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A NOETIC FRACTAL-FRACTIONAL DYNAMICAL FORMALISM FOR HISTORY-DEPENDENT SEOUL VIRUS-STREPTOCOCCUS PYOGENES DUAL-PATHOGEN INTERACTION WITH PHYSICS-INFORMED LEARNING

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Abstract. Seoul virus is an important rodent-borne hantavirus that can cause serious illness in humans, while *Streptococcus pyogenes* is a clinically significant bacterial pathogen associated with a wide range of infections and severe complications. The interaction between these two infections is not well understood from a mathematical modeling perspective. In this work, we develop a fractal–fractional model to investigate the transmission and co-infection dynamics of Seoul virus and *Streptococcus pyogenes*. The model is described using the Atangana–Baleanu–Caputo fractal–fractional derivative so that the effects of memory and the nonuniform nature of disease transmission can be represented more realistically than in conventional integer-order models. Several mathematical properties of the proposed system are studied, including the existence and uniqueness of solutions

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and the behavior of the disease-free equilibrium. The basic reproduction number is obtained to identify the threshold conditions associated with the spread of the infections, and the Ulam–Hyers stability of the model is also examined. In addition to the theoretical analysis, a physics-informed neural network is constructed to approximate the solutions of the fractal–fractional system. The governing equations and initial conditions are incorporated into the neural-network training process, allowing the model structure to guide the numerical approximation. The simulations illustrate the influence of the fractional and fractal parameters on the evolution of the co-infection system and show that the PINN approach can reproduce the model dynamics with good accuracy. The findings suggest that combining fractal–fractional operators with physics-informed learning offers a useful way to study co-infectious disease systems in which memory, heterogeneity, and nonlinear interactions play an important role.

Keywords: fractal fractional; existence and uniqueness; Ulam–Hyers stability; co-infection model; physics-informed neural network.

2020 AMS Subject Classification: 92C60, 26A33, 65L05, 68T07.

1. INTRODUCTION

Seoul virus and *Streptococcus pyogenes* are two clinically important pathogens with distinct modes of transmission and disease characteristics. Seoul virus is a rodent-borne hantavirus that is maintained primarily in rodent populations and can cause serious disease in humans, including hemorrhagic fever with renal syndrome. In contrast, *Streptococcus pyogenes*, commonly referred to as Group A *Streptococcus* (GAS), is a bacterial pathogen responsible for a broad range of human infections, from relatively mild illnesses to severe invasive conditions. Although considerable attention has been given to the epidemiology and transmission of these pathogens individually, their behavior when occurring together has received comparatively little attention from a mathematical modeling perspective. Understanding such interactions is important because the presence of one pathogen may alter the progression and transmission characteristics of another through changes in host susceptibility, disease progression, or immune response. This limited understanding provides a clear motivation for developing a mathematical framework capable of describing the transmission and interaction of Seoul virus and *S. pyogenes* within a common epidemiological setting.

Seoul virus is primarily maintained in rodent reservoirs, particularly Norway rats, which can sustain persistent infection and provide repeated opportunities for transmission to humans. Easterbrook et al. [9] demonstrated the important role of regulatory T-cell activity in the chronic

infection of Seoul virus, providing evidence for mechanisms that may contribute to persistent viral infection within rodent populations. Hinson et al. [14] further reported the importance of aggressive encounters and bite wounds, particularly among male Norway rats, in the transmission of Seoul virus. These findings indicate that behavioral and ecological characteristics of rodent populations can influence the persistence and transmission of the virus. In addition, Knust et al. [15, 27] investigated a multistate Seoul virus outbreak in the United States associated with breeding facilities and home-based laboratories, demonstrating the continuing potential for rodent-to-human transmission and emphasizing the importance of surveillance. Similarly, Swanink et al. [26] reported the first indigenous case of Seoul virus infection in the Netherlands, further demonstrating that the geographical occurrence of the virus may extend beyond traditionally recognized endemic settings.

Streptococcus pyogenes is a Gram-positive, aerotolerant bacterium characterized by chains of non-motile and non-spore-forming cocci [4]. Although it may be present as part of the normal human microbial community, *S. pyogenes* can behave as an opportunistic pathogen and cause a broad spectrum of clinical infections. The bacterium produces clear zones of complete red-cell lysis on blood agar and belongs to Lancefield group A based on its specific antigenic characteristics [21]. Its name is derived from Greek terms associated with its chain-forming and pus-producing properties [21]. Laboratory methods used to distinguish *S. pyogenes* from other streptococcal species include biochemical and diagnostic tests such as the catalase and pyrrolidonyl arylamidase tests [25].

The global burden associated with GAS infections is substantial, with an estimated 700 million cases occurring annually and approximately 650,000 cases developing into severe or invasive infections [3]. Although the overall mortality associated with these infections is relatively low, mortality among invasive cases can approach 25% [3]. Delayed or inadequate treatment may result in serious complications, including sepsis and multiple-organ failure [8, 28]. Historically, *S. pyogenes* has also been associated with scarlet fever, a toxin-mediated disease resulting from the production of streptococcal exotoxins [10]. These epidemiological characteristics demonstrate the importance of understanding not only the individual transmission processes of

the two pathogens but also the possible consequences of their interaction within a shared host population.

Despite the epidemiological importance of Seoul virus and *S. pyogenes*, mathematical studies describing their combined transmission dynamics remain limited. Most conventional epidemic models are constructed using integer-order differential equations, which describe the present state of a system primarily through instantaneous rates of change. Such formulations may not adequately represent transmission processes in which previous infection history, delayed responses, persistence, or heterogeneous interactions influence the current dynamics. This limitation is particularly relevant for Seoul virus because of its persistent circulation in rodent reservoirs and for bacterial infections whose progression may depend on previous exposure and host-response mechanisms. Therefore, there is a need for a mathematical formulation that can incorporate temporal memory and irregular transmission characteristics within the same modeling framework.

Fractional calculus provides an alternative approach because fractional-order derivatives introduce nonlocal effects and allow the current state of a system to depend on its previous history[29]. This feature can be useful when modeling biological processes in which memory, persistence, delayed responses, or hereditary effects cannot be adequately represented by classical derivatives. However, fractional-order modeling alone does not necessarily account for irregular or heterogeneous structures in transmission processes[13]. Fractal–fractional calculus provides a broader framework by combining the memory characteristics of fractional operators with fractal behavior. Consequently, it offers additional flexibility for representing complex epidemiological systems in which both historical dependence and structural heterogeneity may influence disease evolution.

In this study, the Atangana–Baleanu–Caputo (ABC) fractal–fractional derivative is adopted to formulate the co-infection model. The use of the ABC operator is motivated by its ability to incorporate nonlocal memory effects through a nonsingular kernel while allowing the model to retain the influence of fractal characteristics. This is particularly relevant to the present problem because the transmission of Seoul virus involves persistent infection within rodent reservoirs, while the dynamics of *S. pyogenes* may involve nonlinear host–pathogen interactions

and different stages of infection. The proposed formulation therefore goes beyond a direct replacement of an integer-order derivative with a fractional derivative by providing a framework in which memory and fractal features can be considered simultaneously.

At the same time, conventional numerical techniques may become computationally demanding when applied to nonlinear fractal–fractional systems because of their nonlocal structure. Recent developments in artificial neural networks have provided alternative computational tools for approximating complex nonlinear biological systems. Deep artificial neural networks can learn complicated relationships from data with relatively few explicit assumptions. However, their dependence on sufficiently large and reliable datasets and their limited ability to preserve underlying mechanistic laws can restrict their applicability to epidemiological systems [16][20]. These limitations have encouraged the development of hybrid approaches that combine mechanistic mathematical models with the approximation capability of neural networks.

Physics-Informed Neural Networks (PINNs) are particularly attractive in this context because the governing differential equations can be incorporated directly into the training procedure. Rather than relying solely on observational data, PINNs use the mathematical structure of the underlying system to constrain the learned solution. This feature is especially useful for epidemiological models for which complete observational datasets may not always be available. PINN-based approaches have been successfully applied to several problems in engineering and biological sciences, including bioprocess modeling and system optimization [31, 23, 18, 30]. Their ability to incorporate governing laws into the learning process has also motivated their application to infectious-disease modeling, particularly in situations where available epidemiological data are limited [19, 17, 5, 6].

The above discussion reveals an important methodological gap. Existing epidemiological models generally focus on individual pathogens, while conventional integer-order formulations may not adequately capture memory-dependent and heterogeneous transmission processes. On the other hand, fractional models introduce memory effects but may not simultaneously represent fractal characteristics of the underlying system. Moreover, the use of physics-informed neural networks for solving fractal–fractional co-infection models involving both a rodent-borne

viral infection and a bacterial infection remains insufficiently explored. These limitations motivate the development of a model that combines co-infection dynamics, fractal–fractional memory effects, and physics-informed computational learning within a single framework.

To address this gap, we formulate a new fractal–fractional mathematical model for the co-infection dynamics of Seoul virus and *Streptococcus pyogenes* using the Atangana–Baleanu–Caputo derivative. The proposed model is designed to describe the transmission and progression of the two infections while accounting for the nonlocal and fractal characteristics of the underlying epidemiological processes. The mathematical properties of the model are investigated by establishing the existence and uniqueness of its solutions. The disease-free equilibrium is identified, and the basic reproduction number is derived to determine the threshold conditions associated with disease invasion and persistence. In addition, Ulam–Hyers stability is analyzed to examine the robustness of the model with respect to small perturbations. To complement the theoretical analysis, a physics-informed neural network is developed for approximating the solutions of the proposed fractal–fractional system. The governing equations and initial conditions are incorporated into the learning process so that the neural network remains consistent with the underlying epidemiological dynamics.

The main contribution of this work is therefore the integration of three complementary components: a mathematical representation of Seoul virus and *S. pyogenes* co-infection, an ABC fractal–fractional formulation capable of representing memory and fractal effects, and a PINN-based computational framework for solving the resulting nonlocal system. This combination provides an alternative approach for investigating co-infection systems whose dynamics may involve persistence, memory, nonlinear interactions, and heterogeneous transmission characteristics.

The remainder of this paper is organized as follows. Sections 1 and 2 introduce the mathematical preliminaries and definitions required for the fractal–fractional Atangana–Baleanu–Caputo formulation. Sections 3 proposed transmission model for the co-infection of Seoul virus and *Streptococcus pyogenes* is developed in the subsequent section. The existence and uniqueness of the model solutions are established using a fixed-point approach in Section 4. Section 5 focuses on the disease-free equilibrium and the derivation of the basic reproduction number. The

Ulam–Hyers stability of the system is investigated in Section 6. Numerical simulations and the corresponding results are presented in Section 7. Section 8 describes the physics-informed neural network framework used to approximate the solutions of the proposed fractal–fractional model. Finally, Section 9 summarizes the principal findings and outlines possible directions for future research.

