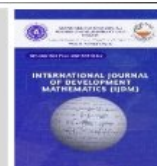




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Mathematical Analysis of Drought-Induced and Maize Streak Virus Dynamics in Non-Genetically Modified Maize Plants

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Abstract

Drought stress and Maize Streak Virus (MSV) are major constraints to maize production, particularly in regions where water scarcity and vector-mediated virus transmission occur simultaneously. This study develops and analyzes a mathematical model of the dynamics of drought stress and MSV transmission in non-genetically modified maize to enhance understanding of their combined effects on maize productions. The basic reproduction number was derived using the next-generation matrix (NGM) method to quantify the average number of secondary infections. The global stability of the system were established using Castillo-Chavez method, respectively. It was shown that the disease-free equilibrium is both locally and globally asymptotically stable when . Sensitivity analysis was performed using the normalized forward sensitivity index to

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identify key parameters influencing disease transmission. Parameters with positive sensitivity indices increase the risk of outbreaks, whereas those with negative indices reduce the likelihood of sustained transmission. Numerical simulations were conducted to examine the temporal dynamics of the model compartments and assess the influence of the vector-to-maize transmission parameter. The simulations showed a rapid decline in the susceptible non-GM maize population, while drought-exposed and MSV-related compartments exhibited substantial changes during the early stages of the simulation. The population suffering from both drought and MSV increased considerably, reaching approximately 17,500 plants around

days 65-70 before gradually declining. Parameter variation was performed by varying the vector-to-maize transmission parameter, from 0.25 to 1.00. The findings suggest that the interaction between drought and MSV can impose a substantial and persistent burden on non-GM maize. Effective management therefore requires an integrated approach involving vector control, early MSV detection, improved drought management, and the development of maize varieties with enhanced drought tolerance and MSV resistance. The model provides a useful baseline for assessing the potential benefits of genetically modified maize and other intervention strategies for reducing the combined effects of drought and MSV on maize production.

1 Introduction

Maize (*Zea mays L.*) is one of the most important cereal crops worldwide, serving as a staple food for humans, a major component of livestock feed, and a valuable raw material for numerous agro-industrial products. Globally, maize contributes significantly to food security, employment, and economic growth owing to its high productivity, wide adaptability, and diverse utilization. In sub-Saharan Africa (SSA), maize constitutes the primary staple food for millions of households and provides income for both smallholder and commercial farmers. Consequently, improving maize productivity remains central to achieving sustainable agricultural development and food security in the region [Bazarra et al. \(2022\)](#). Despite its global importance, the combined effects of biotic and abiotic stresses increasingly threaten maize production. Among the abiotic constraints, drought is regarded as one of the most devastating factors limiting maize productivity. Water deficit adversely affects seed germination, leaf expansion, photosynthetic efficiency, nutrient uptake, flowering, grain filling, and ultimately crop yield. The increasing frequency and severity of drought episodes associated with climate change have further intensified production losses, particularly in rain-fed agricultural systems that dominate many African countries. Studies have shown that the reproductive stage of maize is especially sensitive to drought, making water stress a major obstacle to achieving stable grain production under changing climatic conditions ([Ayembillah et al. \(2022\)](#); [Ackora-Prah et al. \(2023\)](#)).

In addition to drought stress, viral diseases constitute another major constraint to maize production. Among these diseases, Maize Streak Virus (MSV) remains the most economically

important viral disease of maize in Africa. MSV belongs to the genus *Mastrevirus* within the family *Geminiviridae* and is transmitted primarily by leafhoppers of the genus *Cicadulina* in a persistent manner. Infection is characterized by chlorotic streaks on leaves, reduced chlorophyll content, severe stunting, poor ear formation, and substantial grain yield reduction. Under favourable environmental conditions and when susceptible cultivars are grown, yield losses may approach 100%, posing serious threats to food security and rural livelihoods across SSA ([Acharya et al. \(2003\)](#); [Ado et al. \(2007\)](#)). The epidemiology of MSV is strongly influenced by environmental conditions that affect both the host plant and the insect vector. Climatic factors such as temperature, rainfall, humidity, and drought modify the abundance, survival, dispersal, and feeding behaviour of *Cicadulina* leafhoppers, thereby influencing virus transmission dynamics. Drought and irregular rainfall patterns have been identified as important drivers of MSV outbreaks because they alter vector ecology and increase crop susceptibility. Consequently, climate variability not only affects maize physiology directly but also indirectly promotes disease development through enhanced vector activity and host vulnerability ([Collins, \(2026\)](#)). The simultaneous occurrence of drought stress and MSV infection presents a complex challenge because the two stresses interact in ways that amplify their individual effects. Drought weakens plant defence mechanisms, reduces physiological resilience, and alters metabolic processes that influence susceptibility to pathogen invasion. Conversely, MSV infection further impairs photosynthesis, disrupts cellular metabolism, and diminishes the plant's capacity to tolerate water limitation. The combined effects of drought and viral infection therefore often result in greater reductions in biomass accumulation and grain yield than either stress acting independently. Understanding these interactions is essential for designing effective crop management strategies capable of improving maize productivity under climate change.

Mathematical modelling has become an indispensable tool for understanding plant disease epidemiology and evaluating disease management strategies. Differential equation models provide a quantitative framework for investigating disease transmission mechanisms, host interactions, threshold dynamics, and intervention strategies that may be difficult or expensive to evaluate experimentally. Through stability analysis, reproduction number estimation, sensitivity analysis, and numerical simulation, mathematical models provide valuable insights into disease persistence and control. In recent years, several deterministic, stochastic, and fractional-order models have been developed to study the transmission dynamics of MSV and related plant diseases ([Collins, & Duffy \(2022\)](#); [FAO \(2022\)](#); [Flood \(2010\)](#)). For example, [Erentein et al. \(2022\)](#) formulated and analysed a deterministic compartmental model for MSV transmission, deriving the basic reproduction number and investigating the local and global stability of the disease-free equilibrium. [Collins, & Duffy \(2022\)](#) extended MSV modelling by incorporating stochastic effects to evaluate uncertainty in disease dynamics, while subsequent fractional-order models demonstrated that memory effects can substantially influence disease progression and optimal control strategies [Sutanto et al. \(2024\)](#). More recently, [Collins, \(2026\)](#) developed a mathematical model describing maize yield under the co-infection of maize streak virus and maize stripe virus, highlighting the importance of integrating epidemiological dynamics with crop productivity. Although considerable progress has been made in modelling MSV epidemiology, most existing studies primarily focus on virus transmission and vector

dynamics without explicitly incorporating drought-induced physiological stress into the disease process. Likewise, many drought models investigate crop growth under water limitation but neglect the simultaneous influence of viral infection Josie (2023).

Consequently, there remains a significant gap in the mathematical understanding of the coupled effects of drought stress and MSV infection on the dynamics of non-genetically modified maize. Developing an integrated framework capable of capturing these interacting processes is important for predicting disease behaviour under changing environmental conditions and for informing sustainable crop management strategies. Motivated by this gap, the present study develops a deterministic mathematical model describing the dynamics of non-genetically modified maize plants under the combined influence of drought stress and Maize Streak Virus infection. The proposed model incorporates the interactions among healthy and infected maize plants, leaf hopper vectors, and drought-dependent plant responses. Theoretical analyses are performed to establish the positivity and boundedness of solutions, determine the disease-free equilibrium, derive the basic reproduction number, and investigate the stability properties of the system. Furthermore, sensitivity analysis and numerical simulations are conducted to quantify the influence of key epidemiological and environmental parameters on disease transmission and maize productivity. The findings provide useful insights into the interaction between climatic stress and plant viral diseases and offer a quantitative framework for supporting integrated disease management and sustainable maize production under climate variability.

