EUROPEAN JOURNAL OF PURE AND APPLIED MATHEMATICS
2026, Vol. 19, Issue 2, Article Number 6482
ISSN 1307-5543 – ejpam.com
Published by New York Business Global

Theoretical and Computational Analysis of Coupled System of Multiple Sclerosis Using Non-Singular Type Derivative

Ghazala Nazir[1,*], Rahmat Ali Khan[2], Faheem Yaqoob[3], Sadique Ahmad[4], Mohammed A. Elaffendi[4], Abdelhamied Ashraf Ateya[4]

[1] *Department of Mathematics, Mohi-ud-Islamic University, Narian Sharif, AJK, Pakistan*
[2] *Department of Mathematics, University of Malakand, Chakdara Dir(L) 18000, KPK, Pakistan*
[3] *Department of Computer Sciences, Faculty of Basic Science, University of Poonch, Rawalakot Azad Kashmir, Pakistan*
[4] *EIAS Data Science and Blockchain Laboratory, College of Computer and Information Sciences, Prince Sultan University, Riyadh 11586, Saudi Arabia*

Abstract. In this study, we develop and analyze a mathematical model for multiple sclerosis (MS), a progressive neurological disorder of the central nervous system. One of its main properties is the dissemination of lesions in time and space. The model is constructed within the framework of non-singular fractional derivatives of non-integer order. Using nonlinear functional analysis, we establish key qualitative properties of the system, including existence and stability of solutions. The Banach and Krasnoselskii fixed point theorems are applied to demonstrate solution existence, while stability is investigated through the Hyers-Ulam approach. For the numerical approximation of the model's compartments, an Adams-Bashforth method is employed, and simulations are presented to illustrate the dynamic behavior of the system under varying fractional orders. The results indicate that modeling with non-singular fractional derivatives provides a powerful framework for exploring neurodegenerative disorders and holds potential for enhancing strategies to predict and manage the progression of MS.

2020 Mathematics Subject Classifications: 26A33, 34A08, 92C60, 47H10

Key Words and Phrases: Neural disease model, non-singular derivative, existence theory, fractional differential equations, stability analysis

*Corresponding Author.
DOI: https://doi.org/10.29020/nybg.ejpam.v19i2.6482

Email addresses: ghazalanazir732@gmail.com (G. Nazir), rahmat_alipk@yahoo.com (R. A. Khan), faheemyaqoob@uper.edu.pk (F. Yaqoob), saahmad@psu.edu.sa (S. Ahmad), affendi@psu.edu.sa (M. A. Elaffendi), aateya@psu.edu.sa (A. A. Ateya)

1. Introduction

Currently, the area devoted to epidemiology has great attention. Various diseases like HIV, HBV, Ebola, malaria, etc, have been investigated from various aspects. Controlling these diseases in society is a great challenge since ancient times. Human beings continuously have been trying to save society from such diseases. Here it is remarkable that acquired chronic neurological disease known as multiple sclerosis is increasingly affecting young, adults, and in many countries like Australia affects three times more women than men. The said disease is one of the neural diseases. The aforesaid infection is an autoimmune affiliated neurological syndrome comprising the vital nervous system, primarily destroying oligodendrocytes and consequently upsetting the neural function in the brain and spine. Symptoms include gait disturbance, impaired vision, and other neurological infections. The type and inflexibility of symptoms differ from one case to another from a slight case that needs no treatment to a severe case with disturbing diurnal activities that need relapse preventative therapies. For instance, authors have conducted some reviewers in this regard by analyzing existing literature about the aforementioned disease in [1] and [2]. Also, some further detail about the neural disease can be found in [3] and [4].Moreover [5] discussed the dual-path network for automated polyp segmentation in colonoscopy and Researchers, therefore, use various tools to investigate such diseases for treatment, awareness of the public, and cure purposes. In this regard, mathematical epidemiology has got considerable attention recently. Various epidemic disease models have been studied in [6]. Since approximately, three million people around the world have had the neurological disease. The reason for the said disease is quite unsatisfying and numerous aspects have been planned to describe the tools of the syndrome, similar as inheritable, immune, viral, or ecological. As compared to men, the said disease is more than three times in women.

Various diseases are investigated by the use of mathematical models. The powerful tools to study and analyze various diseases from different perspectives are known as mathematical models. Recently neurological diseases are severer threat to men, women as well as to young people throughout the world. Several research articles have been published on the mathematical models of the mentioned disease. For instance, authors [7] have investigated a simple mathematical model for relapsing-remitting the said infection. Therefore, authors [8] have studied the following three compartments model as

$$\begin{cases} \dfrac{d\ell_1}{dt} = r\ell_1(1-\ell_1) - b\ell_1\ell_3, \\ \dfrac{d\ell_2}{dt} = b\ell_1\ell_3 - a\ell_2, \\ \dfrac{d\ell_3}{dt} = c\ell_2 - d\ell_1\ell_3 - k\ell_3, \\ \ell_1(0) = \ell_{1,0},\ \ell_2(0) = \ell_{2,0},\ \ell_3(0) = \ell_{3,0}. \end{cases} \tag{1}$$

Here we explained that ℓ_1 stands for the ratio of the robust nerve cells at time t, ℓ_2 denoted the density of the affected nerve cells and ℓ_3 denotes the toxic effector's nerve cells. The other parameters have been explained in Table 1. The proposed multiple

Nomenclature	Description
$\ell_{1,0}$	Initial data of robust nerve cells
$\ell_{2,0}$	Inial data for affected nerve cells
$\ell_{3,0}$	Initial data for toxic effectors nerves
r	Growth rate of the healthy cells
k	Death rate of virus
a	Death rate of cells
b	Attack rate on healthy cells
c	Cache rate of the virus
d	The rate at which virus is affected by the healthy cells

Table 1: Nomenclature and description of the model (2).

sclerosis (MS) model captures important biological characteristics of the disease and provides insight into how the immune system interacts with neural tissues over time. The inclusion of fractional-order derivatives introduces memory effects, which are biologically meaningful as they reflect the long-term dependence of neurological damage and immune responses. In MS, the immune system does not act instantaneously; instead, cumulative past events (such as prior relapses or inflammatory activity) influence the present state of the disease. This phenomenon is well represented in the fractional-order framework. Recently authors [9] have investigated a model for automated diagnosis of amyotrophic lateral sclerosis. They have used neural networks with fractional position updated based on electromyogram and firefly algorithm. In the same line, authors [10] have investigated the psychological disease by using artificial intelligence tools. Authors [11] have established some theoretical analysis on multiple sclerosis recently. Authors [12] have induced control of neuro-inflammation in a preclinical model of multiple sclerosis which included a promising simple, effective, non-invasive, and low-cost therapy. Currently, authors [13] have discussed microstructure-weighted connectomics in the aforesaid disease. The superficial white matter integrity has been identified in the said disease in early stages in [14]. Moreover [15] also discussed the human papilloma virus (HPV) disease with new perspectives of fractional calculus. It is important to note that ordinary calculus has not been able to adequately describe certain features of mathematical models. Consequently, non-integer-order derivatives can provide a more thorough definition of memory and genetic traits. Because a derivative of non-integer order is a definite integral that provides the whole range of a function. As a specific case, the aforementioned spectrum maintains its integer counterpart. Additionally, compared to integer order, analogous operators offer greater degree of freedom. A thorough overview, background, and basic findings have been examined in [16], [17], and [18]. Additionally, some intriguing uses of the mentioned field have been studied [19], [20], [21]. Given the significance of fractional calculus, [22] and [21] have examined mathematical models and their optimization under the aforementioned idea. In [23], [24], and [25], several mathematical models for examining fractional order dynamics have been examined. There is currently no unique definition for differential op-

