Mathematical Modelling and Control

http://www.aimspress.com/journal/mmc

MMC, 2(4): 153–164
DOI:10.3934/mmc.2022016
Received: 04 July 2022
Revised: 15 October 2022
Accepted: 16 November 2022
Published: 16 December 2022

Research article

Mathematical modeling of the dynamics of maize streak virus disease (MSVD)

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Abstract: In this paper, a deterministic ordinary differential equations model for the transmission dynamics of maize streak virus disease (MSVD) in maize plants is proposed and analyzed qualitatively. Using the next generation matrix approach, the basic reproduction number, $\mathcal{R}_0$ with respect to the MSVD free equilibrium is found to be $4.7065 \approx 5$. The conditions for local stability of the disease-free equilibrium and endemic equilibrium points were established. From the results, the disease-free equilibrium point was found to be unstable whenever $\mathcal{R}_0 > 1$ and the endemic equilibrium point was found to be locally asymptotically stable whenever $\mathcal{R}_0 > 1$. The sensitivity indices of various parameters with respect to the MSVD eradication or spreading were determined. It was found that b ,β_1 ,β_{11} , and β_2 are the parameters that are directly related to $\mathcal{R}_0$, and H_2, μ, μ_1 and γ are inversely related to $\mathcal{R}_0$. Numerical simulation was performed and displayed graphically to justify the analytical results.

Keywords: stability analysis; compartmental model; basic reproduction number, bifurcation and sensitivity analysis; numerical simulation

1. Introduction

Maize is one of the most important cereal crops cultivated worldwide and it represents a staple food for a significant proportion of the world's population. There is no native toxins reported for the genus Zea [1]. Maize was first introduced by Portuguese merchants in the 16th century to Africa through Ghana [2]. In Sub-Saharan Africa, maize provides staple food for about 50% of the population and provides 50% of basic calories [3]. Maize serves as a major staple food for many Ghanaian households. It is predominantly cultivated under rain-fed agriculture by poorly resourced subsistence farmers in the coastal Savannah, forest, transition and guinea Savannah zones [4]. The production of maize in Ghana accounts for about 50% of the total cereals produced, with an estimated 30% losses due to pests and diseases [5].

Fuller et al [6] described the disease condition as "mealic variegation" at the beginning of the twentieth century which was later renamed maize streak virus. The Maize Streak Virus Disease (MSVD) is one of the common diseases of maize. MSVD is caused by a virus known as the maize streak virus (MSV), a species of genus Mastrevirus of the family Geminiviridae [7]. MSVD is transmitted by the vector of genus cicadulina mbila Naude of the leafhopper species [2]. MSVs have single-component, spherical, single-stranded DNA genomes of approximately 2700 bases in MSV and all associated grass mastreviruses, encapsulated in $22 \times 38nm$ geminate particles consisting of two incomplete T=1 icosahedra with 22 pentameric capsomers composed of a single acapsid protein of 32kD [2]. They have characteristically twinned (geminate) particle morphology that is stable at pH 4-8 [2]. Geminiviruses have agronomic importance globally [8]. They were responsible for the major economic crisis for many subsistence farmers in Sub-Saharan Africa [9]. Epidemics resulting in economic losses have been reported in over 20 African countries including Nigeria, Sudan, Ghana, Cameroon, Togo, Burkina Faso, Tanzania, Benin and Ethiopia. The MSV-A is the common strain of the virus responsible for causing MSVD in Ghana and throughout Sub-Saharan Africa [10]. Apart from the Strain MSV-A, there are 10 other known strains of the virus MSV-B-MSV-K causing MSVD in wheat, barley, oat, millet and many other wild grass species [2]. A survey conducted in November/December, 2010 across the forest and transition zones in Ghana on virulence of the MSV-A shows that, in 79 well-distributed maize farms, the mean MSV incidence was 18.544% and the symptom results score was 2.956 (1-no symptom and 5-extremely severe symptom) [10]. This shows that, there is no correlation between these two variables. 51 genome sequences were determined through phylogenetic analysis of clone MSV-A isolates [10]. Environmental factors are the main causes of MSVD epidemics.

MSVD affects mainly the leaves, the younger leaves to be precise. Matured leaves remain healthy throughout the infection period. Symptoms of the disease include minute, pale and circular spots on the lower and exposed portions of the younger leaves. Maize plants infected at an early stage usually become stunted in growth causing bareness, interveinal necrosis, chlorosis and consequently, death of affected plants. However, the effect is less pronounced when it affects matured plants.

Various methods have been used to control the incidence of MSVD. These include early planting when virus inoculum loads are low, the use of chemicals/insecticides such as carbofuran with its residual effects, development and use of streak-resistant variety of maize [2], suppression through biological agents to control the pathogens [11], physical removal of diseased plants and use of ultraviolet (UV) radiation [12] among others.

Maize streak virus disease (MSVD) in maize plants, like many other plant diseases is still a major problem in Ghana and Sub-Saharan Africa despite efforts made to reduce the incidence of the disease. The disease has caused reduced crop yield and worsened the economic lives of many families that depend on farming for their survival in the country [4].

Over the years, researchers have used mathematical models as a stop-gap measure in dealing with infectious diseases as they provide better understanding of the dynamics of diseases. Several models have since been proposed and used to study the dynamics of MSVD.

Shi, et al [13] developed a Susceptible-Infected-Removed (SIR) compartmental deterministic epidemic model which describes the MSVD with the aim to investigate the effect of insect vectors on the spread of the disease. Using the next generation matrix method, the formula for the basic reproduction number, $\mathcal{R}_0$ was obtained and this helped to establish the stability of the disease-free equilibrium and the endemic equilibrium. From the results of the study, the disease-free equilibrium is globally asymptotically stable whenever $\mathcal{R}_0 \leq 1$ and the unique endemic equilibrium is globally asymptotically stable provided that $\mathcal{R}_0 > 1$.

