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Research Article

Ecoepidemiological Model and Analysis of MSV Disease Transmission Dynamics in Maize Plant

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In this paper, an ecoepidemiological deterministic model for the transmission dynamics of maize streak virus (MSV) disease in maize plant is proposed and analysed qualitatively using the stability theory of differential equations.The basic reproduction number with respect to the MSV free equilibrium is obtained using next generation matrix approach. The conditions for local and global asymptotic stability of MSV free and endemic equilibria are established. The model exhibits forward bifurcation and the sensitivity indices of various embedded parameters with respect to the MSV eradication or spreading are determined. Numerical simulation is performed and dispalyed graphically to justify the analytical results.

1. Introduction

Maize (*Zea mays L.*) is grown globally across temperate and tropical zones, spanning all continents [1]. It is the most widely grown and consumed staple crop in Africa with more than 100 million Africans depending on it as their main food source which is annually planted over an area of 15.5 million hectares [2, 3]. In Ethiopia, maize is the most cereal gown which ranks first in yield per hectare and is grown in all 11 administrative regions [4]. It is the primary food, averaging slightly more than 20% of daily caloric intake. About 9 million smallholder farmers grow maize and 75% of the maize produced is consumed as food [5, 6]. The reports of Ethiopian Commodity Exchange show that three-fourth of maize produced is used for household expenditure; only about ten percent is marketed and the remaining is used for seed, in-kind expenses for labor and animal feed [6].

Maize production is constrained by many abiotic and biotic factors of which maize streak disease (MSD) is the major biotic threat in Ethiopia. It is the most destructive and devastating disease of maize in Sub-Saharan Africa which is caused by maize streak virus (MSV; genus *Mastrevirus*, family *Geminiviridae*) [3, 7] including Ethiopia [4]. MSV is a major constraint to maize and over 80 other crops species [8] including oats, wheat, sorghum, millet, finger millet, and sugarcane [2, 9]. MSD is a major threat to cereal crops amongst smallholder farmers in Sub-Saharan Africa causing up to US $ 480 million losses annually [10]. MSD is a viral disease which has single-component, circular, ssDNA [2, 3].

MSV has been reported to be the most economically significant causing 100% yield loss if infection occurs in the first three weeks of planting maize [3, 11]. It is irregular in nature and transmitted in a persistent manner by leafhoppers in the genus *Cicadulina* [2, 5]. Globally, 22 species of Cicadulina leafhoppers have been reported, of which 18 are found in Africa. *Cicadulina mbila* is the most predominant vector and the most important in the epidemiology of the virus [2] from the 8 known vectors of MSV in the genus. In Ethiopia, five of these 22 known species of *Cicadulina* have been recorded [4].

Mathematical modeling is an important tool used in analyzing the dynamics of infectious diseases. Several models have been formulated and analyzed to explain the dynamics of plant disease transmission. The authors in [12] investigated the impacts of foliar diseases on maize plant population

Table 1: Description of parameters of the MSV model (1).

parameter	Description
β_1	Predation and infection rate of Infected Leafhopper on Susceptible Maize plant
β_2	Predation and infection rate of Susceptible Leafhopper on Infected Maize plant
b	Conversion rate of Infected Leafhopper
q	Recruitment rate of Susceptible Leafhopper
K	Carrying capacity
C	Half saturation rate of Susceptible Leafhopper with Infected Maize plant
A	Half saturation rate of Susceptible maize with Infected plant
μ_1	Death rate of infected maize
μ_2	Death rate of susceptible leafhopper
μ_3	Death rate of infected leafhopper
r	Intrinsic growth rate of Maize

dynamics from the developed epidemiological mathematical model. They also applied optimal control theory with chemical, cultural, and disease resistance as a control intervention. In [13], the authors derived a continuous epidemiological model of African cassava mosaic virus disease in which the dynamics are within a locality, of healthy and infected cassava and of infective and noninfective whitefly vectors. The authors in [14] reviewed a differential equation model on plants by adjusting to a specific plant disease model which was a general model of [15]. Optimal control theory to a continuous deterministic epidemiological Cassava brown streak disease model with chemical and uprooting as control measures was applied by the author [16]. In [17], the authors proposed and analyzed an SI-SEI maize disease model which is a combination of both the host and the vector population models and has been formulated to study and analyse the dynamics of maize lethal necrosis disease.

Motivated by references [12, 16, 17], in this paper, we present a deterministic model to study and analyze the dynamics of MSV in the maize plant population. We believe that the results of our research work will be useful indicating suitable means of controlling the disease transmission or rather eradicate it. This may ensure maximum maize harvest for farmers for food security. The organization of this paper is as follows. In Section 2, the mathematical analysis of the model including determining the invariant region where the model is mathematically and epidemiologically well posed is presented; basic reproduction number $\mathfrak{R}_0$ is obtained by using the next generation matrix method; the local stability of the equilibria is obtained by the Jacobian matrix method; and, by the method of Castillo- Chavez, the global stability of the disease-free equilibrium is investigated; we determine the endemic equilibrium point of the model and the local stability of the equilibria by the Jacobian matrix method; and, by the method of constructing a Lyapunov function, the global stability of the endemic equilibrium is investigated. At the end of Section 3, we determine the bifurcation and sensitivity analysis. In Section 4, we give some numerical simulations to prove our theoretical results with a brief discussion. In the final section, there is a conclusion.

2. Model Description and Formulation

The model we introduce consists of two populations: the maize population and the leafhopper vector population. Both populations have two subclasses: susceptible and infected. At time t, let S(t) denote the density of the susceptible maize and I(t) denote the density of the infected maize. The susceptible and infected leafhopper vector densities are denoted by H(t) and Y(t), respectively.

If there is no leafhopper predator population and no disease, the host population grows logistically with intrinsic growth rate r and environmental carrying capacity $K(K > 0)$. In the presence of the disease in maize population, the infected host population contributes to the susceptible host population growth towards the carrying capacity $K(K > 0)$. The susceptible leafhopper vectors are recruited at rate q and move to infected leafhopper subgroup by eating infected maize plant at a rate β_2 and its natural death rate is μ_2. The infected leafhopper has a natural death rate μ_3. The disease spreads to the susceptible host when it comes in contact with the MSV pathogen infected leafhopper vector. Susceptible host plants move to the infected class following contact with infected leafhopper at a per capita rate β_1. The host, once became infected, never recovers and gives no or very low yield of maize. The infected maize plants have death rate μ_1 due to the disease. Furthermore, the disease can not be transmitted horizontally and vertically in both populations and it is not genetically inherited. The predation functional response of the leafhopper towards susceptible maize is assumed to follow Michaelis-Menten kinetics and is modelled using a Holling type II [18] functional form with predation and infection coefficients β_1, β_2 and half saturation constants A and C. All the parameters and their descriptions are listed in Table 1.

With regard to the above considerations, we have the compartmental flow diagram shown in Figure 1. From the flow chart (Figure 1), the model will be governed by the following system of differential equation equations:

$$\frac{dS}{dt} = rS\left(1 - \frac{S+I}{K}\right) - \frac{\beta_1 SY}{A+S}$$

$$\frac{dI}{dt} = \frac{\beta_1 SY}{A+S} - \mu_1 I$$

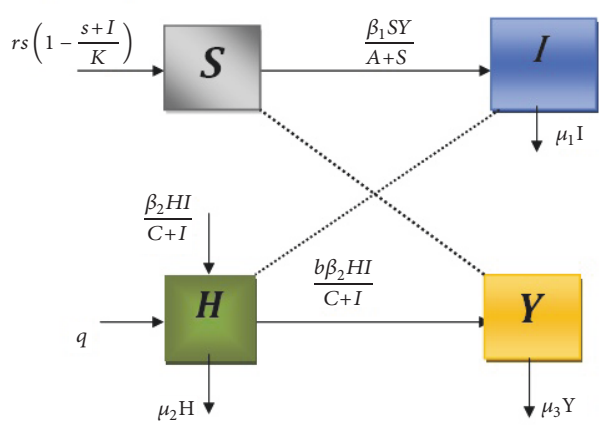

Figure 1: Compartmental diagram for the transmission dynamics of MSV.

$$\frac{dH}{dt} = q - \frac{\beta_2 IH}{C+I} - \mu_2 H$$
$$\frac{dY}{dt} = \frac{b\beta_2 IH}{C+I} - \mu_3 Y \tag{1}$$

With the initial condition

$$S(0) = S_0 \geq 0,$$
$$I(0) = I_0 \geq 0,$$
$$H(0) = H_0 \geq 0, \tag{2}$$
$$Y(0) = Y_0 \geq 0$$

3. Model Analysis

3.1. Positivity of Solutions. For model (1) to be ecoepidemiologically meaningful and well posed, it is necessary to prove that all solutions of system with positive initial data will remain positive for all times $t > 0$. This will be established by the following theorem.

