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GLOBAL BEHAVIOUR OF A TIME FRACTIONAL ORDER SPATIOTEMPORAL EPIDEMIC MODEL WITH BEDDINGTON-DEANGELIS INCIDENCE RATE AND VACCINATION STRATEGY

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Abstract. In this paper, we describe the transmission dynamics of a fractional order of an SVEIR epidemic model with reaction–diffusion and Beddington-DeAngelis incidence rate. The basic reproduction number $\mathscr{R}_0$ is obtained according to the next generation matrix. The local stability of the disease free equilibrium is discussed. Further, utilizing the Lyapunov function method, it has been demonstrated that the global stability of each equilibrium: free equilibrium and endemic equilibrium, is mainly based on the fundamental reproduction number $\mathscr{R}_0$. Finally, numerical simulations were executed to justify the theoretical findings.

Keywords: Global stability; Reaction-diffusion systems; Lyapunov function; Time-fractional.

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1. INTRODUCTION

The use of mathematical analysis and modelling is essential in the study of various infectious diseases, as it helps to gain a deeper understanding of their transmission dynamics and enables the evaluation of control strategies. In epidemiology, there are typically three main categories that describe infectious diseases: susceptible, infected, and removed (recovered) individuals,

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which allow for basic descriptions of the disease. In the literature there is a number of mathematical numbers such as SIS [2], SIR [17, 19, 37], SEIR [18, 9], SVEIR [13], SIRS [31], and many more have been proposed to control the spread of disease.

Many traditional models primarily focus on the temporal variable $'t'$ [31, 2, 17, 19, 37, 18, 9]. However, it's important to note that infection propagation is influenced by more than just time. So, in this paper, we take into account the spatial structure since it is considered as an important factor that affects the spatial spreading of disease due to the carrier hosts of infectious sources randomly moving in space. Therefore, several authors also incorporate the spatial variable $'x'$ into their analyses and study the influence of the spatial aspect and mobility of host populations on the dynamics of diseases [26, 33, 34, 35, 29, 32].

Vaccination is one of the effective control measures to prevent and weaken the transmission of infectious diseases. Currently, various modeling studies have been made to explain the effect of vaccination on the spread of diseases [32, 22, 24, 12, 30, 13].

On the other hand, incidence rate is a crucial component in simulating the dynamics of epidemic systems. Nonetheless, standard incidence or bilinear incidence functions are used in a lot of disease models. So, since applying varied incidence rates has the ability to change the system's behavior, in our work we take the incidence rate as Beddington-DeAngelis type: $\frac{\beta SI}{1+aS+bI}$. Here β is the transmission rate, a is a measure of inhibition effect, such as preventive measure taken by susceptible individuals, and b is a measure of inhibition effect such as treatment with respect to infectives. This incidence rate includes the three forms: The first one is the bilinear incidence βSI [11, 39]. The second one is the saturated incidence rate of the form $\frac{\beta SI}{1+aS}$ [1, 38]. The third one is the saturated incidence rate of the form $\frac{\beta SI}{1+bI}$ [15, 3, 31].

Classical differentiation and integration are generalized to any order using fractional differentiation. This is very pertinent to modeling the spread of epidemics because the time-fractional derivative serves as a non-local operator, introducing memory effects where a system's response becomes dependent on its recent history. This non-integer differentiation is crucial for capturing the memory and hereditary properties, offering a more realistic approach to epidemic models. The inclusion of fractional-order derivatives, with their inherent memory effects, allows for

the integration of all past information, enhancing the accuracy of predicting and modeling epidemics. As a result, many authors [7, 16, 20, 23, 36, 5] have begun to study epidemic models using fractional differential equations.

In 2018, Gao and Huang [10] conducted a study on the model described below:

$$
\left\{
\begin{array}{l}
\dfrac{dS}{dt} = \Lambda - \dfrac{\beta SI}{1+aS+bI} - (\eta+\mu)S + \omega V, \\
\dfrac{dV}{dt} = \eta S - (\omega+\mu)V, \\
\dfrac{dE}{dt} = \dfrac{\beta SI}{1+aS+bI} - (\delta+\mu)E, \\
\dfrac{dI}{dt} = \delta E - (\gamma+d+\mu)I, \\
\dfrac{dR}{dt} = \gamma I - \mu R.
\end{array}
\right. \tag{1}
$$

All the parameters in model (1) are positive. The variables S, E, I, R, and V represent the respective counts of susceptible, exposed, infectious, recovered, and vaccinated individuals at time t. Table 1 provides the biological interpretations of the remaining parameters.

The model's stability results show that if $\mathscr{R}_0 < 1$, the disease-free equilibrium is globally asymptotically stable and if $\mathscr{R}_0 > 1$, model (1) has an endemic equilibrium $(S^*, E^*, I^*, R^*, V^*)$ which is globally asymptotically stable.

In the real world and during an epidemic, a variety of variables may influence the disease outbreak and are not included in the model formulation. Examples include the population's fear of infection and weather patterns. As it is utilized to comprehend many real-world situations, this phenomenon can be modeled by substituting a fractional differential derivative for the ordinary differential derivative. For the model (1), the state at any time t does not depend on the previous history. Moreover, since the spatial structure influence the spreading of disease and describe the reality well as the fractional derivative, so motivated by the work of [10] in this paper we consider a fractional $SVEIR$ epidemic model with diffusion and Beddington-DeAngelis type incidence rate. The spreading dynamic of the epidemic is then governed by the following fractional system

$$
\begin{cases}
{}^{C}D_t^{\alpha} S(t,x) = d_S \Delta S(t,x) + \Lambda - \dfrac{\beta SI}{1+aS+bI} - (\eta+\mu)S(t,x) + \omega V(t,x), \\
{}^{C}D_t^{\alpha} V(t,x) = d_V \Delta V(t,x) + \eta S(t,x) - (\omega+\mu)V(t,x), \\
{}^{C}D_t^{\alpha} E(t,x) = d_E \Delta E(t,x) + \dfrac{\beta SI}{1+aS+bI} - (\delta+\mu)E(t,x), \\
{}^{C}D_t^{\alpha} I(t,x) = d_I \Delta I(t,x) + \delta E(t,x) - (\gamma+d+\mu)I(t,x), \\
{}^{C}D_t^{\alpha} R(t,x) = d_R \Delta R(t,x) + \gamma I(t,x) - \mu R(t,x),
\end{cases} \tag{2}
$$

${}^{C}D_t^{\alpha}$ is the Caputo fractional-order derivative with $0 < \alpha \leq 1$ and Δ denotes the Laplacian operator. We consider system (2) with initial conditions:

$$
S(0,x) = \psi_1(x) \geq 0, \quad V(0,x) = \psi_2(x) \geq 0, \quad E(0,x) = \psi_3(x) \geq 0, \tag{3}
$$

$$
I(0,x) = \psi_4(x) \geq 0, \quad R(0,x) = \psi_5(x) \geq 0, \quad \text{for} \quad x \in \Omega
$$

We acknowledge that the self-contained nature of the model (2) involves dynamics within its boundaries, yet there is an absence of emigration. Consequently, the homogeneous Neumann boundary conditions with no-flux are applied.

$$
\frac{\partial S}{\partial \nu} = \frac{\partial V}{\partial \nu} = \frac{\partial E}{\partial \nu} = \frac{\partial I}{\partial \nu} = \frac{\partial R}{\partial \nu} = 0, \quad \text{for} \quad x \in \partial\Omega, \tag{4}
$$

with $\frac{\partial}{\partial \nu}$ denotes the outward normal derivative on $\partial\Omega$, where Ω is a bounded domain in $\mathbb{R}^n$.