2. PRELIMINARIES

This section outlines the basic framework of the fractal-fractional (FF) operator and introduces the key definitions necessary for deriving the main results of this work. Furthermore, in the context of this study, we make use of the function space $y(t) \in C([0, 1]) \rightarrow \mathbb{R}$, where the magnitude of the function is evaluated through the norm $\|y\| = \max_{t \in [0, 1]} |y(t)|$.

Definition 2.1. [2, 1] Let $y \in C((a, b), \mathbb{R})$ be a function that is fractal differentiable on (a, b) . Considering a generalized Mittag-Leffler kernel, with a fractal order $0 < \wp \leq 1$ and a fractional dimension $0 < \Theta \leq 1$, the FF derivative of $y(t)$ is defined as follows:

$${}_0^{FFM}D_t^{\wp, \Theta}(y(t)) = \frac{\mathcal{AB}(\wp)}{(1 - \wp)} \frac{d}{dt^\Theta} \int_0^t y(u) \mathcal{E}_\wp\left(\frac{-\wp}{1 - \wp}(t - u)^\wp\right) du,$$

where $AB(\wp) = 1 - \wp + \frac{\wp}{\gamma(\wp)}$ and $\frac{dy(u)}{du^\Theta} = \lim_{s \rightarrow u} \frac{y(t) - y(u)}{t^\Theta - u^\Theta}$.

Definition 2.2. [2, 1] Atangana-Baleanu defines the FF integral of $y(t)$ with a Mittag-Leffler type kernel and a fractional order $0 < \wp \leq 1$ as follows:

$${}_0^{FFM}I_s^{\wp, \Theta}(y(t)) = \frac{\wp^\Theta}{AB(\wp)\gamma(\wp)} \int_0^t u^{\Theta-1} y(u) (t - u)^{\wp-1} du + \frac{\Theta(1 - \wp)t^{\Theta-1}}{AB(\wp)} y(t).$$

3. MODEL FORMULATION

This model explores the transmission dynamics of Seoul Virus and Streptococcus pyogenes within a human population, explicitly accounting for the emergence of coinfection and its clinical progression. The total human population is structured into epidemiological compartments according to infection status and treatment stage. Individuals are recruited into the susceptible class S_h at a constant rate Π and are subject to natural mortality at rate μ . Susceptible individuals acquire infection through effective contact with infectious individuals and progress to the

exposed classes E_{HV} or E_{SC} , corresponding to Seoul Virus and streptococcal infection, respectively, governed by the forces of infection γ_{HV} and γ_{SC} . After an incubation period, exposed individuals move to the infectious classes I_{HV} and I_{SC} at rates σ_{HV} and σ_{SC} . Infected individuals may initiate treatment at rates ϕ_{HV} and ϕ_{SC} , transitioning to the treatment compartments T_{HV} and T_{SC} , from which recovery occurs at rates ρ_{HV} and ρ_{SC} , leading to the recovered classes R_{HV} and R_{SC} .

The model further incorporates coinfection dynamics by allowing susceptible individuals to enter the exposed coinfection class E_{HS} through the force of infection γ_{HS} , while individuals in the single-infection or exposed classes may acquire a secondary infection and progress to the coinfecting infectious class I_{HS} . The progression from E_{HS} to I_{HS} occurs at rate ϵ_{HS} , and coinfecting individuals may receive treatment at rate ϕ_{HS} , entering the treatment class T_{HS} and subsequently recovering into R_{HS} at rate ρ_{HS} . The parameters ζ_{HV} and ζ_{SC} represent the rates at which individuals infected with one pathogen acquire the second pathogen, while ω_{HV} and ω_{SC} capture the contribution of exposed individuals to coinfection through secondary exposure pathways.

The forces of infection are defined in terms of effective transmission coefficients and population mixing, where β_{HV} , β_{SC} , and β_{HS} denote the transmission rates of Seoul Virus, streptococcal infection, and coinfection, respectively. The modification parameters κ_{HV} and κ_{SC} account for the enhanced or reduced infectiousness of coinfecting individuals in transmitting single infections, while θ_{HV} and θ_{SC} quantify the contribution of singly infected individuals to the spread of coinfection. The total human population size is denoted by N_h . Disease-induced mortality associated with severe infection and coinfection is represented by the parameter τ , which acts on the relevant infectious and treatment compartments. This comprehensive framework enables the assessment of interaction effects between Seoul Virus and *Streptococcus pyogenes* and provides meaningful insight into the epidemiological impact of coinfection on disease transmission and control. Accordingly, the total human population at any time t , denoted by $N_h(t)$, is obtained by summing all epidemiological compartments and is given by

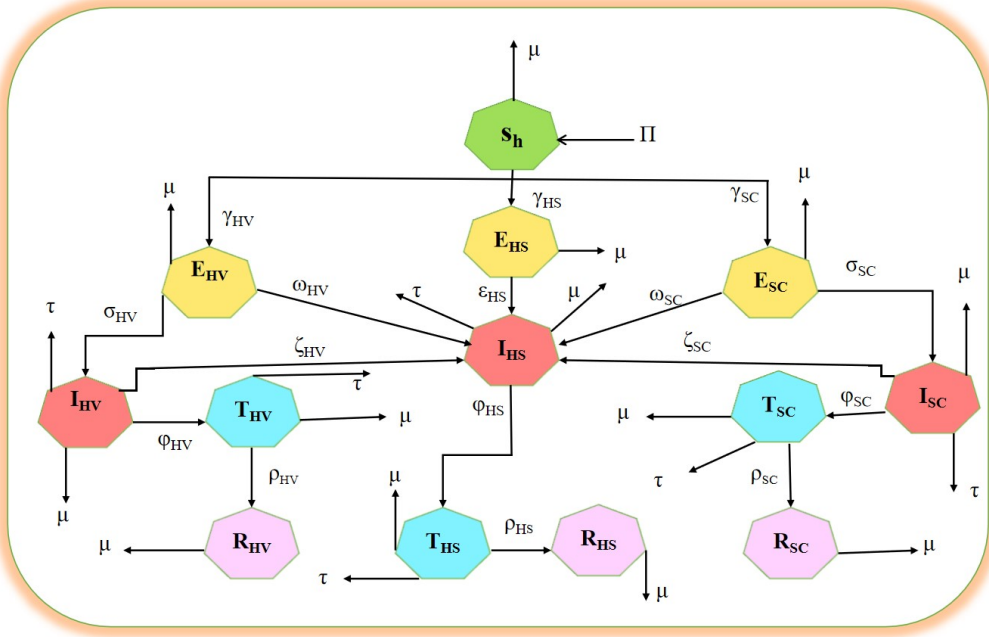


FIGURE 1. Schematic Diagram Illustrating the Structure and Transmission Pathways of the Co-infection Model

$$N_h(t) = S_h + E_{HV} + I_{HV} + T_{HV} + R_{HV} + E_{SC} + I_{SC} + T_{SC} + R_{SC} + E_{HS} + I_{HS} + T_{HS} + R_{HS}.$$

Considering the interaction structure among the epidemiological classes and incorporating memory effects through a time-dependent kernel characterized by the Mittag-Leffler correlation function, the transmission dynamics of Seoul Virus and Streptococcus pyogenes with coinfection are described by a deterministic system of nonlinear fractal-fractional differential equations.

$${}^{FF}D_t^{\varphi, \Theta} S_h = \Pi - (\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu) S_h,$$

$${}^{FF}D_t^{\varphi, \Theta} E_{HV} = \gamma_{HV} S_h - (\sigma_{HV} + \omega_{HV} + \mu) E_{HV},$$

$${}^{FF}D_t^{\varphi, \Theta} I_{HV} = \sigma_{HV} E_{HV} - (\phi_{HV} + \zeta_{HV} + \tau + \mu) I_{HV},$$

$${}^{FF}D_t^{\varphi, \Theta} T_{HV} = \phi_{HV} I_{HV} - (\rho_{HV} + \tau + \mu) T_{HV},$$

$${}^{FF}D_t^{\varphi, \Theta} R_{HV} = \rho_{HV} T_{HV} - \mu R_{HV},$$

$$\begin{aligned}
& {}^{FF}D_t^{\varphi, \Theta} E_{SC} = \gamma_{SC} S_h - (\sigma_{SC} + \omega_{SC} + \mu) E_{SC}, \\
& {}^{FF}D_t^{\varphi, \Theta} I_{SC} = \sigma_{SC} E_{SC} - (\phi_{SC} + \zeta_{SC} + \tau + \mu) I_{SC}, \\
& {}^{FF}D_t^{\varphi, \Theta} T_{SC} = \phi_{SC} I_{SC} - (\rho_{SC} + \tau + \mu) T_{SC}, \\
& {}^{FF}D_t^{\varphi, \Theta} R_{SC} = \rho_{SC} T_{SC} - \mu R_{SC}, \\
(3.1) \quad & {}^{FF}D_t^{\varphi, \Theta} E_{HS} = \gamma_{HS} S_h - (\epsilon_{HS} + \mu) E_{HS}, \\
& {}^{FF}D_t^{\varphi, \Theta} I_{HS} = \epsilon_h E_{HS} + \zeta_{HV} I_{HV} + \zeta_{SC} I_{SC} + \omega_{HV} E_{HV} + \omega_{SC} E_{SC} \\
& \quad - (\phi_{HS} + \tau + \mu) I_{HS}, \\
& {}^{FF}D_t^{\varphi, \Theta} T_{HS} = \phi_{HS} I_{HS} - (\rho_{HS} + \tau + \mu) T_{HS}, \\
& {}^{FF}D_t^{\varphi, \Theta} R_{HS} = \rho_{HS} T_{HS} - \mu R_{HS}.
\end{aligned}$$

with,

$$\begin{aligned}
\gamma_{HV} &= \frac{\beta_{HV}}{N_h} (I_{HV} + \kappa_{HV} I_{HS}), \\
\gamma_{SC} &= \frac{\beta_{SC}}{N_h} (I_{SC} + \kappa_{SC} I_{HS}), \\
\gamma_{HS} &= \frac{\beta_{HS}}{N_h} (I_{HS} + \theta_{HV} I_{HV} + \theta_{SC} I_{SC}),
\end{aligned}$$

The model system is supplemented with non-negative initial conditions corresponding to each epidemiological compartment. At the initial time $t = 0$, the state variables are assumed to satisfy,

$$\begin{aligned}
(3.2) \quad & S_h(0) = S_{h0}, \quad E_{HV}(0) = E_{HV0}, \quad I_{HV}(0) = I_{HV0}, \quad T_{HV}(0) = T_{HV0}, \quad R_{HV}(0) = R_{HV0}, \\
& E_{SC}(0) = E_{SC0}, \quad I_{SC}(0) = I_{SC0}, \quad T_{SC}(0) = T_{SC0}, \quad R_{SC}(0) = R_{SC0}, \\
& E_{HS}(0) = E_{HS0}, \quad I_{HS}(0) = I_{HS0}, \quad T_{HS}(0) = T_{HS0}, \quad R_{HS}(0) = R_{HS0},
\end{aligned}$$

