A Maize Foliar Disease Mathematical Model with Standard Incidence Rate

by Windarto Windarto

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A Maize Foliar Disease Mathematical Model with Standard Incidence Rate

Windarto[1*], Fatmawati[1], and Kamara Mustiko Putri[1]

[1]Department of Mathematics, Faculty of Science and Technology, Universitas Airlangga, Indonesia

Corresponding author e-mail: windarto@fst.unair.ac.id

Abstract. In this paper, we present a mathematical model of maize foliar disease with standard incidence rate. The present model is an improvement model from Collins and Duffy, where Collins and Duffy consider a mathematical model with the bilinear incidence rate. The present model has two equilibria namely the disease-free equilibrium and endemic equilibrium. We find that the disease-free equilibrium is asymptotically stable whenever the basic reproductive ratio is less than one. On the other hand, the endemic equilibrium will exist and be asymptotically stable whenever the basic reproductive ratio is greater than one. Furthermore, we perform numerical simulations to confirm the analytical results.

1. Introduction

Maize or corn (*Zea mays L.*) is one type of food crop planted by farmers. Maize is also one of staple foods in several regions in Indonesia. The main producing areas of corn in Indonesia are West Java, Central Java, East Java, Yogyakarta, Madura, East Nusa Tenggara, North Sulawesi, South Sulawesi and Maluku. Maize is classified in *Plantae* kingdom, *Spermatophyta* phylum, *Angiospermae* subphylum, *Monocotyledonae* class, *Cyperales* order, *Poaceae* family, *Zea* genus, *Zea mays* species [1].

During one life cycle of maize plants, every part of maize is vulnerable to a number of plant diseases. The diseases can reduce the quantity and quality of maize crop yields [2]. Maize diseases could be classified into bacterial diseases, fungal diseases, nematodes/parasitic diseases, virus diseases and virus-like diseases [3]. One of fungal diseases infects maize crop is Northern corn leaf blight. This disease is caused by *Exserohilum turcicum* / *Helminthosporium turcicum* species [4]. Damage to the maize leaf area during grain filling could cause a forty percent reduction in grain yield ([5], [6], and [7]).

Mathematical modelling has significant role in understanding many real problems, including infectious diseases spreading and plant diseases spreading. Suitable mathematical models can be used to analyse the dynamics of plant diseases. Many mathematical models have been constructed and analysed to describe the dynamics of plant disease transmission. Holt et al. developed a mathematical model of African mosaic virus diseases spread. The model from Holt et al. was a host-vector model, where the model contained four compartment, namely healthy cassava, infected cassava, non-infective whitefly vectors and infective whitefly vectors [8]. Jeger et al. also developed a host-vector model for describing plant-virus transmission. Jeger et al. applied a SEIR-type and SEI-type model to analyse the dynamics of host plant population and the dynamics of insect vector population respectively [9]. Jeger et al. improved their previous model to study the interactions between host plant, virus-plant and insect as vector of the virus [10]. Jeger et al. also applied a SIR-type mathematical model to explain direct transmission of plant virus through vector mating [11]. Meng and Li investigated effect of replanting of

healthy plants and removal of infected plants as treatments to control plant diseases spreading through a mathematical model [12].

Some researchers also investigated dynamics of maize diseases through mathematical models. Stewart el al. performed a mathematical simulation of *Fusarium* growth in maize ears after artificial inoculation [13]. Paul and Munkvold used regression technique and artificial neural network for the Prediction of gray leaf spot of maize [14]. Collins and Duffy formulated a mathematical model to study the impacts of maize foliar diseases on maize population [15].

In their model, Collins and Duffy studied the dynamics of susceptible plant, infected plant and pathogen dynamics. Collins and Duffy applied bilinear incidence rate to model reduction rate of susceptible maize due to pathogen infections. Bilinear incidence rate is only accurate in the early phases of an epidemic in a population of medium size ([16], [17]). In this paper, we improve the model from Collins and Duffy by consider standard incidence rate. In the next section, we present a mathematical model of maize foliar model with standard/fractional incidence rate. Then we discuss linear stability of equilibria of the proposed model. Then, we perform numerical simulations to illustrate analytical results of this study. Finally, conclusions of this study are presented in the last section.

