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Mathematical Model on the Dynamics of Bacterial Blight of Rice in the Presence of *Lysobacter antibioticus* Considering Introduction at Different Stages

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Abstract

In this research we formulated the Plants diseases model with the aim of studying the dynamics of the use of *lysobacter antibioticus* for prevention and control of rice bacterial blight. The disease free equilibrium state of the models was also obtained by equating each of the equation of the modified model to zero and simplifying. The basic reproduction number for the model was derived using the next generation matrix approach. Numerical simulation was carried out using MATLAB2018a to virtualize the dynamics of the model. Five numerical experiment was carried out and it was shown that biocontrol help to reduce the population of the pathogen as well as act as treatment for those that are already exposed or infected with the disease. It was also observed that the biocontrol agent provide immunity to rice plants against been infected with the disease. Finally, we observed from the simulation that the earlier the control is introduced the more protection plants will receive.

Keywords: Biocontrol; Lysobacter antibioticus; mathematical model; bacterial blight of rice.

1 Introduction

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Plant disease losses, in addition to hunger and malnutrition, have a huge financial consequence when an epidemic breakout occurs [1]. Plant disease losses have been reduced as a result of the introduction and implementation of disease control techniques. Plant disease control has historically relied on the use of pesticides like fungicides and insecticides on a regular basis. This is due to the fact that pesticides are efficient against a wide range of illnesses and increase agricultural yields. Even though pesticides can be advantageous, there will still be short and long-term risks that can harm the environment and endanger human health, such as cases of acute poisoning, including suicide attempts, mass toxicity from food contamination, chemical mishaps in company, and workers exposed in agriculture, these are the most severe health problems associated with pesticides used for agricultural purposes.

Rice bacterial blight, often known as bacterial blight of rice (BB), is a lethal bacterial disease that is one of the most damaging illnesses of farmed rice (*Oryza sativa* and *O. glaberrima*). Crop losses can reach 75% in extreme outbreaks, and huge areas of rice are afflicted each year. The disease was initially noticed in Kyushu, Japan, in 1884–85, and the pathogenic agent, Xanthomonas oryzae pathovar oryzae (also known as Xoo), was discovered in 1911, when it was still known as Bacillus oryzae. Bacterial blight has been found in rice-growing countries of Asia, the western coast of Africa, Australia, Latin America, and the Caribbean, where it thrives in warm, humid settings [3].

BB is a vascular disorder that causes a chronic infection in the veins, resulting in tannish-grey to white lesions. At the tillering stage, symptoms appear, and disease incidence rises with plant growth, peaking at the flowering period. The more damaging form of the disease, Kresek, causes the entire plant's leaves to turn pale yellow and wilt from seedling to early tillering, resulting in a partial or complete crop failure. The most vulnerable plants are those that are less than 21 days old, and temperatures between 28 and 34°C are ideal for kresek development. Yellow lesions with wavy borders on leaf blades that may extend to the Sheath are indicative of BB. Over time, these lesions will turn a white straw color. In warm and humid settings, a bacterial oozing from affected leaves has been noticed, which adds to the spread of the disease. Though leaf blight can develop at any stage of growth, it is most common between maximal tillering and substantial maturity damage, when kresek is present [4].

Post-flowering infections, such as BB, have a negligible impact on grain output. When bacteria invade during panicle commencement or later phases before to flowering, however, grain growth is severely hampered, resulting in higher in infertility. Bacterial blight manifests itself as water-soaked streaks that stretch from the leaf tips and edges, gradually widening and releasing milky liquid that dries into yellow droplets. The conclusion of the illness is signaled by the appearance of typical grayish white lesions on the leaves as they dry out and die. Seedling leaves become dry and wilty, a condition known as kresek. Diseased seedlings usually die between two to three weeks of disease; adult plants may survive, but yield and quality will be minimal [5].

To limit the harm caused by BB, several disease management strategies like chemical control, host-plant resistance, cropping system modification, and biocontrol have been adopted. Chemical control and host plant resistance, two of the most commonly used management techniques, both have drawbacks. Chemical pesticides are harmful to the environment, and host-plant resistance based on a single gene may not be long-lasting in the field, resulting in repeated resistance breakdowns. Environmentally friendly and long-term control techniques must be developed. Biocontrol is an environmentally friendly, cost-effective, and long-term alternative to BB management. This strategy can also be combined with other management techniques to provide additional protection and maintain rice harvests. Antibiotic microorganisms are thought to be great biocontrol agents because of their evident benefits. They are quiet easy to manage and they grow at a very fast rate to occupy rhizosphere aggressively [6].

However, there are a variety of options for reducing pesticide use, including cultural control, host plant resistance, and biological control (biocontrol). Biocontrols have gotten a lot of press in the previous 30 years. Induced resistance (IR) is a technique that targets the use of plant defensive systems as well as the introduction of biological adversaries to prey to compete and produce a favorable environment for diseases [2].

Because of the great diversity of the pathogen population in its susceptibility to chemicals utilized for control, as well as the detrimental consequences of the chemicals on both humans and the environment, effective chemical control for the management of rice BB has yet to be created. As a result, biocontrol is required, which appears to

be an environmentally benign and cost-effective answer to the major danger to rice agriculture [7]. A number of research on the use of biocontrol for rice BB have been conducted, including one by Guang-Hai et al in [4] discovered that a novel *Lysobacter antibioticus* strain can be employed as a biocontrol agent. However, understanding the dynamics of the interaction between the biocontrol agent (*Lysobacter antibioticus*), the rice plants, and the BB pathogen has received little attention. Hence we develop a mathematical model to study the dynamics of the interaction between biocontrol agent (*Lysobacter antibioticus*), the rice plants, and the BB pathogen.

1.1 Mathematical models

Mathematical modeling is a useful technique for analyzing agricultural issues. The use of mathematical theory to solve the problem provides for a qualitative and quantitative assessment of the problem. The number of research of mathematical modeling applied to the biocontrol system is rapidly increasing nowadays. To have a deeper knowledge of the biocontrol system's dynamical system, one must look at the interactions within the system and comprehend the mechanisms involved.

In order to compare the effects of different models for the effect of host responses to the load of infection on the production of susceptible tissue, Gilligan, Gubbins, and Simons in [8] reformulated a model for botanical epidemics into a SIR form for susceptible (S), infected (I), and removed (R) plant organs. The effects of initial (particulate) inoculum density on the dynamics of disease resulting from primary and secondary infection of wheat by the take-all fungus Gaeumannomyces graminis var. tritici were investigated by Bailey and Gilligan in [9] using a combination of experimentation and mathematical modeling. The dynamical features of models for plant epidemics, particularly soil-borne fungal infection, were examined by [10]. Anggriani, Arumi, Hertini, Istifadah, and Supriatna in [11] Incorporated roguing and replanting with preventive treatment into a mathematical model of plant disease. Anggriani, Arumi, Hertini, Istifadah, and Supriatna in [12] created a deterministic mathematical model in which the plant population is divided into five compartments: Susceptible, Exposed, Infected, Post-Infectious (Removed), and Protected. They also included preventive and curative treatments in the model and analyzed their effects on plant disease transmission dynamics. The equilibrium points and epidemic threshold conditions are calculated analytically. To back up the analytical results, several numerical simulations were provided. They discovered that combining preventive and curative therapies can help reduce the number of diseased plants. Pathak and Maiti in [13] investigated the dynamical behavior of a model for pest biocontrol in which the pest is supposed to be infected with a virus. The model's boundedness and stability are investigated. The impact of time-delay is examined. To highlight their analytical findings, they use numerical simulations. The time delay is observed to have a regulatory impact on the system. Few biocontrol researchers, including Xiao and Van den Bosch in [14] consider that the pest to be managed frequently also infests wild plant species. A model for the effect of a wild host plant species on biologically-based pest control (BBTs) is established and examined in their research. The pest species in question not only feeds on the crop, but it also has a wild host. The model equations are mathematically examined in terms of non-negativity invariance, boundedness of solutions, nature of equilibria, permanence, and global stability. According to Xu, Salama, Jeries, and Jeger in [15], a previously published general mathematic model was employed in a numerical analysis to better understand the dynamics of foliar pathogens in connection to processes, as well as the timing and coverage of biocontrol agent (BCA) applications. It was demonstrated that a BCA with either competition or induced resistance as its principal mechanism of biocontrol was more effective in lowering disease development than a BCA with either mycoparasitism or antibiosis as its mechanism using the model parameter values utilized [15].

