Mathematical Modeling for Controlling Brown Planthopper using Integrated Pest Management

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Abstract: One insect pest that causes enormous damage to rice plants in many countires is the brown planthopper (BPH). Various strategies have been proposed to control BPH outbreaks, such as cultural control, physical control, chemical control and biological control. Integrated pest management (IPM) is a method of controlling pests using a variety of methods to achieve effective and environmentally friendly results. However, the best methods in IPM application are yet to be determined. Since laboratory experiments have limitations in many respects, such as ethics, time, environment, and cost, mathematical modeling is considered as an option which can be used to study the effects of IPM on BPH infestation. In this article, we investigate mathematical models describing management of BPH under IPM strategies. In particular, we study two biological control agents including *Cyrtorhinus lividipennis*, a natural enemy of BPH, and *Metarhizium neoanisopliae*, an entomopathogenic fungus that causes diseases in pests. A data set from laboratory experiments previously published is also used in this study. In addition, we study functional responses describing predator's prey consumption. Finally, mathematical features of the proposed models are analyzed. The existence of equilibrium points, their stability and Hopf bifurcation are proved and numerical results are obtained to illustrate the theoretical results. This study suggests that using IPM is an effective method for rapidly reducing insect populations in the early stages of BPH outbreaks which might contribute to the long-term suppression of BPH.

Key-Words: - Mathematical model, Hopf bifurcation, Brown planthopper, Integrated pest management, Biological control, *Cyrtorhinus lividipennis*, *Metarhizium neoanisopliae*

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1 Introduction

Brown planthopper (BPH), *Nilaparvata lugens*, is a pest that causes damage to rice in many countries, especially Asia, where rice is a staple food, [1]. Hence, various methods of BPH management have been proposed to reduce the damage to rice plants, such as chemical techniques, [2], [3], [4], [5], biological techniques, [6], [7], [8], using resistant rice varieties, [9], and integrated pest management (IPM) principles, [10], [11]. The use of pesticides can cause damage to the environment and increase the insecticide resistance of pests. As an example of chemical pesticides, the effects of nitenpyram were studied on the developmental, reproduction, and survival rates of BPH, and the results of the study showed that insecticide resistance to nitenpyram may cause significant spending on resistant BPH, [3]. Likewise, Wen et al. discussed the effects of triflumezopyrim on BPH and the results showed that the application of triflumezopyrim had a significant effect on the development of BPH, [4]. Additionally, Kang et al. investigated the feeding behavior and life table of *Nilaparvata lugens* and *Sogatella furcifera* by using the control of imidacloprid and sulfoxaflor, [5]. For environmental reasons and studies of pesticide resistance, some research has been conducted on the use of biological pesticides such as using the plant essential oil, [6], natural enemies of BPH, [7], [12], entomopathogenic fungi, [8], and resistant rice varieties, [9]. However, using natural methods such as biological control and resistant rice varieties may not be enough to stop an outbreak of BPH on plants. Therefore, integrated pest management (IPM) has been proposed to determine the suitability of pest control with a combination of various methods to achieve cost-effectiveness. IPM strategies for pest control were studied to illustrate the effect of human management on insects using a variety of methods and to demonstrate the cost-effectiveness of appropriate controls, [10], [11].

There are many natural enemies of BPH, such as: *Crytorrhinus lividipennis*, [7], [12], *Tytthus chinensis*, [12], and *Pardosa pseudoannulata*, [13]. However, the most popular predator is *Crytorrhinus lividipennis* because it is effective in getting rid of BPHs. *Crytorrhinus lividipennis* is an essential insect that plays an important role in reducing planthopper and leafhopper outbreaks. It is a natural predator of the BPH as it attacks BPH eggs in rice fields. *Crytorrhinus lividipennis* can commonly be found in tropical to warm temperate areas, together with BPH outbreak areas, [14]. In areas where BPH outbreaks occur, it is often found that *Crytorrhinus lividipennis* is able to reduce the population density of BPH, and an increase in the number of *Crytorrhinus lividipennis* causes the population density of BPH to decrease as well, [15]. Therefore, in this work, we choose to study *Crytorrhinus lividipennis* as a natural enemy of BPH.

Another biocontrol that will be considered in this paper is the use of a pathogen. We focus on the use of *Metarhizium*, a fungus that is an insect pathogen that can kill pests and which is also known as an entomopathogenic fungus, [16]. Using the *Metarizium* fungus to control BPH is a method of biological prevention and elimination that is effective in the long term and leaves no toxic residue. It is safe for the environment. *Metarizium*'s properties are that it is easy to produce and durable in high environments, being able to live in the soil for years. It is also easy to use by mixing fresh germs into the soil or by mixing with water to spray. It also spreads easily as it can be blown away by the wind or by being attached to people, animals, or insects. Additionally, *Metarhizium* can be used to reduce the population of various pests such as the Rhinoceros beetle (*Oryctes agamemnon arabicus*), [17], the desert locust (*Schistocerca gregaria*), [18], and the BPH (*Nilaparvata lugens*), [19].

Mathematical modeling with ordinary differential equation systems has become a tool used to analyze many biological phenomena. In the past few years, a number of dynamical models of rice plants have been developed. For example, in 2017, [20], published an epidemic model to study the occurrence of disease in rice due to infection by the Southern rice black-stained dwarf virus with the vector, the white-backed planthopper, *Sogatella furcifera*. In 2020, a mathematical model of rice tungro disease with insecticide and biological agent was published by [21]. In 2021, [22], studied a dynamical model of rice blast disease that is affected by tropical climate conditions. In 2023, [23], analyzed a tungro virus disease spread model in rice plants by investigating the characteristics of two viruses, namely, the rice tungro spherical virus and the rice tungro bacilliform virus. In 2024, [24], applied optimal control theory to a vector-borne rice yellow mottle virus disease problem. In the same year, [25], developed mathematical models for the interaction of rice and BPH to illustrate the effects of habitat complexity and monsoon migration.

One of the most prominent recent mathematical models for rice-pest interactions has been proposed by [26], who assessed the effects of IPM, which combined a cultural method and a chemical method, for managing insect pest outbreaks through the use of optimal control. IPM strategies have also been studied in conjunction with mathematical models of other types of plants, for example, by [27], who presented a mathematical model with IPM of *Jatropha curcas* in 2019. In 2021, [28], presented a mathematical model for controlling fall armyworm management on maize biomass by using IPM strategies. The above research has certainly demonstrated many useful results and improved the existing knowledge on IPM strategy modeling.

In this paper, we study a mathematical model for the relationship between susceptible and infected BPHs with IPM treatment by a pathogen *Metarhizium*, a natural enemy of BPH such as *Crytorrhinus lividipennis*, and a chemical pesticide.

The remainder of this paper is organized as follows. In Section 2, we describe the assumptions, the derivation, and the details of the model. In Section 3, we discuss best fitting for six functional responses for *Crytorrhinus lividipennis* consumption on BPH density. In Section 4, the *Metarhizium* fungus will be fitted to the susceptible-infected-removed model to determine the parameters. Next, in Section 5, we prove the positivity and boundedness of the models and derive the equilibrium points and investigate their stability. In Section 6, numerical computations are shown to illustrate the analytical results. Finally, in Section 7, we discuss and summarize the results obtained in this study.

2 Assumptions and Modeling

2.1 Assumptions

Integrating the IPM strategy into the mathematical model is an effective way to consider controlling insect pest outbreaks. Our model focuses on a scenario in which the effects of infections by entomopathogenic fungus (*Metarhizium*), predation by the natural enemy (*Crytorrhinus lividipennis*), and poisoning by chemical insecticides are the main factors controlling BPH populations. The following assumptions are made to formulate our mathematical model:

[H1] The BPH population thrives under abundant rice resources and no other resource limitations. In such a situation, the BPH population can grow or at least approximately grow in an exponential manner.

[H2] Individual BPH is susceptible to getting the disease because the *Metarhizium* fungus affects all BPH stages: egg, nymphal, and adult stages. The fungus *Metarhizium* does not cause significant disease in *Crytorrhinus lividipennis*.

[H3] Infected BPH cannot revert to susceptible BPH. That is, if a BPH has been exposed to the fungus and has symptoms, then it is unable to recover. When infected BPH show symptoms, they become motionless or barely movable, are covered with hard spores, and eventually die.

[H4] Infected BPH cannot produce new offspring.

[H5] *Crytorrhinus lividipennis* attacks mainly BPH eggs to reduce the number of BPH.

[H6] The consumption of BPH eggs by *Crytorrhinus lividipennis* correlates with a functional response.

[H7] Predator ingestion of infected eggs has no significant effect on the system. BPH eggs that are exposed to the fungus die so rapidly that natural enemies cannot hunt them. The natural enemy *Crytorrhinus lividipennis* is more likely to feed on the healthy eggs.

[H8] *Crytorrhinus lividipennis* can consume other food resources (reserve food) to support their population.

[H9] The growth of the natural enemy population, *Crytorrhinus lividipennis*, cannot continue indefinitely as the growth is limited by resource availability and environmental constraints.

[H10] The chemical pesticide affects both susceptible BPH and its natural enemy, but has no significant effect on infected BPH.

2.2 Modeling

We assume that the three variables in the mathematical model are susceptible BPH population density $B_s(t)$ at time t, infected BPH population density $B_i(t)$ at time t, and predatory *Cyrtorhinus lividipennis* population density $P(t)$ at time t. With the assumptions in subsection 2.1, the system of ordinary differential equations governing the population dynamics is given in system (1). A diagram of the model is given in Figure 1.

$$\begin{aligned}\frac{dB_s(t)}{dt} &= rB_s - \beta B_s B_i - f(B_s)P - c_s B_s,\\ \frac{dB_i(t)}{dt} &= \beta B_s B_i - \gamma B_i,\\ \frac{dP}{dt} &= \alpha P\left(1 - \frac{P}{K_p}\right) + \mu f(B_s)P - c_p P,\end{aligned} \tag{1}$$

where r is the intrinsic growth rate of susceptible BPH, representing the difference between the natural birth and death rates. β is the disease transmission coefficient. γ is the mortality rate with the disease. α is the intrinsic growth rate of the predator, representing the birth rate of *Cyrtorhinus lividipennis* supported by food reserves minus the natural death rate of *Cyrtorhinus lividipennis*. K_p is the carrying capacity for *Cyrtorhinus lividipennis* population. μ is the energy conversion rate, which means the rate of energy conversion from consuming prey to reproductive success and production of the new offspring of *Cyrtorhinus lividipennis*. The functional response $f(B_s)$ means the ability of *Cyrtorhinus lividipennis* to consume eggs of susceptible BPH. c_s and c_p are the mortality rates due to chemical insecticide of susceptible BPH and *Cyrtorhinus lividipennis*, respectively. The initial conditions are given by $B_s(t_0) = B_{s,0} \geq 0$, $B_i(t_0) = B_{i,0} \geq 0$ and $P_0(t_0) = P_0 \geq 0$. All parameters in system (1) are positive.

