SABA Publishing

**Journal of Mathematical Analysis and Modeling**
jmam.sabapub.com
ISSN 2709-5924

J Math Anal & Model
(2025)6(1) : 117-143
doi:10.48185/jmam.v6i1.1401

# A Mathematical Model for Effective Fungicide Use in Rice Blast Re-Infection

JIMRISE O. OCHWACH[b,*], BONFACE O. OBITA [a] MARK O. OKONGO [a],

[a] Chuka University, Chuka, Kenya
[b] Mama Ngina University College, Gatundu, Kenyas

• *Received: 10 November 2024* • *Accepted: 24 November 2024* • *Published Online: 15 June 2025*

## Abstract

Rice blast, caused by the pathogenic fungus *Magnaporthe oryzae*, poses a significant threat to rice production, especially in regions like Kenya where rice is both a staple food and a key economic commodity. This study introduces a comprehensive mathematical model designed to investigate the dynamics of rice blast re-infection following fungicide application on infected crops. The model is formulated as a system of ordinary differential equations (ODEs) that categorizes the rice population into five compartments: Susceptible, Protected, Infected, Recovered, and Secondary Infected. The positivity and boundedness of solutions were established, ensuring that the model is both mathematically and biologically well-posed. Using the stability theory of differential equations, the model was analyzed, and the basic reproduction number $R_0$ was derived via the Next Generation Matrix method. The existence of both the Disease-Free Equilibrium (DFE) and the Endemic Equilibrium Point (EEP) was demonstrated. Stability analysis revealed that the DFE is locally and globally stable when $R_0 < 1$, and unstable when $R_0 > 1$. Conversely, the EEP is locally asymptotically stable when $R_0 > 1$. Sensitivity analysis identified the fungicide application rate ($\pi$) as the most influential parameter in reducing rice blast re-infection. Numerical simulations were conducted to support the analytical findings, demonstrating that effective fungicide use can substantially decrease disease prevalence, thereby enhancing rice yield and promoting sustainable agricultural practices. This study contributes meaningfully to the field of plant disease modeling and provides a robust framework for future research into the epidemiology and management of crop diseases.

Keywords: Rice Pest, Mathematical modelling, Rice blast, Stability analysis, Plant pest model, pest control.

## 1. Introduction

Mathematical modeling has emerged as a powerful and indispensable tool for describing and analyzing complex real-world processes. Over the past several decades, this field has undergone rigorous development and extensive investigation by researchers, yielding profound insights and practical solutions. By translating real-world phenomena into mathematical equations, these models enable systematic analysis, predictive capabilities,

*Jimrise O. Ochwach: ojimrise09@gmail.com

and informed decision-making 21.

A key application of mathematical modeling lies in the study of infectious diseases, where it has proven invaluable in designing strategies for control, eradication, and investigation. Its interdisciplinary nature extends to various fields, with mathematical biology gaining significant traction among researchers. Furthermore, the integration of mathematical modeling into agriculture has fostered precision-driven approaches, particularly in crop disease management, addressing critical challenges in food security [3, 16].

The utility of mathematical models extends across disciplines, including music, philosophy, and the social sciences [22]. These models are instrumental in understanding component interactions, explaining system dynamics, and forecasting behavior. Recent advancements have expanded their application into fractal calculus, which has been employed to explore phenomena ranging from SARS-CoV-2 dynamics [1] to financial systems and prey-predator interactions [22]. In the context of health, models have provided critical insights into the dynamics of respiratory infections and HIV/AIDS [18, 25, 9, 24]. Agricultural challenges, such as the control of the false codling moth, have similarly benefited from mathematical approaches [16, 15, 11].

Re-infection poses a significant challenge in disease management, both in public health and agriculture. For instance, Cohen et al. [5] modeled tuberculosis re-infection, demonstrating how a small proportion of infected individuals develop primary disease while others reactivate latent infections over time. Similarly, Wangari et al. [24] studied re-infection dynamics in COVID-19, showing that the stability of infection-free equilibria depends on the basic reproduction number ($R_o$). These studies underscore the importance of robust mathematical frameworks in exploring re-infection dynamics, a relatively underexplored area in agricultural contexts.

In agriculture, where uncertainty is prevalent, reliable and timely forecasting is essential. Crop diseases are among the leading causes of yield reduction, emphasizing the need for prior knowledge of disease onset and severity to mitigate losses. Crop modeling provides not only advanced yield predictions but also early warnings of disease outbreaks, enabling the implementation of appropriate protective measures.

Rice (*Oryza sativa L.*), a staple food for nearly half of the global population, exemplifies the significance of such models. In Kenya, where rice ranks as the third most important crop, its production faces significant threats from diseases like rice blast, caused by the fungus *Pyricularia oryzae* [6]. Reports indicate yield losses of up to 50% in western Kenya, with some fields experiencing losses exceeding 70–80% [15, 14]. Symptoms include lesions on plant shoots, stems, and panicles, leading to severe yield losses, particularly during critical growth stages such as panicle formation [8].

Efforts to manage rice blast have included the application of fungicides, such as Azoxystrobin and Tricyclazole, which have shown effectiveness in reducing disease severity [12, 10]. However, these studies often overlook the dynamic nature of the fungus and the

potential for re-infection. To address these gaps, Castle and Gilligan [4] developed a model exploring fungicide application dynamics, while Anggriani [2] extended this work by integrating the curative and protective functions of fungicides. Despite these advances, assumptions about permanent fungicide effects and immunity after treatment limit the models' applicability to diseases like rice blast, which can recur and cause substantial yield losses.

Given the widespread occurrence and economic impact of rice blast, there is a pressing need for comprehensive mathematical models that capture the dynamics of re-infection following fungicide application. This study aims to develop such a model, contributing to the effective management of rice blast and supporting the sustainability of rice production in Kenya.

## 2. Model Formulation

This study developed a mathematical model to optimize fungicide application in reducing the re-infection of rice blast. The model is formulated using a system of ordinary differential equations (ODEs), with the rice crop population classified into five distinct compartments:

S: The susceptible rice crop, representing seedlings transplanted from the nursery.

P: The rice crop protected by fungicide.

I: The rice crop infected by rice blast for the first time.

R: The recovered rice crop following the application of fungicide on infected plants.

X: The rice crop experiencing secondary infection by the rice blast pathogen.

The model incorporates a recruitment rate $g(s)$, representing the rate at which seedlings are transplanted from the nursery into the system. Additionally, $h$ denotes the natural removal rate across all compartments. This compartmental structure and associated parameters provide a framework for analyzing the dynamics of fungicide effectiveness and the progression of rice blast infections.

### *2.1. Assumptions of Model Development*

1 The fungal disease spread through one population
2 There is a simple density dependent growth of the host up to a carrying capacity;
3 A protectant fungicide affects susceptible hosts, reducing their capacity to become infected;
4 The population is divided into five classes, susceptible, S, infected, I protected, P, recovered, R, and the secondarily infected, X.
5 Primary and secondary infection does not occur at the same time

The seedling from the nursery represented by g(s) leads to production of susceptible class, S. When fungicides are applied on the susceptible rice crops, they become protected of which some protected crops may come back to susceptible class depending on the effectiveness of fungicide applied.

Primary infection takes place when there is interaction between the susceptible and the infected leading to the compartment of the infected (I). On application of fungicide on infected rice crops, some may recover fully from the blast hence class(R) which can again be infected hence goes back to susceptible class.

Primarily infected crops in class (I) may later have the disease spread to other parts of the plant or inner tissues of the plant. This result into secondary infection class(X) of which the parts infected may decay hence class (cX) leading to low produce of the infected plant. Natural death rate affect all classes.

