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A stochastic Differential Equations Model for Lung Cancer Tumor Control and Prevention

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ABSTRACT

In this research, we studied the dynamic behavior of lung cancer to prevent its spread. A new stochastic mathematical model was proposed for this study. To verify the validity of the proposed model, we demonstrated the existence and uniqueness of the solution to the new stochastic model. We explained the conditions that must be met for a person with cancer to be cured by establishing the stochastic basic reproduction number. If $R^s > 1$, this means that the cancer cells in the lung are spreading. On the contrary if $R^s < 1$, this means that the cancer cells are in a state of elimination and the patient is cured. We verified the validity of the results using computer simulations in MATLAB.

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1. INTRODUCTION

Cancer is a term that encompasses a wide range of diseases that can affect any part of the body. It is also called a malignant tumor cancer occurs as a result of the transformation of normal cells into tumor cells in a multi-stage process that progresses from a precancerous lesion to a malignant tumor. These changes result from the interaction of an affected person's genetic factors with three main external factors, which are: physical carcinogens, such as ultraviolet radiation; chemical carcinogens, including components of tobacco and alcohol; and biological carcinogens, such as infections caused by certain bacteria, parasites, and viruses [1,2]. Cancer is a leading cause of death worldwide, killing 10 million people in 2020, accounting for one-sixth of deaths worldwide. The most common types of cancer worldwide are lung cancer, breast cancer, prostate cancer, and colon cancer. To control cancer, it is necessary to know the causes of the disease. It was found that one-third of cancer deaths result from alcohol and tobacco use, high body mass index, low fruit and vegetable intake, lack of physical activity, and air pollution. Air pollution is also a major risk factor for lung cancer [1]. To understand experimental results and explain the biological mechanisms underlying lung cancer progression, numerous mathematical models have been developed to control lung cancer metastasis using principles of mathematical oncology to simulate tumor growth and spread [3,4]. Most mathematical models developed to study lung cancer use compartmental models to track cancer cells, healthy cells, and immune cells, incorporating factors such as genetic mutations, immune system interactions, and drug dynamics[5,6].We note that most of the mathematical models developed to explain the dynamics of lung cancer rely heavily on ordinary differential equations [7, 8, and 9]. In recent years, stochastic differential equations have gained attention and have been used to model many infectious and non-infectious diseases, including coronavirus disease, AIDS, and viral hepatitis [10-19]. In this work, we will introduce environmental stochasticity into a deterministic differential equation model to study how cancer cells spread and interact with immune system cells in the presence of treatment. Several reasons motivated us to use stochastic differential equation models instead of ordinary differential equation models. This is especially true when simulating the spread of lung cancer, as the process of cancer metastasis is actually stochastic, not deterministic. This is because cancer cells interact with immune system cells under the same environmental conditions but produce different outcomes. We can obtain a distribution of expected outcomes by using the stochastic differential equation model multiple

times, making the new stochastic mathematical modeling approach more transparent than deterministic ordinary differential equation models. For example, a deterministic differential equations model will provide a single expected value for the total number of cancer cells at time t. The remaining part of the manuscript is organized as follows: In the second section, we developed a new stochastic mathematical model that explains how cancer cells spread and interact with immune cells in the lung in the presence of treatment, adding the influence of environmental factors in the form of random parameters. In the third section, we prove the existence and uniqueness of the solution to the proposed mathematical model. The fourth section is devoted to deriving the basic reproduction number of the proposed stochastic mathematical model. In fifth section, the main results are presented. Finally, Section Six presents the conclusions and some suggestions.

2. FORMULATING A DETERMINISTIC AND STOCHASTIC MATHEMATICAL MODEL FOR LUNG CANCER CONTROL

Many mathematical models have been developed to understand the dynamics of cancer cells, which ultimately lead to lung cancer. In this paper, we have created a mathematical model that describes the immune system's interaction with cancer cells in the presence of treatment. Our goal in this work is to control lung cancer after detection and treatment by introducing treatments that help generate anti-cancer cells in patients with weak immunity [19].