In the absence of *Metarhizium*, *Cyrtorhinus lividipennis*, and chemical insecticide, the growth dynamics of susceptible BPH population density is to increase exponentially, represented by the term rB_s. Especially, when there is a large amount of food in the area, BPH population is large enough to create a new population quickly. Including the sudden arrival of BPH can lead to a rapid increase in the BPH

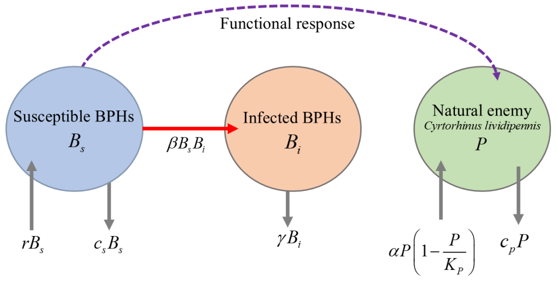


Fig. 1: Schematic diagram of the dynamical model with IPM given by system (1). Susceptible BPH population density at time t is denoted by $B_s(t)$. Infected BPH population density at time t is denoted by $B_i(t)$. Predatory *Cyrtorhinus lividipennis* population density at time t is denoted by $P(t)$.

population. This situation usually occurs in the early stages of a BPH outbreak.

Assumption [H1] means that there is no significant environmental constraints on the population growth of BPH. We define the term $\beta B_s B_i$ to represent the pathogenicity of the fungus *Metarhizium*, as assumed in [H2]. We ignore the recovery term of infected BPH by the assumption [H3]. However, the term γB_i represents the decay of infected BPHs. Assumption [H4] indicates that we neglect the increase in the BPH population dependent on infected BPHs because BPHs infected by the *Metarhizium* fungus often suffer physiological damage that results in a significant reduction in their reproductive ability, [29], [30]. Consequently, infected individuals typically do not contribute to reproduction in this population dynamics. In addition, assumption [H2] also indicates that *Metarhizium* does not cause significant disease in the predator, [31].

When BPH population grows, its natural enemies appear as predators, namely *Cyrtorhinus lividipennis*. *Cyrtorhinus lividipennis* can reduce the population of BPH by consuming BPH eggs, reducing the total population of BPH as assumed in [H5]. From assumption [H6], the functional response is given by $f(B_s)$ expressed in the terms $f(B_s)P$ and $\mu f(B_s)P$. Assumption [H7] is used to define the role between *Cyrtorhinus lividipennis* and infected BPH. It shows that *Cyrtorhinus lividipennis* does not consume eggs of infected BPH, that is, there is no direct relationship between $P(t)$ and $B_i(t)$.

According to assumption [H8], we assume that there are other reserve foods that can support the population of *Cyrtorhinus lividipennis*. Nevertheless, following the assumption [H9], the population growth of *Cyrtorhinus lividipennis* due to reserved foods has limitations represented by the carrying capacity of *Cyrtorhinus lividipennis* given by K_p. Therefore, the term $\alpha P\left(1 - P/K_p\right)$ refers to assumptions [H8] and [H9]. Then the negative density effect is introduced in the per capita growth rate of the enemy population as $\alpha\left(1 - P/K_p\right)$ indicating the logistic growth. Examples of alternative foods include eggs of the whitebacked planthopper (WBPH), *Sogatella furcifera*, [32], [33], and the green leafhopper (GLH), *Nephotettix virescens*, [33].

BPH infected with *Metarhizium* may have an impaired immune system, and this can make them more sensitive to chemical insecticides. On the other hand, the spores that emerge after infection may provide some protection against the chemicals. This, along with the reason that infected insects are usually less motile than susceptible ones, means that they are less likely to come into contact with the chemicals. However, according to assumption [H3], infected BPH dies after a certain time. We therefore assumed that the chemicals had no significant effect on infected BPHs. In addition, there are insecticides that specifically target healthy BPH while having a minimal effect on infected BPH, such as dinotefuran, [34], [35], imidacloprid, [35], [36], and thiamethoxam, [36]. It is also interesting that chemical intervention often affects insects that are natural enemies of BPHs, [37], [38]. Therefore, *Cyrtorhinus lividipennis* are assumed to be affected by the insecticides in this model. Application of chemical insecticide is defined by the assumption [H10] which indicates that the chemical intervention affects susceptible BPH and *Cyrtorhinus lividipennis*, given by the terms $c_s B_s$ and $c_p P$, respectively.

3 Functional Responses of *Cyrtorhinus lividipennis* on BPH

In this subsection, we consider functional response models based on equation (1) to determine the suitability and rationale for observing the consumption behavior of the BPH eggs by the *Cyrtorhinus lividipennis*. We compare six candidate functional responses by fitting parameters to the following models: Holling's type I, Holling's type II, Holling's type III, Holling's type IV, Roger's type, and Ivlev's type, and checking for suitable parameters using statistical techniques. We introduce the functional responses as models for the consumption curve of adult *Cyrtorhinus lividipennis* on BPH egg density.

3.1 Functional response models

The factor denoted by f in (1) represents the effect of the egg consumption by an individual of *Cyrtorhinus lividipennis* on the recruitment of reproductive BPH individuals. Thus, in our modeling, f could be given as a function of the number of eggs produced by the adult BPH population (i.e., B_s). Since the BPH population intrinsically is assumed to grow in the exponential manner with the constant growth rate r, we can assume in addition that the total number of eggs produced by BPH adults is proportional to B_s. Hence, for mathematical simplicity, we give the factor as a function of B_s, $f = f(B_s)$ in (1).

In 1959, [39], presented the simplest expression for the relationship between prey offered and prey consumed as the linear functional response,

$$f_{\text{HollingI}}(x) = a_1 x, \tag{2}$$

where a_1, which is a parameter to be fitted, denotes attack rate on BPH eggs by *Cyrtorhinus lividipennis*. Normally, (2) is known as the Holling's type I or Lotka-Volterra type, [40]. Essentially, (2) describes a situation where a predator's consumption rate increases linearly with prey density, meaning that the higher the prey density, the greater the predator's attack rate. In the same study, Holling also introduced a functional response that adds the parameter of the time to pick up one prey, which can be written in the following form:

$$f_{\text{HollingII}}(x) = \frac{a_2 x}{1 + a_2 b_2 x}, \tag{3}$$

where a_2 is the rate of successful search, and b_2 is a positive value and denotes the time to pick up one BPH, that is, handling time, [39]. Generally, equation (3) is called the Holling's type II functional response, or the Holling's disc equation. The relationship between prey density and predator consumption resembles the shape of a hyperbola with a finite upper bound for the predation rate in terms of the prey density.

According to [41], [42], when the attack rate a_2 in (3), changes from being constant to depending linearly on prey density as $a_3 x$, then the functional response becomes a sigmoid curve that can be written in the form

$$f_{\text{HollingIII}}(x) = \frac{a_3 x^2}{1 + a_3 b_3 x^2}, \tag{4}$$

where a_3 is the attack coefficient, and b_3 is handling time. This form of sigmoid functional response is often called the Hill function with reference to the usage of the formula in the context of chemical kinetics, see for example in [43].

One of the most interesting ecological phenomena between predators and prey is the prey's defense behavior. Prey can defend themselves by various means, for example, by the swarming effect or by the anti-predator behavior of adult prey attacking juvenile predators. However, the prey's defense behavior depends on the population density, [44], [45], [46]. These phenomena can reduce the attack rate or increase the predator's capture time. These behaviors are often encountered in the form of a dome-shaped curve, [46], [47], [48]. This corresponds to Holling's type IV functional response which is given by:

$$f_{\text{HollingIV}}(x) = \frac{a_4 x}{b_4 + x^2}, \tag{5}$$

which takes the maximum value $a_4/(2\sqrt{b_4})$ for $x = \sqrt{b_4}$. Equation (5), which is also called the simplified Monod-Haldane function, was proposed by [47]. Examples of the functional response type IV applied to studying insect societies are given in many papers, [46], [48], [49], [50]. In addition to the functions mentioned earlier, predator food consumption can also depend on the predator consumption velocity, for example, the faster consumption can be assumed to reduce the consumption velocity. An example of such functional response is Roger’s type functional response, which can be written in the following form:

$$f_{\text{Roger}}(x) = x \left(1 - \exp\left(-a_5 \left(1 - b_5 f_{\text{Roger}}(x)\right)\right)\right), \tag{6}$$

where a_5 is the attack rate and b_5 is the handling time, [51], [52]. Equation (6) is sometimes called the Roger's random predator equation, [51]. Moreover, we can use the Lambert W function to solve (6) (for details see, [52]).

In mathematical models, there is another function that has been studied, namely the Ivlev's functional response, which can be expressed in the following form:

$$f_{\text{Ivlev}}(x) = a_6 \left(1 - \exp(-b_6 x)\right), \tag{7}$$

where a_6 denotes the maximum number of attacks that can be made per predator, and b_6 denotes a positive constant, which is a coefficient relating change in prey consumption rate to prey concentration, [53]. Ivlev's functional response can also be obtained from Watt's equation, [39], [54].

In addition to the above models, there are many functional response models in which we are not interested in this study, such as the generalized Holling's type III functional response, [42], the generalized Holling's type IV functional response, [45], [55], and the Roger's random predator type III functional response, [56], [57], [58], because those functions do not adequately explain the biological meaning of BPH and its predators.

3.2 Data collection and processing

The data used in this section to calculate functional responses showing the number of BPHs (*N. lugens*) eggs consumed per *Cyrtorhinus lividipennis* were originally given in Table 1 in the research work of [7]. The following details were given for the data: BPH populations were maintained at the Department of Entomology, Faculty of Agriculture, Khon Kaen University, Thailand. Five densities of eggs of BPH were offered as 5, 10, 20, 30, and 40 eggs per predator. Here, average data on adult male and female stages of *Cyrtorhinus lividipennis* consumption were used.