2. The proposed model

In this section, we proposed a mathematical model of maize foliar disease spread. The proposed model is an improvement of the model from Collins and Duffy [15]. Here, we consider standard incidence rate to explain infection rate of pathogen to susceptible plants. The model was constructed under the following assumptions:

(1) The model consists of three compartments, namely number of susceptible maize population (S), number of infected maize population (I) and number of pathogens population (P).
(2) Susceptible maize populations are planted with constant rate.
(3) Natural death rate of maize population is constant.
(4) Death rate of infected maize plants due to pathogenic infection is constant.
(5) The infection rate of susceptible maize by pathogen is constant.
(6) The increasing rate of pathogen population is proportional to the number of infected maize population.
(7) Death rate of pathogen population is constant.

Transmission diagram of the proposed model is presented in Figure 1.

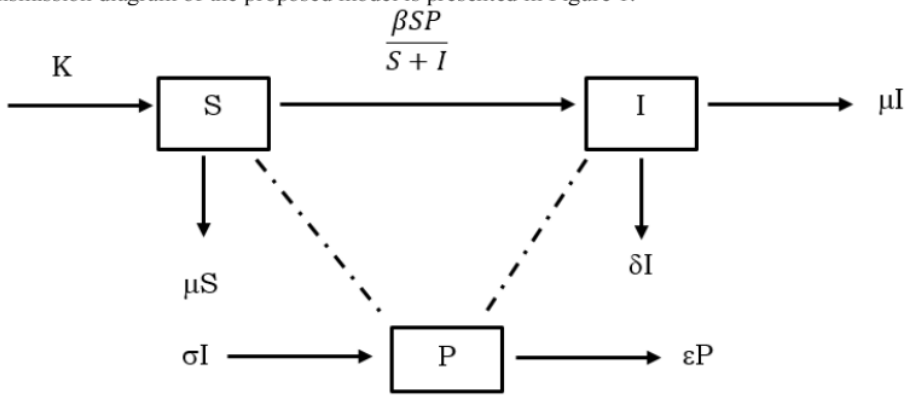

Figure 1. Transmission diagram of maize foliar disease mathematical model.

From the assumptions, the dynamics of maize foliar disease could be described by the following differential equation system:

$$\frac{dS}{dt} = K - \frac{\beta SP}{S+I} - \mu S, \quad (1)$$

$$\frac{dI}{dt} = \frac{\beta SP}{S+I} - (\mu + \delta)I, \tag{2}$$

$$\frac{dP}{dt} = \sigma I - \varepsilon P. \tag{3}$$

The region of biological interest of the model in Eq. (1)-(3) is

$$\Omega := \{(S, I, P) \in R^3, S, I, P \geq 0, S + I > 0\}. \tag{4}$$

All parameters in the model in Eq. $(1) - (3)$ are positive. When the number maize population is constant, the mathematical model with standard incidence rate in eq. (1)-(3) could be simplified into the model with bilinear incidence rate. Hence, the mathematical model with standard incidence rate could be considered as a generalization and an improvement of the model with bilinear incidence rate. Description of the parameters is presented in Table 1.

Table 1. Description of parameters of the proposed model

Parameter	Description
K	Planted rate of susceptible maize
β	Infection rate of susceptible maize by pathogen
μ	Natural death rate of maize population
δ	Death rate of infected maize plants due to pathogenic infection
σ	Increasing/growth rate of pathogen population
ε	Death rate of pathogen population

The differential equation (1) describes the dynamics of susceptible maize population. Susceptible maize population increases because of corn planting corn, and it decreases because of pathogen infection and natural death. The differential equation (2) shows the dynamics of infected maize population. Infected maize population increases due to infection of susceptible maize population by pathogen. On the other hand, infected maize population decreases due to natural deaths and pathogenic infection death. Equation (3) explains the pathogen population dynamics. Pathogen population increases due to the proliferation of pathogens in infected maize populations. On the other hand, pathogen population decreases due to natural death.