erators with non-integer orders. There are several definitions in literature. One definition of a power law uses a singular kernel, whereas the other uses a nonsingular kernel. In 2015 and 2016, respectively, certain new definitions of non-integer order derivatives were introduced. Numerous mathematical models have been studied using the concerned operators [26], [27], [28], and [29]. Non-local non-singular derivatives are another name for the aforementioned operators. In light of this, numerous methods have been developed to solve different derivatives-related problems analytically or numerically. Similarly, in [30], a biological model under the aforementioned derivative was numerically solved using the Adam Bash-Forth approach. For semi-analytical conclusions of different models under the aforementioned derivatives, the authors in [31] have also employed the iterative technique. For qualitative results, such as stability and numerical simulations, the model (1) has not been examined under the $\mathscr{ABC}$ derivative. We point out here that the $\mathscr{ABC}$ derivative is a nonlocal differential operator with a non-singular kernel that may be used to describe a wide range of processes and phenomena. Furthermore, the typical fractional order derivative is insufficient to represent certain types of dissipative phenomena. The non-singular derivatives have thus been employed to great effect (see [31]). Additionally, since the majority of these real-world applications rely on the function's history, nonlocal derivatives are becoming increasingly intriguing and useful in accurately describing the phenomenon in the form of a mathematical model. Additionally, compared to Caputo-Fabrizio's differential operator, which likewise uses a nonlocal kernel but is not a fractional differential operator, the aforementioned differential operator is more broad. However, the aforementioned $\mathscr{ABC}$ derivative has certain drawbacks because differential operators of this type have finalization conditions, therefore the function on the right side of the equation should be subject to an additional assumption.

Since integer order derivative is a local operator while that of fractional order is the global operator. Also derivative of order $\omega \in (0, 1)$ is defiant integral but ordinary derivative at $\omega = 1$. Therefore, in the interpretation of numerical presentation graphically, we see that as $\omega \to 1$, the concerned curves are converging to integer order. Also, the global dynamics cannot be explained by integer order. Fractional differential operators are what enable us to better grasp real-world problems by describing their global dynamics. Although several mathematicians have been studied multiple sclerosis using classical integer-order or singular fractional-order operators, these approaches often fall short in non-local characteristics that are fundamental to the progression of neurodegenerative disorders. To address this limitation, we adopt the Atangana-Baleanu-Caputo (ABC) fractional derivative with its non-singular kernel. Through the development of a three-compartment MS model under this framework, together with existence and stability analysis and supporting numerical simulations, our study advances the mathematical modeling of MS and offers a more realistic description of its progression dynamics Therefore, inspired by aforesaid literature, we study existence theory, stability, and numerical analysis of the following MS

disease model under $\mathscr{ABC}$ derivative with order $\omega \in (0,1]$ as given by

$$\begin{cases} {}^{\mathscr{ABC}}\mathcal{D}_t^{\omega}\ell_1(t) = r\ell_1(1-\ell_1) - b\ell_1\ell_3, \\ {}^{\mathscr{ABC}}\mathcal{D}_t^{\omega}\ell_2(t) = b\ell_1\ell_3 - a\ell_2, \\ {}^{\mathscr{ABC}}\mathcal{D}_t^{\omega}\ell_3(t) = c\ell_2 - (d\ell_1 - k)\ell_3, \end{cases} \tag{2}$$

under the conditions

$$\ell_q(0) \geq 0,\ q = 1,2,3.$$

In order to deduce some sufficient criteria for the existence of a solution to the suggested model, we develop the existence theory using the arguments presented in [32], [33]. We also set some requirements for stability of the U-H type. We simulate our results for some supplied data using the numerical scheme for $\mathscr{ABC}$ type derivative that has been devised in [34] and [35]. Additionally, we refer to [36] for the specific features of the $\mathscr{ABC}$ type derivative. Additional pertinent findings about fractional order models can be found in [37–39].

We have produced our manuscript as follows: In order to provide an overview of the model and methodology that we use, Section 1 is linked to some theory. Some supporting documentation needed for study is included in Section 2. The model's stability results and qualitative analysis are presented in Section 3. We present a method to simulate a series solution to the problem in Section 4. In Section 5, the Matlab graphs and discussion are shown. Section 6 concludes with a brief comment.

2. Preliminaries

We recollect some definitions from [36].

Definition 1. *Let $\varrho \in \mathcal{H}^1(0,T)$ be Hilbert space and $\omega \in (0,1]$, one has*

$${}^{\mathscr{ABC}}\mathcal{D}_t^{\omega}(\varrho(t)) = \int_0^t \frac{\mathscr{ABC}(\omega)}{(1-\omega)}\varrho'(\vartheta)E_{\omega}\left[\frac{-\omega(t-\vartheta)^{\omega}}{1-\omega}\right]d\vartheta.$$

Here, E_{ω} is called as a Mittag-Leffler function and $\mathscr{ABC}(\omega)$ is the normalization function.

Definition 2. *The fractional integral of $\mathscr{ABC}$ is*

$${}^{\mathscr{AB}}\mathcal{I}_t^{\omega}(\varrho(t)) = \frac{(1-\omega)\varrho(t)}{\mathscr{ABC}(\omega)} + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{ABC}(\omega)\Gamma(\omega)}\varrho(\vartheta)d\vartheta.$$

Lemma 1 ([36], Proposition 3)**.** *If $t = 0$, then $\varrho(0) = 0$, therefore, the solutions of*

$$\begin{aligned} {}^{\mathscr{ABC}}\mathscr{D}_t^{\omega}(\varrho(t)) =& \varrho(t),\ \omega \in (0,1], \\ \varrho(0) =& \varrho_0, \end{aligned}$$

is described as

$$\varrho(t) = \varrho_0 + \frac{(1-\omega)\varrho(t)}{\mathscr{ABC}(\omega)} + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{ABC}(\omega)\Gamma(\omega)}\varrho(\vartheta)d\vartheta.$$

Theorem 1. *[33]. Let* $\mathbf{E} \subset \mathbf{W}$, *is closed subset of Banach space, and one has* $\mathbf{A}, \mathbf{B}$, *with*

(i) $\mathbf{A}\varrho_1 + \mathbf{B}\varrho_2 \in \mathbf{E}$;

(ii) $\mathbf{A}$ *is a contractive, and* $\mathbf{B}$ *is completely continuous,*

then $\mathbf{A}\varrho + \mathbf{B}\varrho = \varrho$ *has at least one solution.*

3. Analysis of the Proposed Model

Fixed point theory is a dominant technique to study qualitative behavior for the aforesaid problem. By applying the Krasnoselskii's theorem, we check the qualitative behavior for solutions of problem (2). For this purpose, our considered model (2) takes the form

$$\begin{cases} \phi_1(t, \ell_1, \ell_2, \ell_3) = r\ell_1(1-\ell_1) - b\ell_1\ell_3, \\ \phi_2(t, \ell_1, \ell_2, \ell_3) = b\ell_1\ell_3 - a\ell_2, \\ \phi_3(t, \ell_1, \ell_2, \ell_3) = c\ell_2 - d\ell_1\ell_3 - k\ell_3. \end{cases} \tag{3}$$

Further we use

$$\mathscr{Z}(t) = \begin{cases} \ell_1(t) \\ \ell_2(t)\,, \\ \ell_3(t) \end{cases} \quad \mathscr{Z}_0 = \begin{cases} \ell_{1,0} \\ \ell_{2,0}\,, \\ \ell_{3,0} \end{cases} \quad \mho(t, \mathscr{Z}(t)) = \begin{cases} \phi_1(t, \ell_1, \ell_2, \ell_3) \\ \phi_2(t, \ell_1, \ell_2, \ell_3) \\ \phi_3(t, \ell_1, \ell_2, \ell_3). \end{cases} \tag{4}$$

