

# Optimal Control Model of *Verticillium lecanii* Application in the Spread of Yellow Red Chili Virus

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**Abstract:** - In this study, we developed a model of yellow viral disease of red chili plants that are spread through whitefly bugs (*Bemisia tabaci*). In addition, we used optimal control theory with Pontryagin's minimum principle to determine the optimal control of *Verticillium lecanii* (*V. lecanii*) applications so as to minimize the costs incurred in reducing the intensity of the spread of yellow viral diseases. The results showed that *V. lecanii* was sufficiently applied for 15 days with the application of 90% of the prescribed dose to minimize the costs incurred by farmers in the cultivation of red chili plants.

**Key-Words:** - Optimal control, Pontryagin's Minimum Principle, Mathematical Modeling, *V. lecanii*, Yellow Virus, Red Chili, Plant Disease.

## 1 Introduction

*Capsicum annuum* (*C. annuum*) is the second most important horticultural plant after tomatoes, grown in tropical regions such as Indonesia. Besides having important economic values, *C. annuum* also has spicy taste and high nutritional value [1-10]. The high vitamin C, provitamin A, and calcium contained in *C. annuum* cause many people to consume these vegetable fruits [4, 5, 6, 10]. Furthermore, *C. annuum* has pharmacological functions as antineoplastic, antidiabetic, antifungal, antiviral, antibacterial, antioxidant, antiangiogenic, analgesic, vasodilation, gastroprotective activity, and anti-obesity [3, 4, 6].

The high benefits and needs of *C. annuum* require farmers to cultivate red chili plants. Farmers often experience heavy losses due to various obstacles when cultivating red chili plants, such as plant disease infection.

One of the problems of red chili plant disease often encountered by farmers is the emergence of a yellow virus caused by the Gemini virus. Symptoms caused by the Gemini virus vary depending on the genus and species of the infected plant. Symptoms of yellow virus in red chili plant first appear on young leaves or shoots in the form of yellow spots around the leaf bone, then develop into yellow leaf bones. Symptoms continue until almost all young leaves or shoots are bright yellow, and some yellow mixed with green leaves, concave and narrow, smaller and thicker [11-16].

Gemini virus can be transmitted through insects, namely *Bemisia tabaci* (*B. tabaci*) [12, 13, 14, 16, 17, 18]. The insect gets a virus when taking food from infected red chili plants, then spreads it in its body fluids, so that when sucking food from healthy plants, the virus enters the body of the red chili plant [17].

To control this problem, entomopathogenic fungi (*V. lecanii*) can be used as population control of *B. tabaci* [18-21]. However, if too many *V. lecanii* are applied, it will generate a large amount of costs. Therefore, to minimize the costs incurred in controlling *B. tabaci* population to reduce the intensity of yellow virus disease in red chili plants, another knowledge is needed to analyze it. One of which is to use mathematical modeling which then looks for optimal model control.

Many researchers conducted research on mathematical models of plant diseases, including modeling for vector-borne diseases with direct transmission carried out [22] and then developed with regard to a one-time delay [23]. On the other hand, [24] created and analyzed epidemic models of host vector plants with monotonous and bilinear cases. Then [25] creates and analyzed mathematical models of dispersion diseases transmitted by insects by observing climate change, as well as conducting numerical simulations to understand the behavior of mathematical models from previous research [26].

Mathematical modeling of plant diseases transmitted by vectors by linking predator-prey models to host pathogens is discussed by [27],

transient [28] combining predator-prey models with host vectors to be tested for influence indirectly from predators against host vector dynamics. Modeling of plant diseases caused by viruses has been discussed by [29-30], who consider that the development of epidemics can be limited by the limitations of the virus in the early stages of the epidemic, namely when plants are susceptible to health [29]. Then, they developed model for virus transmission and the dynamics of disease spread from interactions between plant-virus-vector-parasitoid [30].

Meanwhile, [31] analyzed mathematical models of interactions between host plants, soil-borne pathogens, and microbial antagonists in controlling the virus. Then, minimum hybrid models combined from herbivore-plants with susceptible-infected models are discussed by [32] and models of vector-borne-transmitted plant diseases have been discussed by [33] using a fractional derivative method.

