

Modeling Maize Streak Disease with Time Delay and Random Effects: Implications for Insecticide-Based Control

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ABSTRACT

Maize streak disease (MSD) remains a major constraint to maize production, particularly in sub-Saharan Africa, where its spread is strongly influenced by environmental, agronomic, and vector-related factors. This paper develops a stochastic delayed model for MSD transmission dynamics. The model incorporates a discrete time delay to represent incubation and vector-mediated transmission, while stochastic perturbations are included to describe environmental variability and random fluctuations in disease spread. The qualitative analysis shows that the model is biologically well posed, with positive and bounded solutions. The disease-free and endemic equilibria are derived, and the effective reproduction number is obtained using the next-generation matrix method. Also, the sensitivity of parameters is studied rigorously using the given data. The stochastic formulation leads to a modified threshold, indicating that random perturbations can reduce disease persistence and may contribute to disease extinction under suitable conditions. The standard numerical method, Euler–Maruyama, and the nonstandard numerical method, namely the nonstandard finite difference (NSFD) method, are used to solve the stochastic MSD model due to the unavailability of a closed-form solution for the model. Unfortunately, the Euler–Maruyama method has some limitations and is dependent on the discretization step size, whereas the NSFD method is independent and provides more reliable results. Moreover, numerical simulations based on a stochastic nonstandard finite difference scheme are used to illustrate the theoretical results and the effects of key parameters, including transmission rate, delay, and insecticide efficacy. The limited MSD data from 2020–2025 are used only for illustrative trend analysis. Overall, the proposed stochastic delayed model provides a useful framework for understanding MSD dynamics and assessing insecticide-based control strategies under uncertainty.

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1 Introduction

Maize streak disease (MSD) is still a major challenge in maize production especially in sub-Saharan Africa, where the disease incidence is highly dependent on environment, agronomic, spatial, and seasonal factors [1,2]. From field and molecular studies, it has been found that the incidence of MSD is not uniform in different parts of the country and in different seasons, and the distribution of maize streak virus (MSV) strains is not uniform in different agro-ecological zones [3]. Additionally, local surveys have shown that the prevalence of the disease is strongly correlated with the environmental factors and vector densities [4]. The observations presented here demonstrate that MSD transmission is a dynamic process that is influenced by climate, vector activity, and vector control. Several useful mathematical models with determinism have been developed and used to gain insight in the mechanisms of MSD transmission, equilibrium behavior and possible intervention [5]. Additionally, optimal control analysis has demonstrated that the disease burden may be reduced more effectively by using multiple intervention strategies than by using a single intervention strategy [6]. Optimal control models that are based on cost-effectiveness have also aided the linkage of theoretical disease modeling to practical agricultural decision making [7]. Deterministic models, however, do not usually account for randomness in the fields and the time until the disease occurs, which can be crucial in plant-vector disease systems. Thus, delay and stochastic effects have become important extensions in the modeling of plant diseases. Delay in MSD dynamics could be the time needed to acquire the virus, incubation, latent infection development, effective vector mediated transmission. Stochastic effects also are important because of the random variation of weather, vector densities, crop management and field insecticide performance. To account for this randomness and time-lag, stochastic delay modelling studies have been undertaken for infection processes more recently [8,9]. These studies suggest that delay and randomness can have significant impacts on persistence, extinction, and the timing of outbreaks. A number of other non-classical modeling approaches have been also developed for MSD and related epidemiological systems. In order to describe the memory and nonlocal effects in MSD transmission, fractional-order models have been introduced in [10] and advanced numerical techniques have been proposed to enhance the stability and accuracy of fractional MSD models in [11]. When the dynamics of the complex diseases cannot be captured with the integer-order models commonly used, fractal-fractional models have also been introduced in the literature [12,13]. The trend of generalized modeling tools in plant disease epidemiology is reflected in these advances. Biologically and agriculturally, several studies have been conducted on tested resilient maize varieties, molecular breeding, and induced mutation techniques in resistance to maize streak virus (MSV) [14,15]. Recent reviews have highlighted the need to include genetic, agronomic, surveillance, and mathematical modeling practices for a sustainable MSD control strategy [16]. Furthermore, remote sensing, imaging, and sensor-based techniques have facilitated better monitoring and early detection of MSD and other maize diseases at a large scale [17,18]. This, along with a number of new reports of geographic distribution and heavy infection levels in various African areas, solidifies the idea that MSD is still a persistent threat on the agricultural sector [19,20]. The authors investigated a mathematical model and examined the qualitative behavior of the model, such as stability and also numerical analysis using real data [21]. In recent years, variable-order fractional models and fractal-fractional numerical schemes have also been found to be helpful for the modeling of complex nonlinear dynamical systems with memory and nonlocal effects [22,23]. The works are relevant to the increasing use of generalized operators and sophisticated numerical techniques for modelling complex biological and physical processes. In epidemiological systems with memory effects like the smoking dynamics with relapse and TB–HIV co-infection, generalized operators are applied to complex disease

modeling, which is motivated by fractional and fractal-fractional models, as mentioned in [24,25]. These developments notwithstanding, there remains a need for an MSD model which incorporates insecticide modified transmission, a biologically meaningful delay, stochastic environmental effects and a numerical method that maintains the nonnegative nature of plant population variables. This work fills this gap, by proposing a stochastic delayed MSD model with an insecticide treated plant class. The rate $1 - \gamma$ is the factor by which the treatment of insects does not provide complete immunity in the model, but rather reduces the likelihood of them spreading an infection. This assumption is biologically sound as the primary effect of insecticide is the reduction of vector activity and transmission pressure, and not directly of the maize plants.

The main contributions of this work are as follows. It first introduces a delayed MSD model using susceptible, insecticide treated, exposed, infected, and recovered classes of plants. Secondly, the effective reproduction number is calculated and the disease-free and endemic equilibria are studied. Third, the deterministic model is extended to a stochastic delay system and a stochastic threshold is used to discuss extinction and persistence under random perturbations. Fourth, a stochastic nonstandard finite difference scheme is designed which maintains the nonnegativity and boundedness of the numerical solution. Finally, the qualitative behavior of the proposed scheme is compared with the Euler–Maruyama scheme and it is concluded that the NSFD approach is more appropriate for this application due to the better respect of the biological constraints of the MSD model. Compared with existing stochastic delay epidemic models, the novelty of the present work lies in its MSD-specific formulation. The model combines insecticide-modified vector transmission, a biologically interpretable incubation delay, stochastic environmental perturbations, and a positivity-preserving stochastic NSFD discretization in a single framework. In particular, the insecticide-treated class is not assumed to be fully immune; instead, insecticide reduces the effective vector-mediated infection rate through the factor $1 - \gamma$. This makes the proposed framework biologically more suitable for maize streak disease than a direct application of a standard epidemic delay model. The paper is organized as follows. Section 2 presents the delayed MSD model. Section 3 studies the qualitative analysis of the model, including the positivity and boundedness of solutions. Section 4 derives the equilibrium states and the effective reproduction number.

Section 5 presents the statistical analysis of MSD data. Section 6 presents a stochastic delayed version of the MSD model. Section 7 presents the stochastic NSFD method and simulations. Finally, Section 8 gives the conclusion and future research directions.

2 Delay Model Formulation

The deterministic system is extended by introducing a discrete time delay in order to improve the biological realism of the maize streak disease (MSD) model. In plant–vector disease transmission, infection does not become epidemiologically effective immediately after contact with infectious plants or vectors. Instead, a finite period is required for virus acquisition, incubation, and effective transmission. Therefore, a delay $\tau > 0$ is introduced, so that the present force of infection depends on the infected population at the previous time $t - \tau$. Biologically, the delay τ represents the time required for virus acquisition, incubation, latent infection development, and effective vector-mediated transmission. Thus, contact with infected plants or infectious vectors does not produce immediate new infections. Instead, the infection process becomes epidemiologically effective after a finite time interval. This justifies the use of the delayed term $I(t - \tau)$ in the force of infection.

The maize plant population is divided into five compartments: $S(t)$, $F(t)$, $E(t)$, $I(t)$, and $R(t)$, representing susceptible, insecticide-treated, exposed, infected, and recovered plants, respectively.

