality $x \leq 2(x + 1 - \ln x)$, for any $x > 0$, one obtains

$$\begin{aligned} dV_1 \leq & \Big[A + 2b + \rho + \theta + \frac{1}{2}\sum_{i=1}^{4}\sigma_i^2 + 2\,(S\, + 1 - \ln S\,) + 2\left(\frac{k_1}{a} + \mu + \gamma\right)(I_1 + 1 - \ln I_1) \\ & + 2(\frac{k_2}{a} + \delta)\,(I_2 + 1 - \ln I_2) + 2\,(R + 1 - \ln R)\Big]dt + \sigma_1(S\, - 1)dW_1 + \sigma_2(I_1 - 1)dW_2 \\ & + \sigma_3(I_2 - 1)dW_3 + \sigma_4(R - 1)dW_4, \end{aligned}$$

which means that

$$dV_1 \leq K(1 + V_1)dt + \sigma_1(S\, - 1)dW_1 + \sigma_2(I_1 - 1)dW_2 + \sigma_3(I_2 - 1)dW_3 + \sigma_4(R - 1)dW_4, \tag{3.4}$$

where $K = \max\{K_1, K_2\}$, $K_1 = A + 2b + \rho + \theta + \frac{1}{2}\sum_{i=1}^{4}\sigma_i^2$ and $K_2 = \max\left\{2, 2(\frac{k_1}{a} + \mu + \gamma), 2(\frac{k_2}{a} + \delta)\right\}$. For $t_1 \leq T$, integrating both sides of (3.4) from 0 to $t_1 \wedge \tau_s$ and then taking the expectation leads to

$$\begin{aligned} & EV_1\,(S\,(t_1 \wedge \tau_s), I_1(t_1 \wedge \tau_s), I_2(t_1 \wedge \tau_s), R(t_1 \wedge \tau_s)) \\ & \leq V_1\,(S\,(0), I_1(0), I_2(0), R(0)) + KE\int_0^{t_1\wedge\tau_s}(1 + V_1)\;dt \\ & \leq V_1\,(S\,(0), I_1(0), I_2(0), R(0)) + KT + K\int_0^{t_1\wedge\tau_s} EV_1\,(S\,(t_1 \wedge \tau_s), I_1(t_1 \wedge \tau_s), I_2(t_1 \wedge \tau_s), R(t_1 \wedge \tau_s))\;\;dt. \end{aligned}$$

Following [25, 26, 29, 44], applying Grownwall's inequality, one gets

$$EV_1\,(S\,(t_1 \wedge \tau_s), I_1(t_1 \wedge \tau_s), I_2(t_1 \wedge \tau_s), R(t_1 \wedge \tau_s)) \leq [V_1\,(S\,(0), I_1(0), I_2(0), R(0)) + K\,T]\,e^{KT} = K_3.$$

The rest of the proof is similar to [25, 26, 29] and hence is omitted here. This complete the proof. □

The above theorem shows that the stochastic HCV system (3.1) have positive global solution remain in $\mathbb{R}_+^4$ with probability one. The non-explosion characteristic in an epidemic model is essential but often insufficient. Hence, one needs to indicate that the solution will be ultimately bounded with a large probability. In the following, we will establish the stochastically ultimate boundedness property of the HCV system (3.1).

**Lemma 2.** *Let $N(t) = S\,(t)+I_1(t)+I_2(t)+R(t)$, then for any given initial value $(S\,(0), I_1(0), I_2(0), R(0)) \in \mathbb{R}_+^4$, the following inequality holds:*

$$\lim_{t\to\infty} N(t) < \infty \;\; a.s.$$

*Proof.* It follows from HCV model (3.1) that

$$dN(t) = (A - bN(t))dt + \sigma_1 S\,(t)dW_1(t) + \sigma_2 I_1(t)dW_2(t) + \sigma_3 I_2(t)dW_3(t) + \sigma_4 R(t)dW_4(t) \tag{3.5}$$

