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Reproduction Number and Stability of a Caputo Fractional Derivative Immuno-Metabolic Infection Model for Cats with Obesity and T2DM Effects

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Abstract: We have study chronic inflammation, obesity and type 2 diabetes mellitus (T2DM) [33] are increasingly recognized as key immuno-metabolic determinants of host susceptibility, infection progression, and recovery in companion animals. Motivated by these mechanisms, we formulate a fractional-order immuno-metabolic epidemic model for infection transmission in cats stratified by metabolic health status. The model employs Caputo derivatives of order $0 < \xi < 1$ to capture memory-dependent immunological and inflammatory dynamics, including adipokine-driven chronic inflammation, obesity-induced metabolic impairment, and T2DM-associated immune dysfunction. The feline population is divided into metabolically healthy and metabolically impaired groups, each with susceptible, infected, and recovered classes, together with an additional variable describing systemic inflammation. Using the next-generation matrix method, we derive the basic reproduction number R_0, explicitly decomposing contributions from healthy and metabolically impaired hosts. Analytical results show that metabolic impairment and inflammation enhance transmission through increased susceptibility and infectiousness. Within a fractional Lyapunov framework, the disease-free equilibrium is locally and globally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. The proposed model provides a rigorous mathematical framework for quantifying how chronic inflammation and metabolic disorders modulate infection dynamics in feline populations, underscoring the importance of incorporating immuno-metabolic factors into epidemic models to improve disease burden prediction and design targeted interventions for cats with obesity and T2DM. And plot the figure by the help of python.
Keywords: Fractional order(Caputo Fractional Derivative), T2DM Effects, Stability analysis, Reproduction Number.

1. Introduction

Metabolic dysregulation and infectious diseases are increasingly recognized as interconnected processes affecting both human and veterinary health. Accumulating evidence demonstrates that obesity, chronic low-grade inflammation, and type 2 diabetes mellitus (T2DM) substantially impair immune function, modifying susceptibility to infection and influencing disease severity and outcomes [9, 16, 22]. In humans, these immuno-metabolic interactions have been widely documented, but studies in companion animals, particularly domestic cats, remain relatively limited [21, 26, 30]. Recent epidemiological surveys reveal a growing prevalence of obesity and T2DM in domestic cats due to lifestyle changes, decreased physical activity, and altered dietary patterns [33]. Obesity in cats is strongly associated with insulin resistance and hyperglycemia, leading to chronic T2DM, which in turn compromises innate and adaptive immunity. Affected cats exhibit higher circulating levels of pro-inflammatory cytokines, persistent systemic inflammation, and impaired antiviral defense, which can exacerbate the course of infectious diseases [13]. This bidirectional relationship between metabolic dysfunction and infection underscores the need for models that integrate both physiological and epidemiological factors. Classical epidemic models typically partition host populations based solely on infection status and demographic characteristics, overlooking physiological factors such as obesity, chronic inflammation, and T2DM, which can crucially influence disease dynamics [25, 27]. In feline populations, the coexistence of metabolically healthy and impaired individuals creates complex feedback loops: metabolic dysfunction increases susceptibility to infection, infection accelerates inflammatory processes, and persistent inflammation further worsens metabolic health. These

interactions can amplify disease transmission, maintain persistent infection cycles, and complicate control measures. Systemic inflammation, often fueled by excess adipose tissue acting as an endocrine organ, plays a central role in linking obesity, T2DM, and immune dysregulation [32]. Chronic inflammation impairs immune memory and antiviral responses, prolonging infection and slowing recovery in domestic cats. Such processes highlight the importance of incorporating immuno-metabolic feedbacks in predictive epidemic models. Fractional-order calculus, especially Caputo derivatives, has recently gained prominence in biological and epidemiological modeling due to its ability to capture memory and hereditary effects [1, 3, 6, 12, 31, 34]. Fractional epidemic models have been shown to improve the fit to empirical data and produce more realistic predictions for diseases with cumulative or delayed effects, making them particularly suitable for modeling the slow progression of metabolic dysfunction and chronic inflammation in cats [7, 15, 20]. In this work, we develop a fractional-order immuno-metabolic epidemic model for domestic cats that integrates infection dynamics, metabolic state transitions, inflammation-driven susceptibility, and T2DM-related immune dysfunction. The proposed framework partitions the host population into metabolically distinct compartments and explicitly models transitions due to obesity and T2DM, with susceptibility functions modulated by inflammation and governed by Caputo fractional derivatives. Major contributions of this study include: (i) formulation of a seven-compartment model for cats, (ii) analytical derivation and interpretation of equilibrium states, (iii) computation of a basic reproduction number incorporating metabolic and inflammatory effects, (iv) stability analysis of the disease-free equilibrium, and (v) assessment of how obesity and T2DM influence the persistence of infection. To the best of our knowledge, this model is the first to unify fractional epidemic theory with immuno-metabolic mechanisms in domestic cats, providing a robust framework for veterinary epidemiology and feline health management.Type 2 diabetes mellitus (T2DM) in domestic cats not only affects metabolic homeostasis but also has significant epidemiological implications for infectious disease dynamics. Cats with T2DM commonly exhibit insulin resistance and chronic hyperglycemia, conditions that compromise both innate and adaptive immune responses [33]. Hyperglycemia and associated metabolic dysregulation promote the overproduction of pro-inflammatory cytokines such as TNF-α, IL-6, and IL-1β, creating a state of persistent systemic inflammation [30]. This cytokine dysregulation impairs leukocyte function, reduces the efficacy of pathogen clearance, and delays recovery from infections. Epidemiologically, such immuno-metabolic impairment increases susceptibility to common feline pathogens, prolongs infectious periods, and may enhance secondary transmission within domestic and shelter populations [14,26]. Consequently, T2DM acts as both a direct and indirect driver of infection risk and persistence, highlighting the necessity of incorporating metabolic and inflammatory parameters into feline epidemic models. Understanding these mechanisms is essential for predicting outbreak dynamics, optimizing preventive strategies, and tailoring clinical interventions for metabolically compromised cats.

1.1. Structure of Paper

The rest of the paper proceeds as follows: Section 1 introduces the mathematical formulation of the Immuno-Metabolic Infection Model for Cats with Obesity and T2DM Effects, Section 2 preliminaries, Section 3 Analysis of the Model, Section 4 non negativity and uniform boundedness Section 5 details the stability analysis, Section 6 basic reproduction number, Section 7 numerical simulations and diagrams.

1.2. Preliminaries: Caputo derivative

Let $0 < \xi < 1$ be the fractional order. The Caputo fractional derivative of order ξ for a sufficiently smooth function $f(t)$ is defined [18, 19] by

$$ {}^{C}D_{0^+}^{\xi} f(t) = \frac{1}{\Gamma(1-\xi)} \int_0^t \frac{f'(s)}{(t-s)^{\xi}}\, ds, \qquad t > 0, $$

where $\Gamma(\cdot)$ is the Gamma function. For $\xi = 1$ this reduces to the standard first derivative.

2. Mathematical Model Formulation

Based on the biological mechanisms, we formulate a compartmental immuno-metabolic epidemic model that integrates the effects of chronic inflammation, obesity and type 2 diabetes mellitus (T2DM) in cats on infection dynamics. The model accounts for the bidirectional interactions between metabolic dysregulation and immune system activation.

2.1. Model Structure

We divide the total host population $N(t)$ into two major subpopulations: metabolically healthy and metabolically impaired individuals (the latter representing those with obesity and/or T2DM) in cats. Each subpopulation is further classified according to the epidemiological status susceptible, infected, or recovered. Hence,

$$N(t) = S_H(t) + I_H(t) + R_H(t) + S_M(t) + I_M(t) + R_M(t),$$

where the subscripts H and M denote the metabolically healthy and metabolically impaired groups, respectively. An additional variable, $A(t)$ represents the systemic level of inflammation driven by adipokines and cytokines.

Model Equations

The model dynamics are governed by the following nonlinear system of differential equations [2]:

$$\begin{cases} \frac{dS_H}{dt} &= \Lambda - \lambda_H S_H - \rho S_H + \phi S_M - \mu S_H, \\ \frac{dI_H}{dt} &= \lambda_H S_H - (\gamma_H + \alpha_H + \mu) I_H, \\ \frac{dR_H}{dt} &= \gamma_H I_H - (\mu + \eta) R_H + \eta R_M, \\ \frac{dS_M}{dt} &= \rho S_H - \lambda_M S_M - \phi S_M - \mu S_M, \\ \frac{dI_M}{dt} &= \lambda_M S_M - (\gamma_M + \alpha_M + \mu) I_M, \\ \frac{dR_M}{dt} &= \gamma_M I_M - (\mu + \eta) R_M + \eta R_H, \\ \frac{dA}{dt} &= c_O(S_M + I_M + R_M) + p_H I_H + p_M I_M - \delta_A A. \end{cases} \tag{1}$$

Biological Interpretation

The system captures several biological mechanisms consistent with immunometabolic evidence:

- Chronic inflammation (A) is sustained by cytokines secreted from infected cells $(p_H I_H, p_M I_M)$ and adipose tissue (c_O) with metabolic clearance at rate δ_A.
- The metabolically impaired class (S_M, I_M, R_M) experiences higher baseline inflammation and reduced immune efficiency.
- Increased inflammatory load enhances susceptibility $\sigma(A)$, reduces recovery γ_M and increases disease-induced mortality α_M.
- Transitions between metabolic states are governed by ρ (progression to obesity/T2DM) and ϕ (reversal via therapeutic intervention).