2 Description of the Non-Genetically Modified Maize Model

The model describes the dynamics of MSV in maize plants under drought conditions and vector interactions. The total population of maize at time t denoted by $N_m(t)$, is subdivided into seven compartments: susceptible maize plants $M_s(t)$, maize exposed to drought $M_{ed}(t)$, maize exposed to MSV $M_{em}(t)$, maize exposed to both drought and MSV $M_{dm}(t)$, maize suffering severe drought $M_{sd}(t)$, maize infected with MSV $M_{im}(t)$, and maize suffering both severe drought and MSV infection $S_{dm}(t)$. The vector population is divided into susceptible vectors $V_s(t)$ and infectious vectors $V_i(t)$. The environmental condition is classified into drought-free environment. Thus, The entire population, represented by:

$$N_m(t) = M_s(t) + M_{ed}(t) + M_{em}(t) + M_{dm}(t) + M_{sd}(t) + M_{im}(t) + S_{dm}(t) \quad (1)$$

$$N_v(t) = V_s(t) + V_i(t) \quad (2)$$

The susceptible compartment $M_s(t)$ is increased through recruitment in planting of maize at a rate π_m . It is decreased by natural death (at a rate $\mu_m M_s$), and by effective contact between susceptible maize population and drought and MSV and a rate λ_d and λ_m respectively. Such that

$$\frac{dM_s}{dt} = \pi_m - (\lambda_d + \lambda_m + \mu_m)M_s \quad (3)$$

Where $\lambda_m = \frac{\beta_{vm}V_i(\theta E + \theta_d E_d)}{N_v}$, $\lambda_v = \frac{\beta_{mv}(M_{im} + \tau S_{dm})(\theta E + \theta_d E_d)}{N_m}$, $\lambda_d = \alpha D_e$.

The exposed to drought class $M_{ed}(t)$ is increased by the maize population which become affected by drought at a rate λ_d and it is reduced by the maize plants population which

become infected by MSV at a rate λ_m . It is further reduced by $(\sigma_1 + \phi_1 + \mu_m)M_{ed}$ where σ_1 is the rate of progression from M_{ed} to M_{sd} , μ_m , is the natural death rate and ϕ_1 is the rate of progression from drought exposed class M_{ed} to M_{dm} , so that

$$\frac{dM_{ed}}{dt} = \lambda_d M_s - \lambda_v M_{ed} - (\sigma_1 + \phi_1 + \mu_m)M_{ed} \quad (4)$$

The exposed to drought class $M_{ed}(t)$ is increased by the maize plants population which become infected by MSV at a rate λ_m and it is reduced by the maize population which become affected by the drought at a rate λ_d . It is further reduced by $(\sigma_2 + \phi_2 + \mu_m)M_{em}$ where σ_2 is the rate of progression from M_{em} to M_{im} , μ_m , is the natural death rate and ϕ_2 , is the progression rate from M_{em} to M_{dm} , so that

$$\frac{dM_{em}}{dt} = \lambda_m M_s - \lambda_d M_{em} - (\sigma_2 + \phi_2 + \mu_m)M_{em} \quad (5)$$

The maize plants that are exposed to both drought and MSV class $M_{dm}(t)$ is increased by the maize plants population which become exposed to both MSV and drought at a rate $\phi_1 \lambda_{dm}$. It is reduced by $(\gamma_2 + \mu_m)M_{dm}$ where γ_2 is the rate of progression from M_{dm} to S_{dm} , μ_m , is the natural death rate, so that

$$\frac{dM_{dm}}{dt} = \phi_1 \lambda_{dm} M_{ed} + \phi_2 M_{em} - (\gamma_2 + \mu_m)M_{dm} \quad (6)$$

The population of the maize plants that suffers from severe drought, $M_{sd}(t)$ is increased by the rate of progression, σ_1 from M_{ed} to M_{sd} , and it is reduced by γ_1 is the rate of progression from M_{sd} to S_{dm} and μ_m , is the natural death rate and the disease-induced death rate at a rate δ_1 , so that

$$\frac{dM_{sd}}{dt} = \sigma_1 M_{ed} - (\gamma_1 + \delta_1 + \mu_m)M_{sd} \quad (7)$$

The population of the maize plants that are infectious with MSV, $M_{im}(t)$ is increased by σ_2 the rate of progression from M_{em} to M_{im} , and it is reduced by γ_3 is the rate of progression from M_{im} to S_{dm} and μ_m , is the natural death rate and the disease-induced death rate at a rate δ_2 , so that

$$\frac{dM_{im}}{dt} = \sigma_2 M_{em} - (\gamma_3 + \delta_2 + \mu_m)M_{im} \quad (8)$$

The population of the maize plants that suffers severe drought and infectious with MSV, $S_{dm}(t)$ is increased by γ_1 the rate of progression from M_{sd} to S_{dm} , by γ_2 is the rate of progression from M_{dm} to S_{dm} and by γ_3 is the rate of progression from M_{im} to S_{dm} , μ_m , is the natural death rate and the disease-induced death rate at a rate δ_3 , so that

$$\frac{dS_{dm}}{dt} = \gamma_1 M_{sd} + \gamma_2 M_{dm} + \gamma_3 M_{im} - (\delta_3 + \mu_m)S_{dm} \quad (9)$$

The infected vector compartment $V_i(t)$ is increased by effective contact between susceptible vectors population a rate λ_v . It is decreased by natural death at a rate $\mu_v V_i$, and so that

$$\frac{dV_i}{dt} = \lambda_v V_s - \mu_v V_i \quad (10)$$

The environmental drought-free compartment $E(t)$ decreases by the rate at which drought pushes plants into stress at a rate κE and increases due to recovery from drought at a rate ηE_d . So that

$$\frac{dE}{dt} = \eta E_d - \kappa E \quad (11)$$

The environmental with drought compartment $E_d(t)$ is increases by which the drought pushes plants into stress at a rate κE and decreases due to recovery from drought at a rate ηE_d . So that

$$\frac{dE_d}{dt} = \kappa E - \eta E_d \quad (12)$$

Table 1. Description of State Variables

Variable	Description
$N_m(t), N_v(t)$	Total populations of maize and vectors
$M_s(t)$	Susceptible genetically modified maize plants
$M_{ed}(t)$	Non genetically modified maize exposed to drought
$M_{em}(t)$	Non genetically modified maize exposed to MSV
$M_{dm}(t)$	Non genetically modified maize exposed to both drought and MSV
$M_{sd}(t)$	Non genetically modified maize suffering severe drought
$M_{im}(t)$	Non genetically modified maize infected with MSV
$S_{dm}(t)$	Non genetically modified maize plants suffering severe drought and MSV infection
$H(t)$	Harvested maize
$V_s(t)$	Susceptible vectors
$V_i(t)$	Infected vectors
$E(t)$	Drought-free environment
$E_d(t)$	Drought-affected environment

