Stability analysis for yellow virus disease mathematical model of red chili plants

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Abstract. One of the main obstacles in the cultivation of red chili plants (*Capsicum annuum*) is the yellow virus disease caused by the Gemini virus. The virus is transmitted through insect vectors which can be controlled using insect pathogenic fungi (*Verticillium lecanii*). To understand the dynamics of the spread of the yellow virus in *Capsicum annuum* (*C. annuum*), we have formulated and analyzed a mathematical model of plant disease. From this model, the value of the Basic Reproduction Number (*ℜ₀*) is presented, which is an essential quantity in an epidemiological model. The obtaining results indicate that when the value of *ℜ₀* is greater than one, the endemic equilibrium point is asymptotically stable. However, if the value of *ℜ₀* is smaller than one, then the endemic equilibrium point is unstable. Also, we provide numerical simulations to illustrate the results.

1. Introduction

*Capsicum annuum* (red chili) is a horticultural product that has an important economic value in Indonesia. Apart from being used as a vegetable or cooking spice, *C. annuum* can also increase farmers' income because it is one of the highest-priced horticultural products [1-4]. Along with the increasing population and the development of industries that require *C. annuum* raw materials, the need for *C. annuum* in Indonesia from year to year continues to increase. This is due to problems with pests and plant diseases that cause low productivity [5-7].

One of the problems in the cultivation of *C. annuum* is exposure to the Gemini virus, which is a yellow virus that can attack *C. annuum* that are transmitted through insect vectors. He can catch the virus when he sucks on an infected *C. annuum*, then the virus spreads into his body so that when he sucks food from a susceptible plant, the virus enters the body of the *C. annuum*. This viral infection can cause symptoms such as yellow, stunted, and rolled (cupping) leaves [2, 3, 8, 9, 10]. This problem can be controlled using insect pathogenic fungi (*Verticillium lecanii*). The *V. lecanii* is an effective entomopathogen fungal biological agent for controlling *B. tabaci*. Recently the use of *V. lecanii* has been widely developed to reduce the use of pesticides [11-14]. Application of *V. lecanii* on *C. annuum* is done by spraying suspensions on the lower surface of the leaves [15]. But if too much use of *V. lecanii*, it can inhibit the growth of the chili plant. To see the effectiveness of using *V. lecanii*, it requires other knowledge to get a more thorough analysis. One of them is using a mathematical model [16].

Many researchers have developed mathematical models for plant diseases, including [17-19] which consider the transmission of the disease as vector-host interaction. The combination of the prey-predator
mathematical model with the host-vector theory was analyzed by Ruiqing [20], whereas a host-vector epidemic model with non-linear and bilinear events was analyzed by Seema [21]. Anggriani formulated a mathematical model of plant disease by considering protective and curative factors [22], analyzed fungicide applications as protection and curative [23], and analyzed the effect of insecticide use on Tungro disease vectors [24]. Wang has analyzed the dynamics of vector-borne disease models with the age of infection and prevalence rates globally [25]. Meanwhile, Anggriani discussed the optimal control of fungicide effects [26-27]. In this study we will discuss and analyze the dynamic model of yellow virus in *C. annuum* by considering insect vector.

2. Model Construction
To formulate a mathematical model, assumptions are used, such as *A* and *BN*, respectively, are designated as notations for insect vector populations and *C. annuum*. The population of *C. annuum* which is divided into four compartments, namely susceptible vegetative *C. annuum* (*S_v*(t)), infected vegetative *C. annuum* (*I_v*(t)), susceptible generative *C. annuum* (*S_g*(t)), and Infected generative *C. annuum* (*I_g*(t)), while insect vectors are divided into two compartments, susceptible insects (*S_BT*(t)) and infected insects (*I_BT*(t)).

*C. annuum* that are susceptible to vegetative and generative periods can be infected if the infected insect vector sucks the susceptible *C. annuum*, respectively, with an infection rate of *β_1* and *β_2*. Meanwhile, susceptible *C. annuum* in vegetative periods that are not sucked by the infected insect vector will grow into susceptible during the generative period with growth rates denoted *α*. In addition, all population of *C. annuum* are given *V. lecanii* which is denoted by *δ_p*, *C. annuum* can die naturally at the rate of *μ_p*, and infected *C. annuum* could not recover. Whereas susceptible insect vector populations can be infected if they suck infected *C. annuum* during the vegetative or generative period, with the infection rates denoted by *γ_1* and *γ_2*, respectively, and these vectors can die naturally or as a result of applying *V. lecanii*, with the speed of death denoted by *μ_l* and *θ_l* [28]. This problem described as in Figure 1.

