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Research Article

Nonlinear Fractional Stochastic Delay Modeling and Computational Analysis of Herpes Simplex Virus Type II Dynamics

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Abstract: Herpes simplex virus (HSV) is a widespread infection responsible for painful blisters and ulcers. According to the World Health Organization, approximately 519.5 million people aged 15-49 years (13.3%) worldwide are infected with herpes simplex virus type II (HSV-II), the primary cause of genital herpes. In this study, we develop a nonlinear stochastic fractional delay differential equation (SFDDE) model to describe the transmission dynamics of HSV-II in a human population. The population is divided into susceptible $S(t)$, exposed $E(t)$, asymptomatic $A(t)$, symptomatic $I(t)$, HSV-infected $H(t)$, and recovered $R(t)$compartments. The model's fundamental properties, including existence, uniqueness, positivity, and boundedness of solutions, are established. Local and global stability analyses are conducted around the HSV-free and HSV-present equilibrium points, and the basic reproduction number is derived using the next-generation matrix method along with sensitivity analysis. Numerical simulations based on a stochastic nonstandard finite difference (NSFD) scheme confirm the theoretical results and demonstrate the stability of the proposed model. These findings highlight the importance of nonlinear fractional stochastic modeling in understanding and controlling HSV-II transmission dynamics.

Keywords: Nonlinear model, dynamical analysis, stability and sensitivity analysis, Numerical simulation, Results.
Mathematics Subject Classification: 65M06; 65M12; 34K05; 34K20; 35K55

1. Introduction

In [1], the authors provided the analysis for transmission of Herpes Simplex Virus II with deterministic model of nonlinear differential equations.
In [2], the authors analyzed different types of viruses like HSV 1 and HSV 2 with using the world health organization (WHO) reported cases. In [3], the authors demonstrated the Aids and HSV-2 infection and also analyses the dynamics and relation between co-infection. In [4], the authors stated different numerical schemes for the control of HSV disease in human population and make more realistic strategies for their dynamics. In [5], the authors exhibited the advanced mathematical model construction for the Herpes disease which vastly spread in humans. In [6], the authors addressed that from 2000 to 2020 five priority areas from a WHO workshop were identified. Data on the author's country, gender, authorship position, and funding source were gathered through manuscript reviews and online searches. The data was analyzed using IBM SPSS V.26. In [7], the authors analyzed HSV-1 and HSV-2 infections to find differences based on gender at birth, age, and site of infection. Investigated the influence of comorbidities and pregnancy on infection type. Performed detailed analysis of data to reveal these differences. In [8], the authors provided a systematic search of MEDLINE, CINAHL, Global Health, and Cochrane databases (2000–2020) was conducted to identify the relevant English-language studies coming from LMICs. Two researchers independently screened and extracted predefined variables, organizing data into Excel. Analysis was carried out using IBM SPSS V.26. In [9], The authors evaluated immunocompetent adults who were diagnosed with meningitis at a tertiary care hospital in Korea. The study covered the period between 2016 and 2018. Data analysis was done on clinical patterns and outcomes. In [10], the authors carried out a retrospective analysis on 21,210, 49,494, and 32,457 outpatients and inpatients between 1 day to 17 years old. Patients had been subjected to nucleic acid testing for HSV-2, EBV, and CMV. Study duration from January 2018 to December 2023. In [11], the authors assessed Cebus apella (C. apella), a New World primate, using a genital infection model for HSV-2. All four animals' vaginal swabs were used to cultivate HSV-2 for nine to fourteen days after intravaginal inoculation. All the monkeys were initially seronegative for HSV-2. In [12], the authors collected a total of 145 individuals with quantified genital herpes severity, and their HSV genital shedding rate was measured. In [13], the authors examined the evidence of disease transition within individuals and make control strategies for the HSV-II. In [14], the authors provided the mathematical model with vaccinated parameter for the control of HSV-II. In [15], the authors found HSV-2 UL24 is very potent in suppressing the activation of the IFN-β promoter. It results in drastically decreased levels of IFN-β at both mRNA and protein levels. Results demonstrate the inhibitory function of UL24 in the IFN-β signaling pathway. In [16], the authors founded Chemical and physical stress intermittently reactivate latent viruses in the dorsal root ganglia (DRG). This results in recurrent viral shedding in the genital mucosal epithelium. As a consequence, symptomatic patients

suffer episodes of genital herpes. In [17], the authors adapted a dynamic HIV transmission model for South Africa, incorporating HSV-2 to model its synergistic impact on HIV. Assessed the implications of (i) cohort vaccination of 9-year-olds with a prophylactic vaccine to reduce susceptibility to HSV-2, and (ii) therapeutic vaccination of symptomatically HSV-2 infected subjects to reduce HSV shedding. In [18], the authors addressed an optimal control problem applied to a coinfection model of HIV and HSV-2. The model is set up by formulating a system of ordinary differential equations. It delves into the management and control dynamics of this coinfection. In [19], the authors utilized probability trees to model the natural history of genital herpes (GH) caused by HSV-1 and HSV-2 infections among those aged 18–49 years. Insights into infection dynamics and disease outcomes were thereby offered. In [20], the authors adhered to the systematic review of the Cochrane and PRISMA guidelines, which considered all publications from both the Chinese and English bibliographic systems. All sources that were considered were published up to March 18th, 2023. In [24], the authors studied a mathematical model of tumor development and therapy utilizing virotherapy. In [28-29], the authors conducted numerical studies on a fractional-order nonlinear SIR–SI model for dengue fever epidemics and a fuzzy fractional human liver model using a novel double-parametric approach.

The first-ever HSV-II model has been developed in this study which takes into account fractional memory effects, stochastic perturbations, and explicit time delays all at the same time. The model is the result of rigorous analytical results and a stochastic NSFD scheme that preserves the structure of the original model. The new insight provided by this integrated framework to long-term and uncertain HSV-II dynamics is more than that of the existing models. By integrating temporal delays, fractional calculus, and stochastic processes, the stochastic delayed methodology helps scientists create more accurate models of complicated systems.

This paper is organized as follows. Section 1 presents a focused review of the literature on Herpes Simplex Virus–II, including its epidemiology and underlying biological mechanisms. Section 2 describes the formulation of the proposed model and provides analytical results, including existence, uniqueness, positivity, boundedness, the basic reproduction number, local and global stability, and sensitivity analysis. Section 3 introduces the stochastic nonstandard finite difference (NSFD) numerical scheme and discusses its qualitative properties. Section 4 presents graphical simulations illustrating the dynamical behavior of the model along with their biological interpretation and real-world significance. Finally, Section 5 summarizes the main findings, highlights the contributions of the study, and outlines potential directions for future research.

2 Model Formulation

This paper presents the dynamical system of ordinary differential equations in formulating the dynamics of the human population in the presence of Human

Immunodeficiency Virus (HIV) treatment. The present study considered a model that comprises six compartments of the human population like susceptible individuals $S(t)$, exposed individuals $E(t)$, asymptomatic individuals $A(t)$, symptomatic Individuals $I(t)$, Herpes Simplex Virus-II Individuals $H(t)$ and recovered individuals $R(t)$, respectively.

$$N(t) = S(t) + E(t) + A(t) + I(t) + H(t) + R(t).$$

This recruit susceptible into the population at a rate Λ. Individuals in the susceptible sub-compartment move to the exposed sub-compartment at a per capita rate η of becoming infectious (we recall that $1/\eta$ is approximately the length of the latent period). Exposed individuals progress to the symptomatic sub-compartment with probability ρ, and to the asymptomatic sub-compartment with probability $(1 - \rho)$. Asymptomatic individuals are usually considered to be infectious, though at a lower rate qA.

The susceptible individuals are acquiring HSV-II infection with the force of infection $\beta[I + qA]$ where, β is the contact rate and q is the transmission coefficient for the asymptomatic individuals. Here, q denotes the *relative transmission rate* of asymptomatic individuals compared to symptomatic individuals, accounting for reduced or enhanced infectiousness of asymptomatic cases. If $q > 1$ then, the asymptomatic infect susceptible is more likely than infective. If $q = 1$, then asymptomatic and infective have an equal chance to infect the susceptible but if $q < 1$ then, the infective has a good chance to infect the susceptible than the asymptomatic. Some of the asymptomatic and symptomatic individuals progress to Herpes simplex virus-II at a rate φ, ϕ respectively and others recover naturally through the body's immune system at a rate γ,θ respectively. These herpes simplex virus-II individuals get treated at the rate of δ and move to the subcompartment of recovery. Here also, the recovered individuals might lose their immunity and recover to the susceptible

sub-compartment ω rate. The individual will suffer from death because of diseases when it attains the HSV-II stage at ξ rate. For all these sub compartments the mortality rate of individuals is noted as μ. All parameters and the variables taken for this model are kept to be positive.
The system of nonlinear stochastic fractional delay differential equations is described as follows:

$$ {}_0^cD_t^\alpha[S] = \Lambda^\alpha - \beta^\alpha S(t-\tau)(I(t-\tau) + q^\alpha A(t-\tau))e^{-\mu^\alpha \tau} - \mu^\alpha S + \omega^\alpha R + \sigma_1 S d(B). \quad (1) $$

$$ {}_0^cD_t^\alpha[E] = \beta^\alpha S(t-\tau)(I(t-\tau) + q^\alpha A(t-\tau))e^{-\mu^\alpha \tau} - (\eta^\alpha + \mu^\alpha)E + \sigma_2 E d(B). \quad (2) $$

$$ {}_0^cD_t^\alpha[A] = (1-\rho^\alpha)\eta^\alpha E - (\varphi^\alpha + \gamma^\alpha + \mu^\alpha)A + \sigma_3 A d(B). \quad (3) $$

$$ {}_0^cD_t^\alpha[I] = \rho^\alpha \eta^\alpha E - (\phi^\alpha + \theta^\alpha + \mu^\alpha)I + \sigma_4 I d(B). \quad (4) $$

$$ {}_0^cD_t^\alpha[H] = \varphi^\alpha A + \phi^\alpha I - (\delta^\alpha + \xi^\alpha + \mu^\alpha)H + \sigma_5 H d(B). \quad (5) $$

$$ {}_0^cD_t^\alpha[R] = \gamma^\alpha A + \theta^\alpha I + \delta^\alpha H - (\omega^\alpha + \mu^\alpha)R + \sigma_6 R d(B). \quad (6) $$

under the initials;

$$ S(0) \geq 0, E(0) \geq 0, A(0) \geq 0, I(0) \geq 0, H(0) \geq 0, R(0) \geq 0, \; t \geq 0, \tau < t. $$

Where $\sigma_i; (i = 1,2,3,4,5,6)$ fluctuation and $B(t)$ is the Brownian motion with time delay τ.

