Available online at www.qu.edu.iq/journalcm
JOURNAL OF AL-QADISIYAH FOR COMPUTER SCIENCE AND MATHEMATICS
ISSN:2521-3504(online) ISSN:2074-0204(print)

Persistence and Extinction in Stochastic Model of an Oncolytic Virotherapy by Tumor-Virus Interaction with Statistical Analysis

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ARTICLE INFO

Article history:
Received: 08/07/2025
Rrevised form: 29/07/2025
Accepted : 17/08/2025
Available online: 30/90/2025

Keywords:
Stochastic differential equation,
Statistical analysis,
The Cancerous tumor,
Targeted chemotherapy,
Oncolytic viruses.

ABSTRACT

In this paper, we consider the study of the persistence and extinction of cancer cells in the stochastic system that describes the relationship between cancer cells and the viral effect on the growth of cancer cells. The sufficient conditions for the extinction of cancer cells and for the survival of normal cells are proposed. In addition, we provide a statistical analysis of the stochastic model by studying the expected value, conditional expected value, variance, and conditional variance to solve the system. Finally, a numerical simulation of the system is introduced in order to illustrate the results.

MSC..

https://doi.org/10.29304/jqcsm.2025.17.32438

1. Introduction

Since the late 1880s, viruses have attracted considerable interest as possible agents of tumor destruction. According to the history of oncolytic viruses, physicians have seen that some cancer patients do experience remission following a viral infection [26]. Therefore, the viral therapy method has received significant attention from researchers interested in studying cancer tumor treatments. The chief problem that the researchers sought to answer is how to undermine the ability of those viruses that cause the disease so that they convert appropriate as medicines [6].

It turns out that viruses can eliminate cancer patients' tumor materials in the right circumstances. It has been established that the harm done to tumorous matter is significantly more severe than the harm done to normal host matter. The majority of these viruses were deemed unsafe for use in cancer treatment due to their pathogenicity. However, most viruses may have their pathogenicity removed without losing their oncolytic effectiveness thanks to adaptability and genetic engineering approaches [32].

Viruses used in this treatment can only reproduce specifically in cancer cells, leaving healthy normal cells mostly unharmed. The newly released viruses from the lysed cells have the potential to cause several infection cycles by infecting nearby or distant tumor cells. Much work has recently been done to comprehend the molecular mechanics

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and dynamics of oncolytic virus cytotoxicity. These initiatives offered an intriguing potential substitute treatment strategy to aid in the recovery of cancer patients. However, both the immune response and the virus-cancer interaction play a complex role in the results of virotherapy ([17, 18, 33, 36]). The majority of cancer treatments currently in use were created empirically [21]. Nonetheless, a number of mathematical models have lately been created to explain how such interactions will turn out. ([4-7,17–20, 38, 41]). In order to investigate the dynamics of virotherapy, other models and methods are being developed. ([2, 23, 33, 36, 37–41]). Many traditional mathematical models, such as reaction-diffusion models and Lotka-Volterra models, which generally assume that populations are well-mixed, have been used to build a number of mathematical models of virotherapy. However, it is evident that the spatial constituent and native communications are essential in populace expansion (see, for example, [19]), therefore this may not be the case. In general, there hasn't been any experimental evidence to support many of the modeling techniques used today. An in computer model that can spatially explicitly depict the dynamics between the virus and tumor populations has been developed in order to solve this issue [5, 6]. The qualitative characteristics of the model from [5, 6]are examined in [1], along with the qualitative of fixed points and the long-term behavior of the solution. Significant features of the model make it intriguing from a mathematical and clinical standpoint. In [28], the cells were separated into three categories: normal cells, cancer cells, and responsive cells. The stochastic mathematical model of the formation of cancerous tumors with targeted treatment was presented. The system's long-term behavior and stability were examined. It has been demonstrated that the tumor-free equilibrium state is nearly universally stable under specific circumstances.

2. Primaries

We present the following definitions [8-13] and hypotheses [15, 16] for the ease of research into the necessary circumstances of tumor cell extinction and tumor survival under the influence of microenvironment white noise.

(i) If $\lim_{t\to+\infty} y(t) = 0$, the tumor cell $y(t)$ will go extinct as soon as possible.

(ii) If $\langle y(t)\rangle_* = \lim_{t\to+\infty} \inf\langle y(t)\rangle > 0$, then the tumor cell $y(t)$ will be very persistent in the mean a.s.

(iii) $\langle y(t)\rangle = \frac{1}{t}\int_0^t y(s)\,ds, \quad \langle y(t), y(t)\rangle = \frac{1}{t}\int_0^1 y^2(s)\,ds.$

Definition 2.1 [27,29,30]: If the stochastic process $\{x(t)\}_{t_0\le t\le T}$ is admitted to the following axioms, then $x(t) \in \mathbb{R}^d$a solution of the following stochastic differential equation (SDE)

$$dX_t = f(x(t),t)dt + g(x(t),t)dB_t \qquad (1)$$

(a) The function $\{x(t)\}$ is continuous and $\mathcal{F}_t$-adapted

(b) $\{f(x(t),t)\} \in \mathcal{L}^1([t_0,T];R^d)$ and $g(x(t),t)\} \in \mathcal{L}^2([t_0,T];R^{d\times m})$;

(c) Equation (1.2) is true for all t in $[t_0,T]$ with full measure.