Non-linear regression analysis was performed on this data using the *fitnlm* function in MATLAB to model the relationship between the independent and dependent variables using the six functional responses in (2)-(7). The independent variable x consisted of five values: 5, 10, 20, 30, and 40. The dependent variable $f(x)$ was calculated as the mean of the data from two groups, adult male and adult female of *Cyrtorhinus lividipennis*. For the *fitnlm* function, the constant(s) to be fitted were constrained by lower and upper bounds, and initial guesses were provided. Subsequently, the output of the fitting parameter command *fitnlm* estimated values of the model coefficient(s) for each model.

The statistical techniques that were applied in this work to determine the suitability of functional responses for data were the sum of square errors (SSE), the coefficient of determination (R-squared; R^2), the adjusted coefficient of determination (adjusted R-squared; $\mathrm{R}^2_{\text{adj}}$), and the root-mean-square error (RMSE). In addition, model selection criteria that are commonly used in statistics, such as the Akaike information criterion (AIC), and the corrected Akaike information criterion (AICc), [59], [60], were used to select parameters. Let n indicate the total number of data points, p indicate the number of parameters in the model. $\hat{y}_i$ signify the predicted value, y_i signify the observed value, and $\bar{y}$ signify the mean of the observed values. The SSE was computed by

$$\mathrm{SSE} = \sum_{i=1}^{n} (y_i - \hat{y}_i)^2 . \tag{8}$$

The R^2 was calculated by

$$\mathrm{R}^2 = 1 - \frac{\sum_{i=1}^{n}(y_i - \hat{y}_i)^2}{\sum_{i=1}^{n}(y_i - \bar{y})^2}. \tag{9}$$

The adjusted R^2 was calculated by

$$\mathrm{R}^2_{\text{adj}} = 1 - \left(1 - \mathrm{R}^2\right) \frac{n-1}{n-p-1}. \tag{10}$$

The RMSE was calculated by

$$\mathrm{RMSE} = \sqrt{\frac{1}{n}\sum_{i=1}^{n}(y_i - \hat{y}_i)^2}. \tag{11}$$

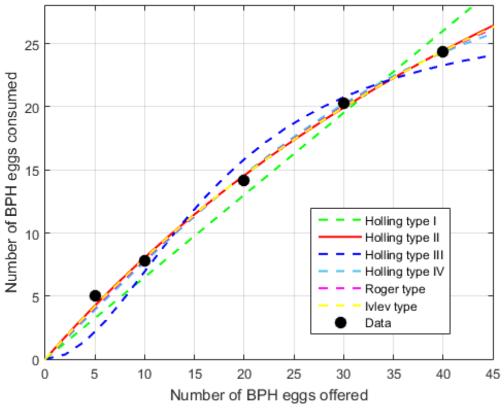


Fig. 2: Plots of six functional responses with data points.

The AIC was calculated by

$$\mathrm{AIC} = n \ln\left(\frac{\mathrm{SSE}}{n}\right) + 2p. \tag{12}$$

The AICc was computed by

$$\mathrm{AICc} = \mathrm{AIC} + \frac{2p(p+1)}{n-p-1}. \tag{13}$$

3.3 Results of fitting functional responses

We show the results of adult *Cyrtorhinus lividipennis* consumption on BPH eggs by using the disc method in Table 1. The results indicate that Holling's Type II functional response has the highest R^2 and adjusted-R^2 values. Similarly, Holling's Type II has the lowest values for SEE and RMSE. However, we should also consider the AIC and AICc values of functional responses where the AICc value of Holling's type II functional response was the lowest. Therefore, the Holling's type II was taken as the best fitting model for adult *Cyrtorhinus lividipennis*. A plot of all functional responses for adult *Cyrtorhinus lividipennis* is shown in Figure 2.

4 Pathogen Virulence Assay

In this section, we exploited the fit of the cumulative number of BPH deaths in the treated group under laboratory conditions for obtaining the transmission rate and mortality rate of BPH due to *Metarhizium* using the susceptible-infected-removed (SIR) model.

4.1 Data collection and processing

BPH population data was gathered from three distinct geological regions in Thailand: Phetchabun province in the Central region, Buri Ram province in the Northeast region, and Phatthalung province

Table 1. Summary of theoretical values of consumption of adult *Cyrtorhinus lividipennis* from all functional responses.

Statistical Test	Holling's Type I	Holling's Type II	Holling's Type IIII	Holling's Type IV	Reger's Type	Ivlev's Type
Estimated Parameters	$a_1 = 0.6509$	$a_2 = 0.9009$ $b_2 = 0.0131$	$a_3 = 0.0928$ $b_3 = 0.0362$	$a_4 = 4303$ $b_4 = 5480$	$a_5 = 1.856$ $b_5 = 0.0202$	$a_6 = 44.81$ $b_6 = 0.0196$
SSE	9.2548	0.8931	13.194	1.4369	1.0729	0.9375
RMSE	1.5211	0.5456	2.0971	0.6921	0.598	0.559
R^2	0.9652	0.9966	0.9504	0.9946	0.9960	0.9965
R^2_{adj}	0.9652	0.9955	0.9338	0.9928	0.9946	0.9953
AIC	19.2679	9.5769	23.041	11.9547	10.494	9.8192
AICc	20.6012	15.5769	29.041	17.9547	16.494	15.8192

in the South region, during 2021-2022. Data on *Metarhizium neoanisopliae* strain BCC4849 was obtained from the BIOTEC Culture Collection (BCC), Thailand. The sample consisted of 270 BPH individuals. The BPH mortality was recorded daily over a span of 7 days. The BPH data before and after infection with *Metarhizium* fungus are given in Figure 3.

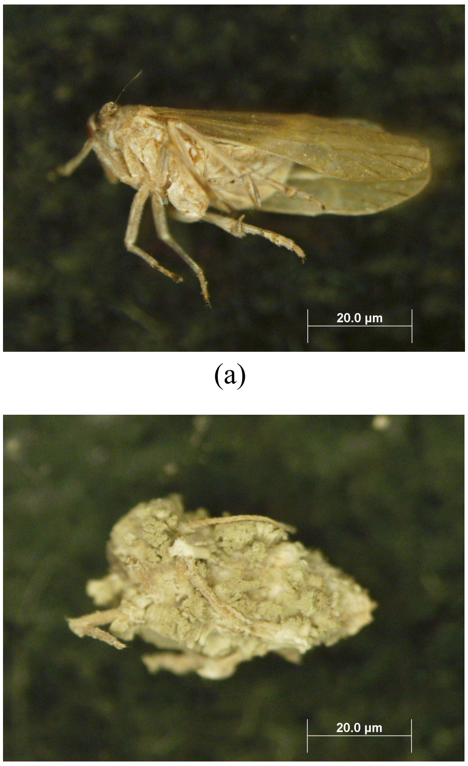
For parameter estimation, the two Matlab functions used in this section were *ode45* and *fminsearch*. The *ode45* function, based on the 4th and 5th-order Runge-Kutta integration method known as the Dormand-Prince pair, [61], was employed to numerically solve the dynamical system. The function *fminsearch*, based on the Nelder-Mead simplex direct search method, [62], was used for parameter optimization to fit experimental data.

4.2 Model based on experimental data

The data included in our experimental design is explained in subsection 4.1. We considered an SIR model, [63], [64], based on experiments to determine the infection rate or severity of *Metarhizium* fungus. In a practical experiment, it was assumed that when we introduce fungus, every BPH in the trial unit comes into contact with the fungus.

Let the susceptible BPH population density B_s denote the population of BPH individuals exposed to *Metarhizium* but not necessarily infected. The infected BPH population density is denoted by B_i. The removed BPH population, B_r, denotes BPHs that died from exposure to the *Metarhizium* fungus and have been removed from the system. Removed BPH individuals cannot return to the susceptible or infected class.

Based on model (1) in Section 2, we considered the effect of *Metarhizium* fungus only; that is, the system (1) excluded the variable of predator ($P(t) \equiv 0$). In the experiment, the number of susceptible BPHs cannot be increased by producing new populations because our experimental period of 7 days was too short for a gestation period of BPHs. We therefore assumed that there was no growth term of exposed BPH that is $r = 0$. There is no chemical effect in this section $c_s = 0$. All BPH individuals were exposed to the fungus, so we assumed that BPH mortality occurred only due to the fungal toxins, with β representing the transmission rate from exposed group to infected group and γ representing the BPH mortality rate in the infected group. The numbers of BPH in the populations in



(a)

(b)

Fig. 3: The characteristics of *Metarhizium* fungal infestation on BPH: (a) Before treatment with *Metarhizium*, (b) After treatment with *Metarhizium*.

the laboratory were established before the experiment and remained constant throughout the study. The total BPH population was given by $B_t = B_s + B_i + B_r$, which satisfies the differential equation $B_t' = 0$. The experiment contained 270 BPHs, that is, $B_t = 270$.

The number of dead BPH was counted daily and they were removed from the system. The model used in this section was given by

$$\begin{aligned}\frac{dB_s(t)}{dt} &= -\beta B_s B_i,\\ \frac{dB_i(t)}{dt} &= \beta B_s B_i - \gamma B_i,\\ \frac{dB_r(t)}{dt} &= \gamma B_i,\end{aligned} \tag{14}$$

where parameters β and γ can be determined by fitting the differential system (14) to the laboratory data with the initial conditions $B_{s,0} \geq 0$, $B_{i,0} \geq 0$, $B_{r,0} \geq 0$ at the initial time t_0. The number of removed BPH B_r was obtained by counting the number of dead BPH from day 1 to day 7.

4.3 Results of fitting data Metarhizium

Parameters β and γ for the SIR model shown in (14) were fitted using *ode45* and *fminsearch* functions from Matlab with guessed values as $\beta = 0.5$, and $\gamma = 0.5$.

For setting the initial conditions, we considered that at the start $(t = 0)$ of the experiment no death had occurred. Therefore, we set $B_r(0) = 0$. BPH death may occur on day 1. However, BPH can be infectious from day one. We considered the lowest infection on the first day to be 1 individual to observe the spread of *Metarhizium* fungus, so $B_i(0) = 1$ and $B_s(0) = 269$.

After obtaining the parameters β and γ, the basic reproduction number $(\mathcal{R}_0)$ is given by $\mathcal{R}_0 = \beta B_t/\gamma$. Median lethal time (LT$_{50}$) can be calculated by estimating $B_r(t) \geq B_t/2$ and calculating the smallest t value corresponding to the time that BPH populations approaches 50% mortality.

