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Stability of Stochastic Model for Hepatitis C Transmission with an Isolation Stage

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Abstract. In this paper we construct and investigate stability features of two stochastic hepatitis C models with an isolation stage which are obtained by an introduction of stochastic perturbations into the deterministic model for hepatitis C with an isolation stage. One of the stochastic models has only disease–free equilibrium and the other endemic equilibrium state. Aforementioned equilibriums belong to the equilibriums of corresponding deterministic system. For both of models, first of all, we prove the existence and uniqueness of global positive stochastic solution. Thereafter, by using suitable Lyapunov functions, we investigate stability properties of both models. We close the paper with numerical simulation with reliable data of hepatitis C transmission to illustrate our theoretical results.

1. Introduction and Motivation

Hepatitis C (HCV) is one of several viruses that is caused by viral hepatitis. Hepatitis means inflammation of the liver. The liver is a vital organ that processes nutrients, filters the blood, and fights infections. Inflammation of the liver affects its function. Hepatitis is often caused by a virus, and to the smaller extent, it can be also caused by heavy alcohol use, toxins, some medications, and certain medical conditions.

Hepatitis C spreads only through exposure to an infected person's blood. The primary route of transmission in the developed world is intravenous drug use, while in the developing world the main methods are blood transfusions and unsafe medical procedures. The cause of transmission remains unknown in 20% of cases. Intravenous drug use is a major risk factor for hepatitis C in many parts of the world. As it is stated in [12], 25 of 77 countries reviewed, were found to have prevalence of hepatitis C in the intravenous drug user population of between 60% and 80%. Twelve countries had rates greater than 80%. It is believed that ten million intravenous drug users are infected with hepatitis C, and the highest absolute totals have China (1.6 million), the United States (1.5 million) and Russia (1.3 million). Blood transfusion or organ transplants without HCV screening carry significant risks of infection. This is why some countries instituted universal screening which decreased the risk of infection. However, the low risk remains as there is a period of about 11-70 days between the potential blood donor's acquiring hepatitis C and the blood's testing positive depending on the method. On the other side, in some countries, especially in developing ones, screening

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for hepatitis C is not done due to the cost. Those who have experienced a needle stick injury from someone who was HCV positive have about a 1.8% chance of subsequently contracting the disease themselves (see [19]). Hospital equipment has also been documented as a method of transmission of hepatitis C, including reuse of needles and syringes, multiple-use of medication vials, infusion bags and improperly sterilized surgical equipment, among others, as pointed out in [1].

Up to 85% of individuals who are initially (acutely) infected with hepatitis C will fail to eliminate the virus and will become chronically infected. It is estimated that around 170 million people worldwide, and around 100 000 in Serbia, have chronic hepatitis C. However, this infection was only demonstrated in 25% cases because the symptoms of the disease were either absent or uncharacteristic, and they were detected most often by chance during routine blood testing.

The theoretical study of the spread od hepatitis C has a long history. In their research, authors divide the population of infected individuals in the classes. In [3] infected population is divided into acutely infected individuals and chronic carriers of the disease, while in [5, 7] besides two distinct infection stages (acute and chronic), there is an isolation compartment. For models defined there, authors carry out stability analysis of the equilibrium states.

All papers mentioned above deal with deterministic models. However, variability and uncertainty which may manifest through unpredictability of person–to–person contact is better described by stochastic models, as it is highlighted in [5]. Hence, authors formulate stochastic epidemic model of hepatitis C using a continuous time Markov chain and compare dynamics od deterministic and stochastic models. Beside the approach via Markov chains, from biological and epidemiological perspective, random effects can be expressed in Itô or Stratonovich stochastic integrals. The Stratonovich integral lacks the important property of the Itô integral, which does not "look into the future". In many real-world applications, one only has information about past events, and hence the Itô interpretation is more natural. Thus, on the basis of deterministic model presented in [5], we construct five-state stochastic model by using system od stochastic differential equations of Itô type.

Hence, let us briefly describe the deterministic model considered by Imran et al. [5]. They formulated a SIR epidemiological model in such a way to divide infected persons at time t into three compartments: acute $A(t)$, chronic $C(t)$ and isolated $Q(t)$, and thus, they obtained five–stages model where the total population size at time t, $N(t)$, in addition to the infected individuals, also consists of susceptible $S(t)$ and recovered $R(t)$ ones. For the model, the following assumptions are made: all new borne individuals are susceptible and susceptible population has a constant recruitment rate Π, all the infected individuals develop the acute stage of hepatitis C first at rate λ, individuals from all three infected stages are capable of transmitting the disease, infected individuals from acute stage either progress to chronic stage of the disease at rate ξ or recover naturally at rate κ, individuals who are chronically infected with hepatitis C may recover from the disease at rate ψ or move to isolated stage at rate α, all isolated individuals can recover at rate γ, and individuals from the recovered class become susceptible over time at rate ω. Also, all individuals may die at natural death rate μ, or from the disease, where δ_a, δ_c and δ_q are disease–induced rates for acute, chronic and isolated individuals, respectively.

Thus, the dynamics of hepatitis C is given by the system of ordinary differential equations:

$$\begin{aligned}
\frac{dS(t)}{dt} &= \Pi+\omega R(t)-\lambda S(t)-\mu S(t),\\
\frac{dA(t)}{dt} &= \lambda S(t)-(\xi+\kappa+\mu+\delta_a)\,A(t),\\
\frac{dC(t)}{dt} &= \xi A(t)-(\alpha+\psi+\mu+\delta_c)\,C(t),\\
\frac{dQ(t)}{dt} &= \alpha C(t)-\left(\gamma+\mu+\delta_q\right)Q(t),\\
\frac{dR(t)}{dt} &= \kappa A(t)+\psi C(t)+\gamma Q(t)-(\omega+\mu)\,R(t),
\end{aligned} \tag{1}$$

with initial conditions $S(0) = S_0$, $A(0) = A_0$, $C(0) = C_0$, $Q(0) = Q_0$, $R(0) = R_0$, and $\lambda = \lambda(t) = \beta\left[\frac{\eta A(t)+C(t)+\zeta Q(t)}{N(t)}\right]$,

$t \geq 0$. From the epidemiological point of view, $\lambda(t)$ is a function that models transition rate from the class of susceptible individuals to the class of infectious individuals and is called force of infection. For large classes of infectious diseases it is more realistic to represents the force of infection as a fraction of infectious class with respect to the number of total population than as a absolute number of infectious subjects. Since we have three infectious classes that do not transmit the disease at the same intensity, $\lambda(t)$ is a function of $A(t)$, $C(t)$, $Q(t)$ and $N(t)$. In definition of $\lambda(t)$ there are also positive constants, related to the classes of infectious populations, which are described as follows:

β - effective contact rate,
η - modification parameter for infectiousness of acute individuals,
ζ - modification parameter for infectiousness of isolated individuals.