The solution $\{x(t)\}$ is considered unique if any other solution $\{\tilde{x}\,(t)\}$ is undifferentiated from $\{x(t)\}$.

$$\mathbb{P}\{x(t) = \tilde{x}\,(t), for\ all\ t_0 \le t \le T\} = 1$$

Remark 2.2 [27,29,30]: (a) Use $x(t;t_0,x_0)$ to represent the solution of equation (1). Then, for each s in the interval $[t_0,T]$,

$$x(t) = x(s) + \int_s^t f(x(r),r)dr + \int_s^t g(x(r),r)dB(r) \text{ on } s \le t \le T \qquad (2)$$

However, because this is a stochastic differential equation with the initial value $x(s) = x(s;t_0,x_0)$, and because the solution is represented by $x\big(t;s,x(s;t_0,x_0)\big)$, the semigroup axiom is satisfied by the solution of (1).

$$x(s;t_0,x_0) = x\big(t;s,x(s;t_0,x_0)\big), \qquad t_0 \le s \le t \le T\,. \qquad (3)$$

(b) As long as they are adjusted, the coefficients f, g can generally depend on ω. For additional information, read Gihman and Skorohod [3].

Proposition 2.3 [27,35]: Consider the SDE

$$dx = (ax + c)dt + (bx + d)d\beta,\ x(0) = x_0 \qquad (4)$$

where a, b, c, and d are constants and β represents a typical Brownian motion. The solution of (4) is provided by

$$dx = \psi(t)(x_0 + (c - bd)\int_0^1 \frac{1}{\psi(s)}ds + d\int_0^1 \frac{1}{\psi(s)}d\beta(s)) \qquad (5)$$

where

$$\psi(t) = \exp\left(\left(a - \tfrac{1}{2}b^2\right)t + b\beta(t)\right) \qquad (6)$$

Because they may be used to describe strictly positive processes, scalar linear SDEs with multiplicative noise are extremely common in financial applications. The Black-Scholes model is an illustration of one of these SDEs.

Theorem 2.4 [29,30]The solution $X(t)$ of the nonhomogeneous linear stochastic differential equation

$$dX(t) = [f_1(t) + f_2(t)X(t)]dt + [g_1(t) + g_2(t)X(t)]dW(t) \qquad (7)$$

can be written

$$X(t) = X_0(t)\left\{X(t) + \int_0^t X_0^{-1}(s)[f_1(s) - g_1(s)g_2(s)]\,ds + \int_0^t X_0^{-1}(s)g_1(s)\,dW(s)\right\} \quad (8)$$

were

$$X_0(t) = \exp\left\{\int_0^t \left[f_2(s) - \frac{1}{2}g_2^2(s)\right]ds + \int_0^t g_2(s)\,dW(s)\right\}.$$

Proposition 2.5 [29,30] Consider the SDE

$$dX_t = rX_t(K - X_t)dt + \sigma X_t dB_t\ ; X_t = x > 0. \qquad (9)$$

(a) The solution to the SDE is provided by

$$X_t = \frac{\exp\left\{\left(rK - \frac{1}{2}\sigma^2\right)t + \sigma B_t\right\}}{x^{-1} + r\int_0^t \exp\left\{\left(rK - \frac{1}{2}\sigma^2\right)s + \sigma B_s\right\}ds};\ t \ge 0. \qquad (10)$$

(b) The conditional expectation and the conditional variance of the solution X_t are given by

$$E[x(t+s)/x(t)] = K + e^{-rs}(x(t) - K) \qquad (11)$$

and

$$Var[x(t+s)/x(t)] = \frac{\sigma^2 x(t)}{2r}(1 - e^{-2rs}) \qquad (12)$$

All types of cells are killed by conventional chemotherapy medications at varying rates, and this can have serious side effects like anemia, exhaustion, hair loss, and more. Targeted chemotherapy, on the other hand, focuses primarily on cancer cells in order to minimize adverse effects.

3. Model Formulation

This section focuses to study the deterministic and stochastic model of our main problem.

3.1 Deterministic Model

The model being examined is a traditional three-species Lotka-Volterra system. These systems have been crucial in simulating interspecies competition, which has a big influence on research into various opposition representations in medicine ecology and biology. For example, see [21, 22, 32]. Three different cell types are involved in our concept: normal cells denoted as x, cancer cells y, and infected cancer cells z.

The relationship between tumor growth and viral infection of tumor cells is described by this mean-field model, which is based on predator-prey interactions.