2.2. Fractional-order Model with Caputo Derivative

Fractional model equations Replace each time derivative by the Caputo derivative of order ξ. The fractional immune metabolic epidemic model reads [2, 33] explain fig.(1, 12)

$$\begin{cases} {}^{C}D^{\xi}_{0^+} S_H(t) &= \Lambda - \lambda_H(t)\, S_H(t) - \rho\, S_H(t) + \phi\, S_M(t) - \mu\, S_H(t), \\ {}^{C}D^{\xi}_{0^+} I_H(t) &= \lambda_H(t)\, S_H(t) - (\gamma_H + \alpha_H + \mu)\, I_H(t), \\ {}^{C}D^{\xi}_{0^+} R_H(t) &= \gamma_H\, I_H(t) - (\mu + \eta)\, R_H(t) + \eta\, R_M(t), \\ {}^{C}D^{\xi}_{0^+} S_M(t) &= \rho\, S_H(t) - \lambda_M(t)\, S_M(t) - \phi\, S_M(t) - \mu\, S_M(t), \\ {}^{C}D^{\xi}_{0^+} I_M(t) &= \lambda_M(t)\, S_M(t) - (\gamma_M + \alpha_M + \mu)\, I_M(t), \\ {}^{C}D^{\xi}_{0^+} R_M(t) &= \gamma_M\, I_M(t) - (\mu + \eta)\, R_M(t) + \eta\, R_H(t), \\ {}^{C}D^{\xi}_{0^+} A(t) &= c_O\big(S_M(t) + I_M(t) + R_M(t)\big) + p_H I_H(t) + p_M I_M(t) - \delta_A A(t). \end{cases} \tag{2}$$

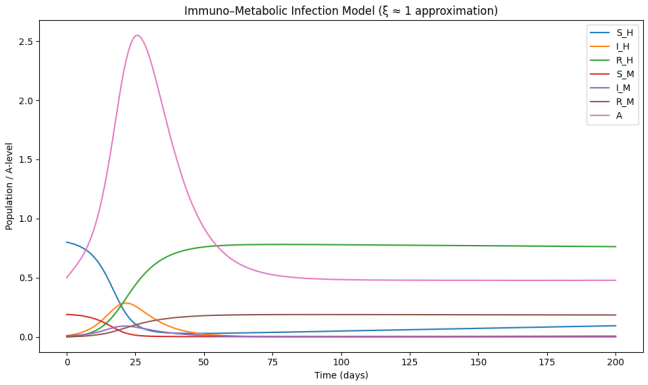

Figure 1: This 2d graph depicts an immuno-metabolic infection model.

Figure.(1) depicts an immuno-metabolic infection model showing the interaction between host compartments (S_H, L_H, R_H), metabolic states (S_M, I_M, R_M), and pathogen load (A) over 200 days. The pathogen population A exhibits a rapid spike around day 25, followed by decay, while the recovered host population R_H steadily increases and stabilizes, indicating successful immune clearance with some individuals reaching recovered metabolic states.

3. Existence, Uniqueness and Positivity of Solutions

Theorem 3.1. *Consider the Caputo fractional-order system of order* $0 < \xi < 1$ *[1, 2, 3].*

Proof. We are seeking for a sufficient condition for the presence and uniqueness of the proposed model solutions in the region $\Theta \times [0, \mathscr{T}]$, where
$\Theta = (S_H(t), I_H(t), R_H(t), S_M(t), I_M(t), R_M(t), A(t)) \in \mathbb{R}^7$
$from$: $\max ||S_H(t)||, ||I_H(t)||, ||R_H(t)||, ||S_M(t)||, ||I_M(t)||, ||R_M(t)||, ||A(t)||$. The method employed is used consider a mapping

$$
\begin{aligned}
{}^{C}D^{\xi}_{0^+} S_H(t) &= \mathscr{F}_1(t, X), \\
{}^{C}D^{\xi}_{0^+} I_H(t) &= \mathscr{F}_2(t, X), \\
{}^{C}D^{\xi}_{0^+} R_H(t) &= \mathscr{F}_3(t, X), \\
{}^{C}D^{\xi}_{0^+} S_M(t) &= \mathscr{F}_4(t, X), \\
{}^{C}D^{\xi}_{0^+} I_M(t) &= \mathscr{F}_5(t, X), \\
{}^{C}D^{\xi}_{0^+} R_M(t) &= \mathscr{F}_6(t, X), \\
{}^{C}D^{\xi}_{0^+} A(t) &= \mathscr{F}_7(t, X),
\end{aligned}
$$

where $X(t) = (S_H, I_H, R_H, S_M, I_M, R_M, A)^{\top}$

$$
||\mathscr{F}(Y)-\mathscr{F}(\hat{Y})|| = \begin{cases}
|\mathscr{F}_1(Y)-\mathscr{F}_1\hat{Y}|+|\mathscr{F}_2(Y)-\mathscr{F}_2\hat{Y}|+|\mathscr{F}_3(Y)-\mathscr{F}_3\hat{Y}|+|\mathscr{F}_4(Y)-\mathscr{F}_4\hat{Y}| \\
+|\mathscr{F}_5(Y)-\mathscr{F}_5\hat{Y}|+|\mathscr{F}_6(Y)-\mathscr{F}_6\hat{Y}|+|\mathscr{F}_7(Y)-\mathscr{F}_7\hat{Y}| \\
=|(\Lambda-\lambda_H(t)S_H-\rho S_H+\phi S_M-\mu S_H)-(\Lambda-\lambda_H(t)\hat{S_H}-\rho\hat{S_H}+\phi\hat{S_M}-\mu\hat{S_H})| \\
+|(\lambda_H(t)S_H-(\gamma_H+\alpha_H+\mu)I_H)-(\lambda_H(t)\hat{S_H}-(\gamma_H+\alpha_H+\mu)\hat{I_H})| \\
+|(\gamma_H I_H-(\mu+\eta)R_H+\eta R_M)-(\gamma_H\hat{I_H}-(\mu+\eta)\hat{R_H}+\eta\hat{R_M})| \\
+|(\rho S_H-\lambda_M(t)S_M-\phi S_M-\mu S_M)-(\rho\hat{S_H}-\lambda_M(t)\hat{S_M}-\phi\hat{S_M}-\mu\hat{S_M})| \\
+|(\lambda_M(t)S_M-(\gamma_M+\alpha_M+\mu)I_M)-(\lambda_M(t)\hat{S_M}-(\gamma_M+\alpha_M+\mu)\hat{I_M})| \\
+|(\gamma_M I_M-(\mu+\eta)R_M+\eta R_H)-(\gamma_M\hat{I_M}-(\mu+\eta)\hat{R_M}+\eta\hat{R_H})| \\
+|(c_O(S_M+I_M+R_M)+p_H I_H+p_M I_M-\delta_A A)-(c_O(\hat{S_M}+\hat{I_M}+\hat{R_M}) \\
+p_H\hat{I_H}+p_M\hat{I_M}-\delta_A\hat{A})| \\
\leq(\lambda_H(t)+\rho+\mu)|S_H-\hat{S_H}|+\phi|S_M-\hat{S_M}|+\lambda_H|S_H-\hat{S_H}| \\
+(\gamma_H+\alpha_H+\mu)|I_H-\hat{I_H}|+\gamma_H|I_H-\hat{I_H}|+(\mu+\eta)|R_H-\hat{R_H}|+\eta|R_M-\hat{R_M}| \\
+\rho|S_H-\hat{S_H}|+(\lambda_M(t)+\phi+\mu)|S_M-\hat{S_M}|+\lambda_M(t)|S_M-\hat{S_M}| \\
+(\gamma_M+\alpha_M+\mu)|I_M-\hat{I_M}|+(\gamma_M)|I_M-\hat{I_M}|+(\mu+\eta)|R_M-\hat{R_M}| \\
+\eta|R_H-\hat{R_H}|+p_H|I_H-I_H|+p_M|I_M-\hat{I_M}|+\delta_A|A-\hat{A}|+c_O|S_M-\hat{S_M}| \\
+c_O|I_M-\hat{I_M}|+c_O|R_M-\hat{R_M}| \\
\leq(2\lambda_H(t)+2\rho+\mu)|S_H-\hat{S_H}|+(2\gamma_H+\alpha_H+\mu+p_H)|I_H-\hat{I_H}| \\
+(\mu+2\eta)|R_H-\hat{R_H}|+(2\phi+2\lambda_M(t)+\mu+c_O)|S_M-\hat{S_M}| \\
+(2\gamma_M+\alpha_M-\mu+p_M+c_O)|I_M-\hat{I_M}|+(2\eta+\mu+c_O)|R_M-\hat{R_M}|+\delta_A|A-\hat{A}| \\
\leq\mathscr{F}_1|S_H-\hat{S_H}|+\mathscr{F}_2|I_H-\hat{I_H}|+\mathscr{F}_3|R_H-\hat{R_H}|+\mathscr{F}_4|S_M-\hat{S_M}| \\
+\mathscr{F}_5|I_M-\hat{I_M}|+\mathscr{F}_6|R_M-\hat{R_M}|+\mathscr{F}_7|A-\hat{A}| \\
\leq L\|\mathscr{F}(Y)-\mathscr{F}(\hat{Y})\|
\end{cases} \tag{3}
$$

where max $=(\mathscr{F}_1,\mathscr{F}_2,\mathscr{F}_3,\mathscr{F}_4,\mathscr{F}_5,\mathscr{F}_6,\mathscr{F}_7)$

$$
\begin{aligned}
\mathscr{F}_1 &= 2\lambda_H(t)+2\rho+\mu, \\
\mathscr{F}_2 &= 2\gamma_H+\alpha_H+\mu+p_H, \\
\mathscr{F}_3 &= \mu+2\eta, \\
\mathscr{F}_4 &= 2\phi+2\lambda_M(t)+\mu+c_O, \\
\mathscr{F}_5 &= 2\gamma_M+\alpha_M-\mu+p_M+c_O, \\
\mathscr{F}_6 &= 2\eta+\mu+c_O, \\
\mathscr{F}_7 &= \delta_A
\end{aligned} \tag{4}
$$

$$
\|\mathscr{F}(Y)-\mathscr{F}(\hat{Y})\| = \sum_{\text{components}} c_j\,|(y_j-\hat{y}_j)| \;\leq\; \Big(\max_j c_j\Big) \sum_{\text{components}} |y_j-\hat{y}_j|. \tag{5}
$$

Continuous dependence.. Uniqueness and continuous dependence on initial data follow directly from the Lipschitz condition and the fractional Grönwall inequality. □

3.1. Existence of Solution

If all parameters are biologically bounded and recruitment and mortality terms satisfy $\frac{dN}{dt} \leq \Lambda - \mu N$, then the solution exists globally for all $t \geq 0$.

3.2. Positivity of solutions.

Consider the i-th component:

$$
X_i(t) = X_{i0} + \frac{1}{\Gamma(\xi)} \int_0^t (t-s)^{\xi-1} F_i(s, X(s))\, ds.
$$

Each F_i satisfies a *quasi-positivity* condition:

$$
F_i(t,X) \geq 0 \quad \text{whenever } X_i = 0,\ X_j \geq 0\ (j \neq i).
$$

This holds because any negative term in F_i is proportional to X_i itself (e.g., $-\lambda_H S_H$, $-\mu S_H$). Therefore, if $X_i(0) \geq 0$, then $X_i(t) \geq 0$ for all $t \in [0,T]$. A contradiction argument using the integral form excludes the possibility of any component becoming negative.

