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A stochastic KANCE model for lung cancer tumor control

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Abstract:

Lung cancer is considered a serious disease affecting human health. It is affected by various biological and environmental factors. These factors influence the growth and spread of the disease in the human body. In this research , we addressed the dynamics of lung cancer and modeled these dynamics by building a stochastic mathematical model based on stochastic differential equations (SDEs). This model combines, the cancer cells(K) ,the T-cells(A) , the dendritic cells(N) , the cytokines(C) , and the cytokine inhibitors(E). The main objective of our model is to study the effect of random factors on the interaction between disease and immunity. We try to identify the conditions under which the disease persists or disappears. In this research , we used Ito's theory to analyze disease stability . The basic reproduction number is deduced , which shows us whether the disease is in remission or growing. We also proved the existence of the solution and its uniqueness to our model to confirm the validity of the (KANCE) model.

Keywords: Stochastic differential equations (SDEs),A stochastic model ,Lung cancer , Basic reproduction number, wiener process.

Note: The research is based on an M.A thesis or a PhD dissertation :yes

Introduction:

One of the most common diseases today is cancer , which can affect any organ in the body . One-sixth of deaths worldwide are caused by cancer, and one of its most common types is lung cancer , considered one of the most dangerous cancers for human life , often leading to death . Cancer cells arise from the interaction of an individual's genetic factors with three main external factors: biological carcinogens like Infection caused by a bacterial injury , parasites , and viruses; Cancer-causing physical agents such as ultraviolet radiation; and Cancer-causing chemical agents , which are components of tobacco and alcohol[1,2] . Numerous mathematical models have been developed to study and clarify biological mechanisms underlying lung cancer development and to control its metastasis[3-5]. These models rely on the principles of mathematical oncology to simulate The prevalence and growth of cancer cells in the lung[6,7]. Most modern mathematical models developed for lung cancer studies are segmented models that track cancer cells, healthy cells, and dendritic cells, taking into account factors such as immune system interactions, drug dynamics, and genetic mutations[7-11]. However, all previous mathematical models ignore factors that can weaken immunity and contribute to lung cancer development, such as smoking, alcohol consumption, UV exposure, excess weight, and lack of exercise. These factors also affect treatment effectiveness[11-19]. To represent these factors that influence immunity and disease progression, we proposed a stochastic mathematical model that represents the role of these factors in system stability and also describes the interaction of cancer cells with immune system cells in the available of treatment. Furthermore, the distribution of the outcomes can be obtained using the stochastic differential equation model multiple times, making this new stochastic mathematical modeling approach more transparent than deterministic differential equation models. For example, a deterministic differential equation model would provide only one predicted value for the total number of cancer cells at time t[19-26]. This new model provides valuable insights for public health strategies and treatment planning. We organized the remainder of the manuscript as follows: In the section 2, presents some basic definitions and

theories related to random differential equations. In the section 3, the mathematical model was formulated, and a table was also presented that includes all the parameters that appeared in the model.

To investigate the activities of cancer cells and the lung's ability to heal, the basic proliferation number was presented in Section 4. In the section 5, the existence and uniqueness of the solution to the proposed mathematical model were proven. A main results were presented in section 6. The section 7, was devoted to the conclusion.

Preliminaries

To learn how to use the stochastic differential equations model to understand the dynamic movement of cancer cells, we will present in this section some definitions and theories related to stochastic differential equations.

Definition (1)

A stochastic process W(t) , $t \in [0, \infty)$ is said to be wiener process if it satisfies

i) $P(W(0) = 0) = 1,$

ii) For $0 < t_0 < t_1 < \cdots < t_n$, the increments

$W(t_1) - W(t_0), \dots, W(t_n) - W(t_{n-1})$ are independent.

iii) For any t and $l > 0, W(t + l) - W(t)$ has a Gaussian distribution with mean 0 and variance l.