Density of susceptible host, S is produced from seedlings from nursery represented by g(s) and their number also increased by seedlings initially protected by fungicide whose effectiveness has expired $(\epsilon P)$ and the recovered infected host that are susceptible to the disease again $(\mu R)$. The susceptible class is reduced by natural death rate(hS) , the effectiveness of the fungicide applied hence protecting the crop $(\pi S)$ leading to class (P) and primary infection brought by interaction between susceptible and the infected crops $(\beta_p SI)$. This leads to equation 2.1:

$$\frac{dS}{dt} = g(s) + \epsilon P + \mu R - \beta_p SI - \pi S - hS \tag{2.1}$$

Density of the infected host (I) is as a result of interaction between susceptible and infected and the number of infected class is reduced by natural death rate (hI) and recovered initially infected crops (mI) hence equation 2.2:

$$\frac{dI}{dt} = \beta_p SI - mI - aI - hI \tag{2.2}$$

Density of recovered host is produced by total effectiveness of fungicide applied on infected rice crop which may again be susceptible to blast again and the number minimized by death rate as shown by equation 2.3:

$$\frac{dR}{dt} = mI - hR - \mu R \tag{2.3}$$

Density of secondary infected crops are as a result of blast attacking rice crop for the second time since blast may attack the leaves and later the neck just before heading which may result to decay of part infected (cX) leading to low produce hence equation 2.4:

$$\frac{dX}{dt} = aI - hX - cX \tag{2.4}$$

Density of protected crops are as a result of fungicide applied to susceptible rice crop for a given effective period after which they may be attacked by rice blast hence equation 2.5:

$$\frac{dP}{dt} = \pi S - \epsilon P - hp \tag{2.5}$$

The five equations can therefore be written as,

$$\begin{aligned}
\frac{dS}{dt} &= g(s) + \epsilon P + \mu R - \beta_p SI - \pi S - hS \\
\frac{dI}{dt} &= \beta_p SI - mI - aI - hI \\
\frac{dR}{dt} &= mI - hR - \mu R \\
\frac{dX}{dt} &= aI - hX - cX \\
\frac{dP}{dt} &= \pi S - \epsilon P - hp
\end{aligned} \tag{2.6}$$

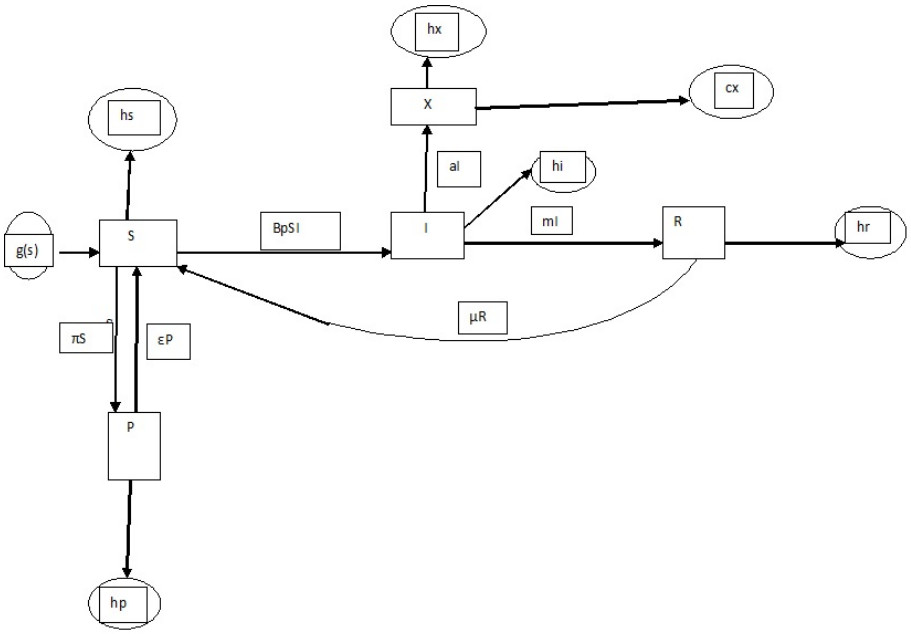


Figure 1: Model Flow Chart

### *2.2. Model Parameter and Values*

| Parameter | Definition | Source | Source |
|---|---|---|---|
| $\mu$ | co-efficient for R going to S | 0.5 | Aminiel et al(2015) |
| $\epsilon$ | rate of damage of fungicide given to plant | 0.0001 | Estimated |
| $\pi$ | rate of fungicide applicatio | 0.1 | Angriani(2018) |
| $\beta_s$ | rate of secondary infection | 0.075 | Angriani(2018) |
| $\beta_p$ | rate of primary infection | 0.00005 | Estimated |
| h | rate of removal of all classes | 0.00025 | Estimated |
| c | rate of decay of inoculum | 0.8 | Angriani(2018) |
| g(s) | production of susceptible | 0.95 | Estimated |
| a | rate of secondary infection | 0.2 | Estimated |
| m | rate of recovery of infected host | 0.9 | Estimated |

Table 1: Model parameters and Values

## 3. Model Analysis

### *3.1. Positivity and Boundedness of Solution*

This study establishes the well posedness of the model by showing that its solutions are positive and bounded.A system is well posed if the solutions of a system remain non-negative for all non-negative initial conditions,the solution exists, unique and depends on the model parameters and the initial conditions [20]. The model (2.6) is replicated below for easy reference.

$$\begin{aligned}
\frac{dS}{dt} &= g(s) + \epsilon P + \mu R - \beta_p SI - \pi S - hS \\
\frac{dI}{dt} &= \beta_p SI - mI - aI - hI \\
\frac{dR}{dt} &= mI - hR - \mu R \\
\frac{dX}{dt} &= aI - hX - cX \\
\frac{dP}{dt} &= \pi S - \epsilon P - hp
\end{aligned} \tag{3.1}$$

### *3.2. Boundedness of Solution*

The study shows that the solutions are bounded in invariant region $\gamma$ where $\gamma = (S, I, R, X, P) : N \leqslant \frac{g(s)}{h}$

**Theorem 1** The solutions of the model are contained in the feasible region $\gamma$

**Proof**

Adding the system of five equations , we have:

$N = S + I + R + X + P$

$$\frac{dN}{dt} = [g(s) + \epsilon P + \mu R - \beta_p SI - \pi S - hS] + [\beta_p SI - mI - aI - hI] +$$
$$[mI - hR - \mu R] + [aI - hX - cX] + [\pi S - \epsilon P - hP]$$
$$\frac{dN}{dt} = g(s) - h(S + I + R + X + P) - cX$$
$$\frac{dN}{dt} = g(s) - hN - cX$$
$$\frac{dN}{dt} < g(s) - hN$$
$$\frac{dN}{dt} + hN \leqslant g(s)$$

Using the integrating factor $e^{ht}$ to solve

$$N \leqslant \frac{g(s)}{h} + Ce^{-ht}$$

At $t = 0$

$$N(0) - \frac{g(s)}{h} < C$$

Substituting

$$N \leqslant \frac{g(s)}{h} + N(0) - \frac{g(s)}{h}e^{-ut}$$

Where N(0) is the initial population As $t \longrightarrow \infty$

Which implies

$$0 \leqslant N \leqslant g(s)$$

Therefore the solutions are bounded in the invariant region

$$\gamma = S(t), I(t), R(t), X(t), P(t) \in R_+^5 : S(t) + I(t) + R(t) + X(t) + P(t) \leqslant 0$$

*3.3. Positivity of Solutions*

Since the model considers a plant population, we assume that all parameters are non-negative and that all the solutions with positive initial values will remain positive for $t \geqslant 0$

**Theorem 2** Let the initial conditions be $S(0) \geqslant 0$,$I(0) \geqslant 0$,$R(0) \geqslant 0$,$X(t) \geqslant 0$ and $P(0) \geqslant 0 \in \gamma$, then the solutions S(t),I(t),R(t),X(t) and P(t) of system 2.1 are positive for all $t \geqslant 0$