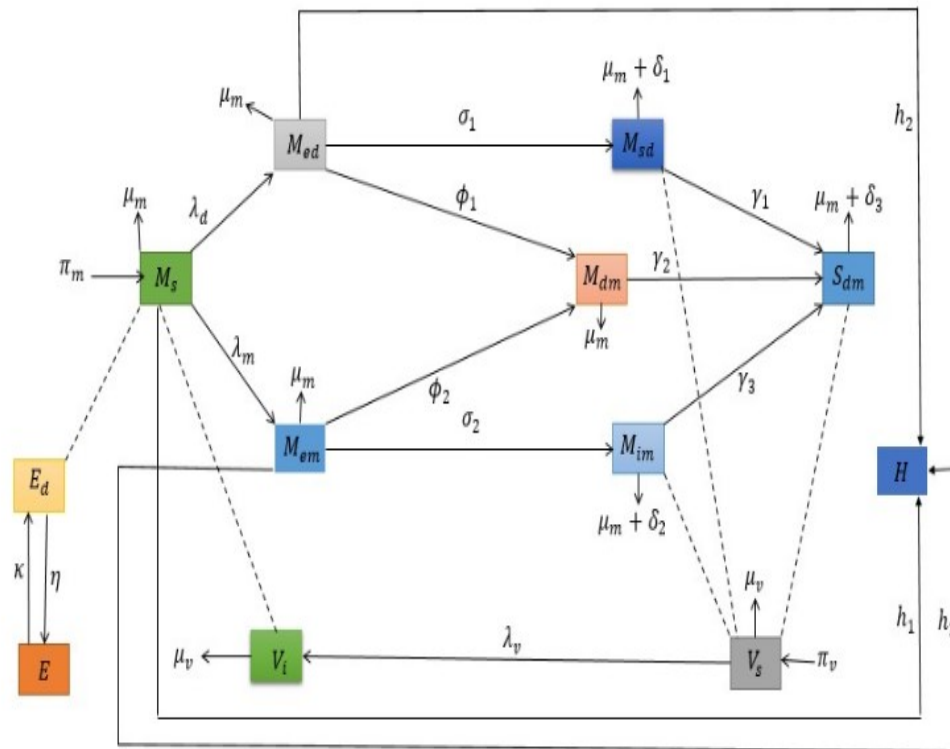


Figure 1. Flow Diagram of Non-Genetically Maize Model.

Table 2: Parameters description of the non-genetically maize plants model

Parameter	Description
π_m, π_v	Recruitment rate of maize plants and vectors respectively.
μ_m, μ_v	Natural mortality rate of maize and vectors respectively.
β_{mv}, β_{vm}	Effective risk rate of maize and vectors respectively.
$\gamma_1, \gamma_2, \gamma_3$	Progression rate of M_{sd} , M_{dm} and M_{im} respectively.
σ_1, σ_2	Progression rate of M_{ed} and M_{em} respectively.
α	Drought impact factor.
θ, θ_d	Environmental effects for drought-free and drought-affected.
$\delta_1, \delta_2, \delta_3$	Disease-induced death rate due to drought, virus and both drought and virus respectively.
ϕ_1	The rate of progression from drought exposed class M_{ed} to M_{dm}
ϕ_2	The progression rate from M_{em} to M_{dm}
h_1, h_2, h_3	Harvesting of M_s , M_{ed} and M_{em} respectively.

The non-genetically maize model equations are:

$$\left\{ \begin{array}{l} \frac{dM_s}{dt} = \pi_m - (\lambda_d + \lambda_m + \mu_m)M_s, \\ \frac{dM_{ed}}{dt} = \lambda_d M_s - (\sigma_1 + \phi_1 + \mu_m)M_{ed}, \\ \frac{dM_{em}}{dt} = \lambda_m M_s - (\sigma_2 + \phi_2 + \mu_m)M_{em}, \\ \frac{dM_{dm}}{dt} = \phi_1 M_{ed} + \phi_2 M_{em} - (\gamma_2 + \mu_m)M_{dm}, \\ \frac{dM_{sd}}{dt} = \sigma_1 M_{ed} - (\gamma_1 + \delta_1 + \mu_m)M_{sd}, \\ \frac{dM_{im}}{dt} = \sigma_2 M_{em} - (\gamma_3 + \delta_2 + \mu_m)M_{dm}, \\ \frac{dS_{dm}}{dt} = \gamma_1 M_{sd} + \gamma_2 M_{dm} + \gamma_3 M_{dm} - (\delta_3 + \mu_m)S_{dm}, \\ \frac{dV_s}{dt} = \pi_v - (\lambda_v + \mu_v)V_s, \\ \frac{dV_i}{dt} = \lambda_v V_s - \mu_v V_i, \\ \frac{dE}{dt} = \eta E_d - \kappa E, \\ \frac{dE_d}{dt} = \kappa E - \eta E_d. \end{array} \right. \quad (13)$$

Where $\lambda_m = \frac{\beta_{vm}V_i(\theta E + \theta_d E_d)}{N_v}$, $\lambda_v = \frac{\beta_{mv}(M_{dm} + \tau S_{dm})(\theta E + \theta_d E_d)}{N_m}$, $\lambda_d = \alpha D_e$

Subject to the following initial condition:

$$\left\{ \begin{array}{l} M_s(0) \geq 0, M_{ed}(0) \geq 0, M_{em}(0) \geq 0, M_{im}(0) \geq 0, M_{sd}(0) \geq 0, M_{dm}(0) \geq 0, \\ V_s(0) \geq 0, V_i(0) \geq 0, E(0) \geq 0, E_d(0) \geq 0. \end{array} \right. \quad (14)$$

2.1 Basic Properties of the non-genetically modified maize plants model

The invariant region and positivity of solution of dynamical system (13) is given, coupled with the fact that, the model describes maize plants and vector population. Hence, the study claims the following results.

2.2 Invariant region of the non-genetically modified maize plants model

In this section, we get a region in which the solution of equations (1) is bounded. To achieve this, we first consider the total population of the non-genetically modified maize plants. N_m , where

$$N_m = M_s(t) + M_{ed}(t) + M_{em}(t) + M_{dm}(t) + M_{sd}(t) + M_{im}(t) + S_d(t) \quad (15)$$

Then, differentiating N_m both sides of equation (1) with respect to t leads to

$$\frac{dN_m}{dt} = \frac{dM_s(t)}{dt} + \frac{dM_{ed}(t)}{dt} + \frac{dM_{em}(t)}{dt} + \frac{dM_{dm}(t)}{dt} + \frac{dM_{sd}(t)}{dt} + \frac{dM_{im}(t)}{dt} + \frac{dS_d(t)}{dt} \quad (16)$$

Simplifying equation (16) leads to

$$\frac{dN_m}{dt} = \pi_m - \mu_m N_m - \delta_1 M_{sd} - \delta_2 M_{im} - \delta_3 S_{dm} \quad (17)$$

Similarly, the total population of the vector given by

$$N_v(t) = V_s(t) + V_i(t) \quad (18)$$

Further simplification of equation (18) resulted into

$$N_v(t) = \pi_v - \mu_v N_v \quad (19)$$

Theorem 1: *The closed $\Omega_m = \left(X \in \mathbb{R}_+^{11} : N_m \leq \frac{\pi_m}{\mu_m}, N_v \leq \frac{\pi_v}{\mu_v} \right)$ is positively invariant and attracting with respect to the model (13).*

Where $X = (M_s, M_{ed}, M_{em}, M_{dm}, M_{sd}, M_{im}, S_{dm}, V_s, V_i, E, E_d)$