Therefore, our mathematical model will be as follows:

$$\begin{aligned}
\frac{dT(t)}{dt} &= (r(1-n)T - \alpha T - \theta CT),\\
\frac{dC(t)}{dt} &= (\theta CT + \rho CD - kC),\\
\frac{dD(t)}{dt} &= (m - \rho CD - \omega D - \tau D),\\
\frac{dY(t)}{dt} &= (\tau D - dY),\\
\frac{dZ(t)}{dt} &= (dY - aZ).
\end{aligned} \tag{1}$$

We note that our model, like other previous mathematical models, ignores factors that enhance patients' immunity, as stated on the World Health Organization's website on cancer [1]. These factors include quitting smoking, maintaining a healthy weight, following a balanced diet including fruits and vegetables, exercising regularly, reducing alcohol consumption, and avoiding exposure to ultraviolet radiation, especially from tanning beds. These factors also affect the effectiveness of treatment, as does the patient's psychological state, which varies from one patient to another. All of these reasons prompted us to introduce random parameters into the proposed mathematical model. Therefore, the model is as follows:

$$\begin{aligned}
dT(t) &= (r(1-n)T - \alpha T - \theta CT)dt - \sigma_1 CTdW(t),\\
dC(t) &= (\theta CT + \rho CD - kC)dt + \sigma_1 CTdW(t),\\
dD(t) &= (m - \rho CD - \omega D - \tau D)dt - \sigma_2 CDdW(t),\\
dY(t) &= (\tau D - dY)dt - \sigma_3 YdW(t),\\
dZ(t) &= (dY - aZ)dt + \sigma_3 YdW(t).
\end{aligned} \tag{2}$$

with initial conditions

$$T(0) \geq 0, C(0) \geq 0, D(0) \geq 0, Y(0) \geq 0, Z(0) \geq 0. \tag{3}$$

To illustrate the true role of each parameter used in the first and second mathematical models, we present Table 1.

Table 1. Parameters of the model and their meanings

Parameters	Description
$\boldsymbol{T(t)}$	cancer cells
$\boldsymbol{C(t)}$	is the CD8+ T cells
$\boldsymbol{D(t)}$	Are the dendritic cells
$\boldsymbol{Y(t)}$	Represent cytokines, which are signaling proteins that help control inflammation in your body.
$\boldsymbol{Z(t)}$	Is the anti-PD-L1 inhibitor.
$\boldsymbol{r}$	Lung cells are susceptible to infection
$\boldsymbol{(1-n)}$	Represents the possibility of finding a treatment that limits the growth of the cancerous tumor.
$\boldsymbol{\alpha}$	It represents the percentage of cancer cells that are eliminated from the body by dendritic cells.
$\boldsymbol{\theta}$	Represents the rate at which CD8+ T cells eliminate lung cancer cells.
$\boldsymbol{\sigma_1}$	Represents the random parameter in the system.
$\boldsymbol{k}$	Refers to the rate of CD8+ T cell demise
$\boldsymbol{\sigma_2}$	Represents the random parameter in the system.

$\boldsymbol{\sigma_3}$	Represents the random parameter in the system.
$\boldsymbol{\rho}$	Refers to the rate at which CD8+ T cells become activated by dendritic cells.
$\boldsymbol{\omega}$	Represents the death rate of dendritic cells.
$\boldsymbol{m}$	It represents the sources responsible for generating dendritic cells.
$\boldsymbol{\tau}$	The source of Y(t) to reduce the dendritic cells
$\boldsymbol{d}$	Represents the rate of increase in the strength of the immune system.
$\boldsymbol{a}$	The natural mortality rate for PD-L1 inhibitors.
$\boldsymbol{W_1(t), W_2(t), W_3(t)}$	Represents independent Brownian motions.

3. EXISTENCE AND UNIQUENESS

In this section, we will prove the existence and uniqueness of the solution of the proposed stochastic differential equation model for studying lung cancer control and prevention.

