MODELLING THE EFFECT OF FUSARIUM OXYSPORUM IN TRANSMISSION DYNAMICS OF FUSARIUM WILT DISEASE OF CASHEW PLANTS IN CONTEXT OF SOUTH-EASTERN TANZANIA

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Abstract: Cashew nuts are the most important cash crop in Tanzania's south-eastern region, where cashew nut farming is the primary source of income for the majority of people. Aside from their importance, cashew plants are vulnerable to Fusarium wilt, a devastating disease. In particular study a system of equations for the model is formulated and analyzed qualitatively using the stability theorem of ordinary differential equation. The stability analysis indicates that the system is stable under the specified conditions. Furthermore, the analysis shows that an increase in transmission is determined by the rate of contact between susceptible plants and infected plants via root contact, whereas numerical results show that decomposed infected plants contribute to the growth of Fusarium oxysporum, increasing the disease outbreak.

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1. INTRODUCTION

Fusarium wilt is the most destructive plant disease affecting various plants worldwide [1]–[3]. This disease was first recorded in Australia 1876 affecting bananas [4]. Fusarium wilt has been the most damaging agricultural disease affecting numerous plants across the globe, including cotton, tomatoes and conifers[5]–[9]. On the other hand Fusarium wilt also affects cashew plants and was first confirmed to be a destructive disease in various parts of Tanzania in 2012, including Magawa village in the Coast region, Nanganga, Lindumbe, and Mnongodi villages in Mtwara region [2]. The cashew plant is grown all over the world and produces cashew apples and cashew nuts.

The continents that produce Cashew nuts are Asia, Africa, South America and Australia, where 40% of global production comes from Africa. Tanzania is the third largest producer in Africa and accounts for about 20% of Africa's total production [10]. Most of the production in Tanzania (about 80%) comes from the south-eastern part of the country, Lindi and Mtwara regions [11].

Cashew nut is the main source of income for many families in the south-eastern regions of Tanzania, but cashew nut also accounts for up to 10% of the overall amount of foreign exchange earnings in Tanzania [12]. Regardless of high quantity contribution to the nation and continent at large, the production of Cashew nut in the south-eastern region of Tanzania is below   production standard of Cashew nut [11].

The standard production of cashew nut is 800 kg per hectare while the actual production decreases from 800 kg to 500 kg per hectare. Several factors including drought, decreasing soil fertility, insect pests and diseases have been attributed to the failure to achieve the ultimate production goal [13], [14]. The disease factor, among others, is the key reason that influences the production of cashew nuts. Pestalotia leaf stain, Fusarium wilt, cashew leaf and nut blight, anthracnose, dieback, damping off, and cashew powdery mildew disease are the most significant cashew plant diseases [11]. Cashew plants are affected by Fusarium wilt disease, which contributes to harvest losses of up to 100% if not managed and thus reduces yield [2].

The Fusarium wilt disease is induced by Fusarium Oxysporum fungus [5], [6], [15], [16]. The infection is initiated by chlamydospores through roots or wound penetration [7]. At any stage of

the plant growth the symptoms of Fusarium wilt may appear. The symptoms of Fusarium wilt disease is the yellowing and wilting of the leaves, xylem vessels develop into slightly red color and are noticeable as lines or dots within the stem [1], [16]–[18]. The entire leaves wilt and ultimately the tree dies within two to three weeks from the onset of symptoms [2], [19]. The focus was on understanding the evolution of different states associated with disease transmission.

Mathematical modelling play a vital role in analyzing the dynamics of disease transmission in various population including plants. The study by [20], [21] analyzed disease in plants and suggested the possible control measures. On other hand, [22], [23] investigated the epidemiological model of fungus in plants and analyzed the disease severity and provide enough information to make appropriate decisions. This study therefore intends to evaluate the impact of Fusarium oxysporum in transmission of Fusarium wilt disease in cashew plants in South-eastern Tanzania.

2. MATHEMATICAL MODEL FORMULATION

We formulate mathematical model for Fusarium wilt disease by considering two populations, Cashew plants and Fusarium Oxysporum fungus population. Four compartment variables comprise the cashew plants as susceptible plants (S), exposed plants (E), infected plants (I) and recovered plants (R). Owing to the disease dynamics, the plant may shift from one compartment to another. The cashew plant, which is free of the disease but can be infected when it comes into contact with fungus, is susceptible plant. The root of susceptible plant exposed through its contact with the infected soil or infected Cashew plant via roots or wounds [3], [24], [25], [26], [27]. The total population of Cashew plants is denoted by $N = S + E + I + R$. The population of Fusarium oxysporum contains two subclasses namely, Macroconidia spores (M) and Chlamydospores (C). Macroconidia spores are the spores that appear on the surface of dead infected plants and then transform into Chlamydospores before they return to the soil [1], [17]. The model formulated under the assumption that, the growth rate of Cashew plants population

follows logistic function with growth rate r and carrying capacity k_1. The plants and fungus contact is assumed to follow the simple mass action with β as a force of infection

$$\beta = \left(\frac{\tau_1 C}{d + C} + \tau_2 I \right) S$$

where by τ_1 and τ_2 are the effective contact rate between susceptible cashew plant with chlamydospores fungus from the soil and susceptible cashew plant with infected cashew plant [3].

The parameter d represents the saturation constant rate of Chlamydospores fungus into the soil. Progression rate of exposed cashew plants to infected represent by ω. The infected cashew plant may die naturally at the rate of α or may die from the disease for a period of two to three weeks at the rate of σ. The treated infected Cashew plants may recover after 120 days at the rate of ρ and become susceptible at the rate of φ [19].

The Macroconidia spores follows logistic function with growth rate a and carrying capacity k_2. Fungus (Microconidia) attack parenchymatous tissue of the dead plants and sporulate profusely on the surface of dead plants as a Macroconidia spores at the rate of ψ [17], [28], [29]. The parameter ψ is defined as $\psi = \upsilon E + \gamma I$ where υ and γ are transformation rates of Microconidia to Macroconidia from exposed and infected plants respectively. After the decomposition of dead cashew plants, Macroconidia spores transformed into Chlamydospores fungus at the rate of η [30]. Chlamydospores leaves the compartment through decay at the rate of μ [1].

