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## A MATHEMATICAL MODEL FOR THE MLND DYNAMICS AND SENSITIVITY ANALYSIS IN A MAIZE POPULATION

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**ABSTRACT.** Maize Lethal Necrosis disease (MLND) is a viral disease that can cause fatal damage to the crop of maize plants. This is very common in East Africa countries and Democratic Republic of Congo (DRC). In this manuscript, a mathematical model has been developed to study and analyze the dynamics of the MLND in the maize crop population. The disease free (DFE) and endemic equilibrium (EE) points of the model has been computed and the basic reproduction number ( $R_0$ ) derived using the next generation matrix method. We performed sensitivity analysis by using parametric values from literature and estimated ones. We found that the rates of transmission,  $\lambda_{mo}$ ,  $\beta_o$  and  $\beta_{mm}$  are the most positively sensitive parameters. Numerical simulations were also performed to verify the analytical results. Thus, this research work recommends that deliberate strategic intervention should be targeted on the disease transmission rates which are significant for MLND transmission in order to eradicate the disease or reduce the intensity of the disease transmission in the maize population.

### 1. INTRODUCTION

Research teams from various countries in Africa are under pressure of finding solution to threaten food security which is due maize lethal necrosis diseases (MLND). MLND is a new serious disease which emerged for the first time in Kenya in 2011 and spread to Tanzania and Uganda in the year 2012 [1]. In these occasions a number of damage and losses were recorded. The disease is caused by maize chlorotic mottle virus (MCMV) and sugarcane mosaic virus (SCMV) [2] or co infections of MCMV and any other types of viruses such as Wheat Streak Mosaic Virus (WSMV) or Maize Dwarf Mosaic Virus (MDMV) [2] form MLND.

The transmission of MLND is attributable to various insects such as maize thrips, rootworms, leaf beetles and leaf hoppers. However, research findings indicate the disease transmission through seeds normally occurs at very low rates [2]. Also, the MLND virus may spread through soil, infected plant debris and aphid vectors [3, 4, 5].

Mathematical modeling has also been a significant mainstay in the development of strategic

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*Key words and phrases.* Maize, Dynamics, Sensitivity, Parameters, Model, Analysis.

intervention programs towards disease control in the plant crops population. Several deterministic models have been established to describe and analyze the dynamics of diseases in plant populations, but few correlate to this studies. Thus mathematical modeling has not fully been employed as an alternative way to describe and analyze the dynamics of the disease under consideration in our case. We present a deterministic model to study and analyze the dynamics of MNLD in the maize population in this paper. We believe that the results of our research work will be useful in finding suitable means of controlling the disease transmission, or rather eradicate it. This may ensure farmers and peasants maximum maize harvest for food security.

## 2. MODEL FORMULATION

This paper presents an SI-SEI-type model of host and vector populations that incorporates: The maize host population, which is categorized as Susceptible sub-population ( $S_m$ ); Exposed maize sub-population ( $E_m$ ) and Infected maize sub-population ( $I_m$ ). The model includes: the vector population, which is categorized as Susceptible vector sub-population ( $S_v$ ) and Infected vector sub-population ( $I_v$ ). It also includes: virus in the environment ( $P_o$ ). In the model, there is no recruitment rate since it describes a single season and no plantation of maize plants. The class  $S_m$  declines constantly at the rate ( $\eta$ ), which is due to force of infection between virus in the environment ( $P_o$ ); direct contact of maize to maize ( $I_m$ ) and vector from infected maize plants ( $I_v$ ). The infected maize plants finally increases the number of exposed plants ( $E_m$ ) at the same constant rate ( $\eta$ ). However, some susceptible maize plants remain in the exposed state while the remaining ones immediately progress to infected maize class, ( $I_m$ ) at a constant rate ( $\alpha_m$ ). Then the infected maize has disease-induced constant death rate  $\sigma_m$ . There is no natural death in the host plants population for all three classes as maize plants never naturally die till harvested at the end of season. The infected maize and the environment transmit the MNLD virus to the susceptible vector,  $S_v$  at constant rates ( $\beta_{mv}$ ) and ( $\beta_o$ ) respectively. The susceptible vectors are constantly recruited at the rate  $\Lambda$ ; constantly progress to infected vector class with mass action  $\lambda(P_o, I_m)$ , with proportionality constant rate  $\lambda$  and die naturally at a constant rate  $\mu_v$ . The infected vectors also naturally die at the same constant rate  $\mu_v$ . The MNLD virus in the environment are recruited at the rate proportional to ( $\theta P_o$ ) and from the infected maize plants at a constant rate ( $\sigma$ ), and then die naturally at the rate ( $\mu_o$ ).

In the formulation of our model the following assumptions were considered:

- i Pathogen from infected maize plants reaches the vegetative environment by vectors and through shedding which is due to wind, people, rain and birds.
- ii Transmission of infection from the vegetative environment to susceptible maize is through vectors and shedding which is due to wind, people, rain and birds.

- iii Some maize plants are exposed but resist the MLND throughout the season.
- iv Once the vectors become the carriers of pathogens, it is for the whole of their life.
- v The population is heterogeneous. That is to say, the individuals which compose the population can be grouped into different classes in accordance to their epidemiological state.
- vi Each susceptible individual in the class has equal chance to be infected by contagious individuals if it happens they come in contact.
- vii There are no immigrants and expatriates (emigrants). The only pathway of entering into the population is through sowing seeds and the only way of exit is through death from MLND-related causes.

2.1. **Model variables and parameters.** A complete explanation of the variables and parameters that are used in the model are summarized and described in Tables 1 and 2, respectively.

Table 1: Variables and their description

Variable	Description	Units
$N_m$	Total number of maize plants population	plant $ha^{-1}$
$N_v$	Total number of vector population	vector $ha^{-1}$
$S_m$	Susceptible population maize plants	plant $ha^{-1}$
$S_v$	Susceptible vector population	vector $ha^{-1}$
$I_m$	Infected population maize plants	plant $ha^{-1}$
$I_v$	Infected vector population for maize plants	vector $ha^{-1}$
$E_m$	Exposed maize plants with latent incubation period	plant $ha^{-1}$
$P_o$	Other plants carrier of pathogens (vegetative environment)	cells $ha^{-1}$

Table 2: Parameters and their description

Parameter	Description	Units
$\beta_{mv}$	Contact rate from infected maize to susceptible vector population	ha $cell^{-1} day^{-1}$
$\mu_o$	Natural death rate of vector within environment	ha $plants^{-1} day^{-1}$
$\beta_o$	Contact rate of other plants (not maize) carrying pathogen with maize	ha $cell^{-1} day^{-1}$
$\beta_{ov}$	Contact rate of virus in the environment to the	