Theorem.1 The solution $(T(t), C(t), D(t), Y(t), Z(t))$ of the proposed stochastic differential equation model (2) for studying lung cancer control and prevention is unique on $t \geq 0$ for any initial value $(T(0), C(0), D(0), Y(0), Z(0)) \in R_+^5$ furthermore the solution will stay in R_+^5 almost surely, i.e. $(T(t), C(t), D(t), Y(t), Z(t)) \in R_+^5$ for all $t \geq 0$ with probability one.

Proof: For every given initial value $(T(0), C(0), D(0), Y(0), Z(0)) \in R_+^5$, the coefficient of the equation are locally Lipchitz Continuous.

There will be one local solution.$(T(t), C(t), D(t), Y(t), Z(t))$ for $t \in [0, \beta_e)$, where β_e is the perfect moment to stop. To demonstrate that this solution is universal, we prove that $\beta_e = \infty$ almost surely, let $k_0 \geq 0$ be big enough, so that $T(0), C(0), D(0), Y(0)$ and $Z(0)$ all lie within the interval $\left[\frac{1}{k_0}, k_0\right]$ for every integer $k \geq k_0$.

The stopping time will be defined as follows:

$$\beta_k = \left\{t \in [0, \beta_e): \min\{T(t), C(t), D(t), Y(t), Z(t)\} \leq \frac{1}{k} \text{ or } max\{T(t), C(t), D(t), Y(t), Z(t)\} \geq k \right\}. \quad (4)$$

During this research, we will take into consideration that $\inf\emptyset = \infty$ as usual $\emptyset$ means the empty set. Where $\beta_k$is increasing as $k \to \infty$.

Set $\beta_\infty = \lim_{k\to\infty} \beta_k$, whence $\beta_\infty \leq \beta_e$ with probability one .If we are able to demonstrate that $\beta_\infty = \infty$ with probability one, then $\beta_e = \infty$ and $(T(t), C(t), D(t), Y(t), Z(t)) \in R_+^5$, almost surely for all $t \geq 0$. Put otherwise, all we have to demonstrate to finish the proof is that $\beta_\infty = \infty$ with probability one or almost surely, If this assumption is not true, this means that there are two constants $L > 0$ and $\varepsilon \in (0,1)$ so that

$$P\{\beta_\infty \leq L\} > \epsilon, \quad (5)$$

That means there is an integer $k_1 \geq k_0$ such that

$P\{\beta_k \leq L\} \geq \epsilon$ for any integer $k \geq k_1$. (6)

Define a C^2 – function $v: R_+^5 \to R_+$ by $v(x)$ =T+C+D+Y+Z+1-(logT+logC+logD+logY+logZ), this proposed function is a positive function according to the following rule $u + 1 - \log(u) \geq 0, \forall u > 0$. By applying Ito's formula to the proposed function and using the coefficients of the proposed equations model, we obtain:

$$dv(T,C,D,Y,Z) = \{(1-\frac{1}{T})[r(1-n)T - \alpha T - \theta CT]$$
$$+\left(1-\tfrac{1}{C(t)}\right)[(\theta CT + \rho CD - kC)] +\left(1-\tfrac{1}{D(t)}\right)$$
$$\left[(m - \rho CD - \omega D - \tau D)\right] + \left(1-\frac{1}{Y(t)}\right)[\tau D - dY] + \left(1-\frac{1}{Z(t)}\right)[dY - aZ] + \frac{1}{2}\sigma_1^2 C^2 + \frac{1}{2}\sigma_1^2 T^2 + \frac{1}{2}\sigma_2^2 C^2$$
$$+\frac{1}{2}\sigma_3^2 + \frac{\sigma_3^2 Y^2}{2Z^2}\Big] dt + \left(\sigma_1 C - \sigma_1 T + \sigma_2 C - \sigma_2 CD + \sigma_3 - \frac{\sigma_3 Y}{Z}\right) dW(t)$$
$$= \left[(r(1-n)T - \alpha T - \theta CT) + \left((\theta CT + \rho CD - kC)\right) + \left((m - \rho CD - \omega D - \tau D)\right) + (\tau D - dY) + (dY - aZ)\right] + \left[-r + rn + \alpha + \theta C - \theta T - \rho D + K - \frac{m}{D} + \rho C + \omega + \tau - \frac{(\tau D(t) - dY(t))}{Y(t)} - \frac{(dY - aZ)}{Z(t)}\right]$$
$$+\left(\sigma_1 C - \sigma_1 T + \sigma_2 C - \sigma_2 CD + \sigma_3 - \tfrac{\sigma_3 Y}{Z}\right) dW(t)$$