Alemneh, et al [14] proposed and analyzed qualitatively an eco-epidemiological deterministic model for the transmission dynamics of maize streak virus(MSV) disease in maize plant using the stability theory of differential equations. In the model formulation, two populations were considered; the maize population and the leafhopper vector population. These populations were sub-divided into susceptible and infected. The susceptible and infected maize densities at time, t were $S(t)$ and $I(t)$ respectively while that of the leafhopper vector are $H(t)$ and $Y(t)$ respectively. From the results of the study, control intervention strategies reduce the disease infection of maize population. The model shows that the spread of the disease largely 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 leaf hoppers and it should be done before the arrival of leafhopper or uprooting and burning the infected maize from the field.

Similarly, in an attempt to deal with MSVD, [15] developed a mathematical model on optimal control and cost-effectiveness analysis of maize streak virus pathogen interaction with pest invasion in maize plants. The model was formulated based on Susceptible(S)-Infected(I) and Susceptible(H)-Exposed(Y)-Infected(P) compartmental deterministic model for the maize host and leafhopper vector populations respectively. Prevention (u_1), quarantine (u_2) and chemical control (u_3) were used as the maize streak virus control strategies. It was found that the combination of prevention and quarantine is the best effective strategies in terms of cost as well as health benefits.

Alemneh, et al [16] formulated and analyzed an optimal deterministic eco-epidemiological model for the dynamics of maize streak virus (MSV) and examined the best strategy to fight maize population from maize streak disease (MSD). Three control interventions, namely prevention u_1, quarantine u_2 and chemical control u_3 were incorporated into the model to determine the best control strategies for MSD. Similarly, from the perspective of cost-effective strategy, the combination of prevention and quarantine was found to be the best cost-effective strategy from the other integrated strategies. Another interesting study of MSVD can be found in [17].

To the best of our knowledge, no consideration was given to the latent period before infection in the literature. This paper draws its motivation from the works in [14–16] to present a compartmental deterministic model that incorporates this period called exposed period to describe the spread and control of Maize Streak Virus Disease (MSVD).

The organization of this paper is as follows; Section 2 presents of the proposed model. Section 3 presents the qualitative analysis of the model. Section 4 contains results of numerical simulations performed to support analytical results. Section 5 presents the summary and conclusion based on the findings of the paper.

2. Model formulation

In the model formulation, both the maize host plant population and the transmission vector, brownish white leafhopper are considered. Based on Susceptible (S), Exposed (E) and Infected (I) compartmental model, the proposed model for this study is SEI for the study of the dynamics of MSVD in maize host population and SI model for the study of the dynamics of MSVD in leafhopper vector population. The total population of the maize host $M(t)$ is put into three compartments at time t, namely, Susceptible maize plants (S), Exposed maize plants (E) and Infected maize plants (I). On the other hand, the total population of leafhoppers $L(t)$ vector is put into two compartments at time, t namely, Susceptible leafhopper (Q) and Infected leafhopper (P). Therefore, $M(t) = S(t) + E(t) + I(t)$ and $L(t) = Q(t) + P(t)$.

In the absence of the leafhopper vector, the maize host population grows logistically with intrinsic growth rate r_1. Susceptible maize plants with natural resistance to viral infections do not become infected after the pathogens have been deposited on them by the infected leafhopper vector. Those without any immunity, become exposed at a rate β_1 in the presence of infected leafhoppers. Exposed maize plants become infectious at a rate, δ. Similarly, Infected Leafhopper becomes infectious at a rate β_{11}. Once infected, the maize plants are removed and burnt at a rate γ. Infected maize plants die naturally at a rate μ. Young and susceptible leafhopper recruited by birth into the environment at a rate b, become infected at a rate, β_2 after coming into contact with an infected maize host.

Susceptible and infected leafhoppers have natural death rates μ_1. It is further assumed that only healthy maize seeds are sowed in the farmland of carrying capacity, K, no deaths as a result of infection before harvesting and the infected leafhopper vectors remain infectious after they become infected with the virus.

Considering the above assumptions, the model is given by the following equations ;

$$\left.\begin{array}{l}
\frac{dS}{dt} = r_1\lambda S - \frac{\beta_1 SP}{H_1+S} - \frac{\beta_{11}SI}{H_1+S},\\
\frac{dE}{dt} = r_2\lambda E + \frac{(\beta_1 P+\beta_{11}I)S}{H_1+S} - \delta E\\
\frac{dI}{dt} = \delta E - (\gamma+\mu)I,\\
\frac{dQ}{dt} = b - \frac{\beta_2 IQ}{H_2+I} - \mu_1 Q,\\
\frac{dP}{dt} = \frac{\beta_2 IQ}{H_2+I} - \mu_1 P,\\
\lambda = \left(1 - \frac{S+E+I}{K}\right),\\
\text{with nonnegative initial conditions.}
\end{array}\right\} \quad (2.1)$$

The equations of the total population of the maize plants and the total population of the leafhopper vectors are

$$\left.\begin{aligned} \frac{dM}{dt} &= (r_1 S + r_2 E)\left(1 - \frac{M}{K}\right) - (\gamma + \mu)I, \\ \frac{dL}{dt} &= b - \mu_1 L. \end{aligned}\right\} \quad (2.2)$$

The dynamics of the spread of MSVD is depicted in Figure 1.

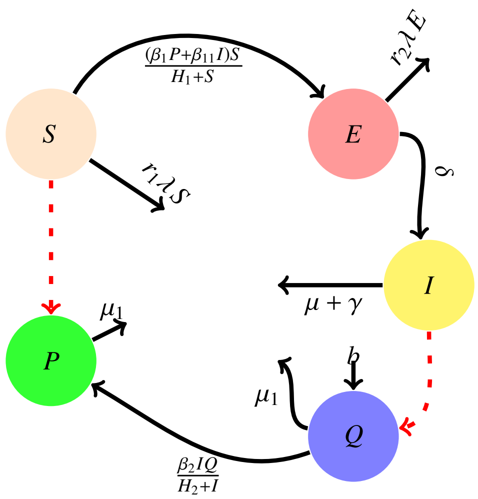

Figure 1. Schematic Diagram of the MSVD Model (2.1)

The model parameters are summarized in Table 1.

