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Nonlocal and nonsingular kernel-based fractional dynamics of infectious disease with waning immunity

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Abstract

The burden of infections is a critical public health issue, and addressing it requires a multifaceted approach involving healthcare systems, governments, and communities. In this work, the dynamics of Japanese encephalitis (JE) is constructed with waning immunity using the Atangana-Baleanu fractional derivative. We introduce the fundamental theory related to the proposed fractional operator for the evaluation of the dynamics. The recommended model of the infection is examined for the essential results and the endemic indicator $\mathcal{R}_0$ is determined with the help of next generation matrix approach. The existence and uniqueness of the solution of fractional-order system are evaluated via fixed point theory. Moreover, a newly developed numerical method is applied to iteratively solve our fractional-order model. Numerical simulations suggest that the management strategies are productive in reducing the prevalence of JE in humans, mosquitoes, and pigs. The fractional-order derivative provides more precise and realistic insights into the dynamics of JE, as evidenced by numerical findings that demonstrate the impact of various input factors on infection dynamics. Our research underscores the role of different factors of the recommended system for the management and control of the infection.

Keywords: Infectious disease, fractional dynamics, fixed-point theory, existence of solution, disease control, public health.

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1. Introduction

Encephalitis is an infection or immune response that causes inflammation in active areas of the brain. Symptoms can include headaches, a stiff neck, light sensitivity, mental confusion, and convulsions. Japanese encephalitis (JE), a vector-borne disease caused by the Japanese encephalitis virus (JEV), primarily affects populations in Asia, with research on the virus spanning over 90 years [20]. Encephalitis epidemics have been reported in Japan since the 1870s, with significant outbreaks occurring roughly every decade. A major outbreak in 1924 led to more than 6,000 cases [9]. JEV infects animals and is transmitted

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by Culex mosquitoes, with a prevalence throughout the Pacific Rim and eastern and southern Asia. Several other neurotropic flaviviruses with similar clinical, epidemiological, and virological characteristics are found worldwide [14]. One such flavivirus, the West Nile virus, is present in parts of Europe, Africa, and the Middle East, and is commonly associated with rash, joint discomfort, fever, and neurological symptoms. In 1996, an encephalitis outbreak due to West Nile virus occurred in Romania [18]. Human infection with JEV typically occurs when individuals reside in or visit areas near the virus's enzootic cycle, which exists in both rural and urban fringe regions.

Mathematical models are valuable tools for understanding the transmission of infectious diseases and providing effective control interventions for these infections [1, 17]. These modeling approaches involve constructing mathematical frameworks that characterize the spread of the JE virus across various populations, including humans, animals, and mosquitoes. The application of mathematical modeling to biological phenomena has emerged as a significant field of research [13]. In 2009, the first model of JE infection was introduced and analyzed using mathematical tools [10]. Subsequent studies proposed an eight-equation ordinary differential equation (ODE) model to describe JE transmission, focusing on the determination of the endemic indicator and the stability of the system around equilibrium points [11]. Additionally, between 2006 and 2010, researchers utilized mathematical modeling and likelihood-based methods to investigate the disappearance and recurrence of JE cases in humans [21]. This study recommended the removal of vaccinated human and pig compartments from prior models of JE transmission [6]. The modeling of Japanese encephalitis (JE) continues to evolve, providing critical insights into the disease's transmission dynamics and potential control measures. This study aims to develop an epidemic model to analyze the dynamics of JE infection while considering the effects of waning immunity. Furthermore, it seeks to investigate the impact of various parameters on transmission dynamics and propose effective control strategies to mitigate disease spread.

Fractional derivatives have emerged as a vital tool in the modeling of epidemic dynamics due to their ability to capture complex behaviors that traditional integer-order models may overlook [8]. These derivatives allow for a more nuanced representation of memory and hereditary effects in biological systems, reflecting the reality that the dynamics of infectious diseases often depend not only on the current state but also on past states [3, 15]. In epidemic models, fractional derivatives can effectively incorporate phenomena such as waning immunity, heterogeneous population interactions, and the nonlocal nature of disease transmission. By enabling a more accurate portrayal of the underlying processes, fractional calculus enhances our understanding of epidemic spread, facilitating the development of more effective control strategies and public health interventions [7, 16]. Consequently, integrating fractional derivatives into epidemic models represents a significant advancement in mathematical epidemiology, providing deeper insights into the dynamics of infectious diseases and aiding in the formulation of timely and informed responses to outbreaks. In this study, we present the nonlocal and nonsingular kernel-based fractional dynamics of Japanese encephalitis (JE) with waning immunity to capture the complex phenomena of the infection more accurately than traditional models.

This work is outlined as: The theory and results of fractional calculus are established in Section 2. In Section 3, we formulate the dynamics of the infection with non-singular and non-local kernel. The recommended fractional system of the infection is also investigated in Section 3 and the endemic indicator is determined. Using fixed point theory, Section 4 establishes the existence and uniqueness of solutions. In Section 5, a numerical scheme for the model analysis that illustrates the system's solution routes is provided. Ending remarks are included in Section 6.

2. Theory and Concepts

The definitions and main conclusions of the Caputo fractional derivatives and Atangana-Baleanu, as described in [2, 12], will be introduced in this section.

Definition 2.1. For a given $f : [p, q] \to \mathbb{R}$, the Caputo operator of order υ is defined as:

$$ {}^{C}_{p}D^{\upsilon}_{t}(u(t)) = \frac{1}{\eta(n-\upsilon)} \int_{p}^{t} (t-\kappa)^{n-\upsilon-1} u^{n}(\kappa) d\kappa, $$

for $\upsilon \in (n-1, n)$, where $n \in \mathbf{Z}$.

Definition 2.2. Take f in a manner that $f \in H^1(p, q)$, $q > p$, and $\in [0, 1]$, then the ABC derivative is defined as:

$$ {}^{ABC}_{p}D^{\upsilon}_{t} f(t) = \frac{B(\upsilon)}{1-\upsilon} \int_{p}^{t} f'(\kappa) E_{\upsilon} \left[\frac{(t-\kappa)^{\upsilon}}{1-\upsilon} (-\upsilon) \right] d\kappa. $$

Definition 2.3. The integral of ABC operator is symbolized by ${}^{ABC}_{p}I^{\upsilon}_{t} f(t)$ and is described as follows:

$$ {}^{ABC}_{p}I^{\upsilon}_{t} f(t) = \frac{1-\upsilon}{B(\upsilon)} f(t) + \frac{\upsilon}{\eta(\upsilon)B(\upsilon)} \int_{p}^{t} (t-\kappa)^{\upsilon-1} f(\kappa) d\kappa. $$

As υ approaches 0, it is clear that the original function is obtained.