The class $F(t)$ denotes plants under insecticide-based protection. This class should not be interpreted as a fully immune class. Since MSD is transmitted by insect vectors, insecticide is assumed to reduce vector pressure and vector-mediated transmission rather than directly immunizing the plant. Hence, treated plants can still become infected, but at a reduced rate $(1 - \gamma)\beta F(t)I(t - \tau)e^{-\mu\tau}$, where $\gamma \in (0, 1]$ measures insecticide efficacy. Thus, $\gamma = 1$ corresponds to complete protection against vector-mediated infection, while smaller values of γ represent partial protection. The parameter ω describes the waning or loss of insecticide protection, so $F(t)$ represents temporary vector suppression rather than permanent immunity.

The total population is given by

$$N(t) = S(t) + F(t) + E(t) + I(t) + R(t). \quad (1)$$

The interaction and flow of model parameters are presented in Fig. 1.

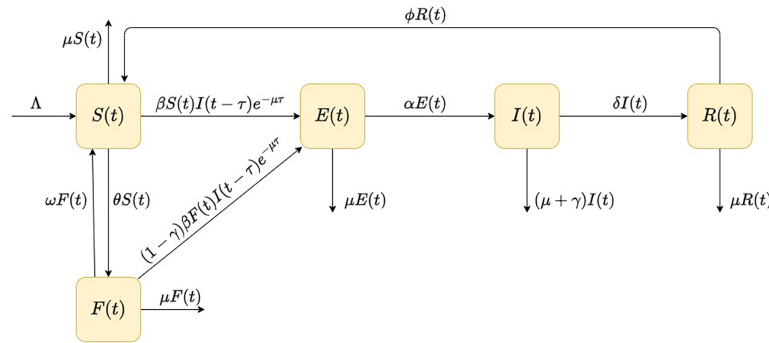


Figure 1: Flowchart of maize streak disease (MSD)

In the presence of the delay τ , the infection incidence is described by the delayed terms $\beta S(t)I(t - \tau)e^{-\mu\tau}$ and $(1 - \gamma)\beta F(t)I(t - \tau)e^{-\mu\tau}$. The factor $e^{-\mu\tau}$ represents survival during the delay period. The delayed MSD model is therefore formulated as follows:

$$\frac{dS(t)}{dt} = \Lambda + \omega F(t) + \phi R(t) - \beta S(t)I(t - \tau)e^{-\mu\tau} - \theta S(t) - \mu S(t), \quad (2)$$

$$\frac{dF(t)}{dt} = \theta S(t) - (1 - \gamma)\beta F(t)I(t - \tau)e^{-\mu\tau} - \omega F(t) - \mu F(t), \quad (3)$$

$$\frac{dE(t)}{dt} = \beta S(t)I(t - \tau)e^{-\mu\tau} + (1 - \gamma)\beta F(t)I(t - \tau)e^{-\mu\tau} - (\alpha + \mu)E(t), \quad (4)$$

$$\frac{dI(t)}{dt} = \alpha E(t) - (\delta + \sigma + \mu)I(t), \quad (5)$$

$$\frac{dR(t)}{dt} = \delta I(t) - (\phi + \mu)R(t). \quad (6)$$

Since the system involves a delay, the model requires initial history functions instead of simple initial values. Thus, the system is supplemented with the following conditions:

$S(\vartheta) = \psi_1(\vartheta)$, $F(\vartheta) = \psi_2(\vartheta)$, $E(\vartheta) = \psi_3(\vartheta)$, $I(\vartheta) = \psi_4(\vartheta)$, $R(\vartheta) = \psi_5(\vartheta)$, for $\vartheta \in [-\tau, 0]$, where each function $\psi_i(\vartheta) \geq 0$ is continuous in the interval $[-\tau, 0]$.

3 Qualitative Analysis of the Delay Model

In this section, we investigate the fundamental analytical properties of the delayed maize streak disease model.

3.1 Feasible Region

We define the biologically feasible region for the system as

$$\Omega = \left\{ (S, F, E, I, R) \in \mathbb{R}_+^5 : 0 \leq N(t) \leq \frac{\Lambda}{\mu} \right\}. \quad (7)$$

The set Ω represents all non-negative states for which the total population of maize remains bounded by the carrying threshold $\frac{\Lambda}{\mu}$.

3.2 Positivity and Boundedness

Theorem 1: For non-negative continuous initial functions on $[-\tau, 0]$, the solution of system (2)–(6) remains non-negative and bounded for all $t \geq 0$.

Proof: From system (2)–(6), each equation satisfies a lower bound of the form

$$\dot{X}(t) \geq -c_X(t)X(t), \quad X \in \{S, F, E, I, R\},$$

where $c_X(t) \geq 0$. Hence, by Gronwall's inequality,

$$S(t), F(t), E(t), I(t), R(t) \geq 0, \quad t \geq 0.$$

Moreover, for

$$N(t) = S(t) + F(t) + E(t) + I(t) + R(t),$$

we have

$$\dot{N}(t) = \Lambda - \mu N(t) - \sigma I(t) \leq \Lambda - \mu N(t).$$

Therefore,

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}), \quad \limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}.$$

Thus, the feasible region

$$\Omega = \left\{ (S, F, E, I, R) \in \mathbb{R}_+^5 : 0 \leq N(t) \leq \frac{\Lambda}{\mu} \right\}$$

is positively invariant. $\square$

4 Equilibrium States and Reproduction Number

At the disease-free equilibrium, $E = I = R = 0$, and hence

$$\mathcal{E}_0 = \left(\frac{\Lambda(\omega + \mu)}{\mu(\theta + \omega + \mu)}, \frac{\theta \Lambda}{\mu(\theta + \omega + \mu)}, 0, 0, 0 \right).$$

For the endemic equilibrium $\mathcal{E}^* = (S^*, F^*, E^*, I^*, R^*)$, $I^* > 0$, we obtain

$$E^* = \frac{\delta + \sigma + \mu}{\alpha} I^*, \quad R^* = \frac{\delta}{\phi + \mu} I^*, \quad F^* = \frac{\theta S^*}{(1 - \gamma)\beta I^* e^{-\mu\tau} + \omega + \mu}.$$

Using the next-generation matrix method [26], the effective reproduction number is

$$\mathcal{R}_e = \frac{\alpha\beta e^{-\mu\tau} (S^0 + (1 - \gamma)F^0)}{(\alpha + \mu)(\delta + \sigma + \mu)},$$

where $S^0 = \frac{\Lambda(\omega + \mu)}{\mu(\theta + \omega + \mu)}$, $F^0 = \frac{\theta\Lambda}{\mu(\theta + \omega + \mu)}$. Therefore,

$$\mathcal{R}_e = \frac{\alpha\beta\Lambda e^{-\mu\tau} (\omega + \mu + (1 - \gamma)\theta)}{\mu(\alpha + \mu)(\delta + \sigma + \mu)(\theta + \omega + \mu)}. \quad (8)$$

4.1 Sensitivity Analysis

The normalized forward sensitivity index of $\mathcal{R}_e$ with respect to a parameter p is defined by

$$\Upsilon_p^{\mathcal{R}_e} = \frac{\partial \mathcal{R}_e}{\partial p} \times \frac{p}{\mathcal{R}_e}.$$

Using Eq. (8), the sensitivity indices are obtained as

$$\begin{aligned} \Upsilon_\beta^{\mathcal{R}_e} &= 1, & \Upsilon_\Lambda^{\mathcal{R}_e} &= 1, & \Upsilon_\alpha^{\mathcal{R}_e} &= \frac{\mu}{\alpha + \mu}, & \Upsilon_\delta^{\mathcal{R}_e} &= -\frac{\delta}{\delta + \sigma + \mu}, & \Upsilon_\sigma^{\mathcal{R}_e} &= -\frac{\sigma}{\delta + \sigma + \mu}, \\ \Upsilon_\gamma^{\mathcal{R}_e} &= -\frac{\gamma\theta}{\omega + \mu + (1 - \gamma)\theta}, & \Upsilon_\omega^{\mathcal{R}_e} &= \frac{\omega\gamma\theta}{(\omega + \mu + (1 - \gamma)\theta)(\theta + \omega + \mu)}, \\ \Upsilon_\theta^{\mathcal{R}_e} &= -\frac{\gamma\theta(\omega + \mu)}{(\omega + \mu + (1 - \gamma)\theta)(\theta + \omega + \mu)}, \end{aligned}$$

and

$$\Upsilon_\mu^{\mathcal{R}_e} = \mu \left[\frac{1}{\omega + \mu + (1 - \gamma)\theta} - \frac{1}{\mu} - \frac{1}{\alpha + \mu} - \frac{1}{\delta + \sigma + \mu} - \frac{1}{\theta + \omega + \mu} \right].$$

The sensitivity analysis of the model parameters is presented in Table 1 and shown graphically in Fig. 2. The sensitivity results show that β and Λ are the main parameters that increase $\mathcal{R}_e$, indicating that stronger transmission and larger susceptible recruitment promote MSD spread. In contrast, γ and θ have strong negative effects, showing that higher insecticide efficacy and wider treatment coverage reduce vector-mediated infection. The negative indices of δ , σ , and μ indicate that faster removal, recovery, or natural loss of infected plants lowers disease persistence. Thus, reducing vector contact and improving insecticide performance are the most effective control directions suggested by the sensitivity analysis.