Here, the densities of susceptible, vaccinated, latent, infected and recovered individuals at time t and spatial location x are denoted by $S(t,x)$, $V(t,x)$, $E(t,x)$, $I(t,x)$ and $R(t,x)$, respectively.

The rest of this paper is structured as follows: In section 2, we give some preliminaries about fractional calculus. In section 3, we determine the basic reproduction number and the existence of the equilibria. In section 4, we discuss the local and global stability of model system at the disease-free and endemic equilibrium points. In section 5, we illustrate our theoretical results by numerical simulation.

TABLE 1. The parameters description used in model

Parameter	The physical interpretation
Λ	Recruitment rate
β	Transmission rate
η	The vaccination rate coefficient
μ	Natural death rate
δ	The rate at which exposed individuals become infectious
ω	The rate of losing immunity
γ	The recovery rate
d	Death rate due to the disease
a	The proportion constant related to susceptible individuals
b	The proportion constant related to infectious individuals
d_i $i = S,V,E,I,R$	Diffusion rate of S,V,E,I and R respectively

2. PRELIMINARIES

In this section, we present the definition of Caputo fractional-order derivative, and some useful lemmas are recalled for next analysis.

Definition 2.1. *(*[25]*). The fractional integral of order α for a function $f(t)$ is defined as*

$$I^{\alpha} f(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} f(s) ds,$$

where $t \geq 0$, $\alpha > 0$, $\Gamma(.)$ is the gamma function, $\Gamma(t) = \int_0^{\infty} x^{t-1} e^{-x} dx$.

Definition 2.2. *(*[25]*). The Caputo fractional derivative of order α for the function $f(x) \in \mathscr{C}^n([0,\infty),\mathbb{R})$ is defined by*

$${}^{C}D_t^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t \frac{f^{(n)}(s)}{(t-s)^{\alpha-n+1}} ds,$$

where $t \geq 0$, and n is a positive integer such that $n-1 \leq \alpha < n$.

Furthermore, when $0 < \alpha < 1$,

$${}^{C}D_t^{\alpha} f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{f'(s)}{(t-s)^{\alpha}} ds.$$

Definition 2.3. *([25]). The Mittag-Leffler type function with one parameter is defined as follows*

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

Lemma 2.4. *([28]). Let Ψ be a positive function defined by $\Psi(y) = y - \ln(y) - 1$, $y > 0$ and $y(t) \in \mathbb{R}+^*$ is a continuous differentiable function for all $\alpha \in [0,1]$ and $t \geqslant 0$,*

$${}^C D_t^\alpha \left[y^* \Psi \left(\frac{y(t)}{y^*} \right) \right] \leqslant \left(1 - \frac{y^*}{y(t)} \right) {}^C D_t^\alpha y(t), \quad y^* \in \mathbb{R}_+^*.$$

3. Basic Reproduction Number and Existence of Equilibrium

In this section, we will examine the presence of both the disease-free equilibrium and the endemic equilibrium within the framework of model (2). Given that the equation for R operates independently of the other equations, we can establish the following subsystem.

$$\begin{cases} {}^C D_t^\alpha S(t,x) = d_S \Delta S(t,x) + \Lambda - \dfrac{\beta SI}{1+aS+bI} - (\eta+\mu)S(t,x) + \omega V(t,x), \\ {}^C D_t^\alpha V(t,x) = d_V \Delta V(t,x) + \eta S(t,x) - (\omega+\mu)V(t,x), \\ {}^C D_t^\alpha E(t,x) = d_E \Delta E(t,x) + \dfrac{\beta SI}{1+aS+bI} - (\delta+\mu)E(t,x), \\ {}^C D_t^\alpha I(t,x) = d_I \Delta I(t,x) + \delta E(t,x) - (\gamma+d+\mu)I(t,x), \end{cases} \tag{5}$$

It is easy to check that model (5) always has the disease-free equilibrium $P^0 = (S^0, V^0, 0, 0)$, where

$$S^0 = \frac{(\omega+\mu)\Lambda}{\mu(\eta+\omega+\mu)} \quad \text{and} \quad V^0 = \frac{\eta\Lambda}{\mu(\eta+\omega+\mu)}.$$

To investigate the presence and distinctiveness of the endemic equilibrium, denoted as $P^* = (S^*, V^*, E^*, I^*)$, we initiate our analysis by examining the fundamental reproductive number, $\mathscr{R}_0$, of model (5). indeed, This quantity is recognized as the expected average number of new infection cases created by an average infectious individual (over their period of infectivity) within a population that is entirely composed of susceptible individuals. To calculate basic reproduction number we will use the method presented in [27] given by Van Den Driessche and Watmough.

Let $X = (S,V,E,I)^T$. So model (5) can be written as ${}^C D_t^\alpha X = \mathscr{F}(X) - \mathscr{V}(X)$, where

$$\mathscr{F}(X) = \begin{pmatrix} 0 \\ 0 \\ \dfrac{\beta SI}{1+aS+bI} \\ 0 \end{pmatrix}, \quad \mathscr{V}(X) = \begin{pmatrix} \Lambda - (\eta+\mu)S + \omega V \\ \eta S - (\omega+\mu)V \\ -(\delta+\mu)E \\ \delta E - (\gamma+d+\mu)I \end{pmatrix}$$

The transition matrix V and the new infection matrix F, which are the Jacobian of $\mathscr{F}$ and $\mathscr{V}$ evaluated at P^0 respectively, are provided by

$$F = \begin{pmatrix} 0 & \frac{\beta S^0}{1+aS^0} \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} -(\delta+\mu) & 0 \\ \delta & -(\gamma+d+\mu) \end{pmatrix},$$

so spectral radius of the next generation matrix $-FV^{-1}$ can be found as,

$$\rho(-FV^{-1}) = \frac{\delta\beta\Lambda(\omega+\mu)}{(\delta+\mu)(\gamma+d+\mu)(\mu(\eta+\omega+\mu)+a\Lambda(\omega+\mu))}.$$

Hence, the fundamental reproductive number $\mathscr{R}_0$, for model (5) can be determined as follows:

$$\mathscr{R}_0 = \frac{\delta\beta\Lambda(\omega+\mu)}{(\delta+\mu)(\gamma+d+\mu)(\mu(\eta+\omega+\mu)+a\Lambda(\omega+\mu))}. \tag{6}$$

If $\mathscr{R}_0 > 1$, the system (5) has a unique endemic equilibrium point $P^* = (S^*,V^*,E^*,I^*)$ with,

$$S^* = \frac{(\omega+\mu)(\delta\Lambda - (\delta+\mu)(\gamma+d+\mu)I^*)}{\delta\mu(\eta+\omega+\mu)}.$$

$$V^* = \frac{\eta S^*}{\omega+\mu}.$$

$$E^* = \frac{(\gamma+d+\mu)I^*}{\delta}.$$

$$I^* = \frac{\delta\Lambda[\mu(\eta+\omega+\mu)+a\Lambda(\omega+\mu)](\mathscr{R}_0-1)}{\mathscr{R}_0\mu(\delta+\mu)(\gamma+d+\mu)(\eta+\omega+\mu)(\mathscr{R}_0-1)+b\Lambda\delta\mu(\eta+\omega+\mu)}.$$

3.1. Sensitivity analysis of $\mathscr{R}_0$. When a virus spreads rapidly it becomes important to find ways to control it, i.e., to increase or decrease certain parameters that affect the infection's ability to propagate. Sensitivity analysis is one method to evaluate each parameter's impact for the spread of disease.

