

A MATHEMATICAL MODEL FOR MAIZE STREAK VIRUS TRANSMISSION: FRACTIONAL APPROACH

SOUMAYE HARO*, ELISEE GOUBA, BOUKARY OUEDRAOGO

Laboratory for Numerical Analysis, Computer Science and Biomathematics (LANIBIO), Joseph Ki-Zerbo University,
Burkina Faso

*Corresponding author: soumaye_haro@ujkz.bf

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ABSTRACT. In this paper, we have formulated a Caputo fractional order model for the transmission dynamics of maize streak virus (MSV). The use of fractional differential equations in the sense of Caputo to model maize streak disease allows for the incorporation of long-term memory effects influencing the epidemic dynamics. This approach provides a more accurate representation of the slow and recurrent behaviors observed in the field, while enhancing the precision of predictions and the effectiveness of disease management strategies. After mathematical analysis, it appears that the proposed model is well-posed from both mathematical and epidemiological perspectives. The existence of a unique disease-free equilibrium has been proven. An explicit formula, dependent on the model parameters, has been obtained for the basic reproduction number R_0 used in epidemiology. The conditions for local and global asymptotic stability of the disease-free equilibrium point of the MSV have been established. Furthermore, we determined the sensitivity indices of the parameters with respect to R_0 in order to identify the most influential parameter and provide the best conditions for reducing the transmission of MSV disease in maize. Finally, numerical simulations were performed to illustrate and validate our theoretical results.

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Key words and phrases. MSV; fractional-order model; stability; sensitivity analysis; numerical simulations.

1. INTRODUCTION

Maize (*Zea mays* L.) is cultivated throughout the world, in both temperate and tropical zones, and is present on all continents [24]. In Africa, it is the most widespread and consumed food crop, serving as the main food source for more than 100 million people. Each year, its cultivation covers approximately 15.5 million hectares [19, 20]. Among all viral diseases affecting maize in Africa, infection caused by maize streak virus (MSV) stands out as the most widespread and devastating [40]. This virus is responsible for severe disease in many regions of the continent [41]. The symptoms were initially described as “mealie variegation” [42], before being redefined as “maize streak disease,” abbreviated

as MSV [43]. Maize production is limited by various abiotic and biotic factors. Among these factors is maize streak disease (MSD), which is one of the main biotic constraints in Burkina Faso [33]. This disease, considered the most destructive and devastating to maize in sub-Saharan Africa, particularly in Burkina Faso, is caused by the virus (MSV) [20, 33, 34]. In addition to maize, MSV poses a significant threat to more than 80 crop species [35], including oats, wheat, sorghum, millet, pearl millet, and sugarcane [19, 36]. MSD, which appeared in Burkina Faso in the 1990s [33], is a viral disease characterized by a genome consisting of a single-stranded circular DNA with a single component [19, 20]. For smallholder farmers in sub-Saharan Africa, MSD causes considerable economic losses, estimated at nearly US 480 million per year [37]. In some cases, the economic damage caused by this disease reaches 100% yield loss when infection occurs within the first three weeks after sowing [20, 25]. The maize streak virus (MSV) is considered to be the most economically damaging virus, causing yield losses of up to 100%. Its transmission, which is persistent but irregular, is carried out by leafhoppers of the genus *Cicadulina* [19, 26]. Thus far, 22 species of *Cicadulina* have been identified worldwide, including 18 in Africa. Among the eight known vectors of MSV in this genus, *Cicadulina mbila* is recognized as the most important and predominant in the epidemiology of the virus [19]. Mathematical modeling is the process of constructing mathematical objects whose behaviors or properties correspond in some way to a particular real-world system. In this description, a mathematical object could be a system of equations, a stochastic process, a geometric or algebraic structure, a simulation algorithm implemented on a computer, etc. The term real-world system could refer to a physical system, a financial system, a social system, an ecological system, etc [48]. Various models have been proposed and studied in the literature to describe and better understand disease transmission processes in plants. In [27], the authors analyzed the impact of leaf diseases on maize population dynamics using a mathematical epidemiological model. They also applied optimal control theory, considering different control strategies, including chemical and cultural methods and the use of resistant varieties. In [28], a continuous epidemiological model of African cassava mosaic disease was formulated, taking into account the local dynamics of healthy and infected cassava plants as well as infected and uninfected whitefly vectors. Furthermore, the authors of [29] revisited a differential equation model applied to plants, which they adapted to a specific disease based on a general model proposed in [30]. Finally, optimal control theory was also applied in [31] to a continuous deterministic epidemiological model of cassava brown streak disease, incorporating the application of chemicals and the removal of infected plants as control measures. In [32], an SI-SEI epidemiological model was proposed and studied for maize disease, simultaneously integrating host and vector populations. This framework allowed for an in-depth analysis of the dynamics of lethal necrosis in maize. In [33], the authors propose a deterministic eco-epidemiological model to study the transmission dynamics of MSV in the maize population. Models built from integer operators do not take into account memory effects present in

the system, due to their purely local nature. To better represent these effects in the dynamics studied, and drawing inspiration from the work presented in [27,31–33], we propose a fractional-order SI–SEI model based on the Caputo derivative to analyze the dynamics of MSV within maize populations. We believe that the results of this research will provide valuable insights for the implementation of effective strategies to control and even eradicate the transmission of the disease. Such an approach would help to ensure optimal yields for farmers and, consequently, enhance food security. This paper is organized as follows: In section 2, we present some preliminary mathematics. In section 3, we describe and formulate the model. In section 4, We performed a mathematical analysis of the model: First, we determine the invariant domain that guarantees both mathematical and epidemiological consistency of the model. Next, we calculate the basic reproduction number R_0 using the next-generation matrix method, as well as the disease-free equilibrium state E_0 . Next, the local stability of this disease-free equilibrium point is analyzed using the Jacobian matrix. In addition, the Castillo-Chavez method is used to study the global stability of this equilibrium. Finally, a sensitivity analysis is presented to identify the parameters that most influence R_0 and therefore the transmission dynamics of MSV. In section 5, we perform numerical simulations to verify our theoretical results. Section 6 is devoted to the conclusion.

2. PRELIMINARIES

In this section, we focus on the definitions and properties of two specific functions of fractional calculus: the Gamma function and the Mittag-Leffler function, which we will use in this paper. Furthermore, we introduce the fractional integration operator as well as several definitions of the most commonly used fractional derivatives, in particular the Caputo derivative, whose most important properties we discuss. (for more details, see [5–8]).

2.1. Some definitions and theorems.

Definition 1. [5] [*Euler’s gamma function*] The gamma function, denoted $\Gamma(\cdot)$, generalizes the factorial $n!$ and is defined by:

$$\Gamma(x) = \int_0^{+\infty} t^{x-1} e^{-t} dt; \quad R_e(x) > 0. \quad (1)$$

with $\Gamma(1) = 1$ and $\Gamma(0^+) = +\infty$.

Proposition 1. Euler’s gamma function satisfies the following propositions:

- (1) $\Gamma\left(\frac{1}{2}\right) = \int_0^{\infty} t^{-\frac{1}{2}} e^{-t} dt = \sqrt{\pi}$.
- (2) $\Gamma(1) = \int_0^{\infty} e^{-t} dt = 1$.
- (3) Integration by parts gives us: $\Gamma(x+1) = \int_0^{\infty} t^x e^{-t} dt = x\Gamma(x)$.

(4) Euler's gamma function generalizes the factorial, we have $\Gamma(n+1) = n!$.

Definition 2. [5] [Mittag-Leffler function] The concept of the Mittag-Leffler function was first introduced by the mathematician in 1903 [44]. This function is a generalization of the exponential function, for which it plays the same role. The Mittag-Leffler function has a single parameter α and is denoted by $\mathbf{E}_\alpha(\cdot)$. It is defined by

$$\mathbf{E}_\alpha(z) = \sum_{k=0}^{+\infty} \frac{z^k}{\Gamma(\alpha_k + 1)}, z \in \mathbb{C}, \alpha > 0$$

The two-parameter Mittag-Leffler function was introduced by Agarwal [12]. This function also plays a key role in fractional calculus theory and is defined by: the following series expansion:

$$\mathbf{E}_{\alpha,\beta}(z) = \sum_{k=0}^{+\infty} \frac{z^k}{\Gamma(\alpha^k + \beta)}, z \in \mathbb{C}, \alpha, \beta > 0 \quad (2)$$

Property 1. The Mittag-Leffler function has properties, the most important of which are:

- (1) $E_{1,1}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(k+1)} = \sum_{k=0}^{\infty} \frac{z^k}{k!} = e^z$.
- (2) $E_{2,1}(z^2) = \sum_{k=0}^{\infty} \frac{z^{2k}}{\Gamma(2k+1)} = \sum_{k=0}^{\infty} \frac{z^{2k}}{2k!} = \cosh(z)$.
- (3) $E_{2,2}(z^2) = \sum_{k=0}^{\infty} \frac{z^{2k}}{\Gamma(2k+2)} = \frac{1}{z} \sum_{k=0}^{\infty} \frac{z^{2k+1}}{(2k+1)!} = \frac{\sinh}{z}$.
- (4) $E_{\alpha,1}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k+1)} = E_\alpha(z)$.

Definition 3. Let $\alpha > 0$, the Caputo fractional derivative of order α , denoted by ${}^C D^\alpha$, of a function f is defined by:

$$\begin{aligned} {}^C D^\alpha f(t) &= I^{n-\alpha} \left(\left(\frac{d}{dt} \right)^n f \right) (t) \\ &= \frac{1}{\Gamma(n-\alpha)} \int_a^t (t-\tau)^{n-\alpha-1} f^{(n)}(\tau) d\tau \end{aligned}$$

where $t \geq 0, n-1 < \alpha < n \in \mathbb{Z}^+$ and $f^{(n)}(\tau)$ denotes the n th derivative of f .