Preliminaries: The basic definitions are as follows;

Definition 1: For a function $q \in C_n$, the Caputo fractional derivative of order $\alpha \in (n-1, n), n \in N$ is;

$$ {}_0^cD_t^\alpha q(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t \frac{q^n(T)dT}{(t-T)^{\alpha+1-n}}. $$

Definition 2: For the function $q(t)$, the expression describes the equivalent fractional integral with order $\alpha > 0$.

$$I_t^{\alpha} q(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t - T)^{\alpha-1} q(T) dT.$$

Where "Γ" is the gamma function displayed.

Definition 3: A function $f(t, y)$ satisfies a Lipshitz condition in the variable y on set $D \subset R^2$ if a constant $L > 0$ exists with

$$\|f(t, y_1) - f(t, y_2)\| \leq L\|y_1 - y_2\|,$$

whenever (t, y_1), $(t, y_2)$are in D, L is Lipshitz constant.

2.1 Existence and Uniqueness

In this part, the existence and uniqueness of the model (1-6) are established with $\sigma_i = 0; i = 1,2,3,4,5,6$. For this,

$$S(t) = S_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - s)^{\alpha-1} \hbar_1(s, S) ds. \quad (7)$$

$$E(t) = E_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - s)^{\alpha-1} \hbar_2(s, E) ds. \quad (8)$$

$$A(t) = A_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - s)^{\alpha-1} \hbar_3(s, A) ds. \quad (9)$$

$$I(t) = I_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - s)^{\alpha-1} \hbar_4(s, I) ds. \quad (10)$$

$$H(t) = H_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - s)^{\alpha-1} \hbar_5(s, H) ds. \quad (11)$$

$$R(t) = R_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t - s)^{\alpha-1} \hbar_6(s, R) ds. \quad (12)$$

The functions listed in system (7–12) under the integral are

$$\hbar_1(t, S) = \Lambda^{\alpha} - \beta^{\alpha} S(I + q^{\alpha} A) e^{-\mu^{\alpha} \tau} - \mu^{\alpha} S + \omega^{\alpha} R. \quad (13)$$

$$\hbar_2(t, E) = \beta^{\alpha} S(I + q^{\alpha} A) e^{-\mu^{\alpha} \tau} - (\eta^{\alpha} + \mu^{\alpha}) E. \quad (14)$$

$$\hbar_3(t, A) = (1 - \rho^{\alpha}) \eta^{\alpha} E - (\varphi^{\alpha} + \gamma^{\alpha} + \mu^{\alpha}) A. \quad (15)$$

$$\hbar_4(t, I) = \rho^{\alpha} \eta^{\alpha} E - (\phi^{\alpha} + \theta^{\alpha} + \mu^{\alpha}) I. \quad (16)$$

$$\hbar_5(t, H) = \varphi^{\alpha}A + \phi^{\alpha}I - (\delta^{\alpha} + \xi^{\alpha} + \mu^{\alpha})H. \quad (17)$$

$$\hbar_6(t, R) = \gamma^{\alpha}A + \theta^{\alpha}I + \delta^{\alpha}H - (\omega^{\alpha} + \mu^{\alpha})R. \quad (18)$$

Let $X(t) = (S(t),E(t),A(t),I(t),H(t),R(t))$ and define the operator T on the Banach space $C([-\tau,T],R^6)$ with the supremum norm $\| X \|_{\infty} = \sup_{t\in[-\tau,T]} \| X(t) \|$ by $(TX)(t) = X(0) + \frac{1}{\Gamma(\alpha)}\int_0^t (t - s)^{\alpha-1} h(s,X(s),X(s - \tau))\, ds$, where $h = (\hbar_1,\hbar_2,\hbar_3,\hbar_4,\hbar_5,\hbar_6)$. Existence and uniqueness follow if h is Lipschitz in X on a bounded region G and the contraction condition $\frac{L T^{\alpha}}{\Gamma(\alpha+1)} < 1$ holds for some $T > 0$, where L is the Lipschitz constant.

Furthermore, it is assumed that E_1, ε_2, ε_3, ε_4,ε_5 and ε_6 exist as positive constants and that $S(t)$, $E(t)$,$A(t)$,$I(t)$,$H(t)$ and $R(t)$ are non-negative limiting functions.
Such that

$$\|S(t)\| \le \varepsilon_1, \|E(t)\| \le \varepsilon_2,\|A(t)\| \le \varepsilon_3,\|I(t)\| \le \varepsilon_4,,\|H(t)\| \le \varepsilon_5 \text{ and },\|R(t)\| \le \varepsilon_6.$$

Theorem 1: Show that Lipschitz conditions fulfill by kernel $\hbar_i;(i = 1,2,3,4,5,6)$ for each model (1-6) with $\sigma_i = 0;i = 1,2,3,4,5,6$, when $0 \le W = \max\{1,2,3,4,5,6\} < 1$.

Proof: For a detailed proof, see Appendix A, where Equations (19)-(42) are derived and used in the proof.

Theorem 2: Prove that: (i) The system of equations (25-30) has a definite uniform function. (ii) if there exists at least a $t_* > 1$ such that $\frac{\xi_1}{\Gamma(\alpha)} < 1$. For the model system with $\sigma_i = 0;i = 1,2,3,4,5,6$ there exists at least a solution if $\frac{\xi_1}{\Gamma(\alpha)} < 1$ for $i = 1,2,3,4,5,6$.

Proof: For a detailed proof, see Appendix B, where Equations (43)-(64) are derived and used in the proof.

Theorem 3: The system (1-6) demonstrates the uniqueness with $\sigma_i = 0$; $(i = 1,2,3,4,5,6)$ if $\left(1 - \frac{\xi_1}{\Gamma(\alpha)}(t)\right) > 0$.

Proof: For a detailed proof, see Appendix C, where Equations (65)-(72) are derived and used in the proof.

2.2 Positivity and Boundedness

Theorem 4: Assume the initial history functions satisfy $(S(t),E(t),A(t),I(t),H(t),R(t)) \geq 0, t \in [-\tau,0]$, and all parameters are nonnegative. For $\sigma_i = 0$, $(i = 1,\ldots,6)$, the solution of system (1-6) remains nonnegative for all $t > 0$, i.e., $(S(t),E(t),A(t),I(t),H(t),R(t)) \in R_+^6, \forall t > 0$.

Proof. Using the Caputo fractional formulation, each state variable admits the equivalent Volterra integral form. For example,

$$S(t) = S(0) + \frac{1}{\Gamma(\alpha)}\int_0^t (t - s)^{\alpha-1} F_S(s,X(s),X(s - \tau))\, ds,$$

and similarly, for E,A,I,H,R, where $X = (S,E,A,I,H,R)$ and F_S denotes the right-hand side of (1). Now suppose (for contradiction) that some component becomes negative. Let $t^* > 0$ be the first time such that at least one component hits zero and then attempts to cross into the negative region. At time t^*, that component equals zero, while all other components remain nonnegative by minimality of t^*, and the delayed terms $X(t^* - \tau)$ are also nonnegative because the history is nonnegative.

On each boundary face, the corresponding right-hand side is nonnegative under nonnegative states. In particular, evaluating the drift terms on the boundary yields:

- if $S(t^*) = 0$, then $F_S(t^*) = \Lambda^\alpha + \omega^\alpha R(t^*) \geq 0$;
- if $E(t^*) = 0$, then $F_E(t^*) = \beta^\alpha S(t^* - \tau)(I(t^* - \tau) + q^\alpha A(t^* - \tau))e^{-\mu^\alpha \tau} \geq 0$;
- if $A(t^*) = 0$, then $F_A(t^*) = (1 - \rho^\alpha)\eta^\alpha E(t^*) \geq 0$;
- if $I(t^*) = 0$, then $F_I(t^*) = \rho^\alpha \eta^\alpha E(t^*) \geq 0$;

- if $H(t^*) = 0$, then $F_H(t^*) = \phi^\alpha A(t^*) + \varphi^\alpha I(t^*) \geq 0$;
- if $R(t^*) = 0$, then $F_R(t^*) = \gamma^\alpha A(t^*) + \theta^\alpha I(t^*) + \delta^\alpha H(t^*) \geq 0$.

Therefore, at the first hitting time t^*, the Caputo integral representation implies the solution cannot decrease past zero (fractional comparison), which contradicts the assumption that a component crosses into negative values. Hence all components remain nonnegative for all $t > 0$.