**Proof**

From the first equation of 2.6

$$\frac{dS}{dt} = g(s) + \epsilon P + \mu R - \beta_p SI - \pi S - hS$$

$$\frac{dS}{dt} \geqslant -(\beta p + \pi + h)S$$

By separation of variables and integrating both sides

$$\frac{\int dS}{dt} \geqslant \int -(\beta_P I + \pi + h)dt$$

$$\ln S(t) \geqslant -(\beta_P I + \pi + h)t + C$$

$$S(t) \geqslant \exp^{-}(\beta_P I + \pi + h)t \times \exp^{c}$$

Taking $\exp^{c}$ to be A

$$S(t) \geqslant A \exp^{-(\beta_P I + \pi + h)t}$$

Using the initial conditions $t = 0$, $S(0) = A$ ,
Substituting A

$$S(t) \geqslant S(0) \exp^{-}(\beta_P + \Pi + h)t \tag{3.2}$$

Therefore
$S(t) \geqslant 0$ for all $t = 0$ for all $t \geqslant 0$
From the above it follows that from equations two,three, four and five respectively that $I(t) \geqslant 0$,$R(t) \geqslant 0$,$X(t) \geqslant 0$ and $P(t) \geqslant 0$ for all $t \geqslant 0$

*3.4. Equilibria Analysis*

An equilibrium point is defined by [19] as a constant solution of a model system. The equilibrium points of a model system are obtained by setting the right hand side of differential equations to zero and solving each to get a constant solution. Epidemiological models usually have two equilibrium points, namely, the disease free equilibrium and endemic equilibrium. The existence of equilibrium points points of the model is determined with respect to the basic reproduction number, derived using the next generation matrix approach. Stability analysis of the model is done to determine the conditions for the spread of blast re-infection.

*3.4.1. Disease Free Equilibrium* $(E_o)$

The disease free equilibrium $(E_o)$ is a point where the disease is not present in the population and therefore $\frac{dI}{dt}, \frac{dR}{dt}, \frac{dX}{dt}$ and $\frac{dP}{dt} = 0$
Considering system 2.6 when there is no blast, we get

$$\begin{aligned}
\frac{ds}{dt} &= g(S) - h(S) \\
\frac{dI}{dt} &= 0 \\
\frac{dR}{dt} &= 0 \\
\frac{dX}{dt} &= 0 \\
\frac{dP}{dt} &= 0
\end{aligned} \tag{3.3}$$

Solving the first equation 4.1

$$0 = g(S) - hS$$

$$S = \frac{g(s)}{h}$$

Thus the disease free equilibrium of the system $(E_o)$ is given by $(\frac{g(s)}{h}, 0, 0, 0, 0)$

*3.4.2. Basic Reproduction Number*

Basic Reproduction Number,$(R_0)$ is defined by [Ochwach et al., 7] as the average number of secondary infections caused by a single infections individual during his or her entire life time as an infective when introduced into purely susceptible population. In this study secondary infection is treated as blast re-infection. The basic reproduction number measures the potential spread of blast re-infection within rice population. If $R_0 < 1$, then each re-infected rice crop will produce less than one re-infected rice crop on average hence re-infection of rice blast will not spread.

On the other hand when $R_0 > 1$ , then every re-infected rice crop in its entire life will cause more than one re-infections on average hence re-infection invades entire population.In this study we seek for $R_0 < 1$ so that we control blast re-infection spread in the rice population . This study uses Next Generation Matrix to derive $R_0$ [23] Consider the next generation matrix made up of matrices F and V, such that

$$G = FV^1$$

$$F = \frac{\delta f_i}{\delta x_j(x_o)}$$

$$V = \frac{\delta v_i}{x_j(x_o)}$$

Where $x_o$ is DFE point.
$f_i$ is the re-infection matrix(rate of appearence of new re-infections in compartment i)
$v_i$ is the transition matrix( rate of transfer of individuals from compartment i by all other means)

The basic reproduction number is given as the dominant eigenvalue

$$R_o = \rho FV^{-}1$$

The re-infection compartments are I and X. We use second and the fourth equations of 4.2.1 to compute $R_O$

$$\frac{dI}{dt} = \beta_p SI - mI - aI - hI$$

$$\frac{dX}{dt} = aI - (C + h)$$

$$F_i = \begin{pmatrix} \beta_p SI \\ aI \end{pmatrix}$$

$$V_i = \begin{pmatrix} (m+a+h)I \\ (c+h)X \end{pmatrix}$$

Calculating the Jacobian matrix at the disease free equilibrium $(\frac{g}{h}, 0, 0, 0, 0)$

$$F = \begin{pmatrix} \beta_p \frac{g(s)}{h} & 0 \\ a & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} m+a+h & 0 \\ 0 & c+h \end{pmatrix}$$

On solving the inverse of the matrix V, we get

$$V^1 = \begin{pmatrix} \frac{1}{m+a+h} & 0 \\ 0 & \frac{1}{c+h} \end{pmatrix}$$

Therefore

$$FV^{-}1 = \begin{pmatrix} \beta_p \frac{g(s)}{h(m+a+h)} & 0 \\ \frac{a}{m+a+h} & 0 \end{pmatrix}$$

$$= \begin{pmatrix} \frac{\beta_p g(s)}{hm+ha+h^2} & 0 \\ \frac{a}{m+a+h} & 0 \end{pmatrix}$$

Eigen values of $FV^{-}1$ are

$$\begin{pmatrix} 0 & \frac{\beta_p g(s)}{h^2+ha+hm} \end{pmatrix}$$

$$R_0 = \rho FV^{-}1$$

$$R_0 = \frac{\beta_p g(s)}{h^2 + ha + hm} \tag{3.4}$$

*3.5. Stability Analysis*

*3.5.1. Local Stability Analysis of Disease Free Equilibrium*$(E_o)$

To determine the local stability of Disease Free Equilibrium point, the variation Jacobian matrix at equilibrium point $JE_o$ of the model system 2.6 is developed and is given by:

$$JE_o = \begin{pmatrix} -(\pi+h) & \frac{-\beta_p g}{h} & \mu & 0 & \epsilon \\ 0 & \beta_p \frac{g}{h} - (m+a+h) & 0 & 0 & 0 \\ 0 & m & -(h+\mu) & 0 & 0 \\ 0 & a & 0 & -(h+c) & 0 \\ \pi & 0 & 0 & 0 & -\epsilon - h \end{pmatrix}$$

The stability of the Disease Free Equilibrium point can be analysed by studying the behaviour of $JE_o$. All its eigenvalues should have negative parts for local stability of Disease Free Equilibrium. By using mathematica software, we have the following eigenvalues:

$$\lambda_1 = -(c + h)$$

$$\lambda_2 = -h$$

$$\lambda_3 = -(a + m + h - \beta_p \frac{g}{h})$$

$$\lambda_4 = -(h + \Pi + \epsilon)$$

$$\lambda_5 = -(h + \mu)$$

Condition
For the root $\lambda_3$ to be negative

$$a + m + h \leqslant \beta_p \frac{g}{h}$$

The following result is a proof of local stability of the disease free equilibrium and the proof applies techniques used in [23].

**Theorem 3**

For disease free equilibrium to be locally asymptotically stable $R_0 < 1$

**Proof** The proof applies [23] techniques. One of the eigen values of the $JE_o$ is $\lambda 1 = \frac{-ch-h^2}{h}$

we find other eigen values by checking the signs of the eigenvalues of the block matrix given by,

$$\begin{pmatrix} \frac{\beta_p g(s)}{h-m-a-h} & 0 \\ a & -c-h \end{pmatrix}$$

Now let $T_r$ be Trace of A and K be the determinant of A and consider the linear system $x'(t) = A \times t$ where

$$A = \begin{pmatrix} a & b \\ c & d \end{pmatrix}$$

The following conditions can be shown

a )If $K < 0$ , the characteristic roots of A will have opposite signs

b ) If $K > 0$ and $\delta = Tr - 4K \geqslant 0$, the characteristic roots of matrix A will have same sign.
The roots will be negative if $Tr < 0$ and positive if $Tr > 0$

c ) If $K > 0$ ,$\delta < 0$ and $Tr \neq 0$, then the characteristic roots of A will be imaginary with negative real part if $Tr < 0$ and a positive real part if $Tr > 0$

d ) If $K > 0$ and $Tr = 0$, matrix A will have purely imaginary roots.