*Continued on next page*

Table 2 – *Continued from previous page*

Parameter	Description	Units
	susceptible vectors	ha $cell^{-1} day^{-1}$
$\beta_{vm}$	Contact rate from virus carrying vectors to $S_m$ maize population	ha $cell^{-1} day^{-1}$
$\beta_{mm}$	Contact rate of contagious maize to susceptible maize population	ha $cell^{-1} day^{-1}$
$\lambda_{vo}$	Contact rate from virus carrying vectors to the vegetative environment	ha $cell^{-1} day^{-1}$
$\lambda_{mo}$	Contact rate from infectious maize plants to the vegetative environment	ha $cell^{-1} day^{-1}$
$\Lambda$	Recruitment rate into susceptible vector population	cells $day^{-1}$
$\eta$	Force of infection rate of 3 masses of action due vectors, $I_m$ and virus in $P_o$	ha $cell^{-1} day^{-1}$
$\lambda$	Force of infection rate of combined masses of action due infected maize and virus in the environment.	ha $cell^{-1} day^{-1}$
$\mu_v$	Natural death rate of vector population	ha $plants^{-1} day^{-1}$
$\sigma_m$	Death rate due maize infection	$plants^{-1} day^{-1}$
$\sigma$	Contribution of infected maize to the growth of viruses.	cells $plants^{-1} day^{-1}$
$\theta$	Contribution of infected plants to the growth of viruses in the environment	cells $plants^{-1} day^{-1}$
$\alpha_m$	Progression rate of exposed maize plant to be infectious	plant $day^{-1}$

**2.2. Model flow chart.** Using assumptions of the model and defined variables/parameters, the dynamics of maize lethal necrosis disease (MLND) in maize population can be shown as the flow chart in Figure 1 depicts.

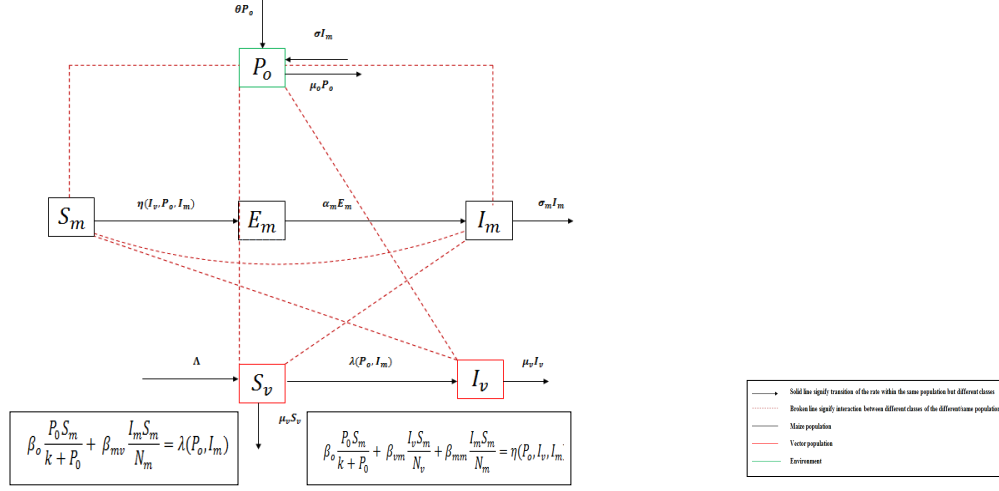


FIGURE 1. Schematic of MLND with susceptible maize plants population which interacts with forces of infection from viruses in the environment ( $P_o$ ), infected maize ( $I_m$ ) and infected vector ( $I_v$ ) populations. Epidemic starts from susceptible maize ( $S_m$ ) and moves to exposed class ( $E_m$ ) and infected class ( $I_m$ ) respectively.

**2.3. Equations of the model.** Considering the compartmental diagram as described by Figure 1, we formulate basic mathematical model which shows transmission dynamics for MLND using the following differential equations:

$$\begin{aligned}
 (1) \quad & \frac{dS_m}{dt} = -\left(\beta_o \frac{P_o}{k + P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m}\right) S_m, \\
 (2) \quad & \frac{dE_m}{dt} = \left(\beta_o \frac{P_o}{k + P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m}\right) S_m - \alpha_m E_m, \\
 (3) \quad & \frac{dI_m}{dt} = \alpha_m E_m - \sigma_m I_m, \\
 (4) \quad & \frac{dS_v}{dt} = \Lambda - \left(\beta_{ov} \frac{P_o}{k + P_o} + \beta_{mv} \frac{I_m}{N_m}\right) S_v - \mu_v S_v, \\
 (5) \quad & \frac{dI_v}{dt} = \left(\beta_{ov} \frac{P_o}{k + P_o} + \beta_{mv} \frac{I_m}{N_m}\right) S_v - \mu_v I_v, \\
 (6) \quad & \frac{dP_o}{dt} = \lambda_{mo} \frac{I_m}{N_m} + \lambda_{ov} \frac{I_v}{N_v} - \mu_o P_o,
 \end{aligned}$$

with initial conditions:

$$S_m > 0, E_m \geq 0, I_m \geq 0, P_o \geq 0, S_v \geq 0, I_v \geq 0.$$

Note that the total number of maize plants population is given by

$$(7) \quad N_m = S_m + E_m + I_m,$$

while the total number of vector population is given by

$$(8) \quad N_v = S_v + I_v.$$

**2.4. Basic properties of the model.** In order to see if the model is mathematically and epidemiologically well posed to study the MLND dynamics, we considered two basic properties of the model. We used Box invariant of Metzler Matrix to show the existence of invariant region [6, 7] and basic standard method to prove the positivity of solutions.

**2.4.1. Invariant region.** We proof the invariant region as follows: The system of the model (1) can be written as

$$\frac{dX}{dt} = A(X)X + F \text{ with } X = (S_m, E_m, I_m, S_v, I_v, P_o)^T \text{ and the constant term } F = (0, 0, 0, \lambda, 0, 0)^T.$$

That is

$$A(X) = \begin{pmatrix} -\mathcal{F}_m & 0 & 0 & 0 & 0 & 0 \\ \mathcal{F}_m & -\alpha_m & 0 & 0 & 0 & 0 \\ 0 & \alpha_m & -\sigma_m & 0 & 0 & 0 \\ 0 & 0 & 0 & -\mathcal{F}_v - \mu_v & 0 & 0 \\ 0 & 0 & 0 & \mathcal{F}_v & -\mu_v & 0 \\ 0 & 0 & \frac{\lambda_{mo}}{N_m} & 0 & \frac{\lambda_{ov}}{N_m} & -\mu_o \end{pmatrix}$$

and

$$\mathcal{F}_m = (\beta_o \frac{P_o}{k + P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m}),$$

$$\mathcal{F}_v = (\beta_{ov} \frac{P_o}{k + P_o} + \beta_{mv} \frac{I_m}{N_m}).$$

$A(X)$  is a Metzler Matrix  $\forall X \in R_+^6$  which all off diagonal terms are non negative and  $F \geq 0$ . The system  $\frac{dX_m}{dt} = A(X)X + F$  is positive invariant in  $R_+^6$ . We can conclude that the feasible region  $\Omega$  is a set of  $\Omega = (S_m, E_m, I_m, S_v, I_v, P_o) \in R_+^6$  with initial data  $S_m > 0, E_m \geq 0, I_m \geq 0, S_v \geq 0, I_v \geq 0, P_o \geq 0$ . Hence, the solution stays in the region if it started in the region.