To reduce the disease, we specifically employ the definition of the sensitivity index, which provides the significance of the variable related with each model parameter [6].

Definition 3.1. *The $\mathscr{R}_0$ sensitivity index with respect to x is defined by*

$$\Upsilon_x^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial x}\frac{x}{\mathscr{R}_0}. \tag{7}$$

One may determine whether a parameter increases (positive sign) or decreases (negative sign) the value of $\mathscr{R}_0$ based on the sign of each index.

To compute the sensitivity index for $\mathscr{R}_0$ in relation to the parameters of the model, we will use the expression of $\mathscr{R}_0$ provided in (6).

$$\Upsilon_\beta^{\mathscr{R}_0} = 1.$$

$$\Upsilon_\Lambda^{\mathscr{R}_0} = \frac{\mu(\eta+\omega+\mu)}{\mu(\eta+\omega+\mu)+a\Lambda(\omega+\mu)}.$$

$$\Upsilon_\delta^{\mathscr{R}_0} = \frac{\mu}{\delta+\mu}.$$

$$\Upsilon_d^{\mathscr{R}_0} = \frac{-d}{\gamma+d+\mu}.$$

$$\Upsilon_\gamma^{\mathscr{R}_0} = \frac{-\gamma}{\gamma+d+\mu}.$$

$$\Upsilon_\mu^{\mathscr{R}_0} = \mu\left[\frac{\mu\eta}{(\omega+\mu)(\mu(\eta+\omega+\mu)+a\Lambda(\omega+\mu))} - \frac{\gamma+d+\delta+2\mu}{(\delta+\mu)(\gamma+d+\mu)} - \frac{\eta+\omega+\mu}{\mu(\eta+\omega+\mu)+a\Lambda(\omega+\mu)}\right].$$

$$\Upsilon_\omega^{\mathscr{R}_0} = \frac{\eta\mu\omega}{(\omega\mu)(\mu(\eta+\omega+\mu)+a\Lambda(\omega+\mu))}.$$

$$\Upsilon_a^{\mathscr{R}_0} = \frac{-\Lambda a(\omega+\mu)}{\mu(\eta+\omega+\mu)+a\Lambda(\omega+\mu)}.$$

$$\Upsilon_\eta^{\mathscr{R}_0} = \frac{-\mu\eta}{\mu(\eta+\omega+\mu)+a\Lambda(\omega+\mu)}.$$

We observe that $\mathscr{R}_0$ is increasing with β, Λ, δ and ω while it is decreasing with d, γ, a and η but we cannot say anything about other parameter μ.

4. Stability Analysis of the Equilibria

4.1. Local stability of the disease free equilibrium. In this section, we will discuss the stability of the disease-free equilibrium P^0 of the model (2).

Theorem 4.1. *If $\mathscr{R}_0 \leq 1$ the disease free equilibrium point P^0 is locally asymptotically stable.*

Proof. In the presence of diffusion, the stability of P^0 reduces to applying [8, Theorem 1] to the linearizing operator $\mathscr{L} = D\Delta + A$. Note that A is the Jacobian matrix evaluated at the equilibrium point.

The equilibrium point $P^0 = (\frac{(\omega+\mu)\Lambda}{\mu(\eta+\omega+\mu)}, \frac{\eta\Lambda}{\mu(\eta+\omega+\mu)}, 0, 0)$ satisfies

$$\begin{cases} 0 = d_S\Delta S + \Lambda - \frac{\beta S^* I^*}{1+aS^*+bI^*} - (\eta+\mu)S^* + \omega V^*, \\ 0 = d_V\Delta V + \eta S^* - (\omega+\mu)V^*, \\ 0 = d_E\Delta E + \frac{\beta S^* I^*}{1+aS^*+bI^*} - (\delta+\mu)E^*, \\ 0 = d_I\Delta I + \delta E^* - (\gamma+d+\mu)I^*, \end{cases}$$

with Neumann boundaries

$$\frac{\partial S}{\partial \nu} = \frac{\partial V}{\partial \nu} = \frac{\partial E}{\partial \nu} = \frac{\partial I}{\partial \nu} = 0 \quad \text{for} \quad x \in \partial\Omega.$$

The linearizing operator may be given as follows:

$$\mathscr{L}(P^0) = \begin{pmatrix} d_S\Delta - (\eta+\mu) & \omega & 0 & \frac{-\beta S^0}{1+aS^0} \\ \eta & d_V\Delta - (\omega+\mu) & 0 & 0 \\ 0 & 0 & d_E\Delta - (\delta+\mu) & \frac{\beta S^0}{1+aS^0} \\ 0 & 0 & \delta & d_I\Delta - (\gamma+d+\mu) \end{pmatrix}.$$

Let $(\lambda_i)_i$ denotes the indefinite sequence of positive eigenvalues for the Laplacian operator Δ over Ω, with Neumann boundary conditions defined by $0 = \lambda_0 < \lambda_1 \leq \lambda_2 \leq \lambda_3 \leq \cdots \leq \nearrow^{+\infty}$,

see [4]. The stability of P^0 depends on the eigenvalues of the matrices

$$J_i(P^0) = \begin{pmatrix} -d_S\lambda_i - (\eta+\mu) & \omega & 0 & \frac{-\beta S^0}{1+aS^0} \\ \eta & -d_V\lambda_i - (\omega+\mu) & 0 & 0 \\ 0 & 0 & -d_E\lambda_i - (\delta+\mu) & \frac{\beta S^0}{1+aS^0} \\ 0 & 0 & \delta & -d_I\lambda_i - (\gamma+d+\mu) \end{pmatrix}.$$

The associated characteristic equation can be expressed as follows:

$$det(J_i(P^0) - XI) = [X^2 + p_1X + p_2][X^2 + p_3X + p_4],$$

with

$$\begin{aligned} p_1 &= \delta + \gamma + d + 2\mu + \lambda_i(d_E + d_I) \\ p_2 &= (\delta+\mu)(\gamma+\mu)(1-\mathscr{R}_0) + \lambda_i d_I(\delta+\mu) + \lambda_i d_E(\gamma+d+\mu) + \lambda_i^2 d_E d_I \\ p_3 &= \eta + \omega + 2\mu + \lambda_i d_S + \lambda_i d_V \\ p_4 &= \mu(\eta+\omega+\mu) + \lambda_i d_V(\eta+\mu) + \lambda_i d_S(\omega+\mu) + \lambda_i^2 d_S d_V. \end{aligned}$$

All of the coefficients p_i, $i = 1,2,3,4$ of the characteristic equation are positive since $\mathscr{R}_0 \leq 1$. Then, the application of the Routh-Hurwitz Theorem confirms that all the roots X have negative real parts, signifying that $|\arg(X)| > \frac{\pi}{2} > \frac{\alpha\pi}{2}$. Then, we conclude the local asymptotic stability of P^0. □

4.2. Global stability of the disease free equilibrium. In this section, we investigate the global stability of the disease-free equilibrium P^0 for system (5) by constructing proper Lyapunov function.