Table1: Parameters Description, ref. [5]

Parameter	Description	Value	Unit
r	Proliferation of normal cells	0.5	$1/h$ cell
a	Death rate of normal population	0.2	$1/h$ cell
s	Proliferation of the uninfected cells	1.0	$mm^3\ h/$ cell
b	Death rate of uninfected population	0.1	$1/h$ cell
c	Proliferation of the infected cells	1.2	$mm^3\ h/$ cell
d	Death rate of the infected cells	0.1	$1/h$ cell
σ_1		0.1	Estimate
σ_2		0.7	Estimate
σ_3		0.2	Estimate

This rivalry can have a geographical impact on normal cells, even though the important communication is mostly among infected and uninfected cells. As a result, the fundamental dynamics of such an interaction can be captured

by the three model compartments. The following situation is used to formulate the model: The virotherapy in question can be modeled using a net and knots filled with the three different kinds of cells in addition to empty ones. Although infested cells may lone assault as well as inhabit a knot occupied via a cancer cell since the virus is designed to bout individual cancer cells and travels from cell to cell, while a cancer or normal cell multiplies, the recently formed cell must inhabit a near void knot ([31,34]).

Assuming that the growth and death rates of the three cell types can be modified, as well as that viral infection parameters can be set, the model assumes that the virus arrival times vary but follow the Poisson process with time to the next event being exponentially distributed ([5, 6]).

The model, described above, is governed by the following system of differential equations, where all of the parameters are nonnegative:

$$\begin{cases} \frac{dx}{dt} = & rx\,(1 - x - y - z) - ax \\ \frac{dy}{dt} = & sy\,(1 - x - y - z) - by - cyz \\ \frac{dz}{dt} = & cyz - \delta z \end{cases} \qquad (13)$$

and the initial conditions are: $x(0) = x_0 > 0$ ، $y(0) = y_0 > 0$ ،and $z(0) = z_0 > 0$:

where a, b, and d stand for the corresponding population's death rates, and r for proliferation. The model was fitted to data from in vitro investigations and makes the assumption that mass action kinetics occur ([5, 6]).

3.2 Stochastic Model

The stochastic model that corresponding to (13) can be formulated as follows:

It may happen that the effector cells' natural death rate (d_1), intrinsic growth rate of tumor cells (r_1) , maximum carrying capacity of tumor cells $(1/b_1)$, normal cells' growth rate (r_2) and decay rate of targeted chemo-drug (d_2) are not completely known but subject to some random environmental effects, so that

$$r(t) \longmapsto r(t) + \sigma_1 \dot{W}_1,\ s(t) \longmapsto s(t) + \sigma_2 \dot{W}_2 \text{ , and } \delta(t) \longmapsto \delta(t) - \sigma_3 \dot{W}_3,$$

where the exact behavior of the noise terms $\sigma_i \dot{W}_i$,(such that $B_i(t)$ represent the customary independent Brownian motions and $\sigma_i > 0, i = 1,2,3)$ are unknown only their probability distribution. The functions $r(t), s(t)$, and $\delta(t)$, are assumed to be nonrandom and constants. Thus the system (13) becomes

$$\begin{cases} dx = & [rx\,(1-x) - rx(y+z) - ax]dt + [\sigma_1 x\,(1-x) - \sigma_1 x(y+z)]dW_1 \\ dy = & [sy\,(1-y) - sy(x+z) - (by + cyz)]dt + [\sigma_2 y\,(1-y) - \sigma_2 y(x+z)]\,dW_2 \\ dz = & (cy - \delta)zdt + \sigma_3 z dW_3 \end{cases}$$

(14)

4. Persistence and Extinction

In the study of long-term population behavior, Communities may behave differently depending on the level of noise y, either extinction or persistence. So in this item we will assume the persistence and extinction of $y(t)$.

Theorem 4.1: The densities of tumor cells $y(t)$ *in* (14) **,** *satisfied:*

$$\lim_{t\to+\infty} \sup \frac{1}{t} \ln y(t) \le 0 \quad \text{almost surely,} \qquad (15)$$

with positive initial value $y(0)$.

Proof: By the second equation of (14) and the Itô's formula, we get

$$d \ln y(t) = [sy\,(1\ -\ y\,) - sy(\,x + \,z) - (\,by + \,cyz)]dt + [\sigma\_2\, y\,(1\ -\ y\,) - \sigma\_2\, y(\,x + \,z)]\, dW\_2$$

$$\le [sy\,(1\ -\ y\,)]dt + \sigma_2 dW_2 \qquad (16)$$

Construct a comparison system:

$$d \ln \bar{y} = \left[s\,(1\ -\ \bar{y}\,) - \frac{\sigma_2^2}{2}\right] dt + \sigma_2 dW_2,\ \bar{y}_0 = \bar{y}(0).$$

Define $V_1 = e^t \ln \bar{y}$. Applying Itô's formula, it is obtained that

$$dV_1 = d(e^t \ln \bar{y}) = e^t d \ln \bar{y} + \ln \bar{y}\, d\, e^t$$

$$= e^t \left[\ln \bar{y} + s(1 - \bar{y}) - \frac{\sigma_2^2}{2}\right] dt + e^t \sigma_2 dW_1(t)$$

Now, $dV_1 = \ LV_1\, dt \ + \ e^t \sigma_2 dW_2(t),$ where $LV_1 = e^t \left[\ln \bar{y} + s(1 - \bar{y}) - \frac{\sigma_2^2}{2}\right]$.