The calculated results are shown in Table 2. In addition, the visualization of the model is also shown in Figure 4.

5 Mathematical Results of the Model with IPM

According to the model (1) with the Holling's type II functional response in Section 3, we can express the main model used in this study as follows:

Table 2. Estimation from SIR model using initial conditions $B_{s,0}(0) = 269$, $B_{i,0}(0) = 1$ and $B_{r,0}(0) = 0$ and guessed parameters $\beta = 0.5$, $\gamma = 0.5$. We also exhibit the basic reproduction number $\mathcal{R}_0$ and the median lethal time LT$_{50}$.

Parameters Estimated	β	0.0107
	γ	1.0387
Goodness-of-fit	RMSE	7.0032
	R^2	0.9948
$\mathcal{R}_0$		2.781
LT$_{50}$ (days)		3.90

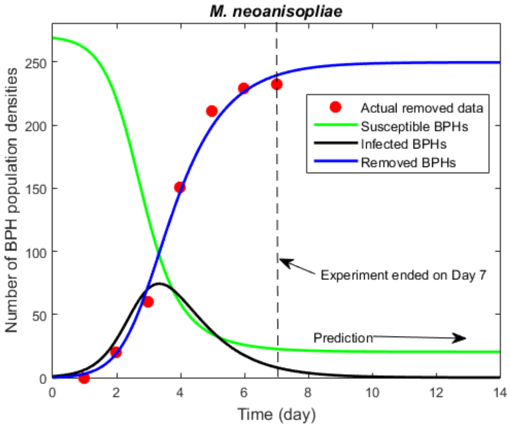


Fig. 4: This figure shows plot of the system (14). Estimated parameters used to plot are shown in Table 2.

$$\begin{aligned}\frac{dB_s(t)}{dt} &= rB_s - \beta B_s B_i - \frac{aB_sP}{1+abB_s} - c_sB_s,\\ \frac{dB_i(t)}{dt} &= \beta B_s B_i - \gamma B_i,\\ \frac{dP}{dt} &= \alpha P\left(1-\frac{P}{K_p}\right) + \mu\frac{aB_sP}{1+abB_s} - c_pP,\end{aligned} \tag{15}$$

where a is the rate of successful search and b is the handling time given by (3) with $B_{s,0} \geq 0$, $B_{i,0} \geq 0$ and $P_0 \geq 0$. All parameters are defined in section 2.2.

5.1 Positivity and boundeness

Theorem 5.1 *For given initial condition $W(0) \in \mathbb{R}^3_+$, the system* (15) *has a unique solution. Further, the trajectories of* (15) *starting at $W(0)$ are positive and uniformly bounded.*

Proof. Let $W(0) = (B_s(0), B_i(0), P(0)) \in \mathbb{R}^3_+$. Before proving the existence and uniqueness of a solution, we will prove that if solutions exist, they are non-negative and bounded. We prove the non-negativity of the trajectories of (15) by solving the first equation of system (15). We get the following relationship:

$$\begin{aligned}B_s(t) = B_s(0)\exp\Bigg[\int_0^t \Big(r - \beta B_i(\tau) \\ - \frac{aP(\tau)}{1+abB_s(\tau)} - c_s\Big)d\tau\Bigg].\end{aligned} \tag{16}$$

Therefore, $B_s(t) \geq 0$ if $B_s(0) \geq 0$ and $B_s(t) > 0$ if $B_s(0) > 0$. The proof for $B_i(t)$ and $P(t)$ can be conducted in the same manner.

We let the function $W(t) = B_s(t) + B_i(t) + P(t)$ and $\sigma \in \mathbb{R}$. Accordingly, we get the derivative with respect to time as:

$$\begin{aligned}\frac{dW(t)}{dt} + \sigma W(t) =& \alpha P\left(1 - \frac{P}{K_p}\right) \\ &- (1-\mu)\frac{aB_sP}{1+abBs} \\ &+ \left[\sigma - (c_s - r)\right]B_s \\ &+ (\sigma - \gamma)B_i + (\sigma - c_p)P.\end{aligned} \tag{17}$$

We choose $\sigma = \min\{c_s - r, \gamma, c_p\}$, with $c_s > r$ and $0 < \mu \leq 1$. We have

$$\frac{dW(t)}{dt} + \sigma W(t) \leq \alpha P\left(1 - \frac{P}{K_p}\right) \leq \frac{\alpha K_p}{4}. \tag{18}$$

Hence, we have

$$W(t) \leq \exp(-\sigma t)W(0) + (1 - \exp(-\sigma t))\frac{\alpha K_p}{4\sigma}, \tag{19}$$

for $t \geq 0$. Thus it holds for $t \geq 0$ that

$$W(t) \leq \max\left[W(0), \frac{\alpha K_p}{4\sigma}\right] < \infty, \tag{20}$$

and therefore the solution is bounded. □

5.2 Existence of equilibrium points

The model (15) has the following six equilibrium points:

(i) The trivial equilibrium $\mathcal{E}_0(0,0,0)$ always occurs. This corresponds to the case of BPH and *Cyrtorhinus lividipennis* not appearing in the system.

(ii) The BPH free equilibrium is expressed as $\bar{\mathcal{E}}(0, 0, \frac{K_p}{\alpha}(\alpha - c_p))$. This is an interesting equilibrium point because it describes the situation where BPHs disappear from the system but *Cyrtorhinus lividipennis* are available. This equilibrium is valid if $\alpha > c_p$, that is, the intrinsic growth rate of the predator based on food reserves α, which is the difference between the *Cyrtorhinus lividipennis*'s birth rate supported by eating other foods and their natural mortality rate, is greater than chemical effect c_p. Conversely, if the growth rate of *Cyrtorhinus lividipennis* supported by alternative food is lower than the rate of destruction by chemicals $\alpha \leq c_p$, the BPH free equilibrium $\bar{\mathcal{E}}$ does not exist.

(iii) The predator free equilibrium $\tilde{\mathcal{E}}(\frac{\gamma}{\beta}, \frac{r-c_s}{\beta}, 0)$. This means that there are no predator *Cyrtorhinus lividipennis* left to sustain the system. This equilibrium is feasible if $r > c_s$, otherwise $\tilde{\mathcal{E}}$ does not exist.

(iv) The infection free equilibrium $\hat{\mathcal{E}}\left(\hat{B}_s, 0, \hat{P}\right)$. This is associated with the case that the infected BPH has been eliminated, but susceptible BPH and *Cyrtorhinus lividipennis* still remain. The expression of $\hat{B}_s$ can be implicitly written as

$$\hat{B}_s = \frac{1}{ab}\left(\frac{a\hat{P}}{r - c_s} - 1\right). \tag{21}$$

Then, $\hat{B}_s$ is feasible if

$$0 < r - c_s < a\hat{P}, \tag{22}$$

and $\hat{P}$ is the positive root of the following equation:

$$\hat{P}^2 - \frac{K_p}{\alpha}\left(\alpha - c_p + \frac{\mu}{b}\right)\hat{P} + \frac{\mu K_p(r - c_s)}{\alpha ab} = 0. \tag{23}$$

Since $r - c_s > 0$, the solution $\hat{P}$ of equation (23) does not have a positive single real root. Therefore, two positive real roots of equation (23) denoted by $\hat{P}_{1,2}$ can be written in the following form

$$\begin{aligned}\hat{P}_{1,2} =& \frac{1}{2}\Bigg\{\frac{K_p}{\alpha}\left(\alpha - c_p + \frac{\mu}{b}\right) \\ &\pm\sqrt{\frac{K_p^2}{\alpha^2}\left(\alpha - c_p + \frac{\mu}{b}\right)^2 - \frac{4\mu K_p(r - c_s)}{\alpha ab}}\Bigg\}.\end{aligned} \tag{24}$$

Solution (24) must satisfy the following conditions:

$$r - c_s < \frac{abK_p}{4\mu\alpha}\left(\alpha - c_p + \frac{\mu}{b}\right)^2, \tag{25}$$

and

$$\alpha - c_p + \frac{\mu}{b} > 0. \tag{26}$$

Therefore, if the conditions (25) and (26) are satisfied along with $\hat{P}_1 > (r - c_s)/a$ and $\hat{P}_2 > (r - c_s)/a$, then from (22), the infection free equilibrium point $\hat{\mathcal{E}}$ can exist as two points simultaneously. With the analysis on the inequalities of (22), (25), and (26), we can find the following conditions for the existence of $\hat{\mathcal{E}}$, i.e., if and only if

$$0 < \frac{r-c_s}{a} < \frac{K}{\alpha}(\alpha - c_p) \quad \text{or} \quad \begin{cases} \frac{r-c_s}{a} < \frac{K}{\alpha}(\alpha - c_p); \\ 0 < \alpha - c_p < \frac{\mu}{b}, \end{cases}$$

then the infection free equilibrium point $\hat{\mathcal{E}}$ uniquely exists with $\hat{P} = \hat{P}_2$ given by (24). If and only if

$$\begin{cases} \max\left[0, \frac{K_p}{\alpha}(\alpha - c_p)\right] < \frac{r-c_s}{a} < \frac{bK_p}{4\mu\alpha}\left(\alpha - c_p + \frac{\mu}{b}\right)^2; \\ -\frac{\mu}{b} < \alpha - c_p < \frac{\mu}{b}, \end{cases}$$

we have two points simultaneously as the infection free equilibrium point $\hat{\mathcal{E}}$ where $\hat{P} = \hat{P}_1$ and $\hat{P} = \hat{P}_2$ given by (24), respectively.

