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Research article

A stochastic SICR model for hepatitis C virus transmission with Ornstein-Uhlenbeck process

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Abstract: This paper proposes a stochastic susceptible-acute-chronic-recovery (SICR) model for hepatitis C virus (HCV) transmission, in which environmental fluctuations in the transmission rate are modeled by a logarithmic Ornstein-Uhlenbeck process. This formulation preserves the positivity of parameters while capturing the mean-reverting stochasticity. A sufficient condition for disease extinction is established in terms of a stochastic threshold $\mathcal{R}_0^e$: if $\mathcal{R}_0^e < 1$, then the infection becomes extinct almost surely. Conversely, when $\mathcal{R}_0^s > 1$, the model admits at least one ergodic stationary distribution, which implies a long-term persistence of the disease. Furthermore, under the condition $\mathcal{R}_0 > 1$, an explicit probability density function for the stationary distribution near the quasi-endemic equilibrium is derived, thus providing a characterization of stationary fluctuations. Numerical simulations are conducted to validate the theoretical findings and to elucidate the influence of noise parameters on the disease dynamics. The results demonstrate that environmental fluctuations can sustain HCV transmission even when the deterministic reproduction number is below unity, thus underscoring the critical role of stochasticity in HCV transmission.

Keywords: hepatitis C virus; stochastic epidemic model; Ornstein-Uhlenbeck process; extinction; stationary distribution; probability density function
Mathematics Subject Classification: 34F05, 37H10, 60J70, 92B05

1. Introduction

Hepatitis C virus (HCV) infection remains a major global health burden, with approximately 50 million chronic cases and 1.0 million new infections annually [1]. In 2022, an estimated 242,000 deaths–mostly from cirrhosis and hepatocellular carcinoma–were attributed to HCV, making it a leading cause of liver-related mortality worldwide. The natural history of HCV infection is divided into acute and chronic phases. Acute infection is usually asymptomatic and rarely life-threatening, with spontaneous clearance occurring in 15% to 45% of infected persons within six months. Chronic

infection develops in the remaining 55% to 85% of cases, among whom the 20-year risk of cirrhosis ranges from 15% to 30% [1]. HCV transmission primarily occurs through exposure to infected blood. Major routes of transmission include unsafe medical procedures, needle sharing, transfusion of blood or blood products, mother-to-child transmission, and sexual contact [2]. In contrast to hepatitis B, no effective vaccine is currently available against HCV, thus posing substantial challenges for prevention [3–5]. Although the advent of direct-acting antiviral agents has significantly improved cure rates for HCV, high treatment costs and limited healthcare resources continue to hinder global disease control efforts [6].

Mathematical models have long been recognized as essential tools to elucidate the transmission dynamics of infectious diseases, a paradigm that holds true for HCV. The foundation of HCV modeling traces back to the seminal work of Neumann et al. [7], who introduced a system of ordinary differential equations in 1998 to describe the interplay among healthy hepatocytes, infected cells, and free virus. Building upon this foundation, subsequent research recognized that HCV infection is characterized by distinct acute and chronic phases, calling for more refined model structures. Yuan and Yang [8] first incorporated both acute and chronic stages into an SEIV model, highlighting the contribution of chronic infection to disease persistence. Zhang and Zhou [9] further extended these frameworks by permitting reinfection from the recovered class, thereby accounting for the fact that protective immunity following HCV infection is not fully established.

The most relevant deterministic baseline for our work is the susceptible-acute-chronic-recovery (SICR) model of HCV proposed by Cui et al. [10], which explicitly differentiates between acute and chronic HCV infections. This model captures the clinically important feature that acute patients may experience spontaneous recovery, while chronic patients do not. The model is formulated as follows:

$$\begin{cases} \frac{\mathrm{d}S}{\mathrm{d}t} = \Lambda - \beta S(I + C) - aS, \\ \frac{\mathrm{d}I}{\mathrm{d}t} = \beta S(I + C) - (a + \gamma)I, \\ \frac{\mathrm{d}C}{\mathrm{d}t} = p\gamma I - (a + \mu)C, \\ \frac{\mathrm{d}R}{\mathrm{d}t} = (1 - p)\gamma I - aR, \end{cases} \tag{1}$$

where S, I, C, and R denote the susceptible, acutely infected, chronically infected, and recovered populations, respectively. The parameter Λ represents the recruitment rate, β represents the transmission rate, a represents the natural death rate, p represents the proportion of acute infections that progress to chronicity, and μ represents the disease-induced death rate for chronic patients. The parameter γ denotes the rate at which acute infection is resolved–this includes both spontaneous recovery (with probability $1 - p$) and progression to chronic infection (with probability p). Within this framework, the basic reproduction number

$$\mathcal{R}_0 = \frac{\beta\Lambda}{a(a + \gamma)} + \frac{\beta\Lambda p\gamma}{a(a + \gamma)(a + \mu)},$$

functions as a threshold parameter for global dynamics: When $\mathcal{R}_0 < 1$, the infection-free equilibrium is globally asymptotically stable, leading to disease extinction; conversely, when $\mathcal{R}_0 > 1$, a unique endemic equilibrium exists and is globally asymptotically stable, indicating disease persistence. A key clinically relevant feature of this model is that acute patients may experience spontaneous recovery, while chronic patients do not, thus positioning it as an appropriate baseline for further extensions.

Concurrent developments have centered on incorporating host immune responses into modeling frameworks [11–13]. Wodarz [14] examined the role of cytotoxic T lymphocytes (CTL) and antibody responses in HCV dynamics, whereas subsequent studies have incorporated the effects of cytokines, including IFN-γ and IL-2 [15], as well as the essential role of helper T cells in sustaining CTL activity [16]. These modeling efforts have revealed that the magnitude and timing of immune responses significantly influence the disease outcomes, from spontaneous clearance to chronic progression.

Despite the valuable insights offered by deterministic models, these approaches inherently neglect the stochastic fluctuation characteristics of real-world epidemiological processes. Variability in environmental conditions, social behaviors, seasonal factors, and the inherent randomness of pathogen transmission can profoundly influence the disease dynamics, thereby potentially modifying the threshold conditions and producing outcomes that deviate from deterministic expectations. For instance, Liu and Guo [17] studied stochastic within-host HCV dynamics. Qi et al. [18] analyzed a nonlinear HCV model under random fluctuations. Authors in [19, 20] examined the effects of protection awareness and environmental variability. Alnafisah and El-Shahed [21] compared deterministic and stochastic HCV models with different viral genome types. Rajasekar et al. [22] considered a chronically infected treated population. Yu and Ma [23] studied a dysentery model, and later [24] an avian influenza model. Yang et al. [25] investigated rate-induced tipping in a coupled human-environment system. However, the direct white-noise perturbation approach suffers from two major drawbacks. First, it may compromise the biological positivity: directly adding Gaussian noise to parameters can produce negative values, which are epidemiologically meaningless. Second, it treats fluctuations as uncorrelated in time, thereby ignoring the fact that environmental changes often exhibit mean-reverting behavior (e.g., seasonal effects or long-term control measures). To overcome this limitation while maintaining analytical tractability, several studies have turned to mean-reverting processes to model parameter fluctuations. Dong et al. [26] used a logarithmic Ornstein-Uhlenbeck (O-U) process for an HIV model. Liu [27] applied it to a maize streak virus model. Zhang and Su [28] analyzed a stochastic HIV model with general incidence. Shi and Jiang [29] studied an SIS model with generalized nonlinear incidence. Zhou et al. [30] derived explicit probability densities for a stochastic SIQRS model.