Similarly, letting the initial data be  $\{S_m(0), E_m(0), I_m(0), I_v(0), P_o(0)\} \in \Omega$  and using standard method, the solution set  $\{S_m, E_m, I_m, S_v, I_v, P_o\}$  of the model system (1) was shown to be non negative  $\forall t \geq 0$ . It was therefore concluded that the solution set  $\Omega = \{S_m, E_m, I_m, S_v, I_v, P_o\}$  of the model system (1) is non-negative for all  $t > 0$  and so it is sufficient to study the dynamics of MLND.



### 3. MODEL ANALYSIS

**3.1. Existence of disease free equilibrium point.** The disease free equilibrium (DFE) point of the model system (1) is given by

$$(9) \quad E^0 = (S_m, E_m, I_m, S_v, I_v, P_o) = (N_m, 0, 0, \frac{\Lambda}{\mu_v}, 0, 0).$$

When there is no disease, there will be susceptible maize population plants,  $N_m$ . On the other hand in the susceptible vector population there will be new reproduction of vectors  $\Lambda$  and dying  $\mu_v$ ,

where  $N_m$  is the constant with adoption value of 44,000 [8] maize population in one hector.

**3.2. The basic reproduction number  $R_o$ .** Analysis of the equilibrium point, is carried out by considering spectral radius of the matrix  $FV^{-1}$  called threshold quantity. The basic reproduction number is the measure of secondary contagious number as a results of one contagious maize plant in the susceptible maize population.

With the help of Next Generation Matrix, the approach adopted, we determine the basic reproduction number [9]. We consider the population distinguished by six different classes which are  $S_m, E_m, I_m, S_v, I_v$  and  $P_o$ .

Firstly we arrange the system to get group of infectious classes only that is  $(E_m, I_m, I_v, P_o)$ . Once again we assume  $f_j(x)$  be the rate of introduction of new infectious (transmission) in compartment i,  $v_j^+(x)$  be the transmission after new infectious (transition rate by all other means) and  $v_j^-(x)$  rate of transfer of individual out of compartment j.

The disease transmission model comprised of the system of equations

$$X_j' = f_j(x) - v_j(x),$$

where

$$v_j(x) = v_j^+(x) - v_j^-(x).$$

To obtain the matrices  $F$  and  $V$  of dimension 'n'by'n' we differentiate vectors  $f_j(x)$  and  $v_j(x)$  respectively.

$$F = \left( \frac{\partial f_j(x)}{\partial x_i} \right),$$

$$V = \left( \frac{\partial v_j(x)}{\partial x_i} \right),$$

with

$$1 \leq j, i \leq n,$$

where  $x_o$  is a vector column from DFE. From the model equations

$$(f_j(x_o)) = \begin{pmatrix} F_m \\ 0 \\ F_v \\ 0 \end{pmatrix} \text{ and, } (v_j(x_o)) = \begin{pmatrix} \alpha_m E_m \\ -\alpha_m E_m + \sigma_m I_m \\ \mu_v S_v \\ \lambda_{mo} \frac{I_m}{N_m} + \lambda_{ov} \frac{I_v}{N_v} - \mu_o P_o \end{pmatrix},$$

$$F_m = (\beta_o \frac{P_o}{k+P_o} + \beta_{vm} \frac{I_v}{N_v} + \beta_{mm} \frac{I_m}{N_m}) S_m,$$

$$F_v = (\beta_{ov} \frac{P_o}{k+P_o} + \beta_{mv} \frac{I_m}{N_m}) S_v.$$

Differentiate  $(F_m, F_v, 0, 0)^T$  with respect to  $(E_m, I_m, I_v, P_o)^T$  and obtain

$$F = \begin{pmatrix} 0 & \beta_{mm} \frac{S_m}{N_m} & \beta_{vm} \frac{S_m}{N_v} & \beta_o \frac{k S_m}{(k+P)^2} \\ 0 & 0 & 0 & 0 \\ 0 & \beta_{mv} \frac{S_v}{N_m} & 0 & \beta_{ov} \frac{k S_v}{(k+P)^2} \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

At disease free  $S_m = N_m$  which is the number of maize grains sowed and germinated in a maize plant, and  $S_v = \frac{\Lambda}{\mu_v}$  as from

$$\frac{dS_v}{dt} = \Lambda - (\beta_{ov} \frac{P_o}{k+P_o} + \beta_{mv} \frac{I_m}{N_m}) S_v - \mu_v S_v.$$

Hence matrix  $F$  reduces to

$$F = \begin{pmatrix} 0 & \beta_{mm} & \frac{\beta_{vm} N_m}{N_v} & \frac{\beta_o N_m}{k} \\ 0 & 0 & 0 & 0 \\ 0 & \beta_{mv} \frac{\Lambda}{N_m \mu_v} & 0 & \beta_{ov} \frac{\Lambda}{\mu_v K} \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

$$\text{Again differentiate } v_j(x_o) = \begin{pmatrix} \alpha_m E_m \\ -\alpha_m E_m + \sigma_m I_m \\ \mu_v S_v \\ \lambda_{mo} \frac{I_m}{N_m} + \lambda_{ov} \frac{I_v}{N_v} - \mu_o P_o \end{pmatrix}$$

with respect to  $(E_m, I_m, I_v, P_o)^T$  to obtain matrix  $V$ .

