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Optimal Control Analysis of Rice Blast Disease with Environmental Pathogen Pool and Yield Dynamics

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ABSTRACT

The fungus *Magnaporthe oryzae* drives rice blast disease, a primary constraint on global rice production that often leads to yield losses exceeding 30%. This study develops and analyses a mathematical model of rice blast transmission to enlighten disease mitigation strategies. The model divides the rice plant population into susceptible, exposed, infectious, and removed classes, while explicitly incorporating a dynamic environmental pathogen pool and cumulative yield dynamics to capture the compounding drivers of an outbreak. We establish the positivity and boundedness of the model solutions, determine its equilibrium points, derive the basic reproduction number (R_0), and perform a sensitivity analysis to identify key transmission parameters. We apply Pontryagin's maximum principle to design an optimal control framework to reduce infection rates in the plant population and associated intervention costs. Time-dependent controls include resistant variety deployment, fungicide application, roguing of symptomatic plants, and fertiliser and irrigation enhancement. Simulation results demonstrate that fungicide application constitutes the foundational control measure, achieving approximately 84% reduction in peak infection and enabling epidemic elimination when combined with other strategies. Resistant variety deployment serves as a critical yield multiplier, transforming aggressive roguing from a catastrophically destructive practice into a highly productive integrated strategy. Furthermore, fertilizer/irrigation enhancement acts as an essential catalyst, requiring a threshold level ($u_4 \geq 0.2$) to convert disease suppression into realized productivity gains; without such enhancement, control investments become counterproductive. These findings indicate that optimal rice blast management requires balancing epidemiological suppression with agronomic productivity. Ultimately, the ideal strategy is contingent on whether farmers prioritize maximum yield potential or production stability, suggesting that moderate rouging rates can optimize outcomes in resistance-based integrated control systems.

KEYWORDS: Rice Blast Disease, Optimal Control, Pontryagin's Maximum Principle, Roguing.

I. INTRODUCTION

Among global cereal crops, few match the agricultural significance of rice (*Oryza sativa*), which serves as the staple diet for more than 50% of the world's population. It plays a vital role in ensuring food security, reducing poverty, and supporting economic development, particularly in developing countries where agriculture remains a major source of livelihood. In many African and Asian countries, rice contributes significantly to household nutrition and national food supply. Consequently, increasing rice productivity has become a major priority for governments, policymakers, and agricultural stakeholders

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seeking to meet the food demands of a rapidly growing population. Despite its importance, a variety of environmental stressors and living pests continually jeopardize rice yields. Of all the biological threats to global rice production, few are as destructive as rice blast disease, caused by the fungus *Magnaportheoryzae.* The disease infects rice plants at different growth stages, causing lesions on leaves, stems, nodes, and panicles, thereby reducing photosynthetic efficiency, grain quality, and overall yield. Severe outbreaks of rice blast disease frequently inflict devastating damage on final crop harvests, sometimes exceeding 30%, thereby posing a serious threat to sustainable rice production and global food security. To mitigate the impact of rice blast disease, several management strategies have been proposed and implemented. These include the use of resistant rice varieties, fungicide application, roguing (removal of infected plants), proper irrigation practices, and fertilizer enhancement programs. Resistant varieties reduce rice plants' susceptibility to infection, fungicides suppress pathogen growth and disease progression, and roguing helps reduce disease transmission by removing infected plants from the field. Similarly, fertiliser and irrigation management improve plant vigour and enhance productivity. However, the effectiveness of these interventions depends on the intensity, timing, and combination of their implementation. Therefore, determining the most efficient and economically feasible disease management strategy remains an important challenge.

Mathematical modelling has emerged as essential for deciphering the underlying dynamics of crop epidemics and evaluating the effectiveness of intervention strategies. In epidemiological modelling, susceptible plants are healthy plants capable of contracting the disease; exposed plants are infected plants that have not yet developed visible symptoms; infected plants are those actively expressing disease symptoms; and removed plants represent plants that have acquired protection against further disease progression. In the context of rice blast disease, environmental pathogen spores play a crucial role in disease transmission, and cumulative rice yield serves as an important indicator of agricultural productivity. By applying optimal control theory, researchers can systematically evaluate trade-offs between public health impacts and the financial toll of management efforts while maximizing agricultural output. Several researchers have investigated the transmission dynamics and management of rice blast disease using mathematical models. [9] developed mathematical approaches for understanding plant disease epidemics and highlighted the importance of disease modelling in crop protection. [10] examined the biological mechanisms underlying plant infection by *Magnaporthe oryzae* and emphasized the devastating effects of rice blast disease on crop productivity. [3] proposed a daily epidemiological model for seasonal forecasting and early warning of rice blast outbreaks in South Korea. [4] developed a rice blast disease model under tropical climatic conditions and demonstrated the influence of environmental factors on disease spread. Subsequently, [7] incorporated climatic variables into rice blast disease transmission models, while [8] extended these frameworks to investigate disease severity and progression under varying environmental conditions. Recent studies have also explored the influence of control measures on rice production. [2] developed an analytical model to project crop yields under the pressure of rice blast epidemics and investigated the effects of fertilizer application, resistant varieties, mechanical control, and chemical control on rice productivity. The study demonstrated that effective implementation of disease management strategies can significantly improve rice yield and contribute to food security. Furthermore, experimental studies by [5] and [1] confirmed the effectiveness of fungicides in suppressing rice blast disease, while [6] highlighted the economic benefits of reducing disease-related losses.

Although these studies have contributed significantly to understanding rice blast disease dynamics and management, important research gaps remain. Most existing models focus primarily on disease forecasting, parameter estimation, climatic influences, or yield prediction. In particular, the model

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developed by [2] examined the effects of various control measures on rice yield but did not formulate an optimal control framework to determine the most cost-effective combination of interventions over time. Furthermore, many existing rice blast disease models fail to account for environmental factors (pathogen dynamics), cumulative rice yield, and multiple management tactics within a single, cohesive optimal control system. Consequently, there remains a limited understanding of how to optimally combine resistant variety deployment, fungicide application, roguing effort, and fertilizer enhancement to reduce disease prevalence, suppress pathogen density, and maximize rice productivity.

Motivated by these limitations, this study develops a deterministic optimal control model for rice blast disease transmission that incorporates susceptible, exposed, infected, and removed rice plant populations together with environmental pathogen dynamics and cumulative rice yield. The model integrates four time-dependent control measures, namely resistant variety deployment, fungicide application, roguing effort, and fertilizer enhancement effort. The study aims to investigate the effectiveness of these interventions in reducing disease transmission, minimizing pathogen accumulation, improving rice yield, and lowering management costs. Using Pontryagin's Maximum Principle, the work seeks to determine optimal intervention strategies to promote sustainable rice production and improve food security.