In view of (3), and (4), the system (2) yields

$$\begin{aligned} {}^{\mathscr{A}\mathscr{B}\mathscr{C}}\mathcal{D}_t^{\omega}[\mathscr{Z}(t)] &= \mho(t, \mathscr{Z}(t)), \\ \mathscr{Z}(0) &= \mathscr{Z}_0,\ \mathcal{J} = [0, T]. \end{aligned} \tag{5}$$

In view of the Definition 2, the problem (5) can be transformed as

$$\mathscr{Z}(t) = \mathscr{Z}_0 + \Big[\mho(t, \mathscr{Z}(t))\Big] \frac{(1-\omega)}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} + \frac{\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \int_0^t (t-\vartheta)^{\omega-1} \mho(\vartheta, \mathscr{Z}(\vartheta)) d\vartheta,\ t \in \mathcal{J}. \tag{6}$$

Here, we define $\mathbf{W} = \mathcal{C}(\mathcal{J} \times \mathcal{R}^3, \mathcal{R})$ with the norm $\|\mathscr{Z}\| = \sup_{t \in \mathcal{J}}\{|\mathscr{Z}(t)| :\ \mathscr{Z} \in \mathbf{W}\}$. Following assumptions are stated for onward analysis:

(A_1) For constant $\Omega_{\mho} > 0$, one has

$$|\mho(t, \mathscr{Z}) - \mho(t, \widehat{\mathscr{Z}}(t))| \leq \Omega_{\mho} |\mathscr{Z} - \widehat{\mathscr{Z}}|.$$

(A_2) For constants $\Lambda_{\mho} > 0, \nabla_{\mho} >, 0$ we have $|\mho(t, \mathscr{Z}(t))| \leq \Lambda_{\mho}|\mathscr{Z}| + \nabla_{\mho}$.

Theorem 2. *Problem* (5) *has at least one solution under assumption* $(\mathbb{A}_2)$*, if* $\Omega_{\mho} < \mathcal{M}(\omega)$. *Consequently, the concerned model* (2) *has at least one solution.*

Proof. If $\mathbf{E} = \{\mathscr{Z} \in \mathbf{W} : \|\mathscr{Z}\| \leq r\} \subset \mathbf{W}$ be closed bounded set, then we describe the operators as

$$\begin{aligned} \mathbf{A}(\mathcal{G})(t) &= G_0 + \left[\Phi(t, \mathscr{Z}(t))\right] \frac{(1-\omega)}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)}, \\ \mathbf{B}(\mathcal{G})(t) &= \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} \Phi(\vartheta, \mathscr{Z}(\vartheta)) d\vartheta. \end{aligned} \tag{7}$$

To show the operator $\mathbf{A}$ in equation (7) is contraction, let $\mathscr{Z}, \widehat{\mathscr{Z}} \in \mathbf{W}$, we have

$$\begin{aligned} \|\mathbf{A}(\mathcal{G}) - \mathbf{A}(\widehat{\mathscr{Z}})\| &= \sup_{t\in\mathcal{J}} |\mathbf{A}(\mathcal{G})(t) - \mathbf{A}(\widehat{\mathscr{Z}})(t)|, \\ &= \sup_{t\in\mathcal{J}} \left| \frac{(1-\omega)}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} \left[\mho(t, \mathscr{Z}) - \mho(t, \widehat{\mathscr{Z}})\right] \right|. \end{aligned}$$

From which, we have

$$\|\mathbf{A}(\mathcal{G}) - \mathbf{A}(\widehat{\mathscr{Z}})\| \leq \frac{(1-\omega)\Omega_{\mho}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} |\mathscr{Z} - \widehat{\mathscr{Z}}|,$$

which represents that A is contractive.

To prove that $\mathbf{B}$ is completely continuous operator, let

$$\begin{aligned} |\mathbf{B}(\mathscr{Z})(t)| &= \left| \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \mathscr{Z}(\vartheta, u, v, w) d\vartheta \right|, \\ &\leq \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \left| \mho(\vartheta, \mathscr{Z}(\vartheta)) \right| d\vartheta. \end{aligned} \tag{8}$$

Taking supremum over $\mathcal{J}$ of equation (8) which yields

$$\begin{aligned} \|\mathbf{B}(\mathcal{G})\| &\leq \sup_{t\in\mathcal{J}} \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} |\mho(\vartheta, \mathscr{Z}(\vartheta))| d\vartheta, \\ &\leq \int_0^T \frac{\omega(T-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \left[\Lambda_{\mho}\|\mathscr{Z}\| + \nabla_{\mho}\right] d\vartheta, \\ &\leq \frac{T^{\omega}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \left(\Lambda_{\mho} r + \nabla_{\mho}\right). \end{aligned} \tag{9}$$

Hence $\mathbf{B}$ is bounded in (9). Let $t_2 > t_1$, then one has

$$\begin{aligned} |\mathbf{B}(\mathcal{G})(t_2) - \mathbf{B}(\mathcal{G})(t_1)| &= \left| \int_0^{t_1} \frac{\omega[(t_1-\vartheta)^{\omega-1} - (t_2-\vartheta)^{\omega-1}]}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \mho(\vartheta, \mathscr{Z}) d\vartheta + \int_{t_1}^{t_2} \frac{\omega(t_2-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \mho(\vartheta, \mathscr{Z}) d\vartheta \right|, \\ &\leq \frac{1}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} (\Lambda_{\mho} r + \nabla_{\mho})(t_2^{\omega} - t_1^{\omega} + (t_2 - t_1)^{\omega}). \end{aligned} \tag{10}$$

From (10), we see that

$$|\mathbf{B}\mathscr{Z}(t_2) - \mathbf{B}\mathscr{Z}(t_1)| \to 0, \text{ as } t_2 \to t_1.$$

Also **B** is uniformly continuous and bounded. therefore **B** is equi-continuous and hence **B** relatively compact. Hence criteria of Theorem 1 full filled. Thus the proposed model has at least one solution.

Theorem 3. *Under assumption (A_1) and continuity of Φ if the condition $\frac{(1+T^{\omega})\Omega_{\mho}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} < \Gamma(\omega)$ satisfies, then the problem* (2) *has a unique solution.*

Proof. Let $\mathcal{V} : \mathbf{W} \to \mathbf{W}$ be an operator define by

$$\mathcal{V}(\mathcal{G})(t) = \mathscr{Z}_0 + \frac{(1-\omega)}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)}\Big[\mho(t, \mathscr{Z}(t))\Big] + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\Phi(\vartheta, \mathscr{Z}(\vartheta))d\vartheta.$$

Let $\mathscr{Z}, \widehat{\mathscr{Z}} \in \mathbf{W}$, then

$$\begin{aligned}
\|\mathcal{V}(\mathcal{G}) - \mathcal{V}(\widehat{\mathscr{Z}})\| &= \sup_{t\in\mathcal{J}}\Big|\mathcal{V}(\mathcal{G})(t) - \mathcal{V}(\widehat{\mathscr{Z}})(t)\Big|, \\
&\leq \sup_{t\in\mathcal{J}}\left|\frac{(1-\omega)}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)}\Big[\mho(t, \mathscr{Z}(t)) - \mho(t, \widehat{\mathscr{Z}}(t))\Big]\right| \\
&+ \sup_{t\in\mathcal{J}}\int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\Big|\mho(\vartheta, \mathscr{Z}(\vartheta)) - \mho(\vartheta, (\widehat{\mathscr{Z}}(\vartheta)))\Big|d\vartheta, \\
&\leq \frac{(1-\omega)}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)}\Omega_{\mho}\Big\|\mathscr{Z} - \widehat{\mathscr{Z}}\Big\| + \frac{T^{\omega}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\Omega_{\mho}\Big\|\mathscr{Z} - \widehat{\mathscr{Z}}\Big\|, \\
\text{which implies that } \|\mathcal{V}(\mathcal{G}) - \mathcal{V}(\widehat{\mathscr{Z}})\| &\leq \frac{(1+T^{\omega})\Omega_{\mho}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\Big\|\mathscr{Z} - \widehat{\mathscr{Z}}\Big\|. \qquad (11)
\end{aligned}$$

Hence the proof.