(v) The interior equilibrium $\mathcal{E}^*(B_s^*, B_i^*, P^*)$. This corresponds to the case that BPH and *Cyrtorhinus lividipennis* remain, where

$$\begin{aligned} B_s^* &= \frac{\gamma}{\beta}, \\ B_i^* &= \frac{1}{\beta}\Bigg\{ r - c_s \\ &\quad - \frac{K_p \beta a}{\alpha(\beta + ab\gamma)}\left[\alpha - c_p + \frac{\mu a \gamma}{\beta + ab\gamma}\right]\Bigg\}, \\ P^* &= \frac{K_p}{\alpha}\left[\alpha - c_p + \frac{\mu a \gamma}{\beta + ab\gamma}\right], \end{aligned} \tag{27}$$

Therefore, $\mathcal{E}^*$ exists if

$$0 < \frac{K_p \beta a}{\alpha(\beta + ab\gamma)}\left[\alpha - c_p + \frac{\mu a \gamma}{\beta + ab\gamma}\right] < r - c_s. \tag{28}$$

5.3 Local stability of equilibrium points

In this subsection, we discuss the stability of the equilibrium points of system (15) derived in the previous subsection. We first obtain the Jacobian matrix of the system at an equilibrium point $\mathcal{E} = (B_s, B_i, P)$ given by

$$J(\mathcal{E}) = \begin{bmatrix} G_1 & -\beta B_s & -\frac{aB_s}{1+abB_s} \\ \beta B_i & -\beta B_s - \gamma & 0 \\ G_2 & 0 & G_3 \end{bmatrix}. \tag{29}$$

where $G_1 = r - c_s - \beta B_i - \frac{aP}{1+abB_s} + \frac{a^2 bB_s P}{(1+abB_s)^2}$, $G_2 = \frac{\mu a P}{1+abB_s} - \frac{\mu a^2 b B_s P}{(1+abB_s)^2}$, and $G_3 = \alpha - c_p - \frac{2\alpha P}{K_p} + \frac{\mu a B_s}{1+abB_s}$. Making use of the eigenvalues of the Jacobian matrix (29) for each of the feasible equilibria obtained in the previous section, we can obtain the condition for their local stability. We show the results in this section as five theorems which we will use to check local stability in the following section of numerical simulations.

Theorem 5.2 *The trivial equilibrium point* $\mathcal{E}_0$ *of system* (15) *is locally asymptotically stable if* $r < c_s$ *and* $\alpha < c_p$.

Proof. The stability of the equilibrium point $\mathcal{E}_0$ of model (15) can be obtained by computing the Jacobian matrix at point $\mathcal{E}_0$ as follows:

$$J(\mathcal{E}_0) = \begin{bmatrix} r - c_s & 0 & 0 \\ 0 & -\gamma & 0 \\ 0 & 0 & \alpha - c_p \end{bmatrix}. \tag{30}$$

Since the Jacobian is diagonal, the eigenvalues are the diagonal elements. Therefore,

$$\lambda_1 = -\gamma < 0,\ \lambda_2 = r - c_s,\ \lambda_3 = \alpha - c_p. \tag{31}$$

Therefore, $\mathcal{E}_0$ is locally asymptotically stable if $r < c_s$ and $\alpha < c_p$. ☐

Theorem 5.3 *When the BPH free equilibrium point* $\bar{\mathcal{E}}$ *of system* (15) *exists, it is locally asymptotically stable if* $r - c_s < \frac{aK_p}{\alpha}(\alpha - c_p)$. *Otherwise, if* $r - c_s > \frac{aK_p}{\alpha}(\alpha - c_p)$, *it is unstable.*

Proof. The Jacobian matrix of system (15) at $\bar{\mathcal{E}}$ is expressed as

$$J(\bar{\mathcal{E}}) = \begin{bmatrix} r - c_s - \frac{aK_p}{\alpha}(\alpha - c_p) & 0 & 0 \\ 0 & -\gamma & 0 \\ \frac{\mu a K_p}{\alpha}(\alpha - c_p) & 0 & c_p - \alpha \end{bmatrix}. \tag{32}$$

Since the Jacobian is lower tridiagonal, the eigenvalues are the diagonal elements and therefore

$$\begin{aligned} &\lambda_1 = -\gamma < 0, \qquad \lambda_2 = -(\alpha - c_p) < 0, \\ &\lambda_3 = r - c_s - \frac{aK_p}{\alpha}(\alpha - c_p). \end{aligned} \tag{33}$$

Therefore, $\bar{\mathcal{E}}$ is locally asymptotically stable if $\alpha > c_p$ and $r - c_s - \frac{aK_p}{\alpha}(\alpha - c_2) < 0$. ☐

Theorem 5.4 *When the predator free equilibrium* $\tilde{\mathcal{E}}$ *of system* (15) *exists, it is unstable if* $\alpha - c_p + \frac{\mu a \gamma}{ab\gamma + \beta} > 0$. *In contrast, it is locally asymptotically stable only if* $\alpha - c_p + \frac{\mu a \gamma}{ab\gamma + \beta} \leq 0$. *The solution has an oscillatory behavior around the equilibrium.*

Proof. The Jacobian matrix of system (15) at $\tilde{\mathcal{E}}$ is expressed as

$$J(\tilde{\mathcal{E}}) = \begin{bmatrix} 0 & -\gamma & -\frac{\gamma a}{ab\gamma + \beta} \\ r - c_s & 0 & 0 \\ 0 & 0 & \alpha - c_p + \frac{\mu a \gamma}{ab\gamma + \beta} \end{bmatrix}. \tag{34}$$

The eigenvalues of (34) are the lowest diagonal element and the eigenvalues of the top right 2×2 block. Therefore the eigenvalues are:

$$\lambda_1 = \alpha - c_p + \frac{\mu\, a\gamma}{ab\gamma + \beta}, \tag{35}$$

and

$$\lambda_{2,3} = \pm i\sqrt{\gamma\,(r - c_s)}. \tag{36}$$

Therefore, when the equilibrium $\tilde{\mathcal{E}}$ exists, it is unstable if $\alpha - c_p + \frac{\mu\, a\gamma}{ab\gamma+\beta} > 0$. We cannot determine the stability when $\alpha - c_p + \frac{\mu\, a\gamma}{ab\gamma+\beta} < 0$, because of the purely imaginary eigenvalues. However, if the equilibrium $\tilde{\mathcal{E}}$ is locally asymptotically stable, it must be satisfied that $\alpha - c_p + \frac{\mu\, a\gamma}{ab\gamma+\beta} \leq 0$. ☐

Theorem 5.5 *The infection free equilibrium $\hat{\mathcal{E}}$ is locally asymptotically stable if*

$$n_1 > 0, \quad n_2 > 0 \quad \textit{and} \quad \beta\hat{B}_s < \gamma, \tag{37}$$

where n_1 and n_2 are the coefficients of λ in the characteristic equation of the variational matrix of system (15) *evaluated at $\hat{\mathcal{E}}$ which can be arranged in the form*

$$\left(\lambda^2 + n_1\lambda + n_2\right) = 0. \tag{38}$$

Proof. The Jacobian matrix of model (15) at $\hat{\mathcal{E}}$ can be expressed as

$$J(\hat{\mathcal{E}}) = \begin{bmatrix} \frac{a^2b\hat{B}_s\hat{P}}{(1+ab\hat{B}_s)^2} & -\beta\hat{B}_s & -\frac{a\hat{B}_s}{1+ab\hat{B}_s} \\ 0 & \beta\hat{B}_s - \gamma & 0 \\ \Xi & 0 & -\frac{\alpha\hat{P}}{K_p} \end{bmatrix}, \tag{39}$$

where $\Xi = \frac{\mu a\hat{P}}{1+ab\hat{B}_s} - \frac{\mu a^2b\hat{B}_s\hat{P}}{(1+ab\hat{B}_s)^2}$.

The characteristic equation of (39) is

$$\Big(\lambda - (\beta\hat{B}_s - \gamma)\Big)\left(\lambda^2 + n_1\lambda + n_2\right) = 0, \tag{40}$$

where

$$\begin{aligned} n_1 =& \frac{\alpha\hat{P}}{K_p} - \frac{a^2b\hat{B}_s\hat{P}}{(1+ab\hat{B}_s)^2}, \\ n_2 =& -\frac{a^2b\hat{B}_s\hat{P}}{(1+ab\hat{B}_s)^2}\frac{\alpha\hat{P}}{K_p} + \frac{a\hat{B}_s}{1+ab\hat{B}_s}\Xi. \end{aligned} \tag{41}$$

If n_1 and n_2 in (40) are such that $n_1 > 0$ and $n_2 > 0$ along with $\beta\hat{B} - \gamma < 0$, then the equilibrium point $\hat{\mathcal{E}}$ has local asymptotic stability. ☐

Theorem 5.6 *The equilibrium point $\mathcal{E}^*$ is locally asymptotically stable if the following conditions hold:*

$$m_1 > 0, \;\; m_2 > 0, \;\; m_3 > 0, \;\; \textit{and} \;\; m_1m_2 - m_3 > 0, \tag{42}$$

where m_1, m_2, m_3 are the coefficients of λ in the characteristic equation of the variational matrix of system (15) *evaluated at $\mathcal{E}^*$ which can be written in the form*

$$\lambda^3 + m_1\lambda^2 + m_2\lambda + m_3 = 0. \tag{43}$$

Proof. The Jacobian matrix of system (15) at $\mathcal{E}^*$ is given by

$$J(\mathcal{E}^*) = \begin{bmatrix} \frac{a^2bB_s^*P^*}{(1+abB_s^*)^2} & -\gamma & -\frac{aB_s^*}{1+abB_s^*} \\ \beta B_i^* & 0 & 0 \\ \frac{\mu aP^*}{1+abB_s^*} - \frac{\mu a^2bB_s^*P^*}{(1+abB_s^*)^2} & 0 & -\frac{\alpha P^*}{K_p} \end{bmatrix}. \tag{44}$$

Then, the characteristic equation of the matrix $J(\mathcal{E}^*)$ can be obtained in the following form

$$\lambda^3 + m_1\lambda^2 + m_2\lambda + m_3 = 0, \tag{45}$$

where

$$\begin{aligned} m_1 =& \frac{\alpha P^*}{K_p} - \frac{a^2bB_s^*P^*}{(1+abB_s^*)^2}, \\ m_2 =& \frac{a^2bB_s^*P^*}{(1+abB_s^*)^2}\left(\beta B_i^*\right) + \gamma\beta B_i^* \\ &+ \frac{aB_s^*}{1+abB_s^*}\left(\frac{\mu aP^*}{1+abB_s^*} - \frac{\mu a^2bB_s^*P^*}{(1+abB_s^*)^2}\right), \\ m_3 =& \frac{\gamma\beta B_i^*\alpha P^*}{K_p} > 0. \end{aligned} \tag{46}$$

Using the Routh-Hurwitz criterion for the necessary and sufficient conditions for the locally asymptotic stability of system (15) at $\mathcal{E}^*$, if

$$m_i > 0, \, (i = 1, 2, 3), \quad \text{and} \quad m_1m_2 > m_3, \tag{47}$$

hold, then the equilibrium point $\mathcal{E}^*$ is locally asymptotically stable. ☐

5.4 Bifurcation Analysis

We consider parameter β, which represents the fungus's potential to spread from the susceptible group to the infected group, as the bifurcation parameter to observe the long-term effect of the fungus infection on population dynamics. We next consider the stability of the system (15) at the coexistence equilibrium point $\mathcal{E}^*$.