Table 1: Sensitivity indices of $\mathcal{R}_e$.

Parameter	Index	Effect
β	1.0000	Increase
Λ	1.0000	Increase
α	0.1057	Increase
δ	-0.5682	Decrease
σ	-0.2841	Decrease
γ	-2.0019	Decrease
ω	0.1183	Increase
θ	-0.8873	Decrease
μ	-0.4844	Decrease

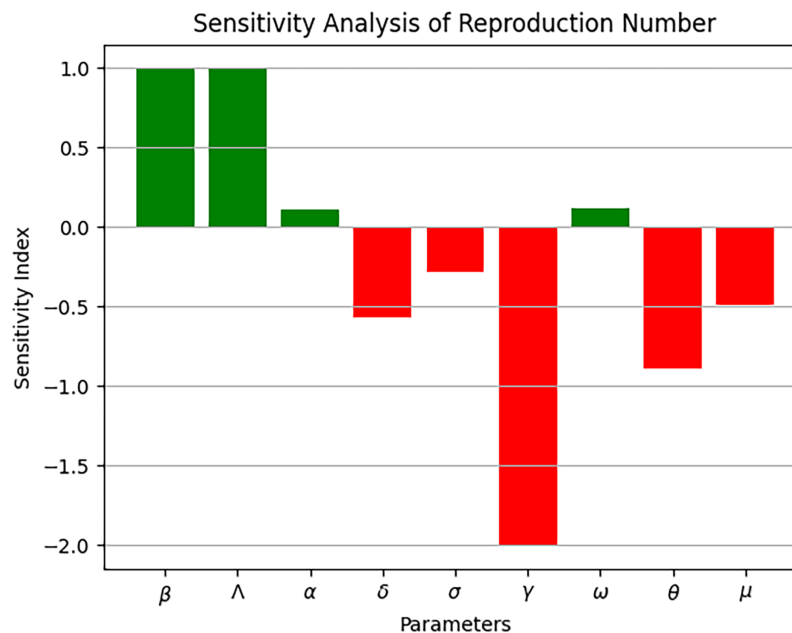


Figure 2: Sensitivity indices of $\mathcal{R}_e$.

5 Statistical Analysis of Maize Streak Disease

To corroborate the proposed stochastic delay model, statistical and graphical analyzes are performed using data on maize streak disease (MSD) incidence between 2020 and 2025. MSD continues to play an significant role in limiting maize production in sub-Saharan Africa, and environmental variability, vector population, and control policies have a significant impact on its dynamics [27]. The observed infection rates (%) during the study period are summarized in Table 2. Data represent yearly averages obtained from agricultural reports and epidemiological observations.

Table 2: Statistics of MSD.

2020	2021	2022	2023	2024	2025
38%	42%	47%	55%	61%	58%

The data show that the prevalence of the disease is on a slight rising trend between 2020 and 2024 but then slightly declines in 2025. It could be related to a better disease management strategy like using insecticides and adopting resistant varieties of maize. The sample mean and variance of the infection data are computed as

$$\bar{I} = 50.17, \quad \text{Var}(I) \approx 77.37,$$

which exhibit observable differences in the prevalence of diseases. This uncertainty warrants the use of stochastic effects in the mathematical model. Fig. 3 shows, the pattern of temporal change in MSD incidence. The figure depicts a steady increasing trend in the level of infection until 2024 and a decrease in 2025. The dotted line shows the linear trend that is fitted, which shows that the general trend is an increase in the disease transmission with time. A three-dimensional surface plot is created to further investigate the impact of the key epidemiological parameters on disease dynamics, and the plot of this form is presented in Fig. 4. This curve represents how the number of the infected persons changes over time and rate of transmission.

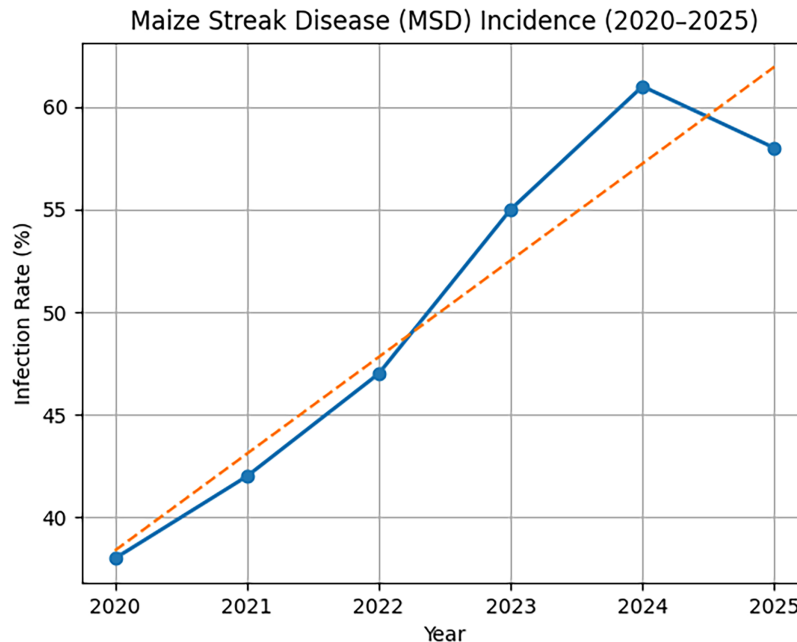

Figure 3: Temporal variation of maize streak disease (MSD) incidence from 2020 to 2025

Fig. 4 surface shows that when the transmission rate values are higher, infection levels increase very rapidly with time. The nonlinear development trend on the surface evidences the simultaneous influence of time evolution and sensitivity of parameters on disease propagation. This proves that minor modifications of the parameters of transmissions can have a great impact on the severity of the outbreak. The assumptions of the stochastic delay model are well supported by the combined

statistical and graphical analyses. The environmental randomness is reflected in the variability of the level of infection and is represented in terms of stochastic perturbations. The slow growth and the lag in responding to the dynamics of the infections justifies the presence of delay terms, whereas the later decrease in the age justifies the correctiveness of the control measures. On the whole, the findings reveal that MSD transmission has stochastic and time-dependent characteristics. Thus, the ability to introduce stochasticity and delay to the modeling framework makes the disease dynamics more realistic and accurate and realistic.

3D Surface Plot of Infection Dynamics

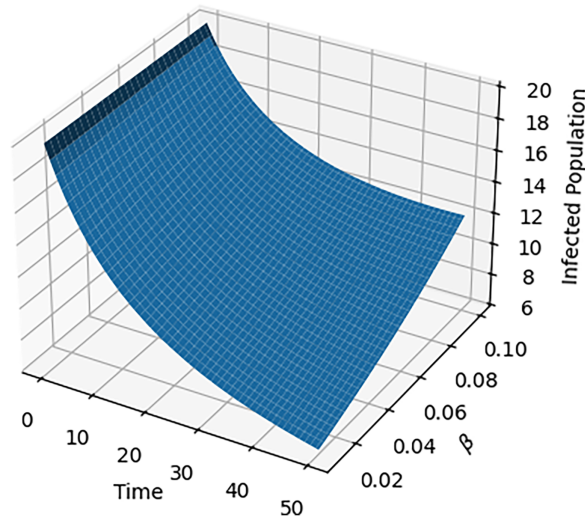


Figure 4: Three-dimensional surface plot showing the variation of the infected population with respect to time and the transmission rate β

6 Stochastic Delayed Model

Let $U(t) = (S(t), F(t), E(t), I(t), R(t))^T$ be the state vector of the delayed model. During a sufficiently short time interval Δt , the possible changes in the state and their corresponding transition probabilities are described in Table 3.