Let the function $\rho.$ such that $\rho \to 0$ at $t \to 0$, and

- $|\rho(t)| < \epsilon$, for $\epsilon > 0$;
- ${}^{\mathscr{A}\mathscr{B}\mathscr{C}}\mathcal{D}_t^{\omega}\mathscr{Z}(t) = \mho(t, \mathscr{Z}(t)) + \rho(t)$.

Lemma 2. *The solutions of the perturbed problem*

$$\begin{aligned}
{}_0^{\mathscr{A}\mathscr{B}\mathscr{C}}\mathcal{D}_t^{\omega}\mathscr{Z}(t) &= \mho(t, \mathscr{Z}(t)) + \rho(t), \\
\mathscr{Z}(0) &= \mathscr{Z}_0,
\end{aligned}$$

satisfy the given equation

$$\left|\mathscr{Z}(t) - \left(\mathscr{Z}_0 + \Big[\mho(t, \mathscr{Z}(t)) - \mho_0\Big]\frac{(1-\omega)}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\mho(\vartheta, \mathscr{Z}(\vartheta))d\vartheta\right)\right| \leq \mathcal{L}_{\mho}\epsilon,$$

where $\mathcal{L}_{\mho} = \frac{\mathcal{F}_{\mho}(1+T)}{\mathcal{M}(\omega)}$.

Theorem 4. *The solutions of problem* (5) *is H- U stable under assumption* (A_2) *with Lemma 2, if* $\mathcal{L}_{\mho} < 1$ *. Therefore, approximate solution of the concerned problem* (2) *is H-U stable.*

Proof. Let $\mathscr{Z} \in \mathbf{W}$ be any solution and $\bar{\mathscr{Z}} \in \mathbf{W}$ be at most one solution, then

$$\begin{aligned}
|\mathscr{Z}(t) - \widehat{\mathscr{Z}}(t)| &= \left| \mathscr{Z}(t) - \left(\mathscr{Z}_0 + \left[\mho(t, \widehat{\mathscr{Z}}(t)) - \mho_0 \right] \frac{(1-\omega)}{\mathcal{M}(\omega)} + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \mho(\vartheta, \widehat{\mathscr{Z}}(\vartheta)) d\vartheta \right) \right|, \\
&\leq \left| \mathscr{Z}(t) - \left(\mathscr{Z}_0 + \left[\mho(t, \mathscr{Z}(t)) - \mho_0 \right] \frac{(1-\omega)}{\mathcal{M}(\omega)} + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \mho(\vartheta, \mathscr{Z}(\vartheta)) d\vartheta \right) \right| \\
&+ \left| \left(\mathscr{Z}_0 + \left[\mho(t, \mathscr{Z}(t)) - \mho_0 \right] \frac{(1-\omega)}{\mathcal{M}(\omega)} + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \mho(\vartheta, \mathscr{Z}(\vartheta)) d\vartheta \right) \right. \\
&- \left. \left(G_0 + \left[\Phi(t, \widehat{\mathscr{Z}}(t)) - \mho_0 \right] \frac{(1-\omega)}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)} \mho(\vartheta, \widehat{\mathscr{Z}}(\vartheta)) d\vartheta \right) \right|, \\
&\leq \mathcal{L}_{\mho}\epsilon + \mathcal{L}_{\mho} \| \mathscr{Z} - \widehat{\mathscr{Z}} \|.
\end{aligned}$$

This implies that

$$\| \mathscr{Z} - \widehat{\mathscr{Z}} \| \leq \frac{\mathcal{L}_{\mho}}{1 - \mathcal{L}_{\mho}} \epsilon.$$

So, the solutions of model (5) is H- U stable.

H–U stability demonstrates the resilience of the system to small perturbations, implying that minor variations in biological processes—such as immune activity, demyelination rates, or repair capacity—do not significantly alter the overall course of the disease. This interpretation highlights that the stability results go beyond mathematical validation, offering meaningful insight into the robustness or fragility of the disease system when subjected to slight biological or therapeutic changes, thereby enhancing the practical significance of our findings

4. Numerical solution of the concerned biological problem

Here, we write the equivalent integral form of our considered model as applying the initial condition together with the operator ${}^{\mathscr{A}\mathscr{B}\mathscr{C}}\mathcal{I}_0^{\omega}$ and convert the fraction model (1) into system of fractional integral equations as below

$$\begin{cases}
\ell_1(t) - \ell_1(0) = {}^{\mathcal{AB}}\mathcal{I}_0^{\omega} \phi_1(t, \ell_1(t), \ell_2(t), \ell_3(t)), \\
\ell_2(t) - \ell_2(0) = {}^{\mathcal{AB}}\mathcal{I}_0^{\omega} \phi_2(t, \ell_1(t), \ell_2(t), \ell_3(t)), \\
\ell_3(t) - \ell_3(0) = {}^{\mathcal{AB}}\mathcal{I}_0^{\omega} \phi_3(t, \ell_1(t), \ell_2(t), \ell_3(t)),
\end{cases} \tag{12}$$

which gives

$$\begin{aligned}
\ell_1(t) &= \ell_{1,0} + \frac{(1-\omega)\phi_1(t,\ell_1(t),\ell_2(t),\ell_3(t))}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\phi_1(\vartheta,\ell_1(\vartheta),\ell_2(\vartheta),\ell_3(\vartheta))d\vartheta,\\
\ell_2(t) &= \ell_{2,0} + \frac{(1-\omega)\phi_2(t,\ell_1(t),\ell_2(t),\ell_3(t))}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\phi_2(\vartheta,\ell_1(\vartheta),\ell_2(\vartheta),\ell_3(\vartheta))d\vartheta, \quad (13)\\
\ell_3(t) &= \ell_{3,0} + \frac{(1-\omega)\phi_3(t,\ell_1(t),\ell_2(t),\ell_3(t))}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} + \int_0^t \frac{\omega(t-\vartheta)^{\omega-1}}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\phi_3(\vartheta,\ell_1(\vartheta),\ell_2(\vartheta),\ell_3(\vartheta))d\vartheta.
\end{aligned}$$

Now we apply two point interpolation polynomial to approximate the nonlinear functions $\phi_i(t,\ell_1(t),\ell_2(t),\ell_3(t)),\ i=1,2,3$ over the interval $[t_{\mathrm{p}},t_{\mathrm{p+1}}]$ as

$$\begin{aligned}
\phi_i(t,\ell_1(t),\ell_2(t),\ell_3(t)) &\cong \frac{\phi_i(t_{\mathrm{p}},\ell_1(t_{\mathrm{p}}),\ell_2(t_{\mathrm{p}}),\ell_3(t_{\mathrm{p}}))(t-t_{\mathrm{p-1}})}{\mathrm{h}}\\
&+ \frac{\phi_i(t_{\mathrm{p-1}},\ell_1(t_{\mathrm{p-1}}),\ell_2(t_{\mathrm{p-1}}),\ell_3(t_{\mathrm{p-1}}))(t-t_{\mathrm{p}})}{\mathrm{h}}. \quad (14)
\end{aligned}$$