Table 1. Description of the Maize streak virus disease model parameters.

Parameter	Description
b	Birth rate of susceptible leafhopper
K	Carrying capacity of the farm
β_1	Predation and Infection rate of Infected Leafhopper on Susceptible Maize plant
β_{11}	Predation and Infection rate of Exposed maize plant on Infected Leafhopper
β_2	Predation and Infection rate of Susceptible Leafhopper on Infected Maize plant
H_1	Half saturation rate of Susceptible maize with Infected plant
H_2	Half saturation rate of Susceptible Leafhopper with Infected Maize plant
μ	Natural death rate of infected maize plant
γ	Removal rate of infected maize plant
μ_1	Natural death rate of susceptible leafhopper
δ	Rate at which exposed maize plants become infectious
r_1	Growth rate of Susceptible Maize
r_2	Growth rate of Exposed Maize

3. Qualitative analysis of the model

3.1. Positivity of solutions

For the MSVD model (2.1) above to be epidemiologically meaningful, it is important to prove that all solutions of the system with non-negative initial data in ϕ will remain positive for all times ($t \geq 0$).

Theorem 3.1. *Let ϕ=(S,E,I,Q,P) $\in \mathbb{R}^5_+$: $S(0) > 0$, $E(0) > 0$, $I(0) > 0$, $Q(0) > 0$, $P(0) > 0$. Then the solution set $(S(t), E(t), I(t), Q(t), P(t))$ of the system (2.1) is positive for all $t \geq 0$.*

Proof. From the first equation of (2.1),

$$\begin{aligned} \frac{dS}{dt} &= r_1 S\left(1 - \frac{S+E+I}{K}\right) - \frac{\beta_1 S P}{H_1+S} - \frac{\beta_{11} S I}{H_1+S} \\ &\leq r_1 S\left(1 - \frac{S}{K}\right) \end{aligned} \quad (3.1)$$

so that we have

$$\frac{dS}{dt} \leq r_1 S\left(1 - \frac{S}{K}\right).$$

Separating variables and solving the above inequality gives

$$S(t) \leq \frac{KS(0)}{e^{-r_1 t}(K - S(0)) + S(0)}$$

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

$$\left.\begin{aligned} E(t) &\leq \frac{KE(0)}{e^{-r_2 t}(K-E(0))+E(0)}, \\ I(t) &\geq I(0)e^{-(\gamma+\mu)t} \geq 0, \\ Q(t) &\geq Q(0)e^{-\mu_1 t} \geq 0, \\ P(t) &\geq P(0)e^{-\mu_1 t} \geq 0. \end{aligned}\right\} \quad (3.2)$$

Since the initial conditions for the system (2.1) above are all nonnegative, they have positive solutions. Therefore, the model is epidemiologically meaningful and well posed. □

3.2. Feasible region

The solution sets of the MSVD model with nonnegative initial data in $\mathbb{R}^5$ are feasible for all $t > 0$ if they enter the invariant region $\phi = \phi_m \times \phi_l \subset \mathbb{R}^3_+ \times \mathbb{R}^2_+$ where $\phi_m = \left\{(S(t), E(t), I(t)) \in \mathbb{R}^3_+ : M(0) \leq M(t) \leq K\right\}$ and $\phi_l = \left\{(Q(t), P(t)) \in \mathbb{R}^2_+ : L(0) \leq L(t) \leq \frac{b}{\mu_1}\right\}$

Proof. Let $\phi = (S, E, I, Q, P) \in \mathbb{R}^5_+$ be any solution of the MSVD model system (2.1) with nonnegative initial conditions. The total maize plant population can be shown to satisfy; $\frac{\mathrm{d}M}{\mathrm{d}t} \le (r_1 S + r_2 E)\left(1 - \frac{M}{K}\right) \le rM\left(1 - \frac{M}{K}\right)$, where $r = \max\{r_1,\ r_2\}$

$$\implies \frac{\mathrm{d}M}{\mathrm{d}t} \le rM\left(1 - \frac{M}{K}\right)$$

Separating the variables gives

$$\frac{\mathrm{d}M}{M\left(1 - \frac{M}{K}\right)} \le rdt \tag{3.3}$$

$$\implies \left(\frac{1}{M} + \frac{1}{K(1 - \frac{M}{K})}\right)\mathrm{d}M \le rdt \tag{3.4}$$

Integrating and evaluating 3.4 at $t = 0, M = M(0)$, we have

$$M(t) \le \frac{KM(0)}{e^{-rt}(K - M(0)) + M(0)}$$

Hence,

$$M(t) \le \frac{KM(0)}{e^{-rt}(K - M(0)) + M(0)}$$

As t approaches ∞, we obtain $0 \le M(t) \le K$.

The population of the maize plants will increase to K as $t \to \infty$. Then $M(0) \le M(t) \le K$ for all t. Therefore,

$$\phi_m = \left\{(S(t), E(t), I(t)) \in R^3_+ : M(0) \le M(t) \le K\right\}$$

Similarly, for leafhopper population, $L(Q, R) = Q(t) + R(t)$. After differentiating L with respect to t and substituting dQ/dt, dR/dt, we have

$$\frac{dL}{dt} = b - \mu_1 Q - \mu_1 P \le b - \mu_1 L \tag{3.5}$$

$$\implies \frac{\mathrm{d}L}{\mathrm{d}t} + \mu_1 L \le b \tag{3.6}$$

Solving and evaluating 3.6 at $t = 0, L(0) = L_0$, we obtained.

$$L(t) \le \frac{b}{\mu_1} + (L_0 - \frac{b}{\mu_1})e^{-\mu_1 t}$$

Then $\frac{b}{\mu_1} \le L(t) \le L(0)$ for all t when $L(0) \ge \frac{b}{\mu_1}$. This means the leafhopper population $L(t)$ reduces to the equilibrium $\frac{b}{\mu_1}$ as $t \to \infty$. However, for $L(0) \le \frac{b}{\mu_1}$, the leafhopper vector population $L(t)$ increases to $\frac{b}{\mu_1}$ as $t \to \infty$. Then, $L(0) \le L(t) \le \frac{b}{\mu_1}$ for all t. The feasible solution set of the leafhopper vector population enters the region $\phi_l = \left\{(Q(t), P(t)) \in \mathbb{R}^2_+ : L(0) \le L(t) \le \frac{b}{\mu_1}\right\}$.