Epidemiologically, the transmission rate β is influenced by factors that fluctuate around a long-term mean: seasonal variations in contact rates, public health interventions that are implemented and then relaxed, and population behavioral changes that revert over time. The O-U process is the simplest continuous-time mean-reverting process, and the logarithmic transformation ensures $\beta(t) > 0$. Based on the deterministic SICR framework of Cui et al. [10], we introduce stochasticity through an O-U process applied to the logarithm of β. Specifically, we model $\ln\beta$ as an O-U process:

$$\mathrm{d}\ln\beta = r(\ln\bar{\beta} - \ln\beta)\mathrm{d}t + \sigma\mathrm{d}B(t), \tag{2}$$

where $\bar{\beta}$ denotes the long-term mean transmission rate, $r > 0$ denotes the speed of mean-reversion, $\sigma > 0$ denotes the noise intensity, and $B(t)$ denotes a standard Brownian motion. Letting

$$x = \ln\beta - \ln\bar{\beta},$$

the resulting stochastic SICR model is given by the following:

$$\begin{cases} \mathrm{d}x = -rx\mathrm{d}t + \sigma \mathrm{d}B(t), \\ \frac{\mathrm{d}S}{\mathrm{d}t} = \Lambda - \bar{\beta}e^{x}S(I+C) - aS, \\ \frac{\mathrm{d}I}{\mathrm{d}t} = \bar{\beta}e^{x}S(I+C) - (a+\gamma)I, \\ \frac{\mathrm{d}C}{\mathrm{d}t} = p\gamma I - (a+\mu)C. \end{cases} \tag{3}$$

Since the dynamics of S, I, and C are completely determined by the first three equations in Model (1), the extinction and persistence of the infection are solely determined by $I(t)$ and $C(t)$. Once $I(t)$ and $C(t)$ are known, $R(t)$ can be explicitly solved from its linear ordinary differential equations. Therefore, omitting R does not affect any of our main results. When $\sigma = 0$, the O-U process reduces to the deterministic case $\beta(t) = \bar{\beta}$, and consequently, model (3) degenerates to the deterministic model (1).

Substantial progress has been made in stochastic HCV modeling, yet key limitations persist: direct white-noise perturbations may compromise the biological positivity [21, 22], and explicit probability density functions are seldom derived [17–20]. To address these gaps, this paper ordinary differential equations the following: (i) proposes a stochastic HCV model where the transmission rate follows a logarithmic O-U process; (ii) derives a sufficient extinction condition $\mathcal{R}_0^e < 1$ that explicitly incorporates the noise intensity σ and the mean-reversion speed r; (iii) proves the existence of an ergodic stationary distribution when $\mathcal{R}_0^s > 1$, thus implying long-term disease persistence; (iv) obtains an explicit expression for the probability density function near the quasi-positive equilibrium when $\mathcal{R}_0 > 1$; (v) numerically demonstrates that environmental fluctuations can sustain HCV transmission even when the deterministic $\mathcal{R}_0 < 1$.

The remainder of this paper is structured as follows: Section 2 presents the preliminaries, including global solution existence and an invariant absorbing domain; Section 3 establishes the extinction condition via the stochastic threshold $\mathcal{R}_0^e$; Section 4 proves disease persistence by demonstrating an ergodic stationary distribution when $\mathcal{R}_0^s > 1$; Section 5 derives the explicit probability density function of the stationary distribution near the quasi-positive equilibrium. Section 6 provides numerical simulations to verify our theoretical results; and finally, we conclude with a discussion of the main findings and their public health implications.

2. Preliminaries

We work on a complete probability space $(\Omega, \mathcal{F}, \{\mathcal{F}\}_{t\geq 0}, \mathbb{P})$ with a filtration $\{\mathcal{F}\}_{t\geq 0}$ that satisfies the usual conditions, and let $B(t)$ be a standard Brownian motion adapted to this filtration. The system state is denoted by $X(t) = (x(t), S(t), I(t), C(t))$, $t \geq 0$, with the initial condition $X_0 \in \mathbb{R} \times \mathbb{R}^3_+$. Standard notation includes $\mathbb{R}^n$ for the Euclidean space and $\mathbb{R}^n_+ = x \in \mathbb{R}^n : x_i > 0, i = 1, \ldots, n$ for its positive cone. For any matrix M, M^T and M^{-1} denote its transpose and inverse, respectively.

To facilitate the analysis of model (3), we recall some essential results for stochastic differential equations. Consider the general equation:

$$\mathrm{d}z(t) = F_1(z,t)\mathrm{d}t + F_2(z,t)\mathrm{d}B(t), \tag{4}$$

where $B(t)$ is an m-dimensional Brownian motion. For any $C^{2,1}$ function $V(z,t)$, the operator $\mathcal{L}$ is given

by the following:

$$\mathcal{L}V(z,t) = \frac{\partial V}{\partial t} + \frac{\partial V}{\partial z}F_1(z,t) + \frac{1}{2}\text{trace}\left(F_2^T(z,t)\frac{\partial^2 V}{\partial z^2}F_2(z,t)\right),$$

where subscripts denote partial derivatives.

Lemma 2.1. *[29] For the logarithmic transmission rate process*

$$\mathrm{d}\ln\beta = r(\ln\bar{\beta} - \ln\beta)\mathrm{d}t + \sigma\mathrm{d}B(t),$$

with positive constants r, σ, $\bar{\beta}$, and standard Brownian motion B(t), the time average satisfies:

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t \beta^k(\tau)\mathrm{d}\tau = \bar{\beta}^k \exp\Big(\frac{k^2\sigma^2}{4r}\Big)\ a.s.,\quad for\ any\ \ k > 0.$$

A direct consequence of the ergodic property is that for $\beta = \bar{\beta}e^x$,

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t e^{kx(\tau)}\mathrm{d}\tau = \exp\Big(\frac{k^2\sigma^2}{4r}\Big)\ a.s.$$

Lemma 2.2. *[31, 32] Model* (3) *admits at least one invariant probability measure (i.e., an ergodic stationary distribution) if there exists a bounded closed domain $D \subset \mathbb{R}^4$ such that*

$$\liminf_{t\to+\infty}\frac{1}{t}\int_0^t \mathbb{P}(\tau, X(\tau), D)\mathrm{d}\tau > 0\ a.s.,$$

where $\mathbb{P}(\tau, X(\tau), D)$ denotes the transition probability of $X(\tau) \in D$.

Lemma 2.3. *[30] Let Θ_0 be the symmetric matrix that solves the real algebraic equation $G_0^2 + A_0\Theta_0 + \Theta_0 A_0^T = 0$, where $G_0 = diag(1,0,0,0)$ and*

$$A_0 = \begin{pmatrix} -\varrho_1 & -\varrho_2 & -\varrho_3 & -\varrho_4 \\ 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \end{pmatrix}.$$

If $\varrho_1 > 0$, $\varrho_3 > 0$, $\varrho_4 > 0$, and $\varrho_1(\varrho_2\varrho_3 - \varrho_1\varrho_4) - \varrho_3^2 > 0$, then Θ_0 is a positive definite matrix given by

$$\Theta_0 = \begin{pmatrix} \frac{\varrho_2\varrho_3 - \varrho_1\varrho_4}{\varrho_3}\varrho & 0 & -\varrho & 0 \\ 0 & \varrho & 0 & -\frac{\varrho_1}{\varrho_3} \\ -\varrho & 0 & \frac{\varrho_1}{\varrho_3}\varrho & 0 \\ 0 & -\frac{\varrho_1}{\varrho_3} & 0 & \frac{\varrho_1\varrho_2 - \varrho_3}{\varrho_3\varrho_4}\varrho \end{pmatrix},$$

with

$$\varrho = \frac{\varrho_3}{2(\varrho_1(\varrho_2\varrho_3 - \varrho_1\varrho_4) - \varrho_3^2)}.$$

Theorem 2.1. *Given $X_0 \in \mathbb{R}\times\mathbb{R}_+^3$, there exists a unique global solution $X(t)$ to model* (3) *for $t \geq 0$, and $X(t) \in \mathbb{R}\times\mathbb{R}_+^3$ almost surely for all $t \geq 0$.*

Proof. Although the coefficients of model (3) are locally Lipschitz, they do not satisfy the linear growth requirement. Hence, the classical existence theorem only ensures a unique local solution $X(t)$ defined on $[0, \tau_e)$ for any prescribed initial condition X_0, with τ_e denoting the explosion time. To verify that the solution exists globally, we must establish $\tau_e = \infty$ with a probability of one.

