

A Mathematical Model of Tumor Growth in Human Body with the Rough Set

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ABSTRACT

Tumors are a significant issue in the world. They are a substantial cause of death and put a heavy load on medical services. Many researchers' have been trying to develop a new medical treatment model for tumors. The growth of tumor cells is uncertain due to their abnormal behavior. The Rough set method is an emerging interventional technique and the most powerful mathematical tool to deal with unpredictable situations. Metastasis dispersal is the procedure by which a few cells from the tumor leave and make another tumor. Subsequently, the danger can scatter through the entire living organism. In this paper, the dynamics of tumor cells are established with the metastasis process in the human body and verified by the Rough set method's technique. This paper develops a connection between applied mathematics, numerical computation, and applications of biological systems.

Keywords: Tumor; Nonlinear; Carrying capacity; Tumor cells; Rough set

1. Introduction

Tumors are a significant issue around the world. They are a significant cause of death and put a heavy load on medical services [1-2]. Many researchers' have been working to develop a novel medical practice model for tumors. The growth of tumor cells is unpredictable due to their irregular behavior [1-2]. Possible signs and reactions of the tumor cell growth include a bump, anomalous dying, unexplained weight decrease, and bowel movements [3]. Tobacco consumption is one of the major causes of around twenty-two percent of

tumor deaths [1]. Another ten percent is due to heaviness, poor diet, lack of physical activity, and unreasonable drinking of liquor [1, 4-5]. A different component includes biological pollutants, contaminations, and ionizing radiation [6]. Various hereditary changes are required before tumors develop [7]. Around ten percent of growth results from gained inherited imperfections from a person's parents [8].

Tumors are recognized by particular signs and screening tests [1]. They are then regularly inquired about by therapeutic imaging and avowed by biopsy [9].

Tumor growths may be prevented by not smoking, keeping up a healthy weight, not drinking too much alcohol, eating vegetables, immunization against certain diseases, and keeping up an essential separation from sunlight [10-11]. Early identification through screening is vital for tumors [12-13].

In past decades, continuous advancement related to tumor analysis has been presented [14]. Several mathematical principles are used to identify and treat the tumor. Models are being applied to examine how tumors form [15] and develop [16-19]. They are being used to customize current treatment, anticipate the viability [20-24], or the blend of different treatments [25-27] and provide insight into the development of resistance [28-29]. Many ODE models are suggested to address tumor development [30-31] and are consistently utilized to make expectations about the sufficiency of growth medications [32].

The growth of tumor cells is uncertain due to their abnormal behavior. The Rough set method is an emerging interventional technique and the most powerful mathematical tool to deal with unpredictable situations. Metastasis dispersal is the procedure by which a few cells from the tumor leave and make another tumor. Subsequently, the threat can spread through in the entire organism.

In this paper, a scientific model is given for tumor cells population development with the human body's metastasis process and approved by the Rough set in uncertain circumstances. In this methodology, the mathematical analysis of the nonlinear behavior of the tumor cells population is set up via carrying capacity with the human body's metastasis process and is approved by the Rough set. This paper develops a connection between applied mathematics, numerical computation, and applications of biological systems. These advances offer novel

insights for tumor growth, further supporting research in tumor cell dynamics.

2. Mathematical Model

In this segment, a mathematical model is given. This model is centered on the number of tumor cells and carrying capacity. The following assumptions are for the development of the model.

2.1 Assumption taken

In this model, three assumptions taken as follow:

(A1) A per capita tumor cell development subject to tumor size concerning carrying limit is given by the logistic model [14].

(A2) The carrying capacity growth is proportional to the tumor surface [33].

(A3) Metastatic discharge is relative to the 2/3 power of essential tumor estimate, which compares to surficial metastatic outflow [34].

2.2 Dynamics of growth of the number of tumors cells in terms of carrying capacity

To find the number of diseased cells in the tumor in a human body is a hard task because of constant changes in tumor growth over time [14]. The number of cells changes according to time [14],

$$\frac{dB}{dt} = \eta_1 B - \eta_2 B \quad (2.1)$$

where B stands for the number of tumor cells at time t (in days), η_1 and η_2 respectively, are producing and dying tumor cells, $\frac{dB}{dt}$ is the per capita growth rate of tumor cells population and $\eta_1 - \eta_2 = \gamma$ represents the tumor population growth.