Then, the solution of Eq (3.5) has the following form

$$N(t) = \frac{A}{b} + \left(N(0) - \frac{A}{b}\right)e^{-bt} + M(t),$$

where

$$M(t) = \int_0^t e^{-b(t-s)}\sigma_1 S(s)dW_1(s) + \int_0^t e^{-b(t-s)}\sigma_2 I_1(s)dW_2(s) + \int_0^t e^{-b(t-s)}\sigma_3 I_2(s)dW_3(s) + \int_0^t e^{-b(t-s)}\sigma_4 R(s)dW_4(s),$$

which is a contiuous local martingal with $M(0) = 0$, almost surely. Define $\Lambda(t) = \frac{A}{b}[1 - e^{-bt}]$ and $U(t) = N(0)[1 - e^{-bt}]$, then we have $N(t) = N(0) + \Lambda(t) - U(t) + M(t)$. Clearly, $\Lambda(t)$ and $U(t)$ are continuous adapted increasing process on $t \geq 0$ with $\Lambda(0) = U(0) = 0$. Then by Theorem 3.9 in [30], we have $\lim_{t\to\infty} N(t) < \infty$ a.s. □

**Remark 3.** *It follows from the first equation of HCV system* (3.1) *that*

$$S(t) - S(0) = At - b\int_0^t S(s)ds - \int_0^t \frac{k_1 S(s)I_1(s)}{a + S(s)} - \int_0^t \frac{k_2 S(s)I_2(s)}{a + S(s)} + \sigma_1 \int_0^t S(s)dW_1(s),$$

*and it follows that*

$$\langle S(t)\rangle \leq \lim_{t\to\infty}\left[\frac{A}{b} - \frac{S(t) - S(0)}{t} + \frac{\sigma_1}{t}\int_0^t S(s)dW_1(s)\right] \leq \frac{A}{b}.$$

**Theorem 4.** *If* $\frac{\sigma_1^2}{2} + \frac{1}{2} < b$, $k_1 + \frac{\sigma_2^2}{2} + \frac{1}{2} < \rho$, $k_2 + \frac{\sigma_3^2}{2} + \frac{1}{2} < \theta$, $\sigma_4^2 + \frac{1}{2} < b$, *then the solutions of* (3.1) *are stochastically ultimate bounded.*

*Proof.* For $(S(t), I_1(t), I_2(t), R(t)) \in \mathbb{R}_+^4$, define the following function

$$V_2(S(t), I_1(t), I_2(t), R(t)) = S(t)^2 + I_1(t)^2 + I_2(t)^2 + R(t)^2.$$

By Itô formula, one has

$$dV_2(S, I_1, I_2, R) = LV_2(S, I_1, I_2, R)dt + 2\sigma_1 S^2 dW_1 + 2\sigma_2 I_1^2 dW_2 + 2\sigma_3 I_2^2 dW_3 + 2\sigma_4 R^2 dW_4,$$

where

$$\begin{aligned} LV_2(S, I_1, I_2, R) =& 2\left(AS - \frac{k_1 S^2 I_1}{a + S} - \frac{k_2 S^2 I_2}{a + S} - bS^2\right) + 2I_1^2\left(\frac{k_1 S}{a + S} + \mu I_2 - \rho\right) \\ &+ 2I_2^2\left(\frac{k_2 S}{a + S} - \mu I_1 - \theta\right) + 2(\gamma I_1 R + \delta I_2 R - bR^2) + \sigma_1^2 S^2 + \sigma_2^2 I_1^2 + \sigma_3^2 I_2^2 + \sigma_4^2 R^2 \\ \leq& (\sigma_1^2 - 2b + 1)S^2 + \left(2k_1 + \sigma_2^2 - 2\rho + 1\right)I_1^2 + \left(2k_2 + \sigma_3^2 - 2\theta + 1\right)I_2^2 + \left(\sigma_4^2 - 2b + 1\right)R^2 \\ &- \left[S(t)^2 + I_1(t)^2 + I_2(t)^2 + R(t)^2\right] + 2\left(AS + \mu I_1^2 I_2 + \gamma I_1 R + \delta I_2 R\right). \end{aligned}$$

Assume $f(S(t), I_1(t), I_2(t), R(t)) = (\sigma_1^2 - 2b + 1)S^2 + \left(2k_1 + \sigma_2^2 - 2\rho + 1\right)I_1^2 + \left(2k_2 + \sigma_3^2 - 2\theta + 1\right)I_2^2 + \left(\sigma_4^2 - 2b + 1\right)R^2 + 2\left(AS + \mu I_1^2 I_2 + \gamma I_1 R + \delta I_2 R\right)$. According to Lemma 2 and Remark 3, one can find that the function $f(S(t), I_1(t), I_2(t), R(t))$ has an upper bound.

