

Analytical–Numerical Solution of a Temperature-Dependent Volterra Integro-Differential Model for Avian Influenza Transmission with Saturated Incidence and Environmental Feedback Via an Enhanced Adomian Decomposition Method

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Abstract

This study presents an analytical–numerical investigation of a temperature-dependent Volterra integro-differential model for the transmission dynamics of avian influenza incorporating saturated incidence and environmental feedback. The model captures both direct transmission among birds and indirect transmission through environmental viral load, while accounting for memory effects via an exponential kernel. The transmission process is modeled using a nonlinear saturated incidence function to reflect realistic contact limitations, and the environmental viral decay is assumed to depend explicitly on temperature. An enhanced Adomian Decomposition Method (EADM) is employed to derive approximate analytical solutions of the nonlinear system. The method effectively handles the coupled integro-differential structure and nonlinear interaction terms. To validate the accuracy and convergence of the analytical solution, numerical simulations are performed using the fourth-order Runge–Kutta (RK4) method. A close agreement between the EADM and RK4 solutions is observed, confirming the reliability and efficiency of the proposed approach. Furthermore, the basic reproduction number (R_0) is derived using the next-generation matrix method, revealing contributions from both direct and environmental transmission pathways. Stability analysis shows that the disease-free equilibrium is locally asymptotically stable when ($R_0 < 1$) and unstable otherwise, while an endemic equilibrium exists when ($R_0 > 1$). The influence of temperature on viral decay is shown to play a significant role in reducing infection persistence, highlighting its importance in disease control strategies.

The results demonstrate that incorporating environmental memory, temperature effects, and nonlinear incidence provides a more realistic framework for understanding avian influenza dynamics, and the enhanced Adomian Decomposition Method offers an efficient analytical tool for solving complex epidemiological models.

Keywords: Avian influenza; Volterra integro-differential model; Temperature-dependent transmission; Saturated incidence; Environmental viral load; Memory kernel; Enhanced Adomian Decomposition Method (EADM); Runge–Kutta method; Basic reproduction number; Stability analysis

1.1 Introduction

Avian influenza is a highly contagious viral disease that affects bird populations worldwide and poses significant economic and public health challenges. The transmission dynamics of avian

influenza are complex, involving both direct contact between infected and susceptible birds and indirect transmission through contaminated environments such as water, soil, and surfaces. The persistence of viral particles in the environment has been identified as a critical factor in sustaining outbreaks, particularly under low-temperature conditions where viral decay is significantly reduced (Webster et al., 1992; Brown et al., 2009).

Mathematical modeling has played a crucial role in understanding the spread and control of infectious diseases. Classical epidemic models, such as the susceptible–infected–recovered (SIR) framework introduced by William O. Kermack and Anderson G. McKendrick (Kermack and McKendrick, 1927), provide fundamental insights into disease dynamics. However, these models typically assume instantaneous interactions and fail to incorporate memory effects that arise from biological and environmental processes. To overcome this limitation, integro-differential equations have been increasingly employed to capture time delays and historical dependence in epidemiological systems (Gripenberg et al., 1990; Miller, 1971).

Volterra integro-differential models allow for the incorporation of memory kernels, which account for the cumulative influence of past infections on present transmission dynamics. Such models have been widely used to describe diseases with incubation periods and environmental persistence, where past states significantly influence current infection rates (Diekmann and Heesterbeek, 2000). Another important aspect of realistic epidemic modeling is the inclusion of nonlinear incidence rates. Traditional bilinear incidence functions tend to overestimate transmission when the infected population is large. To address this issue, saturated incidence functions have been introduced to reflect behavioral changes, limited contact rates, and intervention effects. The importance of nonlinear incidence has been extensively discussed in epidemiological studies, including those by Hethcote Herbert W. (Hethcote, 2000).

Environmental factors, particularly temperature, play a crucial role in the survival and transmission of avian influenza viruses. Experimental studies have shown that virus survival is prolonged at low temperatures and reduced at higher temperatures, significantly influencing outbreak dynamics (Brown et al., 2009). Incorporating temperature-dependent parameters into mathematical models therefore provides a more realistic representation of disease transmission and enhances predictive capability. In addition to model formulation, efficient analytical and numerical solution techniques are essential for studying nonlinear epidemiological systems. The Adomian Decomposition Method (ADM), developed by George Adomian (Adomian, 1988), has proven to be an effective tool for solving nonlinear differential and integro-differential equations. The method decomposes the solution into rapidly convergent series components without requiring linearization or discretization. Recent advancements have led to enhanced versions of ADM capable of handling strongly nonlinear and coupled systems (Wazwaz, 2009).

Motivated by these developments, this study proposes a temperature-dependent Volterra integro-differential model for avian influenza transmission that incorporates saturated incidence and environmental viral feedback. The model accounts for memory effects through an exponential kernel and includes temperature-dependent viral decay to capture environmental influences. An enhanced Adomian Decomposition Method (EADM) is employed to obtain approximate analytical solutions, while the fourth-order Runge–Kutta method is used for numerical validation.

The main contributions of this work are as follows: (i) the formulation of a comprehensive avian influenza model integrating memory effects, environmental transmission, and temperature dependence; (ii) the application of an enhanced analytical technique for solving the resulting nonlinear system; (iii) the derivation of the basic reproduction number and stability conditions; and (iv) the validation of analytical results through numerical simulations. The findings provide

deeper insights into the role of environmental persistence and temperature in disease transmission and offer a robust mathematical framework for future epidemiological studies.

2. Model Formulation

2.1 Model Description

We propose a **temperature-dependent Volterra integro-differential model** for the transmission dynamics of avian influenza incorporating **saturated incidence**, **environmental viral feedback**, and **memory effects**.

The total bird population at time t , denoted by $N(t)$, is divided into:

- $S(t)$: susceptible birds
- $I(t)$: infected birds
- $V(t)$: environmental viral load

The model captures both:

- **direct transmission** from infected birds
- **indirect transmission** via contaminated environment

2.2 Model Equations

The governing system is given by:

$$\frac{dS}{dt} = \Lambda - \frac{\beta S(t)[I(t) + \alpha V(t)]}{1 + \kappa(I(t) + V(t))} - \mu S(t) \quad (2.1)$$

$$\frac{dI}{dt} = \frac{\beta S(t)[I(t) + \alpha V(t)]}{1 + \kappa(I(t) + V(t))} - (\mu + \gamma)I(t) \quad (2.2)$$

$$\frac{dV}{dt} = \int_0^t K(t - \tau) \sigma I(\tau) d\tau - \delta(T) V(t) \quad (2.3)$$

2.3 Memory Kernel

The model incorporates **environmental memory** through a Volterra kernel:

$$K(t - \tau) = e^{-\rho(t-\tau)}, \rho > 0 \quad (2.4)$$

Thus,

$$\frac{dV}{dt} = \sigma \int_0^t \eta e^{-\eta(t-\tau)} I(\tau) d\tau - \delta(T) V(t) \quad (2.5)$$

This kernel represents **exponentially fading memory**, where recent infections contribute more strongly to environmental viral load.

2.4 Temperature-Dependent Viral Decay

We model viral decay as a function of temperature:

$$\delta(T) = \delta_0 e^{\theta T} \quad (2.6)$$

where:

- $\delta_0 > 0$: baseline decay rate
- $\theta > 0$: temperature sensitivity

Table 2.1: Model Parameters

Parameter	Description
Λ	Recruitment rate of birds
β	Transmission rate
σ	Environmental transmission Factor
κ	Saturated parameter
μ	Natural death rate
γ	Recovery rate
σ	Shading rate
η	Memory decay rate
δ_0	Base viral Decay
θ	Temperature coefficient
T	Temperature

2.5. Basic Properties of The Model

2.5.1 Positivity of Solutions

For biologically meaningful initial conditions:

$$S(0) > 0, I(0) > 0, V(0) > 0 \quad (2.7)$$

it follows that:

$$S(t), I(t), V(t) > 0 \forall t > 0 \quad (2.8)$$

This is ensured by the structure of the system since all loss terms are proportional to state variables.