A per capita tumor cell development, subject to tumor size, and concerning carrying limit K , is given by the logistic model [33]. So

$$\frac{dB}{dt} = \gamma B \left(1 - \frac{B}{K}\right) . \quad (2.2)$$

Let $f(B) = \gamma \left(1 - \frac{B}{K}\right)$ and thus per capita growth rate of tumor cell decreases as well as increases. In a real-life situation, the growth of cells cannot grow exponentially because, after some time, the cells' growth will reach a constant position. So now from Eq. (2.2),

$$\frac{dB}{dt} = Bf(B) . \quad (2.3)$$

The carrying limit K is considered as a variable representing the tumor cell growth. So

$$\frac{dK}{dt} = \gamma B^{2/3} . \quad (2.4)$$

The fraction $\frac{2}{3}$ is taken because the carrying capacity is proportional to the tumor surface [34]. Here, we can deduce two cases as follows [14]:

- (i) If $\lim_{t \rightarrow \infty} B(t) = K$, then shows that the growth of the tumor cell converges to the carrying capacity.
- (ii) The relative growth $\frac{1}{B} \frac{dB}{dt}$ decreases with the increase of tumor cell. For the solution of the carrying capacity, now from the Eq. (2.5),

$$\frac{dK}{dt} = \gamma \left\{ \frac{KB_0}{B_0 + (K - B_0)e^{-\gamma t}} \right\}^{2/3} .$$

Moreover, when the above equation is solved, we obtain

$$\gamma dt = K^{-2/3} \left[1 + \frac{2}{3} \left(\frac{K}{B_0} - 1 \right) e^{-\gamma t} \right] dK . \quad (2.5)$$

We simplify the Eq. (2.5),

$$\gamma dt = \left[\left(1 - \frac{2}{3} e^{-\gamma t} \right) K^{-2/3} + \frac{2}{3B_0} e^{-\gamma t} K^{1/3} \right] dK .$$

Now, integrating the above equation we obtain

$$\gamma t + c = \left[\left(1 - \frac{2}{3} e^{-\gamma t} \right) 3K^{1/3} + \frac{1}{2B_0} e^{-\gamma t} K^{4/3} \right] \quad (2.6)$$

at $t = 0, K = K_0$.

So, from Eq. (2.7), we obtain

$$c = K_0^{1/3} + \frac{1}{2B_0} K_0^{4/3} .$$

By Eq. (2.6), we obtain

$$\gamma t + c = \left[(aK^{1/3} + bK^{4/3}) \right], \quad (2.7)$$

where $a = 3 \left(1 - \frac{2}{3} e^{-\gamma t} \right)$ and $b = \frac{1}{2B_0} e^{-\gamma t}$.

This implies that

$$\gamma t + c = abK^{4/3} .$$

That gives

$$K = \left[\frac{\gamma t + c}{ab} \right]^{3/4} . \quad (2.8)$$

where $a = 3 \left(1 - \frac{2}{3} e^{-\gamma t} \right)$, $b = \frac{1}{2B_0} e^{-\gamma t}$ and

$$c = K_0^{1/3} + \frac{1}{2B_0} K_0^{4/3} .$$

This shows that K is the maximum number of tumor cells in the human body at any part; it means, it forms a tumor of the maximum size.

2.3 Tumor cells growth with metastasis process

Metastasis dispersal is the strategy by which a couple of cells from the tumor will

leave and make another tumor. In this way, the threat can disperse through the entire organism [34]. This metastasis procedure is in charge of ninety percent of the patients' demise. Here from Eq. (2.7), if

$$\lim_{t \rightarrow \infty} B(t) = K. \tag{2.9}$$

The growth of tumor cells reaches the carrying capacity. So, it can be said that after reaching the state of carrying capacity, a couple of cells from the tumor will leave and make another tumor [34]. In the case of the absence of the dead cells, the tumor cells grow at a rate proportional to the current population of the tumor cells

$$\frac{dB}{dt} = \mu B. \tag{2.10}$$

The above Eq. (2.10) shows the growth of the tumor cell in any other part of the human body since the metastatic discharge is relative to the 2/3 power of essential tumor estimate, which compares to surficial metastatic outflow [34]. Both primary tumor and metastases make new metastases at the rate given by,

$$\gamma(B) = aB^{2/3}. \tag{2.11}$$

Now, we can also write

$$\gamma(B) = a \left[\frac{KB_0}{B_0 + (K - B_0)e^{-\gamma t}} \right]^{2/3}. \tag{2.12}$$

The above equation shows the tumor cells' growth rate in other parts of the human body with the metastasis process. The tumor cells in this part will get the same carrying capacity again like (2.12) and still with the metastasis process; it will reach like (2.8) and so on.