Strict positivity. If at least one component of X_0 is positive, or if $\Lambda > 0$ introduces inflow into the system, then by continuity and quasi-positivity, all compartments become positive for $t > 0$.

$$N = S_H + I_H + R_H + S_M + I_M + R_M + A.$$

4. Stability Analysis

4.1. Locally Stability

We scrutinize the local stability of the disease free equilibruim (DFE) for the fractional order system with Caputo derivative of order $0 < \xi < 1$.

$$\begin{aligned}
{}^{C}D^{\xi}_{0^+} S_H(t) &= \Lambda - \lambda_H(t)\, S_H(t) - \rho\, S_H(t) + \phi\, S_M(t) - \mu\, S_H(t) = 0,\\
{}^{C}D^{\xi}_{0^+} I_H(t) &= \lambda_H(t)\, S_H(t) - (\gamma_H + \alpha_H + \mu)\, I_H(t) = 0,\\
{}^{C}D^{\xi}_{0^+} R_H(t) &= \gamma_H\, I_H(t) - (\mu + \eta)\, R_H(t) + \eta\, R_M(t) = 0,\\
{}^{C}D^{\xi}_{0^+} S_M(t) &= \rho\, S_H(t) - \lambda_M(t)\, S_M(t) - \phi\, S_M(t) - \mu\, S_M(t) = 0,\\
{}^{C}D^{\xi}_{0^+} I_M(t) &= \lambda_M(t)\, S_M(t) - (\gamma_M + \alpha_M + \mu)\, I_M(t) = 0,\\
{}^{C}D^{\xi}_{0^+} R_M(t) &= \gamma_M\, I_M(t) - (\mu + \eta)\, R_M(t) + \eta\, R_H(t) = 0,\\
{}^{C}D^{\xi}_{0^+} A(t) &= c_O\big(S_M(t) + I_M(t) + R_M(t)\big) + p_H I_H(t) + p_M I_M(t) - \delta_A A(t) = 0.
\end{aligned} \tag{6}$$

In this section, we derive all possible equilibrium points of the proposed fractional-order immuno–metabolic epidemic model [4, 5, 6]. An equilibrium point is any constant vector

$$E = (S_H, I_H, R_H, S_M, I_M, R_M, A)$$

satisfying

$${}^{C}D^{\xi}_{0^+} X(t) = 0, \qquad X \in \{S_H, I_H, R_H, S_M, I_M, R_M, A\},$$

which forces all right-hand sides of the system to zero. Due to the structured interactions between healthy and metabolically impaired classes, the model admits several biologically meaningful equilibrium points.

1. Trivial Equilibrium E_0

The completely population-free state is obtained by setting all compartments to zero:

$$E_0 = (0,0,0,0,0,0,0).$$

2. Axial Equilibrium E_1

This equilibrium represents a population of only susceptible healthy individuals:

$$E_1 = (S_H, 0, 0, 0, 0, 0, 0),$$

where

$$\boxed{S_H = \frac{\Lambda + \phi\, \mathscr{P}}{\lambda_H(t) + \rho + \mu}.}$$

3. Infected Healthy Equilibrium E_2

Here, infection appears only in the healthy class:

$$E_2 = (S_H, I_H, 0, 0, 0, 0, 0),$$

with

$$\boxed{I_H = \frac{(\lambda_H(t)\Lambda + \lambda_H(t)\phi)\, \mathscr{P}}{(\lambda_H(t) + \rho + \mu)(\gamma_H + \alpha_H + \mu)}.}$$

4. Recovered Healthy Equilibrium E_3

The recovered healthy class is now active:

$$E_3 = (S_H, I_H, R_H, 0, 0, 0, 0),$$

where

$$\boxed{R_H = \frac{\gamma_H \lambda_H(t)\,(\Lambda - \phi\,\mathscr{P})}{(\lambda_H(t) + \rho + \mu)(\mu + \eta)} + \eta\mathscr{R}.}$$

5. Impaired Susceptible Equilibrium E_4

The metabolically impaired susceptible population is introduced:

$$E_4 = (S_H, I_H, R_H, S_M, 0, 0, 0), \qquad \boxed{S_M = \mathscr{P}}.$$

6. Impaired Infected Equilibrium E_5

The infected impaired class becomes non-zero:

$$E_5 = (S_H, I_H, R_H, S_M, I_M, 0, 0),$$

with

$$\boxed{I_M = \frac{\lambda_M(t)\mathscr{P}}{\gamma_M + \alpha_M + \mu}.}$$

7. Impaired Recovered Equilibrium E_6

Recovered metabolically impaired hosts are present:

$$E_6 = (S_H, I_H, R_H, S_M, I_M, R_M, 0), \qquad \boxed{R_M = \mathscr{R}}.$$

8. Fully Endemic Equilibrium E_7

All population classes and the inflammatory response are active:

$$E_7 = (S_H, I_H, R_H, S_M, I_M, R_M, A),$$

where the inflammatory level is

$$\boxed{A = \frac{c_O\left(\mathscr{P} + \dfrac{\lambda_M(t)\mathscr{P}}{\gamma_M + \alpha_M + \mu}\right)}{\text{den}}.}$$

The auxiliary quantities $\mathscr{P}$, $\mathscr{Q}$, and $\mathscr{R}$ are given by:

$$\mathscr{P} = \frac{\rho\phi + \lambda_M(t)(1 + \rho + \mu) + \phi\left(\lambda_H(t) + \rho + \mu\right) + \mu\left(\lambda_H(t) + \rho + \mu\right)}{\rho\Lambda},$$

$$\mathscr{Q} = \frac{\gamma_H \lambda_H(t)\,(\Lambda - \phi\,\mathscr{P})}{(\lambda_H(t) + \rho + \mu)(\mu + \eta)},$$

$$\mathscr{R} = \frac{\gamma_M\left(\dfrac{\lambda_M(t)\mathscr{P}}{\gamma_M + \alpha_M + \mu}\right) + \eta\mathscr{Q}}{\mu}.$$

Model System and Jacobian Formulation

We consider the compact form of the fractional-order model governed by the Caputo derivative of order ξ as follows from eq(2):

Jacobian Matrix Formulation

Let

$$F = (F_1, F_2, F_3, F_4, F_5, F_6, F_7)^\top,$$

so that the system can be expressed as

$${}^{C}D_t^{\xi} X = F(X),$$

where $X = (S_H, I_H, R_H, S_M, I_M, R_M, A)^\top$. Then, the Jacobian matrix is given by:

$$J = \frac{\partial F}{\partial X} = \begin{pmatrix} \frac{\partial F_1}{\partial S_H} & \frac{\partial F_1}{\partial I_H} & \frac{\partial F_1}{\partial R_H} & \frac{\partial F_1}{\partial S_M} & \frac{\partial F_1}{\partial I_M} & \frac{\partial F_1}{\partial R_M} & \frac{\partial F_1}{\partial A} \\ \frac{\partial F_2}{\partial S_H} & \frac{\partial F_2}{\partial I_H} & \frac{\partial F_2}{\partial R_H} & \frac{\partial F_2}{\partial S_M} & \frac{\partial F_2}{\partial I_M} & \frac{\partial F_2}{\partial R_M} & \frac{\partial F_2}{\partial A} \\ \frac{\partial F_3}{\partial S_H} & \frac{\partial F_3}{\partial I_H} & \frac{\partial F_3}{\partial R_H} & \frac{\partial F_3}{\partial S_M} & \frac{\partial F_3}{\partial I_M} & \frac{\partial F_3}{\partial R_M} & \frac{\partial F_3}{\partial A} \\ \frac{\partial F_4}{\partial S_H} & \frac{\partial F_4}{\partial I_H} & \frac{\partial F_4}{\partial R_H} & \frac{\partial F_4}{\partial S_M} & \frac{\partial F_4}{\partial I_M} & \frac{\partial F_4}{\partial R_M} & \frac{\partial F_4}{\partial A} \\ \frac{\partial F_5}{\partial S_H} & \frac{\partial F_5}{\partial I_H} & \frac{\partial F_5}{\partial R_H} & \frac{\partial F_5}{\partial S_M} & \frac{\partial F_5}{\partial I_M} & \frac{\partial F_5}{\partial R_M} & \frac{\partial F_5}{\partial A} \\ \frac{\partial F_6}{\partial S_H} & \frac{\partial F_6}{\partial I_H} & \frac{\partial F_6}{\partial R_H} & \frac{\partial F_6}{\partial S_M} & \frac{\partial F_6}{\partial I_M} & \frac{\partial F_6}{\partial R_M} & \frac{\partial F_6}{\partial A} \\ \frac{\partial F_7}{\partial S_H} & \frac{\partial F_7}{\partial I_H} & \frac{\partial F_7}{\partial R_H} & \frac{\partial F_7}{\partial S_M} & \frac{\partial F_7}{\partial I_M} & \frac{\partial F_7}{\partial R_M} & \frac{\partial F_7}{\partial A} \end{pmatrix}.$$

$$J = \frac{\partial F}{\partial X} = \begin{pmatrix} \lambda_H(t) - \rho - \mu & 0 & 0 & \phi & 0 & 0 & 0 \\ \lambda_H(t) & -(\gamma_H - \alpha_H + \mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_H & -(\mu + \eta) & 0 & 0 & \eta & 0 \\ \rho & 0 & 0 & -\lambda_M(t) - \phi - \mu & 0 & 0 & 0 \\ 0 & 0 & 0 & \lambda_M(t) & -(\gamma_M - \alpha_M + \mu) & 0 & 0 \\ 0 & 0 & \eta & 0 & \gamma_M & -(\mu + \eta) & 0 \\ 0 & p_H & 0 & c_O & c_O + p_M & c_O & -\delta_A \end{pmatrix}. \tag{7}$$

Theorem 4.1 (Local Stability of the Zero Equilibrium)**.** *Consider the fractional-order system eq.(2), where all parameters are nonnegative and* $0 < \xi < 1$ *[7, 8, 9] and fig.(2). Then:*

(i) The point $E_0 = (0,0,0,0,0,0,0)$ *is an equilibrium of (2) if and only if* $\Lambda = 0$.

(ii) Assuming $\Lambda = 0$*, the Jacobian matrix at* E_0 *has eigenvalues*

$$\begin{aligned} &-(\rho+\mu), \qquad -(\gamma_H+\alpha_H+\mu), \qquad -(\mu+\eta), \\ &-(\phi+\mu), \qquad -(\gamma_M+\alpha_M+\mu), \qquad -(\mu+\eta), \qquad -\delta_A, \end{aligned}$$

all of which lie in the open left-half of the complex plane. Consequently, by Matignon's stability criterion for Caputo systems, the zero equilibrium E_0 *is locally asymptotically stable for every* $0 < \xi \leq 1$.