Theorem 4.2. *If $\mathscr{R}_0 \leq 1$, then the disease free equilibrium P^0 is globally asymptotically stable.*

Proof. Let $\mathscr{W}_1$ be the positive function defined by

$$\mathscr{W}_1(t,x) = \int_\Omega \left[\frac{\delta}{(\delta+\mu)(\gamma+d+\mu)} E(t,x) + \frac{1}{\gamma+d+\mu} I(t,x) \right] dx.$$

The fractional derivative of order α in the sense of Caputo of $\mathscr{W}_1$ is given by

$$\begin{aligned}
{}^{C}D_t^{\alpha}\mathscr{W}_1(t,x) &= \int_{\Omega}\left[\frac{\delta}{(\delta+\mu)(\gamma+d+\mu)}\,{}^{C}D_t^{\alpha}E(t,x)+\frac{1}{\gamma+d+\mu}\,{}^{C}D_t^{\alpha}I(t,x)\right]dx\\
&= \int_{\Omega}\frac{\delta}{(\delta+\mu)(\gamma+d+\mu)}\left(\frac{\beta SI}{1+aS+bI}-(\delta+\mu)E(t,x)\right)dx\\
&\quad+\int_{\Omega}\frac{1}{\gamma+d+\mu}\left(\delta E(t,x)-(\gamma+d+\mu)I(t,x)\right)dx\\
&\quad+\int_{\Omega}\left(\frac{\delta}{(\delta+\mu)(\gamma+d+\mu)}d_E\Delta E(t,x)+\frac{1}{\gamma+d+\mu}d_I\Delta I(t,x)\right)dx
\end{aligned}$$

According to Green's formula and the boundary conditions, we have

$$\int_{\Omega}\left(\frac{\delta}{(\delta+\mu)(\gamma+d+\mu)}d_E\Delta E(t,x)+\frac{1}{\gamma+d+\mu}d_I\Delta I(t,x)\right)dx=0.$$

Then,

$$\begin{aligned}
{}^{C}D_t^{\alpha}\mathscr{W}_1(t,x) &= \int_{\Omega}\left(\frac{\delta}{(\delta+\mu)(\gamma+d+\mu)}\frac{\beta SI}{1+aS+bI}-I\right)dx\\
&\leq \int_{\Omega}\left(\frac{\delta}{(\delta+\mu)(\gamma+d+\mu)}\frac{\beta S^0}{1+a_1S^0}-1\right)I\,dx\\
&\leq \int_{\Omega}(\mathscr{R}_0-1)\,I\,dx
\end{aligned}$$

If $\mathscr{R}_0\leq 1$, then ${}^{C}D_t^{\alpha}\mathscr{W}_1\leq 0$ for all $S,V,E,I,R>0$: Let $\mathscr{M}_0=\left\{(S,V,E,I,R)\,:{}^{C}D_t^{\alpha}\mathscr{W}_1=0\right\}=\left\{P^0\right\}$. Then by LaSalle's invariance principle [14], P^0 is globally asymptotically stable once $\mathscr{R}_0\leq 1$. □

4.3. Global stability of the endemic equilibrium.

Theorem 4.3. *If $\mathscr{R}_0>1$, the endemic equilibrium point P^* is globally asymptotically stable.*

Proof. Consider the positive function defined by

$$\mathscr{W}_2(t,x)=\int_{\Omega}\left[S^*\Psi\left(\frac{S(t,x)}{S^*}\right)+V^*\Psi\left(\frac{V(t,x)}{V^*}\right)+E^*\Psi\left(\frac{E(t,x)}{E^*}\right)+\frac{\delta+\mu}{\delta}I^*\Psi\left(\frac{I(t,x)}{I^*}\right)\right]dx.$$

The fractional derivative of order α in the sense of Caputo of $\mathscr{W}_2$ is given by

$$\begin{aligned}
{}^{C}D_t^{\alpha}\mathscr{W}_2(t,x)\leq \int_{\Omega}\Big[&\left(1-\frac{S^*}{S(t,x)}\right){}^{C}D_t^{\alpha}S(t,x)+\left(1-\frac{V^*}{V(t,x)}\right){}^{C}D_t^{\alpha}V(t,x)+\left(1-\frac{E^*}{E(t,x)}\right){}^{C}D_t^{\alpha}E(t,x)\\
&+\frac{\delta+\mu}{\delta}\left(1-\frac{I^*}{I(t,x)}\right){}^{C}D_t^{\alpha}I(t,x)\Big]dx
\end{aligned}$$

$$
\begin{aligned}
&\leq \int_\Omega \left(d_S \Delta S(t,x) + d_V \Delta V(t,x) + d_E \Delta E(t,x) + \frac{\delta+\mu}{\delta} d_I \Delta I(t,x) \right) dx \\
&- \int_\Omega \left(\frac{S^*}{S(t,x)} d_S \Delta S(t,x) + \frac{V^*}{V(t,x)} d_V \Delta V(t,x) + \frac{E^*}{E(t,x)} d_E \Delta E(t,x) + \frac{\delta+\mu}{\delta} \frac{I^*}{I(t,x)} d_I \Delta I(t,x) \right) dx \\
&+ \int_\Omega \left(1 - \frac{S^*}{S} \right) \left(\Lambda - \frac{\beta SI}{1+aS+bI} - \eta S - \mu S + \omega V \right) dx \\
&+ \int_\Omega \left(1 - \frac{V^*}{V} \right) (\eta S - \omega V - \mu V)\, dx \\
&+ \int_\Omega \left(1 - \frac{E^*}{E} \right) \left(\frac{\beta SI}{1+aS+bI} - (\delta+\mu)E \right) dx \\
&+ \int_\Omega \frac{\delta+\mu}{\delta} \left(1 - \frac{I^*}{I} \right) (\delta E - (\gamma+d+\mu)I)\, dx
\end{aligned}
$$

According to Green's formula and the boundary conditions, we have

$$
\int_\Omega \left(d_S \Delta S(t,x) + d_V \Delta V(t,x) + d_E \Delta E(t,x) + \frac{\mu+\delta}{\delta} d_I \Delta I(t,x) \right) dx = 0,
$$

and

$$
\begin{aligned}
-\int_\Omega d_S S^* \frac{\Delta S(t,x)}{S(t,x)} dx &= -d_S S^* \int_\Omega \frac{\|\nabla S(t,x)\|^2}{S(t,x)^2} dx. \\
-\int_\Omega d_V V^* \frac{\Delta V(t,x)}{V(t,x)} dx &= -d_V V^* \int_\Omega \frac{\|\nabla V(t,x)\|^2}{V(t,x)^2} dx. \\
-\int_\Omega d_E E^* \frac{\Delta E(t,x)}{E(t,x)} dx &= -d_E E^* \int_\Omega \frac{\|\nabla E(t,x)\|^2}{E(t,x)^2} dx. \\
-\int_\Omega d_I I^* \frac{\Delta I(t,x)}{I(t,x)} dx &= -d_I I^* \int_\Omega \frac{\|\nabla I(t,x)\|^2}{I(t,x)^2} dx.
\end{aligned}
$$