Let $N_1 = \sup f(S(t), I_1(t), I_2(t), R(t)) + 1$. As a result

$$dV_2(S, I_1, I_2, R) = (N_1 - V_2)dt + 2\sigma_1 S^2 dW_1 + 2\sigma_2 I_1^2 dW_2 + 2\sigma_3 I_2^2 dW_3 + 2\sigma_4 R^2 dW_4.$$

By Itô formula, one obtains

$$d(e^t V_2) \leq e^t N_1 dt + e^t \left[2\sigma_1 S^2 dW_1 + 2\sigma_2 I_1^2 dW_2 + 2\sigma_3 I_2^2 dW_3 + 2\sigma_4 R^2 dW_4\right].$$

Integrating both sides of the above equation from 0 to t and then taking expectations, one gets

$$e^t V_2(S(t), I_1(t), I_2(t), R(t)) \leq V_2(S(0), I_1(0), I_2(0), R(0)) + N_1 e^t - N_1,$$

hence

$$\limsup_{t\to\infty} \mathbb{E}[|X(t)|^2] \leq N_1.$$

Using Chebyshev's inequality, one can obtains

$$\mathbb{P}[|X(t)| \geq H] \leq \frac{\mathbb{E}[|X(t)|^2]}{H^2},$$

where $H = \frac{\sqrt{N_1}}{\sqrt{\epsilon}},\ \epsilon > 0$. Then

$$\limsup_{t\to\infty} \mathbb{P}[|X(t)| \geq H] \leq \frac{N_1}{H^2} = \epsilon.$$

This completes the proof. □

In the following, we will establish the conditions for extinction of hepatitis C virus from the stochastic system (3.1). The threshold parameter $R_{01}^s$ defined by $R_{01}^s = \frac{k_1}{\rho+\frac{\sigma_2^2}{2}}$, while the threshold parameter $R_{02}^s$ defined by $R_{02}^s = \frac{k_2}{\theta+\frac{\sigma_3^2}{2}}$ and the threshold parameter $R_{03}^s$ defined by $R_{03}^s = \frac{k_1+k_2}{\rho+\theta}$. The stochastic basic reproduction number $R_0^s$ defined by $R_0^s = \max\left\{R_{01}^s, R_{02}^s, R_{03}^s\right\}$.

**Theorem 5.** *The disease die out exponentially with probability one if* $R_0^s < 1$.

*Proof.* Let $V_3(I_1, I_2) = \ln(I_1 + I_2)$. Using Itô formula, one obtains

$$\begin{aligned}
d(V_3(I_1, I_2)) =& \left[\frac{1}{(I_1+I_2)}\left(\frac{k_1 S I_1}{a+S} + \frac{k_2 S I_2}{a+S} - \rho I_1 - \theta I_2\right) - \frac{\sigma_2^2 I_1^2}{2(I_1+I_2)^2} - \frac{\sigma_3^2 I_2^2}{2(I_1+I_2)^2}\right] dt \\
&+ \frac{\sigma_2 I_1}{(I_1+I_2)} dW_2 + \frac{\sigma_3 I_2}{(I_1+I_2)} dW_3 \\
\leq& \frac{1}{(I_1+I_2)^2}\left[\left(\frac{k_1 S}{(a+S)} - \rho - \frac{\sigma_2^2}{2}\right) I_1^2 + \left(\frac{k_2 S}{(a+S)} - \theta - \frac{\sigma_3^2}{2}\right) I_2^2 + \left(\frac{k_1 S}{(a+S)} - \rho\right) I_1 I_2\right. \\
&\left. + \left(\frac{k_2 S}{(a+S)} - \theta\right) I_1 I_2\right] dt + \frac{\sigma_2 I_1}{(I_1+I_2)} dW_2 + \frac{\sigma_3 I_2}{(I_1+I_2)} dW_3 \\
\leq& \frac{1}{(I_1+I_2)^2}\left[\left(k_1 - \rho - \frac{\sigma_2^2}{2}\right) I_1^2 + \left(k_2 - \theta - \frac{\sigma_3^2}{2}\right) I_2^2 + (k_1 - \rho) I_1 I_2\right. \\
&\left. + (k_2 - \theta) I_1 I_2\right] dt + \frac{\sigma_2 I_1}{(I_1+I_2)} dW_2 + \frac{\sigma_3 I_2}{(I_1+I_2)} dW_3 \\
\leq& \left[(\rho + \frac{\sigma_2^2}{2})(R_{01}^s - 1) + (\theta + \frac{\sigma_3^2}{2})(R_{02}^s - 1) + (\rho + \theta)(R_{03}^s - 1)\right] dt \\
&+ \frac{\sigma_2 I_1}{(I_1+I_2)} dW_2 + \frac{\sigma_3 I_2}{(I_1+I_2)} dW_3 \\
\leq& \left[2(\rho+\theta) + \frac{\sigma_2^2}{2} + \frac{\sigma_3^2}{2}\right](R_0^s - 1)\, dt + \frac{\sigma_2 I_1}{(I_1+I_2)} dW_2 + \frac{\sigma_3 I_2}{(I_1+I_2)} dW_3,
\end{aligned}$$