The mean increment of the state vector is given by

$$\mathbb{E}[\Delta U] = \sum_{i=1}^{13} P_i X_i.$$

Hence,

$$\mathbb{E}[\Delta U] = \begin{pmatrix} \Lambda + \omega F(t) + \phi R(t) - \beta S(t)I(t - \tau)e^{-\mu\tau} - \theta S(t) - \mu S(t) \\ \theta S(t) - (1 - \gamma)\beta F(t)I(t - \tau)e^{-\mu\tau} - \omega F(t) - \mu F(t) \\ \beta S(t)I(t - \tau)e^{-\mu\tau} + (1 - \gamma)\beta F(t)I(t - \tau)e^{-\mu\tau} - (\alpha + \mu)E(t) \\ \alpha E(t) - (\delta + \sigma + \mu)I(t) \\ \delta I(t) - (\phi + \mu)R(t) \end{pmatrix} \Delta t.$$

Table 3: Transition probabilities of the delayed MSD model

Transition	State change X_i	Probability P_i
T_1	[1, 0, 0, 0, 0]	$\Lambda \Delta t$
T_2	[-1, 1, 0, 0, 0]	$\theta S(t) \Delta t$
T_3	[-1, 0, 1, 0, 0]	$\beta S(t) I(t - \tau) e^{-\mu \tau} \Delta t$
T_4	[0, -1, 1, 0, 0]	$(1 - \gamma) \beta F(t) I(t - \tau) e^{-\mu \tau} \Delta t$
T_5	[0, -1, 0, 0, 0]	$\omega F(t) \Delta t$
T_6	[-1, 0, 0, 0, 0]	$\mu S(t) \Delta t$
T_7	[0, -1, 0, 0, 0]	$\mu F(t) \Delta t$
T_8	[0, 0, -1, 0, 0]	$\mu E(t) \Delta t$
T_9	[0, 0, -1, 1, 0]	$\alpha E(t) \Delta t$
T_{10}	[0, 0, 0, -1, 0]	$(\delta + \sigma + \mu) I(t) \Delta t$
T_{11}	[0, 0, 0, 1, -1]	$\phi R(t) \Delta t$
T_{12}	[0, 0, 0, 0, 1]	$\delta I(t) \Delta t$
T_{13}	[0, 0, 0, 0, -1]	$(\phi + \mu) R(t) \Delta t$

Thus, the drift vector is

$$H(U(t), t) = \frac{\mathbb{E}[\Delta U]}{\Delta t},$$

that is,

$$H(U(t), t) = \begin{pmatrix} \Lambda + \omega F(t) + \phi R(t) - \beta S(t) I(t - \tau) e^{-\mu \tau} - \theta S(t) - \mu S(t) \\ \theta S(t) - (1 - \gamma) \beta F(t) I(t - \tau) e^{-\mu \tau} - \omega F(t) - \mu F(t) \\ \beta S(t) I(t - \tau) e^{-\mu \tau} + (1 - \gamma) \beta F(t) I(t - \tau) e^{-\mu \tau} - (\alpha + \mu) E(t) \\ \alpha E(t) - (\delta + \sigma + \mu) I(t) \\ \delta I(t) - (\phi + \mu) R(t) \end{pmatrix}.$$

The covariance matrix of ΔU is

$$\mathbb{E}[\Delta U \Delta U^T] = \sum_{i=1}^{13} P_i X_i X_i^T.$$

Therefore, the diffusion matrix is defined by

$$K(U(t), t) = \sqrt{\frac{\mathbb{E}[\Delta U \Delta U^T]}{\Delta t}}.$$

Hence, the stochastic differential form of the delayed model can be written as follows.

$$dU(t) = H(U(t), t) dt + K(U(t), t) dW(t), \quad (9)$$

where $W(t)$ is a standard Brownian motion vector.

6.1 Covariance Matrix

By employing the transitions summarized in Table 3, we obtain

$$\mathbb{E}[\Delta U \Delta U^T] = \sum_{i=1}^{13} P_i X_i X_i^T = \begin{pmatrix} Q_{11} & Q_{12} & Q_{13} & 0 & Q_{15} \\ Q_{21} & Q_{22} & Q_{23} & 0 & 0 \\ Q_{31} & Q_{32} & Q_{33} & Q_{34} & 0 \\ 0 & 0 & Q_{43} & Q_{44} & Q_{45} \\ Q_{51} & 0 & 0 & Q_{54} & Q_{55} \end{pmatrix} \Delta t,$$

where

$$Q_{11} = P_1 + P_2 + P_3 + P_6, \quad Q_{12} = Q_{21} = -P_2, \quad Q_{13} = Q_{31} = -P_3,$$

$$Q_{15} = Q_{51} = 0, \quad Q_{22} = P_2 + P_4 + P_5 + P_7, \quad Q_{23} = Q_{32} = -P_4,$$

$$Q_{33} = P_3 + P_4 + P_8 + P_9, \quad Q_{34} = Q_{43} = -P_9,$$

$$Q_{44} = P_9 + P_{10} + P_{12}, \quad Q_{45} = Q_{54} = -P_{12}, \quad Q_{55} = P_{11} + P_{12} + P_{13}.$$

Thus, the drift and diffusion terms are completely determined.

6.2 Euler–Maruyama Method

To numerically approximate the stochastic delayed model (9), we employ the Euler–Maruyama scheme [28]. Let the time interval $[0, T]$ be partitioned into N equal subintervals of length Δt , namely

$$0 = t_0 < t_1 < t_2 < \cdots < t_N = T, \quad (10)$$

where

$$t_n = n\Delta t, \quad n = 0, 1, 2, \dots, N.$$

Let

$$U^n = U(t_n) = (S^n, F^n, E^n, I^n, R^n)^T,$$

and define the Brownian increments by

$$\Delta B_n = B(t_{n+1}) - B(t_n), \quad n = 0, 1, \dots, N-1. \quad (11)$$

Each ΔB_n is normally distributed with mean zero and variance Δt .

The Euler–Maruyama approximation of the stochastic delayed system is then given by

$$U^{n+1} = U^n + H(U^n, t_n)\Delta t + K(U^n, t_n)\Delta B_n, \quad (12)$$

where the delay term $I(t_n - \tau)$ is approximated by I^{n-m} , with $m = \tau/\Delta t$, assuming that τ is an integer multiple of Δt .

7 Numerical Methodology

To approximate the stochastic delayed MSD model, we use a stochastic nonstandard finite difference (NSFD) scheme. Let $[0, T]$ be divided into n subintervals with step size $h = T/n$, $t_k = kh$, and $m = \tau/h$. The delay term is approximated by I^{k-m} , and the Brownian increments satisfy $\Delta B_k \sim N(0, h)$.

The stochastic NSFD scheme is given by

$$S^{k+1} = \frac{S^k + h(\Lambda + \omega F^k + \phi R^k + \sigma_1 S^k \Delta B_k)}{1 + h(\beta I^{k-m} e^{-\mu\tau} + \theta + \mu)}, \quad (13)$$

$$F^{k+1} = \frac{F^k + h(\theta S^k + \sigma_2 F^k \Delta B_k)}{1 + h((1 - \gamma)\beta I^{k-m} e^{-\mu\tau} + \omega + \mu)}, \quad (14)$$

$$E^{k+1} = \frac{E^k + h(\beta S^k I^{k-m} e^{-\mu\tau} + (1 - \gamma)\beta F^k I^{k-m} e^{-\mu\tau} + \sigma_3 E^k \Delta B_k)}{1 + h(\alpha + \mu)}, \quad (15)$$

$$I^{k+1} = \frac{I^k + h(\alpha E^k + \sigma_4 I^k \Delta B_k)}{1 + h(\delta + \sigma + \mu)}, \quad (16)$$

$$R^{k+1} = \frac{R^k + h(\delta I^k + \sigma_5 R^k \Delta B_k)}{1 + h(\phi + \mu)}. \quad (17)$$

This denominator form treats the main loss terms nonlocally and helps preserve the non-negativity and boundedness of the numerical solution, which are essential for biological consistency.

Theorem 2: Assume that

$$S^0, F^0, E^0, I^0, R^0 \geq 0,$$

and that the stochastic increments keep the numerators non-negative. Then the NSFD approximations remain non-negative and bounded for all $k \geq 0$.