This study is organized into four main parts. Section I introduces the epidemic model, while Section II details the chosen control methodologies. Numerical simulations and a thorough discussion of the resulting optimal trends are provided in Section III, while Section IV offers a concluding summary of the research.

II. RELATED WORKS

A. Materials and Methods

In this section, we will discuss the basic properties, i.e. the positivity and boundedness of the system, the equilibrium point, the basic reproduction number (R_0) and the sensitivity analysis of the model in the absence of the control measures.

The system of equations (1) symbolizes the total rice plant population at time. t, by $N(t)$, divided into six compartments, namely: the susceptible rice plant compartment, $S(t)$; which are healthy rice plants population; Exposed rice plants compartment are infected plants that have not yet developed visible symptoms, $E(t)$; Infected rice plant compartments are those actively expressing disease symptoms, $I(t)$; Removed rice plants compartment represents plants that have acquired protection against further disease progression, $R(t)$; Environmental pathogen spores compartment, that play a key role in disease transmission, $P(t)$; and Cummulative rice compartment serves as an important indicator of agricultural productivity, $Y(t)$. The total population per unit time t is $N(t)$. The control $u_1(t)$ represents resistant variety deployment (which reduces the susceptibility of rice plants to infection), $u_2(t)$ represents the fungicide application, which suppresses pathogen growth and disease progression, $u_3(t)$ represents a roguing effort that helps reduce disease transmission by removing infected plants from the field, and $u_4(t)$ represents a fertilizer enhancement effort which improves plant vigour and enhances productivity. We consider the following system of six nonlinear differential equations below:

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$$\frac{dS}{dt} = r(1-\frac{N}{u_4 k}) - \beta(1-u_1)\frac{SP}{k+P} - \mu S$$
$$\frac{dE}{dt} = \beta(1-u_1)\frac{SP}{k+P} - (\sigma+\mu)E$$
$$\frac{dI}{dt} = \sigma E - \phi u_2 I - (\mu+\delta+u_3)I$$
$$\frac{dR}{dt} = (\delta+u_3)I - \mu R$$
$$\frac{dP}{dt} = \lambda I(1-\frac{P}{p_{\max}}) - \mu_p(1+\varepsilon u_2)P - \upsilon S\frac{P}{k+P}$$
$$\frac{dY}{dt} = p(\gamma_s u_4 S + \gamma_e(1-\theta_e)E + \gamma_i(1-\theta_i)I)$$

…….(1)

Where $u_4 k = k_0(1+\eta u_4)$.

B. State Variables, Model Parameters, Their Descriptions, Values, and Sources

To facilitate the formulation and analysis of the proposed rice blast disease model, the state variables, model parameters, and control variables employed in the study are summarized in the following tables. The descriptions of the variables and parameters, along with their corresponding values and sources, are provided to support biological interpretation and ensure the reproducibility of the numerical simulations. Parameter values were obtained from published literature where available, while parameters lacking empirical estimates were assumed within biologically realistic ranges and subsequently validated through sensitivity analysis.

Table 1: Description of State Variables

Variables	Descriptions	Units
$S(t)$	Population of Susceptible Rice Plants at time (t)	Plants
$E(t)$	Population of Exposed Rice Plants at time (t)	Plants
$I(t)$	Population of Infected Rice Plants at time (t)	Plants
$R(t)$	Population of Removed Rice Plants at time (t)	Plants
$P(t)$	Environmental Pathogen Spores Density at time (t)	$spores\ m^{-2}$
$Y(t)$	Cumulative Rice Yield at time (t)	Tonnes/ha

Table 2: Description of Model Parameters

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Parameter	Description	Values	Units	Sources
r	Intrinsic growth rate of rice plants	0.16	day^{-1}	[2]
β	Disease transmission coefficient	0.68	day^{-1}	[2]
μ	Natural mortality rate of rice plants	0.001	day^{-1}	[2]
σ	Progression rate from exposed to infected	0.10	day^{-1}	[4]
δ	Disease-induced mortality rate	0.02	day^{-1}	[2]
λ	Pathogen spore production rate	5.00	$spores\ day^{-1}$	[2]
$P_{\max}$	Maximum environmental pathogen carrying capacity	100000	$spores\ m^{-2}$	Assumed
ε	Fungicide effectiveness against pathogen survival	0.80	Dimensionless	[2]
μ_p	Natural pathogen decay rate	0.15	day^{-1}	[2]
η	Fertiliser enhancement coefficient	0.50	Dimensionless	Assumed
γ_s	Yield production rate for susceptible plants	0.05	Tonnes/ha day^{-1}	Assumed
γ_e	Yield production rate for exposed plants	0.03	Tonnes/ha day^{-1}	Assumed
γ_i	Yield production rate for infected plants	0.005	Tonnes/ha day^{-1}	Assumed
θ_e	Yield loss fraction for exposed plants	0.40	Dimensionless	Assumed
θ_i	Yield loss fraction for infected plants	0.70	Dimensionless	Assumed
k_0	Baseline carrying capacity	1000	Plants	[2]
υ	Pathogen removal rate	0.001	day^{-1}	[2]
ϕ	Fungicide treatment efficacy	0.6	day^{-1}	[2]

C. Model Assumptions

The deterministic rice blast disease model developed in this study is based on the following assumptions:

(a). Fertiliser and irrigation enhancement efforts increase the carrying capacity of the rice production system and improve plant productivity.

(b). The disease transmission process follows a saturated incidence function, reflecting the fact that infection rates increase with pathogen density but approach saturation at high spore concentrations.

(c). The deployment of resistant rice varieties reduces the susceptibility of healthy rice plants to infection.

(d). Infected rice plants contribute to the production and accumulation of pathogen spores in the environment.

(e). All rice plants are subject to natural mortality at a rate (μ).

(f). The environmental pathogen population grows through spore production by infected plants and is limited by a maximum carrying capacity $(P_{\max})$.

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(g). Rice yield is generated by susceptible, exposed, and infected rice plants but is reduced by disease severity.

The rice plant population is divided into four epidemiological compartments: susceptible rice plants, exposed rice plants, infected rice plants, and removed rice plants. We define the total rice plant population at time t as $N(t) = S(t) + E(t) + I(t) + R(t)$. Rice plants grow according to a logistic growth model characterized by a baseline growth rate (r) and an environmental ceiling (k).

D. Positivity and Boundedness

The system is qualitatively analyzed better to understand the dynamics of the rice blast model. The plant population cannot be negative. It is therefore necessary to ascertain the positivity of the state variables for future times. The set of solutions of $\{S(t), E(t), I(t), R(t)\}$ of System 1 is positive for $t \geq 0$ with initial conditions $S(0), E(0), I(0), R(0)$ being non-negative.