Definition 4. Let $f \in L^1_{loc}(\mathbb{R}_+)$ be zero for $t < 0$.

We call the Laplace transform of f (if it exists) the mapping defined by:

$$\begin{aligned} L\{f(t)\} : \mathbb{R} &\rightarrow \mathbb{C} \\ s &\mapsto L\{f(t)\}(s) = \int_0^\infty \exp(-st) f(t) dt, \quad s \in \mathbb{C} \end{aligned}$$

We also use the expression $F(s)$ to describe the Laplace transform. We have

$$F(s) = L\{f(t)\} \iff f(t) = L^{-1}\{F(s)\} = \int_{c-i\infty}^{c+i\infty} \exp(st) F(s) ds, \quad c = \operatorname{Re}(s) > c_0,$$

Definition 5. [Laplace transform] The Laplace transform of the two-parameter Mittag-Leffler function is

❶

$$L\left\{t^{\beta-1} E_{\alpha,\beta}(\mp \lambda t^\alpha); s\right\} = \frac{s^{\alpha-\beta}}{s^\alpha \pm \lambda}, \quad \operatorname{Re}(s) > |\lambda|^{\frac{1}{\alpha}}, \lambda \in \mathbb{R}$$

② Let $f \in C^n(\mathbb{R}^+)$, $n \in \mathbb{N}$. Then

$$L\{f^{(n)}(t); s\} = s^n F(s) - \sum_{k=0}^{n-1} s^{n-k-1} f^{(k)}(0).$$

③ Using the same property as before, we obtain the following formula:

$$L\{{}^C D^\alpha f(t); s\} = s^\alpha F(s) - \sum_{k=0}^{n-1} s^{\alpha-k-1} f^{(k)}(0), \quad n-1 \leq \alpha \leq n.$$

Definition 6. [Inverse Laplace transform] The inverse Laplace transform of the function $F(s)$, denoted $f(t) = \mathcal{L}^{-1}(F(s))(t)$, is given by:

$$f(t) = \mathcal{L}^{-1}(F(s))(t) = \frac{1}{2i\pi} \int_{-\infty}^{+\infty} F(s) e^{-st} ds,$$

is such that the integral exists ($\mathbf{R}_e(s) \geq 0$).

Theorem 1. [10][Banach contraction] Let X be a Banach space and

$F : X \rightarrow X$

a contraction, that is, there exists $0 \leq k < 1$ such that: for all

$$x, z \in X, \|F(x) - F(z)\|_X \leq k \|x - z\|_X,$$

then F has a unique fixed point.

Theorem 2. [11]Banach fixed point theorem] Let E be a closed subset

of a Banach space Z and T a mapping associating the set E with E .

Suppose that: $\|T(x) - T(z)\|_E \leq k \|x - z\|_E, \forall x, z \in E, 0 \leq k < 1$, then

- ① there exists a unique vector $x^* \in E$ such that $x^* = T(x^*)$,
- ② x^* can be obtained by the method of successive approximations, starting from any arbitrary initial vector in E .

2.2. Stability. This subsection is devoted to the study of the stability of fractional differential equations in the linear and nonlinear cases. We are particularly interested in autonomous and non-autonomous fractional differential equations of the Caputo type. We begin by presenting the stability result in the case of a linear autonomous fractional differential equation with constant coefficients, as given in [13,16].

2.2.1. The linear case. Consider the following fractional differential equation:

$$\begin{cases} {}^C D^\alpha x(t) = Ax(t), \\ x(0) = x_0, \end{cases} \quad (3)$$

where $x \in \mathbb{R}^n, 0 < \alpha < 1$ and $A \in \mathbb{R}^{n \times n}$ is a matrix.

1. The system 3 is asymptotically stable if and only if the eigenvalues λ of the matrix A satisfy the following condition $|\arg \lambda| > \frac{\alpha\pi}{2}$. Also, if the state vector $x(t)$ tends toward zero with $\|x(t)\| < C_1 t^{-\alpha}$, $t > 0$, $\alpha > 0$.
2. The system 3 is stable if and only if the eigenvalues λ of the matrix A satisfy the following condition $|\arg \lambda| \geq \frac{\alpha\pi}{2}$ and all eigenvalues satisfying $|\arg \lambda| = \frac{\alpha\pi}{2}$ have geometric multiplicity equal to one.

2.2.2. *The nonlinear nonautonomous case.* Consider the following fractional differential equation:

$$\begin{cases} {}^C D^\alpha x(t) = A(t)x(t) + f(x(t)), \\ x(0) = x_0, \end{cases} \quad (4)$$

where $x \in \mathbb{R}^n$, $0 < \alpha < 1$, $A \in \mathbb{R}^{n \times n}$ is a matrix, and $f(x) \in \mathbb{R}^n$ is a continuous nonlinear function. We have the following result:

Theorem 3. [45,46] Let $x = 0$ be the equilibrium point of the system 4 if we have

$$\begin{aligned} 1- & \lim_{t \rightarrow \infty} A(t) = A \\ 2- & f(0) = 0 \text{ and } \lim_{\|x\| \rightarrow 0} \frac{\|f(x)\|}{\|x\|} = 0 \\ 3- & |\arg(\text{spec}(A))| > \alpha \frac{\pi}{2} \text{ and } \|A\| > \frac{N_\alpha}{\alpha} \geq 1, \end{aligned}$$

where N_α (sometimes simply denoted by N) denotes a positive real constant that depends on the fractional order α , used as a stability threshold in the asymptotic analysis of fractional systems. Then the equilibrium point of system 4 is asymptotically stable.

3. DESCRIPTION AND FORMULATION OF THE MODEL

3.1. Description of the model. In this section, we formulate a mathematical model where two populations interact: the maize population and the leafhopper population. To do this, we divided the maize population into two subpopulations: the density of susceptible maize ($S_m(t)$) and the density of infected maize ($I_m(t)$). As for the leafhopper vector population, it is divided into the density of susceptible leafhoppers ($S_v(t)$), the density of exposed leafhoppers ($E_v(t)$), and the density of infected leafhoppers ($I_v(t)$). In the absence of leafhopper predators and disease, the host population undergoes logistic growth characterized by an intrinsic growth rate r and an environmental carrying capacity K that is strictly positive. Conversely, when the disease is present in the maize population, infected individuals participate in the growth dynamics of the susceptible host population, which then tends towards the carrying capacity K . Susceptible leafhoppers are recruited at a rate b_v . When they feed on infected maize plants, they join the infected leafhopper subclass at a rate β_2 . Their natural mortality is described by the rate α_3 , while infected leafhoppers experience natural mortality at the rate α_4 and a

conversion rate b . Both leafhopper population subclasses have a mortality rate α_v due to predators. MSV transmission to the susceptible host occurs when it comes into contact with an infected leafhopper. Thus, susceptible maize plants become infected at a rate β_1 after interaction with a leafhopper carrying the virus. Once infected, the plants do not recover and their maize yield becomes zero or very low. In addition, susceptible and infected maize plants undergo a reduction due to natural mortality at rates of α_1 and α_2 , respectively. Infected maize plants also undergo a reduction due to disease-induced mortality α_m and a rate θ_m , at which they are removed from susceptible individuals. Furthermore, the disease is not transmissible horizontally or vertically in either population and is not genetically hereditary. The functional predation response of leafhoppers on susceptible maize is assumed to follow Michaelis-Menten kinetics and is represented by a Holling II-type function, [39], characterized by the predation and infection coefficients β_1 and β_2 , respectively, and the half-saturation constants A and C . All parameters and their definitions are summarized in table 1 and 2. Based on these considerations, the corresponding compartmental scheme is presented in figure 1.

Parameter	Description
β_1	Predation and infection rates of infected leafhoppers on susceptible maize plants.
K	Carrying capacity .
A	Half-saturation constant of susceptible maize plants with infected plants .
α_1	Natural mortality rate of susceptible maize plants .
α_2	Natural mortality rate of infected maize plants.
α_m	Disease-induced mortality rate of maize plants.
θ_m	Rate at which infected maize plants are removed from susceptible maize plants.
r	Intrinsic growth rate of maize.

TABLE 1. Specific maize parameters used in the model.

Parameter	Description
β_2	Predation and infection rates of susceptible leafhoppers on infected maize plants
b	Conversion rate of infected leafhoppers.
b_v	Recruitment rate of susceptible leafhoppers.
k_v	Rate of progression from the exposed class to the infectious class
C	Half-saturation constant of susceptible leafhoppers with infected maize plants
α_3	Natural mortality rate of susceptible and exposed leafhoppers.
α_4	Natural mortality rate of infected leafhoppers.
α_v	Leafhopper mortality rate due to predators.

TABLE 2. Specific leafhopper parameters used in the model.