Hence writing the system (15) at $t=t_{\mathrm{p+1}}$, and then using the approximations described in (14) for $i=1,2,3$ in (15), and using $\mathrm{h}=t_{\mathrm{p+1}}-t_{\mathrm{p}}$, we have

$$\begin{aligned}
\ell_1(t_{\mathrm{p+1}}) &= \ell_{1,0} + \frac{1-\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)}\phi_1(t,\ell_1(t_{\mathrm{p}}),\ell_2(t_{\mathrm{p}}),\ell_3(t_{\mathrm{p}}))\\
&+ \frac{\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\int_{t_{\mathrm{p}}}^{t_{\mathrm{p+1}}}(t-\vartheta)^{\omega-1}\Bigg[\frac{\phi_1(t_{\mathrm{p}},\ell_1(t_{\mathrm{p}}),\ell_2(t_{\mathrm{p}}),\ell_3(t_{\mathrm{p}}))}{\mathrm{h}}(\vartheta-t_{\mathrm{p-1}})\\
&+ \frac{\phi_1(t_{\mathrm{p-1}},\ell_1(t_{\mathrm{p-1}}),\ell_2(t_{\mathrm{p-1}}),\ell_3(t_{\mathrm{p-1}}))}{\mathrm{h}}(\vartheta-t_{\mathrm{p}})\Bigg]d\vartheta,\\
\ell_2(t_{\mathrm{p+1}}) &= \ell_{2,0} + \frac{1-\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)}\phi_2(t,\ell_1(t_{\mathrm{p}}),\ell_2(t_{\mathrm{p}}),\ell_3(t_{\mathrm{p}})) \quad (15)\\
&+ \frac{\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\int_{t_{\mathrm{p}}}^{t_{\mathrm{p+1}}}(t-\vartheta)^{\omega-1}\Bigg[\frac{\phi_2(t_{\mathrm{p}},\ell_1(t_{\mathrm{p}}),\ell_2(t_{\mathrm{p}}),\ell_3(t_{\mathrm{p}}))}{\mathrm{h}}(\vartheta-t_{\mathrm{p-1}})\\
&+ \frac{\phi_2(t_{\mathrm{p-1}},\ell_1(t_{\mathrm{p-1}}),\ell_2(t_{\mathrm{p-1}}),\ell_3(t_{\mathrm{p-1}}))}{\mathrm{h}}(\vartheta-t_{\mathrm{p}})\Bigg]d\vartheta,\\
\ell_3(t_{\mathrm{p+1}}) &= \ell_{3,0} + \frac{1-\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)}\phi_3(t,\ell_1(t_{\mathrm{p}}),\ell_2(t_{\mathrm{p}}),\ell_3(t_{\mathrm{p}}))\\
&+ \frac{\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)\Gamma(\omega)}\int_{t_{\mathrm{p}}}^{t_{\mathrm{p+1}}}(t-\vartheta)^{\omega-1}\Bigg[\frac{\phi_3(t_{\mathrm{p}},\ell_1(t_{\mathrm{p}}),\ell_2(t_{\mathrm{p}}),\ell_3(t_{\mathrm{p}}))}{\mathrm{h}}(\vartheta-t_{\mathrm{p-1}})\\
&+ \frac{\phi_3(t_{\mathrm{p-1}},\ell_1(t_{\mathrm{p-1}}),\ell_2(t_{\mathrm{p-1}}),\ell_3(t_{\mathrm{p-1}}))}{\mathrm{h}}(\vartheta-t_{\mathrm{p}})\Bigg]d\vartheta.
\end{aligned}$$

Hence after solving above integral system (15), we finally obtain

$$\ell_1(t_{\mathrm{p+1}}) = \ell_1(t_0) + \frac{1-\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)}\phi_1(t,\ell_1(t_{\mathrm{p}}),\ell_2(t_{\mathrm{p}}),\ell_3(t_{\mathrm{p}}))$$

$$
\begin{aligned}
+ \quad & + \frac{\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} \sum_{k=0}^{\mathrm{p}} \Bigg(\frac{\phi_1(t_{\mathrm{p}}, \ell_1(t_{\mathrm{p}}), \ell_2(t_{\mathrm{p}}), \ell_3(t_{\mathrm{p}}))}{\Gamma(\omega+2)} \\
& \times \mathrm{h}^{\omega} \Big[(\mathrm{p}+1-i)^{\omega}(\mathrm{p}-k+2+\omega) - (\mathrm{p}-k)^{\omega}(\mathrm{p}-k+2+2\omega) \Big] \\
& - \frac{\phi_1(t_{\mathrm{p}-1}, \ell_1(t_{\mathrm{p}-1}), \ell_2(t_{\mathrm{p}-1}), \ell_3(t_{\mathrm{p}-1}))}{\Gamma(\omega+2)} \mathrm{h}^{\omega} [(\mathrm{p}+1-k)^{\omega+1} - (\mathrm{p}-k)^{\omega}(\mathrm{p}-k+1+\omega)] \Bigg),
\end{aligned} \tag{16}
$$

$$
\begin{aligned}
\ell_2(t_{\mathrm{p}+1}) &= \ell_2(t_0) + \frac{1-\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} \phi_1(t, \ell_1(t_{\mathrm{p}}), \ell_2(t_{\mathrm{p}}), \ell_3(t_{\mathrm{p}})) \\
+ \quad & + \frac{\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} \sum_{k=0}^{\mathrm{p}} \Bigg(\frac{\phi_1(t_{\mathrm{p}}, \ell_1(t_{\mathrm{p}}), \ell_2(t_{\mathrm{p}}), \ell_3(t_{\mathrm{p}}))}{\Gamma(\omega+2)} \\
& \times \mathrm{h}^{\omega} \Big[(\mathrm{p}+1-i)^{\omega}(\mathrm{p}-k+2+\omega) - (\mathrm{p}-k)^{\omega}(\mathrm{p}-k+2+2\omega) \Big] \\
& - \frac{\phi_1(t_{\mathrm{p}-1}, \ell_1(t_{\mathrm{p}-1}), \ell_2(t_{\mathrm{p}-1}), \ell_3(t_{\mathrm{p}-1}))}{\Gamma(\omega+2)} \mathrm{h}^{\omega} [(\mathrm{p}+1-k)^{\omega+1} - (\mathrm{p}-k)^{\omega}(\mathrm{p}-k+1+\omega)] \Bigg),
\end{aligned} \tag{17}
$$

$$
\begin{aligned}
\ell_3(t_{\mathrm{p}+1}) &= \ell_3(t_0) + \frac{1-\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} \phi_1(t, \ell_1(t_{\mathrm{p}}), \ell_2(t_{\mathrm{p}}), \ell_3(t_{\mathrm{p}})) \\
+ \quad & + \frac{\omega}{\mathscr{A}\mathscr{B}\mathscr{C}(\omega)} \sum_{k=0}^{\mathrm{p}} \Bigg(\frac{\phi_1(t_{\mathrm{p}}, \ell_1(t_{\mathrm{p}}), \ell_2(t_{\mathrm{p}}), \ell_3(t_{\mathrm{p}}))}{\Gamma(\omega+2)} \\
& \times \mathrm{h}^{\omega} \Big[(\mathrm{p}+1-i)^{\omega}(\mathrm{p}-k+2+\omega) - (\mathrm{p}-k)^{\omega}(\mathrm{p}-k+2+2\omega) \Big] \\
& - \frac{\phi_1(t_{\mathrm{p}-1}, \ell_1(t_{\mathrm{p}-1}), \ell_2(t_{\mathrm{p}-1}), \ell_3(t_{\mathrm{p}-1}))}{\Gamma(\omega+2)} \mathrm{h}^{\omega} [(\mathrm{p}+1-k)^{\omega+1} - (\mathrm{p}-k)^{\omega}(\mathrm{p}-k+1+\omega)] \Bigg).
\end{aligned} \tag{18}
$$

5. Results and discussion

For graphical presentation of our approximate solutions of different classes of the proposed model, we use the given numerical values of parameters in Table 2.

Initial data/ Parameters	Numerical value
$\ell_{1,0}$	0.4
$\ell_{2,0}$	0.3
$\ell_{3,0}$	0.1
r	0.05
k	0.1
a	0.13
b	0.025
c	0.2
d	0.01

Table 2: Parameters with numerical values.

