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Analytical Solution of Cancer Cells Interaction with Virotherapy in the Framework of Fractional Derivative Operator

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Abstract: In this paper, we have introduced a mathematical model of virotherapy for medication of tumor cells by using fractional calculus. In the current model four differential conditions are presented which portray cooperation among non-infected tumor cells, infected tumor cells, virions and effector T cells. We will examine the presence and uniqueness of the introduced mathematical model. We will likewise concentrate on the local stability of equilibrium points. Then, we will discuss the numerical solutions to investigate the use of fractional order derivative and different variables. We will likewise plot a few charts to show the outcomes.

Keywords: Fractional derivative, mathematical modelling, numerical solution, Sumudu transform.

1 Introduction

Cancer is one of the burning issue for both developed and developing nations. It has been seen for low and middle income countries, that most of the population does not have well regulated care system for cancer patients. In our study we found that there are main three types of treatment for Cancer - Chemotherapy, Surgery and Radiotherapy. The whole research done by scientists, mathematicians, doctors etc.,in the field of cancers and its treatments comes under the department Oncology. In research work we found the main cause of cancer is abnormal growth of any cells in the human body. The count of sufferers of cancer is readily increasing day and night. Death number due to cancer chart is increasing per year for the whole world. If we talk about the origin of the cancer, it is seen that it originated from Egypt about 3000. Before Christs. It was found in fossilized bone tumors and human mummies. If we talk about cancer, it was the the Greek Physician Hippocrates who explained the word about cancer.

Oncolytic virotherapy is a treatment of cancer, in this treatment cancerous tumor cells are injected with a virus to infect and break down these cells without killing healthy cells [1]. This cancer treatment also give power to the defense system of body by invigorating the immune system [2]. It attacks to the cells as as a virus normally attack the body and works without chemotherapy drugs or any type of radiation. Due to this, it is not susceptible to the same drug resistance and radiation as common treatments. Because of the way virotherapy works, this type of treatment can be used in conjunction with other treatments, it can be given any one of time as before surgery, after surgery , between radiation, during chemotherapy [2].The median treatment duration of virotherapy is three years with planned follow-up, and oncolytic virotherapy avoids the adverse side effects common to other common cancer treatments such as chemotherapy and radiation [3]. For a virus to be acceptable for oncolytic virotherapy, it must be capable of replication and selective infection [4].

In recent years, many researchers and scientist have given their contribution in the field of cancer treatments. If we study the literature we found a systematic growth of cancer treatment model and this time dependent up gradation give better

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results in that field.For detailed analysis see [3]-[15].
The aim of this article is to examine the dynamics of a system of nonlinear differential equations which are used to study the impact of virotherapy in tumors and the role of the specific immune response on cancer control. Mathematical modelling is quite useful to get perfect direction in the research of oncology. The intricacy of disease cancer lends itself well to quantitative methodologies as it presents difficulties and potentially open doors for new turns of events. Mathematical modeling, thus adds to malignant growth research by assisting with explaining systems and giving quantitative expectations that can be approved. Ongoing development of quantitative models resolves many inquiries connected with cancer inception, movement, and metastasis, as well as intratumoral heterogeneity, therapy reaction, and obstruction. Mathematical modeling of malignant growth supplements our exploratory and clinical examinations, yet in addition challenges flow ideal models, rethinks how we might interpret components, propels tumorigenesis and illuminates future exploration in disease science. Mathematical modeling opens new roads for medication research as well as keeps up with the capacity to tackle what is happening of different sorts of cancer. Oncology likewise makes sense of the circumstance where an unexpected flare-up in the quantity of contaminated patients spreads quickly across the populace and influences a huge piece of the human populace. The study looks to track down the reasons for malignant growth and to distinguish and give further developed medicines. Mathematical modeling encompasses the subjective parts of cancer modelling. In mathematical modeling calculus plays an important role. It has been also observed that fractional calculus also shows its importance in many field of science, engineering, biology etc. Fractional calculus is a branch of mathematics that involves the integration and differentiation of arbitrary order [16]-[20]. The prosperity and improvement of fractional calculus has resulted to large number of developments in the fields of chemical sciences, life sciences, biotechnology, physics, medicinal studies and many more (for applications see [21]-[37]). While studying the literature, we can find many fractional derivative operators with the singular kernel, some of them are-Riemann-Liouville, Liouville-Caputo, etc. [16]-[26].
The idea of non-singular kernel made many more developments in the theory of fractional derivatives and integrals. The first use of non-singular kernel in fractional derivation was given by M. Caputo and M. Fabrizio [35]. In the literature, new definitions of fractional derivatives with non-singular kernel are also found. A survey of academicians shows that the core of the power law is the latency and the scale invariant for the mean square displacement. These developments are necessary for a discussion of fluid flow in a heterogeneous medium. The greatest benefit of the fractional differential-coefficient operator with the non-singular kernel is that it changes the power-stretched exponential as the waiting time.