Theorem 1. *Let* $\Omega = \{(S, I, H, Y) \in \Re^4 : S(0) > 0, I(0) > 0, H(0) > 0, Y(0) > 0\}$. *Then the solution set* $(S(t), I(t), H(t), Y(t))$ *of system (1) is positive for all* $t \geq 0$.

Proof. From the first equation of the system

$$\frac{dS}{dt} = rS\left(1 - \frac{S+I}{K}\right) - \frac{\beta_1 SY}{A+S} \leq rS\left(1 - \frac{S}{K}\right) \tag{3}$$

Then we have

$$\frac{dS}{S(1-S/K)} \leq rdt \Longrightarrow$$
$$S(t) \leq \frac{KS(0)}{e^{-rt}(K - S(0)) + S(0)} \tag{4}$$

As t approachers ∞, we obtain $0 \leq S(t) \leq K$. By using the same procedure, we obtained

$$I(t) \geq I(0)\, e^{-\mu_1 t} \geq 0,$$
$$H(t) \geq H(0)\, e^{-\mu_2 t} \geq 0, \tag{5}$$
$$Y(t) \geq Y(0)\, e^{-\mu_3 t} \geq 0$$

Thus the model is meaningful and well posed. Therefore, it is sufficient to study the dynamics of the model in Ω. □

3.2. Invariant Region. Let us determine a region in which the solution of model(1) is bounded. For this model the total maize population is $N_1(S, I) = S(t) + I(t)$. Then, after differentiating N_1 with respect to time and substituting the expression of $dS/dt, dI/dt$, we obtain

$$\frac{dN_1}{dt} = rS\left(1 - \frac{S+I}{K}\right) - \mu_1 I \leq rS - \mu_1 I$$
$$= S(r+1) - (S + \mu_1 I) \leq \widehat{k}(r+1) - \alpha N_1 \tag{6}$$

where $\widehat{k} = \max\{S(0), K\}$ and $\alpha = \min\{1, \mu_1\}$. Then

$$\frac{dN_1}{dt} + dN_1 \leq \widehat{k}(r+1) \tag{7}$$

After solving Eq. (7) and evaluating it as $t \longrightarrow \infty$, we got

$$\Omega_h = \left\{(S, I)\, \varepsilon R_+^2 : N_1(t) \leq \frac{\widehat{k}}{\alpha}(r+1)\right\} \tag{8}$$

Similarly, for leafhopper population $N_2(H, Y) = H(t) + Y(t)$, we get

$$\frac{dN_2}{dt} = q - \mu_2 H - \mu_3 Y \leq q - mN_2 \tag{9}$$

Where $m = \min(\mu_1, \mu_2)$. Then

$$\frac{dN_2}{dt} + mN_2 \leq q \tag{10}$$

After solving Eq. (10) and evaluating it as $t \longrightarrow \infty$, we got

$$\Omega_v = \left\{(H, Y)\, \varepsilon R_+^2 : N_2(t) \leq \frac{q}{m}\right\} \tag{11}$$

Therefore the feasible solution set for the MSV model given by

$$\Omega = \Omega_h \times \Omega_v = \left\{ (S, I, H, Y) \in R_+^4 : N_1(t) \leq \frac{\widehat{k}}{\alpha}(r+1); N_2(t) \leq \frac{q}{m} \right\} \tag{12}$$

is positively invariant, inside which the model is considered to be epidemiologically meaningful and mathematically well posed.

3.3. Disease-Free Equilibrium Point (DFE). The disease-free equilibrium of model (1) is obtained by equating all equations of the model to zero and then letting $I = 0$ and $Y = 0$. Then we get

$$E_0 = \left(K, 0, \frac{q}{\mu_2}, 0 \right) \tag{13}$$

3.4. Basic Reproduction Number. We compute the basic reproduction number $\mathfrak{R}_0$ for the model, to analyze the stability of the equilibrium points. The basic reproduction number, $\mathfrak{R}_0$, measures the expected number of secondary infections that result from one newly infected individual introduced into a susceptible population [16]. We calculate the basic reproduction number $\mathfrak{R}_0$ of the system by applying the next generation operator approach as laid out by [19] and so it is the spectral radius of the next-generation matrix. The first step to get $\mathfrak{R}_0$ is rewriting the model equations starting with newly infective classes:

$$\begin{aligned} \frac{dI}{dt} &= \frac{\beta_1 SY}{A+S} - \mu_1 I \\ \frac{dY}{dt} &= \frac{b\beta_2 IH}{C+I} - \mu_3 Y \end{aligned} \tag{14}$$

Then, by the principle of next-generation matrix, we obtained

$$F = \begin{bmatrix} \dfrac{\beta_1 SY}{A+S} \\ \dfrac{b\beta_2 IH}{C+I} \end{bmatrix}, \qquad V = \begin{bmatrix} \mu_1 I \\ \mu_3 Y \end{bmatrix} \tag{15}$$

Therefore, the basic reproduction number is given as

$$\mathfrak{R}_0 = \sqrt{\frac{b\beta_1\beta_2 Kq}{\mu_1\mu_2\mu_3 C(A+K)}} \tag{16}$$

$\mathfrak{R}_0$ is a threshold parameter that represents the average number of infected vectors and infected hosts caused by a cross-infection of one infectious maize plant and one infectious leafhopper vector when the other population consists of only susceptible population [19]. Two generations are required for transmission of MSV to take place in the maize field; that is why the square root is found in $\mathfrak{R}_0$, that is, from an infectious maize plant to a susceptible leafhopper vector and then from an infectious leafhopper vector to susceptible maize [16]. It is too clear when $\mathfrak{R}_0$ is rewritten as follows:

$$\mathfrak{R}_0 = \sqrt{\frac{\beta_1 K}{\mu_1(A+K)} \frac{b\beta_2 q}{C\mu_2\mu_3}} = \mathfrak{R}_{0h} \times \mathfrak{R}_{0v} \tag{17}$$

Where

(i) $\mathfrak{R}_{0h} = \sqrt{\beta_1 K/\mu_1(A+K)}$ is the maize plants contribution when they infect the leafhopper, and

(ii) $\mathfrak{R}_{0v} = \sqrt{b\beta_2 q/C\mu_2\mu_3}$ is the contribution of the leafhopper population when it infects maize plants.

3.5. Local Stability of DFE

Theorem 2. *The DFE point is locally asymptotically stable if* $\mathfrak{R}_0 < 1$ *and unstable if* $\mathfrak{R}_0 > 1$.