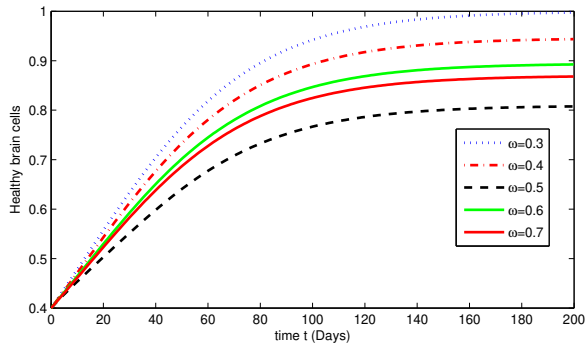

Figure 1: Plot of robust nerve cells at different fractional orders.

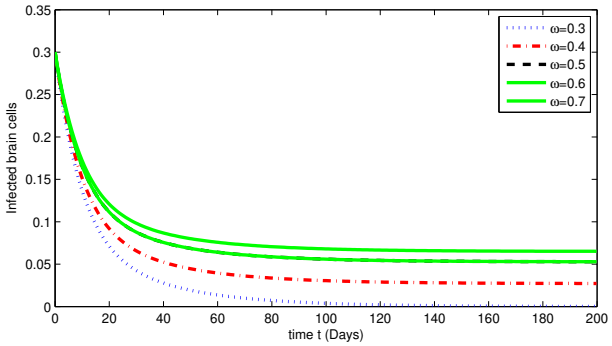

Figure 2: Plot of affected nerve cells at different fractional orders.

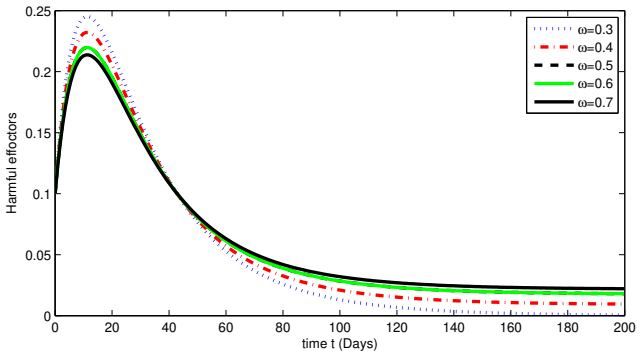

Figure 3: Plot of toxic effectors nerves cells at different fractional orders.

In Figures 1-3, we have presented the approximate solution to the three compartments in the proposed model for distinct values of fractional order ω using Matlab-16. In Figure $1-3$, we have presented plots for the approximate solutions of robust nerve cells, affected nerve cells, and toxic effectors nerve cells at different values of ω. As we increase the values of ω, then the solutions approach to integer order solution. Moreover, with proper cures (vaccination) and physiotherapy, the density of robust nerve cells is increasing with time. The growth and decay process is not the same at different fractional values. Hence, in Figures, 2, and 3 affected and toxic effectors nerve cells are decreasing with time. As we observe that different curves are shown in Figures 1-3 at different values of ω. The decrease in the dynamics is a little bit faster at lower fractional value as compared to the higher fractional order. On the other hand, an increase in dynamics can be observed for larger fractional orders as compared to smaller ones. To observe such behaviors see figures 4, 5, 6.

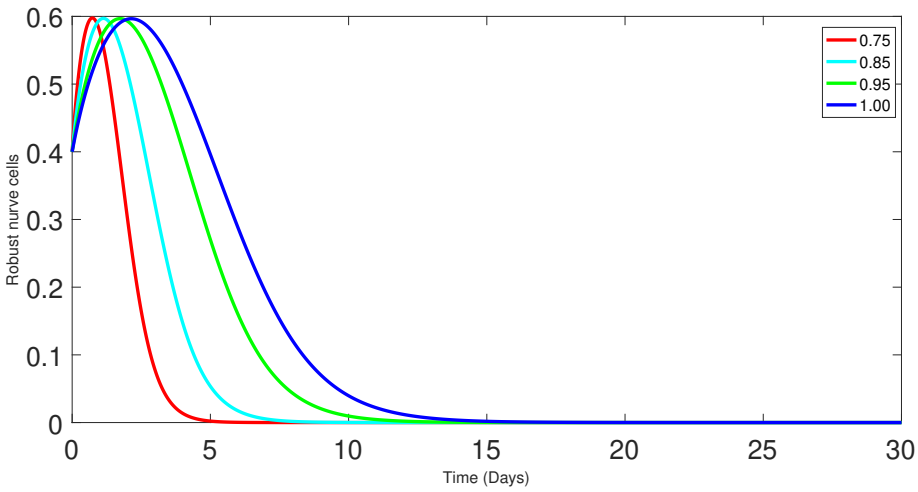

Figure 4: Plot of robust nerve cells at different fractional orders.

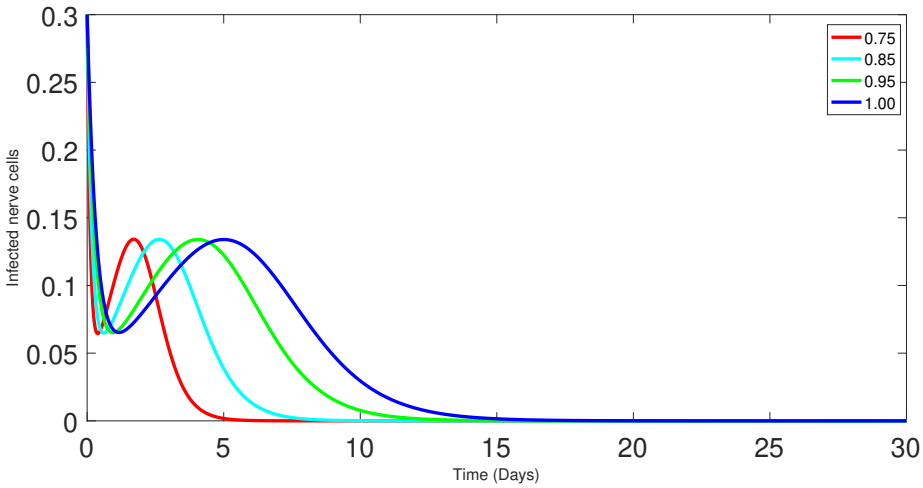

Figure 5: Plot of affected nerve cells at different fractional orders.

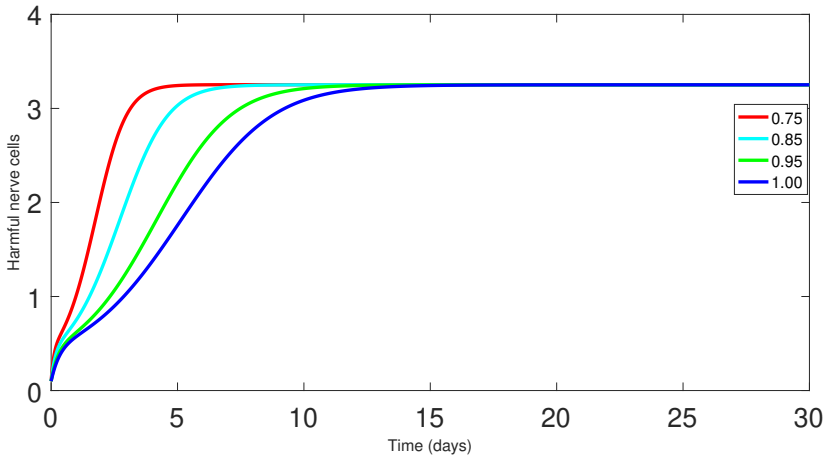

Figure 6: Plot of toxic effectors nerves cells at different fractional orders.

