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Stochastic Differential Equations Model for the Interaction of HCV with Liver Cells

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Abstract

The main objective of this paper is to study the dynamic behavior of the hepatitis C virus to control the spread of this disease. The study was conducted by creating a new stochastic mathematical model. Three equilibriums specific to this system are discussed, namely, disease-free equilibrium, equilibrium in the absence of an antibody response, and equilibrium when immune response CTLs are zero. We also showed conditions that must be met for the injured person to recover by searching for the basic reproduction number. If $R_0 < 1$ it means that the liver will get rid of the virus and heal the infected person. While if $R_0 > 1$ in this case, the infection grows and the disease can invade all liver cells. These results have also been demonstrated by computer simulations.

Keywords: Mathematical modeling of HCV, Basic reproduction number, Hepatitis C virus, Disease-free equilibrium.

نموذج المعادلات التفاضلية التصادفية لتفاعل HCV مع خلايا الكبد

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الخلاصة

الهدف الرئيسي من هذا البحث, هو دراسة السلوك الديناميكي لفيروس التهاب الكبد سي من اجل السيطرة على انتشار هذا المرض. اجريت الدراسة من خلال انشاء نموذج رياضي تصادفي جديد. تمت مناقشة ثلاث توازنات خاصة بهذا النظام,

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وهي التوازن الخالي من الأمراض, والتوازن في غياب استجابة الجسم المضاد, والتوازن عندما تكون الاستجابة المناعية صفر. تم إظهار الشروط التي يجب توافرها حتى يتعافى المصاب من خلال انشاء رقم التكاثر الاساسي . اذا كان رقم التكاثر الاساسي اقل من واحد, فهذا يعني أن الكبد سيتخلص من الفيروس ويشفى الشخص المصاب, وبالعكس اذا كان رقم التكاثر الاساسي اكبر من واحد في هذه الحالة تنمو العدوى ويمكن للمرض ان يغزو جميع خلايا الكبد. كما تم توضيح هذه النتائج من خلال المحاكاة الحاسوبية.

الكلمات المفتاحية: النمذجة الرياضية لفيروس التهاب الكبد سي, عدد التكاثر الاساسي, فيروس التهاب الكبد سي, التوازن الخالي من الأمراض.

Introduction

Hepatitis is an irritation of the liver. This disease may be caused by excessive alcohol consumption, autoimmune diseases, medications, viruses, and bacteria. There are five types of viral hepatitis, including hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), hepatitis D virus (HDV), and hepatitis E virus (HEV). In this manuscript, we will focus our study on hepatitis C because of the huge global health burden caused by this disease. Worldwide, an estimated 58 million people have chronic HCV infection, with approximately 1.5 million new infections annually. It is estimated that 3.2 million adolescents and children suffer from chronic hepatitis C infection. The World Health Organization estimates that in 2019, approximately 290,000 people died from hepatitis C, mostly from cirrhosis and hepatocellular carcinoma[1]. Mathematical models were developed to understand the dynamics of HCV because it helps to interpret the experimental result and to understand the underlying biological mechanism involved in the spreading of the epidemic [2-4]. The vast amount of scientific research that has been done on modeling the interaction of the hepatitis C virus with human hepatocytes has been largely limited to ordinary differential equations (ODEs)[5–10]. In recent years, attention has been paid to stochastic differential equations, and they have been used in modeling various diseases caused by viruses, for example, Coronavirus disease, AIDS, as well as viral hepatitis[11-15].In our paper, we will use a stochastic differential equations model for the interaction of HCV virus with hepatocytes in the presence of immunity. There are many reasons that lead us to use stochastic differential equation models instead of deterministic ordinary differential equations models. Real life is stochastic rather than

deterministic, especially when modeling viral hepatitis outbreak phenomena such as hepatitis C virus dynamics. This is because virus particles that interact with target liver cells are in the same environmental conditions but produce different products. This paper presents the effect of introducing randomness on a deterministic ordinary differential equation model. The new method of mathematical modeling presents more clear results than the deterministic ordinary differential equation models because of using the random differential equations model several times can lead to the expected distribution of the results. For example the total number of cells infected with the HCV virus at time t, while the deterministic differential equations model will give us one expected value. This article is organized as follows. In section 2, We created a new mathematical model that shows the spread of (HCV) disease inside liver cells in the presence of immunity also, a table of all the parameters used during our work is presented. Section 3 describes the basic reproduction number and equilibriums for the new stochastic differential equation model. In section 4, we establish the conditions for which the equilibrium points will be stable or unstable. The main results are presented in Section5. Finally, Section 6 is devoted to the conclusion part and some recommendations.