![Transmission diagram](image)

*Figure 1. Transmission diagram*
The model that can describe the above transmission diagram is:

\[
\begin{align*}
\frac{dS}{dt} &= A - \alpha S - \beta_1(1 - \delta_p)S I_{BT} - \mu_p S, \\
\frac{dI}{dt} &= \beta_1(1 - \delta_p)S I_{BT} - \mu_p I, \\
\frac{dS}{dt} &= \alpha S - \beta_2(1 - \delta_p)S I_{BT} - \mu_p S, \\
\frac{dI}{dt} &= \beta_2(1 - \delta_p)S I_{BT} - \mu_p I, \\
\frac{dS_{BT}}{dt} &= BN - \gamma_1(1 - \delta_p)I S_{BT} - \gamma_2(1 - \delta_p)S_{BT} - \theta I_{BT} S_N - \mu S_{BT}, \\
\frac{dI_{BT}}{dt} &= \gamma_1(1 - \delta_p)I S_{BT} + \gamma_2(1 - \delta_p)S_{BT} - \theta I_{BT} S_N - \mu I_{BT},
\end{align*}
\]

where \( N_p = S_v + I_v + S_g + I_g \) and \( N_v = S_{BT} + I_{BT} \).

3. Model Analysis

3.1. Disease free-equilibrium point

The disease-free equilibrium point of model (1) is obtained by setting all compartments equal to zero. The disease-free equilibrium point is given by:

\[
E_0 = (S^*, I^*, S^*_s, I^*_s, S^*_{BT}, I^*_{BT}) = \left( \frac{A}{\mu + \alpha}, 0, \frac{A\alpha}{\mu (\mu + \alpha)}, 0, \frac{BN}{\theta \delta_p N_r + \mu}, 0 \right).
\]

3.2. Basic Reproduction Number (\( \mathcal{R}_0 \))

We use the next generation method to determine the basic reproduction number (\( \mathcal{R}_0 \)) of the Castillo-Chavez model [27]. Using the notation \( f \) for a matrix of new infections and \( v \) for the matrix transfer, we obtain:

\[
f = \begin{bmatrix} \beta_1(1 - \delta_p)S_v I_{BT} \\ \beta_2(1 - \delta_p)S_g I_{BT} \\ \gamma_1(1 - \delta_p)I_v S_{BT} + \gamma_2(1 - \delta_p)S_{BT} - \theta I_{BT} S_N - \mu I_{BT} \end{bmatrix} \quad \text{and} \quad v = \begin{bmatrix} \mu_p I_v \\ \mu_p I_g \\ \theta I_{BT} S_N + \mu I_{BT} \end{bmatrix}.
\]

The eigen values of the matrix \( (FV^{-1}) \) are:

\[
q_1 = 0 \quad \text{and} \quad q_{2,3} = \pm (1 - \delta_p) \frac{\mathcal{R}_0 (A - \mu p)(\alpha + \theta)(\mu p + \alpha)}{\mu_p (\theta \delta_p N_r + \mu) + \alpha (A \delta_p \theta + \mu \theta)},
\]

where \( F \) and \( V \) are the Jacobian matrices of \( f \) and \( v \) which are evaluated at the disease-free equilibrium point. The basic reproductive number \( \mathcal{R}_0 \) is obtained from spectral radius \( \xi \) (dominant eigen value) of the \( (FV^{-1}) \) matrix denoted by \( \xi \), so that it is equation (3).

\[
\mathcal{R}_0 = \xi (FV^{-1}) = \pm (1 - \delta_p) \frac{\mathcal{R}_0 (A - \mu p)(\alpha + \theta)(\mu p + \alpha)}{\mu_p (\theta \delta_p N_r + \mu) + \alpha (A \delta_p \theta + \mu \theta)}.
\]

3.3. Stability Analysis (Disease-free)

Theorem 1

The disease-free equilibrium of the system (1) is locally asymptotically stable if \( \mathcal{R}_0 < 1 \).

Proof

By following Diekmann [28] substitute (2) into the Jacobian matrix for non-endemic equilibrium points, so that it is obtained:
The positive endemic equilibrium of the system (1) exists whenever \( R_0 > 1 \).

**Proof**

The system (1) have the unique endemic equilibrium \( E_0^* = (S^*_0, I^*_0, I^*_g, S^*_{BT}, I^*_{BT}) \), where:

\[
S^*_0 = \frac{A}{(\mu_p + \alpha)(\delta_p + \delta_{12} N_p + \mu_i)}; \quad I^*_0 = \frac{\beta(1-\delta_i) S^*_0}{\mu_p}; \quad S^* = \frac{\alpha S^*}{\beta(1-\delta_i) I^*_0 + \mu_p}; \quad I^* = \frac{\beta(1-\delta_i) I^*_0}{\mu_p};
\]

\[
S^*_{BT} = \frac{BN}{\gamma(1-\delta_i) 1^*_0 + \gamma(1-\delta_i) 1^*_g + \theta \delta_p N_p + \mu_i}; \quad \text{and } I^*_{BT}, \text{which unique positive solution of the following equation is given as: } f \left( I^*_{BT} \right) = a \left( I^*_{BT} \right)^2 + b \left( I^*_{BT} \right) + c, \text{ where:}
\]
\[ a = \beta_p \beta_i (1 - \delta) \left( \theta \delta N_s + \mu_s \right) \left( A \gamma_i (1 - \delta) + \left( \theta \delta N_s + \mu_s \right) \mu_s \right) > 0, \]

\[ b = (1 - \delta) \left[ \left( \theta \delta N_s + \mu_s \right) \mu_s \left( \mu_v + \beta_i + \alpha \beta_i \right) + A (1 - \delta) \left( \mu_v \beta_i + \mu_i \beta_i \gamma_s \right) \left( \theta \delta N_s + \mu_s \right) + B N v \beta_i \right] \left( 1 - \delta \right) > 0, \]

and \[ c = \left( 1 - \mathcal{R}_0^2 \right) < 0. \]

The endemic equilibrium point in the system (1) will exist if \( \mathcal{R}_0 > 1 \), but if \( \mathcal{R}_0 < 1 \) then the endemic equilibrium point will not exist. This is completed this proof.

