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STOCHASTIC DYNAMICS OF A TARGETED CHEMOTHERAPY-CANCER MODEL

ZAINAB HAYDER HASAN[1,*], IHSAN JABBAR KADHIM[2]

[1]Department of Mathematics, Faculty of Education for Women, University of Kufa, Najaf, Iraq

[2]Department of Mathematics, College of Science, University of Al-Qadisiyah, Diwaniya, Iraq

Abstract: This study aims to provide a stochastic mathematical model of the growth of cancerous tumors with targeted chemotherapy, where the cells were divided into three types of normal cells, cancer cells and responsive cells. The stability and long-terms behavior of the given system was studied. It has been shown, under certain conditions, that the tumor-free equilibrium state is almost globally stable. Accordingly, we conclude that the prescribed therapy can terminate cancer cells and thus the value of the tumor growth rate is obtained. What was also concluded during the study is that if the tumor is small, targeted chemotherapy drugs can be used in a smaller amount to eliminate the tumor from the body with less damage to other healthy cells. And vice versa. Finally, in order to verify our results, we conducted a numerical simulation.

Keywords: random dynamical systems; stability; stochastic differential equation; targeted chemotherapy; the cancerous tumor.

2020 AMS Subject Classification: 37H10.

1. INTRODUCTION

One of the causes of human death is the abnormal growth of body cells, which is called cancer. In order to find new ways to treat cancerous tumors, researchers have been interested in studying the

*Corresponding author

E-mail address: zainabh.alali@uokufa.edu.iq

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dynamics of the growth of cancerous tumors. One of the most important tools used for this purpose is mathematical models. Various of mathematical models describing the development of cancerous tumors and their interactions with other cells have been studied by many researchers [1-4]. In [5] a mathematical model based on tumor growth rate, responsive cell activity, and carrying capacity was presented. In [6], the stability of the immunological reaction to cancer was studied, with the hope of the effect of antibodies that can kill cancer cells directly. Common types of treatment for cancerous tumors are chemotherapy, targeted chemotherapy, radiation therapy, immunotherapy and surgery. It should be noted that targeted chemotherapy is a typical treatment that eliminates cancer cells deprived of any important effect on responsive healthy cells [7].

In order to investigate the efficacy of a chemical drug targeted for the treatment of cancerous tumors, we first construct a system of stochastic differential equations (SDE) that corresponds to the system of ordinary (deterministic) differential equations given in [8]. Next, we investigate the stability of the random dynamic system (RDS) generated from the assumed system of SDE's

Finally, we performed a numerical simulation of the system in order to illustrate the theoretical results.

Definition 1.1 [9, 10]

Let $(\Omega, \boldsymbol{\mathcal{F}}, \mathbb{P})$ be a probability space. The measurable action $\theta: \mathbb{R} \times \Omega \longrightarrow \Omega$ is said to be metric dynamical system (MDS) on $(\Omega, \boldsymbol{\mathcal{F}}, \mathbb{P})$ whenever θ verify $\mathbb{P}(\theta_t B) = \mathbb{P}(B)$ for every $B \in \mathcal{F}$ and $t \in \mathbb{R}$. The MDS is denoted by θ.

Definition 1.2 [9, 10]

The measurable function $\varphi: \mathbb{T} \times \Omega \times X \longrightarrow X$ is said to be co-cycle over the MDS θ if it is verify:

$$\varphi(0, \omega) = id, \varphi(t + s, \omega) = \varphi(t, \theta_s \omega) \circ \varphi(s, \omega), \quad (1)$$

where $t, s \in \mathbb{R}$ and $\omega \in \Omega$.

If, in addition, the functions $\varphi(\cdot, \omega, \cdot): \mathbb{R} \times X \longrightarrow X$ is continuous for every $\omega \in \Omega$, then the co-cycle with the MDS θ called random dynamical system (RDS) over θ.

Definition 1.3 [9, 10]

The affine RDS is a pair (θ, φ) wherever X is a Banach space and:

$$\varphi(t, \omega)x = \Phi(t, \omega)x + \psi(t, \omega) \quad (2)$$

where $\Phi(t, \omega)$ verify Eq. (1) and $\psi : \mathbb{R} \times \Omega \longrightarrow X$ is a measurable function. In case of $\psi(t, \omega) \equiv 0$, then (θ, φ) is called linear and it is written by LRDS.