The eigen values of matrix A are obtained from characteristic equation

$$\lambda^2 - (a+d)\Lambda + (ad - bc) = 0$$
$$\lambda^2 - Tr\lambda + K = 0$$
$$\frac{\lambda = Tr \pm \sqrt{Tr^2 - 4k}}{2}$$

Thus

a ) If $K < 0$ , there exist two real eigen values of opposite signs
b ) If $K > 0$ and $\delta \geqslant 0$, there exist two real eigenvalues of the same sign as the Trace.

c ) If $K > 0$ ,$\delta < 0$ and $Tr \neq 0$,there exist two complex conjugate eigenvalues , $\Lambda = P \pm ir$
d ) If $K > 0$ and $Tr = 0$, there exist two purely imaginary complex conjugate eigenvalues

Using condition b) we can now determine the signs of the other eigenvalues. For the two remaining eigenvalues to be negative, then K< 0 and Tr $< 0$ . We now find conditions that make determinant and trace negative.

$$\frac{(\beta_p g(s)}{h} - (m - a - h)(-c - h)$$

$$\frac{\beta_p g(s)}{h} - (m + a + h)[-c - h]$$

$$\frac{-c(\beta_p g(s)}{h} - (m - a - h) - \frac{h(\beta_p g(s)}{h} - (m - a - h)$$

$$\frac{-c\beta_p g(s)}{h} + cm + ca + ch - \beta_p g(s) + mh + ma + h^2$$

$$= c[m + a + h] + h[m + a + h] - \frac{[c\beta_p g(s)}{h} + \beta_p g(s)] \tag{3.5}$$

For determinant to be greater than one

$$c\beta_p \frac{g(s)}{h} + \beta_p g(s) < c(m + a + h) + h(m + a + h)$$

$$\frac{c\beta_p g(s)}{h} + \beta_p g(s) < (c + h)(m + a + h) \tag{3.6}$$

Dividing both sides by $(c + h)(m + a + h)$

$$\frac{C\beta_p g(s) + h\beta_p g(s)}{h(c+h)(m+a+h)} < 1$$

$$\frac{\beta_p g(s)(c+h)}{h(c+h)(m+a+h)} < 1$$

$$\frac{\beta_p g(s)}{h^2 + ha + hm} < 1 \tag{3.7}$$

Thus $R_0 < 1$

The trace of the block matrix is given by

$$\frac{[\beta_p g(s)}{h} - m - a - h] + (-c - h)$$

$$= \frac{\beta_p g(s)}{h} - m - a - c - 2h$$

$$= \frac{\beta_p g(s) - hm - ha - hc - 2h^2}{h} \tag{3.8}$$

If we make $\beta_p g(s)$ the subject of the formula from the basic reproduction number we get

$$\frac{\beta_p g(s)}{h^2 + ha + hm} = R_0$$

$$\beta_p g(s) = R_o(h^2 + ha + hm) \tag{3.9}$$

Substituting 3.5 and 3.6 gives

$$R_0(h + a + m) - m - a - c - 2h$$

The trace needs to be negative for us to have negative eigenvalues. Therefore for 3.7 to be negative, Ro $< 1$

It can be seen that $JE_O$ has negative eigenvalues only when $R_0 < 1$ and therefore disease free equilibrium is locally asymptotically stable.

*3.6. Global Stability Analysis of Disease Free Equilibrium*$(E_O)$

We use the Castillo- Chaves theorem [23, 11] to investigate the global asymptotic stability of the disease free state. We re-write model system 2.6 as

$$\frac{dx}{dt} = F(X, Z) \tag{3.10}$$

$$\frac{dZ}{dt} = G(X, Z), G(X, 0) = 0$$

Where $X = (S, R, P) \in R^3_+$

denotes non-infectious compartments and $Z = (I, X) \in R^2_+$ denotes infectious classes. The disease free equilibrium of the system now becomes $E_O = (X^*, 0), X^* = \frac{g(S)}{h}$ . To guarantee local asymptotic stability, the following two conditions must be met.

1 $\frac{dX}{dt} = F(X,0), X^*$ is globally asymptotically stable. (GAS)

2 $G(X,Z) = TZ - \hat{G}(X,Z) \geqslant 0$ for $(X,Z) \in T$

Where $T = D_z G(X^*,0)$ is an m matrix( the off element diamond element of T are non-negative) and T is the region where the model is biologically meaningful.

If the system satisfies 1 and 2 then the following theorem holds.

**Theorem 4**

The fixed point $E_o = (X^*,0)$ is globally asymptotic stable equilibrium of 4.4 provided that $R_0 < 1$ and assumptions 1 and 2 are satisfied.

**Proof**

Considering the model system 4.4, we have

$$F(X,Z) = \begin{pmatrix} g(s) + \epsilon P + \mu R - \beta_p SI - \pi S - hS \\ mI - hR - \mu R \\ \Pi S - \epsilon P - hP \end{pmatrix}$$

$$G(X,Z) = \begin{pmatrix} \beta_p SI - mI - aI - hI \\ aI - hX - cX \end{pmatrix}$$

Now

$$F(X,0) = \begin{pmatrix} g(s) + \epsilon P + \mu R - \pi S - hS \\ -hR - \mu R \\ \Pi S - \epsilon P - hP \end{pmatrix}$$

It is clear that $E_0$ is GAS of $\frac{dX}{dT} = F(X,0)$ Hence 1 is satisfied

Now consider condition 2

$$G(X,Z) = \begin{pmatrix} \beta_p SI - mI - aI - hI \\ aI - hX - cX \end{pmatrix}$$

$$G(X,0) \geqslant 0$$

$$T = \begin{pmatrix} \beta_p \frac{g(s)}{h} - m - a - h & 0 \\ a & -c - h \end{pmatrix}$$

$$Z = \begin{pmatrix} I \\ X \end{pmatrix}$$

$$TZ = \begin{pmatrix} \beta_p I \frac{g(s)}{h} - mI - aI - hI \\ aI - cX - hX \end{pmatrix}$$

*3.7. Existence of Endemic Equilibrium Point* $(E_a^*)$

The endemic equilibrium point is obtained when the basic reproduction number is greater than one and therefore blast re-infection spreads in the population. This study denote endemic equilibrium point as $E_a^* = (S^*, I^*, X^*)$. To find this equilibrium point, we equate the right hand side of 4.2.1 to zero

$$g(s) + \epsilon P^* + \mu R^* - \beta_p S^* I^* - \pi S^* - hS^* = 0$$

$$\beta_p S^* I^* - mI^* - aI^* - hI^* = 0 \tag{3.11}$$

$$aI^* - hX^* - cX^* = 0$$

For the existence and uniqueness of endemic equilibrium point $E_a^* = (S^*, I^*, X^*)$ the conditions $S^* > 0$, or $I^* > 0$, or $X^* > 0$ must be satisfied. Solving for $S^*, I^*, X^*$ we get

$$S^* = \frac{g(s) + \epsilon P^* + \mu R^*}{\beta_p I^* + \pi + h}$$

$$I^* = 0$$

$$X^* = \frac{aI^*}{(h+c)}$$

Additive compound matrix approach [13] is used to analyse the local stability of the endemic equilibrium given by $E_a^* = (S^*, I^*, X^*)$ in $\gamma$. Local stability of endemic equilibrium is determined by variation matrix $J(E_a^*)$ of the non linear system.

$$J(E_a^*) = \begin{pmatrix} -(\beta_p I^* + \pi + h) & -\beta_p S^* & 0 \\ \beta_p I^* & -(\beta_p S^* + m + a + h) & 0 \\ 0 & a & -(h+c) \end{pmatrix}$$

**Lemma 1**. Let $J(E_a^*)$ be the variational matrix corresponding to $E_a^*$. If $tr(J(E_a^*))$, $det(J(E_a^*))$ and $det(J^(2)(E_a^*))$ are all negative, then all the eigenvalues of $J(E_a^*)$ have negative real parts.