2 Cancer Cells Interaction with Virotherapy Model For Time Dependent Rate

Kim et al. [13] studied the dynamics of a model which explain the interaction between a tumor cells and oncolytic virus. Their model uses a free virus population which have a virotherapy treatment period as well as growth due to analysis of infected tumor cells. In the cancer cells interaction with Virotherapy model for treatment of cancer, we have thought about the five distinct classes of individuals as u non-infected tumor cells, I infected tumor cells, v virions, τ T-cells, and Π APCs.
By using these parameters Kim et al.[13] gave cancer cells interaction with virotherapy model for time dependent rate [14]

$$\frac{du}{dt} = \alpha u - \beta \frac{uv}{\Lambda} - \omega(\mathrm{I})\frac{u\tau}{\Lambda}, \qquad u(0) = u_0 \tag{1}$$

$$\frac{d\mathrm{I}}{dt} = \beta \frac{uv}{\Lambda} - \chi_{\mathrm{I}}\mathrm{I} - \omega(\mathrm{I})\frac{\mathrm{I}\tau}{\Lambda}, \qquad \mathrm{I}(0) = \mathrm{I}_0 \tag{2}$$

$$\frac{dv}{dt} = U(t) + \gamma\chi_{\mathrm{I}}\mathrm{I} - \chi_v v, \qquad v(0) = v_0 \tag{3}$$

$$\frac{d\tau}{dt} = S_\tau(\mathrm{I}) + p\Pi - \chi_\tau \tau, \qquad \tau(0) = \tau_0 \tag{4}$$

$$\frac{d\Pi}{dt} = S_\Pi(\mathrm{I}) - \chi_\Pi \Pi \qquad \Pi(0) = \Pi_0. \tag{5}$$

where u non-infected tumor cells, I infected tumor cells, v virions, τ T-cells, and Π APCs. In [17], Kim et al. studied the effects of schedules of treatment, doses of various and targeted viruses for behavior of the tumor cell and its population. Abernathy et al. [18], propose the following model describing the contacts between u non-infected tumor cells, I infected tumor cells, v virions, and ε effector T-cells.
We study the mathematical model Nowak, et al.[14] which give mathematical principles of immunology and virology. We

also study the work of Abernathy et al.[15]

$$\begin{array}{ll} \frac{du}{dt} = \alpha u\left(1-\frac{\mathrm{I}+u}{\omega}\right)-\beta uv-\theta_u u\varepsilon, & u(0)=u_0 \\ \frac{d\mathrm{I}}{dt} = \beta uv-\theta_{\mathrm{I}}\mathrm{I}\varepsilon-\chi_{\mathrm{I}}\mathrm{I}, & \mathrm{I}(0)=\mathrm{I}_0 \\ \frac{d\varepsilon}{dt} = c\mathrm{I}-\chi_\varepsilon\varepsilon, & \varepsilon(0)=\varepsilon_0 \\ \frac{dv}{dt} = \Lambda(t)+\gamma\chi_{\mathrm{I}}\mathrm{I}-\chi_v v, & v(0)=v_0. \end{array} \tag{6}$$

The above equation have few coefficients which are defined below

α	Growth rate of non-infected tumor cells
ω	Total carrying capacity of tumor cells
β	growth rate of infected cells
θ_u	Rate of decay of non-infected cells via T-cells
θ_I	Rate of decay of infected cells via T-cells
χ_{I}	Rate of decay for infected cells
χ_ε	Rate of decay for effector T-cells
χ_v	Rate of decay for virions
Λ	The virotherapy dosage
γ	Number of virions released via infected cell lysis
c	Rate of T-cell growth via infected tumor cells