Since the solutions of system (1) must be non-negative, it is required that they are in the cone

$$\Gamma = \left\{(S, A, C, Q, R) \in \mathbb{R}^5 : 0 \leq S+A+C+Q+R \leq \frac{\Pi}{\mu}\right\}. \tag{2}$$

An important quantity for all epidemiological models is the basic reproduction number, or the contact number $\mathcal{R}_0$. It represents the average number of secondary infections that single infectious individual in susceptible population may generate. Susceptible individuals acquire the infection through contact with acute, chronic or isolated individuals. Acutely infected individual produces $\frac{\beta\eta}{k_1}$ new infections since the infection rate is $\beta\eta$, and an average duration of acute stage is $\frac{1}{k_1}$, where $k_1 = \xi + \kappa + \mu + \delta_a$. Similarly, chronic individual may infect $\frac{\beta\xi}{k_1k_2}$ susceptible individuals bearing in mind that infection rate is β, an average duration of chronic phase is $\frac{1}{k_2}$, and probability that acute individual survives and progress to chronic stage is $\frac{\xi}{k_1}$, where $k_2 = \alpha + \psi + \mu + \delta_c$. Finally, infection rate of isolated individuals is $\beta\zeta$, an average duration of infection in isolated stage is $\frac{1}{k_3}$, and probability that acute individual survives and progress to the isolation stage via chronic stage is $\frac{\xi\alpha}{k_1k_2}$, where $k_3 = \gamma + \mu + \delta_q$. Thus,

$$\mathcal{R}_0 = \frac{\beta\left(\eta k_2k_3 + \xi k_3 + \zeta\alpha\xi\right)}{k_1k_2k_3}.$$

Basic reproduction number controls the number of equilibriums of system (1). If $\mathcal{R}_0 \leq 1$ the system has just the disease-free equilibrium state $E^0 = (S^0, A^0, C^0, Q^0, R^0) = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0\right)$. It is apparent that the disease-free equilibrium E^0 is obtained for $A(t) = C(t) = Q(t) = 0$ which implies that $\lambda(t) = 0$. Otherwise, if $\mathcal{R}_0 > 1$, the disease-free equilibrium is still present, but there is also a unique positive endemic equilibrium $E^* = (S^*, A^*, C^*, Q^*, R^*)$, where $S^* = \frac{1}{\lambda^*}\frac{k_1k_2}{\xi}C^*$, $A^* = \frac{k_2}{\xi}C^*$, $Q^* = \frac{\alpha}{k_3}C^*$, $R^* = \frac{1}{k_4}\left(\frac{k_1k_2}{\xi} + \psi\right)C^*$, and $\lambda^* = \beta\left[\frac{\eta A^* + C^* + \zeta Q^*}{N^*}\right]$.

On the basis of model (1) we construct two stochastic models for which we investigate stability properties of the equilibrium states by using the appropriate Lyapunov functions. The method we use relies on the results obtained by Kolmanovskii and Shaikhet [9] and Shaikhet (see [14–17], for instance).

The paper is organized in the following way: In Section 2 we construct a stochastic model that has only the disease free equilibrium state. For such model, we verify that there exists a unique nonnegative global solution and then, we present stability analysis of the disease free equilibrium state by using appropriate Lyapunov function. Section 3 is devoted to another stochastic model, i.e. to model which is constructed in such a way to have endemic equilibrium state succeeded from the deterministic model (1). Again, we prove existence and uniqueness of positive solution and then, by the choice of the suitable Lyapunov function, we determine the conditions for model parameters under which the endemic equilibrium state is stable in probability. In Section 4 we present the numerical simulation of results obtained through the paper in order to show that our theoretical results are compatible with reliable data for hepatitis C transmission. We also give interpretation of the obtained theoretical results and give some possible directions for the future research.

2. Model (3)

In previous section it is already mentioned that the stochastic models are more realistic in describing transmission of hepatitis C then their deterministic analogues. Thus, in this section we involve stochastic perturbation into deterministic system (1). It is assumed that stochastic perturbations are of a white noise type and they are directly proportional to the distances of current states $S(t)$, $A(t)$, $C(t)$, $Q(t)$, $R(t)$ from S^0, A^0, C^0, Q^0, R^0, respectively. This is standard approach which enables that equilibrium state E^0 of stochastic system (3) coincides with the equilibrium state E^0 od deterministic system (1). Stochastic perturbations of this form were proposed for the first time by Beretta et al. [2] for stochastic SIR epidemic model, and latter the idea was applied by different authors for different mathematical models (see [6, 10, 13, 18], among the others, and references cited therein). Thus, the stochastic model we construct has the following form

$$\begin{aligned}
dS(t) &= [\Pi+\omega R(t)-(\lambda+\mu)S(t)]\,dt+\sigma_1\left(S(t)-\frac{\Pi}{\mu}\right)dw_1(t)\\
dA(t) &= [\lambda S(t)-k_1A(t)]\,dt+\sigma_2A(t)dw_2(t)\\
dC(t) &= [\xi A(t)-k_2C(t)]\,dt+\sigma_3C(t)dw_3(t)\\
dQ(t) &= [\alpha C(t)-k_3Q(t)]\,dt+\sigma_4Q(t)dw_4(t)\\
dR(t) &= [\kappa A(t)+\psi C(t)+\gamma Q(t)-k_4R(t)]\,dt+\sigma_5R(t)dw_5(t)
\end{aligned} \tag{3}$$

with initial condition

$$S(0)=S_0,\ A(0)=A_0,\ C(0)=C_0,\ Q(0)=Q_0,\ R(0)=R_0, \tag{4}$$

where k_j, $(j=1,2,3)$ are constants defined in Section 1, $k_4=\omega+\mu$, and $w_i(t)$, $(i=1,2,3,4,5)$ are independent Brownian motions that are defined on complete probability space $(\Omega,\mathcal{F},\{\mathcal{F}_t\}_{t\geq 0},P)$ with the filtration $\{\mathcal{F}_t\}_{t\geq 0}$, satisfying the usual conditions (it is right continuous and increasing, while $\mathcal{F}_0$ contains all P-null sets) and σ_i, $(i=1,2,3,4,5)$ represent intensities of white noise.

2.1. Existence and Uniqueness of Global Solution

To examine dynamical properties of system (3), taking into consideration the context of the system in real life, the existence of a global positive solution is basically needed. In order for a stochastic differential equation to have a unique global solution (i.e. solution that does not explode in finite time) for any given initial data, the coefficients of stochastic differential equation are generally required to satisfy the linear growth condition and local Lipschitz condition [11]. By the following theorem we prove the existence and uniqueness of the global positive solution.

Theorem 2.1. *There is a unique global solution* $(S(t),A(t),C(t),Q(t),R(t))$ *of system (3), on* $t\geq 0$, *for any initial condition (6). This solution will remain in* Γ *with probability 1.*

Proof. Having in mind that for any given initial value (6) the coefficients of the system (3) are locally Lipschitz continuous, there exists a unique local solution $(S(t),A(t),C(t),Q(t),R(t))$ on $t\in[0,\tau_\varepsilon)$, where τ_ε represents the explosion time. In order to prove that this solution has global character, we show that $\tau_\varepsilon=\infty$ a.s.

Let $m_0>0$ be sufficiently large so that each of S_0, A_0, C_0, Q_0 and R_0 lie within the interval $\left[\frac{1}{m_0},m_0\right]$. Then, for each integer $m\geq m_0$, the stopping time τ_m, can be defined as it follows

$$\tau_m=\inf\left\{t\in[0,\tau_\varepsilon):S(t)\notin\left(\frac{1}{m},m\right)\vee A(t)\notin\left(\frac{1}{m},m\right)\vee C(t)\notin\left(\frac{1}{m},m\right)\vee Q(t)\notin\left(\frac{1}{m},m\right)\vee R(t)\notin\left(\frac{1}{m},m\right)\right\},$$

where throughout this paper we set $\inf\emptyset=\infty$ (as usual $\emptyset$ represents the empty set).