Theorem 2.4 ([2]). *The statement below holds true for a function f if $f \in C[p, q]$:*

$$ \| {}^{ABC}_{p}D^{\upsilon}_{t}(f(t)) \| < \frac{B(\upsilon)}{1-\upsilon} \|f(t)\|, \text{ where } \|f(t)\| = max_{p \leqslant t \leqslant q} |f(t)|. $$

Moreover, the condition of Lipschitz is satisfied by the novel ABC operator as given below:

$$ \| {}^{ABC}_{p}D^{\upsilon}_{t} f_1(t) - {}^{ABC}_{p}D^{\upsilon}_{t} f_2(t) \| < \downarrow_1 \| f_1(t) - f_2(t) \|. $$

Theorem 2.5 ([2]). *The following fractional system has a unique solution:*

$$ {}^{ABC}_{p}D^{\upsilon}_{t} f(t) = g(t), $$

given by

$$ f(t) = \frac{1-\upsilon}{B(\upsilon)} g(t) + \frac{\upsilon}{B(\upsilon)\eta(\upsilon)} \int_{p}^{t} g(\kappa)(t-\kappa)^{\upsilon-1} d\kappa. $$

3. Evaluation of the Fractional Dynamics

In this section, we describe the dynamics of Japanese encephalitis (JE), categorizing the population into three groups: pigs, mosquitoes, and humans. The hosts and vector population is indicated by $\mathcal{N}_m$ and $\mathcal{N}_p$, respectively. Three compartments comprise the human population ($\mathcal{N}_h$): susceptible ($\mathcal{S}_h$), infected ($\mathcal{I}_h$), and recovered ($\mathcal{R}_h$). There are two types of mosquitoes in the population ($\mathcal{N}_m$): susceptible ($\mathcal{S}_m$) and infected ($\mathcal{I}_m$). In a similar manner, two groups are identified within the pig population ($\mathcal{N}_p$): susceptible ($\mathcal{S}_p$) and infected ($\mathcal{I}_p$). Let us suppose that the recruitment rate for the human population ($\mathcal{N}_h$) is φ, while the mosquito population ($\mathcal{N}_m$) and pig population ($\mathcal{N}_p$) are considered to have constant, and is symbolized as μ_m and μ_p, respectively.

The transmission of JE take place between pigs, mosquitoes and humans. The pigs and mosquitoes get transmitted with each other and the infected mosquitoes bite the susceptible humans due to which the JE disease transfer to humans. Humans with strong immunity and cure can transfer themselves from the infected class to the recovered class and then to the susceptible class of JE. Hence, our model with the above assumptions for the transmission of JE is as follow:

$$ \begin{cases} \frac{d\mathcal{S}_h}{dt} &= \Xi - \frac{b\beta_{mh}}{\mathcal{N}_h} \mathcal{I}_m \mathcal{S}_h + \rho(1-\tau_1)\mathcal{I}_h + \alpha\mathcal{R}_h - \mu_h \mathcal{S}_h, \\ \frac{d\mathcal{I}_h}{dt} &= \frac{b\beta_{mh}}{\mathcal{N}_h} \mathcal{I}_m \mathcal{S}_h - \rho\mathcal{I}_h - \gamma\mathcal{I}_h - \mu_h \mathcal{I}_h, \\ \frac{d\mathcal{R}_h}{dt} &= \rho\mathcal{I}_h\tau_1 - \alpha\mathcal{R}_h - \mu_h \mathcal{R}_h, \\ \frac{d\mathcal{S}_m}{dt} &= \mu_m \mathcal{N}_m - \frac{c\beta_{pm}}{\mathcal{N}_p} \mathcal{I}_p \mathcal{S}_m - \mu_m \mathcal{S}_m, \\ \frac{d\mathcal{I}_m}{dt} &= \frac{c\beta_{pm}}{\mathcal{N}_p} \mathcal{I}_p \mathcal{S}_m - \mu_m \mathcal{I}_m, \\ \frac{d\mathcal{S}_p}{dt} &= \mu_p \mathcal{N}_p - \frac{c\beta_{mp}}{\mathcal{N}_p} \mathcal{I}_m \mathcal{S}_p - \mu_p \mathcal{S}_p, \\ \frac{d\mathcal{I}_p}{dt} &= \frac{c\beta_{mp}}{\mathcal{N}_p} \mathcal{I}_m \mathcal{S}_p - \mu_p \mathcal{I}_p, \end{cases} \tag{3.1} $$

with appropriate initial condition

$$\mathcal{S}_h(0) \geqslant 0,\ \mathcal{I}_h(0) \geqslant 0,\ \mathcal{R}_h(0) \geqslant 0,\ \mathcal{S}_m(0) \geqslant 0,\ \mathcal{I}_m(0) \geqslant 0, \mathcal{S}_p(0) \geqslant 0,\ \mathcal{I}_p(0) \geqslant 0.$$

The population of human and mosquitoes and pigs are specified as:

$$\begin{aligned}\mathcal{N}_h &= \mathcal{S}_h + \mathcal{I}_h + \mathcal{R}_h,\\ \mathcal{N}_m &= \mathcal{S}_m + \mathcal{I}_m,\\ \mathcal{N}_p &= \mathcal{S}_p + \mathcal{I}_p.\end{aligned}$$

The above model (3.1) in fractional form can written as:

$$\begin{cases} {}^{ABC}_{0}D^{\upsilon}_{t}\mathcal{S}_h &= \Xi - \frac{b\beta_{mh}}{\mathcal{N}_h}\mathcal{I}_m\mathcal{S}_h + \rho\mathcal{I}_h - \rho\mathcal{I}_h\tau_1 + \alpha\mathcal{R}_h - \mu_h\mathcal{S}_h,\\ {}^{ABC}_{0}D^{\upsilon}_{t}\mathcal{I}_h &= \frac{b\beta_{mh}}{\mathcal{N}_h}\mathcal{I}_m\mathcal{S}_h - \rho\mathcal{I}_h - \gamma\mathcal{I}_h - \mu_h\mathcal{I}_h,\\ {}^{ABC}_{0}D^{\upsilon}_{t}\mathcal{R}_h &= \rho\mathcal{I}_h\tau_1 - \alpha\mathcal{R}_h - \mu_h\mathcal{R}_h,\\ {}^{ABC}_{0}D^{\upsilon}_{t}\mathcal{S}_m &= \mu_m\mathcal{N}_m - \frac{c\beta_{pm}}{\mathcal{N}_p}\mathcal{I}_p\mathcal{S}_m - \mu_m\mathcal{S}_m,\\ {}^{ABC}_{0}D^{\upsilon}_{t}\mathcal{I}_m &= \frac{c\beta_{pm}}{\mathcal{N}_p}\mathcal{I}_p\mathcal{S}_m - \mu_m\mathcal{I}_m,\\ {}^{ABC}_{0}D^{\upsilon}_{t}\mathcal{S}_p &= \mu_p\mathcal{N}_p - \frac{c\beta_{mp}}{\mathcal{N}_p}\mathcal{I}_m\mathcal{S}_p - \mu_p\mathcal{S}_p,\\ {}^{ABC}_{0}D^{\upsilon}_{t}\mathcal{I}_p &= \frac{c\beta_{mp}}{\mathcal{N}_p}\mathcal{I}_m\mathcal{S}_p - \mu_p\mathcal{I}_p,\end{cases} \tag{3.2}$$

with non-negative initial values. The symbol ${}^{ABC}_{0}D^{\upsilon}_{t}$ used in the above system (3.2) represents the AB derivative in Caputo sense. The motivation for developing a fractional epidemic model stems from the need to better understand and describe the complex dynamics of infectious disease transmission.

Table 1: Representation of the input factors of JE dynamics with descriptions

Parameter	Interpretation
Ξ	Recruitment rate of host population
α	Losing rate of host immunity
γ	Rate of mortality of host
ρ	Rate of recovery of host
μ_h	Natural death rate of host
μ_m	Rate of recruitment and natural death of mosquitoes
μ_p	Rate of recruitment and natural death of pigs
τ_1	Human's natural recovery rate
b	Transmission's probability from vectors to hosts
c	Average number of bites on pigs by mosquitoes
β_{mh}	Rate of transmission from $\mathcal{I}_m$ to $\mathcal{S}_h$
β_{mp}	Transmission rate between $\mathcal{I}_m$ and $\mathcal{S}_p$
β_{pm}	Transmission rate between $\mathcal{I}_p$ and $\mathcal{S}_m$

Theorem 3.1. *The recommended system (3.2) of infection exhibits positive and bounded solutions for positive initial conditions.*