Proof:

From the system (1), we have; $\frac{dS}{dt} + \mu S \geq r\left(1 - \frac{N}{K}\right)$

……(2)

Using the integrating factor $I.F = e^{\int \mu dt} = e^{\mu t}$. Multiply thoroughly by I.F.

$$\frac{dS}{dt} \cdot e^{\mu t} + \mu S \cdot e^{\mu t} \geq r\left(1 - \frac{N}{K}\right) \cdot e^{\mu t} \Rightarrow \frac{d}{dt}\left(S \cdot e^{\mu t}\right) \geq r\left(1 - \frac{N}{K}\right) \cdot e^{\mu t}$$

Thus, we have; $S(t) \geq S(0)e^{-\mu t} + \left(1 - e^{-\mu t}\right) r\left(\frac{K - N}{\mu K}\right) > 0$.

……(3)

Similarly, by solving for other plant compartments, we have;

$E(t) \geq E(0)e^{-(\sigma+\mu)t} > 0$,……(4)

$I(t) \geq I(0)e^{-(\delta+\mu)t} > 0$, ……..(5)

$R(t) \geq R(0)e^{-\mu t} > 0$ ……(6)

This establishes that $S > 0, E \geq 0, I \geq 0, R \geq 0$ for future time. Thus, positivity of the state variables is ascertained for future time.

E. Rice Blast-free Equilibrium (RBFE)

In this model, the equilibrium points are found by setting the right-hand side of equation (1) equal to zero. The disease-free equilibrium is achieved in the absence of Rice blast disease. Hence, rice blast disease equilibrium $\{S_0, E_0, I_0, R_0, P_0\} = \left\{\frac{kr}{r + \mu k}, 0, 0, 0, 0\right\}$.

F. The Basic Reproduction Number

The basic reproduction number R_0 of the system (1) using the approach of [12] described in the previous section, the basic reproduction number of system (1) is computed as follows:

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$$F=\begin{pmatrix}0 & 0 & \frac{\beta S_0}{k}\\ 0 & 0 & 0\\ 0 & \lambda & 0\end{pmatrix},\qquad V=\begin{pmatrix}\sigma+\mu & 0 & 0\\ -\sigma & \delta+\mu & 0\\ 0 & 0 & \frac{\upsilon S_0}{k}+\mu\end{pmatrix}$$

……(7)

$$V^{-1}=\begin{pmatrix}\frac{1}{\sigma+\mu} & 0 & 0\\ -\frac{\sigma}{(\sigma+\mu)(\delta+\mu)} & \frac{1}{\delta+\mu} & 0\\ 0 & 0 & \frac{k}{\upsilon S_0+\mu k}\end{pmatrix}$$

……(8)

$$FV^{-1}=\begin{pmatrix}0 & 0 & \frac{\beta r}{r+\mu k}\\ 0 & 0 & 0\\ 0 & \lambda & 0\end{pmatrix}\begin{pmatrix}\frac{1}{\sigma+\mu} & 0 & 0\\ \frac{\sigma}{(\sigma+\mu)(\delta+\mu)} & \frac{1}{\delta+\mu} & 0\\ 0 & 0 & \frac{r+\mu k}{(\upsilon+\mu)r+k\mu^2}\end{pmatrix}$$

……(9)

$$FV^{-1}=\begin{pmatrix}0 & 0 & \frac{\beta r}{\upsilon r+\mu(\mu k+r)}\\ 0 & 0 & 0\\ \frac{\lambda\sigma}{(\sigma+\mu)(\delta+\mu)} & \frac{\lambda}{\sigma+\mu} & 0\end{pmatrix}$$

……(10)

R_0 is explicitly computed using $\rho\left(FV^{-1}\right)$, where ρ signifies the spectral radius. Hence,

From the system of equation (1), R_0 defined as

$$R_0=\sqrt{\frac{\beta\sigma\lambda r}{(\sigma+\mu)(\upsilon+\mu)(\upsilon r+\mu(\mu k+r))}}$$

……(11)

G. Sensitivity Analysis of Basic Reproduction Number (R0)

Sensitivity analysis was used to determine the model's fitness to parameter values, that is, to help us identify the parameters with a high impact on the reproduction number R0. Using the approach in [13], we calculated the normalized forward sensitivity indices of R_0. Let

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$$\Upsilon_l^{R_0} = \frac{l}{R_0} \times \frac{\partial R_0}{\partial l} \quad \text{.....(12)}$$

denote the sensitivity index of R_0 with respect to the parameter m. We obtain the results in Table 3.

Table 3: Sensitivity analysis results

Parameters	Description	Sensitivity Index
β	Transmission rate	+0.5000
λ	Sporulation rate	+0.5000
σ	Incubation rate	+0.0050
υ	Spore density rate on plants	-0.9065
μ	Mortality rate	-0.1013
k	Carrying capacity of plants	-0.0028
r	Intrinsic growth rate of plants	+0.0028

Table 3 shows that the basic reproduction numbers R_0 are most sensitive to changes in both λ, β, σ and r. If β, λ, σ , and r increase, R_0 will increase significantly. However, an increase in the parameters, υ, μ, and k will decrease R_0; they all have an inversely proportional relationship with R_0. So, an increase in any of them will decrease R_0. However, the size of the decrease will be proportionally smaller. Given R_0 sensitivity to β, λ, σ , and r, it seems sensible to focus on reducing those parameters and increasing these υ, μ, and k in other to reduce the basic reproduction number. In other words, this sensitivity analysis shows that prevention is better than cure, and avoiding unnecessary transmission can be beneficial. Efforts focused on preventing contact with pathogens are more effective in controlling the spread of rice blast disease.

H. The Optimal Control Problem

In this section, we will investigate optimal control strategies within the model to determine the most effective means of reducing the population of plants infected with rice blast disease. We aim to identify the optimal values. $u^* = (u_1^*, u_2^*, u_3^*, u_4^*)$ of the controls $u = (u_1, u_2, u_3, u_4)$ and the intervention time interval, $[0, T]$ and also minimize the objective function. The control $u_1(t)$ represents resistant variety deployment (which reduces the susceptibility of rice plants to infection) $u_2(t)$; represents the fungicide application (which suppresses pathogen growth and disease progression), $u_3(t)$ represents a roguing effort (that helps reduce disease transmission by removing infected plants from the field), and $u_4(t)$ represents a fertilizer enhancement effort which improves plant vigour and enhances productivity.