Therefore, the feasible solutions set for the MSVD model given by $\phi_m \times \phi_l$ is positively invariant, epidemiologically meaningful and mathematically well-posed in the domain ϕ. Hence, every solution of the model system (2.1) with initial conditions in ϕ remain in ϕ for all $t > 0$. □

3.3. Equilibrium points of the model

The equilibrium points are obtained by setting the right-hand-sides of the model equation (2.1) to zero and finding the state variables. Let $\mathcal{E}^* = (S^*, E^*, I^*, Q^*, P^*)$ be a typical equilibrium point. Therefore the endemic equilibrium $\mathcal{E}^* = (S^*,\ E^*,\ I^*,\ Q^*,\ P^*)$ is such that

$$\left.\begin{aligned} S^* &= \tfrac{K}{2}\left(1 - \tfrac{(\gamma+\mu+\delta)E^* + H_1}{K(\gamma+\mu)}\right) \pm \tfrac{K\Delta}{2r_1}, \\ I^* &= \tfrac{\delta}{\gamma+\mu}E^*, \\ Q^* &= \tfrac{b\left[H_2(\gamma+\mu)+\delta E^*\right]}{\delta(\beta_2+\mu_1)E^*+\mu_1 H_2(\gamma+\mu)}, \\ P^* &= \tfrac{b\delta\beta_2 E^*}{\mu_1\left[\delta(\beta_2+\mu_1)E^*+\mu_1 H_2(\gamma+\mu)\right]}. \end{aligned}\right\} \tag{3.7}$$

where E^* is obtained by solving equation (3.8).

$$\begin{aligned} \mu_1 r_2 (\gamma + \mu) \left[\xi_5 + \delta (\beta_2 + \mu_1) E^*\right] \left[\xi_2 + \Phi(E^*)\right] \left[\xi_3 - \Phi(E^*)\right] \\ -2\left[\xi_4 + \delta^2 \beta_{11} \mu_1 (\beta_2 + \mu_1) E^*\right] \left[\xi_1 + \Phi(E^*)\right] = 0, \end{aligned} \tag{3.8}$$

where

$$\xi_1 = 1 - \tfrac{H_1}{K(\gamma+\mu)}, \qquad \xi_2 = \xi_1 + \tfrac{2H_1}{K},$$
$$\xi_3 = \xi_1 + \tfrac{2}{r_2}(\delta - r_2), \quad \xi_4 = \delta\left(b\beta_1\beta_2 + H_2\beta_{11}\mu_1\right)(\gamma + \mu),$$

$$\Phi(E^*) = -\tfrac{(\gamma+\mu+\delta)E^*}{K(\gamma+\mu)} \pm \tfrac{\Delta}{r_1},$$

$$\left.\begin{aligned} \Delta &= \sqrt{r_1^2\Delta_1^2 - \tfrac{4r_1}{K}\Delta_2 + \tfrac{4r_1^2 H_1}{K}\Delta_3}, \\ \Delta_1 &= \left[1 - \tfrac{(\gamma+\mu+\delta)E^* + H_1(\gamma+\mu)}{K(\gamma+\mu)}\right], \\ \Delta_2 &= \left(\tfrac{b\delta\beta_1\beta_2 E^*}{\mu_1\left[\delta(\beta_2+\mu_1)E^*+\mu_1 H_2(\gamma+\mu)\right]} + \tfrac{\delta\beta_{11}}{\gamma+\mu}E^*\right), \\ \Delta_3 &= \left(1 - \tfrac{(\gamma+\mu+\delta)E^*}{K(\gamma+\mu)}\right). \end{aligned}\right\} \tag{3.9}$$

The following result hold for the model.

Theorem 3.2. *The model (2.1) exhibits at least one endemic equilibrium point if*

$$r_1^2\Delta_1^2 - \frac{4r_1}{K}\Delta_2 + \frac{4r_1^2 H_1}{K}\Delta_3 > 0.$$

The Disease-Free Equilibrium Point (DFE), $\mathcal{E}_0$ is obtained by setting $E^* = 0$ in (3.7), yielding $\mathcal{E}_0 = \left(K,\ 0,\ 0,\ \frac{b}{\mu_1},\ 0\right)$

3.4. The basic reproduction number $\mathcal{R}_0$

Using the next generation matrix method, the analytical formula and numerical value for $\mathcal{R}_0$ is obtained, and this will inform the decision on the presence or otherwise of disease-free equilibrium and endemic equilibrium [18]. It will also help us to determine the spread or otherwise of the disease. To achieve this, the equations are rewritten with newly infective classes as follows;

$$\left.\begin{array}{l} \frac{dE}{dt} = r_2 E\left(1 - \frac{S+E+I}{K}\right) + \frac{(\beta_1 P + \beta_{11} I)S}{H_1+S} - \delta E, \\ \frac{dI}{dt}\, dIt = \delta E - (\gamma+\mu)I, \\ \frac{dP}{dt}\, dPt = \frac{\beta_2 IQ}{H_2+I} - \mu_1 P. \end{array}\right\} \quad (3.10)$$

Using the next generation matrix method, we have

$$\mathcal{R}_0 = \mathcal{R}_{M0} + \sqrt{\mathcal{R}_{M0}^2 + 4\mathcal{R}_{L0}\mathcal{R}_{M10}},$$

where
$\mathcal{R}_{M0} = \frac{K\beta_{11}}{2(H_1+K)(\gamma+\mu)}$, $\mathcal{R}_{M10} = \frac{K\beta_1}{(H_1+K)(\gamma+\mu)}$ and $\mathcal{R}_{L0} = \frac{b\beta_2}{H_2\mu_1^2}$.

3.5. Stability analysis of equilibrium points

The stability analysis is done using the characteristic equation obtained from the Jacobian matrix of the model equation (2.1). Here, stability analysis of the disease-free equilibrium is performed.