Proof. To proof this theorem let us first find the Jacobian matrix of system (1):

$$J = \begin{pmatrix} r\left(1 - \dfrac{2S+I}{K}\right) - \dfrac{\beta_1 AY}{(A+S)^2} & -\dfrac{rS}{K} & 0 & -\dfrac{\beta_1 S}{A+S} \\ \dfrac{\beta_1 AY}{(A+S)^2} & -\mu_1 & 0 & -\dfrac{\beta_1 S}{A+S} \\ 0 & -\dfrac{C\beta_2 H}{(C+I)^2} & -\dfrac{\beta_2 I}{(C+I)} - \mu_2 & 0 \\ 0 & \dfrac{bC\beta_2 H}{(C+I)^2} & \dfrac{b\beta_2 I}{(C+I)} & -\mu_3 \end{pmatrix} \tag{18}$$

Evaluating Eq. (18) at the disease-free equilibrium $E_0 = (K, 0, q/\mu_2, 0)$, we get

$$J = \begin{pmatrix} -r & -r & 0 & -\dfrac{\beta_1 K}{A+K} \\ 0 & -\mu_1 & 0 & \dfrac{\beta_1 K}{A+K} \\ 0 & -\dfrac{\beta_2 q}{C\mu_2} & -\mu_2 & 0 \\ 0 & \dfrac{b\beta_2 q}{C\mu_2} & 0 & -\mu_3 \end{pmatrix} \tag{19}$$

From the Jacobian matrix we obtained a characteristic polynomial as

$$(-\lambda - r)\left(-\lambda - \mu_2\right)\left(\lambda^2 + a_1\lambda + a_2\right) = 0 \tag{20}$$

Where

$$\begin{aligned} a_1 &= \mu_1 + \mu_3 \\ a_2 &= \mu_1\mu_3 - \frac{b\beta_1\beta_2 Kq}{\mu_2 C\left(A+K\right)} \end{aligned} \tag{21}$$

From Eq. (20), we see that

$$\begin{aligned} -\lambda - r &\Longrightarrow \lambda_1 = -r < 0, \\ -\lambda - \mu_2 &\Longrightarrow \lambda_2 = -\mu_2 < 0 \end{aligned} \tag{22}$$

From the last expression, that is

$$\lambda^2 + a_1\lambda + a_2 = 0 \tag{23}$$

we applied Routh-Hurwitz criteria, and by the principle Eq. (23) has strictly negative real root if $a_1 > 0$ and $a_2 > 0$. Clearly we see that $a_1 > 0$ because it is the sum of positive parameters and also

$$a_2 = \mu_1\mu_3 - \frac{b\beta_1\beta_2 Kq}{\mu_2 C\left(A+K\right)} = 1 - \mathfrak{R}_0^2 > 0 \tag{24}$$

Hence the DFE is locally asymptotically stable if $\mathfrak{R}_0 < 1$. □

3.6. Global Stability of DFE. To investigate the global stability of DFE, we used technique implemented by Castillo-Chavez and Song [20] as done in the paper [16]. Thus we rewrite our model (1) in the form

$$\begin{aligned} \frac{dX}{dt} &= F\left(X, Z\right), \\ \frac{dZ}{dt} &= G\left(X, Z\right), \\ G\left(X, 0\right) &= 0 \end{aligned} \tag{25}$$

where $X = (S, H) \in R^2$ denotes uninfected populations and $Z = (I, Y) \in R^2$ denotes the infected population. $E_0 = (X^*, 0)$ represents the disease-free equilibrium of this system. E_0 is a globally asymptotically stable equilibrium for the model if it satisfies conditions (i) and (ii) below:

(i) For $dX/dt = F(X, 0), X^*$ is globally asymptotically stable.

(ii) $dZ/dt = D_Z G(X^*, 0)Z - \widehat{G}(X, Z), \widehat{G}(X, Z) \geq 0$ *for all* $(X, Z) \in \Omega$

where $D_Z G(X^*, 0)$ is an M-matrix (the off diagonal elements are nonnegative) and is also the Jacobian of G(X,Z) taken in (I, Y) and evaluated at $(X^*, 0) = (K, q/\mu_2, 0, 0)$. If system (25) satisfies the above conditions, then the following theorem holds.

Theorem 3. *The equilibrium point $E_0 = (X^*, 0)$ of system (25) is globally asymptotically stable if $\mathfrak{R}_0 \leq 1$ and conditions (i) and (ii) are satisfied.*

Proof. From system (1) we can get $F(X, Z)$ and $G(X, Z)$:

$$\begin{aligned} F\left(X, Z\right) &= \begin{pmatrix} rs\left(1 - \dfrac{S+I}{K}\right) - \dfrac{\beta_1 SY}{A+S} \\ q - \dfrac{\beta_2 IH}{C+I} - \mu_2 H \end{pmatrix}, \\ G\left(X, Z\right) &= \begin{pmatrix} \dfrac{\beta_1 SY}{A+S} - \mu_1 I \\ \dfrac{b\beta_2 IH}{C+I} - \mu_3 Y \end{pmatrix} \end{aligned} \tag{26}$$

Now we consider the reduced system $dX/dt = F(X, 0)$ from condition (i)

$$\begin{aligned} \frac{dS}{dt} &= rs\left(1 - \frac{S}{K}\right), \\ \frac{dH}{dt} &= q - \mu_2 H \end{aligned} \tag{27}$$

$X^* = (K, q/\mu_2)$ is a globally asymptotically stable equilibrium point for the reduced system $dX/dt = F(X, 0)$. This can be verified from the solution of the first equation in Eq. (27); we obtain S(t) = $S(0)K/e^{-rt}(K - S(0)) + S(0)$ which approaches K as $t \longrightarrow \infty$ and from the second equation of Eq. (27) we get $H(t) = q/\mu_2 + (H(0) - q/\mu_2)e^{-\mu_2 t}$ which approaches q/μ_2 as $t \longrightarrow \infty$. We note that this asymptomatic dynamics is independent of the initial conditions in Ω; therefore the convergence of the solutions of the reduced system (27) is global in Ω. Now we compute

$$D_Z G\left(X^*, 0\right) = \begin{pmatrix} -\mu_1 & \dfrac{\beta_1 K}{A+K} \\ \dfrac{b\beta_2 q}{C\mu_2} & -\mu_3 \end{pmatrix} \tag{28}$$

Then, $G(X, Z)$ can be written as

$$G\left(X, Z\right) = D_Z G\left(X^*, 0\right) Z - \widehat{G}\left(X, Z\right) \tag{29}$$

and we want to show $\widehat{G}(X;Z) \geq 0$, which is obtained as

$$\widehat{G}(X,Z) = \begin{pmatrix} \beta_1 Y\left(\frac{K}{A+K} - \frac{S}{A+S}\right) \\ b\beta_2 I\left(\frac{q}{C\mu_2} - \frac{H}{C+I}\right) \end{pmatrix} \tag{30}$$

Here $K \geq S$ and $q/\mu_2 \geq H$. Hence it is clear that $\widehat{G}(X,Z) \geq 0$ for all (X,Z) $\in \Omega$. Therefore, this proves that DFE is globally asymptotically stable when $\mathfrak{R}_0 \leq 1$. □

3.7. The Endemic Equilibrium Point. In the presence of MSD, $S(t) \geq 0; I(t) \geq 0; H(t) \geq 0, Y(t) \geq 0$ the model has an equilibrium point called endemic equilibrium point denoted by $E^* = (S^*, I^*, H^*, Y^*)$. E^* is the steady state solution where MSD persist in the population of maize plants. It can be obtained by equating each equation of the model equal to zero; that is,

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dH}{dt} = \frac{dY}{dt} = 0 \tag{31}$$