The objective function presents the optimal challenge we are working on, specifically focused on pinpointing cost-effective management tactics. We aim to suppress the infected rice population while curbing operational expenditures over the designated time horizon $[0, T]$. The function is given by

$$J(u_1, u_2, u_3, u_4) = \int_0^T e^{-rt} \left(p(\gamma_s u_4 S + \gamma_e (1-\theta_e) E + \gamma_i (1-\theta_i) I) - \frac{1}{2} c_1 u_1^2 - \frac{1}{2} c_2 u_2^2 - \frac{1}{2} c_3 u_3^2 - \frac{1}{2} c_4 u_4^2 \right) dt$$

.....(13)

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Where p represents the price of rice, r represents the discount rate, S is the susceptible rice plant, E is the exposed rice plant, I is the infected rice plant's compartment and c_i, where $i=1,...,4$ represent the control costs coefficients. The issue is to find the optimal functions such that $J(u_1,u_2,u_3,u_4)=\max J=(u_1,u_2,u_3,u_4)$ with, $(u_1,u_2,u_3,u_4)\in U$ and the control set is defined as $U=(u_1,u_2,u_3,u_4)$ or $u_i(t)$ is the Lebesgue measure on $[0,1]$, $0\le u_1,u_2,u_3,u_4\le 1, t\in[0,T]$ subject to the system shown in the model equation, along with the appropriate initial conditions. To address the issue, we should utilize Pontryagin's maximum principle to establish the system's optimality and demonstrate the existence of an optimal control.

I. Characterization of the Optimal Control

In this section, we want to show that an optimal control exists for the system (1).

Theorem 1.1. There exist optimal controls (u_1,u_2,u_3,u_4) and equivalent solutions, $S^*, E^*, I^*, R^*, P^*, Y^*$ that minimize $J(u_1,u_2,u_3,u_4)$ over U.

Proof. The integrand of the objective functional J is a convex function of (u_1,u_2,u_3,u_4), and the system of the dynamic equation satisfies the Lipschitz property for the state variables. So far, the state solutions are bounded. To find the optimal solution, we first establish the Lagrangian and Hamiltonian based on the model equation (1) and the objective functional (2). The Lagrangian for this control problem can be expressed as follows:

$$L(u_1,u_2,u_3,u_4)=e^{-rt}\left(p(\gamma_s u_4 S+\gamma_e(1-\theta_e)E+\gamma_i(1-\theta_i)I)-\frac{1}{2}c_1u_1^2-\frac{1}{2}c_2u_2^2-\frac{1}{2}c_3u_3^2-\frac{1}{2}c_4u_4^2\right)$$

.....(14)

We need to find the minimum values of the Lagrangian and the Hamiltonian. Now, the Hamiltonian H The problem is given as:

$$\begin{aligned}H=&e^{-rt}\left(p(\gamma_s u_4 S+\gamma_e(1-\theta_e)E+\gamma_i(1-\theta_i)I)-\frac{1}{2}c_1u_1^2-\frac{1}{2}c_2u_2^2-\frac{1}{2}c_3u_3^2-\frac{1}{2}c_4u_4^2\right)\\&+\lambda_S\left(r\left(1-\frac{N}{k_0(1+\eta u_4)}\right)-\beta(1-u_1)S\frac{P}{k+P}-\mu S\right)+\lambda_E\left(\beta(1-u_1)S\frac{P}{k+P}-(\sigma+\mu)E\right)\\&+\lambda_I\left(\sigma E-\phi u_2 I-(\mu+\delta+u_3)I\right)+\lambda_R\left((\delta+u_3)I-\mu R\right)\\&+\lambda_P\left(\lambda I(1-\frac{P}{p_{\max}})-\mu_p(1+\varepsilon u_2)P-\upsilon S\frac{P}{k+P}\right)+\lambda_Y\left(p(\gamma_s u_4 S+\gamma_e(1-\theta_e)E+\gamma_i(1-\theta_i)I)\right)\end{aligned}$$

.....(15)

Where $\lambda_S, \lambda_E, \lambda_I, \lambda_R, \lambda_P$, and λ_Y are adjoint variables.

Let us now pinpoint the important requirements that the ideal control policies and their associated state variables need to satisfy, applying Pontryagin's Maximum Principle.

Theorem 1.2: Given an optimal control pair (u_1,u_2,u_3,u_4) and a solution S, E, I, R, P, Y of the corresponding system of equations, there exist adjoint variables $\lambda_S, \lambda_E, \lambda_I, \lambda_R, \lambda_P$, and λ_Y satisfying

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$$\frac{d\lambda_S}{dt}=\left(\frac{\beta(1-u_1)P}{k+P}+\mu\right)\lambda_S-e^{-rt}p\gamma_s u_4-\frac{\beta(1-u_1)P}{k+P}\lambda_E+\frac{\lambda_P\upsilon P}{k+P}-\gamma_s u_4\lambda_Y \quad (16)$$

$$\frac{d\lambda_E}{dt}=(\sigma+\mu)\lambda_E-e^{-rt}p\gamma_e(1-\theta_e)-\sigma\lambda_I-\gamma_e(1-\theta_e)\lambda_Y \quad (17)$$

$$\frac{d\lambda_I}{dt}=(\phi u_2+\delta+\mu+u_3)\lambda_I-e^{-rt}p\gamma_i(1-\theta_i)-(\delta+u_3)\lambda_R-\gamma_i(1-\theta_i)\lambda_Y \quad (18)$$

$$\frac{d\lambda_R}{dt}=\mu\lambda_R \quad (19)$$

$$\frac{d\lambda_P}{dt}=\lambda_P\left(\frac{\lambda I}{P_{\max}}+\mu_P(1+\varepsilon u_2)+\frac{\nu S c}{(c+P)^2}\right)-\frac{\beta(1-u_1)S c(\lambda_E-\lambda_S)}{(c+P)^2} \quad (20)$$

$$\frac{d\lambda_Y}{dt}=0 \quad (21)$$

And the transversality conditions $\lambda_S(T)=\lambda_E(T)=\lambda_I(T)=\lambda_R(T)=\lambda_P(T)=\lambda_Y(T)=0$ as the end conditions at the final time T and the optimal control variables (u_1,u_2,u_3,u_4) are defined as;

$$u_1=\max\left\{0,\min\left(\frac{PS\beta(-\lambda_E+\lambda_S)e^{rt}}{(k+P)c_1},1\right)\right\} \quad (22)$$

$$u_2=\max\left\{0,\min\left(\frac{(-\lambda_P\gamma_P\varepsilon P-\lambda_I\phi I)e^{rt}}{c_2},1\right)\right\} \quad (23)$$

$$u_3=\max\left\{0,\min\left(\frac{I(-\lambda_I+\lambda_R)e^{rt}}{c_3},1\right)\right\} \quad (24)$$