Integrating from 0 to t, we can get that

$$e^t \ln \bar{y}\,(t) - \ln \bar{y}_0 = \int_0^t e^\tau \left[\ln \bar{y}\,(\tau) + s(1 - \bar{y}(\tau)) - \frac{\sigma_2^2}{2}\right] d\tau$$

$$+ \int_0^t e^\tau \sigma_1\, dW_2(\tau).$$

Set $M_1(t) \coloneqq \int_0^t e^\tau \sigma_2\, dB_2(\tau)$, then $\langle M_1(t), M_1(t)\rangle = \int_0^t e^{2\tau} \sigma_2^2\, d\tau$ (the quadratic variation) .So , for any $\bar{y}_0$, $c_1\ c_2 > 0$, we have

$$\mathbb{P}\left\{\sup_{0\le t\le \bar{y}_0} \left[M_1(t) - \frac{c_1}{2}\langle M_1(t), M_1(t)\rangle\right] > c_2\right\} \le e^{-c_1 c_2} \qquad (17)$$

(This follows from the exponential martingale inequality [29,30]). Using the analogous technique as Zhu *et al.* [22], put $\bar{y}_0 = \lambda_0 v, c_1 = \varepsilon e^{-\lambda_0 v}$,

$c_2 = \frac{\theta e^{\lambda_0 v} \ln \lambda_0}{\varepsilon}$, where $\lambda_0 \in \mathbb{N}$, $0 < \varepsilon < 1, \theta > 1, v > 0$. Hence,

$$\mathbb{P}\left\{\sup_{0\le t\le\lambda_0 v}\left[M_1(t)-\frac{\varepsilon e^{-\lambda_0 v}}{2}\langle M_1(t),M_1(t)\rangle\right]>\frac{\theta e^{\lambda_0 v}\ln\lambda_0}{\varepsilon}\right\}\le\lambda_0^{-\theta}$$

By using Borel–Cantalli Lemma, we arise

$$M_1(t)\le\frac{\varepsilon e^{-\lambda_0 v}}{2}\langle M_1(t),M_1(t)\rangle+\frac{\theta e^{\lambda_0 v}\ln\lambda_0}{\varepsilon},\ 0\le t\le\lambda_0 v.$$

Choose $\Omega_0=\bigcap_{i=1}^{2}\Omega_i$ for some $\Omega_i\subset\Omega$. Then $\mathbb{P}(\Omega_0)=1$. Define

$$\lambda_0(\varpi)=\max\{\lambda_0(\varpi),i=1,2,\dots,n\},\forall\varpi\in\Omega_0\,.$$

Hence,

$$\sum_{i=1}^{n}M_1(t)\le\frac{\varepsilon e^{-\lambda_0 v}}{2}\langle M_1(t),M_1(t)\rangle+\frac{\theta e^{\lambda_0 v}\ln\lambda_0}{\varepsilon},\ 0\le t\le\lambda_0 v.$$

satisfies. So, if $0\le t\le\lambda_0 v$, then

$$e^t\ln\bar{y}\,(t)-\ln\bar{y}_0\le\int_0^t e^{\tau}\left[\ln\bar{y}\,(\tau)+s(1-\bar{y}(\tau))+\frac{\sigma_2^2}{2}(\varepsilon e^{s-\lambda_0 v}-1)\right]d\tau$$

$$+\frac{\theta e^{\lambda_0 v}\ln\lambda_0}{\varepsilon}$$

Hence,

$$\ln\bar{y}\,(s)+s(1-\bar{y}(\tau))+\frac{\sigma_2^2}{2}(\varepsilon e^{s-\lambda_0 v}-1)$$

has the supremum for all $t\in[0,\lambda_0 v]$. That is , there is M_1 such that

$$\ln\bar{y}\,(s)+s(1-\bar{y}(\tau))+\frac{\sigma_2^2}{2}(\varepsilon e^{s-\lambda_0 v}-1)\le M_1.$$

For any $(\lambda_0-1)v\le t\le\lambda_0 v$ with $\lambda_0=\lambda_0(\varpi)$,

$$e^t\ln\bar{y}\,(t)-\ln\bar{y}_0\le M_1(e^t-1)+\frac{\theta e^{\lambda_0 v}\ln\lambda_0}{\varepsilon}$$

Then

$$\frac{\ln\bar{y}\,(t)}{\ln t}\le\frac{\ln\bar{y}_0}{e^t\ln t}+\frac{M_1(1-e^{-t})}{\ln t}+\frac{\theta e^{\lambda_0 v}\ln\lambda_0}{\varepsilon}$$

Then

$$\limsup_{t\to\infty}\frac{\ln\bar{y}(t)}{\ln t}\le\frac{\theta e^{v}}{\varepsilon}.$$

Thus, by setting $\theta\uparrow 1,\varepsilon\uparrow 1$ and $\theta\uparrow 1$, t follows that $\limsup_{t\to\infty}\frac{\ln\bar{y}(t)}{\ln t}\le 1$ a.s..