3.5.1. Local stability of disease-free equilibrium(DFE)

Theorem 3.3. *The disease-free equilibrium is locally asymptotically stable whenever $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. The Jacobian matrix of the system (2.1) is given by:

$$J = \begin{pmatrix} \Lambda_1 & \frac{-r_1 S}{K} & \frac{-r_1 S}{K} - \frac{S\beta_{11}}{H_1+S} & 0 & -\frac{\beta_1 S}{H_1+S} \\ \Lambda_2 & \Lambda_3 & \frac{r_2 E}{K} + \frac{\beta_{11} S}{H_1+S} & 0 & \frac{\beta_1 S}{H_1+S} \\ 0 & \delta & -(\gamma+\mu_3) & 0 & 0 \\ 0 & 0 & \frac{\beta_2 H_2 Q}{(H_2+I)^2} & \frac{\beta_2 I}{H_2+I} - \mu_4 & 0 \\ 0 & 0 & \frac{\beta_2 H_2 Q}{(H_2+I)^2} & \frac{\beta_2 I}{H_2+I} & -\mu_5 \end{pmatrix}$$

$$\Lambda_1 = r_1\left(1 - \frac{2S+E+I}{K}\right) - \frac{\beta_1 H_1 P}{(H_1+S)^2} - \frac{\beta_{11} H_1 I}{(H_1+S)^2},$$
$$\Lambda_2 = -\frac{r_2 E}{K} + \frac{(\beta_1 P + \beta_{11} I)H_1}{(H_1+S)^2},$$
$$\Lambda_3 = r_2\left(1 - \frac{S+2E+I}{K}\right) - \delta$$

Evaluating the Jacobian at the disease-free equilibrium, $\mathcal{E}_0 = (K, 0, 0, \frac{b}{\mu_1}, 0)$, we have

$$J(\mathcal{E}_0) = \begin{pmatrix} -r_1 & -r_1 & -r_1 - \frac{K\beta_{11}}{H_1+K} & 0 & -\frac{K\beta_1}{H_1+K} \\ 0 & -\delta & \frac{K\beta_{11}}{H_1+K} & 0 & \frac{K\beta_1}{H_1+K} \\ 0 & \delta & -\gamma-\mu & 0 & 0 \\ 0 & 0 & -\frac{b\beta_2}{H_2\mu_1} & -\mu_1 & 0 \\ 0 & 0 & \frac{b\beta_2}{H_2\mu_1} & 0 & -\mu_1 \end{pmatrix}$$

Clearly, two of the eigenvalues of $J(\mathcal{E}_0)$ are $-r_1$ and $-\mu_1$ and the remainder of the eigenvalues are those of the following sub-matrix;

$$J^1(\mathcal{E}_0) = \begin{pmatrix} -\delta & \frac{K\beta_{11}}{H_1+K} & \frac{K\beta_{11}}{H_1+K} \\ \delta & -\gamma-\mu & 0 \\ 0 & \frac{b\beta_2}{H_2\mu_1} & -\mu_1 \end{pmatrix}.$$

Equivalently, this can be rewritten in terms of $\mathcal{R}_{L0}, \mathcal{R}_{M0}, \mathcal{R}_{M10}$ as

$$J^1(\mathcal{E}_0) = \begin{pmatrix} -\delta & 2\mathcal{R}_{M0}(\gamma+\mu) & \mathcal{R}_{M10}(\gamma+\mu) \\ \delta & -\gamma-\mu & 0 \\ 0 & \mathcal{R}_{L0}\mu_1 & -\mu_1 \end{pmatrix}.$$

The characteristic equation of $J^1(\mathcal{E}_0)$ is given by

$$x^3 + a_1 x^2 + a_2 x + a_3 = 0,$$

where
$a_1 = \delta + \gamma + \mu + \mu_1$,
$a_2 = \delta\gamma + \delta\mu + \delta\mu_1 + \gamma\mu_1 + \mu\mu_1 - 2\mathcal{R}_{M0}\delta\gamma - 2\mathcal{R}_{M0}\delta\mu$,
$a_3 = \delta\gamma\mu_1 + \delta\mu\mu_1 - 2\mathcal{R}_{M0}\delta\gamma$.
By the Routh-Hurwitz criteria, the characteristic polynomial has all its zeros in the left half of the plane if $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1 a_2 > a_3$.

Clearly, the first condition is satisfied and the other two are satisfied whenever $\mathcal{R}_0 \leq 1$. Therefore, all the eigenvalues of the matrix $J^1(\mathcal{E}_0)$ have negative real parts whenever $\mathcal{R}_0 < 1$. Therefore, the disease-free equilibrium point (DFE), E_0 is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if otherwise, concluding the proof. □

3.6. Sensitivity analysis of model parameters

Sensitivity analysis is used to determine parameters with the most influence on the basic reproduction number. This will inform us on the significance of each parameter on the disease transmission. To achieve this, the normalized forward sensitivity index which is defined as

$$\Upsilon^{p}_{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \times \frac{p}{\mathcal{R}_0}$$

for $\mathcal{R}_0$ which differentiably depends on parameter p is used. The sensitivity indices are computed and presented in Table 2.

Table 2. Sensitivity indices for the basic parameters.

Parameter	Sensitivity indices
b	0.4370
K	0
β_1	0.4370
β_{11}	0.1259
β_2	0.4370
H_1	0
H_2	-0.4370
μ	-0.1185
μ_1	-0.4370
δ	0
r_1	0
r_2	0
γ	-0.4444

From Table 2, it can be seen that varying the infection and predation rates of maize and leafhoppers, β_1, β_{11} and β_2 respectively, and the birth rate of susceptible leafhopper b, have a direct relationship with the basic reproduction number, $\mathcal{R}_0$, while the basic reproduction number, $\mathcal{R}_0$ has an inverse relationship with the death rate of infected maize, μ, death rates of susceptible and infected leafhopper, μ_1 and removal rate of infected maize plants γ when they are varied.