4. EXISTENCE AND UNIQUENESS OF THE SOLUTION

In this section, we shall prove the existence and uniqueness of the proposed model using a fixed point approach. By applying the Atangana Baleanu Fractional order Fuzzy integration

operator on the given equation as shown in equation 3.1 and choosing the initial conditions, we may obtain the following: [22]

$$S_h(t) = S_h(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} [\Pi - (\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu)S_h] du \\ + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\Pi - (\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu)S_h],$$

$$E_{HV}(t) = E_{HV}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} [\gamma_{HV}S_h - (\sigma_{HV} + \omega_{HV} + \mu)E_{HV}] du \\ + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\gamma_{HV}S_h - (\sigma_{HV} + \omega_{HV} + \mu)E_{HV}],$$

$$I_{HV}(t) = I_{HV}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} [\sigma_{HV}E_{HV} - (\phi_{HV} + \zeta_{HV} + \tau + \mu)I_{HV}] du \\ + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\sigma_{HV}E_{HV} - (\phi_{HV} + \zeta_{HV} + \tau + \mu)I_{HV}],$$

$$T_{HV}(t) = T_{HV}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} [\phi_{HV}I_{HV} - (\rho_{HV} + \tau + \mu)T_{HV}] du \\ + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\phi_{HV}I_{HV} - (\rho_{HV} + \tau + \mu)T_{HV}],$$

$$R_{HV}(t) = R_{HV}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} [\rho_{HV}T_{HV} - \mu R_{HV}] du \\ + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\rho_{HV}T_{HV} - \mu R_{HV}],$$

$$E_{SC}(t) = E_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} [\gamma_{SC}S_h - (\sigma_{SC} + \omega_{SC} + \mu)E_{SC}] du \\ + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\gamma_{SC}S_h - (\sigma_{SC} + \omega_{SC} + \mu)E_{SC}],$$

$$I_{SC}(t) = I_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} [\sigma_{SC}E_{SC} - (\phi_{SC} + \zeta_{SC} + \tau + \mu)I_{SC}] du \\ + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\sigma_{SC}E_{SC} - (\phi_{SC} + \zeta_{SC} + \tau + \mu)I_{SC}],$$

$$T_{SC}(t) = T_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} [\phi_{SC}I_{SC} - (\rho_{SC} + \tau + \mu)T_{SC}] du$$

$$\begin{aligned}
& + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\varphi_{SC}I_{SC} - (\rho_{SC} + \tau + \mu)T_{SC}], \\
R_{SC}(t) &= R_{SC}(0) + \frac{\wp\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} [\rho_{SC}T_{SC} - \mu R_{SC}] du \\
& + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\rho_{SC}T_{SC} - \mu R_{SC}], \\
E_{HS}(t) &= E_{HS}(0) + \frac{\wp\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} [\gamma_{HS}S_h - (\varepsilon_{HS} + \mu)E_{HS}] du \\
& + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\gamma_{HS}S_h - (\varepsilon_{HS} + \mu)E_{HS}], \\
I_{HS}(t) &= I_{HS}(0) + \frac{\wp\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} [\varepsilon_{HS}E_{HS} + \zeta_{HV}I_{HV} + \zeta_{SC}I_{SC} + \omega_{HV}E_{HV} + \omega_{SC}E_{SC} \\
& - (\varphi_{HS} + \tau + \mu)I_{HS}] du \\
& + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\varepsilon_{HS}E_{HS} + \zeta_{HV}I_{HV} + \zeta_{SC}I_{SC} + \omega_{HV}E_{HV} + \omega_{SC}E_{SC} - (\varphi_{HS} + \tau + \mu)I_{HS}], \\
T_{HS}(t) &= T_{HS}(0) + \frac{\wp\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} [\varphi_{HS}I_{HS} - (\rho_{HS} + \tau + \mu)T_{HS}] du \\
& + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\varphi_{HS}I_{HS} - (\rho_{HS} + \tau + \mu)T_{HS}], \\
R_{HS}(t) &= R_{HS}(0) + \frac{\wp\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} [\rho_{HS}T_{HS} - \mu R_{HS}] du \\
& + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} [\rho_{HS}T_{HS} - \mu R_{HS}].
\end{aligned}$$

$$\mathfrak{R}_1(t, S_h) = \Pi - (\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu)S_h,$$

$$\mathfrak{R}_2(t, E_{HV}) = \gamma_{HV}S_h - (\sigma_{HV} + \omega_{HV} + \mu)E_{HV},$$

$$\mathfrak{R}_3(t, I_{HV}) = \sigma_{HV}E_{HV} - (\varphi_{HV} + \zeta_{HV} + \tau + \mu)I_{HV},$$

$$\mathfrak{R}_4(t, T_{HV}) = \varphi_{HV}I_{HV} - (\rho_{HV} + \tau + \mu)T_{HV},$$

$$\mathfrak{R}_5(t, R_{HV}) = \rho_{HV}T_{HV} - \mu R_{HV},$$

$$\mathfrak{R}_6(t, E_{SC}) = \gamma_{SC}S_h - (\sigma_{SC} + \omega_{SC} + \mu)E_{SC},$$

$$\mathfrak{R}_7(t, I_{SC}) = \sigma_{SC}E_{SC} - (\varphi_{SC} + \zeta_{SC} + \tau + \mu)I_{SC},$$

$$\mathfrak{R}_8(t, T_{SC}) = \varphi_{SC} I_{SC} - (\rho_{SC} + \tau + \mu) T_{SC},$$

$$\mathfrak{R}_9(t, R_{SC}) = \rho_{SC} T_{SC} - \mu R_{SC},$$

$$\mathfrak{R}_{10}(t, E_{HS}) = \gamma_{HS} S_h - (\varepsilon_{HS} + \mu) E_{HS},$$

$$\begin{aligned} \mathfrak{R}_{11}(t, I_{HS}) &= \varepsilon_{HS} E_{HS} + \zeta_{HV} I_{HV} + \zeta_{SC} I_{SC} + \omega_{HV} E_{HV} + \omega_{SC} E_{SC} \\ &\quad - (\varphi_{HS} + \tau + \mu) I_{HS}, \end{aligned}$$

$$\mathfrak{R}_{12}(t, T_{HS}) = \varphi_{HS} I_{HS} - (\rho_{HS} + \tau + \mu) T_{HS},$$

$$\mathfrak{R}_{13}(t, R_{HS}) = \rho_{HS} T_{HS} - \mu R_{HS}.$$

$$\|S_h(t)\| \leq \mathcal{L}_1, \quad \|E_{HV}(t)\| \leq \mathcal{L}_2, \quad \|I_{HV}(t)\| \leq \mathcal{L}_3,$$

$$\|T_{HV}(t)\| \leq \mathcal{L}_4, \quad \|R_{HV}(t)\| \leq \mathcal{L}_5, \quad \|E_{SC}(t)\| \leq \mathcal{L}_6,$$

$$\|I_{SC}(t)\| \leq \mathcal{L}_7, \quad \|T_{SC}(t)\| \leq \mathcal{L}_8, \quad \|R_{SC}(t)\| \leq \mathcal{L}_9,$$

$$\|E_{HS}(t)\| \leq \mathcal{L}_{10}, \quad \|I_{HS}(t)\| \leq \mathcal{L}_{11}, \quad \|T_{HS}(t)\| \leq \mathcal{L}_{12},$$

$$\|R_{HS}(t)\| \leq \mathcal{L}_{13},$$

Theorem 4.1. Assume that hypothesis $(\mathcal{H})$ holds. Then, the nonlinear operators $\mathfrak{R}_i(t, \cdot)$, $i \in \mathbb{N}_1^{13}$, satisfy the Lipschitz condition. Moreover, if the associated Lipschitz constants η_i satisfy $0 < \eta_i < 1$ for all $i \in \mathbb{N}_1^{13}$, then the operator generated by the system is a contraction.

Proof. We first show that the operator $\mathfrak{R}_1(t, S_h)$ satisfies the Lipschitz condition. Let $S_h(t), S_h^*(t) \in L[0, 1]$. Then,

$$\begin{aligned} \|\mathfrak{R}_1(t, S_h) - \mathfrak{R}_1(t, S_h^*)\| &= \|-(\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu)(S_h - S_h^*)\| \\ &\leq (\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu) \|S_h - S_h^*\| \\ &\leq \eta_1 \|S_h - S_h^*\|, \end{aligned}$$

$$\begin{aligned} \|\mathfrak{R}_2(t, E_{HV}) - \mathfrak{R}_2(t, E_{HV}^*)\| &= \|-(\sigma_{HV} + \omega_{HV} + \mu)(E_{HV} - E_{HV}^*)\| \\ &\leq (\sigma_{HV} + \omega_{HV} + \mu) \|E_{HV} - E_{HV}^*\| \\ &\leq \eta_2 \|E_{HV} - E_{HV}^*\|, \end{aligned}$$

$$\|\mathfrak{R}_3(t, I_{HV}) - \mathfrak{R}_3(t, I_{HV}^*)\| \leq (\varphi_{HV} + \zeta_{HV} + \tau + \mu) \|I_{HV} - I_{HV}^*\| = \eta_3 \|I_{HV} - I_{HV}^*\|,$$

$$\begin{aligned}
\|\mathfrak{R}_4(t, T_{HV}) - \mathfrak{R}_4(t, T_{HV}^*)\| &\leq (\rho_{HV} + \tau + \mu) \|T_{HV} - T_{HV}^*\| = \eta_4 \|T_{HV} - T_{HV}^*\|, \\
\|\mathfrak{R}_5(t, R_{HV}) - \mathfrak{R}_5(t, R_{HV}^*)\| &\leq \mu \|R_{HV} - R_{HV}^*\| = \eta_5 \|R_{HV} - R_{HV}^*\|, \\
\|\mathfrak{R}_6(t, E_{SC}) - \mathfrak{R}_6(t, E_{SC}^*)\| &\leq (\sigma_{SC} + \omega_{SC} + \mu) \|E_{SC} - E_{SC}^*\| = \eta_6 \|E_{SC} - E_{SC}^*\|, \\
\|\mathfrak{R}_7(t, I_{SC}) - \mathfrak{R}_7(t, I_{SC}^*)\| &\leq (\varphi_{SC} + \zeta_{SC} + \tau + \mu) \|I_{SC} - I_{SC}^*\| = \eta_7 \|I_{SC} - I_{SC}^*\|, \\
\|\mathfrak{R}_8(t, T_{SC}) - \mathfrak{R}_8(t, T_{SC}^*)\| &\leq (\rho_{SC} + \tau + \mu) \|T_{SC} - T_{SC}^*\| = \eta_8 \|T_{SC} - T_{SC}^*\|, \\
\|\mathfrak{R}_9(t, R_{SC}) - \mathfrak{R}_9(t, R_{SC}^*)\| &\leq \mu \|R_{SC} - R_{SC}^*\| = \eta_9 \|R_{SC} - R_{SC}^*\|, \\
\|\mathfrak{R}_{10}(t, E_{HS}) - \mathfrak{R}_{10}(t, E_{HS}^*)\| &\leq (\varepsilon_{HS} + \mu) \|E_{HS} - E_{HS}^*\| = \eta_{10} \|E_{HS} - E_{HS}^*\|, \\
\|\mathfrak{R}_{11}(t, I_{HS}) - \mathfrak{R}_{11}(t, I_{HS}^*)\| &\leq (\varphi_{HS} + \tau + \mu) \|I_{HS} - I_{HS}^*\| = \eta_{11} \|I_{HS} - I_{HS}^*\|, \\
\|\mathfrak{R}_{12}(t, T_{HS}) - \mathfrak{R}_{12}(t, T_{HS}^*)\| &\leq (\rho_{HS} + \tau + \mu) \|T_{HS} - T_{HS}^*\| = \eta_{12} \|T_{HS} - T_{HS}^*\|, \\
\|\mathfrak{R}_{13}(t, R_{HS}) - \mathfrak{R}_{13}(t, R_{HS}^*)\| &\leq \mu \|R_{HS} - R_{HS}^*\| = \eta_{13} \|R_{HS} - R_{HS}^*\|.
\end{aligned}$$