By the form of stopping time, we conclude that τ_m is increasing when $m\to\infty$. Set $\tau_\infty=\lim_{m\to\infty}\tau_m$. Then, $\tau_\varepsilon=\infty$ a.s. and $(S(t),A(t),C(t),Q(t),R(t))$ is a positive global solution of system (3) a.s. for $t\geq 0$. In case

that this statement is not true, then there exist a pair of constants $T>0$ and $\rho\in(0,1)$ such that $P\{\tau_\infty\le T\}>\rho$. Consequently, for an integer $m_1\ge m_0$, holds that

$$P\{\tau_\infty\le T\}\ge\rho \quad \text{for all} \quad m\ge m_1.$$

Define a C^2-function $V:\mathbb{R}^5_+\to\mathbb{R}_+$ by

$$V(S,A,C,Q,R)=S-1-\ln S+A-1-\ln A+C-1-\ln C+Q-1-\ln Q+R-1-\ln R.$$

Inequality $b-1-\ln b\ge 0$, that holds for any $b>0$ directly implies the nonnegativity of the function above. Using the Itô formula for the system (3), we obtain

$$dV(S,A,C,Q,R)=LV(S,A,C,Q,R)+\frac{S-1}{S}\left(S-\frac{\Pi}{\mu}\right)\sigma_1dw_1+(A-1)\sigma_2dw_2+(C-1)\sigma_3dw_3+(Q-1)\sigma_4dw_4+(R-1)\sigma_5dw_5,$$

where

$$\begin{aligned}LV(S,A,C,Q,R)=&\frac{S-1}{S}(\Pi+\omega R-(\lambda+\mu)S)+\frac{A-1}{A}(\lambda S-k_1A)+\frac{C-1}{C}(\xi A-k_2C)\\&+\frac{Q-1}{Q}(\alpha C-k_3Q)+\frac{R-1}{R}(\kappa A+\psi C+\gamma Q-k_4R)+\frac{1}{2}\cdot\frac{1}{S^2}\left(S-\frac{\Pi}{\mu}\right)^2\sigma_1^2+\frac{1}{2}\sum_{i=2}^{5}\sigma_i^2.\end{aligned}$$

Bearing in mind the definition of constants k_j for $j=\overline{1,4}$ and the fact that $\lambda\le\beta$, we conclude

$$\begin{aligned}LV(S,A,C,Q,R)\le&-\mu S-\left(\Pi+\frac{\Pi}{\mu}\sigma_1^2\right)\cdot\frac{1}{S}+\frac{1}{2}\cdot\left(\frac{\Pi}{\mu}\sigma_1\right)^2\cdot\frac{1}{S^2}-(\mu+\delta_a)A-(\mu+\delta_c)C-(\mu+\delta_q)Q-\mu R\\&-\frac{\omega R}{S}-\frac{\lambda S}{A}-\frac{\xi A}{C}-\frac{\alpha C}{Q}-\frac{\kappa A}{R}-\frac{\psi C}{R}-\frac{\gamma Q}{R}+M,\end{aligned}$$

where $M=\Pi+\beta+\mu+k_1+k_2+k_3+k_4+\frac{1}{2}\sum_{i=1}^{5}\sigma_i^2$. Thus, we obtain that $LV\le K$, where K is a positive constant. Therefore,

$$dV(S,A,C,Q,R)\le Kdt+\frac{S-1}{S}\left(S-\frac{\Pi}{\mu}\right)\sigma_1dw_1+(A-1)\sigma_2dw_2+(C-1)\sigma_3dw_3+(Q-1)\sigma_4dw_4+(R-1)\sigma_5dw_5.$$

The rest of the proof is rather standard for this type of theorems, and, hence, is omitted. □

2.2. Stability in Probability

Our interest in this section is to establish conditions for coefficients of the system (3), under which the hepatitis C will die out in population. In order to ensure elimination of hepatitis C, we prove stability in probability of the disease–free equilibrium of system (3). Definitions and theorems about stability in probability of stochastic differential equations may be found in [4], for example, and all of them are about stability of trivial solution of the considered stochastic differential equation. Thus, let us introduce new variable $X=S-\frac{\Pi}{\mu}$ and obtain the system

$$\begin{aligned}dX(t)&=\left[\omega R(t)-(\lambda+\mu)X(t)-\lambda\frac{\Pi}{\mu}\right]dt+\sigma_1X(t)dw_1(t)\\dA(t)&=\left[\lambda X(t)+\lambda\frac{\Pi}{\mu}-k_1A(t)\right]dt+\sigma_2A(t)dw_2(t)\\dC(t)&=[\xi A(t)-k_2C(t)]\,dt+\sigma_3C(t)dw_3(t)\\dQ(t)&=[\alpha C(t)-k_3Q(t)]\,dt+\sigma_4Q(t)dw_4(t)\\dR(t)&=[\kappa A(t)+\psi C(t)+\gamma Q(t)-k_4R(t)]\,dt+\sigma_5R(t)dw_5(t)\end{aligned}\tag{5}$$

with initial condition

$$X(0)=S_0-\frac{\Pi}{\mu},\ A(0)=A_0,\ C(0)=C_0,\ Q(0)=Q_0,\ R(0)=R_0. \tag{6}$$

In the sequel, we consider equations for infected individuals from (5), i.e. system (7) which represents the dynamics of acute, chronic and isolated individuals

$$\begin{aligned} dA(t)&=\left[\beta\left[\frac{\eta A(t)+C(t)+\zeta Q(t)}{N(t)}\right]X(t)+\beta\left[\frac{\eta A(t)+C(t)+\zeta Q(t)}{N(t)}\right]\frac{\Pi}{\mu}-k_1A(t)\right]dt+\sigma_2A(t)dw_2(t)\\ dC(t)&=[\xi A(t)-k_2C(t)]\,dt+\sigma_3C(t)dw_3(t)\\ dQ(t)&=[\alpha C(t)-k_3Q(t)]\,dt+\sigma_4Q(t)dw_4(t)\end{aligned} \tag{7}$$

with initial condition $(A_0,\ C_0,\ Q_0)$.

Since many problems concerning the stability of equilibrium states of nonlinear stochastic system can be reduced to those about stability of solutions of linear associated system, let us consider the linear form of system (7). For that purpose, we use procedure similar to one described in [17]. Let us denote from system (5) the following functions

$$\begin{aligned} f^1(X,A,C,Q,R)&=\omega R-\mu X-\beta\frac{\eta A+C+\zeta Q}{X+\frac{\Pi}{\mu}+A+C+Q+R}\left(X+\frac{\Pi}{\mu}\right), & g^1(X,A,C,Q,R)&=\sigma_1X,\\ f^2(X,A,C,Q,R)&=\beta\frac{\eta A+C+\zeta Q}{X+\frac{\Pi}{\mu}+A+C+Q+R}\left(X+\frac{\Pi}{\mu}\right)-k_1A, & g^2(X,A,C,Q,R)&=\sigma_2A,\\ f^3(X,A,C,Q,R)&=\xi A-k_2C, & g^3(X,A,C,Q,R)&=\sigma_3C,\\ f^4(X,A,C,Q,R)&=\alpha C-k_3Q, & g^4(X,A,C,Q,R)&=\sigma_4Q,\\ f^5(X,A,C,Q,R)&=\kappa A+\psi C+\gamma Q-k_4R, & g^5(X,A,C,Q,R)&=\sigma_5R.\end{aligned} \tag{8}$$

It is obvious that all functions $f^i(X,A,C,Q,R)$ and $g^i(X,A,C,Q,R)$, $i=\overline{1,5}$, in (8) are differentiable with respect to X,A,C,Q and R. If we use representation for all these functions

$$\begin{aligned} f^i(X,A,C,Q,R)&=f^i(0,0,0,0,0)+(f^i)'_X(0,0,0,0,0)\,X+(f^i)'_A(0,0,0,0,0)\,A+(f^i)'_C(0,0,0,0,0)\,C\\ &\quad+(f^i)'_Q(0,0,0,0,0)\,Q+(f^i)'_R(0,0,0,0,0)\,R+o(X,A,C,Q,R),\\ g^i(X,A,C,Q,R)&=g^i(0,0,0,0,0)+(g^i)'_X(0,0,0,0,0)\,X+(g^i)'_A(0,0,0,0,0)\,A+(g^i)'_C(0,0,0,0,0)\,C\\ &\quad+(g^i)'_Q(0,0,0,0,0)\,Q+(g^i)'_R(0,0,0,0,0)\,R+o(X,A,C,Q,R),\end{aligned}$$

where $o(X,A,C,Q,R)$ is a negligible small term of higher order than one, we obtain the linear part of system (5)