Then we obtain

$$\begin{aligned} I^* &= \frac{rS^*\left(\mathfrak{R}_0^2\mu_1\mu_2\mu_3 CA - S^*\left(b\beta_1\beta_2 q - \mathfrak{R}_0^2\mu_1\mu_2\mu_3\right)\right)}{\mathfrak{R}_0^2\mu_1^2\mu_2\mu_3 CA + rS^*\left(b\beta_1\beta_2 q - \mathfrak{R}_0^2\mu_1\mu_2\mu_3\right)} \\ H^* &= \frac{qc\left(\mu_1 + rS^*/K\right) + qrS^*\left(1 - S^*/K\right)}{\beta_2 rS^*\left(1 - s/K\right) + \beta_2\mu_2\left(\mu_1 + rS^*/K\right)} \\ Y^* &= \frac{qc}{\mu_3}\left(1 - \frac{\mu_2\beta_1\left(\mu_1 + rS^*/K\right) + rS^*\left(1 - S^*/K\right)}{\beta_2 rS^*\left(1 - S^*/K\right) + \mu_2\left(\mu_1 + rS^*/K\right)}\right) \end{aligned} \tag{32}$$

and S^* is the positive root of the equation

$$Q_1 S^4 + Q_2 S^3 + Q_3 S^2 + Q_4 S + Q_5 = 0 \tag{33}$$

where

$$\begin{aligned} Q_1 &= -\mu_1\mu_3\beta_2 r^2 \\ Q_2 &= \mu_1\mu_3\beta_2 r^2\left(\mu_2 - A + 2K\right) + \beta_1 qcr^2\left(\mu_1 - \beta_2\right) \\ Q_3 &= \mu_1\mu_2\mu_3\beta_2 r\left(Ar - Kr + \mu_1 K\right) \\ &\quad - \beta_1\beta_2 qcr\left(\mu_1 K - Kr - \mu_2 r\right) \\ &\quad - \beta_1\mu_1 qcr\left(\beta_1 r - K + Kr\right) \\ &\quad + \mu_1\mu_3\beta_2 Kr^2\left(2A - 1\right) \\ Q_4 &= \beta_1\beta_2\mu_1 qcrK\left(K + 2\mu_2\right) - \beta_1\mu_1^2 qcrK\left(2\beta_1 + K\right) \\ &\quad - \mu_1\mu_3\beta_2 rKA\left(rK + \mu_2 r\right) \\ &\quad + \mu_1\mu_2\mu_3\beta_2 rK\left(\mu_1 A - K\right) \\ Q_5 &= \mu_1^2 K^2\left(\beta_1 qc\left(\beta_2\mu_2\beta_1\mu_1\right) - \mu_2\mu_3\beta_2 rA\right) \end{aligned} \tag{34}$$

3.8. Local Stability of Endemic Equilibrium

Theorem 4. *The endemic equilibrium E^* of system (1) is locally asymptotically stable in Ω if the following conditions hold for $\mathfrak{R}_0 > 1$:*

$$\begin{aligned} r\left(1 - \frac{2S^* + I^*}{K}\right) &> \frac{\beta_1 AY^*}{\left(A + S^*\right)^2} + \mu_1, \\ -\left(\mu_1 + \mu_2\right)\left(C + I^*\right) &> \beta_2 I^*, \\ \frac{bC\beta_1\beta_2 S^* H^*}{\left(A + S^*\right)\left(C + I^*\right)^2} &> \mu_1\mu_2 \end{aligned} \tag{35}$$

Proof. Let us first obtain the Jacobian matrix of system (1):

$$J = \begin{pmatrix} r\left(1 - \frac{2S+I}{K}\right) - \frac{\beta_1 AY}{(A+S)^2} & -\frac{rS}{K} & 0 & -\frac{\beta_1 S}{A+S} \\ \frac{\beta_1 AY}{(A+S)^2} & -\mu_1 & 0 & \frac{\beta_1 S}{A+S} \\ 0 & -\frac{C\beta_2 H}{(C+I)^2} & -\frac{\beta_2 I}{(C+I)} - \mu_2 & 0 \\ 0 & \frac{bC\beta_2 H}{(C+I)^2} & \frac{b\beta_2 I}{(C+I)} & -\mu_3 \end{pmatrix} \tag{36}$$

Evaluating this at the endemic equilibrium $E^* = (S^*, I^*, H^*, Y^*)$, we get

$$J = \begin{pmatrix} A_{11} & A_{12} & A_{13} & A_{14} \\ A_{21} & A_{22} & A_{23} & A_{24} \\ A_{31} & A_{32} & A_{33} & A_{34} \\ A_{41} & A_{42} & A_{43} & A_{44} \end{pmatrix} \tag{37}$$

where

$$\begin{aligned} A_{11} &= r\left(1 - \frac{2S^* + I^*}{K}\right) - \frac{\beta_1 AY^*}{\left(A + S^*\right)^2}, \\ A_{12} &= -\frac{rS^*}{K}, \\ A_{13} &= 0, \end{aligned}$$

$$
\begin{aligned}
A_{14} &= -\frac{\beta_1 S^*}{A+S^*} \\
A_{21} &= \frac{\beta_1 A Y^*}{(A+S^*)^2}, \\
A_{22} &= -\mu_1, \\
A_{23} &= 0, \\
A_{24} &= \frac{\beta_1 S^*}{A+S^*} \\
A_{31} &= 0, \\
A_{32} &= -\frac{C\beta_2 H^*}{(C+I^*)^2}, \\
A_{33} &= -\frac{\beta_2 I^*}{(C+I^*)} - \mu_2, \\
A_{34} &= 0 \\
A_{41} &= 0, \\
A_{42} &= \frac{bC\beta_2 H^*}{(C+I^*)^2}, \\
A_{43} &= \frac{b\beta_2 I^*}{(C+I^*)}, \\
A_{44} &= -\mu_3
\end{aligned}
\tag{38}
$$

The characteristic equation of the Jacobian matrix is given by

$$
\lambda^4 + A\lambda^3 + B\lambda^2 + C\lambda + D = 0 \tag{39}
$$

where

$$
\begin{aligned}
A &= -\left(A_{11} + A_{22} + A_{33} + A_{44}\right) \\
B &= A_{11}A_{22} + A_{11}A_{33} + A_{22}A_{44} \\
&\quad + \left(A_{11} + A_{33}\right)\left(A_{22} + A_{44}\right) + A_{21}A_{12} \\
&\quad + A_{42}A_{24} \\
C &= A_{33}A_{44}\left(A_{11} + A_{22}\right) + A_{11}A_{22}\left(A_{33} + A_{44}\right) \\
&\quad + A_{24}A_{42}\left(A_{11} + A_{33}\right) \\
&\quad + A_{12}A_{21}\left(A_{33} + A_{44}\right) - A_{24}A_{32}A_{43} \\
&\quad - A_{21}A_{14}A_{42} \\
D &= A_{11}A_{33}\left(A_{22}A_{44} - A_{24}A_{42}\right) \\
&\quad - A_{21}A_{14}\left(A_{32}A_{43} - A_{42}A_{33}\right) \\
&\quad + A_{11}A_{24}A_{43}A_{32} - A_{21}A_{12}A_{33}A_{44}
\end{aligned}
\tag{40}
$$

The sufficient conditions for $A > 0, C > 0, D > 0, AB > C, ABC > C^2 + A^2D$ are as follows:

$$
\begin{aligned}
r\left(1 - \frac{2S^* + I^*}{K}\right) &> \frac{\beta_1 A Y^*}{(A+S^*)^2} + \mu_1, \\
-\left(\mu_1 + \mu_2\right)\left(C + I^*\right) &> \beta_2 I^*, \\
\frac{bC\beta_1\beta_2 S^* H^*}{\left(A+S^*\right)\left(C+I^*\right)^2} &> \mu_1\mu_2
\end{aligned}
\tag{41}
$$

Thus, according to the Routh Hurwitz criterion, the characteristic equation (39) will have negative roots or imaginary roots with negative real part for $\mathfrak{R}_0 > 1$; the endemic equilibrium E^* is locally asymptotically stable. □

3.9. Global Stability of Endemic Equilibrium

Theorem 5. *If $\mathfrak{R}_0 > 1$, the endemic equilibrium E^* of the model (1) is globally stable.*

Proof. To establish the global stability of the endemic equilibrium E^*, we consider the following Lyapunov function:

$$
\begin{aligned}
V = \frac{\left(S - S^*\right)^2}{2} &+ \sigma_1\frac{\left(I - I^*\right)^2}{2} + \sigma_2\frac{\left(H - H^*\right)^2}{2} \\
&+ \sigma_3\frac{\left(Y - Y^*\right)^2}{2}
\end{aligned}
\tag{42}
$$

where $\sigma_1, \sigma_2, \sigma_3 > 0$ are to be chosen properly such that $(dV/dt)(E^*) = 0$ and $V(S, I, H, Y) > 0$ for all $(S, I, H, Y) \setminus E^*$.