Formulation of the Model

To understand the behavior of the causative virus (HCV) and how it interacts with liver cells, several mathematical models have been developed, the most important of which is the model developed by [8]. The behavior of the virus was studied in the presence of the treatment, since in this model it was assumed that infected hepatocytes and uninfected T cells proliferate, and we agree with this model because the liver is an organ that regenerates due to the population homeostasis system in the human liver [16]. Thus, any loss of hepatocytes can be compensated for by the proliferation of existing hepatocytes and the mathematical model provided by [8] is as follows:

$$\begin{cases} \frac{dT}{dt} = G + nT\left(1 - \frac{T+I}{T_{max}}\right) - bT - (1-u)wCT \\ \frac{dI}{dt} = (1-u)wCT + nI\left(1 - \frac{T+I}{T_{max}}\right) - sI \\ \frac{dc}{dt} = (1-\mu)qI - \alpha C \end{cases} \quad (1)$$

We note that the mathematical model (1) ignores the immune system responses, which are the most important factor in stimulating therapeutic cells. While, it was proposed by the researchers in the source [9]a mathematical model that deals with the interaction between the virus that causes the disease (HCV) and the immune responses in the host, and it is as follows:

$$\begin{aligned}
\frac{dT}{dt} &= G - bT - wCT \\
\frac{dI}{dt} &= wCT - sI - \beta IY \\
\frac{dC}{dt} &= qI - \alpha C - kcZ \\
\frac{dY}{dt} &= mIY - dY \\
\frac{dZ}{dt} &= \tau CZ - hZ
\end{aligned} \tag{2}$$

In this paper, we introduce a new mathematical model that describes the interaction of viruses with hepatocytes by integrating the mathematical model (1) with the mathematical model (2), and because the process of virus multiplication inside the human body through its entry into liver cells is subject to many complex biological factors, as well as the effectiveness of treatment is variable from one person to another. All these reasons prompted us to introduce random parameters in the new proposed model, which is as follows:

$$\begin{cases}
dT = \left(G + nT\left(1 - \frac{T+I}{T_{max}}\right) - bT - (1-u_1)wCT\right)dt + \sigma_1 u_1 wCT dW_1(t) & (3) \\
dI = \left((1-u_1)wCT + nI\left(1 - \frac{T+I}{T_{max}}\right) - sI - \beta IY\right)dt - \sigma_1 u_1 wCT dW_1(t) & (4) \\
dC = \left((1-u_2)qI - \alpha C - kCZ\right)dt - \sigma_2 u_2 qI dW_2(t) & (5) \\
\frac{dY}{dt} = mIY - dY & (6) \\
\frac{dZ}{dt} = \tau CZ - hZ & (7)
\end{cases}$$

All parameters used in the mathematical model (1) and (2) as well as in the new mathematical model (3-7) are mentioned and illustrated in Table 1

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Table 1: Model States and Model parameters

Parameter	Description
T_{max}	The maximum size of the liver growth.
$I(t)$	Represents the infected cells.
$C(t)$	Represent virus particles.
G	It represents the production rate of uninfected cells per unit time.
n	The highest proliferation rate of infected (I) cells and uninfected (T) cells.
b	It represents the rate of removal of healthy cells per unit time.
w	It represents the rate of transmission of virus particles into liver cells.
s	Represents the rate of removal of infected cells.
β	Represent rate at which CTLs (Y) kills infected cells.
q	It is the maximum amount of infectious virus particles produced from infected cells in the case of a weak or ineffective vaccine.
α	It represents the rate of removal of virus particles per unit of time.
k	Represent rate at which antibody (Z) neutralized the virus particles(c).
m	Expand the rate of CTLs (Y) in response to virus antigen.
d	The removal rate of CTLs (Y) in the absence of antigenic.
τ	The growth rate of antibody (Z) in response to virus particles(c).
h	Natural decay rate of antibody (Z).
$(1-u_1)$	Represents the potential for a vaccine that blocks the interaction between infectious virus particles and healthy target cells (T).
$(1-u_2)$	It represents a treatment that prevents virus particles from gathering properly and this results in a weakened virus that is unable to replicate.
σ_1 & σ_2	Are parameters used to model the stochastic in the evolution.
$W_1(t)$ &$W_2(t)$	It represents independent standard Brownian motions.