Choose n_0 large enough such that all components of X_0 belong to the interval $[\frac{1}{n_0}, n_0]$. For every $n \geq n_0$, define the stopping time

$$\tau_n = \inf\left\{t \in [0, \tau_e)\Big| \max\{X_i\} \geq n \text{ or } \min\{X_i\} \leq \frac{1}{n},\ i = 1, 2, 3, 4\right\},$$

and let

$$\tau_\infty = \lim_{n\to\infty} \tau_n.$$

Clearly, $\tau_\infty \leq \tau_e$ a.s., and it suffices to show $\tau_\infty = \infty$ almost surely.

Introduce the auxiliary function V: $\mathbb{R} \times \mathbb{R}^3_+ \to \mathbb{R}_+$ given by the following:

$$V = (e^x - 1 - x) + \sum_{i=2}^{4}(X_i - 1 - \ln X_i).$$

Applying Itô's formula to V yields the following:

$$\begin{aligned}\mathcal{L}V =& - rx(e^x - 1) + \frac{1}{2}\sigma^2 e^x + \Big(1 - \frac{1}{S}\Big)\Big(\Lambda - \bar{\beta}e^x S(I + C) - aS\Big)\\ &+ \Big(1 - \frac{1}{I}\Big)\Big(\bar{\beta}e^x S(I + C) - (a + \gamma)I\Big) + \Big(1 - \frac{1}{C}\Big)(p\gamma I - (a + \mu)C)\\ \leq& - rx(e^x - 1) + \frac{1}{2}\sigma^2 e^x + \Lambda + 3a + \gamma + \mu + \bar{\beta}e^x(I + C).\end{aligned} \tag{5}$$

The dynamics of the total population are governed by the following:

$$\frac{\mathrm{d}}{\mathrm{d}t}(S + I + C) = \Lambda - a(S + I + C) - (1 - p)\gamma I - \mu C.$$

From this, we deduce that if $S(0)+I(0)+C(0) \leq \frac{\Lambda}{a}$, then $S+I+C \leq \frac{\Lambda}{a}$. Conversely, if $S(0)+I(0)+C(0) > \frac{\Lambda}{a}$, then $S + I + C \leq \frac{\Lambda}{a} \leq S(0) + I(0) + C(0)$. Let $\Delta = \max\left\{\frac{\Lambda}{a}, S(0) + I(0) + C(0)\right\}$; then, by (5), we obtain

$$\begin{aligned}\mathcal{L}V \leq& - rx(e^x - 1) + \frac{1}{2}\sigma^2 e^x + \Lambda + 3a + \gamma + \mu + \bar{\beta}\Delta e^x\\ =&\Lambda + 3a + \gamma + \mu + g(x),\end{aligned}$$

where

$$g(x) = -rx(e^x - 1) + \frac{1}{2}\sigma^2 e^x + \bar{\beta}\Delta e^x.$$

Because $g(x) \to -\infty$ when $x \to +\infty$ and also when $x \to -\infty$, there exists a constant ϕ such that $\sup_{x\in\mathbb{R}}\{g(x)\} \leq \phi$. Thus,

$$\mathcal{L}V \leq \Lambda + 3a + \gamma + \mu + \phi.$$

The remaining arguments follow the standard methodology detailed in [26]. □

Remark 2.1. *As a consequence of Theorem 2.1, the state process $X(t)$ is ultimately bounded. Define the domain:*

$$\Gamma = \left\{X(t) \in \mathbb{R} \times \mathbb{R}_+^3 : 0 < S + I + C \leq \frac{\Lambda}{a}\right\}. \tag{6}$$

Then, Γ is almost surely positively invariant and absorbing for the dynamics. Consequently, we restrict our subsequent analysis to this region.

With the global existence and boundedness of solutions established, we now turn to the central question in epidemic modeling: under what conditions does the infection become extinct? The following section provides a sufficient condition for the almost sure extinction of HCV in terms of a stochastic threshold $\mathcal{R}_0^e$, where

$$\mathcal{R}_0^e = \mathcal{R}_0 + \frac{\left(\frac{\bar{\beta}\Lambda}{a} + (a+\mu)\mathcal{R}_0\right)\left(e^{\frac{\sigma^2}{r}} - 2e^{\frac{\sigma^2}{4r}} + 1\right)^{\frac{1}{2}}}{\min\left\{a+\mu, \frac{\bar{\beta}\Lambda}{a\mathcal{R}_0}\right\}}.$$

3. Extinction of HCV

Theorem 3.1. *For any initial value $X_0 \in \Gamma$, the solution $X(t)$ of model* (3) *satisfies the following:*

$$\limsup_{t\to\infty} \frac{1}{t} \ln\left(\mathcal{R}_0 I + \frac{\bar{\beta}\Lambda}{a(a+\mu)} C\right) < \min\left\{a+\mu, \frac{\bar{\beta}\Lambda}{a\mathcal{R}_0}\right\}(\mathcal{R}_0^e - 1), \text{ a.s.}$$

In particular, if $\mathcal{R}_0^e < 1$, then

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

which implies that HCV undergoes the extinction with a probability of one.

Proof. Consider the nonnegative C^2-function V: $\mathbb{R}_+^2 \to \mathbb{R}_+$ given by the following:

$$V = \alpha_1 I + \alpha_2 C,$$

where α_1 and α_2 will be specified in due course. Applying Itô's formula to V yields the following:

$$\begin{aligned}
\frac{\mathrm{d}\ln V}{\mathrm{d}t} =& \frac{\alpha_1}{V}\left(\bar{\beta}e^x S(I+C) - (a+\gamma)I\right) + \frac{\alpha_2}{V}\left(p\gamma I - (a+\mu)C\right) \\
\leq& \frac{\alpha_1}{V}\left(\frac{\bar{\beta}\Lambda(I+C)}{a} - (a+\gamma)I\right) + \frac{\alpha_2}{V}\left(p\gamma I - (a+\mu)C\right) + \frac{\alpha_1\bar{\beta}\Lambda(I+C)}{aV}\left|e^x - 1\right| \\
=& \frac{1}{V}\left(\frac{\alpha_1\bar{\beta}\Lambda}{a}C - \alpha_2(a+\mu)C\right) + \frac{\alpha_1\bar{\beta}\Lambda(I+C)}{aV}\left|e^x - 1\right| + \frac{1}{V}\left(\frac{\alpha_1\bar{\beta}\Lambda}{a}I - (\alpha_1(a+\gamma) - \alpha_2 p\gamma)I\right).
\end{aligned} \tag{7}$$

Let

$$\alpha_1 = \frac{\bar{\beta}\Lambda}{a(a+\gamma)} + \frac{\bar{\beta}\Lambda p\gamma}{a(a+\gamma)(a+\mu)} = \mathcal{R}_0, \quad \alpha_2 = \frac{\bar{\beta}\Lambda}{a(a+\mu)},$$

such that

$$\frac{\bar{\beta}\Lambda}{a} = \alpha_2(a+\mu) = \alpha_1(a+\gamma) - \alpha_2 p\gamma.$$

Note that $\mathcal{R}_0 < \mathcal{R}_0^e$. Hence, $\mathcal{R}_0^e < 1$ is a sufficient condition for $\mathcal{R}_0 < 1$. It follows from (7) that

$$\begin{aligned}\frac{\mathrm{d}\ln V}{\mathrm{d}t} \leq & \frac{1}{V}\left(\frac{\bar{\beta}\Lambda}{a}(\mathcal{R}_0-1)I + \frac{\bar{\beta}\Lambda}{a}(\mathcal{R}_0-1)C\right) + \frac{\mathcal{R}_0\bar{\beta}\Lambda(I+C)}{aV}\left|e^x-1\right| \\ \leq & \min\left\{a+\mu, \frac{\bar{\beta}\Lambda}{a\mathcal{R}_0}\right\}(\mathcal{R}_0-1) + \left(\frac{\bar{\beta}\Lambda}{a} + (a+\mu)\mathcal{R}_0\right)\left|e^x-1\right|.\end{aligned} \tag{8}$$