Proof. The recommended system (3.2) of infection has positive solutions under suitable initial conditions. To assure the boundedness, we add three equations of the system (3.2) and get

$${}^{ABC}_{0}D^{\upsilon}_{t}(\mathcal{S}_h + \mathcal{I}_h + \mathcal{R}_h) \leqslant \Xi - \mu_h(\mathcal{S}_h + \mathcal{I}_h + \mathcal{R}_h), \tag{3.3}$$

further, Laplace transformation convert equation (3.3) into

$$\begin{aligned}\mathcal{S}_h + \mathcal{I}_h + \mathcal{R}_h \leqslant &\left(\frac{B(\upsilon)}{B(\upsilon) + (1-\upsilon)\mu_h}(\mathcal{S}_h(0) + \mathcal{I}_h(0) + \mathcal{R}_h(0) + \frac{(1-\upsilon)(\Xi)}{B(\upsilon) + (1-\upsilon)\mu_h}\right)D_{\upsilon,1}(-\beta t^{\upsilon})\\ &+ \frac{\upsilon(\Xi)}{B(\upsilon) + (1-\upsilon)\mu_h}D_{\upsilon,\upsilon+1}(-\beta t^{\upsilon}),\end{aligned}$$

where $D_{p,q}$ is Mittag-Leffler function with p, q, and $\beta = \frac{\upsilon\mu_h}{B(\upsilon)+(1-\upsilon)\mu_h}$. Then, applying the result of [5], we have $\mathcal{S}_h(t)+\mathcal{I}_h(t)+\mathcal{R}_h(t) \leqslant \frac{\Xi}{\mu_h} \doteq M_1$, as t tends to infinity. Next add two equation of (3.2) implies:

$$ {}_0^{ABC}D_t^{\upsilon}(\mathcal{S}_m+\mathcal{I}_m) \leqslant \mu_m\mathcal{N}_m - \mu_m(\mathcal{S}_m+\mathcal{I}_m), $$

implies

$$ {}_0^{ABC}D_t^{\upsilon}(\mathcal{S}_m+\mathcal{I}_m) \leqslant A - \mu_m(\mathcal{S}_m+\mathcal{I}_m), \tag{3.4} $$

where $A = \mu_m\mathcal{N}_m$. Then, Laplace transformation convert equation (3.4) into

$$ \begin{aligned} \mathcal{S}_m+\mathcal{I}_m \leqslant & \left(\frac{B(\upsilon)}{B(\upsilon)+(1-\upsilon)\mu_m}(\mathcal{S}_m(0)+\mathcal{I}_m(0)+\frac{(1-\upsilon)(A)}{B(\upsilon)+(1-\upsilon)\mu_m}\right)D_{\upsilon,1}(-\beta t^{\upsilon}) \\ & + \frac{\upsilon(A)}{B(\upsilon)+(1-\upsilon)\mu_m}D_{\upsilon,\upsilon+1}(-\beta t^{\upsilon}), \end{aligned} $$

where $D_{p,q}$ is Mittag-Leffler function with p, q, and $\beta = \frac{\upsilon\mu_m}{B(\upsilon)+(1-\upsilon)\mu_m}$. By using the results of [5], we have $\mathcal{S}_m(t)+\mathcal{I}_m(t) \leqslant \frac{A}{\mu_m} \doteq M_2$, as t tends to infinity. Adding last two equations of model (3.2) as:

$$ {}_0^{ABC}D_t^{\upsilon}(\mathcal{S}_p+\mathcal{I}_p) \leqslant \mu_p\mathcal{N}_p - \mu_p(\mathcal{S}_p+\mathcal{I}_p), \tag{3.5} $$

implies the following

$$ {}_0^{ABC}D_t^{\upsilon}(\mathcal{S}_p+\mathcal{I}_p) \leqslant C - \mu_p(\mathcal{S}_p+\mathcal{I}_p), $$

where $C = \mu_p\mathcal{N}_p$. Then, Laplace transformation convert equation (3.5) into

$$ \begin{aligned} \mathcal{S}_p+\mathcal{I}_p \leqslant & \left(\frac{B(\upsilon)}{B(\upsilon)+(1-\upsilon)\mu_p}(\mathcal{S}_p(0)+\mathcal{I}_p(0)+\frac{(1-\upsilon)(C)}{B(\upsilon)+(1-\upsilon)\mu_p}\right)D_{\upsilon,1}(-\beta t^{\upsilon}) \\ & + \frac{\upsilon(C)}{B(\upsilon)+(1-\upsilon)\mu_p}D_{\upsilon,\upsilon+1}(-\beta t^{\upsilon}), \end{aligned} $$

where $D_{p,q}$ is Mittag-Leffler function with p, q, and $\beta = \frac{\upsilon\mu_p}{B(\upsilon)+(1-\upsilon)\mu_p}$. By applying the reuslts of [5], we have $\mathcal{S}_p(t)+\mathcal{I}_p(t) \leqslant \frac{C}{\mu_p} \doteq M_3$, as $t \to \infty$.

Suppose $M = max(M_1, M_2, M_3)$, then $(\mathcal{S}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{S}_m, \mathcal{I}_m, \mathcal{S}_p, \mathcal{I}_p) \leqslant M$. As a result, the recommended system (3.2) of infection exhibits positive and bounded solutions. □

3.1. *Analysis of the Model*

Here, we focus on the steady-state and the endemic indicator of the system, indicated by $\mathcal{R}_0$. In order to determine the steady states of the system, we first set the derivative to zero as follows: ${}_0^{ABC}D_t^{\upsilon}\mathcal{S}_h = {}_0^{ABC}D_t^{\upsilon}\mathcal{I}_h = {}_0^{ABC}D_t^{\upsilon}\mathcal{R}_h = {}_0^{ABC}D_t^{\upsilon}\mathcal{S}_m = {}_0^{ABC}D_t^{\upsilon}\mathcal{I}_m = {}_0^{ABC}D_t^{\upsilon}\mathcal{S}_p = {}_0^{ABC}D_t^{\upsilon}\mathcal{I}_p = 0$. Then, we have

$$ \begin{cases} 0 &= \Xi - \frac{b\beta_{mh}}{\mathcal{N}_h}\mathcal{I}_m\mathcal{S}_h + \rho\mathcal{I}_h - \rho\mathcal{I}_h\tau_1 + \alpha\mathcal{R}_h - \mu_h\mathcal{S}_h, \\ 0 &= \frac{b\beta_{mh}}{\mathcal{N}_h}\mathcal{I}_m\mathcal{S}_h - \rho\mathcal{I}_h - \gamma\mathcal{I}_h - \mu_h\mathcal{I}_h, \\ 0 &= \rho\mathcal{I}_h\tau_1 - \alpha\mathcal{R}_h - \mu_h\mathcal{R}_h, \\ 0 &= \mu_m\mathcal{N}_m - \frac{c\beta_{pm}}{\mathcal{N}_p}\mathcal{I}_p\mathcal{S}_m - \mu_m\mathcal{S}_m, \\ 0 &= \frac{c\beta_{pm}}{\mathcal{N}_p}\mathcal{I}_p\mathcal{S}_m - \mu_m\mathcal{I}_m, \\ 0 &= \mu_p\mathcal{N}_p - \frac{c\beta_{mp}}{\mathcal{N}_p}\mathcal{I}_m\mathcal{S}_p - \mu_p\mathcal{S}_p, \\ 0 &= \frac{c\beta_{mp}}{\mathcal{N}_p}\mathcal{I}_m\mathcal{S}_p - \mu_p\mathcal{I}_p. \end{cases} \tag{3.6} $$

Now, for the disease-free steady-states (DFE) of the system (3.6), we put the infected ones equal to zero as

$$ \mathcal{I}_h = \mathcal{I}_m = \mathcal{I}_p = 0, $$

and get

$$\mathcal{E}_0 = \left(\mathcal{S}_h^0, \mathcal{I}_h^0, \mathcal{R}_h^0, \mathcal{S}_m^0, \mathcal{I}_m^0, \mathcal{S}_p^0, \mathcal{I}_p^0\right) = \left(\frac{\Xi}{\mu_h}, 0, 0, \mathcal{N}_m, 0, \mathcal{N}_p, 0\right).$$