Consequently, for every $i \in \mathcal{N}_1^{13}$, all of the kernels $\mathfrak{R}_j, j \in \mathcal{N}_1^{13}$ satisfy the Lipschitz condition with constant $\eta_i < 1$. Hence, the proof is completed. Now, the system can be rewritten as follows:

$$\begin{aligned}
S_h(t) &= S_h(0) + \frac{\wp \Theta}{AB(\wp) \Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_1(u, S_h(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_1(t, S_h(t)), \\
E_{HV}(t) &= E_{HV}(0) + \frac{\wp \Theta}{AB(\wp) \Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_2(u, E_{HV}(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_2(t, E_{HV}(t)), \\
I_{HV}(t) &= I_{HV}(0) + \frac{\wp \Theta}{AB(\wp) \Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_3(u, I_{HV}(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_3(t, I_{HV}(t)), \\
T_{HV}(t) &= T_{HV}(0) + \frac{\wp \Theta}{AB(\wp) \Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_4(u, T_{HV}(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_4(t, T_{HV}(t)),
\end{aligned}$$

$$\begin{aligned}
 R_{HV}(t) = & R_{HV}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_5(u, R_{HV}(u)) du \\
 & + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_5(t, R_{HV}(t)),
 \end{aligned}$$

$$\begin{aligned}
 E_{SC}(t) = & E_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_6(u, E_{SC}(u)) du \\
 & + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_6(t, E_{SC}(t)),
 \end{aligned}$$

$$\begin{aligned}
 I_{SC}(t) = & I_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_7(u, I_{SC}(u)) du \\
 & + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_7(t, I_{SC}(t)),
 \end{aligned}$$

$$\begin{aligned}
 T_{SC}(t) = & T_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_8(u, T_{SC}(u)) du \\
 & + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_8(t, T_{SC}(t)),
 \end{aligned}$$

$$\begin{aligned}
 R_{SC}(t) = & R_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_9(u, R_{SC}(u)) du \\
 & + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_9(t, R_{SC}(t)),
 \end{aligned}$$

$$\begin{aligned}
 E_{HS}(t) = & E_{HS}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_{10}(u, E_{HS}(u)) du \\
 & + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_{10}(t, E_{HS}(t)),
 \end{aligned}$$

$$\begin{aligned}
 I_{HS}(t) = & I_{HS}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_{11}(u, I_{HS}(u)) du \\
 & + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_{11}(t, I_{HS}(t)),
 \end{aligned}$$

$$\begin{aligned}
 T_{HS}(t) = & T_{HS}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_{12}(u, T_{HS}(u)) du \\
 & + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_{12}(t, T_{HS}(t)),
 \end{aligned}$$

$$\begin{aligned}
R_{HS}(t) &= R_{HS}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_{13}(u, R_{HS}(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_{13}(t, R_{HS}(t)).
\end{aligned}$$

Together with the initial conditions given by

$$\begin{aligned}
S_{h_0}(t) &= S_h(0), E_{HV_0}(t) = E_{HV}(0), I_{HV_0}(t) = I_{HV}(0), T_{HV_0}(t) = T_{HV}(0), R_{HV_0}(t) = R_{HV}(0), \\
E_{SC_0}(t) &= E_{SC}(0), I_{SC_0}(t) = I_{SC}(0), T_{SC_0}(t) = T_{SC}(0), R_{SC_0}(t) = R_{SC}(0), \\
E_{HS_0}(t) &= E_{HS}(0), I_{HS_0}(t) = I_{HS}(0), T_{HS_0}(t) = T_{HS}(0), R_{HS_0}(t) = R_{HS}(0).
\end{aligned}$$

The recursive formulas corresponding to Equation (5) are given by

$$\begin{aligned}
S_{h_n}(t) &= S_h(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_1(u, S_{h_{n-1}}(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_1(t, S_{h_{n-1}}(t)), \\
E_{HV_n}(t) &= E_{HV}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_2(u, E_{HV_{n-1}}(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_2(t, E_{HV_{n-1}}(t)), \\
I_{HV_n}(t) &= I_{HV}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_3(u, I_{HV_{n-1}}(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_3(t, I_{HV_{n-1}}(t)), \\
T_{HV_n}(t) &= T_{HV}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_4(u, T_{HV_{n-1}}(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_4(t, T_{HV_{n-1}}(t)), \\
R_{HV_n}(t) &= R_{HV}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_5(u, R_{HV_{n-1}}(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_5(t, R_{HV_{n-1}}(t)), \\
E_{SC_n}(t) &= E_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_6(u, E_{SC_{n-1}}(u)) du \\
&\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_6(t, E_{SC_{n-1}}(t)),
\end{aligned}$$

$$\begin{aligned}
 I_{SC_n}(t) &= I_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_7(u, I_{SC_{n-1}}(u)) du \\
 &\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_7(t, I_{SC_{n-1}}(t)), \\
 T_{SC_n}(t) &= T_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_8(u, T_{SC_{n-1}}(u)) du \\
 &\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_8(t, T_{SC_{n-1}}(t)), \\
 R_{SC_n}(t) &= R_{SC}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_9(u, R_{SC_{n-1}}(u)) du \\
 &\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_9(t, R_{SC_{n-1}}(t)), \\
 E_{HS_n}(t) &= E_{HS}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_{10}(u, E_{HS_{n-1}}(u)) du \\
 &\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_{10}(t, E_{HS_{n-1}}(t)), \\
 I_{HS_n}(t) &= I_{HS}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_{11}(u, I_{HS_{n-1}}(u)) du \\
 &\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_{11}(t, I_{HS_{n-1}}(t)), \\
 T_{HS_n}(t) &= T_{HS}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_{12}(u, T_{HS_{n-1}}(u)) du \\
 &\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_{12}(t, T_{HS_{n-1}}(t)), \\
 R_{HS_n}(t) &= R_{HS}(0) + \frac{\wp^\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \mathfrak{R}_{13}(u, R_{HS_{n-1}}(u)) du \\
 &\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \mathfrak{R}_{13}(t, R_{HS_{n-1}}(t)).
 \end{aligned}$$

Theorem 4.2. If the following condition is satisfied, then the model 3.1 admits a solution,

$$\triangle = \max\{\delta_i\} < 1, \quad i \in \mathcal{N}_1^{13}.$$

Proof: Define the following sequences:

$$\wp_{1_n}(t) = S_{h_{n+1}}(t) - S_h(t),$$

$$\wp_{2_n}(t) = E_{HV_{n+1}}(t) - E_{HV}(t),$$

$$\begin{aligned}
\wp_{3_n}(t) &= I_{HV_{n+1}}(t) - I_{HV}(t), \\
\wp_{4_n}(t) &= T_{HV_{n+1}}(t) - T_{HV}(t), \\
\wp_{5_n}(t) &= R_{HV_{n+1}}(t) - R_{HV}(t), \\
\wp_{6_n}(t) &= E_{SC_{n+1}}(t) - E_{SC}(t), \\
\wp_{7_n}(t) &= I_{SC_{n+1}}(t) - I_{SC}(t), \\
\wp_{8_n}(t) &= T_{SC_{n+1}}(t) - T_{SC}(t), \\
\wp_{9_n}(t) &= R_{SC_{n+1}}(t) - R_{SC}(t), \\
\wp_{10_n}(t) &= E_{HS_{n+1}}(t) - E_{HS}(t), \\
\wp_{11_n}(t) &= I_{HS_{n+1}}(t) - I_{HS}(t), \\
\wp_{12_n}(t) &= T_{HS_{n+1}}(t) - T_{HS}(t), \\
\wp_{13_n}(t) &= R_{HS_{n+1}}(t) - R_{HS}(t).
\end{aligned}$$

Then, we obtain

$$\begin{aligned}
\|\wp_{1_n}(t)\| &\leq \frac{\wp\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1}(t-u)^{\wp-1} \|\mathfrak{R}_1(u, S_{h_n}(u)) - \mathfrak{R}_1(u, S_h(u))\| du \\
&\quad + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} \|\mathfrak{R}_1(t, S_{h_n}(t)) - \mathfrak{R}_1(t, S_h(t))\| \\
&\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right] \delta_1 \|S_{h_n} - S_h\| \\
&\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_1^n \|S_{h_1} - S_h\|.
\end{aligned}$$

Similarly, we get,

$$\begin{aligned}
\|\wp_{2_n}(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_2^n \|E_{HV_1} - E_{HV}\|, \\
\|\wp_{3_n}(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_3^n \|I_{HV_1} - I_{HV}\|, \\
\|\wp_{4_n}(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_4^n \|T_{HV_1} - T_{HV}\|,
\end{aligned}$$

$$\begin{aligned}
 \|\wp_5(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_5^n \|R_{HV_1} - R_{HV}\|, \\
 \|\wp_6(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_6^n \|E_{SC_1} - E_{SC}\|, \\
 \|\wp_7(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_7^n \|I_{SC_1} - I_{SC}\|, \\
 \|\wp_8(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_8^n \|T_{SC_1} - T_{SC}\|, \\
 \|\wp_9(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_9^n \|R_{SC_1} - R_{SC}\|, \\
 \|\wp_{10}(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_{10}^n \|E_{HS_1} - E_{HS}\|, \\
 \|\wp_{11}(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_{11}^n \|I_{HS_1} - I_{HS}\|, \\
 \|\wp_{12}(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_{12}^n \|T_{HS_1} - T_{HS}\|, \\
 \|\wp_{13}(t)\| &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right]^n \delta_{13}^n \|R_{HS_1} - R_{HS}\|.
 \end{aligned}$$

Hence, for $\triangle = \max\{\delta_i\} < 1$, the sequence $\{\wp_{i_n}(t)\}$ converges uniformly, and therefore the model 3.1 admits a solution. This completes the proof.