$$\begin{aligned} d\tilde X(t)&=\left[-\mu\tilde X(t)-\beta\left(\eta\tilde A(t)+\tilde C(t)+\zeta\tilde Q(t)\right)+\omega\tilde R(t)\right]dt+\sigma_1\tilde X(t)dw_1(t)\\ d\tilde A(t)&=\left[\beta\left(\eta\tilde A(t)+\tilde C(t)+\zeta\tilde Q(t)\right)-k_1\tilde A(t)\right]dt+\sigma_2\tilde A(t)dw_2(t)\\ d\tilde C(t)&=\left[\xi\tilde A(t)-k_2\tilde C(t)\right]dt+\sigma_3\tilde C(t)dw_3(t)\\ d\tilde Q(t)&=\left[\alpha\tilde C(t)-k_3\tilde Q(t)\right]dt+\sigma_4\tilde Q(t)dw_4(t)\\ d\tilde R(t)&=\left[\kappa\tilde A(t)+\psi\tilde C(t)+\gamma\tilde Q(t)-k_4\tilde R(t)\right]dt+\sigma_5\tilde R(t)dw_5(t),\end{aligned} \tag{9}$$

and from it, the linear part of system (7) is

$$\begin{aligned} d\tilde A(t)&=\left[\beta\left(\eta\tilde A(t)+\tilde C(t)+\zeta\tilde Q(t)\right)-k_1\tilde A(t)\right]dt+\sigma_2\tilde A(t)dw_2(t)\\ d\tilde C(t)&=\left[\xi\tilde A(t)-k_2\tilde C(t)\right]dt+\sigma_3\tilde C(t)dw_3(t)\\ d\tilde Q(t)&=\left[\alpha\tilde C(t)-k_3\tilde Q(t)\right]dt+\sigma_4\tilde Q(t)dw_4(t).\end{aligned} \tag{10}$$

The stability conditions will be obtained by using the appropriate Lyapunov function.

Theorem 2.2. *Let the parameters of system (7) satisfy condition* $\mathcal{R}_0 < 1$ *and*

$$2\beta\eta+\hat{a}\xi+\beta\zeta<2k_1, \tag{11}$$

$$\alpha+\hat{a}\beta\left(1+\frac{\zeta}{2}\right)<2k_2, \tag{12}$$

$$\alpha+\beta\zeta\left(1+\frac{\hat{a}}{2}\right)<2k_3, \tag{13}$$

$$\beta\eta<k_1+k_2, \tag{14}$$

where $\hat{a}$ *is an arbitrary positive number such that*

$$\hat{a}>\frac{2(\beta+\xi)}{k_1+k_2-\beta\eta}. \tag{15}$$

Assume also that

$$\sigma_2^2<2k_1-(2\beta\eta+\hat{a}\xi+\beta\zeta), \tag{16}$$

$$\sigma_3^2<2k_2-\left(\alpha+\hat{a}\beta\left(1+\frac{\zeta}{2}\right)\right), \tag{17}$$

$$\sigma_4^2<2k_3-\left(\alpha+\beta\zeta\left(1+\frac{\hat{a}}{2}\right)\right). \tag{18}$$

Then the trivial solution of system (10) is asymptotically mean square stable.

Proof. Let $V(\tilde{A},\tilde{C},\tilde{Q})=\tilde{A}^2+\tilde{C}^2+\tilde{Q}^2+a\tilde{A}\tilde{C}$ be Lyapunov function, where a is a positive constant that will be chosen later. Applying the differential operator L that is associated to the system (10), we obtain

$$\begin{aligned}LV=&-\left(2k_1-2\beta\eta-a\xi-\sigma_2^2\right)\tilde{A}^2-\left(2k_2-a\beta-\sigma_3^2\right)\tilde{C}^2-\left(2k_3-\sigma_4^2\right)\tilde{Q}^2\\&+\left(2\beta+2\xi-a\left(k_1+k_2-\beta\eta\right)\right)\tilde{A}\tilde{C}+2\beta\zeta\tilde{A}\tilde{Q}+\left(2\alpha+a\beta\zeta\right)\tilde{Q}\tilde{C}.\end{aligned}$$

If we choose the constant a as $\hat{a}$ in (15), where positivity of this number is guaranteed by condition (14), and use elementary inequality $\pm 2xy \le x^2 + y^2$, we obtain

$$LV\le-\left(2k_1-(2\beta\eta+\hat{a}\xi+\beta\zeta)-\sigma_2^2\right)\tilde{A}^2-\left(2k_2-\left(\alpha+\hat{a}\beta\left(1+\frac{\zeta}{2}\right)\right)-\sigma_3^2\right)\tilde{C}^2-\left(2k_3-\left(\alpha+\beta\zeta\left(1+\frac{\hat{a}}{2}\right)\right)-\sigma_4^2\right)\tilde{Q}^2.$$

Conditions (11)-(13) and (16)-(18) ensure that quantities in the brackets multiplying $\tilde{A}^2, \tilde{C}^2, \tilde{Q}^2$ are positive, which completes the proof. □

Now, if we substitute $A=C=Q=0$ in (9), we obtain

$$d\tilde{X}(t)=-\mu\tilde{X}(t)dt+\sigma_1\tilde{X}dw_1(t)$$
$$d\tilde{R}(t)=-k_4\tilde{R}(t)dt+\sigma_5\tilde{R}(t)dw_5(t),$$

where both equations are homogeneous stochastic differential equations that have the trivial solutions $\tilde{X}(t) = 0$ and $\tilde{R}(t) = 0$, for $t \ge 0$. It is well known (see [13], for example) that the solutions of these two equations are asymptotically mean square stable if

$$\sigma_1^2 < 2\mu, \tag{19}$$

$$\sigma_5^2 < 2k_4. \tag{20}$$

Therefore, the trivial equilibrium of system (9) is asymptotically mean square stable under the conditions of Theorem 2.2 and conditions (19) and (20).

The order of nonlinearity of system (5) is greater than one. According to this, based on theoretical results (see [15–17]), all conditions that are contained in formulation of Theorem 2.2, with addition of conditions (19) and (20), are sufficient for stability in probability of trivial solution of system (5), which is equivalent to the fact that under the same conditions the disease-free equilibrium E^0 of system (3) is stabile in probability. Hence, we present the main result in this section without the proof.

Theorem 2.3. *Assume that the parameters of system (3) satisfy all conditions from Theorem 2.2, as well as the conditions (19) and (20). Then the disease-free equilibrium of system (3) is stable in probability.*

3. Model (21)

In this section another stochastic version of deterministic model (1) is considered. Here it is assumed that for basic reproduction number holds $\mathcal{R}_0 > 1$ in order to ensure the existence of endemic equilibrium E^* for system (1). This equilibrium point describes the state in population when hepatitis C persist in population, but there are not outbreaks in sense that number of infected individuals do not explode. As we have already mentioned, environmental fluctuations have a significant influence on transmission of hepatitis C, so, it is reasonable to investigate how these fluctuations affect system (1). The way of stability investigation of our stochastic model in this section is based on the procedure which is explained in detail in Chapter 12.2.3 of [13]. Thus, if we suppose, with the same motivation as in previous section, that the system is exposed to white–noise type stochastic perturbation which intensities are directly proportional to deviations of $S(t)$, $A(t)$, $C(t)$, $Q(t)$ and $R(t)$ from S^*, A^*, C^*, Q^* and R^*, respectively, we obtain stochastic system

$$\begin{aligned}
dS(t) &= [\Pi + \omega R(t) - (\lambda+\mu)S(t)]\,dt + \sigma_1 (S(t)-S^*)\,dw_1(t)\\
dA(t) &= [\lambda S(t) - k_1 A(t)]\,dt + \sigma_2 (A(t)-A^*)\,dw_2(t)\\
dC(t) &= [\xi A(t) - k_2 C(t)]\,dt + \sigma_3 (C(t)-C^*)\,dw_3(t)\\
dQ(t) &= [\alpha C(t) - k_3 Q(t)]\,dt + \sigma_4 (Q(t)-Q^*)\,dw_4(t)\\
dR(t) &= [\kappa A(t) + \psi C(t) + \gamma Q(t) - k_4 R(t)]\,dt + \sigma_5 (R(t)-R^*)\,dw_5(t)
\end{aligned} \tag{21}$$

with initial condition

$$S(0) = s_0,\ A(0) = a_0,\ C(0) = c_0,\ Q(0) = q_0\ , R(0) = r_0, \tag{22}$$

where $w_i(t)$, $i = \overline{1,5}$ are independent Brownian motions defined, as usual, on a complete probability space $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, P)$ with the filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions, and σ_i^2 represent the intensities of $w_i(t)$, $i = \overline{1,5}$.

