



# Novel numerical investigation of the fractional oncolytic effectiveness model with M1 virus via generalized fractional derivative with optimal criterion

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## ABSTRACT

Oncolytic virotherapy is an efficacious chemotherapeutic agent that addresses and eliminates cancerous tissues by employing recombinant infections. M1 is a spontaneously produced oncolytic alphavirus with exceptional specificity and powerful activity in individual malignancies. The objective of this paper is to develop and assess a novel fractional differential equation (FDEs)-based mathematical formalism that captures the mechanisms of oncogenic M1 immunotherapy. The aforesaid framework is demonstrated with the aid of persistence, originality, non-negativity, and stability of systems. Additionally, we also examine all conceivable steady states and the requirements that must exist for them to occur. We also investigate the global stability of these equilibria and the characteristics that induce them to be unstable. Furthermore, the Atangana–Baleanu fractional-order derivative is employed to generalize a treatment of the cancer model. This novel type of derivative furnishes us with vital understanding regarding parameters that are widely used in intricate mechanisms. The Picard–Lindelof approach is implemented to investigate the existence and uniqueness of solutions for the fractional cancer treatment system, and Picard’s stability approach is used to address governing equations. The findings reveal that the system is more accurate when the fractional derivative is implemented, demonstrating that the behaviour of the cancer treatment can be interpreted when non-local phenomena are included in the system. Furthermore, numerical results for various configurations of the system are provided to exemplify the established simulation.

## Introduction

Cancer is a group of disorders in which certain of the immune system’s tissues proliferate indefinitely and metastasize to distant organs. Several genetic alterations are responsible for the development of carcinoma. It could begin in practically any part of the anatomy. While old or impaired tissues normally perish, replacement lymphocytes develop only when the tissue does not require them. Excess lymphocytes have the potential to continue to alienate and cause cancer. When a tumour propagates to other parts of the body (metastasis) [1], it becomes hazardous. That is precisely why, in terms of avoiding translocation, it is imperative to discover malignancy as soon as it is practicable.

Therapy for carcinoma is carefully addressed. Liposuction, radiation, chemotherapeutics, gender reassignment, monoclonal antibodies, and virotherapy are some of the anticancer agents that are administered individually or in tandem. Several of the novel therapeutics involve virotherapy, which involves utilizing a microbe that has been reconfigured. The Oncolytic virus is the name given to this infection [2]. Oncolytic infections infiltrate and kill tumour tissues by exploiting the organism’s reproductive mechanism to propagate them and disseminate them to undamaged tissues in the adjacent neighbourhood, (see Fig. 1). Owing to a clinical study, begomovirus M1, a spontaneously generated and discriminating oncogenic infection attacking zinc-finger

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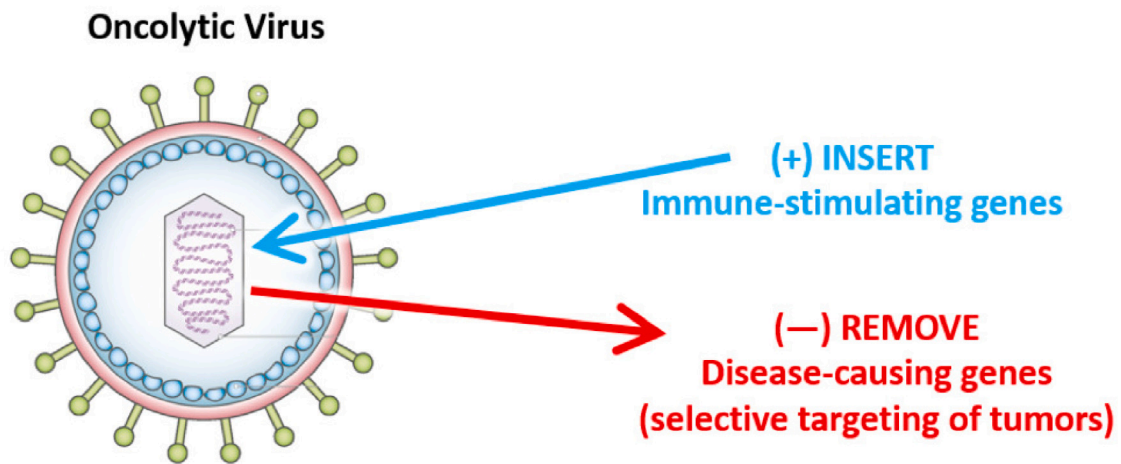


Fig. 1. Oncolytic virus having stimulating and disease-causing genes.

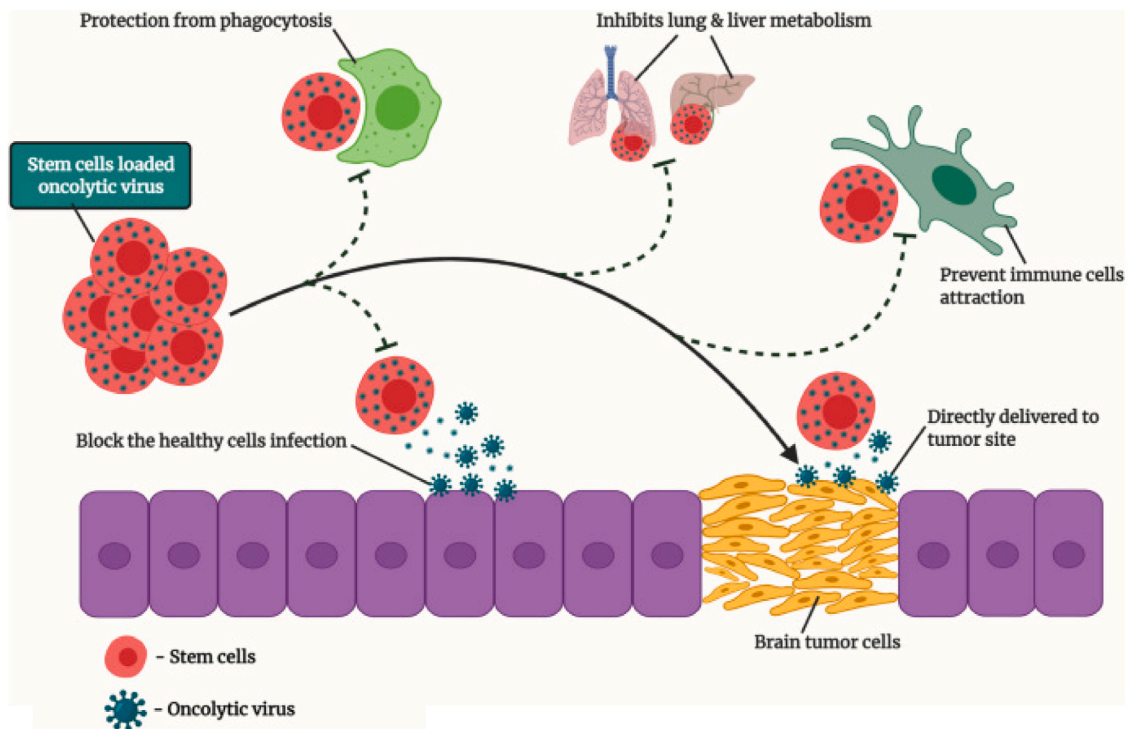


Fig. 2. Relation between the stem cells and oncolytic virus.

antioxidant polypeptide (ZAP) impaired tumour tissues, has significant oncogenic activity and malignant glioma phenotypic expression in cultured cells, in vivo, and ex vivo tests [3]. Wang et al. [4] developed a dynamical framework driven by ordinary DEs that characterize the proliferation of immune tissues, cancer lymphocytes, and the M1 infection under restricted nourishment to simulate the involvement of the M1 infection in oncogenic immunotherapy. Elaiw et al. [5] incorporated spatiotemporal characteristics and an anti-tumour inflammatory system controlled by cytotoxicity *T* lymphoma (CTL) receptors into the system reported in [4]. According to the findings in [5], innate immunity has a detrimental consequence on oncogenic M1 immunotherapy, reducing its efficacy. Oncolytic diseases, like various chemotherapies, have diverse molecular mechanisms that include both internal and external harmful effects on malignant tissues, such as endogenous enzymes, the strengthening of inflammatory microbes, endothelial dysfunction, and neuroplasticity, (Fig. 2) [6,7]. A substantial breakthrough has subsequently been introduced in the implementation arena of fractional

calculus, wherein revolutionary formulations featuring non-singular and non-local kernels are exploited [8–19]. The relatively new attribute recommended tends to make use of the generalized Mittag-Leffler function (M-LF), which includes the central pillar, and the specifics of this pathway frustrate the innovative methodologies to achieve numerous auxiliary intriguing aspects that have been recognized in substantial situations, including mean square compression dependably and broadening variability. Abdeljawad and Baleanu [20] contemplated the qualitative properties of a new nonlocal fractional derivative with M-L nonsingular kernel and its applications. Abdeljawad [21] expounded the fractional operators with generalized M-L kernels and their iterated differintegrals. Jarad et al. [22] discussed a class of ordinary differential equations in the frame of Atangana–Baleanu fractional derivative. Abdeljawad and Al-Mdallal [23] investigated the discrete M-L kernel type fractional difference initial value problem and Gronwall’s inequality. Following its introduction in 2016 by Atangana and Baleanu [24], the innovative fractional derivative operator has also been commonly used

in a variety of areas of science. For a limited space of time, modelling using the AB-fractional derivative results in a complex structure. The M-LF has since been revealed to become a highly powerful and relevant filtration procedure as the strength and exponential principles enable the AB-fractional derivative, in the perspective of Caputo, an inexpensive algebraic tool for modelling extremely sophisticated key scenarios. Because of their phenomenal non-orientation, these formulas are well-known for producing fractional DEs with obvious intentional oddities, such as the Riemann–Liouville and Caputo derivatives [25–31]. We have additionally observed a spike in popularity in numerical modelling within these operators. Nevertheless, calculating these components theoretically causes a slew of processing challenges (see [32–34]).

However, some of the aforementioned scientific methods ignored the reminiscence phenomenon by simply addressing integer-order derivatives. The fractional-order derivative is a valuable device for comprehending recollection and inheritance aspects. For example, Cole [35] exhibited that biological entity cell vesicles exhibit fractional-order transmittance while recollection indicates that the system’s reaction is reliant mostly on the present incarnation as well as on its entire chronology. As a result, despite the fractional derivative, the conventional integer-order derivative does not exhibit this reminiscence phenomenon as it is a localized generator.

The fundamental intention of this investigation is to create a computational formula centred on ABC fractional derivative to analyse how memory affects the mechanics of oncogenic M1 immunotherapy. The innovative approach entails deploying the M1 oncolytic virus to treat cancer, with each phase consisting of four states: the concentrations of nutrients, normal cells, tumour cells, and M1 virus at time  $t$ , respectively. The fundamental aspects of the proposed framework, such as the EU of strategies and experimental validation, are closely examined. A numerical approach relying on Lagrange interpolations is developed to simulate the analysed framework, as well as several biological and scientific explanations are provided. The findings foreshadow the impact of cancer therapy by taking into account M1 oncolytic infection and its interactions.

**Model description and preliminaries**

Now let us review the ABC-fractional derivative operators’ underlying theories and associated ramifications.

**Definition 1 ([24]).** Assume that there be a mapping  $f \in C^1(a, b)$ ,  $b > a$ , with  $0 \leq \varphi \leq 1$ . Then, the AB-fractional derivative is then expressed in Caputo’s viewpoint as shown in:

$${}^a ABC D_t^\varphi f(t) = \frac{ABC(\varphi)}{1-\varphi} \int_a^t \frac{df}{dx} E_\varphi\left(-\frac{\varphi}{1-\varphi}(t-x)^\varphi\right) dx, \tag{1}$$

where  $ABC(\varphi) = 1 - \varphi + \varphi/\Gamma(\varphi)$  indicates normalization function such that  $ABC(0) = ABC(1) = 1$  and  $E_\varphi(z)$  signifies the M-LF presented as

$$E_\varphi(z) = \sum_{\delta=0}^{\infty} \frac{z^\delta}{1+\varphi\delta}, \quad \varphi, \delta \in \mathbb{C}, \quad \Re(\varphi) > 0. \tag{2}$$

**Definition 2 ([24]).** Suppose there be a function following AB-fractional integral form of  $f \in C^1(a, b)$  is as shown in:

$${}^a AB I_t^\varphi f(t) = \frac{1-\varphi}{ABC(\varphi)} f(t) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_a^t f(x)(t-x)^{\varphi-1} dx. \tag{3}$$

Also, the relation between AB-fractional derivative and the Sumudu transform is presented as follows:

$$ST\{D_t^\varphi f(t)\} = \frac{ABC(\varphi)}{1-\varphi} \left(\varphi\Gamma(\varphi+1)E_\varphi\left(\frac{-\mu^\varphi}{1-\varphi}\right)\right) \{ST(f(t)) - f(0)\}. \tag{4}$$

**Proposition 1 ([20]).** For  $f \in C^1(a, b)$ , then the AB-fractional derivative and integral operator holds the Newton–Leibniz identity:

$${}^a AB I_t^\varphi ({}^a ABC D_t^\varphi f(t)) = f(t) - f(a). \tag{5}$$

**Lemma 1 ([24]).** Suppose there be a continuous mapping defined on  $[a, b]$ . Then, the following variant holds true:

$$\|{}^{ABR} D_t^\varphi f(t)\| < \frac{ABC(\varphi)}{1-\varphi} \|f(z_1)\|, \tag{6}$$

where  $\|f(z_1)\| = \max_{z_1 \in [a,b]} |f(z_1)|$ .

**Theorem 1 ([36,37]).** Assume the subsequent time-fractional ordinary DE:

$${}^a ABC D_t^\varphi f(t) = g(t) \tag{7}$$

then (7) has unique solution attaining by applying inverse Laplace transform and convolution property as

$$f(t) = \frac{1-\varphi}{ABC(\varphi)} g(t) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\varsigma)^{\varphi-1} g(\varsigma) d\varsigma. \tag{8}$$

Our next result is the generalized mean value theorem.

**Lemma 2 ([20]).** Suppose  $g(x) \in C[a_1, \bar{b}]$  and considering  ${}^a ABC D_t^\varphi g(x) \in C[\bar{a}, \bar{b}]$  when  $\varphi \in (0, 1)$ . Then we have  $g(x) = g(\bar{a}) + \frac{1}{\Gamma(\varphi)} {}^a ABC D_t^\varphi g(\eta)(x-\bar{a})^\varphi$ , when  $0 \leq \eta \leq x, \forall x \in (\bar{a}, \bar{b})$ .

In view of Lemma 2, if  $g(x) \in [0, \bar{b}]$ ,  ${}^a ABC D_t^\varphi g(x) \in (0, \bar{b})$  and  ${}^a ABC D_t^\varphi g(x) \geq 0, \forall x \in (0, \bar{b}), \varphi \in (0, 1)$ , then there be a mapping  $g(x)$  is nondecreasing and if  ${}^a ABC D_t^\varphi g(x) \leq 0, \forall x \in (0, \bar{b})$ , then the mapping  $g(x)$  is nonincreasing  $\forall x \in (0, \bar{b})$ .

Here, we shall get ready to work on the model’s development presented by Wang et al. [4]. The following is the general paradigm for ongoing study:

$$\begin{cases} \frac{dS}{dt} = \Xi - \lambda S(t) - \rho_1 S(t)R(t) - \rho_2 S(t)Q(t), \\ \frac{dR}{dt} = \delta_1 \rho_1 S(t)R(t) - (\lambda + \epsilon_1)R(t), \\ \frac{dQ}{dt} = \delta_2 \rho_2 S(t)Q(t) - (\lambda + \epsilon_2)Q(t) - \rho_3 Q(t)X(t), \\ \frac{dX}{dt} = \Theta + \delta_3 \rho_3 Q(t)X(t) - (\lambda + \epsilon_3)X(t), \end{cases} \tag{9}$$

where  $\mathcal{N}(t) = S(t) + R(t) + Q(t) + X(t)$  which represents that  $0 \leq \mathcal{N}(t) \leq \frac{\Xi}{\lambda} + \mathcal{N}(0) \exp(-\lambda t)$  with  $\mathcal{N}(0)$  in the initial value. Thus,  $0 \leq \mathcal{N}(t) \leq \Xi/\lambda$ , as  $t \mapsto \infty$ .