Corollary 4.2: According to sumption of Theorem 3.1, we have

$$\limsup_{t\to\infty} \frac{1}{t} \ln\ y(t)\ \leq\ 0\ \text{almust surly.}$$

Proof: In view of Theorem 3.1,

$$\limsup_{t\to\infty} \frac{1}{t} \ln y(t) = \limsup_{t\to\infty} \frac{1}{\ln t} \ln\ y(t) \limsup_{t\to\infty} \frac{1}{t} \ln\ t$$

$$\leq \limsup_{t\to\infty} \frac{1}{t} \ln\ t.$$

Since $\limsup_{t\to\infty} \frac{1}{t} \ln\ t = 0$, then $\limsup_{t\to\infty} \frac{1}{t} \ln y(t) \leq 0$.

Theorem 4.3: Consider *the* tumor cell densities $y(t)$ *in* (1.1)*,*

(i) if $s - \frac{\sigma_1^2}{2} < 0$, *then* the tumor cell densities $y(t)$ *will lean towards to extinct a.s..*

(ii) if $s - \frac{\sigma_1^2}{2} > 0$, *then* the tumor cell densities $y(t)$ *is weakly persistent in the mean almost surly.*

Proof: (i) According to

$$dy \leq sy(1-y)dt + \sigma_2 y dW_2$$

we construction a comparison stochastic system [5]:

$$d\bar{y} = \bar{y}(s - s\bar{y})dt + \sigma_2 \bar{y} dW_2(t)\ ,\ \bar{y}_0 = \bar{y}(0). \qquad (18)$$

The Itô's formula has given us

$$d \ln y = \left(s - s\bar{y} - \frac{\sigma_1^2}{2}\right) dt + \sigma_2 dW_2(t).$$

By performing the integration from 0 to t for the two sides of the equation above, yields

$$\ln \bar{y}\ (t) - \ln \bar{y}_0 = \int_0^t \left[s - s\bar{y}(\tau) - \frac{\sigma_2^2}{2}\right] d\tau + \int_0^t \sigma_2\ dw_2(\tau)$$

$$= \int_0^t \left[s - s\bar{y}(\tau) - \frac{\sigma_2^2}{2}\right] d\tau + \int_0^t \sigma_2\ dW_2(\tau),$$

Thus

$$\bar{y}(t) = \bar{y}_0 \exp\left\{\int_0^t \left[s - s\bar{y}(\tau) - \frac{\sigma_2^2}{2}\right] d\tau + M_1(t)\right\}$$

where $M_1(t) = \int_0^t \sigma_2\ dB_2(\tau)$. According to strong law of large numbers, yields

$$\lim_{t\to+\infty} \sup \frac{M_1(t)}{t} = 0.$$

Thus,

$$\limsup_{t\to\infty} \frac{1}{t} \ln\ \bar{y}(t) \le s - \frac{\sigma_2^2}{2} < 0 \text{ a.s.}.$$

Due to the comparison theorem for SDEs, we get

$$\limsup_{t\to\infty} \frac{1}{t} \ln\ \bar{y}(t) < 0, \text{ then } \lim_{t\to+\infty} y(t) = 0.$$

(ii) Demonstrating the existence of a constant $\alpha > 0$ such that $\langle y(t)\rangle^* \ge \alpha > 0$ is satisfied by any solution of (18).

On the other hand, assume that the outcome is inaccurate. Select ε_1 be arbitrarily tiny in order that

$$-d_2 - \frac{\sigma_2^2}{2} + k\varepsilon_1 < 0\ ,\ s - \frac{\sigma_2^2}{2} - s\varepsilon_1 > 0.$$

Then the solution $(\bar{y}(t), \bar{z}(t))$ exists for any $\varepsilon_1 > 0$ $(\bar{y}(t), \bar{z}(t))$ such that $\mathbb{P}\{\langle \bar{y}(t)\rangle^* < \varepsilon_1\} > 0$.

Consequently,

$$d \ln \bar{z} \le \left(k\bar{y} - d_2 - \frac{\sigma_2^2}{2}\right) dt + \sigma_2 dW_2(t).$$

By performing the integration from 0 to t for the two sides of the equation above and then divide by t, yields

$$\frac{1}{t}(\ln \bar{z}(t) - \ln \bar{z}(0)) \le \frac{1}{t}\int_0^t \left(-d_2 - \frac{\sigma_2^2}{2}\right) d\tau + \frac{1}{t}\int_0^t k\bar{y}(\tau)\, d\tau + \frac{1}{t}\int_0^t \sigma_2\, dW_2(\tau)$$

$$= -d_2 - \frac{\sigma_2^2}{2} + k\frac{1}{t}\int_0^t \bar{y}(\tau)\, d\tau + \frac{M_2(t)}{t}, \qquad (19)$$

where $M_2(t) = \int_0^t \sigma_2\, dW_2(\tau)$. According to strong law of large numbers, $\limsup_{t\to+\infty} \frac{M_2(t)}{t} = 0$. Hence,

$$\lim_{t\to+\infty} \sup \frac{1}{t} \ln \bar{z}(t) \le -d_2 - \frac{\sigma_2^2}{2} + k\varepsilon_1 < 0.$$