The threshold parameter, often represented as $\mathcal{R}_0$, is the average number of secondary infections produced by a single infected individual in a fully susceptible population. This parameter serves as a critical indicator for understanding the potential spread of an infectious disease [4]. To determine $\mathcal{R}_0$, we deal only with infected compartments of the system (3.2) as

$$\begin{cases} {}_0^{ABC}D_t^{\upsilon}\mathcal{I}_h &= \frac{b\beta_{mh}}{\mathcal{N}_h}\mathcal{I}_m\mathcal{S}_h - \rho\mathcal{I}_h - \gamma\mathcal{I}_h - \mu_h\mathcal{I}_h, \\ {}_0^{ABC}D_t^{\upsilon}\mathcal{I}_m &= \frac{c\beta_{pm}}{\mathcal{N}_p}\mathcal{I}_p\mathcal{S}_m - \mu_m\mathcal{I}_m, \\ {}_0^{ABC}D_t^{\upsilon}\mathcal{I}_p &= \frac{c\beta_{mp}}{\mathcal{N}_p}\mathcal{I}_m\mathcal{S}_p - \mu_p\mathcal{I}_p. \end{cases}$$

By using the next-generation method [4], we have the following

$$\mathcal{F} = \begin{bmatrix} 0 & \frac{b\beta_{mh}}{\mathcal{N}_h}\mathcal{S}_h^o & 0 \\ 0 & 0 & \frac{c\beta_{pm}}{\mathcal{N}_p}\mathcal{S}_m^o \\ 0 & \frac{c\beta_{mp}}{\mathcal{N}_p}\mathcal{S}_p^o & 0 \end{bmatrix} \text{ and } \mathcal{V} = \begin{bmatrix} (\rho+\gamma+\mu_h) & 0 & 0 \\ 0 & \mu_m & 0 \\ 0 & 0 & \mu_p \end{bmatrix},$$

this implies that

$$\rho(\mathcal{F}\mathcal{V}^{-1}) = \sqrt{\frac{c^2\mathcal{N}_m\beta_{pm}\beta_{mp}}{\mathcal{N}_p\mu_p\mu_m}},$$

replace $\rho\mathcal{F}\mathcal{V}^{-1}$ by $\mathcal{R}_0$, we have the below endemic indicator:

$$\mathcal{R}_0 = \sqrt{\frac{c^2\mathcal{N}_m\beta_{pm}\beta_{mp}}{\mathcal{N}_p\mu_p\mu_m}}.$$

Theorem 3.2. *The steady state $\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable for other case.*

This result can be smoothly proved using the Jacobian matrix approach for $\mathcal{R}_0 < 1$, which ensures that the infection vanishes from the community.

4. Existence Theory

For the fractional model (3.2), we applied the theory of fixed-point to verify the solution's existence and uniqueness. In order to do this, we rewrite the system (3.2) as follows:

$$\begin{cases} {}_0^{ABC}D_t^{\upsilon}\nu(t) = \mathcal{H}(t, \nu(t)), \\ \nu(0) = \nu_0, \quad 0 < t < T < \infty. \end{cases} \tag{4.1}$$

In system (4.1), states variables are the vectors $\nu(t) = (\mathcal{S}_h, \mathcal{I}_h, \mathcal{R}_h, \mathcal{S}_m, I_m, \mathcal{S}_p, \mathcal{I}_p)$ represents the and $\mathcal{H}$ denotes the vector function which is also continuous and is given as:

$$\mathcal{H} = \begin{pmatrix} \mathcal{H}_1 \\ \mathcal{H}_2 \\ \mathcal{H}_3 \\ \mathcal{H}_4 \\ \mathcal{H}_5 \\ \mathcal{H}_6 \\ \mathcal{H}_7 \end{pmatrix} = \begin{pmatrix} \Xi - \frac{b\beta_{mh}}{\mathcal{N}_h}\mathcal{I}_m\mathcal{S}_h + \rho\mathcal{I}_h - \rho\mathcal{I}_h\tau_1 + \alpha\mathcal{R}_h - \mu_h\mathcal{S}_h \\ \frac{b\beta_{mh}}{\mathcal{N}_h}\mathcal{I}_m\mathcal{S}_h - \rho\mathcal{I}_h - \gamma\mathcal{I}_h - \mu_h\mathcal{I}_h \\ \rho\mathcal{I}_h\tau_1 - \alpha\mathcal{R}_h - \mu_h\mathcal{R}_h \\ \mu_m\mathcal{N}_m - \frac{c\beta_{pm}}{\mathcal{N}_p}\mathcal{I}_p\mathcal{S}_m - \mu_m\mathcal{S}_m \\ \frac{c\beta_{pm}}{\mathcal{N}_p}\mathcal{I}_p\mathcal{S}_m - \mu_m\mathcal{I}_m \\ \mu_p\mathcal{N}_p - \frac{c\beta_{mp}}{\mathcal{N}_p}\mathcal{I}_m\mathcal{S}_p - \mu_p\mathcal{S}_p \\ \frac{c\beta_{mp}}{\mathcal{N}_p}\mathcal{I}_m\mathcal{S}_p - \mu_p\mathcal{I}_p \end{pmatrix},$$

state variables with initial condition: $w_0(t) = (\mathcal{S}_h(0), \mathcal{I}_h(0), \mathcal{R}_h(0), \mathcal{S}_m(0), \mathcal{I}_m(0), \mathcal{S}_p(0), \mathcal{I}_p(0))$. The Lipschitz condition $\mathcal{H}$, is described below

$$\|\mathcal{H}(t, \nu_1(t)) - \mathcal{H}(t, \nu_2(t))\| \leqslant \mathcal{N}\|\nu_1(t) - \nu_2(t)\|. \tag{4.2}$$

The system (3.2) of the fractional model is examined for existence and uniqueness in the following result:

Theorem 4.1. *The solution of* (3.2) *is unique if the criteria listed below is met*

$$\frac{(1-\upsilon)}{ABC(\upsilon)}\mathcal{N} + \frac{\upsilon}{\Gamma(\upsilon)ABC(\upsilon)}T_{max}^{\upsilon}\mathcal{N} < 1. \tag{4.3}$$

Proof. First, we use the ABC fractional integral of (4.1) to get the non-linear Volterra integral equation, we obtain the below

$$\nu(t) = \frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}(t, \nu(t)) + \nu_0 + \frac{\upsilon}{\Gamma(\upsilon)ABC(\upsilon)}\int_0^t \mathcal{H}(\xi, \nu(\xi))(t-\xi)^{\upsilon-1}d\xi. \tag{4.4}$$

Suppose $V = (0, T)$ and the operator $\Upsilon : \mathcal{C}(W, R^7) \to \mathcal{C}(V, R^7)$ presented as

$$\Upsilon[\nu(t)] = \nu_0 + \mathcal{H}(t, \nu(t))\frac{1-\upsilon}{ABC(\upsilon)} + \frac{\upsilon}{\Gamma(\upsilon)ABC(\upsilon)}\int_0^t (t-\xi)^{\upsilon-1}\mathcal{H}(\nu(\xi), \xi,)d\xi.$$