3. Analysis of the proposed model

The model in Eq. (1)-(3) has two equilibria, namely pathogen-free equilibrium (E_1) and pathogen equilibrium (E_2). The pathogen-free equilibrium is given by

$$E_1 := (S_1, I_1, P_1) = \left(\frac{K}{\mu}, 0, 0\right). \tag{5}$$

The pathogen equilibrium is given by

$$E_2 := (S_2, I_2, P_2) = \left(\frac{K\varepsilon}{(\beta\sigma - \delta\varepsilon)}, \frac{K\varepsilon(R-1)}{(\beta\sigma - \delta\varepsilon)}, \frac{K\sigma(R-1)}{(\beta\sigma - \delta\varepsilon)}\right), \tag{6}$$

where

$$R = \frac{\beta\sigma}{(\mu+\delta)\varepsilon}. \tag{7}$$

The pathogen-free equilibrium always exists. The pathogen equilibrium exists if and only if the threshold parameter $R > 1$. Here the threshold parameter R in eq. (7) is basic reproductive ratio of the proposed model in eq. (1)-(3).

The Jacobian matrix of the proposed model in Eq. (1)-(3) is given by

$$J = \begin{bmatrix} -\frac{\beta P}{S+I} + \frac{\beta SP}{(S+I)^2} - \mu & \frac{\beta SP}{(S+I)^2} & -\frac{\beta S}{S+I} \\ \frac{\beta P}{S+I} - \frac{\beta SP}{(S+I)^2} & -\frac{\beta SP}{(S+I)^2} - (\mu + \delta) & \frac{\beta S}{S+I} \\ 0 & \sigma & -\varepsilon \end{bmatrix}. \tag{8}$$

Stability of the pathogen-free equilibrium is presented in Theorem 1.

Theorem 1. *The pathogen-free equilibrium E_1 is locally asymptotically stable if the basic reproductive ration is less than one. Moreover, the pathogen-free equilibrium is unstable if the basic reproductive ratio is greater than one.*

Proof. The Jacobian matrix of the proposed model in eq. (1)-(3) evaluated at the pathogen-free equilibrium E_1 is given by

$$J(E_1) = \begin{bmatrix} -\mu & 0 & -\beta \\ 0 & -(\mu+\delta) & \beta \\ 0 & \sigma & -\varepsilon \end{bmatrix}. \quad (9)$$

Eigenvalues of $J(E_1)$ are obtained from the following characteristic polynomial

$$(\lambda+\mu)(\lambda^2 + (\mu+\delta+\varepsilon)\lambda + (\mu+\delta)\varepsilon - \beta\sigma) = 0. \quad (10)$$

Hence eigenvalues of $J(E_1)$ are $\lambda_1 = -\mu$ and the zeros of the following quadratic polynomial

$$(\lambda^2 + (\mu+\delta+\varepsilon)\lambda + (\mu+\delta)\varepsilon - \beta\sigma) = 0.$$

By using the Routh-Hurwitz theorem, all eigenvalues of $J(E_1)$ are negative or complex eigenvalues with negative real parts if and only if $R = \frac{\beta\sigma}{(\mu+\delta)\varepsilon} < 1$. Therefore, the pathogen-free equilibrium is locally asymptotically stable if the basic reproductive ratio is less than one.

If the basic reproductive ratio is greater than one, then the characteristic polynomial in eq. (10) has one positive roots. Hence, the Jacobian matrix $J(E_1)$ has one positive eigenvalue. Consequently, the pathogen-free equilibrium is unstable. This completes the proof. ■

Global stability of the pathogen-free equilibrium is presented in Theorem 2.