Definition 1.4 [9, 10]

Let (X, d) be a metric space. The multi-valued map $M: \Omega \longrightarrow 2^X/\{\emptyset\}$ is said to be a random set if $\rho: \Omega \longrightarrow \mathbb{R}^+$, where $\rho(\omega) := dist_X(x, M(\omega))$ is measurable function for every $x \in X$. When $M(\omega)$ is closed (compact) in X for each $\omega \in \Omega$, then $M(\omega)$ is called a closed (resp. compact) random set.

Definition 1.5 [9, 10]

Consider the RDS (θ, φ). If the random variable $u: \Omega \mapsto X$ satisfy $u(\theta_t \omega) = \varphi(t, \omega)u(\omega)$ where $t \geq 0, \omega \in \Omega$ then it is called an equilibrium of (θ, φ).

Definition 1.6 [9, 10]

Consider the LRDS (θ, φ). If there is $\Omega^* \in \mathcal{F}$ with $\mathbb{P}(\Omega^*) = 1$, then the smallest number λ satisfy:

$$\lambda(\omega, x) := \lim_{t \to +\infty} \frac{1}{t} log\ \|\varphi(t, \omega)x\|, \omega \in \Omega^*, t > 0 \tag{3}$$

is called the Lyapunov exponent for (θ, φ).

2. MODEL FORMULATION

Traditional chemotherapy drugs cause side effects such as hair loss, fatigue, anemia, and others, because these drugs eliminates all types of cells at different rates. As for targeted chemotherapy, it mainly targets cancer cells so that there are few side effects. The description of this by $-kTC$, k represents the rate of attachment of chemotherapeutic drugs targeted at cancer cells. As in [7], we introduce the parameter η, in order to quantity the efficiency of targeted chemotherapy on competent and normal immune cells, so:

$$\begin{aligned} \frac{dI}{dt} &= s + \frac{\rho IT}{\sigma + T} - c_1 IT - d_1 I - a_1(1-\eta)CI, \\ \frac{dT}{dt} &= r_1 T(1 - b_1 T) - c_2 IT - c_3 TN - a_2 CT, \\ \frac{dN}{dt} &= r_2 N(1 - N) - c_4 TN - a_3(1-\eta)CN, \\ \frac{dC}{dt} &= u - d_2 C - kTC, \end{aligned} \tag{4}$$

and $I(0) = I_0 > 0, T(0) = T_0 > 0, N(0) = N_0 > 0$ and $C(0) = C_0 > 0$ are the initial conditions. $I(t)$ show the effector cell densities at time $t, T(t)$ represent tumor cells at time t, $N(t)$ represent normal cells at time t and $C(t)$ represent amount of targeted chemo drug managed at time t. The stochastic model that corresponding to Eq.(4) can be formulated as follows:

The natural death rate (d_1), intrinsic growth rate (r_1), maximum carrying capacity ($1/b_1$), growth

rate (r_2) of normal cells, and decay rate (d_2) of the targeted chemo-drug of effector cells may not all be known and may be influenced by random environmental factors. so that from Theorem 7.1 and Lemma 8.2 in [4], we have $d_1(t) \longmapsto d_1(t) + \sigma_1\dot{B}_1$, $r_1(t) \longmapsto r_1(t) + \sigma_2\dot{B}_2$, $b_1(t) \longmapsto b_1(t) + \sigma_3\dot{B}_3$, $r_2(t) \longmapsto r_2(t) + \sigma_4\dot{B}_4$ and $d_2(t) \longmapsto d_2(t) + \sigma_5\dot{B}_5$, where the exact behavior of the noise terms $\sigma_i\dot{B}_i$, (such that $B_i(t)$ represent the customary independent Brownian motions and $\sigma_i > 0$, $i = 1,2,3,4$) are unknown only their probability distribution. The functions $d_1(t), r_1(t), b_1(t), r_2(t)$, and $d_2(t)$ are assumed to be nonrandom and constants. Thus Eq. (4) becomes:

$$\begin{aligned} dI &= \left(s + \frac{\rho IT}{\sigma + T} - c_1IT - d_1I - a_1(1-\eta)CI\right)dt - \sigma_1 I dB_1 \\ dT &= [r_1T(1 - b_1T) - c_2IT - c_3TN - a_2CT]dt + \sigma_2 T dB_2 - \sigma_2 b_1 T^2 dB_2 \\ dN &= [r_2N(1-N) - c_4TN - a_3(1-\eta)CN]dt + \sigma_3 N(1-N)dB_3 \\ dC &= (u - d_2C - kTC)dt - \sigma_4 C dB_4 \end{aligned} \tag{5}$$

In the following sections the long-term behavior of Eq. (5) will be studied.