The Basic Reproduction Number (R_0) and equilibriums

To understand the behavior of the virus that causes HCV, and whether the human liver can heal and get rid of HCV, we will use the basic reproduction number, which is indicated by the symbol R_0 which is the number of secondary infections produced by a single infected cell in a hepatocyte population. If $R_0 < 1$, this means that any virus-carrying cell will transmit viruses to less than one cell and this means the liver will get rid of the virus and heal the infected person. Unlike that if $R_0 > 1$ so, each infected cell produces on average more than one new infected cell and in this case, the infection grows and the disease can invade all liver cells. The basic reproduction number (R_0) for the new mathematical model will be as:

$$R_0 = \frac{1}{s}\left(n\left(1-\frac{T_0}{T_{max}}\right)+\frac{(1-u_1-\sigma_1 u_1)(1-u_2-\sigma_2 u_2)wT_0 q}{\propto}\right) \quad (8)$$

To determine the stability of the new model (3 –7), we evaluate the steady states or the equilibrium points of this model. Similar to models (1) and (2), the new model has an equilibrium point called disease- free equilibrium. This represents the nonappearance of virus (i.e.,$C_0 = 0$). So when solving the equations (3)-(7) , we obtain disease- free equilibrium which is as follows:

$$(T_0, I_0, C_0, Y_0, Z_0) = \left(\frac{T_{max}}{2n}\left[n - b \pm \sqrt{(n-b)^2 + \frac{4nG}{T_{max}}}\right], 0,0,0,0\right)$$

When the virus particles ($C_1 \neq 0$) and the antibody response ($Z_1 = 0$) the second equilibrium point will found as follows,

solving equation (6) to get

$$I_1 = \frac{d}{m}, \tag{9}$$

Then, substitute equations (9) and ($Z_1 = 0$) in equation (5) to get

$$C_1 = \frac{(1-u_2)-\sigma_2 u_2 qd}{m\alpha}, \tag{10}$$

Then from equation (3) obtain

$$T_1 = \frac{T_{max}}{2n}\left(n - \frac{nI_1}{T_{max}} - b - (1-u_1)wC_1 + \sigma_1 u_1 wC_1 \pm \sqrt{\left(n - \frac{nI_1}{T_{max}} - b - (1-u_1)wC_1 + \sigma_1 u_1 wC_1\right)^2 - 4\left(\frac{-n}{T_{max}}\right)G}\right) \tag{11}$$

when substitute equation (9)and (10) in equation (11) obtain

$$T_1 = \frac{T_{max}}{2n}\left(\begin{array}{c} n - \frac{nd}{mT_{max}} - b - (1-u_1)w\frac{(1-u_2)-\sigma_2 u_2 qd}{m\alpha} + \sigma_1 u_1 w\frac{(1-u_2)-\sigma_2 u_2 qd}{m\alpha} \\ \pm\sqrt{\left(n - \frac{nd}{mT_{max}} - b - (1-u_1)w\frac{(1-u_2)-\sigma_2 u_2 qd}{m\alpha} + \sigma_1 u_1 w\frac{(1-u_2)-\sigma_2 u_2 qd}{m\alpha}\right)^2 - 4\left(\frac{-n}{T_{max}}\right)G} \end{array}\right)$$

$$\text{so } T_1 = \frac{T_{max}}{2n}\left(H \mp \sqrt{H^2 + U}\right), \tag{12}$$

Where $H = n - \frac{nd}{mT_{max}} - b - (1-u_1)w\frac{(1-u_2)-\sigma_2 u_2 qd}{m\alpha} + \sigma_1 u_1 w\frac{(1-u_2)-\sigma_2 u_2 qd}{m\alpha}$,

and $U = \frac{4nG}{T_{max}}$

when substitute $((T_1, I_1 \& C_1)$ in equation (4) to get

$$Y_1 = \frac{(1-u_1)wC_1\,T_1 + n\,I_1\left(1 - \frac{T_1 + I_1}{T_{max}}\right) - sI_1 - \sigma_1 u_1 wC_1\,T_1}{\beta I_1} \tag{13}$$