At the endemic equilibrium we have

$$
\begin{aligned}
\Lambda &= \frac{\beta_1 S^* I^*}{1+aS^*+bI^*} + \eta S^* + \mu S^* - \omega V^*, \\
\eta S^* &= (\omega+\mu) V^*, \\
(\delta+\mu) E^* &= \frac{\beta S^* I^*}{1+aS^*+bI^*}, \\
E^* &= \frac{\gamma+d+\mu}{\delta} I^*.
\end{aligned}
$$

Then

$$
\begin{aligned}
&{}^C D_t^\alpha \mathscr{W}_2(t,x) \\
&\leq -\int_\Omega \left[d_S S^* \frac{\|\nabla S(t,x)\|^2}{S(t,x)^2} + d_V V^* \frac{\|\nabla V(t,x)\|^2}{V(t,x)^2} + d_E E^* \frac{\|\nabla E(t,x)\|^2}{E(t,x)^2} + \frac{\delta+\mu}{\delta} d_I I^* \frac{\|\nabla I(t,x)\|^2}{I(t,x)^2} \right] dx
\end{aligned}
$$

$$
\begin{aligned}
&+\int_\Omega \Big(\left(1-\frac{S^*}{S}\right)\Big[\frac{\beta S^* I^*}{1+aS^*+bI^*}+\eta S^*+\mu S^*-\omega V^*-\frac{\beta SI}{1+aS+bI}-\eta S-\mu S+\omega V\Big] \\
&\quad+\left(1-\frac{V^*}{V}\right)(\eta S-\omega V-\mu V)+\left(1-\frac{E^*}{E}\right)\left(\frac{\beta SI}{1+aS+bI}-(\delta+\mu)E\right) \\
&\quad+\left(1-\frac{I^*}{I}\right)\left((\delta+\mu)E-\frac{(\delta+\mu)(\gamma+d+\mu)}{\delta}I\right)\Big)dx \\
\leq &-\int_\Omega \left[d_S S^*\frac{\|\nabla S(t,x)\|^2}{S(t,x)^2}+d_V V^*\frac{\|\nabla V(t,x)\|^2}{V(t,x)^2}+d_E E^*\frac{\|\nabla E(t,x)\|^2}{E(t,x)^2}+\frac{\delta+\mu}{\delta}d_I I^*\frac{\|\nabla I(t,x)\|^2}{I(t,x)^2}\right]dx \\
&+\int_\Omega \Big[\frac{-\mu}{S}(S-S^*)^2+\frac{\beta S^* I^*}{1+aS^*+bI^*}+\mu V^*-\frac{S^*}{S}\frac{\beta S^* I^*}{1+aS^*+bI^*}-\mu V^*\frac{S^*}{S}+\omega V^*+\mu V^* \\
&\quad+\frac{\beta S^* I}{1+aS+bI}-\omega V\frac{S^*}{S}-\mu V-\omega V^*\frac{S}{S^*}\frac{V^*}{V}-\mu V^*\frac{S}{S^*}\frac{V^*}{V}+\omega V^*+\mu V^* \\
&\quad-\frac{E^*}{E}\frac{\beta SI}{1+aS+bI}+(\delta+\mu)E^*-(\delta+\mu)E^*\frac{I}{I^*}-(\delta+\mu)E\frac{I^*}{I}+(\delta+\mu)E^*\Big]dx \\
\leq &-\int_\Omega \left[d_S S^*\frac{\|\nabla S(t,x)\|^2}{S(t,x)^2}+d_V V^*\frac{\|\nabla V(t,x)\|^2}{V(t,x)^2}+d_E E^*\frac{\|\nabla E(t,x)\|^2}{E(t,x)^2}+\frac{\delta+\mu}{\delta}d_I I^*\frac{\|\nabla I(t,x)\|^2}{I(t,x)^2}\right]dx \\
&+\int_\Omega \Big[\frac{-\mu}{S}(S-S^*)^2+\mu V^*\left(3-\frac{S^*}{S}-\frac{V}{V^*}-\frac{SV^*}{S^*V}\right)+\omega V^*\left(2-\frac{SV^*}{S^*V}-\frac{S^*V}{SV^*}\right) \\
&\quad+\frac{\beta S^* I^*}{1+aS^*+bI^*}\left(3-\frac{S^*}{S}-\frac{EI^*}{E^*I}-\frac{I}{I^*}\right)+\frac{\beta S^* I}{1+aS+bI}\left(1-\frac{SE^*}{S^*E}\right)\Big]dx
\end{aligned}
$$

Since the arithmetic mean is always less than the geometric mean then:

$$3-\frac{S^*}{S}-\frac{V}{V^*}-\frac{SV^*}{S^*V}\leq 0,$$

$$2-\frac{SV^*}{S^*V}-\frac{S^*V}{SV^*}\leq 0,$$

$$3-\frac{S^*}{S}-\frac{EI^*}{E^*I}-\frac{I}{I^*}\leq 0,$$

and

$$1-\frac{SE^*}{S^*E}\leq 0.$$

Therefore,

$${}^C D_t^\alpha \mathscr{W}_2(t,x)\leq 0.$$

Further, we can conclude that ${}^C D_t^\alpha \mathscr{W}_2(t,x)=0$ if $S=S^*$, $V=V^*$, $E=E^*$ and $I=I^*$, thus by the principle of LaSalle invariance [14], if $\mathscr{R}_0>1$ the endemic equilibrium point P^* is globally asymptotically stable.

□

5. Numerical Simulation

In this section, we provide graphical representations that validate our theoretical discoveries. To numerically integrate the system (2)-(3), we employ forward finite difference approximations to discretize the time-fractional derivative and centered finite difference schemes to approach the Laplacian operator in one-dimensional space. Furthermore, we consider the domain $\Omega = [0, 15]$. This approach yields a high level of accuracy with a time order of $2 - \alpha$ and a spatial order of 2, as detailed in [21].

5.1. Numerical Simulation for $\mathscr{R}_0 \leq 1$. In this simulation, we consider the parameter values: $\Lambda = 25$, $\beta = 0.0354$, $\mu = 0.2$, $\delta = 0.3$, $\omega = 0.02$, $\eta = 0.4$, $\gamma = 0.056$, $a = 0.8$, $b = 0.4$, $d = 0.1 \times 10^{-5}$ and $d_S = d_V = d_E = d_I = d_R = 0.0000005$. Based on these parameter values, we have $\mathscr{R}_0 = 0.1 \leq 1$ and $P^0 = (44.35, 80.64, 0, 0, 0)$. For the initial conditions we take $(S(0,x), V(0,x), E(0,x), I(0,x), R(0,x)) = (100, 10, 0, 6, 0)$.