Further, we consider the following ABC-fractional derivative model in the form of DEs to be presented as follows:

$$\begin{cases} {}^a ABC D_t^\varphi S(t) = \Xi - \lambda S(t) - \rho_1 S(t)R(t) - \rho_2 S(t)Q(t), \\ {}^a ABC D_t^\varphi R(t) = \delta_1 \rho_1 S(t)R(t) - (\lambda + \epsilon_1)R(t), \\ {}^a ABC D_t^\varphi Q(t) = \delta_2 \rho_2 S(t)Q(t) - (\lambda + \epsilon_2)Q(t) - \rho_3 Q(t)X(t), \\ {}^a ABC D_t^\varphi X(t) = \Theta + \delta_3 \rho_3 Q(t)X(t) - (\lambda + \epsilon_3)X(t), \end{cases} \tag{10}$$

where  $S(t)$ ,  $R(t)$ ,  $Q(t)$  and  $X(t)$  are the specific dietary, regular cells, cancer hepatocytes, and M1 viral contents at time  $t$ , respectively.  $\Xi$  and  $\Theta$  are the enlistment variables for nutritional and M1 viral levels, respectively. In addition,  $\Theta$  denotes the lowest efficacious dose of drug. The material is used at frequencies of  $\rho_1 SR$  and  $\rho_2 SQ$ , respectively, by regular and malignant tissues. The population development of regular cellular components of absorbing the food is  $\delta_1 \rho_1 SR$ , whereas the proliferation speed of cancer cells is  $\delta_2 \rho_2 SQ$ . The viral penetrates and eliminates cancerous tissues at a frequency of  $\rho_3 QX$ , and it reproduces at a frequency of  $\delta_3 \rho_3 QX$ . The similar effects of food and bacterium discharge are represented by the factor  $\lambda$ . The spontaneous mortality rates of immune tissues, cancerous cells, and M1 infection are represented by the variables  $\epsilon_1, \epsilon_2$  and  $\epsilon_3$ . The ABC fractional derivative having  $\varphi \in (0, 1]$  is denoted by the symbol  ${}^a ABC D_t^\varphi$  which illustrates the higher capacity.

It is worth noting that the ODE mathematical analysis for proving the M1 virus’s high oncogenic efficiency [4] is a particular instance of the system provided by component (10), and it is enough to choose  $\varphi = 1$ . Additionally, we suppose that the starting requirements (11) of (10) are met to argue that our system is scientifically well-posed:

$$S(0) = \phi_1(0) \geq 0, \quad R(0) = \phi_2 \geq 0, \quad Q(0) = \phi_3 \geq 0, \quad X(0) = \phi_4 \geq 0. \tag{11}$$

**Table 1**  
Table of listed components and associated interpretations.

Parameters	Explanation	Data estimated [38]
$S(t)$	Level of nutritional concentration in time $t$	
$R(t)$	Number of normal cells in time $t$	
$Q(t)$	Number of tumour cells in time $t$	
$X(t)$	Number of M1 virus in time $t$	
$\Xi$	Nutrient recruitment rate	0.02
$\Theta$	Minimum effective dosage of M1 virus	0.01
$\lambda$	Washout constant rate of nutrient and bacteria	0.02
$\epsilon_1$	Natural death rate constants of normal cells	0.01
$\epsilon_2$	Natural death rate of tumour cells	0.008
$\epsilon_3$	Natural death rate of M1 virus	0.01
$\rho_1$	Rate of normal cells after consuming the nutrients	0.03
$\rho_2$	Rate of tumour cells after consuming the nutrients	0.03
$\rho_3$	Rate of virus infects and kills tumour cells	0.01
$\delta_1$	Typical cell growth rate as a function of nutrition consumption	0.8
$\delta_2$	Growth rate of tumour cells as a result of consuming the nutrient	0.5
$\delta_3$	Replicates rate	0.8

**Theorem 2.** The domain of the AB system (10) that is outbreaks-sustainable is determined by

$$\bar{\mathcal{D}} =: \left\{ (S, R, Q, X) \in \mathbb{R}_+^4 : 0 \leq S + R + Q + X \leq \mathcal{N} \leq \frac{\Xi}{\lambda} \right\}. \tag{12}$$

The validity and originality of scenario (10) have now been established, and all that is required to demonstrate that the collection specified in (12) is positively consistent. The demonstration of Theorem 2 will be developed on the basis of a subsequent argument.

**Theorem 3.** The solution of the proposed fractional-order model (10) along ICs is unique and bounded in  $\bar{\mathcal{D}}$ .

**Proof.** The existence and uniqueness of the solution of system (10) on the time interval  $(0, \infty)$  can be obtained by the process discussed in the work of Lin [39]. Subsequently, we have to explain the non-negative region  $\mathbb{R}_+^4$  is positively invariant region. From model (10), we find

$$\begin{cases} {}_0^{ABC}D_t^\varphi S \Big|_{S=0} = \Xi \geq 0, \\ {}_0^{ABC}D_t^\varphi R \Big|_{R=0} = 0, \\ {}_0^{ABC}D_t^\varphi Q \Big|_{Q=0} = 0, \\ {}_0^{ABC}D_t^\varphi X \Big|_{X=0} = \Theta \geq 0. \end{cases} \tag{13}$$

If  $(S, R, Q, X) \in \mathbb{R}_+^4$ , the solution  $[S, R, Q, X]$  cannot escape from the hyperplanes  $S = 0$ ,  $R = 0$ ,  $Q = 0$ , and  $X = 0$ . Also, on each hyperplane bounding the non-negative orthant, the vector field points into  $\mathbb{R}_+^4$ , i.e., the domain  $\mathbb{R}_+^4$  is a positively invariant set.  $\square$

**Lemma 3.** The oncolytic efficacy model (10) with non-negative ICs in region  $\bar{\mathcal{D}}$  is positively invariant

**Proof.** By adding the human population in a model (10), the rate of change of total population is,

$$\begin{aligned} {}_0^{ABC}D_t^\varphi \mathcal{N}(t) &= (\Xi + \Theta) - \lambda S(t) - \rho_1(1 - \delta_1)S(t)R(t) \\ &\quad - \rho_2(1 - \delta_2)S(t)Q(t) - \rho_3(1 - \delta_3)Q(t)X(t) \\ &\quad - (\lambda + \epsilon_1)R - (\lambda + \epsilon_2)Q - (\lambda + \epsilon_3)X \\ &\leq \Xi - \lambda \mathcal{N}(t) \end{aligned}$$

Implementing the Laplace transform yields to

$$\begin{aligned} \mathcal{L} \left\{ {}_0^{ABC}D_t^\varphi \mathcal{N}(t) + \lambda \mathcal{N}(t) \right\} &\leq \mathcal{L} \{ \Xi \} \\ \mathcal{L}(\mathcal{N}) \left( (1 - \sigma)s^\varphi - \frac{\varphi \sigma}{1 - \varphi} \right) - s^{\varphi-1} \mathcal{N}(0) &\leq \frac{1 - \varphi}{ABC(\varphi)} \left( s^\varphi + \frac{\varphi}{1 - \varphi} \right) \frac{\Xi}{s} \\ &\leq \left( 1 - \frac{\varphi \sigma}{(1 - \sigma)(1 - \varphi)} \right)^{-1} \left\{ \frac{1 - \varphi}{(1 - \sigma)(1 - \varphi)} \left( \frac{1 - \varphi + \varphi s^{-\varphi}}{1 - \varphi} \right) \frac{\Xi}{s} + \frac{\mathcal{N}(0)}{s(1 - \sigma)} \right\}, \end{aligned}$$

where  $\sigma = \frac{\lambda(\varphi-1)}{ABC(\varphi)}$ . The response is provided by employing the inverse Laplace transform as follows:

$$\begin{aligned} \mathcal{N}(t) &= \frac{\Xi}{\lambda} - \frac{\Xi}{\lambda(1 - \sigma)} \frac{d}{dt} \int_0^t E_\varphi \left( \frac{\sigma \varphi}{(1 - \sigma)(1 - \varphi)} (t - x)^\varphi \right) dx \\ &\quad + \frac{\mathcal{N}(0)}{1 - \sigma} E_\varphi \left( \frac{\sigma \varphi}{(1 - \sigma)(1 - \varphi)} t^\varphi \right), \end{aligned}$$

where  $E_{\varphi_1, \varphi_2}$  indicates the M-L function. Considering the assumption that the M-L function exhibits asymptotic characteristics, we have

$$E_{\varphi_1, \varphi_2}(\theta) \approx \sum_{k=1}^{\infty} \theta^{-k} / \Gamma(\varphi_2 - \varphi_1 k) + \mathcal{O}(|\theta|^{-1-w}), \quad |\theta| \mapsto \infty, \quad \frac{\varphi_2^\xi}{2} < |Arg \theta| \leq \xi,$$

it is not hard to perceive that  $\mathcal{N}(t) \mapsto \Xi/\lambda$  as  $t \mapsto \infty$ . Ultimately, (12) is the biologically viable in the desired domain of system (10).  $\square$

*Equilibrium points (EPs) and stability results*

Next, we shall figure out where our system (10) has EPs. Any equilibrium position of framework (10), by definition, meets the required algebraic expressions:

$$\Xi - \lambda S(t) - \rho_1 S(t)R(t) - \rho_2 S(t)Q(t) = 0, \tag{14}$$

$$\delta_1 \rho_1 S(t)R(t) - (\lambda + \epsilon_1)R(t) = 0, \tag{15}$$

$$\delta_2 \rho_2 S(t)Q(t) - (\lambda + \epsilon_2)Q(t) - \rho_3 Q(t)X(t) = 0, \tag{16}$$

$$\Theta + \delta_3 \rho_3 Q(t)X(t) - (\lambda + \epsilon_3)X(t) = 0, \tag{17}$$

Utilizing (15), this shows that  $R = 0$  or  $S = (\lambda + \epsilon_1)/\delta_1 \rho_1$ . Analogously, (16) allows to  $Q = 0$  or  $\delta_2 \rho_2 S = \lambda + \epsilon_2 + \rho_3 X$ :

(a) Choosing  $R = 0$  and  $Q = 0$ , then  $S = \Xi/\lambda$  and  $X = \Theta/(\lambda + \epsilon_3)$ . Hence, model (10) has an EP

$$\mathcal{E}_0 = (S_0, 0, 0, X_0) = \left( \Xi/\lambda, 0, 0, \Theta/(\lambda + \epsilon_3) \right).$$

(b) Choosing  $R \neq 0$  and  $Q = 0$ , then  $S = \lambda + \epsilon_1/\delta_1 \rho_1$ ,  $X = \Theta/(\lambda + \epsilon_3)$  and  $R = (\Phi_1 - 1)d/\rho_1$ , where

$$\Phi_1 = \frac{\Xi \delta_1 \rho_1}{\lambda(\lambda + \epsilon_1)}. \tag{18}$$

This figure shows the tendency of immune tissues to process food. It is known as the absorbing quantity [4]. When  $\Phi_1 > 1$ , then (10) has an EP

$$\mathcal{E}_1(S, R, 0, X) = \left( (\lambda + \epsilon_1)/\delta_1 \rho_1, (\Phi_1 - 1)d/\rho_1, 0, \Theta/(\lambda + \epsilon_3) \right).$$

(c) Choosing  $R = 0$  and  $Q \neq 0$ , then  $S = (\rho_3 X + \lambda + \epsilon_2)/\delta_2 \rho_2$ ,  $Q = -(\lambda + \epsilon_2)/(\delta_2 \rho_2 - \rho_3 X)$  and

$$b_1 X^2 + b_2 X + b_3 = 0, \tag{19}$$

where  $\bar{a} = \rho_3(\delta_3\rho_3\lambda + \rho_2(\lambda + \epsilon_3))$ ,  $a_2 = c_1(\lambda + \epsilon_2)/\rho_3 - \rho_2\rho_3(\Theta + \delta_2\delta_3\Xi)$ ,  $c_3 = -\Theta\rho_2(\lambda + \epsilon_2)$ . Note that  $c_1 > 0$  and  $a_3 < 0$ , therefore, we have  $D = c_2^2 - 4c_1c_3$ . Therefore, (19) has two roots as  $\mathbf{X}_\pm$ . It is clear that as  $\mathbf{X}_+ > 0$  and  $\mathbf{X}_- < 0$ , whenever  $\mathbf{X} > 0$ , the  $\mathbf{X} = \mathbf{X}_+$ . It suffices that  $\mathbf{S} > 0$ . Also,  $\mathbf{Q} \implies \Phi_2 > 1 + \Theta\rho_3/(\lambda + \epsilon_2)(\lambda + \epsilon_3)$ , where

$$\Phi_2 = \frac{\Xi\delta_2\rho_2}{\lambda(\lambda + \epsilon_2)}. \tag{20}$$

This calculation gives the cancer tissues' capability of accumulating nutrition. It is also described as the amount of resources absorbed by cancer cells. So, the model (10) has also EP when  $\Phi_2 > 1 + \Theta\rho_3/(\lambda + \epsilon_2)(\lambda + \epsilon_3)$ . This EP is indicated by

$$\mathcal{E}_2(\mathbf{S}_2, 0, \mathbf{Q}_2, \mathbf{X}_2) = \left( (\rho_3\mathbf{X}_2 + \lambda + \epsilon_2/\delta_2\rho_2), 0, -\lambda/\rho_2 + \Xi\delta_2/(\epsilon_2 + \lambda + \rho_3\mathbf{X}_2), \mathbf{X}_+ \right).$$

(d) Choosing  $\mathbf{R} \neq 0$  and  $\mathbf{Q} \neq 0$ , then  $\mathbf{S} = (d + \epsilon_1)/\delta_1\rho_1$  and  $\mathbf{X} = (\lambda + \epsilon/\rho_3)(\Phi_2/\Phi_1 - 1)$  as  $\mathbf{X} > 0 \implies \Phi_2 > \Phi_1$ .

Utilizing (17), we attain  $\mathbf{Q} = \frac{\mathbf{X}(\lambda + \epsilon_3) - \Theta}{\delta_3\rho_3\mathbf{X}}$ . Analogously,  $\mathbf{Q} > 0$  allows TO  $\Phi_2 < \Phi_1 + \frac{\Xi\delta_1\rho_1\rho_2}{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$ . Plugging  $\mathbf{S}$  and  $\mathbf{Q}$  in (14), we get

$$\mathbf{R} = \frac{(\lambda + \epsilon_2)(\Phi_2/\Phi_1 - 1)(\Xi\delta_1\delta_2\rho_1\rho_3 - \delta_3\rho_3\lambda(\lambda + \epsilon_1) - \rho_2(\lambda + \epsilon_1)(\lambda + \epsilon_3))}{\delta_3\rho_1\rho_3(\Phi_2/\Phi_1 - 1)(\lambda + \epsilon_1)(\lambda + \epsilon_2)} + \frac{\rho_2\rho_3\Theta}{\delta_3\rho_1\rho_3(\Phi_2/\Phi_1 - 1)(\lambda + \epsilon_2)} \tag{21}$$

This demonstrates that model (10) has another EP, which is presented as

$$\mathcal{E}_3(\mathbf{S}_3, \mathbf{R}_3, \mathbf{Q}_3, \mathbf{X}_3) = \left( \frac{\lambda + \epsilon_1}{\delta_1\rho_1}, \frac{(\lambda + \epsilon_2)(\Phi_2/\Phi_1 - 1)(\Xi\delta_1\delta_2\rho_1\rho_3 - \delta_3\rho_3\lambda(\lambda + \epsilon_1) - \rho_2(\lambda + \epsilon_1)(\lambda + \epsilon_3))}{\delta_3\rho_1\rho_3(\Phi_2/\Phi_1 - 1)(\lambda + \epsilon_1)(\lambda + \epsilon_2)}, \frac{\rho_2\rho_3\Theta}{\delta_3\rho_1\rho_3(\Phi_2/\Phi_1 - 1)(\lambda + \epsilon_2)}, \frac{\mathbf{X}_3(\lambda + \epsilon_3) - \Theta}{\delta_3\rho_3\mathbf{X}_3}, \frac{(\lambda + \epsilon_2)(\Phi_2/\Phi_1 - 1)}{\rho_3} \right). \tag{22}$$

The accompanying outcome summarizes all of the preceding situations.

**Theorem 4.** Suppose there be  $\Phi_1$  and  $\Phi_2$  described in (18) and (20). Then

(a1) Model (10) is generally in a non-competitive equilibria  $\mathcal{E}_0(\mathbf{S}_0, 0, 0, \mathbf{X}_0)$ .

(b1) Model (10) is generally in a non-cancer equilibria  $\mathcal{E}_1(\mathbf{S}, \mathbf{R}, 0, \mathbf{X})$  when  $\Phi_1 > 1$ .

(c1) Model (10) is generally in a therapeutic breakdown equilibria  $\mathcal{E}_2(\mathbf{S}_2, 0, \mathbf{Q}_2, \mathbf{X}_2)$  when  $\Phi_2 > 1 + \frac{\rho_2\Theta}{(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$ .

(d1) Model (10) is generally in a limited accomplishment equilibria  $\mathcal{E}_3(\mathbf{S}_3, \mathbf{R}_3, \mathbf{Q}_3, \mathbf{X}_3)$  when  $\Phi_2 > \Phi_1 + \frac{\delta_1\rho_1\rho_3\Xi\Theta}{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$ .

The experimental investigation of the equilibria  $\mathcal{E}_0, \mathcal{E}_1, \mathcal{E}_2$  and  $\mathcal{E}_3$  is the core objective of this segment.