Now to simplify the analysis, Abernathy et al.convert it in non-dimensional model by setting few parameters. The non-dimensional model is

$$\begin{array}{ll} \frac{du}{dt} = u(1-\mathrm{I}-u)-uv-u\varepsilon, & u(0)=u_0 \\ \frac{d\mathrm{I}}{dt} = uv-\theta\mathrm{I}\varepsilon-\chi_{\mathrm{I}}\mathrm{I}, & \mathrm{I}(0)=\mathrm{I}_0 \\ \frac{d\varepsilon}{dt} = c\mathrm{I}-\chi_\varepsilon\varepsilon, & \varepsilon(0)=\varepsilon_0 \\ \frac{dv}{dt} = \Lambda(t)+\gamma\chi_{\mathrm{I}}\mathrm{I}-\chi_v v, & v(0)=v_0. \end{array} \tag{7}$$

3 The Qualitative Analysis of the Model

In this part of the paper, we study the model and find the positive and bounded region for the state variables. We also find the steady state results of the model and stability of these results.

3.0.1 **Boundedness and Positivity of the Model**

Theorem 1.*Consider all the parameters are positive, then in the results of the model $u(t),v(t),I(t),\varepsilon(t)$ are all positive for all $t>0$.*

*Proof.*Let us consider Δ=$Sup\{t>0: u_0,I_0,\varepsilon_0,v_0\geq 0\}$. Now first we consider the equation

$$\frac{du}{dt} = u(1-\mathrm{I}-u)-uv-u\varepsilon,$$

or

$$\frac{du}{dt} = u\phi_1(u,\mathrm{I},\varepsilon,v) \tag{8}$$

where $\phi_1(u,\mathrm{I},\varepsilon,v)=(1-\mathrm{I}-u)-v-\varepsilon$, or

$$\frac{du}{u} = \phi_1(u,\mathrm{I},\varepsilon,v)\,dt \tag{9}$$

on solving we get

$$u(t) = u(0)\exp\left(\int_0^t \phi_1(u,\mathrm{I},\varepsilon,v)ds\right) \tag{10}$$

on using the initial condition, we get

$$u(t) = u_0\exp\left(\int_0^t \phi_1(u,\mathrm{I},\varepsilon,v)ds\right) \tag{11}$$

Now we consider the next equation

$$\frac{d\mathrm{I}}{dt} = uv - \theta\,\mathrm{I}\varepsilon - \chi_{\mathrm{I}}\mathrm{I}, \tag{12}$$

or

$$\frac{d\mathrm{I}}{dt} \geq -\left(\theta\,\varepsilon + \chi_{\mathrm{I}}\right)\mathrm{I}, \tag{13}$$

where

$$\phi_2(\varepsilon) = -\left(\theta\,\varepsilon + \chi_{\mathrm{I}}\right),$$

hence

$$\mathrm{I}(t) \geq \mathrm{I}(0)\exp\left(\int_0^t \phi_2(\varepsilon)ds\right) \tag{14}$$

on using the initial condition, we get

$$\mathrm{I}(t) \geq \mathrm{I}_0\exp\left(\int_0^t \phi_2(\varepsilon)ds\right) \tag{15}$$

Similarly, the third equation is

$$\frac{d\varepsilon}{dt} = c\mathrm{I} - \chi_\varepsilon\varepsilon, \tag{16}$$

or

$$\frac{d\varepsilon}{dt} \geq -\chi_\varepsilon\varepsilon,$$

where

$$\phi_3(\varepsilon) = -\chi_\varepsilon\varepsilon$$

on simplification we get

$$\varepsilon(t) \geq \varepsilon(0)\exp\left(\int_0^t \phi_3(\varepsilon)ds\right) \tag{17}$$

on using the initial condition, we get

$$\varepsilon(t) \geq \varepsilon_0\exp\left(\int_0^t \phi_3(\varepsilon)ds\right) \tag{18}$$