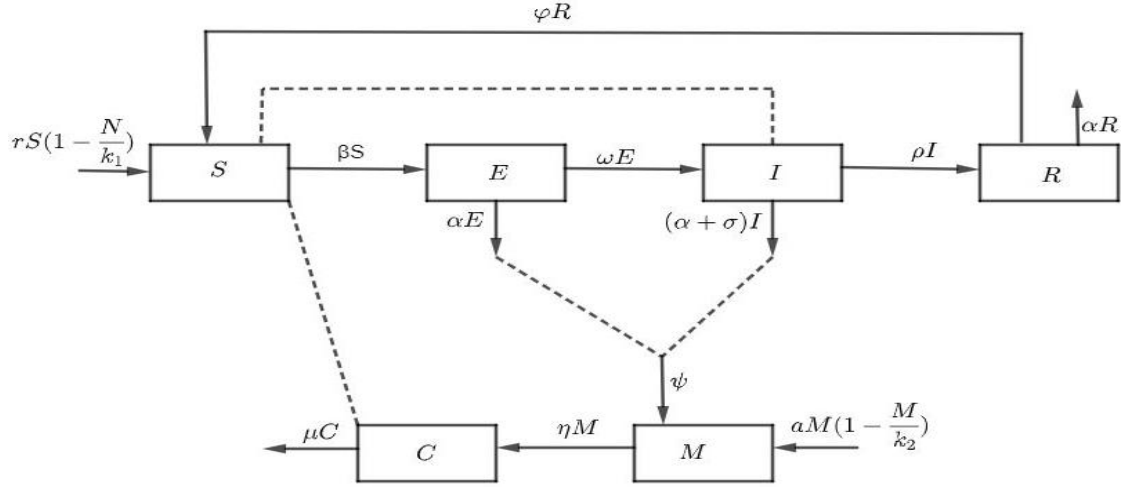Basing on the assumptions made, the following flow chart represents the summary and model formulation

Figure 1: Flow chart showing the dynamics of Fusarium wilt disease in the cashew plan

Model equations

From the Figure 1, we formulate ordinary differential equations as:

$$\frac{dS}{dt} = rS\left(1 - \frac{N}{k_1}\right) + \varphi R - \left(\frac{\tau_1 C}{d + C} + \tau_2 I\right)S$$

$$\frac{dE}{dt} = \left(\frac{\tau_1 C}{d + C} + \tau_2 I\right)S - (\alpha + \omega)E$$

$$\frac{dI}{dt} = \omega E - (\alpha + \sigma + \rho)I \tag{1}$$

$$\frac{dR}{dt} = \rho I - (\alpha + \varphi)R$$

$$\frac{dM}{dt} = \upsilon E + \gamma I + aM\left(1 - \frac{M}{k_2}\right) - \eta M$$

$$\frac{dC}{dt} = \eta M - \mu M$$

Subject to the following initial conditions:

$$S \geq 0, E \geq 0, I \geq 0, R \geq 0, M \geq 0, C \geq 0$$

3. METHODS AND SOLUTION

3.1. Positivity and Boundedness of the Model

The set off points $x_i = (x_1, x_2, x_3, x_4, x_5, x_6)$ in $\Re_+^6$ have positive coordinates which the solution of

the model (1) is represented by $\Re_+^6$. With the initial values $x^0 = (x_1^0, x_2^0, x_3^0, x_4^0, x_5^0, x_6^0)$ we

investigate the system of equation (1).We formulate the following lemma using the approach of

Lungu et al, [31]:

Lemma1. The system (1) can be transformed into following differential inequalities

$$\frac{dx_i}{dt} \geq A_i x_i + \sum_{j=1}^n B_{ij} x_j + \varepsilon, (i = 1, 2, 3..., n), \tag{2}$$

Where $B_{ij} \geq 0, \varepsilon \geq 0$. If $x_i(0) \geq \varepsilon$ then $x_i(t) \geq 0$ for all $t > 0$ and $1 \leq i \leq n$. We assume that

$\varepsilon > 0$ it approximates the system with the sequence $\varepsilon = \varepsilon \downarrow 0$

Proof. Assume that $x_i(0) \geq \varepsilon$ for $1 \leq i \leq 6$ is not true. As a result, there is a minimal integer

$t_0 > 0$ such that $x_i(t) > 0$ for $1 \leq i \leq n$, $0 \leq t \leq t_0$, $x_i(t_0) = 0$ for at least i say $i = i_0$. Then x_{i0} is a

decreasing function at $t = t_0$, resulting in $\dfrac{dx_{i0}}{dt}(t_0) \leq 0$. \tag{3}

At $x_{i0}(t)$, the differential inequality (3) becomes

$$\frac{dx_{i0}}{dt}(t_0) \geq \sum_{j=1}^n B_{ij} x_i + \varepsilon \geq \varepsilon > 0 \tag{4}$$

This is direct contradiction to our lemma (1). For the state variable in our model, we always take

$$S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0, M(0) \geq 0, C(0) \geq 0 \tag{5}$$

Hence in the region $\Re_+^6$ the model equation is epidemiologically significant and may be utilized

to analyze Fusarium Wilt in Cashew plants.

Stead state

The model (1) has the disease free equilibrium DFE as $\phi^0 = \left(S^0, 0, 0, 0, 0, 0 \right) = \left(k_1, 0, 0, 0, 0, 0 \right)$

The basic reproduction number $\mathfrak{R}_0$, is defined as the average number of secondary infections produced by infectious individual in its life time [32]. The computation of reproduction number was done by next generation method as proposed by Van den Driessche and Watmough [33].

The model basic reproduction number obtained by choosing the largest eigenvalue in absolute terms that is the spectra radius of the matrix FV^{-1} (next generation matrix) is given by

$$\mathfrak{R}_0 = \mathfrak{R}_C + \mathfrak{R}_I$$

$$\mathfrak{R}_C = \frac{k_1 \eta \tau_1 \left(\gamma \omega + \upsilon \left(\alpha + \rho + \sigma \right) \right)}{d \mu \left(\eta - a \right) \left(\alpha + \rho + \sigma \right) \left(\alpha + \omega \right)} \quad \text{and} \quad \mathfrak{R}_I = \frac{k_1 \omega \tau_2}{\left(\alpha + \rho + \sigma \right) \left(\alpha + \omega \right)}$$

Hence

$$\mathfrak{R}_0 = \frac{k_1 \eta \tau_1 \left(\gamma \omega + \upsilon \left(\alpha + \rho + \sigma \right) \right)}{d \mu \left(\eta - a \right) \left(\alpha + \rho + \sigma \right) \left(\alpha + \omega \right)} + \frac{k_1 \omega \tau_2}{\left(\alpha + \rho + \sigma \right) \left(\alpha + \omega \right)}$$

The basic reproduction number generated via two causative sources that is contaminated environment and infected cashew plants. The the average length the time used for the chlamydospores to be saturated in the soil $\frac{1}{d}$ and the average length of time spends for transformation process from Macroconidia spores to Chlamydospores

$\frac{\eta \left(\left(\alpha + \rho + \sigma \right) \alpha + \left(\sigma + \alpha \right) \omega \right)}{\mu \left(\eta - a \right)}$ attributes the $\mathfrak{R}_0$.