In addition, the theory and analysis of plant pathology as well as some material about plant disease epidemics can be found in [34-35], whereas mathematical modeling involving protection and care has also been carried out, including [36], by conducting mathematical modeling to determine the effectiveness of fungicides in influencing invasion dynamics and resistance of plant pathogens. Meanwhile, [37] have constructed an application model of fungicide as a protection and curative vector. Then, they have created a mathematical model for plant disease by performing maintenance of rouging, replanting, and preventive, and discussed the dynamics of the spread of disease with curative and preventive methods [38] in a two-stage disease model [39].

Furthermore, [40] analyzed the effects of insecticides for infected plants and [41] determined optimal control of mathematical models of plant diseases to see the effectiveness of fungicide applications.

After studying the ideas of previous researchers, we discussed the optimal control of the use of *V. lecanii* to see its effectiveness in reducing the intensity of yellow viral diseases so as to minimize the costs incurred by farmers in chili cultivation.

## 2 Dynamic Model

In constructing the model of the spread of yellow virus from this study, there are several assumptions used, including:

- 1) The population of red chili plants is constant.

- 2) The population of red chili plants is divided into four classes, namely red chili plants that are susceptible to the vegetative phase ( $S_v$ ), infected red chili plants in the vegetative phase ( $I_v$ ), red chili plants that are susceptible to the generative phase ( $S_g$ ), and infected red chili plants in the generative phase ( $I_g$ ).
- 3) The insect population is divided into two classes, namely susceptible insects ( $S_{BT}$ ) and infected insects ( $I_{BT}$ ).
- 4) Plants that are susceptible to both vegetative and generative phases can be infected if they interact directly with infected insects.
- 5) Insects that are susceptible can be infected if they interact directly with infected plants both in the vegetative and generative phase.
- 6) Infected plants and insects cannot recover.
- 7) All plant populations were given *V. lecanii*.

The variables and parameters used can be seen in Table 1.

Table 1. The definition of variables and parameters

Variable/ Parameter	Definition
$N_p$	Population of chili plants
$N_v$	Population of <i>B. tabaci</i>
$S_v$	Susceptible of chili plants in the vegetative phase
$I_v$	Infected chili plants in the vegetative phase
$S_g$	Susceptible chili plants in the generative phase
$I_g$	Infected chili plants in the generative phase
$S_{BT}$	Susceptible <i>B. tabaci</i>
$I_{BT}$	Infected <i>B. tabaci</i>
$A$	Recruitment of chili plants
$B$	Recruitment of <i>B. tabaci</i>
$\alpha$	Rate of growth from vegetative to generative phase
$\beta_1$	Rate of infected chili plants in the vegetative phase
$\beta_2$	Rate of infected chili plants in the generative phase
$\gamma_1$	Rate of <i>B. tabaci</i> infection when taking infected plant food in the vegetative phase
$\gamma_2$	Rate of <i>B. tabaci</i> infection when taking infected plant food in the generative phase
$\delta_p$	<i>V. lecanii</i>
$\mu_p$	The death rate of chili plants
$\mu_t$	The natural death rate of <i>B. tabaci</i>

Variable/ Parameter	Definition
$\theta_i$	The death rate of <i>B. tabaci</i> due to curative intervention

The schematic diagram of the spread of yellow virus in chili plants involving *V. lecanii* can be described as shown in Fig. 1

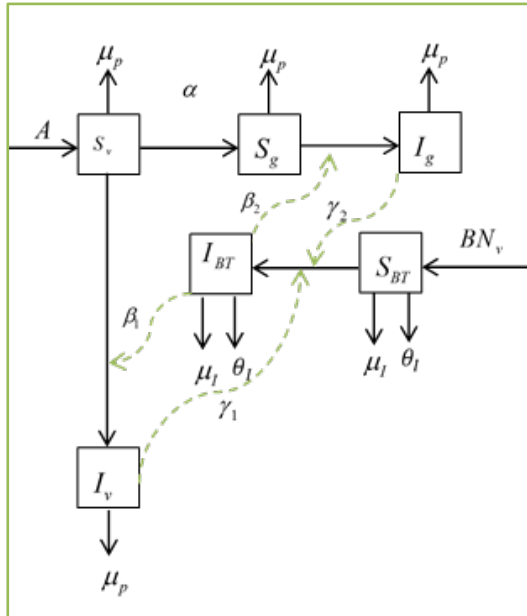


Fig.1: Schematic diagram of the spread of yellow virus in chili plants involving *V. lecanii*