$$u_4=\max\{0,\min(\psi),1\} \quad (25)$$

$$\psi=\frac{\left(3^{2/3}\left(\frac{1}{2}\left(3^{2/3}\left(e^{-3rt}k_0^2\left(\sqrt{27}\left(Ne^{-3rt}A_1r\lambda_S\right)^{1/2}A_9\right)\eta e^{2rt}+A_2\right)\right)^{2/3}2^{2/3}e^{rt}\right)+k_0\left(A_8 3^{1/3}\left(e^{-3rt}k_0^2\left(\left(\sqrt{27}A_3\right)\right)\right)\right)\right)A_5\eta^2A_4}{\left(9\left(e^{-3rt}k_0^2\left(\begin{array}{l}\left(\sqrt{27}\left(Ne^{-3rt}A_6r\lambda_S\right)^{1/2}\sqrt{3}c_4+2S^2k_0\eta\gamma_s^2\lambda_Y^2\left(Sp\eta\gamma_s+c_4\right)\right)\eta e^{2rt}\\+\frac{2e^{3rt}S^3k_0\eta^3\lambda_Y^3\gamma_s^3}{3}+A_7e^{rt}+\frac{2k_0\left(Sp\eta\gamma_s+c_4\right)^3}{3}\end{array}\right)^{1/3}k_0c_4\eta\right)\right)}$$

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where

$$A_1 = \frac{4S^2k_0\eta^2\lambda_Y^2\gamma_s^2\left(S\eta P\gamma_s + c_4\right)e^{2rt}}{9} + \frac{4e^{-3rt}S^3k_0\eta^3\lambda_Y^3\gamma_s^3}{27} + \left(\frac{4S\lambda_Y\gamma_s\left(S\eta P\gamma_s + c_4\right)^2k_0}{9} + \eta Nc_4^2r\lambda_S\right)\eta e^{rt}$$

$$+ \frac{4k_0\left(S\eta P\gamma_s + c_4\right)^3}{27}$$

$$A_2 = \left(\sqrt{27}\left(Ne^{-3rt}\left(A_1\right)r\lambda_S\right)^{1/2}\sqrt{3}c_4 + 2\lambda_Y^2\gamma_s^2S^2\eta c_0(S\eta P\gamma_s + c_4)\right)\eta e^{2rt} + \frac{2e^{3rt}S^3k_0\eta^3\lambda_Y^3\gamma_s^3}{3} + \left(2S^3k_0\eta^3P^2\lambda_Y\gamma_s^3\right)$$

$$+ \left(4S^2k_0c_4P\lambda_Y\gamma_s^2 + 9Nc_4^2r\lambda_S\right)\eta^2 + 2Sk_0c_4^2\eta\lambda_Y\gamma_s$$

$$A_3 = Ne^{-3rt}\left(\begin{array}{l}\frac{4S^2k_0\lambda_Y^2\gamma_s^2\left(S\eta P\gamma_s + c_4\right)e^{2rt}}{9} + \frac{4e^{3rt}S^3k_0\eta^3\lambda_Y^3\gamma_s^3}{27} \\ + \left(\frac{4S\lambda_Y\gamma_s\left(S\eta P\gamma_s + c_4\right)^2k_0}{9} + \eta Nc_4^2r\lambda_S\right)\eta e^{rt} + \frac{4k_0\left(S\eta P\gamma_s + c_4\right)^3}{27}\end{array}\right)$$

$$A_4 = 2Sk_0c_4^2\eta\lambda_Y\gamma_s)e^{rt} + \frac{2k_0(S\eta P\gamma_s + c_4)^3}{3}))^{1/3} + \left(\left(S\eta P\gamma_s + c_4\right)^2e^{-rt} + \lambda_Y\gamma_sS\eta\left(e^{rt}\lambda_Y\gamma_sS\eta + 2S\eta P\gamma_s + 2c_4\right)\right)k_02^{1/3})))$$

$$A_5 = r\lambda_S)^{1/2}\sqrt{3}c_4 + 2\lambda_Y^2\gamma_s^2S^2\eta k_0\left(S\eta P\gamma_s + c_4\right)\eta e^{2rt} + \frac{2e^{3rt}\lambda_Y^3\gamma_s^3S^3\eta^3k_0}{3} + \left(2S^3\lambda_Y\gamma_s^3\eta^3P^2k_0 + 4S^2k_0c_4P\lambda_Y\gamma_s^2 + 9Nc_4^2r\lambda_S\right)$$

$$A_6 = \left(\begin{array}{l}\frac{4S^2k_0\eta^2\gamma_s^2\lambda_Y^2\left(Sp\eta\gamma_s + c_4\right)e^{2rt}}{9} + \frac{4e^{3rt}S^3k_0\eta^3\lambda_S^3\gamma_s^3}{27} \\ + \left(\frac{4S\lambda_S\gamma_s\left(Sp\eta\gamma_s + c_4\right)^2k_0}{9} + \eta Nc_4^2r\lambda_S\right)\eta e^{rt} + \frac{4k_0\left(Sp\eta\gamma_s + c_4\right)^3}{27}\end{array}\right), \quad A_9 = \sqrt{3}c_4 + 2\lambda_Y^2\omega_S^2S^2\eta c_0(S\eta P\omega_S + c_4)$$

$$A_7 = 2S^3k_0\eta^3p^2\lambda_Y\gamma_s^3 + \left(4S^2k_0c_4\lambda_Y\gamma_s^2 + 9Nc_4^2r\lambda_S\right)\eta^2 + 2sk_0c_4^2\eta\lambda_Y\gamma_s, \qquad A_8 = S\eta P\gamma_s + e^{rt}S\eta\lambda_Y\gamma_s - 2k_4$$

III. RESULTS AND DISCUSSIONS

This section presents numerical simulations of Model (1) to illustrate how variations in key parameters and control inputs influence overall rice yields. We shall use the following initial values $S(0) = 20000$, $E(0) = 200$, $I(0) = 200$, $R(0) = 0$, $P(0) = 10000$ and $Y(0) = 0$. The specific parameters governing our analysis are detailed in Table 2, and the model's structural behaviour is illustrated in the figures that follow.