Theorem 4.3. Under Assumption $\mathcal{H}$, the model 3.1 admits a unique solution provided that

$$\left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right] \delta_i \leq 1, \quad i = 1, 2, \dots, 13.$$

Proof: Assume that there exists another solution

$$(S_h^*(t), E_{HV}^*(t), I_{HV}^*(t), T_{HV}^*(t), R_{HV}^*(t), E_{SC}^*(t), I_{SC}^*(t), T_{SC}^*(t), R_{SC}^*(t), E_{HS}^*(t), I_{HS}^*(t), T_{HS}^*(t), R_{HS}^*(t)),$$

with the same initial conditions.

$$\begin{aligned}
 S_h^*(t) &= S_h^*(0) + \frac{\wp\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \mathfrak{R}_1(u, S_h^*(u)) du \\
 &\quad + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} \mathfrak{R}_1(t, S_h^*(t)).
 \end{aligned}$$

Now, we compute

$$\begin{aligned}
 \|S_h - S_h^*\| &= \frac{\wp\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \|\mathfrak{R}_1(u, S_h(u)) - \mathfrak{R}_1(u, S_h^*(u))\| du \\
 &\quad + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} \|\mathfrak{R}_1(t, S_h(t)) - \mathfrak{R}_1(t, S_h^*(t))\|. \\
 (4.1) \quad \|S_h - S_h^*\| &\leq \frac{\wp\Theta}{AB(\wp)\Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \Phi_1 \|S_h - S_h^*\| du \\
 &\quad + \frac{\Theta(1-\wp)t^{\Theta-1}}{AB(\wp)} \Phi_1 \|S_h - S_h^*\| \\
 &\leq \left[\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right] \Phi_1 \|S_h - S_h^*\|.
 \end{aligned}$$

$$(4.2) \quad \left[1 - \left(\frac{\wp\Theta\Gamma(\Theta)}{AB(\wp)\Gamma(\wp+\Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right) \Phi_1 \right] \|S_h - S_h^*\| \leq 0.$$

The inequality in (4.4) holds only if $\|S_h - S_h^*\| = 0$, which implies $S_h(t) = S_h^*(t)$ for all $t \geq 0$. Applying the same argument to $E_{HV}, I_{HV}, T_{HV}, R_{HV}, E_{SC}, I_{SC}, T_{SC}, R_{SC}, E_{HS}, I_{HS}, T_{HS}$ and R_{HS} , we conclude that the model 3.1 possesses a unique solution. This completes the proof.

5. DISEASE-FREE EQUILIBRIUM AND BASIC REPRODUCTION NUMBER

The disease-free equilibrium (DFE) of system 3.1 is obtained by setting all infected, exposed, treated, and recovered compartments equal to zero, namely

$$E_{HV} = I_{HV} = T_{HV} = R_{HV} = E_{SC} = I_{SC} = T_{SC} = R_{SC} = E_{HS} = I_{HS} = T_{HS} = R_{HS} = 0.$$

From the susceptible human population equation,

$${}^{FF}D_t^{\wp, \Theta} S_h = \Pi - (\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu) S_h,$$

and by equating the right-hand side to zero, we obtain

$$S_h^0 = \frac{\Pi}{\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu}.$$

Hence, the disease-free equilibrium of model 3.1 is given by

$$\mathcal{E}_0 = \left(\frac{\Pi}{\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0 \right).$$

5.1. Basic Reproduction Number. Using the next-generation matrix approach, the infected compartments are

$$(E_{HV}, E_{SC}, E_{HS}).$$

The matrix of new infection terms is given by

$$\mathcal{F} = \begin{pmatrix} \gamma_{HV} S_h \\ \gamma_{SC} S_h \\ \gamma_{HS} S_h \end{pmatrix},$$

while the transition matrix is

$$\mathcal{V} = \begin{pmatrix} (\sigma_{HV} + \omega_{HV} + \mu) E_{HV} \\ (\sigma_{SC} + \omega_{SC} + \mu) E_{SC} \\ (\varepsilon_{HS} + \mu) E_{HS} \end{pmatrix}.$$

Evaluating the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ at the disease-free equilibrium $\mathcal{E}_0$, we obtain

$$F = \begin{pmatrix} \gamma_{HV} S_h^0 & 0 & 0 \\ 0 & \gamma_{SC} S_h^0 & 0 \\ 0 & 0 & \gamma_{HS} S_h^0 \end{pmatrix}, \quad V = \begin{pmatrix} \sigma_{HV} + \omega_{HV} + \mu & 0 & 0 \\ 0 & \sigma_{SC} + \omega_{SC} + \mu & 0 \\ 0 & 0 & \varepsilon_{HS} + \mu \end{pmatrix}.$$

Thus, the next-generation matrix is given by

$$K = FV^{-1}.$$

The basic reproduction number $\mathcal{R}_0$ is the spectral radius of K , which yields

$$\mathcal{R}_0 = \frac{\gamma_{HV} S_h^0}{\sigma_{HV} + \omega_{HV} + \mu} + \frac{\gamma_{SC} S_h^0}{\sigma_{SC} + \omega_{SC} + \mu} + \frac{\gamma_{HS} S_h^0}{\varepsilon_{HS} + \mu}.$$

Substituting the value of S_h^0 , we finally obtain

$$\mathcal{R}_0 = \frac{\Pi}{\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu} \left(\frac{\gamma_{HV}}{\sigma_{HV} + \omega_{HV} + \mu} + \frac{\gamma_{SC}}{\sigma_{SC} + \omega_{SC} + \mu} + \frac{\gamma_{HS}}{\varepsilon_{HS} + \mu} \right).$$

6. ULAM-HYERS STABILITY

In this section, we establish the Ulam-Hyers stability of the fractal-fractional model given by system 3.1. For this purpose, we introduce the following definition [24].

Definition 6.1. The model 3.1 is said to be Ulam–Hyers stable if there exist constants $\zeta_i > 0$, $i = 1, 2, \dots, 14$, such that for every $\ell_i > 0$, $i = 1, 2, \dots, 13$, the inequalities

$$\begin{aligned}
 & \left| {}^{FF}D_t^{\varrho, \Theta} S_h(t) - \Pi_1(t, S_h) \right| \leq \ell_1, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} E_{HV}(t) - \Pi_2(t, E_{HV}) \right| \leq \ell_2, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} I_{HV}(t) - \Pi_3(t, I_{HV}) \right| \leq \ell_3, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} T_{HV}(t) - \Pi_4(t, T_{HV}) \right| \leq \ell_4, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} R_{HV}(t) - \Pi_5(t, R_{HV}) \right| \leq \ell_5, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} E_{SC}(t) - \Pi_6(t, E_{SC}) \right| \leq \ell_6, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} I_{SC}(t) - \Pi_7(t, I_{SC}) \right| \leq \ell_7, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} T_{SC}(t) - \Pi_8(t, T_{SC}) \right| \leq \ell_8, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} R_{SC}(t) - \Pi_9(t, R_{SC}) \right| \leq \ell_9, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} E_{HS}(t) - \Pi_{10}(t, E_{HS}) \right| \leq \ell_{10}, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} I_{HS}(t) - \Pi_{11}(t, I_{HS}) \right| \leq \ell_{11}, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} T_{HS}(t) - \Pi_{12}(t, T_{HS}) \right| \leq \ell_{12}, \\
 & \left| {}^{FF}D_t^{\varrho, \Theta} R_{HS}(t) - \Pi_{13}(t, R_{HS}) \right| \leq \ell_{13},
 \end{aligned}
 \tag{6.1}$$

where $\Pi_i(\cdot)$ denote the right-hand sides of system 3.1. Additionally, assume that system 3.1 admits a unique solution

$$(S_h^*, E_{HV}^*, I_{HV}^*, T_{HV}^*, R_{HV}^*, E_{SC}^*, I_{SC}^*, T_{SC}^*, R_{SC}^*, E_{HS}^*, I_{HS}^*, T_{HS}^*, R_{HS}^*).$$

Remark 6.1. Let $h_1(t)$ be a continuous function such that

$$|h_1(t)| < \ell_1,$$

and

$${}^{FF}D_t^{\wp, \Theta} S_h(t) - \Pi_1(t, S_h) + h_1(t) = 0.$$

Similarly, for $i = 1, 2, \dots, 13$, one may define continuous functions $h_i(t)$ satisfying inequalities 6.1.

Theorem 6.1. Under Assumption $\mathcal{H}$, the fractal–fractional model 3.1 is Ulam–Hyers stable.

Proof: Let $\ell_1 > 0$ and $S_h(t)$ be any function satisfying

$$|{}^{FF}D_t^{\wp, \Theta} S_h(t) - \Pi_1(t, S_h)| \leq \ell_1.$$

From Remark 1, there exists a function $h_1(t)$ such that $|h_1(t)| < \ell_1$ and

$${}^{FF}D_t^{\wp, \Theta} S_h(t) = \Pi_1(t, S_h) - h_1(t).$$

Applying the associated fractal–fractional integral operator, we obtain

$$\begin{aligned} S_h(t) &= S_h(0) + \frac{\wp \Theta}{AB(\wp) \Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \Pi_1(u, S_h(u)) du \\ &\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \Pi_1(t, S_h(t)) \\ &\quad + \frac{\wp \Theta}{AB(\wp) \Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} h_1(u) du + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} h_1(t). \end{aligned}$$

Similarly, the exact solution $S_h^*(t)$ satisfies

$$\begin{aligned} S_h^*(t) &= S_h^*(0) + \frac{\wp \Theta}{AB(\wp) \Gamma(\wp)} \int_0^t u^{\Theta-1} (t-u)^{\wp-1} \Pi_1(u, S_h^*(u)) du \\ &\quad + \frac{\Theta(1-\wp)}{AB(\wp)} t^{\Theta-1} \Pi_1(t, S_h^*(t)). \end{aligned}$$

Taking the norm and using the Lipschitz condition, we get

$$\begin{aligned} \|S_h - S_h^*\| &\leq \left[\frac{\wp \Theta \Gamma(\Theta)}{AB(\wp) \Gamma(\wp + \Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right] \delta_1 \|S_h - S_h^*\| \\ &\quad + \left[\frac{\wp \Theta \Gamma(\Theta)}{AB(\wp) \Gamma(\wp + \Theta)} + \frac{\Theta(1-\wp)}{AB(\wp)} \right] \ell_1. \end{aligned}$$