The rest of the paper is organized as follows: Section 2 the model was formulated and analysed. Section 3 the numerical simulation is carried out and in Section 4 the results were discussed and conclusion drawn.

2 Model Formation and Analysis

2.1 Assumption for the model

i. The plants can be exposed to the pathogen at two stages. When the plant is less than 3 weeks old and when the plant is older than 3 weeks.
ii. Young and matured exposed plants can recover with temporary protection against the pathogen.
iii. Infected matured plants can recover with temporary protection against the pathogen.
iv. There is interaction of the young and matured plants with the pathogen.

v. The biocontrol agents interact with the pathogens, the rice plant.
vi. Susceptible young plant grows to become susceptible matured plant if not exposed to the pathogen.
vii. The disease pathogens are already in the environment as residue from harvest and existing in some wild host.
viii. The rice population is not constant.
ix. Natural death occurs in each compartment.
x. The biocontrol agents release lytic enzymes and antibiotics that suppresses and kill the pathogen.
xi. The biocontrol agents does not on its own become a pathogen for another disease.
xii. Young infected plants cannot recover from the disease once infected.
xiii. Biocontrol agents continues to multiply after application
xiv. The preventive treatment (*Lysobacter antibioticus*) is applied to the young and matured plant.
xv. Susceptible plants that receive preventive treatment enters protected compartment. P_r
xvi. Protected plants $P_r(t)$ have protection from the biocontrol agents but they are not immune to the disease, thus they can loss there protection and become susceptible naturally.
xvii. Other form of control like the use of insecticides is not considered
xviii. There is no rogueing of infected plants.

2.2 Description of the modified model

In this model, we divide the plant population $N(t)$ into 10 compartments namely: $S_y(t)$, the number of plants Susceptible to the pathogen and less than 21 days old; $S_m(t)$, the number of plants susceptible to the pathogen and greater than 21 days old; $E_y(t)$, the number of plants exposed to the pathogen and less than 21 days old; $S_m(t)$, the number of plants susceptible to the pathogen and greater than 21 days old; $E_y(t)$, the number of young plants exposed to the pathogen; $E_m(t)$, the number of matured plants exposed to the pathogen; $K(t)$, the number of young plants infected by pathogen leading to Kresek phase of BB; $L(t)$, the number of matured plants infected by the pathogen leading to the bacterial blight phase of BB; $R_y(t)$, the number of recovered Young plants; $R_m(t)$, the number of recovered matured plants; $P_{ry}(t)$, the number of young plants protected by bio control agent, $P_{rm}(t)$, the number of matured plants protected by biocontrol agent. While $B(t)$, is the population of biocontrol agents and $\mathrm{P}(t)$, is the population of pathogen *Xanthomonas oryzae pathovar oryzae*.

The total population of the plant N(t) is increasing with time because planting is continuous throughout the year. There is natural death and disease induced death. The total rice pollution is assumed to be susceptible at the initial stage before the application of the biocontrol agent to the farm. The susceptible young rice plants $S_y(t)$ after interaction with the biocontrol agent acquire protection from the agent and moved to $P_{ry}(t)$ compartment at the rate ι_y and subsequently move to $P_{rm}(t)$. $S_y(t)$ that fails to acquire protection when they interact with the biocontrol agents become exposed to the disease when they interact with the pathogen at the rate ϕ_y and those that do not acquire protection and also do not become infected with the disease after approximately 21 days will move to the $S_m(t)$ at the rate γ.

The exposed young rice plants $E_y(t)$ either move to the infected compartment $K(t)$ which is the kresek phase after 10 to 15 days of being exposed at a rate ω_y or have an interaction with the biocontrol agent and moved to the recovered young rice plant $R_y(t)$ at the rate α_y. Young rice plants after recovering from the exposure to the disease moved into the susceptible matured plant compartment S_m at the rate φ_y because it is assumed that before the cycle of exposure and recovery to be complete the rice plants should be more than 21 days old. It is assumed that susceptible young plant $S_y(t)$ once infected cannot recover from the disease but will die due to the disease or die naturally.

Susceptible matured plant $S_m(t)$ after interacting with biocontrol agents acquire protection at the rate χ and those that do not acquire the protection if they interact with the disease pathogen becomes exposed to the disease at the rate of ϕ_m. $E_m(t)$ either interact with the biocontrol agents *Lysobacter antibioticus* to recover and move to the recovered compartment at the rate α_m or move to the infected compartment at the rate ω_m. $L(t)$ the infected rice plants can die from the disease or interact with the biocontrol agents *Lysobacter antibioticus* to recover and move to the recovered compartment at the rate ρ. The recovered matured plants R_m becomes susceptible matured plants $S_m(t)$ at the rate φ_m.

The protected rice plants losses its protection naturally after sometime due to environmental factors and other weather conditions to become susceptible matured rice plants. The biocontrol agents *Lysobacter antibioticus*

and the pathogen *Xanthomonas oryzae pathovar oryzae* exhibit a form of a logistic growth and also die out naturally. The recruitment into the pathogen compartment at a rate g and that of the biocontrol agent is n. The whole plants population compartment has natural death rate of δ. r is replanting rate of rice seedlings, ϕ_y Rate of exposure of young susceptible plants to the pathogen, ϕ_m Rate of exposure of matured susceptible plants to the pathogen, θ_y is the rate of exposure of young susceptible plants to the pathogen as result of interactions with infected young plants, θ_m is the rate of exposure of matured susceptible plants to the pathogen as result of interactions with infected matured plants, ω_y is the rate at which exposed young plants get infected, ω_m is the rate at which exposed matured plants becomes infected, α_y is rate of recovery of exposed young plants as a result of interaction between the pathogen and the bio control agent, $\alpha_m$is the rate of recovery of exposed matured plants as a result of interaction between the pathogen and the bio control agent, φ_y is the rate at which the recovered young plants losses it temporary protection acquired from the bio control agent and become susceptible, φ_m is the rate at which the recovered matured plants losses it temporary protection acquired from the biocontrol agent and become susceptible, ι_y is the rate at which young plants acquire protection from bio control agents, ι_m is the rate at which matured plants acquire protection from bio control agents, χ is the rate at which protected matured rice plants loss it protection, χ_y is the rate at which protected young rice plants becomes protected adult plants, γ is the rate at which susceptible young plants grow to become susceptible matured plants, ρ is recovery rate of infected matured plants, β is the growth rate of the pathogen, ψ growth rate of the biocontrol agents, λ is the natural death rate of the pathogen, η is the natural death rate of the biocontrol agents, δ is the natural death rate of the rice plants, μ_y is the death rate of young plant due to disease infection, μ_m is the death rate of matured plant due to disease infection, μ_p is the death rate of the pathogen due to the activities of the biocontrol agent, ϖ is the carrying capacity of the population of biocontrol agents, q is the carrying capacity of the population of the pathogen, $g$is the application rate of biocontrol agent, n is the recruitment rate if disease pathogen