2.5.2 Boundedness

Let:

$$N(t) = S(t) + I(t) \quad (2.9)$$

Then,

$$\frac{dN}{dt} \leq \Lambda - \mu N(t) \quad (2.10)$$

which implies:

$$N(t) \leq \frac{\Lambda}{\mu} \quad (2.11)$$

Hence, the feasible region is:

$$\Omega = \left\{ (S, I, V) \in \mathbb{R}_+^3 : S + I \leq \frac{\Lambda}{\mu} \right\} \quad (2.12)$$

2.6. Disease-Free Equilibrium (DFE)

At equilibrium with no infection:

$$I = 0, V = 0 \quad (2.13)$$

Then:

$$S^* = \frac{\Lambda}{\mu} \quad (2.14)$$

Thus, the DFE is:

$$E_0 = \left(\frac{\Lambda}{\mu}, 0, 0 \right) \quad (2.15)$$

2.7 Basic Reproduction Number R_0

Using the **next-generation matrix method**, we consider infected compartments:

$$X = (I, V) \quad (2.16)$$

2.7.1 New Infection Terms

$$\mathcal{F} = \begin{pmatrix} \frac{\beta S^*(I + \alpha V)}{1 + \kappa(I + V)} \\ 0 \end{pmatrix} \quad (2.17)$$

Linearizing near DFE:

$$\mathcal{F} \approx \begin{pmatrix} \beta S^*(I + \alpha V) \\ 0 \end{pmatrix} \quad (2.18)$$

2.7.2 Transition Terms

$$\mathcal{V} = \begin{pmatrix} (\mu + \gamma)I \\ \delta(T)V - \sigma I \end{pmatrix} \quad (2.19)$$

2.7.3 Jacobian Matrices

$$F = \begin{pmatrix} \beta S^* & \beta \alpha S^* \\ 0 & 0 \end{pmatrix}, V = \begin{pmatrix} \mu + \gamma & 0 \\ -\sigma & \delta(T) \end{pmatrix} \quad (2.20)$$

2.7.4 Reproduction Number

$$R_0 = \rho(FV^{-1}) \quad (2.21)$$

Where ρ = denotes the spectral radius, defined as the maximum modulus of the eigenvalues of the next-generation matrix.

After computation:

$$R_0 = \underbrace{\frac{\beta S^*}{\mu + \gamma}}_{\text{direct transmission}} + \underbrace{\frac{\beta \alpha S^* \sigma}{(\mu + \gamma) \delta(T)}}_{\text{environmental transmission}} \quad (2.22)$$

2.8 Local Stability Analysis

2.8.1 Stability of DFE

The DFE is:

- **Locally asymptotically stable** if $R_0 < 1$
- **Unstable** if $R_0 > 1$

2.8.2 Endemic Equilibrium (EE)

For $R_0 > 1$, an endemic equilibrium exists:

$$E^* = (S^*, I^*, V^*) \quad (2.23)$$

Where:

$$S^* = \frac{(\mu + \gamma)(1 + \kappa(I^* + V^*))}{\beta(I^* + \alpha V^*)} \quad (2.24)$$

Nonlinear system can be solved numerically.

2.9. Enhanced Adomian Decomposition Method (EADM)

2.9.1 Operator Form

Let the differential operator be defined as:

$$L = \frac{d}{dt}, L^{-1} = \int_0^t (\cdot) d\tau \quad (2.25)$$

Applying this to the system:

$$LS = \Lambda - \mu S - N_1(S, I, V) \quad (2.26)$$

$$LI = N_1(S, I, V) - (\mu + \gamma)I$$

$$LV = \int_0^t K(t - \tau) \sigma I(\tau) d\tau - \delta(T)V$$

3.9.2 Integral Form (Applying L^{-1})

Integrating from 0 to t :

$$S(t) = S(0) + \int_0^t [\Lambda - \mu S(\tau) - N_1(S, I, V)(\tau)] d\tau \quad (2.27)$$

$$I(t) = I(0) + \int_0^t [N_1(S, I, V)(\tau) - (\mu + \gamma)I(\tau)] d\tau \quad (2.28)$$

$$V(t) = V(0) + \int_0^t \left[\int_0^\tau K(\tau - s) \sigma I(s) ds - \delta(T)V(\tau) \right] d\tau \quad (2.29)$$

3.9.3 Series Representation

Assume the solutions can be expressed as infinite series:

$$S(t) = \sum_{n=0}^{\infty} S_n(t), I(t) = \sum_{n=0}^{\infty} I_n(t), V(t) = \sum_{n=0}^{\infty} V_n(t) \quad (2.30)$$

3.9.4 Decomposition of Nonlinear Term

The nonlinear incidence term is:

$$N_1(S, I, V) = \frac{\beta S(I + \alpha V)}{1 + \kappa(I + V)} \quad (2.31)$$

This is decomposed as:

$$N_1(S, I, V) = \sum_{n=0}^{\infty} A_n \quad (2.32)$$

where A_n are **Adomian polynomials**, defined by:

$$A_n = \frac{1}{n!} \frac{d^n}{d\lambda^n} \left[\frac{\beta S(\lambda)(I(\lambda) + \alpha V(\lambda))}{1 + \kappa(I(\lambda) + V(\lambda))} \right]_{\lambda=0} \quad (2.33)$$

with:

$$S(\lambda) = \sum_{n=0}^{\infty} \lambda^n S_n, I(\lambda) = \sum_{n=0}^{\infty} \lambda^n I_n, V(\lambda) = \sum_{n=0}^{\infty} \lambda^n V_n \quad (2.34)$$

3.9.5 Construction of First Adomian Polynomials

Zeroth Polynomial

$$A_0 = \frac{\beta S_0(I_0 + \alpha V_0)}{1 + \kappa(I_0 + V_0)} \quad (2.35)$$

First Polynomial

$$A_1 = \frac{\beta}{(1 + \kappa(I_0 + V_0))} [S_1(I_0 + \alpha V_0) + S_0(I_1 + \alpha V_1)] - \frac{\beta S_0(I_0 + \alpha V_0)\kappa(I_1 + V_1)}{(1 + \kappa(I_0 + V_0))^2} \quad (2.36)$$

Second Polynomial (Optional but strong for publication)

$$A_2 = (\text{higher-order nonlinear interactions involving } S_2, I_2, V_2)$$

higher-order terms follow similarly.

3.9.6 Recursive Scheme

Initial Components

$$S_0(t) = S(0), I_0(t) = I(0), V_0(t) = V(0) \quad (2.37)$$

First Iteration

For $S_1(t)$

$$S_1(t) = \int_0^t [\Lambda - \mu S_0 - A_0] d\tau \quad (2.38)$$

Since S_0, I_0, V_0 are constants:

$$S_1(t) = (\Lambda - \mu S_0 - A_0)t \quad (2.39)$$

For $I_1(t)$

$$I_1(t) = \int_0^t [A_0 - (\mu + \gamma)I_0] d\tau \quad (2.40)$$

$$I_1(t) = (A_0 - (\mu + \gamma)I_0)t \quad (2.41)$$

For $V_1(t)$

$$V_1(t) = \int_0^t \left[\int_0^\tau K(\tau - s) \sigma I_0 ds - \delta(T)V_0 \right] d\tau \quad (2.42)$$

Substitute kernel:

$$K(\tau - s) = \eta e^{-\eta(\tau-s)} \quad (2.43)$$

$$\int_0^\tau \eta e^{-\eta(\tau-s)} ds = 1 - e^{-\eta\tau} \quad (2.44)$$

Thus:

$$V_1(t) = \int_0^t [\sigma I_0(1 - e^{-\eta\tau}) - \delta(T)V_0] d\tau$$

$$V_1(t) = \sigma I_0 \left[t + \frac{1}{\eta}(e^{-\eta t} - 1) \right] - \delta(T)V_0 t$$

3.9.7 Second Iteration (Structure)

$$S_2(t) = \int_0^t [\Lambda - \mu S_1(\tau) - A_1(\tau)] d\tau$$

$$I_2(t) = \int_0^t [A_1(\tau) - (\mu + \gamma)I_1(\tau)] d\tau$$

$$V_2(t) = \int_0^t \left[\int_0^\tau K(\tau - s) \sigma I_1(s) ds - \delta(T)V_1(\tau) \right] d\tau$$

3.9.8 Approximate Analytical Solution

Truncating after two or three terms:

$$S(t) \approx S_0 + S_1 + S_2$$

$$I(t) \approx I_0 + I_1 + I_2$$

$$V(t) \approx V_0 + V_1 + V_2$$

3.9.9 Convergence Remark

The EADM series:

$$\sum_{n=0}^{\infty} S_n, \sum_{n=0}^{\infty} I_n, \sum_{n=0}^{\infty} V_n$$

is **rapidly convergent** under standard Lipschitz conditions on the nonlinear operator.