Theorem 2. *If the basic reproductive ratio is equal or less than one, then the pathogen-free equilibrium E_1 is globally asymptotically stable.*

Proof. We define a Lyapunov function

$U: \{(S,I,P) \in \Omega : S > 0\} \to R$ where $U(S,I,P) = I + \frac{\beta}{\varepsilon}P$.

U is a nonnegative function on the domain Ω. Moreover, U attains minimum value when $I = P = 0$. The time derivative of U evaluated at the solution of mathematical model in eq. (1)-(3) is given by

$$\frac{dU}{dt} = \frac{dI}{dt} + \frac{\beta}{\varepsilon}\frac{dP}{dt} = -\frac{\beta IP}{S+I} - \left[(\mu+\delta) - \frac{\beta\sigma}{\varepsilon}\right]I = -\frac{\beta IP}{S+I} - (\mu+\delta)[1-R]I \leq 0.$$

In addition, we find $\frac{dU}{dt} = 0$ if and only if $I = 0$ or $P = 0$. Hence, $I(t) \to 0$ or $P(t) \to 0$ as $t \to \infty$. From eq. (3), $I(t) = 0$ causes $P(t) \to 0$ as $t \to \infty$. Then by using $P(t) = 0$ in eq. (1), we find $S(t) \to \frac{K}{\mu}$ as $t \to \infty$.

Moreover, by using $P(t) = 0$ in eq. (1), we find $S(t) \to \frac{K}{\mu}$ as $t \to \infty$. We also find $I(t) \to 0$ as $t \to \infty$ whenever $P(t) = 0$. As a result, by using LaSalle invariant principle, we find that every solution of the mathematical model in eq.(1)-(3) with initial value in Ω tends to the pathogen-free equilibrium as $t \to \infty$ [18] ■.

Stability of pathogen equilibrium is presented in Theorem 3.

Theorem 3. *The pathogen equilibrium E_2 is locally asymptotically stable whenever the basic reproductive ratio is greater than one.*

Proof. Let $J(E_2)$ be the Jacobian matrix of the proposed model in eq. (1)-(3) evaluated at the pathogen equilibrium E_2. Eigenvalues of $J(E_2)$ satisfies the following characteristic polynomial

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0,$$

where

$$a_1 = \frac{(R-1)\beta\sigma}{R\varepsilon} + 2\mu + \delta + \varepsilon = \frac{\beta\sigma}{\varepsilon} + \mu + \delta + \varepsilon,$$

$$a_2 = \frac{\beta\sigma\mu R(R-1) + \beta\sigma\delta(R-1)^2 + \beta\sigma\varepsilon(R-1)}{R^2\varepsilon} + \mu(\mu+\delta+\varepsilon),$$

$$a_3 = \frac{\beta\sigma\mu(R-1) + \beta\sigma\delta(R-1)^2}{R^2}.$$

It is clear that $a_1, a_2, a_3 > 0$ whenever $R > 1$. By direct calculation we find that

$$a_1a_2 - a_3 = \frac{\beta\sigma\mu(R-1)^2 + \beta\sigma\varepsilon(R-1)}{R^2} + \left(\frac{\beta\sigma}{\varepsilon} + \mu + \delta + \varepsilon\right)\mu(\mu + \delta + \varepsilon)$$
$$+ \left(\frac{\beta\sigma}{\varepsilon} + \mu + \delta\right)\left(\frac{\beta\sigma\mu R(R-1) + \beta\sigma\delta(R-1)^2 + \beta\sigma\varepsilon(R-1)}{R^2\varepsilon}\right) > 0.$$

By using the Routh-Hurwitz theorem, all eigenvalues of $J(E_2)$ are negative or complex eigenvalues with negative real parts if $R > 1$. As a result, the pathogen equilibrium is locally asymptotically stable if $R > 1$. This completes the proof. ■

4. Numerical Simulations

In this section, we present some numerical simulations to describe solution of the proposed model at pathogen-free condition and pathogen condition. Initial conditions of pathogen-free simulation are $S(0) = 200, I(0) = 400, P(0) = 400$. Some type of hybrid maize could be harvested after 97 days. Hence, we choose natural death parameter value $\mu = \frac{1}{97\ day} = 0.0103/\ day$. Here we simulate the proposed model from t = 0 until t = 200 days. Parameter values used in the simulation are shown in Table 2.