The schematic diagram in Fig. 1 is obtained by the following model:

$$\begin{aligned}
 \frac{dS_v}{dt} &= A - \alpha S_v - \beta_1(1 - \delta_p)S_v I_{BT} - \mu_p S_v \\
 \frac{dI_v}{dt} &= \beta_1(1 - \delta_p)S_v I_{BT} - \mu_p I_v \\
 \frac{dS_g}{dt} &= \alpha S_v - \beta_2(1 - \delta_p)S_g I_{BT} - \mu_p S_g \\
 \frac{dI_g}{dt} &= \beta_2(1 - \delta_p)S_g I_{BT} - \mu_p I_g \\
 \frac{dS_{BT}}{dt} &= BN_v - \gamma_1(1 - \delta_p)I_v S_{BT} - \gamma_2(1 - \delta_p)I_g S_{BT} - \theta_i u_1 S_{BT} N_p - \mu_I S_{BT} \\
 \frac{dI_{BT}}{dt} &= \gamma_1(1 - \delta_p)I_v S_{BT} + \gamma_2(1 - \delta_p)I_g S_{BT} - \theta_i \delta_p I_{BT} N_p - \mu_I I_{BT}
 \end{aligned}
 \tag{1}$$

### 3 Optimal Control

The purpose of the dynamic red chili model is to minimize the population of plants infected during vegetative or generative period and insects infected by optimizing *V. lecanii*, which will be solved using the minimum principle of Pontryagin [42]. The objective functions used are as follows:

$$J(u) = \int_{t_0}^{t_1} (A_1 I_v(t) + A_2 I_g(t) + A_3 I_{BT} + A_4 u_1^2(t)) dt \tag{2}$$

The model can be solved using optimal control theory, where  $u_1$  is the rate of giving *V. lecanii* and  $A_i \geq 0$ , for  $i = 1, 2, \dots, 4$  is the cost coefficient, and  $t_f$  is the end time. Quadratic objective functions are used to measure control costs, which are assumed that in reality there is no linear relationship between the impact of intervention and the intervention costs of infected populations (inversion forms a non-linear function) [43].

With constrain:

$$\begin{aligned}
 \frac{dS_v}{dt} &= A - \alpha S_v - \beta_1(1 - u_1)S_v I_{BT} - \mu_p S_v \\
 \frac{dI_v}{dt} &= \beta_1(1 - u_1)S_v I_{BT} - \mu_p I_v \\
 \frac{dS_g}{dt} &= \alpha S_v - \beta_2(1 - u_1)S_g I_{BT} - \mu_p S_g \\
 \frac{dI_g}{dt} &= \beta_2(1 - u_1)S_g I_{BT} - \mu_p I_g \\
 \frac{dS_{BT}}{dt} &= BN_v - \gamma_1(1 - u_1)I_v S_{BT} - \gamma_2(1 - u_1)I_g S_{BT} - \theta_i u_1 S_{BT} N_p - \mu_I S_{BT} \\
 \frac{dI_{BT}}{dt} &= \gamma_1(1 - u_1)I_v S_{BT} + \gamma_2(1 - u_1)I_g S_{BT} - \theta_i u_1 I_{BT} N_p - \mu_I I_{BT}
 \end{aligned}
 \tag{3}$$

Boundary conditions:

$$\begin{aligned}
 t_0 < t < t_1, 0 \leq u_1(t) \leq 1, S_v(0) = S_{v_0} \geq 0, I_v(0) = I_{v_0} \geq 0, S_g(0) = S_{g_0} \geq 0, I_g(0) = I_{g_0} \geq 0, S_{BT}(0) = S_{BT_0} \geq 0, I_{BT}(0) = I_{BT_0} \geq 0.
 \end{aligned}$$