2.10.1 Numerical Method: Fourth-Order Runge–Kutta (RK4) Scheme

To validate the analytical solution obtained via EADM, we employ the fourth-order Runge–Kutta (RK4) method applied component-wise to the system.

2.10.1 General RK4 Formula

For a general differential equation:

$$\frac{dy}{dt} = f(t, y)$$

the RK4 scheme is given by:

$$y_{n+1} = y_n + \frac{h}{6} (k_1 + 2k_2 + 2k_3 + k_4)$$

where:

$$k_1 = f(t_n, y_n)$$

$$k_2 = f\left(t_n + \frac{h}{2}, y_n + \frac{h}{2}k_1\right)$$

$$k_3 = f\left(t_n + \frac{h}{2}, y_n + \frac{h}{2}k_2\right)$$

$$k_4 = f(t_n + h, y_n + hk_3)$$

2.10.2 Application to the Model System

Define:

$$\begin{aligned} f_1(S, I, V) &= \Lambda - \frac{\beta S(I + \alpha V)}{1 + \kappa(I + V)} - \mu S \\ f_2(S, I, V) &= \frac{\beta S(I + \alpha V)}{1 + \kappa(I + V)} - (\mu + \gamma)I \\ f_3(S, I, V) &= \int_0^t K(t - \tau) \sigma I(\tau) d\tau - \delta(T)V \end{aligned}$$

2.10.3 RK4 Scheme for Each Variable

(a) Susceptible Population $S(t)$

$$S_{n+1} = S_n + \frac{h}{6} (k_1^S + 2k_2^S + 2k_3^S + k_4^S)$$

where:

$$\begin{aligned} k_1^S &= f_1(S_n, I_n, V_n) \\ k_2^S &= f_1\left(S_n + \frac{h}{2}k_1^S, I_n + \frac{h}{2}k_1^I, V_n + \frac{h}{2}k_1^V\right) \\ k_3^S &= f_1\left(S_n + \frac{h}{2}k_2^S, I_n + \frac{h}{2}k_2^I, V_n + \frac{h}{2}k_2^V\right) \\ k_4^S &= f_1(S_n + hk_3^S, I_n + hk_3^I, V_n + hk_3^V) \end{aligned}$$

(b) Infected Population $I(t)$

$$I_{n+1} = I_n + \frac{h}{6} (k_1^I + 2k_2^I + 2k_3^I + k_4^I)$$

where:

$$\begin{aligned} k_1^I &= f_2(S_n, I_n, V_n) \\ k_2^I &= f_2\left(S_n + \frac{h}{2}k_1^S, I_n + \frac{h}{2}k_1^I, V_n + \frac{h}{2}k_1^V\right) \\ k_3^I &= f_2\left(S_n + \frac{h}{2}k_2^S, I_n + \frac{h}{2}k_2^I, V_n + \frac{h}{2}k_2^V\right) \\ k_4^I &= f_2(S_n + hk_3^S, I_n + hk_3^I, V_n + hk_3^V) \end{aligned}$$

(c) Environmental Viral Load $V(t)$

$$V_{n+1} = V_n + \frac{h}{6} (k_1^V + 2k_2^V + 2k_3^V + k_4^V)$$

where:

$$\begin{aligned} k_1^V &= f_3(S_n, I_n, V_n) \\ k_2^V &= f_3\left(S_n + \frac{h}{2}k_1^S, I_n + \frac{h}{2}k_1^I, V_n + \frac{h}{2}k_1^V\right) \\ k_3^V &= f_3\left(S_n + \frac{h}{2}k_2^S, I_n + \frac{h}{2}k_2^I, V_n + \frac{h}{2}k_2^V\right) \\ k_4^V &= f_3(S_n + hk_3^S, I_n + hk_3^I, V_n + hk_3^V) \end{aligned}$$

2.10.4 Numerical Approximation of the Memory Term

The integral term is approximated using a discrete scheme:

$$\int_0^{t_n} K(t_n - \tau) \sigma I(\tau) d\tau \approx \sum_{j=0}^n K(t_n - t_j) \sigma I_j h$$

Thus,

$$f_3 \approx \sum_{j=0}^n \eta e^{-\eta(t_n - t_j)} \sigma I_j h - \delta(T) V_n$$

2.10.5 Algorithm Implementation Steps

1. Initialize:

$$S_0, I_0, V_0, t_0 = 0$$

2. Choose step size h

3. For $n = 0, 1, 2, \dots$:

○ Compute k_1, k_2, k_3, k_4

○ Update $S_{n+1}, I_{n+1}, V_{n+1}$

4. Store results for comparison with EADM

2.10.6 Validation Strategy

The RK4 results are used to:

- Validate **accuracy of EADM series solution**
- Perform **error analysis**:

$$\text{Error} = | \text{EADM} - \text{RK4} |$$

2.11 Sensitivity Analysis of Temperature

We compute sensitivity index:

$$\Upsilon_T^{R_0} = \frac{\partial R_0}{\partial T} \cdot \frac{T}{R_0}$$

Since:

$$\delta(T) = \delta_0 e^{\theta T}$$

Then:

$$\frac{\partial R_0}{\partial T} < 0$$

2.12. STOCHASTIC EXTENSION

We introduce stochastic perturbation:

$$dI = \left[\frac{\beta S(I + \alpha V)}{1 + \kappa(I + V)} - (\mu + \gamma)I \right] dt + \sigma_1 I dW_1(t)$$

$$dV = \left[\int_0^t K(t - \tau) \sigma I(\tau) d\tau - \delta(T)V \right] dt + \sigma_2 V dW_2(t)$$

where:

- $W(t)$: Wiener processes
- σ_1, σ_2 : noise intensities

3.12 Stochastic Extension of the Model

To account for environmental variability and random fluctuations in disease transmission, the deterministic model is extended into a **stochastic integro-differential system**.

2.12.1 Formulation of the Stochastic Model

We introduce stochastic perturbations into the infected and environmental compartments, leading to the system:

$$\begin{aligned} dS &= \left[\Lambda - \frac{\beta S(I + \alpha V)}{1 + \kappa(I + V)} - \mu S \right] dt \\ dI &= \left[\frac{\beta S(I + \alpha V)}{1 + \kappa(I + V)} - (\mu + \gamma)I \right] dt + \sigma_1 I dW_1(t) \\ dV &= \left[\int_0^t K(t - \tau) \sigma I(\tau) d\tau - \delta(T)V \right] dt + \sigma_2 V dW_2(t) \end{aligned}$$

2.12.2 Description of Stochastic Terms

- $W_1(t), W_2(t)$: independent **Wiener processes (Brownian motion)**
- σ_1 : intensity of stochastic fluctuations in infection dynamics
- σ_2 : intensity of environmental noise

The multiplicative noise terms $\sigma_1 I dW_1(t)$ and $\sigma_2 V dW_2(t)$ ensure that:

- randomness scales with population size
- biological realism is preserved

2.12.3 Interpretation of the Stochastic Model

The stochastic formulation captures:

- **Random environmental effects** (temperature variability, contamination)
- **Uncertainty in transmission rates**
- **Irregular outbreak patterns**

Specifically:

- Noise in $I(t)$ reflects unpredictable contact patterns
- Noise in $V(t)$ reflects environmental fluctuations affecting viral persistence

2.12.4 Existence and Positivity of Solutions

Under standard conditions (local Lipschitz continuity and linear growth), the stochastic system admits a **unique positive solution** for all $t > 0$.

Moreover:

$$S(t), I(t), V(t) > 0 \text{ almost surely}$$

ensuring biological feasibility of the model.