Hence,

$$\|S_h - S_h^*\| \leq \zeta_1 \ell_1,$$

where

$$\zeta_1 = \frac{\left[\frac{\varrho\Theta\Gamma(\Theta)}{AB(\varrho)\Gamma(\varrho+\Theta)} + \frac{\Theta(1-\varrho)}{AB(\varrho)} \right]}{1 - \left[\frac{\varrho\Theta\Gamma(\Theta)}{AB(\varrho)\Gamma(\varrho+\Theta)} + \frac{\Theta(1-\varrho)}{AB(\varrho)} \right] \delta_1}.$$

Applying the same argument to the remaining state variables, we obtain

$$\|E_{HV} - E_{HV}^*\| \leq \zeta_2 \ell_2, \quad \|I_{HV} - I_{HV}^*\| \leq \zeta_3 \ell_3, \quad \dots, \quad \|R_{HS} - R_{HS}^*\| \leq \zeta_{13} \ell_{13}.$$

Therefore, the fractal–fractional model 3.1 is Ulam–Hyers stable. $\square$

7. NUMERICAL SIMULATION

Numerical simulations were conducted to analyze the dynamics of the proposed fractal- fractional Seoul virus–Streptococcus co-infection model using parameter values and initial conditions derived from available epidemiological data [11, 12]. To reduce computational complexity while preserving the inherent memory and heterogeneity effects, the fractal–fractional operator was approximated by its integer-order representation. The resulting system of ordinary differential equations was solved $t \in [0, 250]$, ensuring stable and accurate solutions. This approach effectively captures both transient behavior and long-term disease evolution, including rapid changes during outbreak peaks and transitions among epidemiological states. The initial population configuration was designed to represent a realistic situation where the majority of the population is susceptible, and only a few are exposed or infected. In particular, we assigned $S_h(0) = 800$, with small initial values for all exposed and infected compartments. This parameterization is centered on the early stages of the outbreak and highlights the role of fractal-fractional dynamics during the initial invasion process.

The recruitment rate into the susceptible compartment was set to $\Pi = 20$, and the natural death rate to $\mu = 0.01$. The transmission rates from the susceptible population to the Seoul Virus, Streptococcus, and severe infection compartments were set to $\gamma_{HV} = 0.3$, $\gamma_{SC} = 0.25$, and $\gamma_{HS} = 0.15$, respectively, to represent moderate transmission rates in line with observed trends. The transition rates from exposed to infected compartments were assigned $\sigma_{HV} = 0.2$ and $\sigma_{SC} = 0.18$, with the rate to severe co-infection $\varepsilon_{HS} = 0.12$. The rate of treatment initiation was set as $\phi_{HV} = 0.1$, $\phi_{SC} = 0.09$, and $\phi_{HS} = 0.11$, representing the response to treatment for the different disease states. The recovery rates from treatment were set as $\rho_{HV} = 0.08$, $\rho_{SC} = 0.07$,

and $\rho_{HS} = 0.09$. The parameters controlling cross-infection and disease progression ω_{HV} , ω_{SC} , ζ_{HV} , and ζ_{SC} were chosen to represent biologically plausible interaction strengths between Seoul Virus and Streptococcus infections. The disease-induced removal rate was fixed at $\tau = 0.02$.

Figure 2 illustrates the susceptible human population $S_h(t)$ dynamics for different values of the fractal parameter $\wp = 0.2, 0.54, 0.8$, and 1.0 , while the fractional order is kept constant at $\Theta = 0.9$. The susceptible population is expected to decrease over time due to infection pressure and natural mortality. Higher $\wp$ values lead to faster depletion of the susceptible population $S_h(t)$, indicating higher contact heterogeneity and faster disease spread. Lower $\wp$ values incorporate higher fractal memory effects, resulting in slower depletion of the susceptible population and a more gradual epidemic process.

The same figure also illustrates the dynamics of the exposed human population related to the Seoul Virus transmission route, $E_{HV}(t)$. This population increases first as susceptible individuals turn into infected ones and then decreases as they turn into the infected group. Higher $\wp$ values result in earlier and more prominent peaks, which represent stronger exposure due to the dominant fractal impact. Lower $\wp$ values result in later and less prominent peaks, emphasizing the role of fractal memory in controlling the process.

Figure 3 illustrates the infected Seoul Virus population, $I_{HV}(t)$. The findings indicate that higher values of the fractal parameter result in more prominent and steeper infection peaks, which represent the fast transition from exposure to infection. As lower $\wp$ values are considered, the infection peak becomes flatter and longer, which emphasizes the role of fractal-fractional memory in controlling the infection process by reducing the sudden outbreak peaks. The treated Seoul Virus population, $T_{HV}(t)$, also behaves in a similar manner, increasing with the rise in infection levels and then decreasing as the recovery process takes over. The Seoul Virus population $R_{HV}(t)$ is depicted in Figure 4. This class grows over time and eventually reaches a steady state. The larger the fractal parameter, the faster the accumulation of recoveries, and the smaller fractal parameters stress the memory-driven recovery process using the fractional operator. This demonstrates the fractal-fractional model's influence on the long-term disease spread.

The exposed Streptococcus population $E_{SC}(t)$ is depicted in Figure 5. This class initially grows and then declines as the population moves towards infection. Increasing the $\wp$ parameter increases the exposed peak, indicating higher susceptibility to fractal influences; smaller $\wp$ parameters stress the exposure process. The infected Streptococcus population $I_{SC}(t)$ is depicted in Figure 5. The larger fractal parameters increase the infection spread and peak; smaller $\wp$ parameters stress the infection process by suppressing the infection peak and extending the infection time, indicating the fractional influence on the infection process.

The treated Streptococcus population $T_{SC}(t)$ closely follows the infection process, with larger $\wp$ parameters increasing the treatment peak and time. Smaller fractal parameters stress the treatment initiation time, indicating the fractal-fractional influence on the treatment initiation time. The recovered Streptococcus population $R_{SC}(t)$ is depicted in Figure 6. This class steadily grows towards equilibrium. Larger fractal parameters stress the recovery process, and smaller parameters stress the memory-driven recovery process using the fractional operator. Figure 7 illustrates the exposed co-infection class $E_{HS}(t)$, which emerges based on the interaction between Seoul Virus and Streptococcus. A larger cross-infection parameter $\wp$ indicates a faster growth rate for the co-exposed class, indicating greater cross-pathway interactions. A smaller $\wp$ indicates less co-infection pressure. The infected co-infection class $I_{HS}(t)$ represents the most critical phase of the infection and is very sensitive to changes in the fractal parameter. A larger $\wp$ indicates a significantly increased infection pressure, indicating the nonlinear and synergistic interactions in co-infection processes.

Figure 8 highlights the treated co-infected class $T_{HS}(t)$. This class represents the infection process, and larger fractal parameters indicate increased treatment demand. Smaller $\wp$ parameters indicate smoother and more delayed treatment processes, indicating memory effects in treating critical co-infections. The recovered co-infected class $R_{HS}(t)$ grows slowly and saturates as time progresses. Larger fractal parameters indicate faster recovery accumulation, while smaller parameters indicate a longer recovery time due to the combined effects of fractal and fractional parameters. Overall, these findings confirm the biological validity and robustness of the fractal-fractional co-infection model.

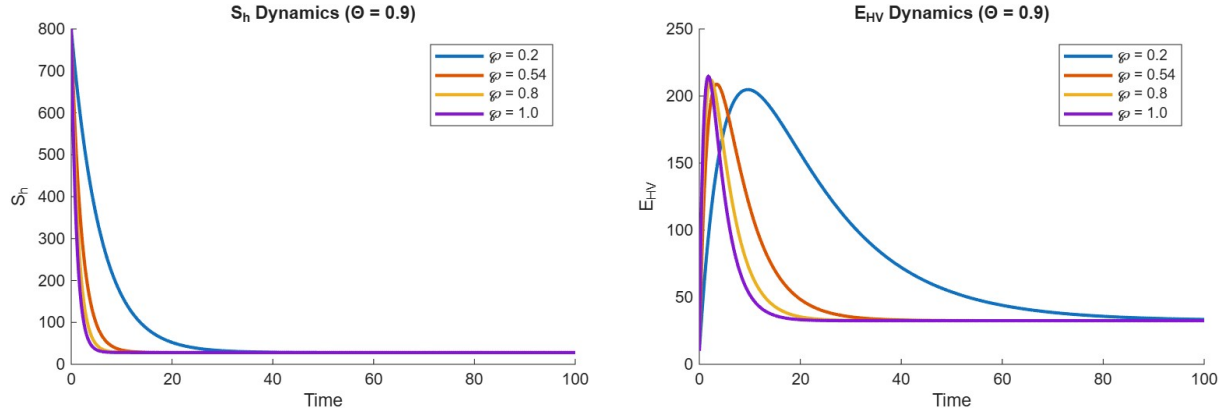


FIGURE 2. Dynamics of Fractal-Fractional Susceptible and Exposed Seoul Virus Population

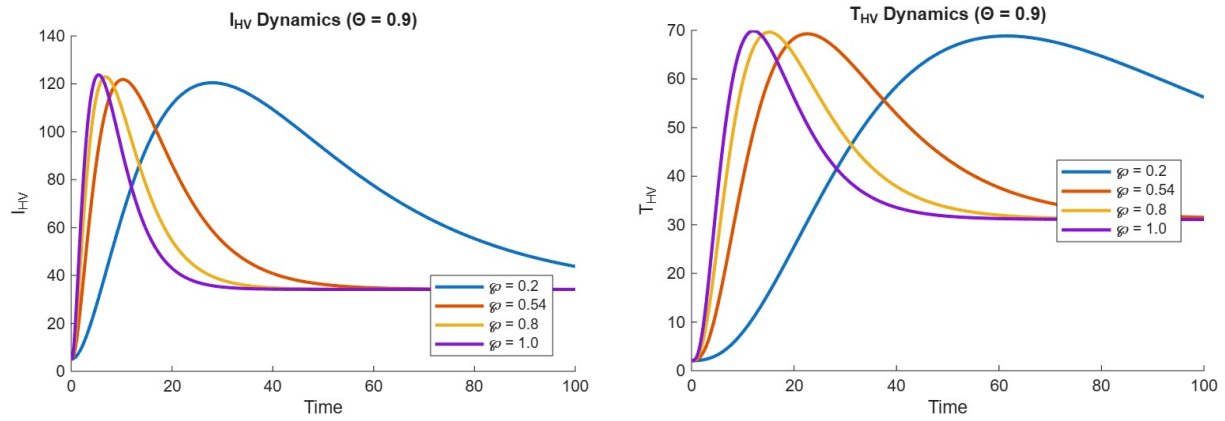


FIGURE 3. Dynamics of Fractal-Fractional Infected and Treated Seoul Virus Population

8. PINN APPROACH FOR SOLVING THE MODEL

8.1. Fractal-Fractional Physics-Informed Neural Network (FF-PINN) Framework. To numerically solve the proposed fractal-fractional Seoul Virus-Streptococcus pyogenes co-infection model, a PINN framework is adopted. Unlike purely data-driven neural networks, the PINN approach explicitly embeds the underlying fractal-fractional epidemiological equations into the training process, ensuring that the learned solution remains consistent with the governing biological laws.