Definition (2) The Mathematical models of SDE is

$$dX(t) = k(X(t), t)dt + z(X(t), t)dW(t),$$
$$t \in [t_0, T], T > 0. \qquad (1)$$

with initial condition $X(t_0) = X_0$, where X_0 is stochastic variable independent of $W(t) - W(t_0)$ and $E(|X_0|^2) < +\infty$, k & z are diffusion and drift coefficient functions respectively. The solution to equation (1) in integral formula takes the following formal

$$X(t) = X_0 + \int_{t_0}^{t} k(X(s), s)ds + \int_{t_0}^{t} z(X(s), s)\, dW(s), t \in [t_0, T], \qquad (2)$$

where the first integral is Riemann integral and the Second integral is stochastic integral.

Definition (3) let $z(t)$ be a continuous stochastic function having derivative in $[\varepsilon, \delta]$ and $W(t), t \geq 0$ denote a wiener process the mathematical formula for stochastic integral will be as follows:

$$\int_{\varepsilon}^{\delta} z(t)dW(t) \equiv \lim_{n\to\infty} \sum_{i=1}^{n} z(t_{i-1})[W(t_i) - W(t_{i-1}] \qquad (3)$$

where $\varepsilon = t_0 < t_1 \ldots < t_n = \delta$ is the subdivision of the area $[\varepsilon, \delta]$.

Theorem(1)

Ito's formula, Assume that Y_t has SDE in the form

$Y_t = k(Y_t, t)dt + z(Y_t, t)dW(t),$

(4)

for all $k, z \in C^{1,2}(L \times R, R)$, suppose that $F: L \times R \to R$ be continuous and has $\frac{\partial F}{\partial t}$, $\frac{\partial F}{\partial Y_t}$ and $\frac{\partial^2 F}{\partial Y_t^2}$ are exist and continuous if $F = F(Y_t, t)$, so F has the SDE $dF = \frac{\partial F}{\partial t}dt + \frac{\partial F}{\partial Y_t}kdt + 0.5\,\frac{\partial^2 F}{\partial Y_t^2}Z^2dt.$

$dF(Y_t, t) =$

$$\left[\frac{\partial F}{\partial t} + \frac{\partial F}{\partial Y_t}k + 0.5\,\frac{\partial^2 F}{\partial Y_t^2}Z^2\right]dt + \frac{\partial F}{\partial Y_t}zdW(t). \qquad (5)$$

The last equation (5) is Ito's formula, and we can express it in its expanded form as

$$dF_n(Y_t, t) = \left[\frac{\partial F_n}{\partial t} + \sum_{i=1}^{m} k_i \frac{\partial F_n}{\partial Y_t} + \frac{1}{2}\sum_{i,j=1}^{m} z_{i1}z_{j1}\frac{\partial^2 F_n}{\partial Y_i \partial Y_j}\right]dt + \sum_{i=1}^{d}\sum_{i=1}^{m} z_{i1}\frac{\partial F_n}{\partial Y_i}dW(t). \qquad (6)$$

where $n = 1,2, \ldots, m$ and $W(t) = (W(t)^1, W(t)^2, \ldots, W(t)^d)^T$.

Formulating the mathematical model

We developed a mathematical model of lung cancer that utilizes cytokines and cytokine inhibitors to boost the immune system . We named this model KANCE .

KANCE incorporates a random factor not found in previous studies . This random factor covers extracellular factors that may contribute to lung cancer . These factors include smoking , air pollution , exposure to certain chemicals or radiation, and anything else that promotes cancer growth[1] .

In this model **K** symbolize the cancer cells ,**A** symbolize the T-cells , **N** symbolize the dendritic cells , **C** symbolize the cytokines , and **E** symbolize the cytokine inhibitors.