Proof: All denominators in the NSFD scheme are positive. Hence, if the numerators are non-negative, then

$$S^{k+1}, F^{k+1}, E^{k+1}, I^{k+1}, R^{k+1} \geq 0.$$

Thus, positivity follows by induction.

Moreover, by adding the discrete equations and using cancellation of transfer terms, we obtain

$$N^{k+1} \leq \frac{N^k + h\Lambda}{1 + h\mu}, \quad N^k = S^k + F^k + E^k + I^k + R^k.$$

Therefore,

$$N^k \leq \max \left\{ N^0, \frac{\Lambda}{\mu} \right\}.$$

Hence, the NSFD solution remains bounded in the feasible region. $\square$

Computational Results

Numerical simulations were performed using parameter values from the literature [29]:

$$\Lambda = 0.97, \quad \beta = 0.022, \quad \mu = 0.013, \quad \theta = 0.12, \quad \omega = 0.002, \quad \delta = 0.05,$$

$$\gamma = 0.9998, \quad \alpha = 0.11, \quad \phi = 0.02, \quad \sigma = 0.025.$$

The stochastic intensities were taken as (0.5, 0.4, 0.3, 0.6, 0.1). The simulations illustrate the effects of stochastic fluctuations, delay, and insecticide-based control on MSD dynamics.

Figs. 5 and 6 compare the stochastic Euler-Maruyama and stochastic NSFD methods for $I(t)$. For $h = 0.01$, both methods converge to the endemic equilibrium. However, for $h = 0.5$, Euler-Maruyama becomes unstable and produces nonphysical behavior, while the stochastic NSFD scheme remains positive, bounded, and convergent. Hence, the NSFD method is more reliable for large step sizes. Fig. 7 confirms that $N(t)$ remains bounded by Λ/μ , supporting the biological feasibility of the model. Figs. 8 and 9 show that delay and stochastic noise strongly influence infection dynamics; larger delay ($\tau = 5$) increases $I(t)$ above 4.5, while strong noise ($\sigma_4 = 0.6$) drives infection toward extinction. Fig. 10 presents a bounded phase portrait of $S(t)$ vs. $I(t)$, indicating stable long-term dynamics. Figs. 11 and 12 show that stochastic RK4 produces large oscillations, whereas Euler-Maruyama and NSFD remain closer to equilibrium, with NSFD giving the most stable approximation. Fig. 13 shows that NSFD and Euler-Maruyama have smaller equilibrium errors than stochastic RK4. Figs. 14 and 15 further confirm that NSFD remains bounded for larger step sizes, while stochastic RK4 may become unstable.

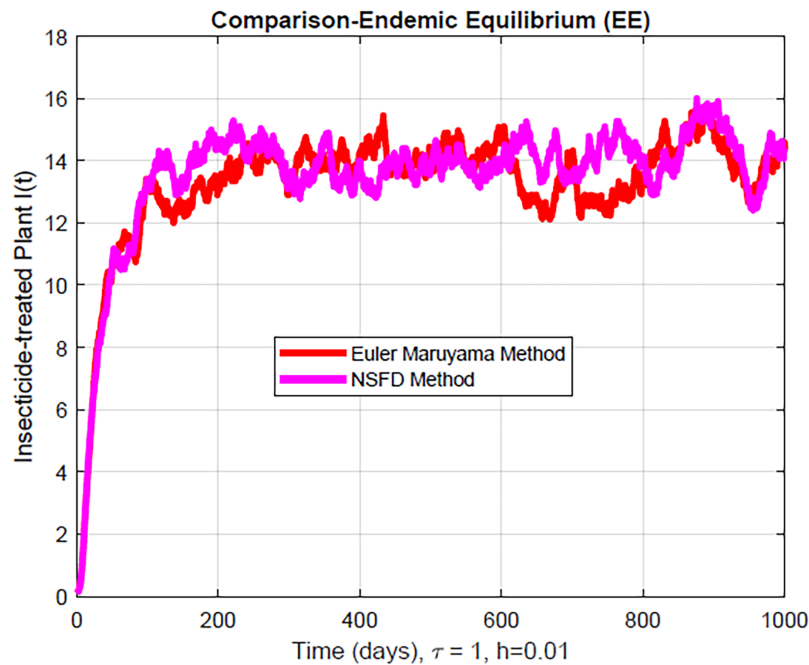


Figure 5: Convergence of Euler-Maruyama and stochastic NSFD methods for small step size $h = 0.01$

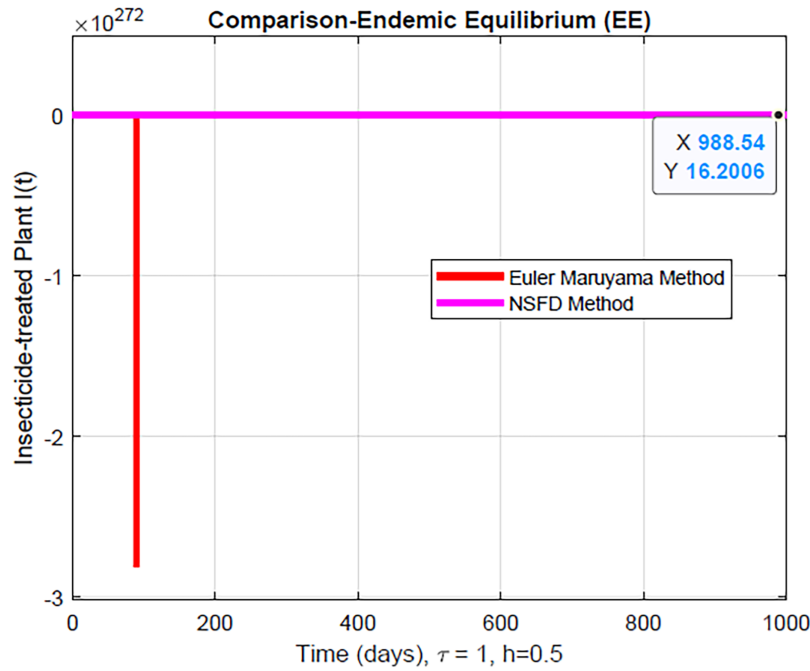


Figure 6: Divergence of Euler–Maruyama method and convergence of stochastic NSFD method for large step size $h = 0.5$

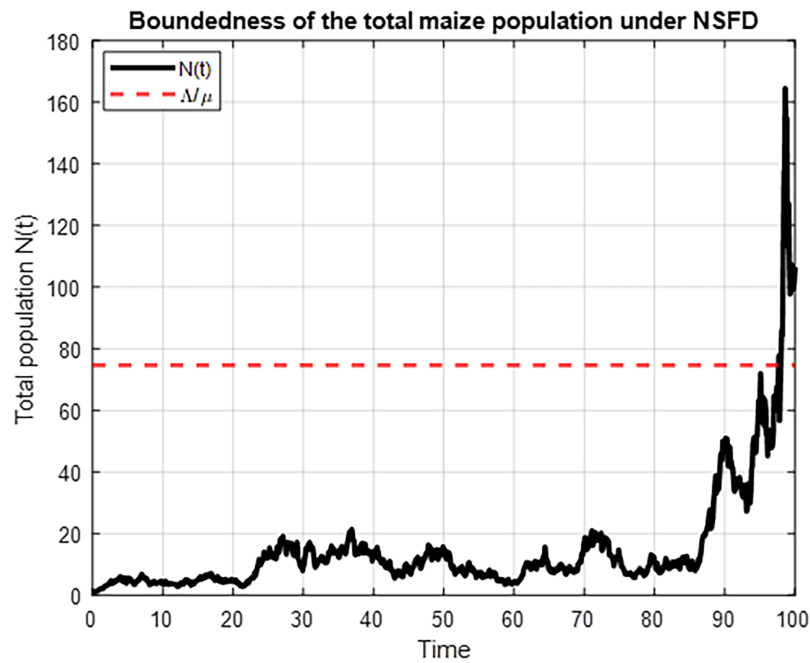


Figure 7: Boundedness of the total population $N(t)$ relative to the theoretical upper bound N/μ