The proposed Physics-Informed Neural Network is designed with a single input corresponding to the temporal variable, while the output layer comprises thirteen neurons representing the

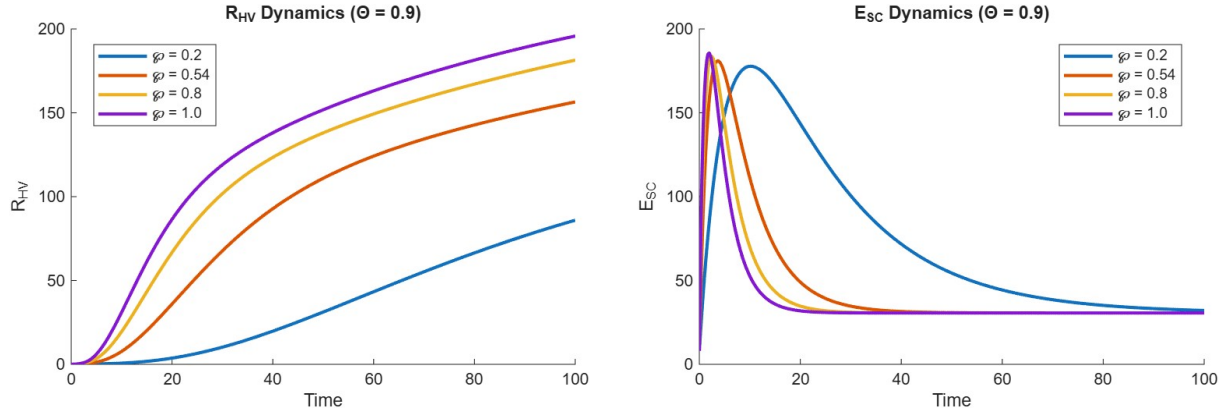


FIGURE 4. Dynamics of Fractal-Fractional Recovered Seoul Virus and Exposed Streptococcus Pyogenes Population

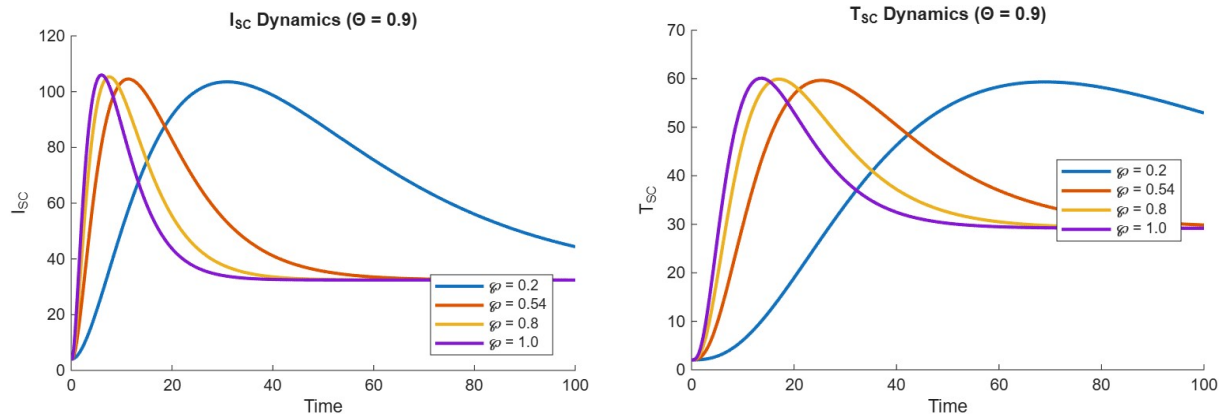


FIGURE 5. Dynamics of Fractal-Fractional Infected and Treated Streptococcus Pyogenes Population

state variables of the co-infection model. The hidden structure consists of three fully connected layers, each containing 50 neurons and equipped with smooth activation functions, which are well suited for capturing the memory and heterogeneity effects inherent to the fractal-fractional operator. Network training is carried out using the Adam optimizer, ensuring efficient and stable convergence in the high-dimensional parameter space. The loss function is formulated as a weighted combination of the residuals of the governing fractal-fractional differential equations, the imposed initial conditions, and the available observational data [7].

8.2. Neural Network Approximation. The PINN is designed to approximate the temporal evolution of all epidemiological compartments in the model. The neural network takes time t

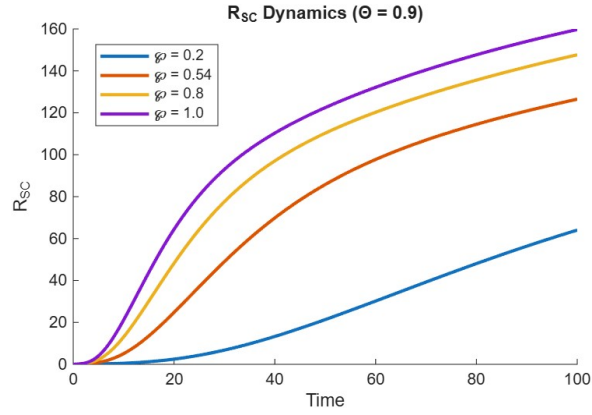


FIGURE 6. Dynamics of Fractal-Fractional Recovered Streptococcus pyogenes Population

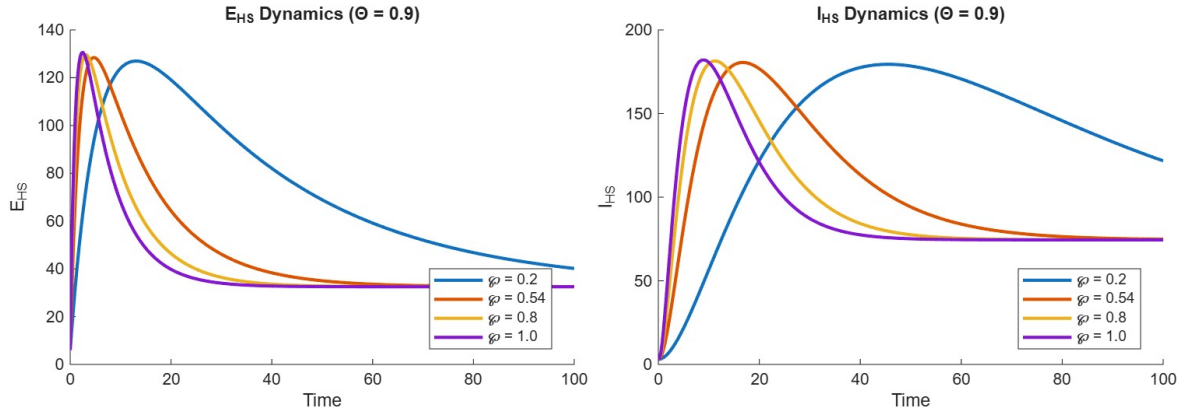


FIGURE 7. Dynamics of Fractal-Fractional Coinfected Exposed and Infected Population

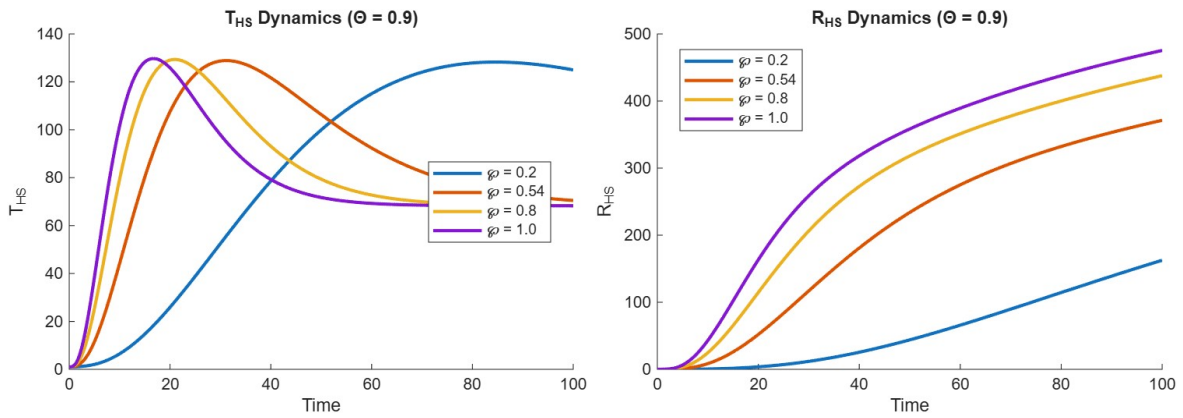


FIGURE 8. Dynamics of Fractal-Fractional Coinfected Treated and Recovered Populations

as input and produces a vector-valued output representing the compartment states, given by

$$\hat{\mathbf{u}}(t) = \begin{bmatrix} \hat{S}_h(t), \hat{E}_{HV}(t), \hat{I}_{HV}(t), \hat{T}_{HV}(t), \hat{R}_{HV}(t), \\ \hat{E}_{SC}(t), \hat{I}_{SC}(t), \hat{T}_{SC}(t), \hat{R}_{SC}(t), \\ \hat{E}_{HS}(t), \hat{I}_{HS}(t), \hat{T}_{HS}(t), \hat{R}_{HS}(t) \end{bmatrix}.$$

Each component of $\hat{\mathbf{u}}(t)$ represents a smooth neural approximation to the corresponding state variable in the fractal–fractional differential equation (FFDE) system. Automatic differentiation is employed to compute the required time derivatives, ensuring high numerical accuracy without discretization errors.

8.3. Fractal-Fractional Residual Construction. The physics-informed component of the PINN is constructed by enforcing the fractal–fractional governing equations at a set of collocation points $\{t_j\}_{j=1}^{N_c}$. For each compartment, a residual function $\mathcal{R}_j(t)$ is defined by substituting the neural network outputs into the FFDEs. For example, the residual associated with the susceptible human population is given by

$$\mathcal{R}_{S_h}(t) = {}^{FF}D_t^{\rho, \Theta} \hat{S}_h(t) - \left(\Pi - (\gamma_{HV} + \gamma_{SC} + \gamma_{HS} + \mu) \hat{S}_h(t) \right),$$

with similar residuals constructed for all remaining compartments. These residuals quantify the degree to which the neural network output violates the underlying epidemiological dynamics.