3.2. Short -Term Behavior of the Disease-Free Equilibrium Point

All the equations in the model system (1) are considered and examined at the disease-free equilibrium point; where the short term behavior is described by using the Jacobian matrix of the model system, necessary and sufficient condition for local stability of the system is evaluated through real parts of the eigenvalues. The system at DFE presented as

$$
J_{\phi^0} = \begin{bmatrix}
-r & 0 & 0 & \varphi & 0 & \dfrac{-k_1\tau_1}{d} \\
0 & -(\alpha+\omega) & \tau_2 & 0 & 0 & \dfrac{k_1\tau_1}{d} \\
0 & \omega & -(\alpha+\sigma+\rho) & 0 & 0 & 0 \\
0 & 0 & \rho & -(\alpha+\varphi) & 0 & 0 \\
0 & \upsilon & \gamma & 0 & -(\eta-a) & 0 \\
0 & 0 & 0 & 0 & \eta & -\mu
\end{bmatrix} \tag{6}
$$

From (6) the first two eigenvalues are $-r$ and $-(\alpha+\varphi)$. Further (6) reduced to:-

$$
D = \begin{bmatrix}
-(\alpha+\omega) & \tau_2 & 0 & \dfrac{k_1\tau_1}{d} \\
\omega & -(\alpha+\sigma+\rho) & 0 & 0 \\
\upsilon & \gamma & -(\eta-a) & 0 \\
0 & 0 & 0 & -\mu
\end{bmatrix} \tag{7}
$$

The eigenvalues of matrix (7) are

$$
-\left(\alpha+\frac{1}{2}(\sigma+\rho+\omega)\right)+\frac{1}{2}\sqrt{\omega(\omega-2\rho-\sigma+4\tau_2)+\rho(\rho+2\sigma+\sigma^2)}
$$

$$
-\left(\alpha+\frac{1}{2}(\sigma+\rho+\omega)\right)-\frac{1}{2}\sqrt{\omega(\omega-2\rho-\sigma+4\tau_2)+\rho(\rho+2\sigma+\sigma^2)}, \quad -\mu \text{ and } -\eta+a
$$

$\lambda_1 < 0$, $\lambda_2 < 0$, $\lambda_3 < 0$, $\lambda_4 < 0$, $\lambda_5 < 0$

$\lambda_6 < 0$ if $\eta > a$ which is obvious since η depends on three variables , a, υ and γ

Since all roots are negative, then the disease free has a permanent behavior.

3.3.Long - Term Behaviors of the Disease-Free Equilibrium Point

Long-term behavior of the disease-free equilibrium point of the Fusarium wilt model is described by considering Metzler matrix method the approach of [34]. We rewrite the model system (1) into transmitting and non-transmitting component. Let us assume that P_n be non-transmitting

components, P_i be transmitting components and $P_{\phi^0,n}$ represents the disease-free points.

$$
\begin{cases}
\dfrac{dP_n}{dt} = A_1\left(P_n - P_{\phi^0,n}\right) + A_2 P_i \\[2mm]
\qquad \dfrac{dP_i}{dt} = A_3 P_i
\end{cases}
\tag{8}
$$

From the given assumption, $P_n = (S,R)^T$, $P_{\phi^0,n} = (k_1,0,0,0,0,0)$, $P_i = (E,I,M,C)$

$$
P_n - P_{\phi^0,n} = \begin{pmatrix} S - k_1 \\ R \end{pmatrix}
$$

The disease-free equilibrium point to have a long -term behavior, we are supposed to verify that

the matrix A_1 has a real and negative eigenvalue and also A_3 is a Metzler matrix. Metzler matrix

is a square matrix with all elements off the diagonal are non-negative. Consider equation (15) we

have

$$
\begin{pmatrix} rS\left(1 - \dfrac{S}{k_1}\right) + \varphi R - \left(\dfrac{\tau_1 C}{d+C} + \tau_2 I\right)S \\[2mm] \rho I - (\alpha + \varphi)R \end{pmatrix} = A_1 \begin{pmatrix} S - k_1 \\ R \end{pmatrix} + A_3 \begin{pmatrix} E \\ I \end{pmatrix}
\tag{9}
$$

and the second equation is

$$
\begin{pmatrix} \left(\dfrac{\tau_1 C}{d+C} + \tau_2 I\right)S - (\alpha + \omega)E \\[2mm] \omega E - (\alpha + \sigma + \rho)I \\[2mm] \upsilon E + \gamma I + aM\left(1 - \dfrac{M}{k_2}\right) \\[2mm] \eta M - \mu C \end{pmatrix} = A_3 \begin{pmatrix} E \\ I \\ M \\ C \end{pmatrix}
\tag{10}
$$

Where by the matrices A_1, A_2 and A_3 are

$$
A_1 = \begin{pmatrix} -r & \varphi \\ 0 & -(\alpha + \varphi) \end{pmatrix}, \quad A_2 = \begin{pmatrix} 0 & -\tau_2 k_1 & 0 & \dfrac{\tau_1 k_1}{d} \\ 0 & \rho & 0 & 0 \end{pmatrix}
$$

$$A_3 = \begin{pmatrix} -(\alpha+\omega) & -\tau_2 k_1 & 0 & \dfrac{\tau_1 k_1}{d} \\ \omega & -(\alpha+\sigma+\rho) & 0 & 0 \\ \upsilon & \gamma & -a+\eta & 0 \\ 0 & 0 & \eta & -\mu \end{pmatrix}$$

The obtained eigenvalues for the A_1 matrix are $-r$ and $-(\alpha+\varphi)$ which verify that the system

$$\frac{dP_n}{dt} = A_1\left(P_n - P_{\phi^0,n}\right) + A_2 P_i$$

Has a long -term behavior at $P_{\phi^0,n}$. Also, A_3 is a Metzler stable matrix since all of its elements in off the main diagonal are non-negative. Hence, the disease-free equilibrium point for Fusarium wilt disease has a long-term behavior and leads to the theorem 2.