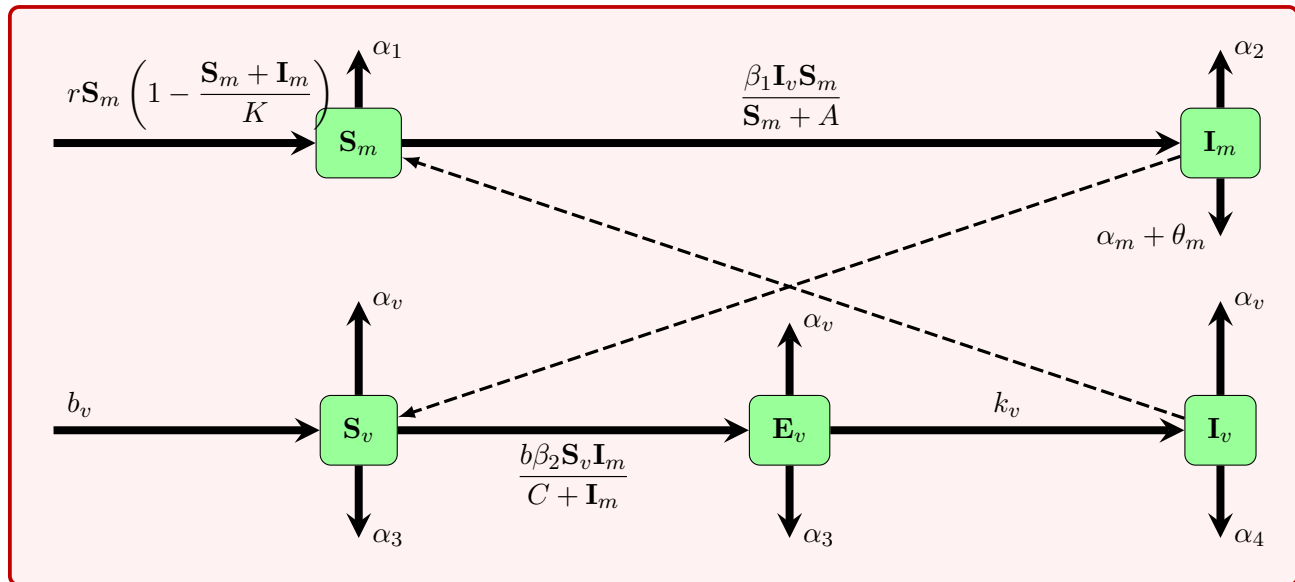


FIGURE 1. Flowchart of the model

3.2. Formulation of the mathematical model of MSV transmission in maize. Based on Figure 1 and the description of the model given above, we establish model (5), represented by a system of differential equations of integer order.

$$\left\{ \begin{array}{l} \frac{dS_m(t)}{dt} = rS_m(t) \left(1 - \frac{S_m(t) + I_m(t)}{K} \right) - \alpha_1 S_m(t) - \beta_1 \frac{I_v(t) S_m(t)}{A + S_m(t)} \\ \frac{dI_m(t)}{dt} = \beta_1 \frac{I_v(t) S_m(t)}{A + S_m(t)} - (\alpha_2 + \alpha_m + \theta_m) I_m(t) \\ \frac{dS_v(t)}{dt} = b_v - (\alpha_v + \alpha_3) S_v(t) - b\beta_2 \frac{S_v(t) I_m(t)}{C + I_m(t)} \\ \frac{dE_v(t)}{dt} = b\beta_2 \frac{S_v(t) I_m(t)}{C + I_m(t)} - (k_v + \alpha_v + \alpha_3) E_v(t) \\ \frac{dI_v(t)}{dt} = k_v E_v(t) - (\alpha_v + \alpha_4) I_v(t). \end{array} \right. \quad (5)$$

Fractional-order differential equations describe many phenomena in various fields of applied science and engineering such as biology, physics, mathematics, etc. Fractional-order derivatives also provide an excellent tool for describing the memory and hereditary properties of various materials and processes [1]. Furthermore, fractional order models capture memory, improve data fidelity, enrich stability analysis, and make models more biologically realistic. Thus, we perturb the deterministic system (5) to obtain a fractional order model in the sense of Caputo. The fractional order model thus obtained is

defined by the system (6).

$$\left\{ \begin{aligned} {}^c_0\mathcal{D}_t^\gamma [\mathbf{S}_m(t)] &= r\mathbf{S}_m(t) \left(1 - \frac{\mathbf{S}_m(t) + \mathbf{I}_m(t)}{K} \right) - \alpha_1\mathbf{S}_m(t) - \beta_1 \frac{\mathbf{I}_v(t)\mathbf{S}_m(t)}{A + \mathbf{S}_m(t)} \\ {}^c_0\mathcal{D}_t^\gamma [\mathbf{I}_m(t)] &= \beta_1 \frac{\mathbf{I}_v(t)\mathbf{S}_m(t)}{A + \mathbf{S}_m(t)} - (\alpha_2 + \alpha_m + \theta_m) \mathbf{I}_m(t) \\ {}^c_0\mathcal{D}_t^\gamma [\mathbf{S}_v(t)] &= b_v - (\alpha_v + \alpha_3) \mathbf{S}_v(t) - b\beta_2 \frac{\mathbf{S}_v(t)\mathbf{I}_m(t)}{C + \mathbf{I}_m(t)} \\ {}^c_0\mathcal{D}_t^\gamma [\mathbf{E}_v(t)] &= b\beta_2 \frac{\mathbf{S}_v(t)\mathbf{I}_m(t)}{C + \mathbf{I}_m(t)} - (k_v + \alpha_v + \alpha_3) \mathbf{E}_v(t) \\ {}^c_0\mathcal{D}_t^\gamma [\mathbf{I}_v(t)] &= k_v \mathbf{E}_v(t) - (\alpha_v + \alpha_4) \mathbf{I}_v(t), \end{aligned} \right. \quad (6)$$

where ${}^c_0\mathcal{D}_t^\gamma$ denotes the fractional derivative in the Caputo sense with $0 < \gamma < 1$.

Applying the fractional integral operator γ in the Caputo sense to the system (6), we obtain the system (7).

$$\left\{ \begin{aligned} \mathbf{S}_m(t) &= \mathbf{S}_m(0) + \frac{1}{\Gamma(\gamma)} \int_0^t (t-u)^{\gamma-1} \left(r\mathbf{S}_m(u) \left(1 - \frac{\mathbf{S}_m(u) + \mathbf{I}_m(u)}{K} \right) - \alpha_1\mathbf{S}_m(u) - \frac{\beta_1\mathbf{I}_v(u)\mathbf{S}_m(u)}{A + \mathbf{S}_m(u)} \right) du \\ \mathbf{I}_m(t) &= \mathbf{I}_m(0) + \frac{1}{\Gamma(\gamma)} \int_0^t (t-u)^{\gamma-1} \left(\beta_1 \frac{\mathbf{I}_v(u)\mathbf{S}_m(u)}{A + \mathbf{S}_m(u)} - (\alpha_2 + \alpha_m + \theta_m) \mathbf{I}_m(u) \right) du \\ \mathbf{S}_v(t) &= \mathbf{S}_v(0) + \frac{1}{\Gamma(\gamma)} \int_0^t (t-u)^{\gamma-1} \left(b_v - (\alpha_v + \alpha_3) \mathbf{S}_v(u) - b\beta_2 \frac{\mathbf{S}_v(u)\mathbf{I}_m(u)}{C + \mathbf{I}_m(u)} \right) du \\ \mathbf{E}_v(t) &= \mathbf{E}_v(0) + \frac{1}{\Gamma(\gamma)} \int_0^t (t-u)^{\gamma-1} \left(b\beta_2 \frac{\mathbf{S}_v(u)\mathbf{I}_m(u)}{C + \mathbf{I}_m(u)} - (k_v + \alpha_v + \alpha_3) \mathbf{E}_v(u) \right) du, \\ \mathbf{I}_v(t) &= \mathbf{I}_v(0) + \frac{1}{\Gamma(\gamma)} \int_0^t (t-u)^{\gamma-1} (k_v \mathbf{E}_v(u) - (\alpha_v + \alpha_4) \mathbf{I}_v(u)) du, \end{aligned} \right. \quad (7)$$

with the initial conditions

$$\mathbf{S}_m(0) \geq 0, \mathbf{I}_m(0) \geq 0, \mathbf{S}_v(0) \geq 0, \mathbf{E}_v(0) \geq 0, \mathbf{I}_v(0) \geq 0.$$

By adding the first two equations of system (6) on the one hand and these last three equations on the other, we obtain respectively the total population of maize plants and that of the Leafhopper vectors:

$${}^c_0\mathcal{D}_t^\gamma [\mathbf{M}_m(t)] = r\mathbf{S}_m(t) \left(1 - \frac{\mathbf{S}_m(t) + \mathbf{I}_m(t)}{K} \right) - (\theta_m + \alpha_m + \alpha_2) \mathbf{I}_m(t) - \alpha_m \mathbf{S}_m(t) \quad (8)$$

and

$${}_0^c \mathcal{D}_t^\gamma [\mathbf{M}_v(t)] = b_v - \alpha_v \mathbf{M}_v(t) - \alpha_3 (\mathbf{S}_v(t) + \mathbf{E}_v(t)) - \alpha_4 \mathbf{I}_v(t). \quad (9)$$

4. MATHEMATICAL ANALYSIS OF THE MODEL

4.1. Boundedness of solutions. In this subsection, we determine a region in which the solution of the model (6) is bounded. To do this, we state the following theorem:

Theorem 4. *The biologically feasible domain of the model (6) given by:*

$$\Omega = \left\{ \left(\mathbf{S}_m(t), \mathbf{I}_m(t), \mathbf{S}_v(t), \mathbf{E}_v(t), \mathbf{I}_v(t) \right) \in \mathbb{R}_+^5 \left| \begin{cases} 0 \leq \mathbf{M}_m(t) \leq \frac{\hat{k}(r+1)}{\eta} \\ 0 \leq \mathbf{M}_v(t) \leq \frac{b_v}{\alpha_v} \end{cases} \right. \right\} \quad (10)$$

is positively invariant and attractive.