Proof:

In the absence of disease-induced death, (i.e. $\delta_1 = \delta_2 = \delta_3 = 0$), and by comparison theorem, the first equation of equation becomes:

$$\frac{dN_m}{dt} \leq \pi_m - \mu_m N_m \quad (20)$$

Solving equation (20) by method of integrating factor and applying initial condition resulted into

$$N_m(t) \leq \frac{\pi_m}{\mu_m} + \left(N_m(0) - \frac{\pi_m}{\mu_m} \right) e^{-\mu_m t} \quad (21)$$

Further simplification of equation (21) resulted into

$$N_m(t) \leq N_m(0)e^{-\mu_m t} + \frac{\pi_m}{\mu_m}(1 - e^{-\mu_m t}) \quad (22)$$

From equation (22), it can be observed that $N_m(t) \rightarrow \left(\frac{\pi_m}{\mu_m} \right)$ as $t \rightarrow \infty$. That is, the total population maize plants size $N_m(t)$ takes off from the value $N_m(0)$ at the initial time $t = 0$ and ends up with the bounded value $\frac{\pi_m}{\mu_m}$ as the time t grows to infinity. Thus, it can be concluded that $N_m(t)$ is bounded within $0 \leq N_H(t) \leq \frac{\Lambda_H}{\mu_H}$.

Solving for the vector population in equation (13), also resulted into

$$N_v(t) \leq N_v(0)e^{-\mu_v t} + \frac{\pi_v}{\mu_v}(1 - e^{-\mu_v t}) \quad (23)$$

From equation 23), it can be observed that $N_v(t) \rightarrow \left(\frac{\pi_v}{\mu_v} \right)$ as $t \rightarrow \infty$. That is, the total vector population size $N_v(t)$ takes off from the value $N_v(0)$ at the initial time $t = 0$ and ends up with the bounded value $\frac{\pi_v}{\mu_v}$ as the time t grows to infinity. Thus, it can be concluded that $N_v(t)$ is bounded within $0 \leq N_v(t) \leq \frac{\pi_v}{\mu_v}$.

Thus, the feasible solution set of the system (13) of the model enters and remains in the

region:

$$\Omega = \left\{ (M_s, M_{ed}, M_{em}, M_{dm}, M_{sd}, M_{im}, S_{dm}, V_s, V_i, E, E_d) \in \mathbb{R}_+^{11} : N_m \leq \frac{\pi_m}{\mu_m}, N_v \leq \frac{\pi_v}{\mu_v} \right\},$$

Hence, our Non-genetically modified maize plants model is well posed and biologically meaningful.

2.3 Positivity of solution for the non-genetically modified maize plants model

This section demonstrated that all solutions to the model equations (13) will remain positive in the future, provided that their corresponding initial values are also positive.

Theorem 2: *Let the initial data of (14) of the model equations (13) is non-negative for all time, $t > 0$.*

Proof: Let $T_1 = \sup \{T > 0 : M_s \geq 0, M_{ed} \geq 0, M_{em} \geq 0, M_{dm} \geq 0, M_{sd} \geq 0, M_{im} \geq 0, S_{dm} \geq 0, V_s \geq 0, V_i \geq 0, E \geq 0, E_d \geq 0 \in [0, T]\}$, thus, $T_1 > 0$. Then it follows from the first equations of (13) after eliminating the positive term that

$$\frac{dM_s}{dt} = -(\alpha D_e + \lambda_m + \mu_m)M_s \quad (24)$$

Applying method of separable variable on equation (13), we have

$$\frac{dM_s}{M_s} \geq -(\lambda_m(z_1)dz_1 + (\alpha D_e + \mu_m))dt > 0 \quad (25)$$

Integrating equation (14), we get

$$\ln M_s(t) \geq - \int_0^T \lambda_m(z_1)dz_1 - (\alpha D_e + \mu_m) \int dt \quad (26)$$

Taking exponential of both sides on equation (26)

$$M_s(t) \geq e^{- \int_0^T \lambda_m(z_1)dz_1 - (\alpha D_e + \mu_m)t + c} \quad (27)$$

where $e^c = C_1$

Simplifying equation (27), we have

$$M_s(t) \geq C_1 e^{- \int_0^T \lambda_m(z_1)dz_1 - (\alpha D_e + \mu_m)t} \quad (28)$$

Applying the initial condition at $t = 0$ on equation (28), we have

$$M_s(0) = C_1 \quad (29)$$

Substitute (29) back into (28) to have

$$M_s(t) \geq M_s(0)e^{-\int_0^T \lambda_m(z_1)dz_1 - (\alpha D_e + \mu_m)t} > 0$$

Applying the same method above to other state variables shows that the solutions are positive for all time $t > 0$.

2.4 Existence of Equilibrium

An equilibrium point is a point where all rate of change with respect to time is constant

At equilibrium state,

$$\frac{dM_s}{dt} = \frac{dM_{ed}}{dt} = \frac{dM_{em}}{dt} = \frac{dM_{dm}}{dt} = \frac{dM_{sd}}{dt} = \frac{dM_{im}}{dt} = \frac{dS_{dm}}{dt} = \frac{dV_s}{dt} = \frac{dV_i}{dt} = \frac{dE}{dt} = \frac{dE_d}{dt} = 0 \quad (30)$$

In this model, we have four (4) different equilibria: we have the drought-free and MSV-free equilibrium, the drought-free and MSV-persistence equilibrium, the drought-persistence and MSV-free equilibrium and lastly the drought-persistence and MSV-persistence equilibrium points. This is an equilibrium point where there is no disease in the maize population but there is persistence of drought. In this case, $\lambda_m = 0, \alpha D_e > 0, \lambda_v = 0$. This implies that $M_{em} = M_{dm} = M_{im} = S_{dm} = V_i = 0$. Applying substitution method, we have the disease-free and drought endemic equilibria given by:

$$\Omega_d^* = \begin{pmatrix} M_s^* \\ M_{ed}^* \\ M_{em}^* \\ M_{dm}^* \\ M_{sd}^* \\ M_{im}^* \\ S_{dm}^* \\ V_s^* \\ V_i^* \\ E^* \\ E_d^* \end{pmatrix} = \begin{pmatrix} \frac{\pi_m}{(\alpha D_e + \mu_m)} \\ \frac{\alpha D_e \pi_m}{(\alpha D_e + \mu_m)(\sigma_1 + \phi_1 + \mu_m)} \\ 0 \\ \frac{\phi_1 \alpha D_e \pi_m}{(\gamma_2 + \mu_m)(\alpha D_e + \mu_m)(\sigma_1 + \phi_1 + \mu_m)} \\ \frac{\sigma_1 \alpha D_e \pi_m}{(\gamma_1 + \mu_m + \delta_1)(\alpha D_e + \mu_m)(\sigma_1 + \phi_1 + \mu_m)} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (31)$$

This represents an equilibrium point characterized by the ongoing presence of disease within the maize population, alongside the continued occurrence of drought. In this case, $\lambda_m > 0, \alpha D_e > 0, \lambda_v > 0$. This implies that $M_{em} \neq 0, M_{dm} \neq 0, M_{im} \neq 0, S_{dm} \neq 0, V_i \neq 0$.