3.4. Long- Term Behavior of Endemic Equilibrium Point

The Long -term behavior of the endemic equilibrium point EE of the model is explored by considering following continuous and differentiable Lyapunov function below

$$L(t) = \sum_{n=1}^{6} V_i(t)\left(y_i - y_i^* \ln y_i\right), V_i > 0$$

Where $V_i(t)$ is a Lyapunov factor, y_i represents a population variable i and y_i^* is the equilibrium point of the model at respective compartment for $i = (1,2,..,6)$ The population variable y_i as $i = (1,2,..,6)$ are $y_1 = S, y_2 = E, y_3 = I, y_4 = R, y_5 = M, y_6 = C$.

The obtained Lyapunov function is

$$L(S,E,I,R,M,C) = V_1\left(y_1 - y_1^* \ln y_1\right) + V_2\left(y_2 - y_2^* \ln y_2\right) + V_3\left(y_3 - y_3^* \ln y_3\right) +$$
$$V_4\left(y_4 - y_4^* \ln y_4\right) + V_5\left(y_5 - y_5^* \ln y_5\right) + V_6\left(y_6 - y_6^* \ln y_6\right)$$

Since the function L is differentiable, then the derivative of $L(t)$ is

$$\frac{dL(t)}{dt} = V_1(t)\left(1 - \frac{y_1^*}{y_1}\right)\frac{dy_1}{dt} + V_2(t)\left(1 - \frac{y_2^*}{y_2}\right)\frac{dy_2}{dt} + V_3(t)\left(1 - \frac{y_3^*}{y_3}\right)\frac{dy_3}{dt}$$
$$+ V_4(t)\left(1 - \frac{y_4^*}{y_4}\right)\frac{dy_4}{dt} + V_5(t)\left(1 - \frac{y_5^*}{y_5}\right)\frac{dy_5}{dt} + V_6(t)\left(1 - \frac{y_6^*}{y_6}\right)\frac{dy_6}{dt}$$

$$V_1(t)ry_1\left(1-\frac{y_1}{k_1}\right)+V_1(t)\varphi y_4-\left(\tau_1 y_3+\frac{\tau_2 y_6}{d+y_6}\right)V_1(t)y_1-V_1(t)ry_1^*\left(1-\frac{y_1}{k_1}\right)-V_1(t)\varphi\frac{y_1^*}{y_1}y_4$$

$$+\left(\tau_1 y_3+\frac{\tau_2 y_6}{d+y_6}\right)V_1(t)y_1^*+\left(\tau_1 y_3+\frac{\tau_2 y_6}{d+y_6}\right)V_2(t)y_1-(\alpha+\omega)V_2(t)y_2-\left(\tau_1 y_3+\frac{\tau_2 y_6}{d+y_6}\right)V_2(t)\frac{y_2^*}{y_2}y_1$$

$$+(\alpha+\omega)V_2(t)y_2^*+V_3(t)\omega y_2-(\alpha+\sigma+\rho)V_3(t)y_3-V_3(t)\omega\frac{y_3^*}{y_3}y_2+(\alpha+\sigma+\rho)V_3(t)y_3^*+V_4(t)\rho y_3$$

$$-(\alpha+\varphi)V_4(t)y_4-V_4(t)\rho\frac{y_4^*}{y_4}y_3+V_4(t)(\alpha+\varphi)y_4^*+V_5(t)\gamma y_3+V_5(t)\upsilon y_2+V_5(t)ay_5\left(1-\frac{y_5}{k_2}\right)$$

$$-V_5(t)\eta y_5-V_5(t)\gamma\frac{y_5^*}{y_5}y_3-V_5(t)\upsilon\frac{y_5^*}{y_5}y_2-V_5(t)ay_5^*\left(1-\frac{y_5}{k_2}\right)+V_5(t)\eta y_5^*+V_6(t)\eta y_5-V_6(t)\mu y_6$$

$$-V_6(t)\eta\frac{y_6^*}{y_6}y_5+V_6(t)\mu y_6^*$$

For simplicity, let $\dfrac{dL(t)}{dt}=A-B$ where A all positive terms are and B are the negative terms;

$$A=\begin{pmatrix} V_1(t)ry_1\left(1-\dfrac{y_1}{k_1}\right)+V_1(t)\varphi y_4+\left(\tau_1 y_3+\dfrac{\tau_2 y_6}{d+y_6}\right)V_1(t)y_1^*+\left(\tau_1 y_3+\dfrac{\tau_2 y_6}{d+y_6}\right)V_2(t)y_1 \\ +(\alpha+\omega)V_2(t)y_2^*+V_3(t)\omega y_2+(\alpha+\sigma+\rho)V_3(t)y_3^*+V_4(t)\rho y_3+V_4(t)(\alpha+\varphi)y_4^* \\ +V_5(t)\gamma y_3+V_5(t)\upsilon y_2+V_5(t)ay_5\left(1-\dfrac{y_5}{k_2}\right)+V_5(t)\eta y_5^*+V_6(t)\eta y_5+V_6(t)\mu y_6^* \end{pmatrix}$$

$$B=-\begin{pmatrix} \left(\tau_1 y_3+\dfrac{\tau_2 y_6}{d+y_6}\right)V_1(t)y_1+V_1(t)ry_1^*\left(1-\dfrac{y_1}{k_1}\right)+V_1(t)\varphi\dfrac{y_1^*}{y_1}y_4+(\alpha+\omega)V_2(t)y_2 \\ +\left(\tau_1 y_3+\dfrac{\tau_2 y_6}{d+y_6}\right)V_2(t)\dfrac{y_2^*}{y_2}y_1+(\alpha+\sigma+\rho)V_3(t)y_3+V_3(t)\omega\dfrac{y_3^*}{y_3}y_2+(\alpha+\varphi)V_4(t)y_4 \\ +V_4(t)\rho\dfrac{y_4^*}{y_4}y_3+V_5(t)\eta y_5+V_5(t)\gamma\dfrac{y_5^*}{y_5}y_3+V_5(t)\upsilon\dfrac{y_5^*}{y_5}y_2+V_5(t)ay_5^*\left(1-\dfrac{y_5}{k_2}\right) \\ +V_6(t)\mu y_6+V_6(t)\eta\dfrac{y_6^*}{y_6}y_5 \end{pmatrix}$$