Proof 1. By applying an appropriate upper bound to the equation (9), we obtain:

$${}_0^c \mathcal{D}_t^\gamma [\mathbf{M}_v(t)] \leq b_v - \alpha_v \mathbf{M}_v(t). \quad (11)$$

Which implies that

$${}_0^c \mathcal{D}_t^\gamma [\mathbf{M}_v(t)] + \alpha_v \mathbf{M}_v(t) \leq b_v. \quad (12)$$

Applying the Laplace transform to (12), we obtain:

$$s^\gamma \mathbf{M}_v(s) - s^{\gamma-1} \mathbf{M}_v(0) + \alpha_v \mathbf{M}_v(s) \leq \frac{b_v}{s}. \quad (13)$$

Which implies that

$$\mathbf{M}_v(s) \leq \frac{b_v}{s(s^\gamma + \alpha_v)} + \frac{s^{\gamma-1}}{s^\gamma + \alpha_v} \mathbf{M}_v(0). \quad (14)$$

As

$$\frac{b_v}{s(s^\gamma + \alpha_v)} = \frac{b_v}{\alpha_v} \left(\frac{1}{s} - \frac{s^{\gamma-1}}{s^\gamma + \alpha_v} \right), \quad (15)$$

Then, by substituting (15) into (14), we obtain:

$$\mathbf{M}_v(s) \leq \frac{b_v}{\alpha_v} \left(\frac{1}{s} - \frac{s^{\gamma-1}}{s^\gamma + \alpha_v} \right) + \frac{s^{\gamma-1}}{s^\gamma + \alpha_v} \mathbf{M}_v(0). \quad (16)$$

Which implies that

$$\mathbf{M}_v(s) \leq \left(\mathbf{M}_v(0) - \frac{b_v}{\alpha_v} \right) \frac{s^{\gamma-1}}{s^\gamma + \alpha_v} + \frac{b_v}{\alpha_v} \left(\frac{1}{s} \right). \quad (17)$$

Using the definition of the Taylor series, we have

$$\frac{s^{\gamma-1}}{s^\gamma + \alpha_v} = \frac{1}{s} \left(\frac{1}{1 + \frac{\alpha_v}{s^\gamma}} \right) = \sum_{k=0}^{\infty} \frac{(-\alpha_v)^k}{s^{k\gamma+1}}. \quad (18)$$

Substituting (18) into (17), we obtain:

$$\mathbf{M}_v(s) \leq \left(\mathbf{M}_v(0) - \frac{b_v}{\alpha_v} \right) \sum_{k=0}^{\infty} \frac{(-\alpha_v)^k}{s^{k\gamma+1}} + \frac{b_v}{\alpha_v} \left(\frac{1}{s} \right). \quad (19)$$

Applying the inverse Laplace transform to the inequality (19), we obtain:

$$\mathbf{M}_v(t) \leq \left(\mathbf{M}_v(0) - \frac{b_v}{\alpha_v} \right) \mathbf{E}_\gamma(-\alpha_v t^\gamma) + \frac{b_v}{\alpha_v}, \quad (20)$$

where $\mathbf{E}_{\gamma, \cdot}(\cdot)$ denotes the Mittag-Leffler function series defined in the preliminaries.

By calculating the limit of $\mathbf{M}_v(t)$ as t tends to $+\infty$, we obtain:

$$\mathbf{M}_v(t) \leq \frac{b_v}{\alpha_v}. \quad (21)$$

By finding an appropriate upper bound for equation (8), we obtain:

$${}_0^c \mathcal{D}_t^\gamma [\mathbf{M}_m(t)] \leq r \mathbf{S}_m(t) - (\theta_m + \alpha_m + \alpha_2) \mathbf{I}_m(t). \quad (22)$$

Now

$$r \mathbf{S}_m(t) - (\theta_m + \alpha_m + \alpha_2) \mathbf{I}_m(t) = \mathbf{S}_m(t) (r + 1) - [\mathbf{S}_m(t) + (\theta_m + \alpha_m + \alpha_2) \mathbf{I}_m(t)]. \quad (23)$$

Therefore, by substituting (23) into (22), we obtain:

$$\begin{aligned} {}_0^c \mathcal{D}_t^\gamma [\mathbf{M}_m(t)] &\leq \mathbf{S}_m(t) (r + 1) - [\mathbf{S}_m(t) + (\theta_m + \alpha_m + \alpha_2) \mathbf{I}_m(t)] \\ &\leq \hat{k} (r + 1) - \eta \mathbf{M}_m(t), \end{aligned}$$

where $\hat{k} = \max\{\mathbf{S}_m(0), K\}$ and $\eta = \min\{1, \alpha_2 + \alpha_m + \theta_m\}$, which implies that

$${}_0^c \mathcal{D}_t^\gamma [\mathbf{M}_m(t)] + \eta \mathbf{M}_m(t) \leq \hat{k} (r + 1). \quad (24)$$

After solving (24) and letting t tend towards $+\infty$, we obtain:

$$\mathbf{M}_m(t) \leq \frac{\hat{k}}{\eta} (r + 1). \quad (25)$$

From the inequalities (21) and (25), we can conclude that the solutions of model (6) are bounded.

Thus, the biologically feasible domain of model (6) is:

$$\Omega = \left\{ \left(\mathbf{S}_m(t), \mathbf{I}_m(t), \mathbf{S}_v(t), \mathbf{E}_v(t), \mathbf{I}_v(t) \right) \in \mathbb{R}_+^5 \left| \begin{cases} 0 \leq \mathbf{M}_m(t) \leq \frac{b_v}{\alpha_v} \\ 0 \leq \mathbf{M}_v(t) \leq \frac{\hat{k} (r + 1)}{\eta} \end{cases} \right. \right\}$$

○

4.2. Existence and uniqueness of solutions. In this subsection, we will show that the system (6) admits a unique solution in the set Ω . To do this, let us state the following theorems:

Theorem 5. The system (6) satisfies the Cauchy Lipschitz continuity conditions.

Proof 2. For the proof, let's write the system (6) in the form:

$$\begin{cases} {}^c\mathcal{D}_t^\gamma [x(t)] = G(t, x), \quad x(0) = x_0 \\ G(t, x) = Bx(t) + f(x(t)) + \Psi, \end{cases} \quad (26)$$

where

$$x(t) = (\mathbf{S}_m(t), \mathbf{I}_m(t), \mathbf{S}_v(t), \mathbf{E}_v(t), \mathbf{I}_v(t))^T, \quad \Psi = (0, 0, b_v, 0, 0)^T,$$

$$x(0) = x_0 = (\mathbf{S}_m(0), \mathbf{I}_m(0), \mathbf{S}_v(0), \mathbf{E}_v(0), \mathbf{I}_v(0))^T,$$

$$B = \begin{pmatrix} -\alpha_1 & 0 & 0 & 0 & 0 \\ 0 & -\alpha_c - \alpha_m - \theta_m & 0 & 0 & 0 \\ 0 & 0 & -\alpha_v - \alpha_3 & 0 & 0 \\ 0 & 0 & 0 & -k_v - \alpha_v - \alpha_3 & 0 \\ 0 & 0 & 0 & k_v & -\alpha_v - \alpha_4 \end{pmatrix}$$

and

$$f(x(t)) = \begin{pmatrix} r\mathbf{S}_m(t) \left(1 - \frac{\mathbf{S}_m(t) + \mathbf{I}_m(t)}{K}\right) - \beta_1 \frac{\mathbf{I}_v(t)\mathbf{S}_m(t)}{A + \mathbf{S}_m(t)} \\ \beta_1 \frac{\mathbf{I}_v(t)\mathbf{S}_m(t)}{A + \mathbf{S}_m(t)} \\ -b\beta_2 \frac{\mathbf{S}_v(t)\mathbf{I}_m(t)}{C + \mathbf{I}_m(t)} \\ b\beta_2 \frac{\mathbf{S}_v(t)\mathbf{I}_m(t)}{C + \mathbf{I}_m(t)} \\ 0 \end{pmatrix}.$$

Let $G(t, x)$ be the function such that:

$$\begin{aligned} G : \mathbb{R}_+ \times \mathbb{R}_+^5 &\longrightarrow \mathbb{R}_+^5 \\ (t, x) &\longmapsto G(t, x(t)) = Bx(t) + f(x(t)) + \Psi. \end{aligned}$$

To demonstrate the existence and uniqueness of the Cauchy problem (26), we will first show that G is locally Lipschitz with respect to $x(t)$, that is to say:

$$\text{for all } x_1(t), x_2(t) \in \mathbb{R}_+^5, \quad \|G(t, x_1(t)) - G(t, x_2(t))\| \leq C \|x_1(t) - x_2(t)\|, \quad \text{for all } C > 0.$$

$$\begin{aligned}
\|G(t, x_1(t)) - G(t, x_2(t))\| &= \|B(x_1(t) - x_2(t)) + f(x_1(t)) - f(x_2(t))\| \\
&\leq \|B(x_1(t) - x_2(t))\| + \|x_1(t) - x_2(t)\| \\
&\leq \|B\| \|x_1(t) - x_2(t)\| + \|x_1(t) - x_2(t)\| \\
&\leq (\|B\| + 1) \|x_1(t) - x_2(t)\| \\
&\leq K_2 \|x_1(t) - x_2(t)\|, \quad K_2 = \|B\| + 1 < \infty.
\end{aligned}$$

Therefore, $G(t, x(t))$ is continuous and bounded.