We will present the KANCE model , which is constructed based on the inclusion of stochastic factors as follows

$$dK(t) = (r(1-n)K - \beta K - \gamma AK)dt - \sigma^{1} AKdW_1(t), \quad (7)$$

$$dA(t) = (\gamma AK + \omega AN - hA)\,dt + \sigma_1 AKdW_1(t), \quad (8)$$

$$dN(t) = (\mu - \omega AN - \rho N - \lambda N)dt - \sigma_2 AN\,dW2\,(t), \quad (9)$$

$$dC(t) = (\lambda N - gC)dt - \sigma_3 CdW_3(t), \quad (10)$$

$$dE(t) = (gC - qE)dt + \sigma_3 C\,dW_3(t). \quad (11)$$

With initial conditions $K(t) \geq 0$, $A(t) \geq 0$, $N(t) \geq 0$, $C(t) \geq 0$, $E(t) \geq 0$. (12)

To illustrate the true role of each parameter used in the proposed mathematical model, we present Table 1.

Table 1 : parameter of the model and their meanings

Parameters	Description
K(t)	Cancer cells
A(t)	CD8+ T cells
N(t)	Dendritic cells
C(t)	The concentration of cytokine
E(t)	The cytokine inhibitors
r	Lung cells are susceptible to infection
(1 – n)	The Capacity factor
β	Rate of cancer cell destruction
γ	Represents the percentage of cancer cell elimination by CD8+ T cells..

h	Nature death rate of A-cells
μ	Nature generation rate of N-cells
ω	It represents the amended at which dendritic cells become inactive by CD8+ T cells,
ρ	Nature destruction rate of N-cells
λ	The percentage of dendritic cell loss
g	The rate at which anti-PD-L1 Inhibitor cells increase
q	Removal rate of cytokine inhibitors
σ_1	First stochastic parameter
σ_2	Second stochastic parameter
σ_3	Third stochastic parameter
$W_1(t), W_2(t), W_3(t)$	Independent Brownie motions

The stochastic Basic reproduction number(R_e^n)

To investigate the behavior of cancer cells and whether the lung is capable of healing and eliminating the disease, we will use the basic reproduction number (R_e^n). If $R_e^n > 1$, this means that the amount of new cancer cells formed is greater than the number of cancer cells eliminated by immunity and treatments, thus indicating disease spread. If $R_e^n < 1$, this means that the amount of cancer cells eliminated is greater than the number of cancer cells formed, indicating the disappearance of cancerous tumors from the lung. The basic reproduction number for this model will be derived from equation (7) as follows:

$$dK(t) = \big(r(1-n)K(t) - \beta K(t) - \gamma AK(t)\big)dt - \sigma_1 AK(t)dW_1(t),$$

we will rewrite equation (7) as

$$\frac{dK(t)}{K(t)} = (r(1-n) - \beta - \gamma A)dt - \sigma_1 AdW_1(t),$$

Then the term $\ln K(t)$ will appear in the solution ,

then we set $V(K^t, t) = \text{Ln. } K(t)$, since

f= $\left(r(1-n)K(t) - \beta K(t) - \gamma AK(t)\right)$ and g= $-\sigma_1 AK(t)$

so by using Ito's formula will get

d $V(K^t, t)$=($\frac{1}{K_t}$ $(r(1-n)K_t - \beta K_t - \gamma AK_t)$ −

$\frac{1}{2}\frac{1}{k_t^2}\sigma_1^2 A^2 k_t^2)\, dt$ $\frac{1}{K_t}\sigma_1 AK_t\, dW_1(t)$

d(ln K_t) = (r (1-n) - β - γ A - $\frac{1}{2}\sigma_1^2 A^2$) dt - σ_1 A $dW_1(t)$

from integration we get

$ln\, K_t - ln\, K_0$ = (r (1-n) - β - γ A - $\frac{1}{2}\sigma_1^2 A^2$) t - σ_1A(W^t-W^0)

ln $\frac{K_t}{k_0}$= (r (1-n) - β - γ A - $\frac{1}{2}\sigma_1^2 A^2$) t − σ_1AWt

K_t= K o $e^{(r(1-n) - \beta - \gamma A - \frac{1}{2}\sigma_1^2 A^2)t - \sigma_1 AWt}$

So the Basic reproduction number R^s is

R_e^n= $[\frac{r(1-n)}{\beta+\gamma A} - \frac{\sigma_1^2 A^2}{2(\beta+\gamma A)}]$.