If $A<B$, then $\dfrac{dL(t)}{dt}\leq 0$. The only possibility of getting $\dfrac{dL(t)}{dt}=0$ is when

$$y_1=y_1^*, y_2=y_2^*, y_3=y_3^*, y_4=y_4^*, y_5=y_5^*, y_6=y_6^*$$

From the above we have

$$y_1 = S, y_2 = E, y_3 = I, y_4 = R, y_5 = M, y_6 = C$$

Since we have two options $\frac{dL(t)}{dt} < 0$ and $\frac{dL(t)}{dt} = 0$, Then the largest compact invariant set in S, E, I, R, M, C is $\frac{dL(t)}{dt} = 0$ as a singleton E^* Which is the endemic equilibrium point of the model equation.

According to LaSalle's invariant principle by Mpeshe et al., [35] endemic equilibrium point E^* has permanent behavior in the interior region of S, E, I, R, M, C.

4. NUMERICAL RESULTS AND DISCUSSION

Sensitivity Analysis of $\mathcal{R}_0$

Sensitivity analysis is carried out to understand the influence of each parameter in the spread of the disease. The computation based on

$$\Upsilon_q^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial q} \times \frac{q}{\mathcal{R}_0}$$

The result indicates that susceptible and infected plants contact rate via root contact is the leading sensitive in disease transmission. Other positive sensitive index is the decomposed diseased plants in the field enhance grows of Fusarium oxysporum fungus in the soil, resulting in an increase in disease outbreak. The rate of transformation of Macroconidia to chlamydospores and treatment rate has the most negative sensitive.

Effect of Chlamydospores on Disease Transmission

The number of plants exposed to the disease appears to increase exponentially as the contact rate between susceptible plants and chlamydospores increases from 600 to 1100 in 9 months. It then fell to 286 after 2.5 years as a result of disease progression, as shown in Figure 2.
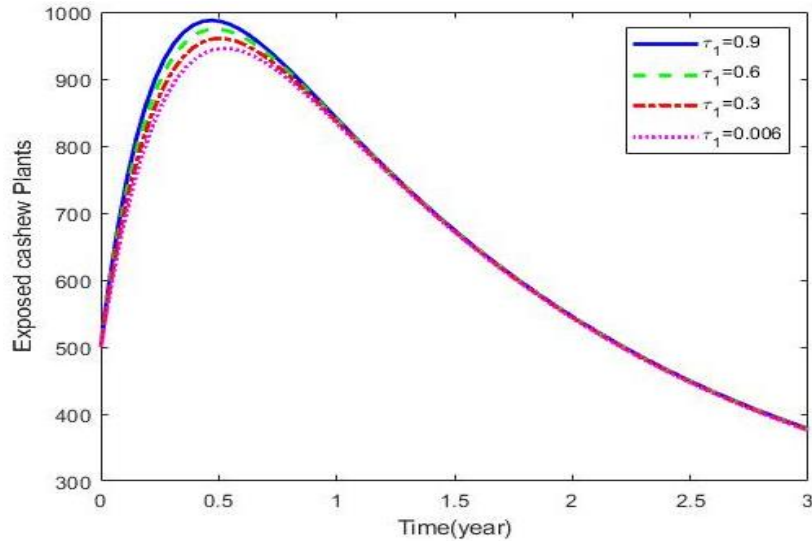
Figure 2. The effect of chlamydospores in disease transmission

Effect of Disease Transmission through Plant Roots contact

As the rate of contact between susceptible cashew plants and infected cashew plants increases, the plants that exposed to diseases increase as shown in figure 2. The rate of contact between plant roots has a significant impact on disease transmission. This implies that as the rate of interaction between plant roots rises, disease transmission tends to increase.
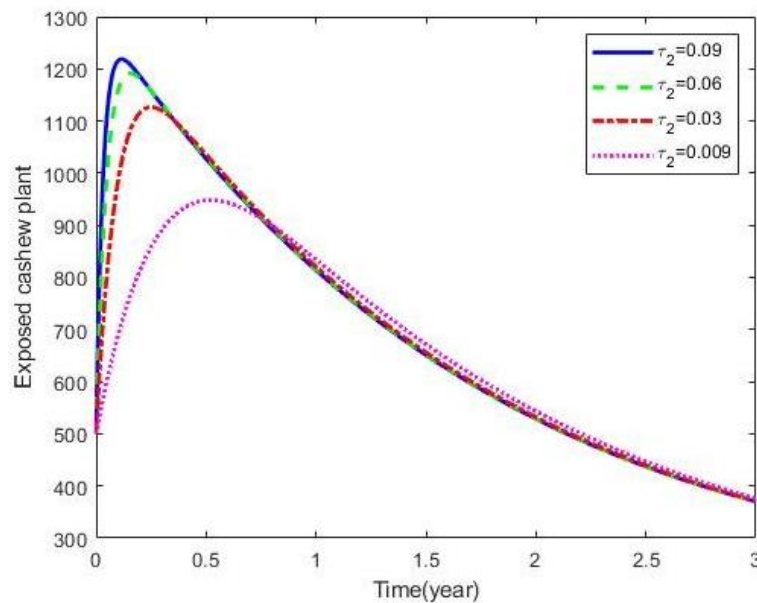