8.4. Loss Function Formulation. The total loss function guiding the FF–PINN training is defined as a weighted sum of three components:

$$(8.1) \quad \mathcal{L} = \lambda_1 \sum_{i=1}^{N_d} \left\| \hat{\mathbf{u}}(t_i) - \mathbf{u}^{\text{data}}(t_i) \right\|^2 + \lambda_2 \sum_{j=1}^{N_c} \left\| \mathcal{R}_j(t_j) \right\|^2 + \lambda_3 \left\| \hat{\mathbf{u}}(0) - \mathbf{u}_0 \right\|^2,$$

where $\mathbf{u}^{\text{data}}(t_i)$ denotes the available observational or synthetic data, $\mathbf{u}_0$ represents the initial conditions, and λ_1 , λ_2 , and λ_3 are positive weighting parameters that balance data fidelity, physical consistency, and initial condition enforcement, respectively.

8.5. Training Dynamics and Convergence Behavior. During the initial phase of training 9, the PINN loss starts at a relatively high magnitude due to the random initialization of the network parameters. Within the first 100-150 epochs, the loss decreases rapidly from the order of 10^1 to below 10^1 , indicating that the network quickly captures the dominant dynamics of the

proposed co-infection model. As the training progresses, the rate of loss reduction becomes more gradual, reflecting continuous improvement in satisfying the physics-based constraints and data consistency.

Around 500 epochs, the loss reaches approximately $10^{0.5}$, demonstrating a substantial reduction in the residual errors associated with the governing equations. Beyond this stage, the loss continues to decline smoothly and monotonically, eventually approaching values close to 10^{-1} by 1000 epochs. This consistent decay of the loss function confirms the stable convergence of the FF-PINN framework and highlights its capability to accurately learn nonlinear interactions and memory effects introduced by the fractal-fractional derivative, while preserving population conservation throughout the training process.

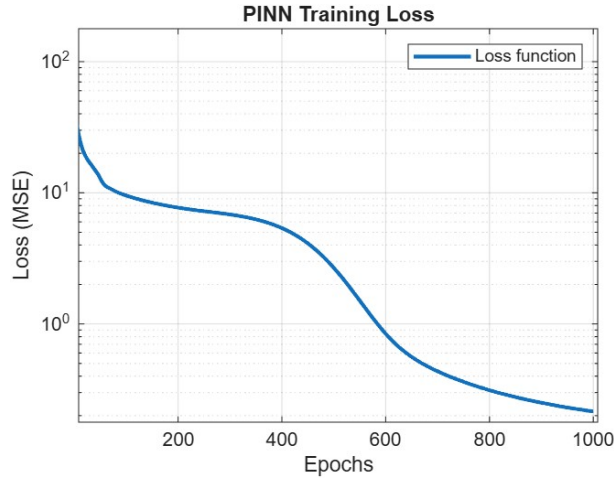


FIGURE 9. The Loss Function with $\varphi = 0.5$ and $\Theta = 0.9$

8.6. Simulation of Forward FF-PINNs. To simulate the dynamics of the proposed fractal-fractional Mutual model of Seoul Virus and Steptococcus Pyogenes, we implemented a forward PINN. The network consisted of three hidden layers with 50 neurons each, trained over 1000 epochs. The temporal domain $t \in [0, 200]$ was discretized into 1000 collocation points, which were used to evaluate the ODE residuals.

The model parameters were chosen according to the simulation study, and the numerical solution of the compartmental ODE system served as the reference data. The loss function was minimized to a tolerance of 0.01, ensuring a high-fidelity approximation of the system dynamics.

Figures 10–16 show the time evolution of the infected, treatment, and recovered compartments, respectively, under different fractal-fractional parameter sets (ρ, Θ) . Markers represent the ODE data, dashed lines indicate the PINN-predicted values, and the shaded region corresponds to a $\pm 5\%$ prediction range.

The effect of the network architecture was also evaluated. A smaller number of neurons can limit the representational capacity, producing a coarse approximation of rapid transitions in infection dynamics. With 50 neurons per hidden layer, the network effectively captured the non-linear dynamics while maintaining reasonable computational cost. The mean squared error between the predicted and ODE data confirmed that the PINN accurately reproduces the compartmental dynamics for the fractal-fractional model.

Overall, the results demonstrate that the forward FF-PINN accurately reproduces the dynamics of the system, validating both the numerical solution and the efficacy of the neural network approach in modeling complex fractional-order epidemic systems.

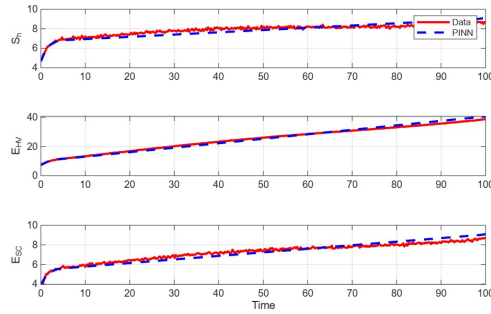


FIGURE 10. Numerical solution of the proposed model obtained via forward PINNs, illustrating the treatment dynamics of the infected population.

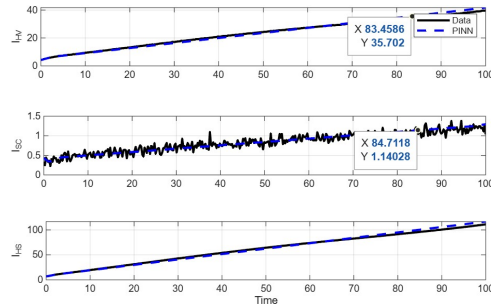


FIGURE 11. Numerical solution of the proposed model obtained via forward PINNs, illustrating the infected population dynamics.

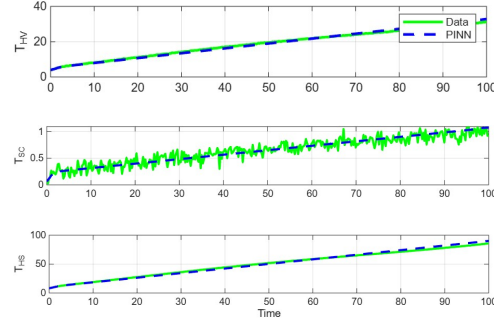


FIGURE 12. Numerical solution of the proposed model obtained via forward PINNs, illustrating the susceptible and exposed dynamics.

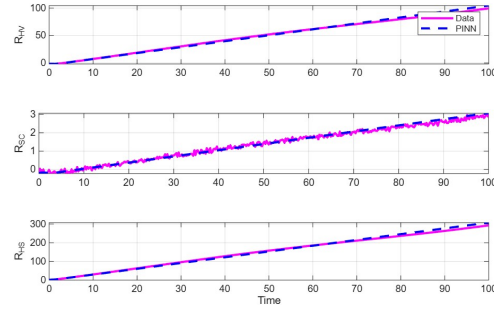


FIGURE 13. Numerical solution of the proposed model obtained via forward PINNs, illustrating the recovery dynamics of the population.

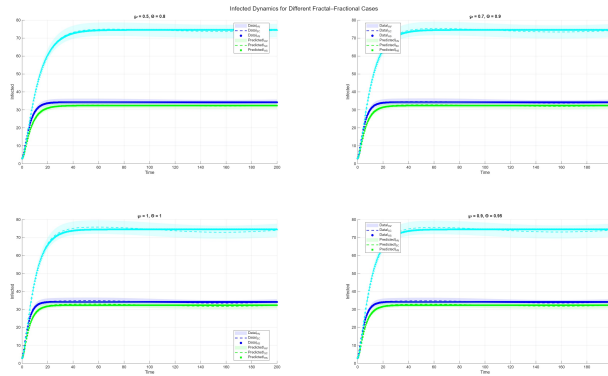


FIGURE 14. FF-PINNs prediction for infected compartments (I_{HV} , I_{SC} , I_{ES}) with hidden dim = 50 and absolute error between data and prediction ($\phi = 0.5$, $\Theta = 0.8$).

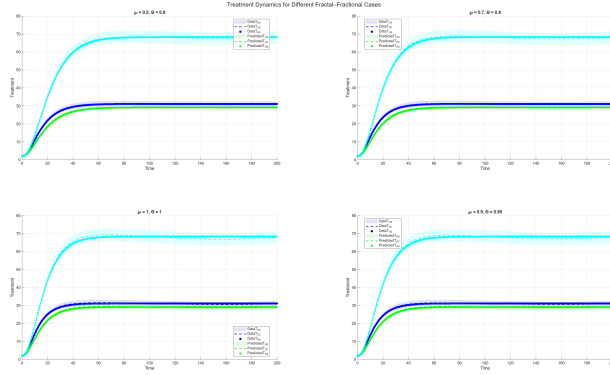


FIGURE 15. FF-PINNs prediction for treatment compartments (T_{HV} , T_{SC} , T_{HS}) with hidden dim = 50 and absolute error between data and prediction ($\wp = 0.5$, $\Theta = 0.8$).

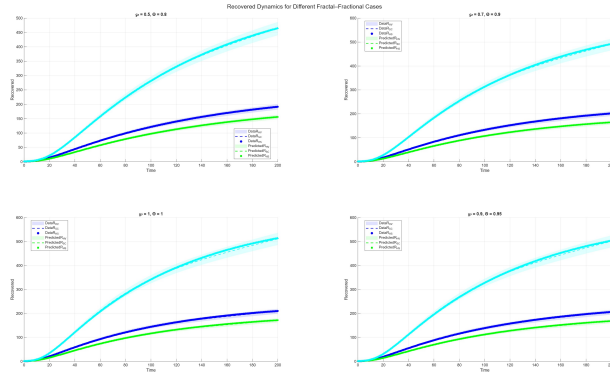


FIGURE 16. FF-PINNs prediction for recovered compartments (R_{HV} , R_{SC} , R_{HS}) with hidden dim = 50 and absolute error between data and prediction ($\wp = 0.5$, $\Theta = 0.8$).

9. CONCLUSION

In this study, a fractal–fractional model was developed to investigate the co-infection dynamics of Seoul virus and *Streptococcus pyogenes*. The use of the Atangana–Baleanu–Caputo fractal–fractional operator allows the model to account for memory effects and the irregular features that may arise in the transmission and progression of infectious diseases. The mathematical analysis establishes the existence and uniqueness of the model solutions and examines their stability properties, while the basic reproduction number provides a threshold measure for understanding the potential spread and persistence of the infections. To obtain numerical

solutions, a Physics-Informed Neural Network was incorporated into the proposed framework, with the governing equations embedded directly into the learning process. The numerical results demonstrate the capability of the PINN approach to approximate the solutions of the fractal–fractional system without depending entirely on conventional numerical discretization or extensive training data. Overall, the proposed framework combines mechanistic epidemiological modeling with physics-informed computational learning and provides a flexible approach for studying co-infection systems involving memory and heterogeneous transmission effects. The methodology can also be adapted to other infectious-disease models and may be useful for investigating alternative transmission scenarios and intervention strategies.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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