Before the dynamical features of stochastic model (21) are investigated, the existence and uniqueness of global positive solution for model (21) should be checked out.

3.1. Existence and Uniqueness of Global Solution

Since we consider an epidemiological model, it is important that system (21) has global and non-negative solution which does not explode in the finite time.

Thus, we give the following result without the proof since it can be proved by using the similar procedure as in the proof of Theorem 2.1.

Lemma 3.1. *For any given initial value* (22) $\in \Gamma$ *there exists a unique global positive solution* $(S(t), A(t), C(t), Q(t), R(t))$ *of system (21), for all* $t \geq 0$ *and the solution will remain in* Γ *with probability 1.*

3.2. Stability in Probability

In the Section 2 we investigated the conditions under which the disease will die out by proving the stochastic stability of disease–free equilibrium of system (3). However, system (1) has another equilibrium state that characterize the case when hepatitis C spreads within the population. This equilibrium point is also an equilibrium point of our stochastic model (21). Besides the assumption $\mathcal{R}_0 > 1$ which ensures existence of endemic equilibrium state E^*, as in [5], we will consider a special case of system (21) where

total population is constant, i.e. $N(t)=N^*$. That is how we are able to reduce system (21) by expressing the variable S as $S=N^*-A-C-Q-R$, and obtain

$$\begin{aligned}
dA(t)&=\left[\beta\left[\frac{\eta A(t)+C(t)+\zeta Q(t)}{N^*}\right](N^*-A(t)-C(t)-Q(t)-R(t))-k_1A(t)\right]dt+\sigma_2\,(A(t)-A^*)\,dw_2(t)\\
dC(t)&=[\xi A(t)-k_2C(t)]\,dt+\sigma_3\,(C(t)-C^*)\,dw_3(t)\\
dQ(t)&=[\alpha C(t)-k_3Q(t)]\,dt+\sigma_4\,(Q(t)-Q^*)\,dw_4(t)\\
dR(t)&=[\kappa A(t)+\psi C(t)+\gamma Q(t)-k_4R(t)]\,dt+\sigma_5\,(R(t)-R^*)\,dw_5(t)
\end{aligned}\tag{23}$$

with initial condition

$$A(0)=a_0,\ C(0)=c_0,\ Q(0)=q_0\,,R(0)=r_0.\tag{24}$$

In the sequel, we establish the conditions for coefficients of system (21) which provide stability in probability of endemic equilibrium state.

In order to investigate stability properties of endemic equilibrium state E^*, we center system (23) at it by introducing new variables $x=A-A^*$, $y=C-C^*$, $z=Q-Q^*$ and $v=R-R^*$. Thus, we obtain transformed stochastic system

$$\begin{aligned}
dx(t)&=\Big[(\beta\eta-k_1)\,(x(t)+A^*)+\beta\,(y(t)+C^*)+\beta\zeta\,(z(t)+Q^*)\\
&\quad-\frac{\beta}{N^*}\left[\eta(x(t)+A^*)+(y(t)+C^*)+\xi(z(t)+Q^*)\right](x(t)+A^*+y(t)+C^*+z(t)+Q^*+v(t)+R^*)\Big]dt+\sigma_2x(t)dw_2(t)\\
dy(t)&=[\xi\,(x(t)+A^*)-k_2\,(y(t)+C^*)]\,dt+\sigma_3y(t)dw_3(t)\\
dz(t)&=[\alpha\,(y(t)+C^*)-k_3\,(z(t)+Q^*)]\,dt+\sigma_4z(t)dw_4(t)\\
dv(t)&=[\kappa\,(x(t)+A^*)+\psi\,(y(t)+C^*)+\gamma\,(z(t)+Q^*)-k_4\,(v(t)+R^*)]\,dt+\sigma_5v(t)dw_5(t)
\end{aligned}\tag{25}$$

with initial condition

$$x(0)=a_0-A^*,\ y(0)=c_0-C^*,\ z(0)=q_0-Q^*,\ v(0)=r_0-R^*.\tag{26}$$

It is obvious that the stability in probability of the trivial solution of system (25) is equivalent to the stability in probability of the endemic equilibrium of system (23).

Before proving the stability of the trivial solution for model (25), with the same motivation as in Subsection 2.2, we first consider it's linear part. For that purpose, as in previous section, we will use that

$$\begin{aligned}
F^i(x,y,z,v)&=F^i(0,0,0,0)+(F^i)'_x(0,0,0,0)\,x+(F^i)'_y(0,0,0,0)\,y+(F^i)'_z(0,0,0,0)\,z+(F^i)'_v(0,0,0,0)\,v+o(x,y,z,v),\\
G^i(x,y,z,v)&=G^i(0,0,0,0)+(G^i)'_x(0,0,0,0)\,x+(G^i)'_y(0,0,0,0)\,y+(G^i)'_z(0,0,0,0)\,z+(G^i)'_v(0,0,0,0)\,v+o(x,y,z,v),
\end{aligned}$$

where $o(x,y,z,v)$ is a negligible small term of higher order than one, while the functions $F^i(x,y,z,v)$ and $G^i(x,y,z,v)$, $i=\overline{1,4}$, are differentiable with respect to x,y,z and v, and defined by

$$\begin{aligned}
F^1(x,y,z,v)&=\left[\frac{\beta}{N^*}(\eta x+y+\zeta z)+\lambda^*\right][S^*-(x+y+z+v)]-k_1(x+A^*)\,, &\quad G^1(x,y,z,v)&=\sigma_2x,\\
F^2(x,y,z,v)&=\xi\,(x+A^*)-k_2\,(y+C^*)\,, &\quad G^2(x,y,z,v)&=\sigma_3y,\\
F^3(x,y,z,v)&=\alpha\,(y+C^*)-k_3\,(z+Q^*)\,, &\quad G^3(x,y,z,v)&=\sigma_4z,\\
F^4(x,y,z,v)&=\kappa\,(x+A^*)+\psi\,(y+C^*)+\gamma\,(z+Q^*)-k_4\,(v+R^*)\,, &\quad G^4(x,y,z,v)&=\sigma_5v.
\end{aligned}$$

The previous functions are obtained by using notation from [5] where the authors denote $Y=\frac{k_2}{\xi}+1+\frac{\alpha}{k_3}+\frac{1}{k_4}\left(\frac{k_1k_2}{\xi}+\psi\right)$ and obtain that $C^*=\frac{\mathcal{R}_0-1}{Y}S^*$, $A^*=\frac{k_2}{\xi}\frac{\mathcal{R}_0-1}{Y}S^*$, $Q^*=\frac{\alpha}{k_3}\frac{\mathcal{R}_0-1}{Y}S^*$, $R^*=\frac{1}{k_4}\left(\frac{k_1k_2}{\xi}+\psi\right)\frac{\mathcal{R}_0-1}{Y}S^*$, and

$$N^*=S^*+A^*+C^*+Q^*+R^*=\frac{\mathcal{R}_0-1}{Y}S^*\left[\frac{k_2}{\xi}+1+\frac{\alpha}{k_3}+\frac{1}{k_4}\left(\frac{k_1k_2}{\xi}+\psi\right)\right]+S^*=\mathcal{R}_0S^*,$$

and, hence, $\frac{\beta S^*}{N^*} = \frac{\beta}{\mathcal{R}_0}$, as well as the definition of λ^*. Thus, the linear part of system (25) is

$$\begin{aligned}
d\tilde{x}(t) &= \left[\left(\frac{\beta}{\mathcal{R}_0}\eta - \lambda^* - k_1\right)\tilde{x}(t) + \left(\frac{\beta}{\mathcal{R}_0} - \lambda^*\right)\tilde{y}(t) + \left(\frac{\beta}{\mathcal{R}_0}\zeta - \lambda^*\right)\tilde{z}(t) - \lambda^*\tilde{v}(t)\right]dt + \sigma_2\tilde{x}(t)dw_2(t) \\
d\tilde{y}(t) &= \left[\xi\tilde{x}(t) - k_2\tilde{y}(t)\right]dt + \sigma_3\tilde{y}(t)dw_3(t) \\
d\tilde{z}(t) &= \left[\alpha\tilde{y}(t) - k_3\tilde{z}(t)\right]dt + \sigma_4\tilde{z}(t)dw_4(t) \\
d\tilde{v}(t) &= \left[\kappa\tilde{x}(t) + \psi\tilde{y}(t) + \gamma\tilde{z}(t) - k_4\tilde{v}(t)\right]dt + \sigma_5\tilde{v}(t)dw_5(t).
\end{aligned} \tag{27}$$

Now, we may proceed by considering the conditions for asymptotic mean square stability of system (27).