Proof. Evaluating eq.(2) at E_0 shows that the first equation reduces to ${}^{C}D^{\xi} S_H = \Lambda$. Hence E_0 can be an equilibrium only when $\Lambda = 0$; this proves (i).

To establish local stability, we linearize the system at E_0, assuming $\Lambda = 0$. Because the incidence terms $\lambda_H S_H$ and $\lambda_M S_M$ vanish at E_0 and their partial derivatives also vanish due to the product structure, the Jacobian matrix becomes block lower-triangular with negative diagonal entries:

$$J(E_0) = \begin{pmatrix} -(\rho+\mu) & 0 & 0 & \phi & 0 & 0 & 0 \\ 0 & -(\gamma_H+\alpha_H+\mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_H & -(\mu+\eta) & 0 & 0 & \eta & 0 \\ \rho & 0 & 0 & -(\phi+\mu) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\gamma_M+\alpha_M+\mu) & 0 & 0 \\ 0 & 0 & \eta & 0 & \gamma_M & -(\mu+\eta) & 0 \\ 0 & p_H & 0 & c_O & p_M & c_O & -\delta_A \end{pmatrix}.$$

Gershgorin's theorem shows that all eigenvalues of $J(E_0)$ have strictly negative real parts, proving the eigenvalue list given above.

For a fractional-order linear system ${}^{C}D^{\xi}x = J(E_0)x$, Matignon's criterion states that the equilibrium is asymptotically stable iff

$$|\arg(\lambda_i)| > \frac{\pi\xi}{2}, \qquad i = 1,\ldots,7.$$

Since each eigenvalue is strictly negative real, we have $\arg(\lambda_i) = \pi$, and therefore

$$\pi > \frac{\pi\xi}{2} \qquad (0 < \xi \leq 1),$$

which confirms local asymptotic stability. This completes the proof. □

Theorem 4.2 (Local stability of the boundary equilibrium $E_1 = (S_H^*, 0, 0, 0, 0, 0, 0)$)**.** *Consider the fractional-order Caputo system eq.(2) with parameters nonnegative and* $0 < \xi < 1$. *Assume the incidence (force of infection) functions are smooth and satisfy the bilinear (mass-action) linearization*

$$\lambda_H \approx \beta_H I_M, \qquad \lambda_M \approx \beta_M I_H$$

near the disease-free / boundary state. Let

$$E_1 = (S_H^*, 0, 0, 0, 0, 0, 0)$$

be a steady state with $S_H^* \geq 0$ *and all other state components zero.*

Then the linearization of the system at E_1 *has spectrum contained in the open left half-plane (equivalently, all eigenvalues are real and strictly negative). Consequently, by Matignon's criterion for linear fractional systems,* E_1 *is* locally asymptotically stable *for every fractional order* $0 < \xi < 1$ *[10, 11, 12]and this fig.(3).*

Proof. We linearize the system at E_1. Denote

$$a = \gamma_H + \alpha_H + \mu > 0, \qquad b = \gamma_M + \alpha_M + \mu > 0.$$

Under the stated linearization of the incidence, the infection-subsystem partial derivatives at E_1 are

$$\left.\frac{\partial(\lambda_H S_H)}{\partial I_M}\right|_{E_1} = \beta_H S_H^*, \qquad \left.\frac{\partial(\lambda_M S_M)}{\partial I_H}\right|_{E_1} = \beta_M S_M^* = 0,$$

since $S_M^* = 0$ at E_1. All other infection-related linearization terms that involve products with zero compartments vanish at E_1.

Assembling the Jacobian $J = Df(E_1)$ in the natural ordering $x = (S_H, I_H, R_H, S_M, I_M, R_M, A)$ yields a matrix whose infection block (rows/columns for I_H, I_M) is upper-triangular:

$$J_I = \begin{pmatrix} -a & \beta_H S_H^* \\ 0 & -b \end{pmatrix}.$$

The remaining entries of J consist of negative diagonal terms coming from transfers and removals (for example $-(\rho+\mu)$, $-(\phi+\mu)$, $-(\mu+\eta)$, $-\delta_A$) and nonnegative off-diagonal transfer entries (e.g. $\rho, \phi, \gamma_H, \gamma_M, \eta, p_H, c_O$), but these do not produce eigenvalues with nonnegative real part because the diagonal damping rates are strictly positive.

From the triangular form of J_I, the eigenvalues associated with the infection subsystem are $-a$ and $-b$, both strictly negative. The rest of the Jacobian has diagonal entries equal to negative outflow rates and, by Gershgorin's theorem (or by direct inspection), all remaining eigenvalues have strictly negative real parts. Thus every eigenvalue λ_i of J satisfies $\Re(\lambda_i) < 0$.

For the linear fractional system

$${}^{C}D^{\xi}y(t) = Jy(t),$$

Matignon's criterion states that the equilibrium is asymptotically stable if every eigenvalue λ_i of J satisfies

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

Since all eigenvalues are real negative, $\arg(\lambda_i) = \pi$ and therefore

$$\pi > \frac{\pi\xi}{2} \quad \text{for all } 0 < \xi \leq 1,$$

so Matignon's condition is satisfied. Hence E_1 is locally asymptotically stable. □

3. Existence and Local Stability of the Human-Only Equilibrium E_2 Consider the fractional Caputo system of order $0 < \xi < 1$ [13, 14, 15] and fig.(3) explained with nonnegative parameters and smooth incidence functions as described. Suppose an equilibrium of the form

$$E_2 = (S_H^*, I_H^*, 0, 0, 0, 0, 0)$$

exists and satisfies the algebraic steady-state conditions.

(a) **Feasibility:** At E_2, the mosquito and environmental equations require

$$\rho S_H^* = 0, \qquad p_H I_H^* = 0,$$

so unless $\rho = p_H = 0$ (or $S_H^* = I_H^* = 0$), such an equilibrium cannot exist.

(b) **Linearization and Stability:** If the feasibility conditions hold, define

$$a = \gamma_H + \alpha_H + \mu, \qquad m = \gamma_M + \alpha_M + \mu,$$

and let

$$F_{11} = \frac{\partial(\lambda_H S_H)}{\partial S_H}\Big|_{E_2}, \qquad F_{12} = \frac{\partial(\lambda_H S_H)}{\partial I_H}\Big|_{E_2}.$$

The Jacobian at E_2 block-decomposes, with the human subsystem

$$J_H = \begin{pmatrix} -(\rho+\mu) - F_{11} & -F_{12} \\ F_{11} & F_{12} - a \end{pmatrix},$$

and all other eigenvalues being negative outflow rates. The RouthŰHurwitz conditions

$$\mathrm{tr}(J_H) = F_{12} - a - (\rho+\mu) - F_{11} < 0, \qquad \det(J_H) = [-(\rho+\mu) - F_{11}](F_{12} - a) + F_{11}F_{12} > 0$$

guarantee that J_H has eigenvalues with negative real parts. If all other outflow rates are positive, every eigenvalue of the full Jacobian satisfies $\Re(\lambda) < 0$, and by Matignon's criterion, E_2 is locally asymptotically stable for all $0 < \xi < 1$.

4. Existence and Local Stability of the Human-only Equilibrium E_3 Consider the fractional Caputo system of order $0 < \xi < 1$ [16, 17, 18] and fig.(4) with nonnegative parameters and smooth incidence functions. Suppose a steady state of the form

$$E_3 = (S_H^*, I_H^*, R_H^*, 0, 0, 0, 0), \qquad S_H^* \geq 0, I_H^* \geq 0, R_H^* \geq 0,$$

exists and satisfies the steady-state conditions.

(i) **Feasibility.** At E_3, evaluating the S_M and A equations yields

$$\rho\, S_H^* = 0, \qquad p_H\, I_H^* = 0.$$

Therefore, if $S_H^* > 0$ and $I_H^* > 0$, we require $\rho = 0$ and $p_H = 0$.

(ii) **Local stability.** If the feasibility constraints hold, define

$$a = \gamma_H + \alpha_H + \mu, \qquad b := \mu + \eta,$$

and the partial derivatives

$$F_{11} = \frac{\partial(\lambda_H S_H)}{\partial S_H}\Big|_{E_3}, \quad F_{12} = \frac{\partial(\lambda_H S_H)}{\partial I_H}\Big|_{E_3}, \quad F_{13} = \frac{\partial(\lambda_H S_H)}{\partial R_H}\Big|_{E_3}.$$

The Jacobian J at E_3 has a human 3×3 block

$$J_H = \begin{pmatrix} -(\rho+\mu) - F_{11} & -F_{12} & -F_{13} \\ F_{11} & F_{12} - a & F_{13} \\ 0 & \gamma_H & -b \end{pmatrix},$$

and all other blocks are decoupled and negative by construction.
The characteristic polynomial of J_H is

$$\chi_H(\lambda) = \lambda^3 + A_1\lambda^2 + A_2\lambda + A_3,$$

with

$$A_1 = -\operatorname{tr}(J_H), \qquad A_2 = \text{sum of principal } 2\times 2 \text{ minors of } -J_H, \qquad A_3 = -\det(J_H).$$

The RouthŰHurwitz criteria for stability are

$$A_1 > 0, \qquad A_2 > 0, \qquad A_1A_2 > A_3.$$

If these hold and all negative outflow rates remain positive, then by Matignon's fractional order criterion, E_3 is locally asymptotically stable for all $0 < \xi < 1$.

5. Existence and Local Stability of the Semi-Vector Equilibrium E_4 Consider the fractional Caputo system of order $0 < \xi < 1$ [19, 20 ,21] and explained in fig.(5) with nonnegative parameters and smooth incidence functions. Suppose there exists a steady state of the form

$$E_4 = (S_H^*, I_H^*, R_H^*, S_M^*, 0, 0, 0), \qquad S_H^*, S_M^* \geq 0, I_H^*, R_H^* \geq 0,$$

satisfying the algebraic steady-state conditions.

(i) **Feasibility.** Evaluating the steady-state equations at E_4 yields

$$\rho S_H^* = (\phi + \mu)S_M^*, \qquad \lambda_M(E_4) = 0 \text{ whenever } S_M^* > 0.$$

Thus, a nontrivial semi-vector equilibrium with $S_M^* > 0$ requires vanishing mosquito force of infection at E_4, typically imposing parameter or incidence constraints.