Integrating inequality (8) over the interval $[0, t]$ and then dividing by t leads to the following:

$$\frac{1}{t}\ln\frac{V(t)}{V(0)} \leq \min\left\{a+\mu, \frac{\bar{\beta}\Lambda}{a\mathcal{R}_0}\right\}(\mathcal{R}_0-1) + \left(\frac{\bar{\beta}\Lambda}{a} + (a+\mu)\mathcal{R}_0\right)\left(\frac{1}{t}\int_0^t \left|e^x-1\right|^2\mathrm{d}s\right)^{\frac{1}{2}}. \tag{9}$$

In view of Lemma 2.1, one has the almost sure convergence

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t \left|e^x-1\right|^2\mathrm{d}s = e^{\frac{\sigma^2}{r}} - 2e^{\frac{\sigma^2}{4r}} + 1.$$

Taking the limit superior as $t \to \infty$ in Eq (9) and invoking the above result, we obtain the following:

$$\begin{aligned}\limsup_{t\to\infty}\frac{\ln V(t)}{t} \leq & \min\left\{a+\mu, \frac{\bar{\beta}\Lambda}{a\mathcal{R}_0}\right\}(\mathcal{R}_0-1) + \left(\frac{\bar{\beta}\Lambda}{a} + (a+\mu)\mathcal{R}_0\right)\left(e^{\frac{\sigma^2}{r}} - 2e^{\frac{\sigma^2}{4r}} + 1\right)^{\frac{1}{2}} \\ = & \min\left\{a+\mu, \frac{\bar{\beta}\Lambda}{a\mathcal{R}_0}\right\}(\mathcal{R}_0^e-1),\ a.s.\end{aligned}$$

Consequently, if $\mathcal{R}_0^e < 1$, then

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

which implies that the infection ultimately disappears from the population in the long run. □

Remark 3.1. *(i) When $\mathcal{R}_0^e < 1$, the condition $\mathcal{R}_0 < 1$ automatically holds due to the fact that $\mathcal{R}_0 < \mathcal{R}_0^e$. Consequently, the extinction of HCV in the stochastic setting implies that the deterministic model also predicts extinction.*

(ii) Theorem 3.1 indicates that the extinction of the disease is determined not only by the basic reproduction number $\mathcal{R}_0^e$ but also by the stochastic volatility σ and the mean-reversion speed r of the logarithmic O-U process. Specifically, a larger r or smaller σ reduces the term $\left(e^{\frac{\sigma^2}{r}} - 2e^{\frac{\sigma^2}{4r}} + 1\right)^{\frac{1}{2}}$, thereby lowering $\mathcal{R}_0^e$ and promoting extinction. This highlights the role of environmental stability in disease control: Reducing fluctuations in the transmission rates can help eliminate the infection even when the deterministic reproduction number exceeds unity.

While Section 3 identified conditions under which HCV goes extinct, the more challenging scenario arises when the infection persists. The following section addresses this by proving the existence of an ergodic stationary distribution when $\mathcal{R}_0^s > 1$, which implies long-term persistence of the disease even in a fluctuating environment. Here,

$$\mathcal{R}_0^s = \frac{\bar{\beta}\Lambda e^{\frac{\sigma^2}{8r}}}{a(a+\gamma)} + \frac{\bar{\beta}\Lambda p\gamma e^{\frac{\sigma^2}{12r}}}{a(a+\gamma)(a+\mu)}.$$

4. Stationary distribution

Theorem 4.1. *If $\mathcal{R}_0^s > 1$, then stochastic model* (3) *admits at least one ergodic stationary distribution $\upsilon(\cdot)$ on Γ.*

Proof. We begin by constructing the following auxiliary function:

$$V_1 = -a_1 \ln S - \ln I - a_2 \ln S - a_3 \ln C,$$

where the positive constants a_1, a_2, and a_3 will be specified in due course. Applying Itô's formula and utilizing the elementary inequality $e^x \leq \theta e^{2x} + \frac{1}{4\theta}$ (with $\theta \geq 0$ being a free parameter), the operator $\mathcal{L}V_1$ can be estimated as follows:

$$\begin{aligned}
\mathcal{L}V_1 =& a_1\left(-\frac{\Lambda}{S} + \bar{\beta}e^x(I+C) + a\right) - \bar{\beta}S\,e^x - \frac{\bar{\beta}SCe^x}{I} + a + \gamma \\
&+ a_2\left(-\frac{\Lambda}{S} + \bar{\beta}e^x(I+C) + a\right) + a_3\left(-\frac{p\gamma I}{C} + a + \mu\right) \\
\leq& a_1\left(-\frac{\Lambda}{S} + \frac{\bar{\beta}\Lambda\theta e^{2x}}{a} + \frac{\bar{\beta}(I+C)}{4\theta} + a\right) - \bar{\beta}S\,e^x - \frac{\bar{\beta}SCe^x}{I} + a + \gamma \\
&+ a_2\left(-\frac{\Lambda}{S} + \frac{\bar{\beta}\Lambda\theta e^{2x}}{a} + \frac{\bar{\beta}(I+C)}{4\theta} + a\right) + a_3\left(-\frac{p\gamma I}{C} + a + \mu\right).
\end{aligned}$$

By further applying the inequality

$$-\frac{\Lambda}{S} - \bar{\beta}S\,e^x \leq -2\sqrt{\bar{\beta}\Lambda e^x}$$

and a similar treatment for the terms that involve I and C, we obtain a more compact upper bound. This yields

$$\begin{aligned}
\mathcal{L}V_1 \leq& -2\sqrt{a_1\bar{\beta}\Lambda e^x} + \frac{a_1\bar{\beta}\Lambda\theta}{a}(e^{2x} - e^{\frac{\sigma^2}{r}}) + a_1\left(a + \frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{r}}\right) + \frac{a_1\bar{\beta}(I+C)}{4\theta} + a + \gamma \\
&- 3\sqrt[3]{a_2a_3\bar{\beta}\Lambda p\gamma e^x} + \frac{a_2\bar{\beta}\Lambda\theta}{a}\left(e^{2x} - e^{\frac{\sigma^2}{r}}\right) + a_2\left(a + \frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{r}}\right) + \frac{a_2\bar{\beta}(I+C)}{4\theta} + a_3(a+\mu) \\
=& -2\left(a_1\bar{\beta}\Lambda e^{\frac{\sigma^2}{8r}}\right)^{\frac{1}{2}} - 3\left(a_2a_3\bar{\beta}\Lambda p\gamma e^{\frac{\sigma^2}{12r}}\right)^{\frac{1}{3}} + a_1\left(a + \frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{r}}\right) + a_2\left(a + \frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{r}}\right) \\
&+ a_3(a+\mu) + a + \gamma + \frac{(a_1+a_2)\bar{\beta}(I+C)}{4\theta} + h_1(x) + h_2(x) + h_3(x),
\end{aligned} \tag{10}$$

where

$$\begin{aligned}
h_1(x) &= \frac{\bar{\beta}\Lambda\theta}{a}(a_1+a_2)\left(e^{2x} - e^{\frac{\sigma^2}{r}}\right), \\
h_2(x) &= 2\left(a_1\bar{\beta}\Lambda\right)^{\frac{1}{2}}\left(e^{\frac{\sigma^2}{16r}} - e^{\frac{x}{2}}\right), \\
h_3(x) &= 3\left(a_2a_3\bar{\beta}\Lambda p\gamma\right)^{\frac{1}{3}}\left(e^{\frac{\sigma^2}{36r}} - e^{\frac{x}{3}}\right).
\end{aligned}$$