Lemma 1: (**Fractional Grönwall Inequality).** Let $u(t)$ be a nonnegative, continuous function on $[0,T]$and assume that

$$u(t) \leq a + b\int_0^t (t-s)^{\alpha-1} u(s)\, ds, 0 < \alpha \leq 1,$$

where $a,b \geq 0$are constants. Then

$$u(t) \leq a\, E_\alpha(b\, t^\alpha), t \in [0,T],$$

where E_α denotes the Mittag-Leffler function

Theorem 5: The system (1-6) under the nonnegative initial conditions is bounded for all $t \geq 0$ when $\sigma_i = 0$, $i = 1,2,3,4,5,6$, and the solution lies in the feasible region

$$G = \left\{(S, E, A, I, H, R) \in R^{+6}; 0 < N \leq \frac{\Lambda^\alpha}{\mu^\alpha}, \forall t \geq 0, \tau < t\right\}.$$

Proof. Let $N(t) = S(t) + E(t) + A(t) + I(t) + H(t) + R(t)$. By summing equations (1-6) and using the nonnegativity of all state variables and parameters, we obtain

$$D_t^\alpha N(t) \leq \Lambda^\alpha - \mu^\alpha N(t).$$

The associated comparison equation

$$D_t^\alpha y(t) = \Lambda^\alpha - \mu^\alpha y(t), y(0) = N(0), \quad (73)$$

has the explicit solution

$$y(t) = \frac{\Lambda^\alpha}{\mu^\alpha} + (N(0) - \frac{\Lambda^\alpha}{\mu^\alpha}) E_\alpha(-\mu^\alpha t^\alpha). \quad (74)$$

By the fractional comparison principle and Lemma 1, it follows that

$$N(t) \leq y(t), \forall\, t \geq 0.$$

Since $E_\alpha(-\mu^\alpha t^\alpha) \to 0$ as $t \to \infty$, we conclude that

$$\limsup_{t\to\infty} N(t) \leq \frac{\Lambda^\alpha}{\mu^\alpha}. \quad (75)$$

Therefore, all solutions of system (1-6) remain bounded and lie in the feasible region G.

2.3. Model Equilibria and Reproduction Number

This part will provide the model equilibria for system (1-6). Therefore,

HSV-Free Equilibrium $= HSVFE = C^0 = (S_0,E_0,A_0,I_0,H_0,R_0) = \left(\frac{\Lambda^\alpha}{\mu^\alpha},0,0,0,0,0\right)$. (76)

HSV-Present Equilibrium $= HSVPE = C^* = (S^*,E^*,A^*,I^*,H^*,R^*)$. (77)

$$S^* = \frac{\Lambda^\alpha+\omega^\alpha R^*}{\beta^\alpha(I^*+q^\alpha A^*)e^{-\mu^\alpha\tau}+\mu^\alpha},\ E^* = \frac{\beta^\alpha S^*(I^*+q^\alpha A^*)e^{-\mu^\alpha\tau}}{(\eta^\alpha+\mu^\alpha)},\ A^* = \frac{(1-\rho^\alpha)\eta^\alpha E^*}{(\varphi^\alpha+\gamma^\alpha+\mu^\alpha)},$$

$$I^* = \frac{\rho^\alpha\eta^\alpha E^*}{(\phi^\alpha+\theta^\alpha+\mu^\alpha)},\ H^* = \frac{\varphi^\alpha A^*+\phi^\alpha I^*}{(\delta^\alpha+\xi^\alpha+\mu^\alpha)},\ R^* = \frac{\gamma^\alpha A^*+\theta^\alpha I^*+\delta^\alpha H^*}{(\omega^\alpha+\mu^\alpha)}.$$

By using Next generation method to get reproduction number for system (1-6) is as follows:

$$R_0 = \frac{\beta^\alpha\Lambda^\alpha\eta^\alpha[q^\alpha(1-\rho^\alpha)(\phi^\alpha+\theta^\alpha+\mu^\alpha)+\rho^\alpha(\varphi^\alpha+\gamma^\alpha+\mu^\alpha)]e^{-\mu^\alpha\tau}}{\mu^\alpha(\eta^\alpha+\mu^\alpha)(\varphi^\alpha+\gamma^\alpha+\mu^\alpha)(\phi^\alpha+\theta^\alpha+\mu^\alpha)}.$$ (78)

The basic reproduction number R_0 plays a threshold role in determining the existence and stability of the equilibrium points. When $R_0 < 1$, the HSV-free equilibrium is locally (and globally) asymptotically stable, indicating that HSV-II cannot invade the population and the infection dies out. When $R_0 > 1$, the HSV-free equilibrium becomes unstable and a unique HSV-present equilibrium emerges, corresponding to persistent disease transmission. Thus, R_0 governs the qualitative transition between disease extinction and persistence in the proposed model.

2.4 Local Stability

The local stability analysis is carried out within the framework of Caputo fractional-order systems. For fractional differential equations of order $0 < \alpha \leq 1$, an equilibrium point is locally asymptotically stable if all eigenvalues λ_i of the corresponding Jacobian matrix satisfy $|\arg(\lambda_i)| > \frac{\alpha\pi}{2}$. This criterion generalizes the classical ODE stability condition and accounts for the memory effects inherent in fractional-order models. Using this fractional stability criterion, we analyze the HSV-free and HSV-present equilibrium points of system (1-6).

Theorem 6: For the Caputo fractional-order system (1–6) with $0 < \alpha \leq 1$ and $\sigma_i = 0 (i = 1,2,3,4,5,6)$, the HSV-free equilibrium C^0 is locally asymptotically stable

if $R_0 < 1$. Conversely, C^0becomes unstable when $R_0 > 1$.

Proof: Linearizing system (1–6) around the HSV-free equilibrium C^0yields the corresponding Jacobian matrix $J(C^0)$. For a Caputo fractional-order system of order $0 < \alpha \leq 1$, an equilibrium point is locally asymptotically stable if all eigenvalues $\lambda_i$of the Jacobian matrix satisfy the fractional-order stability condition

$$|\arg(\lambda_i)| > \frac{\alpha\pi}{2}.$$

Applying this criterion to $J(C^0)$, it follows that all eigenvalues satisfy the above condition when the basic reproduction number $R_0 < 1$, ensuring local asymptotic stability of the HSV-free equilibrium. Conversely, when $R_0 > 1$, at least one eigenvalue violates the fractional stability condition, and the HSV-free equilibrium becomes unstable. Hence, under the fractional-order framework, the stability of $C^0$is completely governed by the threshold parameter R_0.

Theorem 7: For the Caputo fractional-order system (1–6) with $0 < \alpha \leq 1$and $\sigma_i = 0 (i = 1,2,3,4,5,6)$, the HSV-present equilibrium C^*exists when $R_0 > 1$and is locally asymptotically stable provided that all eigenvalues $\lambda_i$of the Jacobian matrix evaluated at C^*satisfy

$$|\arg(\lambda_i)| > \frac{\alpha\pi}{2}.$$

Proof: After linearizing Jacobian matrix of the system (1-6), the 6^{th} order polynomial follows as

$$\lambda^6 + A_5\lambda^5 + A_4\lambda^4 + A_3\lambda^3 + A_2\lambda^2 + A_1\lambda + A_0 = 0.$$

Here.

$$A_5 = (a_7 + a_9) + (a_5 + a_{10}) + (a_1 + a_{11})$$

$$A_4 = a_9a_7 + a_5a_{10} + (a_5 + a_{10})(a_9 + a_7) - a_2a_7 - a_2a_6 - a_3a_8 + (a_1 + a_{11})$$

$$[(a_7 + a_9)(a_5 + a_{10})] + a_1a_{11}.$$

$$A_3 = (a_7a_9)(a_5 + a_{10}) + (a_7 + a_9)(a_5a_{10}) - (a_9 + a_{10})(a_2a_6) - (a_7 + a_{10})(a_3a_8) +$$

$$(a_1 + a_{11})[a_9a_7 + a_5a_{10} + (a_5 + a_{10})(a_9 + a_7) - a_2a_7 - a_2a_6 - a_3a_8] + a_1a_{11}$$

$$[(a_7 + a_9) + (a_5 + a_{10}) + (a_1 + a_{11})] - a_4(a_2a_6 + a_3a_8).$$

$$A_2 = (a_1 + a_{11})$$

$$[(a_7a_9)(a_5 + a_{10}) + (a_7 + a_9)(a_5a_{10}) - (a_9 + a_{10})(a_2a_6) - (a_7 + a_{10})(a_3a_8) + (a_1 + a_{11})]$$

$$+ a_1a_{11}[a_9a_7 + a_5a_{10} + (a_5 + a_{10})(a_9 + a_7) - a_2a_7 - a_2a_6 - a_3a_8] - a_4$$

$$(a_2a_6a_9 + a_3a_8a_7) - a_4(a_{10} + a_{11})(a_2a_6 + a_3a_8).$$

$$A_1 = a_9a_7a_5a_{10} - a_2a_6a_9a_{10} - a_3a_8a_{10}a_7 + (a_1 + a_{11})$$

$$[a_9a_7a_5a_{10} - a_2a_6a_9a_{10} - a_3a_8a_{10}a_7] + a_1a_{11}$$

$$[(a_7a_9)(a_5 + a_{10}) + (a_7 + a_9)(a_5a_{10}) - (a_9 + a_{10})(a_2a_6) - (a_7 + a_{10})(a_3a_8)] - a_4$$

$$(a_{10} + a_{11})(a_2a_6a_9 + a_3a_8a_7) - a_4a_{10}a_{11}(a_2a_6a_9 + a_3a_8a_7).$$

$$A_0 = a_1a_{11}[a_9a_7a_5a_{10} - a_2a_6a_9a_{10} - a_3a_8a_{10}a_7].$$

$$a_1 = \beta^{\alpha}S^*(I^* + q^{\alpha}A^*)e^{-\mu^{\alpha}\tau} + \mu^{\alpha},\ a_2 = \beta^{\alpha}S^*q^{\alpha}e^{-\mu^{\alpha}\tau},\ a_3 = \beta^{\alpha}S^*e^{-\mu^{\alpha}\tau},\ a_4 = \beta^{\alpha}$$

$$(I^* + q^{\alpha}A^*)e^{-\mu^{\alpha}\tau},\ a_5 = (\eta^{\alpha} + \mu^{\alpha}), a_6 = (1 - \rho^{\alpha})\eta^{\alpha},\ a_7 = (\varphi^{\alpha} + \gamma^{\alpha} + \mu^{\alpha}),\ a_8 = \rho^{\alpha}$$

$$\eta^{\alpha},\ a_9 = (\phi^{\alpha} + \theta^{\alpha} + \mu^{\alpha}),\ a_{10} = (\delta^{\alpha} + \xi^{\alpha} + \mu^{\alpha}),\ a_{11} = (\omega^{\alpha} + \mu^{\alpha}).$$

Since the 6th order polynomial with A_5, A_4, A_3, A_2, A_1 and A_0 are positive coefficient with $A_5A_4 > A_3$, $(A_5A_4 - A_3)(A_5A_2 - A_3A_4) > A_1A_5^2$, $A_5A_2 > A_3A_4$. So, by the Routh-Hurwitz Criterion satisfies when $R_0 > 1$. Hence C^* of the given system (1-6) is LAS.