Hence.

$$dv(T,C,D,Y,Z) \leq [m + \alpha + k + \omega + \tau + rT + rn + \theta C + \rho C + +\left(\sigma_1 C - \sigma_1 T + \sigma_2 C - \sigma_2 CD + \sigma_3 - \tfrac{\sigma_3 Y}{Z}\right) dW(t) = Q. \quad (7)$$

Consequently

$$E[v(\mathrm{T}(t)(\beta_k \wedge L), \mathrm{C}(t)(\beta_k \wedge L), \mathrm{D}(t)(\beta_k \wedge L), \mathrm{Y}(t)(\beta_k \wedge L), \mathrm{Z}(t)(\beta_k \wedge L)]$$
$$\leq v\big(T(0), C(0), D(0), Y(0), Z(0)\big) + E\left[\int_0^{\beta_k \wedge L} Q dt\right]$$
$$\leq v\big(T(0), C(0), D(0), Y(0), Z(0)\big) + QL. \quad (8)$$

Let $\Omega_k = \beta_k \leq L$ for $k \geq k_1$ and via equation (6) $P(\Omega_k) \geq \epsilon$. Note that for each $\omega \in \Omega_k$, there is at least one $\mathrm{T}(t)(\beta_k, \omega), \mathrm{C}(t)(\beta_k, \omega), \mathrm{D}(t)(\beta_k, \omega), \mathrm{Y}(t)(\beta_k, \omega), \mathrm{Z}(t)(\beta_k, \omega)$ that is equivalent k or $\frac{1}{k}$,and so $v\big((\mathrm{T}(t)(\beta_k), \mathrm{C}(t)(\beta_k), \mathrm{D}(t)(\beta_k), \mathrm{Y}(t)(\beta_k),\ \mathrm{Z}(t)(\beta_k)\big)$ is no less then $k - 1 - \log k$ or $\left(\frac{1}{k}\right) - 1 + \log k$. As a result

$$v\big((\mathrm{T}(t)(\beta_k), \mathrm{C}(t)(\beta_k), \mathrm{D}(t)(\beta_k), \mathrm{Y}(t)(\beta_k), \mathrm{Z}(t)(\beta_k)\big)$$
$$\geq E(k - 1 - \log k) \wedge \left(\left(\frac{1}{k}\right) - 1 + \log k\right). \quad (9)$$

Consequently, equations (6) and (8) imply that

$$v\big(T(0), C(0), D(0), Y(0), Z(0)\big) + QL \geq E\left[1_{\Omega_{(\omega)}} v\big((\mathrm{T}(t)(\beta_k), \mathrm{C}(t)(\beta_k), \mathrm{D}(t)(\beta_k), \mathrm{Y}(t)(\beta_k), \mathrm{Z}(t)(\beta_k)\big)\right]$$
$$\geq \epsilon\left[(k - 1 - \log k) \wedge \left(\left(\frac{1}{k}\right) - 1 + \log k\right)\right]. \quad (10)$$

Where $1_{\Omega_{(\omega)}}$ is the indicator function of Ω. Letting $k \to \infty$ leads to the contradiction

$\infty > v\big(T(0), C(0), D(0), Y(0), Z(0)\big) + QL = \infty$ This means $\beta_\infty = \infty$ with probability one. ■

4. THE STOCHASTIC BASIC REPRODUCTION NUMBER (R^s)

To understand the dynamics of cancer cells, which lead to the formation of masses or tumor tissue that hinders the lung from functioning properly, we will use the random basic reproduction number, which is symbolized in this manuscript as ($\boldsymbol{R^s}$). This number represents the number of cancer cells responsible for forming cancerous tumors in the lung. If it is $R^s < 1$, it means that the lung will get rid of the cancer cells and the patient will be cured. Conversely, if it is $R^s > 1$, it means that the cancer cells are spreading and could infect all parts of the lung. The stochastic basic reproduction number can be derived as follows:

If we take the equation for cancer cells

$dT(t) = (r(1-n)T - \alpha T - \theta CT)dt - \sigma_1 CT dW(t)$,

and reformulate it as

$$\frac{dT(t)}{T(t)} = (r(1-n) - \alpha - \theta C)dt - \sigma_1 C dW(t)$$

So, the part $\ln T(t)$ must appear in the solution, in this case, we set $V(T(t), t) = \ln T(t)$ and when we apply Ito's formula[14] well get

$$d\,V(T(t), t) = \left[0 + (r(1-n) - \alpha - \theta C)T(t)\left(\frac{1}{T(t)}\right) - \frac{1}{2}{\sigma_1}^2 C^2 T(t)^2 \left(\frac{t}{T(t)^2}\right)\right] dt$$
$$+ \sigma_1 CT(t)\left(\frac{1}{T(t)}\right) dW(t),$$

so $d\,V(T(t), t) = \left[(r(1-n) - \alpha - \theta C) - \frac{1}{2}{\sigma_1}^2 C^2\right] dt + \sigma_1 C dW(t)$ when both sides of the equation are integrated, we obtain

$\ln T(t) - \ln T(0) = \left[(r(1-n) - \alpha - \theta C) - \frac{1}{2}{\sigma_1}^2 C^2\right] t + \sigma_1 C\big(W(t) - W(0)\big)$ since $W(0) = 0$. So the solution for equation of cancer cells is

$$T(t) = T_0 exp\left[(r(1-n) - \alpha - \theta C) - \frac{1}{2}{\sigma_1}^2 C^2\right] t + \sigma_1 C\big(W(t)\big)$$

So, the stochastic basic reproduction number is

$$\boldsymbol{R^s} = \left[\frac{r(1-n)}{\alpha + \theta C} - \frac{\sigma_1^2 C^2}{2(\alpha + \theta C)}\right] \quad (11)$$

5. MAIN RESULTS

To illustrate the role of restrictions on the spread of cancer, which we symbolize with the symbol $(1-n)$, and therapies that stimulate the immune system to produce CD8+ T cells that eliminate cancer cells, as well as the positive role of increasing immunity by quitting smoking, not consuming alcohol, exercising, and

avoiding exposure to ultraviolet rays, these positive factors are symbolized by random parameters $\sigma_1, \sigma_2 \& \sigma_3$. We will look at two examples to illustrate the role of the above restrictions.

Example (1): If the treatment is late and ineffective and the random parameters are small, the parameter values, will be as in the Table 2.

Table 2. Values of the proposed parameters that control system stability

Parameter	The value
r	$100000 * 10^{-2} day^{-1}$
$\boldsymbol{\alpha}$	$2000 * 10^{-1} day^{-1}$
C	$1000 * 10^{-2} day^{-1}$
$\boldsymbol{\theta}$	$1000 * 10^{-2} cells^{-1} mm^{-3} day^{-1}$
$\boldsymbol{\sigma_1}$	$1 * 10^{-2}$
$\boldsymbol{\sigma_2}$	$2 * 10^{-2}$
$\boldsymbol{\sigma_3}$	$3 * 10^{-2}$
n	$1 * 10^{-1}$
$\boldsymbol{T(0)}$	$10000 * 10^{-1} day^{-1}$
$\boldsymbol{D(0)}$	$10000 * 10^{-1} day^{-1}$
$\boldsymbol{\rho}$	$100 * 10^{-1} day^{-1}$
$\boldsymbol{k}$	$1000 * 10^{-1} day^{-1}$

First, we have to find the basic random reproduction number $\boldsymbol{R^s}$,by substituting the values of the parameters in equation (11) as,

$$\frac{100000*10^{-2}(0.9)}{2000*10^{-1}+1000*10^{-2}*1000*10^{-2}} - \frac{0.0001*100}{2(2000*10^{-1}+1000*10^{-2}*1000*10^{-2}} = 3.$$