Hence,

$$V = \begin{pmatrix} \alpha_m & 0 & 0 & 0 \\ -\alpha_m & \sigma_m & 0 & 0 \\ 0 & 0 & \mu_v & 0 \\ 0 & -\frac{\lambda_{mo}}{N_m} & -\frac{\lambda_{ov}}{N_v} & \mu_o \end{pmatrix}.$$

We find the inverse of  $V$  i.e

$$V^{-1} = \begin{pmatrix} \frac{1}{\alpha_m} & 0 & 0 & 0 \\ \frac{1}{\alpha_m} & \frac{1}{\sigma_m} & 0 & 0 \\ 0 & 0 & \frac{1}{\mu_v} & 0 \\ \frac{\lambda_{mo}}{N_m \sigma_m \mu_o} & \frac{\lambda_{mo}}{N_m \sigma_m \mu_o} & \frac{\lambda_{ov}}{N_v \mu_v \mu_o} & \frac{1}{\mu_v} \end{pmatrix}.$$

And multiplying matrix  $F$  and  $V^{-1}$  we obtain

$$FV^{-1} = \begin{pmatrix} \frac{\beta_{mm}}{\sigma_m} + \frac{\beta_o \lambda_{mo}}{k \sigma_m \mu_o} & \frac{\beta_{mm}}{\sigma_m} + \frac{\beta_o \lambda_{mo}}{k \sigma_m \mu_o} & \frac{\beta_{vm} N_m}{N_v \mu_v} + \frac{\lambda_{vo} \beta_o N_m}{k N_v \mu_v \mu_o} & \frac{\beta_o N_m}{k \mu_o} \\ 0 & 0 & 0 & 0 \\ \frac{\beta_{mv} \Lambda}{N_m \sigma_m \mu_v} + \frac{\lambda_{mo} \beta_{ov} \Lambda}{k N_m \sigma_m \mu_v \mu_o} & \frac{\beta_{mv} \Lambda}{N_m \sigma_m \mu_v} + \frac{\lambda_{mo} \beta_{ov} \Lambda}{k N_m \sigma_m \mu_v \mu_o} & \frac{\lambda_{vo} \beta_{ov} \Lambda}{k N_v \mu_v^2 \mu_o} & \frac{\beta_{ov} \Lambda}{k \mu_v \mu_o} \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

According to [10] the matrix can be reduced into 2 by 2 matrix. [11] The dominant eigenvalue of this matrix is  $R_o$ , which can be obtained from the trace and determinant of that matrix as

$$K = \begin{pmatrix} \frac{\beta_{mm}}{\sigma_m} + \frac{\beta_o \lambda_{mo}}{k \sigma_m \mu_o} & \frac{\beta_{vm} N_m}{N_v \mu_v} + \frac{\beta_o N_m \lambda_{vo}}{k N_v \mu_v \mu_o} \\ \frac{\beta_{mv} \Lambda}{N_m \mu_v \sigma_m} + \frac{\beta_{ov} \Lambda \lambda_{mo}}{k \mu_v N_m \sigma_m \mu_o} & \frac{\beta_{ov} \Lambda \lambda_{vo}}{k \mu_o N_v \mu_v^2} \end{pmatrix}.$$

$$Trace(K) = \frac{\beta_{mm}}{\sigma_m} + \frac{\beta_o \lambda_{mo}}{k \sigma_m \mu_o} + \frac{\lambda_{vo} \beta_{ov} \Lambda}{k N_v \mu_v^2 \mu_o}$$

or in a simple form:

$$Trace(K) = \frac{N_m \mu_v^2 (k \beta_{mm} \mu_o + \beta_o \lambda_{mo}) + \lambda_{vo} \beta_{ov} \Lambda}{k N_v \mu_v^2 \mu_o}.$$

$$Det(K) = - \frac{\Lambda (k \beta_{mv} \beta_{vm} \mu_o - \beta_{mm} \beta_{ov} \lambda_{vo} + \beta_{mv} \beta_o \lambda_{vo} + \beta_{ov} \beta_{vm} \lambda_{mo})}{k \mu_o N_v \mu_v^2 \sigma_m}.$$

But according to [10] we have this formula for

$$R_o = \rho(K) = \frac{1}{2} \left( Trace(K) + \sqrt{Trace(K)^2 - 4det(K)} \right).$$

We substitute trace and determinant in the formula above and obtain a condensed form as shown below.

$$R_o = \rho(K) = \frac{1}{2} \left( \frac{\beta_{mm}}{\sigma_m} + \frac{\beta_o \lambda_{mo}}{k \sigma_m \mu_o} + \frac{\Lambda \lambda_{vo} \beta_{ov}}{N_v k \mu_v^2 \mu_o} \right)$$

$$(10) \quad + \frac{1}{2} \sqrt{\left( \frac{\beta_{mm}}{\sigma_m} + \frac{\beta_o \lambda_{mo}}{k \sigma_m \mu_o} + \frac{\Lambda \lambda_{vo} \beta_{ov}}{N_v k \mu_v^2 \mu_o} \right)^2 + 4 \frac{\Lambda}{N_v} \left( \frac{k \beta_{mv} \beta_{vm} \mu_o - \beta_{mm} \beta_{ov} \lambda_{vo} + \beta_{mv} \beta_o \lambda_{vo} + \beta_{ov} \beta_{vm} \lambda_{mo}}{k \mu_o \mu_v^2 \sigma_m} \right)}.$$

**3.3. Endemic equilibrium point.** Endemic equilibrium point of the model system (1)  $E^*$  is a steady state solution where by the disease persists in the population or an equilibrium comprised of diseased sub-populations i.e.  $E_m \neq 0$ ,  $I_m \neq 0$ ,  $I_v \neq 0$  and  $P_o \neq 0$ . By equating all expressions for the equations in the model system (1) to zero and let  $E^* = (S_m^*, E_m^*, I_m^*, S_v^*, I_v^*, P_o^*)$  we compute the system and arrive to the cubic polynomial equation as follows.

$$(11) \quad P(I^*) = AI^{*3} + BI^{*2} - DI^* - C$$

where

$$\begin{aligned}
A &= \beta_{mm}\mu_v^2\mu_o N_v \lambda_{mo}(\alpha_m + \sigma_m), \\
B &= (\alpha_m + \sigma_m)\beta_o\mu_v^2\mu_o N_v^2 N_m \lambda_{mo} + \beta_{mv}\mu_v\mu_o N_m \lambda_{mo}\Lambda(\alpha_m + \sigma_m) + \beta_{mm}\mu_v^2\mu_o^2 N_v^2 N_m k(\alpha_m + \sigma_m) \\
&\quad + \beta_{mm}\mu_o\mu_v N_m N_v \lambda_{ov}\Lambda(\alpha_m + \sigma_m) > -\beta_{mm}\mu_v^2\mu_o N_m N_v \lambda_{mo}\alpha_m - \sigma_m\mu_v^2\mu_o N_v^2 \alpha_m \lambda_{ov}, \\
D &= -\beta_o\mu_o\mu_v N_m^2 N_v \lambda_{ov}\Lambda(\alpha_m + \sigma_m) + \beta_{mv}\mu_v\mu_o^2 N_m^2 N_v \Lambda\alpha_m k(\alpha_m + \sigma_m) \\
&\quad - \beta_{mv}\mu_o N_m^2 \Lambda^2 \alpha_m k(\alpha_m + \sigma_m) - \beta_o\mu_v^2\mu_o N_m^2 N_v^2 \lambda_{mo}\alpha_m \\
&\quad - \beta_{mm}\mu_v\mu_o N_m^2 N_v \Lambda\alpha_m - \beta_{mm}\mu_v^2\mu_o^2 N_m^2 N_v^2 k\alpha_m \\
&\quad - \mu_v\mu_o N_m^2 N_v \lambda_{mo}\Lambda\sigma_m\alpha_m - \mu_v^2\mu_o^2 N_m^2 N_v^2 k\sigma_m\alpha_m, \\
C &= -\beta_o\mu_v\mu_o N_m^3 N_v \lambda_{ov}\Lambda\alpha_m - \beta_{mv}\mu_v\mu_o^2 N_m^3 N_v \lambda_{ov}k\alpha_m - \beta_{mv}\mu_o N_m^3 N_v \lambda_{ov}\Lambda^2 \alpha_m.
\end{aligned}$$