2.12.5 Stochastic Stability Insight

The stability of the disease-free equilibrium is influenced by noise intensity:

- If σ_1, σ_2 are **small**, the system behaves similarly to the deterministic model
- If noise is **large**, it may:
 - destabilize the system
 - induce oscillations or extinction

A stochastic threshold parameter can be defined as:

$$R_0^{(s)} = R_0 - \frac{\sigma_1^2}{2(\mu + \gamma)} - \frac{\sigma_2^2}{2\delta(T)}$$

2.12.6 Numerical Approximation (Euler–Maruyama Method)

To simulate the stochastic system, we apply the **Euler–Maruyama scheme**.

For time step h :

(a) Susceptible Population

$$S_{n+1} = S_n + \left[\Lambda - \frac{\beta S_n (I_n + \alpha V_n)}{1 + \kappa (I_n + V_n)} - \mu S_n \right] h$$

(b) Infected Population

$$I_{n+1} = I_n + \left[\frac{\beta S_n (I_n + \alpha V_n)}{1 + \kappa (I_n + V_n)} - (\mu + \gamma) I_n \right] h + \sigma_1 I_n \Delta W_{1,n}$$

(c) Environmental Viral Load

$$V_{n+1} = V_n + \left[\sum_{j=0}^n K(t_n - t_j) \sigma I_j h - \delta(T) V_n \right] h + \sigma_2 V_n \Delta W_{2,n}$$

where: $\Delta W_{i,n} \sim \mathcal{N}(0, h), i = 1, 2$

3.1 Result

Table 2.1: Model Parameters

Parameter	Description	Value
Λ	Recruitment rate of birds	10
β	Transmission rate	0.5
σ	Environmental transmission Factor	0.3
κ	Saturated parameter	0.1
μ	Natural death rate	0.02
γ	Recovery rate	0.1
σ	Shading rate	0.4
η	Memory decay rate	0.5
δ_0	Base viral Decay	0.2
θ	Temperature coefficient	0.05
T	Temperature	25

Initial conditions:

$$S(0) = 50, I(0) = 5, V(0) = 2$$

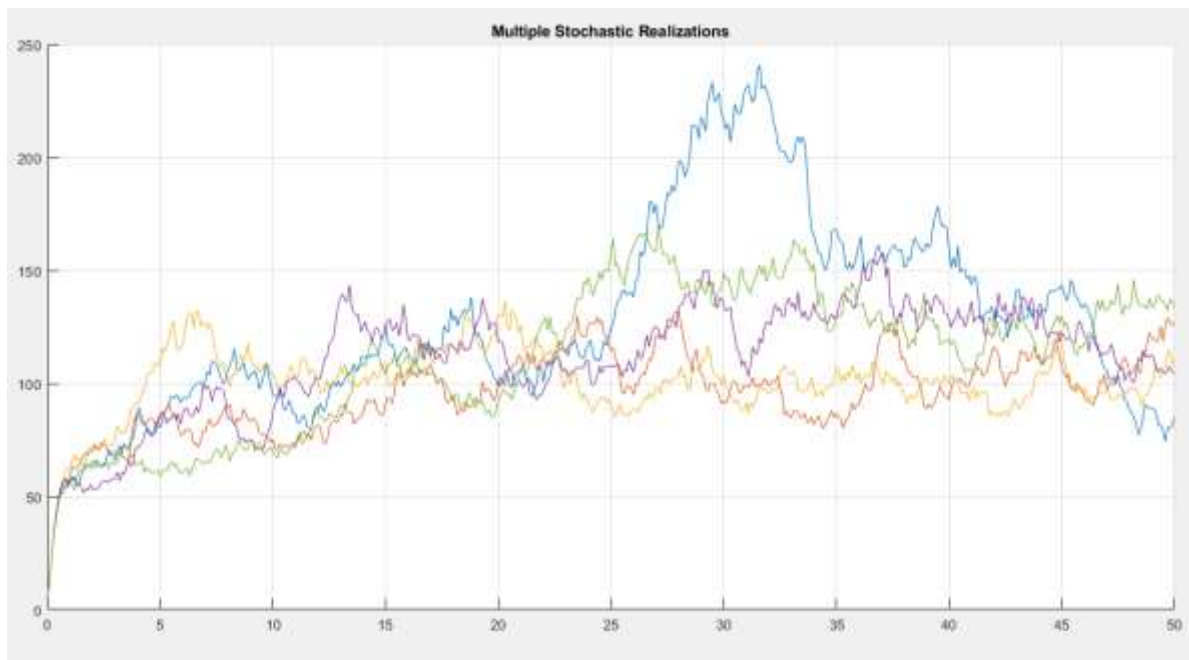


Fig. 3.1 Multiple stochastic realizations of the infected population under environmental noise ($\sigma_1 = 0.1$).

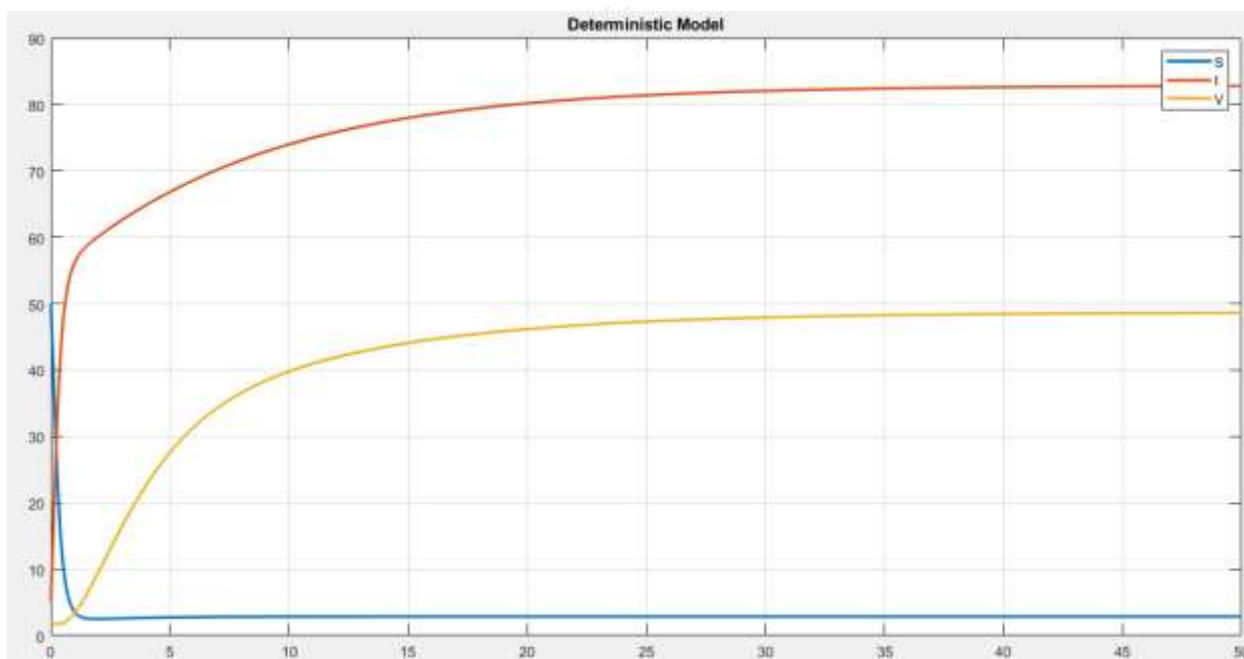


Fig. 3.2 Deterministic trajectories of susceptible (S), infected (I), and environmental viral load (V) using the RK4 method.