using integration from 0 to $t$, one obtains

$$\ln\,(I_1(t)+I_2(t)) \leq \ln\,(I_1(0)+I_2(0)) + \left[2(\rho+\theta)+\frac{\sigma_2^2}{2}+\frac{\sigma_3^2}{2}\right](R_0^s-1)\,t + \int_0^t \frac{\sigma_2 I_1}{2(I_1+I_2)}dW_2 + \int_0^t \frac{\sigma_3 I_2}{2(I_1+I_2)}dW_3.$$

Applying strong law of large numbers for local martingales one gets

$$\limsup_{t\to\infty}\frac{\ln\,(I_1(t)+I_2(t))}{t} \leq \left[2(\rho+\theta)+\frac{\sigma_2^2}{2}+\frac{\sigma_3^2}{2}\right](R_0^s-1) < 0,$$

as a result, diseases $I_1(t)$ and $I_2(t)$ die out and tend to zero exponentially a.s., if $R_0^s < 1$. □

**Remark 6.** *Theorem 3 indicates that the disease die out exponentially a.s., if $R_0^s < 1$ with the consequence that the recover class $R(t)$ also goes to extinction a.s.*

## 4. Numerical simulations

In this part, the numerical results will be compared with the theorems formulated in the previous sections. The interactions between populations classes will be simulated by the following parameters [32]: $N = 1000000, \mu = 0.02, a = 1, b = 0.02, k_1 = 0.00001, k_2 = 0.00002, \gamma = 0.001, \delta = 0.001$. To give some numerical finding to the HCV system (3.1), we use the Milstein method mentioned in [2, 3, 15]. The stochastic hepatitis virus C system (3.1) reduces to the following discrete system

$$\begin{aligned}
S_{(j+1)} &= x_j + h\left(A - \frac{k_1 S_j I_{1j}}{a+S_j} - \frac{k_2 S_j I_{2j}}{a+S} - bS_j\right) + \sigma_1 S_j\sqrt{h}\epsilon_{1j} + \frac{\sigma_1^2}{2}S_j\left[\epsilon_{1j}^2-1\right]h,\\
I_{1(j+1)} &= I_{1j} + h\left(\frac{k_1 S_j I_{1j}}{a+S_j} + \mu I_{1j}I_{2j} - bI_{1j} - \gamma I_{1j}\right) + \sigma_2 I_{1j}\sqrt{h}\epsilon_{2j} + \frac{\sigma_2^2}{2}I_{1j}\left[\epsilon_{2j}^2-1\right]h,\\
I_{2(j+1)} &= I_{2j} + h\left(\frac{k_2 S_j I_{2j}}{a+S_j} - \mu I_{1j}I_{2j} - bI_{2j} - \delta I_{2j}\right) + \sigma_3 I_{2j}\sqrt{h}\epsilon_{3j} + \frac{\sigma_3^2}{2}I_{2j}\left[\epsilon_{3j}^2-1\right]h,\\
R_{(j+1)} &= R_j + h\left(\gamma I_{1j} + \delta I_{2j} - bR_j\right) + \sigma_4 R_j\sqrt{h}\epsilon_{4j} + \frac{\sigma_4^2}{2}R_j\left[\epsilon_{4j}^2-1\right]h,
\end{aligned} \tag{4.1}$$

where $h$ is a positive time increment and $\epsilon_{ij}, (i = 1, 2, 3, 4)$ are independent random Gaussian variables $N(0, 1)$. One can note that for the given parameters, the value of the stochastic basic reproduction number $R_0^s = 0.00095$. As a result, the conditions of Theorem 5 are verified and the disease die out exponentially with probability one if $R_0^s < 1$. Figure 1 represents the dynamical behavior of model (3.1) when the noise strength law ($\sigma_i = 0.01$). For $k_1 = k_2 = 0.0308$, the conditions of Theorem 5 are verified as $R_0^s = 1.130769 > 1$ as shown in Figure 2. It is shown that the trajectories of the stochastic system (3.1) oscillates around the coexistence equilibrium of the deterministic system (1.1).