3. BIOLOGICALLY ACCEPTABLE

In this section we will show whether the solutions of Eq. (5) are realistic or not, for all parameters adopted in the model. In order to make sure that the solutions are positive and constrained, we will use the principle of random comparison [9], so we have:

$$\frac{dI}{dt} = s + \frac{\rho IT}{\sigma + T} - c_1IT - \left(d_1 + \sigma_1\dot{B}_1\right)I - a_1(1-\eta)CI \le s - \left(d_1 + \sigma_1\dot{B}_1\right)I$$

So, $dI \le d_1\left(\frac{s}{d_1} - I\right)dt + \sigma_1 I dB_1$. Now, the solution of the SDE $dI^* = d_1\left(\frac{s}{d_1} - I^*\right)dt + \sigma_1 I^* dB_1$ is provided by:

$$I^*(t) = \Phi(t)\left\{I^*(0) + s\int_0^t \Phi^{-1}(\tau)d\tau\right\},$$

where $\Phi(t) = exp\left\{-(d_1 + \frac{\sigma_1^2}{2})t + \sigma_1 B_1(t)\right\}$. Then:

$$I(t) \le I^*(0)\, exp\left\{-(d_1 + \frac{\sigma_1^2}{2})t + \sigma_1 B_1(t)\right\}$$

$$+s\int_0^t exp\left\{-\left(d_1 + \frac{\sigma_1^2}{2}\right)(t-\tau) - \sigma_1(B_1(t) - B_1(\tau))\right\}d\tau$$

Hence, $\lim\limits_{t\to\infty} \sup I(t,\omega) \le \frac{s}{\left(d_1 + \frac{\sigma_1^2}{2}\right)}$.

Equation in the right hand side models a process which naturally falls back to its equilibrium level of s.

$$\frac{dT}{dt} = \left(r_1 + \sigma_2\dot{B}_2\right)T(1 - b_1T) - c_2IT - c_3TN - a_2CT \leq \left(r_1 + \sigma_2\dot{B}_2\right)T(1 - b_1T)$$

Then $dT \leq r_1b_1T\left(\frac{1}{b_1} - T\right)dt + \sigma_2TdB_2$. Now, consider the SDE:

$$dT^* = r_1b_1T^*\left(\frac{1}{b_1} - T^*\right)dt + \sigma_2T^*dB_2$$

Then

$$T^*(t) = \frac{exp\left\{\left(r_1 - \frac{1}{2}\sigma_2^2\right)t + \sigma_2B_2(t)\right\}}{(T^*(0))^{-1} + r_1b_1\int_0^t exp\left\{\left(r_1 - \frac{1}{2}\sigma_2^2\right)\tau + \sigma_2B_2(\tau)\right\}d\tau}$$

So

$$\lim_{t\to\infty} sup\, T(t) \leq \frac{\left(r_1 - \frac{1}{2}\sigma_2^2\right)}{r_1b_1}$$

Similarly, the SDE:

$$dN = [r_2N(1 - N) - c_4TN - a_3(1 - \eta)CN]dt + \sigma_3N(1 - N)dB_3,$$

implies that $dN \leq r_2N(1 - N)dt + \sigma_3NdB_3$. Consider the SDE:

$$dN^* = r_2N^*(1 - N^*)dt + \sigma_3N^*dB_3,$$

this equation has the exact solution given by:

$$N^*(t) = \frac{exp\left\{\left(r_2 - \frac{1}{2}\sigma_3^2\right)t + \sigma_3B_3(t)\right\}}{N^{-1} + r_2\int_0^t exp\left\{\left(r_2 - \frac{1}{2}\sigma_3^2\right)\tau + \sigma_3B_3(\tau)\right\}d\tau}$$

So

$$\lim_{t\to\infty} sup\, N(t) \leq \frac{\left(r_2 - \frac{1}{2}\sigma_3^2\right)}{r_2}$$

Finally,

$$\frac{dC}{dt} = u - \left(d_2 + \sigma_5\dot{B}_5\right)C - kTC \leq u - \left(d_2 + \sigma_5\dot{B}_5\right)C$$