Hence, our disease endemic and drought endemic equilibrium points is given as:

$$\Omega^{****} = \begin{pmatrix} M_s^{****} \\ M_{ed}^{****} \\ M_{em}^{****} \\ M_{dm}^{****} \\ M_{sd}^{****} \\ M_{im}^{****} \\ S_{dm}^{****} \\ V_s^{****} \\ V_i^{****} \\ E^{****} \\ E_d^{****} \end{pmatrix} = \begin{pmatrix} \frac{\pi_m}{\lambda_m^{****} + g_1} \\ \frac{\alpha D_e \pi_m}{g_2 (\lambda_m^{****} + g_1)} \\ \frac{\pi_m \lambda_m^{****}}{(\sigma_1 + \phi_1 + \mu_m) g_1} \\ \frac{\lambda_m^{****} \pi_m}{g_3 (\lambda_m^{****} + g_1)} \\ \frac{\pi_m (g_3 \phi_1 \alpha D_e + g_2 \phi_2 \lambda_m^{****})}{g_2 g_3 g_4 (\lambda_m^{****} + g_1)} \\ \frac{\sigma_1 \alpha D_e \pi_m}{g_1 g_2 (\lambda_m^{****} + g_1)} \\ \frac{\sigma_2 \lambda_m^{****} \pi_m}{g_1 g_3 (\lambda_m^{****} + g_1)} \\ \frac{\gamma_1 M_{sd}^{****} + \gamma_2 M_{dm}^{****} + \gamma_3 M_{im}^{****}}{g_7} \\ \frac{\pi_v}{\lambda_v^{****} - \mu_v} \\ \frac{\lambda_v^{****} \pi_v}{\mu_v (\lambda_v^{****} + \mu_v)} \\ \frac{\lambda_v^{****} \pi_v}{\mu_v (\lambda_v^{****} + \mu_v)} \\ \frac{\eta E_d^{****}}{\kappa} \\ \frac{\kappa E^{****}}{\eta} \end{pmatrix} \quad (32)$$

2.5 Basic reproduction number of the non-genetically maize plants model

This section studies the basic reproduction number, which is a threshold parameter that controls the spread of a disease. To obtain the basic reproduction number, we apply the next generation approach described in [Van Den Driessche & Watmough \(2002\)](#). The associated incidence term F and the transfer term V for the model are respectively obtained as:

$$F_m = \begin{pmatrix} M_{em} \\ M_{dm} \\ M_{im} \\ S_{dm} \\ V_i \end{pmatrix} = \begin{pmatrix} \frac{\beta_{vm} V_i (\theta E + \theta_d E_d) M_s}{N_v} \\ 0 \\ 0 \\ 0 \\ \frac{\beta_{mv} (M_{im} + \tau S_{dm}) (\theta E + \theta_d E_d) V_s}{N_m} \end{pmatrix}, \quad V_m = \begin{pmatrix} (\sigma_2 + \phi_2 + \mu_m) M_{em} \\ -\phi_1 M_{ed} - \phi_2 M_{em} + (\mu_m + \gamma_2) M_{dm} \\ -\sigma_2 M_{em} + (\gamma_3 + \delta_2 + \mu_m) M_{im} \\ -\gamma_1 M_{sd} - \gamma_2 M_{dm} - \gamma_3 M_{im} + (\mu_m + \delta_3) S_{dm} \\ \mu_v V_i \end{pmatrix} \quad (33)$$

Finding the Jacobian for the incidence and transfer terms evaluated at drought and MSV-free equilibrium points resulted into

$$F_m = \begin{pmatrix} 0 & 0 & 0 & 0 & \frac{\beta_{vm} x M_s^0}{N_v^0} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_{mv} x V_s^0}{N_m^0} & \frac{\beta_{mv} x \tau V_s^0}{N_m^0} & 0 \end{pmatrix} \quad \text{and} \quad V_m = \begin{pmatrix} g_3 & 0 & 0 & 0 & 0 \\ -\phi_2 & g_4 & 0 & 0 & 0 \\ -\sigma_2 & 0 & g_6 & 0 & 0 \\ 0 & -\gamma_2 & -\gamma_3 & g_7 & 0 \\ 0 & 0 & 0 & 0 & \mu_v \end{pmatrix} \quad (34)$$

Finding the inverse matrix of the transfer term, we have

$$V_m^{-1} = \begin{pmatrix} \frac{1}{g_3} & 0 & 0 & 0 & 0 \\ \frac{\phi_2}{g_3 g_4} & \frac{1}{g_4} & 0 & 0 & 0 \\ \frac{\sigma_2}{g_3 g_6} & 0 & \frac{1}{g_6} & 0 & 0 \\ \frac{\gamma_3 \sigma_2 g_4 + \gamma_2 \phi_2 g_6}{g_3 g_4 g_6 g_7} & \frac{\gamma_2}{g_7 g_4} & \frac{\gamma_3}{g_6 g_7} & \frac{1}{g_7} & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{\mu_v} \end{pmatrix}, \quad (35)$$

where

$$\left. \begin{aligned} g_1 &= (\alpha D_e + \mu_m), & g_2 &= (\sigma_1 + \phi_1 + \mu_m), & g_3 &= (\sigma_2 + \phi_2 + \mu_m), \\ g_4 &= (\gamma_2 + \mu_m), & g_6 &= (\gamma_3 + \mu_m + \delta_2), & g_7 &= (\mu_m + \delta_3) \end{aligned} \right\}$$

Thus, the effective reproductive number of system model (13) is calculated from the

spectral radius $\rho(FV^{-1})$, therefore, the non-genetic MSV reproduction number is

$$R_0^{ng} = \frac{\sqrt{g_4 g_7 g_3 g_6 \beta_{vm} \mu_v \beta_{mv} x (\sigma_2 (\gamma_3 + g_7) g_4 + \tau g_6 \gamma_2 \phi_2)}}{g_4 g_3 g_6 g_7 \mu_v} \quad (36)$$

2.6 Global stability of the non-genetically modified maize plant disease-free and drought-free equilibrium points

In this section, we examined the global stability of the drought and disease-free equilibrium points that are free from both disease and drought for the model (1) was investigated using the theorem by Castillo et al. (2002)

Lemma 1 Consider a model system written in the form

$$\left. \begin{aligned} \frac{dX}{dt} &= F(X, Z) \\ \frac{dZ}{dt} &= G(X, Z), (X, 0) = 0 \end{aligned} \right\} \quad (37)$$

where $X = (M_s, M_{ed}, M_{sd}, V_s, E, E_d)$ and $Z = (M_{em}, M_{dm}, M_{im}, S_{dm}, V_i)$ with the components of $X \in \mathbb{R}^6$ denoting the uninfected population and the components of $Z \in \mathbb{R}^5$ denoting infected population.

The disease free equilibrium is now denoted as:

$$\Omega^0 = \begin{pmatrix} \frac{\pi_m}{\mu_m} \\ 0 \\ 0 \\ \frac{\pi_v}{\mu_v} \\ 0 \\ 0 \end{pmatrix} \quad (38)$$

The following two conditions H_1 and H_2 must be met to guarantee a global asymptotic stability:

Assume that:

$$(H_1). \quad \text{For } \frac{dX}{dt} = F(X, 0) \text{ is globally asymptotically stable} \quad (36)$$

$$(H_2). \quad G(X, Z) = PZ - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0, \text{ For } (X, Z) \in \Omega_m \quad (37)$$

where $P = D_Z G(X^0, 0)$ is a M -matrix (the off-diagonal elements of P are non-negative) and Ω is the region where the model makes biological sense. Then, the $DDFE$, X^* is globally asymptotically stable if $R_0 < 1$ (Castillo-Chavez et al. 2002).