So, the new model converge to the second equilibrium point which is as follows

$(T_1, I_1, C_1, Y_1, Z_1)$

$$= \left(\frac{T_{max}}{2n}\left(H \mp \sqrt{H^2 + U}\right), \frac{d}{m}, \frac{(1-u_2)-\sigma_2 u_2 qd}{m\alpha}, \frac{(1-u_1)wC_1\,T_1 + n\,I_1\left(1 - \frac{T_1 + I_1}{T_{max}}\right) - sI_1 - \sigma_1 u_1 wC_1\,T_1}{\beta I_1}, 0\right)$$

The third infected equilibrium state observed, when CTLs response equal to zero (i.e., $Y_2 = 0$),

solving equation (7) to get

$$C_2 = \frac{h}{\tau} \tag{14}$$

Then, substitute equation (14) in equation (5) to get

$$I_2 = \frac{\propto h + khZ_2}{\left((1 - u_2 - \sigma_2 u_2)q\tau\right)} \tag{15}$$

When substitute equation (14) and (15) in equation (5) to get

$$Z_2 = \frac{(1 - u_2 - \sigma_2 u_2)qI_2 - \propto C_2}{kC_2} \tag{16}$$

When using equation (11) obtain

$$T_2 = \frac{T_{max}}{2n}\left(n - \frac{nI_2}{T_{max}} - b - (1 - u_1)wC_2 + \sigma_1 u_1 wC_2 \pm \sqrt{\left(n - \frac{nI_2}{T_{max}} - b - (1 - u_1)wC_2 + \sigma_1 u_1 wC_2\right)^2 + 4\left(\frac{n}{T_{max}}\right)G}\right)$$

this implies that

$$T_1 = \frac{T_{max}}{2n}\left(F \mp \sqrt{F^2 + U}\right), \tag{17}$$

Where

F= $n - \frac{nI_2}{T_{max}} - b - (1 - u_1)wC_2 + \sigma_1 u_1 wC_2$ and U= $\left(\frac{4nG}{T_{max}}\right)$

So, model (3-7) converge to third equilibrium point which is

$$(T_2, I_2, C_2, Y_2, Z_2) = \left(\begin{matrix}\frac{T_{max}}{2n}\left(F \mp \sqrt{F^2 + U}\right), \frac{\propto h + khZ_2}{\left((1 - u_2 - \sigma_2 u_2)q\tau\right)}, \frac{h}{\tau}, \\ 0, \frac{(1 - u_2 - \sigma_2 u_2)qI_2 - \propto C_2}{kC_2}\end{matrix}\right)$$

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Stability of Equilibriums

In this section, we establish the conditions for which the equilibrium points will be stable or unstable. For this purpose first, we will investigate stability analysis of the disease-free equilibrium. The Jacobin matrix of the new model (3-7) is given as follows:

J(T,I,C,Y,Z)=

$$\begin{bmatrix} n\left(1-\frac{2T+I}{T_{max}}\right)-b-(1-u_1)wC+\sigma_1u_1wC\ , & \frac{-nT}{T_{max}}\ , & -(1-u_1)wT+\sigma_1u_1wT, & 0\ , & 0 \\ (1-u_1)wC-\frac{nI}{T_{max}}-\sigma_1u_1wC, & n\left(1-\frac{T+2I}{T_{max}}\right)-s-\beta Y, & (1-u_1)wT-\sigma_1u_1wT\ , -\beta I\ , & 0 & \\ 0\ , & (1-u_2-\sigma_2u_2)q\ , & -\propto -kZ, & 0, & -KC \\ 0, & mY, & 0, & mI-d, & 0 \\ 0\ , & 0\ , & 0\ , & 0\ , & \tau C-h \end{bmatrix}$$

The Jacobin matrix of the uninfected steady state will be as,

$J_0(T_0,0,0,0,0) =$

$$\begin{bmatrix} n\left(1-\frac{2T_0}{T_{max}}\right)-b, & \frac{-nT_0}{T_{max}}, -(1-u_1)wT_0+\sigma_1u_1wT_0, & 0, & 0 \\ 0, & n\left(1-\frac{T_0}{T_{max}}\right)-s\ , & (1-u_1)wT_0-\sigma_1u_1wT_0, & 0, & 0 \\ 0, & (1-u_2-\sigma_2u_2)q, & -\propto, & 0, & 0 \\ 0, & 0, & 0, & -d, & 0 \\ 0, & 0, & 0, & 0, & -h \end{bmatrix}$$