So, we have $dC \leq d_2\left(\frac{u}{d_2} - C\right)dt + \sigma_5CdB_5$. Now, the solution of the SDE:

$$dC^* = d_2\left(\frac{u}{d_2} - C^*\right)dt + \sigma_4C^*dB_4,$$

is given by:

$$C^*(t) = \Phi(t)\left\{C^*(0) + s\int_0^t \Phi^{-1}(\tau)d\tau\right\}$$

where:

$$(t) = exp\left\{\int_0^t \left[-d_2 - \frac{\sigma_4^2}{2}\right]d\tau + \int_0^t \sigma_4\, dB_4(\tau)\right\}$$

$$= exp\left\{-(d_4 + \frac{\sigma_4^2}{2})t + \sigma_4 B_4(t)\right\}$$

Then:

$$(t) \le C(0)\, exp\left\{-(d_2 + \frac{\sigma_4^2}{2})t + \sigma_4 B_4(t)\right\}$$

$$+u\int_0^t exp\left\{-\left(d_2 + \frac{\sigma_4^2}{2}\right)(t-\tau) - \sigma_4(B_4(t) - B_4(\tau))\right\}d\tau$$

Hence,

$$\lim_{t\to\infty} \sup C(t,\omega) \le \frac{u}{\left(d_2 + \frac{\sigma_4^2}{2}\right)}.$$

Equation in the right hand side models a process which naturally falls back to its equilibrium level of u. So, we have:

$$\Delta = \{(I,T,N,C) \in \mathbb{R}^4 : I \le \lambda_1, T \le \lambda_2, N \le \lambda_3, C \le \lambda_4\}$$

where

$$\lambda_1 := \frac{s}{\left(d_1 + \frac{\sigma_1^2}{2}\right)},\ \lambda_2 := \frac{\left(r_1 - \frac{1}{2}\sigma_2^2\right)}{r_1 b_1},\ \lambda_3 := \frac{\left(r_2 - \frac{1}{2}\sigma_3^2\right)}{r_2}, \text{ and } \lambda_4 := \frac{u}{\left(d_2 + \frac{\sigma_4^2}{2}\right)}\ .$$

We conclude from the above discussion that the domain region Δ is positive, and this indicates that the model in Eq. (2) is biologically acceptable.

4. STABILITY ANALYSIS

Here we will discuss the long-term behavior of the RDS generated by Eq. (5) and examine the random attractors.

The equation $dI^* = (-d_1 I^* + s)dt + \sigma_1 I^* dB_1$ generates the affine RDS (θ, φ^*), where φ^* is provided by:

$$\varphi^*(t,\omega)I^* = \Phi^*(t,\omega)I^* + \psi^*(t,\omega) \qquad (6)$$

where:

$$\Phi^*(t,\omega)I^* = I^* exp\left\{-\left(d_1 + \frac{\sigma_1^2}{2}\right)t + \sigma_1 B_1(t)\right\} \tag{7}$$

and

$$\psi^*(t,\omega) = s\int_0^t \exp\left\{-\left(d_1 + \frac{\sigma_1^2}{2}\right)(t-\tau) + \sigma_1\big(B_1(t) - B_1(\tau)\big)\right\} d\tau \tag{8}$$

Since the (topological) Lyapunov exponent $\lambda := -\left(d_1 + \frac{\sigma_1^2}{2}\right)$ for (θ, Φ^*) is negative, then (θ, Φ^*) is dissipative [10] inside the universe made up of all tempered R subsets. Thus from Definition 3.13 and Remark 3.14 in [11] the RDS (θ, Φ^*) is strong dissipative. From Proposition 1.9.2 and Remark 1.9.2 in [10] we can conclude that (θ, Φ^*) admits a unique equilibrium.

$$u(\omega) = s\int_{-\infty}^0 exp\left\{\left(d_1 + \frac{\sigma_1^2}{2}\right)\tau - \sigma_1 B_1(\tau)\right\} d\tau \tag{9}$$

From Proposition 1.9.3 in [10] $u(\omega)$ is exponentially stable. Now, since $dI \leq dI^*$, so u is super-equilibrium of RDS (θ, φ). Now:

$$T(t) = \frac{exp\left\{\left(r_1 - \frac{1}{2}\sigma_2^2\right)t + \sigma_2 B_2(t)\right\}}{T^{-1} + r_1 b_1 \int_0^t exp\left\{\left(r_1 - \frac{1}{2}\sigma_2^2\right)\tau + \sigma_2 B_2(\tau)\right\} d\tau} \tag{10}$$