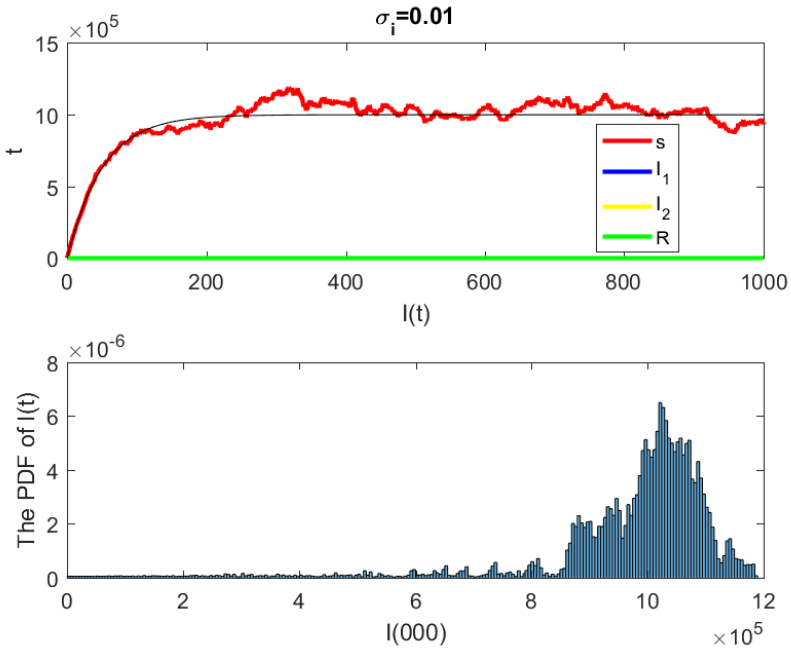


**Figure 1.** Fluctuation in susceptible population with with $R_0^s = 0.00095 < 1$.

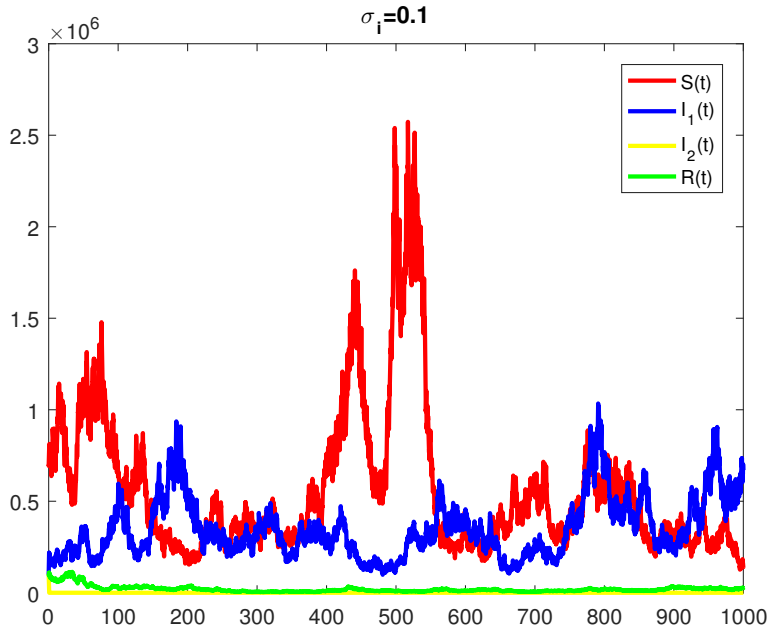


**Figure 2.** The evolution of infected and recovered individuals with $R_0^s = 1.3968 > 1$.

## 5. Conclusions

The novelty of this study is that we introduced a new compartment and saturated incidence rate into the classical hepatitis C virus genome model. A deterministic and stochastic model for hepatitis C with different types of virus genomes is proposed and analyzed. We perform a stochastic extension of the deterministic model to study the fluctuation between environmental factors. Firstly, the existence of a unique global positive solution for the stochastic model is investigated. Secondly, sufficient conditions for the extinction of hepatitis C virus from the stochastic system are obtained. Theoretical results are illustrated using numerical simulations. Theoretical and numerical results show that the smaller white noise can ensure the persistence of susceptible and infected populations while the larger white noise can lead to the extinction of populations. For the deterministic model, some sufficient conditions are obtained to ensure the stability of the equilibrium points. The results show that when the basic reproduction number $R_0 < 1$, the deterministic model is asymptotic stable around the free disease equilibrium point $E_0$. By introducing the basic reproduction number $R_0$ and the stochastic basic reproduction number $R_0^s$, the conditions that cause the disease to die out are indicated. Biologically, it can be noted that by social distancing with hepatitis C patients, this can lead to a fluctuation in the value of $k_1$ and $k_2$, and thus $R_0^s$ becomes less than one, which leads to the disappearance of the epidemic.

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## Conflict of interest

The authors declare that there is no conflict of interests.

## References

1. S. Allegretti, I. M. Bulai, R. Marino, M. A. Menandro, K. Parisi, Vaccination effect conjoint to fraction of avoided contacts for a SARS-CoV-2 mathematical model, *Math. Model. Numer. Simul. Appl.*, **1** (2021), 56–66. https://doi.org/10.53391/mmnsa.2021.01.006
2. Y. Alnafisah, The implementation of milstein scheme in two-dimensional SDEs using the fourier method, *Abstr. Appl. Anal.*, **2018** (2018). https://doi.org/10.1155/2018/3805042
3. Y. A. Alnafisah, Comparison between milstein and exact coupling methods using MATLAB for a particular two-dimensional stochastic differential equation, *J. Inform. Sci. Eng.*, **36** (2020), 1223–1232.
4. I. Bashkirtseva, L. Ryashko, T. Ryazanova, Analysis of regular and chaotic dynamics in a stochastic eco-epidemiological model, *Chaos Soliton. Fract.*, **131** (2020), 109549. https://doi.org/10.1016/j.chaos.2019.109549

5. B. Berrhazi, M. El Fatini, A. Lahrouz, A. Settati, R. Taki, A stochastic SIRS epidemic model with a general awareness-induced incidence, *Physica A*, **512** (2018), 968–980. https://doi.org/10.1016/j.physa.2018.08.150