Next, the Hamiltonian function is determined:

$$\begin{aligned}
 H &= A_1 I_v(t) + A_2 I_g(t) + A_3 I_{BT} + A_4 u_1^2(t) + \\
 &\lambda_1(A - \alpha S_v - \beta_1(1 - u_1)S_v I_{BT} - \mu_p S_v) + \\
 &\lambda_2(\beta_1(1 - u_1)S_v I_{BT} - \mu_p I_v) + \lambda_3(\alpha S_v - \\
 &\beta_2(1 - u_1)S_g I_{BT} - \mu_p S_g) + \lambda_4(\beta_2(1 - u_1)S_g I_{BT} - \\
 &\mu_p I_g) + \lambda_5(BN_v - \gamma_1(1 - u_1)I_v S_{BT} - \gamma_2(1 - \\
 &u_1)I_g S_{BT} - \theta_i u_1 S_{BT} N_p - \mu_I S_{BT}) + \lambda_6(\gamma_1(1 - \\
 &u_1)I_v S_{BT} + \gamma_2(1 - u_1)I_g S_{BT} - \theta_i u_1 I_{BT} N_p - \\
 &\mu_I I_{BT})
 \end{aligned}
 \tag{4}$$

with  $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5$ , and  $\lambda_6$  is a co-state variable or often referred to as Lagrange multiplier.

According to the minimum principle of Pontryagin, the Hamiltonian function must satisfy

$$\dot{x}(t) = \begin{bmatrix} \dot{S}_v(t) \\ \dot{I}_v(t) \\ \dot{S}_g(t) \\ \dot{I}_g(t) \\ \dot{S}_{BT}(t) \\ \dot{I}_{BT}(t) \end{bmatrix}, \quad \hat{\lambda}(t) = \begin{bmatrix} \dot{\lambda}_1(t) \\ \dot{\lambda}_2(t) \\ \dot{\lambda}_3(t) \\ \dot{\lambda}_4(t) \\ \dot{\lambda}_5(t) \\ \dot{\lambda}_6(t) \end{bmatrix}, \quad \text{and stationary}$$

conditions.

State condition:

$$\begin{aligned}
 \frac{\partial S_v(t)}{\partial t} = \frac{\partial H}{\partial \lambda_1} &= (A - \alpha S_v - \beta_1(1 - u_1)S_v I_{BT} - \mu_p S_v) \\
 \frac{\partial I_v(t)}{\partial t} = \frac{\partial H}{\partial \lambda_2} &= (\beta_1(1 - u_1)S_v I_{BT} - \mu_p I_v)
 \end{aligned}$$

$$\begin{aligned} \frac{\partial S_g(t)}{\partial t} &= \frac{\partial H}{\partial \lambda_3} = (\alpha S_v - \beta_2(1 - u_1)S_g I_{BT} - \mu_p S_g) \\ \frac{\partial I_g(t)}{\partial t} &= \frac{\partial H}{\partial \lambda_4} = (\beta_2(1 - u_1)S_g I_{BT} - \mu_p I_g) \\ \frac{\partial S_{BT}(t)}{\partial t} &= \frac{\partial H}{\partial \lambda_5} = (BN_v - \gamma_1(1 - u_1)I_v S_{BT} - \gamma_2(1 - u_1)I_g S_{BT} - \theta_I u_1 S_{BT} N_p - \mu_I S_{BT}) \\ \frac{\partial I_{BT}(t)}{\partial t} &= \frac{\partial H}{\partial \lambda_6} = (\gamma_1(1 - u_1)I_v S_{BT} + \gamma_2(1 - u_1)I_g S_{BT} - \theta_I u_1 I_{BT} N_p - \mu_I I_{BT}) \end{aligned} \tag{5}$$

co-state condition:

$$\begin{aligned} \hat{\lambda}_1 &= -\frac{\partial H}{\partial S_v} = -\lambda_1(-\alpha - \beta_1(1 - u_1)I_{BT}) - \lambda_2 \beta_1(1 - u_1)I_{BT} - \lambda_3 \alpha + \lambda_5 \theta_I u_1 S_{BT} + \lambda_6 \theta_I u_1 I_{BT} \\ \hat{\lambda}_2 &= -\frac{\partial H}{\partial I_v} = -A_1 - \lambda_1 \mu_p + \lambda_2 \mu_p - \lambda_5(-\gamma_1(1 - u_1)S_{BT} - \theta_I u_1 S_{BT}) - \lambda_6(\gamma_1(1 - u_1)S_{BT} - \theta_I u_1 I_{BT}) \\ \hat{\lambda}_3 &= -\frac{\partial H}{\partial S_g} = -\lambda_1 \mu_p - \lambda_3(-\beta_2(1 - u_1)I_{BT} - \mu_p) - \lambda_4 \beta_2(1 - u_1)I_{BT} + \lambda_5 \theta_I u_1 S_{BT} + \lambda_6 \theta_I u_1 I_{BT} \\ \hat{\lambda}_4 &= -\frac{\partial H}{\partial I_g} = -A_2 I_g(t) - \lambda_1 \mu_p + \lambda_4 \mu_p - \lambda_5(BN_v - \gamma_2(1 - u_1)S_{BT} - \theta_I u_1 S_{BT}) - \lambda_6(\gamma_2(1 - u_1)S_{BT} - \theta_I u_1 I_{BT}) \\ \hat{\lambda}_5 &= -\frac{\partial H}{\partial S_{BT}} = -\lambda_5(B - \gamma_1(1 - u_1)I_v - \gamma_2(1 - u_1)I_g - \theta_I u_1 N_p - \mu_I) - \lambda_6(\gamma_1(1 - u_1)I_v + \gamma_2(1 - u_1)I_g) \\ \hat{\lambda}_6 &= -\frac{\partial H}{\partial I_{BT}} = -A_3 - \lambda_1(-\beta_1(1 - u_1)S_v) - \lambda_2(\beta_1(1 - u_1)S_v) - \lambda_3(\beta_2(1 - u_1)S_g) - \lambda_4(\beta_2(1 - u_1)S_g) - \lambda_6(-\theta_I u_1 N_p - \mu_I) \end{aligned}$$

Stationary condition:

$$u_1 = \frac{1}{2A_4} (\lambda_6 I_{BT} N_p \theta_I - \lambda_1 I_{BT} S_v \beta_1 + \lambda_2 I_{BT} S_v \beta_1 - \lambda_3 I_{BT} S_g \beta_2 + \lambda_4 I_{BT} S_g \beta_2 - \lambda_5 I_g S_{BT} \gamma_2 + \lambda_6 I_g S_{BT} \gamma_2 - \lambda_5 I_v S_{BT} \gamma_1 + \lambda_6 S_{BT} I_v \gamma_1 + \lambda_5 N_p S_{BT} \theta_I)$$

Since  $0 \leq u_1 \leq 1$ , the optimal control is thus:

$$u_1 = \max \left\{ \min \left[ \frac{1}{2A_4} (\lambda_6 I_{BT} N_p \theta_I - \lambda_1 I_{BT} S_v \beta_1 + \lambda_2 I_{BT} S_v \beta_1 - \lambda_3 I_{BT} S_g \beta_2 + \lambda_4 I_{BT} S_g \beta_2 - \lambda_5 I_g S_{BT} \gamma_2 + \lambda_6 I_g S_{BT} \gamma_2 - \lambda_5 I_v S_{BT} \gamma_1 + \lambda_6 S_{BT} I_v \gamma_1 + \lambda_5 N_p S_{BT} \theta_I), 1 \right], 0 \right\}$$

### 4 Numerical Simulation

To provide illustrative examples of red chili plants and *B. tabaci* populations with *V. lecanii* control or without *V. lecanii* control, we use the

assumed parameter values and initial values as shown in the Table. 2.