The obtained polynomial function with cubic degree indicates the existence of endemic equilibrium points. Using Maple 18 (32 bit) we solve the polynomial equation to obtain three distinct roots of which one is real and the other two are complex.

We rely on the real root and define an equilibrium point which is comprised of epidemic populations and given by the endemic equilibrium point as

$$(12) \quad (E^e) = \begin{pmatrix} S_m^* \\ E_m^* \\ I_m^* \\ S_v^* \\ I_v^* \\ P_o^* \end{pmatrix} = \begin{pmatrix} \Lambda_1 \\ \Lambda_2 \\ \Lambda_3 \\ \Lambda_4 \\ \Lambda_5 \\ \Lambda_6 \end{pmatrix},$$

where

$$\begin{aligned}
\Lambda_1 &= N_m - \left(\frac{\alpha_m + \sigma_m}{\alpha_m}\right) \frac{af^2 - 6ag - 6a^2 fl}{6a^2 f}, \\
\Lambda_2 &= \frac{\sigma_m}{\alpha_m} \left(\frac{af^2 - 6ag - 6a^2 fl}{6a^2 f}\right), \\
\Lambda_3 &= \frac{Af^2 - 6ag - 6a^2 fl}{6a^2 f}, \\
\Lambda_4 &= \frac{\Lambda}{\frac{\beta_{ov}P_o}{k+P_o} + \frac{\beta_{mv}I_m^*}{N_m} + \mu_v}, \\
\Lambda_5 &= \frac{\left(\frac{\beta_{ov}P_o}{k+P_o} + \frac{\beta_{mv}I_m^*}{N_m} + \mu_v\right)^2}{\Lambda\mu_v}, \\
\Lambda_6 &= \frac{\lambda_{mo}I_m^*}{\mu_o N_m} + \frac{\lambda_{ov}\Lambda}{\mu_o\mu_v N_v}
\end{aligned}$$

and

$$f = \frac{1}{6} \frac{\sqrt[3]{108 CA^2 + 36 (D) BA + 12 \sqrt{3} \sqrt{27 A^2 C^2 + 18 ABC (D) + 4 AD^3 - 4 B^3 C - B^2 D^2 A - 8 B^3}}}{A},$$

$$g = A \sqrt[3]{108 CA^2 + 36 (D) BA + 12 \sqrt{3} \sqrt{27 A^2 C^2 + 18 ABC (D) + 4 AD^3 - 4 B^3 C - B^2 D^2 A - 8 B^3}},$$

$$l = 2 A (D) - \frac{2}{3} B^2,$$

$$a = A.$$

**3.4. Sensitivity analysis.** Sensitivity analysis can applied to test the ability of the model in relation to prediction of model parameters[11]. In this section we use sensitivity analysis to determine the impact of parameters on  $R_o$ . In order to determine an excellent way that can reduce maize death and morbidity due MLND, it is crucially important to understand the proportional importance of factors that are liable for the transmission and prevalence of the infection.

Table 3 shows the parameter values of maize disease model. The parameters are taken from the previous studies that correspond to this study, existing information and through approximation.

To perform sensitivity analysis of  $R_o$  we need to first determine sensitivity indices's for each of parameters.

We define sensitivity indices's as partial derivative of  $R_o$  with respect to a given parameter as multiplied by the ratio of given parameter to the basic reproduction number  $R_o$ .

Mathematically, this definition is given by:

$$(13) \quad r_{m_i}^{R_o} = \left( \frac{\partial R_o}{\partial m_i} \right) \times \frac{m_i}{R_o}.$$

The approach used in equation (13) above is called normalized forward sensitivity indices of  $R_o$  with respect to parameters  $m_i$ , where  $m_i$  is any parameter from  $R_o$ .

**3.5. Parameters estimation and adoption.** In this subsection, we will review some journals to get proper parameters and where necessary approach of estimation will be used.

The primary infection rate of other plants population rather than maize can be greater or equivalent to 0.02 i.e 0.04,0.06,0.08... [12]

Transmission of Maize Chlorotic Mottle Virus by Chrysomelid Beetles and its cleoptera species of 8 classes was estimated [13]. From acquisition to inoculation is 8 days. The rates of these different classes were 0.390,0.905,0.052,0.024,0.170,0.139,0.156 and 0. Finding the average we obtain 0.2295 and again divide by 8 days and results to our one of transmission rate  $\beta_{mm}$  0.0286875.

Leafhopper transmission rate of ACMV 0.020(standard) 0.005 to 0.100 (range)  $(diseasedplant)^{-1}day^{-1}$  and transmission rate of EACMV 0.030 (standard) 0.01 to 0.140 (range)  $(diseasedplant)^{-1}day^{-1}$  [14]. Although MCMV could be transmitted mechanically and by seeds at very low rate

(0.008 0.04), the insects have crucial role in disease transmission and as the matter of fact leafhoppers are responsible by settling on the crops[15].Therefore, from this description we can estimate some of our model parameters between such interval as from 0.005 to 0.100 as we can see in the Table 3.

Vector carrying capacity per plant  $k = 50$ ,Vector birth rate 0.11.2, Vector death rate 0.05,Disease-induced mortality rate (roguing) 0.02 [16].

Other plants transmission and roguing rate are 0.0064 and 0.00033 respectively[17]. Maize recruitment rate 276.8212,contact rate of maize plants with pathogen  $\beta$  0.0024,net decay rate of pathogens 0.85,contributions of  $I(t)$  to the growth of pathogen 0.0018, natural death rate of  $S_m$  0.00033113 ,death rate of  $I_m$  due to foliage disease 0.00099338,total population of 44,000 maize plants per ha and maturity of maize plants from day one the harvest day is 151 days[8].It can also be noted that 90 days are also applicable for short season.