Theorem 3.2. *Assume that the parameters of model (27) satisfy $\mathcal{R}_0 > 1$ and conditions*

$$\lambda^* > \frac{\frac{\beta}{\mathcal{R}_0}\zeta}{1 - \frac{\gamma}{k_3 + k_4}}, \tag{28}$$

$$\gamma < 2c^*k_3, \tag{29}$$

$$\varepsilon^* < \frac{2c^*k_3 - \gamma}{a^*(\lambda^* - \frac{\beta}{\mathcal{R}_0}\zeta) - \frac{\kappa}{2}}, \tag{30}$$

$$2a^*\left(\frac{\beta}{\mathcal{R}_0}\eta + \frac{\lambda^*}{2\varepsilon^*}\right) + e^*\xi < 2a^*\left(k_1 + \lambda^* + \frac{1}{2\varepsilon^*}\frac{\beta}{\mathcal{R}_0}\zeta\right) + \frac{\kappa}{2\varepsilon^*}, \tag{31}$$

$$e^*\left(\frac{\beta}{\mathcal{R}_0} - \lambda^*\right) < 2b^*k_2, \tag{32}$$

where a^*, b^*, c^*, d^* and e^* are positive constants defined as

$$\begin{aligned}
a^* &= \frac{\kappa(k_3 + k_4)}{2\gamma\lambda^*} \\
b^* &= \frac{\left(\frac{(k_3+k_4)\psi}{\gamma} + \alpha\right)\left(k_1 + k_2 + \lambda^* - \frac{\beta}{\mathcal{R}_0}\eta\right) - \frac{(k_3+k_4)\kappa}{\gamma}\left(\frac{\beta}{\mathcal{R}_0} - \lambda^*\right)}{2\xi\lambda^*} \\
c^* &= \frac{\left(\frac{(k_3+k_4)\psi}{\gamma} + \alpha\right)\left(\lambda^* - \frac{\beta}{\mathcal{R}_0}\zeta\right) - \lambda^*\psi}{2\alpha\lambda^*} \\
d^* &= \frac{k_3 + k_4}{2\gamma} \\
e^* &= \frac{1}{\lambda^*}\left(\frac{(k_3 + k_4)\psi}{\gamma} + \alpha\right).
\end{aligned} \tag{33}$$

Also, let intensities of noise satisfy

$$\sigma_2^2 < \frac{2a^*\left(k_1 + \lambda^* - \frac{\beta}{\mathcal{R}_0}\eta - \frac{1}{2\varepsilon^*}\left(\lambda^* - \frac{\beta}{\mathcal{R}_0}\zeta\right)\right) - e^*\xi + \frac{\kappa}{2\varepsilon^*}}{a^*}, \tag{34}$$

$$\sigma_3^2 < \frac{2b^*k_2 - e^*\left(\frac{\beta}{\mathcal{R}_0} - \lambda^*\right)}{b^*}, \tag{35}$$

$$\sigma_4^2 < \frac{2c^*k_3 - 2a^*\varepsilon^*\left(\lambda^* - \frac{\beta}{\mathcal{R}_0}\zeta\right) - \gamma + \varepsilon^*\kappa}{c^*}, \tag{36}$$

$$\sigma_5^2 < 2k_4. \tag{37}$$

Then, the trivial equilibrium of system (27) is asymptotically mean square stable.

Proof. Using the Lyapunov function,

$$V(\tilde{x},\tilde{y},\tilde{z},\tilde{v}) = a\tilde{x}^2 + b\tilde{y}^2 + c\tilde{z}^2 + d\tilde{v}^2 + e\tilde{x}\tilde{y} + \tilde{z}\tilde{v},$$

where a, b, c, d, e, are positive constants that will be chosen in the sequel, we calculate operator LV for system (27) and obtain

$$\begin{aligned} LV = &-\tilde{x}^2\left[2a\left(\lambda^* + k_1 - \frac{\beta}{\mathcal{R}_0}\eta - \frac{\sigma_2^2}{2}\right) - e\xi\right] - \tilde{y}^2\left[2b\left(k_2 - \frac{\sigma_3^2}{2}\right) - e\left(\frac{\beta}{\mathcal{R}_0} - \lambda^*\right)\right] - \tilde{z}^2\left[2c\left(k_3 - \frac{\sigma_4^2}{2}\right) - \gamma\right] \\ &- 2d\tilde{v}^2\left[k_4 - \frac{\sigma_5^2}{2}\right] + \tilde{x}\tilde{y}\left[2a\left(\frac{\beta}{\mathcal{R}_0} - \lambda^*\right) + 2b\xi - e\left(k_1 + k_2 + \lambda^* - \frac{\beta}{\mathcal{R}_0}\eta\right)\right] + \tilde{x}\tilde{z}\left[2a\left(\frac{\beta}{\mathcal{R}_0}\zeta - \lambda^*\right) + \kappa\right] \\ &+ \tilde{x}\tilde{v}\left[-2a\lambda^* + 2d\kappa\right] + \tilde{y}\tilde{z}\left[2c\alpha + e\left(\frac{\beta}{\mathcal{R}_0}\zeta - \lambda^*\right) + \psi\right] + \tilde{y}\tilde{v}\left[2d\psi - e\lambda^* + \alpha\right] + \tilde{z}\tilde{v}\left[2d\gamma - (k_3 + k_4)\right]. \end{aligned}$$

In order to annul the quantities in the brackets multiplying $\tilde{x}\tilde{y}$, $\tilde{x}\tilde{v}$, $\tilde{y}\tilde{z}$, $\tilde{y}\tilde{v}$ and $\tilde{z}\tilde{v}$, we choose constants a, b, c, d, e to be as a^*, b^*, c^*, d^*, e^* defined in (33), respectively. Thus, we obtain

$$\begin{aligned} LV = &-\tilde{x}^2\left[2a^*\left(\lambda^* + k_1 - \frac{\beta}{\mathcal{R}_0}\eta - \frac{\sigma_2^2}{2}\right) - e^*\xi\right] - \tilde{y}^2\left[2b^*\left(k_2 - \frac{\sigma_3^2}{2}\right) - e^*\left(\frac{\beta}{\mathcal{R}_0} - \lambda^*\right)\right] \\ &- \tilde{z}^2\left[2c^*\left(k_3 - \frac{\sigma_4^2}{2}\right) - \gamma\right] - 2d^*\tilde{v}^2\left[k_4 - \frac{\sigma_5^2}{2}\right] + \tilde{x}\tilde{z}\left[2a^*\left(\frac{\beta}{\mathcal{R}_0}\zeta - \lambda^*\right) + \kappa\right]. \end{aligned}$$