Theorem 4: The non-genetically modified ma-free equilibrium point $\Omega_0 = (X^*, 0)$ of the

system model (1) is globally asymptotically stable provided $R_0^{ng} < 1$ and the conditions (H1) and (H2) are satisfied.

Proof: from the model (1) we can get $F(X, Z)$ and $G(X, Z)$

$$F(X, Z) = \begin{pmatrix} \pi_m - (\lambda_d + \lambda_m + \mu_m)M_s \\ \alpha D_e - (\sigma_1 + \phi_1 + \mu_m)M_{ed} \\ \pi_v - \lambda_v V_s - \mu_v V_s \\ -\kappa E \\ \kappa E_d \end{pmatrix} \quad (38)$$

$$G(X, Z) = \begin{pmatrix} \lambda_m M_s - (\sigma_2 + \phi_2 + \mu_m)M_{em} \\ \phi_2 M_{em} - (\gamma_2 + \mu_m)M_{dm} \\ \sigma_2 M_{em} - (\gamma_3 + \delta_2 + \mu_m)M_{im} \\ \gamma_2 M_{dm} + \gamma_3 M_{im} - (\delta_3 + \mu_m)S_{dm} \\ \lambda_v V_s - \mu_v V_i \end{pmatrix} \quad (39)$$

At disease free equilibrium, equation (37) becomes

$$F(X, 0) = \begin{pmatrix} \pi_m - \mu_m M_s \\ 0 \\ 0 \\ \pi_v - \mu_v V_s \\ 0 \\ 0 \end{pmatrix} \quad (40)$$

From the system (40), it can be seen that

$\Omega^0 = \left\{ \frac{\pi_m}{\mu_m}, 0, 0, 0, 0, 0, \frac{\pi_v}{\mu_v}, 0, 0, 0 \right\}$ is Globally-Asymptotically Stable (GAS).

This can be verified from the solutions, namely:

$$\begin{aligned} M_s(t) &= \frac{\pi_m}{\mu_m} + \left[M_s(0) - \frac{\pi_m}{\mu_m} \right] e^{-\mu_m t} \\ V_s(t) &= \frac{\pi_v}{\mu_v} + \left[V_s(0) - \frac{\pi_v}{\mu_v} \right] e^{-\mu_v t} \\ 0 \\ E(t) &= E(0)e^{-\kappa t} \\ E_d(t) &= E_d(0)e^{-\eta t} \end{aligned} \quad (41)$$

As $t \rightarrow \infty$, the solutions

$$(M_{ed}(t), M_{em}(t), M_{dm}(t), M_{sd}(t), M_{im}(t), S_{dm}(t), V(t)_i, E(t), E_d) = \left\{ \frac{\pi_m}{\mu_m}, 0, 0, 0, 0, 0, \frac{\pi_v}{\mu_v}, 0, 0, 0 \right\}$$

which implies a global convergence of the system (1) in Ω_m , and this satisfied condition H_1 .

The second condition demands that:

$$\hat{G}(X, Z) = PZ - G(X, Z) \quad (42)$$

and that $\hat{G}(X, Z) \geq 0, \quad \forall (X, Z) \in \Omega_m$

we already know that from equation (39),

$$G(X, Z) = \begin{pmatrix} \lambda_m M_s - (\sigma_2 + \phi_2 + \mu_m) M_{em} \\ \phi_2 M_{em} - (\gamma_2 + \mu_m) M_{dm} \\ \sigma_2 M_{em} - (\gamma_3 + \delta_2 + \mu_m) M_{im} \\ \gamma_2 M_{dm} + \gamma_3 M_{im} - (\delta_3 + \mu_m) S_{dm} \\ \lambda_v V_s - \mu_v V_i \end{pmatrix} \quad (39)$$

$$\hat{G}(X, Z) = PZ - G(X, Z). \quad (40)$$

where P is an $n \times n$ matrix, Z is a column vector and $G(X, Z)$ is a column vector formed from the infectious compartments.

By computing the Jacobian of equation (39) and evaluating at disease-free equilibrium, gives

$$P = \begin{pmatrix} -g_3 & 0 & 0 & 0 & \frac{\beta_{vm} x M_s^0}{N_v^0} \\ \phi_2 & -g_4 & 0 & 0 & 0 \\ \sigma_2 & 0 & -g_6 & 0 & 0 \\ 0 & \gamma_2 & \gamma_3 & -g_7 & 0 \\ 0 & 0 & \frac{\beta_{mv} x V_s^0}{N_v^0} & \frac{\beta_{mv} x \tau V_s^0}{N_v^0} & -\mu_v \end{pmatrix} \quad (43)$$

It can be observed that the matrix P is a metzler matrix because all its off-diagonal elements are non-negative. Thus multiplying the matrix P and Z gives

$$PZ = \begin{pmatrix} -g_3 & 0 & 0 & 0 & \frac{\beta_{vm} x M_s^0}{N_v^0} \\ \phi_2 & -g_4 & 0 & 0 & 0 \\ \sigma_2 & 0 & -g_6 & 0 & 0 \\ 0 & \gamma_2 & \gamma_3 & -g_7 & 0 \\ 0 & 0 & \frac{\beta_{mv} x V_s^0}{N_v^0} & \frac{\beta_{mv} x \tau V_s^0}{N_v^0} & -\mu_v \end{pmatrix} \begin{pmatrix} M_{em} \\ M_{dm} \\ M_{im} \\ S_{dm} \\ V_i \end{pmatrix} \quad (44)$$

Evaluating equation (44), gives

$$PZ = \begin{pmatrix} \frac{\beta_{vm}V_iM_s^0}{N_m^0} - g_3M_{em} \\ -g_4M_{dm} + \phi_2M_{em} \\ \sigma_1M_s - g_5M_{sd} \\ \sigma_2M_{em} - g_6M_{im} \\ \gamma_2M_{dm} + \gamma_3M_{im} - g_7S_{dm} \\ \frac{\beta_{mv}V_s^0xM_{im}}{N_v^0} + \frac{\beta_{mv}x\tau V_s^0S_{dm}}{N_v^0} - \mu_vV_i \end{pmatrix} \quad (45)$$

Now, using $\hat{G}(X, Z) = PZ - G(X, Z)$ one obtains the following

$$\hat{G}(X, Z) = \begin{pmatrix} (\beta_{vm}xV_i) \left(\frac{M_s^0}{N_v^0} - \frac{M_s}{N_v} \right) \\ 0 \\ 0 \\ 0 \\ (\beta_{mv}x)(M_{im} + \tau S_{dm}) \left(\frac{V_s^0}{N_m^0} - \frac{V_s}{N_m} \right) \end{pmatrix} \quad (46)$$

In the system (1), the total population is bounded by $\left(\frac{\pi_m}{\mu_m}, \frac{\pi_v}{\mu_v}\right)$ and $(M_{em}, M_{dm}, M_{im}, S_{dm}, V_i) \leq (M_s^0, V_s^0)$. Hence, it is clear that the matrix $\hat{G}(X, Z) \geq 0$

since $\frac{M_s^0}{N_m^0} \geq \frac{M_s}{N_m}$, $\frac{V_s^0}{N_v^0} \geq \frac{V_s}{N_v}$. Also, it is obvious that the matrix P is an M-matrix which satisfies the conditions H_1 and H_2 , we conclude that the system (1) is Globally Asymptotically Stable, (GAS) if $R_0^{ng} < 1$.