(ii) **Local Stability.** Suppose (i) holds. Let

$$a = \gamma_H + \alpha_H + \mu, \quad b = \mu + \eta,$$

and define the partial derivatives at E_4 as

$$F_{11} = \frac{\partial(\lambda_H S_H)}{\partial S_H}, \quad F_{12} = \frac{\partial(\lambda_H S_H)}{\partial I_H}, \quad F_{13} = \frac{\partial(\lambda_H S_H)}{\partial R_H}, \quad F_{14} = \frac{\partial(\lambda_H S_H)}{\partial S_M},$$
$$G_{41} = \frac{\partial(\lambda_M S_M)}{\partial S_H}, \quad G_{42} = \frac{\partial(\lambda_M S_M)}{\partial I_H}, \quad G_{43} = \frac{\partial(\lambda_M S_M)}{\partial R_H}, \quad G_{44} = \frac{\partial(\lambda_M S_M)}{\partial S_M}.$$

The Jacobian's principal block for (S_H, I_H, R_H, S_M) is

$$J_4 = \begin{pmatrix} -(\rho+\mu) - F_{11} & -F_{12} & -F_{13} & \phi - F_{14} \\ F_{11} & F_{12} - a & F_{13} & F_{14} \\ 0 & \gamma_H & -b & 0 \\ \rho + G_{41} & G_{42} & G_{43} & -(\phi+\mu) + G_{44} \end{pmatrix},$$

while the remaining rows/columns are decoupled with strictly negative diagonal elements.
Denote the characteristic polynomial of J_4 as

$$\chi_4(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4,$$

where a_1, a_2, a_3, a_4 are determined by J_4. The equilibrium E_4 is locally asymptotically stable if the quartic RouthŰHurwitz conditions hold:

$$a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0, a_1a_2 > a_3, a_1a_2a_3 > a_1^2a_4 + a_3^2.$$

If these and all decoupled negative rates are satisfied, every eigenvalue has negative real part, and Matignon's criterion ensures stability for all $0 < \xi < 1$.

6. Existence and Local Stability of the Semi-Endemic Equilibrium E_5 Consider the fractional Caputo system of order $0 < \xi < 1$ [22, 23, 24] and fig.(6) explained with nonnegative parameters and smooth incidence functions. Suppose an equilibrium

$$E_5 = (S_H^*, I_H^*, R_H^*, S_M^*, I_M^*, 0, 0), \quad S_H^*, S_M^* \geq 0, \quad I_H^*, I_M^*, R_H^* \geq 0,$$

satisfies the steady-state conditions.

(1) Feasibility conditions. The following necessary algebraic relations hold:

$$\begin{aligned} 0 &= \rho S_H^* - \lambda_M(E_5) S_M^* - (\phi + \mu) S_M^*, \\ 0 &= \lambda_M(E_5) S_M^* - (\gamma_M + \alpha_M + \mu) I_M^*, \\ 0 &= \gamma_M I_M^* + \eta R_H^*. \end{aligned}$$

Nonnegativity implies:

- If $\gamma_M > 0$, then $I_M^* = 0$.
- If $\eta > 0$, then $R_H^* = 0$.

Hence existence requires either zero values of some states or special parameter relations.

(2) Linearization and Jacobian. Define

$$a = \gamma_H + \alpha_H + \mu, \quad m = \gamma_M + \alpha_M + \mu, \quad b = \mu + \eta,$$

and the partial derivatives evaluated at E_5:

$$F_{1j} = \frac{\partial(\lambda_H S_H)}{\partial x_j}, \quad G_{4j} = \frac{\partial(\lambda_M S_M)}{\partial x_j}, \quad j = 1, \ldots, 7,$$

with $x = (S_H, I_H, R_H, S_M, I_M, R_M, A)$.

The Jacobian block for $(S_H, I_H, R_H, S_M, I_M)$ is

$$J_5 = \begin{pmatrix} -(\rho+\mu) - F_{11} & -F_{12} & -F_{13} & \phi - F_{14} & -F_{15} \\ F_{11} & F_{12} - a & F_{13} & F_{14} & F_{15} \\ 0 & \gamma_H & -b & 0 & 0 \\ \rho + G_{41} & G_{42} & G_{43} & -(\phi+\mu) + G_{44} & G_{45} \\ 0 & 0 & 0 & G_{54} & G_{55} - m \end{pmatrix}.$$

(3) Local stability criterion. The fractional linearized system reads

$${}^{C}D^{\xi} y(t) = J_5 y(t),$$

with decoupled stable equations for remaining variables. The equilibrium E_5 is locally asymptotically stable if and only if all eigenvalues λ of J_5 satisfy $\Re(\lambda) < 0$, ensuring via Matignon's criterion stability for all $0 < \xi < 1$.

(4) Sufficient stability condition. If all diagonal entries satisfy

$$-(\rho+\mu) - F_{11} < 0, \quad F_{12} - a < 0, \quad -b < 0, \quad -(\phi+\mu) + G_{44} < 0, \quad G_{55} - m < 0,$$

and the off-diagonal terms are sufficiently small, then Gershgorin's theorem guarantees all eigenvalues lie in the left half-plane, implying stability.

7. Existence and Local Stability of the Full HostŰVector Equilibrium Consider the Caputo fractional-order system (order $0 < \xi < 1$) [25, 26, 27] and fig.(7) explained of eq.(2) with nonnegative parameters and smooth incidence functions λ_H, λ_M. Assume equilibrium

$$E_6 = (S_H^*, I_H^*, R_H^*, S_M^*, I_M^*, R_M^*, 0), \quad S_H^*, I_H^*, R_H^*, S_M^*, I_M^*, R_M^* \geq 0.$$

(i) **Feasibility for $A^* = 0$.** The A-equation at equilibrium implies

$$0 = c_O(S_M^* + I_M^* + R_M^*) + p_H I_H^* + p_M I_M^*.$$

Since all terms are nonnegative, this holds only if

$$\boxed{c_O = p_H = p_M = 0} \quad \text{or} \quad S_M^* = I_M^* = R_M^* = I_H^* = 0.$$

Thus, a full hostŰvector equilibrium with nonzero infected/vector compartments and $A^* = 0$ requires vanishing pollution/source terms.

(ii) **Linearization at E_6.** Writing the state as

$$x = (S_H, I_H, R_H, S_M, I_M, R_M, A)^\top,$$

define partials at E_6:

$$F_{1j} := \frac{\partial(\lambda_H S_H)}{\partial x_j}, \quad G_{4j} := \frac{\partial(\lambda_M S_M)}{\partial x_j}, \quad j = 1, \ldots, 7.$$

The Jacobian $J = Df(E_6)$ is

$$\begin{pmatrix} -(\rho+\mu)-F_{11} & -F_{12} & -F_{13} & \phi - F_{14} & -F_{15} & -F_{16} & -F_{17} \\ F_{11} & F_{12} - (\gamma_H + \alpha_H + \mu) & F_{13} & F_{14} & F_{15} & F_{16} & F_{17} \\ 0 & \gamma_H & -(\mu+\eta) & 0 & 0 & \eta & 0 \\ \rho + G_{41} & G_{42} & G_{43} & -(\phi+\mu) + G_{44} & -G_{45} & -G_{46} & -G_{47} \\ 0 & 0 & 0 & G_{44} & G_{45} - (\gamma_M + \alpha_M + \mu) & G_{46} & G_{47} \\ 0 & 0 & \eta & G_{44} & \gamma_M & -(\mu+\eta) & 0 \\ 0 & p_H & 0 & c_O & p_M & c_O & -\delta_A \end{pmatrix}.$$

(iii) **Local Stability Criterion.** The fractional linearized system near E_6 is

$${}^C D^\xi y(t) = J(E_6) y(t).$$

Matignon's theorem states local asymptotic stability for $0 < \xi < 1$ if and only if all eigenvalues λ of $J(E_6)$ satisfy

$$|\arg(\lambda)| > \frac{\pi\xi}{2}.$$

A sufficient practical check is

$$\Re(\lambda) < 0 \quad \text{for all eigenvalues } \lambda.$$

(v) **Sufficient Condition.** If

$$\begin{cases} -(\rho+\mu) - F_{11} < 0, \\ F_{12} - (\gamma_H + \alpha_H + \mu) < 0, \\ -(\mu+\eta) < 0, \\ -(\phi+\mu) + G_{44} < 0, \\ G_{45} - (\gamma_M + \alpha_M + \mu) < 0, \\ -(\mu+\eta) < 0, \\ -\delta_A < 0, \end{cases} \qquad \text{and} \quad \Re(d_i) + R_i < 0 \text{ for each row } i,$$

where d_i are diagonal entries and R_i sum of off-diagonal magnitudes of $J(E_6)$, then all eigenvalues have negative real parts and E_6 is locally asymptotically stable for all $0 < \xi \leq 1$.

8. Existence and Local Stability of the Full Equilibrium E_7

Consider the Caputo fractional-order system (order $0 < \xi < 1$) from eq(2) with smooth incidence functions λ_H, λ_M and nonnegative parameters [28, 29, 30] and explained in fig.(8) . Let

$$E_7 = (S_H^*, I_H^*, R_H^*, S_M^*, I_M^*, R_M^*, A^*), \quad S_H^*, I_H^*, R_H^*, S_M^*, I_M^*, R_M^*, A^* \geq 0,$$

be an equilibrium of the system.

(i) **Equilibrium relation for A^*.** The equilibrium value A^* satisfies

$$A^* = \frac{c_O(S_M^* + I_M^* + R_M^*) + p_H I_H^* + p_M I_M^*}{\delta_A} \geq 0.$$

(ii) **Jacobian at E_7.** With state ordering $x = (S_H, I_H, R_H, S_M, I_M, R_M, A)^\top$, define partial derivatives at E_7:

$$F_{1j} = \frac{\partial(\lambda_H S_H)}{\partial x_j}, \quad G_{4j} = \frac{\partial(\lambda_M S_M)}{\partial x_j}, \quad j = 1, \ldots, 7,$$

and abbreviations $a = \gamma_H + \alpha_H + \mu$, $m = \gamma_M + \alpha_M + \mu$, $b = \mu + \eta$.
The Jacobian $J(E_7)$ is

$$\begin{pmatrix} -(\rho+\mu) - F_{11} & -F_{12} & -F_{13} & \phi - F_{14} & -F_{15} & -F_{16} & -F_{17} \\ F_{11} & F_{12} - a & F_{13} & F_{14} & F_{15} & F_{16} & F_{17} \\ 0 & \gamma_H & -b & 0 & 0 & \eta & 0 \\ \rho + G_{41} & G_{42} & G_{43} & -(\phi+\mu) + G_{44} & -G_{45} & -G_{46} & -G_{47} \\ 0 & 0 & 0 & G_{44} & G_{45} - m & G_{46} & G_{47} \\ 0 & 0 & \eta & G_{44} & \gamma_M & -b & 0 \\ 0 & p_H & 0 & c_O & p_M & c_O & -\delta_A \end{pmatrix}.$$

(iii) **Local stability via Matignon's criterion.** The linearized system is

$${}^C D^\xi y(t) = J(E_7) y(t).$$

By Matignon's theorem, E_7 is locally asymptotically stable for $0 < \xi \leq 1$ if every eigenvalue λ of $J(E_7)$ satisfies

$$|\arg(\lambda)| > \frac{\pi\xi}{2}.$$

A sufficient condition is that all eigenvalues have strictly negative real parts, i.e.,

$$\Re(\lambda) < 0.$$

(iv) **Practical stability tests.** Recommended are:

- *Gershgorin discs / diagonal dominance:* If for each row i,

$$\Re(d_i) + R_i < 0, \quad R_i = \sum_{j \neq i} |J_{ij}|,$$

then all eigenvalues lie in the open left half-plane, ensuring stability.