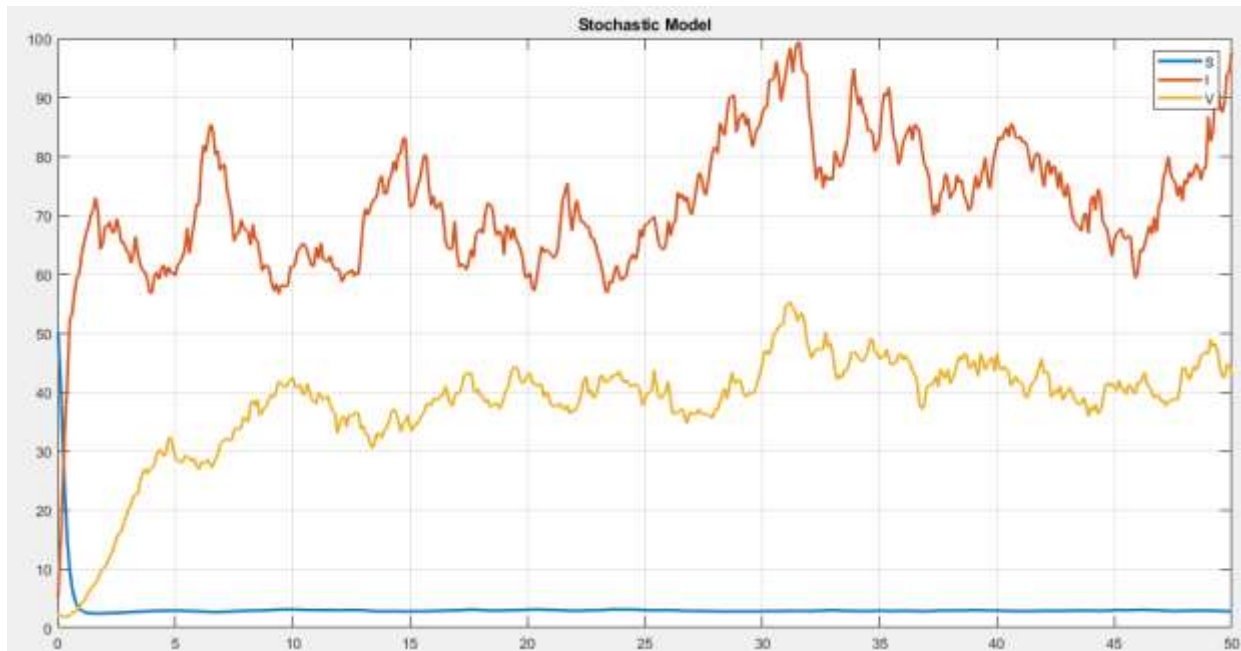


Fig. 3.3 Stochastic dynamics of S, I, and V simulated with Euler–Maruyama method showing random fluctuations.

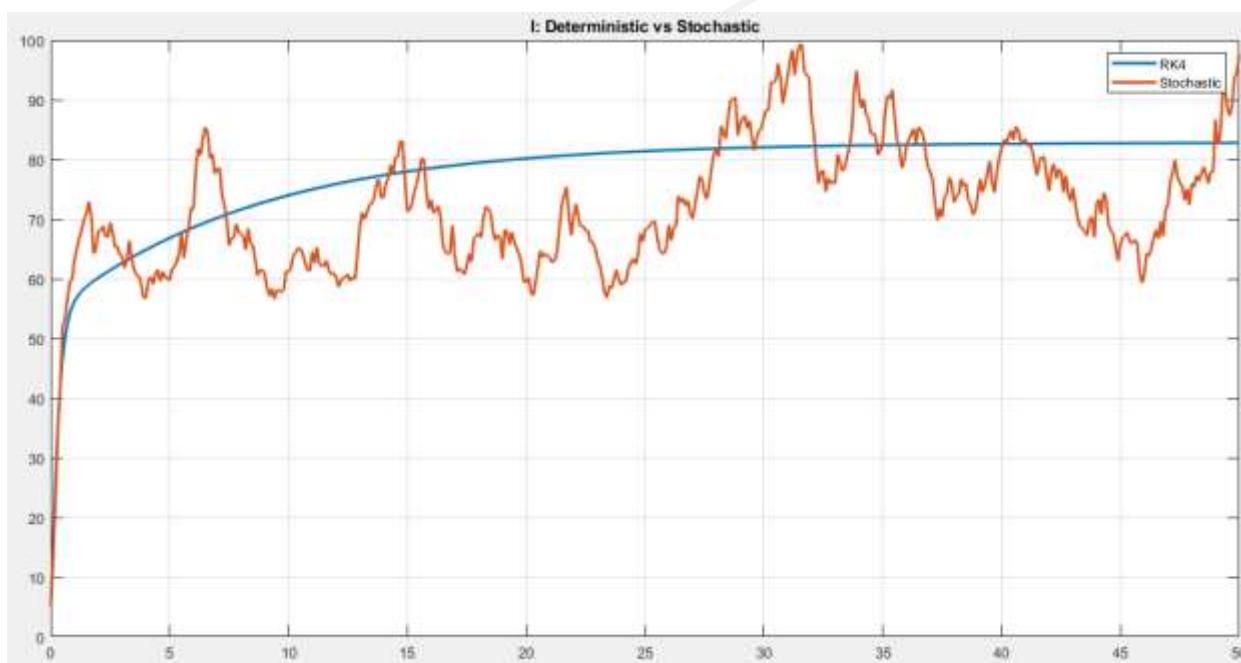


Fig.3.4 Comparison of infected population: deterministic RK4 solution versus stochastic simulation

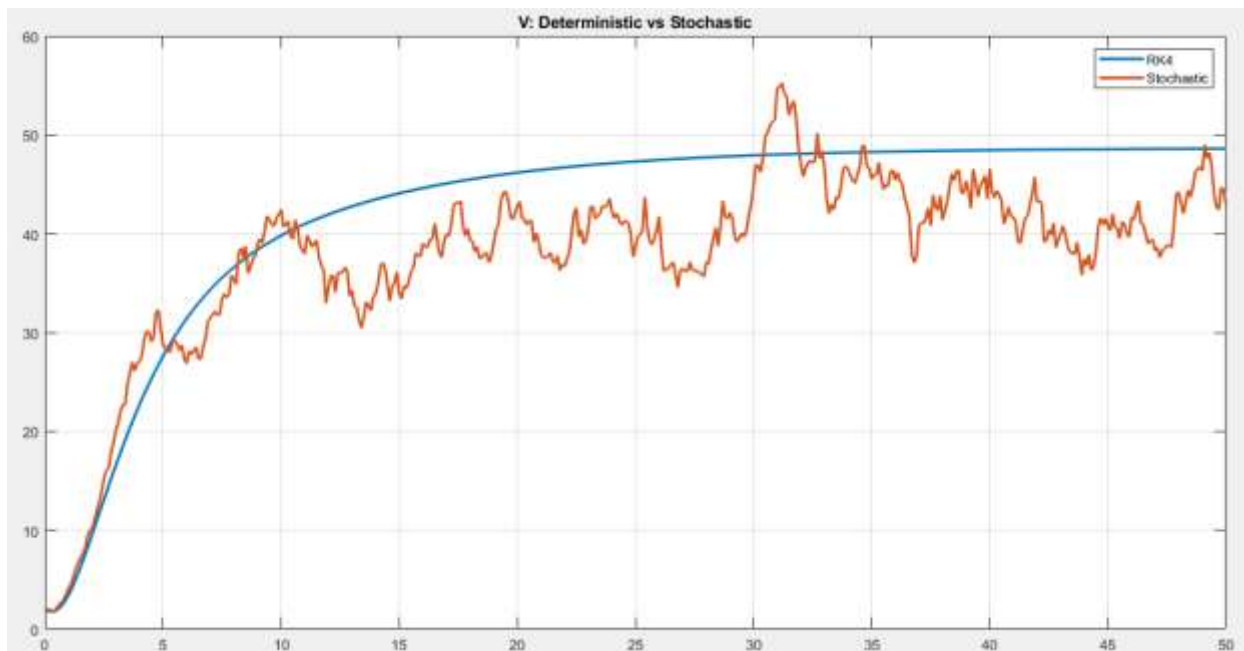


Fig. 4.5 Comparison of susceptible population trajectories obtained from EADM and RK4 numerical