2.5 Global Stability

In this part, we analyzed the stability of the model (1-6) at both equilibrium points like HSV-Free Equilibrium and HSV-Present Equilibrium point in the sense of global.

Theorem 8: The system (1-6) is GAS at C^0 when $R_0 < 1$ with $\sigma_i = 0$; $(i = 1,2,3,4,5,6)$.

Proof: Define the Lyapunov function $L: G \to R$,

$$L(t) = \left[S - S_0 - S_0 \log \frac{S}{S_0}\right] + E + A + I + H + R.$$

$$ {}_0^cD_t^\alpha L(t) = \left[\frac{S-S_0}{S}\right] {}_0^cD_t^\alpha S + {}_0^cD_t^\alpha E + {}_0^cD_t^\alpha A + {}_0^cD_t^\alpha I + {}_0^cD_t^\alpha H + {}_0^cD_t^\alpha R. $$

$$ {}_0^cD_t^\alpha L(t) = \left[\frac{S-S_0}{S}\right]\left(\Lambda^\alpha - \beta^\alpha S(I + q^\alpha A)e^{-\mu^\alpha \tau} - \mu^\alpha S + \omega^\alpha R\right) + $$

$$ \left(\beta^\alpha S(I + q^\alpha A)e^{-\mu^\alpha \tau} - (\eta^\alpha + \mu^\alpha)E\right) + ((1 - \rho^\alpha)\eta^\alpha E - (\varphi^\alpha + \gamma^\alpha + \mu^\alpha)A) + $$

$$ (\rho^\alpha \eta^\alpha E - (\phi^\alpha + \theta^\alpha + \mu^\alpha)I) + (\varphi^\alpha A + \phi^\alpha I - (\delta^\alpha + \xi^\alpha + \mu^\alpha)H) + $$

$$ (\gamma^\alpha A + \theta^\alpha I + \delta^\alpha H - (\omega^\alpha + \mu^\alpha)R). $$

$$ {}_0^cD_t^\alpha L(t) \le - (\Lambda^\alpha + \omega^\alpha R)\frac{(S-S_0)^2}{SS_0} - \mu^\alpha I\left(1 - \frac{\beta^\alpha S e^{-\mu^\alpha \tau}}{\mu^\alpha}\right) - \mu^\alpha A\left(1 - \frac{\beta^\alpha S q^\alpha e^{-\mu^\alpha \tau}}{\mu^\alpha}\right) - (\xi^\alpha + \mu^\alpha)H - $$

$$ (\omega^\alpha + \mu^\alpha)R. $$

This implies that ${}_0^cD_t^\alpha L(t) \le 0$ when $R_0 < 1$, and ${}_0^cD_t^\alpha L(t) = 0$ only at C^0. Therefore, by the fractional-order extension of LaSalle's invariance principle (for Caputo systems), the HSV-free equilibrium C^0 is globally asymptotically stable for $R_0 < 1$ when $\sigma_i = 0$.

Theorem 9: The system (1-6) is GAS at C^* when $R_0 > 1$ with $\sigma_i = 0; i = 1,2,3,4,5,6,$

Proof: Let the Lyapunov function $Z: G \to R$,

$$ Z = k_1\left(S - S^* - S^*\log\left(\frac{S}{S^*}\right)\right) + k_2\left(E - E^* - E^*\log\left(\frac{E}{E^*}\right)\right) + k_3\left(A - A^* - A^*\log\left(\frac{A}{A^*}\right)\right) + k_4 $$

$$ \left(I - I^* - I^*\log\left(\frac{I}{I^*}\right)\right) + k_5\left(H - H^* - H^*\log\left(\frac{H}{H^*}\right)\right) + k_6\left(R - R^* - R^*\log\left(\frac{R}{R^*}\right)\right). $$

$$ {}_0^cD_t^\alpha Z = k_1\left(\frac{S-S^*}{SS^*}\right){}_0^cD_t^\alpha S + k_2\left(\frac{E-E^*}{EE^*}\right){}_0^cD_t^\alpha E + k_3\left(\frac{A-A^*}{AA^*}\right){}_0^cD_t^\alpha A + k_4\left(\frac{I-I^*}{II^*}\right){}_0^cD_t^\alpha I + k_5\left(\frac{H-H^*}{HH^*}\right){}_0^cD_t^\alpha $$

$$ H + k_6\left(\frac{R-R^*}{RR^*}\right){}_0^cD_t^\alpha R. $$

$$ {}_0^cD_t^\alpha Z = - k_1(\Lambda^\alpha + \omega^\alpha R)\frac{(S-S^*)^2}{SS^*} - k_2\left(\beta^\alpha S(I + q^\alpha A)e^{-\mu^\alpha \tau}\right)\frac{(E-E^*)^2}{EE^*} - k_3((1 - \rho^\alpha)\eta^\alpha E)\frac{(A-A^*)^2}{AA^*} $$

$$ - k_4(\rho^\alpha \eta^\alpha E)\frac{(I-I^*)^2}{II^*} - k_5(\varphi^\alpha A + \phi^\alpha I)\frac{(H-H^*)^2}{HH^*} - k_6(\gamma^\alpha A + \theta^\alpha I + \delta^\alpha H)\frac{(R-R^*)^2}{RR^*}. $$

If we choose $k_i = 1$ where $(i = 1,2,3,4,5,6)$

$$ {}_0^cD_t^\alpha Z = - (\Lambda^\alpha + \omega^\alpha R)\frac{(S-S^*)^2}{SS^*} - \left(\beta^\alpha S(I + q^\alpha A)e^{-\mu^\alpha \tau}\right)\frac{(E-E^*)^2}{EE^*} - ((1 - \rho^\alpha)\eta^\alpha E)\frac{(A-A^*)^2}{AA^*} - $$

$$ (\rho^\alpha \eta^\alpha E)\frac{(I-I^*)^2}{II^*} - (\varphi^\alpha A + \phi^\alpha I)\frac{(H-H^*)^2}{HH^*} - (\gamma^\alpha A + \theta^\alpha I + \delta^\alpha H)\frac{(R-R^*)^2}{RR^*}. $$

Therefore, since ${}_0^C D_t^\alpha Z \le 0$ and ${}_0^C D_t^\alpha Z = 0$ if and only if $S = S^*, E = E^*, A = A^*, I = I^*, H = H^*, R = R^*$, it follows from the fractional LaSalle invariance principle for Caputo fractional systems that the HSV-present equilibrium C^* is globally asymptotically stable for $R_0 > 1$ when $\sigma_i = 0$.

2.6. Sensitivity Analysis

This part will cover the sensitivity of model parameter of R_0. We will identify the sensitive parameter indices in terms of space with respect to the reproduction number R_0.

$$V_{\Lambda^\alpha} = \frac{\Lambda^\alpha}{R_0} \times \frac{\partial R_0}{\partial \Lambda^\alpha} = 1 > 0,\ V_{\beta^\alpha} = \frac{\beta^\alpha}{R_0} \times \frac{\partial R_0}{\partial \beta^\alpha} = 1 > 0,\quad V_{\eta^\alpha} = \frac{\eta^\alpha}{R_0} \times \frac{\partial R_0}{\partial \eta^\alpha} = \frac{\mu^\alpha}{\mu^\alpha + \eta^\alpha} > 0,$$

$$V_{q^\alpha} = \frac{q^\alpha}{R_0} \times \frac{\partial R_0}{\partial q^\alpha} = \frac{q^\alpha[(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha) + \rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)]}{q^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha) + \rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)} > 0,$$

$$V_{\rho^\alpha} = \frac{\rho^\alpha}{R_0} \times \frac{\partial R_0}{\partial \rho^\alpha} = \frac{\rho^\alpha[-q^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha) + \rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)]}{q^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha) + \rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)} > 0,$$

$$V_{\gamma^\alpha} = \frac{\gamma^\alpha}{R_0} \times \frac{\partial R_0}{\partial \gamma^\alpha} = \frac{-q^\alpha\gamma^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha)}{(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)[q^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha) + \rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)]} < 0,$$

$$V_{\varphi^\alpha} = \frac{\varphi^\alpha}{R_0} \times \frac{\partial R_0}{\partial \varphi^\alpha} = -\frac{q^\alpha\varphi^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha)}{(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)[q^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha) + \rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)]} < 0\ ,$$

$$V_{\theta^\alpha} = \frac{\theta^\alpha}{R_0} \times \frac{\partial R_0}{\partial \theta^\alpha} = -\frac{\theta^\alpha\rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)}{(\phi^\alpha + \theta^\alpha + \mu^\alpha)[q^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha) + \rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)]} < 0,$$

$$V_{\phi^\alpha} = \frac{\phi^\alpha}{R_0} \times \frac{\partial R_0}{\partial \phi^\alpha} = -\frac{\theta^\alpha\rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)}{(\phi^\alpha + \theta^\alpha + \mu^\alpha)[q^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha) + \rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)]} < 0\ ,$$

$$V_{\mu^\alpha} = \frac{\mu^\alpha}{R_0} \times \frac{\partial R_0}{\partial \mu^\alpha} = -$$