2.7 Sensitivity analysis of model parameters

In this section, sensitivity analysis of the parameters of the reproduction number model (13) was examined. This helps in checking and identifying the parameters that have impact on the basic reproductive number. To determine the impact, R_0 was partially differentiated with respect to the model parameters in equation (13). In an attempt to compute the sensitivity index, the technique outlined by [Bolaños, & Edmeades, \(1993\)](#) was adopted. This technique develops a formula to obtain the sensitivity index of all the basic parameters, defined as;

$$\Delta_y^{R_0^{ng}} = \frac{\partial R_0^{ng}}{\partial y} \times \frac{y}{R_0^{ng}} \quad (51)$$

where y represents all the basic parameters to be tested. The sensitivity of R_0^{ng} to each of the parameters in R_0^{ng} were calculated using normalized Sensitivity index. The results shows that the parameters have both positive and negative effects on R_0^{ng} . Positive SI values, such as $\beta_{vm}, \beta_{mv}, \sigma_{md}, \sigma_m, \theta, \theta_d, \tau$, it reveal that an increase in these parameters values increases R_0^{ng} , which led to the infection attacking the population. While the parameters $\gamma_{2m}, \pi_m, \pi_v, \delta_2, \delta_3, \phi_2$ have negative SI, that is, an increase in these parameters

values decrease R_0^{ng} , and as a result, the infection gradually fades from the population.

3 Numerical simulations

We performed numerical simulation of the model equations using the inbuilt RK–ode45 function in MATLAB R2021a to study the behavior of the system and effects of infection. The initial condition employed are:

$$M_s(0) = 950, M_{ed}(0) = 50, M_{em}(0) = 10, M_{dm}(0) = 5, M_{im}(0) = 8, \\ S_{dm}(0) = 2, V_s(0) = 450, V_i(0) = 50, E(0) = 0.8, E_d(0) = 0.2$$

Figures 1 and 2 are the graphs generated from numerical simulations carried out on the model equations.

3.1 Experiment One: Model Dynamics to Variation in the Vector-to-Maize Transmission Rate Over Time t

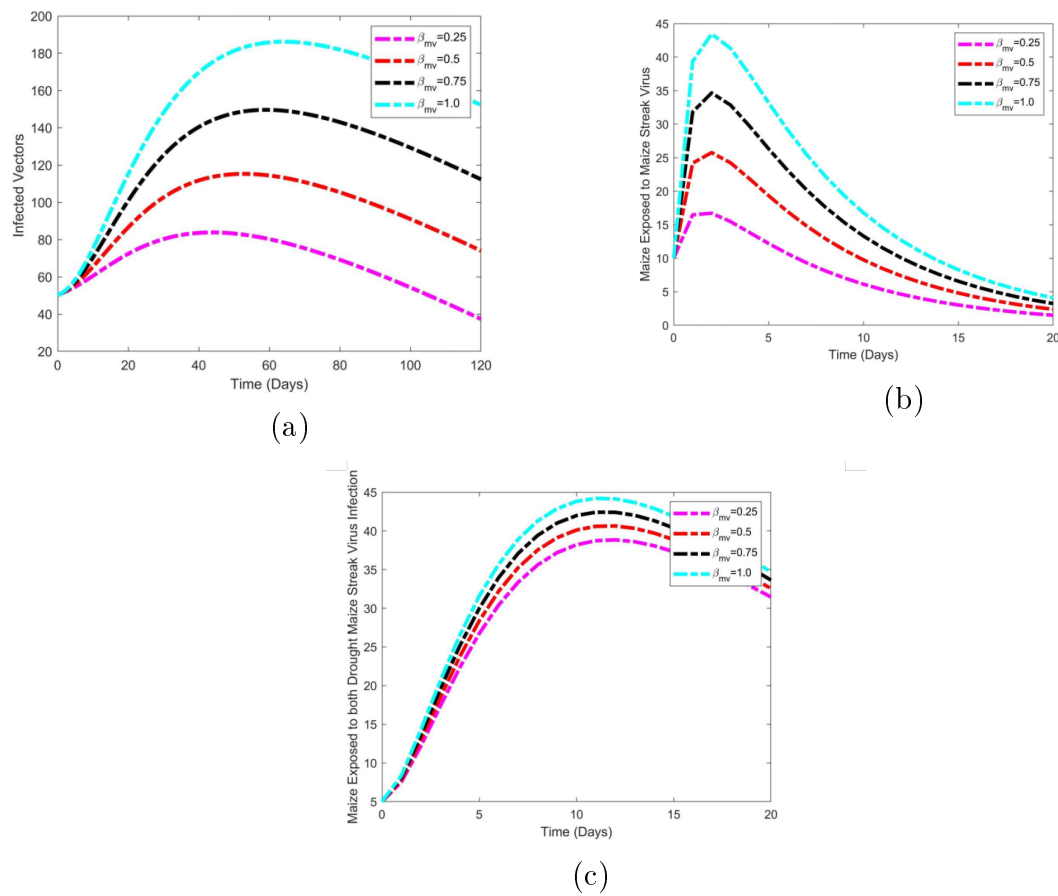


Figure 1: Effect of Infected vector on Maize Streak, Maize exposed to virus and Naize exposed to both drought streak Environment infected vectors at time t