(13)

Existence and uniqueness

In this section, we discuss the existence and uniqueness of the solution to the proposed stochastic differential equation for lung tumor propagation.

Theorem : The solution (K(t) , A(t) , N(t) ,C(t) , E(t)) to this proposed model of lung cancer is unique on to t ≥ 0 for every initial value (K(0) , A(0) , N(0) ,C(0) , E(0)) ∈ R_+^5 and the solution will persist within R_+^5 with one possibility . i.e. (K(t) , A(t) , N(t) ,C(t) , E(t)) ∈ R_+^5 ∀ t ≥ 0 a.s. (almost surely).

Proof : The equation's coefficients are locally Lipschitz continuous for all possible initial population sizes (K(0) , A(0) , N(0) , C(0) , E(0)) ∈ R_+^5 . there is one solution (K(t) , A(t) , N(t) ,C(t) , E(t)) on t ∈ [0 ,S_e) where S_e is the moment of explosion .

To confirm that the solution is global , we demonstrate that S_e = ∞ a.s

let $a_0 \geq 0$ be big enough , so that K(0) , A(0) , N(0) , C(0) and E(0) all are situated within the given interval $[\frac{1}{a_0} , a_0]$ for every integer $a \geq a_0$.

The stopping time is formally defined as:

$S^a = \{t \in [0 , S_e] : \min \{ K(t) , A(t) , N(t) , C(t) , E(t) \} \leq \frac{1}{a}$
or $\max \{ K(t) , A(t) , N(t) , C(t) , E(t) \} \geq a \}$.
(14)

During this research we consider $\inf \emptyset = \infty$ as usual $\emptyset$ denote the empty set .

Where S^a is growing as $a \to \infty$.

Now set $S_\infty = \lim_{a\to\infty} S^a$,where $S_\infty \geq s_e$ a.s .

If we are able to prove that $S_\infty = \infty$ a.s then $s_e = \infty$ and
$(K(t) , A(t) , N(t) , C(t) , E(t)) \in R_+^5$ almost surely for all $t \geq 0$.

Otherwise all we have to explain to finish the proof is that $S_\infty = \infty$ a.s for if this assertion is untrue , after which there are two constants B>0 and $\varepsilon \in (0,1)$ such that

$P\{ S_\infty \leq B \} > \varepsilon$ .Thus an integer exist $a_1 \geq a_0$ such that

$P\{S^a \leq B\} \geq \varepsilon$. For every one $a \geq a_1$.
(15)

Describe a C^2- function V: $R_+^5 \to R_+$ by
$V(K,A,N,C,E) = [k+1- \log k] +[A+1-\log A] + [N+1-\log N] +[C+1-\log C]+[E+1-\log E]$
(16)

This function's non-negative ,
it is clear from $X+1-\log X \geq 0 , \forall X > 0$.

Applying Ito's formula we get

$$dV(K,A,N,C,E) = [(1-\frac{1}{K})(r\,(1-n)\,K - \beta\,K - \gamma\,AK) + (1-\frac{1}{A})\,(\gamma AK + \omega AN - hA) + (1-\frac{1}{N})\,(\mu - \omega AN - \rho N - \lambda N) + (1-\frac{1}{C})\,(\lambda N - gC) + (1-\frac{1}{E})\,(gC - qE) + \frac{1}{2}\sigma_1^2 A^2 + \frac{1}{2}\sigma_1^2 K^2 + \frac{1}{2}\sigma_2^2 A^2 + \frac{1}{2}\sigma_3^2 + \frac{1}{2E^2}\sigma_3^2 C^2]dt + (1-\frac{1}{K})(-\sigma_1 AK)\,dW_1 + (1-\frac{1}{A})\,(\sigma_1 AK)dW_1 + (1-\frac{1}{N})\,(-\sigma_2 AN)dW_2 + (1-\frac{1}{C})(-\sigma_3 C)dW_3 + (1-\frac{1}{E})\,(\sigma_3 C)dW_3 .$$