$$\frac{(\mu^\alpha)^2[q^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha)^2[3(\mu^\alpha)^2 + 2(\varphi^\alpha + \gamma^\alpha + \eta^\alpha)\mu^\alpha] + \rho^\alpha[3(\mu^\alpha)^2 + 2(\phi^\alpha + \theta^\alpha + \eta^\alpha)\mu^\alpha + \eta^\alpha(\varphi^\alpha + \gamma^\alpha)](\varphi^\alpha + \gamma^\alpha + \mu^\alpha)^2}{[q^\alpha(1-\rho^\alpha)(\phi^\alpha + \theta^\alpha + \mu^\alpha) + \rho^\alpha(\varphi^\alpha + \gamma^\alpha + \mu^\alpha)]}$$

$$< 0\ ,$$

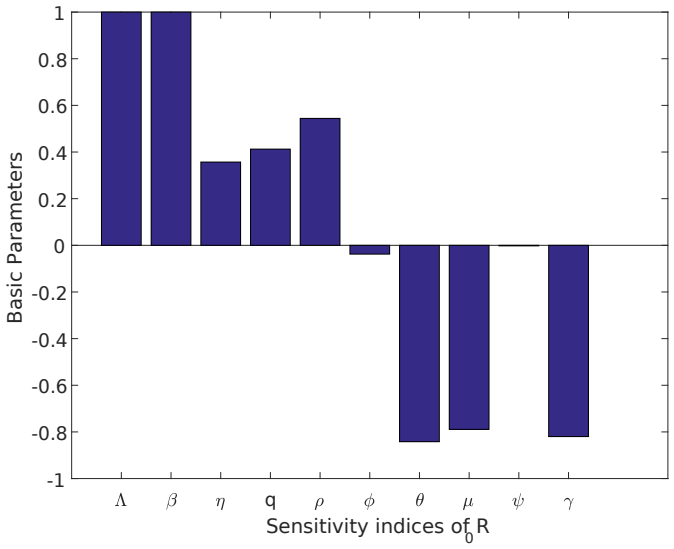

Fig. 1: Sensitivity indices of reproduction number (R_0).

Table 1: Values and signs of parameter's sensitivity:

Parameters	Signs	Values
Λ^{α}	Positive	1
β^{α}	Positive	1
η^{α}	Positive	0.357
q^{α}	Positive	0.4125
ρ^{α}	Positive	0.5442
ϕ^{α}	Negative	-0.0378
θ^{α}	Negative	-0.842
μ^{α}	Negative	-0.789
φ^{α}	Negative	-0.0023
γ^{α}	Negative	-0.82

To discuss the graphs for the Herpes simplex virus, which is in the middle of understanding HSV transmission dynamics. Fig. 1 shows the sensitivity indices of the reproduction number (R_0) concerning different model parameters. Positive sensitivity indices for the parameters mean that they are directly related to R_0, and an increase in these parameters increases the reproduction number and enhances the spread of the disease. On the other hand, negative sensitivity indices for the parameters indicate an inverse relationship, where an increase in these parameters decreases R_0 and reduces the transmission of the disease. Table 1 confirms these dynamics to be biologically relevant because the positively sensitive parameters likely would reflect factors like transmission rates or population interactions, while negative ones might reflect intervention-related factors such as recovery rate or vaccine effects. Understanding those relationships enables targeted control strategies since focus could be placed on the reduction of positively sensitive parameters along with enhancing the negatively sensitive ones that could help manage the HSV-II spread effectively.

3. Numerical Methods

We propose a generalized stochastic fractional technique for the solution of the

stochastic fractional-order system (1-6) like a NSFD scheme (see [21]). In all cases, t denotes the temporal step size.

3.1 Non-Standard Finite Difference (NSFD) Scheme

Consider the stochastic fractional delayed system (1-6) with Caputo derivative of order $0 < \alpha \leq 1$. Using the integral representation of the Caputo derivative, a general equation of the form $D_t^{\alpha} X(t) = F(X(t)) - L(X(t)) + \sigma X(t)\, dB(t)$ can be written in a time-stepping form where the deterministic increment is scaled by $\frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}$. Following the nonstandard finite difference (NSFD) rules of Mickens, we discretize the gain terms explicitly and treat the loss terms implicitly through a denominator in order to preserve positivity and boundedness for any step size. For the stochastic part, we apply the Euler-Maruyama approximation by replacing the Brownian increment with $\Delta B_i = B_i(t_{n+1}) - B_i(t_n) \sim N(0,\Delta t)$, which yields multiplicative noise terms of the form $\sigma_i X^n \Delta B_i$. Implementing these principles componentwise for S,E,A,I,H,R, and evaluating the delayed mortality survival factor $e^{-\mu^{\alpha}\tau}$ consistently, leads to the stochastic fractional NSFD scheme given by Eqs. (79)-(84).

$$S^{n+1} = \frac{S^n + \frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}(\Lambda^{\alpha} + \omega^{\alpha} R^n + \sigma_1 S^n \Delta B_1)}{1 + \frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}\left(\beta^{\alpha}(I^n + q^{\alpha} A^n) e^{-\mu^{\alpha}\tau} + \mu^{\alpha}\right)} \quad (79)$$

$$E^{n+1} = \frac{E^n + \frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}\left(\beta^{\alpha} S^n (I^n + q^{\alpha} A^n) e^{-\mu^{\alpha}\tau} + \sigma_2 E^n \Delta B_2\right)}{1 + \frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}(\eta^{\alpha} + \mu^{\alpha})} \quad (80)$$

$$A^{n+1} = \frac{A^n + \frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}((1-\rho^{\alpha})\eta^{\alpha} E^n + \sigma_3 A^n \Delta B_3)}{1 + \frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}(\varphi^{\alpha} + \gamma^{\alpha} + \mu^{\alpha})} \quad (81)$$

$$I^{n+1} = \frac{I^n + \frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}(\rho^{\alpha}\eta^{\alpha} E^n + \sigma_4 I^n \Delta B_4)}{1 + \frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}(\phi^{\alpha} + \theta^{\alpha} + \mu^{\alpha})} \quad (82)$$

$$H^{n+1} = \frac{H^n + \frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}(\varphi^{\alpha} A^n + \phi^{\alpha} I^n + \sigma_5 H^n \Delta B_5)}{1 + \frac{\Delta t^{\alpha}}{\Gamma(\alpha+1)}(\delta^{\alpha} + \xi^{\alpha} + \mu^{\alpha})} \quad (83)$$

$$R^{n+1} = \frac{R^n + \frac{\Delta t^\alpha}{\Gamma(\alpha+1)}(\gamma^\alpha A^n + \theta^\alpha I^n + \delta^\alpha H^n + \sigma_6 R^n \Delta B_6)}{1 + \frac{\Delta t^\alpha}{\Gamma(\alpha+1)}(\omega^\alpha + \mu^\alpha)} \quad (84)$$

Here $\Delta t > 0$ is the time step and ΔB_i are independent Brownian increments with mean zero and variance Δt. Furthermore, we investigate the properties of fractional NSFD of model (79-84).

Theorem 10: (Positivity). The system (79-84) satisfies the positivity under the initial conditions.

Proof: The model (79-84) is free from negative term. Thus, the initial condition also positive, as desired.

Theorem 11: (Boundedness). The system (79-84) satisfies the boundedness under the initial conditions for $K(n,\alpha) \geq 0$, such that $S^n, E^n, A^n, I^n, H^n, R^n \in [0, K(n,\alpha)]$, for each $n \in N$.

Proof: The sum of model (79-84) for the bondedness is as follows;

$$\left(1 + \frac{\Delta t^\alpha}{\Gamma(\alpha+1)}(\mu^\alpha)\right)S^{n+1} + \left(1 + \frac{\Delta t^\alpha}{\Gamma(\alpha+1)}(\mu^\alpha)\right)E^{n+1} + \left(1 + \frac{\Delta t^\alpha}{\Gamma(\alpha+1)}(\mu^\alpha)\right)A^{n+1} +$$

$$\left(1 + \frac{\Delta t^\alpha}{\Gamma(\alpha+1)}(\mu^\alpha)\right)I^{n+1} + \left(1 + \frac{\Delta t^\alpha}{\Gamma(\alpha+1)}(\xi^\alpha + \mu^\alpha)\right)H^{n+1} + \left(1 + \frac{\Delta t^\alpha}{\Gamma(\alpha+1)}(\mu^\alpha)\right)R^{n+1} \leq$$

$$(S^n + E^n + A^n + I^n + H^n + R^n) + \frac{\Delta t^\alpha}{\Gamma(\alpha+1)}(\Lambda^\alpha) + \frac{\Delta t^\alpha}{\Gamma(\alpha+1)}$$

$$(\sigma_1 S^n \Delta B_1 + \sigma_2 E^n \Delta B_2 + \sigma_3 A^n \Delta B_3 + \sigma_4 I^n \Delta B_4 + \sigma_5 H^n \Delta B_5 + \sigma_6 R^n \Delta B_6).$$

The proof relies on mathematical induction, with $K(n+1,\alpha)$ serving as the end point of sequence of identities and inequalities.

4 Graphical Simulation

In this part, we use graphical simulations for fractional model (1-6). To that end, the model parameters are fixed as given in Table 2 (see [1]). Moreover, in all simulations, $\Delta B_i \sim N(0,\Delta t)$ are taken as independent Wiener increments, and the noise intensities were fixed at $\sigma_i = 0.3$. To investigate the combined effect of memory and delay, simulations for different fractional orders were conducted. α while changing the delay parameter τ over various representative values. This allows comparison of disease dynamics under interchangeable enumerate all (α,τ) combinations and point out how explicit delays change the persistence effects induced by fractional memory. The numerical findings confirm that increasing τ generally delays peaks and prolongs persistence while decreasing

α enhances long-term memory effects.

Table: 2 Values of parameters.