**Theorem 5.** Suppose there be a non-competitive equilibria  $\mathcal{E}_0$  is globally asymptotically stable for

$$\Phi_2 \leq 1 + \frac{\Theta\rho_3}{(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$$

or  $\Phi_1 \leq 1$ . Furthermore, it is unstable if

$$\Phi_2 > 1 + \frac{\Theta\rho_3}{(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$$

or  $\Phi_1 > 1$ .

**Proof.** To prove the consequence for non-competitive equilibria, for this, suppose the following Lyapunov functional depend on the parameter  $\varphi = x - 1 - \ln x, \forall x > 0$ :

$$\Delta(t) = \mathbf{S}_0\wp\left(\frac{\mathbf{S}(t)}{\mathbf{S}_0}\right) + \frac{1}{\delta_2\delta_3}\mathbf{X}_0\wp\left(\frac{\mathbf{X}(t)}{\mathbf{X}_0}\right) + \frac{1}{\delta_1}\mathbf{R}(t) + \frac{1}{\delta_2}\mathbf{Q}(t),$$

The derivative of  $\Theta$  in the direction of model (10) is presented by

$$\frac{d\Delta}{dt} \leq \left(\frac{\mathbf{S} - \mathbf{S}_0}{\mathbf{S}}\right)\frac{d\mathbf{S}}{dt} + \frac{1}{\delta_2\delta_3}\left(\frac{\mathbf{X} - \mathbf{X}_0}{\mathbf{X}}\right)\frac{d\mathbf{X}}{dt} + \frac{1}{\delta_1}\frac{d\mathbf{R}}{dt} + \frac{1}{\delta_2}\frac{d\mathbf{Q}}{dt}$$

$$= \left(\frac{\mathbf{S} - \mathbf{S}_0}{\mathbf{S}}\right)(\Xi - \mathbf{S}\lambda - \rho_1\mathbf{S}\mathbf{R} - \rho_2\mathbf{S}\mathbf{T} - 1) + \frac{1}{\delta_2\delta_3}\left(\frac{\mathbf{X} - \mathbf{X}_0}{\mathbf{X}}\right)(\Theta + \delta_3\rho_3\mathbf{Q}\mathbf{X} - (\lambda + \epsilon_3)\mathbf{X}) + \frac{1}{\delta_1}(\delta_1\rho_1\mathbf{S}\mathbf{R} - (\lambda + \epsilon_1)\mathbf{R}) + \frac{1}{\delta_2}(\delta_2\rho_2\mathbf{S}\mathbf{Q} - (\lambda + \epsilon_2)\mathbf{Q} - \rho_3\mathbf{Q}\mathbf{X}).$$

Utilizing  $\mathbf{S}_0 = \Xi/\lambda$  and  $\mathbf{X}_0 = \Theta/(\lambda + \epsilon_3)$ , we find

$$\frac{d\Delta}{dt} \leq \frac{-\lambda}{\mathbf{S}}(\mathbf{S} - \mathbf{S}_0)^2 + \mathbf{R}(\Phi_1 - 1)\frac{\lambda + \epsilon_1}{\delta_1} - \frac{\lambda + \epsilon_3}{\delta_2\delta_3}\frac{(\mathbf{X} - \mathbf{X}_0)^2}{\mathbf{X}} + \frac{\lambda + \epsilon_2}{\delta_2}\left(\Phi_2 - 1 - \frac{\Theta\rho_3}{(\lambda + \epsilon_2)(\lambda + \epsilon_3)}\right).$$

This illustrates that  $\frac{d\Delta}{dt} \leq 0$  when  $\Phi_1 \leq 1$  and  $\Phi_2 \leq 1 + \frac{\Theta\rho_3}{(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$ . It is clear that  $\frac{d\Theta}{dt} = 0$  iff  $\mathbf{S} = \mathbf{S}_0, \mathbf{R} = 0, \mathbf{Q} = 0$  and  $\mathbf{X} = \mathbf{X}_0$ . Thus, the maximum invariant set is in  $\{(\mathbf{S}, \mathbf{R}, \mathbf{Q}, \mathbf{X}) \mid \frac{d\Delta}{dt} = 0\}$  is the set  $\mathcal{E}_0$ . Taking into consideration Lasalle's invariance theorem [40], conclude that  $\mathcal{E}_0$  is globally asymptotically stable for  $\Phi_1 \leq 1$  and  $\Phi_2 \leq 1 + \frac{\Theta\rho_3}{(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$ .

In case  $\mathcal{E}_0$  has a dynamical characteristic, it has to be examined further. Whenever  $\Phi_1 > 1$  or  $\Phi_2 > 1 + \frac{\Theta\rho_3}{(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$ . We do this by computing the characteristic equation at  $\mathcal{E}_0$ , which is supplied by

$$v_1 = -\lambda, v_2 = -\lambda - \epsilon_3, v_3 = \frac{(\Phi_2 - 1)(\lambda + \epsilon_2) - \rho_3\Theta}{\lambda + \epsilon_3}, v_4 = (\Phi_1 - 1)(\lambda + \epsilon_1). \tag{23}$$

From above, we observe that  $v_1, v_2 < 0$  and  $v_3 > 0$  as  $\Phi_2 \leq 1 + \frac{\Theta\rho_3}{(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$  and  $v_4 > 0$  if  $\Phi_1 > 1$ . Finally,  $\mathcal{E}_0$  is unstable if  $\Phi_1 > 1$  or  $\Phi_2 > 1 + \frac{\Theta\rho_3}{(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$ . □

In an analogous manner, we can find the stability criteria for other EPs  $\mathcal{E}_1, \mathcal{E}_2$  and  $\mathcal{E}_3$ .

**Theorem 6.** Assume that  $\Phi_1 > 1$ . Then the tumour-free equilibrium  $\mathcal{E}_1$  is globally asymptotically stable if

$$\Phi_2 \leq \Phi_1 + \frac{\Xi\Theta\delta_1\rho_1\rho_3}{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)(\lambda + \epsilon_3)}, \tag{24}$$

otherwise it is unstable.

**Proof.** Assume the subsequent Lyapunov functional:

$$\Delta_1(t) = \mathbf{S}_1\wp\left(\frac{\mathbf{S}(t)}{\mathbf{S}_1}\right) + \frac{1}{\delta_1}\mathbf{R}_1\wp\left(\frac{\mathbf{R}(t)}{\mathbf{R}_1}\right) + \frac{1}{\delta_2}\mathbf{Q}_1(t) + \frac{1}{\delta_2\delta_3}\mathbf{X}_1\wp\left(\frac{\mathbf{X}(t)}{\mathbf{X}_1}\right). \tag{25}$$

Therefore we have

$$\frac{d\Delta_1}{dt} \leq \left(1 - \frac{\mathbf{S}_1}{\mathbf{S}}\right)(\Xi - \lambda\mathbf{S} - \rho_1\mathbf{S}\mathbf{R} - \rho_2\mathbf{S}\mathbf{Q}) + \frac{1}{\delta_1}\left(1 - \frac{\mathbf{R}_1}{\mathbf{R}}\right)(\delta_1\rho_1\mathbf{S}\mathbf{R} - (\lambda + \epsilon_1)) + \frac{1}{\delta_2}(\delta_2\rho_2\mathbf{S}\mathbf{Q} - (\lambda + \epsilon_2)\mathbf{Q} - \rho_3\mathbf{Q}\mathbf{X}) + \frac{1}{\delta_2\delta_3}\left(1 - \frac{\mathbf{X}_1}{\mathbf{X}}\right)(\Theta + \delta_3\rho_3\mathbf{Q}\mathbf{X} - (\lambda + \epsilon_3)\mathbf{X}). \tag{26}$$

Setting  $\mathbf{X}_1 = \Theta/(\lambda + \epsilon_3)$  and  $\mathbf{S}_1 = (\lambda + \epsilon_2)/\delta_1\rho_1$ , we get

$$\frac{d\Delta_1}{dt} \leq \lambda\left(1 - \frac{\mathbf{S}_1}{\mathbf{S}}\right)^2 + \rho_1\mathbf{S}_1\mathbf{R}_1\left(\frac{2\mathbf{S}\mathbf{S}_1 - \mathbf{S}_1^2 - \mathbf{S}^2}{\mathbf{S}\mathbf{S}_1}\right) + \left(\rho_1\mathbf{S}_1 - \frac{\lambda + \epsilon_1}{\delta_1}\right)\mathbf{R} + \left(\frac{\rho_2(\lambda + \epsilon_1)}{\delta_1\rho_1} - \frac{\lambda + \epsilon_2}{\delta_2} - \frac{\Theta\rho_3}{\delta_2(\lambda + \epsilon_3)}\right)\mathbf{Q} + \frac{\Theta}{\delta_2\delta_3}\left(\frac{2\mathbf{X}\mathbf{X}_1 - \mathbf{X}_1^2 - \mathbf{X}^2}{\mathbf{X}\mathbf{X}_1}\right) = \frac{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)}{\Xi\delta_1\delta_2\rho_1}\left(\Phi_2 - \Phi_1 - \frac{\Xi\Theta\delta_1\rho_1\rho_2}{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)(\lambda + \epsilon_3)}\right)\mathbf{Q} - (\lambda + \rho_1\mathbf{R}_1)\frac{(\mathbf{S} - \mathbf{S}_1)^2}{\mathbf{S}} - \frac{\Theta}{\delta_2\delta_3}\frac{(\mathbf{X} - \mathbf{X}_1)^2}{\mathbf{X}\mathbf{X}_1}. \tag{27}$$

Then,  $\frac{d\Delta_1}{dt} \leq 0$ , when

$$\Phi_2 \leq \Phi_1 + \frac{\Xi\Theta\rho_1\rho_3}{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)(\lambda + \epsilon_3)}. \tag{28}$$

Clearly,  $\frac{d\Phi_1}{dt} = 0$  if and only if  $\mathbf{S} = \mathbf{S}_1$ ,  $\mathbf{R} = \mathbf{R}_1$ ,  $\mathbf{Q} = 0$  and  $\mathbf{X} = \mathbf{X}_1$ . Thus, the maximum invariant set in  $\{(\mathbf{S}, \mathbf{R}, \mathbf{Q}, \mathbf{X}) \mid \frac{d\Phi_1}{dt} = 0\}$  is the set  $\mathcal{E}_1$ . Taking into consideration Lasalle's invariance theorem [40], conclude that  $\mathcal{E}_1$  is globally asymptotically stable for

$$\Phi_2 \leq \Phi_1 + \frac{\Xi \Theta \rho_1 \rho_3}{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)(\lambda + \epsilon_3)}.$$

On contrary, the characteristic equation at  $\mathcal{E}_1$ , which is supplied by

$$(\lambda + \epsilon_1 + \nu)(\delta_2 \rho_2 \mathbf{S}_1 - \lambda - \epsilon_2 - \rho_3 \mathbf{X}_1 - \nu)F(\nu) = 0, \tag{29}$$

where  $F(\nu) = (\lambda + \nu + \rho_1 \mathbf{R})(\lambda + \epsilon_1 + \nu - \delta_1 \rho_1 \mathbf{S}_1) + \delta_1 \rho_1^2 \mathbf{R}_1 \mathbf{S}_1$ . Therefore, the eigenvalues of (29) are

$$\begin{aligned} \nu_1 &= \delta_2 \rho_2 \mathbf{S}_1 - \lambda - \epsilon_2 - \rho_3 \mathbf{X}_1 \\ &= \frac{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)}{\Xi \delta_1 \rho_1} \left( \Phi_2 - \Phi_1 - \frac{\Xi \Theta \delta_1 \rho_1 \rho_3}{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)(\lambda + \epsilon_3)} \right). \end{aligned} \tag{30}$$

It is clear that  $\nu_1 > 0$  then  $\Phi_2 > \Phi_1 + \frac{\Xi \Theta \delta_1 \rho_1 \rho_3}{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$ . Which shows the un-stability of  $\mathcal{E}_1$ . This completes the proof.  $\square$

**Theorem 7.** Assume that  $\Phi_2 > 1 + \frac{\Theta \rho_3}{(\lambda + \epsilon_1)(\lambda + \epsilon_2)}$  and  $\Phi_2 > \Phi_1$ . Then the treatment failure equilibrium  $\mathcal{E}_2$  is globally asymptotically stable if

$$\Phi_1 + \frac{\Theta \rho_3}{\delta_3 \lambda(\lambda + \epsilon_2)((\Phi_2/\Phi_1) - 1)} \leq 1 + \frac{\rho_2(\lambda + \epsilon_3)}{\delta_3 \rho_3 \lambda}. \tag{31}$$

Otherwise, it is unstable.

**Proof.** Assume the subsequent Lyapunov functional:

$$A_2(\mathbf{t}) = S_2 \wp\left(\frac{\mathbf{S}(\mathbf{t})}{S_2}\right) + \frac{1}{\delta_1} \mathbf{R}_1(\mathbf{t}) + \frac{1}{\delta_2} \mathbf{Q}_2(\mathbf{t}) \wp\left(\frac{\mathbf{Q}(\mathbf{t})}{\mathbf{Q}_2}\right) + \frac{1}{\delta_2 \delta_3} \mathbf{X}_2 \wp\left(\frac{\mathbf{X}(\mathbf{t})}{\mathbf{X}_2}\right). \tag{32}$$

Then, we have

$$\begin{aligned} \frac{dA_2}{dt} &\leq \lambda S_2 \left(1 - \frac{S_2}{S}\right)^2 + \rho_2 S_2 \mathbf{R}_2 \left(\frac{2SS_1 - S_2^2 - S^2}{SS_2}\right) + \left(\rho_1 S_2 - \frac{\lambda + \epsilon_1}{\delta_1}\right) \mathbf{R} \\ &\quad + \left(\rho_2 S_2 - \frac{(\lambda + \epsilon_2)}{\delta_2} - \frac{\rho_3}{\delta_2} \mathbf{X}_2\right) \mathbf{Q} + \frac{\Theta}{\delta_2 \delta_3} \left(\frac{2\mathbf{X}\mathbf{X}_2 - \mathbf{X}_2^2 - \mathbf{X}^2}{\mathbf{X}\mathbf{X}_2}\right) \\ &= \rho_1 (S_2 - S_3) - \frac{\Theta}{\delta_2 \delta_3} \frac{(\mathbf{X} - \mathbf{X}_2)^2}{\mathbf{X}\mathbf{X}_2} - (\lambda + \rho_2 \mathbf{R}_2) \frac{(S - S_2)^2}{S}. \end{aligned} \tag{33}$$

Simple computations gives

$$\begin{aligned} S_2 - S_3 &= \Xi \delta_1 \delta_3 \rho_1 \rho_3 (\lambda + \epsilon_2) ((\Phi_2/\Phi_1) - 1) + \Theta \rho_2 \rho_3 (\lambda + \epsilon_1) \\ &\quad - \rho_2 (\lambda + \epsilon_1)(\lambda + \epsilon_2)(\lambda + \epsilon_3) ((\Phi_2/\Phi_1) - 1) \\ &\quad - \delta_3 \rho_3 \lambda (\lambda + \epsilon_1)(\lambda + \epsilon_2) ((\Phi_2/\Phi_1) - 1). \end{aligned} \tag{34}$$

Thus,  $S_2 - S_3 < 0$  implies that  $\Phi_1 + \frac{\Theta \rho_3}{\delta_3 \lambda(\lambda + \epsilon_2)((\Phi_2/\Phi_1) - 1)} \leq 1 + \frac{\rho_2(\lambda + \epsilon_3)}{\delta_3 \rho_3 \lambda}$ .

Ultimately,  $\frac{dA_2}{dt} \leq 0$ , when  $\Phi_1 + \frac{\Theta \rho_3}{\delta_3 \lambda(\lambda + \epsilon_2)((\Phi_2/\Phi_1) - 1)} \leq 1 + \frac{\rho_2(\lambda + \epsilon_3)}{\delta_3 \rho_3 \lambda}$ .