After simplifying Ito's formula we get

$$dV(K,A,N,C,E) = r\,(1-n) - \beta\,K - hA + \mu - \rho N - qE - \frac{r(1-n)}{K} + \beta + \gamma A - \gamma k - \omega N + h - \frac{\mu}{N} + \omega\,A + \rho + \lambda - \frac{\lambda N}{C} + g - \frac{gC}{E} + q + \frac{1}{2}\,\sigma_1^2 A^2 + \frac{1}{2}\,\sigma_1^2 K^2 + \frac{1}{2}\,\sigma_2^2 A^2 + \frac{1}{2}\,\sigma_3^2 + \frac{1}{2E^2}\,\sigma_3^2 C^2 + (\sigma_1 A - \sigma_1 K)\,dW_1 + (\sigma_2 A - \sigma_2 AN)dW_2 + (\sigma_3 - \frac{\sigma_3}{E}C)\,dW_3 .$$

Hence $dV(K,A,N,C,E) \le r(1-n) + \mu + \beta + \gamma A + h + \omega\,A + \rho + \lambda + g + q + \frac{1}{2}\,\sigma_1^2 A^2 + \frac{1}{2}\,\sigma_1^2 K^2 + \frac{1}{2}\,\sigma_2^2 A^2 + \frac{1}{2}\,\sigma_3^2 + \frac{1}{2E^2}\,\sigma_3^2 C^2 + (\sigma_1 A - \sigma_1 K)dW_1 + (\sigma_2 A - \sigma_2 AN)\,dW_2 + (\sigma_3 - \frac{\sigma_3 C}{E})\,dW_3 = H.$

where H represents only the positive terms of the equation .Consequently $E[K(t)\,(S_a \wedge B), A(t)(S_a \wedge B)$,N(t)$(S_a \wedge$ B),C(t) $(S_a \wedge$ B),E(T) $(S_a \wedge$ B)$] \le$ V[(K(0) , A(0) , N(0) , C(0) , E(0)]+E[$\int_0^{(S_a \wedge B)} H\,dt \le$V[(K(0) , A(0) , N(0) , C(0) , E(0)]
+HB (17)

Let $\Omega_a = S_a \le$ B for a $\ge a_1$ and via equation (16) P$(\Omega_a) \ge \varepsilon$.

Observe that for each $\mathfrak{G} \in \Omega_a$ there exist at least one K(t)(S_a,$\mathfrak{G}$) , A(t) (S_a,$\mathfrak{G}$) ,N(t) (S_a,$\mathfrak{G}$) ,C(t) (S_a,$\mathfrak{G}$) ,E(t) (S_a,$\mathfrak{G}$)that is equivalent a or $\frac{1}{a}$ and so V[K(t)(S_a) ,A(t) (S_a) ,N(t) (S_a) ,C(t)(S_a) ,E(t)(S_a)] is not less than a-1-log a or $\frac{1}{a}$ -1+log a .

AS a result

V[K(t)(S_a) , A(t) (S_a) , N(t) (S_a) , C(t)(S_a),E(t)(S_a)]

≥E(a-1-loga)^($\frac{1}{a}$ -1+log a) . (18)

Consequently ,Equations (16) and (17) imply that

V[(K(0) , A(0) , N(0) , C(0) , E(0)]+HB

≥ E[$1_{\Omega(\mathfrak{G})}$V[K(t)(S_a) ,A(t) (S_a) ,N(t) (S_a) ,C(t)(S_a) ,E(t)(S_a)]

≥Ɛ[(a-1-loga)^ ($\frac{1}{a}$ -1+log a)] (19)