Consequently, it is locally Lipschitzian with respect to $x(t)$. □

Theorem 6. [15,16] (Picard-Lindelof)

Let $0 < \gamma < 1$, $I = [0, T^*] \subset \mathbb{R}$ and $J = \{x(t) - x(0) \mid |x(t) - x(0)| \leq k\}$. Let $g : I \times J \rightarrow \mathbb{R}$ be a continuous and bounded function, that is, there exists a unique constant $M > 0$ such that $|g(t, x(t))| \leq M$.

If $kK_2 < M$, then $x \in C([0, T^*])$ is a solution to the problem (26), $T^* = \min \left(T, \left(\frac{k\Gamma(\gamma + 1)}{M} \right)^{\frac{1}{\gamma}} \right)$.

Proof 3. Consider $T = \{x \in C([0, T^*]) : \|x(t) - x(0)\| \leq k\}$. Since $T \subseteq \mathbb{R}$, it follows that T is a closed set; therefore, T is a complete metric space. Hence, the problem (26) can be rewritten as follows:

$${}_0^c \mathcal{D}_t^{-\gamma} [{}_0^c \mathcal{D}_t^\gamma [x(t)]] = {}_0^c \mathcal{D}_t^{-\gamma} [g(t, x(t))],$$

which implies that

$$x(t) = x(0) + {}_0^c \mathbf{I}_t^\gamma [g(t, x(t))],$$

that is to say

$$x(t) = x(0) + \frac{1}{\Gamma(\gamma)} \int_0^t (t-s)^{\gamma-1} g(s, x(s)) ds. \quad (27)$$

To solve problem (26), consider equation (27), equivalent to a Volterra integral equation, and determine an operator G^* defined in T by:

$$G^*[x(t)] = x(0) + \frac{1}{\Gamma(\gamma)} \int_0^t (t-s)^{\gamma-1} g(s, x(s)) ds. \quad (28)$$

It is necessary to verify that the operator defined in (28) satisfies the conditions of the Banach-Picard contraction theorem. First, we demonstrate that the operator G^* is invariant on the set T .

To this end, we proceed with the following calculation:

$$\begin{aligned}
 |G^*[x(t)] - x(0)| &= \left| \frac{1}{\Gamma(\gamma)} \int_0^t (t-s)^{\gamma-1} g(s, x(s)) ds \right| \\
 &\leq \frac{1}{\Gamma(\gamma)} \int_0^t (t-s)^{\gamma-1} |g(s, x(s))| ds \\
 &\leq \frac{1}{\Gamma(\gamma)} \int_0^t (t-s)^{\gamma-1} M ds \\
 &\leq \frac{M}{\Gamma(\gamma+1)} t^\gamma \\
 &\leq \frac{M}{\Gamma(\gamma+1)} (T^*)^\gamma \\
 &\leq \frac{M}{\Gamma(\gamma+1)} \times \frac{k\Gamma(\gamma+1)}{M}.
 \end{aligned}$$

So, we have:

$$|G^*[x(t)] - x(0)| \leq k. \quad (29)$$

The inequality (29) is also equivalent to

$$x(0) - k \leq G^*[x(t)] \leq x(0) + k, \text{ pour tout } t \in [0; T^*].$$

Therefore, the operator G^* is a defined function from T to itself. Next, we will show that

G^* is a contraction on T .

$$\begin{aligned}
 |G^*[x_1] - G^*[x_2]| &= \left| \frac{1}{\Gamma(\gamma)} \int_0^t (t-s)^{\gamma-1} [g(s; x(s)) - g(s; x_2(s))] ds \right| \\
 &\leq \frac{1}{\Gamma(\gamma)} \int_0^t (t-s)^{\gamma-1} K \|x_1 - x_2\| ds \\
 &\leq \frac{K}{\Gamma(\gamma+1)} \|x_1 - x_2\| t^\gamma \\
 &\leq \frac{K}{\Gamma(\gamma+1)} \|x_1 - x_2\| (T^*)^\gamma \\
 &\leq \frac{K_2}{\Gamma(\gamma+1)} \|x_1 - x_2\| \times \frac{k\Gamma(\gamma+1)}{M}.
 \end{aligned}$$

So

$$|G^*[x_1] - G^*[x_2]| \leq \frac{kK_2}{M} \|x_1 - x_2\|. \quad (30)$$

According to the hypothesis of theorem 1, $K_2k \leq M$ implying that $\frac{K_2k}{M} \leq 1$, we can write that:

$$|G^*[x_1] - G^*[x_2]| \leq \|x_1 - x_2\|, \quad (31)$$

Therefore, T is a contraction. By theorem 2, T has a unique fixed point. Therefore, the problem (26) has a unique solution ○

4.3. Disease-free equilibrium. To determine the disease-free equilibrium of model (6), we set all the equations of the system to zero, then consider $\mathbf{I}_m(t) = \mathbf{E}_v(t) = \mathbf{I}_v(t) = 0$. We therefore obtain:

$$\mathbf{E}_0 = \left(\frac{K}{r} (r - \alpha_1), 0, \frac{b_v}{\alpha_v + \alpha_3}, 0, 0 \right). \quad (32)$$

4.4. Basic reproduction number $\mathbf{R}_0$. We determine the basic reproduction number $\mathbf{R}_0$ of model (6) in order to study the stability of the equilibrium points. This number measures the expected number of secondary infections resulting from the introduction of a single newly infected individual into a susceptible population [4]. We compute the basic reproduction rate $\mathbf{R}_0$ of the system by applying the next-generation operator approach, as defined in [3]; hence, it corresponds to the spectral radius of the next-generation matrix. By using this next-generation matrix method, we obtain:

$$\mathbf{F} = \begin{pmatrix} 0 & 0 & \frac{\beta_1 K (r - \alpha_1)}{Ar + K (r - \alpha_1)} \\ \frac{b\beta_2 b_v}{C (\alpha_v + \alpha_3)} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{ and } \mathbf{V} = \begin{pmatrix} \alpha_2 + \alpha_m + \theta_m & 0 & 0 \\ 0 & k_v + \alpha_v + \alpha_3 & \\ 0 & -k_v & \alpha_v + \alpha_4 \end{pmatrix}.$$

Therefore, the basic reproduction number $\mathbf{R}_0$ is given by:

$$\mathbf{R}_0 = \sqrt{\frac{\beta_1 K (r - \alpha_1) b\beta_2 b_v k_v}{C [Ar + K (r - \alpha_1)] (\alpha_2 + \alpha_m + \theta_m) (\alpha_v + \alpha_3) (\alpha_v + \alpha_4) (k_v + \alpha_v + \alpha_3)}}. \quad (33)$$

$\mathbf{R}_0$ is a threshold parameter that quantifies the average number of new infections (in vectors and maize plants) generated by a cross-interaction between an infected maize plant and an infected leafhopper, in a context where all other plants and insects are susceptible. The spread of MSV in a maize field requires two transmission cycles: first, the virus passes from an infected maize plant to a healthy leafhopper, and then from this newly infected leafhopper to a healthy maize plant. This two-step process explains the presence of a square root in the expression for $\mathbf{R}_0$, as it reflects the succession of these two transmissions necessary for a complete infection to occur in the population. Thus, $\mathbf{R}_0$ can be rewritten as:

$$\mathbf{R}_0 = \mathbf{R}_{0m} \times \mathbf{R}_{0v} \quad (34)$$

where

❶ $\mathbf{R}_{0m} = \sqrt{\frac{\beta_1 K (r - \alpha_1)}{[Ar + K (r - \alpha_1)] (\alpha_2 + \alpha_m + \theta_m)}}$ is the average number of secondary infections produced by a single infected leafhopper in a population of all susceptible maize plants,

❷ $\mathbf{R}_{0v} = \sqrt{\frac{b\beta_2 b_v k_v}{C (\alpha_v + \alpha_3) (\alpha_v + \alpha_4) (k_v + \alpha_v + \alpha_3)}}$ is the average number of secondary infections produced by a single infected maize plant in a population of all susceptible leafhoppers.

4.5. Stability of the disease-free equilibrium.

4.5.1. Local stability of the disease-free equilibrium.

Theorem 7. *The disease-free equilibrium point $\mathbf{E}_0$ is locally and asymptotically stable when $\mathbf{R}_0 < 1$ and unstable when $\mathbf{R}_0 > 1$.*

Proof 4. *To demonstrate this theorem, we determine the Jacobian matrix of the system (6).*

This Jacobian matrix $\mathcal{J}$ of the system (6) evaluated at the equilibrium point $\mathbf{E}_0$ is given by:

$$\mathcal{J}(\mathbf{E}_0) = \begin{pmatrix} \alpha_1 - r & \alpha_1 - r & 0 & 0 & \frac{-\beta_1 K (r - \alpha_1)}{Ar + K (r - \alpha_1)} \\ 0 & -(\alpha_2 + \alpha_m + \theta_m) & 0 & 0 & \frac{\beta_1 K (r - \alpha_1)}{Ar + K (r - \alpha_1)} \\ 0 & \frac{-bb_v \beta_2}{C (\alpha_v + \alpha_3)} & -(\alpha_v + \alpha_3) & 0 & 0 \\ 0 & \frac{bb_v \beta_2}{C (\alpha_v + \alpha_3)} & 0 & -(k_v + \alpha_v + \alpha_3) & 0 \\ 0 & 0 & 0 & k_v & -\alpha_v - \alpha_4 \end{pmatrix}.$$

From the Jacobian matrix at the point $\mathbf{E}_0$, we obtain the characteristic polynomial $P(\lambda)$ defined by:

$$P(\lambda) = (\lambda + r - \alpha_1) (\lambda + \alpha_v + \alpha_3) (\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3), \quad (35)$$

where

$$a_1 = 2\alpha_v + \alpha_2 + \alpha_3 + \alpha_4 + \alpha_m + \theta_m + k_v,$$

$$a_2 = (\alpha_v + \alpha_4) (\alpha_2 + \alpha_m + \theta_m) + (\alpha_v + \alpha_4) (k_v + \alpha_v + \alpha_3) + (\alpha_2 + \alpha_m + \theta_m) (k_v + \alpha_v + \alpha_3), \text{ and}$$

$$a_3 = (\alpha_v + \alpha_4) (\alpha_2 + \alpha_m + \theta_m) (k_v + \alpha_v + \alpha_3) (1 - \mathbf{R}_0^2).$$

Solving $P(\lambda) = 0$, we obtain $\lambda_1 = -(r - \alpha_1) < 0$ and $\lambda_2 = -(\alpha_v + \alpha_3) < 0$, so

$$|Arg(\lambda_i)|_{i \in \{1,2\}} = \pi > \frac{\pi\gamma}{2}, \quad 0 < \gamma < 1.$$

From the third factor of (35), that is to say

$$\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0, \quad (36)$$

we apply the Routh-Hurwitz criteria.