3.5. Endemic-equilibrium point

**Theorem 3**

The endemic equilibrium-point of system (1) is locally asymptotically stable if \( \mathcal{R}_0 > 1 \).

**Proof**

Since the endemic stability proving in the system (1) is quite complex in analysis, it is possible to prove this theorem through numerical simulations (see Figure 4 and Figure 5), from the results of numerical simulations it is proven that the endemic equilibrium system is local asymptotic if \( \mathcal{R}_0 > 1 \). This completes the proof.

4. Numerical Simulation

To illustrate the dynamics of the spread of yellow virus in each compartment, we use the parameter values and the initial conditions for each of the respective compartments shown in Table 1.

**Table. 1** The value of variables and parameters

| Variable/Parameter | Value (\( \mathcal{R}_0 < 1 \)) | Value (\( \mathcal{R}_0 > 1 \)) | Variable/Parameter | Value (\( \mathcal{R}_0 < 1 \)) | Value (\( \mathcal{R}_0 > 1 \)) |
|--------------------|---------------------------------|---------------------------------|--------------------|---------------------------------|---------------------------------|
| \( N_p \)         | 800                             | 800                             | \( \alpha \)       | 0,7                             | 0,7                             |
| \( N_v \)         | 400                             | 400                             | \( \beta_1 \)      | 0,01                            | 0,01                            |
| \( S_v \)         | 500                             | 500                             | \( \beta_2 \)      | 0,01                            | 0,01                            |
| \( I_v \)         | 150                             | 150                             | \( \gamma_1 \)     | 0,25                            | 0,25                            |
| \( S_g \)         | 350                             | 350                             | \( \gamma_2 \)     | 0,3                             | 0,3                             |
| \( I_g \)         | 100                             | 100                             | \( \delta_p \)     | 0,7                             | 0,3                             |
| \( S_B \)         | 300                             | 300                             | \( \mu_p \)        | 0,3                             | 0,3                             |
| \( I_B \)         | 100                             | 100                             | \( \mu_i \)        | 0,7                             | 0,7                             |
| \( A \)           | 100                             | 100                             | \( \theta_i \)     | 0,5                             | 0,5                             |
| \( B \)           | 100                             | 100                             |                     |                                 |                                 |

The dynamics of the spread of yellow virus in \( C.\ annuum \) is shown in Figure 2 until Figure 5 using the Maple 18 software with initial values and parameter values in Table 1.

From Figure 2, it can be seen that the population of infected \( C.\ annuum \) in the vegetative period and in the generative period will become extinct when \( \mathcal{R}_0 < 1 \). Whereas in Figure 3, it can be seen that the population of insect vector which is susceptible is reduced and insect vector that is infected will become extinct when \( \mathcal{R}_0 < 1 \). From Figure 4 and Figure 5, it can be seen that the population of susceptible \( C.\ annuum \) is decline and the infected \( C.\ annuum \) are increasing, and that the infected insect vector does not extinct when \( \mathcal{R}_0 > 1 \). The effectiveness of using \( V.\ lecanii \) for all populations in infected compartments can be seen in Figure 6 until Figure 8. This illustration used Maple 18 software with initial values and parameter values in Table 1.
Figure 2. The dynamical of population *C. annuum* when $R_0 < 1$

Figure 3. The dynamical of population insect vector when $R_0 < 1$

Figure 4. The dynamical of population *C. annuum* when $R_0 > 1$

Figure 5. The dynamical of population insect vector when $R_0 > 1$

Figure 6. Sensitivity analysis for infected *C. annuum* in vegetative periods

Figure 7. Sensitivity analysis for infected *C. annuum* in generative periods

Figure 8. Sensitivity analysis for infected insect vector
From Figures 6 until Figure 8, it can be seen that the *C. annuum* that are infected in the vegetative and generative periods, and the infected insect vector will become extinct after *V. lecanii* is applied at speeds greater than or equal to 0.5. However, it can be seen, if the rate of *V. lecanii* is 0.5, it requires a longer time than the *V. lecanii* rate of 0.7.

5. Conclusion
The yellow virus spread model in *C. annuum* using *V. lecanii* as control of disease transmission has two equilibrium points, namely non-endemic and endemic points. The non-endemic point will be asymptotically stable if $r_0 < 1$ and will have a unique endemic equilibrium point if $r_0 > 1$. The simulation results show that the application of *V. lecanii* is effective in reducing yellow virus infection in *C. annuum*.

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