3.7. Bifurcation analysis

Let $v = [v_1, v_2, v_3, v_4, v_5]$ and $w = [w_1, w_2, w_3, w_4, w_5]^T$ respectively be the left and right eigenvectors for the jacobian $J(\mathcal{E}_0)$. Then

$$\begin{aligned} w_1 &= -\frac{(H_1+K)r_1w_2(\gamma+\mu)+\delta w_2((\beta_1\beta_2 b+\beta_{11})K+(H_1+K)r_1)}{(\gamma+\mu)(H_1+K)r_1}, \\ w_3 &= \frac{\delta w_2}{\gamma+\mu}, w_4 = -\frac{\beta_2 b\delta w_2}{{\mu_1}^2H_2(\gamma+\mu)}, w_5 = \frac{\beta_2 b\delta w_2}{\mu_1^2H_2(\gamma+\mu)} \\ v_1 &= 0,\ v_3 = v_2,\ v_4 = 0,\ v_5 = \frac{\beta_1 K v_2}{(H_1+K)\mu_1} \end{aligned}$$

where w_2 and v_2 are selected such that $\mathbf{w}\cdot\mathbf{v} = \mathbf{1}$.

The bifurcation coefficients are thus obtained using:

$$\mathbf{a} = \sum_{i,j,k=1}^{n} v_k w_i w j \frac{\partial^2 f_k}{\partial x_i \partial\beta^*} \text{ and } \mathbf{b} = \sum_{i,k=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial\beta^*}.$$

Hence

$$\mathbf{a} = -2\, v_2 w_2 \left\{ \eta_1 \left(\frac{\delta\beta_{11}H_1(\mu_1^2H_2-\beta_2 b)}{\mu_1^2H_2(H_1+K)^2(\gamma+\mu)} - \frac{r_2}{K} \right) + \frac{w_2 r_2(\gamma+\mu+\delta)}{K(\gamma+\mu)} + \frac{\beta_1 K\delta^2 w_2\beta_2 b(\mu_1+\beta_2)}{(H_1+K)(\gamma+\mu)^2\mu_1^3{H_2}^2} \right\} < 0,$$

and

$$\mathbf{b} = v_2 w_2 \left(\frac{\delta(\mu_1^2H_2+\beta_2 b)\beta_1 H_1}{\mu_1^2H_2(\gamma+\mu)(H_1+K)^2} - 2\frac{\eta\beta_1 H_1}{(H_1+K)^2} + \frac{\eta r_2}{K} - \frac{r_2(4\gamma+4\mu+K\delta)}{K^2(\gamma+\mu)} - \frac{((2\mu_1+\beta_2)b-\mu_1^2H_2)\beta_1 K\delta\beta_2}{\mu_1^3{H_2}^2(H_1+K)(\gamma+\mu)} \right).$$

Where

$$\eta_1 = \frac{(H_1+K)r_1w_2\mu_1^2H_2(\gamma+\mu)+\delta w_2(\beta_1 K\beta_2 b+(r_1H_1+r_1K+\beta_{11}K)\mu_1^2H_2)}{\mu_1^2H_2(\gamma+\mu)(H_1+K)r_1}$$

$$\eta = \frac{\mu_1^2H_2(\gamma+\mu)(H_1+K)r_1+\delta(\beta_1 K\beta_2 b+(r_1H_1+r_1K+\beta_{11}K)\mu_1^2H_2)}{\mu_1^2H_2(\gamma+\mu)(H_1+K)r_1}$$

Clearly $\mathbf{a} < 0$ and therefore, the nature of the bifurcation depends on the sign of $\mathbf{b}$. The following hold concerning $\mathbf{b}$.

1. $b > 0$ if and only if

$$\frac{\delta(\mu_1^2H_2+\beta_2 b)\beta_1 H_1}{\mu_1^2H_2(\gamma+\mu)(H_1+K)^2} + \frac{\eta r_2}{K} > 2\frac{\eta\beta_1 H_1}{(H_1+K)^2} + \frac{r_2(4\gamma+4\mu+K\delta)}{K^2(\gamma+\mu)} + \frac{((2\mu_1+\beta_2)b-\mu_1^2H_2)\beta_1 K\delta\beta_2}{\mu_1^3{H_2}^2(H_1+K)(\gamma+\mu)}.$$

2. $\mathbf{b} < 0$ if and only If

$$\frac{\delta(\mu_1^2H_2+\beta_2 b)\beta_1 H_1}{\mu_1^2H_2(\gamma+\mu)(H_1+K)^2} + \frac{\eta r_2}{K} < 2\frac{\eta\beta_1 H_1}{(H_1+K)^2} + \frac{r_2(4\gamma+4\mu+K\delta)}{K^2(\gamma+\mu)} + \frac{((2\mu_1+\beta_2)b-\mu_1^2H_2)\beta_1 K\delta\beta_2}{\mu_1^3{H_2}^2(H_1+K)(\gamma+\mu)}$$

The following results establish theorem 3.4.

Theorem 3.4. *Whenever* $\boldsymbol{b} > 0$ *the system exhibits forward bifurcation and whenever* $\boldsymbol{b} < 0$ *the system exhibits backward bifurcation.*

3.8. Numerical simulation

In order to show the impact of each parameter on the spread or otherwise of the disease, simulation on the basic parameters is performed by varying the values of the rate of predation and infection of susceptible maize plants by infected leafhopper, β_1 , rate of predation and infection of exposed maize plants by infected leafhopper, β_{11} , rate of infection of leafhopper by infected maize, β_2 , natural death rate in leafhoppers, μ_1 , removal rate of infected maize, γ and recruitment rate of susceptible leafhoppers, b , while keeping all other parameter values constant. The results of the simulations are presented in Figures 2, 3, 4, 5, 6, 7 and 8. Figure 2 shows a time series plot of susceptible, exposed, infected maize, and susceptible and infected leafhopper . It is observed that the susceptible maize population decreases exponentially towards the endemic equilibrium point as the population of exposed and infected plants increases. The exposed maize population however, increases exponentially towards the endemic equilibrium point. This is due to the presence of the disease in maize and leafhopper population. Again, the infected maize population marginally increases with time.