The characteristic equation about $J_0(T_0,0,0,0,0)$ is $|J_0(T_0,0,0,0,0)-\lambda I|=0$

$$\begin{vmatrix} n\left(1-\frac{2T_0}{T_{max}}\right)-b-\lambda, & \frac{-nT_0}{T_{max}}, -(1-u_1)wT_0+\sigma_1u_1wT_0, 0, 0 \\ 0, & n\left(1-\frac{T_0}{T_{max}}\right)-s-\lambda\ , & (1-u_1)wT_0-\sigma_1u_1wT_0, 0, 0 \\ 0, & (1-u_2-\sigma_2u_2)q, & -\propto-\lambda, & 0, & 0 \\ 0, & 0, & 0, & -d-\lambda, & 0 \\ 0, & 0, & 0, & 0, & -h-\lambda \end{vmatrix}=0$$

Clearly, the roots of the characteristic equation or the eigenvalue are $\lambda_1=-h$, $\lambda_2=-d$, $\lambda_3=-b+n[1-\frac{2T_0}{T_{amx}}]$, and the other two eigenvalues are determined by the quadratic equation.

$\lambda^2 + a_1\lambda + a_2 = 0$, (18)

Where $a_1 = -\left(n - \frac{nT_0}{T_{max}} - s - \propto\right)$

and $a_2 = -\propto \left(n - \frac{nT_0}{T_{max}} - s\right) - (1 - u_1 - \sigma_1 u_1)(1 - u_2 - \sigma_2 u_2) w T_0 q$.

Obviously, $a_1 > 0$ and $a_1 > 0$ if and only if $R_0 < 1$Hence all the eigenvalue have negative real parts if and only if $R_0 < 1$. So $J_0(T_0, 0,0,0,0)$ is locally asymptotically stable for $R_0 < 1$and unstable for $R_0 > 1$.

Main Results

In this section, we carried out some numerical simulations to support our analytical results by using computer simulations. We found from analytical results $I(t)$ and $C(t)$ are exponentially stable, and $\lim_{t\to\infty} I(t) = 0, \& \lim_{t\to\infty} C(t) = 0$, if $R_0 < 1$. While $I(t)$ and $C(t)$ are unstable if $R_0 > 1$. For this purpose, we will present two examples that illustrate the role of immune responses, active therapy, and stochastic parameters in stabilizing the system**.**

Example (1): Let us choose the parameter values as follows:

PARAMETER	THE VALUE
T_{max}	$1.0 * 10^7\ cells\ ml^{-1}$
G	$1.0 * 10^5 cells\ ml^{-1} day^{-1}$
n	$0.1\ day^{-1}$
b	$1.0 * 10^{-2}\ day^{-1}$
w	$4 * 10^{-7}\ ml day^{-1} virions^{-1}$
s	$0.1\ day^{-1}$
β	$6.4 * 10^{-2} day^{-1}$
q	$4.0 * 10^0\ virions\ cells^{-1} day^{-1}$
α	$5.0 * 10^0\ day^{-1}$
k	$2.0 * 1 day^{-1}$
m	$4.4 * 10^{-7} day^{-1}$
d	$1.0 * 10^{-2} day^{-1}$
τ	$1.0 * 10^{-5} day^{-1}$
h	$0.01 day^{-1}$
(u_1)	$0.9\ unit\ less$
(u_2)	$0.9\ unit\ less$
σ_1	0.01
σ_2	0.01

We first calculate the basic reproduction number, It must be less than 1, and find it as follows:

Since

$$R_0 = \frac{1}{s}\left(n\left(1-\frac{T_0}{T_{max}}\right)+\frac{(1-u_1-\sigma_1 u_1)(1-u_2-\sigma_2 u_2)wT_0 q}{\propto}\right)$$

So when you compensate the parameters in the R_0 We will find

$$R_0 = 10\left(0.1\left(1-\frac{10^6}{10^7}\right)+\frac{(0.091)(0.091)*4*10^{-7}10^6*4}{5}\right)$$

So $R_0 = 0.9264992 < 1.$

If we take the equation (4)

$$dI = \left((1-u_1)wCT + nI\left(1-\frac{T+I}{T_{max}}\right) - sI - \beta IY\right)dt - \sigma_1 u_1 wCTdW_1(t)$$

So by using Ito's formula[11] and substitution the parameter values ,we have the solution of equation (4) as $I(t) = 10^6 e^{-(0.0498)t}$. So the infected cells $I(t)$tend to zero exponentially in 70 days . The computer simulation programs Fig. 1, by using MATLAB, support these results clearly.