**Theorem 4.5**. If $R_0 > 1$, the endemic equilibrium $E_a^*$ of the model 4.2.1 is locally asymptotically stable in $\gamma$

**Proof**

From Jacobian matrix $J(E_a^*)$ we have $tr(J(E_a^*)) = -(\beta_p I + \beta_p S + \Pi + 3h + m + a + c)<0$

$$det(J(E_a^*)) = -(c+h)(I^*S^*\beta_p^2 + (-h-\pi-I^*\beta_p)(-a-h-m-\beta_p S^*))$$

Hence the trace and determinant of the jacobian matrix $J(E_a^*)$ are all negative.

**Lemma 2**. Let M and N be subset of $J^(2)(E_a^*)$. The (M,N) entry of $U_ij(J^(2)(E_a^*)$ is the coefficient of K in the expansion of the determinant of the sub-matrix of $J(E_a^*) + KI$ index by row in M and column in N

**Proof**

The sub- matrix of $J(E_a^*) + KI$ is given as

$$J(E_a^*) = \begin{pmatrix} -(\beta_p I^* + \pi + h) + K & -\beta_p S^* & 0 \\ \beta_p I^* & -(\beta_p S^* + m + a + h) + K & 0 \\ 0 & a & -(h+c) + K \end{pmatrix}$$

The sub-matrix of $J(E_a^*) + KI$ indexed by rows and columns is given by:

$$\begin{pmatrix} -(\beta_p I^* + \pi + h) + K & -\beta_p S^* \\ \beta_p I^* & -(\beta_p S^* + m + a + h) + K \end{pmatrix}$$

The co-efficient of K in the determinant of this matrix is

$$-\beta_p I^* - \pi - 2h - \beta_p S^* - m - a$$

and thus the (1,1) entry of $U_ij$ is

$$-\beta_p I^* - \pi - 2h - \beta_p S^* - m - a$$

Other entries were obtained by same method. Entry (1,2) is given by

$$\begin{pmatrix} -(\beta_p I^* + \pi + h) + K & 0 \\ \beta_p I^* & 0 \end{pmatrix}$$

The co-efficient of K in the determinant is

0

Entry (1,3)

$$\begin{pmatrix} -\beta_p S^* & 0 \\ -(\beta_p S^* + m + a + h) + K & 0 \end{pmatrix}$$

The co-efficient of this matrix is

0

Entry (2,1)

$$\begin{pmatrix} -(\beta_p I^* + \pi + h) + K & -\beta_p S^* \\ 0 & a \end{pmatrix}$$

The co-efficient of K is a Entry (2,2)

$$\begin{pmatrix} -(\beta_p I^* + \pi + h) + K & 0 \\ 0 & -(h + c) \end{pmatrix}$$

The co-efficient of K in the determinant of this matrix is $-h - c$ Entry (2,3)

$$\begin{pmatrix} -\beta_p S^* & 0 \\ a & -(h + c) + K \end{pmatrix}$$

The co-efficient of K in the determinant of this matrix is
$-\beta_p S$

Entry (3,1)

$$\begin{pmatrix} \beta_p I^* & -(\beta_p S^* + m + a + h) + K \\ 0 & a \end{pmatrix}$$

The co-efficient of K in the determinant of this matrix is 0
Entry (3,2)

$$\begin{pmatrix} -(\beta_p I^* & 0 \\ 0 & -(h + c) + K \end{pmatrix}$$

The co-efficient of K in the determinant of this matrix is $\beta_p I$
Entry (3,3)

$$\begin{pmatrix} -(\beta_p S^* + m + a + h) + K & 0 \\ a & -(h + c) + K \end{pmatrix}$$

The co-efficient K in the determinant of this matrix is $-\beta_p S - m - a - 2h - c$

Therefore $J^{(}2)(E_a^2)$ is

$$\begin{pmatrix} -\beta_p I^* - \pi - \beta_p S - a - m - 2h & 0 & 0 \\ a & -h - c & -\beta_p S \\ 0 & \beta_p I & -\beta_p S - m - a - c - 2h \end{pmatrix}$$

$$\det(J^{(}2)(E_a^*)) = -(a+2h+m+\pi+\beta_p I+\beta_p S)(ac+c^2+ah+3ch+2h^2+cm+hm+cS\beta_p+hS\beta_p+IS\beta_p^2)$$

Thus according to Lemma 1, the disease Endemic Equilibrium Point $E_a^*$ of the model is locally asymptotically stable

### *3.8. Numerical Illustration of Stability Points of the Model*

To illustrate the disease free equilibrium and endemic equilibrium, numerical simulations of $R_O < 1$ and $R_O > 1$ are shown in the figure 2 and 3

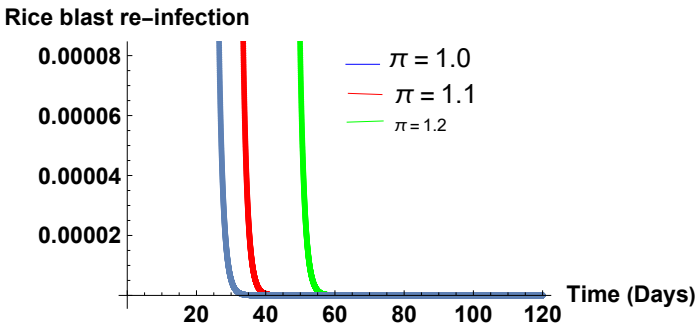


Figure 2: analysis of $R_O < 1$

Figure 2 shows that when $R_O < 1$, all trajectories of rice blast re-infection converges to zero regardless of values of $\pi$. Therefore disease free equilibrium $E_O$ is asymptotically stable

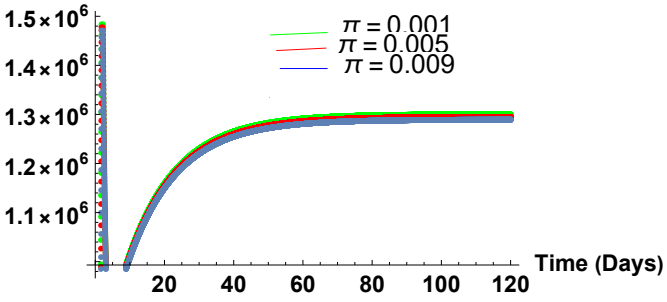


Figure 3: Numerical solutions when $R_O > 0$

Figure 3 shows that when $R_O > 1$, all trajectories of rice blast re-infection converges to $(M_a^*)$ regardless of values of $\pi$. Therefore the endemic equilibrium is asymptotically stable