2.3 Modified model equations

$$\frac{dS_y}{dt} = r(C - N) - \Delta_y S_y - \gamma S_y - \delta S_y - \iota_y S_y B \tag{2.1}$$

$$\frac{dS_m}{dt} = \gamma S_y + \varphi_y R_y + \varphi_m R_m - \Delta_m S_m - \delta S_m + \chi P_{rm} - \iota_m S_m B \tag{2.2}$$

$$\frac{dE_y}{dt} = \Delta_y S_y - \alpha_y E_y B - \omega_y E_y - \delta E_y \tag{2.3}$$

$$\frac{dE_m}{dt} = \Delta_m S_m - \alpha_m E_m B - \omega_m E_m - \delta E_m \tag{2.4}$$

$$\frac{dK}{dt} = \omega_y E_y - (\delta + \mu_y)K \tag{2.5}$$

$$\frac{dL}{dt} = \omega_m E_m - \rho L B - (\delta + \mu_m)L \tag{2.6}$$

$$\frac{dR_y}{dt} = \alpha_y E_y B - \varphi_y R_y - \delta R_y \tag{2.7}$$

$$\frac{dR_m}{dt} = \alpha_m E_m B + \rho L B - \varphi_m R_m - \delta R_m \tag{2.8}$$

$$\frac{dP_{ry}}{dt} = \iota_y S_y B - \chi_y P_{ry} - \delta P_{ry} \tag{2.9}$$

$$\frac{dP_{rm}}{dt} = \iota_m S_m B + \chi_y P_{ry} - \chi P_{rm} - \delta P_{rm} \tag{2.10}$$

$$\frac{dP}{dt} = gP + \beta P\left(1 - \frac{P}{q}\right) - (\lambda + \mu_p B)P \tag{2.11}$$

$$\frac{dB}{dt} = nB + \psi B\left(1 - \frac{B}{\varpi}\right) - \eta B \tag{2.12}$$

Where $\Delta_y = (\theta_y K + \phi_y P)$, $\Delta_m = (\theta_m L + \phi_m P)$

And the following parameters are defined in terms of t. The functions are selected base of the behavior of the function at every given time as described in section 2.2.

$$r(t) = \begin{cases} r & \text{if } t \leq 3 \\ 0 & \text{if } t > 3 \end{cases}, \quad \chi_y(t) = \begin{cases} \chi_y & \text{if } t \geq 3 \\ 1 & \text{if } t > 6 \end{cases}, \quad \varphi_y(t) = \begin{cases} \varphi_y & \text{if } t \geq 3 \\ 1 & \text{if } t > 6 \end{cases},$$

$$\gamma(t) = \begin{cases} \gamma & \text{if } t \geq 3 \\ 1 & \text{if } t > 6 \end{cases}$$

2.4 Existence of solution

If $f(t,x)$ has continuous partial derivatives $\frac{\partial f_i}{\partial x_j}$ on a bounded closed convex domain R (i.e convex set of real numbers), where R is used to denote real numbers, then it satisfies a Lipschitz condition in R. Our interest is in the domain:

$$1 \leq \epsilon \leq R$$

So we look for a bounded solution of the form $0 < \epsilon < \infty$.

To establish the existence and uniqueness of the equations

Let R^n denote the domain

$$|t - t_0| \leq a, \|x - x_0\| \leq b, x = (x_1, x_2, \dots, x_n) \tag{2.4.1}$$

Suppose $f(t,x)$ satisfies the Lipschitz condition

$$\|f(t, x_1) - f(t, x_2)\| \leq k\|x_1 - x_2\| \tag{2.4.2}$$

Let D denote the domain defined in (2.4.1) such that (2.4.2) and Lipschitz condition hold. Then there exist a solution of model system of equation (2.1)-(2.12) which is bounded in the domain D.

Proof:

Let equation (2.1)-(2.12) be equal to $f_1 - f_{12}$ respectively. We show that: $\frac{\partial f_i}{\partial x_j}, i, j = 1,2,3, \dots 12$ are continuous and bounded. That is the partial derivatives are continuous and bounded. We explore the following partial derivatives for all the model equations.

From equation (3.2.1) we have

$$f_1 = r(C - N) - (\theta_y K + \phi_y P) S_y - \gamma S_y - \delta S_y - \iota_y S_y B$$

$$\frac{\partial f_1}{\partial s_y} = -\theta_y K - \phi_y P - \gamma - \delta - \iota_y B$$

$$\left|\frac{\partial f_1}{\partial s_y}\right| = \left|-\theta_y K - \phi_y P - \gamma - \delta - \iota_y B\right| < \infty$$

$$\frac{\partial f_1}{\partial s_m} = 0, \left|\frac{\partial f_1}{\partial s_m}\right| = |0| < \infty$$

$$\frac{\partial f_1}{\partial E_y} = 0, \left|\frac{\partial f_1}{\partial E_y}\right| = |0| < \infty$$

$$\frac{\partial f_1}{\partial E_m} = 0, \left|\frac{\partial f_1}{\partial E_m}\right| = |0| < \infty$$

$$\frac{\partial f_1}{\partial K} = 0, \left|\frac{\partial f_1}{\partial K}\right| = |0| < \infty$$

$$\frac{\partial f_1}{\partial L} = 0, \left|\frac{\partial f_1}{\partial L}\right| = |0| < \infty$$

$$\frac{\partial f_1}{\partial R_y} = 0, \left|\frac{\partial f_1}{\partial R_y}\right| = |0| < \infty$$

$$\frac{\partial f_1}{\partial R_m} = 0, \left|\frac{\partial f_1}{\partial R_m}\right| = |0| < \infty$$

$$\frac{\partial f_1}{\partial P_{ry}} = 0, \left|\frac{\partial f_1}{\partial P_{ry}}\right| = |0| < \infty$$

$$\frac{\partial f_1}{\partial P_{rm}} = 0, \left|\frac{\partial f_1}{\partial P_{rm}}\right| = |0| < \infty$$

$$\frac{\partial f_1}{\partial P} = -\phi_y S_y, \left|\frac{\partial f_1}{\partial P}\right| = \left|-\phi_y S_y\right| < \infty$$

$$\frac{\partial f_1}{\partial B} = -\iota_y S_y, \left|\frac{\partial f_1}{\partial B}\right| = \left|-\iota_y S_y\right| < \infty$$

We established that all the partial derivatives are continuous and bounded; hence by Theorem I we can say that there exists a unique solution of (3.2.1-3.2.12) in the region D.

2.5 Disease free equilibrium point

The total plant population at every given time is N

$$N = S_y + S_m + E_y + E_m + L + K + R_y + R_m + P_{ry} + P_{rm} \tag{2.13}$$

At disease free equilibrium state they can be no death due to the disease.

Therefore, substituting and simplifying we have $N = \frac{rC}{r+\delta}$ (2.14)

At disease free equilibrium (DFEP) we have

$$\frac{dS_y}{dt} = \frac{dS_m}{dt} = \frac{dE_y}{dt} = \frac{dE_m}{dt} = \frac{dK}{dt} = \frac{dL}{dt} = \frac{dR_y}{dt} = \frac{dR_m}{dt} = \frac{dP_r}{dt} = \frac{dP}{dt} = \frac{dB}{dt} = 0 \tag{2.15}$$

Substituting equation (2.1) – (2.12) into (2.15) an simplifying we have DFEP as

$$E_0 = \left(\frac{r\delta C}{(\gamma+\delta)(r+\delta)}, \frac{\gamma r\delta C}{\delta(\gamma+\delta)(r+\delta)}, 0,0,0,0,0,0,0,0,0\right)$$

2.6 Reproduction number for rice plants

To derive the reproduction number for the rice plant we used the next generation matrix as used by (Namawejje, 2011) and we considered equations (2.1) to (2.12). From the outline equations above new infections arise in (2.3) and (2.4) as a result of the interaction between the pathogen, the infected matured plant, infected young plants, susceptible matured plants and susceptible young plants.