Under condition (28) the term in the bracket that multiply $\tilde{x}\tilde{z}$ is negative. Hence, by using elementary inequality $\pm 2xy \le \varepsilon x^2 + \frac{y^2}{\varepsilon}$, $\varepsilon > 0$, we conclude that

$$\begin{aligned} LV = &-\tilde{x}^2\left[2a^*\left(\lambda^* + k_1 - \frac{\beta}{\mathcal{R}_0}\eta - \frac{1}{2\varepsilon}\left(\lambda^* - \frac{\beta}{\mathcal{R}_0}\zeta\right) - \frac{\sigma_2^2}{2}\right) + \frac{1}{2\varepsilon}\kappa - e^*\xi\right] - \tilde{y}^2\left[2b^*\left(k_2 - \frac{\sigma_3^2}{2}\right) - e^*\left(\frac{\beta}{\mathcal{R}_0} - \lambda^*\right)\right] \\ &- \tilde{z}^2\left[2c^*\left(k_3 - \frac{\sigma_4^2}{2}\right) - a^*\varepsilon\left(\lambda^* - \frac{\beta}{\mathcal{R}_0}\zeta\right) - \gamma + \varepsilon\frac{\kappa}{2}\right] - 2d\tilde{v}^2\left[k_4 - \frac{\sigma_5^2}{2}\right]. \end{aligned}$$

If we choose ε as ε^* in (30), it is a positive constant regarding condition (29). Bearing in mind conditions (31)–(37), all the terms in brackets multiplying $\tilde{x}^2$, $\tilde{y}^2$, $\tilde{z}^2$ and $\tilde{v}^2$ are positive, which completes the proof. □

If we introduce new variable $u = S - S^*$, bearing in mind that $N = N^*$, we can transform the first equation of system (21) into

$$du(t) = \left[\Pi + \omega(v + R^*) - \left(\frac{\beta}{N^*}(\eta x + y + \zeta z) + \lambda^* + \mu\right)(u + S^*)\right]dt + \sigma_1 u dw_1(t),$$

and it's linear part is

$$d\tilde{u}(t) = \left[-(\lambda^* + \mu)\,\tilde{u} - \frac{\beta}{\mathcal{R}_0}\eta\tilde{x} - \frac{\beta}{\mathcal{R}_0}\tilde{y} - \frac{\beta}{\mathcal{R}_0}\zeta\tilde{z} + \omega\tilde{v}\right]dt + \sigma_1\tilde{u}dw_1(t).$$

As in the previous section, we can now substitute $\tilde{x} = \tilde{y} = \tilde{z} = \tilde{v} = 0$ in the last equation and get homogeneous stochastic differential equation

$$d\tilde{u}(t) = -(\lambda^* + \mu)\,\tilde{u}(t)dt + \sigma_1\tilde{u}(t)dw_1(t),$$

which has the trivial solutions $\tilde{u}(t) = 0$. This solution is asymptotically mean square stable if

$$\sigma_1^2 < 2(\mu + \lambda^*), \tag{38}$$

which is in more details explained in [13].

It is already highlighted that sufficient conditions for asymptotic mean square stability of the trivial solution of linear system coincide with sufficient conditions for stability in probability of the trivial solution of the corresponding nonlinear system.

Hence, sufficient conditions for stability in probability of endemic equilibrium state E^* of system (21) are presented in the following theorem, without the proof.

Theorem 3.3. *Assume that the parameters of system (21) satisfy all conditions from Theorem 3.2, as well as the condition (38). Then the endemic equilibrium E^* of system (21) is stable in probability.*

4. Numerical Simulations and Conclusions

In order to confirm theoretical results that are discussed in Sections 2 and 3, a numerical simulation for systems (3) and (21) is made. For numerical simulation, we use the Euler-Maruyama approximate method (see [8]) to simulate the solutions of the considered equations[1)].

For simulation purpose we use reliable data presented in [5] to describe the dynamics of hepatitis C transmission.

From the conditions of Theorem 2.3, it is obvious that if effective contact rate β does not exceed certain value, force of infection λ is not so strong and we can expect extinction of the disease from population. Thus, let the model parameters for system (3) be:

$$\begin{aligned} &\Pi = 0.12,\ \gamma = 0.18,\ \kappa = 0.2,\ \omega = 0.95,\ \mu = \frac{1}{21\,900},\ \xi = 0.7,\ \alpha = 0.15,\ \psi = 0.05, \\ &\delta_a = 0.000233,\ \delta_c = 0.00233,\ \delta_q = 0.0.001667,\ \eta = 0.5,\ \zeta = 0.1,\ \beta = 0.1369. \end{aligned} \tag{39}$$

Initial value is

$$S_0 = 2\,000,\ A_0 = 200,\ C_0 = 600,\ Q_0 = 120,\ R_0 = 100. \tag{40}$$

Having in mind the expression for reproduction number, it is not difficult to compute that $\mathcal{R}_0 = 0.6454 < 1$.

Moreover, from conditions (16)–(20) of Theorem 2.3, we can choose intensities of the noise to be

$$\sigma_1^2 = 0.00009,\ \sigma_2^2 = 0.102,\ \sigma_3^2 = 0.0169,\ \sigma_4^2 = 0.06,\ \sigma_5^2 = 0.005. \tag{41}$$

Parameters (39) and (41) satisfy all the conditions from Theorem 2.3, which means that equilibrium state $E^0 = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0\right)$ of system (3) is stable in probability, i.e. under these conditions we can expect elimination of hepatitis C from population. This can be seen in Figures 1–3. In all plots the unit of time is one day.

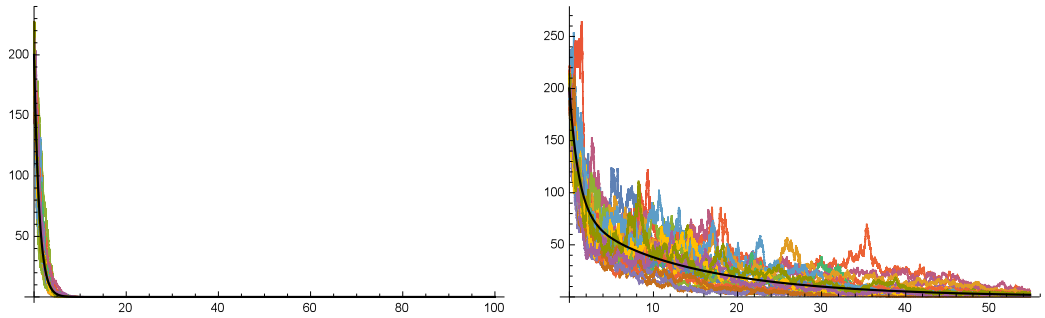

Figure 1: Deterministic (black line) and 25 stochastic trajectories for acute individuals A(t) of models (1) and (3) with initial conditions (40) for parameter values (39) and (41).

[1)] All simulations are made by using *MATHEMATICA* programme.

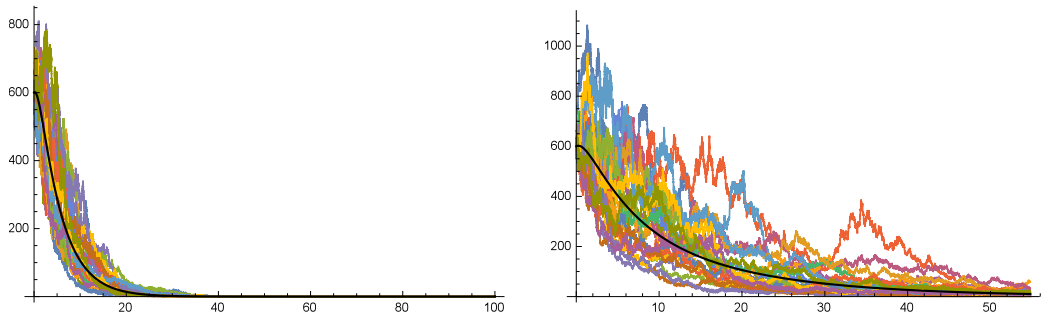

Figure 2: Deterministic (black line) and 25 stochastic trajectories for chronic individuals C(t) of models (1) and (3) with initial conditions (40) for parameter values (39) and (41).