By Proposition 6.6.1 [10], Eq. (10) induce the RDS (θ, φ) where:

$$\varphi(t,\omega)T = \begin{cases} \dfrac{exp\left\{\left(r_1 - \frac{1}{2}\sigma_2^2\right)t + \sigma_2 B_2(t)\right\}}{T^{-1} + r_1 b_1 \int_0^t exp\left\{\left(r_1 - \frac{1}{2}\sigma_2^2\right)\tau + \sigma_2 B_2(\tau)\right\} d\tau}, & T > 0 \\ 0 & T = 0 \end{cases}$$

This RDS is strictly order-preserving in $\mathbb{R}^+$. From Corollary 6.6.1 [10], we conclude that the random set $A(\omega) = [0, u(\omega)]$ is a random attractor for (θ, φ) in $\mathbb{R}^+$, where $u(\omega) \geq 0$ is a random equilibrium.

Consequently,

$$A(\omega) = \begin{cases} \{0\}, & r_1 < 0 \\ \big[0, u_{\alpha,\beta,N}(\omega)\big], & r_1 > 0 \end{cases}$$

where:

$$u_{\alpha,\beta,N}(\omega) \coloneqq r_1 b_1 \int_{-\infty}^{0} exp\{r_1\tau + \sigma_2 B_2(\tau,\omega)\}\, d\tau$$

In addition, based on Proposition 1.9.3 [10], it can be shown that there exists a $\gamma > 0$ with:

$$\lim_{t\to\infty} e^{\gamma}\left|\varphi(t,\theta_{-t}\omega)x - u_{\alpha,\beta,N}(\omega)\right| = 0, \forall\, x > 0, \omega \in \Omega \tag{11}$$

Eq. (11) induces the RDS $(\theta,\bar{\varphi})$ in $\mathbb{R}$ (which is strictly order-preserving) when $N = 2m+1$ is odd for every $m \geq 1$ (by Proposition 6.6.1 in [10]). The random set:

$$A(\omega) = \begin{cases} \{0\}, & \alpha < 0 \\ \left[-u_{\alpha,\beta,N}(\omega), u_{\alpha,\beta,N}(\omega)\right], & \alpha > 0 \end{cases}$$

is the random attractor of $(\theta,\bar{\varphi})$. In the latter case $u_{\alpha,\beta,N}(\omega)$ (resp. $-u_{\alpha,\beta,N}(\omega)$) is globally stable random equilibrium and $u_0 \equiv 0$ is an unstable random equilibrium. So, as α increases through 0, we detect a pitchfork bifurcation [9].

5. NUMERICAL RESULTS

Here, we conduct a numerical simulation so as to make our conclusions more realistic and verify the results obtained and find out how much they correspond to reality. The analogous estimation equations are:

$$I_{k+1} = I_k + \left(s + \frac{\rho I_k T_k}{\sigma + T_k} - c_1 I_k T_k - d_1 I_k - a_1(1-\eta)C_k I_k\right)\Delta t - \sigma_i I_k \sqrt{\Delta t}\xi_{k,i} - \frac{\sigma_i^2 I_k}{2}(\xi_{k,i}^2 - 1)\Delta t$$

$$T_{k+1} = T_k + [r_1 T_k(1 - b_1 T_k) - c_2 I_k T_k - c_3 T_k N_k - a_2 C_k T_k]\Delta t + \sigma_2 T_k \sqrt{\Delta t}\xi_{k,i} + \frac{\sigma_2^2 T_k}{2}(\xi_{k,i}^2 - 1)\Delta t] - \sigma_2 T_k^2 \sqrt{\Delta t}\xi_{k,i} - \frac{\sigma_2^2 T_k^2}{2}(\xi_{k,i}^2 - 1)\Delta t]$$

$$N_{k+1} = N_k + r_2 N_k(1 - N_k) - c_4 T_k N_k - a_3(1-\eta)C_k N_k + \sigma_3 N_k dB_3 - \sigma_3 N_k^2 dB_3 + \sigma_3 N_k \sqrt{\Delta t}\xi_{k,i} + \frac{\sigma_3^2 N_k}{2}(\xi_{k,i}^2 - 1)\Delta t] - \sigma_3 N_k^2 \sqrt{\Delta t}\xi_{k,i} - \frac{\sigma_3^2 N_k^2}{2}(\xi_{k,i}^2 - 1)\Delta t]$$

$$C_{k+1} = C_k + (u - d_2 C_k - k T_k C_k)\Delta t - \sigma_4 C_k \sqrt{\Delta t}\xi_{k,4} + \frac{\sigma_4^2 C_k}{2}(\xi_{k,4}^2 - 1)\Delta t$$

We used the parameter values in Table 1 as in [12, 13] in conducting all numerical simulations. Parameter units were selected arbitrarily.