By direct calculation, the derivative of V along the solution curve of system (1) yields

$$
\begin{aligned}
\frac{dV}{dt} &= \left(S - S^*\right)\frac{dS}{dt} + \sigma_1\left(I - I^*\right)\frac{dI}{dt} \\
&\quad + \sigma_2\left(H - H^*\right)\frac{dH}{dt} + \sigma_3\left(Y - Y^*\right)\frac{dY}{dt}
\end{aligned}
\tag{43}
$$

Now substituting equations of model (1), we get

$$
\begin{aligned}
&= \left(S - S^*\right)\left[rS\left(1 - \frac{S+I}{K}\right) - \frac{\beta_1 S Y}{A+S}\right] \\
&\quad + \sigma_1\left(I - I^*\right)\left[\frac{\beta_1 S Y}{A+S} - \mu_1 I\right] \\
&\quad + \sigma_2\left(H - H^*\right)\left[q - \frac{\beta_2 I H}{C+I} - \mu_2 H\right] \\
&\quad + \sigma_3\left(Y - Y^*\right)\left[\frac{b\beta_2 I H}{C+I} - \mu_3 Y\right]
\end{aligned}
\tag{44}
$$

By rearranging we obtain

$$\begin{aligned}\frac{dV}{dt} &= -\left(S-S^*\right)^2\left[r\left(-1+\frac{S+I}{K}\right)+\frac{\beta_1 Y}{A+S}\right]\\ &\quad-\sigma_1\left(I-I^*\right)^2\left[\frac{-\beta_1 SY}{I\left(A+S\right)}+\mu_1\right]\\ &\quad-\sigma_2\left(H-H^*\right)^2\left[-\frac{q}{H}+\frac{\beta_2 I}{C+I}+\mu_2\right]\\ &\quad-\sigma_3\left(Y-Y^*\right)^2\left[-\frac{b\beta_2 IH}{Y\left(C+I\right)}+\mu_3\right]\end{aligned} \tag{45}$$

We now choose

$$\begin{aligned}\sigma_1 &= \frac{I\left(A+S\right)}{\mu_1 I\left(A+S\right)-\beta_1 SY},\\ \sigma_2 &= \frac{H\left(C+I\right)}{\mu_2 H\left(C+I\right)-q\left(C+I\right)+\beta_2 IH},\\ \sigma_3 &= \frac{Y\left(C+I\right)}{\mu_3 Y\left(C+I\right)-b\beta_2 IH}\end{aligned} \tag{46}$$

Thus, $(dV/dt)(S,I,H,Y) \leq 0$ and an endemic equilibrium point is globally stable. Also $dV/dt = 0$, if and only if $S = S^*, I = I^*, H = H^*, Y = Y^*$. Therefore, the largest compact invariant set in $\{(S^*,I^*,H^*,Y^*) \in \Omega : dV/dt = 0\}$ is the singleton E^*, where E^* is the endemic equilibrium of the system (1). By LaSalle's invariant principle [21], it implies that E^* is globally asymptotically stable in Ω. □

3.10. Bifurcation Analysis. A bifurcation is a qualitative change in the nature of the solution trajectories due to a parameter change. The point at which this change takes place is called a bifurcation point. At the bifurcation point, a number of equilibrium points, or their stability properties, or both, change. We investigate the nature of the bifurcation by using the method introduced in [22], which is based on the use of the center manifold theory in [22].

Theorem 6 (Castillo-Chavez & Song [22]). *Let us consider a general system of ODE's with a parameter ϕ:*

$$\begin{aligned}&\frac{dx}{dt} = f\left(x,\phi\right),\\ &f : R^n \times R \longrightarrow R^n,\ f \in C^2\left(R^n \times R\right)\end{aligned} \tag{47}$$

where $x = 0$ is an equilibrium point for the system in Eq. (47). That is $f(0,\phi) \equiv 0$ for all ϕ. Assume the following.

***M_1**: $A = D_x f(0,0) = ((\partial f/\partial x_j)(0,0))$ is the linearization matrix of the system given by (47) around the equilibrium 0 with ϕ evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts.*

***M_2**: Matrix A has a nonnegative right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue. Let f_k be the k^{th} component of f and*

$$\begin{aligned}a &= \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)\\ b &= \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0)\end{aligned} \tag{48}$$

The local dynamics of (52) around 0 are totally determined by a and b.

(i) *$a > 0, b > 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$, 0 is unstable and there exists a negative, locally asymptotically stable equilibrium.*

(ii) *$a < 0, b < 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable; when $0 < \phi \ll 1$, 0 is locally asymptotically stable equilibrium, and there exists a positive unstable equilibrium.*

(iii) *$a > 0, b < 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$, 0 is stable, and a positive unstable equilibrium appears.*

(iv) *$a < 0, b > 0$. When ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.*

In particular, if $a < 0$ and $b > 0$, then the bifurcation is forward; if $a > 0$ and $b > 0$, then the bifurcation is backward. Using this approach, the following result may be obtained.

Theorem 7. *The model in system (1) exhibits forward bifurcation at $R_0 = 1$.*

Proof. We prove, using center manifold theorem [22], the possibility of bifurcation at $\mathfrak{R}_0 = 1$. Let $S = x_1$, $I = x_2$, $H = x_3$ and $Y = x_4$. In addition, using vector notation $x = (x_1, x_2, x_3, x_4)^T$, and $dx/dt = F(x)$, with $F = (f_1, f_2, f_3, f_4)^T$, then model in system (1) is rewritten in the form

$$\begin{aligned}\frac{dx_1}{dt} &= rx_1\left(1-\frac{x_1+x_2}{K}\right)-\frac{\beta_1 x_1 x_4}{A+x_1}\\ \frac{dx_2}{dt} &= \frac{\beta_1 x_1 x_4}{A+x_1}-\mu_1 x_2\\ \frac{dx_3}{dt} &= q-\frac{\beta_2 x_2 x_3}{C+x_2}-\mu_2 x_3\\ \frac{dx_4}{dt} &= \frac{b\beta_2 x_2 x_3}{C+x_2}-\mu_3 x_4\end{aligned} \tag{49}$$

We consider the predation and transmission rate β_1 as bifurcation parameters so that $\mathfrak{R}_0 = 1$ if

$$\beta_1 = \beta_1^* = \frac{\mu_1\mu_2\mu_3 C\left(A+K\right)}{b\beta_2 qK} \tag{50}$$

The disease-free equilibrium is given by $(x_1 = K, x_2 = 0; x_3 = q/\mu_2, x_4 = 0)$. Then the linearizion matrix of Eq. (49) at a disease-free Equilibrium is given by

$$J = \begin{pmatrix} -r & -r & 0 & -\dfrac{\beta_1^* K}{A+K} \\ 0 & -\mu_1 & 0 & \dfrac{\beta_1^* K}{A+K} \\ 0 & -\dfrac{\beta_2^* q}{C\mu_2} & -\mu_2 & 0 \\ 0 & \dfrac{b\beta_2^* q}{C\mu_2} & 0 & -\mu_3 \end{pmatrix} \tag{51}$$

The right eigenvector, $w = (w_1, w_2, w_3, w_4)^T$, associated with this simple zero eigenvalue can be obtained from $Jw = 0$. The system becomes

$$\begin{aligned} -rw_1 - rw_2 - \frac{\beta_1^* K}{A+K} w_4 &= 0 \\ -\mu_1 w_2 + \frac{\beta_1^* K}{A+K} w_4 &= 0 \\ \frac{\beta_2^* q}{C\mu_2} w_2 - \mu_2 w_3 &= 0 \\ \frac{b\beta_2^* q}{C\mu_2} w_2 - \mu_3 w_4 &= 0 \end{aligned} \tag{52}$$