Table 3 summaries the estimated and collected data as follows:-

Table 3: Parameters values for MLND model.

<b>Parameters</b>	<b>Values</b>	<b>Units</b>	<b>Reference/Source</b>
$k$	10	ha $plant^{-1}$	estimated
$\beta_o$	0.06	ha $cells^{-1}day^{-1}$	[12]
$\beta_{ov}$	0.06	ha $cells^{-1}day^{-1}$	[12]
$N_v$	2,800	ha $ha^{-1}$	estimated
$\beta_{mm}$	0.286875	ha $cells^{-1}day^{-1}$	[13]
$\lambda_{vo}$	0.07486	ha $cells^{-1}day^{-1}$	estimated
$\lambda_{mo}$	0.010	ha $cells^{-1}day^{-1}$	estimated
$\Lambda$	0.95	ha $vectors^{-1}day^{-1}$	estimated
$\mu_v$	0.05	ha $vectors^{-1}day^{-1}$	[16]
$\sigma_m$	0.02	ha $plants^{-1}day^{-1}$	[16]
$\mu_o$	0.0005	ha $pathogens^{-1}day^{-1}$	estimated
$\beta_{mv}$	0.0024	ha $cells^{-1}day^{-1}$	[14]
$\beta_{vm}$	0.024	ha $cells^{-1}day^{-1}$	[14]
$\alpha_m$	0.020	ha $cells^{-1}day^{-1}$	[15]
$N_m$	44,000	ha $ha^{-1}$	[8]
$\sigma$	0.0018	ha $cells^{-1}day^{-1}$	[8]

The numerical value of  $R_o$  referred from equation (10) is given by  $R_o = 7.54$  .

Based on basic reproduction number in (10) and general formula of the sensitivity analysis in (13) we summaries the results of sensitivity indices's obtained as tabulated in Table 4.

Table 4: Sensitivity indices determined based on parameter values.

Parameters	Values	Sensitivity indices
$k$	10	-0.8122148385
$\beta_o$	0.06	0.7996312039
$\beta_{ov}$	0.06	0.01258363435
$N_v$	2,800	-0.01293729282
$\beta_{mm}$	0.286875	0.1869621680
$\lambda_{vo}$	0.07486	0.01246795692
$\lambda_{mo}$	0.010	0.7997468812
$\Lambda$	0.95	0.01293729256
$\mu_v$	0.05	-0.02587458659
$\sigma_m$	0.02	-0.9870627077
$\mu_o$	0.0005	-0.8122148376
$\beta_{mv}$	0.0024	0.00035365821
$\beta_{vm}$	0.024	0.00046933564

From Table 4 we discover that the most positively sensitive parameter is the rate of interaction between contagious maize sub-population and environment  $\lambda_{mo}$ . In fact any movement of pathogens either from infected/environment to environment/infected cause much damage maize population. The parameter adequate or close to the most positively sensitive is the interaction between virus in the environment to susceptible maize sub-population  $\beta_o$ , to this juncture there is no doubt that environment plays crucial role in transmitting MLND. There is also positive moderate transmission of disease through maize to maize contact which is due to adequate contact rate of infected to susceptible maize sub-populations  $\beta_{mm}$ , this is probably because there is a bit distance apart between one line and the other. The less sensitive positive parameters are  $\Lambda$ ,  $\lambda_{vo}$ ,  $\beta_{ov}$ ,  $\beta_{mv}$  and  $\beta_{vm}$ .

It can also be noted that, the most negatively sensitive parameter is the disease induced death rate  $\sigma_m$  and the negatively adequate rates are natural mortality rate  $\mu_o$  and the carrying capacity of virus in the environment  $k$ , while the less negatively sensitive parameters are mortality natural death of vector  $\mu_v$  and total number of vectors  $N_v$ .

It is important to note that, the increase of any of positively sensitive parameter increases  $R_o$  and decreasing any of these decreases  $R_o$  as well, while decreasing any of the negatively sensitive parameter increases  $R_o$  and increasing any of them decreases  $R_o$  too.

The epidemiological importance of this is to give an insights on selection of control parameters in which the most positively/negatively sensitive and adequate parameters can be chosen

for control strategies.

**3.6. Simulation of the basic model.** In this subsection we will deal with basic model simulations. We will see the behavior of MLND dynamics when endemic exists and show how maize populations react towards vectors population and environment as well. Furthermore, we will show the effects of most sensitive and adequate parameters in regards to basic reproduction number.

All simulations are carried out using MATLAB R2013a and HP Windows 7 Professional/ Intel(R) Core (TM)i3-4005U CPU @1.70GHz 1.70GHz Computer machine.

**3.7. Dynamics of populations simulation.** We simulate maize, vectors and viruses in the environment populations before looking into behavior and formation MLND based on 151 days the maize duration from planting to the harvesting day.

From Figures 2 and 3: We observe six different lines as an indication of various populations. The green, cyan, black are three different lines which compose maize populations of at risk (susceptible), exposed and infested population respectively. The susceptible maize population decelerates exponentially to acquire endemic equilibrium level as they die due MLND which is the effects of maize to maize infection, virus in the environment and infected vector population. The exposed maize population assumes parabolic curve as it increases exponentially to a certain maximum point before exponential deceleration to the a certain endemic level. This behavior explain the incubation period that some resistant maize plants takes after exposed as it keeps increasing before turn slowly into infection class.

The red line is the susceptible vectors and the blue line is infested vectors. The susceptible vectors decrease exponentially due to natural death and acquisition of infestation from severely infested maize, vectors and environment and finally acquire the endemic equilibrium level. The infested vectors forms parabolic curve as they do raise and drop exponentially to the endemicity level. The raising to the maximum point implies decrease of susceptible vectors add members to the infestation vectors and some might come from environment and infested maize. On the other hand the exponential dropping indicates no more susceptible vectors which means recruitment rate is much less as compared to both dying and infection rate.

The virus in the environment is indicated by magenta line which as we can see extend exponentially to a certain maximum point and proceed a constant endemic level with unusual number of days nearly 20 years. This behavior explain the fact that viruses in the environment never ends even after maize extinction. The constant endemic level of viruses in the environment indicates that no more supply from both vector population as well as maize population following starvation of hunger after maize plants extinction.