(v) **Sufficient explicit condition.** If the diagonal terms satisfy

$$-(\rho+\mu) - F_{11} < 0, \quad F_{12} - a < 0, \quad -b < 0, \quad -(\phi+\mu) + G_{44} < 0, \quad G_{45} - m < 0, \quad -b < 0, \quad -\delta_A < 0,$$

and the Gershgorin inequalities $\Re(d_i) + R_i < 0$ hold for each row, then E_7 is locally asymptotically stable for all $0 < \xi < 1$.

4.2. Global Stability of the Disease-Free Equilibrium

Theorem 4.3. *The disease free equilibrium $(I_H, I_M) = (0,0)$ is globally asymptotically stable at*

$$E^* = (S_H^*, I_H^*, R_H^*, S_M^*, I_M^*, R_M^*, A^*),$$

provided [31, 32, 33] and explained in fig.(9)

$$\frac{\beta S_H^0}{\Gamma_H} + \frac{\beta \theta^2 S_M^0}{\Gamma_M} < 1. \tag{8}$$

Proof. Define the Lyapunov functional

$$V(I_H, I_M) = \frac{I_H}{\Gamma_H} + \frac{I_M}{\Gamma_M},$$

which is positive definite on $\mathbb{R}^2_{\geq 0}$ and vanishes only at $(0,0)$.

Using linearity of the Caputo derivative,

$${}^{C}D_t^{\xi} V = \frac{1}{\Gamma_H} {}^{C}D_t^{\xi} I_H + \frac{1}{\Gamma_M} {}^{C}D_t^{\xi} I_M.$$

Substituting the subsystem dynamics gives

$$\begin{aligned} {}^{C}D_t^{\xi} V &= \frac{1}{\Gamma_H}\Big(\beta S_H^0(I_H + \theta I_M) - \Gamma_H I_H\Big) + \frac{1}{\Gamma_M}\Big(\beta\theta S_M^0(I_H + \theta I_M) - \Gamma_M I_M\Big) \\ &= \left(\frac{\beta S_H^0}{\Gamma_H} + \frac{\beta\theta S_M^0}{\Gamma_M}\right) I_H + \left(\frac{\beta\theta S_H^0}{\Gamma_H} + \frac{\beta\theta^2 S_M^0}{\Gamma_M}\right) I_M - (I_H + I_M). \end{aligned}$$

Let

$$a = \frac{\beta S_H^0}{\Gamma_H} + \frac{\beta\theta^2 S_M^0}{\Gamma_M}.$$

Since

$$\frac{\beta\theta S_M^0}{\Gamma_M} \leq \frac{\beta\theta^2 S_M^0}{\Gamma_M}, \qquad \frac{\beta\theta S_H^0}{\Gamma_H} \leq \frac{\beta S_H^0}{\Gamma_H} + \frac{\beta\theta^2 S_M^0}{\Gamma_M},$$

we obtain the conservative bound

$${}^{C}D_t^{\xi} V \leq a(I_H + I_M) - (I_H + I_M) = -(1-a)(I_H + I_M).$$

Since

$$c_1(I_H + I_M) \leq V(I_H, I_M) \leq c_2(I_H + I_M), \qquad c_1, c_2 > 0,$$

we obtain

$${}^{C}D_t^{\xi} V \leq -\frac{1-a}{c_2} V.$$

If the inequality (8) holds, then $1 - a > 0$, and by the standard fractional comparison lemma for Caputo derivatives,

$$V(t) \longrightarrow 0 \qquad \text{as } t \to \infty.$$

Hence $I_H(t) \to 0$ and $I_M(t) \to 0$, proving global asymptotic stability of the disease-free equilibrium. □

5. Basic reproduction number of $\mathscr{R}_0$

We extract the infected subsystem [34] and explained in fig.(9)

$$X_I = (I_H, I_M)^{\top}$$

and rewrite the model eq.(2) in the standard nextŰgeneration form

$${}^{C}D_t^{\xi} X_I = F(X) - V(X), \qquad 0 < \xi \leq 1,$$

where:

- $F(X)$ accounts for the appearance of new infections,
- $V(X)$ represents transitions between infected compartments (outflow minus inflow).

Matrix F

New infections are given by

$$F = \begin{pmatrix} \lambda_H^0 S_H^0 \\ \lambda_M^0 S_M^0 \end{pmatrix} = \begin{pmatrix} \beta S_H^0 (I_H + \theta I_M) \\ \beta \theta S_M^0 (I_H + \theta I_M) \end{pmatrix}.$$

Differentiating with respect to I_H and I_M at the DFE ($I_H = I_M = 0$) yields:

$$F = \begin{pmatrix} \beta S_H^0 & \beta \theta S_H^0 \\ \beta \theta S_M^0 & \beta \theta^2 S_M^0 \end{pmatrix}.$$

Matrix *V*

The transition terms for the infected compartments are

$$V = \begin{pmatrix} (\gamma_H + \alpha_H + \mu) I_H \\ (\gamma_M + \alpha_M + \mu) I_M \end{pmatrix}.$$

Thus,

$$V = \begin{pmatrix} \Gamma_H & 0 \\ 0 & \Gamma_M \end{pmatrix}, \qquad \Gamma_H = \gamma_H + \alpha_H + \mu, \quad \Gamma_M = \gamma_M + \alpha_M + \mu.$$

Hence

$$V^{-1} = \begin{pmatrix} \Gamma_H^{-1} & 0 \\ 0 & \Gamma_M^{-1} \end{pmatrix}.$$

The next-generation matrix is

$$K = FV^{-1} = \begin{pmatrix} \dfrac{\beta S_H^0}{\Gamma_H} & \dfrac{\beta \theta S_H^0}{\Gamma_H} \\ \dfrac{\beta \theta S_M^0}{\Gamma_M} & \dfrac{\beta \theta^2 S_M^0}{\Gamma_M} \end{pmatrix}.$$

Now find basic reproduction number

Since K is a 2×2 matrix, the spectral radius is

$$\mathscr{R}_0 = \rho(K) = \frac{1}{2}\left[\mathrm{Tr}(K) + \sqrt{(\mathrm{Tr}(K))^2 - 4\det(K)}\right].$$

Compute the trace:

$$\mathrm{Tr}(K) = \frac{\beta S_H^0}{\Gamma_H} + \frac{\beta \theta^2 S_M^0}{\Gamma_M}.$$

For this model the determinant vanishes:

$$\det(K) = \frac{\beta^2 \theta^2 S_H^0 S_M^0}{\Gamma_H \Gamma_M} - \frac{\beta^2 \theta^2 S_H^0 S_M^0}{\Gamma_H \Gamma_M} = 0.$$

Therefore,

$$\mathscr{R}_0 = \frac{\beta S_H^0}{\Gamma_H} + \frac{\beta \theta^2 S_M^0}{\Gamma_M}.$$

Using the DFE values

$$S_H^0 = \frac{\Lambda}{\rho + \mu}, \qquad S_M^0 = \frac{\Lambda \rho}{(\rho + \mu)(\phi + \mu)},$$

we obtain the compact expression

$$\boxed{\mathscr{R}_0 = \frac{\beta \Lambda}{\rho + \mu}\left[\frac{1}{\Gamma_H} + \frac{\theta^2 \rho}{(\phi + \mu)\Gamma_M}\right]}$$

with

$$\Gamma_H = \gamma_H + \alpha_H + \mu, \qquad \Gamma_M = \gamma_M + \alpha_M + \mu.$$

6. Numerical Analysis

To investigate the impact of type 2 diabetes mellitus (T2DM) on feline infection dynamics and to validate the theoretical results derived above, numerical simulations were performed using the fixed-step Predictor-Corrector method. Specifically, the fractional Adams-Bashforth-Moulton scheme was employed to approximate the Caputo-type fractional derivatives. Let ${}^{C}D_t^{\xi} F_j(t)$ denote the Caputo fractional derivative of order $\xi > 0$ for the state variable $F_j(t)$, with initial conditions

$$F_j^{(r)}(0) = F_{j,0}^{(r)}, \quad r = 0,1,2,\ldots,\lceil \alpha \rceil - 1, \quad j \in \mathbb{N}.$$

Under the Caputo interpretation, this derivative can be expressed equivalently as a Volterra-type integral:

$$F_j(t) = \sum_{r=0}^{\lceil \xi \rceil - 1} \frac{F_{j,0}^{(r)} t^r}{r!} + \frac{1}{\Gamma(\alpha)} \int_0^t (t-u)^{\alpha-1} g_j(u, F_j(u))\, du,$$

where g_j represents the right-hand side of the corresponding fractional differential equation. Model parameters for numerical simulations were selected based on experimental and observational data from feline studies (Table.1) and explain with graphs (1-12). The simulations included fractional orders ξ close to 1 to capture the memory effects relevant to T2DM progression and chronic inflammation in cats. Stability analyses were performed across different fractional orders to examine the overall population dynamics and identify potential equilibrium behavior. Furthermore, the occurrence of Hopf bifurcation was explored to illustrate oscillatory or periodic behavior in infection levels, particularly in populations affected by T2DM-mediated immuno-metabolic impairment. Cats with T2DM typically exhibit insulin resistance and chronic hyperglycemia, which compromise both innate and adaptive immune responses [33]. Hyperglycemia and metabolic dysregulation lead to overproduction of pro-inflammatory cytokines such as TNF-ξ, IL-6, and IL-1β, establishing persistent systemic inflammation [30]. This cytokine imbalance impairs leukocyte activity, reduces pathogen clearance, and prolongs recovery from infections. From an epidemiological perspective, such immuno-metabolic alterations increase susceptibility to feline pathogens, extend infectious periods, and may elevate secondary transmission within domestic or shelter populations [14, 26]. Incorporating these mechanisms into fractional-order epidemic models allows for a more realistic representation of infection dynamics in metabolically compromised cats, providing critical insights for outbreak prediction, preventive strategies, and targeted clinical interventions.