So $\lim_{t\to+\infty} \bar{z}(t) = 0$. Furthermore,

$$d \ln \bar{y}(t) = \left[s - \bar{y}(t) - \frac{\sigma_1^2}{2}\right] dt + \sigma_2 dW_2(t)$$

Consequently,

$$\frac{1}{t}[\ln \bar{y}(t) - \ln \bar{y}(0)] = \frac{1}{t}\int_0^t \left(s - \frac{\sigma_1^2}{2}\right) d\tau - \frac{1}{t}\int_0^t s\bar{y}(\tau)\, d\tau + \frac{M_2(t)}{t}$$

$$= s - \frac{\sigma_1^2}{2} - \frac{1}{t}\int_0^t s\bar{y}(\tau)\, d\tau + \frac{M_2(t)}{t}.$$

By the strong law of large numbers, $\limsup_{t\to+\infty} \frac{M_2(t)}{t} = 0$ is certified. Hence,

$$\limsup_{t\to+\infty} \frac{1}{t}\ln \bar{y}(t) = s - \frac{\sigma_2^2}{2} + s\varepsilon_1 > 0.$$

This resulted in a conflict with Theorem 3.1. Hence, $\langle y(t)\rangle^* > 0$.

Theorem 4.4: *If* $(s - \frac{\sigma_2^2}{2}) < c_3(\frac{\sigma_1^2}{2} - r)$, *then the density of normal cells* $x(t)$ *extinct a.s..*

Proof: If It is clear from the comments that $\langle x(t)\rangle^* < 0$, when $r - \frac{\sigma_1^2}{2} \le 0$. According to the same method as inequality (19), we get

$$d\ln \bar{x}(t) = \left[r - \bar{x}(t) - \frac{\sigma_1^2}{2}\right]dt + \sigma_1 dW_1(t)$$

Consequently,

$$\frac{1}{t}[\ln \bar{x}(t) - \ln \bar{x}(0)] = \frac{1}{t}\int_0^t \left(r - \frac{\sigma_1^2}{2}\right)ds - \frac{1}{t}\int_0^t \bar{x}(s)\,ds + \frac{M_3(t)}{t}$$

$$= r - \frac{\sigma_1^2}{2} - \frac{1}{t}\int_0^t \bar{x}(s)\,ds + \frac{M_3(t)}{t}.$$

By the strong law of large numbers, $\limsup_{t\to+\infty} \frac{M_3(t)}{t} = 0$ is certified. Hence,

$$\limsup_{t\to+\infty} \frac{1}{t}\ln \bar{x}(t) = r - \frac{\sigma_1^2}{2} \le 0.$$

So $\lim_{t\to\infty} \bar{x}(t) = 0$. Then $\lim_{t\to\infty} x(t) = 0$. Furthermore, if $r - \frac{\sigma_1^2}{2} > 0$, there exists $\delta > 0$ for all $\varepsilon_2 > 0$ such that $\frac{M_2(t)}{t} \le \varepsilon_2$ for $t > \delta$. Then

$$\ln \bar{x}(t) - \ln \bar{x}(0) \le \int_0^t \left(r - \frac{\sigma_1^2}{2}\right)ds - \int_0^t \bar{x}(s)\,ds + \frac{M_2(t)}{t}$$

$$\le \left(r - \frac{\sigma_1^2}{2} + \varepsilon_2\right)t - \int_0^t \bar{x}(s)\,ds$$

It follows that $\langle \bar{x}(t)\rangle^* \le \left(r - \frac{\sigma_1^2}{2} + \varepsilon_2\right)$. Let $\varepsilon_2 \longrightarrow 0$, then

$\langle \bar{x}(t)\rangle^* \le \left(r - \frac{\sigma_1^2}{2}\right)$. Therefore

$$\limsup_{t\to\infty} \frac{1}{t}\ln\, y(t) \le s - \frac{\sigma_2^2}{2} + c_3\langle \bar{x}(t)\rangle^*$$

$$\leq (s - \tfrac{\sigma_2^2}{2}) + c_3\left(r - \tfrac{\sigma_1^2}{2}\right)$$

Then $\limsup_{t\to+\infty} \frac{1}{t}\ln \bar{x}(t) < 0$. As a result $\lim_{t\to\infty} \bar{x}(t) = 0$.

5. Statistical Analysis

This section will cover our study of the statistical analysis for the stochastic differential equations that given in (14). As in **[**29,30**]**, the expected of the densities of effector cells is**:**

$$E[z(t,\omega)] \leq \tfrac{s}{d_1} - \left(\tfrac{s}{d_1} - z(0)\right)e^{-d_1 t} \quad \text{then} \quad \lim_{t\to\infty} E[z(t,\omega)] \leq \tfrac{s}{d_1}.$$

and the variance is

$$Var\,[z(t,\omega)] \leq \tfrac{\sigma_3^2}{2d_1}(1 - e^{-2d_1 t}).$$

Mean and variance that are asymptotically conditional. The mean and variance of the process z, conditioned on the starting value $z(0)$, exist since $z(0)$ has a finite second moment.