Parameters	Values	Source [1]
Λ	0.0015	Assumed
μ	0.002	Assumed
β	10.68	Estimated
ϕ	0.004	[1]
φ	0.003	[1]
γ	0.058	[1]
θ	0.089	[1]
ω	0.09	[1]
ξ	0.001	[1]
η	0.9	Estimated
q	0.4	Fitted
δ	0.078	[1]
ρ	0.048	[1]
σ_i	$0 \leq i \leq 1$	Fitted

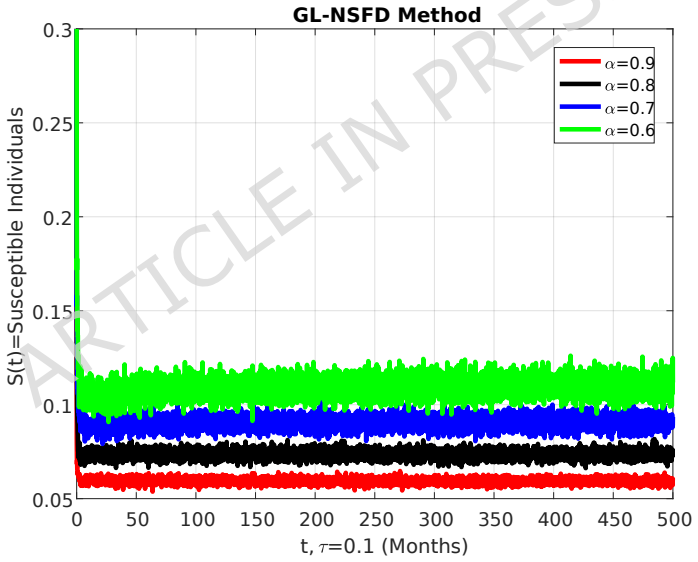

Fig. 2: Impact of "α" on S(t) at τ = 0.1.

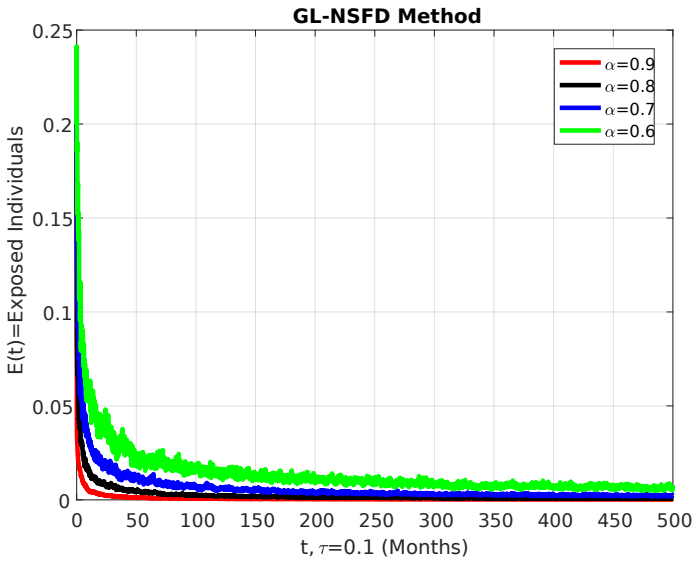

Fig. 3: Impact of "α" on E(t) at τ = 0.1.

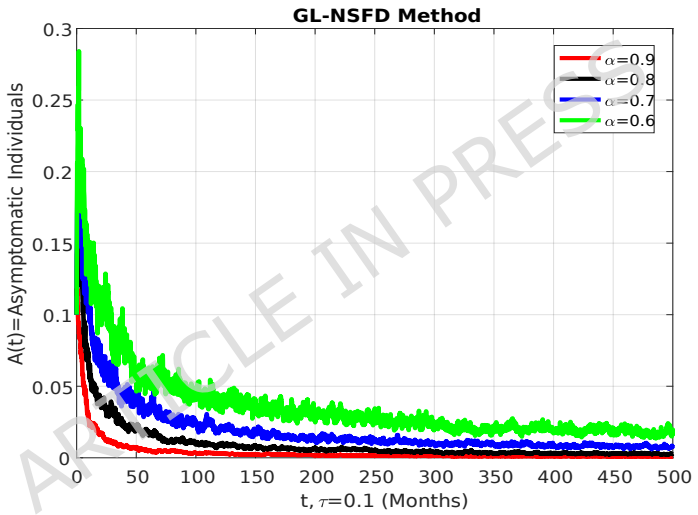

Fig. 4: Impact of "α" on A(t) at τ = 0.1.

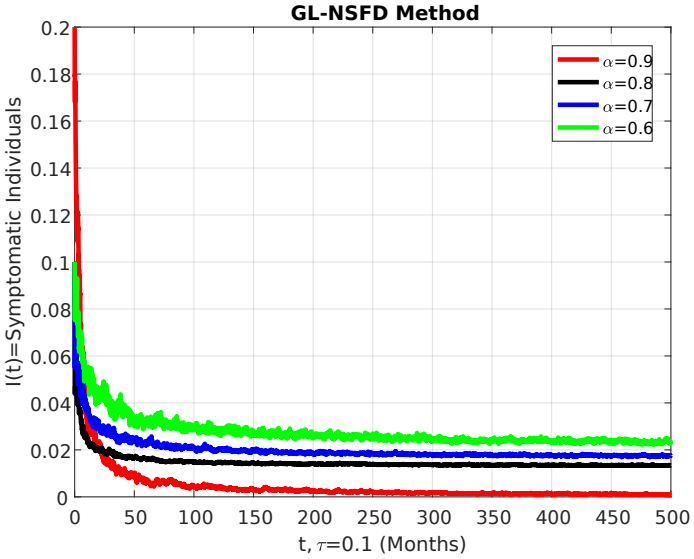

Fig. 5: Impact of "α" on I(t) at τ = 0.1.

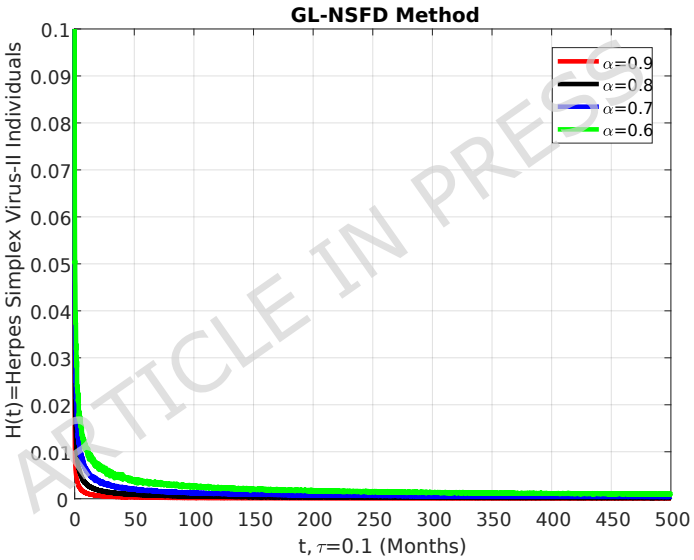

Fig. 6: Impact of "α" on H(t) at τ = 0.1.

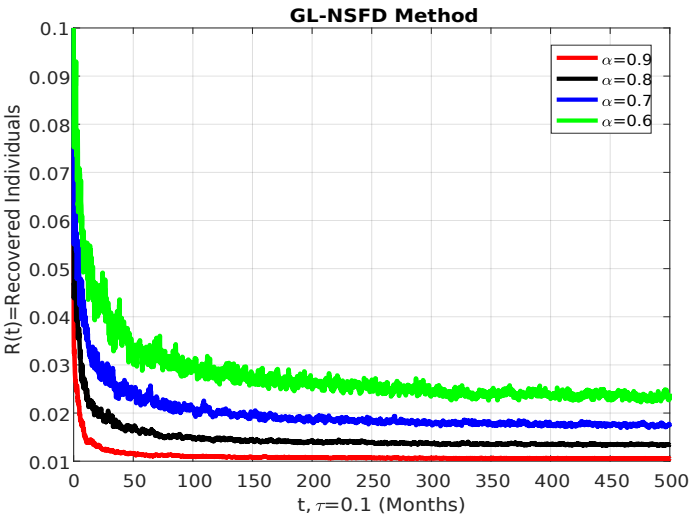

Fig. 7: Impact of "α" on R(t) at τ = 0.1.

4.1 Discussion

The graphical discussion for Herpes Simplex-II Virus (HSV-II) addresses the dynamics of how disease relates to various fractional order values, affecting the different compartments in the model with time delay. Fig. 2 represents the time profile of the susceptible population, as it is affected by changes in fractional orders. A decrease in fractional order implies a significant memory effect on the system that gradually reduces the rate of decline in the susceptible population, reflecting persistence over time for those at risk due to fractional time delay effects. In Fig. 3, the fractional order heavily affects the rate at which exposed individuals transition to the asymptomatic or symptomatic phases. As the fractional order increases, the exposed population drops more sharply, implying more rapid disease progression. The graph for asymptomatic individuals (Fig. 4) exhibits the variability of the peak and persistence of this compartment with fractional order. The lower fractional orders lead to prolonged peaks that suggest a delay in the recovery or progression due to the time-delay effect in the system. In Fig. 5 symptomatic individuals show more significant sensitivity to changes in fractional orders. The delay parameter increases the oscillatory behavior and seems to indicate fluctuations of disease prevalence over time when memory effects and delays interact. Fig. 6 for the viral load shows how fractional dynamics affect the prevalence of the virus. Lower fractional orders lead to sustained viral activity, which reflects the combined effects of delayed immune response and the persistence mechanisms inherent in the disease.