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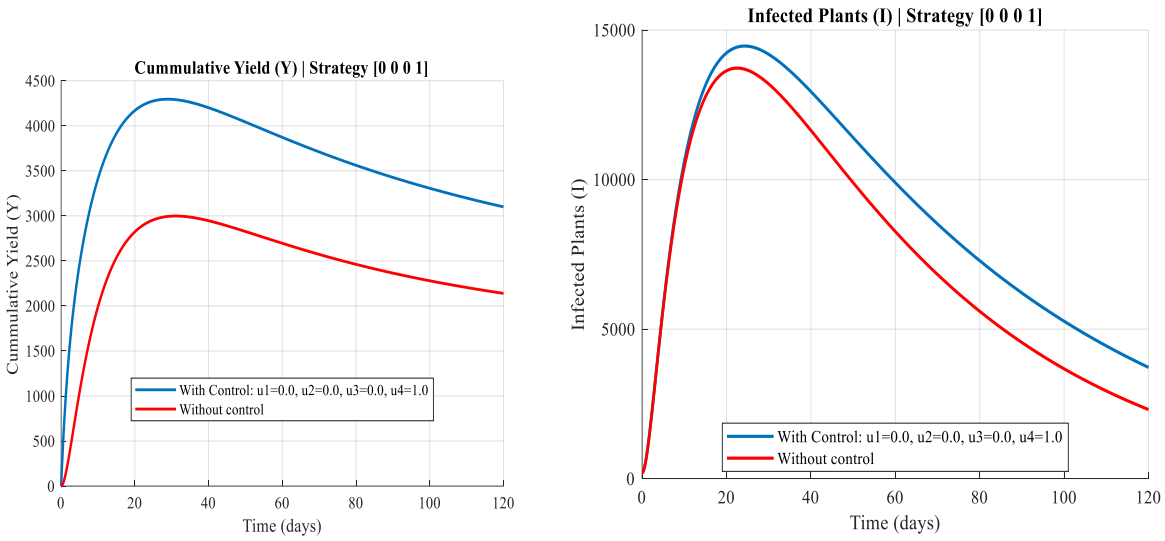
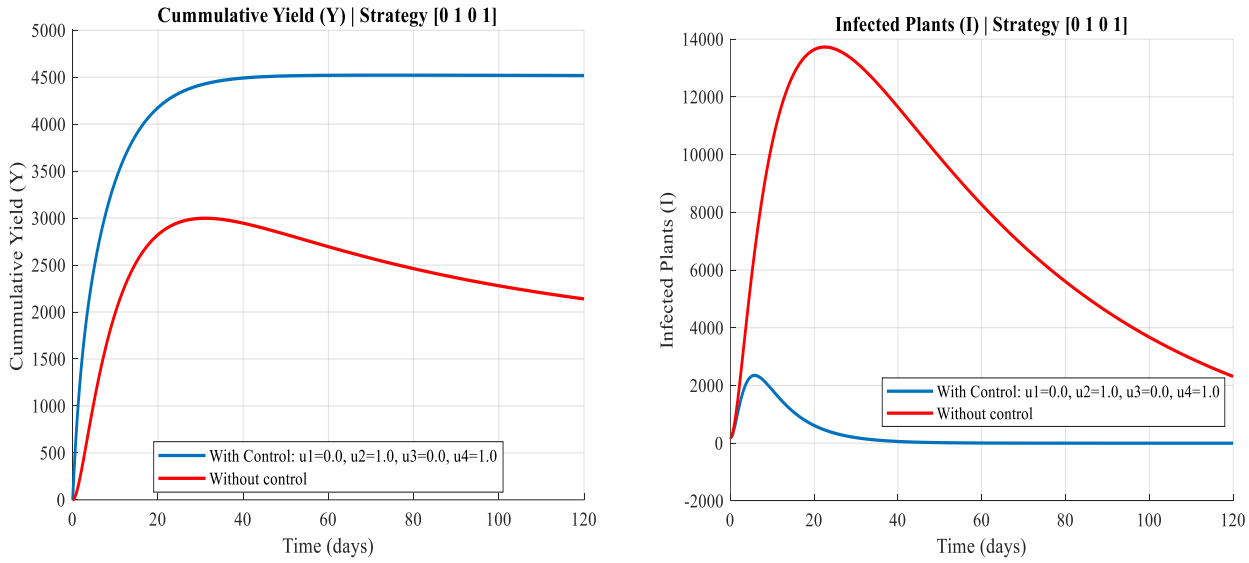


(a) (b)

Figure 1: Dynamics of the effect of Fertilizer/irrigation enhancement on cumulative yield (a) and Infected plant (b)



(a) (b)

Figure 2: Dynamics of the Effect of Fungicide Application Rate and Fertilizer/Irrigation Enhancement on Cumulative Yield (a) and Infected Plant (b)

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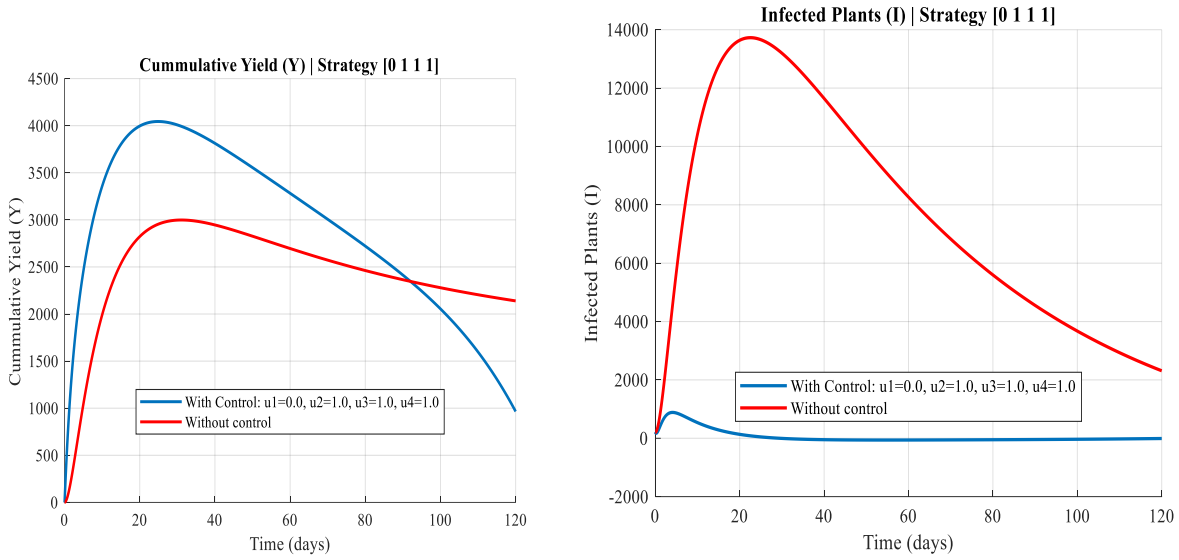
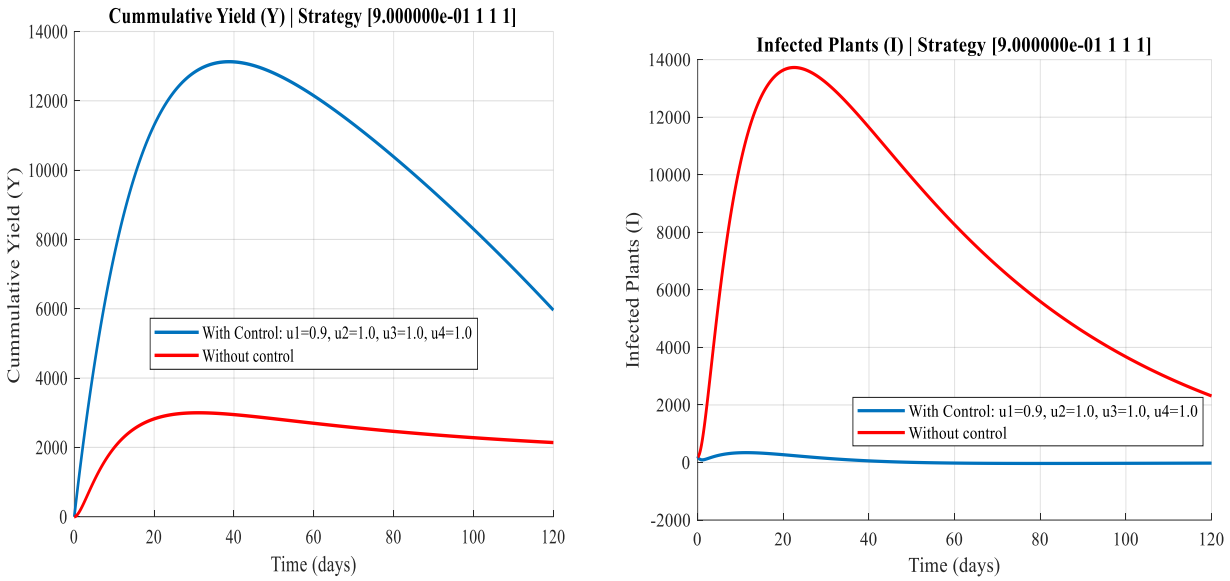