Let

$$\begin{aligned} a_1\left(a+\frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{4r}}\right) &= \frac{\bar{\beta}\Lambda e^{\frac{\sigma^2}{8r}}}{a+\frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{4r}}}, \\ a_2\left(a+\frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{r}}\right) &= a_3(a+\mu) = \frac{\bar{\beta}\Lambda p\gamma e^{\frac{\sigma^2}{12r}}}{\left(a+\frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{r}}\right)(a+\mu)}. \end{aligned} \tag{11}$$

Substituting (11) into (10) yields the following:

$$\begin{aligned} \mathcal{L}V_1 \leq & -\frac{\bar{\beta}\Lambda e^{\frac{\sigma^2}{8r}}}{a+\frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{r}}} - \frac{\bar{\beta}\Lambda p\gamma e^{\frac{\sigma^2}{12r}}}{\left(a+\frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{r}}\right)(a+\mu)} + a + \gamma \\ & + \frac{(a_1+a_2)\bar{\beta}(I+C)}{4\theta} + h_1(x) + h_2(x) + h_3(x) \\ = & -(a+\gamma)(\mathcal{R}_0^s(\theta)-1) + \frac{(a_1+a_2)\bar{\beta}(I+C)}{4\theta} + \sum_{i=1}^{3} h_i(x), \end{aligned} \tag{12}$$

where

$$\mathcal{R}_0^s(\theta) = \frac{\bar{\beta}\Lambda e^{\frac{\sigma^2}{8r}}}{\left(a+\frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{r}}\right)(a+\gamma)} + \frac{\bar{\beta}\Lambda p\gamma e^{\frac{\sigma^2}{12r}}}{\left(a+\frac{\bar{\beta}\Lambda\theta}{a}e^{\frac{\sigma^2}{r}}\right)(a+\gamma)(a+\mu)}.$$

Note that when $\theta \to 0$, $\mathcal{R}_0^s(\theta) \to \mathcal{R}_0^s$.

Define the following:

$$V_2 = V_1 + a_4 C,$$

where $a_4 = \frac{(a_1+a_2)\bar{\beta}}{4\theta(a+\mu)}$. This together with Eq (12) leads to the following:

$$\mathcal{L}V_2 \leq -(a+\gamma)(\mathcal{R}_0^s(\theta)-1) + a_5 I + \sum_{i=1}^{3} h_i(x); \tag{13}$$

here, $a_5 = \frac{(a_1+a_2)\bar{\beta}}{4\theta} + a_4 p\gamma$.

Define the following:

$$V_3 = e^x - 1 - x - \ln S - \ln C - \ln\left(\frac{\Lambda}{a} - S - I - C\right),$$

which implies

$$\begin{aligned} \mathcal{L}V_3 = & -rx(e^x-1) + \frac{\sigma^2}{2}e^x - \frac{\Lambda}{S} + \bar{\beta}e^x(I+C) + a - \frac{p\gamma I}{C} \\ & + a + \mu + \frac{\Lambda - a(S+I+C) - (1-p)\gamma I - \mu C}{\frac{\Lambda}{a} - S - I - C} \\ \leq & -rx(e^x-1) + \left(\frac{\bar{\beta}\Lambda}{a} + \frac{\sigma^2}{2}\right)e^x - \frac{\Lambda}{S} - \frac{p\gamma I}{C} + 3a + \mu - \frac{(1-p)\gamma I}{\frac{\Lambda}{a} - S - I - C}. \end{aligned} \tag{14}$$

Now, we define the following:

$$\hat{V} = MV_2 + V_3,$$

where M is a sufficiently large constant that satisfies

$$-M(a+\gamma)(\mathcal{R}_0^s(\theta)-1) + \sup_{x\in\mathbb{R}}\{h_4(x)\} \le -1,$$

where

$$h_4(x) = -rx(e^x-1) + \left(\frac{\bar{\beta}\Lambda}{a} + \frac{\sigma^2}{2}\right)e^x + 3a + \mu. \tag{15}$$

Given that $\hat{V} \to \infty$ when approaching the boundary of Γ, a non-negative C^2 function can be constructed as follows:

$$V = \hat{V} - \hat{V}_{min},$$

where the constant $\hat{V}_{\min}$ denotes the minimum of $\hat{V}$. Combining Eqs (13) and (14) yields the following:

$$\mathcal{L}V \le F(X) + M\sum_{i=1}^{3} h_i(x),$$

where

$$F(X) = -M(a+\gamma)(\mathcal{R}_0^s(\theta)-1) + a_5MI - \frac{\Lambda}{S} - \frac{p\gamma I}{C} - \frac{(1-p)\gamma I}{\frac{\Lambda}{a} - S - I - C} + h_4(x). \tag{16}$$

As S or $C \to 0$, Eq (16) gives $F(X) \to -\infty$. When $I \to 0$, $F(X) \to -M(a+\gamma)(\mathcal{R}_0^s(\theta)-1) + \sup_{x\in\mathbb{R}}\{h_4(x)\}$. Additionally, $F(X) \to -\infty$ as $x \to \pm\infty$ or $S + I + C \to \frac{\Lambda}{a}$. Consequently, one can identify a compact subset $D \subset \Gamma$ such that $F(X) \le -1$ for any $X \in D^c$.

For any initial value $X_0 \in \Gamma$, applying Itô's integral together with mathematical expectation to the function V yields the following:

$$\begin{aligned} 0 \le& \frac{\mathbb{E}V(X)}{t} = \frac{\mathbb{E}V(X_0)}{t} + \frac{1}{t}\int_0^t \mathbb{E}(\mathcal{L}V(X(\tau)))\mathrm{d}\tau \\ \le& \frac{\mathbb{E}V(X_0)}{t} + \frac{1}{t}\int_0^t \mathbb{E}(F(X(\tau)))\mathrm{d}\tau + M\mathbb{E}\left(\frac{1}{t}\int_0^t \sum_{i=1}^{3} h_i(x(\tau))\mathrm{d}\tau\right). \end{aligned} \tag{17}$$

By virtue of Lemma 2.1, we obtain the following:

$$\lim_{t\to\infty}\mathbb{E}\left(\frac{1}{t}\int_0^t h_i(x(\tau))\mathrm{d}\tau\right) = 0, \quad i = 1,2,3.$$

Moreover, it follows from Eq (16) that there exists a constant Π such that $F(X) \le \Pi$ holds uniformly for all $X \in \Gamma$. Combining the above estimates with (17) leads to the following:

$$\begin{aligned} 0 \le& \liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{E}(F(X(\tau)))\mathrm{d}\tau \\ =& \liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{E}(F(X(\tau)))\mathbf{I}_{X\in D}\mathrm{d}\tau + \liminf_{t\to\infty}\frac{1}{t}\int_0^t \mathbb{E}(F(X(\tau)))\mathbf{I}_{X\in D^c}\mathrm{d}\tau \end{aligned}$$

$$\leq \Pi \liminf_{t\to\infty} \frac{1}{t}\int_0^t \mathbb{P}\{X(\tau)\in D\}\mathrm{d}\tau - \liminf_{t\to\infty} \frac{1}{t}\int_0^t \mathbb{P}\{X(\tau)\in D^c\}\mathrm{d}\tau$$
$$\leq (\Pi+1)\liminf_{t\to\infty} \frac{1}{t}\int_0^t \mathbb{P}\{X(\tau)\in D\}\mathrm{d}\tau - 1.$$

From the above inequality, we immediately deduce the following:

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t \mathbb{P}(\tau, X(\tau), D)\mathrm{d}\tau \geq \frac{1}{\Pi+1} > 0, \ a.s.$$

Therefore, by virtue of Lemma 2.2, model (3) admits at least one ergodic stationary distribution $\upsilon(\cdot)$ on the invariant set Γ. □

Remark 4.1. *The condition $\mathcal{R}_0^s > 1$ ensures the existence of an ergodic stationary distribution, which implies that HCV persists in the population in the long run. Compared to the deterministic threshold $\mathcal{R}_0$, the stochastic threshold $\mathcal{R}_0^s$ incorporates the effect of environmental noise through the factor $e^{\frac{\sigma_2}{8r}}$ and $e^{\frac{\sigma_2}{12r}}$. This shows that even if $\mathcal{R}_0 < 1$, HCV may still persist if the noise intensity σ is large or the mean-reversion speed r is small. Conversely, damping environmental fluctuations can help stabilize the system and potentially push it toward extinction.*

Having established the existence and ergodicity of the stationary distribution for mdel (3) in Section 4, we now further derive its explicit density function.