Clearly,  $\frac{dA_2}{dt} = 0$  if and only if  $\mathbf{S} = S_2$ ,  $\mathbf{R} = 0$ ,  $\mathbf{Q} = \mathbf{Q}_2$  and  $\mathbf{X} = \mathbf{X}_2$ . Thus, the maximum invariant set in  $\{(\mathbf{S}, \mathbf{R}, \mathbf{Q}, \mathbf{X}) \mid \frac{dA_2}{dt} = 0\}$  is the set  $\mathcal{E}_2$ . Taking into consideration Lasalle's invariance theorem [40], conclude that  $\mathcal{E}_2$  is globally asymptotically stable for

$$\Phi_1 + \frac{\Theta \rho_3}{\delta_3 \lambda(\lambda + \epsilon_2)((\Phi_2/\Phi_1) - 1)} \leq 1 + \frac{\rho_2(\lambda + \epsilon_3)}{\delta_3 \rho_3 \lambda}.$$

On contrary, the characteristic equation at  $\mathcal{E}_2$ , which is supplied by

$$(\delta_1 \rho_1 S_2 - \lambda - \epsilon_1 - \mu)F_1(\nu) = 0, \tag{35}$$

where  $F_1(\nu) = (\nu + \rho_2 \mathbf{R}_2 + \lambda)(\lambda + \epsilon_2 + \nu + \rho_3 \mathbf{X}_2 - \delta_2 \rho_2 S_2)(\delta_3 \rho_3 \mathbf{Q}_2 - \lambda - \epsilon_3 - \nu) - \delta_3 \rho_3^2 \mathbf{Q}_2 \mathbf{X}_2$ . Therefore, the eigenvalues of (35) are

$$\nu_2 = \delta_1 \rho_1 S_2 - \lambda - \epsilon_1 = \delta_1 \rho_1 (S_2 - S_3). \tag{36}$$

It is clear that  $\nu_2 > 0$  then  $\Phi_1 + \frac{\Theta \rho_3}{\delta_3 \lambda(\lambda + \epsilon_2)((\Phi_2/\Phi_1) - 1)} \geq 1 + \frac{\rho_2(\lambda + \epsilon_3)}{\delta_3 \rho_3 \lambda}$ . Which shows the un-stability of  $\mathcal{E}_2$ . This completes the proof.  $\square$

**Theorem 8.** The partial success equilibrium  $\mathcal{E}_3$  is globally asymptotically stable if  $\Phi_2 > \Phi_1 + \frac{\Xi \Theta \delta_1 \rho_1 \rho_3}{\lambda(\lambda + \epsilon_1)(\lambda + \epsilon_2)(\lambda + \epsilon_3)}$ .

**Proof.** Assume the subsequent Lyapunov functional:

$$\begin{aligned} A_3(\mathbf{t}) &= S_3 \wp\left(\frac{\mathbf{S}(\mathbf{t})}{S_3}\right) + \frac{1}{\delta_1} \mathbf{R}_3 \wp\left(\frac{\mathbf{R}(\mathbf{t})}{\mathbf{R}_3}\right) + \frac{1}{\delta_2} \mathbf{Q}_3(\mathbf{t}) \wp\left(\frac{\mathbf{Q}(\mathbf{t})}{\mathbf{Q}_3}\right) + \frac{1}{\delta_2 \delta_3} \mathbf{X}_3 \wp\left(\frac{\mathbf{X}(\mathbf{t})}{\mathbf{X}_3}\right). \end{aligned} \tag{37}$$

Then, we have

$$\begin{aligned} \frac{dA_3}{dt} &\leq \left(1 - \frac{S_3}{S}\right) (\Xi - \lambda S - \rho_1 \mathbf{S}\mathbf{R} - \rho_2 S\mathbf{Q}) \\ &\quad + \frac{1}{\delta_1} \left(1 - \frac{\mathbf{R}_3}{\mathbf{R}}\right) (\rho_3 \delta_1 \mathbf{S}\mathbf{R} - (\lambda + \epsilon_1) \mathbf{R}) \\ &\quad + \frac{1}{\delta_2} \left(1 - \frac{\mathbf{Q}_3}{\mathbf{Q}}\right) (\rho_2 \delta_2 S\mathbf{Q} - (\lambda + \epsilon_2) \mathbf{Q} - \rho_3 \mathbf{Q}\mathbf{X}) \\ &\quad + \frac{1}{\delta_2 \delta_3} \left(1 - \frac{\mathbf{X}_3}{\mathbf{X}}\right) (\Theta + \rho_3 \delta_3 \mathbf{Q}\mathbf{X} - (\lambda + \epsilon_3) \mathbf{X}). \end{aligned} \tag{38}$$

Setting  $\Xi = \lambda S_3 + \rho_2 S_3 \mathbf{Q}_3 + \rho_1 S_3 \mathbf{R}_3$ ,  $\rho_3 \mathbf{Q}_3 \mathbf{X}_3 / \delta_2 = \left(\frac{\lambda + \epsilon_3}{\delta_2 \delta_3}\right) \mathbf{X}_3 - \frac{\Theta}{\delta_2 \delta_3}$ ,  $\rho_1 S_3 \mathbf{R}_3 = \left(\frac{\lambda + \epsilon_1}{\delta_1}\right) \mathbf{R}_3$  and  $\rho_2 S_3 \mathbf{Q}_3 = \left(\frac{\lambda + \epsilon_2}{\delta_2}\right) \mathbf{Q}_3 + \frac{\rho_3}{\delta_2} \mathbf{Q}_3 \mathbf{X}_3$ , we have

$$\begin{aligned} \frac{dA_3}{dt} &\leq \lambda S_3 \left(1 - \frac{S_3}{S}\right)^2 + \rho_2 S_3 \mathbf{R}_3 \left(\frac{2SS_3 - S_3^2 - S^2}{SS_3}\right) + \left(\rho_1 S_3 - \frac{\lambda + \epsilon_1}{\delta_1}\right) \mathbf{R} \\ &\quad + \left(\rho_2 S_3 - \frac{(\lambda + \epsilon_2)}{\delta_2} - \frac{\rho_3}{\delta_2} \mathbf{X}_3\right) \mathbf{Q} + \frac{\Theta}{\delta_2 \delta_3} \left(\frac{2\mathbf{X}\mathbf{X}_1 - \mathbf{X}_1^2 - \mathbf{X}^2}{\mathbf{X}\mathbf{X}_1}\right) \\ &\quad + \rho_1 S_3 \mathbf{R}_3 \left(\frac{2SS_3 - S_3^2 - S^2}{SS_3}\right) \\ &= -\frac{\Theta}{\delta_2 \delta_3} \frac{(\mathbf{X} - \mathbf{X}_3)^2}{\mathbf{X}\mathbf{X}_3} - (\lambda + \rho_2 \mathbf{R}_3 + \rho_1 \mathbf{Q}_3) \frac{(S - S_3)^2}{S}. \end{aligned} \tag{39}$$

Therefore,  $\frac{dA_3}{dt} \leq 0$  if and only if  $\mathbf{S} = S_3$  and  $\mathbf{X} = \mathbf{X}_3$ . By an easy calculation, we can prove that  $\frac{dA_3}{dt} = 0$  if and only if  $\mathbf{S} = S_3$ ,  $\mathbf{R} = \mathbf{R}_3$ ,  $\mathbf{Q} = \mathbf{Q}_3$  and  $\mathbf{X} = \mathbf{X}_3$ . Following the LaSalle's invariant approach [40], we conclude that  $\mathcal{E}_3$  is globally asymptotically stable under the assumptions that this point holds.  $\square$

### A fractional model for oncolytic efficacy in the ABC derivative sense

The DEs framework presents the methodological strategy that encompasses the assumptions with the demonstration of MI virus's robust oncolytic effectiveness, followed by the ABC-fractional derivative operator

$$\begin{cases} {}^{ABC}D_t^\varphi \mathbf{S}(\mathbf{t}) = Y_1(\mathbf{t}, \mathbf{S}), \\ {}^{ABC}D_t^\varphi \mathbf{R}(\mathbf{t}) = Y_2(\mathbf{t}, \mathbf{R}) \\ {}^{ABC}D_t^\varphi \mathbf{Q}(\mathbf{t}) = Y_3(\mathbf{t}, \mathbf{Q}) \\ {}^{ABC}D_t^\varphi \mathbf{X}(\mathbf{t}) = Y_4(\mathbf{t}, \mathbf{X}) \end{cases} \tag{40}$$

with ICs stated in (11)

#### Existence and uniqueness of the fractional model for oncolytic efficacy

Here, the (40) demonstrates that the fractional order model for cancer therapy is a nonlinear model. The fixed point methodology is employed to investigate the presence of solutions. Because of this, we define  $\Omega = [0, \mathbf{t}]$  such that  $\mathcal{W} = \mathcal{U}(\Omega) \times \mathcal{U}(\Omega) \times \mathcal{U}(\Omega) \times \mathcal{U}(\Omega)$  as a Banach space  $\mathcal{U}(\Omega) = \mathbb{C}[0, \mathbf{t}]$  of real-valued continuous mappings on the domain  $\Omega$  having the norm:

$$\|(\mathbf{S}, \mathbf{R}, \mathbf{Q}, \mathbf{X})\| = \|\mathbf{S}\| + \|\mathbf{R}\| + \|\mathbf{Q}\| + \|\mathbf{X}\|. \tag{41}$$

Here,  $\|\mathbf{S}\| = \sup\{|\mathbf{S}(\mathbf{t})| : \mathbf{t} \in \Omega\}$ ,  $\|\mathbf{R}\| = \sup\{|\mathbf{R}(\mathbf{t})| : \mathbf{t} \in \Omega\}$ ,  $\|\mathbf{Q}\| = \sup\{|\mathbf{Q}(\mathbf{t})| : \mathbf{t} \in \Omega\}$ ,  $\|\mathbf{X}\| = \sup\{|\mathbf{X}(\mathbf{t})| : \mathbf{t} \in \Omega\}$ .

The AB fractional order integral can be used to transform the model (40) to a Volterra type integral equation. The implementation of Theorem 1 yields the following.

$$\begin{aligned}
 S(t) - S(0) &= \frac{1-\varphi}{ABC(\varphi)} \left\{ \Xi - \lambda S(t) - \rho_1 S(t)R(t) - \rho_2 S(t)Q(t) \right\} \\
 &+ \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} \left\{ \Xi - \lambda S(t) - \rho_1 S(t)R(t) - \rho_2 S(t)Q(t) \right\} d\zeta, \\
 R(t) - R(0) &= \frac{1-\varphi}{ABC(\varphi)} \left\{ \delta_1 \rho_1 S(t)R(t) - (\lambda + \varepsilon_1)R(t) \right\} \\
 &+ \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} \left\{ \delta_1 \rho_1 S(t)R(t) - (\lambda + \varepsilon_1)R(t) \right\} d\zeta, \\
 Q(t) - Q(0) &= \frac{1-\varphi}{ABC(\varphi)} \left\{ \delta_2 \rho_2 S(t)Q(t) - (\lambda + \varepsilon_2)Q(t) - \rho_3 Q(t)X(t) \right\} \\
 &+ \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \\
 &\times \int_0^t (t-\zeta)^{\varphi-1} \left\{ \delta_2 \rho_2 S(t)Q(t) - (\lambda + \varepsilon_2)Q(t) - \rho_3 Q(t)X(t) \right\} d\zeta, \\
 X(t) - X(0) &= \frac{1-\varphi}{ABC(\varphi)} \left\{ \Theta + \delta_3 \rho_3 Q(t)X(t) - (\lambda + \varepsilon_3)X(t) \right\} \\
 &+ \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} \left\{ \Theta + \delta_3 \rho_3 Q(t)X(t) - (\lambda + \varepsilon_3)X(t) \right\} d\zeta. \quad (42)
 \end{aligned}$$

The accompanying terms are defined for clarity

$$\begin{cases}
 Y_1(t, S) = \Xi - \lambda S(t) - \rho_1 S(t)R(t) - \rho_2 S(t)Q(t), \\
 Y_2(t, R) = \delta_1 \rho_1 S(t)R(t) - (\lambda + \varepsilon_1)R(t), \\
 Y_3(t, Q) = \delta_2 \rho_2 S(t)Q(t) - (\lambda + \varepsilon_2)Q(t) - \rho_3 Q(t)X(t), \\
 Y_4(t, X) = \Theta + \delta_3 \rho_3 Q(t)X(t) - (\lambda + \varepsilon_3)X(t),
 \end{cases} \quad (43)$$

**Theorem 9.** The kernels  $Y_i$ ,  $i = 1, 2, 3, 4$  holds the subsequent Lipschitz assumption. Then, if  $Y_1(a, S(a)) = 0$  and contraction variant satisfies

$$0 \leq \vartheta_i < 1, \quad i = 1, 2, 3, 4. \quad (44)$$

**Proof.** Assume that the kernel  $Y_1(t, S) = \Xi - \lambda S(t) - \rho_1 S(t)R(t) - \rho_2 S(t)Q(t)$ . Also, let  $S$  and  $\bar{S}$  are two distinct mappings, then we have

$$\begin{aligned}
 &\|Y_1(t, S) - Y_1(t, \bar{S})\| \\
 &= \|-\lambda(S(t) - \bar{S}(t)) - \rho_1 R(t)(S(t) - \bar{S}(t)) - \rho_2 Q(t)(S(t) - \bar{S}(t))\| \\
 &\leq \|S(t) - \bar{S}(t)\| (\lambda + \rho_1 \|R(t)\| + \rho_2 \|Q(t)\|) \\
 &\leq \|S(t) - \bar{S}(t)\| (\lambda + \rho_1 \kappa_2 + \rho_2 \kappa_3) \\
 &\leq \vartheta \|S(t) - \bar{S}(t)\|. \quad (45)
 \end{aligned}$$

By choosing  $\vartheta = \lambda + \rho_1 \kappa_2 + \rho_2 \kappa_3$ , where  $\kappa_1 = \max_0 i \in I \|S(t)\|$ ,  $\kappa_2 = \max_0 i \in I \|R(t)\|$ ,  $\kappa_3 = \max_0 i \in I \|Q(t)\|$ ,  $\kappa_4 = \max_0 i \in I \|X(t)\|$  are the bounded mappings, then we attain

$$\|Y_1(t, S) - Y_1(t, \bar{S})\| \leq \vartheta \|S(t) - \bar{S}(t)\|. \quad (46)$$

As a result, the Lipschitz criteria applies for  $Y_1$ , and if  $0 \leq \vartheta_i < 1$  is a contraction for  $Y_1$ . Additional kernels can be addressed utilizing the analogous technique, as shown below:

$$\begin{aligned}
 \|Y_2(t, R) - Y_2(t, \bar{R})\| &\leq \vartheta_2 \|R(t) - \bar{R}(t)\|, \\
 \|Y_3(t, Q) - Y_3(t, \bar{Q})\| &\leq \vartheta_3 \|Q(t) - \bar{Q}(t)\|, \\
 \|Y_4(t, X) - Y_4(t, \bar{X})\| &\leq \vartheta \|X(t) - \bar{X}(t)\|. \quad (47)
 \end{aligned}$$

The verification is now finalized.  $\square$

Therefore, the mechanism (43) can be represented as using the kernels from (10)

$$S(t) = S(0) + \frac{1-\varphi}{ABC(\varphi)} Y_1(t, S) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} Y_1(\zeta, S) d\zeta,$$

$$\begin{aligned}
 R(t) &= R(0) + \frac{1-\varphi}{ABC(\varphi)} Y_2(t, R) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} Y_2(\zeta, R) d\zeta, \\
 Q(t) &= Q(0) + \frac{1-\varphi}{ABC(\varphi)} Y_3(t, Q) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} Y_3(\zeta, Q) d\zeta, \\
 X(t) &= \\
 &X(0) + \frac{1-\varphi}{ABC(\varphi)} Y_4(t, X) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} Y_4(\zeta, X) d\zeta. \quad (48)
 \end{aligned}$$

Further, the recursive relationship that follows is presented as

$$\begin{aligned}
 S_r(t) &= \frac{1-\varphi}{ABC(\varphi)} Y_1(t, S_{r-1}) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} Y_1(\zeta, S_{r-1}) d\zeta, \\
 R_r(t) &= \frac{1-\varphi}{ABC(\varphi)} Y_2(t, R_{r-1}) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} Y_2(\zeta, R_{r-1}) d\zeta, \\
 Q_r(t) &= \frac{1-\varphi}{ABC(\varphi)} Y_3(t, Q_{r-1}) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} Y_3(\zeta, Q_{r-1}) d\zeta, \\
 X_r(t) &= \\
 &\frac{1-\varphi}{ABC(\varphi)} Y_4(t, X_{r-1}) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} Y_4(\zeta, X_{r-1}) d\zeta, \quad (49)
 \end{aligned}$$

as well as adequate ICs are

$$S_0(t) = S(0), \quad R_0(t) = R(0), \quad Q_0(t) = Q(0), \quad X_0(t) = X(0). \quad (50)$$

Here, we get the preceding by implementing the difference between the succeeding components

$$\begin{aligned}
 \mathbb{A}_r(t) &= S_r(t) - S_{r-1}(t) \\
 &= \frac{1-\varphi}{ABC(\varphi)} (Y_1(t, S_{r-1}) - Y_1(t, S_{r-2})) \\
 &+ \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} (Y_1(\zeta, S_{r-1}) - Y_1(\zeta, S_{r-2})) d\zeta, \\
 \mathbb{B}_r(t) &= R_r(t) - R_{r-1}(t) \\
 &= \frac{1-\varphi}{ABC(\varphi)} (Y_2(t, R_{r-1}) - Y_2(t, R_{r-2})) \\
 &+ \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} (Y_2(\zeta, R_{r-1}) - Y_2(\zeta, R_{r-2})) d\zeta, \\
 \mathbb{C}_r(t) &= Q_r(t) - Q_{r-1}(t) \\
 &= \frac{1-\varphi}{ABC(\varphi)} (Y_3(t, Q_{r-1}) - Y_3(t, Q_{r-2})) \\
 &+ \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} (Y_3(\zeta, Q_{r-1}) - Y_3(\zeta, Q_{r-2})) d\zeta, \\
 \mathbb{D}_r(t) &= X_r(t) - X_{r-1}(t) \\
 &= \frac{1-\varphi}{ABC(\varphi)} (Y_4(t, X_{r-1}) - Y_4(t, X_{r-2})) \\
 &+ \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} (Y_4(\zeta, X_{r-1}) - Y_4(\zeta, X_{r-2})) d\zeta. \quad (51)
 \end{aligned}$$