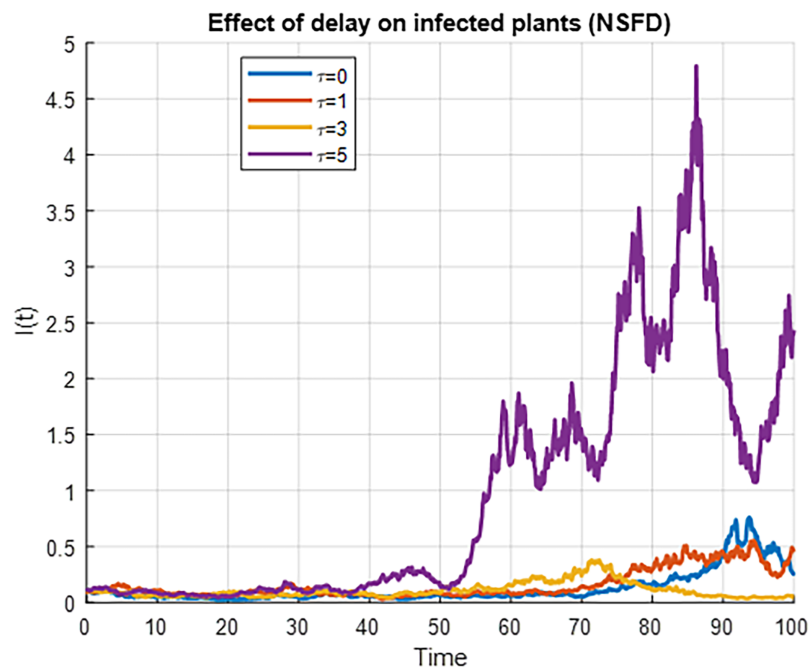


Figure 8: Impact of delay ($\tau = 0, 1, 3, 5$) on the infected population $I(t)$

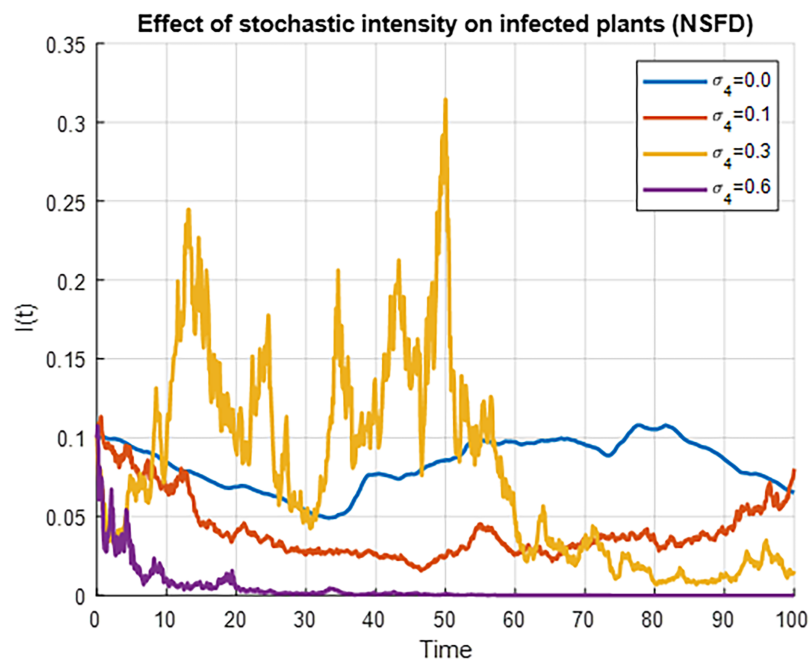


Figure 9: Influence of stochastic intensity σ_4 on the infected population $I(t)$ for different noise levels

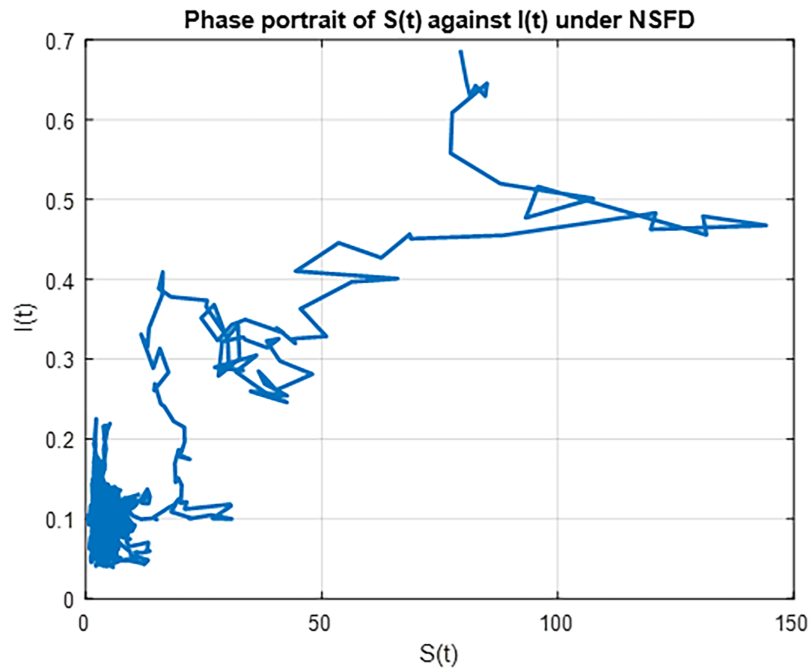


Figure 10: Phase portrait of the susceptible population $S(t)$ vs. the infected population $I(t)$ under stochastic delay dynamics

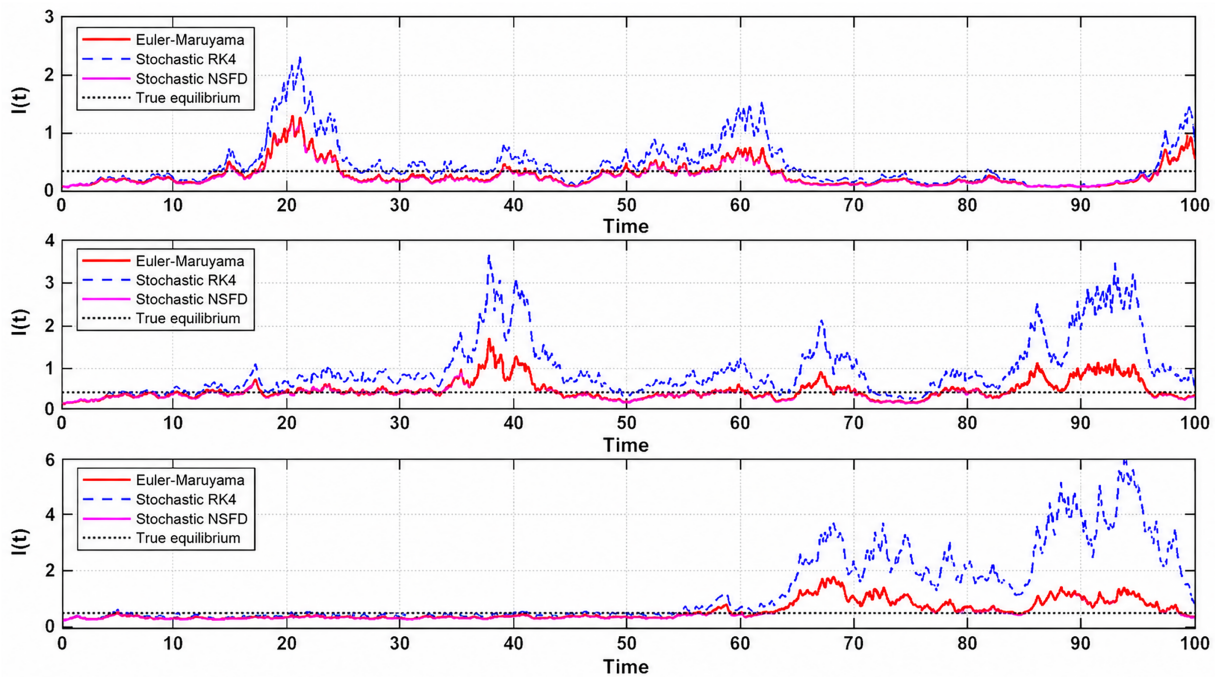


Figure 11: Comparison of Euler–Maruyama, stochastic RK4, and stochastic NSFD methods for the infected population $I(t)$ under different small step sizes $h = 0.01$, $h = 0.05$, and $h = 0.1$