The chaotic dynamics of the suggested model is also examined in Figures 7, 8, 9, and 10. To demonstrate the model's memory and heredity features, we select several fractional order values. These simulations show that when the fractional order value varies, so does the disease's evolution. We observe that at lower fractional orders, the model reaches stability faster than at higher orders. Furthermore, when the fractional order increases, the graphs of each class resemble the dynamics of the integer order model. Consequently, we draw the conclusion that the suggested model is more sophisticated and comprehensive than the traditional paradigm. Furthermore, the method enables a quantitative evaluation of how fractional orders affect the solution trajectory. At lower fractional orders, stronger memory effects emerge in the system, resulting in slower disease progression and longer remission periods. In contrast, higher orders enhance immune activation and intensify the severity of relapses. The Adams–Bashforth scheme successfully reproduces these variations, demonstrating its effectiveness for fractional epidemiological modeling.

From a biological standpoint, the numerical outcomes obtained through the AB method are valuable for assessing therapeutic interventions. For instance, an increase in the treatment-related parameter reduces both the amplitude and frequency of oscillations in the solution, corresponding to fewer and less severe relapses. Quantitatively, the results indicate that even small improvements in relapse-preventive therapy can significantly lower the long-term disease burden.

In summary, the Adams–Bashforth method serves as a reliable quantitative approach for approximating the MS model. It maintains numerical stability under realistic parameter settings, effectively captures the nonlinear and memory-dependent characteristics of

the disease, and provides a practical framework for evaluating treatment strategies. Thus, it bridges the gap between theoretical modeling and clinical interpretation.

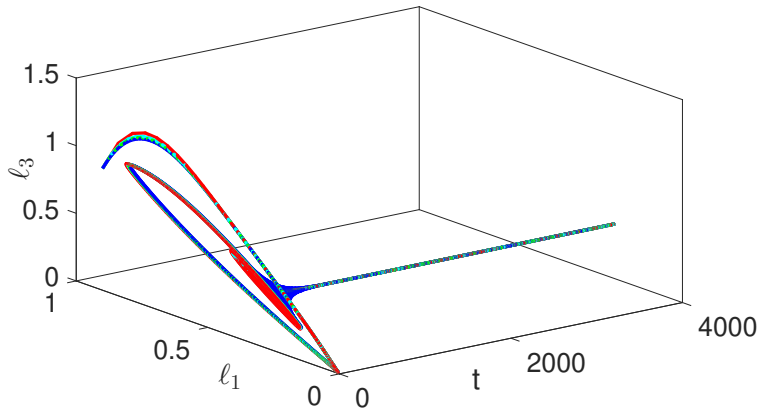

Figure 7: chaotic dynamics of the proposed model at different fractional orders.

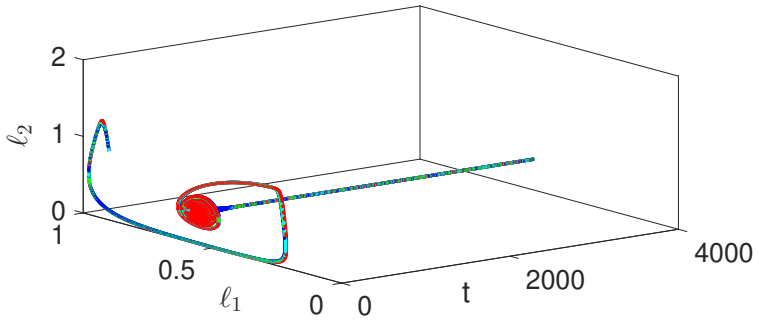

Figure 8: chaotic dynamics of the proposed model at different fractional orders.

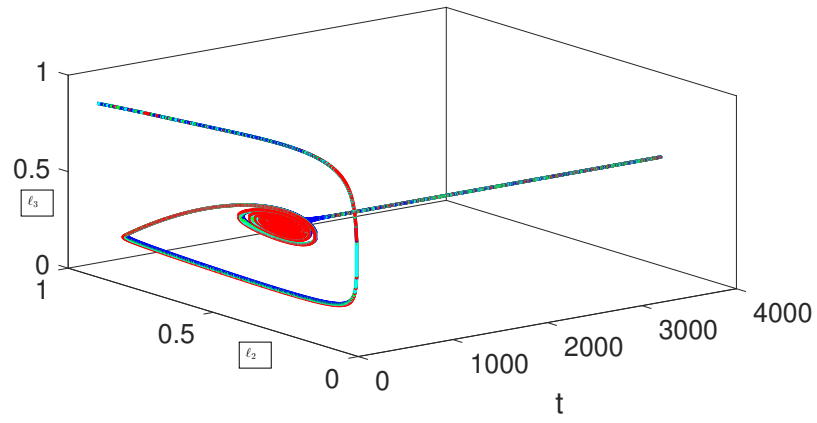

Figure 9: chaotic dynamics of the proposed model at different fractional orders.

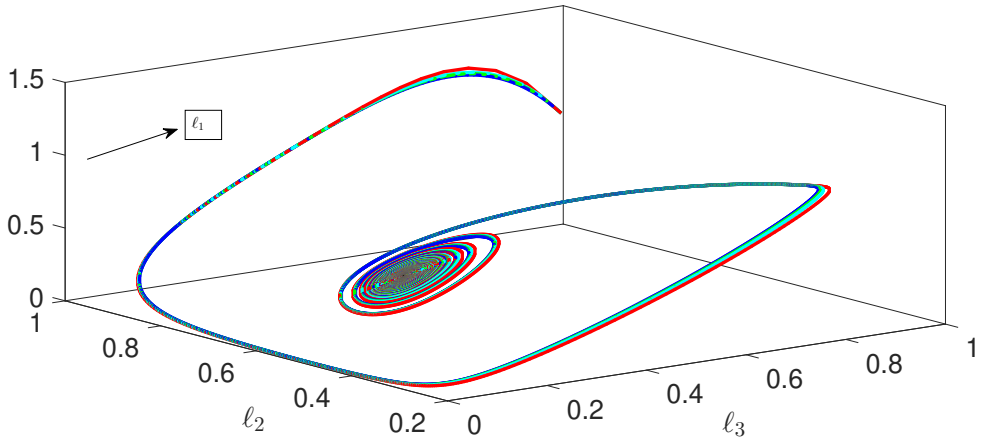

Figure 10: chaotic dynamics of the proposed model at different fractional orders.

6. Conclusion

A comprehensive analysis of the neural disease model has been performed within the framework of the $\mathscr{ABC}$ fractional derivative, which captures memory and hereditary effects more effectively than classical operators. Rigorous theoretical results have been derived concerning the existence of approximate solutions to the proposed nonlinear system, together with meaningful findings on Ulam–Hyers (U–H) stability, ensuring the robustness of the model under small perturbations. For the numerical investigation, the Adams–Bashforth method was extended and applied with biologically relevant parameter values to obtain approximate solutions. The numerical simulations, displayed graphically for various fractional orders, reveal how changes in the fractional order significantly influence the dynamics of different compartments. In particular, lower fractional orders amplify memory effects, leading to prolonged inflammatory responses and delayed recovery, whereas higher orders generate sharper transitions resembling classical dynamics. These results provide valuable insights into the progression of multiple sclerosis and illustrate the potential of fractional operators in capturing complex disease behaviors.

Acknowledgements

Authors are thankful to Prince Sultan University for support and paying the APC.

References

[1] Nina Shigesi, Marina Kvaskoff, Shona Kirtley, Qian Feng, Hai Fang, Julian C Knight, Stacey A Missmer, Nilufer Rahmioglu, Krina T Zondervan, and Christian M Becker. The association between endometriosis and autoimmune diseases: a systematic review and meta-analysis. *Human reproduction update*, 25(4):486–503, 2019.