3.2 Experiment Two: Model Dynamics to Variation in the Vector-to-Maize Transmission Rate Over Time t

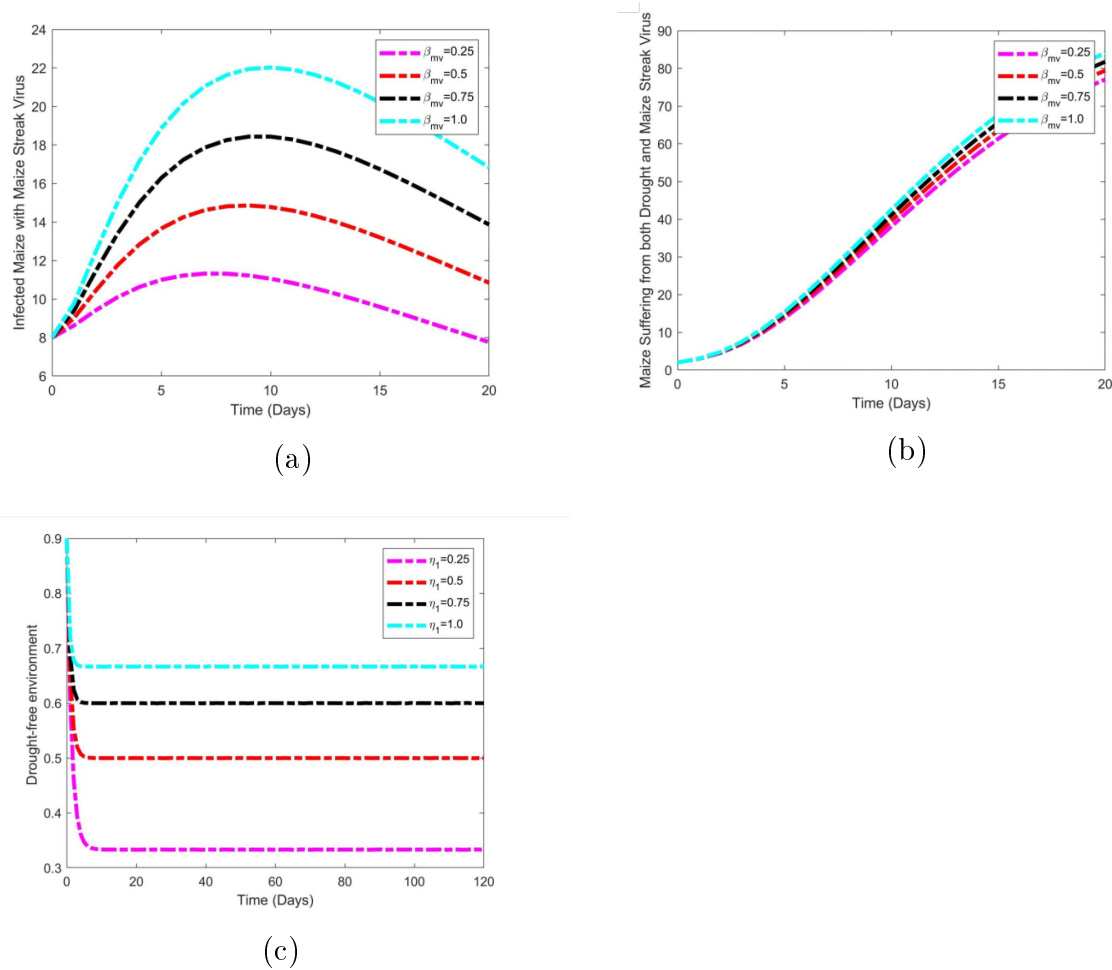


Figure 2: Effect of Infected Maize on Streak virus, Maize suffering from drought and recovery rate from drought on drought-free Environment infected vectors at time t

3.3 Discussion on Simulated Results

The variation of the vector-to-maize transmission parameter, β_{vm} , from 0.25 to 1.00 shows a consistent increase in MSV-related outcomes in the non-GM maize model. As β_{vm} increases, the transmission of MSV from infected vectors to maize becomes more effective, resulting in higher populations in the affected compartments. As shown in Figure 1 (a), increasing β_{vm} produces a substantial increase in the infected vector population. The peak rises from approximately 144 infected vectors at $\beta_{vm} = 0.75$ to about 187 vectors at $\beta_{vm} = 1.00$. The higher transmission values also maintain a larger infected-vector population throughout the simulation, demonstrating the strong influence of β_{vm} on vector infection. Similarly, Figure 1(b) shows that higher values of β_{vm} increase the number of maize plants exposed to MSV. The peak increases from approximately 17 plants for $\beta_{vm} = 0.25$ to about 43 plants for $\beta_{vm} = 1.00$. This indicates that increased vector-to-maize transmission directly increases the number of plants exposed to the virus. The effect is also evident in the infected maize population (Figure 1(c)). The peak increases from approximately 11 plants at $\beta_{vm} = 0.25$ to about 22 plants at $\beta_{vm} = 1.00$. Moreover,

the higher- β_{vm} trajectories remain above the lower transmission scenarios throughout the simulation, indicating that increased transmission not only raises the peak infection but also contributes to a greater infection burden over time.

For maize exposed to both drought and MSV (Figure 2(a)), the four trajectories also increase with β_{mv} , although the differences are relatively small. The peaks range from approximately 39 plants at $\beta_{mv} = 0.25$ to 44 plants at $\beta_{mv} = 1.00$. This relatively narrow separation suggests that both MSV transmission and the independent drought-related processes in the model influence this compartment. Finally, Figure 2(b) shows the effect of β_{mv} on maize suffering from both drought and MSV. All trajectories increase throughout the 20-day period, with the highest transmission rate producing the largest affected population. By day 20, the population increases from approximately 77 plants at $\beta_{mv} = 0.25$ to about 83 plants at $\beta_{mv} = 1.00$. The relatively close trajectories again indicate that, although vector transmission contributes to the combined burden, drought dynamics and other transition processes also play substantial roles.

4 Conclusion

The findings from the non-genetically modified (non-GM) maize model show that drought and Maize Streak Virus (MSV) can place considerable pressure on maize plants, especially when the two stresses occur together. The simulation showed a rapid reduction in the number of healthy susceptible plants, while the number of plants affected by drought and MSV changed considerably during the early part of the simulation. One of the most important findings was the large increase in the number of maize plants suffering from both drought and MSV, reaching about 17,500 plants around days 65-70. Although this number later declined, a considerable population remained affected throughout the simulation. This suggests that the combined effect of drought and MSV may be more persistent than either stress considered alone. The model also showed a gradual decline in both susceptible and infected vectors. This means that the potential for continued MSV transmission becomes weaker over time. At the same time, the environmental results showed that drought conditions developed rapidly and remained relatively stable, providing a continuing source of stress for the maize plants. Overall, the results suggest that non-GM maize can be particularly vulnerable when drought and MSV occur at the same time. The findings highlight the need to consider both factors together when developing strategies to protect maize production. The model also provides a useful baseline for assessing whether GM maize, particularly varieties designed to tolerate drought or resist MSV, can perform better under similar conditions. Taken together, Figures 1(a – c) demonstrate that increasing m_v consistently increases the MSV burden in the non-GM maize system. The strongest response occurs in the infected-vector and MSV-exposed maize populations, while the effect on the combined drought-MSV compartments is comparatively moderate. These results identify m_v as an important parameter governing MSV transmission and suggest that reducing vector-to-maize transmission through effective vector management could substantially reduce MSV exposure and infection in non-GM maize.

5 Recommendations

Based on these findings, several practical measures can be considered: Manage drought and MSV together. Since the model shows that maize can experience both stresses simultaneously, management should not focus on MSV or drought alone. An integrated approach is likely to provide better protection. Act early. The simulations show that major changes occur during the early stages of the simulation. Early monitoring and intervention could therefore help prevent drought stress and MSV infection from becoming widespread. Control MSV vectors. Reducing the number of virus-carrying vectors can help limit the spread of MSV among susceptible maize plants. Regular field monitoring can help farmers identify increasing vector populations early. Improve water management. Where possible, practices such as timely planting, water conservation, irrigation, mulching, and improved soil-water management should be encouraged to reduce the effects of drought on maize. Promote drought-tolerant and MSV-resistant varieties.

The vulnerability observed in the non-GM maize population supports continued efforts to develop and use maize varieties that can withstand drought and resist MSV infection. Strengthen field monitoring. Farmers and agricultural extension workers should monitor maize fields for early signs of drought stress, MSV infection, and increases in vector populations. Early detection can make control measures more effective. Compare non-GM and GM maize. The present model can serve as a baseline for the GM maize model. Comparing the two models will help determine whether GM maize reduces the number of plants affected by drought, MSV, or both simultaneously. Validate the model with field data. Future studies should compare the model results with actual observations of maize populations, drought conditions, MSV infection, vector abundance, and crop yield. This will improve confidence in the model and make its results more useful for agricultural planning. In summary, the model suggests that protecting non-GM maize from drought and MSV requires early, integrated management. Combining better water management, vector control, disease monitoring, and the development of resistant or tolerant maize varieties could help reduce the combined burden of drought and MSV and improve maize production.

Availability of data and material

Not Applicable.

Competing Interest

No competing interest is hereby declared by the authors.

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