From Eq. (52) we obtain

$$\begin{aligned} w_1 &= -\left(1 + \frac{\mu_1}{r}\right) w_2, \\ w_2 &= w_2 > 0, \\ w_3 &= \frac{\beta_2 q}{C\mu_1^2} w_2, \\ w_4 &= \frac{b\beta_2 q}{C\mu_2\mu_3} w_2 \end{aligned} \tag{53}$$

Here we have taken into account the expression for β_1^*. Next we compute the left eigenvector, $v = (v_1, v_2, v_3, v_4)$; associated with this simple zero eigenvalue can be obtained from $vJ = 0$ and the system becomes

$$\begin{aligned} -rv_1 &= 0 \\ -rv_1 - \mu_1 v_2 - \frac{\beta_2^* q}{C\mu_2} v_3 + \frac{b\beta_2^* q}{C\mu_2} v_4 &= 0 \\ -\mu_2 v_3 &= 0 \\ -\frac{\beta_1^* K}{A+K} v_1 + \frac{\beta_1^* K}{A+K} v_2 - \mu_3 v_4 &= 0 \end{aligned} \tag{54}$$

From equation of Eq. (54), we obtain

$$\begin{aligned} v_1 &= v_3 = 0, \\ v_4 &= \frac{C\mu_1\mu_2}{b\beta_2 q} v_2, \end{aligned} \tag{55}$$

Here we have taken into account the expression for β_1^*, where v_2 is calculated to ensure that the eigenvectors satisfy the condition $v.w = 1$. Since the first and third components of v are zero, we do not need the derivatives of f_1 and f_3. From the derivatives of f_2 and f_4, the only ones that are nonzero are the following:

$$\begin{aligned} \frac{\partial^2 f_2}{\partial x_1 \partial x_4} &= \frac{\partial^2 f_2}{\partial x_4 \partial x_1} = \frac{\beta_1^* A}{\left(A + x_1^*\right)^2}, \\ \frac{\partial^2 f_4}{\partial x_2 \partial x_3} &= \frac{\partial^2 f_4}{\partial x_3 \partial x_2} = \frac{b\beta_2}{C} \\ \frac{\partial^2 f_4}{\partial x_2^2} &= -2\frac{b\beta_2 C x_3^*}{\left(C + x_2^*\right)^3}, \\ \frac{\partial^2 f_2}{\partial x_4 \partial \beta_1} &= \frac{x_1^*}{A + x_1^*} \end{aligned} \tag{56}$$

and all the other partial derivatives of f_2 and f_4 are zero. The direction of the bifurcation at $\mathfrak{R}_0 = 1$ is determined by the signs of the bifurcation coefficients a and b, obtained from the above partial derivatives, given, respectively, by

$$\begin{aligned} a = {} & v_2 w_4 w_1 \frac{\beta_1^* A}{\left(A + x_1^*\right)^2} + v_2 w_1 w_4 \frac{\beta_1^* A}{\left(A + x_1^*\right)^2} \\ & + v_4 w_3 w_2 \frac{b\beta_2}{C} + v_4 w_3 w_2 \frac{b\beta_2}{C} \\ & + v_4 w_2 w_2 \left(-2\frac{b\beta_2 C x_3^*}{\left(C + x_2^*\right)^3}\right) \\ = {} & 2v_2 w_4 w_1 \frac{\beta_1^* A}{\left(A + x_1^*\right)^2} + 2v_4 w_3 w_2 \frac{b\beta_2}{C} \\ & - 2v_4 w_2 w_2 \frac{b\beta_2 C x_3^*}{\left(C + x_2^*\right)^3} \end{aligned} \tag{57}$$

Hence

$$a = \left(-2\mu_1 v_2 w_2^2\right)\left[\frac{A\left(1 + \mu_1/r\right)}{K\left(A+K\right)} - \frac{\beta_2}{C\mu_2} + \frac{\mu_1}{C}\right] < 0, \tag{58}$$

$$b = v_2 w_4 \frac{x_1^*}{A + x_1^*} = v_2 w_4 \frac{K}{A+K} > 0 \tag{59}$$

As the coefficient b is always positive and the sign of the coefficient a is negative, MSD model exhibits a forward bifurcation and there exists at least one stable endemic equilibrium when $\mathfrak{R}_0 > 1$. Using expression for I^* in the endemic equilibrium, we plotted a forward bifurcation diagram in Figure 7. We used a set of estimated and assumed parameters in Table 3. □

Table 2: Sensitivity indices table.

parameter symbol	Sensitivity indices
β_1	0.5
β_2	0.5
b	0.5
q	0.5
K	0.39998×10^{-4}
μ_1	-0.5
μ_2	-0.5
μ_3	-0.5
C	-0.5
A	-0.39998×10^{-4}

3.11. Sensitivity Analysis of Model Parameters. We carried out sensitivity analysis, on the basic parameters, to check and identify parameters that can impact the basic reproductive number. Sensitivity analysis notifies us on how significant each parameter is to disease transmission. To go through sensitivity analysis, we followed the approach defined by [23] like in [24]. This technique develops a formula to obtain the sensitivity index of all the basic parameters, defined as follows.

Definition 8. The normalized forward sensitivity index of a variable, g, that depends differentiably on a parameter, p, is defined as

$$\Lambda_P^g = \frac{\partial g}{\partial p} \times \frac{p}{g} \tag{60}$$

for p represents all the basic parameters. Here we have $\mathfrak{R}_0 = \sqrt{b\beta_1\beta_2 Kq/\mu_1\mu_2\mu_3 C(A+K)}$. For example the sensitivity index of $\mathfrak{R}_0$ to β_1 is

$$\begin{aligned}\Lambda_{\beta_1}^{\mathfrak{R}_0} &= \frac{\partial \mathfrak{R}_0}{\partial \beta_1} \times \frac{\beta_1}{\mathfrak{R}_0} = \frac{1}{2\sqrt{b\beta_1\beta_2 Kq/\mu_1\mu_2\mu_3\,(A+K)}} \\ &\cdot \frac{b\beta_2 Kq}{\mu_1\mu_2\mu_3 C\,(A+K)} \frac{\beta_1}{\mathfrak{R}_0} = \frac{1}{2} \geq 0\end{aligned} \tag{61}$$

And we do this in a similar fashion for the remaining parameters.

3.12. Interpretation of Sensitivity Indices. The sensitivity indices of the basic reproductive number with respect to main parameters are found in Table 2. Those parameters that have positive indices (b_1, β_1, β_2, K *and* q) show that they have great impact on expanding the disease in the community if their values are increasing. Due to the reason that the basic reproduction number increases as their values increase, it means that the average number of secondary cases of infection increases in the community. And also those parameters in which their sensitivity indices are negative (C, A, μ_1, μ_2 *and* μ_3) have an effect of minimizing the burden of the disease in the community as their values increase while the others are left constant. And also as their values increase, the basic reproduction number decreases, which leads to minimizing the endemicity of the disease in the community.

Table 3: Parameter values for the MSV model.

parameter symbol	Value day^{-1}	Source
β_1	0.45	[2]
β_2	0.04	[2]
q	0.02	Assumed
K	10,000	Assumed
μ_1	0.008	Assumed
μ_2	0.0303	[3]
μ_3	0.0303	[3]
b	0.45	Assumed
A	0.4	Assumed
C	0.6	Assumed
r	0.005	Assumed

4. Numerical Simulation

Numerical simulations of the model (1) are carried out, in order to illustrate some of the analytical results of the study. A set of reasonable parameter values is given in Table 3. These parameter values were obtained from literature and some of them were assumed. We used S(0) = 1000, I(0) = 20, H(0) = 100, Y(0) = 0, as initial values and parameter values in Table 3 for simulation of MSV model in addition to parameter values in Table 3.