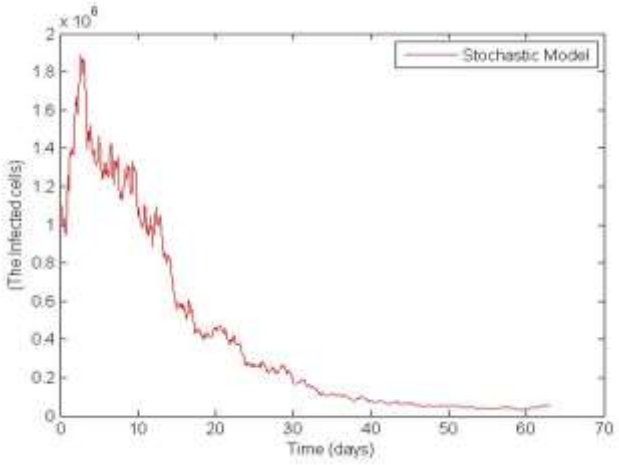

Figure 1: The infected cells goes to zero exponentially in 70 days when $R_0 < 1$,

If we take the equation (5)

$$dC = \big((1-u_2)qI - \alpha C - kCZ\big)dt - \sigma_2 u_2 qI dW_2(t)$$

Also by using Ito's formula[11] and substitution the parameter values in equation (5) ,we have the solution of equation (5) as $C(t) = 10^6 e^{-\left(\frac{3}{2}\right)t}$. So the virus particles $C(t)$ tend to zero exponentially in 70 days.

The computer simulation programs Fig. 2, by using MATLAB, support these results clearly.

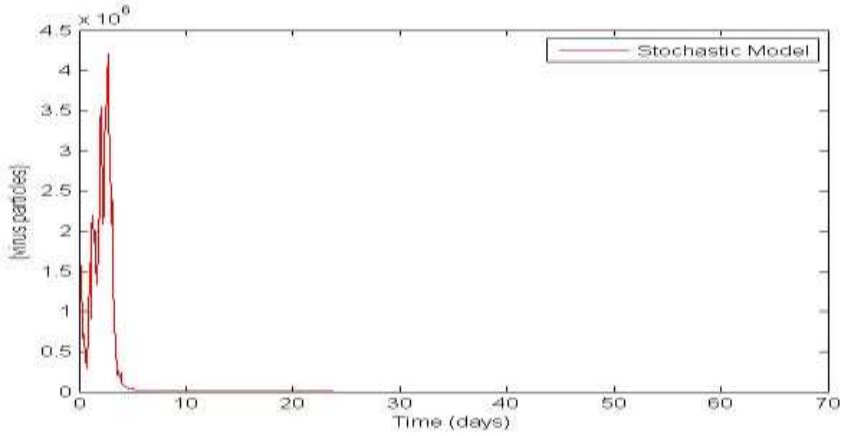

Figure 2: The virus particles goes to zero exponentially in 70 days when $R_0 < 1$,

While if we take the equation (3)

$$dT = \left(G + nT\left(1 - \frac{T+I}{T_{max}}\right) - bT - (1-u_1)wCT\right)dt + \sigma_1 u_1 wCT dW_1(t)$$

So when we solve equation (3) after substituting the parameters, we find the general solution of the stochastic differential equation is $T(t) = 10^6 e^{9999.9t}$. We note from this result that healthy cells $T(t)$ did not go to zero exponentially when $t \to \infty$. We will obtain these results by computer simulation as in Figure. 3.