TABEL 1. Values of the parameter that are used in the simulation.

Parameters	Definition	Values
s	The body has a steady population of effector cells.	0.05
ρ	Maximum number of tumor cells staffing effector cells	1
σ	Decrease in half of the spread term's capability	0.4
d_1	Effector cells' rate of natural death	0.2
r_1	Necessary tumor cell growth rate	0.4
r_2	Standard rate of cell growth	0.35
$1/b_1$	Maximal tumor cell carrying capacity	1/1.5
d_2	Target chemotherapy drug's rate of degradation	0.05
a_1	Kill rate of the targeted chemotherapy drug's effector cell	0.2
a_2	Tumor cell death rate from a targeted chemotherapy medication	0.5
a_3	Kill rate of a targeted chemotherapy medication on a normal cell	0.25
c_1	Tumor cells' growth rate in effector cells	0.2
c_2	Immune cells' contribution to the tumor cells' rate of demise	0.3
c_3	Degradation rate of tumor cells as a result of normal cells	0.2
c_4	The pace at which tumor cells destroy normal cells	0.25
η	Efficacy of the specific chemotherapy medication	0.01
k	Percentage of tumor cell adhesion to certain chemotherapy medications	0.01
u	Drug parameter	0.019 or 0.020 or 0.021

We consider three cases in order to examine the effects of drugs on responsive cells, cancer cells and normal cells. In each case we will use the initial values are: $I(0) = 0.6;\ T(0) = 0.4;\ N(0) = 0.9$ and $C(0) = 0.1$ with tumor growth rate, $r_1 = 0.4$.

- $u = 0{:}\,019, \sigma = (\sigma_1 = 0.1, \sigma_2 = 0.7, \sigma_3 = 0.2, \sigma_4 = 0.05)$ *as shown in Figure 1.*

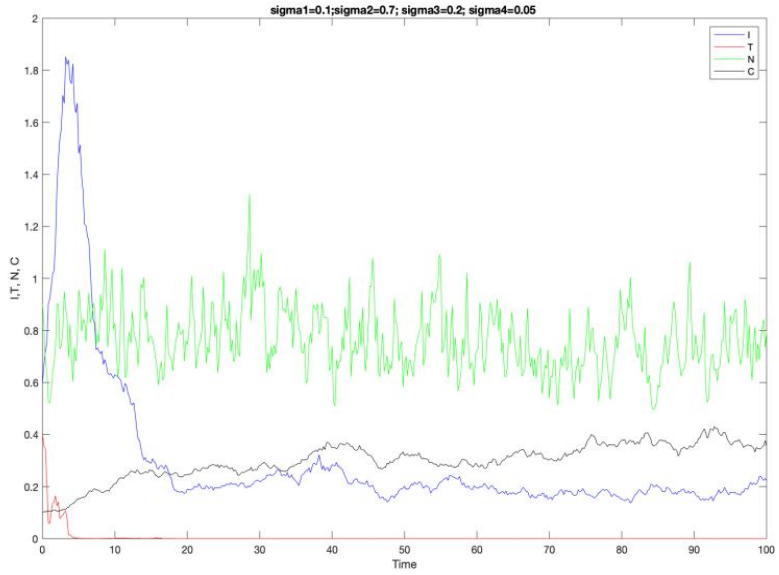

Figure 1 Numerical simulations of Eq. (4) with drug dose $u = 0.019$.