6. Y. Cai, Y. Kang, W. Wang, A stochastic SIRS epidemic model with nonlinear incidence rate, *Appl. Math. Comput.*, **305** (2017), 221–240. https://doi.org/10.1016/j.amc.2017.02.003
7. Y. Cai, X. Mao, Stochastic prey-predator system with foraging arena scheme, *Appl. Math. Model.*, **64** (2018), 357–371. https://doi.org/10.1016/j.apm.2018.07.034 https://doi.org/10.1080/17442508.2019.1612897
8. Z. Chang, X. Meng, X. Lu, Analysis of a novel stochastic SIRS epidemic model with two different saturated incidence rates, *Physica A*, **472** (2017), 103–116. https://doi.org/10.1016/j.physa.2017.01.015
9. J. Cresson, B. Puig, S. Sonner, Stochastic models in biology and the invariance problem, *Discrete Cont. Dyn.-B*, **21** (2016), 2145. https://doi.org/10.3934/dcdsb.2016041
10. J. Cresson, S. Sonner, A note on a derivation method for sde models: Applications in biology and viability criteria, *Stoch. Anal. Appl.*, **36** (2018), 224–239. https://doi.org/10.1080/07362994.2017.1386571
11. B. Daşbaşı, Stability analysis of an incommensurate fractional-order SIR model, *Math. Model. Numer. Simul. Appl.*, **1** (2021), 44–55. http://doi.org/10.53391/mmnsa.2021.01.005
12. T. Feng, Z. Qiu, X. Meng, Dynamics of a stochastic hepatitis C virus system with host immunity, *Discrete Cont. Dyn.-B*, **24** (2019), 6367. https://doi.org/10.3934/dcdsb.2019143
13. C. Gokila, M. Sambath, K. Balachandran, Y. K. Ma, Analysis of stochastic predator-prey model with disease in the prey and Holling type II functional response, *Adv. Math. Phys.*, **2020** (2020). https://doi.org/10.1155/2020/3632091
14. Z. Hammouch, M. Yavuz, N. Özdemir, Numerical solutions and synchronization of a variable-order fractional chaotic system, *Math. Model. Numer. Simul. Appl.*, **1** (2021), 11–23. https://doi.org/10.53391/mmnsa.2021.01.002
15. D. J. Higham, An algorithmic introduction to numerical simulation of stochastic differential equations, *SIAM Review*, **43** (2001), 525–546. https://doi.org/10.1137/S0036144500378302
16. J. L. Horsley-Silva, H. E. Vargas, New therapies for hepatitis C virus infection, *Gastroenterol. Hepat.*, **13** (2017), 22.
17. R. Ikram, A. Khan, M. Zahri, A. Saeed, M. Yavuz, P. Kumam, Extinction and stationary distribution of a stochastic COVID-19 epidemic model with time-delay, *Comput. Biol. Med.*, **141** (2022), 105115. https://doi.org/10.1016/j.compbiomed.2021.105115
18. J. Jiang, S. Gong, B. He, Dynamical behavior of a rumor transmission model with Holling-type II functional response in emergency event, *Physica A*, **450** (2019), 228–240. https://doi.org/10.1016/j.physa.2015.12.143
19. T. Kar, P. K. Mondal, Global dynamics and bifurcation in delayed SIR epidemic model, *Nonlinear Anal.-Real*, **12** (2011), 2058–2068. https://doi.org/10.1016/j.nonrwa.2010.12.021
20. A. Khan, G. Hussain, A. Yusuf, A. H. Usman, A hepatitis stochastic epidemic model with acute and chronic stages, *Adv. Differ. Equ.*, **2021** (2021), 1–10. https://doi.org/10.1186/s13662-021-03335-7