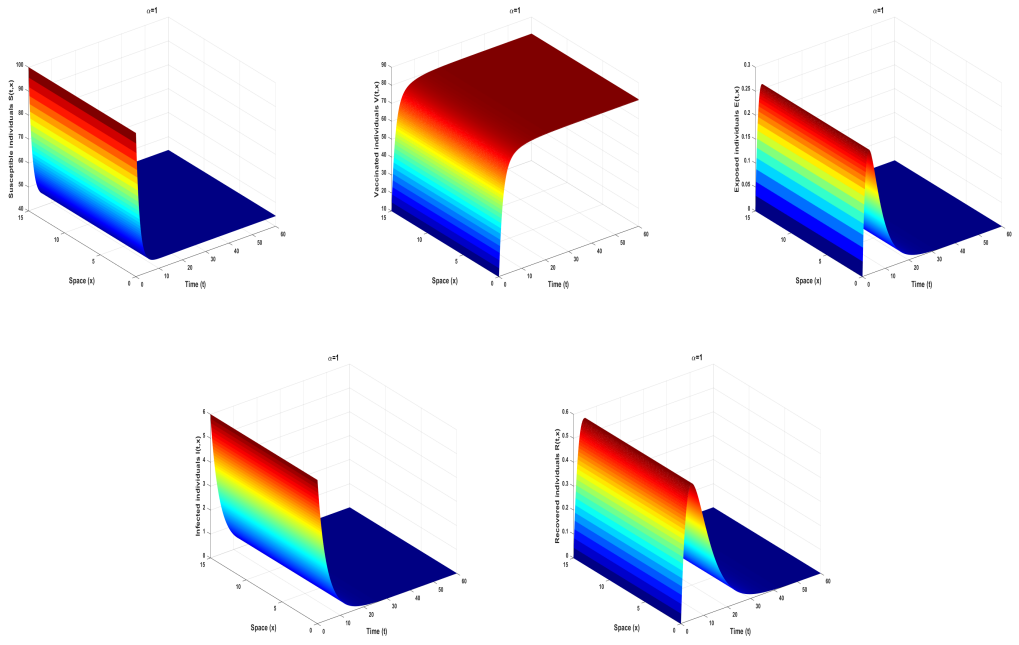

FIGURE 1. The dynamics of the system (2) showing the stability of the free equilibrium P^0 for $\alpha = 1$.

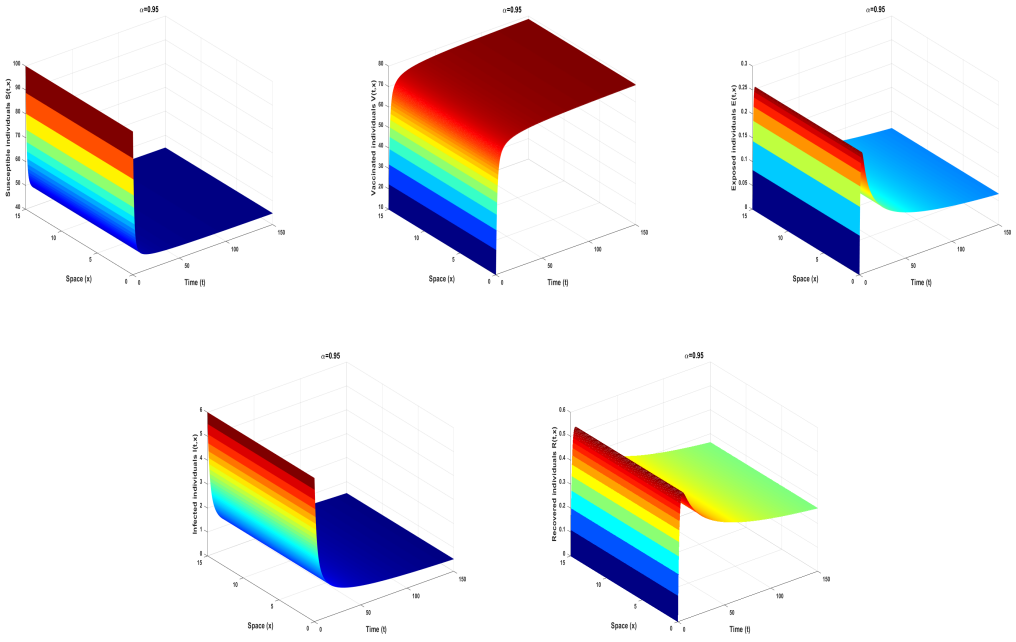

FIGURE 2. The dynamics of the system (2) showing the stability of the free equilibrium P^0 for $\alpha = 0.95$.

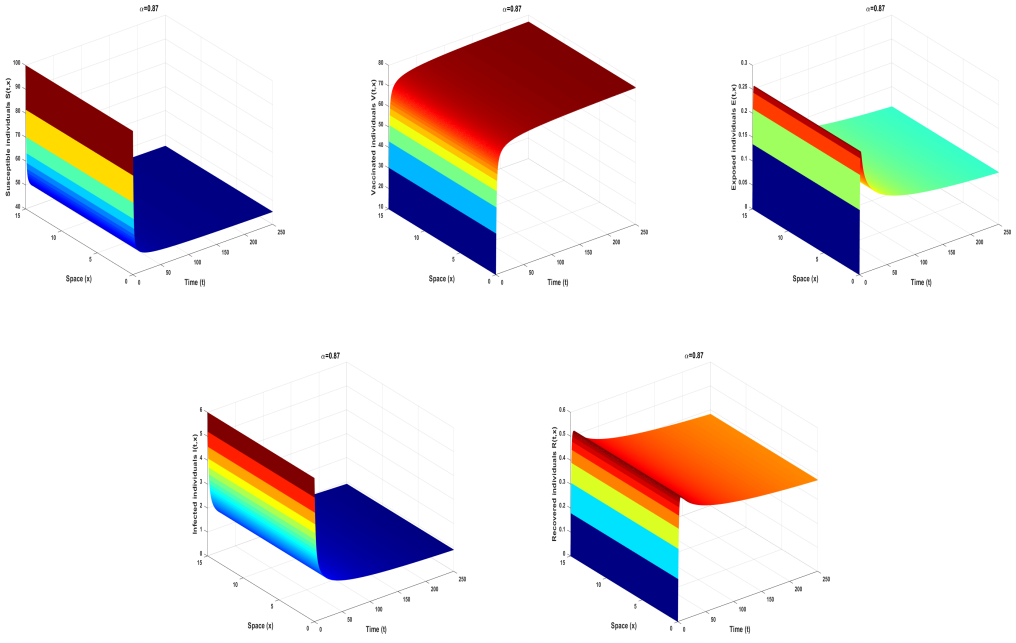

FIGURE 3. The dynamics of the system (2) showing the stability of the free equilibrium P^0 for $\alpha = 0.87$.