$$f = \begin{bmatrix} (\theta_y K + \phi_y P)S_y \\ (\theta_m L + \phi_m P)S_m \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{2.16}$$

$$v = -\begin{bmatrix} -\alpha_y E_y B - \omega_y E_y - \delta E_y \\ -\alpha_m E_m B - \omega_m E_m - \delta E_m \\ \omega_y E_y - (\delta + \mu_y)K \\ \omega_m E_m - \rho L B - (\delta + \mu_m)L \\ g + \beta P\left(1 - \frac{P}{q}\right) - (\lambda + \mu_p B)P \end{bmatrix}$$

$$v = \begin{bmatrix} \alpha_y E_y B + \omega_y E_y + \delta E_y \\ \alpha_m E_m B + \omega_m E_m + \delta E_m \\ -\omega_y E_y + (\delta + \mu_y)K \\ -\omega_m E_m + \rho L B + (\delta + \mu_m)L \\ -g - \beta P\left(1 - \frac{P}{q}\right) + (\lambda + \mu_p B)P \end{bmatrix} \tag{2.17}$$

$F = \frac{\partial f_i(D_0)}{\partial x_i}$ and $V = \frac{\partial V_i(D_0)}{\partial x_i}$. The corresponding Jacobian matrices of F and V are the matrices of the derivatives of f_i and V_i with respect $E_m, L\ and\ P$ at disease free equilibrium point.

$$FV^{-1} = \begin{bmatrix} Y & 0 & \frac{S_y{}^*\theta_y}{(\delta+\mu_y)} & 0 & -\frac{S_y{}^*\phi_y}{n+\beta-\lambda-\mu_p B} \\ 0 & Z & 0 & \frac{\theta_m S_m{}^*}{(\rho B+\delta+\mu_m)} & -\frac{S_m{}^*\phi_m}{n+\beta-\lambda-\mu_p B} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$Y = \frac{S_y{}^*\theta_y\omega_y}{(\alpha_y B + \omega_y + \delta)(\delta + \mu_y)}$$

$$Z = \frac{S_m{}^*\theta_m\omega_m}{(\alpha_m B + \omega_m + \delta)(\rho B + \delta + \mu_m)}$$

The eigenvalues are

$$\frac{r\delta C\theta_y\omega_y}{\left(\left(\gamma+\delta+\iota_y\left(\frac{\varpi n+\varpi\psi-\varpi\eta}{\psi}\right)\right)(r+\delta)\right)(\alpha_y B+\omega_y+\delta)(\delta+\mu_y)},\ 0, \frac{S_m{}^*\theta_m\omega_m}{(\alpha_m B+\omega_m+\delta)(\rho B+\delta+\mu_m)}$$

Determinant of FV^{-1} is equal to zero so therefore $\mathscr{R}_0 = Trace\ of\ FV^{-1}$.

$$\mathscr{R}_0 = \frac{r\delta C\theta_y\omega_y}{\left(\left(\gamma+\delta+\iota_y\left(\frac{\varpi n+\varpi\psi-\varpi\eta}{\psi}\right)\right)(r+\delta)\right)(\alpha_y B+\omega_y+\delta)(\delta+\mu_y)} + \frac{S_m{}^*\theta_m\omega_m}{(\alpha_m B+\omega_m+\delta)(\rho B+\delta+\mu_m)} \tag{2.18}$$

The reproduction number shows the number of secondary rice bacterial blight cases produced by a single infected rice plant during her life time in the absence of any intervention strategy. The value of $\mathscr{R}_0$ can indicate the circumstance in which an epidemic is possible. When $\mathscr{R}_0 > 1$ then the disease is likely to result in an epidemics hence urgent intervention is needed but if $\mathscr{R}_0 < 1$ then the diseases is likely to die out after some time.

2.7 Local stability at disease free equilibrium point E_1

Theorem:

The disease free state E_1 is locally asymptotically stable if all the eigenvalues are negative

Proof:

The Jacobean matrix of the system (3.2.1) to (3.2.12) evaluated at disease free point is obtained as

$$J(E_1) = \begin{bmatrix} A_1 & 0 & 0 & 0 & A_{11} & 0 & 0 & 0 & 0 & 0 & A_{27} & A_{32} \\ A_2 & A_4 & 0 & 0 & 0 & A_{14} & A_{18} & A_{20} & 0 & A_{24} & A_{28} & A_{33} \\ 0 & 0 & A_6 & 0 & A_{12} & 0 & 0 & 0 & 0 & 0 & A_{29} & 0 \\ 0 & 0 & 0 & A_8 & 0 & A_{15} & 0 & 0 & 0 & 0 & A_{30} & 0 \\ 0 & 0 & A_7 & 0 & A_{13} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & A_9 & 0 & A_{16} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & A_{19} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & A_{10} & 0 & A_{17} & 0 & A_{21} & 0 & 0 & 0 & 0 \\ A_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & A_{22} & 0 & 0 & A_{35} \\ 0 & A_5 & 0 & 0 & 0 & 0 & 0 & 0 & A_{23} & A_{26} & 0 & A_{36} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & A_{31} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & A_{37} \end{bmatrix}$$

$A_1 = -(\gamma + \delta + \iota_y B)$, $A_2 = \gamma$, $A_3 = \iota_y B$, $A_4 = -\delta - \iota_m B$, $A_5 = \iota_m B$, $A_6 = -(\alpha_y B + \omega_y + \delta)$, $A_7 = \omega_y$, $A_8 = -(\alpha_m + \omega_m + \delta)$, $A_9 = \omega_m$, $A_{10} = \alpha_m B$, $A_{11} = -\theta_y S_y$, $A_{12} = \theta_y S_y$, $A_{13} = -(\delta + \mu_y)$, $A_{14} = -\theta_m S_m$, $A_{15} = \theta_m S_m$, $A_{16} = -\rho B - (\delta + \mu_m)$, $A_{17} = \rho B$, $A_{18} = \varphi_y$, $A_{19} = -(\varphi_y + \delta)$, $A_{20} = \varphi_m$, $A_{21} = -(\varphi_m + \delta)$, $A_{22} = -\chi_y - \delta$, $A_{23} = \chi_y$, $A_{24} = \chi$, $A_{26} = -\chi - \delta$, $A_{27} = -\phi_y S_y$, $A_{28} = -\phi_m S_m$, $A_{29} = \phi_y S_y$, $A_{30} = \phi_m S_m$, $A_{31} = g + \beta - \lambda - \mu_p B$, $A_{32} = -\iota_y S_y$, $A_{33} = -\iota_m S_m$, $A_{35} = \iota_y S_y$, $A_{36} = \iota_m S_m$, $A_{37} = n + \psi - \frac{2B}{C} - \eta$

$$\lambda_1 = \tfrac{1}{2}A_6 + \tfrac{1}{2}A_{13} + \tfrac{1}{2}\sqrt{{A_6}^2 - 2A_6A_{13} + {A_{13}}^2 + 4A_7A_{12}}$$

$$\lambda_1 = \tfrac{1}{2}(-\alpha_y B - \omega_y - \delta) + \tfrac{1}{2}(-\delta - \mu_y) + \tfrac{1}{2}\sqrt{(-\alpha_y B - \omega_y - \delta)^2 - 2(-\alpha_y B - \omega_y - \delta)(-\delta - \mu_y) + (-\delta - \mu_y)^2 + 4\omega_y\theta_y S_y}$$

$$\lambda_1 = \tfrac{1}{2}\sqrt{B^2{\alpha_y}^2 - 2B\alpha_y\mu_y + 2B\alpha_y\omega_y + {\mu_y}^2 - 2\mu_y\omega_y + {\omega_y}^2 + 4\omega_y\theta_y S_y} - \tfrac{1}{2}\mu_y - \tfrac{1}{2}\omega_y - \delta - \tfrac{1}{2}B\alpha_y$$

$$\lambda_2 = \frac{1}{2}A_6 + \frac{1}{2}A_{13} - \frac{1}{2}\sqrt{{A_6}^2 - 2A_6A_{13} + {A_{13}}^2 + 4A_7A_{12}}$$