$\mathbb{E}[z(t)/z(0)] = z(0)e^{-d_1 t} + \frac{s}{d_1}(1 - e^{-d_1 t})$ and

$$Var[z(t)/z(0)] = \begin{cases} e^{-d_1 t}2s\left[tz(0) - \frac{1}{d_1}z(0) - t\frac{s}{d_1}\right] + z(0)^2 - e^{-2d_1 t}\left(z(0) - \frac{s}{d_1}\right)^2 + \left(\frac{s}{d_1}\right)^2, & \frac{\sigma_3^2}{d_1} = 1 \\ 4e^{-d_1 t}\frac{s}{d_1}\left[\frac{s}{d_1} - z(0)\right] - e^{-2d_1 t}\left(z(0) - \frac{s}{d_1}\right)^2 + 2\frac{s^2}{d_1}t - 3\left(\frac{s}{d_1}\right)^2 + 2\frac{s}{d_1}z(0) + z(0)^2, & \frac{\sigma_3^2}{d_1} = 2 \\ \frac{\left(\frac{s}{d_1}\right)^2\frac{\sigma_3^2}{d_1}}{2 - \frac{\sigma_3^2}{d_1}} + 2\frac{\sigma_3^2}{d_1}\frac{(z(0) - \frac{s}{d_1})\frac{s}{d_1}}{1 - \frac{\sigma_3^2}{d_1}}e^{-d_1 t} - e^{-d_1 t}\left(z(0) - \frac{s}{d_1}\right)^2 & \\ \quad + e^{(\sigma_3^2 - 2d_1)t}\left[z(0)^2 - \frac{2z(0)\frac{s}{d_1}}{1 - \frac{\sigma_3^2}{d_1}} + \frac{2\left(\frac{s}{d_1}\right)^2}{(2 - \frac{\sigma_3^2}{d_1})(1 - \frac{\sigma_3^2}{d_1})}\right], & \text{otherwise} \end{cases}$$

Since $\frac{1}{d_1} > 0$, from ($\mathbb{E}[z(t)/z(0)]$). Thus, the asymptotic mean of z must exist. It is provided by

$$\mathbb{E}[z_\infty(t)] \coloneqq \lim_{t\to\infty} \mathbb{E}[z(t)/z(0)] = \tfrac{s}{d_1}.$$

From ($Var[z(t)/z(0)]$), it implies that the asymptotic variance of z exists under the constraint $\frac{\sigma_3^2}{d_1} < 2$. It is provided by

$$Var[z_\infty(t)] \coloneqq \lim_{t\to\infty} Var[z(t)/z(0)] = \frac{\left(\frac{s}{d_1}\right)^2\frac{\sigma_3^2}{d_1}}{2 - \frac{\sigma_3^2}{d_1}} = \frac{\left(\frac{s}{d_1}\right)^2}{\frac{2d_1}{\sigma_3^2} - 1}$$

Similarly , $E[x(t)] \leq \frac{1}{1+e^{-r_2 t}}$ so $\lim_{t\to\infty} E[x(t)] \leq 1$,

$$E[x(t+s)/x(t)] = 1 + e^{-r_2 s}(x(t) - 1)$$

and

$$Var[x(t+s)/x(t)] = \frac{\sigma_3^2}{2}x(t)(1-e^{-2r_r s}).$$

Also, $E[z(t)] \le e^{-d_2 t}\left(z(0)-\frac{\alpha}{d_2}\right)+\frac{\alpha}{d_2}$ and so $\lim_{t\to\infty} E[z(t)] \le \frac{\alpha}{d_2}$.

6. Numerical Similation of The Stochastic Model

Here, we do a numerical simulation to validate the findings, assess their realism, and strengthen the validity of our conclusions. The analogous estimation equations are:

$$x_{k+1} = x_k + [rx_k(1-x_k) - rx_k(y_k+z_k) - ax_k]\Delta t$$

$$+[x_k(1-x_k) - x_k(y_k+z_k)]\left[\sigma_1\sqrt{\Delta t}\xi_{k,1} + \frac{\sigma_1^2}{2}(\xi_{k,1}^2-1)\Delta t\right]$$

$$y_{k+1} = y_k + [sy_k(1-y_k) - sy_k(x_k+z_k) - (by_k + cy_k z_k)]\Delta t$$

$$+[\sigma_2 y_k(1-y_k) - \sigma_2 y_k(x_k+z_k)]\left[\sigma_2\sqrt{\Delta t}\xi_{k,2} + \frac{\sigma_i^2}{2}(\xi_{k,2}^2-1)\Delta t\right]$$

$$z_{k+1} = z_k + (cy_k - \delta)z_k\Delta t + z_k\left[\sigma_3\sqrt{\Delta t}\xi_{k,3} + \frac{\sigma_3^2}{2}(\xi_{k,3}^2-1)\Delta t\right].$$

The values of the parameters shown Table 1 are used as considered in the study [4] in conducting all numerical simulations. Parameter units were selected arbitrarily.