Finally, Fig. 7 illustrates the recovery dynamics and how fractional orders make the return to the recovered state delayed. Lower orders elongate the recovery period due to strong memory and time-delay effects, while higher orders exhibit recovery rates. This means that by changing the fractional order, one can simulate the different behaviors of diseases such as prolonged exposure phases, oscillatory symptomatic peaks, and delayed recovery. Such insights are crucial for long-term disease control and the planning of effective interventions. By

adjusting the fractional order, one can simulate varying disease behaviors, such as prolonged exposure phases, oscillatory symptomatic peaks, and delayed recovery. These insights are important for understanding long-term disease control and planning effective interventions. In the context of HSV-II, fractional-order memory reflects the cumulative influence of past infection history, including immune persistence, latency, and recurrent reactivation, on current transmission dynamics. Lower values of the fractional order α indicate stronger dependence on historical states, resulting in slower progression and prolonged persistence. This effect differs from the discrete delay τ, which represents a specific biological waiting time, such as incubation or delayed response.
In this model, explicit delay would mainly reflect biological delays of waiting processes, such as incubation and host response delays. Stochastic perturbations capture random variability in transmission and immune dynamics. These components introduce uncertainty and heterogeneity that cannot be modeled by a deterministic model in isolation.

5 Conclusion

In this study, the dynamics of Herpes Simplex Virus–II (HSV-II) were investigated through a stochastic fractional delay differential model incorporating essential epidemiological factors. The model guarantees the existence, uniqueness, positivity, and boundedness of solutions. Two equilibrium states were identified: HSV-Free Equilibrium (HSV-FE) and HSV-Present Equilibrium (HSV-PE), with the basic reproduction number calculated via the Next-Generation Matrix method. Both local and global stability analyses were performed, along with a sensitivity analysis to examine the influence of key parameters on disease transmission. Numerical simulations based on the Stochastic Non-Standard Finite Difference (NSFD) scheme were conducted for various fractional orders α, demonstrating improved positivity, boundedness, and stability compared to standard fractional-order modeling methods. The theoretical findings were further validated through graphical simulations, providing deeper insights into HSV-II dynamics and supporting informed public health decision-making. Overall, the combination of advanced mathematical modeling and robust computational simulations provides a valuable framework for understanding complex biological systems and predicting disease dynamics. The suggested framework might be expanded in future work by availing probabilistic safety and control-theoretic concepts, e.g., risk-aware analysis and adaptive control strategies, which have been proven to work well in complicated engineered systems]25-27]. These types of methods could even further improve the robustness and real-time usability of HSV-II modeling when the uncertainty is present.

Declaration of Conflicts of Interest: The authors affirm they have no conflicts of interest to disclose concerning the current study.

Data Availability: The datasets analyzed during the current study are available from the corresponding author upon reasonable request.

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Appendix A:

Proof: First, we analyze the Lipschitz's condition for the function $\hbar_1(t, S)$. For this, we take help with the following S and S_1:

$$\|\hbar_1(t, S) - \hbar_1(t, S_1)\| =$$

$$\left\|\left(\Lambda^{\alpha} - \beta^{\alpha} S(I + q^{\alpha} A)e^{-\mu^{\alpha}\tau} - \mu^{\alpha} S + \omega^{\alpha} R\right) - \left(\Lambda^{\alpha} - \beta^{\alpha} S_1(I + q^{\alpha} A)e^{-\mu^{\alpha}\tau} - \mu^{\alpha} S_1 + \omega^{\alpha} R\right)\right\|.$$

$$\|\hbar_1(t, S) - \hbar_1(t, S_1)\| = \left\|\left(\beta^{\alpha}(S - S_1)(I + q^{\alpha} A)e^{-\mu^{\alpha}\tau} + \mu^{\alpha}(S - S_1)\right)\right\|.$$

$\|\hbar_1(t, S) - \hbar_1(t, S_1)\| \leq \left\|\beta^{\alpha}(S - S_1)(I + q^{\alpha}A)e^{-\mu^{\alpha}\tau}\right\| + \|\mu^{\alpha}(S - S_1)\|.$

$\|\hbar_1(t, S) - \hbar_1(t, S_1)\| \leq \left(\beta^{\alpha}e^{-\mu^{\alpha}\tau}\|I + q^{\alpha}A\| + \mu^{\alpha}\right)\|S - S_1\|.$

$\|\hbar_1(t, S) - \hbar_1(t, S_1)\| \leq \left(\beta^{\alpha}e^{-\mu^{\alpha}\tau}(\varepsilon_4 + q^{\alpha}\varepsilon_3) + \mu^{\alpha}\right)\|S - S_1\|.$

$\|\hbar_1(t, S) - \hbar_1(t, S_1)\| \leq \xi_1\|S - S_1\|.$

Since, $\xi_1 = \left(\beta^{\alpha}e^{-\mu^{\alpha}\tau}(\varepsilon_4 + q^{\alpha}\varepsilon_3) + \mu^{\alpha}\right)$. Lipschitz's condition is satisfied. Next, for $\hbar_2(t, E)$ consider E and E_1.

$\|\hbar_2(t, E) - \hbar_2(t, E_1)\| =$

$\left\|\left(\beta^{\alpha}S(I + q^{\alpha}A)e^{-\mu^{\alpha}\tau} - (\eta^{\alpha} + \mu^{\alpha})E\right) - \left(\beta^{\alpha}S(I + q^{\alpha}A)e^{-\mu^{\alpha}\tau} - (\eta^{\alpha} + \mu^{\alpha})E_1\right)\right\|.$

$\|\hbar_2(t, E) - \hbar_2(t, E_1)\| = \|((\eta^{\alpha} + \mu^{\alpha})(E - E_1))\|.$

$\|\hbar_2(t, E) - \hbar_2(t, E_1)\| \leq (\eta^{\alpha} + \mu^{\alpha})\|E - E_1\|.$

$\|\hbar_2(t, E) - \hbar_2(t, E_1)\| \leq \xi_2\|E - E_1\|.$

For, $\xi_2 = (\eta^{\alpha} + \mu^{\alpha})$. Lipschitz condition is satisfied.

Next, for $\hbar_3(t, A)$ analyzing for A and A_1.

$\|\hbar_3(t, A) - \hbar_3(t, A_1)\| =$
$\|((1 - \rho^{\alpha})\eta^{\alpha}E - (\varphi^{\alpha} + \gamma^{\alpha} + \mu^{\alpha})A) - ((1 - \rho^{\alpha})\eta^{\alpha}E - (\varphi^{\alpha} + \gamma^{\alpha} + \mu^{\alpha})A_1)\|.$
$\|\hbar_3(t, A) - \hbar_3(t, A_1)\| = \|(\varphi^{\alpha} + \gamma^{\alpha} + \mu^{\alpha})(A - A_1)\|.$

$\|\hbar_3(t, A) - \hbar_3(t, A_1)\| \leq (\varphi^{\alpha} + \gamma^{\alpha} + \mu^{\alpha})\|A - A_1\|.$

$\|\hbar_3(t, A) - \hbar_3(t, A_1)\| \leq \xi_3\|A - A_1\|.$

For, $\xi_3 = (\varphi^{\alpha} + \gamma^{\alpha} + \mu^{\alpha})$. Lipschitz condition is satisfied.

Next, for $\hbar_4(t, I)$ analyzing for I and I_1.

$\|\hbar_4(t, I) - \hbar_4(t, I_1)\| = \|(\rho^{\alpha}\eta^{\alpha}E - (\phi^{\alpha} + \theta^{\alpha} + \mu^{\alpha})I) - (\rho^{\alpha}\eta^{\alpha}E - (\phi^{\alpha} + \theta^{\alpha} + \mu^{\alpha})I_1)\|.$

$\|\hbar_4(t, I) - \hbar_4(t, I_1)\| = \|(\phi^{\alpha} + \theta^{\alpha} + \mu^{\alpha})(I - I_1)\|.$

$\|\hbar_4(t, I) - \hbar_4(t, I_1)\| \leq (\phi^{\alpha} + \theta^{\alpha} + \mu^{\alpha})\|I - I_1\|.$

$\|\hbar_4(t, I) - \hbar_4(t, I_1)\| \leq (\phi^\alpha + \theta^\alpha + \mu^\alpha)\|I - I_1\|.$

For, $\xi_4 = (\phi^\alpha + \theta^\alpha + \mu^\alpha)$. Lipschitz condition is satisfied.

Next, for $\hbar_5(t, H)$ analyzing for H and H_1.

$\|\hbar_5(t, H) - \hbar_5(t, H_1)\| =$
$\|(\varphi^\alpha A + \phi^\alpha I - (\delta^\alpha + \xi^\alpha + \mu^\alpha)H) - (\varphi^\alpha A + \phi^\alpha I - (\delta^\alpha + \xi^\alpha + \mu^\alpha)H_1)\|.$
$\|\hbar_5(t, H) - \hbar_5(t, H_1)\| = \|((\delta^\alpha + \xi^\alpha + \mu^\alpha)(H - H_1))\|.$

$\|\hbar_5(t, H) - \hbar_5(t, H_1)\| \leq (\delta^\alpha + \xi^\alpha + \mu^\alpha)\|H - H_1\|.$

For, $\xi_5 = (\delta^\alpha + \xi^\alpha + \mu^\alpha)$. Lipschitz condition is satisfied.

Next, for $\hbar_6(t, R)$ analyzing for R and R_1.

$\|\hbar_6(t, R) - \hbar_6(t, R_1)\| =$
$\|(\gamma^\alpha A + \theta^\alpha I + \delta^\alpha H - (\omega^\alpha + \mu^\alpha)R) - (\gamma^\alpha A + \theta^\alpha I + \delta^\alpha H - (\omega^\alpha + \mu^\alpha)R_1)\|.$
$\|\hbar_6(t, R) - \hbar_6(t, R_1)\| = \|((\omega^\alpha + \mu^\alpha)(R - R_1))\|.$

$\|\hbar_6(t, R) - \hbar_6(t, R_1)\| \leq (\omega^\alpha + \mu^\alpha)\|R - R_1\|.$

$\|\hbar_6(t, R) - \hbar_6(t, R_1)\| \leq \xi_6\|R - R_1\|.$

For, $\xi_6 = (\omega^\alpha + \mu^\alpha)$. Lipschitz condition is satisfied.