Now we consider the next equation

$$\frac{dv}{dt} = \Lambda(t) + \gamma\chi_{\mathrm{I}}\mathrm{I} - \chi_v v \tag{19}$$

or

$$\frac{dv}{dt} \geq -\chi_v v, \tag{20}$$

on solving we get

$$v(t) \geq v(0)\exp\left(-\chi_v t\right). \tag{21}$$

on using the initial condition, we get

$$v(t) \geq v_0\exp\left(-\chi_v t\right). \tag{22}$$

4 Definition and Pre-Requirements

In this section, we will define some fractional derivative operators those will be used in our work

Definition 1.*[35] Let f is an integrand on $\mathbb{R}$, $t > 0$, $0 < \eta < 1$, the Caputo-Fabrizio fractional differential-coefficient of order η is defined as*

$${}_0^{CF}\zeta_t^{\eta}(f(t)) = \frac{N(\eta)}{1-\eta}\int_0^t exp\left(\frac{-\eta(t-\tau)}{1-\eta}\right)f'(\tau)d\tau. \tag{23}$$

Where ${}_0^{CF}\zeta_t^{\eta}$ addresses the fractional Caputo-Fabrizio differential-coefficient of order η and $N(\eta)$ is a normalization function with respect to $N(0) = N(1) = 1$.

Definition 2.*[35] Let f be an integrable function on $\mathbb{R}$, $t>0$, $0<\eta<1$, the Caputo-Fabrizio time fractional integrand of order η is specified as*

$$^{\mathscr{C}\mathscr{F}}\mathscr{J}^{\eta}{}_{t}(f(t))=\frac{2(1-\eta)}{N(\eta)(2-\eta)}f(t)+\frac{2\eta}{N(\eta)(2-\eta)}\int_{0}^{t}f(\tau)d\tau. \tag{24}$$

Where, $N(\eta)$ is the normalization function with respect to $N(0)=N(1)=1$.

Definition 3.*[23] The Mittag-Leffler function is viewed as the speculation of the exponential function and is characterized as:*

$$E_{\alpha}(t)=\sum_{n=0}^{\infty}\frac{t^{n}}{\Gamma(n\alpha+1)},\quad \alpha>0,\quad \alpha,t\in\mathbb{R}. \tag{25}$$

Definition 4.*Let f(t) be a function of variable t then Sumudu transform of Caputo-Fabrizio fractional differential coefficient of f(t) is*

$$ST({}_{0}^{CF}D_{t}^{\eta})(f(t))=M(\eta)\left[\frac{ST(f(t))-f(0)}{1-\eta+\eta u}\right]. \tag{26}$$

for detail analysis of Sumudu transform see [28],[29],[40].

5 Interconnection of Virotherapy Model with Cancer Cells in the Foundation of Caputo-Fabrizio Differential-Coefficient Operator

We will now extend the cancer cells interaction [39] with virotherapy model by using Caputo-Fabrizio derivative operator. The concepts of fractional derivatives [17]-[20] are extremely valuable in the examination of this present reality issues which show global behaviours. Distinct fractional differential-coefficient operators have their own importance. The fractional differential coefficient with Singular kernel was defined by Riemann-Liouville [17] and this was the first one which has been granted in the area of fractional calculus, after that several enhancements were made in this area which involves definitions of fractional differential coefficient having kernel which are not singular, was presented by Caputo [17]. This definition depends on the idea of power law, therefore it isn't helpful for the issues that indicate the blurring memory process. Caputo ans Fabrizio [35] made some improvements and recommended the definition that can manage the interaction that shows blurring memory process because of the exponential decay accompanied with Delta-Dirac characteristic. For the detailed applications of fractional calculus we can see [21]-[34]. Now, We will present a detailed inspection of this fractional mathematical model in the sense of Caputo-Fabrizio fractional differential-coefficient operators. The solution of cancer cells interaction with virotherapy model and their numerical solution obtained.
In this paper, we consider a time-fractional mathematical model of Cancer Cells Interaction with Virotherapy in the foundation of Caputo-Fabrizo fractional operator