Observe what follows

$$S_r(t) = \sum_{i=1}^r \mathbb{A}_i(t), \quad R_r(t) = \sum_{i=1}^r \mathbb{B}_i(t), \quad Q_r(t) = \sum_{i=1}^r \mathbb{C}_i(t), \quad X_r(t) = \sum_{i=1}^r \mathbb{D}_i(t). \quad (52)$$

Next, implement the norm criterion and the triangle inequality to (51), which yields the corresponding consequence as follows:

$$\begin{aligned}
 \|\mathbb{A}_r(t)\| &= \|S_r(t) - S_{r-1}(t)\| \\
 &\leq \frac{1-\varphi}{ABC(\varphi)} \|Y_1(t, S_{r-1}) - Y_1(t, S_{r-2})\| \\
 &+ \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \left\| \int_0^t (t-\zeta)^{\varphi-1} (Y_1(\zeta, S_{r-1}) - Y_1(\zeta, S_{r-2})) d\zeta \right\|.
 \end{aligned}$$

Since the kernel meets the Lipschitz criteria, we have the following

$$\begin{aligned}
 \|S_r(t) - S_{r-1}(t)\| &\leq \frac{1-\varphi}{ABC(\varphi)} Y_1 \|S_{r-1} - S_{r-2}\| \\
 &+ \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} Y_1 \|S_{r-1} - S_{r-2}\| d\zeta.
 \end{aligned}$$

Consequently, we get the following:

$$\|\mathbb{A}_r(t)\| \leq \frac{1-\varphi}{\text{ABC}(\varphi)} Y_1 \|\mathbb{A}_{r-1}(t)\| + \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} Y_1 \int_0^t (t-\varsigma)^{\varphi-1} \|\mathbb{A}_{r-1}(t)\| d\varsigma. \tag{53}$$

Applying a similar technique, we acquire

$$\begin{aligned} \|\mathbb{B}_r(t)\| &\leq \frac{1-\varphi}{\text{ABC}(\varphi)} Y_2 \|\mathbb{B}_{r-1}(t)\| \\ &\quad + \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} Y_2 \int_0^t (t-\varsigma)^{\varphi-1} \|\mathbb{B}_{r-1}(t)\| d\varsigma, \\ \|\mathbb{C}_r(t)\| &\leq \frac{1-\varphi}{\text{ABC}(\varphi)} Y_3 \|\mathbb{C}_{r-1}(t)\| \\ &\quad + \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} Y_3 \int_0^t (t-\varsigma)^{\varphi-1} \|\mathbb{C}_{r-1}(t)\| d\varsigma, \\ \|\mathbb{D}_r(t)\| &\leq \frac{1-\varphi}{\text{ABC}(\varphi)} Y_4 \|\mathbb{D}_{r-1}(t)\| \\ &\quad + \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} Y_4 \int_0^t (t-\varsigma)^{\varphi-1} \|\mathbb{D}_{r-1}(t)\| d\varsigma. \end{aligned}$$

Now, we prove a revolutionary theorem by acquiring the preceding outcomes.

**Theorem 10.** *The fractional model of oncolytic efficacy (10) considering ABC derivative operator has a solution, if for  $t_{\max}$  the subsequent assumption holds*

$$\frac{1-\varphi}{\text{ABC}(\varphi)} Y_i + \frac{t_{\max}}{\text{ABC}(\varphi)\Gamma(\varphi)} Y_i < 1, \quad \text{for } i = 1, 2, 3. \tag{54}$$

**Proof.** Utilizing the assumption of bounded mappings of  $\mathbf{S}(t)$ ,  $\mathbf{R}(t)$ ,  $\mathbf{Q}(t)$  and  $\mathbf{X}(t)$ . Moreover, the kernels  $Y_i$ ,  $i = 1, 2, 3, 4$  fulfils the Lipschitz assumption from (53), then

$$\begin{aligned} \|\mathbb{A}_r(t)\| &\leq \|\mathbf{S}(0)\| \left\{ \frac{1-\varphi}{\text{ABC}(\varphi)} Y_1 + \frac{t_{\max}^\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} Y_1 \right\}^r, \\ \|\mathbb{B}_r(t)\| &\leq \|\mathbf{R}(0)\| \left\{ \frac{1-\varphi}{\text{ABC}(\varphi)} Y_2 + \frac{t_{\max}^\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} Y_2 \right\}^r, \\ \|\mathbb{C}_r(t)\| &\leq \|\mathbf{Q}(0)\| \left\{ \frac{1-\varphi}{\text{ABC}(\varphi)} Y_3 + \frac{t_{\max}^\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} Y_3 \right\}^r, \\ \|\mathbb{D}_r(t)\| &\leq \|\mathbf{X}(0)\| \left\{ \frac{1-\varphi}{\text{ABC}(\varphi)} Y_4 + \frac{t_{\max}^\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} Y_4 \right\}^r. \end{aligned} \tag{55}$$

Because identity (52) and a continuous mapping occur, we must illustrate that the foregoing mappings are the findings of the suggested oncolytic model (10). For this, we analyse

$$\begin{aligned} \mathbf{S}(t) - \mathbf{S}(0) &= \mathbf{S}_r - \mathbb{E}_r(t), \\ \mathbf{R}(t) - \mathbf{R}(0) &= \mathbf{R}_r - \mathbb{F}_r(t), \\ \mathbf{Q}(t) - \mathbf{Q}(0) &= \mathbf{Q}_r - \mathbb{G}_r(t), \\ \mathbf{X}(t) - \mathbf{X}(0) &= \mathbf{X}_r - \mathbb{H}_r(t). \end{aligned} \tag{56}$$

Further, we illustrate that the infinite term  $\|\mathbb{E}_\infty(t)\| \mapsto 0$ . Thus, we have

$$\begin{aligned} \|\mathbb{E}_r(t)\| &\leq \left\| \frac{1-\varphi}{\text{ABC}(\varphi)} (Y_1(t, \mathbf{S}) - Y_1(t, \mathbf{S}_{r-1})) \right. \\ &\quad \left. + \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \int_0^t (t-\varsigma)^{\varphi-1} (Y_1(\varsigma, \mathbf{S}) - Y_1(\varsigma, \mathbf{S}_{r-1})) d\varsigma \right\|. \end{aligned}$$

It follows that

$$\begin{aligned} \|\mathbb{E}_r(t)\| &\leq \frac{1-\varphi}{\text{ABC}(\varphi)} \|Y_1(t, \mathbf{S}) - Y_1(t, \mathbf{S}_{r-1})\| \\ &\quad + \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \int_0^t (t-\varsigma)^{\varphi-1} \|Y_1(\varsigma, \mathbf{S}) - Y_1(\varsigma, \mathbf{S}_{r-1})\| d\varsigma \\ &\leq \frac{1-\varphi}{\text{ABC}(\varphi)} Y_1 \|\mathbf{S} - \mathbf{S}_{r-1}\| + \frac{Y_1 t^\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \|\mathbf{S} - \mathbf{S}_{r-1}\|. \end{aligned}$$

By repeating a similar technique, we have

$$\|\mathbb{E}_r(t)\| \leq \left\{ \frac{1-\varphi}{\text{ABC}(\varphi)} + \frac{t^\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \right\}^{r+1} Y_1^r \|\mathbf{S} - \mathbf{S}_{r-1}\|^r, \tag{57}$$

Adjusting  $t = t_{\max}$ , then we have

$$\|\mathbb{E}_r(t)\| \leq \left\{ \frac{1-\varphi}{\text{ABC}(\varphi)} + \frac{t_{\max}^\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \right\}^{r+1} Y_1^r \|\mathbf{S} - \mathbf{S}_{r-1}\|^r, \tag{58}$$

Now implementing limit, we achieve  $\|\mathbb{E}_r(t)\| \mapsto 0$ . Analogously, one can obtain  $\|\mathbb{F}_r(t)\| \mapsto 0$ ,  $\|\mathbb{G}_r(t)\| \mapsto 0$ ,  $\|\mathbb{H}_r(t)\| \mapsto 0$ , which assures the existence of the solution of the fractional model oncolytic efficacy.

We now investigate a contraction strategy for the validity of the results of the hypothesized fractional model for oncolytic efficacy (10). Permit a framework of responses to emerge for this, assuming that there is a system of solutions for (10),  $\tilde{\mathbf{S}}(t)$ ,  $\tilde{\mathbf{R}}(t)$ ,  $\tilde{\mathbf{Q}}(t)$  and  $\tilde{\mathbf{X}}(t)$ . Then we have

$$\begin{aligned} \mathbf{S}(\mathbf{Q}) - \tilde{\mathbf{S}}(\mathbf{Q}) &= \frac{1-\varphi}{\text{ABC}(\varphi)} (Y_1(t, \mathbf{S}) - Y_1(t, \tilde{\mathbf{S}})) \\ &\quad + \frac{\varphi}{\Gamma(\varphi)\text{ABC}(\varphi)} \int_0^t (t-\varsigma)^{\varphi-1} (Y_1(\varsigma, \mathbf{S}) - Y_1(\varsigma, \tilde{\mathbf{S}})) d\varsigma. \end{aligned} \tag{59}$$

Implementing norm on (59), we acquire

$$\begin{aligned} \|\mathbf{S}(\mathbf{Q}) - \tilde{\mathbf{S}}(\mathbf{Q})\| &\leq \frac{1-\varphi}{\text{ABC}(\varphi)} \|Y_1(t, \mathbf{S}) - Y_1(t, \tilde{\mathbf{S}})\| \\ &\quad + \frac{\varphi}{\Gamma(\varphi)\text{ABC}(\varphi)} \int_0^t (t-\varsigma)^{\varphi-1} \|Y_1(\varsigma, \mathbf{S}) - Y_1(\varsigma, \tilde{\mathbf{S}})\| d\varsigma. \end{aligned}$$

In view of the Lipschitz assumption for the kernel, we have

$$\|\mathbf{S}(\mathbf{Q}) - \tilde{\mathbf{S}}(\mathbf{Q})\| \leq \frac{1-\varphi}{\text{ABC}(\varphi)} Y_1 \|\mathbf{S} - \tilde{\mathbf{S}}\| + \frac{Y_1 t^\varphi}{\Gamma(\varphi)\text{ABC}(\varphi)} \|\mathbf{S} - \tilde{\mathbf{S}}\|, \tag{60}$$

which permits us to write

$$\|\mathbf{S}(\mathbf{Q}) - \tilde{\mathbf{S}}(\mathbf{Q})\| \left( 1 - \frac{1-\varphi}{\text{ABC}(\varphi)} Y_1 + \frac{Y_1 t^\varphi}{\Gamma(\varphi)\text{ABC}(\varphi)} \right) \leq 0, \tag{61}$$

and

$$\|\mathbf{S}(\mathbf{Q}) - \tilde{\mathbf{S}}(\mathbf{Q})\| = 0 \implies \mathbf{S}(\mathbf{Q}) = \tilde{\mathbf{S}}(\mathbf{Q}). \tag{62}$$

Hence, (61) and (62) demonstrate that the system (10) has a unique solution. We can acquire the unique result for the  $\mathbf{r}$ ,  $\mathbf{Q}$  and  $\mathbf{X}$  using the equivalent technique. As a result, the solution of the fractional model of oncolytic efficacy (10) is unique.

Since the precise result of the suggested framework is hard to ascertain. Both ends of (9) are transformed using the straightforward and inverse Sumudu transforms (4), we have

$$\begin{cases} \mathbf{S}(t) = \mathbf{S}(0) \\ + \text{ST}^{-1} \left\{ \frac{1-\varphi}{\text{ABC}(\varphi)\Gamma(\varphi+1)E_\varphi\left(\frac{-t^\varphi}{1-\varphi}\right)} \text{ST} \left\{ \Xi - \lambda \mathbf{S}(t) - \rho_1 \mathbf{S}(t)\mathbf{R}(t) - \rho_2 \mathbf{S}(t)\mathbf{Q}(t) \right\} \right\}, \\ \mathbf{R}(t) = \mathbf{R}(0) \\ + \text{ST}^{-1} \left\{ \frac{1-\varphi}{\text{ABC}(\varphi)\Gamma(\varphi+1)E_\varphi\left(\frac{-t^\varphi}{1-\varphi}\right)} \text{ST} \left\{ \delta_1 \rho_1 \mathbf{S}(t)\mathbf{R}(t) - (\lambda + \varepsilon_1)\mathbf{R}(t) \right\} \right\}, \\ \mathbf{Q}(t) = \mathbf{Q}(0) + \text{ST}^{-1} \left\{ \frac{1-\varphi}{\text{ABC}(\varphi)\Gamma(\varphi+1)E_\varphi\left(\frac{-t^\varphi}{1-\varphi}\right)} \right. \\ \left. \times \text{ST} \left\{ \delta_2 \rho_2 \mathbf{S}(t)\mathbf{Q}(t) - (\lambda + \varepsilon_2)\mathbf{Q}(t) - \rho_3 \mathbf{Q}(t)\mathbf{X}(t) \right\} \right\}, \\ \mathbf{X}(t) = \mathbf{X}(0) \\ + \text{ST}^{-1} \left\{ \frac{1-\varphi}{\text{ABC}(\varphi)\Gamma(\varphi+1)E_\varphi\left(\frac{-t^\varphi}{1-\varphi}\right)} \text{ST} \left\{ \Theta + \delta_3 \rho_3 \mathbf{Q}(t)\mathbf{X}(t) - (\lambda + \varepsilon_3)\mathbf{X}(t) \right\} \right\}. \end{cases}$$



The following recursive scheme can be written as

$$\left\{ \begin{aligned} &S_{(r)}(t) = S(0) \\ &+ ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} ST \left\{ \Xi - \lambda S(t) - \rho_1 S(t)R(t) - \rho_2 S(t)Q(t) \right\} \right\}, \\ &N_{(r)}(t) = R(0) \\ &+ ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} ST \left\{ \delta_1 \rho_1 S(t)R(t) - (\lambda + \varepsilon_1)R(t) \right\} \right\}, \\ &Q_{(r)}(t) = Q(0) + ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} \right. \\ &\quad \left. \times ST \left\{ \delta_2 \rho_2 S(t)Q(t) - (\lambda + \varepsilon_2)Q(t) - \rho_3 Q(t)X(t) \right\} \right\}, \\ &X_{(r)}(t) = X(0) \\ &+ ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} ST \left\{ \Theta + \delta_3 \rho_3 Q(t)X(t) - (\lambda + \varepsilon_3)X(t) \right\} \right\}. \end{aligned} \right.$$

Here,  $Q = \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})}$  represents the Lagrange multiplier. By employing limit as  $r$  approaches to  $\infty$ , then we can attain the approximate solutions  $S(t) = \lim_{r \rightarrow \infty} S_{(r)}(t)$ ;  $R(t) = \lim_{r \rightarrow \infty} R_{(r)}(t)$ ,  $Q(t) = \lim_{r \rightarrow \infty} Q_{(r)}(t)$  and  $X(t) = \lim_{r \rightarrow \infty} X_{(r)}(t)$ .  $\square$

*Stability analysis of the fractional model for oncolytic efficacy*

Suppose that there be a complete metric space  $(\chi, \bar{d})$  having  $\mathcal{W} : \chi \mapsto \chi$ . Consider a Picard iteration  $\mathcal{W}(y_r) = y_{r+1}$  having a set of fixed points  $F(\mathcal{W}) \neq \emptyset$  of  $\mathcal{W}$  such that  $\lim_{r \rightarrow \infty} y_r = w \in F(\mathcal{W})$ . Suppose there be a sequence  $\{y_r\} \in \chi$  such that  $\lim_{r \rightarrow \infty} \bar{d}(x_r, \mathcal{W}x_r) = 0, \implies x_r \mapsto w$ , then the sequence  $y_{r+1} = y_r$  is Picard  $\mathcal{W}$ -stable. For further investigation (see [41]).