Next, equation (4.4) transforms as:

$$\nu(t) = \Upsilon[\nu(t)]. \tag{4.5}$$

In this case, $\|.\|_V$ denotes the supremum norm on V, and is provided by

$$\|\nu(t)\|_V = \sup_{t\in V}\|\nu(t)\|, \quad \nu(t) \in \mathcal{C}.$$

It is obvious that a Banach space with the norm $\|.\|_V$ is formed by $\mathcal{C}(V, R^7)$. Additionally, it satisfies the below

$$\left\|\int_0^t \nu(\xi)\mathcal{G}(t, \xi)d\xi\right\| \leqslant T\|\mathcal{G}(t, \xi)\|_V\|\nu(t)\|_V, \tag{4.6}$$

considering $\mathcal{G}(t, \xi) \in \mathcal{C}(V^2, R)$, $\nu(t) \in \mathcal{C}(V, R^7)$, in a way that

$$\|\mathcal{G}(t, \xi)\|_V = \sup_{t,\xi\in V}|\mathcal{G}(t, \xi)|.$$

Applying (4.5), we get

$$\begin{aligned}\|\Upsilon[\nu_1(t)] - \Upsilon[\nu_2(t)]\|_V \leqslant \Bigg\| &\frac{(1-\upsilon)}{ABC(\upsilon)}(\mathcal{H}(t, \nu_1(t)) - \mathcal{H}(t, \nu_2(t)) + \frac{\upsilon}{ABC(\upsilon)\Gamma(\upsilon)} \\ &\times \int_0^t (\mathcal{H}(\xi, \nu_1(\xi)) - \mathcal{H}(\xi, \nu_2(\xi)))(t-\xi)^{\upsilon-1}d\xi\Bigg\|_V.\end{aligned}$$

The following is obtained using triangular inequality (4.2) and (4.6):

$$\|\Upsilon[\nu_1(t)] - \Upsilon[\nu_2(t)]\|_V \leqslant \left(\frac{(1-\upsilon)\mathcal{N}}{ABC(\upsilon)} + \frac{\upsilon}{\Gamma(\upsilon)ABC(\upsilon)}\mathcal{N}T_{max}^{\upsilon}\right)\|\nu_1(t) - \nu_2(t)\|_V.$$

Consequently, we obtain

$$\|\Upsilon[\nu_1(t)] - \Upsilon[\nu_2(t)]\|_V \leqslant B\|\nu_1(t) - \nu_2(t)\|_V,$$

in which

$$B = \frac{(1-\upsilon)\mathcal{N}}{ABC(\upsilon)} + \frac{\upsilon}{ABC(\upsilon)\Gamma(\upsilon)}\mathcal{N}T_{max}^{\upsilon}.$$

In this case, Υ is contraction if it satisfies the requirement in (4.3). Consequently, the system (4.1) has a unique solution. □

5. Numerical Method for Solutions

Here, a numerical scheme is presented to highlight the solution pathways of the system (3.2) of the infection. This approach is obtained by combining a two-step Lagrange polynomial [19]. This approach was then used to develop an iterative numerical technique for the system (3.2) of JE. Through the fundamental theorem, we have the following

$$\nu(t)-\nu(0)=\frac{\upsilon}{\Gamma(\upsilon)\times ABC(\upsilon)}\int_0^t(t-\xi)^{\upsilon-1}\mathcal{G}(\xi,\nu(\xi)d\xi+\frac{(1-\upsilon)}{ABC(\upsilon)}\mathcal{G}(t,\nu(t)).$$

At time $t=t_{n+1}$, where $n=0,1,2,...$, gives:

$$\begin{aligned}\nu(t_{n+1})-\nu(0)=&\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}(t_n,\nu(t_n))\\&+\frac{\upsilon}{ABC(\upsilon)\times\Gamma(\upsilon)}\int_0^{t_{n+1}}\mathcal{H}(\xi,\nu(\xi))(t_{n+1}-\xi)^{\upsilon-1}d\xi,\\=&\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}(t_n,\nu(t_n))\\&+\frac{\upsilon}{ABC(\upsilon)\times\Gamma(\upsilon)}\sum_{\nu=0}^{n}\int_{t_\nu}^{t_{\nu+1}}\mathcal{H}(\xi,\nu(\xi))(t_{n+1}-\xi)^{\upsilon-1}d\xi.\end{aligned}\tag{5.1}$$

Over the interval $[t_\nu,t_{\nu+1}]$, we approximate $\mathcal{H}(\xi,\nu(\xi))$ as:

$$\mathcal{H}(\xi,\nu(\xi))\cong P_k(\xi)=\frac{\mathcal{H}(t_\nu,\nu(t_\nu))}{h}(\xi-t_{\nu-1})-\frac{\mathcal{H}(t_{\nu-1},\nu(t_{\nu-1}))}{h}(\xi-t_\nu).$$

Then, the equation (5.1) can expressed in the below way

$$\begin{aligned}\nu(t_{n+1})=&\nu(0)+\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}(t_n,\nu(t_n))\\&+\frac{\upsilon}{ABC(\upsilon)\times\Gamma(\upsilon)}\sum_{\nu=0}^{n}\Big(\frac{\mathcal{H}(t_\nu,\nu(t_\nu))}{h}\int_{t_\nu}^{t_{\nu+1}}(\xi-t_{\nu-1})(t_{n+1}-\xi)^{\upsilon-1}d\xi\\&-\frac{\mathcal{H}(t_{\nu-1},\nu(t_{\nu-1}))}{h}\int_{t_\nu}^{t_{\nu+1}}(\xi-t_\nu)(t_{n+1}-\xi)^{\upsilon-1}d\xi\Big).\end{aligned}$$

After simplifying, we obtain the following

$$\begin{aligned}\nu(t_{n+1})=&\nu(t_0)+\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}(t_n,\nu(t_n))\\&+\frac{\upsilon}{ABC(\upsilon)}\sum_{\nu=0}^{n}\Big(\frac{h^\upsilon\mathcal{H}(t_\nu,\nu(t_\nu))}{\Gamma(\upsilon+2)}((\upsilon-\nu+2+n)(1-\nu+n)^\upsilon-(2\upsilon+n-\nu+2)(n-\nu)^\upsilon)\\&-\frac{h^\upsilon\mathcal{H}(t_{\nu-1},\nu(t_{\nu-1}))}{\Gamma(\upsilon+2)}((1-\nu+n)^{\upsilon+1}-(n+1-\nu+\upsilon)(n-\nu)^\upsilon)\Big).\end{aligned}$$

We obtain the recursive formulas as:

$$\begin{aligned}\mathcal{S}_h(t_{n+1})=&\mathcal{S}_h(t_0)+\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}_1(t_n,\nu(t_n))\\&+\frac{\upsilon}{ABC(\upsilon)}\sum_{\nu=0}^{n}\Big(\frac{h^\upsilon\mathcal{H}_1(t_\nu,\nu(t_\nu))}{\Gamma(\upsilon+2)}((1+n-\nu)^\upsilon(\upsilon-\nu+n+2)-(n-\nu)^\upsilon(2\upsilon-\nu+2+n))\\&-\frac{h^\upsilon\mathcal{H}_1(t_{\nu-1},\nu(t_{\nu-1}))}{\Gamma(\upsilon+2)}((1+n-\nu)^{\upsilon+1}-(n-\nu)^\upsilon(\upsilon-\nu+n+1))\Big),\end{aligned}$$

$$\begin{aligned}\mathcal{I}_h(t_{n+1}) =&\mathcal{I}_h(t_0)+\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}_2(t_n,\nu(t_n))\\&+\frac{\upsilon}{ABC(\upsilon)}\sum_{\nu=0}^{n}\Big(\frac{h^\upsilon\mathcal{H}_2(t_\nu,\nu(t_\nu))}{\Gamma(\upsilon+2)}((\upsilon-\nu+2+n)(n+1-\nu)^\upsilon-(n-\nu)^\upsilon(2\upsilon-\nu+2+n))\\&-\frac{h^\upsilon\mathcal{H}_2(t_{\nu-1},\nu(t_{\nu-1}))}{\Gamma(\upsilon+2)}((1+n-\nu)^{\upsilon+1}-(\upsilon+n+1-\nu)(n-\nu)^\upsilon)\Big),\end{aligned}$$