5. Probability density

Let $E_+^* = (0, S^*, I^*, C^*)$ be the quasi-positive equilibrium of stochastic model (3), defined by the following system:

$$\begin{cases} \Lambda - \bar{\beta}S^*(I^*+C^*) - aS^* = 0, \\ \bar{\beta}S^*(I^*+C^*) - (a+\gamma)I^* = 0, \\ p\gamma I^* - (a+\mu)C^* = 0, \end{cases} \tag{18}$$

where

$$S^* = \frac{\Lambda}{a\mathcal{R}_0}, \ \ I^* = \frac{a(a+\mu)}{\beta(a+\mu+p\gamma)}(\mathcal{R}_0-1), \ \ C^* = \frac{ap\gamma}{\beta(a+\mu+p\gamma)}(\mathcal{R}_0-1).$$

Then E_+^* exists if and only if $\mathcal{R}_0 > 1$. Linearizing model (3) around E_+^* yields the following:

$$\mathrm{d}Y(t) = AY(t)\mathrm{d}t + G\mathrm{d}B(t), \tag{19}$$

where $Y = (x, S-S^*, I-I^*, C-C^*)^T$, $G = \mathrm{diag}(\sigma, 0, 0, 0)$, and

$$A = \begin{pmatrix} -r & 0 & 0 & 0 \\ -a_{21} & -a_{22} & -a_{23} & -a_{23} \\ a_{21} & a_{32} & -a_{33} & a_{23} \\ 0 & 0 & a_{43} & -a_{44} \end{pmatrix},$$

with the auxiliary coefficients defined as follows:

$$a_{21} = (a+\gamma)I^*, \ \ a_{22} = a + \frac{(a+\gamma)I^*}{S^*}, \ \ a_{23} = \bar{\beta}S^*,$$

$$a_{32} = \frac{(a+\gamma)I^*}{S^*},\ \ a_{33} = a+\gamma-\bar{\beta}S^*,\ \ a_{43} = p\gamma,\ \ a_{44} = a+\mu.$$

Theorem 5.1. *If the threshold parameter satisfies $\mathcal{R}_0 > 1$, then for any initial value $X_0 \in \Gamma$, the stationary distribution of model* (3) *near the equilibrium E_+^* is approximately Gaussian. Its probability density function is given by the following:*

$$\Psi(X) = (2\pi)^{-2}|\Sigma|^{-\frac{1}{2}} \exp\left\{-\frac{1}{2}(X-E_+^*)\Sigma^{-1}(X-E_+^*)^T\right\},$$

where Σ is a positive definite matrix defined as follows:

$$\Sigma = (p_1\sigma)^2(J_2J_1)^{-1}\Theta_0[(J_2J_1)^{-1}]^T,$$

with p_1, J_1, J_2, and Θ_0 being specified in the proof.

Proof. In view of [33, 34], system (19) admits a unique explicit solution given by the following:

$$Y(t) = e^{At}Y(0) + \int_0^t e^{A(t-s)}G\mathrm{d}B(s).$$

Since the martingale $\int_0^t e^{A(t-s)}G\mathrm{d}B(s)$ follows a Gaussian distribution $N_4(0,\Sigma_0(t))$ at time t, where

$$\Sigma_0(t) = \int_0^t e^{A(t-s)}G^2e^{A^T(t-s)}\mathrm{d}s,$$

it follows that $Y(t)$ is Gaussian distributed as $N_4(e^{At}Y(0),\Sigma_0(t))$. Under the condition $\mathcal{R}_0 > 1$, the positive equilibrium of the deterministic counterpart for model (3) is globally asymptotically stable, which implies that the matrix A is Hurwitz. Then, using the stability theory of the linear equation [35], we obtain the following:

$$\lim_{t\to+\infty} e^{At}Y(0) = 0,\ \ \Sigma = \lim_{t\to+\infty}\Sigma_0(t) = \int_0^{+\infty} e^{At}G^2e^{A^Tt}\mathrm{d}t.$$

The matrix Σ is clearly positive semi-definite as G^2 is positive semi-definite.

To derive the equation satisfied by Σ, we compute the following:

$$\int_0^{+\infty}\frac{\mathrm{d}}{\mathrm{d}t}\left(e^{At}G^2e^{A^Tt}\mathrm{d}t\right) = -G^2.$$

On the other hand, direct differentiation gives

$$\frac{\mathrm{d}}{\mathrm{d}t}\left(e^{At}G^2e^{A^Tt}\right) = Ae^{At}G^2e^{A^Tt} + e^{At}G^2e^{A^Tt}A^T,$$

and integrating from 0 to $+\infty$ yields

$$\int_0^{+\infty}\frac{\mathrm{d}}{\mathrm{d}t}\left(e^{At}G^2e^{A^Tt}\mathrm{d}t\right) = A\Sigma + \Sigma A^T.$$

Hence, Σ satisfies the algebraic equation

$$G^2 + A\Sigma + \Sigma A^T = 0. \tag{20}$$

Now, we proceed to derive the explicit expression for Σ through a series of matrix transformations.

First, to facilitate the elimination of certain elements, we introduce a transformation matrix J_1 defined as

$$J_1 = \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 1 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix}.$$

By applying this transformation, we obtain the following:

$$A_1 = J_1 A J_1^{-1} = \begin{pmatrix} -r & 0 & 0 & 0 \\ -a_{21} & a_{23} - a_{22} & -a_{23} & -a_{23} \\ 0 & \gamma & -(a+\gamma) & 0 \\ 0 & -a_{43} & a_{43} & -a_{44} \end{pmatrix}.$$

Next, we introduce a second transformation matrix

$$J_2 = \begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & p & 1 \end{pmatrix},$$

which yields

$$A_2 = J_2 A_1 J_2^{-1} = \begin{pmatrix} -r & 0 & 0 & 0 \\ -a_{21} & a_{23} - a_{22} & a_{23}(p-1) & -a_{23} \\ 0 & \gamma & -(a+\gamma) & 0 \\ 0 & 0 & p\mu & -a_{44} \end{pmatrix}.$$

Finally, we construct a standardized transformation matrix J_3 of the form

$$J_3 = \begin{pmatrix} p_1 & p_2 & p_3 & p_4 \\ 0 & p\gamma\mu & -(a+\gamma+a_{44})p\mu & a_{44}^2 \\ 0 & 0 & p\mu & -a_{44} \\ 0 & 0 & 0 & 1 \end{pmatrix},$$

where the coefficients are given by

$$\begin{aligned} p_1 &= -a_{21} p\gamma\mu, \\ p_2 &= (a_{23} - a_{22} - a_{44} - a - \gamma) p\gamma\mu, \\ p_3 &= (a_{23}(p-1)\gamma + (a+\gamma)a_{44} + (a+\gamma)^2 + a_{44}^2) p\mu, \\ p_4 &= a_{23}\gamma(a+\gamma) - a_{44}^3. \end{aligned}$$