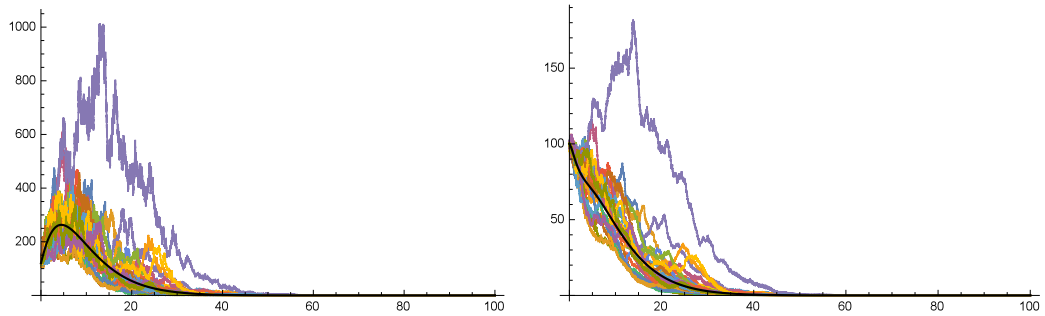

Figure 3: Deterministic (black line) and 25 stochastic trajectories for isolated $Q(t)$ (left) and recovered individuals $R(t)$ (right) of models (1) and (3) with initial conditions (40) for parameter values (39) and (41).

An interesting result was obtained when we try to plot susceptible individuals in order to see that $S(t) \to \frac{\Pi}{\mu}$. More precisely, when we take intensity of the noise from (41) everything was as we expected to be, but what was not expected is that intensities of the noise $\sigma_1^2 = 0.28$ and larger also give us stability in probability of equilibrium state E_0, and we show plot of 25 stochastic trajectories of susceptible individuals $S(t)$ in Figure 4.

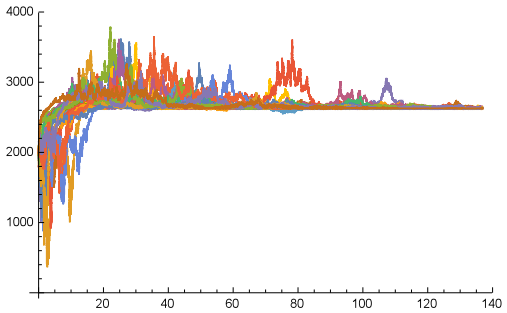

Figure 4: Stochastic trajectories (25) for susceptible individuals $S(t)$ of model (3) with initial conditions (40) for parameter values (39) and $\sigma_1^2 = 0.28$, $\sigma_2^2 = 0.102$, $\sigma_3^2 = 0.0169$, $\sigma_4^2 = 0.06$, $\sigma_5^2 = 0.005$.

On the other hand, if we enhance β, infection becomes stronger and then hepatitis C will persist in population. Setting $\Pi = 1$, $\beta = 0.5703$, and other model parameters with the same value as in (39), we obtain $\mathcal{R}_0 = 2.6887 > 1$ which means that there exists the endemic equilibrium E^* of system (21).

In this case, we use initial value

$$s_0 = 600,\ a_0 = 20,\ c_0 = 60,\ q_0 = 12,\ r_0 = 10, \tag{42}$$

and the force of infection $\lambda = 0.1843$. From conditions (34)–(38) of Theorem 3.3 we set

$$\sigma_1^2 = 0.3,\ \sigma_2^2 = 0.09,\ \sigma_3^2 = 0.00022,\ \sigma_4^2 = 0.00006,\ \sigma_5^2 = 0.005. \tag{43}$$

It is easy to check out that all the conditions of Theorem 3.3 hold, and thus, we expect that the solution of model will tend to endemic equilibrium $E^* = (357.755, 73.2509, 253.214, 209.15, 68.3816)$, which means that hepatitis C will persist in population.

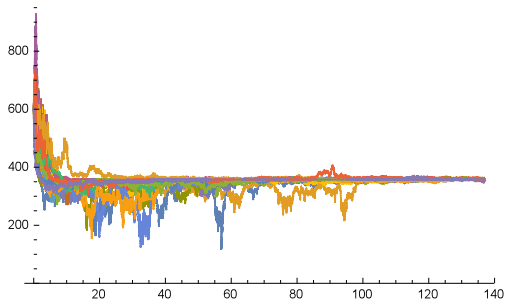

Figure 5: Stochastic trajectories (25) for susceptible individuals $S(t)$ of model (21) with initial conditions (42) for parameter values $\Pi = 1$, $\beta = 0.5703$, and other parameters are the same as in (39), $\lambda = 0.1843$ and (43).

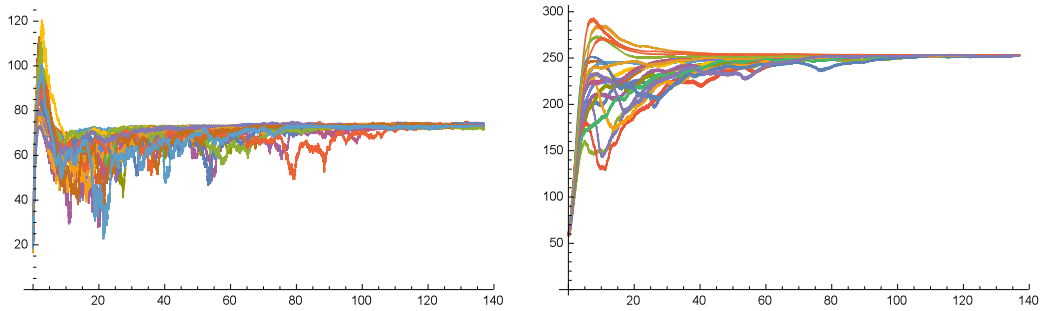

Figure 6: Stochastic trajectories (25) for acute $A(t)$ (left) and chronic individuals $C(t)$ (right) of model (3) with initial conditions (42) for parameter values $\Pi = 1$, $\beta = 0.5703$, and other parameters are the same as in (39), $\lambda = 0.1843$ and (43).

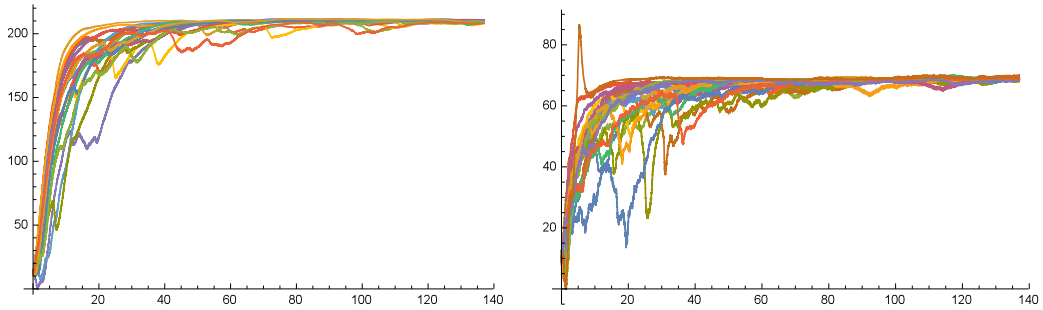

Figure 7: Stochastic trajectories (25) for isolated $Q(t)$ (left) and recovered individuals $R(t)$ (right) of model (3) with initial conditions (42) for parameter values $\Pi = 1$, $\beta = 0.5703$, and other parameters are the same as in (39), $\lambda = 0.1843$ and (43).

Another important parameter, that can help to reduce the number of infective population of Hepatitis C is isolation rate of chronically infected individuals α. Increasing the rate of isolation class decreases the time to disease extinction, regardless of the value of effective contact rate β.

From biological point of view, it is important to control the disease. Thus it would be useful to see how much the effective contact rate may be increased until the outbreak of disease, and this can be interesting topics for some further investigations.

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