$$\lambda_2 = \tfrac{1}{2}(-\alpha_y B - \omega_y - \delta) + \tfrac{1}{2}(-\delta - \mu_y) - \tfrac{1}{2}\sqrt{(-\alpha_y B - \omega_y - \delta)^2 - 2(-\alpha_y B - \omega_y - \delta)(-\delta - \mu_y) + (-\delta - \mu_y)^2 + 4\omega_y\theta_y S_y}$$

$$\lambda_2 = -\tfrac{1}{2}\sqrt{B^2{\alpha_y}^2 - 2B\alpha_y\mu_y + 2B\alpha_y\omega_y + {\mu_y}^2 - 2\mu_y\omega_y + {\omega_y}^2 + 4\omega_y\theta_y S_y} - \tfrac{1}{2}\mu_y - \tfrac{1}{2}\omega_y - \delta - \tfrac{1}{2}B\alpha_y$$

$$\lambda_3 = \frac{1}{2}A_8 + \frac{1}{2}A_{16} + \frac{1}{2}\sqrt{{A_8}^2 - 2A_8A_{16} + {A_{16}}^2 + 4A_9A_{15}}$$

$$\lambda_3 = \frac{1}{2}(-\alpha_m B - \omega_m - \delta) + \frac{1}{2}(-\rho B - \delta - \mu_m) + \frac{1}{2}\sqrt{(-\alpha_m B - \omega_m - \delta)^2 - 2(-\alpha_m B - \omega_m - \delta)(-\rho B - \delta - \mu_m) + (-\rho B - \delta - \mu_m)^2 + 4\omega_m \theta_m S_m}$$

$$\lambda_4 = \frac{1}{2}A_8 + \frac{1}{2}A_{16} - \frac{1}{2}\sqrt{{A_8}^2 - 2A_8A_{16} + {A_{16}}^2 + 4A_9A_{15}}$$

$$\lambda_4 = \frac{1}{2}(-\alpha_m B - \omega_m - \delta) + \frac{1}{2}(-\rho B - \delta - \mu_m) - \frac{1}{2}\sqrt{(-\alpha_m B - \omega_m - \delta)^2 - 2(-\alpha_m B - \omega_m - \delta)(-\rho B - \delta - \mu_m) + (-\rho B - \delta - \mu_m)^2 + 4\omega_m \theta_m S_m}$$

$$\lambda_5 = \frac{1}{2}A_4 + \frac{1}{2}A_{26} + \frac{1}{2}\sqrt{{A_4}^2 - 2A_4A_{26} + {A_{26}}^2 + 4A_5A_{24}}$$

$$\lambda_5 = \frac{1}{2}(-\delta - \iota_m B) + \frac{1}{2}(-\chi - \delta) + \frac{1}{2}\sqrt{(-\delta - \iota_m B)^2 - 2(-\delta - \iota_m B)(-\chi - \delta) + (-\chi - \delta)^2 + 4\iota_m B\chi}$$

$$\lambda_5 = \frac{1}{2}\sqrt{(\chi + B\iota_m)} - \frac{1}{2}\chi - \frac{1}{2}B\iota_m - \delta$$

$$\lambda_6 = \frac{1}{2}A_4 + \frac{1}{2}A_{26} - \frac{1}{2}\sqrt{{A_4}^2 - 2A_4A_{26} + {A_{26}}^2 + 4A_5A_{24}}$$

$$\lambda_6 = \frac{1}{2}(-\delta - \iota_m B) + \frac{1}{2}(-\chi - \delta) - \frac{1}{2}\sqrt{(-\delta - \iota_m B)^2 - 2(-\delta - \iota_m B)(-\chi - \delta) + (-\chi - \delta)^2 + 4\iota_m B\chi}$$

$$\lambda_6 = -\frac{1}{2}\sqrt{(\chi + B\iota_m)} - \frac{1}{2}\chi - \frac{1}{2}B\iota_m - \delta$$

$$\lambda_7 = -(\varphi_m + \delta)$$

$$\lambda_8 = -\chi_y - \delta$$

$$\lambda_9 = g + \beta - \lambda - \mu_p B$$

$$\lambda_{10} = -(\varphi_y + \delta)$$

$$\lambda_{11} = n + \psi - \frac{2B}{C} - \eta$$

$$\lambda_{12} = -(\gamma + \delta + \iota_y B)$$

Hence showed that all the eigenvalues of $J(E_1)$ are negative. Hence the system is locally asymptotically stable.

3 Numerical Solution and Discussion

In this section, we used an inbuilt Matlab 2018a function ode45 to simulate our model. Matlab 2018a is a graphical user interface used for solutions, simulation and visualization of solution curves which solves system of Ordinary Differential Equations (ODEs) using the fourth-order Runge Kutta method. The results of the numerical solution carried out were interpreted and discussed. Some of the baseline values were estimated base on the work of [16].

Table 1. Parameters and baseline value of numerical solution

Parameters	Baseline value	Remark/Source
r	0.125	Estimated
ϕ_y	0.001	Estimated
ϕ_m	0.001	Estimated
θ_y	0.06	[11]
θ_m	0.06	[11]
ω_y	0.17	[11]
ω_m	0.87	Estimated
α_y	0.00006	Estimated
α_m	0.002	Estimated
φ_y	0.5	Estimated
φ_m	0.82	Estimated
ι_y	0.02	Estimated
ι_m	0.3	Estimated
χ	0.002	Estimated
χ_y	0.067	Estimated
γ	0.067	Estimated
ρ	0.00002	Estimated
β	0.992	Estimated
ψ	0.992	Estimated
λ	0.48	Estimated
η	0.02	Estimated
δ	0.004	[11]
μ_y	0.9	Estimated
μ_m	0.003	Estimated
μ_p	0.00018	Estimated
ϖ	2387014640356.4	Estimated
q	2387014640356.4	Estimated
g	0.125	Estimated
n	0.125	Estimated

Experiment One: An experiment to investigate the impact of the recruitment rate of biocontrol agent on the pathogen population.

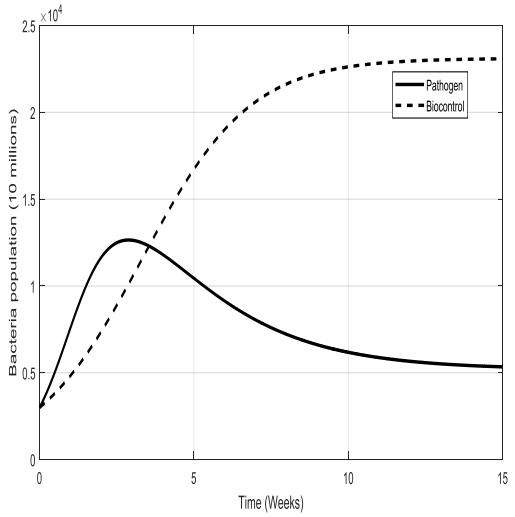

Fig. 1. (a)

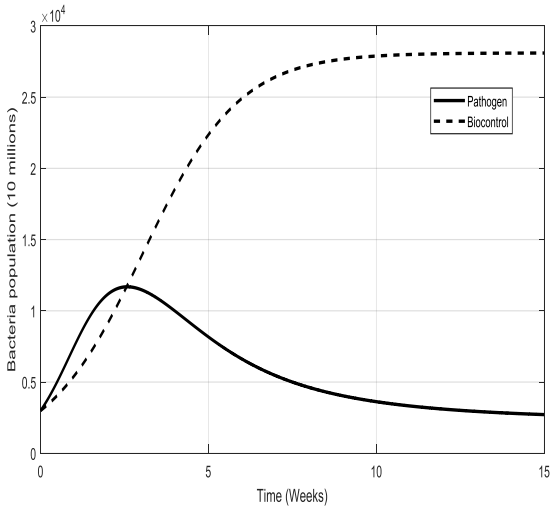

Fig. 1. (b)