Then, we obtain the following:

$$A_0 = J_3 A_2 J_3^{-1} = \begin{pmatrix} -\varrho_1 & -\varrho_2 & -\varrho_3 & -\varrho_4 \\ 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \end{pmatrix},$$

with the coefficients ϱ_i $(i = 1, 2, 3, 4)$ defined as

$$\begin{aligned}
\varrho_1 &= r + a_{22} + a_{33} + a_{44}, \\
\varrho_2 &= a_{22}(a_{33} + a_{44}) + a_{33}a_{44} - a_{23}a_{43} + a_{23}a_{32} + r(a_{22} + a_{33} + a_{44}), \\
\varrho_3 &= a_{22}(a_{33}a_{44} - a_{23}a_{43}) + a_{23}a_{32}(a_{43} + a_{44}) + r(a_{22}(a_{33} + a_{44}) + a_{33}a_{44} - a_{23}a_{43} + a_{23}a_{32}), \\
\varrho_4 &= r(a_{22}(a_{33}a_{44} - a_{23}a_{43}) + a_{23}a_{32}(a_{43} + a_{44})).
\end{aligned}$$

Under the combined transformation $J = J_3 J_2 J_1$, Eq (20) can be equivalently rewritten as

$$JG^2J^T + A_0 J\Sigma J^T + J\Sigma J^T A_0^T = 0,$$

i.e.,

$$G_0^2 + A_0\Theta_0 + \Theta_0 A_0^T = 0, \tag{21}$$

where $G_0 = (1, 0, 0, 0)$, and $\Theta_0 = \frac{J\Sigma J^T}{(p_1\sigma)^2}$. Since A is Hurwitz, it can be shown that $\varrho_i > 0$ for $i = 1, 2, 3, 4$ and that the stability condition $\varrho_1(\varrho_2\varrho_3 - \varrho_1\varrho_4) > \varrho_3^2$ holds. According to Lemma 2.3, the matrix Θ_0 defined by Eq (21) is a positive definite matrix. Therefore,

$$\Sigma = (p_1\sigma)^2 (J_3J_2J_1)^{-1}\Theta_0[(J_3J_2J_1)^{-1}]^T,$$

which is also positive definite. This completes the proof. □

Remark 5.1. *The explicit form of the probability density function derived in Theorem 5.1 provides a characterization of the stationary fluctuations around the quasi-endemic equilibrium. The positive definiteness of* Σ *guarantees that the distribution is non-degenerate and that all compartments exhibit variability around their equilibrium values. We note that the Gaussian density derived in Theorem 5.1 is an approximation for the original nonlinear model, exact only for the linearized dynamics.*

6. Numerical simulations

In this section, numerical simulations are performed to validate the theoretical results and to explore the impact of stochastic fluctuations on HCV transmission dynamics. The parameters are chosen to qualitatively illustrate the theoretical results, not to fit specific epidemiological data. Unless otherwise specified, the baseline parameters are set as $\Lambda = 0.3$, $a = 0.03$, $\beta = \bar{\beta} = 0.01$, $\gamma = 0.4$, $p = 0.3$, and $\mu = 0.01$. Calibration with real-world HCV data (e.g., World Health Organization [1]) is deferred to future work. The initial condition is taken as $(S(0), I(0), C(0)) = (9, 0.02, 0.005)$ for extinction scenarios and $(6, 0.5, 0.06)$ for persistence scenarios.

6.1. *Extinction and persistence of HCV*

To validate the extinction condition in Theorem 3.1, we set $r = 0.3$ and $\sigma = 0.01$, yielding $\mathcal{R}_0$=0.93<1 and $\mathcal{R}_0^e$=0.97<1. As shown in Figure 1, both the deterministic and stochastic models exhibit a rapid decline in the acutely infected I and chronically infected C populations over time, approaching zero. The phase portraits in Figure 2 further confirm that the trajectories converge to the infection-free equilibrium, consistent with the theoretical prediction of disease extinction.

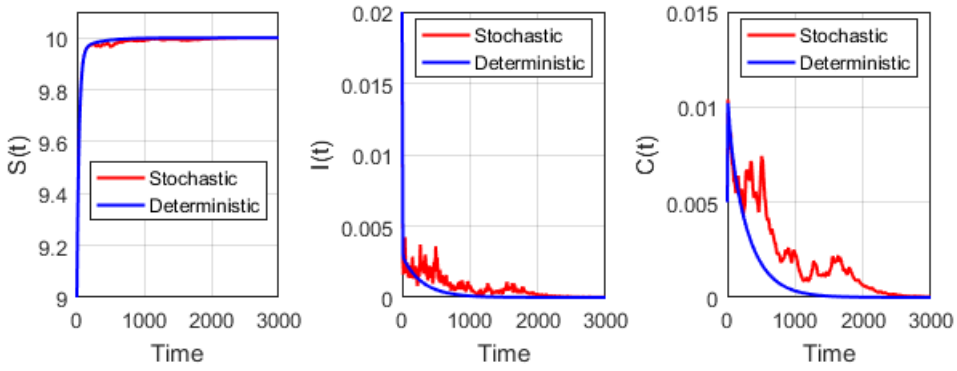

Figure 1. Time series plots of HCV extinction under deterministic and stochastic environments.

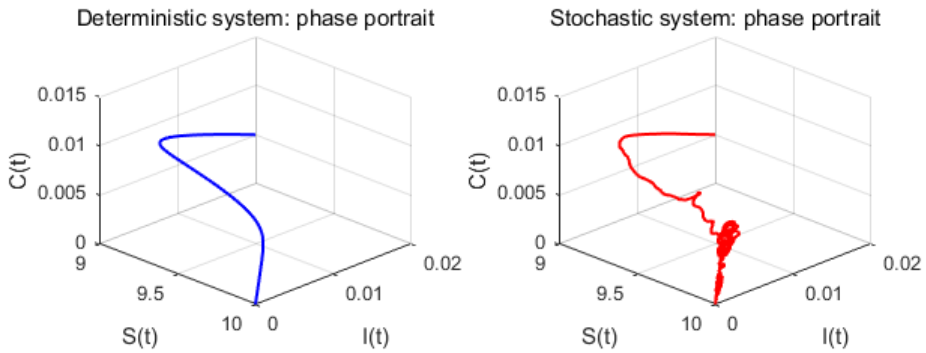

Figure 2. Phase portraits of HCV extinction under deterministic and stochastic environments.

In contrast, for the persistence case, we choose $r = 0.25$ and $\sigma = 0.95$, thus giving $\mathcal{R}_0^s = 1.3 > 1$ while $\mathcal{R}_0 < 1$. Under this parameter regime, Theorem 4.1 guarantees the existence of an ergodic stationary distribution. The time series in Figure 3 show sustained oscillations in the infected populations without extinction, and the lower half-plane of the figure presents the corresponding probability density, thus illustrating the variability around the quasi-endemic state. The phase portraits in Figure 4 further demonstrate that the system does not settle to a fixed point but instead fluctuates within a bounded region, thus indicating long-term persistence.

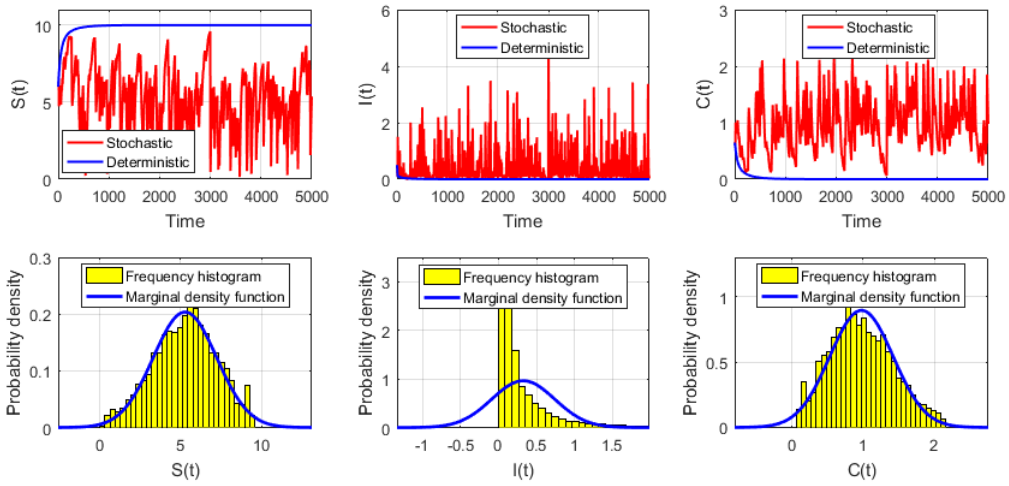

Figure 3. Time series plots of HCV persistence (upper half-plane) and the corresponding probability density (lower half-plane).