❶ $a_1 = 2\alpha_v + \alpha_2 + \alpha_3 + \alpha_4 + \alpha_m + \theta_m + k_v > 0$ because it is the sum of positive parameters.

❷ If $\mathbf{R}_0 < 1$, $a_3 = (\alpha_v + \alpha_4) (\alpha_2 + \alpha_m + \theta_m) (k_v + \alpha_v + \alpha_3) (1 - \mathbf{R}_0^2) > 0$.

$$\begin{aligned} \textcircled{3} \quad a_1 a_2 - a_3 &= (\alpha_v + \alpha_4 + \alpha_2 + \alpha_m + \theta_m) (\alpha_v + \alpha_4) (\alpha_2 + \alpha_m + \theta_m) + \\ &(\alpha_v + \alpha_4 + \alpha_2 + \alpha_m + \theta_m) [(k_v + \alpha_v + \alpha_3) (\alpha_v + \alpha_4 + \alpha_2 + \alpha_m + \theta_m)] + \\ &(\alpha_v + \alpha_4 + \alpha_2 + \alpha_m + \theta_m) (k_v + \alpha_v + \alpha_3)^2 + \\ &\mathbf{R}_0^2 (\alpha_2 + \alpha_m + \theta_m) (\alpha_v + \alpha_4) (k_v + \alpha_v + \alpha_3) > 0. \end{aligned}$$

Since the Routh–Hurwitz criteria are satisfied, the three roots of equation (36) have strictly negative real parts, that is, $|\text{Arg}(\lambda_i)|_{i \in \{3,4,5\}} = \pi > \frac{\pi\gamma}{2}$, $0 < \gamma < 1$.

In conclusion, the equilibrium point $\mathbf{E}_0$ is locally and asymptotically stable if $\mathbf{R}_0 < 1$. $\quad \bigcirc$

4.5.2. Global Stability of the Disease-Free Equilibrium. To study the global stability of the disease-free equilibrium, we use the approach developed by Castillo-Chavez and Song [2].

To this end, we rewrite model (6) in the following form:

$$\begin{cases} {}^c_0\mathcal{D}_t^\gamma [\mathbf{X}] = F(\mathbf{X}, \mathbf{Y}), \\ {}^c_0\mathcal{D}_t^\gamma [\mathbf{Y}] = G(\mathbf{X}, \mathbf{Y}), \quad G(\mathbf{X}, 0) = 0, \end{cases} \quad (37)$$

where $0 < \gamma < 1$, $\mathbf{X} = (\mathbf{S}_m, \mathbf{S}_v) \in \mathbb{R}^2$ denotes the populations of uninfected individuals, and

$\mathbf{Y} = (\mathbf{I}_m, \mathbf{E}_v, \mathbf{I}_v) \in \mathbb{R}^3$ denotes the population of infected individuals.

$\mathbf{E}_0 = (X^*, 0)$ represents the disease-free equilibrium of system (6). The equilibrium point $\mathbf{E}_0$ is said to be globally asymptotically stable for model (6) if it satisfies the following two conditions:

- i) For ${}^c_0\mathcal{D}_t^\gamma [\mathbf{X}] = F(\mathbf{X}, 0)$, then the equilibrium point $\mathbf{X}^*$ is globally and asymptotically stable,
- ii) ${}^c_0\mathcal{D}_t^\gamma [\mathbf{Y}] = {}^c_0\mathcal{D}_Y^\gamma G(\mathbf{X}^*, 0) \mathbf{Y} - \tilde{G}(\mathbf{X}, \mathbf{Y})$, $\tilde{G}(\mathbf{X}, \mathbf{Y}) \geq 0$ for all $(\mathbf{X}, \mathbf{Y}) \in \Omega$,

where ${}^c_0\mathcal{D}_Y^\gamma G(\mathbf{X}^*, 0)$ is an (M)-matrix, that is, a matrix whose off-diagonal elements are non-negative (positive). Moreover, this matrix is the Jacobian matrix of $G(\mathbf{X}, \mathbf{Y})$ evaluated at $(\mathbf{X}^*, 0)$. If system (37) satisfies the two conditions above, then the following theorem holds.

Theorem 8. The equilibrium point $\mathbf{E}_0 = (\mathbf{X}_0, 0)$ of system (37) is globally and asymptotically stable if $\mathbf{R}_0 \leq 1$ and if both conditions (i) and (ii) are satisfied.

Proof 5. For the model (6), we have two functions $F(\mathbf{X}, \mathbf{Y})$ and $G(\mathbf{X}, \mathbf{Y})$ which are data respectively defined by:

$$F(\mathbf{X}, \mathbf{Y}) = \begin{pmatrix} r\mathbf{S}_m(t) \left(1 - \frac{\mathbf{S}_m(t) + \mathbf{I}_m(t)}{K}\right) - \alpha_1 \mathbf{S}_m(t) - \beta_1 \frac{\mathbf{I}_v(t) \mathbf{S}_m(t)}{A + \mathbf{S}_m(t)} \\ b_v - (\alpha_v + \alpha_3) \mathbf{S}_v(t) - b\beta_2 \frac{\mathbf{S}_v(t) \mathbf{I}_m(t)}{C + \mathbf{I}_m(t)} \end{pmatrix} \quad (38)$$

and

$$G(\mathbf{X}, \mathbf{Y}) = \begin{pmatrix} \beta_1 \frac{\mathbf{I}_v(t) \mathbf{S}_m(t)}{A + \mathbf{S}_m(t)} - (\alpha_2 + \alpha_m + \theta_m) \mathbf{I}_m(t) \\ b\beta_2 \frac{\mathbf{S}_v(t) \mathbf{I}_m(t)}{C + \mathbf{I}_m(t)} - (k_v + \alpha_v + \alpha_3) \mathbf{E}_v(t) \\ k_v \mathbf{E}_v(t) - (\alpha_v + \alpha_4) \mathbf{I}_v(t) \end{pmatrix} \quad (39)$$

The condition (i) implies that ${}^c_0\mathcal{D}_t^\gamma[\mathbf{X}] = F(\mathbf{X}, 0)$. Which implies that

$${}^c_0\mathcal{D}_t^\gamma[\mathbf{S}_m(t)] = r\mathbf{S}_m(t) \left(1 - \frac{\mathbf{S}_m(t)}{K}\right) - \alpha_1 \mathbf{S}_m(t) \quad (40a)$$

$${}^c_0\mathcal{D}_t^\gamma[\mathbf{S}_v(t)] = b_v - (\alpha_v + \alpha_3) \mathbf{S}_v(t) \quad (40b)$$

$\mathbf{X}^* = \left(\frac{K}{r}(r - \alpha_1), 0, \frac{b_v}{\alpha_v + \alpha_3}, 0\right)$ is an equilibrium point that is globally and asymptotically stable for the system (40). Indeed, solving equation (40b) gives:

$$\mathbf{S}_v(t) = \frac{b_v}{\alpha_v + \alpha_3} + \left(\mathbf{S}_v(0) - \frac{b_v}{\alpha_v + \alpha_3}\right) \mathbf{E}_\gamma[-(\alpha_v + \alpha_3)t^\gamma]. \quad (41)$$

Taking the limit, we obtain:

$$\lim_{t \rightarrow +\infty} \mathbf{S}_v(t) = \frac{b_v}{\alpha_v + \alpha_3}. \quad (42)$$

For the system (40a), let's choose a Lyapunov function $L(\mathbf{S}_m(t))$ defined by

$$L(\mathbf{S}_m(t)) = (\mathbf{S}_m(t) - \mathbf{S}_m^*)^2, \mathbf{S}_m^* = \frac{K}{r}(r - \alpha_1).$$

L is a positive and zero function if and only if $\mathbf{S}_m(t) = \mathbf{S}_m^*$.

Using the inequalities for fractional derivatives of convex functions, we have:

$${}^c_0\mathcal{D}_t^\gamma[L(\mathbf{S}_m(t))] = -\frac{2r}{K} \mathbf{S}_m(t) (\mathbf{S}_m(t) - \mathbf{S}_m^*)^2.$$

So ${}^c_0\mathcal{D}_t^\gamma[L(\mathbf{S}_m(t))] \leq 0$, $L(\mathbf{S}_m(t))$ is a decreasing function. Moreover, $L(\mathbf{S}_m(t))$ is bounded below by 0, therefore it is convergent. By the Lyapunov stability principle for fractional-order systems, $(\mathbf{S}_m(t))$ converges globally to $(\mathbf{S}_m^*(t))$. Consequently, the solution of the reduced system (40) converges globally in the domain Ω .