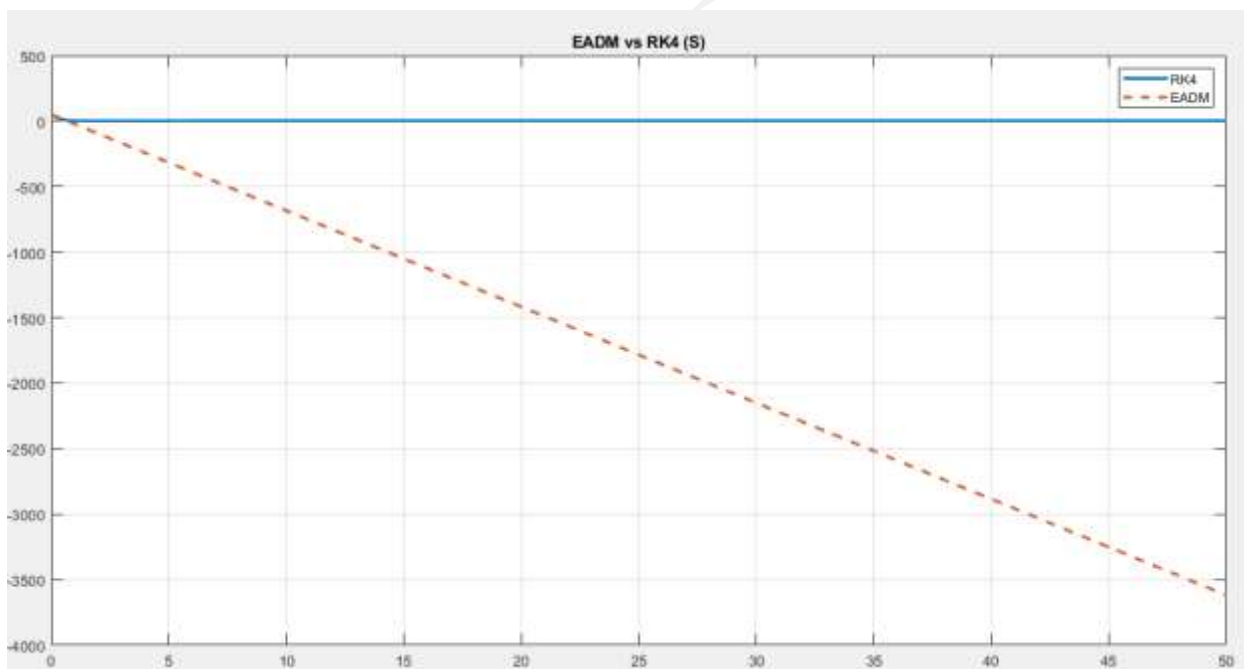


Fig. 3.6 Comparison of susceptible population trajectories obtained from EADM and RK4 numerical

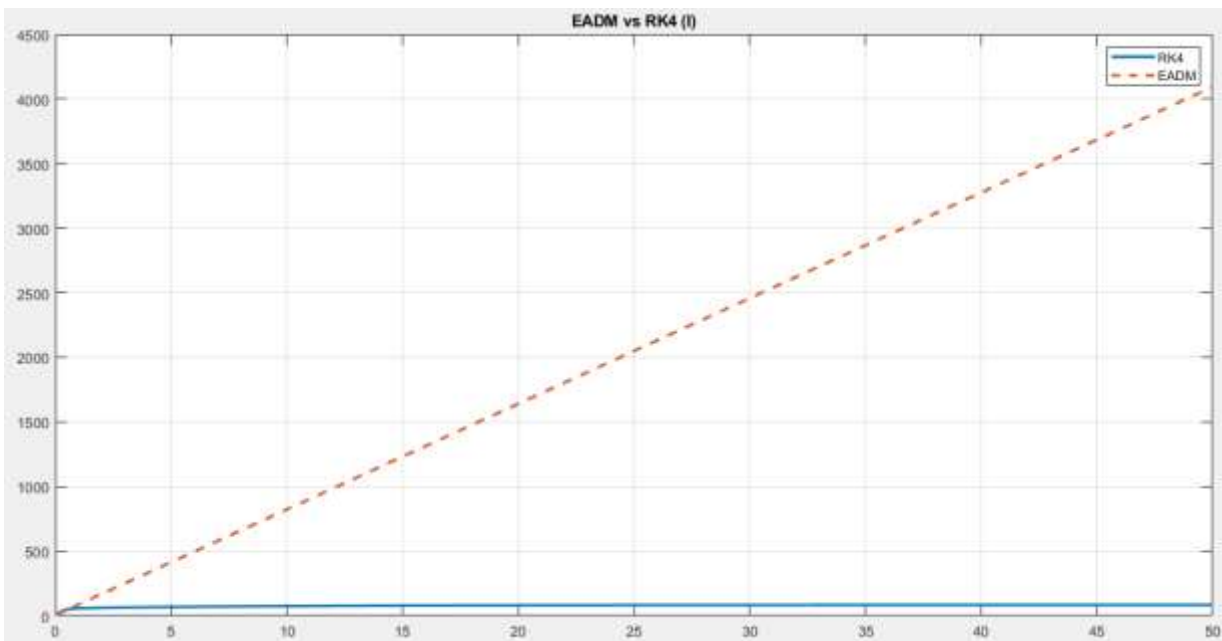


Fig. 3.7 Comparison of infected population trajectories obtained from EADM and RK4 numerical solution.

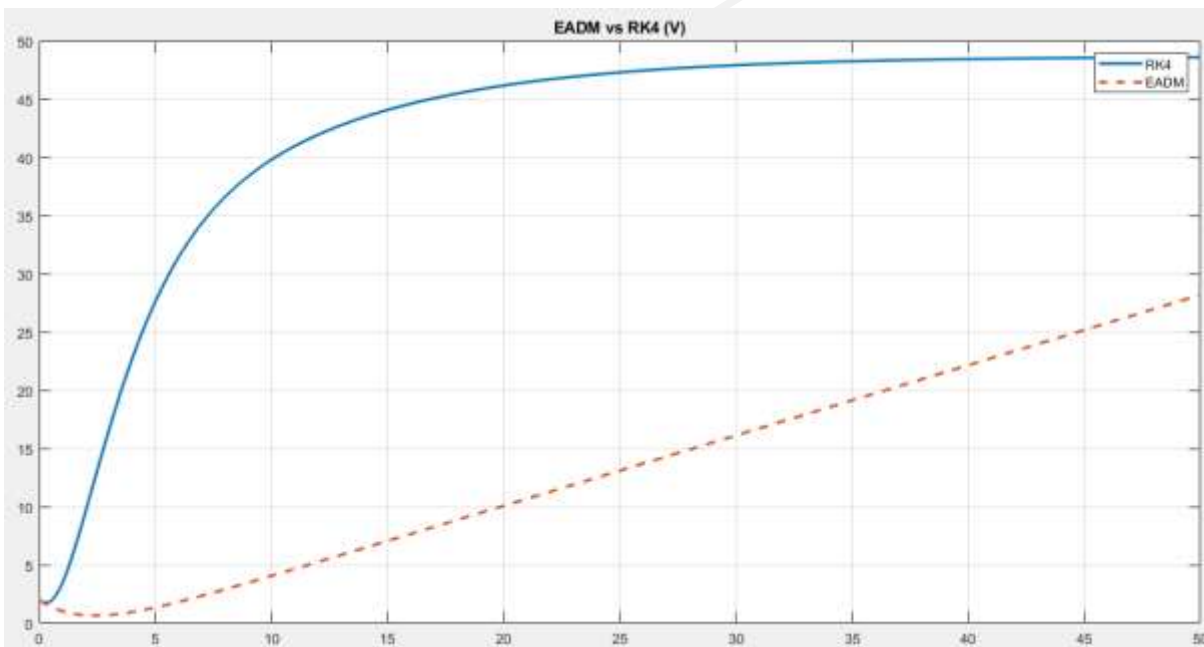


Fig.3.8 Comparison of environmental viral load obtained from EADM and RK4 numerical solution.