Figure 3. Effect of roots contact in disease transmission

Effect of Decomposed Infected Death Plant on disease transmission

Figures 4 show that as the number of disease-related deaths rises, the number of infected plants decreases. The increase in disease-related deaths from 0.002 to 0.5 has resulted in a reduction in the infection population from 900 to 390 in three years. This means that as the number of infected plants declines due to disease-related death, the number of fungi in the soil rises due to the presence of decomposing plants, increasing Fusarium oxysporum.
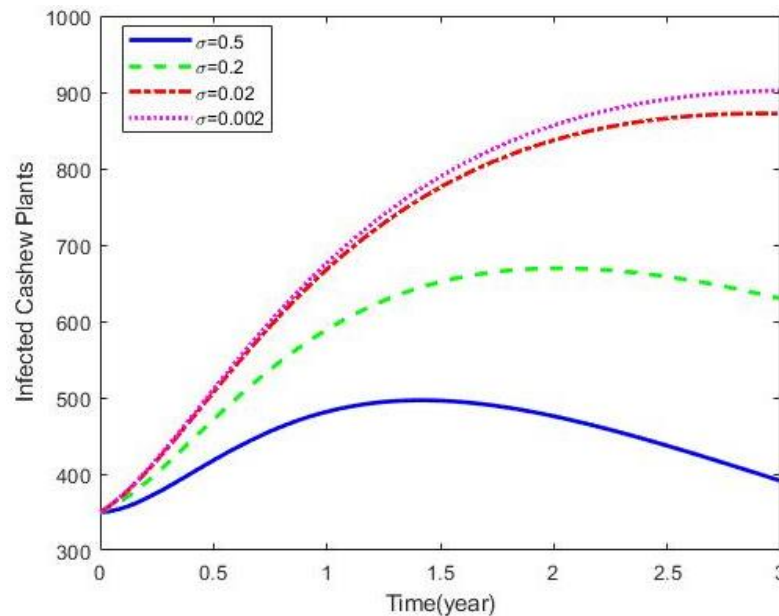
Figure 4. Effect of decomposed infected dead plants

Effect of Decomposed Infected Death Plant on growth of Fusarium Oxysporum

The amount of Macroconidia population increases as the number of decomposed death infected plants in the field increases, as shown in Figure 6. The Macroconidia population increased from 400 to 950 in five years as the transformation rate of Microconidia to Macroconidia from decomposed infected dead plants was 0.9, and the Macroconidia population decreased from 400 to 295 in five years as the transformation rate was reduced to 0.08. The increase in decomposed dead infected plants in the field causes an increase in Macroconidia population, which leads to an increase in the chlamydospores fungus population in the field.
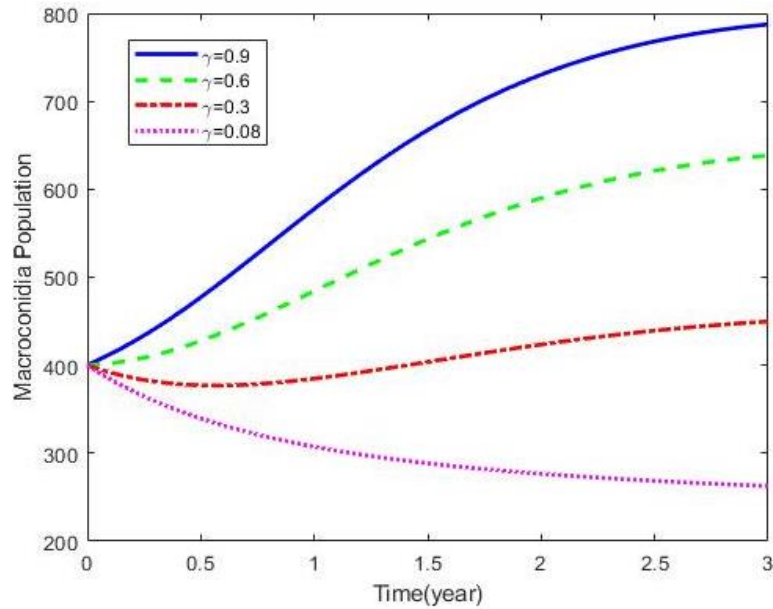
Figure 5, Effect of decomposed infected death plant on growth of Macroconidia

Effect of Fusarium Oxysporum in Disease Dynamics

As the number of exposed plants gets bigger, the infected plants raises as well, due to the progression rate of exposed to infected plants, while Macroconidia decrease as Macroconidia transform into chlamydospores, as shown in Figure 6. Then, as time passes, the exposed plants become fewer, while the infected become more numerous due to an increase in progression rate, and Macroconidia become fewer as the transformation rate to chlamydospores increases.
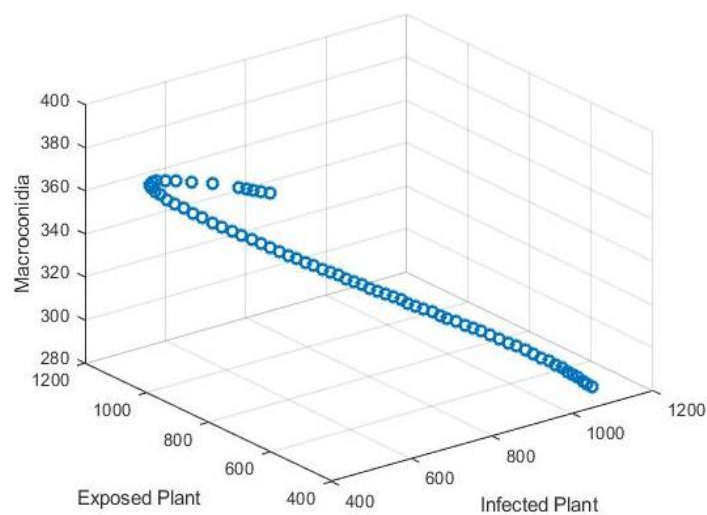
Figure 7. Effect of Macroconidia on disease transmission.
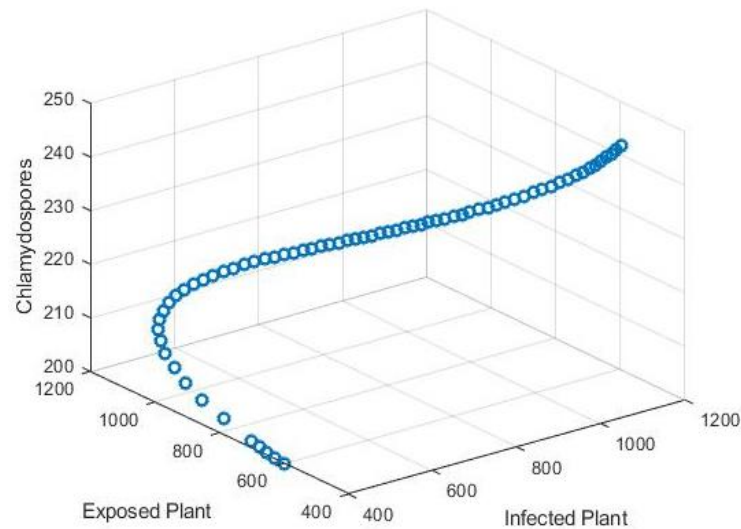
Figure 8. Effect of chlamydospores on disease transmission