$$\begin{aligned}
&^{\mathscr{C}\mathscr{F}}\zeta_{t}^{\eta}u(t)=u(1-\mathrm{I}-u)-uv-u\varepsilon, && u(0)=u_{0}\\
&^{\mathscr{C}\mathscr{F}}\zeta_{t}^{\eta}\mathrm{I}(t)=uv-\theta\mathrm{I}\varepsilon-\chi_{\mathrm{I}}\mathrm{I}, && \mathrm{I}(0)=\mathrm{I}_{0}\\
&^{\mathscr{C}\mathscr{F}}\zeta_{t}^{\eta}\varepsilon(t)=c\mathrm{I}-\chi_{\varepsilon}\varepsilon, && \varepsilon(0)=\varepsilon_{0}\\
&^{\mathscr{C}\mathscr{F}}\zeta_{t}^{\eta}v(t)=\Lambda(t)+\gamma\chi_{\mathrm{I}}\mathrm{I}-\chi_{v}v, && v(0)=v_{0}.
\end{aligned} \tag{27}$$

where $^{\mathscr{C}\mathscr{F}}\zeta_{t}^{\eta}$ is the Caputo -Fabrizio fractional differential-coefficient of order η. Our target of this paper is to illustrate the analytical solutions of these time fractional cancer model with the help of Sumudu transform iteration method.

6 Analytical Results of the Interaction of Cancer Cells with Virotherapy in the Framework of Fractional Differential-Coefficient Operator Using Sumudu Transform Iteration Method

In this section, we find the analytical results of the interlinkage of cancer cells with virotherapy in the structure of fractional differential-coefficient operator using Sumudu transform iteration method. The exact solution of system of equations describing the model is quite typical. So, we use the Sumudu Transform together with iterative technique.
Now first we take Sumudu transform, we get

$$[ST\left[^{\mathscr{C}\mathscr{F}}\zeta_{t}^{\eta}u(t)\right]=ST\left[u(t)(1-\mathrm{I}(t)-u(t))-u(t)v(t)-u(t)\varepsilon\right],$$

or

$$ST(u(t)) = u(0) + \frac{(1-\eta+\eta p)}{M(\eta)} ST\left[u(t)\left(1-\mathrm{I(t)}-u(t)\right)-u(t)v(t)-u(t)\varepsilon\right],$$

on taking inverse Sumudu Transform, we get

$$u(t) = u_0 + ST^{-1}\left[\frac{(1-\eta+\eta p)}{M(\eta)} ST\left\{u(t)\left(1-\mathrm{I(t)}-u(t)\right)-u(t)v(t)-u(t)\varepsilon\right\}\right], \tag{28}$$

we can obtain rest on the same manner;

$$\mathrm{I}(t) = \mathrm{I}_0 + ST^{-1}\left[\frac{(1-\eta+\eta p)}{M(\eta)} ST\left\{u(t)v(t)-\theta\,\mathrm{I(t)}\varepsilon-\chi_{\mathrm{I}}\mathrm{I(t)}\right\}\right], \tag{29}$$

and

$$\varepsilon(t) = \varepsilon_0 + ST^{-1}\left[\frac{(1-\eta+\eta p)}{M(\eta)} ST\left\{c\mathrm{I}(t)-\chi_{\varepsilon}\varepsilon(t)\right\}\right], \tag{30}$$

also

$$v(t) = v_0 + ST^{-1}\left[\frac{(1-\eta+\eta p)}{M(\eta)} ST\left\{\Lambda\,(t)+\gamma\chi_{\mathrm{I}}\mathrm{I}(t)-\chi_v v(t)\right\}\right]. \tag{31}$$

Then we get the following recurrence relations

$$\begin{aligned} u_{n+1}(t) = u_0 + ST^{-1}\left[\tfrac{(1-\eta+\eta p)}{M(\eta)} ST\left\{\left(u_n(t)-\sum_{m=0}^{n} u_m(t)\mathrm{I}_{n-m}(t)-\sum_{m=0}^{n} u_m(t)u_{n-m}(t)\right)\right.\right. \\ \left.\left.-\sum_{m=0}^{n} u_m(t)v_{n-m}(t)-u_n(t)\varepsilon\right\}\right], \end{aligned} \tag{32}$$