From the equality (39), we have:

$${}^c_0\mathcal{D}_Y^\gamma G(\mathbf{X}^*, 0) = \begin{pmatrix} -(\alpha_2 + \alpha_m + \theta_m) & 0 & \frac{\beta_1 K(r - \alpha_1)}{Ar + K(r - \alpha_1)} \\ \frac{b\beta_2 b_v}{C(\alpha_v + \alpha_3)} & -(k_v + \alpha_v + \alpha_3) & 0 \\ 0 & k_v & -(\alpha_v + \alpha_4) \end{pmatrix}$$

and

$$\tilde{G}(\mathbf{X}, \mathbf{Y}) = \begin{pmatrix} \beta_1 \mathbf{I}_v \left(\frac{K(r - \alpha_1)}{Ar + K(r - \alpha_1)} - \frac{\mathbf{S}_m}{A + \mathbf{S}_m} \right) \\ b\beta_2 \mathbf{I}_m \left(\frac{b_v}{C(\alpha_v + \alpha_3)} - \frac{\mathbf{S}_v}{C + \mathbf{I}_m} \right) \\ 0 \end{pmatrix}.$$

Since $\frac{b_v}{\alpha_v + \alpha_3} \geq \mathbf{S}_v$ and $\frac{K}{r}(r - \alpha_1) \geq \mathbf{S}_m$, then it is evident that $\tilde{G}(\mathbf{X}, \mathbf{Y}) \geq 0$ for all $(\mathbf{X}, \mathbf{Y}) \in \Omega$, therefore $\mathbf{E}_0$ is globally and asymptotically stable when $\mathbf{R}_0 \leq 1$ $\bigcirc$

4.6. Sensitivity analysis of the model parameters. We proceed with a sensitivity analysis of the different parameters of $\mathbf{R}_0$ to identify those that most strongly influence the basic reproduction number. This analysis allows us to evaluate the relative importance of each parameter in the transmission process of maize disease. To this end, we adopt the methodology proposed in [17], which was also used in [18]. This approach leads to the establishment of a formula for calculating the sensitivity index associated with each parameter of $\mathbf{R}_0$. This formula is given by the following definition:

Definition 7. The normalized sensitivity index (or forward sensitivity index) of a variable $\mathbf{R}_0$, which depends differentiably on a parameter q , is defined as:

$$\Lambda_q^{\mathbf{R}_0} = \frac{\partial \mathbf{R}_0}{\partial q} \times \frac{q}{\mathbf{R}_0}, \quad (43)$$

where q represents a parameter of $\mathbf{R}_0$.

By applying the same procedure to the other parameters of $\mathbf{R}_0$, we obtain table 3 and 4, which summarizes the sensitivity indices of the basic reproduction number.

Parameter symbol	Value day ⁻¹	Sensitivity index	Source
K	10000	+0.0001	[38]
A	0.4	-0.0001	[38]
θ_m	0.008	-0.04	[38]
r	0.0005	$+0003 \times 10^{-7}$	[38]
α_m	0.08	-0.41	[38]
β_1	0.45	+0.5	[19]
α_2	0.008	-0.04	Assumed
α_1	0.0004	-0.0003	Assumed

TABLE 3. Maize parameters, their values and their sensitivity index.

Parameter symbol	Value day ⁻¹	Sensitivity index	Source
b_v	0.02	+0.5	[38]
b	0.45	+0.5	[38]
k_v	0.8	+0.19	[38]
C	0.6	-0.5	[38]
α_4	0.0303	-0.02	[20]
α_3	0.0303	-0.03	[20]
β_2	0.001	+0.5	Assumed
α_v	0.5	-1.13	Assumed

TABLE 4. Leafhopper parameters, their values and their sensitivity index.

4.7. Interpretation of sensitivity index. The parameters with positive sensitivity index ($\beta_1, \beta_2, b, b_v, k_v, K$) colored blue in table 3 and 4 indicate that an increase in their values promotes the spread of the disease within maize plants. Indeed, the resulting increase in the basic reproduction number implies an increase in the average number of secondary cases of infection in maize plants. Conversely, the parameters with negative sensitivity indices ($\alpha_1, \alpha_2, \alpha_3, \alpha_4, \alpha_v, \alpha_m, \theta_m, r, C, A$) colored green in table 3 and 4 contribute to reducing disease transmission when their values increase. The sensitivity analysis of $\mathbf{R}_0$ for Maize Streak Virus (MSV) indicates that certain parameters significantly influence disease spread in maize and leafhopper populations. The sensitivity analysis of $\mathbf{R}_0$ for Maize Streak Virus (MSV) indicates that certain parameters significantly influence disease spread in maize and leafhopper populations. Parameters with positive sensitivity index such as b, b_v, β_1 and β_2 contribute to increasing $\mathbf{R}_0$. In contrast, the mortality rate of leafhoppers due to predators α_v , with a negative sensitivity index, tends to decrease $\mathbf{R}_0$. These results highlight targeted control strategies to limit MSV spread: reducing interactions between infected leafhoppers and susceptible maize plants, controlling the recruitment and reproduction of susceptible leafhoppers, and enhancing the impact of natural predators to increase leafhopper mortality. By mitigating the effect of positively contributing parameters while strengthening negatively contributing ones, the propagation of MSV in maize and leafhopper populations can be effectively controlled.

5. NUMERICAL RESULTS OF THE MODEL

In this section, numerical simulations of model (6) are carried out to illustrate the analytical results. We determine the numerical scheme for the model using the iterative Euler method [21–23]. Considering the last equation of system (7) and setting $t = t_{n+1}$, with $n \in \mathbb{N}$, we obtain:

$$\mathbf{I}_v(t_{n+1}) = \mathbf{I}_v(0) + \frac{1}{\Gamma(\gamma)} \int_0^{t_{n+1}} (t_{n+1} - u)^{\gamma-1} (k_v \mathbf{E}_v(u) - (\alpha_v + \alpha_4) \mathbf{I}_v(u)) du \quad (44)$$

Consider $\Delta t = \frac{T}{n_T}$, $n_T \in \mathbb{N}$ the time discretization step, $t_h = h\Delta t$ and

$$f_5(s, u(s))_{[t_h, t_{h+1}]} \approx f_5(t_h, u(t_h)), \quad u(t) = (\mathbf{S}_m(t), \mathbf{I}_m(t), \mathbf{S}_v(t), \mathbf{E}_v(t), \mathbf{I}_v(t)) \quad (45)$$

with $f_5(t, u(t)) = k_v \mathbf{E}_v(t) - (\alpha_v + \alpha_4) \mathbf{I}_v(t)$. Applying (45) to (44), we obtain:

$$\mathbf{I}_v(t_{n+1}) = \mathbf{I}_m(0) + \frac{\Delta t^\gamma}{\Gamma(\gamma+1)} \sum_{h=0}^n [(n-h+1)^\gamma - (n-h)^\gamma] (k_v \mathbf{E}_v(t_h) - (\alpha_v + \alpha_4) \mathbf{I}_v(t_h)). \quad (46)$$

By proceeding in the same way to all the equations of the system (6), we obtain the following approximate system (47).

$$\left\{ \begin{array}{l} \mathbf{S}_m(t_{n+1}) = \mathbf{S}_m(0) + \frac{\Delta t^\gamma}{\Gamma(\gamma+1)} \sum_{h=0}^n [(n-h+1)^\gamma - (n-h)^\gamma] \\ \quad \times \left(r \mathbf{S}_m(t_h) \left(1 - \frac{\mathbf{S}_m(t_h) + \mathbf{I}_m(t_h)}{K} \right) - \alpha_1 \mathbf{S}_m(t_h) - \beta_1 \frac{\mathbf{I}_v(t_h) \mathbf{S}_m(t_h)}{A + \mathbf{S}_m(t_h)} \right) \\ \mathbf{I}_m(t_{n+1}) = \mathbf{I}_m(0) + \frac{\Delta t^\gamma}{\Gamma(\gamma+1)} \sum_{h=0}^n [(n-h+1)^\gamma - (n-h)^\gamma] \\ \quad \times \left(\beta_1 \frac{\mathbf{I}_v(t_h) \mathbf{S}_m(t_h)}{A + \mathbf{S}_m(t_h)} - (\alpha_2 + \alpha_m + \theta_m) \mathbf{I}_m(t_h) \right) \\ \mathbf{S}_v(t_{n+1}) = \mathbf{S}_v(0) + \frac{\Delta t^\gamma}{\Gamma(\gamma+1)} \sum_{h=0}^n [(n-h+1)^\gamma - (n-h)^\gamma] \\ \quad \times \left(b_v - (\alpha_v + \alpha_3) \mathbf{S}_v(t_h) - b\beta_2 \frac{\mathbf{S}_v(t_h) \mathbf{I}_m(t_h)}{C + \mathbf{I}_m(t_h)} \right) \\ \mathbf{E}_v(t_{n+1}) = \mathbf{E}_v(0) + \frac{\Delta t^\gamma}{\Gamma(\gamma+1)} \sum_{h=0}^n [(n-h+1)^\gamma - (n-h)^\gamma] \\ \quad \times \left(b\beta_2 \frac{\mathbf{S}_v(t_h) \mathbf{I}_m(t_h)}{C + \mathbf{I}_m(t_h)} - (k_v + \alpha_v + \alpha_3) \mathbf{E}_v(t_h) \right) \\ \mathbf{I}_v(t_{n+1}) = \mathbf{I}_m(0) + \frac{\Delta t^\gamma}{\Gamma(\gamma+1)} \sum_{h=0}^n [(n-h+1)^\gamma - (n-h)^\gamma] (k_v \mathbf{E}_v(t_h) - (\alpha_v + \alpha_4) \mathbf{I}_v(t_h)) \end{array} \right. \quad (47)$$

The numerical results were obtained from an implementation carried out using Python.