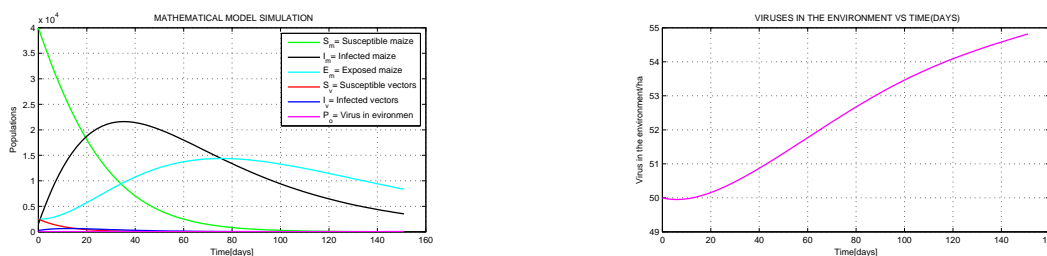


FIGURE 2. with sub-figure 2a and 2b respectively

This Figure is showing fully simulated model and partly in the environment with the MLND dynamics of sub-populations/ha

**3.8. The effects of parameters on basic reproduction number.** We refer Figures 4, 5 and 6 to illustrates the effect of the most sensitive and moderate positive indexes as well as the most sensitive and moderate negative indexes parameters on the basic reproduction number.

In Sub-figures 4a and 5b we note  $\lambda_{mo}$  is the most sensitive positive parameter, that the increase of this transmission rate result to the quick increase of the basic reproduction number. It imply that the interaction of infested sub-population (maize/pathogens) might have significant effects on maize population. Similarly, the maize death rate due to disease  $\sigma_m$  is the most sensitive negative parameter, that any reduction from it (stop dying of maize) make basic reproduction number experience significant exponential retardation. In other words, susceptible maize population will always remain constant if there is no infestation. From Sub-figures 4b and 5a we observe significant steep slope increase of basic reproduction number as the increase of each of these parameters ( $\beta_o$  and  $\beta_{mm}$  as proceeds additional positive increments. The reason behind these rapid positive increments might be the main causative of MLND arises from the contact rate from infected maize to other vegetative plants and interaction of infested to susceptible maize rates.

Sub-figures 6a and 6b illustrates the principal effects of natural death rate of vectors in the environment and the carrying capacity. Both of these moderate rate reduces exponentially to zero as basic reproduction number do and to the contrary is also true. This behavior gives an indication that killing of vectors both in the environment and its population will have significant effects on MLND reduction if not eradication of the whole epidemic since any mortality rate does not favor pathogens but maize population.

**3.9. Discussion and conclusion.** The SI-SEI maize disease model which is a combination of both the host and vector population models has been formulated to study and analyses the dynamics of MLND. It is comprised of six compartments including susceptible, infected and exposed of maize population while of the vector is susceptible and infected with the last class viruses in the environment.

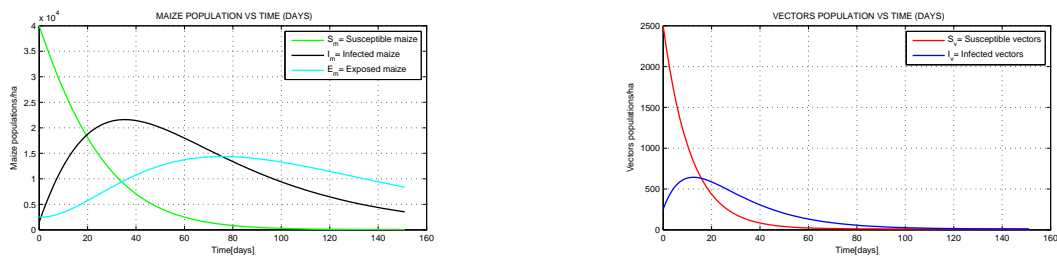


FIGURE 3. with sub-figure 3a,3b respectively

This figure depicts the MLND dynamics partly in maize and partly in vector model sub-populations/ha

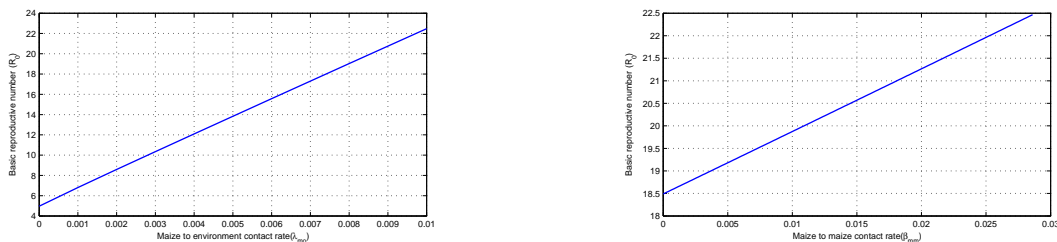


FIGURE 4. Sub-figure 4a,4b respectively

This figure illustrates the effect of  $\lambda_{mo}$  and  $\beta_{mm}$  on the basic reproduction number

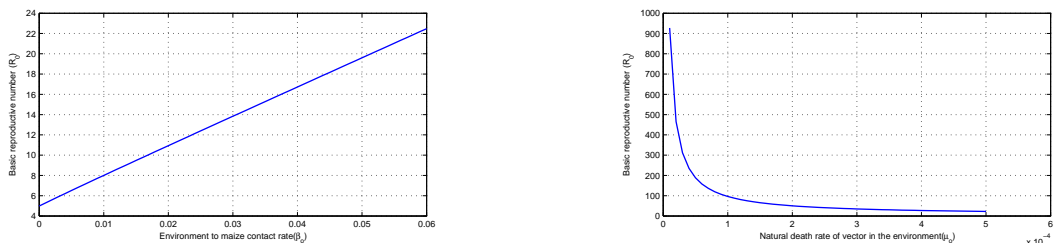


FIGURE 5. Sub-figure 5a,5b respectively

And this show the effects of  $\beta_o$  and  $\mu_o$  on the basic reproduction number  $R_o$

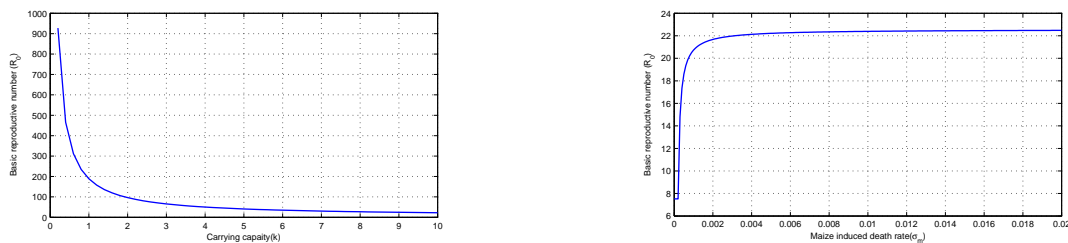


FIGURE 6. Sub-figure 6a,6b

The last Figure which illustrates the effect of carrying capacity and maize disease induced death rate on the  $R_o$

The disease free equilibrium ( $DFE$ ) was determined which showed that in the absence of disease only constant susceptible maize population exists with recruitment and naturally dying of vectors. Other analytical results shows that the system has unique endemic equilibrium point ( $EE$ ) as it can be referenced from equation (12). The basic reproduction number  $R_o$  determined was 7.54 which show that disease is endemic, cementing the point that the system has unique endemic equilibrium point. From the literature review data were collected and summarized in Table 3 which are important for model simulations, sensitivity analysis. Table 4 summaries the work of sensitivity analysis with indexes that are most, adequate and least positively and negatively sensitive crucially for transmission of MLND.