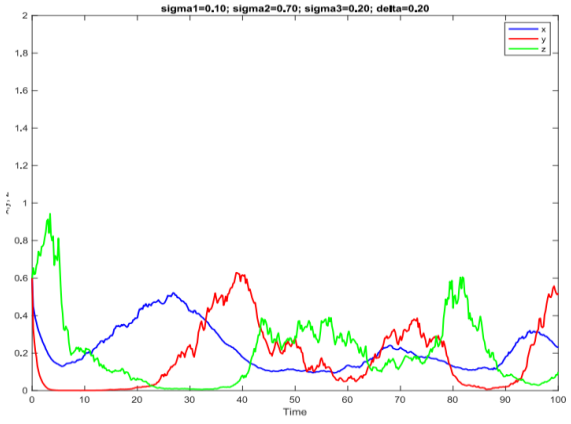

Figure 1: Dynamics of the model with initial values $x_0 = 0.6$, $y_0 = 0.6$, and $z_0 = 0.6$.

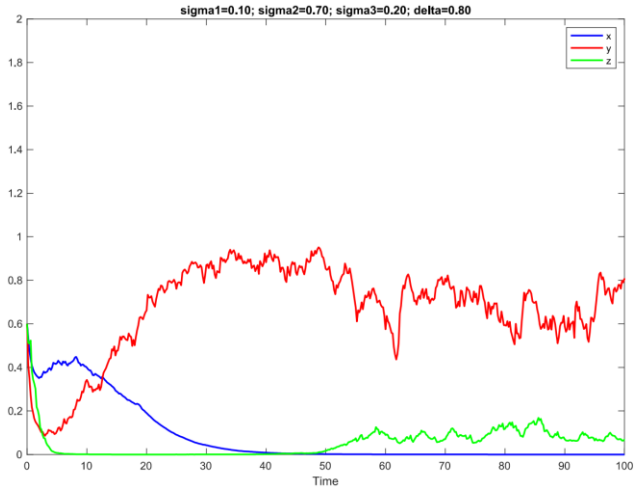

Figure 2: Dynamics of the model with initial values $x_0 = 0.5, y_0 = 0.5$, and $z_0 = 0.6$.

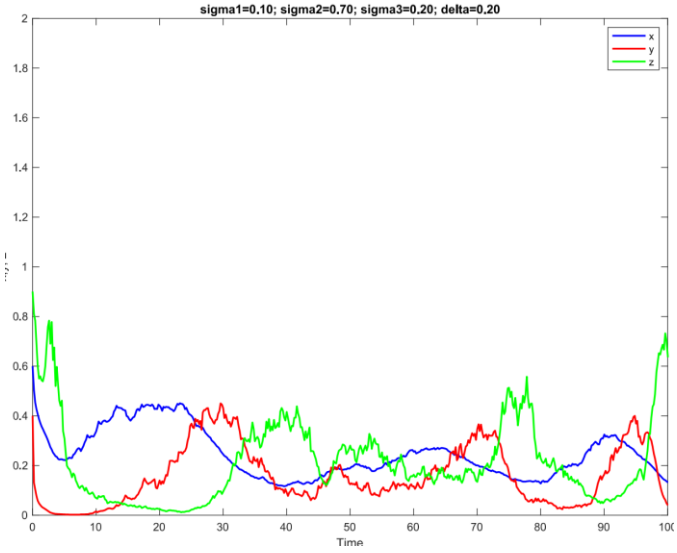

Figure 3: Dynamics of the model with initial values $x_0 = 0.6, y_0 = 0.4$, and $z_0 = 0.09$.

7. Discussion

In this paper, we discussed the persistence and extinction of cancer cells in the human body under the influence of targeted chemotherapy, which is considered one of the best methods of treating tumors, because it targets cancer cells without affecting other healthy cells. Where we used the mathematical model represented by the system of coincidental differential equations (14) as in [15].

8. Conclusion

There are a number of conclusions that were reached during the research. We have shown that the stochastic model provides more accurate results than the corresponding deterministic model. Through the stochastic model, we were able to calculate the expected value of the stochastic system solution, which allows us to predict tumor behavior with high accuracy. In addition to calculating the variance of the system's solution, we can also determine the

probability distribution that describes the solution to the stochastic system. Finally, some conditions on the parameters that ensure the extinction of cancerous tumors are given below:

(i) if $s - \frac{\sigma_1^2}{2} < 0$, *then the* densities of tumor cells $y(t)$ *will lean towards to extinct a.s..*

(ii) if $s - \frac{\sigma_1^2}{2} > 0$, *then the* densities of tumor cells $y(t)$ *is weakly persistent in the mean a.s..*

(iii) If $(s - \frac{\sigma_2^2}{2}) < c_3(\frac{\sigma_1^2}{2} - r)$, *then the density of normal cells* $x(t)$ *will lean towards extinct a.s..*

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