Theorem 5.7 *If there exists a bifurcation point at* $\beta = \beta^*$ *for the equilibrium point* $\mathcal{E}^*(B_s^*, B_i^*, P^*)$ *expressed in* (27)*, then a Hopf bifurcation can occur under the following conditions:*

$$m_2(\beta^*) > 0, \quad m_3(\beta^*) > 0, \quad \mathcal{H}(\beta^*) = 0, \qquad (48)$$

and

$$\left.\frac{d\mathcal{H}(\beta)}{d\beta}\right|_{\beta=\beta^*} \neq 0, \qquad (49)$$

where $\mathcal{H} = m_3 - m_1 m_2$ *and the coefficients* m_1, m_2 *and* m_3 *are formulated in* (46)*.*

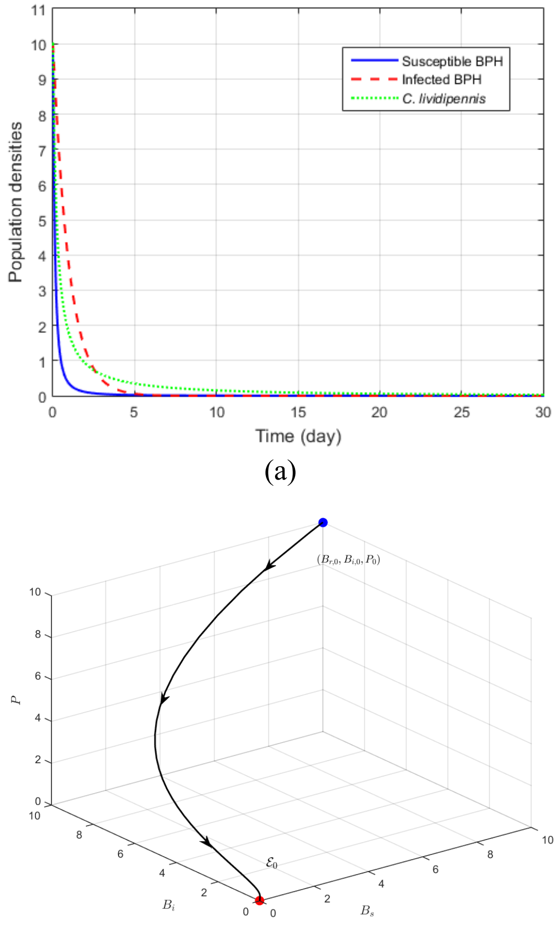
Proof. The proof of this theorem is similar to Theorem 3.6 in [25]. Therefore, we omit the details of this proof here. ☐

6 Numerical Simulations

In this section, we use numerical simulations of model (15) to illustrate the theoretical results presented in section 5. We also give a discussion of the numerical results. For the parameter values a and b in Holling's type II functional response, we used the values given in Table 1 in section 3. The other parameters whose values are required are listed in subsection 2.2 after equation (1). We selected sets of suitable values as follows. There are many reports in the literature on the intrinsic growth rate of BPHs (r) such as [4], [65], [66], [67], [68], [69], with values varying based on the experimental conditions, data collection methods, and factors such as temperature and rice varieties. Here, we picked $r = 0.1803$, presented in [4]. For the disease transmission rate (β) and the death rate of infected BPH (γ) that are indicators of the severity of the *Metarhizium* fungus, we used *M. neoanisopliae* as shown in Table 2, Section 4. For parameters including conversion rate (μ) and intrinsic growth rate (α) of *Cyrtorhinus lividipennis*, we used values from research findings for *Cyrtorhinus lividipennis* in [70], [71]. We assumed values for parameters c_s, c_p and K_p which appeared suitable.

Following the above methods, we obtained our first parameter set $\mathcal{S}_1 := \{\beta = 0.0107, \gamma = 1.0387,\ a = 0.9009,\ b = 0.0131, r = 0.1803,\ \alpha = 0.05,\ c_s = 0.2, c_p = 0.1,\ \mu = 0.123, K_p = 0.1\}$. For this set, the equilibrium $\mathcal{E}_0$ always occurs and since the system (15) reaches equilibrium if $r > c_s$ and $\alpha > c_p$, which is proved in Theorem 5.2, a numerical solution should converge to $\mathcal{E}_0$. Using the parameter set $\mathcal{S}_1$ with the initial values $B_{s,0} = B_{i,0} = P_0 = 10$, we obtained the simulation of (15) for $\mathcal{E}_0$ shown in Figure 5.

We then slid the intrinsic growth rate of *Cyrtorhinus lividipennis* from $\alpha = 0.05$ to $\alpha = 0.5$ in the parameter set $\mathcal{S}_1$. We then had the parameter set $\mathcal{S}_2 := \{\beta = 0.0107, \gamma = 1.0387,\ a = 0.9009,\ b = 0.0131,\ r = 0.1803,\ \alpha = 0.5,\ c_s = 0.2, c_p = 0.1,\ \mu = 0.123, K_p = 0.1\}$. Based on $\mathcal{S}_1$, we see that the rate of insect destruction in $\mathcal{S}_2$ has been decreased since the use of chemicals has decreased. Using the parameter set $\mathcal{S}_2$ with the initial values $B_{s,0} = B_{i,0} = P_0 = 0.1$, we obtained the simulation of (15) shown in Figure 6 which converges to the equilibrium point $\bar{\mathcal{E}}$ as proved in Theorem 5.3.

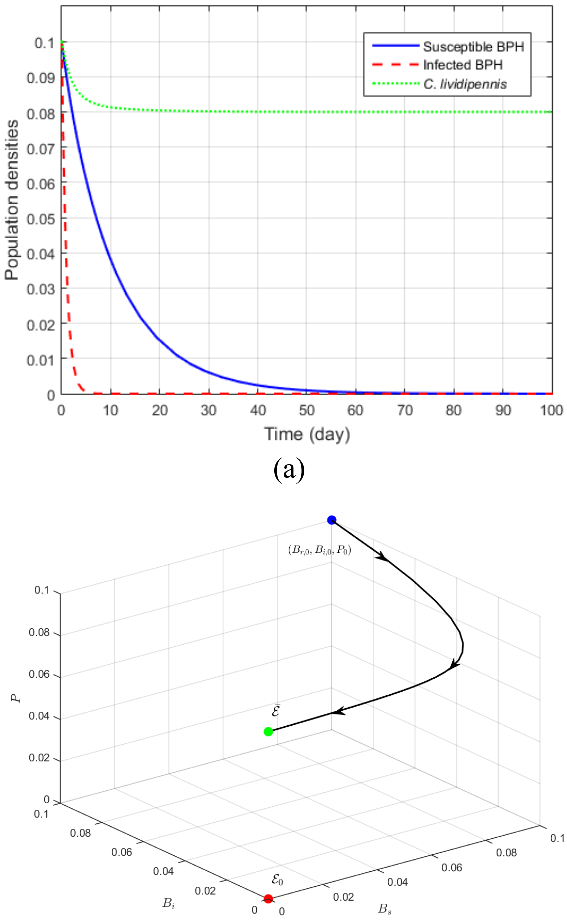


(a)

(b)

Fig. 5: Solution behavior of system (15) when the parameter set $\mathcal{S}_1$ and the initial values $B_{s,0} = B_{i,0} = P_0 = 10$ are used. The eigenvalues are $\lambda_1 = -1.03$, $\lambda_2 = -0.019$ and $\lambda_3 = -0.05$. The solution is locally asymptotically stable at the equilibrium point $\mathcal{E}_0(0, 0, 0)$: (a) Time series solutions of B_s, B_i, and P, (b) Phase plot of B_s, B_i, and P.

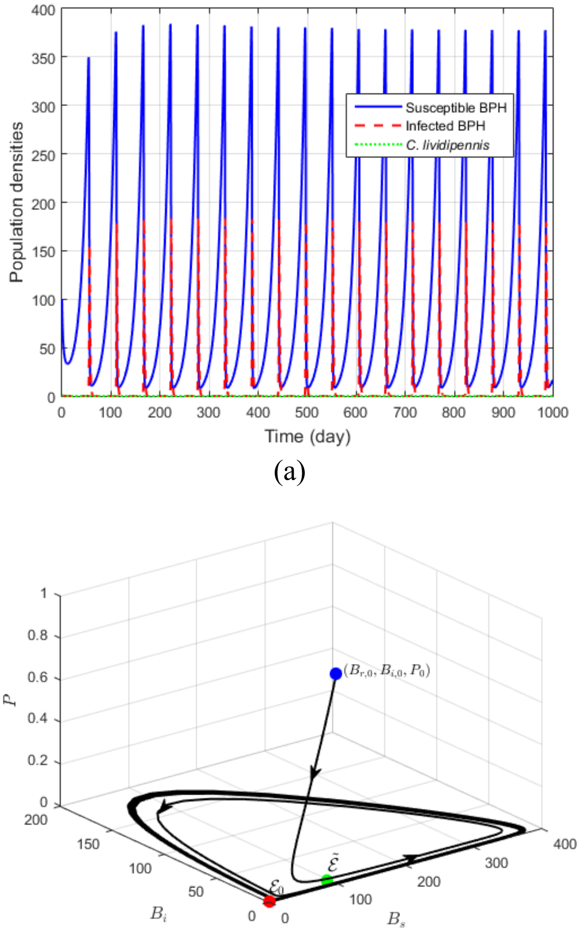
For the next set, we reduced the parameter of the *Cyrtorhinus lividipennis*'s ability to hunt eggs of BPH to ($\mu = 0.001$), to get the parameter set $\mathcal{S}_3 := \{\beta = 0.0107, \gamma = 1.0387,\ a = 0.9009,\ b =$



(a)

(b)

Fig. 6: Solution behavior of system (15) when the parameter set $\mathcal{S}_2$ and the initial values $B_{s,0} = B_{i,0} = P_0 = 0.1$ are used. The eigenvalues are $\lambda_1 = -1.03$, $\lambda_2 = -0.009$ and $\lambda_3 = -0.4$. The solution is locally asymptotically stable at the equilibrium point $\bar{\mathcal{E}}(0, 0, 0.08)$: (a) Time series solutions of B_s, B_i, and P, (b) Phase plot of B_s, B_i, and P.