$$\begin{aligned}\mathcal{R}_h(t_{n+1}) =&\mathcal{R}_h(t_0)+\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}_3(t_n,\nu(t_n))\\&+\frac{\upsilon}{ABC(\upsilon)}\sum_{\nu=0}^{n}\Big(\frac{h^\upsilon\mathcal{H}_3(t_\nu,\nu(t_\nu))}{\Gamma(\upsilon+2)}((-\nu+1+n)^\upsilon(\upsilon+n-\nu+2)-(n-\nu+2+2\upsilon)(n-\nu)^\upsilon)\\&-\frac{h^\upsilon\mathcal{H}_3(t_{\nu-1},\nu(t_{\nu-1}))}{\Gamma(\upsilon+2)}((n-\nu+1)^{\upsilon+1}-(\upsilon+n+1-\nu)(n-\nu)^\upsilon)\Big),\end{aligned}$$

$$\begin{aligned}\mathcal{S}_m(t_{n+1}) =&\mathcal{S}_m(t_0)+\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}_4(t_n,\nu(t_n))\\&+\frac{\upsilon}{ABC(\upsilon)}\sum_{\nu=0}^{n}\Big(\frac{h^\upsilon\mathcal{H}_4(t_\nu,\nu(t_\nu))}{\Gamma(\upsilon+2)}((\upsilon+n-\nu+2)(n-\nu+1)^\upsilon-(n-\nu)^\upsilon(2\upsilon-\nu+2+n))\\&-\frac{h^\upsilon\mathcal{H}_4(t_{\nu-1},\nu(t_{\nu-1}))}{\Gamma(\upsilon+2)}((n+1-\nu)^{\upsilon+1}-(\upsilon+n+1-\nu)(n-\nu)^\upsilon)\Big),\end{aligned}$$

$$\begin{aligned}\mathcal{I}_m(t_{n+1}) =&\mathcal{I}_m(t_0)+\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}_5(t_n,\nu(t_n))\\&+\frac{\upsilon}{ABC(\upsilon)}\sum_{\nu=0}^{n}\Big(\frac{h^\upsilon\bigtriangledown_4(t_\nu,\nu(t_\nu))}{\Gamma(\upsilon+2)}((\upsilon+n-\nu+2)(n+1-\nu)^\upsilon-(2\upsilon-\nu+2+n)(n-\nu)^\upsilon)\\&-\frac{h^\upsilon\bigtriangledown_4(t_{\nu-1},\nu(t_{\nu-1}))}{\Gamma(\upsilon+2)}((n-\nu+1)^{\upsilon+1}-(n-\nu)^\upsilon(\upsilon+n+1-\nu))\Big),\end{aligned}$$

$$\begin{aligned}\mathcal{S}_p(t_{n+1}) =&\mathcal{S}_p(t_0)+\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}_6(t_n,\nu(t_n))\\&+\frac{\upsilon}{ABC(\upsilon)}\sum_{\nu=0}^{n}\Big(\frac{h^\upsilon\mathcal{H}_6(t_\nu,\nu(t_\nu))}{\Gamma(\upsilon+2)}((1-\nu+n)^\upsilon(n+\upsilon-\nu+2)-(n-\nu+2+2\upsilon)(n-\nu)^\upsilon)\\&-\frac{h^\upsilon\mathcal{H}_6(t_{\nu-1},\nu(t_{\nu-1}))}{\Gamma(\upsilon+2)}((1-\nu+n)^{\upsilon+1}-(\upsilon+n-\nu+1)(n-\nu)^\upsilon)\Big),\end{aligned}$$

and

$$\begin{aligned}\mathcal{I}_p(t_{n+1}) =&\mathcal{I}_p(t_0)+\frac{1-\upsilon}{ABC(\upsilon)}\mathcal{H}_7(t_n,\nu(t_n))+\\&\frac{\upsilon}{ABC(\upsilon)}\sum_{\nu=0}^{n}\Big(\frac{h^\upsilon\mathcal{H}_7(t_\nu,\nu(t_\nu))}{\Gamma(\upsilon+2)}((1-\nu+n)^\upsilon(\upsilon-\nu+n+2)-(n-\nu+2+2\upsilon)(n-\nu)^\upsilon)\\&-\frac{h^\upsilon\mathcal{H}_7(t_{\nu-1},\nu(t_{\nu-1}))}{\Gamma(\upsilon+2)}((1-\nu+n)^{\upsilon+1}-(\upsilon-\nu+n+1)(n-\nu)^\upsilon)\Big).\end{aligned}$$

The suggested fractional system of the infection is represented graphically using these approximations. We will carry out many simulations for the suggested system in order to understand the dynamic behaviour of our JE fractional model. For simulation proposals, the parameter values are assumed while the state variable values are taken to be $\mathcal{S}_h = 500, \mathcal{I}_h = 50, \mathcal{R}_h = 30, \mathcal{S}_m = 300$, $\mathcal{I}_m = 120$, $\mathcal{S}_p = 400$ and $\mathcal{I}_p = 150$. We ran four scenarios with the main objectives of estimating infection levels, evaluating the

relevance of these numbers, and forecasting efficient control strategies for avoiding JE infections in order to see how various configurations affected the system's output.

Different scenarios were performed with the main objectives of estimating infection levels, assessing the relevance of these values, and forecasting efficient control strategies to mitigate JE infections. In the first scenario, illustrated in Figures 1-3, we demonstrated the impact of fractional-order dynamics on the system, particularly highlighting the system's sensitivity to changes in the memory index. This parameter notably influenced infection levels, with observed reductions as the memory index varied. The value of υ is assumed to be 0.8, 0.9, and 1.0 in the first simulation presented in Figure 1 while the values of fractional parameters are taken to be 0.6, 0.7 and 0.7 in the Figure 2. In Figure 3, we assumed the value of υ to be 0.4, 0.5 and 0.6 in order to show the impact this parameter on the infection level.

In Figure 4, the impact of biting rate on the solution pathways has been shown where we assumed 0.24, 0.34 and 0.44. It is noticed that the increase of this parameter is dangerous for the system. Therefore, we suggested to control this factor through some control measures. The third and fourth scenarios, shown in Figures 5 and 6, involved varying parameters α and ρ to evaluate their influence on JE infection rates, respectively. In Figure 5, α is taken to be 0.023, 0.033 and 0.43 while ρ is assumed to be 0.0215, 0.0315 and 0.0415 in Figure 6. Our findings indicate that immunity loss contribute significantly in complicating efforts to control infections, particularly in diseases where waning immunity affects individual susceptibility over time. As immunity diminishes, individuals become more vulnerable to reinfection, which sustains transmission within the population and can even lead to outbreaks if left unchecked. Additionally, the biting rate directly influences the infection's spread, as higher biting rates facilitate more opportunities for transmission and intensify the risk of infection. Interestingly, our study highlights the utility of fractional parameters in controlling infection dynamics. The inclusion of fractional derivatives allows for a more accurate representation of the memory effect within the system. Thus, the combination of managing immunity decay, controlling the biting rate, and utilizing fractional calculus offers a comprehensive approach to effectively mitigating the infection's impact within affected populations.