- $u = 0{:}\,020, \sigma = (\sigma_1 = 0.1, \sigma_2 = 0.7, \sigma_3 = 0.2, \sigma_4 = 0.05)$ *as shown in Figure 2.*

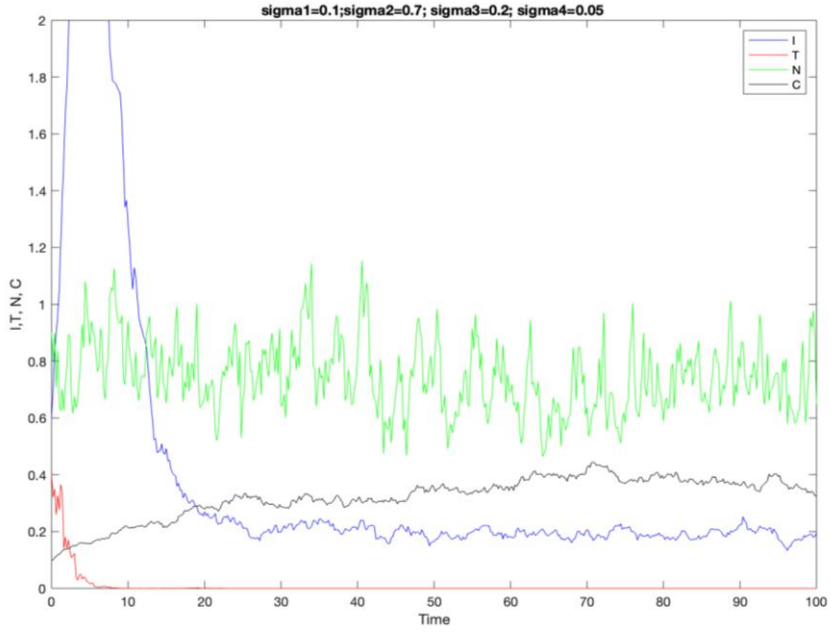

Figure 2 Numerical simulations of Eq. (4) with drug dose $u = 0.020$.

- $u = 0{:}\,021, \sigma = (\sigma_1 = 0.1, \sigma_2 = 0.7, \sigma_3 = 0.2, \sigma_4 = 0.05)$ as shown in Figure 3.

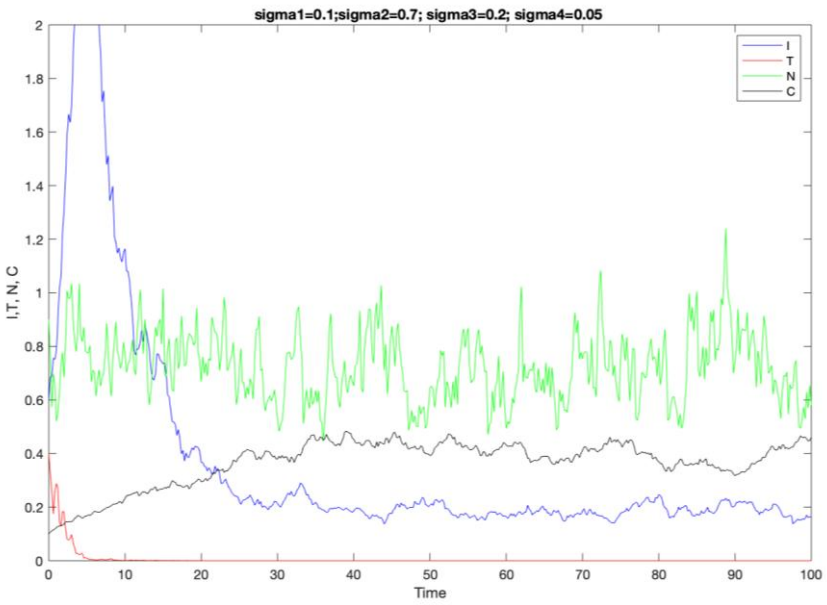

Figure 3 Numerical simulations of Eq. (4) with drug dose, $u = 0.021$.

6. DISCUSSION

As it is known that the enzymatic reactions of proteins are affected by environmental changes in the biochemical system, this is what prompted us to integrate the effects of white noise into the immunological model of tumors, in order to arrive at an accurate discussion of the fluctuations of cell dynamics.

7. CONCLUSION

We examined a mathematical model involving of a system of SDE describing the growth rate of cancerous tumors, taking into account the responsive cells, the reaction of normal cells under the influence of targeted chemotherapy. First, we showed that the model is biologically acceptable, then performed a stability analysis of the system so as to discover the dynamic behavior of the targeted chemotherapy. Finally, from numerical simulations we conclude that if the tumor size is small, then the prescribed treatment can kill cancer cells without significant effect on other healthy cells and vice versa.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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