We find that the value of the random reproduction number $\boldsymbol{R^s} = 3 > 1$. Because the $\boldsymbol{R^s}$ value is greater than one, this indicates that the cancer has spread in the lung. To confirm this result, we will take the cancer cells equation.

$$dT(t) = (r(1-n)T - \alpha T - \theta CT)dt - \sigma_1 CT dW(t).$$

Here, we will find the solution to the equation by applying Ito's random formula as follows

$$T(t) = T_0 exp\left[(r(1-n) - \alpha - \theta C) - \frac{1}{2}{\sigma_1}^2 C^2\right]t + \sigma_1 C\big(W(t)\big)$$

After substituting the values of the parameters in the equation.

$$T(t) = 1000exp\left[(100000 * 10^{-2}(0.9) - 2000 * 10^{-1} - 1000 * 10^{-2} * 1000 * 10^{-2}) - \frac{1}{2}0.0001 * 100\right]t$$

We find that the solution to the cancer cells equation is $T(t) = 1000e^{599.95t}$. This confirms the result we obtained from ($\boldsymbol{R^s}$), which is that the cancerous disease is spreading. To confirm our results, we will use a computer simulation using MATLAB. After using the computer simulation, we find that the disease is spreading and expanding in the lung, as shown in Figure 1 below:

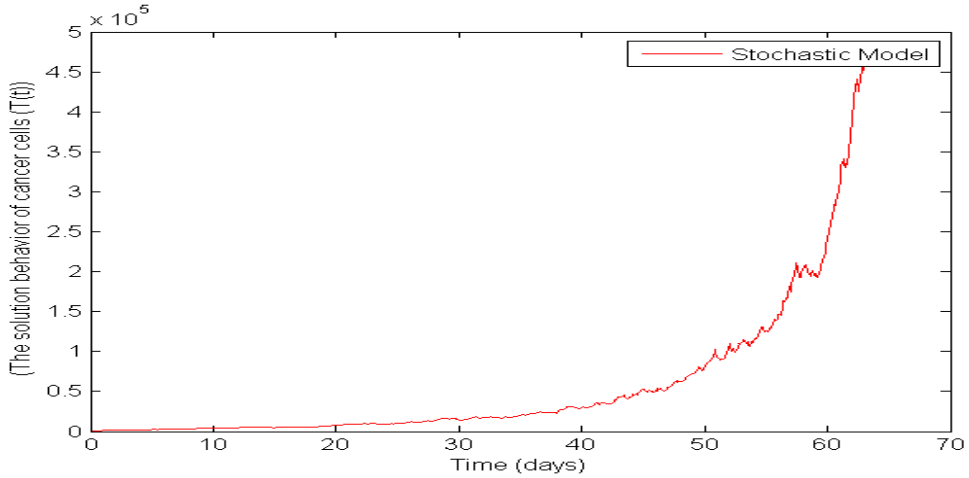

Figure 1. Computer simulations show that cancer cells do not tend towards zero exponentially when $\boldsymbol{R^s} = 3 > 1$.

Example.2: Unlike the previous example, if the treatment is early in the disease onset, it will be effective. Additionally, if the random parameters are relatively large, the parameter values will be as shown in the Table 3.

Table 3. The proposed transaction values after optimization that control the system stability

Parameter	The value
r	$100000 * 10^{-2} day^{-1}$
$\boldsymbol{\alpha}$	$2000 * 10^{-1} day^{-1}$
C	$1000 * 10^{-2} day^{-1}$
$\boldsymbol{\theta}$	$1000 * 10^{-2} cells^{-1} mm^{-3} day^{-1}$
$\boldsymbol{\sigma_1}$	$1 * 10^{-1}$
$\boldsymbol{\sigma_2}$	$2 * 10^{-1}$
$\boldsymbol{\sigma_3}$	$3 * 10^{-1}$
n	$9 * 10^{-1}$
$\boldsymbol{T(0)}$	$10000 * 10^{-1} day^{-1}$
$\boldsymbol{D(0)}$	$10000 * 10^{-1} day^{-1}$
$\boldsymbol{\rho}$	$100 * 10^{-1} day^{-1}$
$\boldsymbol{k}$	$1000 * 10^{-1}\ day^{-1}$