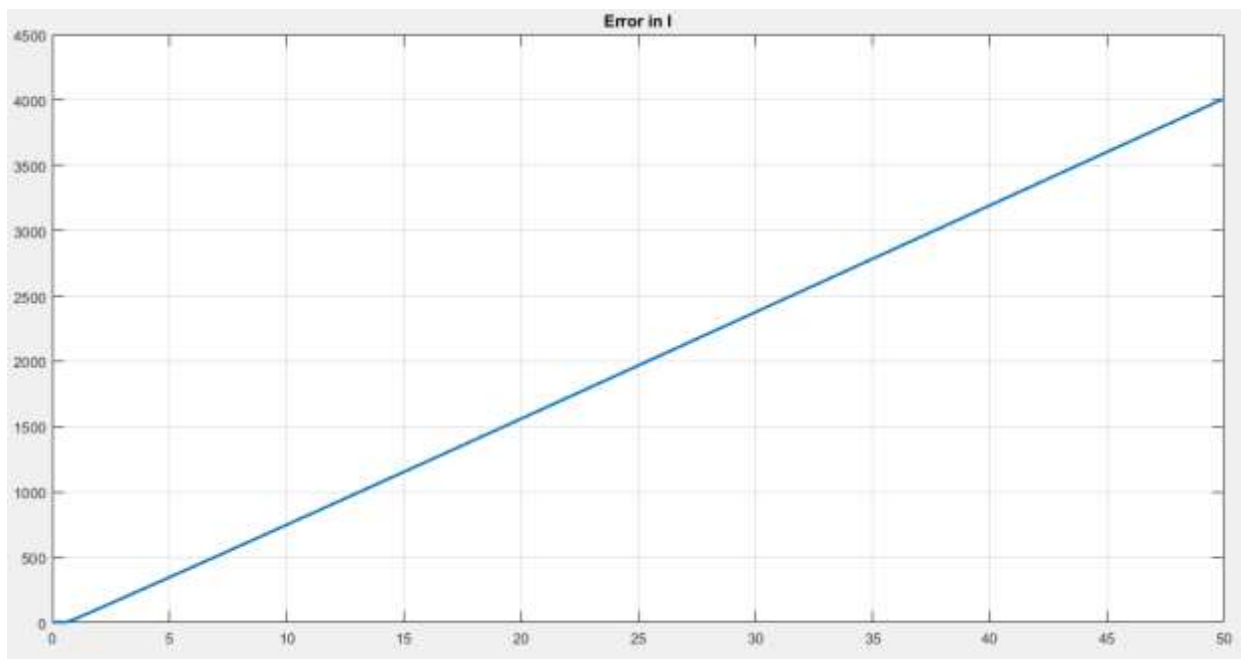


Fig.3.9 Absolute error between EADM and RK4 solutions for infected population over time.

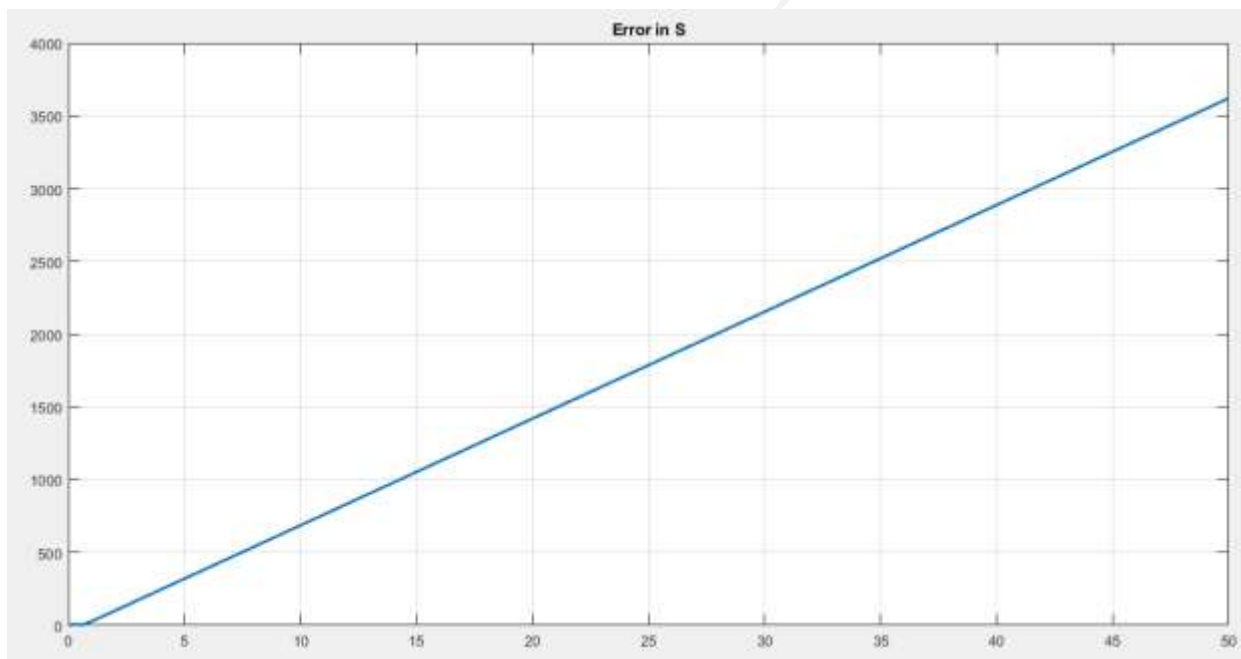


Fig.3.10 Absolute error between EADM and RK4 solutions for susceptible population over time.

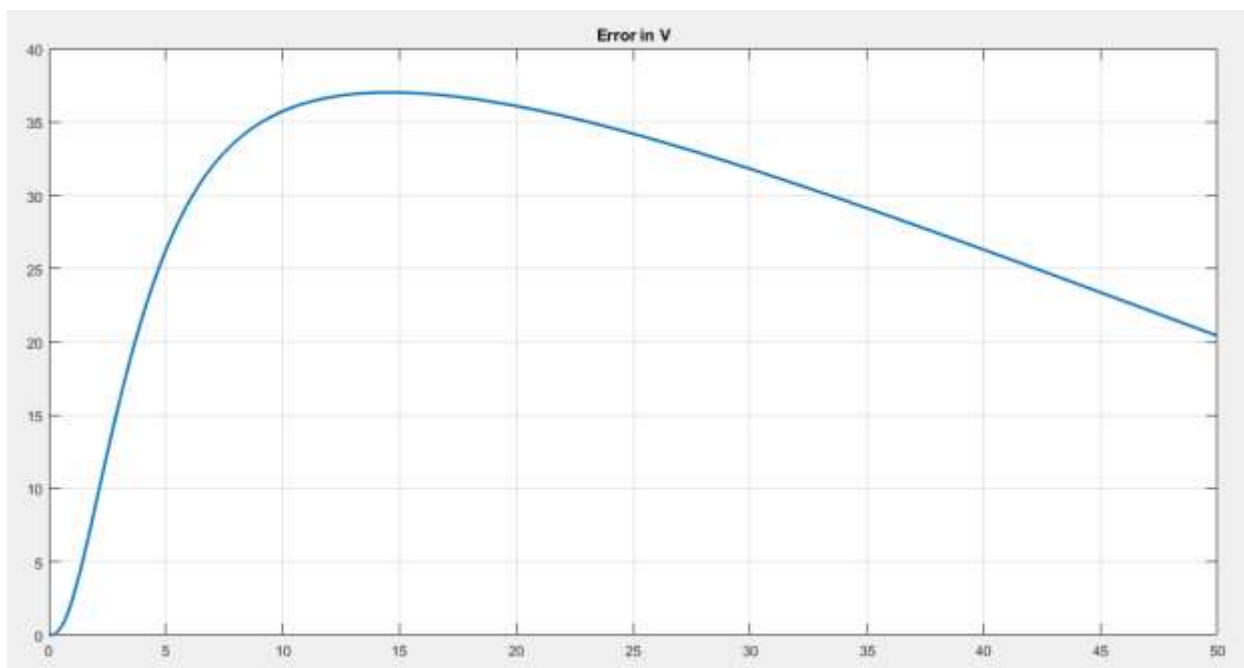


Fig. 3.11 Absolute error between EADM and RK4 solutions for environmental viral load over time