Table 2. The initial and parameters value

Variable/Parameter	Value	Variable/Parameter	Value
$N_p$	80	$\alpha$	0.07
$N_v$	40	$\beta_1$	0.001
$S_v$	50	$\beta_2$	0.001
$I_v$	10	$\gamma_1$	0.025
$S_g$	30	$\gamma_2$	0.02
$I_g$	10	$\delta_p$	0.2
$S_{BT}$	30	$\mu_p$	0.03
$I_{BT}$	10	$\mu_I$	0.07
$A$	10	$\theta_I$	0.05
$B$	10		

By using parameter and initial values in Table. 2, then population dynamics using *V. lecanii* controls or those without using *V. lecanii* controls can be described as shown in Fig. 2 to Fig. 7.

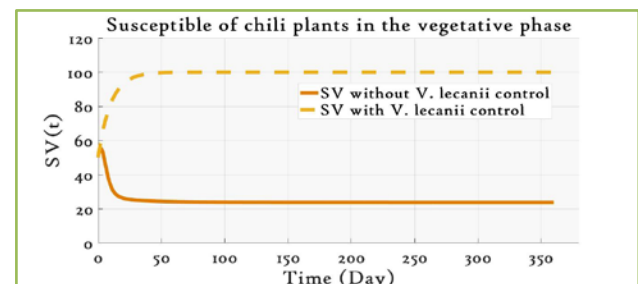


Fig.2: Population of susceptible red chili plants in the vegetative phase

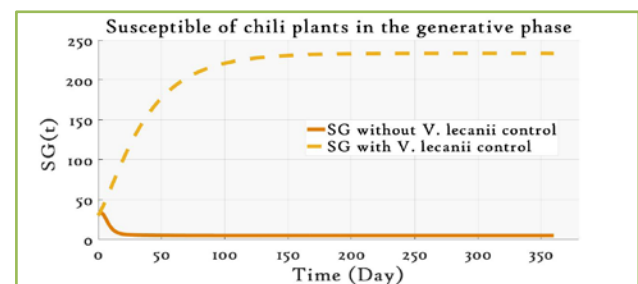


Fig.3: Population of susceptible red chili plants in the generative phase

From Fig. 2 and Fig. 3 shows that if the red chili plants without *V. lecanii* will decreases but if the red chili plants with *V. lecanii* the population of red chili plants will increases. This happens because when a red chili plant with *V. lecanii*, infected *B. tabaci* cannot transmit the virus to susceptible plants (in the vegetative and generative phase), so the plant population increases. But when red chili plants without *V. lecanii*, infected *B. tabaci* can transmit the virus to susceptible plants, so that susceptible

plants (in the vegetative and generative phase) will be reduced, because these plants become infected plants.

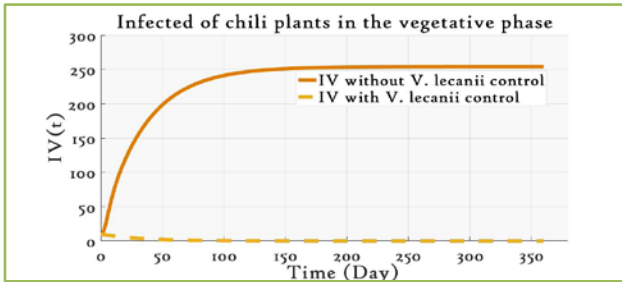


Fig.4: Population of infected red chili plants in the vegetative phase

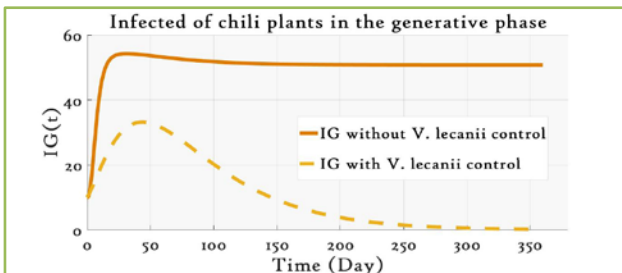


Fig.5: Population of infected red chili plants in the generative phase

Conversely, for infected red chili plant populations (in the vegetative and generative phases) after day 50, the population of red chili plants with *V. lecanii* will decrease, whereas if the population of red chili plants without *V. lecanii* the population of infected red chili plant will increase. This happens because when given *V. lecanii* susceptible plants cannot be infected by infected *B. tabaci*. (see Fig.4 and Fig.5).