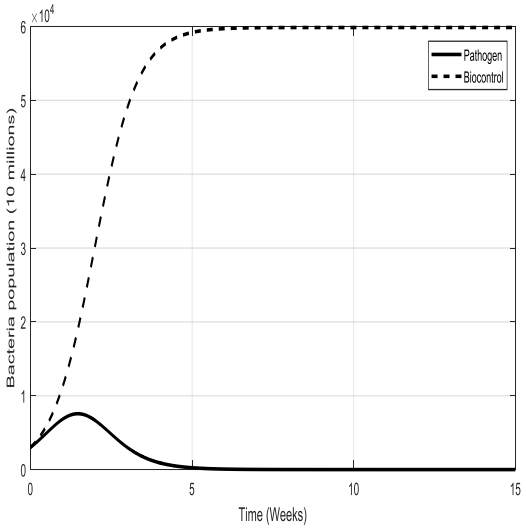

Fig. 1. (c)

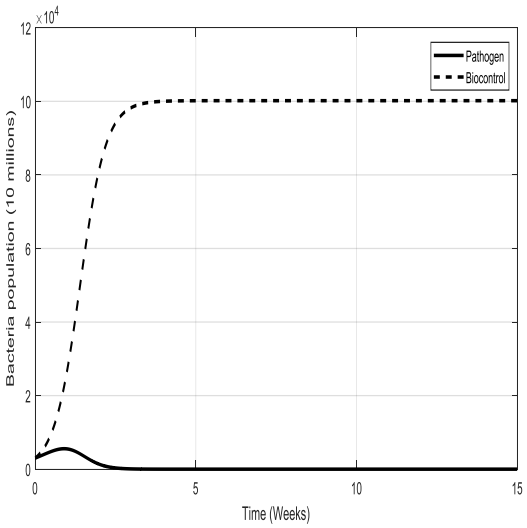

Fig. 1. (d)

Fig. 1. The Dynamics of the population of pathogen as a result of the introduction of biocontrol agent (a) shows pathogen and biocontrol agent population when $g = 0.00125$, (b) shows pathogen and biocontrol agent population when $g = 0.0125$, (c) shows pathogen and biocontrol agent population when $g = 0.9125$ (d) shows the pathogen and biocontrol agent population when $g = 1.9125$

As show in Fig. 1 above the rate at which the biocontrol agent *(Lysobacter antibioticus)* agent is been recruited has a significant impact on the population of the pathogen. From Fig. 1a it is shown that when the value of $g = 0.00125$ the pathogen will grow rapidly up to about 120000(10 millions) after about three weeks before it will start falling but won't fall to zero. Likewise in Fig. 1b when $g = 0.0125$ but in Fig. 1c when the value of $g = 0.9125$ the population of pathogen will only rise to its pick of about 6000(10millions) in less than three weeks before it will start falling and reach zero in about five weeks. Similarly, from Fig. 1d when the value of g is set at *1.9125* the population will only rise to its pick of about 1000(10millions) before it will die out to zero pathogen in less than two weeks.

Experiment Two: An experiment to investigate the impact of biocontrol on Exposed and Infected Plants populations.

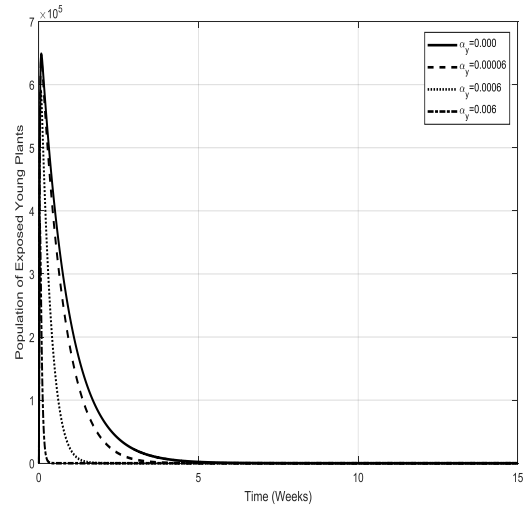

Fig. 2. (a)

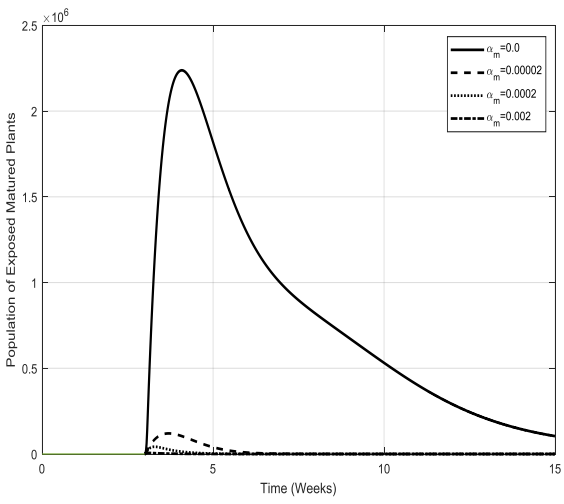

Fig. 2. (b)

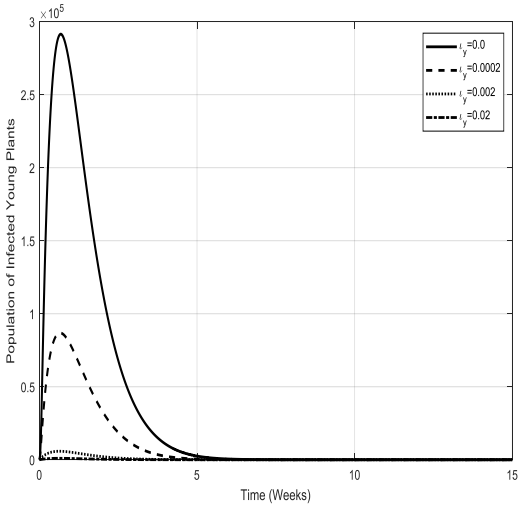

Fig. 2. (c)

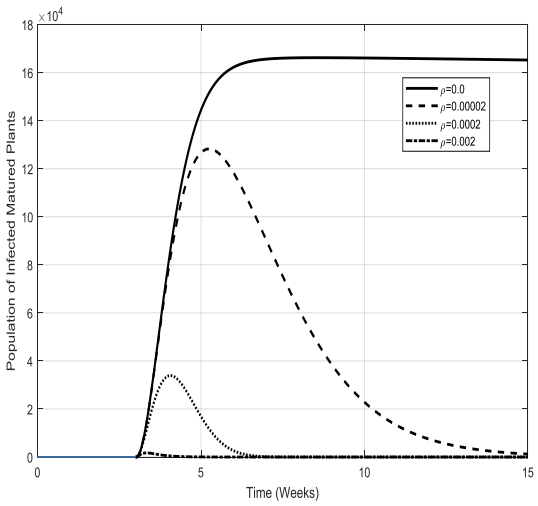

Fig. 2. (d)

Fig. 2. The Dynamics of the population of Exposed and Infected Plants population as a result of the introduction of biocontrol (a) shows Exposed Young Plants population varying the value of α_y, (b) shows Exposed Matured Plants population varying the value of α_m, (c) shows Infected Young Plants population varying the value of ι_y (d) shows Infected Matured Plants population varying the value of ρ

The graph of Fig. 2a shown above shows that as the value of α_y the recovery rate is increasing as a result of increase interaction between exposed young plants and the biocontrol agent the less time it will take for the exposed population to all recovered and tends to zero. Specifically, when the value of $\alpha_y = 0.0006$ it will take approximately four weeks for the population to move out of the compartment but on increasing the value of α_y to 0.006, in less than a week all the young plants population will move out of the compartment.

Considering the graph of Fig. 2b as shown above we observe that as the value of α_m is increasing the less time it will take for the matured plants to recover thereby reducing the population of Exposed Matured plants rapidly to Zero. When the value of $\alpha_m = 0$ it is shown that the exposed matured plants population will rise to as high as 1620000 before falling but setting $\alpha_m = 0.00002$ the population rise to only about 100000 before falling to zero. Further increasing the value of α_m we observe the less the number of exposed matured plants.