Finally, numerical simulation is done to illustrate the dynamics behavior of MLND in maize population and how some most sensitive parameters affects  $R_o$ . It also suggest that viruses in the environment, infected maize and vector populations plays significance role in bringing in infection force which contribute largely to the dynamics of MLND in the maize population. This situation can be seen from Figures 2 and 3 where susceptible maize and vectors decreases exponentially while others assumes exponential curves to a certain point in time by increasing before they decline and find convergence point of a certain unique endemic equilibrium points. Another evidence is taken from Figures 4, 5 and 6 where by  $R_o$  increases/decreases either with steep slopes or exponential curves as we vary by the most or adequate positively/negatively sensitive parameters. Lastly, the closer observation on basic reproduction number, sensitivity analysis and simulation indicates that  $\lambda_{mo}$ ,  $\beta_o$  and  $\beta_{mm}$  parameters are main agents of transmission and persistence of epidemic and also very important in finding how and where to implement the control policies for the elimination of MLND. The numerical solutions predict the danger of losing almost all crops if control measures are not put into consideration since without intervention the population will vary for sometimes and ultimately they all keep approaching to endemic points and after a number of days all susceptible maize will be infected and die of disease.

In general, we formulated and analyzed a mathematical model for dynamics of MLND for a single season maize populations. This model is quite simple to start with in studying and analyzing the dynamics of maize lethal necrosis disease in regards to maize populations as there are a lot of complications if we consider in depth the epidemiological process of MLND. Therefore study will add value to body of knowledge, policy makers and act as a bridge for researchers who would like to study the dynamics of MLND mathematically.

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#### REFERENCES

- [1] I. Adams, D. Miano, Z. Kinyua, A. Wangai, E. Kimani, N. Phiri, R. Reeder, V. Harju, R. Glover, U. Hany, *et al.*, “Use of next-generation sequencing for the identification and characterization of maize chlorotic mottle virus and sugarcane mosaic virus causing maize lethal necrosis in kenya,” *Plant Pathology*, vol. 62, no. 4, pp. 741–749, 2013.
- [2] A. Wangai, M. Redinbaugh, Z. Kinyua, D. Miano, P. Leley, M. Kasina, G. Mahuku, K. Scheets, and D. Jeffers, “First report of maize chlorotic mottle virus and maize lethal necrosis in kenya,” *Journal of Virological Methods*, vol. 240, pp. 49–53, 2017.
- [3] M. Gowda, B. Das, D. Makumbi, R. Babu, K. Semagn, G. Mahuku, M. S. Olsen, J. M. Bright, Y. Beyene, and B. M. Prasanna, “Genome-wide association and genomic prediction of resistance to maize lethal necrosis disease in tropical maize germplasm,” *Theoretical and Applied Genetics*, vol. 128, no. 10, pp. 1957–1968, 2015.
- [4] F. H. Kiruwa, T. Feyissa, and P. A. Ndakidemi, “Insights of maize lethal necrotic disease: A major constraint to maize production in east africa,” *African Journal of Microbiology Research*, vol. 10, no. 9, pp. 271–279, 2016.
- [5] D. W. Miano, “Maize lethal necrosis disease: A real threat to food security in the eastern and central africa region,” 2014.
- [6] J. Kahuru, L. Luboobi, and Y. Nkansah-Gyekye, “Stability analysis of the dynamics of tungiasis transmission in endemic areas,” *Asian Journal of Mathematics and Applications*, vol. 2017, pp. 1–24, 2017.
- [7] A. Abate and A. Tiwari, “The concept of box invariance for special classes of dynamical systems,” in *Proceedings of the 45th IEEE Conference on Decision and Control*, Citeseer, 2007.
- [8] O. C. Collins and K. J. Duffy, “Optimal control of maize foliar diseases using the plants population dynamics,” *Acta Agriculturae Scandinavica, Section B Soil & Plant Science*, vol. 66, no. 1, pp. 20–26, 2016.
- [9] R. C. Ngeleja, L. S. Luboobi, and Y. Nkansah-Gyekye, “Modelling the dynamics of bubonic plague with yersinia pestis in the environment,” *Communications in Mathematical Biology and Neuroscience*, vol. 2016, pp. 1–24, 2016.

- [10] O. Diekmann, J. Heesterbeek, and M. Roberts, “The construction of next-generation matrices for compartmental epidemic models,” *Journal of the Royal Society Interface*, pp. 873–885, 2009.
- [11] S. Edward, D. Kuznetsov, and S. Mirau, “Modeling and stability analysis for a varicella zoster virus model with vaccination,” *Applied and Computational Mathematics*, vol. 3, no. 4, pp. 150–162, 2014.
- [12] L. V. Madden and F. Van Den Bosch, “A population-dynamics approach to assess the threat of plant pathogens as biological weapons against annual crops using a coupled differential-equation model, we show the conditions necessary for long-term persistence of a plant disease after a pathogenic microorganism is introduced into a susceptible annual crop,” *BioScience*, vol. 52, no. 1, pp. 65–74, 2002.
- [13] L. Nault, W. Styer, M. Coffey, D. Gordon, L. Negi, C. Niblett, *et al.*, “Transmission of maize chlorotic mottle virus by chrysomelid beetles,” *Phytopathology*, vol. 68, no. 7, pp. 1071–1074, 1978.
- [14] X.-S. Zhang, J. Holt, and J. Colvin, “Synergism between plant viruses: a mathematical analysis of the epidemiological implications,” *Plant Pathology*, vol. 50, no. 6, pp. 732–746, 2001.
- [15] M. Zhao, H. Ho, Y. Wu, Y. He, and M. Li, “Western flower thrips (*frankliniella occidentalis*) transmits maize chlorotic mottle virus,” *Journal of Phytopathology*, vol. 162, no. 7-8, pp. 532–536, 2014.
- [16] M. Jeger, Z. Chen, G. Powell, S. Hodge, and F. Van den Bosch, “Interactions in a host plant-virus-vector-parasitoid system: Modelling the consequences for virus transmission and disease dynamics,” *Virus research*, vol. 159, no. 2, pp. 183–193, 2011.
- [17] Z. Zhonghua and S. Yaohong, “Stability and sensitivity analysis of a plant disease model with continuous cultural control strategy,” *Journal of Applied Mathematics*, vol. 2014, pp. 1–15, 2014.

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