The susceptible leafhopper population decreases exponentially to the endemic equilibrium point due to natural death and infections from infected maize and MSV from the environment. The infected leafhopper population on the other hand, increases exponentially to form a parabolic curve to the endemic equilibrium point and then decreases over time. Figure 3 shows time series

Table 3. Parameter values for the MSVD model.

Parameter Symbol	Value day^{-1}	Source
b	0.02	[14]
K	10000	[14]
β_1	0.45	[15]
β_{11}	0.04	Assumed
β_2	0.04	[15]
H_1	0.4	[14]
H_2	0.6	[14]
μ	0.008	[15]
μ_1	0.0303	[14]
δ	0.018	[15]
r_1	0.005	[14]
r_2	0.002	Assumed
γ	0.03	[19]

plots of susceptible, exposed and infected maize plants with different values of the rate of predation and infection of susceptible maize plants by infected leafhopper β_1. It is observed that, increasing (decreasing) the infection rate of infected leafhopper on susceptible maize plant β_1, decreases (increases) the susceptible, but increases (decreases) the exposed and infected populations. Figure 4 shows time series plots of susceptible, exposed and infected maize with different values of the rate of predation and infection of exposed maize plants by infected leafhopper, β_{11}. It

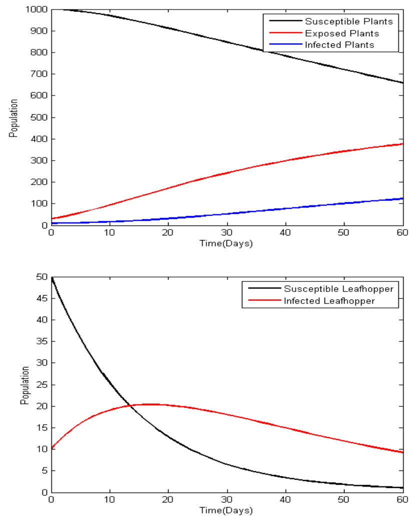

Figure 2. Time series plots of Susceptible, Exposed and Infected maize as well as Susceptible and Infected Leafhopper.

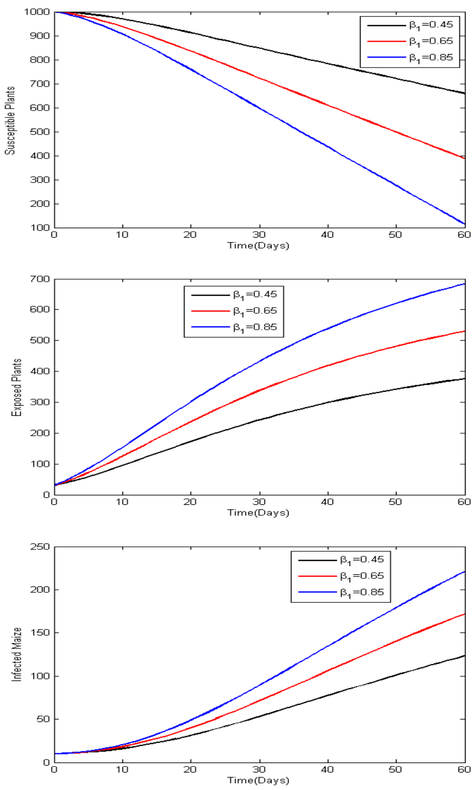

Figure 3. Time series plots of Susceptible, Exposed and Infected maize with different values of β_1.

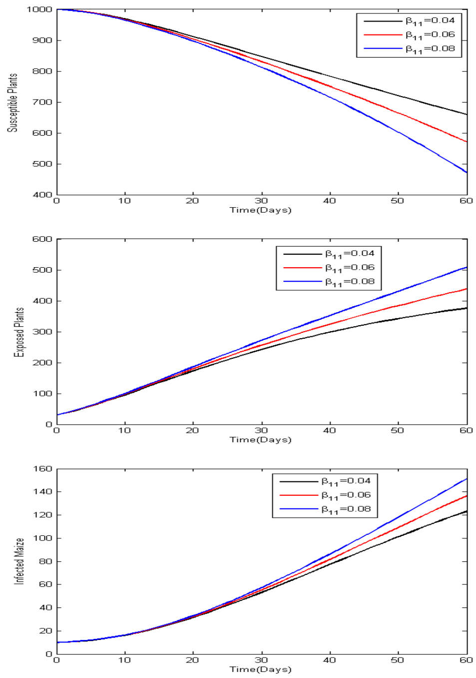

Figure 4. Time series plots of Susceptible, Exposed and Infected maize with different values of β_{11}.

can be seen that, increasing (decreasing) infection rate of exposed maize plant on infected leafhopper also decreases (increases) the susceptible, but increases (decreases) the exposed and infected populations.

Figure 5 shows time series plots of susceptible, exposed and infected maize with different values of the removal rate of infected maize, γ. It is observed that increasing (decreasing) the value of γ increases (increases) the population of susceptible maize plants but decreases (increases) the population of the exposed and infected maize plants. Figure 6 shows time series plots of susceptible, exposed and infected maize with different values of the recruitment rate of susceptible leafhoppers, b. It is observed that increasing (decreasing) b reduces (raises) the population of susceptible maize plants but increases (decreases) the population of exposed and infected maize plants.