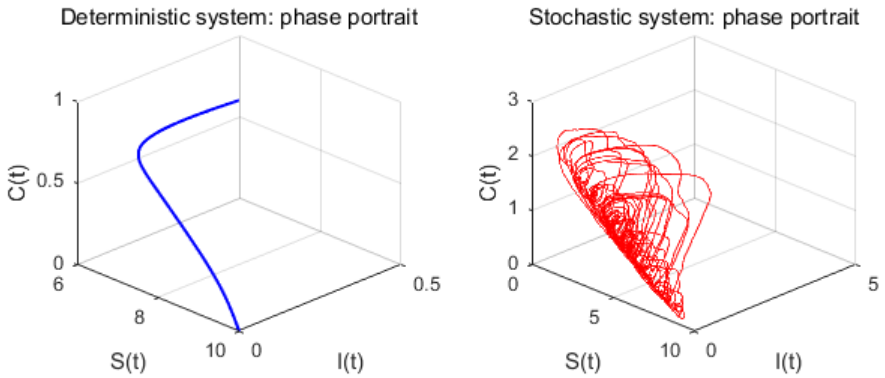

Figure 4. Phase portraits of HCV persistence under deterministic and stochastic environments.

These simulations highlight a key epidemiological insight: environmental noise can qualitatively alter the disease outcomes. Even when the deterministic model predicts extinction ($\mathcal{R}_0 < 1$), sufficiently strong stochastic fluctuations can sustain HCV transmission.

6.2. Combined effect of noise parameters

We further explore the joint influence of the mean-reversion speed r and noise intensity σ on the transmission dynamics through contour plots of $\mathcal{R}_0^e$ and $\mathcal{R}_0^s$, as shown in Figure 5. The left panel of Figure 5 shows the region $R_0^e < 1$ (below the solid red curve), where stochastic extinction is guaranteed. The right panel illustrates the region $R_0^s > 1$ (above the red curve), where an ergodic stationary distribution exists, thus implying long-term disease persistence. Figure 5 demonstrates that both thresholds increase with σ but decrease with r. This indicates that a faster mean reversion (larger r) suppresses the stochastic enhancement of persistence, thereby reducing the likelihood of disease survival in a fluctuating environment. Conversely, a larger environmental variability (σ) poses a

greater challenge to disease elimination than to disease establishment. A notable feature is the existence of a parameter region where $R_0 < 1$ yet $R_0^s > 1$, thereby confirming that stochasticity can enable persistence under conditions that would otherwise lead to extinction. This result underscores the importance of accounting for environmental variability in disease control strategies, as fluctuations in the transmission rates can significantly shift the threshold for disease elimination.

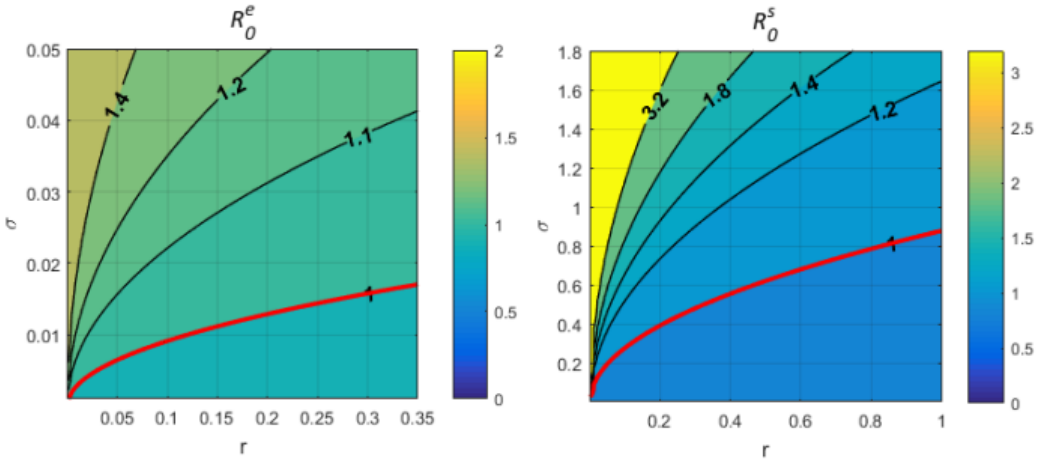

Figure 5. Contour diagrams which illustrate the dependence of (left) $\mathcal{R}_0^e$ and (right) $\mathcal{R}_0^s$ on the parameters r and σ. The solid red curves denote the critical thresholds $\mathcal{R}_0^e = 1$ and $\mathcal{R}_0^s = 1$, respectively.

7. Conclusions

In this study, we developed a stochastic SICR model for HCV transmission by incorporating a logarithmic O-U process to account for environmental fluctuations in the transmission rate. This approach overcomes the biological limitations of traditional white noise perturbation methods by preserving the parameter positivity while capturing the mean-reverting stochasticity. We systematically investigated the dynamics of the model, thereby focusing on the conditions for disease extinction and persistence.

A sufficient condition for an almost sure extinction of HCV is established in terms of a stochastic threshold $\mathcal{R}_0^e$. When $\mathcal{R}_0^e < 1$, the infected populations exponentially converge to zero with a probability of one, thus ensuring disease eradication. In contrast, when $\mathcal{R}_0^s > 1$, the model admits at least one ergodic stationary distribution, thus indicating a long-term persistence of the infection. Notably, these two thresholds incorporate the effects of noise intensity σ and mean-reversion speed r, thereby revealing that environmental fluctuations can qualitatively alter the disease outcomes. One of the key contributions of this work is the derivation of an explicit probability density function for the stationary distribution near the quasi-endemic equilibrium when $\mathcal{R}_0 > 1$. This expression provides a characterization of stationary fluctuations and enables further statistical inference, such as parameter estimation and uncertainty quantification. Numerical simulations corroborate the theoretical findings and highlight the nontrivial role of stochasticity.

The deterministic basic reproduction number $\mathcal{R}_0$ determines the asymptotic behavior of the deterministic model: disease extinction when $\mathcal{R}_0 < 1$ and endemic persistence when $\mathcal{R}_0 > 1$. In our

stochastic model, the thresholds $\mathcal{R}_0^e$ and $\mathcal{R}_0^s$ are modifications of $\mathcal{R}_0$ that incorporate the noise intensity σ and mean-reversion speed r. Obviously, $\mathcal{R}_0^e > \mathcal{R}_0$ and $\mathcal{R}_0^s > \mathcal{R}_0$ always. Hence, both stochastic thresholds are larger than the deterministic threshold. This implies that environmental noise makes extinction more difficult to achieve and can sustain HCV transmission under conditions where the deterministic model predicts extinction (i.e., when $\mathcal{R}_0 < 1$ but $\mathcal{R}_0^s > 1$). From a public health perspective, our results suggest two strategies for HCV control: (i) reduce transmission rate variability through consistent infection control practices; and (ii) increase the mean-reversion speed r via rapid response systems (e.g., surveillance, contact tracing, isolation).

We acknowledge several limitations. First, the transmission rate exogenously fluctuates without behavioral feedback [36]. Second, the ODE model omits spatial heterogeneity and age structure [37]. Third, the thresholds R_0^e and R_0^s are only sufficient conditions; the sharp thresholds remain unknown. Fourth, calibration with real data is deferred. Future work will address these limitations by incorporating behavioral responses, age structure, spatial heterogeneity, and structure-preserving numerical methods following Raza et al. [38].

Author contributions

S. He: writing-original draft, software, formal analysis; Y. Ma: conceptualization, validation, writing-review and editing, funding acquisition. All authors have read and approved the final version of the manuscript for publication

Use of Generative-AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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