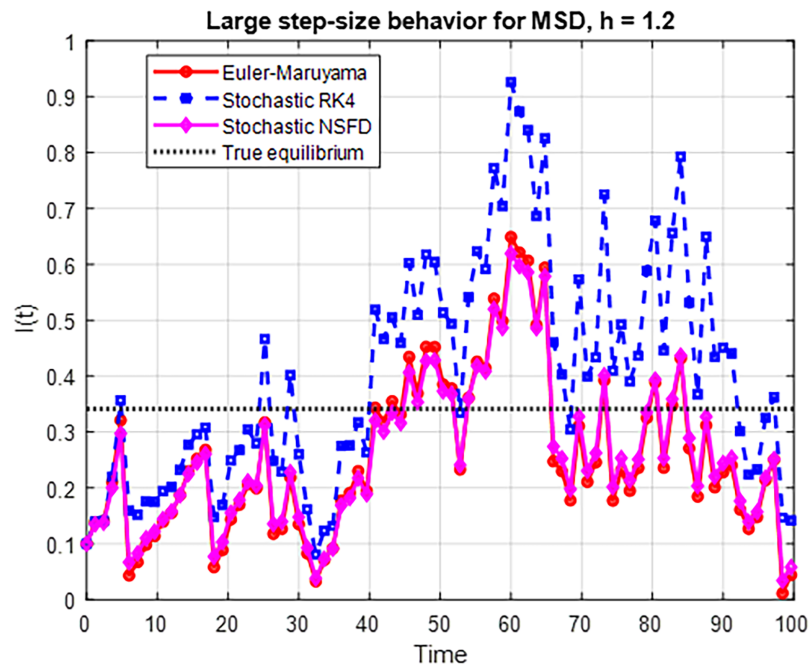


Figure 12: Comparison of Euler–Maruyama, stochastic RK4, and stochastic NSFD methods for the infected population $I(t)$ under stochastic delay dynamics

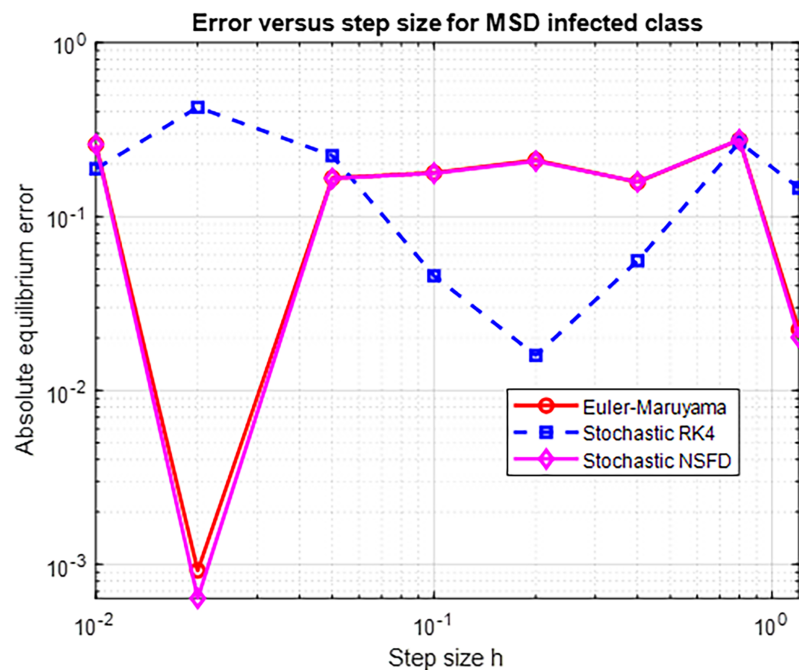


Figure 13: Absolute equilibrium error of Euler–Maruyama, stochastic RK4, and stochastic NSFD methods for different step sizes h

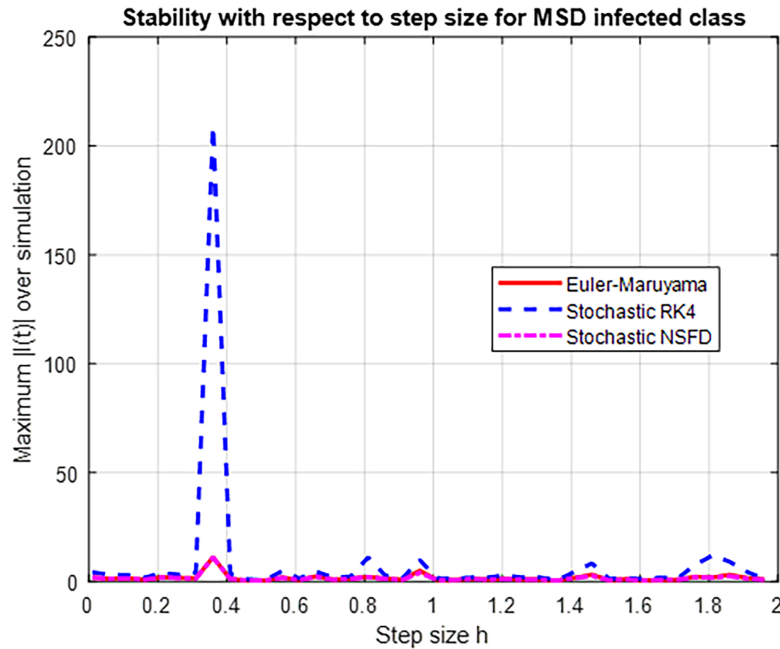


Figure 14: Maximum infection level obtained by Euler-Maruyama, stochastic RK4, and stochastic NSFD methods for varying step sizes h

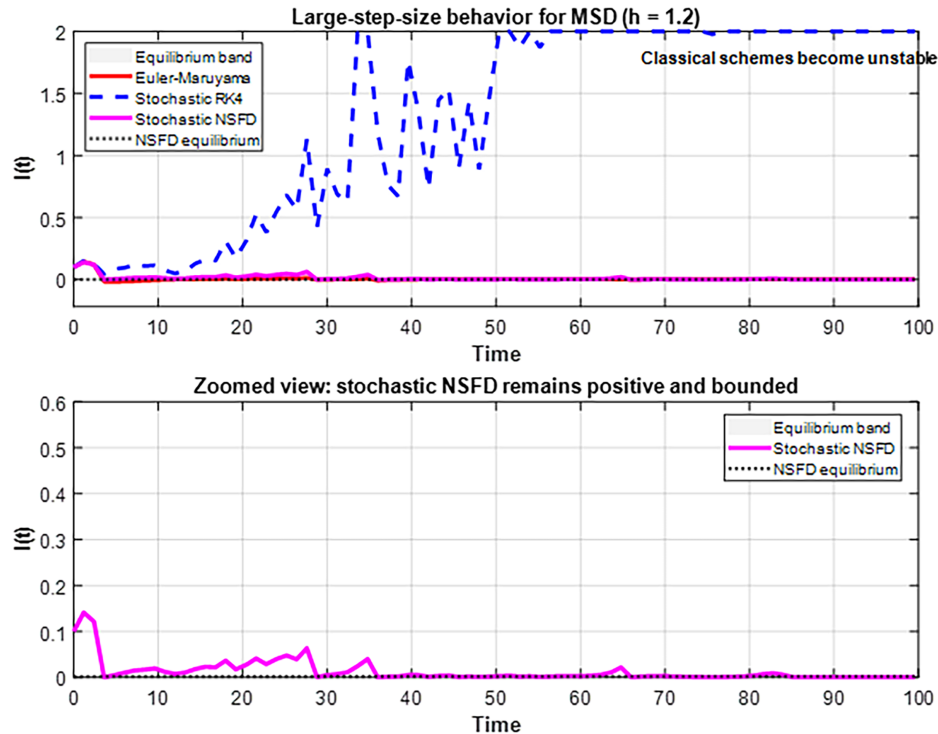


Figure 15: Comparison of numerical stability between classical schemes and the stochastic NSFD method under large step sizes

8 Conclusion

A stochastic delayed model of maize streak disease (MSD) was formulated and analyzed in this study. The model includes a discrete delay to account for incubation and vector-mediated transmission, and the stochastic perturbations account for field-level and environmental fluctuations. The class considered to be only temporary protection from the insecticide acting as a vector and not immunity. The qualitative analysis revealed that the positivity and boundedness of the model solutions are maintained. The next-generation matrix method was used to obtain the disease-free equilibrium, endemic equilibrium and effective reproduction number. The results of the stability analysis show that the disease-free equilibrium is stable when the reproduction number is less than 1 and is unstable when the reproduction number is greater than 1, thus allowing for the possibility of disease persistence when the reproduction number is greater than 1. The stochastic extension also demonstrated that random perturbations could alter the disease threshold and could potentially play a role in disease extinction in appropriate settings. The effects of the transmission rate, efficacy of insecticide, delay and noise intensity on MSD dynamics were demonstrated by numerical simulations with the stochastic NSFD scheme. The NSFD method was demonstrated to be biologically feasible with respect to positivity and boundedness, and compared qualitatively to the Euler–Maruyama method. The dataset for the MSD six years long and was only used for illustrative trend analysis, and not for statistical inference or parameter calibration. Future work will focus on rigorous parameter estimation using larger field datasets, uncertainty quantification, spatial effects, climate-driven transmission, and detailed convergence and stability analysis of the proposed numerical scheme.