Table 2. Parameter values used in the simulation

Parameter	Value	Source
K	10 plants / day	Assumption
β	0.1 / (pathogen . day)	Assumption
μ	0.0103 / day	Assumption
δ	0.0206 / day	Assumption
σ	0.0143 / day	[15]
ε	0.1236 / day	[15]

From Table 2, we find the basic reproductive ratio R is R = 0.3744. In this condition, pathogen growth and the infection rate is lower than the pathogen removal rate (ε) and the infectious removal rate (μ+δ). This condition yields the pathogen-free condition. Consequently, infectious maize population and pathogen population tend to zero for enough long time. This situation is illustrated by the simulation result of pathogen-free condition (see the Figure 2).

We also perform numerical simulation for the pathogen condition. Parameter values for this simulation are shown in Table 2 except for parameter β. Here we choose β = 0.5. Hence we find the basic reproductive ration R is $R = 1.8721$. In this condition, pathogen growth and the infection rate is greater than the pathogen removal rate (ε) and the infectious removal rate (μ+δ). This condition yields the persistence of pathogen condition. Consequently, infectious maize population and pathogen population are always persist in the system. In addition, infectious maize population and pathogen population tend to the pathogen equilibrium values for enough long time. This situation is illustrated by the simulation result of pathogen condition (see the Figure 3).

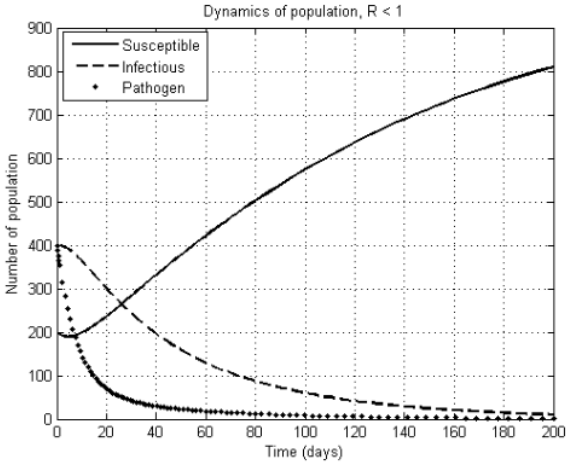

Figure 2. Dynamics of maize disease spread mathematical model for pathogen-free condition

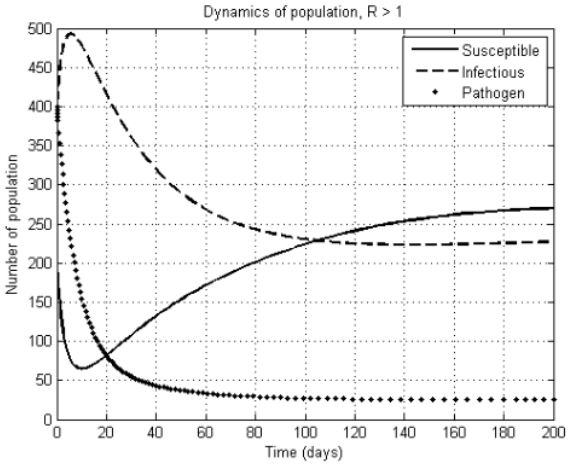

Figure 3. Dynamics of maize disease spread mathematical model for pathogen condition

5. Conclusion

In this paper, we have discussed a mathematical model of maize foliar disease spread with standard incidence rate. The proposed model has two equilibria, namely pathogen-free equilibrium and pathogen equilibrium. We found that property of the proposed model was completely determined by basic reproductive ration of the model.

Acknowledgment

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PAGE 5

PAGE 6

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