**Theorem 11 ([41]).** *Suppose there be a complete metric space  $(\chi, \bar{d})$  and also there be a nonempty fixed point  $F(\mathcal{W})$  of  $\mathcal{W} : \chi \mapsto \chi$ , then there exists  $c_1 \geq 0, c_2 \in [0, 1)$  such that*

$$\bar{d}(\mathcal{W}y_1, \mathcal{W}\delta_1) \leq c_1 \bar{d}(\mathcal{W}y_1, y_1) + c_2 \bar{d}(y_1, \delta_1), \quad \forall y_1 \in \chi, \tag{63}$$

where  $\delta_1 \in F(\mathcal{W})$  having  $\lim_{r \rightarrow \infty} \bar{d}(x_r, \mathcal{W}x_r) = 0$ , then we say that Picard iteration is  $\mathcal{W}$ -stable.

**Theorem 12.** *Consider a self-map  $\mathcal{W}$  presented as*

$$\begin{aligned} \mathcal{W}[S_{(r)}(t)] &= S_{r+1}(t) = S_r(t) + ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} \right. \\ &\quad \left. \times ST \left\{ \Xi - \lambda S_{(r-1)}(t) - \rho_1 S_{(r-1)}(t)R_{(r-1)}(t) - \rho_2 S_{(r-1)}(t)Q_{(r-1)}(t) \right\} \right\}, \\ \mathcal{W}[R_{(r)}(t)] &= R_{r+1}(t) = R_r(t) + ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} \right. \\ &\quad \left. \times ST \left\{ \delta_1 \rho_1 S_{(r-1)}(t)R_{(r-1)}(t) - (\lambda + \varepsilon_1)R_{(r-1)}(t) \right\} \right\}, \\ \mathcal{W}[Q_{(r)}(t)] &= Q_{r+1}(t) = Q_r(t) + ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} \right. \\ &\quad \left. \times ST \left\{ \delta_2 \rho_2 S_{(r-1)}(t)Q_{(r-1)}(t) - (\lambda + \varepsilon_2)Q_{(r-1)}(t) - \rho_3 Q_{(r-1)}(t)X_{(r-1)}(t) \right\} \right\}, \\ \mathcal{W}[X_{(r)}(t)] &= X_{r+1}(t) = X_r(t) + ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} \right. \\ &\quad \left. \times ST \left\{ \Theta + \delta_3 \rho_3 Q_{(r-1)}(t)X_{(r-1)}(t) - (\lambda + \varepsilon_3)X_{(r-1)}(t) \right\} \right\} \end{aligned} \tag{64}$$

is  $\mathcal{W}$ -stable on  $\mathcal{L}^1(\bar{a}, \bar{b})$  if

$$\begin{aligned} &\|\mathcal{W}(S_r(t)) - \mathcal{W}(S_{m_1}(t))\| \\ &\leq \|S_r(t) - S_{m_1}(t)\| \left\{ 1 - \lambda - \rho_1 \varpi_1(\xi) \varpi_2(\xi) - \rho_2 \varpi_1(\xi) \varpi_3(\xi) \right\}, \\ &\|\mathcal{W}(R_r(t)) - \mathcal{W}(R_{m_1}(t))\| \end{aligned}$$

$$\begin{aligned} &\leq \|R_r(t) - R_{m_1}(t)\| \left\{ \delta_1 \rho_1 \varpi_1(\xi) \varpi_3(\xi) - (\lambda + \varepsilon_1) \varpi_3(\xi) \right\}, \\ &\|\mathcal{W}(Q_r(t)) - \mathcal{W}(Q_{m_1}(t))\| \\ &\leq \|Q_r(t) - Q_{m_1}(t)\| \left\{ \delta_2 \rho_2 \varpi_1(\xi) \varpi_3(\xi) - (\lambda + \varepsilon_2) \varpi_3(\xi) - \rho_3 \varpi_3(\xi) \varpi_4(\xi) \right\}, \\ &\|\mathcal{W}(X_r(t)) - \mathcal{W}(X_{m_1}(t))\| \\ &\leq \|X_r(t) - X_{m_1}(t)\| \left\{ \delta_3 \rho_3 \varpi_4(\xi) \varpi_3(\xi) - (\lambda + \varepsilon_3) \varpi_4(\xi) \right\}. \end{aligned}$$

**Proof.** By means of the given hypothesis, also employing the norm on both sides, we have

$$\begin{aligned} &\|\mathcal{W}(S_r(t)) - \mathcal{W}(S_{m_1}(t))\| \\ &= \|S_r(t) - S_{m_1}(t) + ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} \right. \\ &\quad \left. \times ST \left\{ \left( \Xi - \lambda S_r(t) - \rho_1 S_r(t)R_r(t) - \rho_2 S_r(t)Q_r(t) \right) \right. \right. \\ &\quad \left. \left. - \left( \Xi - \lambda S_{m_1}(t) - \rho_1 S_{m_1}(t)R_{m_1}(t) - \rho_2 S_{m_1}(t)Q_{m_1}(t) \right) \right\} \right\} \| \\ &\leq \|S_r(t) - S_{m_1}(t)\| + \|ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} \right. \\ &\quad \left. \times ST \left\{ \left( -\lambda(S_r(t) - S_{m_1}(t)) - \rho_1(S_r(t)R_r(t) - S_{m_1}(t)R_{m_1}(t)) \right. \right. \right. \\ &\quad \left. \left. - \rho_2(S_r(t)Q_r(t) - S_{m_1}(t)Q_{m_1}(t)) \right) \right\} \right\} \|. \end{aligned} \tag{65}$$

It follows that

$$\begin{aligned} &\|\mathcal{W}(S_r(t)) - \mathcal{W}(S_{m_1}(t))\| \\ &\leq \|S_r(t) - S_{m_1}(t)\| \left\{ 1 - \lambda - \rho_1 \varpi_1(\xi) \varpi_2(\xi) - \rho_2 \varpi_1(\xi) \varpi_3(\xi) \right\}. \end{aligned} \tag{66}$$

Analogously, we have

$$\begin{aligned} &\|\mathcal{W}(R_r(t)) - \mathcal{W}(R_{m_1}(t))\| \\ &\leq \|R_r(t) - R_{m_1}(t)\| \left\{ \delta_1 \rho_1 \varpi_1(\xi) \varpi_3(\xi) - (\lambda + \varepsilon_1) \varpi_3(\xi) \right\}, \\ &\|\mathcal{W}(Q_r(t)) - \mathcal{W}(Q_{m_1}(t))\| \\ &\leq \|Q_r(t) - Q_{m_1}(t)\| \left\{ \delta_2 \rho_2 \varpi_1(\xi) \varpi_3(\xi) - (\lambda + \varepsilon_2) \varpi_3(\xi) - \rho_3 \varpi_3(\xi) \varpi_4(\xi) \right\}, \\ &\|\mathcal{W}(X_r(t)) - \mathcal{W}(X_{m_1}(t))\| \\ &\leq \|X_r(t) - X_{m_1}(t)\| \left\{ \delta_3 \rho_3 \varpi_4(\xi) \varpi_3(\xi) - (\lambda + \varepsilon_3) \varpi_4(\xi) \right\}, \end{aligned} \tag{67}$$

where  $\varpi_1, \varpi_2, \varpi_3, \varpi_4$  are the  $ST^{-1} \left\{ \frac{1-\varphi}{ABC(\varphi)\Gamma(\varphi+1)E_{\varphi}(\frac{-\mu\varphi}{1-\varphi})} ST(\cdot) \right\}$ .

Thus, utilizing Theorem 11, we concluded that  $\mathcal{W}$  is Picard  $\mathcal{W}$ -stable according to the foregoing findings.  $\square$

**Numerical analysis and mathematical modelling**

In this section, we will use the model (10) which has been numerically simulated by [42] using the ABC fractional derivative of order  $\varphi$ . The method is used to obtain approximate solutions to the proposed model.

Assuming the subsequent DE of oncolytic efficacy in ABC operator form

$$\begin{cases} {}_0^{ABC}D_t^\varphi \Lambda(t) = \Psi(t, \Lambda(t)), \\ \Lambda(0) = \Lambda_0. \end{cases} \tag{68}$$

The fractional integral representation of the aforesaid initial value problem is as follows:

$$\Lambda(t) - \Lambda(0) = \frac{1-\varphi}{ABC(\varphi)} \Psi(t, \Lambda(Q)) + \frac{\varphi}{ABC(\varphi)\Gamma(\varphi)} \int_0^t (t-\zeta)^{\varphi-1} \Psi(\zeta, \Lambda(\zeta)) d\zeta. \tag{69}$$

Inserting  $\mathbf{t} = \mathbf{t}_{r+1}$ ,  $r = 1, 2, 3, \dots$ , then (69) diminishes to the subsequent

$$\begin{aligned} \Lambda(\mathbf{t}_{r+1}) - \Lambda(0) &= \frac{1-\varphi}{\text{ABC}(\varphi)} \Psi(\mathbf{t}_r, \Lambda(\mathbf{t}_r)) \\ &+ \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \int_0^{\mathbf{t}_{r+1}} (\mathbf{t}_{r+1} - \zeta)^{\varphi-1} \Psi(\zeta, \Lambda(\zeta)) d\zeta. \end{aligned} \quad (70)$$

Taking into consideration  $\Psi(\zeta, \Lambda(\zeta))$  and incorporating the Lagrange polynomial interpolation with two step [42], we acquire the approximation below on  $[\mathbf{t}_\ell, \mathbf{t}_{\ell+1}]$

$$\begin{aligned} P_\ell &= \Psi(\zeta, \Lambda(\zeta)) \\ &= \frac{\zeta - \mathbf{t}_{\ell-1}}{\mathbf{t}_\ell - \mathbf{t}_{\ell-1}} \Psi(\mathbf{t}_\ell, \Lambda(\mathbf{t}_\ell)) - \frac{\zeta - \mathbf{t}_\ell}{\mathbf{t}_\ell - \mathbf{t}_{\ell-1}} \Psi(\mathbf{t}_{\ell-1}, \Lambda(\mathbf{t}_{\ell-1})) \\ &= \frac{\zeta - \mathbf{t}_{\ell-1}}{h} \Psi(\mathbf{t}_\ell, \Lambda(\mathbf{t}_\ell)) - \frac{\zeta - \mathbf{t}_\ell}{h} \Psi(\mathbf{t}_{\ell-1}, \Lambda(\mathbf{t}_{\ell-1})) \\ &\approx \frac{\zeta - \mathbf{t}_{\ell-1}}{h} \Psi(\mathbf{t}_\ell, \Lambda_\ell) - \frac{\zeta - \mathbf{t}_\ell}{h} \Psi(\mathbf{t}_{\ell-1}, \Lambda_{\ell-1}). \end{aligned} \quad (71)$$

Employing Lagrange polynomial interpolation on (70), then we have

$$\begin{aligned} \Lambda_{r+1} &= \Lambda(0) + \frac{1-\varphi}{\text{ABC}(\varphi)} \Psi(\mathbf{t}_r, \Lambda(\mathbf{t}_r)) + \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \\ &\times \sum_{\ell=0}^r \left\{ \frac{\Psi(\mathbf{t}_\ell, \Lambda_\ell)}{h} \int_0^{\mathbf{t}_\ell} \mathbf{t}_\ell^{\ell+1} (\zeta - \mathbf{t}_{\ell-1}) (\mathbf{t}_{r+1} - \zeta)^{\varphi-1} \Psi(\zeta, \Lambda(\zeta)) d\zeta \right. \\ &\left. - \frac{\Psi(\mathbf{t}_{\ell-1}, \Lambda_{\ell-1})}{h} \int_0^{\mathbf{t}_\ell} \mathbf{t}_\ell^{\ell+1} (\zeta - \mathbf{t}_\ell) (\mathbf{t}_{r+1} - \zeta)^{\varphi-1} \Psi(\zeta, \Lambda(\zeta)) d\zeta. \right. \end{aligned} \quad (72)$$

After simplifying the integral in (72), we attain

$$\begin{aligned} \Lambda_{r+1} &= \Lambda(0) + \frac{1-\varphi}{\text{ABC}(\varphi)} \Psi(\mathbf{t}_r, \Lambda(\mathbf{t}_r)) + \frac{\varphi}{\text{ABC}(\varphi)} \\ &\times \sum_{\ell=0}^r \left\{ \frac{h^\varphi \Psi(\mathbf{t}_\ell, \Lambda_\ell)}{\Gamma(\varphi+2)} ((\mathbf{r}+1-\ell)^\varphi (\mathbf{r}-\ell+2+\varphi) - (\mathbf{r}-\ell)^\varphi (\mathbf{r}-\ell+2+2\varphi)) \right. \\ &\left. - \frac{h^\varphi \Psi(\mathbf{t}_{\ell-1}, \Lambda_{\ell-1})}{\Gamma(\varphi+2)} ((\mathbf{r}+1-\ell)^{\varphi+1} - (\mathbf{r}-\ell)^\varphi (\mathbf{r}-\ell+1+\varphi)) + \mathfrak{R}_r^\varphi, \right. \end{aligned} \quad (73)$$

where  $\mathfrak{R}_r^\varphi$  represent the remainder term and is defined as

$$\begin{aligned} \mathfrak{R}_r^\varphi &= \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \sum_{\ell=0}^r \int_0^{\mathbf{t}_\ell} \mathbf{t}_\ell^{\ell-1} \frac{(\zeta - \mathbf{t}_\ell)(\zeta - \mathbf{t}_{\ell-1})}{2!} \\ &\times \frac{\partial^2}{\partial \mu^2} [\Psi(\zeta, \Lambda(\zeta))]_{\zeta=\varepsilon_\mu} (\mathbf{t}_{r+1} - \zeta)^{\varphi-1} d\zeta. \end{aligned} \quad (74)$$

The numerical technique for the oncolytic efficacy system (10) encompassing the AB-fractional integral operator is presented as

$$\begin{aligned} \mathbf{S}(\mathbf{t}) &= \mathbf{S}(0) + \frac{1-\varphi}{\text{ABC}(\varphi)} Y_1(\mathbf{t}, \mathbf{S}(\mathbf{t})) \\ &+ \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \int_0^{\mathbf{t}} (\mathbf{t} - \zeta)^{\varphi-1} Y_1(\zeta, \mathbf{S}(\zeta)) d\zeta, \\ \mathbf{R}(\mathbf{t}) &= \mathbf{R}(0) + \frac{1-\varphi}{\text{ABC}(\varphi)} Y_2(\mathbf{t}, \mathbf{R}(\mathbf{t})) \\ &+ \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \int_0^{\mathbf{t}} (\mathbf{t} - \zeta)^{\varphi-1} Y_2(\zeta, \mathbf{R}(\zeta)) d\zeta, \\ \mathbf{Q}(\mathbf{t}) &= \mathbf{Q}(0) + \frac{1-\varphi}{\text{ABC}(\varphi)} Y_3(\mathbf{t}, \mathbf{Q}(\mathbf{t})) \\ &+ \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \int_0^{\mathbf{t}} (\mathbf{t} - \zeta)^{\varphi-1} Y_3(\zeta, \mathbf{Q}(\zeta)) d\zeta, \\ \mathbf{X}(\mathbf{t}) &= \mathbf{X}(0) + \frac{1-\varphi}{\text{ABC}(\varphi)} Y_4(\mathbf{t}, \mathbf{X}(\mathbf{t})) \\ &+ \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \int_0^{\mathbf{t}} (\mathbf{t} - \zeta)^{\varphi-1} Y_4(\zeta, \mathbf{X}(\zeta)) d\zeta, \end{aligned} \quad (75)$$

supplemented to ICs

$$\mathbf{S}(0) = \mathbf{R}(0) = \mathbf{Q}(0) = \mathbf{X}(0) = 0. \quad (76)$$