From Fig. 2c we can see from the graph showing the population of Infected Young Plants that if the value of ι_y increase as a result of the increase interaction between susceptible young plants and the biological control agent it has an indirect effect on the population of infected young plants. When the value of $\iota_y = 0.0002$ over 800000 will be infected but increasing the value to 0.02 only a few will be infected.

Lastly, when we considered the graph in Fig. 2d we observed that when there is no biocontrol the population of Infected Matured Plants will grow rapidly and reach a pick and maintain that values but on the introduction of biocontrol to the farm when $\rho = 0.00002$ after about 15 weeks they will be no infected matured plants in the farm. Increasing the value of $\rho = 0.02$ we observed that the population of Infected Matured Plants in the farm will be approximately zero almost from the onset.

Experiment Three: An experiment to investigate the impact of biocontrol on Protected Plant Population

From the graph shown in Fig. 3a above we observe that when the value of $\iota_y = 0$ the population of Protected Young Plants will be equal to zero. But when $\iota_y = 0.0002$ the population increase steeply to 3800000 before falling when the value of ι_y is further increase to 0.02 the population will rise to 10900000 therefore, as the value of ι_y is increasing the population of Protected Young Plants will be increasing as well.

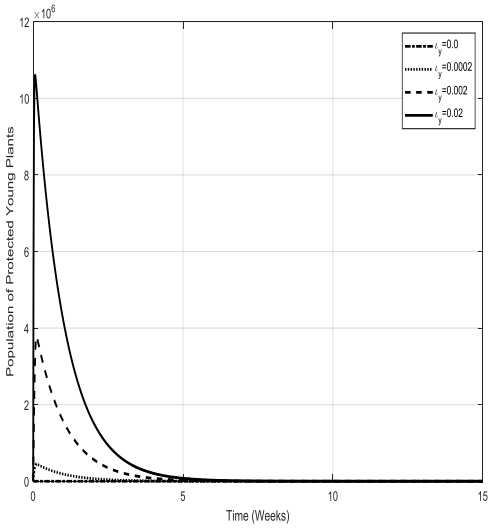

Fig. 3. (a)

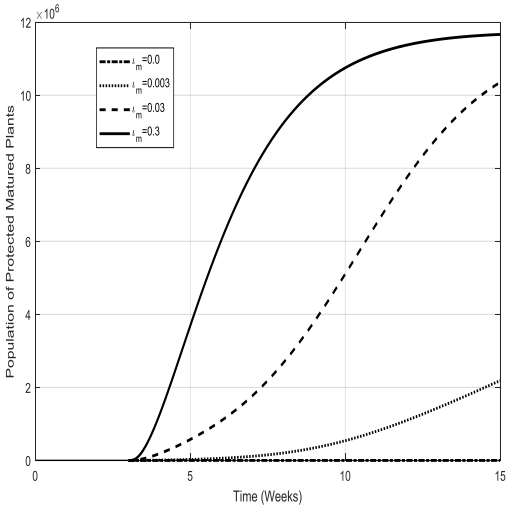

Fig. 3. (b)

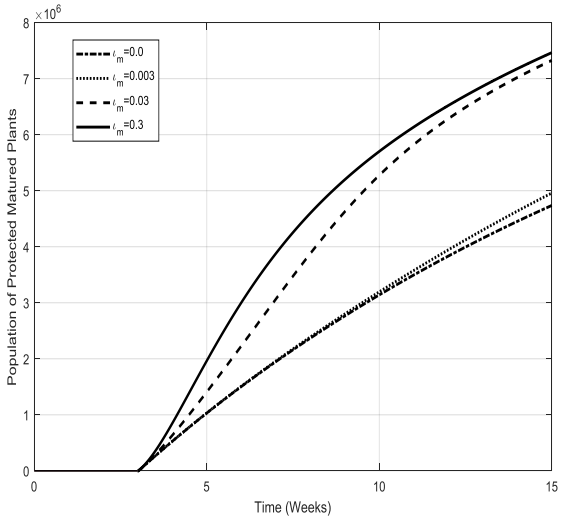

Fig. 3. (c)

Fig. 3. The Dynamics of the population of Protected Plants as a result of the introduction of biocontrol (a) shows Protected Young Plants population varying the value of ι_y, (b) shows Protected Matured Plants population varying the value of ι_m and setting $\iota_y = 0$, (c) shows Protected Matured Plants population varying the value of ι_m

Fig. 3b shows that when the value of $\iota_y = 0$ increasing the value of ι_m increase the population of Protected Matured plants drastically but when the both ι_y and ι_m are equal to zero the population of Protected Matured Plants will also be zero. But from the graph in Fig. 3c we observe that when the value of ι_y is not equal to zero the effect of ι_m on the Protected Plant Population is not so drastic.

Experiment Four: An experiment to compare the plant population with and without biological control.

From Fig. 4a in the presence of biological control we observe that the population of Exposed Young and Matured Plants, as well and the Infected Young and Matured all eventually tends to zero after a given period of time where in the absence of biocontrol as in show in Fig. 4b the population of infected matured plants will persist in the plant population leading to the disease been endemic in the farm if not controlled.

Considering Fig. 4c & d we observe that in the presence of biological control in (c) the population of Protected Plants will be increasing while that of infected plants will diminish to zero. But looking at (d) the reverse is the case with the population of infected plants growing why that of protected plants is equal to zero.

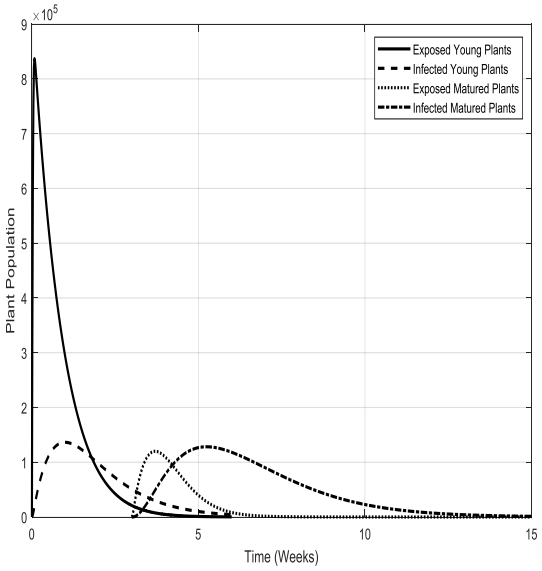

Fig. 4. (a)

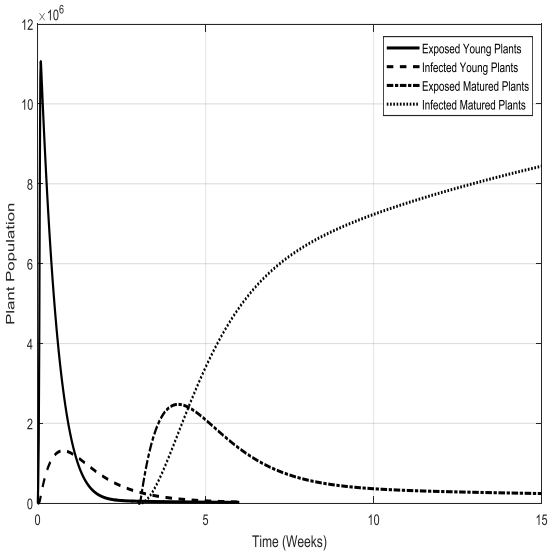

Fig. 4. (b)