[2] Raffaele Nardone, Viviana Versace, Luca Sebastianelli, Francesco Brigo, Stefan Golaszewski, Monica Christova, Leopold Saltuari, and Eugen Trinka. Transcranial magnetic stimulation and bladder function: a systematic review. *Clinical Neurophysiology*, 130(11):2032–2037, 2019.

[3] Isabel J Crane and John V Forrester. Th1 and th2 lymphocytes in autoimmune disease. *Critical Reviews in Immunology*, 25(2), 2005.

[4] Leszek Blach-Olszewska. Mechanisms of over-activated innate immune system regulation in autoimmune and neurodegenerative disorders. *Neuropsychiatr Disease Treatment*, 3(3), 2007.

[5] N. Khan S Hussain M I Khattak M khan, I Ullah. Adpnet: Attention-driven dual-path network for automated polyp segmentation in colonoscopy. *Image and vision Computing*, 162, 2025.

[6] Allen. Mathematical epidemiology. *Berlin*, 1945, 2008.

[7] Elettrebya M F and Ahmed E. A simple mathematical model for relapsing-remitting multiple sclerosis (rrms). *Medical Hypotheses*, 135(2020), 2020.

[8] Sivasamy P and Kumar V R. Approximate analytical solution of relapsing remitting multiple sclerosis using homotopy perturbation method. *National Volatiles & Essential Oils*, 8(4), 2021.

[9] K. Kamalanand A. Bakiya and V. Rajinikanth. Automated diagnosis of amyotrophic lateral sclerosis using electromyograms and firefly algorithm based neural networks with fractional position update. *Physical and Engineering Sciences in Medicine*, 44(4):1095–1105., 2021.

[10] Manar Alqudah, Kamal Shah, Fatemah Mofarreh, and Thabet Abdeljawad. Mathematical modeling of psychological disease by using artificial intelligence tools. *Fractals*, 2025.

[11] L Brusini. Multivariate data analysis suggests the link between brain microstructure and cognitive impairment in multiple sclerosis. *International Symposium on Biomedical Imaging*, 2021.

[12] Schiavi. Non-invasive quantification of inflammation, axonal and myelin injury in multiple sclerosis. *Brain*, 144(1):213–223., 2021.

[13] González. Intranasal delivery of interferon-β-loaded nanoparticles induces control of neuroinflammation in a preclinical model of multiple sclerosis: A promising simple, effective, non-invasive, and low-cost therapy. *Journal of Controlled Release*, 331(2021):443–459., 2021.

[14] S Bosticardo. Microstructure-weighted connectomics in multiple sclerosis. *Brain Connectivity*, 22(1):6–17., 2022.

[15] Thabet Abdeljawad, Nadeem Khan, Bahaaeldin Abdalla, Asma Al-Jaser, Manar Alqudah, and Kamal Shah. A mathematical analysis of human papilloma virus (hpv) disease with new perspectives of fractional calculus. *Alexandria Engineering Journal*, 125:575–599, 2025.

[16] K Buyukturkoglu. Machine learning to investigate superficial white matter integrity in early multiple sclerosis. *Journal of Neuroimaging*, 32(1):36–47., 2022.

[17] I Podlubny. Fractional differential equations. *Mathematics in Science and Engineering*, 32(1), 2022.

[18] H Srivastava A Kilbas and J Trujillo. Theory and application of fractional differential equations. *North Holland Mathematics Studies*, 204(1), 2006.

[19] O I Marichev A A Kilbas and S G Samko. Fractional integrals and derivatives (theory and applications). *Gordon & Breach, New York*, 32(1), 1993.

[20] D L Suthar G Agarwal K S Nisar N Pareek, A Gupta. Homotopy analysis approach to study the dynamics of fractional deterministic lotka-volterra model. *Arab Journal of Basic and Applied Sciences*, 29(1):121–128., 2022.

[21] Z U Khan F M Khan, A Ali. On existence and semi-analytical results to fractional order mathematical model of covid-19. *Arab Journal of Basic and Applied Sciences*, 29(1):40–52., 2022.

[22] F Jarad K Shah, T Abdeljawad. On a nonlinear fractional order model of dengue fever disease under caputo-fabrizio derivative. *Alexendria Engineering Journal*, 59(4):2305–2313., 2020.

[23] R A Attia. Unstable novel and accurate soliton wave solutions of the nonlinear

biological population model. *Arab Journal of Basic and Applied Sciences*, 29(1):19–25., 2022.

[24] T Abdeljawad M Al-Refai. Analysis of the fractional diffusion equations with fractional derivative of non-singular kernel. *Advances in Difference Equations*, 2017, 2017.

[25] S Arshad S Siddiqui, S Parveen. Numerical solution of atangana-baleanu-caputo time-space fractional diffusion equation. *Arab Journal of Basic and Applied Sciences*, 29(1):129–137., 2022.

[26] H Shatha. tangana-baleanu fractional framework of reproducing kernel technique in solving fractional population dynamics system. *Chaos Solitons Fractals*, 133(2020):109–624., 2020.

[27] I Koca A Atangana. Chaos in a simple nonlinear system with atangana-baleanu derivatives with fractional order. *Chaos Solitons Fractals*, 89(2016):447–454 ., 2016.

[28] V F Morales-Delgado J F Gómez-Aguilar, A Atangana. Morales-delgado, electrical circuits rc, lc, and rl described by atangana-baleanu fractional derivatives. *International Journal of Circuit Theory and Applications*, 45(11), 2017.

[29] J F Gómez-Aguilar A Atangana. Numerical approximation of riemann-liouville definition of fractional derivative: from riemann-liouville to atangana-baleanu. *Numerical Methods in Partial Differential Equations*, 34(5):1502–1523., 2018.

[30] T Abdeljawad M A Alqudah K Shah, B Abdalla. A fractal-fractional order model to study multiple sclerosis: a chronic disease. *Fractals*, 32(2), 2024.

[31] M I Syam and M Al-Refai. Fractional differential equations with atangana–baleanu fractional derivative: analysis and applications. *Choas Soliton & Fractals*, 2(2019), 2019.

[32] I Ullah K Shah T Abdeljawad B Abdalla M Khan, N Khan. A novel fractal fractional mathematical model for hiv/aids transmission stability and sensitivity with numerical analysis. *Scientific Reports*, 15(1), 2025.

[33] A Debbouche R A Khan G Nazir, K Shah. Study of hiv mathematical model under nonsingular kernel type derivative of fractional order. *Choas Soliton & Fractals*, 139(2020), 2020.

[34] A Ullah Z A Khan N Khan, A Ali. Mathematical analysis of neurological disorder under fractional order derivative. *AIMS Math*, 8(8):18846–18865., 2023.

[35] R Kumar B Ghanbari, S Kumar. A study of behaviour for immune and tumor cells in immunogenetic tumour model with non-singular fractional derivative. *Choas Soliton & Fractals*, 133(2020), 2020.

[36] T Abdeljawad. A lyapunov type inequality for fractional operators with nonsingular mittag-leffler kernel. *Journal of Inequalities and Applications*, 2017(1):1–11., 2017.

[37] A Din A Zeb S Djilali Z A Khan Q T Ain, N Anjum. On the analysis of caputo fractional order dynamics of middle east lungs coronavirus (mers-cov) model. *Alexandria Engineering Journal*, 61(7):5123–5131., 2022.

[38] M I Ullah M Alqudah T Abdeljawad Q T Ain, A Khan. On fractional impulsive system for methanol detoxification in human body. *Choas Soliton & Fractals*, 160(2022), 2022.

[39] S Karim M Nadeem P Kandege Mwanakatwe Q T Ain, T Sathiyaraj. Kandege mwanakatwe, abc fractional derivative for the alcohol drinking model using two-scale

fractal dimension. *Complexity*, 2022(2022):11 pages., 2022.