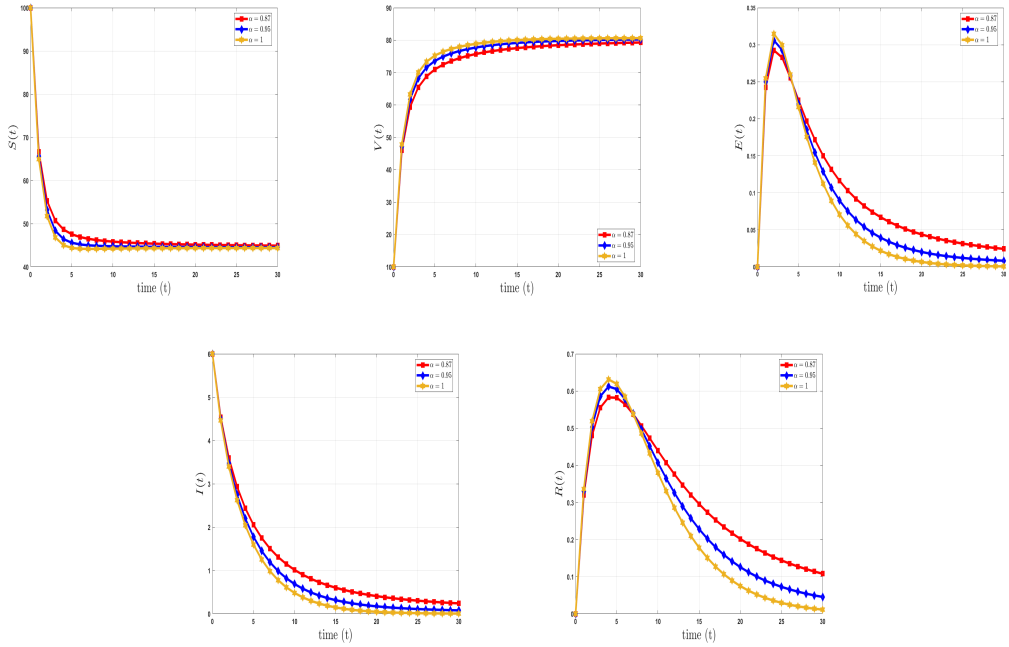

FIGURE 4. The dynamics of the system (2) for x fixed and $\alpha = 0.87, 0.95, 1$ in the case $\mathscr{R}_0 \leq 1$.

In the figures (1), (2) and (3) we observe that the spatio-temporal dynamics convergence toward the free equilibrium P^0 for different values of $\alpha = 1, 0.95, 0.87$. So according to analysis result, P^0 is a globally asymptotically stable. In Figure (4), we have fixed the space variable x to show the effect of the order α along the dynamics of the solution. We notice that all the solutions are globally asymptotically stable for different values of α not just for $\alpha = 1$. We also notice that the solution for $\alpha = 1$ quickly converges to the equilibrium point P^0. Because fractional derivatives capture reality effectively, we can conclude that it takes more time for the epidemic to become stable.

5.2. Numerical Simulation for $\mathscr{R}_0 > 1$. In this simulation, we consider the parameter values: $\Lambda = 25$, $\beta = 0.254$, $\mu = 0.6$, $\delta = 0.5$, $\omega = 0.22$, $\eta = 0.02432$, $\gamma = 0.003$, $a = 0.04$, $b = 0.02$, $d = 0.1 \times 10^{-5}$ and $d_S = d_V = d_E = d_I = d_R = 0.0000005$. Based on these parameter values, we have $\mathscr{R}_0 = 2.95 > 1$ and $P^* = (6.83, 0.202, 0.078, 15.66, 0.078)$. For the initial conditions we take $(S(0,x), V(0,x), E(0,x), I(0,x), R(0,x)) = (100, 10, 0, 6, 0)$.

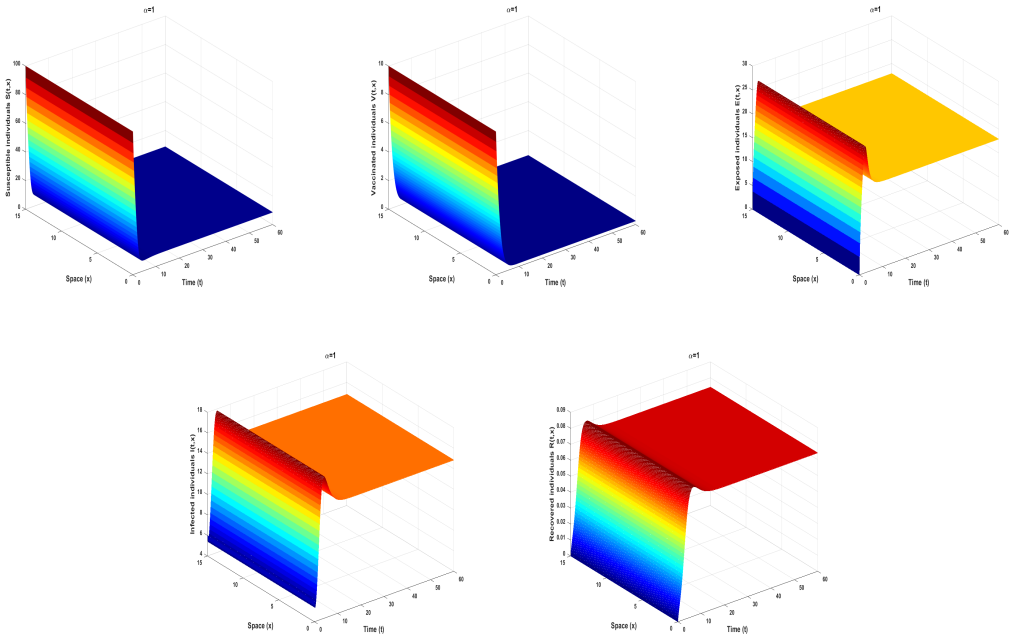

FIGURE 5. The dynamics of the system (2) showing the stability of the endemic equilibrium P^* for $\alpha = 1$.

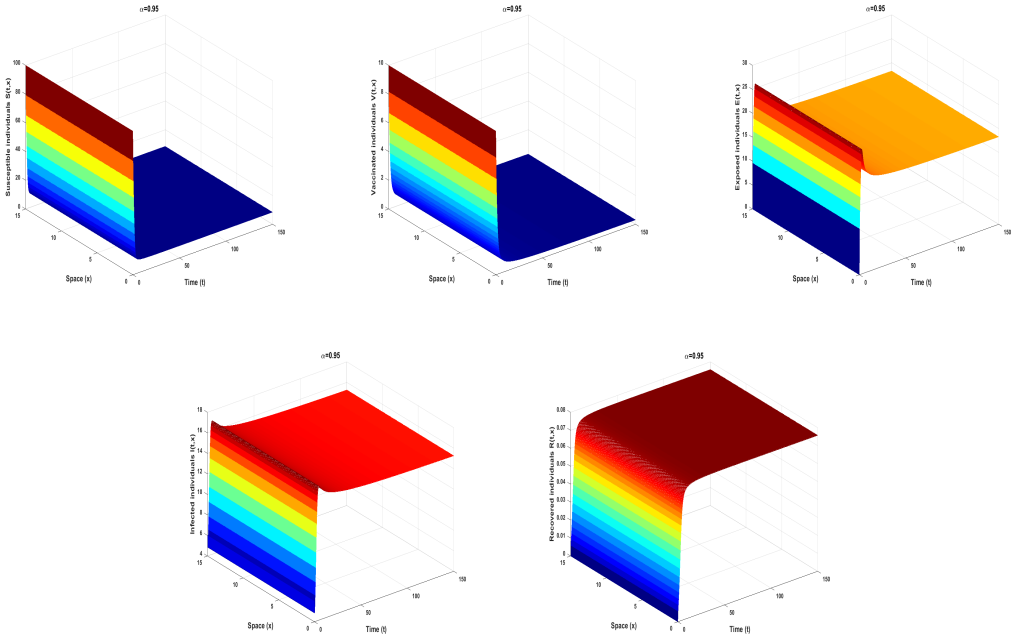

FIGURE 6. The dynamics of the system (2) showing the stability of the endemic equilibrium P^* for $\alpha = 0.95$.