Figure 7 shows that as the number of macroconidia increases, the number of exposed and infected plants increases. This is because the rate at which exposed plants become infected, as well as the decomposition of dead infected plants, increases the amount of fungus in the soil. Figure 8 shows that the number of exposed plants decreases, while the number of infected plants and chlamydospores increases, as more exposed plants become infected, and more decomposed dead infected plants increase the fungus in the soil.
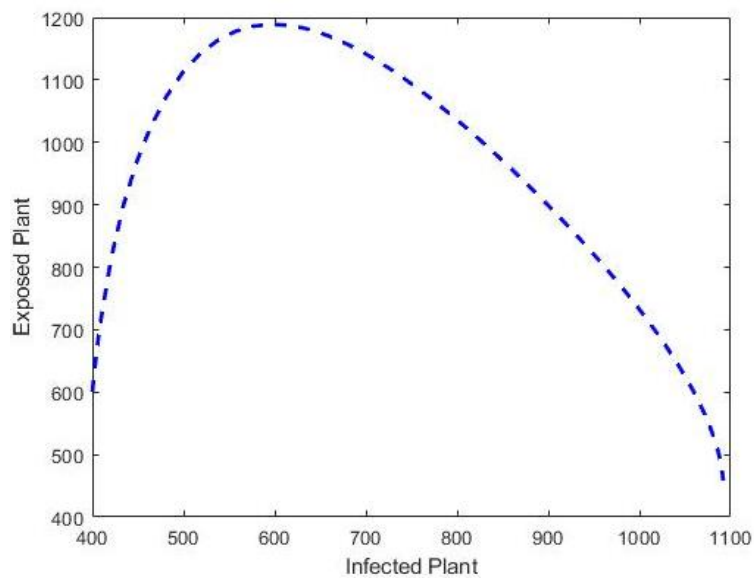
Figure 9. The comparison between infected plants with exposed plant.

Exposed plants increases at first due to the incubation period, then decrease after the fungus mature, while infected plants continue to increase over three years as shown in figure 9.
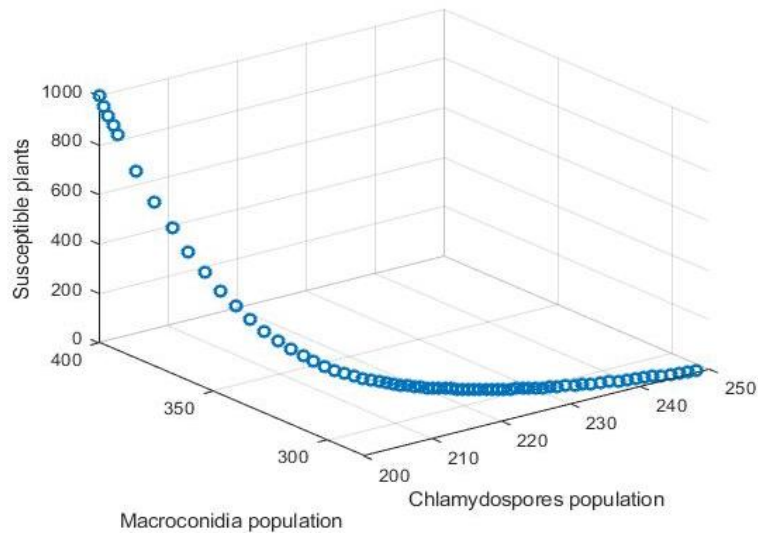

Figure 10. The effect of Fusarium oxysporum fungus on cashew plants.

The number of chlamydospores in the soil increases while Macroconidia decreases due to the rate of transformation to chlamydospores, and susceptible plants decrease as a result of disease exposure as shown in figure 10.
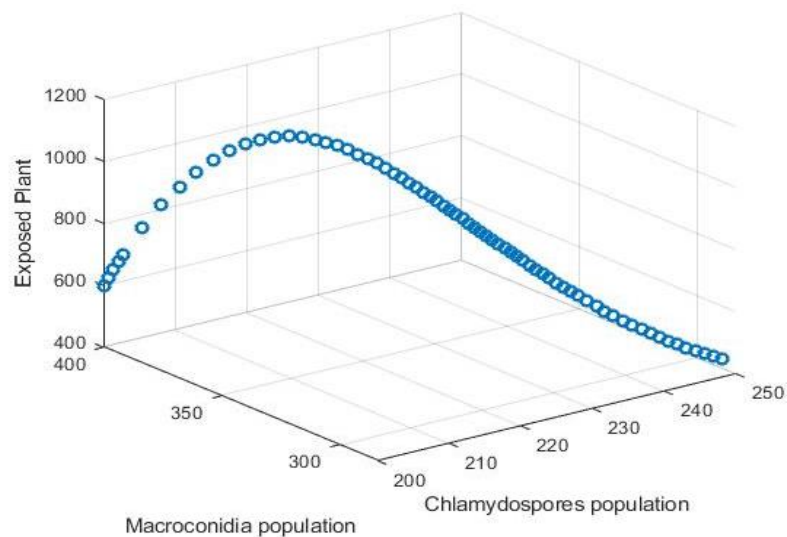

Figure 11. Effect of Fusarium oxysporum fungus on exposed plants

As the number of chlamydospores increases in the soil, the number of Macroconidia decreases due to the rate of transformation to chlamydospores, whereas exposed plants increase at first before decreasing as a result of progression to infected plants, as shown in Figure 11.

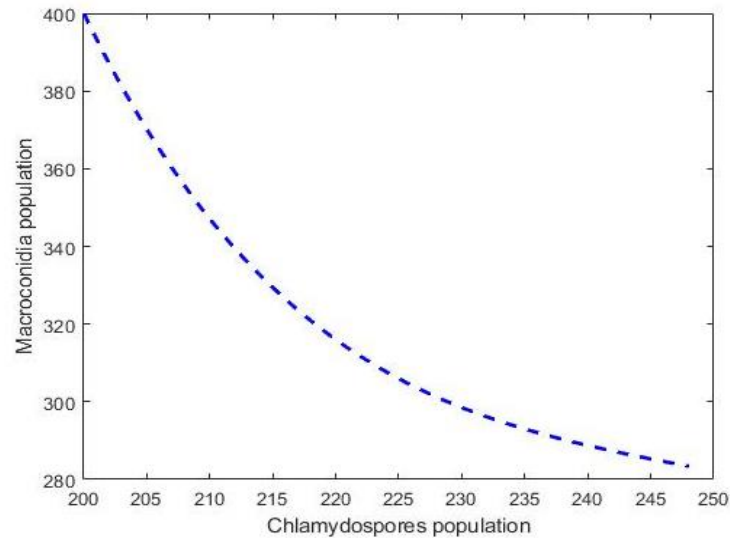**Effect of Macroconidia in the growth of Chlamydospores**

Figure 12. Comparison between Fusarium oxysporum.