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Availability of Data and Materials: All data used for the model development and numerical results are available within the manuscript and through the cited references.

Ethics Approval: Not applicable.

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References

1. Asare-Bediako E, Kvarnheden A, Van Der Puije GC, Taah KJ, Agyei Frimpong K, Amenorpe G, et al. Spatio-temporal variations in the incidence and severity of maize streak disease in the Volta region of Ghana. *J Plant Pathol Microbiol.* 2017;8(3):3. doi:10.4172/2157-7471.1000401.
2. Blankson D, Asare-Bediako E, Frimpong KA, Ampofo E, Taah KJ, Van der Puije GC. Incidence and severity of maize streak disease: the influence of tillage, fertilizer application and maize variety. *Afr J Agric Res.* 2018;13(12):551–60.
3. Charles AK, Muiru WM, Miano DW, Kimenju JW. Distribution of common maize diseases and molecular characterization of maize streak virus in Kenya. *J Agric Sci.* 2019;11(4):47. doi:10.5539/jas.v11n4p47.
4. Mbong GA, Suh C, Sime HD, Anoumaa M, Fonkou T, Laure Chimi Nkombo L, et al. Phytosanitary situation of maize streak virus in the main maize production zones of Cameroon; 2021;12(4):339–53.
5. Ayembillah AO, Seidu B, Bornaa CS. Mathematical modeling of the dynamics of maize streak virus disease (MSVD). *Math Model Control.* 2022;2(4):153–64. doi:10.3934/mmc.2022016.
6. Alemneh HT, Kassa AS, Godana AA. An optimal control model with cost effectiveness analysis of maize streak virus disease in maize plant. *Infect Dis Model.* 2021;6(4):169–82. doi:10.1016/j.idm.2020.12.001.
7. Alemneh HT, Makinde OD, Theuri DM. Optimal control model and cost effectiveness analysis of maize streak virus pathogen interaction with pest invasion in maize plant. *Egypt J Basic Appl Sci.* 2020;7(1):180–93. doi:10.1080/2314808x.2020.1769303.
8. Collins O. Mathematical model analysis for maize yield under co-infection of maize streak virus and maize stripe virus diseases with control measures. *J Nig Soc Phys Sci.* 2026;8(2):3112. doi:10.46481/jn-sps.2026.3112.
9. Iqbal S, Shahid N, Raza A, Lampart M, Ahmed N, Baleanu D, et al. Computational analysis of stochastic delay dynamics in maize streak virus. *PLoS One.* 2025;20(12):e0337556. doi:10.1371/journal.pone.0337556.
10. Ameen IG, Baleanu D, Ali HM. Different strategies to confront maize streak disease based on fractional optimal control formulation. *Chaos Solitons Fractals.* 2022;164(3):112699. doi:10.1016/j.chaos.2022.112699.
11. Liu Q. Dynamical analysis of a stochastic maize streak virus epidemic model with logarithmic Ornstein–Uhlenbeck process. *J Math Biol.* 2024;89(3):30. doi:10.1007/s00285-024-02127-3.
12. Ackora-Prah J, Seidu B, Okyere E, Asamoah JKK. Fractal-fractional caputo maize streak virus disease model. *Fractal Fract.* 2023;7(2):189. doi:10.3390/fractalfract7020189.
13. Malar MC, Gayathri M, Manickam A. A novel study on the maize streak virus epidemic model using caputo-fabrizio fractional derivative. *Contemp Math.* 2023;4(3):435–52. doi:10.37256/cm.4320232383.
14. Sime SS, Menkir A, Adetimirin VO, Gedil M, Kumar PL. Validation of diagnostic markers for streak virus disease resistance in maize. *Agriculture.* 2021;11(2):130. doi:10.3390/agriculture11020130.
15. Afram Y, Amenorpe G, Bediako EA, Darkwa AA, Shandu SF, Labuschagne MT, et al. Induction of genetic variability of maize genotypes through radiation revealed mutants resistant to maize streak disease. *Appl Radiat Isot.* 2024;207(2):111279. doi:10.1016/j.apradiso.2024.111279.
16. Mushayi M, Shimelis H, Derera J, Tesfamariam SA. Breeding for resistance to maize streak virus: challenges, progress and future directions: a review. *Front Plant Sci.* 2025;16:1590870. doi:10.3389/fpls.2025.1590870.
17. Choshi TJ, Dhau I, Mashao F. Enhancing maize streak virus detection: a comparative analysis of sentinel-2 MSI and landsat 9 OLI data across vegetative and reproductive growth stages. *Geocarto Int.* 2025;40(1):2480701. doi:10.1080/10106049.2025.2480701.

18. Pfordt A, Paulus S. A review on detection and differentiation of maize diseases and pests by imaging sensors. *J Plant Dis Prot.* 2024;132(1):40. doi:10.1007/s41348-024-01019-4.
19. Santa Cruz FC, Domalanta JC, Minguez LT, Nieto FGM, Alviar KB. First report of maize streak virus affecting maize in the Philippines. *New Dis Rep.* 2024;50(1):e12302. doi:10.1002/ndr2.12302.
20. Fouad NMI, Barro M, Bangratz M, Sérémé D, Filloux D, Fernandez E, et al. Old foe, new host: epidemiology, genetic diversity, and pathogenic characterization of maize streak virus in rice fields from Burkina Faso. *Phytobiomes J.* 2025;9(1):32–44.
21. Nivetha S, Ghosh M. Mathematical modeling and cost-effective intervention strategies for diabetes management: a data-driven and numerical analysis approach. *PLoS One.* 2026;2021(1):e0339463. doi:10.1371/journal.pone.0339463.
22. Ahmed KIA, Adam HDS, Almutairi N, Saber S. Analytical solutions for a class of variable-order fractional Liu system under time-dependent variable coefficients. *Results Phys.* 2024;56(11):107311. doi:10.1016/j.rinp.2023.107311.
23. Almutairi N, Saber S. Application of a time-fractal fractional derivative with a power-law kernel to the Burke-Shaw system based on Newton's interpolation polynomials. *MethodsX.* 2024;12(5):102510. doi:10.1016/j.mex.2023.102510.
24. Qu H, Rahman MU, Arfan M. Fractional model of smoking with relapse and harmonic mean type incidence rate under Caputo operator. *J Appl Math Comput.* 2023;69(1):403–20. doi:10.1007/s12190-022-01747-6.
25. Liu X, Ahmad S, Rahman MU, Nadeem Y, Akgül A. Analysis of a tb and hiv co-infection model under Mittag-Leffler fractal-fractional derivative. *Phys Scr.* 2022;97(5):054011. doi:10.1088/1402-4896/ac645e.
26. Raza A, Awrejcewicz J, Rafiq M, Ahmed N, Mohsin M. Stochastic analysis of nonlinear cancer disease model through virotherapy and computational methods. *Mathematics.* 2022;10(3):368. doi:10.3390/math10030368.
27. Food and Agriculture Organization. Maize production and plant disease reports in Africa. 2025 [cited 2026 Jan 1]. Available from: <https://www.fao.org>.
28. Shoaib Arif M, Raza A, Abodayeh K, Rafiq M, Bibi M, Nazeer A. A numerical efficient technique for the solution of susceptible infected recovered epidemic model. *Comput Model Eng Sci.* 2020;124(2):477–91. doi:10.32604/cmes.2020.011121.
29. Mrope FM, Kigodi OJ, Jeeva N, Manivel M. Mathematical modeling of the impact of insecticides on the transmission dynamics of maize streak disease. *Model Earth Syst Environ.* 2025;11(4):264. doi:10.1007/s40808-025-02443-7.