21. P. Kumar, V. S. Erturk, Dynamics of cholera disease by using two recent fractional numerical methods, *Math. Model. Numer. Simul. Appl.*, **1** (2021), 102–111. https://doi.org/10.53391/mmnsa.2021.01.010

22. G. Lan, S. Yuan, B. Song, The impact of hospital resources and environmental perturbations to the dynamics of SIRS model, *J. Franklin I.*, **358** (2021), 2405–2433. https://doi.org/10.1016/j.jfranklin.2021.01.015

23. D. Lestari, N. Y. Megawati, N. Susyanto, F. Adi-Kusumo, Qualitative behaviour of a stochastic hepatitis C epidemic model in cellular level, *Math. Biosci. Eng.*, **19** (2022), 1515–1535. https://doi.org/10.3934/mbe.2022070

24. J. Li, K. Men, Y. Yang, D. Li, Dynamical analysis on a chronic hepatitis C virus infection model with immune response, *J. Theor. Biol.*, **365** (2015), 337–346. https://doi.org/10.1016/j.jtbi.2014.10.039

25. J. Li, M. Shan, M. Banerjee, W. Wang, Stochastic dynamics of feline immunodeficiency virus within cat populations, *J. Franklin I.*, **353** (2016), 4191–4212. https://doi.org/10.1016/j.jfranklin.2016.08.004

26. L. Li, W. Zhao, Deterministic and stochastic dynamics of a modified Leslie-Gower prey-predator system with simplified Holling-type IV scheme, *Math. Biosci. Eng.*, **18** (2021), 2813–2831.

27. Q. Li, F. Cong, T. Liu, Y. Zhou, Stationary distribution of a stochastic HIV model with two infective stages, *Physica A*, **554** (2020), 124686. https://doi.org/10.1016/j.physa.2020.124686

28. Q. Liu, D. Jiang, T. Hayat, A. Alsaedi, B. Ahmad, A stochastic SIRS epidemic model with logistic growth and general nonlinear incidence rate, *Physica A*, **551** (2020), 124152. https://doi.org/10.1016/j.physa.2020.124152

29. P. S. Mandal, M. Banerjee, Stochastic persistence and stationary distribution in a Holling-Tanner type prey-predator model, *Physica A*, **391** (2012), 1216–1233. https://doi.org/10.1016/j.physa.2011.10.019

30. X. Mao, *Stochastic differential equations and applications*, Elsevier, 2007.

31. I. Moneim, M. Al-Ahmed, G. Mosa, Stochastic and monte carlo simulation for the spread of thehepatitis B, *Aust. J. Basic Appl. Sci.*, **3** (2009), 1607–1615.