The figures below show the approximate solutions of model (47) based on the Euler method for different values of γ . The parameter values, presented in table 2, are either drawn from the literature or assumed to be biologically realistic [47]. Numerical simulations were performed using the initial conditions $\mathbf{S}_m(0) = 1000$; $\mathbf{I}_m(0) = 20$; $\mathbf{S}_v(0) = 100$; $\mathbf{E}_v(0) = 10$ and $\mathbf{I}_v(0) = 0$, taking $T = 60$ days as the final time.

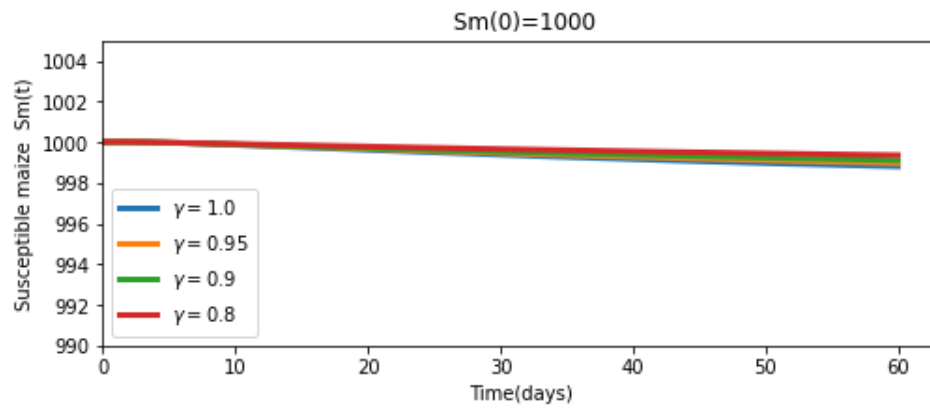


FIGURE 2. Dynamics of the susceptible maize when $\gamma \in \{1, 0.95, 0.80, 0.60\}$.

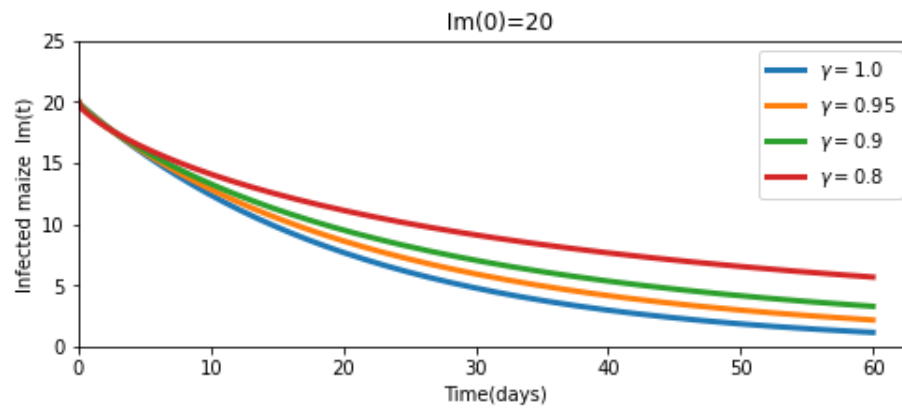


FIGURE 3. Dynamics of the infectious maize when $\gamma \in \{1, 0.95, 0.80, 0.60\}$

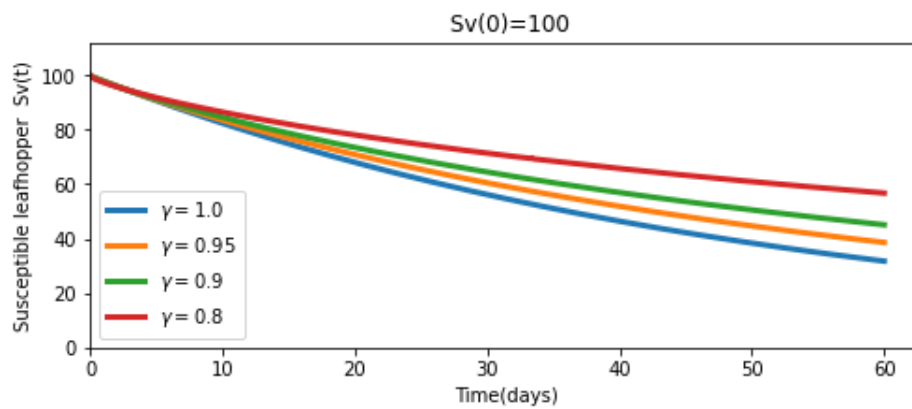


FIGURE 4. Dynamics of the susceptible leafhopper when $\gamma \in \{1, 0.95, 0.80, 0.60\}$

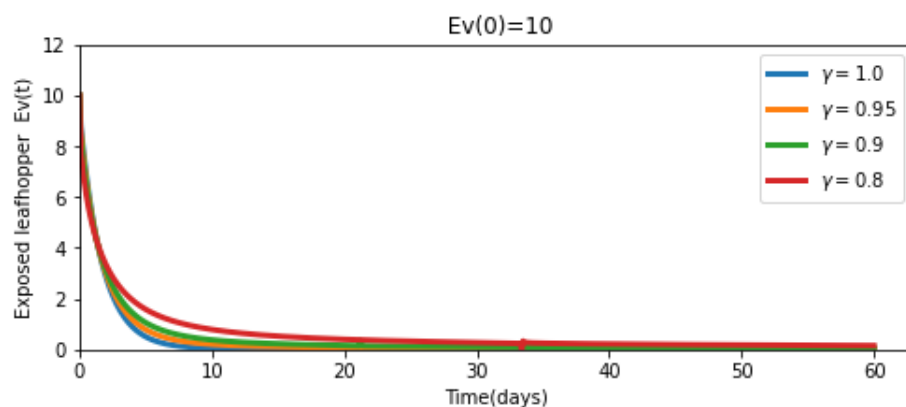


FIGURE 5. Dynamics of the exposed leafhopper when $\gamma \in \{1, 0.95, 0.80, 0.60\}$

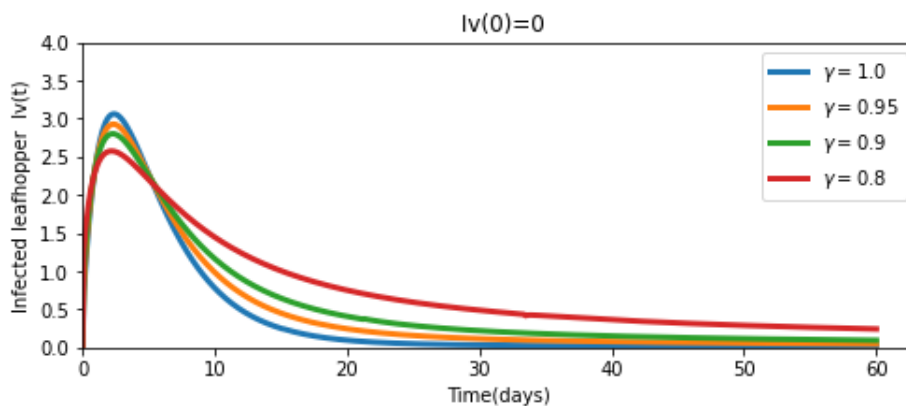


FIGURE 6. Dynamics of the infectious leafhopper when $\gamma \in \{1, 0.95, 0.80, 0.60\}$

The figures 2, 3, 4, 5, and 6 illustrate, respectively, the dynamics of susceptible maize, infectious maize, susceptible leafhoppers, exposed leafhoppers, and infectious leafhoppers for various values of the fractional order γ . In the susceptible maize compartment, we observe a decrease in the number of individuals as the fractional order increases, which becomes particularly noticeable when $\gamma = 1$. In the compartments of infectious maize, susceptible leafhoppers, exposed leafhoppers, and infectious leafhoppers, the number of individuals also decreases as γ varies. For $\gamma = 1$, corresponding to the classical (non-fractional) model, the decline of the infected populations is faster, and the curves approach the horizontal axis more clearly. Biologically, this indicates that the disease will disappear more rapidly in the maize and leafhopper populations. The results indicate that the fractional-order model, by incorporating long-term memory effects, describes the transmission dynamics of MSD in maize more accurately than the classical deterministic model. Indeed, variations in the fractional order γ produce distinct trajectories, providing a finer representation of MSD spread and a more precise evaluation of control strategies.

6. CONCLUSION

In this paper, we developed a fractional-order epidemiological model in the sense of Caputo to study the transmission dynamics of Maize Streak Disease (MSD), caused by the Maize streak virus (MSV). The mathematical analysis demonstrated that the model is well-posed from both epidemiological and theoretical perspectives. We then determined the basic reproduction number R_0 and the disease-free equilibrium, for which local stability was investigated. A sensitivity analysis of the parameters of R_0 was performed, highlighting the parameters that have the greatest influence on the spread of the disease. Finally, numerical simulations of the fractional-order model were performed for different values of the fractional order, confirming the analytical results and showing that the disease dies out when $R_0 < 1$, while it persists when $R_0 > 1$.

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