6.1. Discrete Adams-Bashforth-Moulton Scheme

To numerically solve the fractional-order system representing T2DM-influenced infection dynamics in cats, we applied the fixed-step Adams-Bashforth-Moulton (ABM) predictor-corrector method. Let $F_j^n \approx F_j(t_n)$ denote the numerical approximation of the state variable $F_j(t)$ at time $t_n = nh$, with step size h. The predictor-corrector formulas are given as follows:

Predictor (Adams-Bashforth).

$$\hat{F}_j^{n+1} = F_j^0 + \frac{h^{\xi}}{\Gamma(\xi+1)} \sum_{k=0}^{n} b_{k,n+1}\, g_j(t_k, F_j^k), \quad j = 1,2,\ldots,m, \tag{9}$$

where the weights $b_{k,n+1}$ are defined by

$$b_{k,n+1} = (n+1-k)^{\xi} - (n-k)^{\xi},$$

and $g_j(t_k, F_j^k)$ represents the right-hand side of the fractional differential equation for the j-th compartment.

Corrector (Adams-Moulton).

$$F_j^{n+1} = F_j^0 + \frac{h^{\xi}}{\Gamma(\xi+2)} \left[g_j(t_{n+1}, \hat{F}_j^{n+1}) + \sum_{k=0}^{n} a_{k,n+1}\, g_j(t_k, F_j^k) \right], \tag{10}$$

with weights

$$a_{k,n+1} = (n+1-k)^{\xi+1} - (n-k)^{\xi+1}.$$

This iterative scheme was applied to all compartments of the model: susceptible, infected, and recovered states for both metabolically healthy and T2DM-affected cats, as well as the inflammation state. The fractional order ξ close to 1 was used to reflect the memory effects of chronic hyperglycemia and systemic inflammation associated with T2DM. This approach allows the numerical solution to capture the delayed immuno-metabolic responses and the resulting impact on infection propagation in feline populations.

Table 1: Suggested parameter values (order of magnitude) for an immuno metabolic infection model in domestic cats.

Parameter	Suggested value (units)	Comments / Rationale
Λ	5×10^{-4} (cats/day)	Recruitment (birth/entry) into susceptible pool; adjust to population scale
μ	2.1×10^{-4} (day^{-1})	Natural mortality: $\approx 1/(13\ \text{yr} \times 365)$ (mean lifespan 13 yr).
β	0.20 (day^{-1})	Baseline transmission rate (order of magnitude reference from cat transmission studies).
θ	1.30 (dimensionless)	Relative infectiousness of metabolically impaired cats (e.g., 30% higher)
$\sigma(A)$	$\sigma(A) = 1 + \kappa A,\ \kappa = 0.50$	Inflammation-dependent susceptibility multiplier (baseline 1; per-unit sensitivity)
γ_H	0.12 (day^{-1})	Recovery rate for healthy cats (mean infectious period $\approx$ 8–9 days).
γ_M	0.08 (day^{-1})	Recovery rate for metabolically impaired cats (longer recovery)
α_H	1×10^{-3} (day^{-1})	Disease-induced mortality, healthy hosts (low baseline)
α_M	5×10^{-3} (day^{-1})	Disease-induced mortality, impaired hosts (higher risk)
ρ	5.0×10^{-4} day^{-1}	Rate healthy $\rightarrow$ metabolically impaired (≈ 0.18 yr^{-1}); tune by prevalence data
ϕ	1.0×10^{-4} (day^{-1})	Rate impaired $\rightarrow$ healthy (behavioral/clinical recovery / weight loss interventions)
p_H	1.0 (A-units/day)	Cytokine production by infected healthy cats (model units)
p_M	2.0 (A-units/day)	Cytokine production by infected impaired cats (higher per-capita)
c_O	0.5 (A-units/day)	Chronic inflammatory contribution from adipose tissue (baseline)
δ_A	0.20 (day^{-1})	Clearance rate of systemic inflammation (half-life $\sim$3.5 days)

Figures: 1.

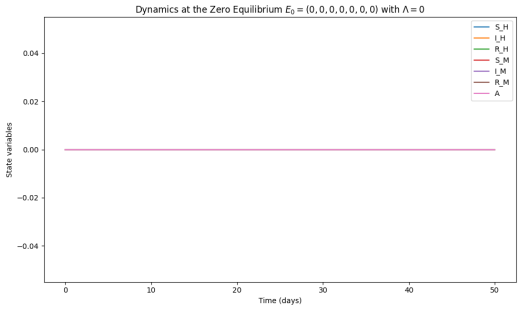

Figure 2: All initial conditions are exactly the equilibrium point E_0

2.

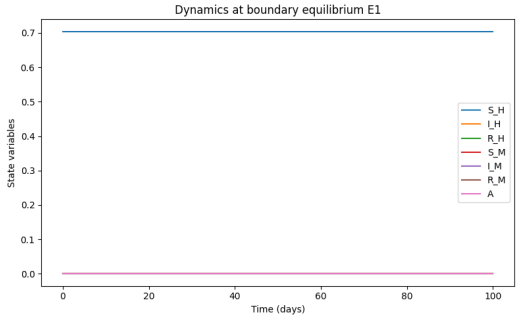

Figure 3: Dynamics at the boundary equilibrium E_1.

In fig.(3) illustrate the numerical simulation confirms that the system quickly settles at the boundary equilibrium E_1, where the host susceptible population S_H remains constant while all infected, recovered, and vector populations (I_H, R_H, S_M, I_M, R_M) remain at zero. The inflammatory marker $A(t)$ also stays at zero, indicating the absence of infection-driven activation. This behaviour is consistent with the analytical stability properties of E_1, which represents a disease-free state with only the susceptible host class persisting.

3.

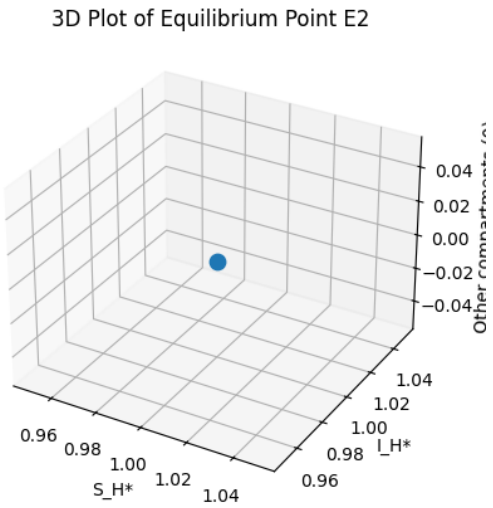

Figure 4: 3D representation of the boundary equilibrium E_2.

In fig.(4) explained the point shown represents the boundary equilibrium $E_2 = (S_H^*, I_H^*, A^*)$ in the reduced three-dimensional space. Here, S_H^* and I_H^* are the nonzero susceptible and infected host components, while the remaining state variables, including the inflammatory marker A^*, remain zero at equilibrium. This plot illustrates the position of E_2 in state space and highlights that infection persists in the host population while the vector population remains extinct.

4.

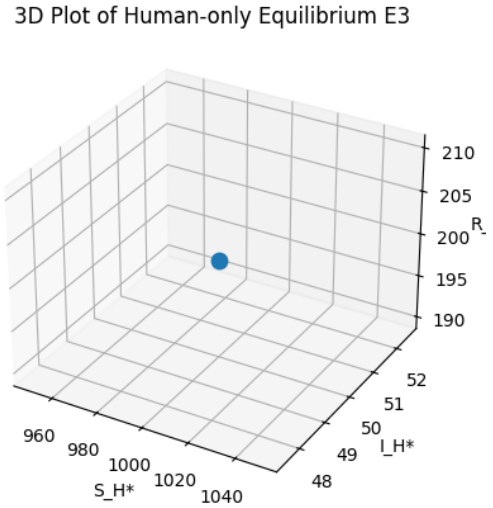

Figure 5: The 3D plot illustrates the human only equilibrium point E_3.

The fig.(5) 3D plot illustrates the human-only equilibrium point E_3 in terms of the susceptible (S_H^*), infected (I_H^*), and recovered (R_H^*) human populations. The point marks the steady-state values where the human subsystem remains in equilibrium without mosquito interaction.

5.

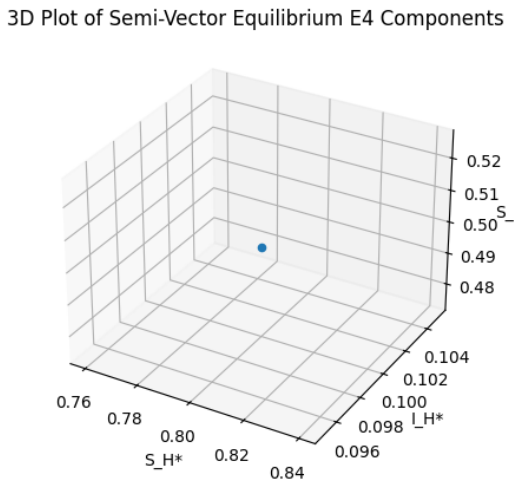

Figure 6: The semi-vector equilibrium point E_4 using the components S_H^*, I_H^*.

The fig(6) 3D plot displays the semi-vector equilibrium point E_4 using the components S_H^*, I_H^*, and S_M^* that characterize the coexistence of humans and vectors. It highlights the steady-state values where both populations persist with low-level infections and stable dynamics. **6.**

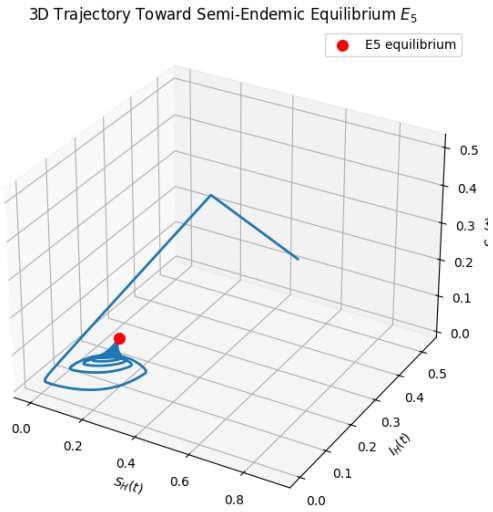

Figure 7: Semi-endemic equilibrium E_5.