32. I. Moneim, G. Mosa, Modelling the hepatitis C with different types of virus genome, *Comput. Math. Method. M.*, **7** (2006), 3–13. https://doi.org/10.1080/10273660600914121

33. Y. Mu, W. C. Lo, Stochastic dynamics of populations with refuge in polluted turbidostat, *Chaos Soliton. Fract.*, **147** (2021), 110963. https://doi.org/10.1016/j.chaos.2021.110963

34. P. A. Naik, Z. Eskandari, H. E. Shahraki, Flip and generalized flip bifurcations of a two-dimensional discrete-time chemical model, *Math. Model. Numer. Simul. Appl.*, **1** (2021), 95–101. https://doi.org/10.53391/mmnsa.2021.01.009

35. F. Özköse, M. Yavuz, Investigation of interactions between COVID-19 and diabetes with hereditary traits using real data: A case study in Turkey, *Comput. Biol. Med.*, **141** (2022), 105044. https://doi.org/10.1016/j.compbiomed.2021.105044

36. F. Özköse, M. Yavuz, M. T. Şenel, R. Habbireeh, Fractional order modelling of omicron SARS-CoV-2 variant containing heart attack effect using real data from the United Kingdom, *Chaos Soliton. Fract.*, **157** (2022), 111954. https://doi.org/10.1016/j.chaos.2022.111954

37. T. A. Phan, J. P. Tian, B. Wang, Dynamics of cholera epidemic models in fluctuating environments, *Stoch. Dynam.*, **21** (2021), 2150011. https://doi.org/10.1142/S0219493721500118

38. S. Rajasekar, M. Pitchaimani, Qualitative analysis of stochastically perturbed SIRS epidemic model with two viruses, *Chaos Soliton. Fract.*, **118** (2019), 207–221. https://doi.org/10.1016/j.chaos.2018.11.023

39. R. Shi, T. Lu, C. Wang, Dynamic analysis of a fractional-order model for hepatitis B virus with Holling II functional response, *Complexity*, **2019** (2019). https://doi.org/10.1155/2019/1097201

40. G. Song, Dynamics of a stochastic population model with predation effects in polluted environments, *Adv. Differ. Equ.*, **2021** (2021), 1–19. https://doi.org/10.1186/s13662-021-03297-w

41. X. Wang, C. Wang, K. Wang, Extinction and persistence of a stochastic SICA epidemic model with standard incidence rate for HIV transmission, *Adv. Differ. Equ.*, **2021** (2021), 1–17. https://doi.org/10.1186/s13662-021-03392-y

42. X. Wang, Y. Tan, Y. Cai, K. Wang, W. Wang, Dynamics of a stochastic HBV infection model with cell-to-cell transmission and immune response, *Math. Biosci. Eng.*, **18** (2021), 616–642. https://doi.org/10.3934/mbe.2021034

43. Z. Wang, M. Deng, M. Liu, Stationary distribution of a stochastic ratio-dependent predator-prey system with regime-switching, *Chaos Soliton. Fract.*, **142** (2021), 110462. https://doi.org/10.1016/j.chaos.2020.110462

44. C. Wei, J. Liu, S. Zhang, Analysis of a stochastic eco-epidemiological model with modified Leslie-Gower functional response, *Adv. Differ. Equ.*, **2018** (2018), 1–17. https://doi.org/10.1186/s13662-018-1540-z

45. C. Xu, G. Ren, Y. Yu, Extinction analysis of stochastic predator-prey system with stage structure and crowley-martin functional response, *Entropy*, **21** (2019), 252. https://doi.org/10.3390/e21030252

46. M. Yavuz, N. Sene, Stability analysis and numerical computation of the fractional predator-prey model with the harvesting rate, *Fractal Fract.*, **4** (2020), 35. https://doi.org/10.3390/fractalfract4030035

47. J. Z. Zhang, Z. Jin, Q. X. Liu, Z. Y. Zhang, Analysis of a delayed SIR model with nonlinear incidence rate, *Discrete Dyn. Nat. Soc.*, **2008** (2008). https://doi.org/10.1155/2008/636153

48. Q. Zhang, X. Wen, D. Jiang, Z. Liu, The stability of a predator-prey system with linear mass-action functional response perturbed by white noise, *Adv. Differ. Equ.*, **2016** (2016), 1–24. https://doi.org/10.1186/s13662-016-0776-8


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