In the above expression, since K is the carrying capacity of tumor cells, that means K is the maximum number of tumor cells in the human body, which shows that it forms the tumor of the maximum size.

3. Result of the Model

Eq. (2.8) shows the expression of the carrying capacity. The number of cells increases with time (days), and after some time growth of the number of tumor cells reaches the constant state (Fig. 1). By changing the integral width, the number of tumor cells increases with time (days), and after some time the constant behavior is shown in Figs. (1)-(3).

If tumor cells' growth reaches the carrying capacity, then a few cells from the tumor leave and make another tumor. In the case of the absence of the dead cells, the tumor cells grow at a rate proportional to the current population of the tumor cells.

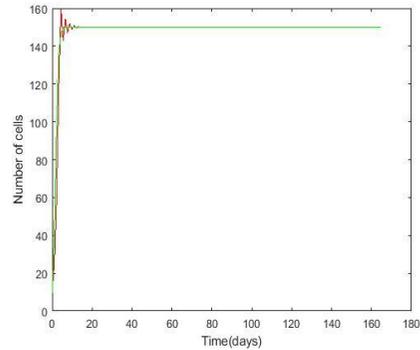


Fig. 1. Tumor cell population growth as a function of time shows that the number of tumor cells increased with time (days), and after some time, the growth of the tumor cells shows constant behavior.

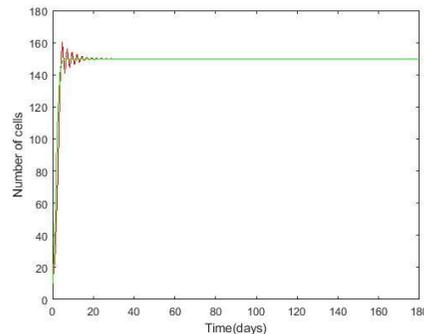


Fig. 2. Tumor cell population growth as a function of time shows that the number of tumor cells increased with time (days), and after some time, the growth of the tumor cells shows constant behavior.

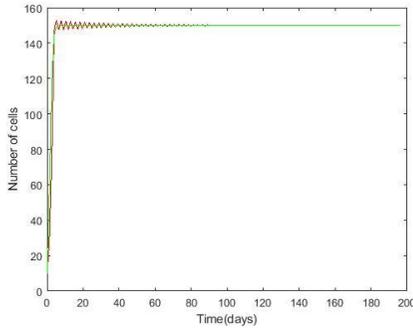


Fig. 3. Tumor cell population growth as a function of time shows that the number of tumor cells increased with time (days), and after some time, the growth of the tumor cells shows constant behavior.

4. The Rough Set

The area of the Rough set applications utilized today is considerably more extensive than before, basically in the zones of the drug, investigation of database traits, and process control. The Rough set has a few covers with different strategies for information examination, e.g., cluster investigation, fuzzy sets, statistics, proof hypothesis. [35-37].

4.1 Validation with the rough set

The data [38] used for describing the model is further relevant because we only require the estimated quantity of tumor cells with time (days) for the validation of the model, and in seeking for the expected quantity of tumor cells, we noticed this preliminary data in the precise form. The data is robust and very proper for work, so we have used it for the model. This work can be utilized in the same type of trial data.

The data [38] of the number of tumor cells and its approximation are taken from the real world, and by using Rough Set Exploration System (RSES 2.2.2) [39], it is observed that the amount of tumor cells grows with time (days), and later sometimes the growth of the tumors cells explicates the consistent form (Fig. 4).

4.2 The mechanism used for the rough set

A data frame comprising information $S = (\rho, \sigma)$, where ρ is the nonempty finite collection of objects and \mathcal{G} is the nonempty finite collection of attributes, $\mathcal{G} \subseteq \rho$ and $\lambda \subseteq \sigma$. The two sets $\lambda_*(\mathcal{G})$ and $\lambda^*(\mathcal{G})$ represent the lower and upper approximation of \mathcal{G} , respectively, and are defined as follows:

$$\lambda_*(\mathcal{G}) = \bigcup_{x \in \rho} \{\lambda(x) : \lambda(x) \subseteq \mathcal{G}\}$$

$$\lambda^*(\mathcal{G}) = \bigcup_{x \in \rho} \{\lambda(x) : \lambda(x) \cap \mathcal{G} \neq \emptyset\}.$$

The set

$$\lambda N_\lambda(\mathcal{G}) = \lambda^*(\mathcal{G}) - \lambda_*(\mathcal{G}),$$

is defined as the boundary region of \mathcal{G} [35-37]. If $\lambda N_\lambda(\mathcal{G}) = \emptyset$ then ν is crisp or exact with respect to λ ; and if $\lambda N_\lambda(\mathcal{G}) \neq \emptyset$, ν is rough or inexact with respect to λ [35-37].