Where $1_{\Omega(\mathfrak{G})}$ is the indictor function of Ω . Let a → ∞ and this is contradiction

∞ > V[(K(0) , A(0) , N(0) , C(0) , E(0)]+HB = ∞

This means S_∞ = ∞ a.s , ■

Main results

In this section, we will numerically prove, using computer simulation, that if R_e^n > 1, this means system instability and disease persistence, and also if R_e^n < 1, this means system stability and recovery of the patient. To illustrate the role of random parameters in system stability and also treatments that stimulate the immune system to produce CD8+ T cells that eliminate cancer cells, we will provide two examples.

Example 1

In this example we will use a weak constraint and small randomness and we will show that the disease is spreading.

r =10000 , β =500 , A=100 , γ =10 , σ_1^2= 0.0001 , n =0.1

The solution to the model will be according to these imposed values as follows

$R_e^n = [\frac{r(1-n)}{\beta+\gamma A} - \frac{\sigma_1^2 A^2}{2(\beta+\gamma A)}]$.

$R_e^n = \frac{10000(0.9)}{500+1000} - \frac{0.0001(10000)}{2(500+1000)}$

$R_e^n = 5.9997 > 1$ Since the basic reproduction number is greater than one, this means the disease will persist and spread. To confirm this fact, we take equation (7).

$dK(t) = (r(1-n)K - \beta K - \gamma AK)dt - \sigma_1 AKdW_1(t)$ by using Ito's formula on equation (7) we get the solution as

$K(t) = K_0 \; e^{(r(1-n) - \beta - \gamma A - \frac{1}{2}\sigma_1^2 A^2)t}$

and substitution the imposed values we get $K(t) = K_0 \; e^{(599.95)t}$ Here, the exponential value moves away from zero, confirming the continued spread of cancer cells in the lung. To illustrate the result, we use MATLAB software as shown in Figure 1.

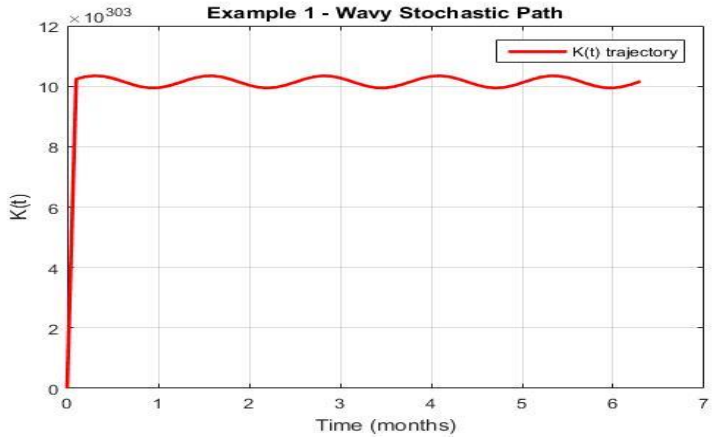


Figure 1 . shows the spread of cancer cells in the lung .

Example 2:

In this example, we will utilize strong constraints and introduce large randomness, demonstrating that the disease gradually shrinks and eventually disappears.

$r = 10000$, $\beta = 500$, $A = 100$, $\gamma = 10$, $\sigma_1^2 = 0.01$, $n = 0.9$

The solution to the model will be according to these imposed values as follows

$R_e^n = \frac{10000(0.1)}{500+1000} - \frac{0.01(10000)}{2(500+1000)}$

$R_e^n = 0.6333333 < 1$, Since the basic reproduction number is less than one, this means that the cancer cells are decreasing, indicating that the tumor is shrinking and eventually disappearing. We take equation (7)

$$dK(t) = (r(1-n)K - \beta K - \gamma AK)dt - \sigma^{1} AKdW_1(t)$$

by using Ito's formula on equation (1) we get

$K(t) = K_0 \; e^{(r(1-n) - \beta - \gamma A - \frac{1}{2}\sigma_1^2 A^2)t}$

and substitute the imposed values, we get

$K(t) = K_0 \; e^{(-550)t}$ Here, the exponential value approaches zero, indicating a decrease in cancer cells. To illustrate the result, we use MATLAB software as shown in Figure 2.