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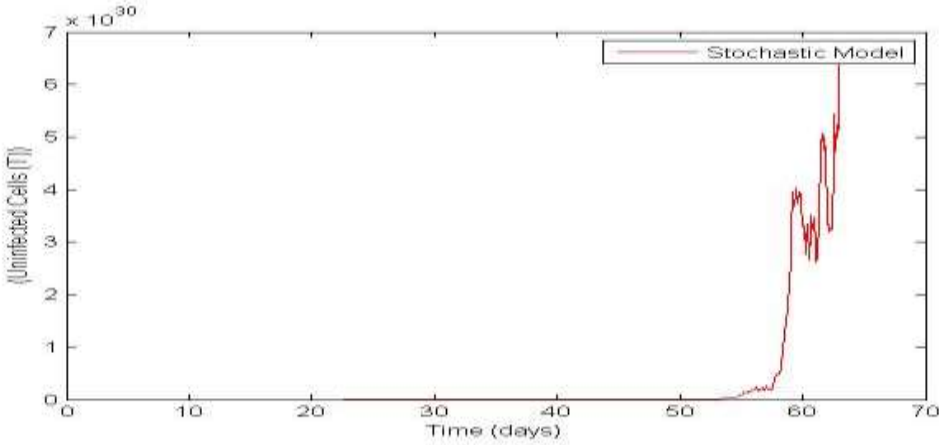

Figure 3: The uninfected cells did not go to zero exponentially in 70 days when $R_0 < 1$,

Example (2). In this example, we will discuss the patient's condition when the efficacy of treatment is poor, the immunity represented by lymphocytes (CTLs) is weak, as well as the stochastic variance (σ_1) and (σ_2) are not big sufficiently. So the parameters will be as follows:

PARAMETER	THE VALUE
T_{max}	$1.0 * 10^7\ cells\ ml^{-1}$
G	$1.0 * 10^5 cells\ ml^{-1} day^{-1}$
n	$0.1\ day^{-1}$
b	$1.0 * 10^{-2}\ day^{-1}$
w	$5 * 10^{-7}\ ml day^{-1} virions^{-1}$
s	$0.1\ day^{-1}$
β	$6.4 * 10^{-2} day^{-1}$
q	$5.0 * 10^0\ virions\ cells^{-1} day^{-1}$
α	$4.0 * 10^0\ day^{-1}$
k	$2.0 * 1 day^{-1}$
m	$4.4 * 10^{-7} day^{-1}$
d	$1.0 * 10^{-2} day^{-1}$
τ	$1.0 * 10^{-5} day^{-1}$
h	$0.01 day^{-1}$
(u_1)	$0.1\ unit\ less$
(u_2)	$0.2\ unit\ less$
σ_1	0.001
σ_2	0.001

When finding the basic reproduction number by substituting the values of the parameters in equation (8), we find that $R_0 = 5.3983 > 1$. This result means that the disease will turn into a chronic disease, and to clarify this result we take equation (4)

$$dI = \left((1-u_1)wCT + nI\left(1 - \frac{T+I}{T_{max}}\right) - sI - \beta IY \right) dt - \sigma_1 u_1 wCT dW_1(t)$$

So by using Ito's formula and substitution the parameter values, we have the solution of equation (4) is $I(t) = 10^6 e^{(3.59982)t}$. So the infected cells $I(t)$ do not tend to zero exponentially when $t \to \infty$.Will support these result by computer simulation as in Figure. 4.