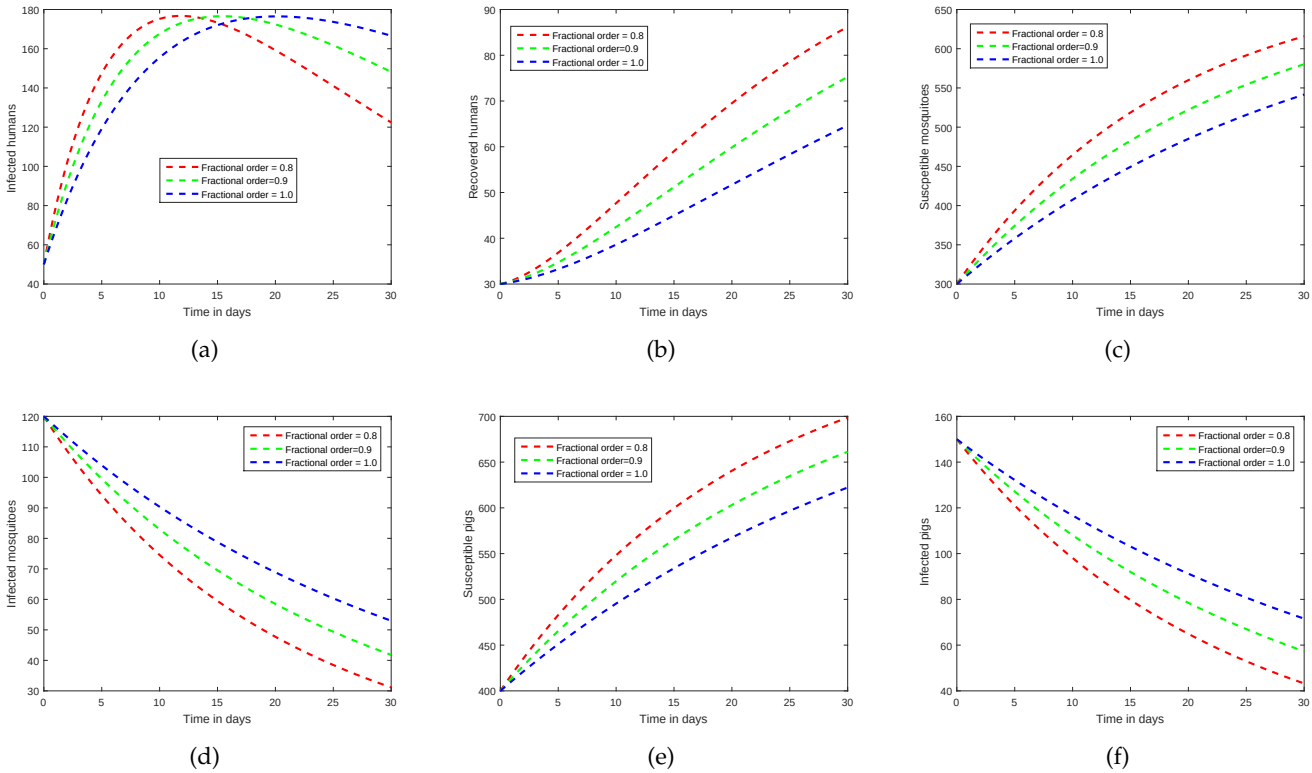


Figure 1: Plotting the solution pathways of the proposed model of the infection with the variation of input factor υ, i.e. $\upsilon = 0.8, 0.9, 1$.

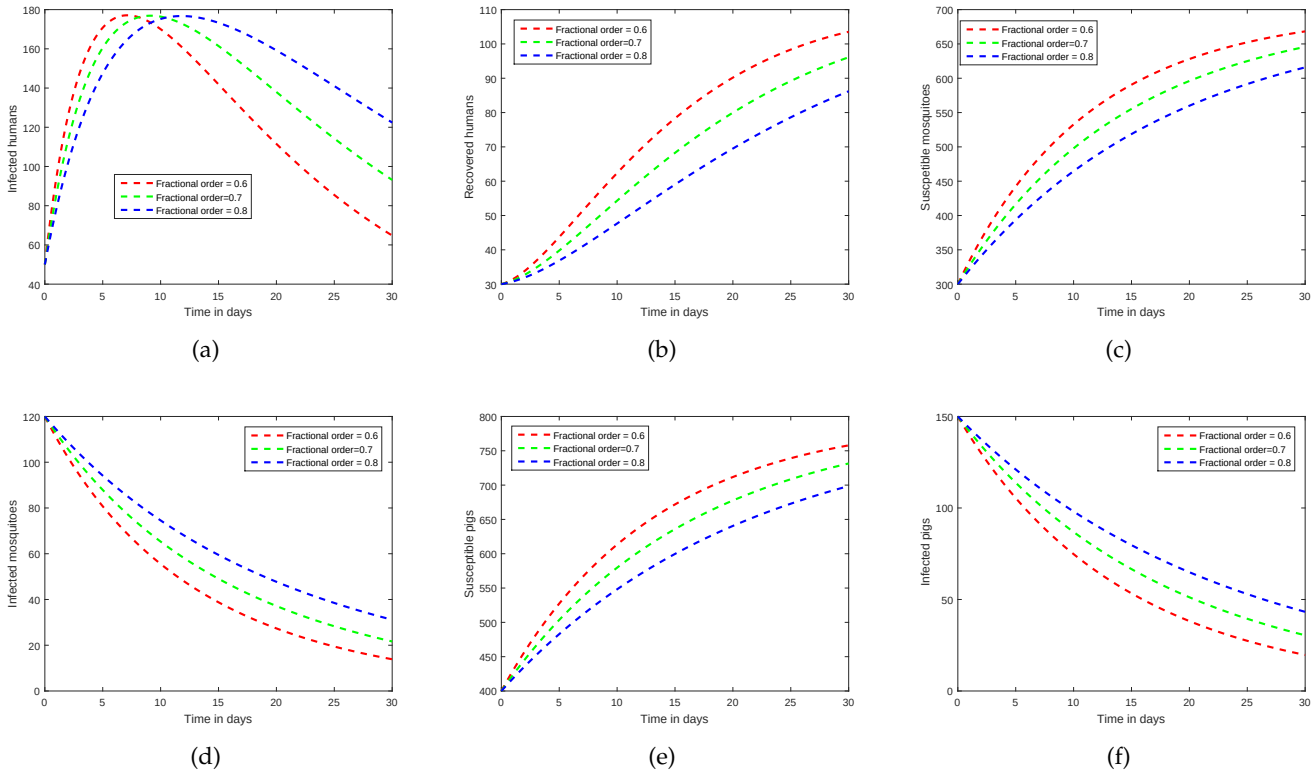


Figure 2: Plotting the solution pathways of the proposed model of the infection with the variation of input factor υ, $\upsilon = 0.6, 0.7, 0.8$.