(a)

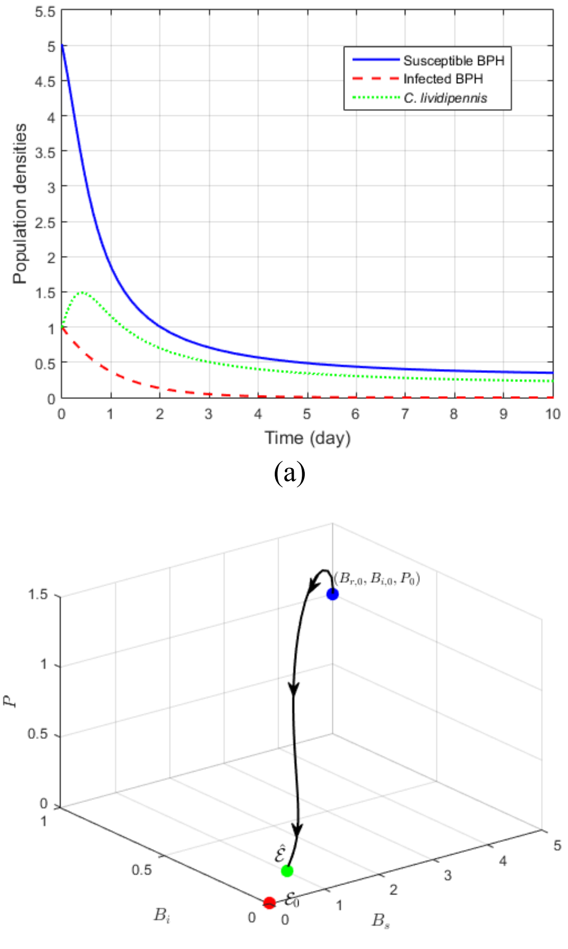
(b)

Fig. 7: Solution behavior of system (15) when the parameter set $\mathcal{S}_3$ and the initial values $B_{s,0} = 100$, $B_{i,0} = P_0 = 1$ are used. The eigenvalues are $\lambda_1 = -0.049$, $\lambda_{2,3} = \pm 0.288i$. The solution oscillates around the equilibrium point $\tilde{\mathcal{E}}(97.07, 7.50, 0)$: (a) Time series solutions of B_s, B_i, and P, (b) Phase plot of B_s, B_i, and P.

$0.0131, r = 0.1803, \alpha = 0.05, c_s = 0.1, c_p = 0.1, \mu = 0.001, K_p = 0.1\}$. Using the parameter set $\mathcal{S}_3$ with the initial values $B_{s,0} = 100, B_{i,0} = P_0 = 1$, we obtained the simulation of (15) shown in Figure 7 which oscillates around $\tilde{\mathcal{E}}$ as proved in Theorem 5.4.

To visualize the stability of the infection free equilibrium point $\hat{\mathcal{E}}$, we substituted $\alpha = 0.2, c_s = 0.001, c_p = 0.08$ and $\mu = 0.98$ into the parameter set $\mathcal{S}_1$. We obtained the set $\mathcal{S}_4 := \{\beta = 0.0107, \gamma = 1.0387, a = 0.9009, b = 0.0131, r = 0.1803, \alpha = 0.2, c_s = 0.001, c_p = 0.08, \mu = 0.98, K_p = 0.1\}$. Using the parameter set $\mathcal{S}_4$ with the initial values $B_{s,0} = 5, B_{i,0} = P_0 = 1$, the simulation of (15) shown in Figure 8 converges to $\hat{\mathcal{E}}$ as proved in Theorem 5.5.

For the stability analysis of the coexistence equilibrium $\mathcal{E}^*$, we assumed an increase in the intrinsic growth rate of BPH (r) and a decrease in the BPH egg consumption ability of *Cyrtorhinus lividipennis* (a). These changes cause the BPH population to remain in the system. When the BPH population is large enough, the food source for the *Cyrtorhinus lividipennis* is sufficient and it remains in the system as well. Based on parameter set $\mathcal{S}_1$, we change the parameters to $r = 0.8$ and $a = 0.1$. Then, we have a new set of parameters: $\mathcal{S}_5 := \{\beta = 0.0107, \gamma = 1.0387, a = 0.1, b = 0.0131, r = 0.8, \alpha = 0.05, c_s = 0.2, c_p =$



(a)

(b)

Fig. 8: Solution behavior of system (15) when the parameter set $\mathcal{S}_4$ and the initial values $B_{s,0} = 5$, $B_{i,0} = P_0 = 1$ are used. The eigenvalues are $\lambda_1 = -1.0352$, $\lambda_{2,3} = -0.199 \pm 0.099i$ with the conditions in Theorem 5.5 as $n_1 = 0.40 > 0$ and $n_2 = 0.049 > 0$. The solution is locally asymptotically stable at the equilibrium point $\hat{\mathcal{E}}(0.317, 0, 0.199)$: (a) Time series solutions of B_s, B_i, and P, (b) Phase plot of B_s, B_i, and P.

$0.1, \mu = 0.123, K_p = 0.1\}$. Using the parameter set $\mathcal{S}_5$ with the initial values $B_{s,0} = 5$, $B_{i,0} = P_0 = 1$, the simulation of (15) for $\mathcal{E}^*$ shown in Figure 9 converges to $\mathcal{E}^*$ as proved in Theorem 5.6. Note that the parameter values in $\mathcal{S}_5$ give eigenvalues of the Jacobian as

$$\lambda_1 = -0.889, \quad \text{and} \quad \lambda_{2,3} = -0.049 \pm 0.702i. \quad (50)$$

Since all real parts are negative, $\mathcal{E}^*$ is locally asymptotically convergent. Also, the Routh-Hurwitz conditions given in the caption of Figure 9 are satisfied.

According to parameter set $\mathcal{S}_5$, we obtained the bifurcation point $\beta^* = 0.108$ satisfying the conditions presented in Theorem 5.7 as

$$\begin{aligned} m_2(\beta^*) &= 0.006 > 0, \quad m_3(\beta^*) = 0.61 > 0, \\ \mathcal{H}(\beta^*) &= 0, \quad \left.\frac{d\mathcal{H}(\beta)}{d\beta}\right|_{\beta=\beta^*} = 0.0015 \neq 0. \end{aligned} \quad (51)$$

In addition, for $\beta = \beta^* = 0.108$, we obtain eigenvalues

$$\lambda_1 = -0.066, \quad \text{and} \quad \lambda_{2,3} = 0 \pm 0.781i. \quad (52)$$

In Figure 9, we used $\beta = 0.0107$ in parameter set $\mathcal{S}_5$. Therefore, Figure 9 presents the case of $\beta < \beta^*$. Next, we choose $\beta = 0.2$, satisfying $\beta^* < \beta$, to present the instability around the equilibrium point $\mathcal{E}^*$. Using initial values $B_{s,0} = B_{i,0} = 10$, $P_0 = 0.1$, parameter set $\mathcal{S}_5$ and $\beta = 0.2$, we obtain the following eigenvalues:

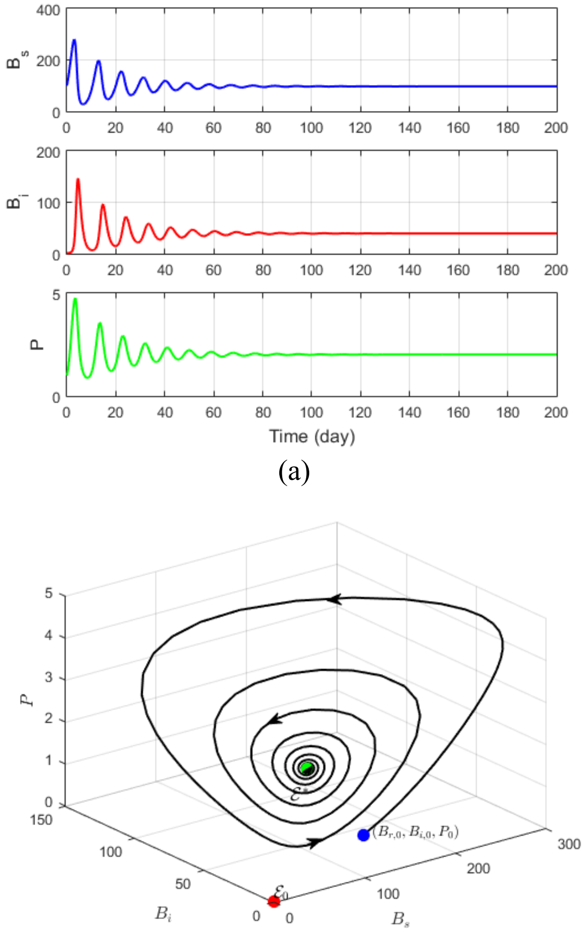
$$\lambda_1 = -0.013, \text{ and } \lambda_{2,3} = 7 \times 10^{-6} \pm 0.78i, \quad (53)$$

with the conditions in Theorem 5.6 as $m_1 = 0.01 > 0$, $m_2 = 0.62 > 0$, $m_3 = 0.008 > 0$, but $m_1 m_2 - m_3 = -8 \times 10^{-6} < 0$. Therefore, the solution is unstable at the equilibrium point $\mathcal{E}^*(5.19, 2.98, 0.026)$ as shown in Figure 10.

7 Discussion and Conclusions

In this article, we present a mathematical model of BPH, with IPM strategies consisting of biological control and chemical control. We used two types of biological control: a natural enemy of BPH and an entomopathogenic fungus. Specifically, *Cyrtorhinus lividipennis* is considered to be a natural enemy of BPH, and an entomopathogenic fungus is studied as *Metarhizium* fungus. In addition, we add a chemical control in our model.