From the left-hand side of Figure 2, the susceptible maize population decelerates exponentially to acquire endemic equilibrium level as they die due to infected leafhopper vector population. The infected maize population assumes parabolic curve as it increases exponentially to a certain maximum point before exponential deceleration to the certain endemic level.

From the right-hand side of Figure 2, the susceptible vectors decrease exponentially due to natural death and acquisition of infestation from severely infected maize and MSV from the environment and finally acquire the endemic equilibrium level and the infected vectors form a parabolic curve as they do raise and drop exponentially to the endemicity level.

Figure 3 shows the simulation of infected maize and susceptible maize for different value of β_1. We can see from the figure that, increasing the infection and predation rate of infected leafhopper on susceptible maize β_1, the basic reproduction number increases, which leads to an increase in the number of infective maize and on the other hand the number of susceptible maize population decrease.

Figure 4 shows the simulation of infected maize and infected leafhopper for different value of β_2. We can see from the figure that, increasing the infection and predation rate of susceptible leafhopper on infected maize β_2, the basic reproduction number increases, which will lead to an increase in the number of the infected maizes as well as the number of infected leafhoppers.

Figure 5 shows simulation of infected leafhopper and susceptible leafhopper for a different value of μ_2 and μ_3. We can see that increasing the death rate of the leafhopper population reduces the basic reproduction number. Due to

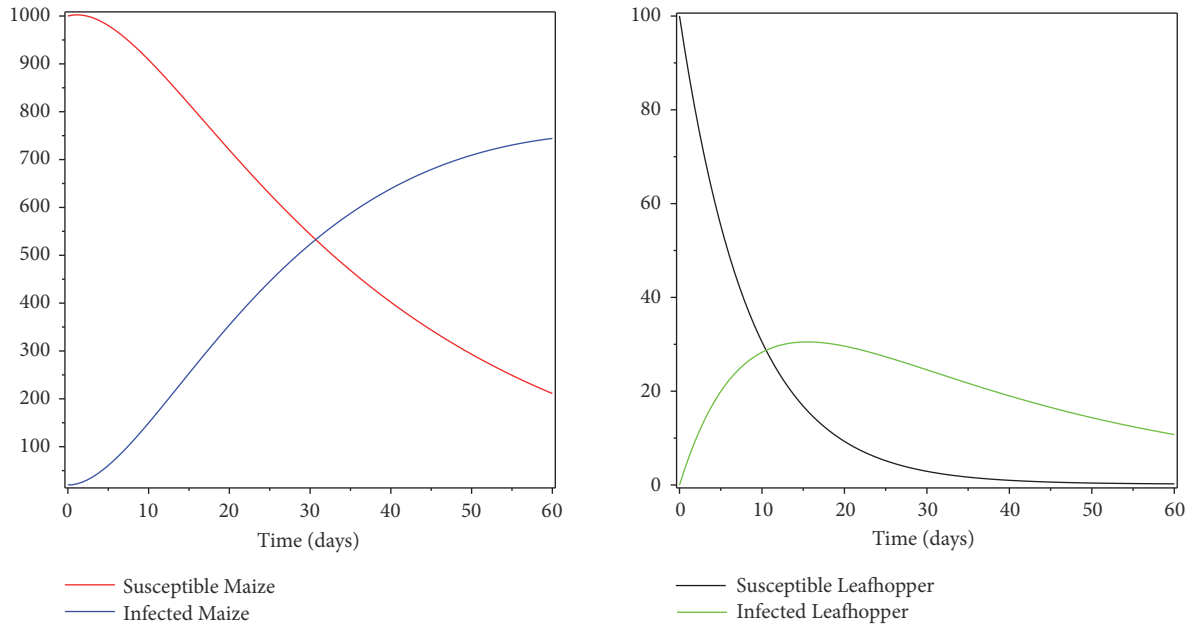

Figure 2: Simulation results of susceptible and infected population of maize and leafhopper when $\mathfrak{R}_0 > 1$.

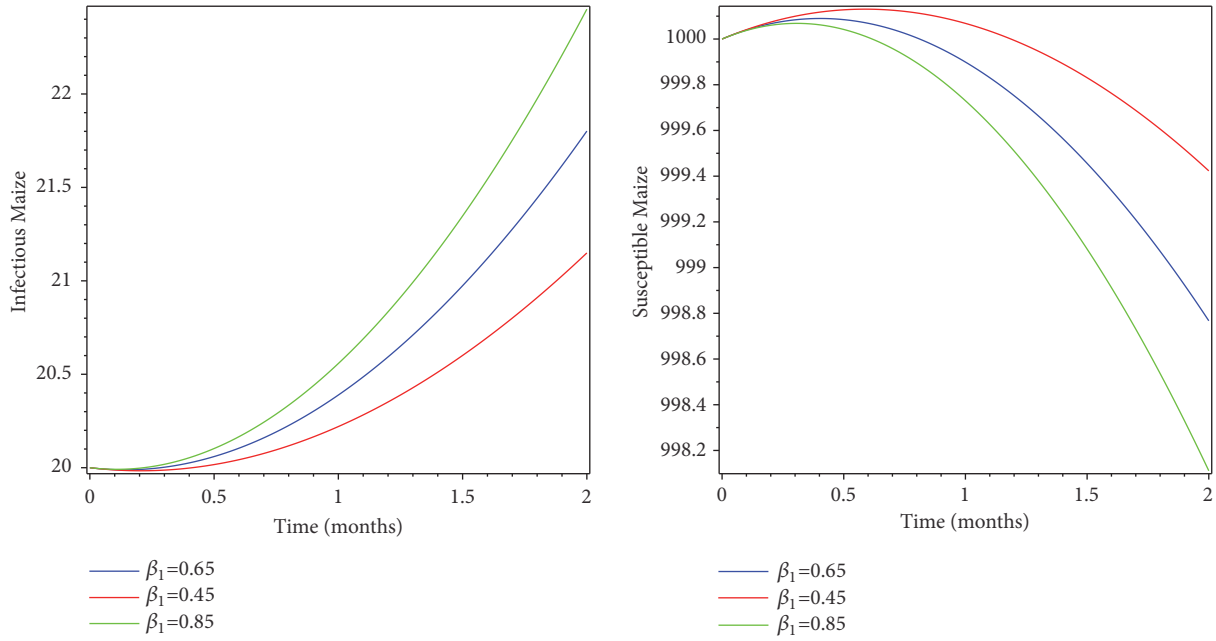

Figure 3: Simulation of infected maize and susceptible maize with different value of β_1.

the indirect relation of $\mathfrak{R}_0$ and μ_2 and μ_3 it leads to a decrease in the population of the leafhopper.

Figure 6 shows simulation of infected maize for different value of μ_1. We can see that increasing the death rate of infected and infectious maize μ_1 reduces the reproduction number. This leads to a decrease in the infection rate of maize.