Figure 7 shows time series plots of susceptible, exposed and infected maize plants with different values of the rate of infection of leafhopper by infected maize, β_2. It is observed that, increasing (decreasing) the infection rate of Susceptible leafhopper on infected maize plant β_2 increases (decreases)

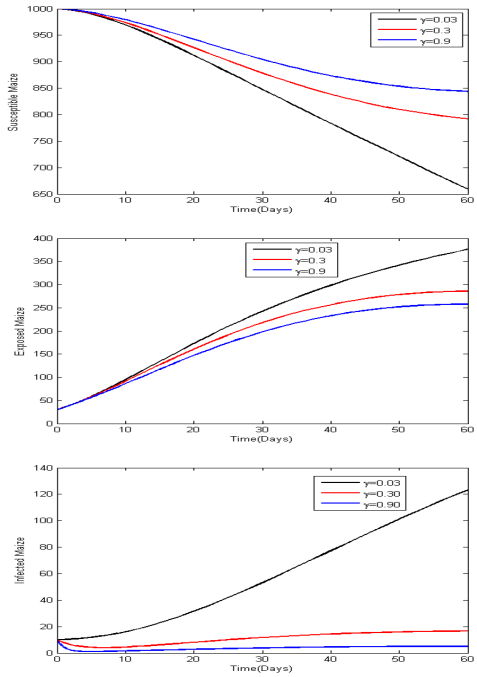

Figure 5. Time series plots of Susceptible, Exposed and Infected Maize with different values of γ.

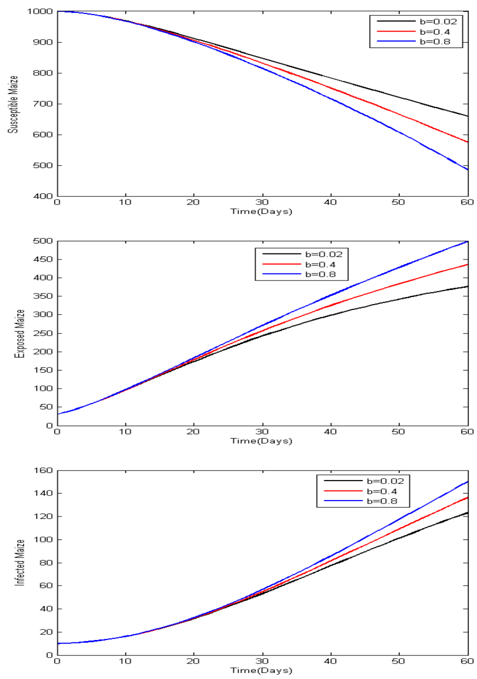

Figure 6. Time series plots of Susceptible, Exposed and Infected Maize with different values of b.

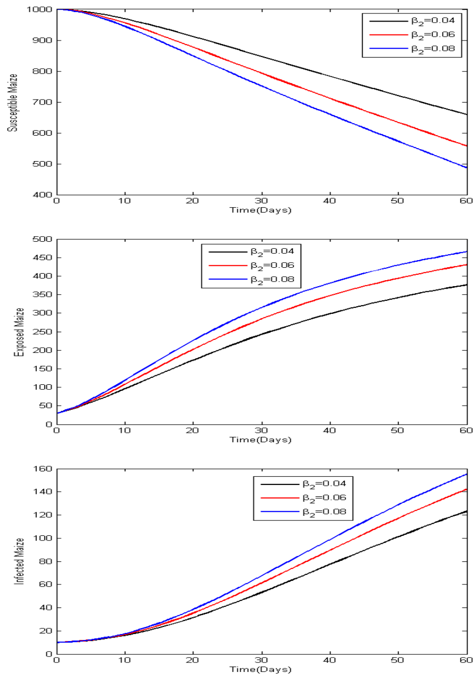

Figure 7. Time series plots of Susceptible, Exposed and Infected Maize with different values of β_2.

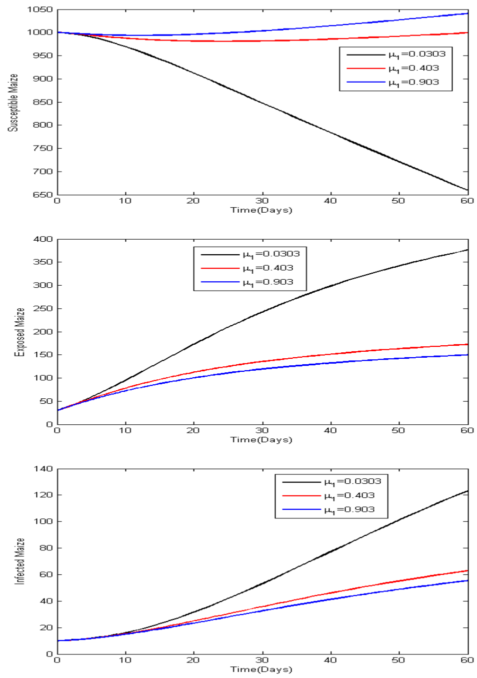

Figure 8. Time series plots of Susceptible, Exposed and Infected maize with different values of μ_1.

the population of exposed and infected maize plants but it leads to a decrease in the population of susceptible maize plants.

Figure 8 shows time series plots of susceptible, exposed and infected maize with different values of natural death rate in leafhoppers, μ_1. It is observed that, increasing (decreasing) the death rate of susceptible leafhopper increases (decreases) the population of susceptible maize but decreases the population of both the exposed and infected maize. There will be same effect when the death rate of the infected leafhopper is varied.

4. Summary and conclusions

The study proposed and analyzed a mathematical model of the dynamics of maize streak virus disease (MSVD). It showed that the system was uniformly bounded and positive. The disease-free and endemic equilibrium points and their local stability analysis was also carried out. In the model analysis, sensitivity analysis of each parameter was carried out to give a clear picture of the impact of each factor on the spread or otherwise of the disease. Finally, analytical results were confirmed by numerical simulations with realistic parameter values. It showed that varying the infection and predation rates of maize and leafhoppers, β_1, β_{11} and β_2 respectively, and the birth rate of susceptible leafhopper b, have a direct relationship with the basic reproduction number $\mathcal{R}_0$, while varying the death rate of infected maize, μ, death rate of susceptible and infected leafhopper, μ_1 and removal rate of infected maize plants have an inverse relationship with the basic reproduction number $\mathcal{R}_0$. The model shows that the spread of the disease largely depends on how these parameters are manipulated. We conclude that parameters which are directly related to the reproduction number must be minimized while we consider increasing those that are inversely related to the reproduction number.

Data availability

The data used in this study are from literature and they have been duly cited in Table 2 and relevant places in this paper.

Conflict of interest

All authors declare no conflicts of interest in this paper.

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