In view of numerical technique (73) to (75), we attain

$$\begin{aligned} \mathbf{S}_{r+1}(\mathbf{t}) &= \mathbf{S}(0) + \frac{1-\varphi}{\text{ABC}(\varphi)} Y_1(\mathbf{t}_r, \mathbf{S}(\mathbf{t}_r)) + \frac{\varphi}{\text{ABC}(\varphi)} \\ &\times \sum_{\ell=0}^r \left\{ \frac{h^\varphi Y_1(\mathbf{t}_\ell, \mathbf{S}_\ell)}{\Gamma(\varphi+2)} ((\mathbf{r}+1-\ell)^\varphi (\mathbf{r}-\ell+2+\varphi) - (\mathbf{r}-\ell)^\varphi (\mathbf{r}-\ell+2+2\varphi)) \right. \\ &\left. - \frac{h^\varphi Y_1(\mathbf{t}_{\ell-1}, \mathbf{S}_{\ell-1})}{\Gamma(\varphi+2)} ((\mathbf{r}+1-\ell)^{\varphi+1} - (\mathbf{r}-\ell)^\varphi (\mathbf{r}-\ell+1+\varphi)) + \mathfrak{R}_{r1}^\varphi, \right. \end{aligned} \quad (77)$$

$$\begin{aligned} \mathbf{R}_{r+1}(\mathbf{t}) &= \mathbf{R}(0) + \frac{1-\varphi}{\text{ABC}(\varphi)} Y_2(\mathbf{t}_r, \mathbf{R}(\mathbf{t}_r)) + \frac{\varphi}{\text{ABC}(\varphi)} \\ &\times \sum_{\ell=0}^r \left\{ \frac{h^\varphi Y_2(\mathbf{t}_\ell, \mathbf{R}_\ell)}{\Gamma(\varphi+2)} ((\mathbf{r}+1-\ell)^\varphi (\mathbf{r}-\ell+2+\varphi) - (\mathbf{r}-\ell)^\varphi (\mathbf{r}-\ell+2+2\varphi)) \right. \\ &\left. - \frac{h^\varphi Y_2(\mathbf{t}_{\ell-1}, \mathbf{R}_{\ell-1})}{\Gamma(\varphi+2)} ((\mathbf{r}+1-\ell)^{\varphi+1} - (\mathbf{r}-\ell)^\varphi (\mathbf{r}-\ell+1+\varphi)) + \mathfrak{R}_{r2}^\varphi, \right. \end{aligned} \quad (78)$$

$$\begin{aligned} \mathbf{Q}_{r+1}(\mathbf{t}) &= \mathbf{Q}(0) + \frac{1-\varphi}{\text{ABC}(\varphi)} Y_3(\mathbf{t}_r, \mathbf{Q}(\mathbf{t}_r)) + \frac{\varphi}{\text{ABC}(\varphi)} \\ &\times \sum_{\ell=0}^r \left\{ \frac{h^\varphi Y_3(\mathbf{t}_\ell, \mathbf{Q}_\ell)}{\Gamma(\varphi+2)} ((\mathbf{r}+1-\ell)^\varphi (\mathbf{r}-\ell+2+\varphi) - (\mathbf{r}-\ell)^\varphi (\mathbf{r}-\ell+2+2\varphi)) \right. \\ &\left. - \frac{h^\varphi Y_3(\mathbf{t}_{\ell-1}, \mathbf{Q}_{\ell-1})}{\Gamma(\varphi+2)} ((\mathbf{r}+1-\ell)^{\varphi+1} - (\mathbf{r}-\ell)^\varphi (\mathbf{r}-\ell+1+\varphi)) + \mathfrak{R}_{r3}^\varphi \right. \end{aligned} \quad (79)$$

and

$$\begin{aligned} \mathbf{X}_{r+1}(\mathbf{t}) &= \mathbf{X}(0) + \frac{1-\varphi}{\text{ABC}(\varphi)} Y_4(\mathbf{t}_r, \mathbf{X}(\mathbf{t}_r)) + \frac{\varphi}{\text{ABC}(\varphi)} \\ &\times \sum_{\ell=0}^r \left\{ \frac{h^\varphi Y_4(\mathbf{t}_\ell, \mathbf{X}_\ell)}{\Gamma(\varphi+2)} ((\mathbf{r}+1-\ell)^\varphi (\mathbf{r}-\ell+2+\varphi) - (\mathbf{r}-\ell)^\varphi (\mathbf{r}-\ell+2+2\varphi)) \right. \\ &\left. - \frac{h^\varphi Y_4(\mathbf{t}_{\ell-1}, \mathbf{X}_{\ell-1})}{\Gamma(\varphi+2)} ((\mathbf{r}+1-\ell)^{\varphi+1} - (\mathbf{r}-\ell)^\varphi (\mathbf{r}-\ell+1+\varphi)) + \mathfrak{R}_{r4}^\varphi. \right. \end{aligned} \quad (80)$$

where  $\mathfrak{R}_{r1}^\varphi, \dots, \mathfrak{R}_{r4}^\varphi$  are presented as

$$\begin{aligned} \mathfrak{R}_{r1}^\varphi &= \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \sum_{\ell=0}^r \int_0^{\mathbf{t}_\ell} \mathbf{t}_\ell^{\ell-1} \frac{(\zeta - \mathbf{t}_\ell)(\zeta - \mathbf{t}_{\ell-1})}{2!} \\ &\times \frac{\partial^2}{\partial \zeta^2} [Y_1(\zeta, \Lambda(\zeta))]_{\zeta=\varepsilon_\mu} (\mathbf{t}_{r+1} - \zeta)^{\varphi-1} d\zeta, \\ \mathfrak{R}_{r2}^\varphi &= \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \sum_{\ell=0}^r \int_0^{\mathbf{t}_\ell} \mathbf{t}_\ell^{\ell-1} \frac{(\zeta - \mathbf{t}_\ell)(\zeta - \mathbf{t}_{\ell-1})}{2!} \\ &\times \frac{\partial^2}{\partial \zeta^2} [Y_2(\zeta, \Lambda(\zeta))]_{\zeta=\varepsilon_\mu} (\mathbf{t}_{r+1} - \zeta)^{\varphi-1} d\zeta, \\ \mathfrak{R}_{r3}^\varphi &= \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \sum_{\ell=0}^r \int_0^{\mathbf{t}_\ell} \mathbf{t}_\ell^{\ell-1} \frac{(\zeta - \mathbf{t}_\ell)(\zeta - \mathbf{t}_{\ell-1})}{2!} \\ &\times \frac{\partial^2}{\partial \zeta^2} [Y_3(\zeta, \Lambda(\zeta))]_{\zeta=\varepsilon_\mu} (\mathbf{t}_{r+1} - \zeta)^{\varphi-1} d\zeta, \\ \mathfrak{R}_{r4}^\varphi &= \frac{\varphi}{\text{ABC}(\varphi)\Gamma(\varphi)} \sum_{\ell=0}^r \int_0^{\mathbf{t}_\ell} \mathbf{t}_\ell^{\ell-1} \frac{(\zeta - \mathbf{t}_\ell)(\zeta - \mathbf{t}_{\ell-1})}{2!} \\ &\times \frac{\partial^2}{\partial \zeta^2} [Y_4(\zeta, \Lambda(\zeta))]_{\zeta=\varepsilon_\mu} (\mathbf{t}_{r+1} - \zeta)^{\varphi-1} d\zeta. \end{aligned} \quad (81)$$

The mathematical findings for (77)–(80) are now reported. The settings of biological components supplied by Wang et al. [4] were implemented in this oncolytic efficacy model (9).

### Results and discussion

Immunotherapy is at the cutting edge of contemporary cancer treatment. With varying degrees of success, innovative medicines have been developed that address all three components of cancer pathogenesis: tumour, niche, and impervious mechanism. Oncolytic viruses are new flaviviruses that are being used in combination for initial and salvage treatment. In an attempt to strengthen improved and more appropriate tumour therapies, numerical strategies have been implemented to assist in comprehending the intricate mechanisms of oncolytic virotherapy. Following that, a slew of numerical models (detailed in the preamble) were built to characterize the oncolytic efficacy model (10). Here, we designed a fractional model of oncolytic efficacy based on fractional epidemic assumptions [10,11] and integrated monitoring indicators in this investigation. The method incorporates the chance of recidivism, which fluctuates irrespective of how long the individual has been on treatment, via the use of the ABC fractional derivative operator, incorporating the variation in parameters represented in Table 1.

To substantiate the simulated predictions from the preceding parts, we perform several numerical computations. As for this explanation, the components of framework (10) gravitate to  $\mathcal{E}_0(\mathbf{S}_0, 0, 0, \mathbf{X}_0) = (\frac{\Xi}{\lambda}, 0, 0, \frac{\varphi}{\lambda+\varepsilon_3})$  in this instance, see, Plot 3 and 4, which is compatible

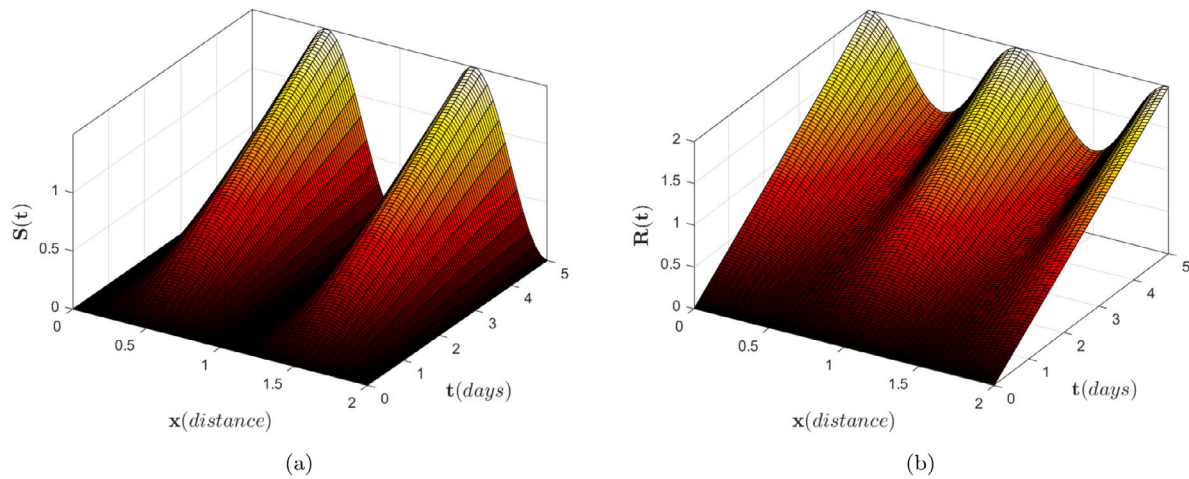


Fig. 3. Three-dimensional illustration of oncolytic efficacy model (10) when  $\phi_1 > 1$  or  $\phi_2 > 1 + \frac{\theta p_3}{(\lambda + \epsilon_2)(\lambda = \epsilon_3)}$ . The non-competitive EP  $\mathcal{E}_0$  is globally asymptotically stable to demonstrate the spatiotemporal factors of nutrient and normal cells, respectively.

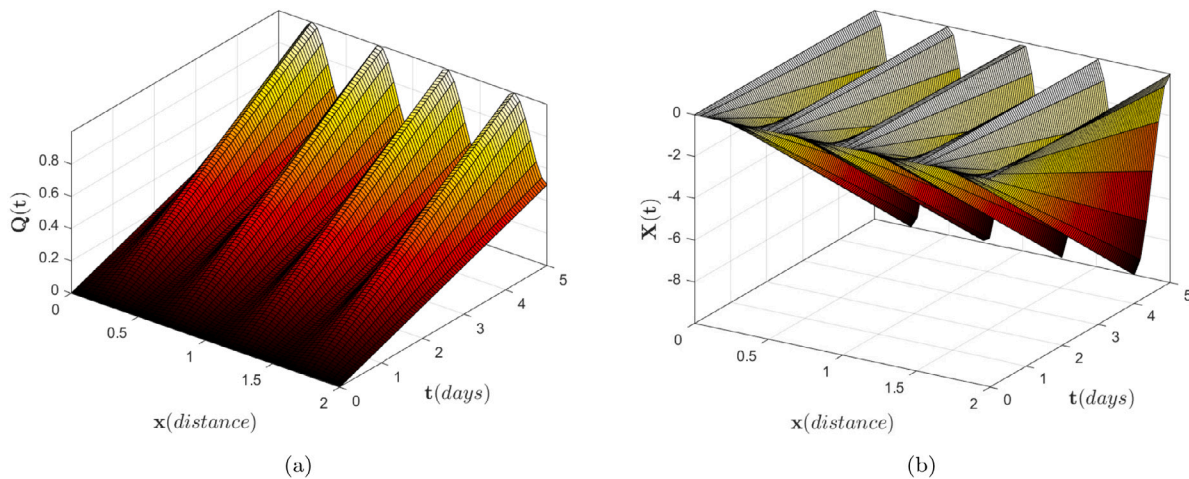


Fig. 4. Three-dimensional illustration of oncolytic efficacy model (10) when  $\phi_1 > 1$  or  $\phi_2 > 1 + \frac{\theta p_3}{(\lambda + \epsilon_2)(\lambda = \epsilon_3)}$ . The non-competitive EP  $\mathcal{E}_0$  is globally asymptotically stable to demonstrate the spatiotemporal factors of tumour cells and free M1 virus, respectively.

with Theorem 5. The nutrition is insufficient to sustain the cancerous and normal tissue communities, resulting in eventual annihilation. This could also be an instance where, in the absence of an immunity reaction, there has been intense rivalry between healthy and cancerous tissues, and the procedure destroyed the cancerous tissues, rendering them unable to maintain regular organisms, resulting in decompensation.

For the ABC fractional oncolytic efficacy model,  $S(t)$ ,  $R(t)$ ,  $Q(t)$  and  $X(t)$  are the specific dietary, regular cells, cancer hepatocytes, and M1 viral contents at time  $t$ , respectively.  $\Xi = 0.02$  and  $\Theta = 0.01$  are the enlistment variables for nutritional and M1 viral levels, respectively. Fig. 5(a)–(b) shows the association of EP  $\mathcal{E}_1(S, R, Q, X)$  in the dearth of an innate reaction, the oncolytic M1 virus therapy unable to decimate cancerous tissue, resulting in the elimination of healthy cells when ICs  $(S, R, Q, X) = (0.36, 0.24, 0.12, 0.12)$ . Meanwhile, Fig. 6(a)–(b) depicts that rise in tumour cells and free M1 virus. As a result, this circumstance poses a serious risk to the service user. This inspection concludes that people with acute or influenced drug therapy after tissue allografts have a clinically important improved incidence of virtually each type of tumour tissue that supplies potent, effective treatments for the pivotal involvement of invulnerable surveillance in tumorigenesis and malignant transformation. Figs. 7–8 illustrates the correlation of EP  $\mathcal{E}_2(S, 0, Q, X)$  when the defensive reaction is absent,

the oncolytic M1 virotherapy is completely successful in eliminating the tumour utilizing ICs  $(S, R, Q, X) = (0.24, 0.16, 0.08, 0.08)$ , resulting in the restoration of healthy tissues and improved wellness outcomes. As a result, the M1 virus is able to regulate the tumour despite being influenced by the immunological reaction targeting tumour tissues. The smallest efficacious dose required to eradicate the tumour is calculated. Furthermore, Figs. 9–10 demonstrates the preciseness of EP  $\mathcal{E}_3(S, R, Q, X)$ , when the anti-tumour innate immunity is active, the tumour is controlled, and the number of cancerous germs decreases, involving the ICs  $(S, R, Q, X) = (1.29, 0.39, 0.29, 0.29)$ . Because oncogenes rely on cancerous tissues for proliferation, this reduction could result in oncolytic virus deterioration and, as a result, therapy rejection. In a nutshell, both the antibody reaction and oncogenic virotherapy were unable to maintain adequate tissues and save the patient’s life in this circumstance.

Finally, Figs. 11–12 represents the response of nutrient, normal tissues, tumour tissues and free M1 virus utilizing the ICs  $(S, R, Q, X) = (0.5, 0.34, 0.29, 0.29)$ . Throughout this situation, the tumour is controlled by the anti-tumour innate invulnerability, that reduces the number of cancerous lymphocytes while increasing the number of healthy tissues. As a result, oncogenic virotherapy can no longer consistently defend the tumour, and the reduction in cancerous growth corresponds to a drop in oncolytic pathogen generation. However, this stratagem

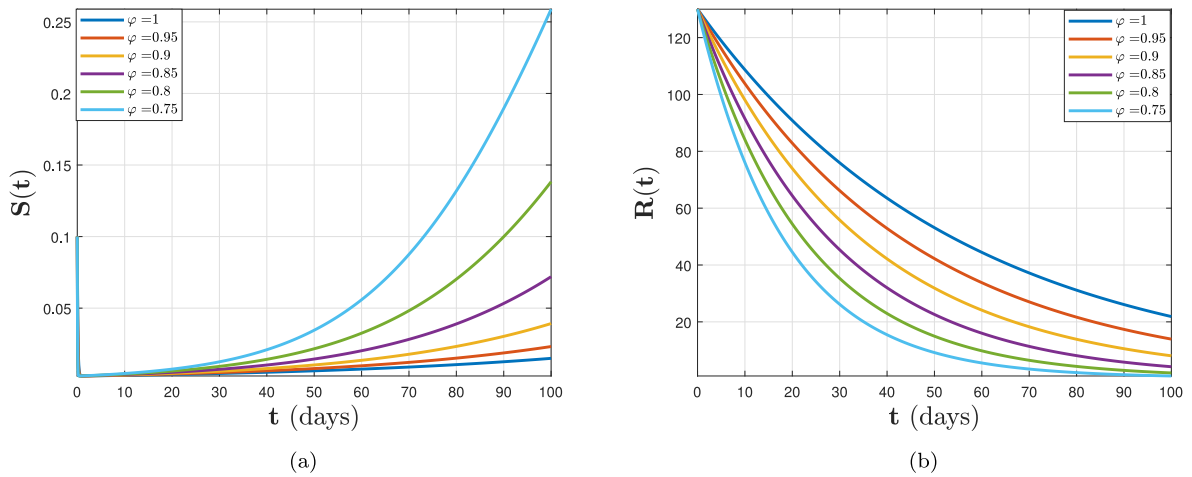


Fig. 5. Two-dimensional representation of fractional model of oncolytic efficacy (10) for multiple fractional orders utilizing the ICs  $(S, R, Q, X) = (0.36, 0.24, 0.12, 0.12)$  of (a) nutrient (b) normal cells.