5. Discussions and Conclusions

In this paper, we have proposed and analysed an ecoepidemiological mathematical model of MSV. We considered a Holling type II functional response which is biologically realistic. We showed that the system was uniformly bounded and

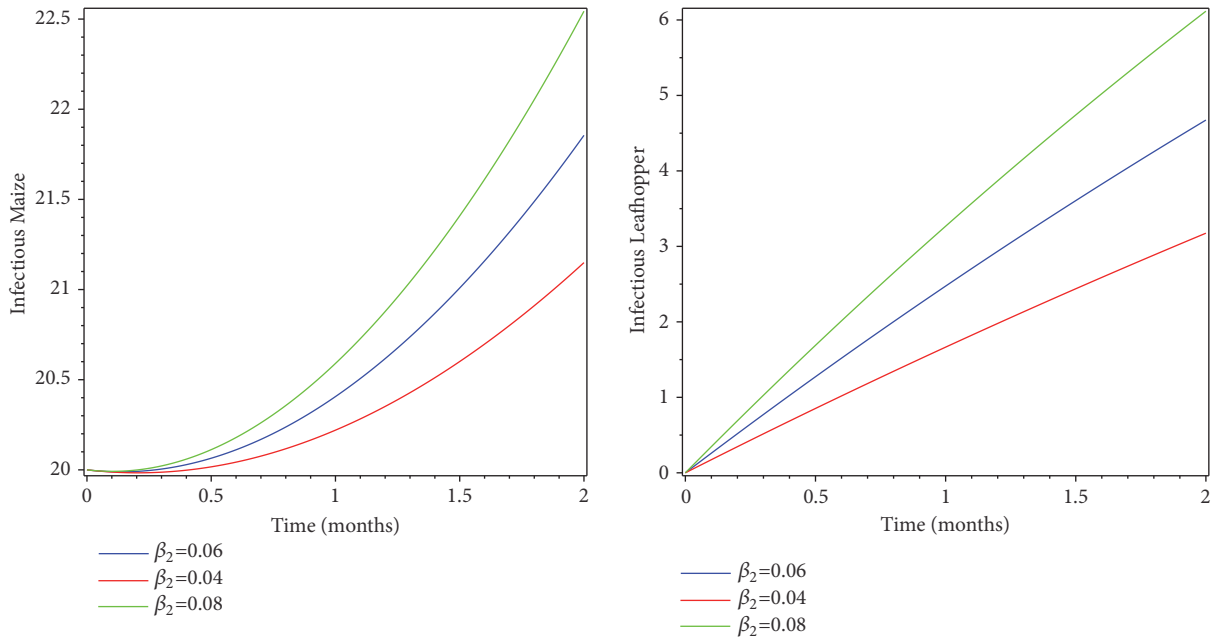

Figure 4: Simulation of infected maize and infected leafhopper with different value of β_2.

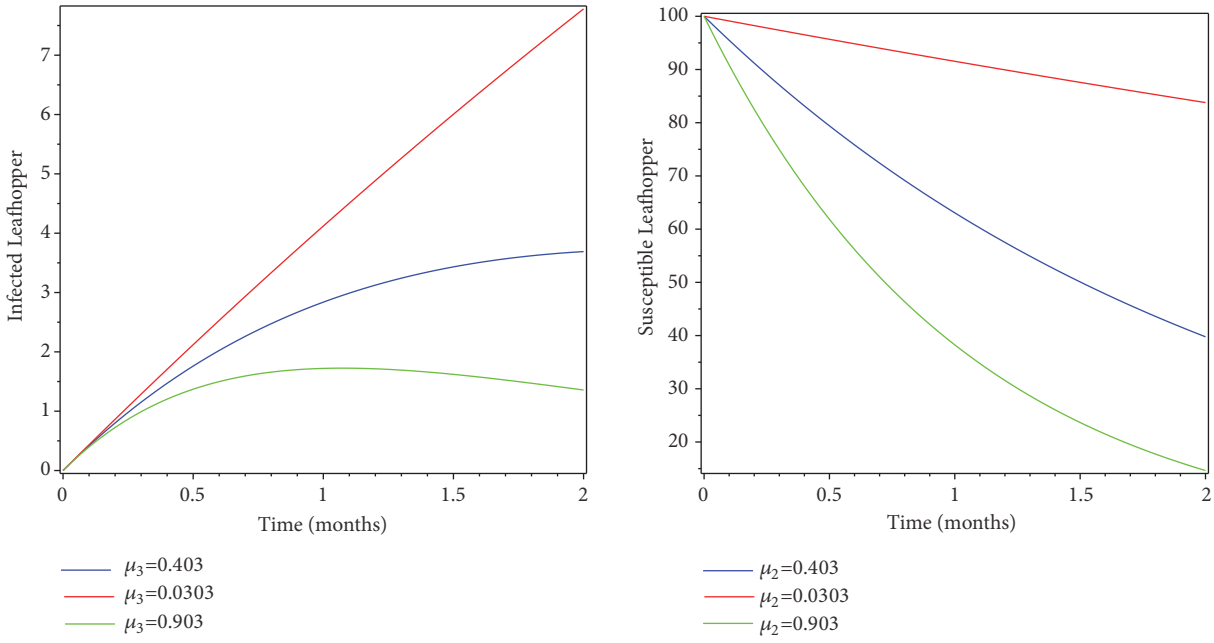

Figure 5: Simulation of infected leafhopper and susceptible leafhopper with different value of μ_2 and μ_3.

positive. We found the disease-free and endemic equilibrium points and their local and global stability analysis has been investigated. The bifurcation analysis of the model is shown. The model analysis also shows the sensitivity of parameters to the disease persistence and dying out.

Finally, analytical results were confirmed by numerical simulation with realistic parameter values. We showed that increasing the infection and predation rates, β_1 and β_2, makes an increase of basic reproduction number which leads to the increase of the number of infected maize population. However, increasing death rate of infected maize and leafhopper population decreases the reproduction number which in turn means that the disease dies out from the maize population. Thus, from the results of this paper, control intervention strategies reduces the disease infection of maize population. The model shows that the spread of the disease largely

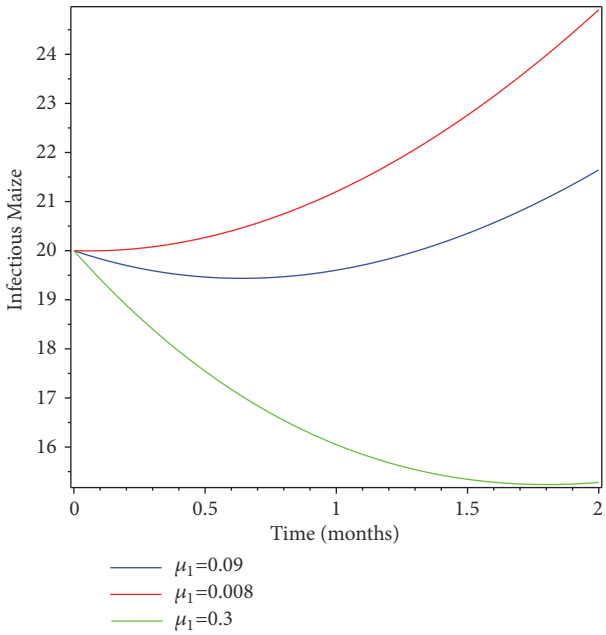

Figure 6: Simulation of the MSV model with different value of μ_1.

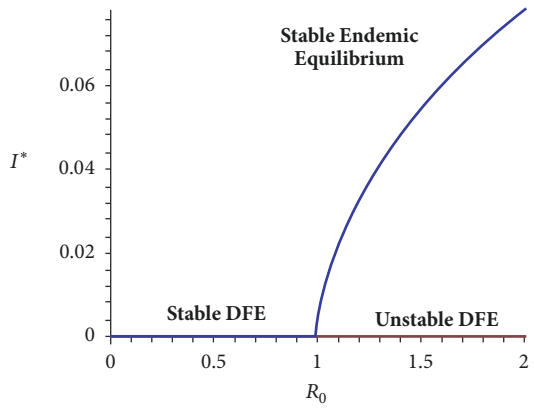

Figure 7: Forward bifurcation of MSV model (1).

depends on the infection and predation rates β_1 and β_2; therefore efforts should be made to minimize the contact of infected maize and susceptible leafhopper and MSV infected maize should be treated either using insecticide chemical to reduce the infection rate of leafhoppers and it should be done before the arrival of leafhopper or uprooting and burning the infected maize from the field. This implies that, to get the best and cost-effective control strategy, we should apply optimal control theory. Thus, we come next with a paper applying the optimal and cost-effective strategies to identify the best and cost-effective strategy for this model.

Data Availability

The data supporting this deterministic model are from previous published articles and they have been duly cited in this paper. These published articles are cited in Table 3 and relevant places in this paper.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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