*3.9. Sensitivity Analysis of Model parameters*

The basic reproduction number is very important in the effort required to eradicate a disease. We carry out sensitivity analysis of the basic reproduction number with respect to the model parameters to access the relative impact of each of the parameters in the disease. This will enable us to determine which intervention strategy is most effective in the control of blast re-infection. The normalized forward sensitivity index is used to calculate sensitivity indices. We define the normalized forward sensitivity index of the basic reproduction number with respect to parameter, A as:

$$S_A^R o = \frac{\delta R_0}{\delta A} \times \frac{A}{R_0}$$

Therefore , the sensitivity index of $R_0$ on parameter $\beta_p$ is given by:

$$S_{\beta_p}^R o = \frac{\delta R_0}{\delta} \times \frac{\beta_p}{R_0}$$

$$= \frac{g(s)}{h^2 + ha + hm} \times \frac{\beta_p (h^2 + ha + hm)}{\beta_p g(s)}$$

$$= 1 > 0$$

Likewise the sensitivity index of g(s) is given by:

$$S_{g(s)}{}^R o = \frac{\delta R_0}{\delta g(s)} \times \frac{g(s)}{R_0}$$

$$= \frac{\beta_p}{h^2 + ha + hm} \times \frac{g(s)(h^2 + ha + hm)}{\beta_p g(s)}$$

$$= 1 > 0$$

Likewise the sensitivity index of a is given by

$$S_a^R o = \frac{\delta R_0}{\delta a} \times \frac{a}{\delta R_0}$$

$$\frac{\delta R_0}{\delta a} = \frac{0[h^2 + ha + hm] - h[\beta_p g(s)]}{(h^2 + ha + hm)^2}$$

$$\frac{= -h\beta_p g(s)}{(h^2 + ha + hm)^2}$$

$$\frac{\delta R_0}{\delta a} \times \frac{a}{R_0} = \frac{-h\beta_p g(s)}{(h^2 + ha + hm)^2} \times \frac{a(h^2 + ha + hm)}{\beta_p g(s)}$$

$$= \frac{-ha}{h^2 + ha + hm}$$

$$= \frac{-a}{h + a + m}$$

Substituting parameters values in table 2

$$\frac{-0.2}{1.10025}$$

$$= -0.18178$$

Likewise the sensitivity index of m is given by:

$$S_m^R o = \frac{\delta Ro}{\delta m} \times \frac{m}{\delta Ro}$$

$$\frac{\frac{\delta Ro}{\delta m} = 0(h^2 + ha + hm) - h[\beta_p g(s)]}{(h^2 + ha + hm)^2}$$

$$\frac{= -h\beta_p g(s)}{(h^2 + ha + hm)^2}$$

$$\frac{= -h\beta_p g(s)}{(h^2 + ha + hm)^2}$$

$$\frac{\delta Ro}{\delta m} \times \frac{m}{\delta Ro} = \frac{-h\beta_p g(s)}{(h^2 + ha + hm)^2} \times \frac{m(h^2 + hahm)}{\beta_p g(s)}$$

$$\frac{= -hm}{(h^2 + ha + hm)}$$

$$= \frac{-m}{h + a + m}$$

Substituting the parameter values in table 4.2,

$$\frac{-0.9}{1.10025}$$

$$= -0.8180$$

Likewise the sensitivity index of h is given by

$$S_h^R o = \frac{\delta Ro}{\delta h} \times \frac{h}{Ro}$$

$$\frac{\delta Ro}{\delta h} = \frac{0(h^2 + ha + hm) - \beta_p g(s)[2h + a + m]}{(h^2 + ha + hm)^2}$$

$$= \frac{-\beta_p g(s)[2h + a + m]}{(h^2 + ha + hm)^2}$$

$$\frac{\delta Ro}{\delta h} \times \frac{h}{\delta Ro} = \frac{-\beta_p g(s)[2h + am]}{(h^2 + ha + hm)^2} \times \frac{h(h^2 + ha + hm)}{(\beta_p g(s))}$$

$$= \frac{-(2h + a + m)}{h^2 + ha + hm}$$

Substituting the parameter values in table 2,

$$\frac{-1.1005}{0.0002750625}$$

$$= -4000.91$$

Table 2
Sensitivity indices of model parameters to Ro

| Parameter | Index |
|---|---|
| $\beta_p$ | +1.000 |
| g(s) | +1.000 |
| a | -0.18178 |
| m | -0.8180 |
| h | -4000.91 |

Table 2: Sensitivity indices of model parameters to $R_0$

The table above shows that parameters $\beta p$, $g(s)$ increases the value of $R_0$ when they are increased as they have positive indices , implying they increase the rate of blast re-infection in rice population. The parameters a, m and h decrease the value of $R_0$ when they are increased as they have negative indices implying that they reduce blast re-infection rate in rice population.

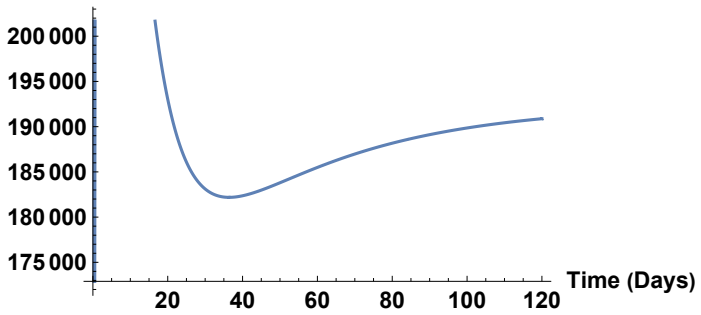


Figure 4: Effect of parameters with negative indices on re-infection of blast on rice population when their values are high. (a=0.2, m= 0.9 , h= 0.025)

Figure 4 shows that when the parameter values of a( Rate of secondary infection), m (rate of recovery of infected host) and h(rate of removal) are increased the production increases. This implies that they reduce the values of $R_0$ when they are increased hence increasing the production.

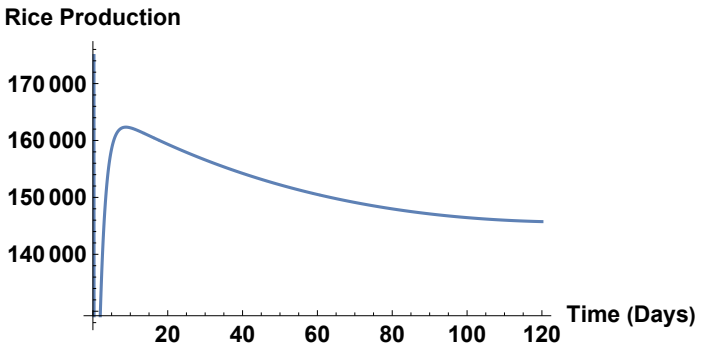


Figure 5: Effect of parameters with negative indices on blast re-infection on rice production when their values are low.( a= 0.01, m= 0.1 , h= 0.00025)

Figure 5 shows that when the parameter values of a (Rate of secondary infection), m( rate of recovery of infected host) and h( rate of removal) are decreased, the production reduces. This implies that they increase the values reproduction number $R_O$ when they are reduced leading to a decrease in rice production.

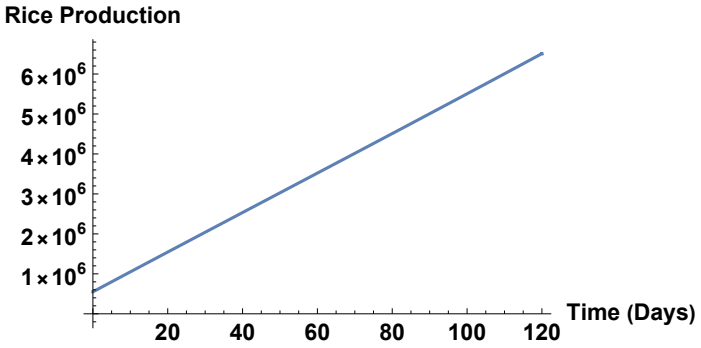


Figure 6: Effect of parameters with positive indices on rice production when their values are low. ($\beta_p = 0.0000005, g(s) = 0.1$)

Figure 6 shows that when parameter values of $\beta_p$(rate of primary infection) and g(s) (production of susceptible host) are decreased , the rice production increases. This implies that they reduce the value of reproduction number $R_O$ when they are reduced hence increased production.