The Rough set depends on the hypothesis that every event is linked to some of the data; during data processing, discretization is a vital tool for dealing with imprecision when applying the Rough set [35-37].

5. Discussion and Conclusion

Tumors are the principal cause of death and put significant weight on the medical practice because of the disease's enduring aspects. The area of the Rough set applications utilized today is considerably more extensive than before, basically in the zones of the drug, investigation of database traits, and process control.

In circumstances of the modern knowledge and former model presented for the tumor cells, the model has included the growth of the tumor cells in terms of carrying capacity and metastasis process.

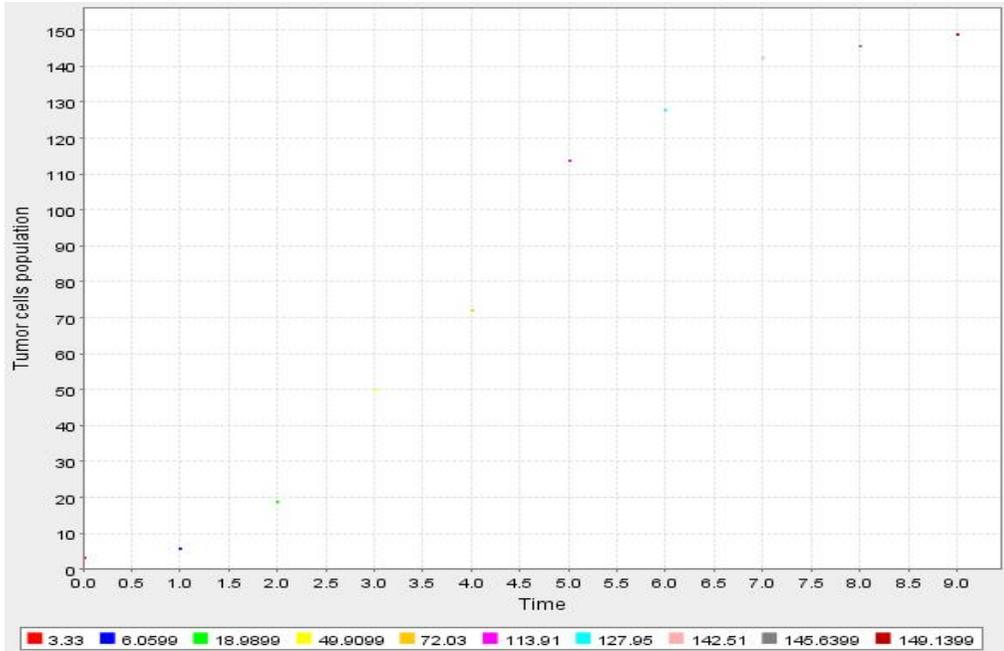


Fig. 4. Tumor cell population growth as a function of time shows that the number of tumor cells increased with time (days), and the growth of the tumor cells show constant behavior by the Rough set exploration system (RSES 2.2.2).

tumor cell growth with the metastasis process, which was approved using the Rough set. We observed the association in the tumor cells and time such that the tumor cells grow, and after some period of time, approach steady-state in a continuous curve graph (Fig. 1). By changing the equidistance width, the tumor cells increase with time (days), and, after some period of time, they shows the same constant behavior in the zigzag curve form in Figs. (1-3). In all cases, the same result is obtained.

In Rough set, by using the Rough Set Exploration System (RSES 2.2.2), we found the association between the tumor cells and time such that the tumor cells grow with time and approach the steady-state (Fig. 4).

The simulation process using MATLAB and the Rough Set Exploration System (RSES 2.2.2) exhibits the same results, i.e., the tumor cells grow with time and approach the steady-state.

Hence, we have established a mathematical model for tumor cells growth with the metastasis process in the human body, validated by the Rough set.

Therefore, these advances offer novel insights for tumor growth, further supporting research in tumor cell dynamics.

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