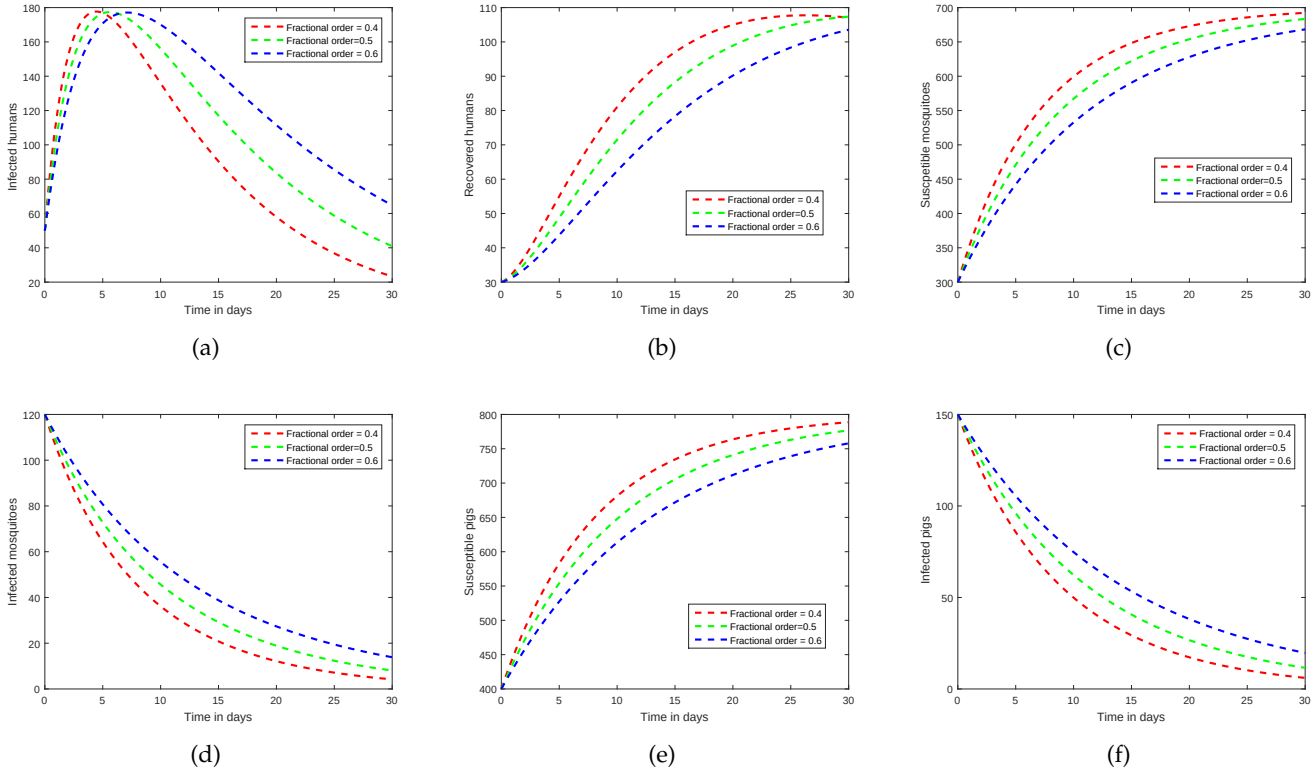
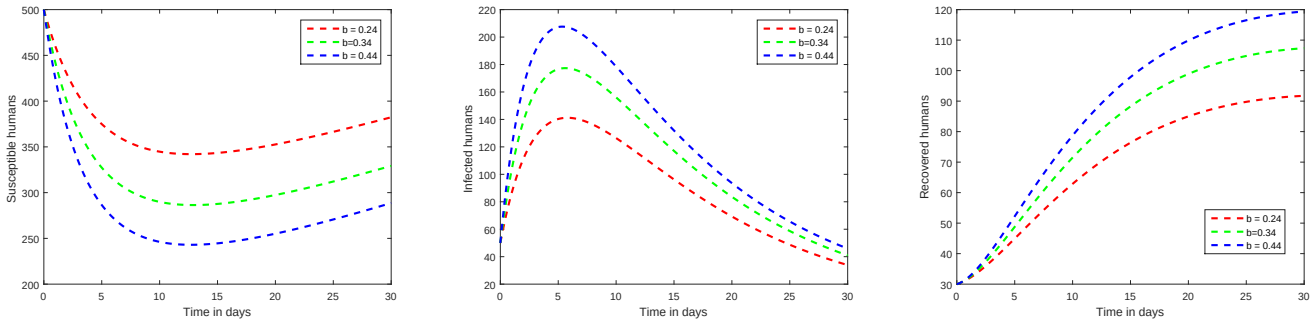
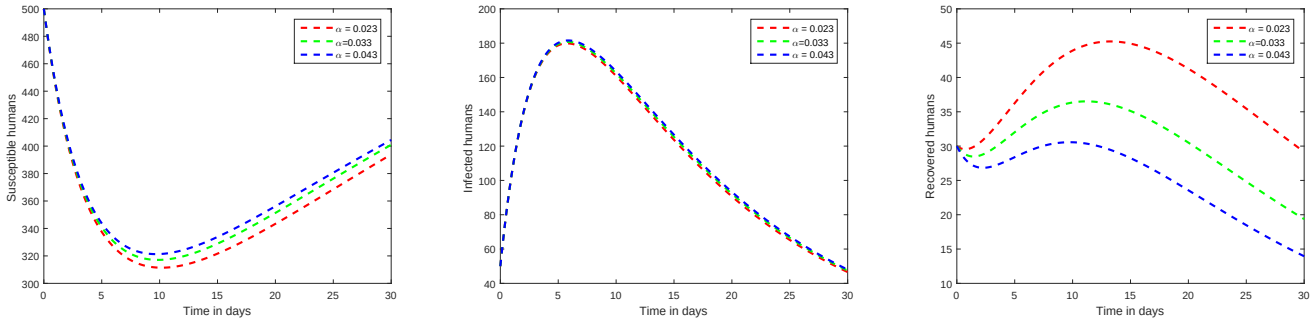


Figure 3: Representation of the time series of the proposed model of JE with different values of input factor υ, i.e. $\upsilon = 0.4, 0.5, 0.6$.



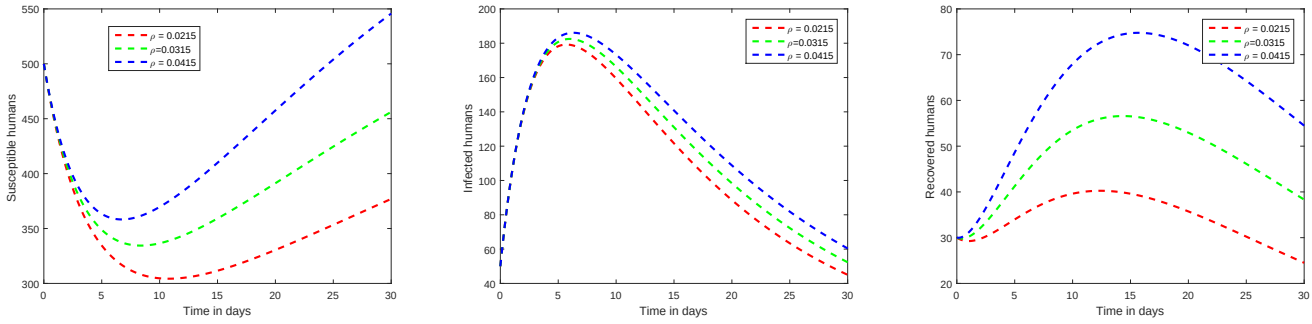
(a) (b) (c)

Figure 4: Graphical view analysis of the proposed Japanese encephalitis infection model with different values of input parameter b, i.e. $b = 0.24, 0.34, 0.44$.



(a) (b) (c)

Figure 5: A graphical depiction of the suggested Japanese encephalitis infection model, specifically with changing the parameter α, i.e., $\alpha = 0.023, 0.033, 0.043$, is used for analysis.



(a) (b) (c)

Figure 6: Graph-based examination of the suggested JE infection model utilising varying input parameter ρ values, namely $\rho = 0.0215, 0.0315, 0.0415$.

6. Conclusion

The burden of infectious diseases remains a significant global health challenge, particularly in low- and middle-income countries where healthcare resources are often limited. Therefore, it is important to examine infectious diseases through modeling to understand the dynamics of infections and develop effective control strategies. In this work, we structured the dynamics of Japanese encephalitis (JE) by using the Atangana-Baleanu fractional derivative with the effect of waning immunity. The related theory of fractional calculus has been presented to investigate the model. We computed $\mathcal{R}_0$ for the suggested fractional model using the next-generation technique. Analytical skills are used to investigate different

aspects of the solution of the system of JE infection. We examined the existence and uniqueness of the solution of fractional-order system using fixed-point theory and a recently developed numerical method for iterative solutions. The effect of various parameters on the dynamics of the infection was demonstrated through numerical analysis. The findings of this work visualized the key factors of the system and suggested effective control measures. In future work, the framework of impulsive differential equations will be utilized to investigate the effects of pulse vaccination on infection dynamics.

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