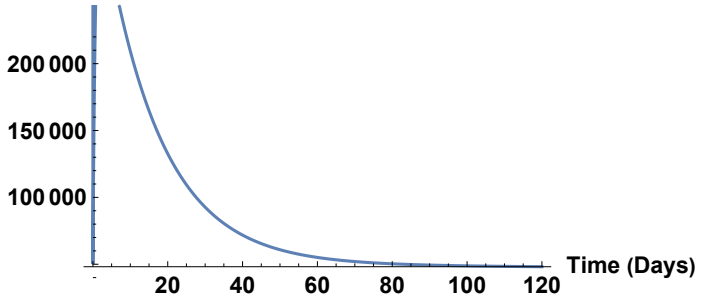


Figure 7: Effects of parameters with positive indices on rice production when their values are high. ($\beta_p = 0.5, g(s) = 0.95$)

Figure 7 shows that when the parameter values of $\beta_p$ (rate of primary infection) and g(s) ( production of susceptible host) are increased the production reduces. This implies that they increase the value of reproduction number $R_O$ hence reducing the production.

## 4. Numerical Simulations

Numerical simulations in this study involved a computer ran calculation of model equations. The simulations are significant in understanding the behaviour of the system whose mathematical solutions are too complex as in most nonlinear systems. The parameter values were got from existing literature or estimated.

This study therefore aimed at providing a mathematical numerical solution to the blast re-infection challenge, by mathematically modeling for the most appropriate fungicide application for blast re-infection control.

The effectiveness of the fungicide on the blast re-infection are numerically simulated below in figure 8, 9, 10 and 11:

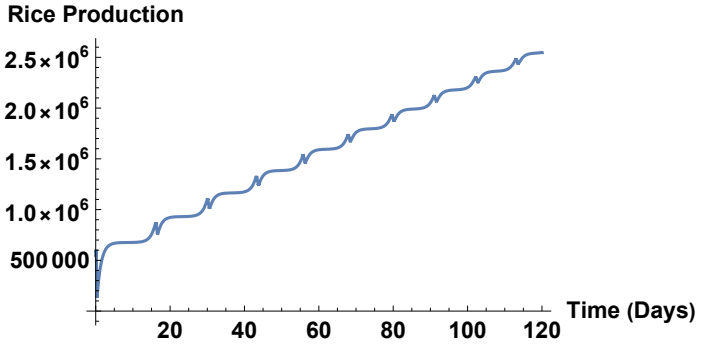


Figure 8: Rice production in the absence of rice blast re-infection and fungicide use ($\mu = 0, \alpha = 0, \pi = 0$)

Figure 8 shows that in the absence rice blast re-infection, rice production increases with time since blast interfere with rice growth and there is no fungicide applied since there is no blast.

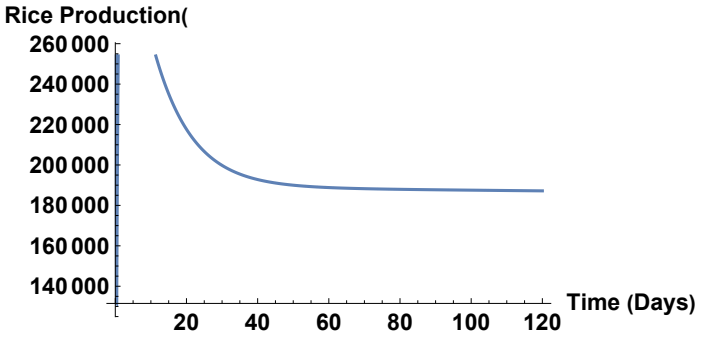


Figure 9: Rice production in the presence of rice blast but no fungicide intervention($\pi = 0$)

Figure 9 shows that with presence of rice blast and no fungicide intervention, the production goes down with time since rice blast interferes with normal carrying capacity of rice crop.

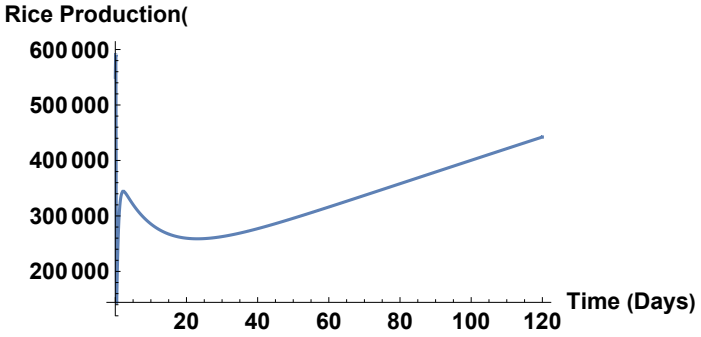


Figure 10: Rice production with fungicide applied once

The production increases with time.However the production is low since the crops are only protected within the early growth stage and infection at later stages may not be controlled.

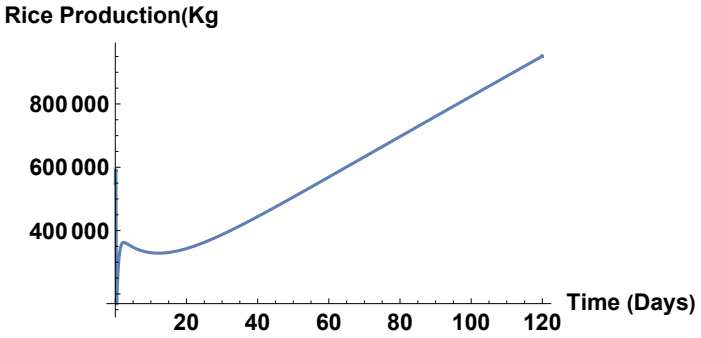


Figure 11: Rice production with fungicide applied three times

As the number of fungicide application increases, the production increases with time since the rice crops are protected from rice blast for a longer period of their growth stages.

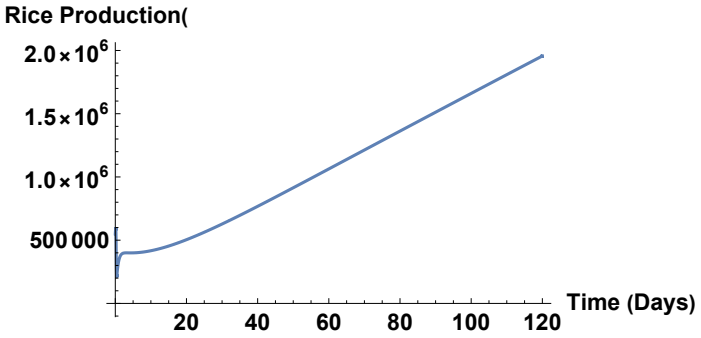


Figure 12: Rice production with fungicide applied seven times

Figure 12 above shows that rice production increases to the maximum with fungicide applied seven times on the rice crops.This is because rice crops are protected throughout growth stages. Production below seven times fungicide application is slightly lower since at a given period the crop may not be protected hence possible rice blast attack lowering production.

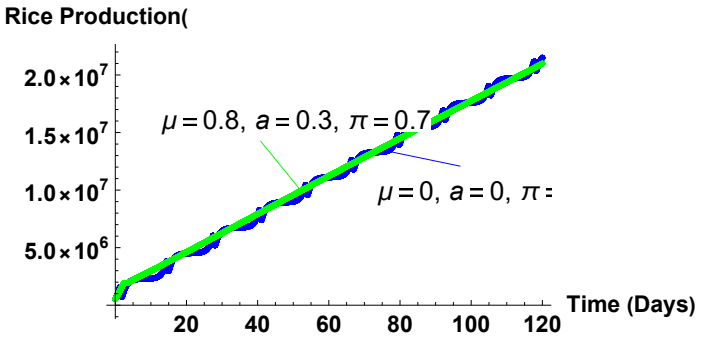


Figure 13: Threshold parameters($\mu = 0.8, \alpha = 0.3, \pi = 0.7$).