(a) (b)

Figure 3: Dynamics of the Effect of Fungicide Application Rate, Roguing Rate of Infected Rice Plants, and Fertiliser/Irrigation Enhancement on Cumulative Yield (a) and Infected plant (b)



(a) (b)

Figure 4: Dynamics of the Effect of Resistance Varieties Deployment, Fungicide Application Rate, Roguing Rate of Infected Rice Plants, and Fertiliser/Irrigation Enhancement on Cumulative Yield (a) and Infected Plant (b)

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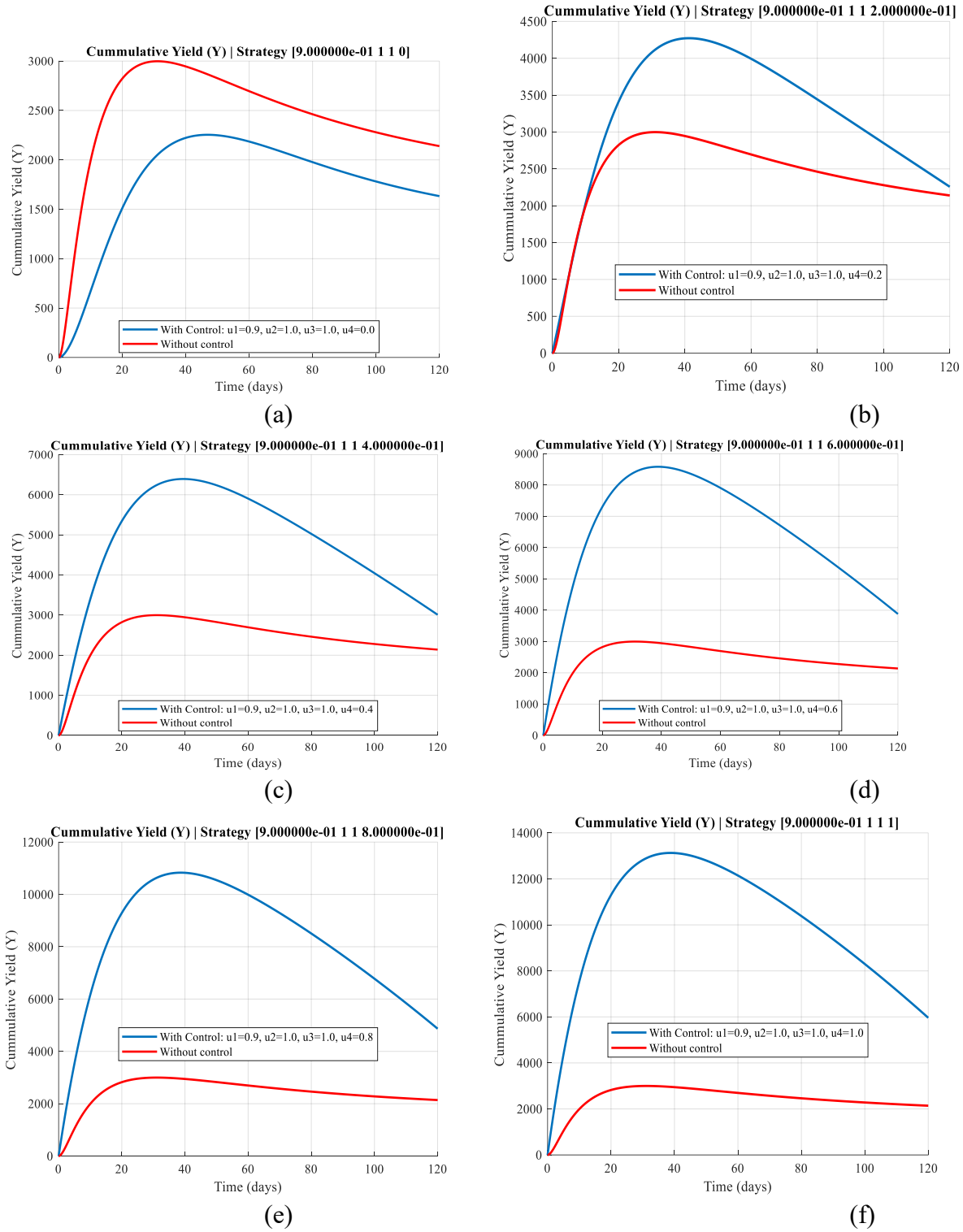

Figure 5: Dynamics of the increase of Fertilizer/Irrigation Enhancement while Observing all Control Measures on Cumulative Yield.

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In Figure 1, the strategy (Fertilizer/irrigation enhancement) represents a yield-maximizing but disease-tolerating approach. Fertilizer/irrigation enhancement alone does not suppress the rice epidemic; in fact, it worsens the infection rate, but the agronomic benefits more than compensate, resulting in substantially higher cumulative yields. This suggests that u_4 is not a rice blast control measure but rather a production-intensification input that operates orthogonally to disease dynamics.