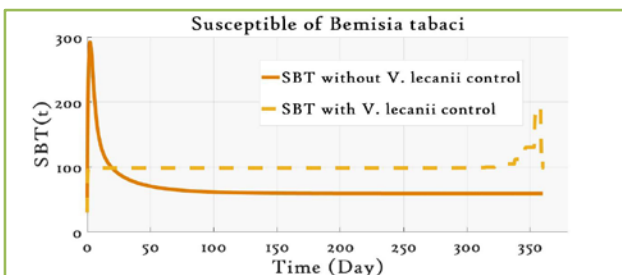


Fig.6: Population of susceptible *B. tabaci*

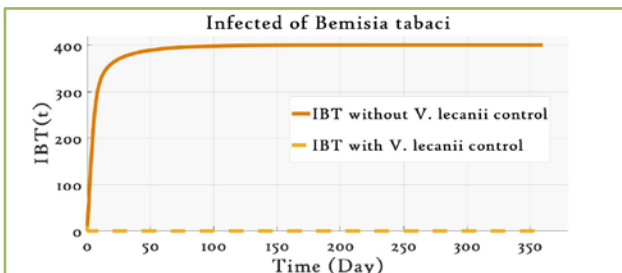


Fig.7: Population of infected *B. tabaci*

Fig. 6 shows that the susceptible *B. tabaci* population experienced a higher increase when the red chili plants with *V. lecanii* compared to if the red chili plants without *V. lecanii*. This is because the susceptible *B. tabaci* cannot be infected when taking food from infected chili plants (interacting directly with infected chili plants), but when the red chili plants without *V. lecanii*, *B. tabaci* can be infected because it takes food from the chili plants infected red (interacts directly with infected red chili plants). In contrast, the infected population of *B. tabaci* continues to increase if the population of red chili plants without *V. lecanii*. But if the red chili with *V. lecanii*, the *B. tabaci* population is almost extinct (see Fig. 7).

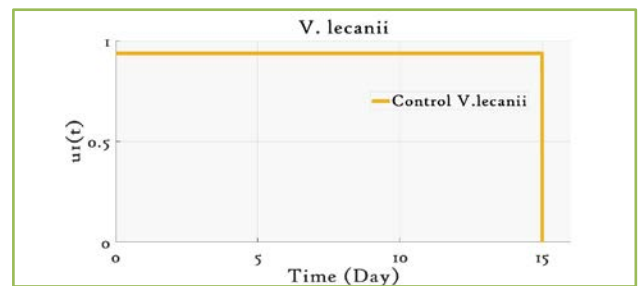


Fig.8: Control Optimal

Fig. 8 shows the level of application of *V. lecanii* as a *B. tabaci* controller to reduce the intensity of the spread of yellow viruses with minimum costs incurred. On Fig. 8, it can be seen that Application of *V. lecanii* is sufficient to be carried out for 15 days with the application of 90% of the dose determined for the costs incurred by farmers in the cultivation of red chili plants.

### 5 Conclusion

In this paper, we have made a model for the spread of yellow viruses in red chili plants by applying *V. lecanii*. Then, we have determined optimal control of the use of *V. lecanii* and provided numerical simulations as an example illustration of populations of red chili plants and *B. tabaci*. Numerical simulation results show that the population of infected plants (in the vegetative and generative phases) decreases due to the red chili plants with *V. lecanii*, which results in an endangered population of *B. tabaci*. But when the red chili plants without *V. lecanii*, the population of infected plants (in the vegetative and generative phases) increases. As a result, the population of susceptible red chili plants (in the vegetative and generative phases) will continue to increase if the red chili plants with *V. lecanii*. In addition, the

simulation results provided also showed that the level of application of *V. lecanii* as a *B. tabaci* controller can reduce the intensity of the spread of the yellow virus. Application of *V. lecanii* is sufficient to be carried out for 15 days with the application of 90% of the prescribed dose to minimize the costs incurred by farmers in the cultivation of red chili plants.

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