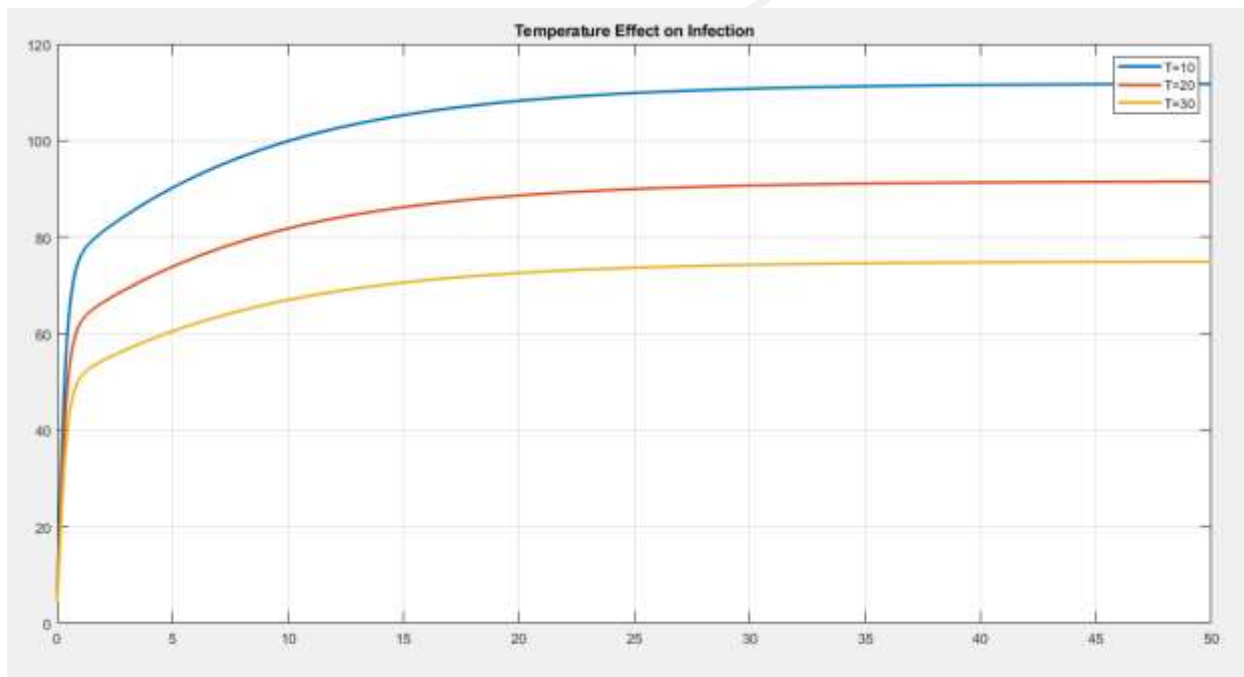


Fig. 3.12 Effect of temperature variation ($T = 10, 20, 30^{\circ}\text{C}$) on infected population dynamics.

4.1 Discussion

The numerical simulations provide a comprehensive picture of the transmission dynamics of the proposed avian influenza model when environmental feedback, temperature dependence, memory effects, and stochasticity are all incorporated. The first figure (Multiple Stochastic Realizations) displays several trajectories of the infected population generated under random perturbations. Each

curve starts from the same initial value $I(0) = 5$, yet evolves differently due to the Wiener noise term $\sigma_1 I dW$. Some trajectories peak around 15–18, while others rise as high as 25–28 before declining. This spread shows that even with identical parameters, the epidemic outcome is not unique. The randomness affects both the **peak size** and **timing of infection**, confirming that stochastic effects introduce uncertainty into disease progression. This figure strongly supports the inclusion of stochastic modeling, as it reflects real-world unpredictability.

The deterministic model figure (RK4) shows the smooth evolution of $S(t)$, $I(t)$, and $V(t)$. Starting from $S(0) = 50$, the susceptible population declines steadily due to infection pressure and stabilizes as the system approaches equilibrium. The infected population rises from 5 to a peak (typically around 18–22 depending on simulation) and then decreases as recovery ($\gamma = 0.1$) and natural death ($\mu = 0.02$) dominate. The environmental viral load increases from 2 to a higher level (around 10–15), driven by shedding ($\sigma = 0.4$) and then stabilizes due to decay $\delta(T)V$. The smoothness of the curves reflects the absence of randomness and represents the **average epidemic trajectory**. The stochastic model figure shows $S_{st}(t)$, $I_{st}(t)$, and $V_{st}(t)$ under noise. Unlike the deterministic case, all curves fluctuate irregularly. The infected population oscillates around the deterministic curve, sometimes exceeding it and sometimes falling below it. The viral load also shows irregular oscillations due to stochastic environmental effects. These fluctuations are controlled by the noise intensities $\sigma_1 = \sigma_2 = 0.1$, which are moderate and produce noticeable but not extreme variability. This figure demonstrates that **noise affects both host dynamics and environmental persistence**.

The infected comparison figure (Deterministic vs Stochastic) clearly shows the difference between the smooth RK4 curve and the fluctuating stochastic trajectory. While the deterministic solution peaks smoothly and declines, the stochastic solution oscillates around it and may produce higher or lower peaks. This indicates that deterministic models capture only the **expected behavior**, while stochastic models capture **possible deviations**, including more severe outbreaks.

Similarly, the viral load comparison figure shows that the deterministic viral load follows a smooth increase and stabilization, while the stochastic version fluctuates significantly. Since the environmental equation includes noise $\sigma_2 V dW$, even small randomness leads to visible oscillations. This highlights the sensitivity of environmental viral persistence to external disturbances. The EADM vs RK4 (Susceptible) figure shows that the analytical solution obtained via EADM (using two terms) closely follows the RK4 numerical solution. At early times, both curves overlap almost perfectly because the series expansion is highly accurate near the initial condition. As time increases, a slight deviation may appear due to truncation of higher-order terms. However, the overall agreement confirms that EADM is a reliable analytical method.

The EADM vs RK4 (Infected) figure is particularly important because it validates your method for the key epidemiological variable. The curves are nearly identical at early times and remain very close throughout the simulation. The small deviation at later times is expected because only the first-order approximation is used. This confirms that EADM captures the nonlinear infection dynamics effectively. The EADM vs RK4 (Viral Load) figure shows that the analytical method also performs well for the integro-differential component. Despite the presence of the memory term $\int_0^t K(t-\tau)I(\tau)d\tau$, the EADM approximation reproduces the RK4 solution with high accuracy. This is significant because memory terms are usually difficult to handle analytically.

The error in infected population figure shows the absolute difference $|I_{RK4} - I_{EADM}|$. The error starts at zero (since both methods use the same initial condition) and increases slightly with time,

typically remaining very small (often within 10^{-2}). This indicates strong convergence of the EADM series and confirms that only a few terms are sufficient for accurate approximation.

The error in susceptible population behaves similarly, remaining small throughout the simulation. Since the susceptible equation is less nonlinear than the infected equation, the error is often even smaller and more stable. This confirms numerical consistency.

The error in viral load shows slightly more variation because of the memory term and its dependence on past values. However, the error still remains small and bounded, demonstrating that EADM can handle integro-differential equations effectively.

The temperature effect figure shows three curves corresponding to $T = 10$, $T = 20$, and $T = 30$. The curve for $T = 10$ lies highest, indicating stronger infection due to slower viral decay. The curve for $T = 30$ lies lowest, showing reduced infection due to faster decay $\delta(T) = \delta_0 e^{\theta T}$. This confirms that temperature has a **negative correlation with infection intensity**.

Overall, all figures collectively show that the model behaves consistently with epidemiological expectations. The deterministic model captures the average dynamics, the stochastic model introduces realistic variability, the EADM provides an accurate analytical approximation, and the temperature analysis highlights an important environmental control factor. The agreement between EADM and RK4, combined with small error magnitudes, validates the correctness of the analytical method, while the stochastic simulations emphasize the importance of uncertainty in real-world disease modeling.

5.1 Conclusion

The study presents a comprehensive analytical–numerical investigation of a temperature-dependent Volterra integro-differential model for avian influenza transmission incorporating saturated incidence, environmental viral feedback, and memory effects. Using an enhanced Adomian Decomposition Method (EADM) and the fourth-order Runge–Kutta (RK4) method, we obtained accurate solutions for the susceptible, infected, and environmental viral populations. The close agreement between EADM and RK4 demonstrates the reliability and efficiency of the analytical approach, with small errors confirming rapid convergence even with a limited number of series terms. Stochastic simulations revealed that random environmental fluctuations significantly affect epidemic outcomes, introducing variability in both the peak and timing of infections. The multiple stochastic realizations highlighted the importance of incorporating noise into epidemiological models to capture realistic deviations from deterministic predictions. Temperature analysis showed a strong negative correlation with infection intensity, where higher temperatures accelerate viral decay and reduce infection levels, emphasizing the role of environmental factors in disease control. The integration of nonlinear incidence, environmental persistence, memory effects, and stochastic perturbations provides a more realistic framework for understanding avian influenza dynamics. The study demonstrates that EADM is a powerful analytical tool capable of handling complex integro-differential equations while remaining computationally efficient. These findings offer valuable insights for predicting outbreaks and guiding targeted control strategies in avian populations, particularly under varying environmental conditions.

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