The fungicide application and fertilizer/irrigation enhancement Strategy represents a highly effective integrated approach, as shown in Figure 2, where fungicide provides the essential foundation for disease suppression, and fertilizer/irrigation enhancement capitalizes on healthy crop status to maximize and stabilize yields. The epidemic is not simply managed but eradicated from the system, while cumulative yield reaches the highest level observed (approximately 4,500) and remains at that level indefinitely. This strategy has successfully reconciled disease control with production intensification.

Moreover, Figure 3 depicts the fungicide application, roguing, and fertilizer/irrigation enhancement strategy, which functions as a warning example of over-management. While the combination of fungicide, roguing, and fertilizer/irrigation achieves the lowest observed infection levels, it yields the worst long-term yield outcome among the strategies. There is no need of adding roguing to an already effective fungicide regime is vastly outweighed by the destructive impact of removing productive plants from the system.

Figure 4 shows that implementing all four control strategies simultaneously drives the system toward its maximum yield potential. This strategy yields near-total suppression of the disease, characterized by an approximate 97% reduction in peak infection and complete eradication by day 30. Under these conditions, crop yield peaks at roughly 13,000 units, a considerable improvement over the uncontrolled scenario, which plateaus at just 2,100 units. However, a post-peak decline of about 62% indicates that aggressive roguing (u_3) becomes economically inefficient, even when combined with genetic resistance of plants. Consequently, future optimization frameworks ought to investigate intermediate roguing intensities (e.g., $0.3 \leq u_3 \leq 0.5$). Moderating this parameter could minimize the financial and physical costs of plant removal while sustaining the high-efficiency baselines of u_1, u_2, and u_4, ultimately stabilizing the 13,000 peak yields through to the final harvest.

Lastly, in Figure 5, the data across the six visualizations track cumulative productivity over a 120-day cultivation cycle for early-maturing rice varieties, comparing managed regimes (u_1, u_2, u_3) with an unmanaged baseline (u_4) at varying levels of agricultural inputs. The results show that nutrient and water enhancement (u_4) is the primary driver of economic viability in this integrated management framework. When inputs are withheld ($u_4 = 0$), active intervention yields a negative return; the managed curve tops out at a mere 2,300, both lower and later than the unmanaged baseline (2,900). This happens because the compounding expenditures of cultivating resistant strains, spraying fungicides, and pulling infected crops are not offset by input-driven growth. This relationship reverses once u_4 reaches 0.2, at which point managed yields climb to 4,300, easily outperforming the unmanaged baseline of 3,000. This performance gap widens progressively with each incremental increase in u_4, stepping up significantly at $u_4 = 0.4$ (6,500 vs 3,000) and at $u_4 = 0.6$ (8,500 vs 3,000), before climbing to 11,000 vs 3,200 at $u_4 = 0.8$. At full input capacity ($u_4 = 1.0$), the managed system peaks at 13,000, while unmanaged productivity plateaus around 3,000. Crucially, the unmanaged baseline stays relatively static across all input levels. This indicates that intensifying irrigation and fertilization in the absence of disease suppression fails to boost output, likely because denser crop tops accelerate pathogen transmission and wipe out potential gains. Conversely, the managed curves show both higher peaks and steeper post-peak declines as inputs rise, indicating accelerated biomass production and earlier rice maturation, typical of intensive farming.

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Ultimately, u_4 serves as an operational catalyst for the remaining control variables (u_1–u_3). Mitigating disease yields zero or negative returns in resource-poor environments; it becomes highly profitable only when abundant water and nutrients enable protected plants to reach their full genetic potential. While this highlights a minimum input threshold for management strategies to become cost-effective, the shrinking ratio of peak output to input volume (from 21,500 at u_4=0.2 to 13,000 at u_4=1.0) reveals clear diminishing marginal returns. This points to an ideal input window of $0.4 \leq u_4 \leq 0.6$, where growers can secure a substantial yield premium (6,500–8,500) while optimizing input efficiency.

Future work will focus on calibrating the model with field data on Rice Blast incidence, particularly using outbreak records from Ebonyi State, Nigeria. Our findings further indicate that fertilizer and irrigation must be implemented alongside other intervention such as fungicide application and the use of resistance varieties to effectively determine disease suppression and tangible yield gains. This aligns with earlier studies [2, 11] which identified chemical control as the most effective strategy for managing Rice Blast.

IV. CONCLUSION

This study shows a mathematical model that predicts the dynamics of Rice Blast disease transmission. We establish the model's mathematical soundness and biological relevance by demonstrating the positivity and boundedness of its solutions. Furthermore, we determine the model's disease-free equilibrium point and derive the basic reproduction number (R_0) to evaluate the transmission potential. Also, a sensitivity analysis and an optimal control strategy are presented to guide effective intervention. The optimal control background provides a powerful, organized approach to managing rice blast disease by determining time-dependent intervention strategies that minimize both disease burden and associated costs. This approach fit in epidemiological dynamics with economic considerations, enabling decision-makers to identify the most productive allocation of limited resources among multiple control measures, including the use of resistant varieties, fungicide application, roguing of infected plants, and cultural practices such as fertilizer/irrigation management. By solving the optimality system derived from Pontryagin's maximum principle, one can obtain control profiles which adapt to the developing state of the epidemic, ensuring interventions are applied precisely when most effective. Based on the extensive analysis of all control strategies, the optimal approach for managing rice blast disease requires a carefully balanced integration of genetic resistance, chemical protection, and fertilizer/irrigation enhancement, without excessive roguing. Fungicide application (u_2) acts as the non-negotiable foundation, achieving approx. 84% reduction in infection, even in isolation, and epidemic elimination when combined with other measures. Resistance variety deployment (u_1) is the critical yield multiplier; without it, aggressive roguing becomes catastrophically destructive, collapsing final yields to approx. 950, whereas u_1 at 0.9 transforms the same strategy into the highest-yielding option, peaking at 13,000. Fertilizer and irrigation enhancement acts as the essential catalyst: at $u_4 = 0$, control investments are wasted and even counterproductive, but at $u_4 \geq 0.2$, it unlocks synergistic yield gains that scale monotonically with input level. The paradoxical finding is that the simplest strategy, fungicide plus fertilizer/irrigation without roguing, delivers the most stable outcome (approx. 4,500 yields with zero post-peak decline). In contrast, the most complex strategy (all control measures) maximizes peak production (approx. 13,000) but suffers a 62% decline due to persistent roguing penalties. Therefore, risk-averse producers should adopt [0 1 0 1] for predictable returns, while yield-maximizing producers should deploy [0.9 1 1 1] and explore reducing u_3 below 1.0 to reduce steep post-peak yield erosion.

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