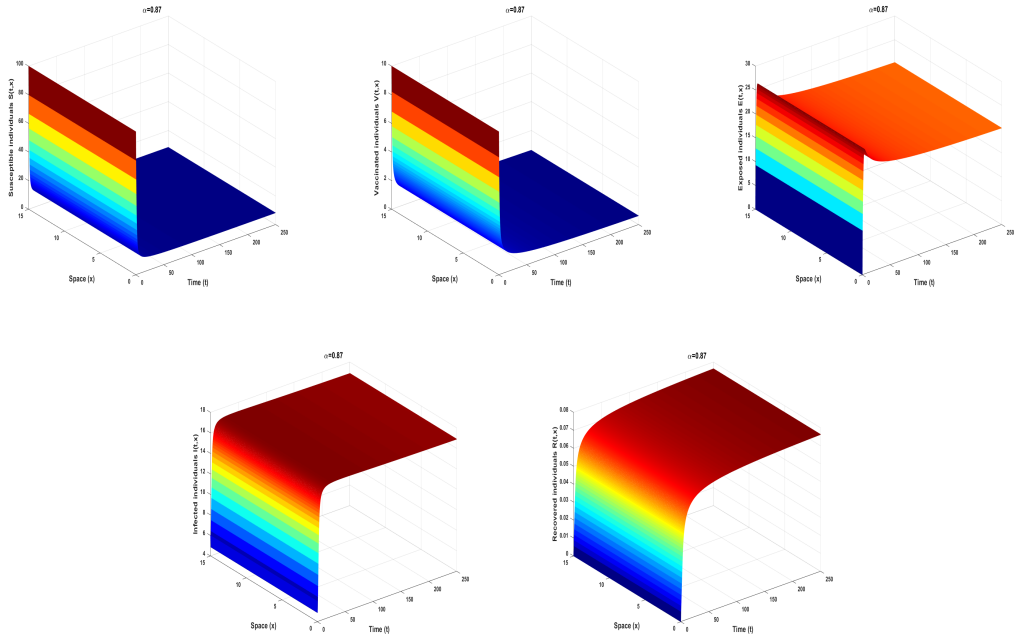

FIGURE 7. The dynamics of the system (2) showing the stability of the endemic equilibrium P^* for $\alpha = 0.87$.

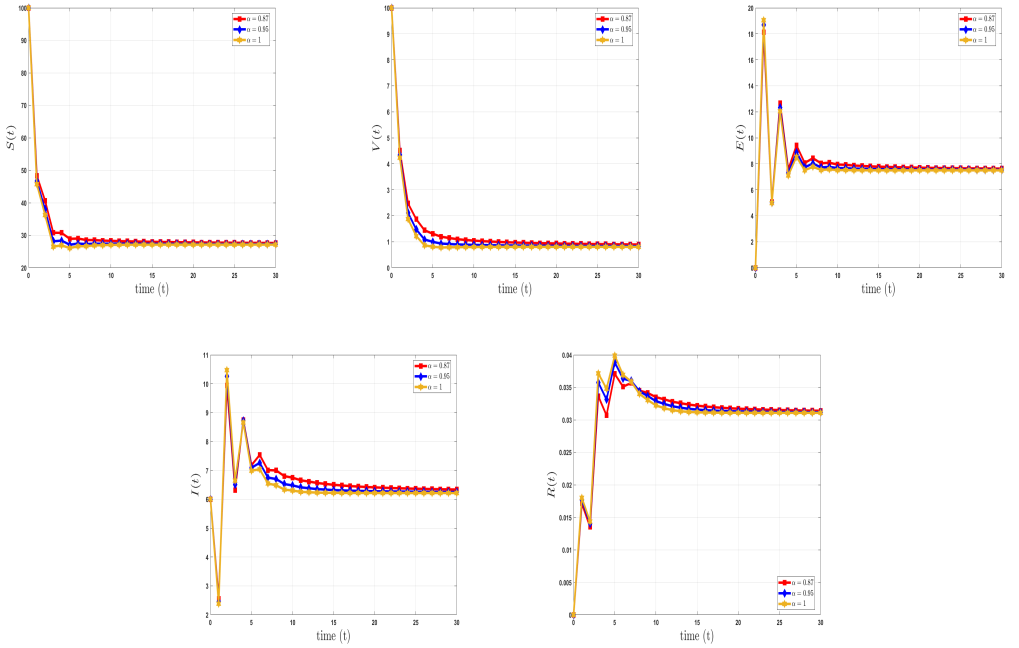

FIGURE 8. The dynamics of the system (2) for x fixed and $\alpha = 0.87, 0.95, 1$ in the case $\mathscr{R}_0 > 1$.

In the figures (5), (6) and (7) we observe that the spatio-temporal dynamics convergence toward the endemic equilibrium P^* for different values of $\alpha = 0.87, 0.95, 1$. So according to analysis result, P^* is a globally asymptotically stable. which means biologically that the infection persists. In Figure (8), we have fixed the space variable x to show the effect of the order α along the dynamics of the solution.

CONCLUSION

In this paper, we have focused on analyzing the qualitative behavior of fractional order $SVEIR$ model with diffusion and Beddington-DeAngelis incidence rate. First, we have determined the basic reproduction number $\mathscr{R}_0$ which plays a crucial role in shaping and influencing the overall global dynamics of our proposed model. After proving the existence of the two equilibrium points for the model, namely the free equilibrium denoted as P^0 and the endemic equilibrium P^*, we proceeded to confirm local stability of the disease free equilibrium P^0 and the global stability of the disease free equilibrium P^0 and the endemic equilibrium P^* by utilizing the Lyapunov function method. Based on our theoretical analysis, we were able to establish the stability of the equilibria not only for the case of the integer derivative $(\alpha = 1)$ but also for the entire range of $0 < \alpha \leq 1$. This finding reaffirms the universality and applicability of our system. It's important to emphasize that global asymptotic stability is observed across a spectrum of α values, and it's not limited to just $\alpha = 1$. In addition, it's noteworthy that the solution for $\alpha = 1$ exhibits a rapid convergence towards the equilibrium point. This interesting numerical experiment leads us to the conclusion that the fractional derivative order significantly influences the speed of convergence towards the equilibrium point. This effect can be attributed to the inherent memory characteristics associated with fractional derivatives. We conclude then that the fractional derivative order describe reality well since the epidemic takes a longer duration to be stable.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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