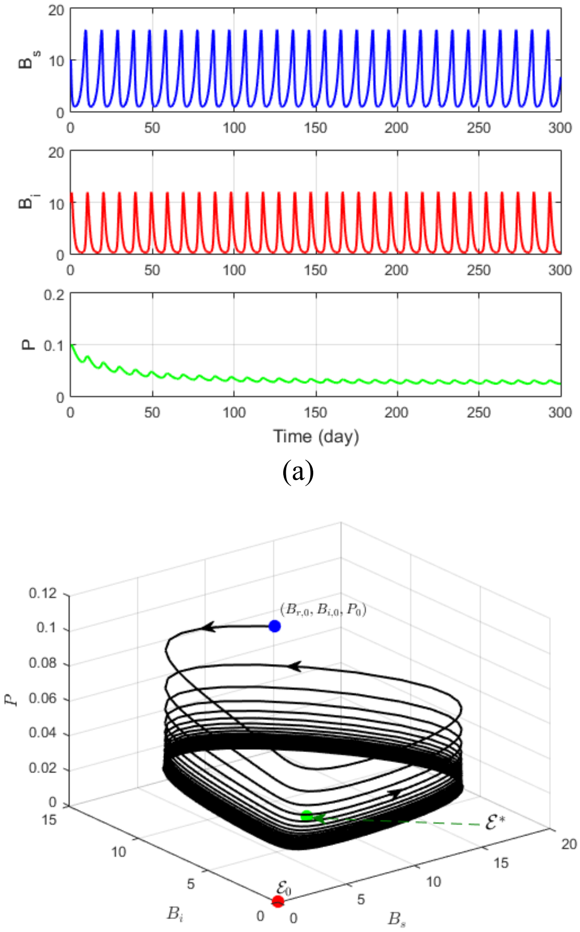
Under assumptions [H1]-[H10] in Section 2, we constructed a model that includes the IPM strategy shown in (1). We defined the BPH population into two groups: the susceptible group B_s and the infected group B_i, while the predator population



(a)

(b)

Fig. 9: Solution behavior of system (15) when the parameter set $\mathcal{S}_5$, the initial values $B_{s,0} = 100$, $B_{i,0} = P_0 = 1$ are used. The eigenvalues are $\lambda_1 = -0.889$, $\lambda_{2,3} = -0.049 \pm 0.702i$ with the conditions in Theorem 5.6 as $m_1 = 0.989 > 0$, $m_2 = 0.585 > 0$, $m_3 = 0.441 > 0$, and $m_1 m_2 - m_3 = 0.137 > 0$. Additionally, this figure simultaneously presents in the case of $\beta < \beta^*$. Therefore, solutions is locally asymptotically stable at the equilibrium point $\mathcal{E}^*(97.07, 39.33, 2.01)$: (a) Time series solutions of B_s, B_i, and P, (b) Phase plot of B_s, B_i, and P.



(a)

(b)

Fig. 10: Solution behavior of system (15) when $\beta^* < \beta$ with the initial values $B_{s,0} = B_{i,0} = 10$, $P_0 = 0.1$ are used. The obtained solution is unstable at the equilibrium point $\mathcal{E}^*(5.19, 2.98, 0.026)$: (a) Time series solutions of B_s, B_i, and P, (b) Phase plot of B_s, B_i, and P.

was represented by the variable P. To observe the behavior of severe BPH outbreaks, we assumed that the BPH population increases exponentially, as defined by the term rB_s, meaning that in the early stages of an outbreak, BPHs can increase their population exponentially without limitations from food or space as assumed in assumption [H1]. Furthermore, to observe long-term IPM control, we disregarded the carrying capacity variable of the BPH population. There is no clear evidence that infected individuals with *Metarhizium* can return to being susceptible BPH again. *Metrhizium* is a fungus that targets specific insect species and has no effect on *Crytorrhinus lividipennis*. We then used the assumptions [H2]-[H7] to construct a model for the epidemic ability of *Metarhizium*. Also, because *Crytorrhinus lividipennis* can eat other small insects in the ecosystem, we assumed [H8] and [H9]. The chemical effect was added with the assumption [H10] that the severity of a chemical depends on the population density. Therefore, the effect of chemical control on BPH and its predator were represented by $c_s B_s$ and $c_p P$ respectively. However, we neglect to analyze the stability of system (1), instead, we define a functional response before analyzing the stability of the system.

In Section 3 we defined the functional response function, which is an important function in studying the dynamics of predator-prey systems. Functional response has been studied to estimate the predator's hunting ability on prey density. Functional responses have been used in entomological research to measure the ability of predators to kill insects under various conditions. Six functions presented in subsection 3.1, Holling's type I, Holling's type II, Holling's type III, Holling's type IV, Roger's type, and Ivlev's type, were fitted to data. Adult *Crytorrhinus lividipennis* was able to consume significantly more BPH eggs than nymphs *Crytorrhinus lividipennis*. Therefore, we were only interested in adult *Crytorrhinus lividipennis* that have important implications for controlling BPH populations with the data reported in [7]. We found that Holling's type II functional response best fits the data with attack rate $a = 0.9009$ and handling time $b = 0.0131$ as shown in Table 1. Thus, we defined $f(B_s) = aB_s/(1+abB_s)$ in system (1), and we obtained the system (15).

An SIR model was developed in Section 4 to evaluate fungal performance. We were able to calculate the $\mathcal{R}_0$ value of an entomopathogenic fungus, *Metarhizium neoanisopliae*, as shown in Table 2. This shows that the use of *M. neoanisopliae* fungus under laboratory conditions cannot cause the BPH population to disappear from the system. This study indicates that using fungus alone can reduce the number of BPHs but cannot stop BPH infestation in the long term, especially when the BPH population has increased exponentially or through immigration. Therefore, in the following section of the study, we examined the model (15) that incorporates IPM in Section 5.

The results from the analysis of model with IPM (15) show that the system has positivity and boundeness as proved in Theorem 5.1. The details of equilibrium points and their stability have been presented in subsections 5.2 and 5.3 respectively. Moreover, numerical results were used to illustrate the theoretical results presented in Section 6.

The equilibrium point $\mathcal{E}_0(0,0,0)$ indicates that when BPH growth rate is less than the insecticide efficiency ($r < c_s$), then BPH disappears from the system. By Theorem 5.2, if the insecticide is very effective, it can also cause *Cyrtorhinus lividipennis* to become extinct ($\alpha < c_p$).

Cyrtorhinus lividipennis, which is the main natural enemy of BPH, can eat other small insects. With sufficient food reserves, its population can survive, which will be beneficial for managing the next generation of BPHs. According to the existence condition for $\bar{\mathcal{E}}(0, 0, \frac{K_p}{\alpha}(\alpha - c_p))$, $\alpha > c_p$, and the stability conditions in Theory 5.3, we found that the equilibrium point is $\bar{\mathcal{E}}$, which means that BPH becomes extinct when a chemical and *Cyrtorhinus lividipennis* with sufficient food reserves are used simultaneously.

On the other hand, if the chemical has enough effect on *Cyrtorhinus lividipennis* ($\alpha + \mu a\gamma/(ab\gamma + \beta) < c_p$) but has little effect on the BPH ($r > c_s$), it may cause the BPH to remain in the system but *Cyrtorhinus lividipennis* to disappear as was the case proved in Theorem 5.4 for the predator free equilibrium $\tilde{\mathcal{E}}(\frac{\gamma}{\beta}, \frac{r-c_s}{\beta}, 0)$.

It was found that the infection free equilibrium $\hat{\mathcal{E}}\left(\hat{B}_s, 0, \hat{P}\right)$ can occur at two points simultaneously when the conditions in equations (22), (25), and (26) are satisfied. Additionally, the stability condition was proved in Theorem 5.5.

For the interior equilibrium $\mathcal{E}^*(B_s^*, B_i^*, P^*)$, we showed that a Hopf bifurcation could occur that satisfied the conditions in Theorem 5.7 at $\beta = \beta^*$. If $\beta < \beta^*$, the system is locally asymptotically stable at $\mathcal{E}^*$ with the conditions in Theorem 5.6. Otherwise, the system is unstable at $\mathcal{E}^*$ if $\beta^* < \beta$. These results also follow from the changing of the real part of eigenvalues $\lambda_{2,3}$ as shown in Equations (50), (52), and (53).

For some initial values, the equilibrium points $\tilde{\mathcal{E}}$ and $\mathcal{E}^*$ indicate that the population of susceptible BPHs depends on the severity of the fungus as $B_s \to \gamma/\beta$ as $t \to \infty$. Therefore, under some conditions, both infected and susceptible groups can coexist in the system with the number of susceptible BPHs remaining over time depending on the severity parameters of infection with the fungus β, and γ.

Therefore, from the results of this study, we recommend the use of an IPM control method that combines fungi, *Cyrtorhinus lividipennis*, and a chemical pesticide as a means of quickly eradicating insects in the early stages of an outbreak and of possibly causing BPHs to become extinct in the long term. The important factor that reduces pests is the use of chemicals. The severity of a chemical has a great effect on the model we study. If the chemicals affect *Cyrtorhinus lividipennis*, it could cause them to become extinct as well. However, without the use of chemicals, the population of *Cyrtorhinus lividipennis* must be large enough to cause long-term extinction. In addition, the use of pathogenic fungus also has long-term effects on the system. In cases where we want to avoid the effects of chemicals as much as possible, fungi and insect predators should be applied together to eliminate insect pests.

In fact, there are many factors affecting BPH populations, such as species of rice, migration, temperature, humidity, other insect predators, habitat complexity, etc. In the future, we intend to include some of these factors in the model to be more realistic. For example, using other insect predators such as *Paederus fuscifes* and wolf spider *Pardosa pseudoannulata*. Using other kinds of fungus such as *Beauveria bassiana* and *Metarhizium* spp., including specifying the specific type of chemical. Additionally, pest management may be achieved through the repeated release of chemical or fungal pesticides and natural enemies of BPH, which can lead to a model that incorporates an impulsive model to observe periodic pest eradication. In addition, the model presented in this work can be further improved to the stochastic differential model, [72], and the delay-differential model, [73]. Ultimately, we hope that this research will provide a guide for the analysis and control of BPH outbreaks in rice fields.

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The authors wrote, reviewed and edited the content as needed and have not utilized artificial intelligence (AI) tools. The authors take full responsibility for the content of the publication.

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H.S., S.S., S.K., and W.J. conceived and supervised the study. N.K., H.S., N.T., and S.S. conducted the mathematical analysis. N.K., H.S., N.T., S.S., S.K., and W.J. conducted the writing of the original manuscript, reviewing and editing of the revised manuscript. The final manuscript was approved by all authors.

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Data Availability

The entire data collected for this study were analyzed, interpreted, and incorporated into this article.

Ethical Approval

The ethical approval for animal care and use for scientific research in this study was obtained by the Ethical Review Committee of the National Center for Genetic Engineering and Biotechnology, Thailand, license number U1-08414-2562.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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