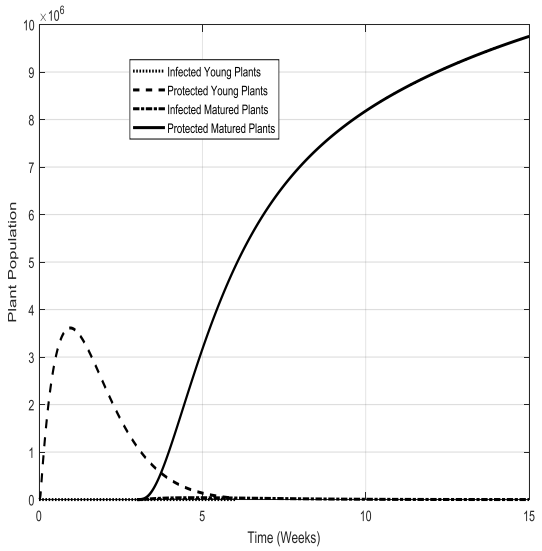

Fig. 4. (c)

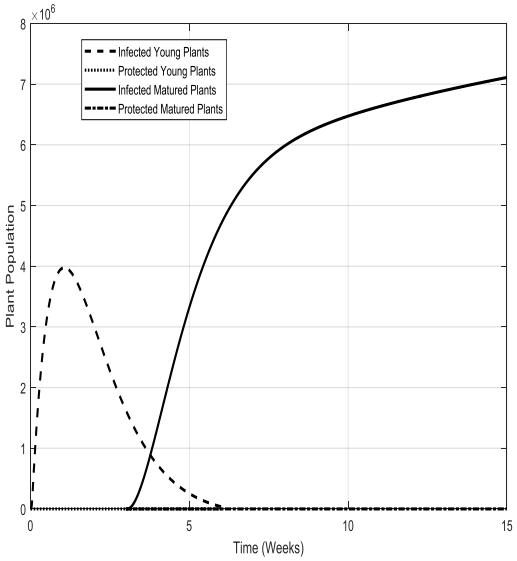

Fig. 4. (d)

Fig. 4. The Dynamics of plant population with and without biocontrol (a) shows plants population with $\iota_m = 0.3,\ \iota_y = 0.02, \rho = 0.00002, \alpha_y = 0.00006$, (b) shows plants population with $\iota_m = \iota_y = \rho = \alpha_y = \alpha_m = 0$, (c) shows plants population with $\iota_m = 0.3,\ \iota_y = 0.02, \rho = 0.00002, \alpha_y = 0.00006$, (d) shows plants population with $\iota_m = \iota_y = \rho = \alpha_y = \alpha_m = 0$

Experiment Five: An experiment to investigate the effect of the time of application of biocontrol on the population of plants.

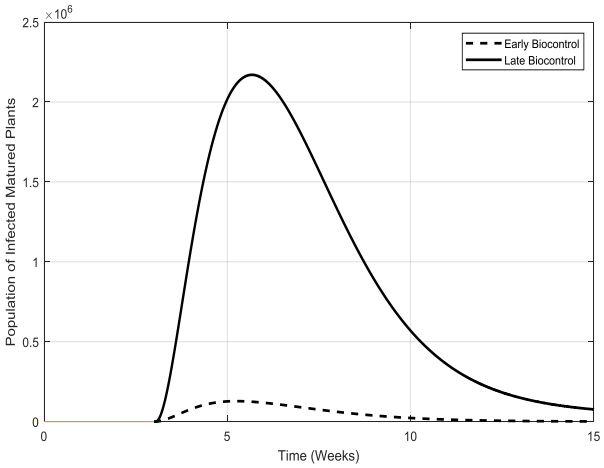

Fig. 5. (a)

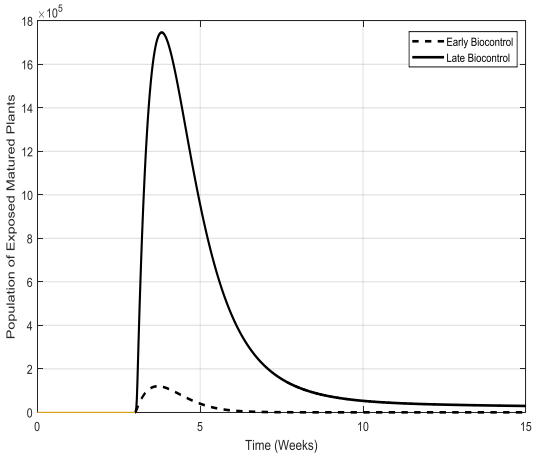

Fig. 5. (b)

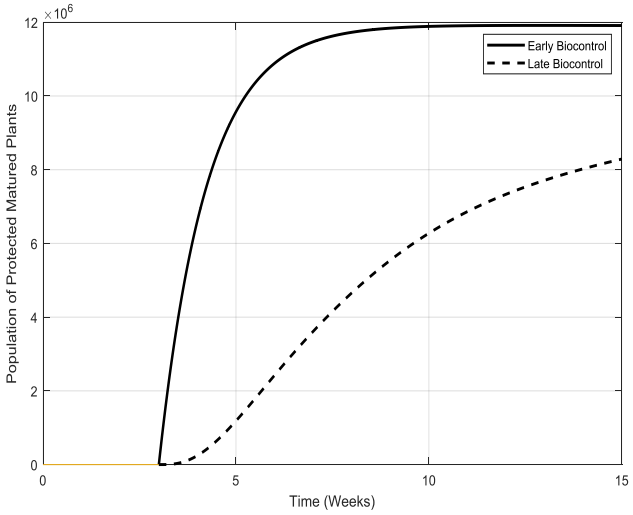

Fig. 5. (c)

Fig. 5. The dynamics of plant population considering time of biocontrol application (a) shows the population of infected matured plants, (b) shows the population of exposed matured plants, (c) shows the population of protected matured plants

From Fig. 5a, we can see from the graph that when biocontrol is applied when the plants are less than three weeks old population of Infected plants in the population when the plants matured is just about 200000 but if left until when the plant has matured before the application of control the population of Infected Matured Plants can go to as high as 2100000.

Considering the graph on Fig. 5b which display the population of Exposed Matured plants at both early and late control, we observe that if the control is applied early when the plants are still young, the population of plants that are likely to be exposed to the disease when they become matured is going to be drastically lower compare to been applied when the plants are already matured.

The graph on Fig. 5c shows the population of Protected Matured Plants, as shown when the control is applied when the plants are still young at about 5 weeks almost all the plants in the farm will be protected from the disease but when we wait until the plants are matured before the introduction of biocontrol some of the plants will still be susceptible to the disease even at 15 weeks.

4 Discussion of Finding

From the result of experiment one, we can say that the biocontrol agent has a significant impact on the population of the pathogen which can possibly lead to total eradication of the disease pathogen from the farm.

The results of experiment two shows that the biocontrol agent can be used for treatment of bacterial blight of rice since on its introduction the population of both the exposed and infected rice plants population decreases drastically to a point in which the population is entirely free from the disease.

Experiment three on the other hand looks at the possibility of using the biocontrol agent as a means of providing immunity to the plants. And from the result obtain it can be seen that the biocontrol agent can not only be used for treatment but can also be used to provide immunity for the plant from been infected with the disease.

In experiment four we try to look at how the plant population will behave in the presence and absence of the biocontrol to ascertain our findings in experiment two and three. And it was observed concurrently that in the absence of any form of control the population of exposed and infected rice plants increase exponentially but in its presence, the population of exposed and infected rice plants reduced drastically which is similar with our result from experiment two and three.

Finally, in the last experiment we tried to find out if the time of application of the biocontrol plays any part in the population of exposed and infected plants. And it was observed that if the biocontrol is introduced when the plants are still young it will help to give a fast and wide spread protection to the plants than when applied late when most of them must have been either exposed or infected with the disease already.

5 Conclusion

In conclusion, a model to study the dynamics of the effect of biocontrol agent on bacteria blight of rice was developed and the dynamics was studied using Matlab 2018a and it was observed that using *Lysobacter antibioticus* as a biocontrol agent the pathogen causing bacterial blight of rice can be completely eradicated from the farm. Our result also shows that the time of application of the biocontrol is very vital. If the biocontrol are applied when the plants are relatively young it will help to protect more rice plants from getting infected compare to if applied at a later stage.

Competing Interests

Authors have declared that no competing interests exist.

References

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