Next, there is constant in (13–18).

$$S_n(t) = \frac{1}{\Gamma(\alpha)}\int_0^t (t - s)^{\alpha-1}\hbar_1(s, S_{n-1})ds. \quad (19)$$

$$E_n(t) = \frac{1}{\Gamma(\alpha)}\int_0^t (t - s)^{\alpha-1}\hbar_2(s, E_{n-1})ds. \quad (20)$$

$$A_n(t) = \frac{1}{\Gamma(\alpha)}\int_0^t (t - s)^{\alpha-1}\hbar_3(s, A_{n-1})ds. \quad (21)$$

$$I_n(t) = \frac{1}{\Gamma(\alpha)}\int_0^t (t - s)^{\alpha-1}\hbar_4(s, I_{n-1})ds. \quad (22)$$

$$H_n(t) = \frac{1}{\Gamma(\alpha)}\int_0^t (t - s)^{\alpha-1}\hbar_5(s, H_{n-1})ds. \quad (23)$$

$$R_n(t) = \frac{1}{\Gamma(\alpha)}\int_0^t (t - s)^{\alpha-1}\hbar_6(s, R_{n-1})ds. \quad (24)$$

Remaining variation is as follows:

$$\psi_{n-1}(t) = (S_n(t) - S_{n-1}(t)) = \frac{1}{\Gamma(\alpha)}\int_0^t (\hbar_1(s, S_{n-1}) - \hbar_1(s, S_{n-2}))ds. \quad (25)$$

$$\varphi_{n-1}(t) = (E_n(t) - E_{n-1}(t)) = \frac{1}{\Gamma(\alpha)}\int_0^t (\hbar_2(s, E_{n-1}) - \hbar_2(s, E_{n-2}))ds. \quad (26)$$

$$\vartheta_{n-1}(t) = (A_n(t) - A_{n-1}(t)) = \frac{1}{\Gamma(\alpha)}\int_0^t (\hbar_3(s, A_{n-1}) - \hbar_3(s, A_{n-2}))ds. \quad (27)$$

$$\varpi_{n-1}(t) = (I_n(t) - I_{n-1}(t)) = \frac{1}{\Gamma(\alpha)}\int_0^t (\hbar_4(s, I_{n-1}) - \hbar_4(s, I_{n-2}))ds. \quad (28)$$

$$\Psi_{n-1}(t) = (H_n(t) - H_{n-1}(t)) = \frac{1}{\Gamma(\alpha)}\int_0^t (\hbar_5(s, H_{n-1}) - \hbar_5(s, H_{n-2}))ds. \quad (29)$$

$$\zeta_{n-1}(t) = (R_n(t) - R_{n-1}(t)) = \frac{1}{\Gamma(\alpha)}\int_0^t (\hbar_6(s, R_{n-1}) - \hbar_6(s, R_{n-2}))ds. \quad (30)$$

Therefore, we have

$$S_n(t) = \sum_{i=0}^{n} \psi_i(t). \quad (31)$$

$$E_n(t) = \sum_{i=0}^{n} \varphi_i(t). \quad (32)$$

$$I_n(t) = \sum_{i=0}^{n} \vartheta_i(t). \quad (33)$$

$$A_n(t) = \sum_{i=0}^{n} \varpi_i(t). \quad (34)$$

$$H_n(t) = \sum_{i=0}^{n} \Psi_i(t). \quad (35)$$

$$R_n(t) = \sum_{i=0}^{n} \zeta_i(t). \quad (36)$$

Let,

$$\|\psi_n(t)\| = \|S_n(t) - S_{n-1}(t)\|.$$

$$\|\psi_n(t)\| = \frac{1}{\Gamma(\alpha)}\int_0^t (\hbar_1(s, S_{n-1}) - \hbar_1(s, S_{n-2}))ds.$$

$$\|\psi_n(t)\| = \frac{\xi_1}{\Gamma(\alpha)}\int_0^t \|S_n(t) - S_{n-1}(t)\|ds.$$

$$\|\psi_n(t)\| = \frac{\xi_1}{\Gamma(\alpha)}\int_0^t \psi_{n-1}(t)ds. \quad (37)$$

Similarly,

$$\|\varphi_n(t)\| = \frac{\xi_2}{\Gamma(\alpha)}\int_0^t \varphi_{n-1}(t)ds. \quad (38)$$

$$\|\vartheta_n(t)\| = \frac{\xi_3}{\Gamma(\alpha)}\int_0^t \vartheta_{n-1}(t)ds. \quad (39)$$

$$\|\varpi_n(t)\| = \frac{\xi_4}{\Gamma(\alpha)}\int_0^t \varpi_{n-1}(t)ds. \quad (40)$$

$$\|\Psi_n(t)\| = \frac{\xi_5}{\Gamma(\alpha)}\int_0^t \Psi_{n-1}(t)ds. \quad (41)$$

$$\|\zeta_n(t)\| = \frac{\xi_6}{\Gamma(\alpha)}\int_0^t \zeta_{n-1}(t)ds. \quad (42)$$

As required.

Appendix B.

Proof: Consider the $S(t)$, $E(t)$,$A(t)$,$I(t)$,$H(t)$ and $R(t)$ are bounded. Then

$$\|\psi_n(t)\| \leq \|S(0)\|\left\|\frac{\xi_1}{\Gamma(\alpha)}(t)\right\|^n. \quad (43)$$

$$\|\vartheta_n(t)\| \leq \|E(0)\|\left\|\frac{\xi_2}{\Gamma(\alpha)}(t)\right\|^n. \quad (44)$$

$$\|\psi_n(t)\| \leq \|A(0)\|\left\|\frac{\xi_3}{\Gamma(\alpha)}(t)\right\|^n. \quad (45)$$

$$\|\varpi_n(t)\| \leq \|I(0)\|\left\|\frac{\xi_4}{\Gamma(\alpha)}(t)\right\|^n. \quad (46)$$

$$\|\Psi_n(t)\| \leq \|H(0)\|\left\|\frac{\xi_5}{\Gamma(\alpha)}(t)\right\|^n. \quad (47)$$

$$\|\zeta_n(t)\| \leq \|R(0)\|\left\|\frac{\xi_6}{\Gamma(\alpha)}(t)\right\|^n. \quad (48)$$

Since, $S(t)$, $E(t)$,$A(t)$,$I(t)$,$H(t)$ and $R(t)$ will converge because the system (31-36) exists and consistent. For this, consider n changes as $A_n(t)$, $B_n(t)$, $C_n(t)$, $D_n(t)$, $X_n(t)$ and $Y_n(t)$. Thus,

$$S(t) - S(0) = S_n(t) - A_n(t). \quad (49)$$

$$E(t) - E(0) = E_n(t) - B_n(t). \quad (50)$$

$$A(t) - A(0) = A_n(t) - C_n(t). \quad (51)$$

$$I(t) - I(0) = I_n(t) - D_n(t). \quad (52)$$

$$H(t) - H(0) = H_n(t) - X_n(t). \quad (53)$$

$$R(t) - R(0) = R_n(t) - Y_n(t). \quad (54)$$

The result of Lipschitz condition for (ξ_1) and the triangle inequality, and $\hbar_1$ for $i = 1,2,3,4,5,6$, fulfills the Lipschitz condition.

$$\|A_n(t)\| = \frac{1}{\Gamma(\alpha)}\int_0^t(\hbar_1(s, S_{n-1}) - \hbar_1(s, S_{n-2}))ds.$$

$$\|A_n(t)\| \leq \frac{\xi_1}{\Gamma(\alpha)}\|S_n(t) - S_{n-1}(t)\|. \quad (55)$$

with repetition of (55),

$$\|A_n(t)\| \leq \left\|\frac{\xi_1}{\Gamma(\alpha)}(t)\right\|^{n+1} \varepsilon_1. \quad (56)$$

Next, at t_*, one acquires

$$\|A_n(t)\| \leq \left\|\frac{\xi_1}{\Gamma(\alpha)}(t_*)\right\|^{n+1} \varepsilon_1. \quad (57)$$

Assuming $n \to \infty$ as the limit.

$$\lim_{n\to\infty}\|A_n(t)\| \leq \lim_{n\to\infty}\left\|\frac{\xi_1}{\Gamma(\alpha)}(t_*)\right\|^{n+1} \varepsilon_1. \quad (58)$$

By applying the hypothesis $\frac{\xi_1}{\Gamma(\alpha)}(t_*) < 1$, we get

$$\lim_{n\to\infty}\|A_n(t)\| = 0. \quad (59)$$

Similarly,

$$\|B_n(t)\| \to 0. \quad (60)$$

$$\|C_n(t)\| \to 0. \quad (61)$$

$$\|D_n(t)\| \to 0. \quad (62)$$

$$\|X_n(t)\| \to 0. \quad (63)$$

$$\|Y_n(t)\| \to 0. \quad (64)$$

As desired.

Appendix C

Proof: Examine how the sets S_1, E_1, A_1, I_1, H_1, and R_1 represent the solutions to (1-6).

$$\|S(t) - S_1(t)\| = \frac{1}{\Gamma(\alpha)}\int_0^t (\hbar_1(s, S) - \hbar_1(s, S_1))ds.$$

$$\|S(t) - S_1(t)\| \leq \frac{\xi_1}{\Gamma(\alpha)}\|S(t) - S_1(t)\|. \quad (65)$$

After simplifying,

$$\left(1 - \frac{\xi_1}{\Gamma(\alpha)}(t)\right)\|S(t) - S_1(t)\| \leq 0. \quad (66)$$

By applying the hypothesis $\left(1 - \frac{\xi_1}{\Gamma(\alpha)}(t)\right) > 0$, we have from (66) yield.

$$\|S(t) - S_1(t)\| = 0. \quad (67)$$

It follows from this because $S(t) = S_1(t)$.

Similarly,

$$E(t) = E_1(t). \tag{68}$$

$$A(t) = A_1(t). \tag{69}$$

$$I(t) = I_1(t). \tag{70}$$

$$H(t) = H_1(t). \tag{71}$$

$$R(t) = R_1(t). \tag{72}$$

Hence proved.