Figure 13 shows the threshold parameters.Fungicide applied seven times through the growth period of rice gives equivalent yields as if there were no rice blast.Beyond seven times, there is no change in the production graph.

## 5. Conclusion

In this study, we developed a comprehensive mathematical model to analyze the dynamics of rice blast re-infection following fungicide application. Our model, structured around a system of Ordinary Differential Equations, effectively categorizes the rice population into five distinct classes: Susceptible, Protected, Infected, Recovered, and Secondary Infected. Through rigorous stability analysis, we established the conditions under which the disease-free equilibrium (DFE) and endemic equilibrium (EEP) are stable, particularly emphasizing the critical role of the Basic Reproduction Number ($R_o$) in determining the model's behavior.

Analysis of endemic equilibrium shows its existence when $R_0 > 1$ and is also asymptotically stable. This shows that when $R_o > 1$ , the rice blast re-infection persists and spreads in the entire population. Sensitivity analysis of the model show that the parameters a(rate of secondary infection), m(rate of recovery of infected host) and h(rate of removal of all classes) should be increased in order to reduce the basic reproduction number hence minimize the rice blast re-infection to the entire rice population. The parameters $\beta_p$ (rate of primary infection) and g(s) (production of susceptible host) should be reduced in order to reduce basic reproduction number. Numerical analysis of the model supports the fact that both the disease free equilibrium and endemic equilibrium are stable. It also shows that reduction in rice blast re-infection increases rice production greatly.

The findings indicate that when $R_o$ is less than one, the DFE is both locally and globally stable, suggesting that effective fungicide application can significantly mitigate the prevalence of rice blast. Conversely, when $R_o$ exceeds one, the EEP becomes locally asymptotically stable, highlighting the potential for disease resurgence if fungicide application is not managed judiciously. Our sensitivity analysis further identified the rate of fungicide application ($\pi$) as a pivotal parameter in controlling rice blast re-infection.

This research not only contributes to the existing body of knowledge on plant disease management but also provides a robust framework for future investigations into the epidemiology of crop diseases. The implications of our findings are particularly relevant for agricultural practices in regions heavily impacted by rice blast, such as Kenya. By optimizing fungicide use, we can enhance rice yields and promote sustainable agricultural practices, ultimately contributing to food security and economic stability in affected regions.

Future research should explore the integration of additional factors, such as environmental conditions and farmer behavior, into the model to further refine our understanding of rice blast dynamics. Additionally, empirical validation of the model through field studies will be essential to confirm the theoretical predictions and enhance the practical applicability of our findings.

## Conflict of Interest

The authors declare that there are no conflict of interest with respect to the publication of this article.

## Acknowledgements

We thank the administrative staff of the Department of Physical Sciences of Chuka University for their hospitality and assistance with matters related to our research work.

## References

[1] Almalahi, M. A., Panchal, S. K., Shatanawi, W., Abdo, M. S., Shah, K., and Abodayeh, K. (2021). Analytical study of transmission dynamics of 2019-ncov pandemic via fractal fractional operator. *Results in physics*, 24:104045.

[2] Anggriani, N. et al. (2015). Global stability for a susceptible-infectious epidemic model with fractional incidence rate. *Applied Mathematical Sciences*, 9(76):3775–3788.
[3] Bryen, D. N., Heake, G., Semenuk, A., and Segal, M. (2010). Improving web access for individuals who rely on augmentative and alternative communication. *Augmentative and Alternative Communication*, 26(1):21–29.
[4] Castle, M. D. and Gilligan, C. A. (2012). An epidemiological framework for modelling fungicide dynamics and control.
[5] Cohen, T., Colijn, C., Finklea, B., and Murray, M. (2007). Exogenous re-infection and the dynamics of tuberculosis epidemics: local effects in a network model of transmission. *Journal of the Royal Society Interface*, 4(14):523–531.
[6] Couch, B. C. and Kohn, L. M. (2002). A multilocus gene genealogy concordant with host preference indicates segregation of a new species, magnaporthe oryzae, from m. grisea. *Mycologia*, 94(4):683–693.
[7] Diekmann, O., Heesterbeek, J. A. P., and Metz, J. A. J. (1990). On the definition and the computation of the basic reproduction ratio r 0 in models for infectious diseases in heterogeneous populations. *Journal of mathematical biology*, 28:365–382.
[8] Gitau, A. N. (2005). Iirr and act, 2005. conservation agriculture. a manual for farmers and extension workers in africa. international institute of rural re-construction.
[9] Gómez-Acevedo, H. and Li, M. Y. (2005). Backward bifurcation in a model for htlv-i infection of cd4+ t cells. *Bulletin of Mathematical Biology*, 67:101–114.
[10] Gouramanis, G. D. (1995). Biological and chemical control of rice blast disease (pyricularia oryzae) in northern greece. *CIHEAM-Options Mediterraneennes*, 15.
[11] Jimrise, O., Okongo, M., and Muraya, M. (2023). Stability analysis of a sterile insect technique model for controlling false codling moth. *Journal of Mathematical Analysis and Modeling*, 4(1):78–105.
[12] Magar, P. B., Acharya, B., and Pandey, B. (2015). Use of chemical fungicides for the management of rice blast (pyricularia grisea) disease at jyotinagar, chitwan, nepal. *International Journal of Applied Sciences and Biotechnology*, 3(3):474–478.
[13] Massawe, L. N., Massawe, E. S., and Makinde, O. D. (2015). Modelling infectiology of dengue epidemic. *Applied and Computational Mathematics*, 4(3):192–206.
[14] Muriithi, C., Mugai, E., and Kihurani, A. (2017). Effect of nitrogen and silicon on management of rice blast (pyricularia oryzae) in mwea irrigation scheme of kenya.
[15] Obita, B. O., Okongo, M. O., Jimrise, O. O., and Lunani, A. M. (2024). Mathematical modelling for rice blast re-infection. *American Journal of Applied Mathematics*, 10(4):37–49.
[16] Ochwach, J., Okongo, M. O., and Muraya, M. M. (2021). Mathematical model for false codling moth control using pheromone traps. *International Journal of Applied*, 10(2):32–52.
[Ochwach et al.] Ochwach, J. O., Mark, O. O., and Murwayi, A. L. M. On basic reproduction number r0: Derivation and application. *Journal of Engineering and Applied Sciences Technology*, 173:2–14, doi:org/10.47363/JEAST/2023(5).
[18] Okongo, M. O., Lunani, M. A., Menge, B. K., and Ochwach, J. O. (2023). The impact of hiv/aids treatment and counseling on the prevalence of tuberculosis and malaria co infections.
[19] ONYUMA, M. B. (2017). *modelling cholera transmission incorporating media coverage*. PhD thesis, MOI UNIVERSITY.
[20] Sala, A., Ariño, C., and Robles, R. (2019). Gain-scheduled control via convex nonlinear parameter varying models. *IFAC-PapersOnLine*, 52(28):70–75.
[21] Shah, K. and Abdeljawad, T. (2023). On complex fractal-fractional order mathematical modeling of co 2 emanations from energy sector. *Physica Scripta*, 99(1):015226.
[22] Shah, K., Sarwar, M., Abdeljawad, T., et al. (2024). On mathematical model of infectious disease by using fractals fractional analysis. *Discrete and Continuous Dynamical Systems-S*, pages 0–0.
[23] Van den Driessche, P. and Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical biosciences*, 180(1-2):29–48.
[24] Wangari, I. M., Sewe, S., Kimathi, G., Wainaina, M., Kitetu, V., and Kaluki, W. (2021). Mathematical modelling of covid-19 transmission in kenya: A model with reinfection transmission mechanism. *Computational and Mathematical Methods in Medicine*, 2021(1):5384481.
[25] Wanjala, H. M., Okongo, M. O., and Ochwach, J. O. (2023). Impact of media awareness and use of face-masks on infectious respiratory disease. *Pan-American Journal of Mathematics*, 2:15.