The fig(7) 3D trajectory illustrates how the system evolves over time toward the semi-endemic equilibrium E_5 in the $(S_H(t), I_H(t), S_M(t))$ space. The spiraling path shows the transient oscillations before stabilizing at the equilibrium point marked in red.

7.

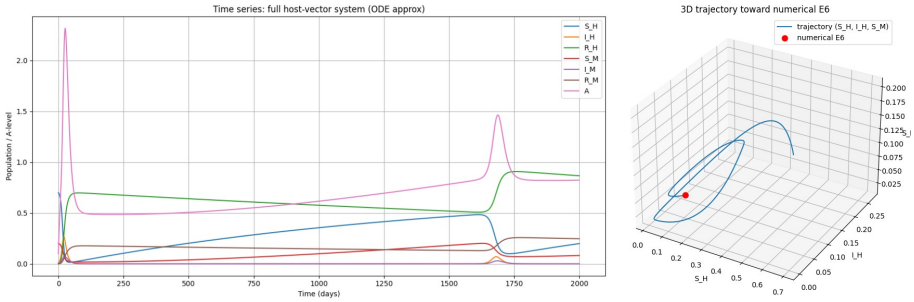

Figure 8: Dynamics of the full host vector immuno-metabolic system.

In fig.(8) illustrate that **Left:** TimeŰseries solutions of all seven state variables $S_H(t)$, $I_H(t)$, $R_H(t)$, $S_M(t)$, $I_M(t)$, $R_M(t)$, and $A(t)$ over a 2000-day simulation window. The system exhibits sharp initial transient peaks, particularly in the inflammatory marker $A(t)$ and the infected classes, followed by a long-term approach toward steady-state levels. **Right:** A 3D phase-space trajectory in the (S_H, I_H, S_M) manifold, showing a spiraling path converging toward the numerically computed endemic equilibrium E_6 (marked by a red point). The contracting spiral indicates local asymptotic stability of E_6 and agrees with the theoretical stability results obtained for the system.

8.

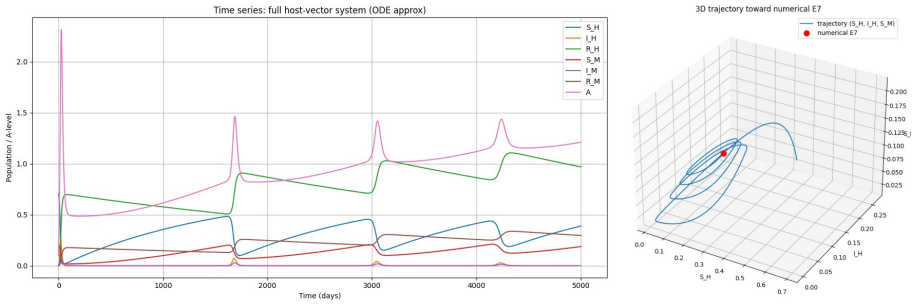

Figure 9: Trajectory of the full host vector immunoŰmetabolic infection model.

In fig.(9) illustrate that timeŰseries dynamics and 3D trajectory of the full hostŰvector immunoŰmetabolic infection model. **Left:** The temporal evolution of all seven state variables $(S_H, I_H, R_H, S_M, I_M, R_M, A)$ is shown over 5000 days. The system exhibits recurrent multiŰscale oscillations driven by the feedback between infection, metabolic impairment, and systemic inflammation. Peaks in the inflammatory marker $A(t)$ coincide with sharp surges in infected classes, while susceptible and recovered populations follow slower adaptive dynamics. **Right:** A 3D phase trajectory in the (S_H, I_H, S_M) space shows a spiraling approach toward the numerically computed endemic equilibrium E_7, marked by a red dot. The shrinking spiral indicates local asymptotic stability of E_7, consistent with the Lyapunov-based global stability analysis.

9.

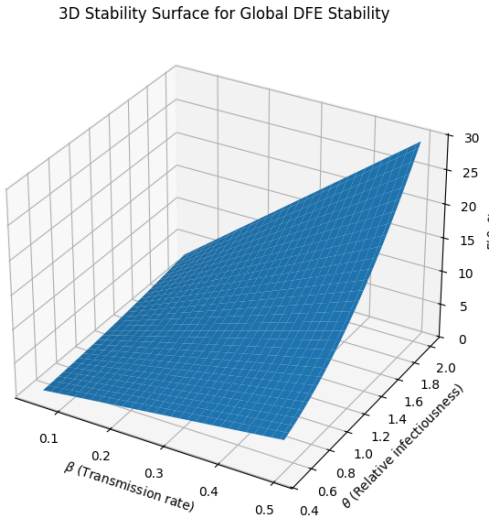

Figure 10: The figure shows how the stability function $F(\beta, \theta)$ changes with the transmission rate β and the infectiousness multiplier θ.

The fig.(10) illustrates how the stability function $F(\beta, \theta)$ varies with the transmission rate β and the infectiousness multiplier θ, using all parameter values listed in Table 1.

10.

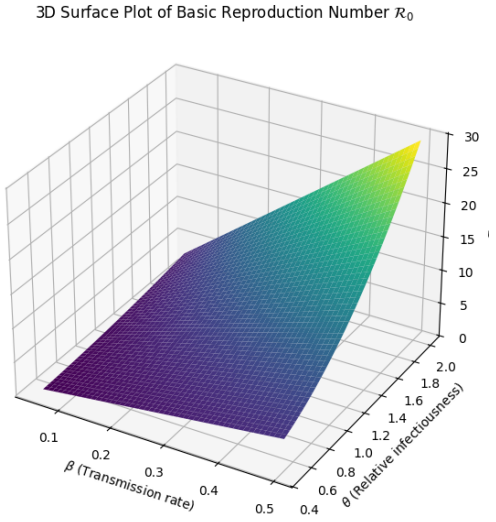

Figure 11: 3D Surface Plot of Basic Reproduction Number $\mathscr{R}_0$.

The 3D fig.(11) surface plot shows how the basic reproduction number $\mathscr{R}_0$ increases with the transmission rate β and the relative infectiousness θ. Higher values of β and θ lead to a steeper rise in $\mathscr{R}_0$, indicating enhanced disease spread potential.

11.

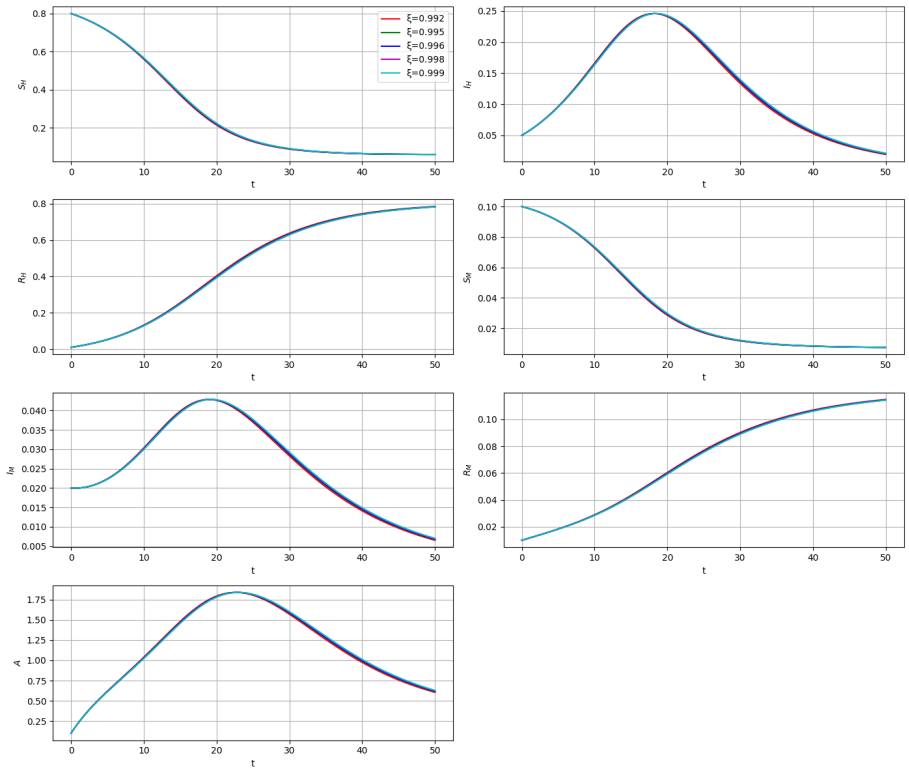

Figure 12: 3D plot of fractional order system.

In fig.(12) explained time evolution of the fractional-order system for different values of the fractional derivative ξ. Each curve corresponds to one of the fractional orders
$\xi = 0.992, 0.995, 0.996, 0.998, 0.999$, and the seven state variables $S_H, I_H, R_H, S_M, I_M, R_M, A$ are shown in separate subplots. Different colors represent different values of ξ.

Conclusion

This study presents a novel fractional-order immuno-metabolic epidemic model that integrates chronic inflammation, obesity, and type 2 diabetes mellitus (T2DM) to investigate their combined effects on infection dynamics in cats. By employing Caputo fractional derivatives, the model effectively captures the memory-dependent immune and metabolic processes that are not representable by classical integer-order models. We derived the basic reproduction number R_0 using the next-generation matrix approach and showed that metabolic impairment significantly increases the pathogenŠs transmission potential. Our stability analysis demonstrates that the disease-free equilibrium is locally and globally asymptotically stable when $R_0 < 1$, whereas the infection persists when $R_0 > 1$. These findings confirm that chronic inflammation and metabolic dysfunction are active drivers that exacerbate disease progression by heightening susceptibility, extending infectious periods, and hampering recovery in feline populations. The proposed framework provides a rigorous mathematical basis for understanding the impact of immuno-metabolic disorders on epidemic outcomes. Biologically, this highlights that inflammation associated with obesity and T2DM is not merely comorbid but fundamentally influences infection dynamics. Therefore, targeted veterinary interventions addressing metabolic health are essential to mitigate infectious disease burden among vulnerable cats. Future research could extend this model by incorporating stochasticity, optimal control strategies involving metabolic and anti-inflammatory therapies, and data-driven parameterization using clinical datasets. Overall, this work advances theoretical insights into immuno-metabolic interactions in epidemics and underscores the importance of metabolic factors in epidemic modeling.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this manuscript.

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Declaration

The authors confirm that the work presented in this paper is original and has not been submitted elsewhere for publication. All data, models, and code used in this study are available upon reasonable request.

Data Availability

The data used in this study are available upon reasonable request.

AuthorsŠ Contributions

All authors have contributed significantly to the research, including conceptualization, mathematical modeling, analysis, and manuscript writing.

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