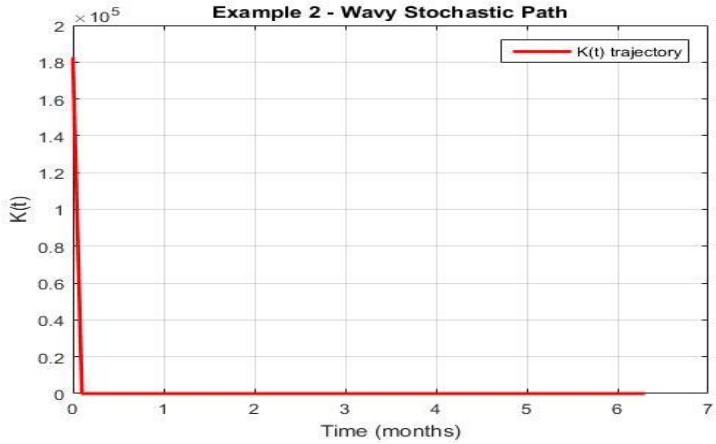


Figure 2. It appears that the cancer cells disappear and the patient recovers.

Conclusion:

In this study, We constructed a mathematical model founded on environmental randomness to explain lung cancer progression. This proposed model incorporates random effects into the deterministic mathematical model to account for factors such as smoking, alcohol consumption, and UV exposure, and we demonstrated the role of these environmental factors in disease progression or remission. It also considers

drugs such as cytokines and cytokine inhibitors as treatments. Furthermore, we demonstrated the role of random parameters in representing the unpredictable way tumors interact with the immune system. Analysis of the basic reproduction number showed that if $R_e^n > 1$, This means that the number of cancer cells removed by immunity, treatments, and environmental factors is less than the number of new cancer cells, and this means that the disease continues, as shown in Figure 1. However, if environmental randomness is high, immunity is high, and treatments are effective, this gives us $R_e^n < 1$, and the tumor will shrink and disappear as shown in Figure 2.

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نموذج (KANCE) التصادفي للسيطرة على ورم سرطان الرئة

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مستخلص البحث:
يُعد سرطان الرئة مرضًا خطيرًا يُؤثر على صحة الإنسان، ويتأثر بعوامل بيولوجية وبيئية متنوعة تُؤثر بدورها على نمو المرض وانتشاره في الجسم. في هذا البحث، تناولنا ديناميكيات سرطان الرئة، وقمنا بنمذجة هذه الديناميكيات من خلال بناء نموذج رياضي تصادفي قائم على المعادلات التفاضلية التصادفية. يجمع هذا النموذج بين الخلايا السرطانية (K)، والخلايا التائية (A)، والخلايا المتغصنة (N)، والسيتوكينات (C)، ومثبطات السيتوكينات (E). يتمثل الهدف الرئيسي لنموذجنا في دراسة تأثير العوامل العشوائية على التفاعل بين المرض والمناعة، ومحاولة تحديد الظروف التي يستمر فيها المرض أو يختفي. في هذا البحث، استخدمنا نظرية إيتو لتحليل استقرار المرض، واستنتجنا معدل التكاثر الأساسي الذي يُبين ما إذا كان المرض في حالة هدوء أو في حالة تفاقم. كما أثبتنا وجود حل فريد لنموذجنا، وذلك لتأكيد صحة نموذج (KANCE).

الكلمات المفتاحية : المعادلات التفاضلية التصادفية، نموذج تصادفي، سرطان الرئة، رقم التكاثر الاساسي، عمليات وينر.

ملاحظة : هل البحث مستل من رسالة ماجستير او اطروحة دكتوراه ؟ نعم