and

$$\begin{aligned} \mathrm{I}_{n+1}(t) = \mathrm{I}_0 \\ +ST^{-1}\left[\tfrac{(1-\eta+\eta p)}{M(\eta)} ST\left\{\sum_{m=0}^{n} u_m(t)v_{n-m}(t)-\theta\sum_{m=0}^{n}\varepsilon_m(t)\mathrm{I}_{n-m}(t)-\chi_{\mathrm{I}}\mathrm{I}_n(t)\right\}\right], \end{aligned} \tag{33}$$

also

$$\varepsilon_{n+1}(t) = \varepsilon_0 + ST^{-1}\left[\frac{(1-\eta+\eta p)}{M(\eta)} ST\left\{c\mathrm{I}_n(t)-\chi_{\varepsilon}\varepsilon_n(t)\right\}\right], \tag{34}$$

as well as

$$v_{n+1}(t) = v_0 + ST^{-1}\left[\frac{(1-\eta+\eta p)}{M(\eta)} ST\left\{\Lambda+\gamma\chi_{\mathrm{I}}\mathrm{I}_n(t)-\chi_v v_n(t)\right\}\right]. \tag{35}$$

Hence we can easily find the result

$$u(t) = \lim_{n\to\infty} u_n(t) \tag{36}$$

$$I(t) = \lim_{n\to\infty} I_n(t) \tag{37}$$

$$\varepsilon(t) = \lim_{n\to\infty} \varepsilon_n(t) \tag{38}$$

and

$$v(t) = \lim_{n\to\infty} v_n(t) \tag{39}$$

7 Numerical Solutions

By using the results defined in equation (32)-(35), we get

$$\begin{aligned} u(0) &= u_0 \\ \mathrm{I}(0) &= \mathrm{I}_0 \\ \varepsilon(0) &= \varepsilon_0 \\ v(0) &= v_0 \end{aligned}$$

and

$$\begin{aligned} u_1(t) &= u_0 + \left[\tfrac{(1-\eta+\eta p)}{M(\eta)}\left(u_0 - u_0\mathrm{I}_0 - {u_0}^2 - u_0 v_0 - \varepsilon u_0\right)\right], \\ \mathrm{I}_1(t) &= \mathrm{I}_0 + \left[\tfrac{(1-\eta+\eta p)}{M(\eta)}\left(u_0 v_0 - \theta\varepsilon_0\mathrm{I}_0 - \chi_{\mathrm{I}}\mathrm{I}_0\right)\right], \\ \varepsilon_1(t) &= \varepsilon_0 + \left[\tfrac{(1-\eta+\eta p)}{M(\eta)}\left(c\mathrm{I}_0 - \chi_\varepsilon\varepsilon_0\right)\right], \\ v_1(t) &= v_0 + \left[\tfrac{(1-\eta+\eta p)}{M(\eta)}\left(\Lambda + \gamma\chi_{\mathrm{I}}\mathrm{I}_0 - \chi_v v_0\right)\right], \\ &\vdots \end{aligned}$$

To get the numerical results of the above discussed model we use the values of distinct variables as carrying capacity (total tumor cells) $3*10^9$, carrying capacity(T-cells per virion per day) 2.9, rate of non-infected becoming infected (per virion per day) $8.9*10^{-13}$, growth rate of non-infected tumor cells (per day) 0.31, rate of decay of non-infected cells via T-cells (per T-cell per day) $1.5*10^{-7}$, rate of decay of infected cells via T-cells (per T-cell per day) $1.5*10^{-7}$, rate of decay for effector T-cells (per cell per day) 0.35, rate of decay for infected cells (per cell per day) 1, rate of decay for virions (per cell per day) 2.3, virions released via infected cell lysis per day 3500, virotherapy dosage virions (per day) varied. We have done some calculations for non-dimensionalized parameter values.Now by using the above mentioned values, we get the numerical outcomes from equation (36)-(39).It is quite useful as we have order of derivative $0 < \eta \leq 1$.

8 Conclusion

By using the Sumudu transform iteration Method, we computed the analytical solutions of Cancer Cells interlinkage with virotherapy in the support of fractional differential-coefficient operator. In this work we have defined a model using the Caputo -Fabrizio fractional differential-coefficient of order η. This work shows the results of the model for fractional order. Which is useful to find the gateway for dosages of an effective virotherapy treatment that are sufficient to reach long term tumor eradication.

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