The indirect proportion of Macroconidia and chlamydospores populations depicted in figure 12. The number of chlamydospores increases as the number of Macroconidia decreases. This is due to the rate at which Macroconidia transform into chlamydospores.

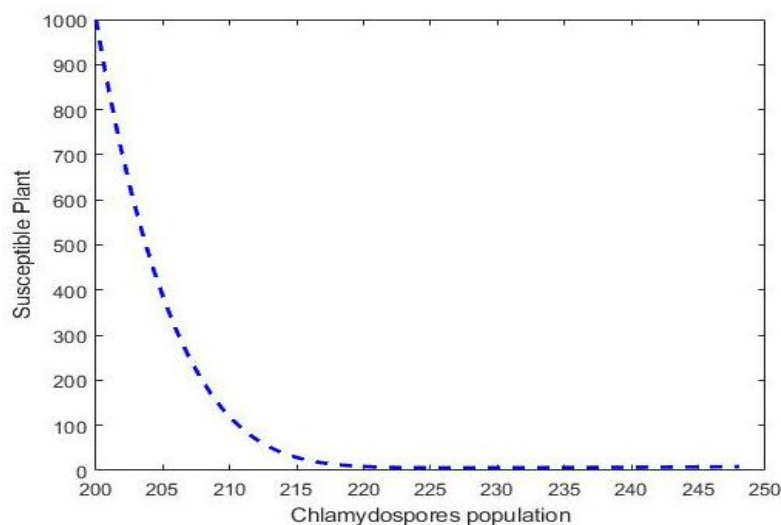**The Relationship between Fusarium Oxysporum and Cashew Plant in Disease Dynamic**

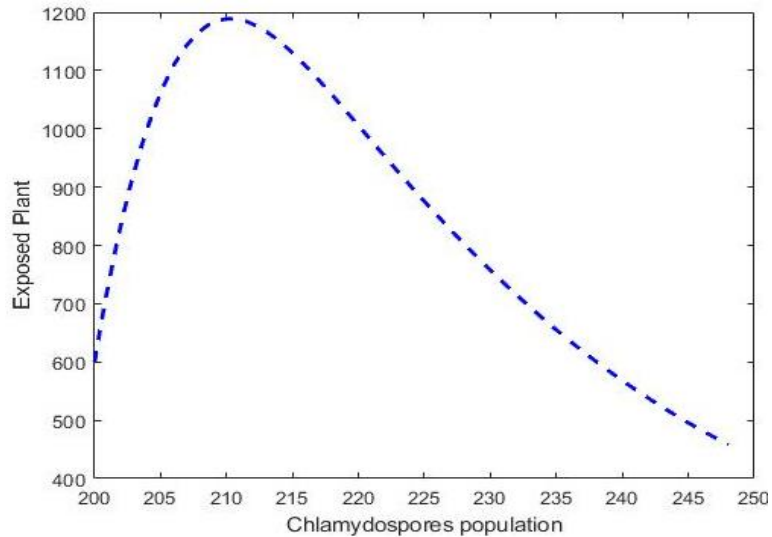Figure 13, effect of Chlamydospores to susceptible plants
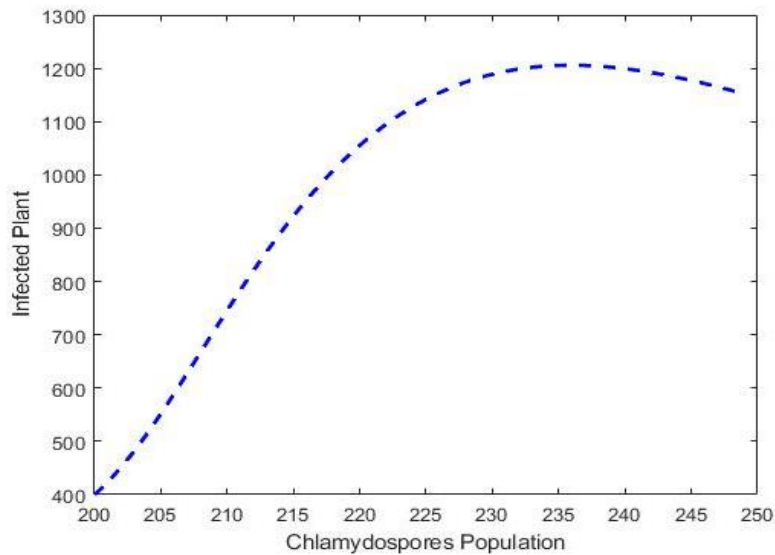
Figure 14, impact of Chlamydospores to exposed plants

Figure 15, relationship between Chlamydospores and infected plant

As the number of chlamydospores in the soil increases, the number of susceptible plants decreases due to plant exposure to the disease, as shown in Figure 13. Figure 14 shows that for the first 8 months, as the chlamydospores increase, so do the exposed plants; however, exposed plants decrease after two years, possibly because exposed plants shift to infected plants after the incubation period. Figure 15 depicts the direct proportion between the two populations; as chlamydospores increase, so do infected plants.

5. CONCLUSION

A specific study investigated and discussed the role of the Fusarium oxysporum fungus in the transmission of Fusarium wilt disease in cashew plants. To gain some quantitative insight into the dynamics of the Fusarium wilt disease, the two populations were used to build a mathematical model that included the cashew plant and the Fusarium oxysporum fungus. The study's findings illustrated that the contact rate of chlamydospores and infected plants with susceptible plants via root contact has a high impact on disease transmission, resulting in significant economic losses, particularly for communities or individuals who rely on cashew farming. The growth of the Fusarium oxysporum fungus in the soil also has an impact on the epidemic. Fusarium oxysporum growth rate is aided by an increase in the decomposition of infected plants in the field.

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CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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