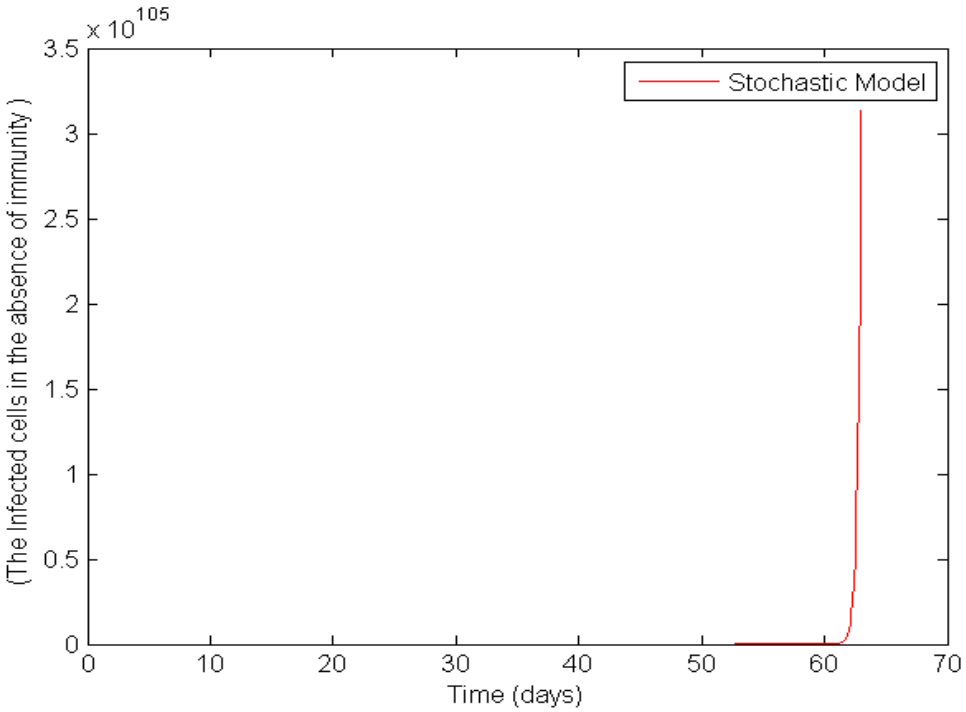

Figure 4: The infected cells not tend to zero exponentially in 70 days when $R_0 > 1$,

Also, when solving equation (5), we find that $C(t) = 10^6 e^{1996t}$. So the virus particles $C(t)$ do not tend to zero exponentially as $t \to \infty$. To illustrate the result, we take Figure. (5).

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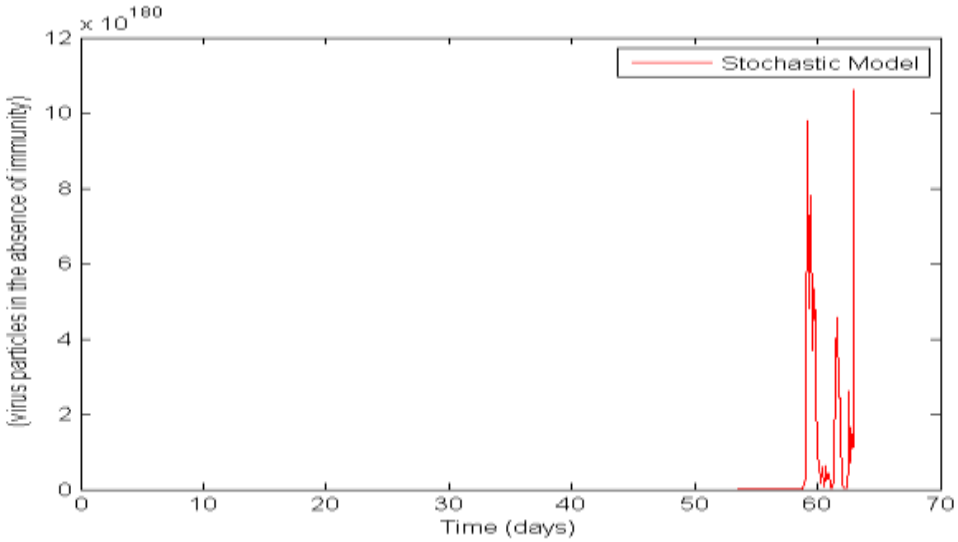

Figure 5: Computer simulation programs virus particles not tend to zero exponentially in 70 days when $R_0 > 1$,

Conclusions

The novelty of our article can be resumed as follows: the behavior of the hepatitis C virus (HCV) is studied by mixing two deterministic systems and introducing environmental stochasticity into them. Through this mathematical model, we proved that if the body has high immunity and the availability of appropriate treatment, and also the stochastic variance σ_1 and σ_2 are big sufficient. This gives $R_0 < 1$. When R_0 is less than one, this means any infected cell will transmission of infected to less than one cell and this means the liver will get rid of the virus and heal the infected person as in Figures 1, 2, and 3. On the contrary, when the immunity is weak, the treatment is ineffective, and the stochastic variance σ_1 and σ_2 are small, this gives $R_0 > 1$, and in this case, each infected cell transmits the infection to more than one healthy cell, This means that the virus will invade all liver cells and the infected person will not recover, as shown in Figures 4 and 5. Because immunity plays an essential role in maintaining the integrity of the liver from viruses. For this reason, immunity must be maintained by avoiding drug use, avoiding drinking alcohol, as well as not drinking and eating foods that have a high sugar content.

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