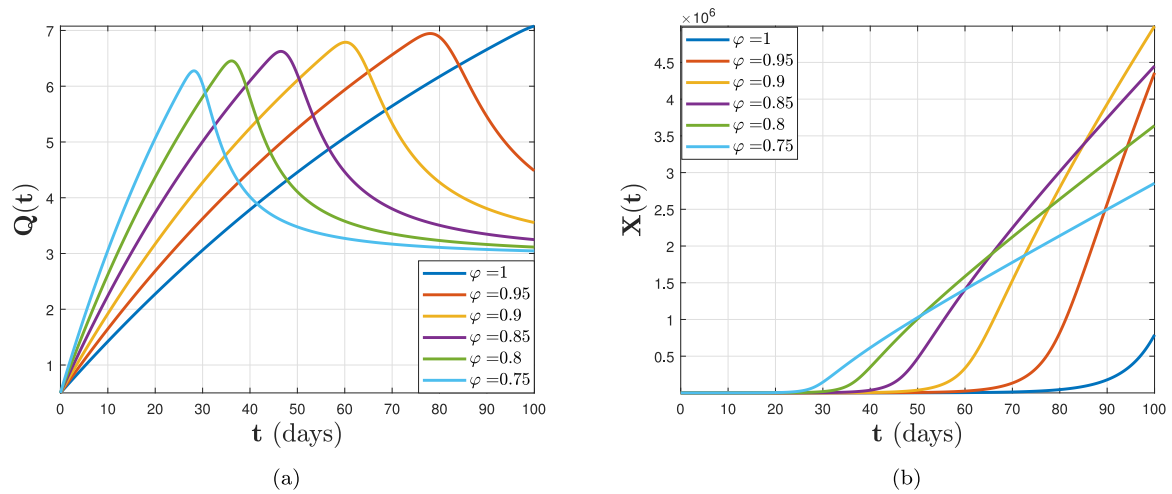


Fig. 6. Two-dimensional representation of fractional model of oncolytic efficacy (10) for multiple fractional orders utilizing the ICs  $(S, R, Q, X) = (0.36, 0.24, 0.12, 0.12)$  of (a) tumour cells (b) free M1 virus.

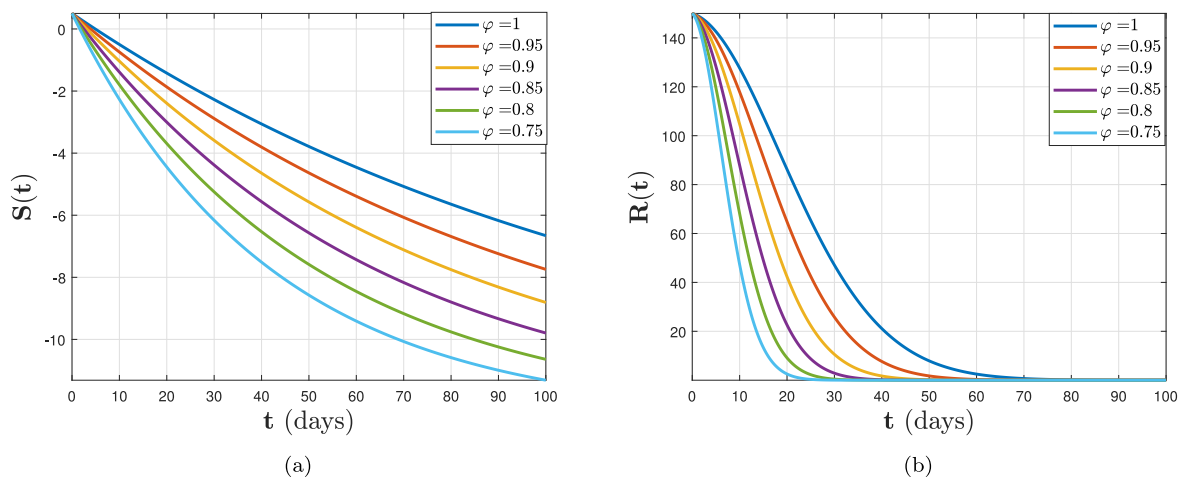


Fig. 7. Two-dimensional representation of fractional model of oncolytic efficacy (10) for multiple fractional orders utilizing the ICs  $(S, R, Q, X) = (0.24, 0.16, 0.08, 0.08)$  of (a) nutrient (b) normal cells.

might be applied to various recipient cellular functions. That is to say, graphics have the potential to effectively enable not only oncotherapy, but also dominating disruption of localized rheumatoid manifestations,

inhibition of innate immunity, and possibly performance expectancy for compensatory or proliferative tissue renovation.

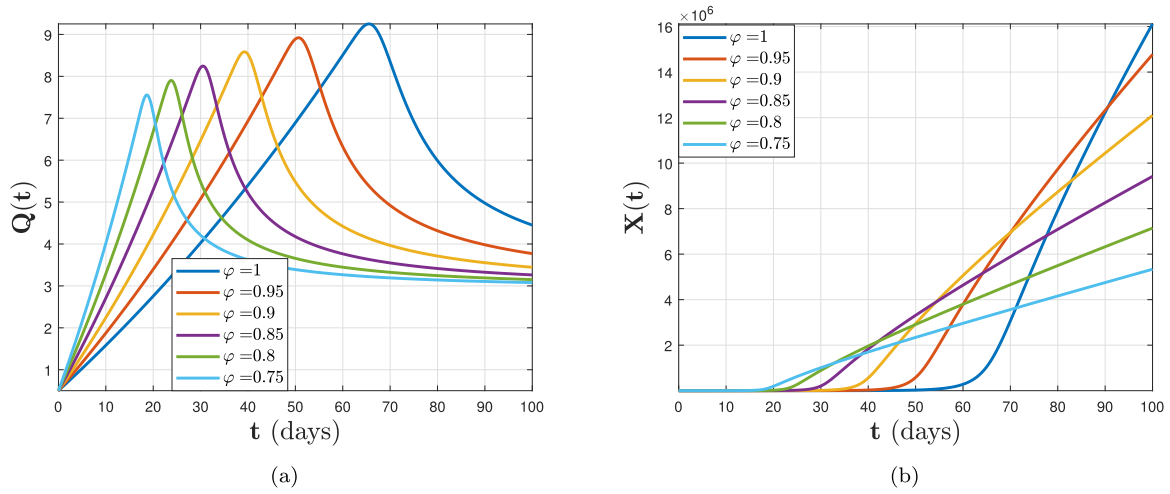


Fig. 8. Two-dimensional representation of fractional model of oncolytic efficacy (10) for multiple fractional orders utilizing the ICs  $(S, R, Q, X) = (0.24, 0.16, 0.08, 0.08)$  of (a) tumour cells (b) free M1 virus.

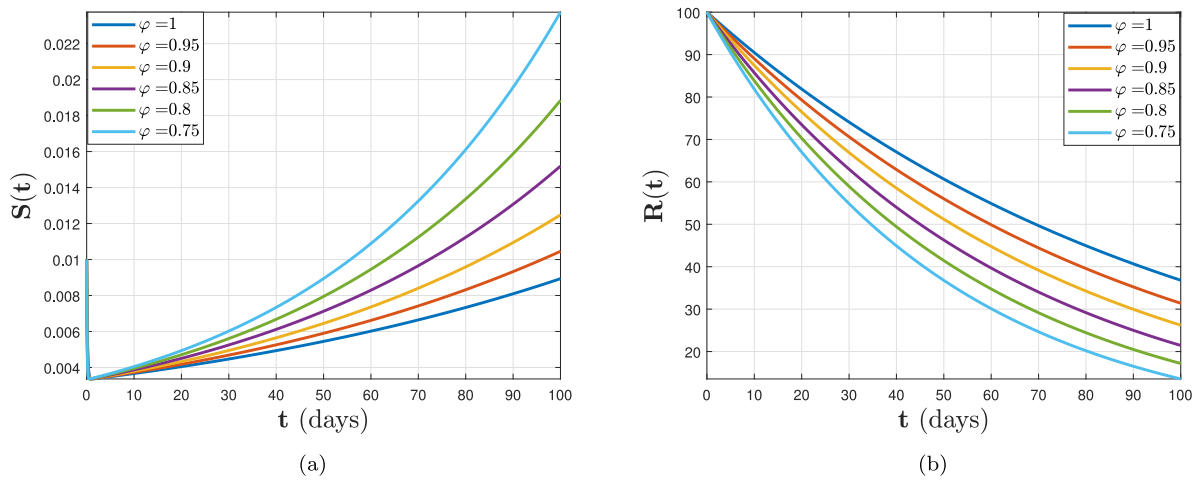


Fig. 9. Two-dimensional representation of fractional model of oncolytic efficacy (10) for multiple fractional orders utilizing the ICs  $(S, R, Q, X) = (1.29, 0.39, 0.29, 0.29)$  of (a) nutrient (b) normal cells.

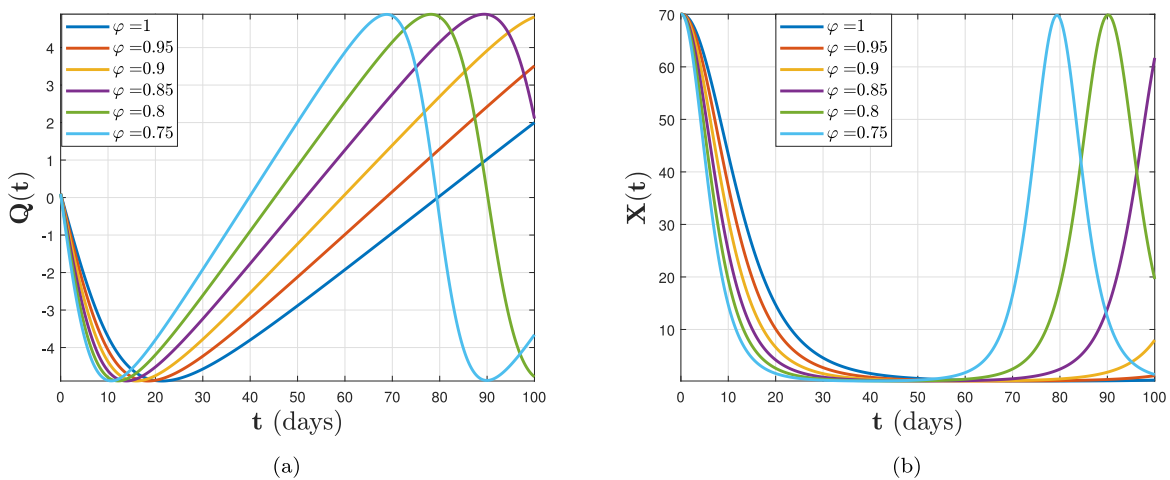


Fig. 10. Two-dimensional representation of fractional model of oncolytic efficacy (10) for multiple fractional orders utilizing the ICs  $(S, R, Q, X) = (1.29, 0.39, 0.29, 0.29)$  of (a) tumour cells (b) free M1 virus.

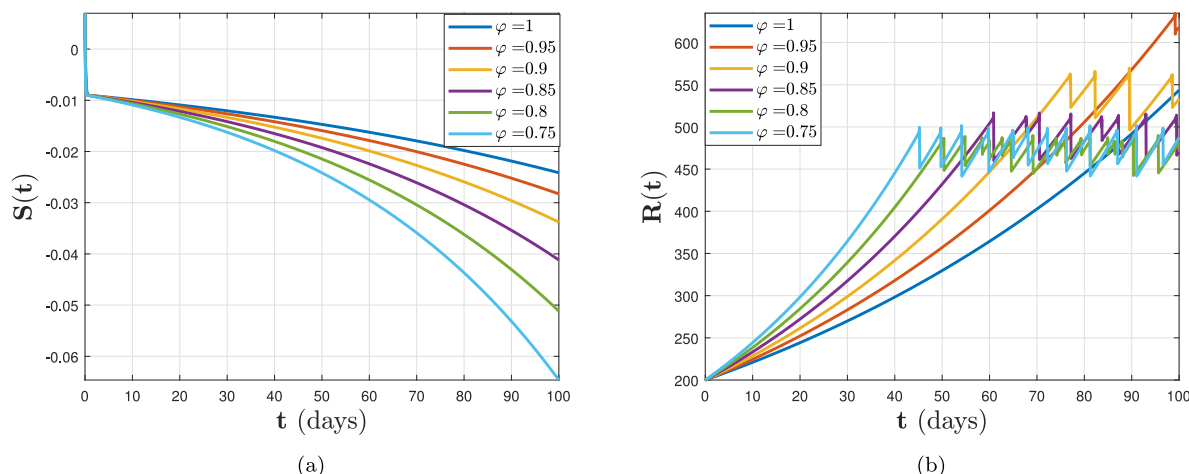


Fig. 11. Two-dimensional representation of fractional model of oncolytic efficacy (10) for multiple fractional orders utilizing the ICs  $(S, R, Q, X) = (0.5, 0.34, 0.29, 0.29)$  of (a) nutrient (b) normal cells.

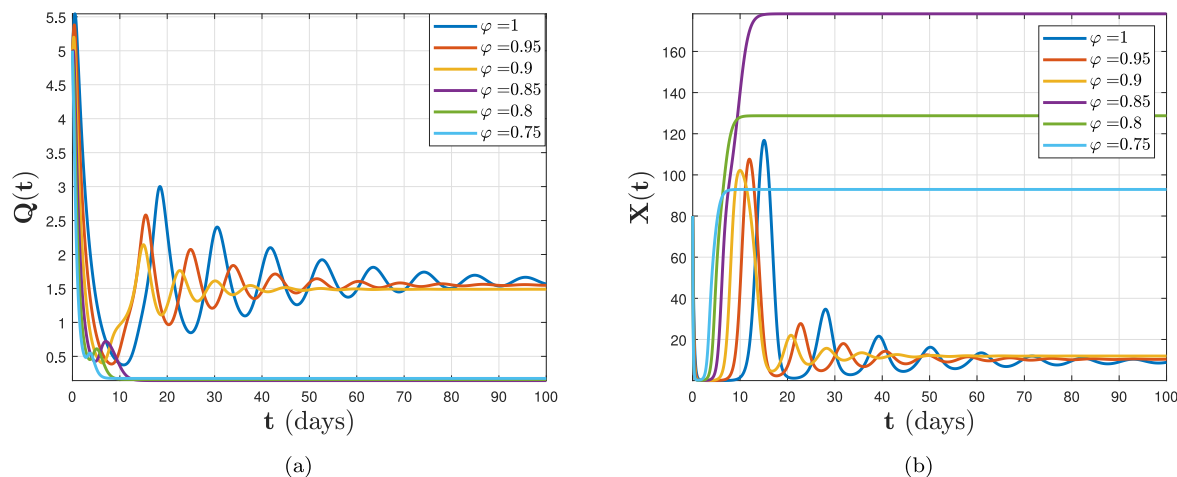


Fig. 12. Two-dimensional representation of fractional model of oncolytic efficacy (10) for multiple fractional orders utilizing the ICs  $(S, R, Q, X) = (0.5, 0.34, 0.29, 0.29)$  of (a) tumour cells (b) free M1 virus.

**Conclusion**

In this investigation, we researched the impact of an oncogenic M1 virotherapy framework in this research, taking into account the memory impact expressed by the ABC fractional derivative. Several mathematical aspects of the aforesaid model are discussed in detail. The four potential EPs in the framework were discovered to be non-competitive equilibrium  $\mathcal{E}_0$ , cancer-free equilibria  $\mathcal{E}_1$ , therapeutic inability equilibria  $\mathcal{E}_2$ , and partially accomplishment equilibria  $\mathcal{E}_3$ . The findings suggest that the M1 infection is partly effective at reducing cancer growth while enhancing immune tissues, perhaps minimizing tumorigenicity and regulating infection severity. According to the aforementioned numerical conclusions, the persistence of the ABC fractional derivative has no relevance to the structural characterization of equilibria. We notice that the fractional order impacts the pace of convergence and the time-frame it requires to reach equilibrium, which is obtained from the numeric computations. It is worth-mentioning that our outcomes discussed all aspects of the oncolytic M1 efficacy model and are more general than the results derived by [4]. The fractional derivative in the perspective of ABC having an M-L kernel is used to derive the outcome of this research. Modelling the behaviour of oncolytic M1 drug treatment using the newly developed fractal-fractional derivative operator [43] could be very interesting. In addition, we will also

incorporate additional biological parameters, including dispersion [44, 45] and immunology [46] into our framework described in (10).

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Not Applicable

**CRediT authorship contribution statement**

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## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Data availability

No data were used to support this study.

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