As in the previous example, calculate the value of the basic random reproduction number $\boldsymbol{R^s}$. We find its value $\boldsymbol{R^s} = 0.33 < 1$, because ($\boldsymbol{R^s}$) is less than one, this means that the cancer cells are in a state of disappearance and that the person with the disease will be cured. To confirm the result we obtained from the random basic reproduction number, we will take the cancer cells equation.

$$dT(t) = (r(1-n)T - \alpha T - \theta CT)dt - \sigma_1 CTdW(t).$$

after that , we will find the solution to the equation by applying Ito's formula as follows

$$T(t) = T_0 exp\left[(r(1-n) - \alpha - \theta C) - \frac{1}{2}{\sigma_1}^2 C^2\right]t + \sigma_1 C\big(W(t)\big)$$

After substituting the values of the parameters in the equation.

$$T(t) = 1000exp\left[(100000 * 10^{-2}(0.1) - 2000 * 10^{-1} - 1000 * 10^{-2} * 1000 * 10^{-2}) - \frac{1}{2}0.01 * 100\right]t$$

Well find the solution to the cancer cells equation is $T(t) = 1000e^{-200.5t}$. The result means that the solution has stabilized, which means the disappearance of the cancerous tumor and the recovery of the person with lung cancer. The stability of the solution was verified using MATLAB, as shown in Figure 2.

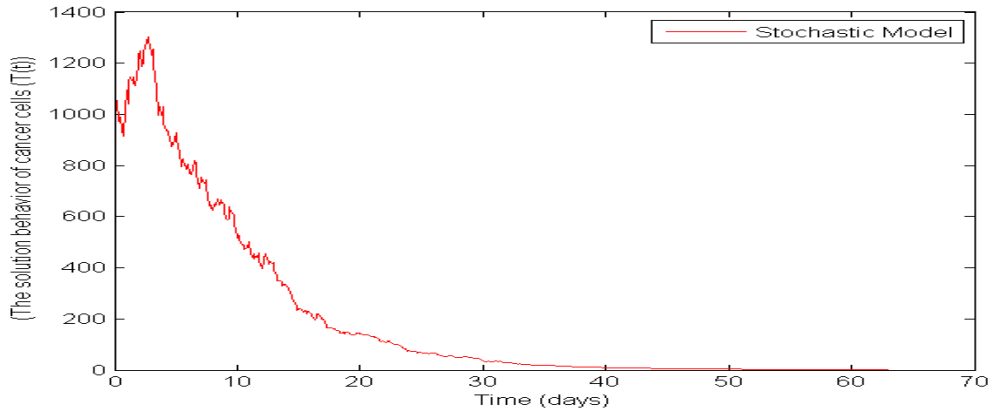

Figure 2. Computer simulations show that cancer cells move significantly toward zero when $\boldsymbol{R^s} = 0.33 < 1$.

6. CONCLUSIONS

The novelty of this research paper is the addition of environmental randomness to the deterministic mathematical model of lung cancer spread. We also discovered the characteristics of the disease and how it spreads within the lung, as well as the interaction of its cells with those of the immune system. In this research, we derived the stochastic basic reproduction number and the conditions necessary for the extinction or spread of cancer cells. In this research, we praised the role of treatments that stimulate the immune system to produce CD8+ T cells in eliminating the disease. We also explained the positive role of increasing immunity by not drinking alcohol and quitting smoking, and avoiding exposure to ultraviolet rays, especially tanning devices and exercising regularly and eating a healthy diet are all beneficial, despite the positive role of these factors play previous researches has not focused on them. Therefore, when the treatment is effective and the random parameters ($\sigma_1, \sigma_2 \& \sigma_3$) are large this gives us $\boldsymbol{R^s} < 1$, which means the disappearance of the disease, as shown in Figure 2. However, if the treatment is ineffective, the random parameters ($\sigma_1, \sigma_2 \& \sigma_3$) weak will get $\boldsymbol{R^s} > 1$ which means the continuation of the disease, as shown in Figure 1.

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