Mathematical Modelling for Rice Blast Re-Infection

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Abstract: Rice is the thirdly most valued cereal crops in Kenya after maize and wheat. The demand for rice in Kenya has increased greatly over the last few years while production has still remained low. This is because rice production is affected by serious constraints especially rice diseases of which the most threatening is rice blast. Rice blast infection and re-infection can occur in different stages of rice growth and therefore need to be controlled. This study aims to developed a mathematical model for rice blast re-infection. The model employs a system of nonlinear ordinary differential equations which is analysed in details for its stability properties. Basic reproduction number R_o for rice blast re-infection was found to be less than one. Numerical simulation of the model is done using Mathematica, and graphical profile of the main variables are depicted. We conclude that rice blast re-infection reduces rice yield and necessary remedy are needed.

Keywords: Mathematical Modeling, Basic Reproduction Ratio, Rice Blast, Epidemiology Model, Epidemic Mode

1. Introduction

Mathematical modelling involves an art of converting a problem from an application area into tractable mathematical formulations whose theoretical and numerical analysis provides insight, answers and guidance useful for the originating application. It also gives precision and direction for problem solution. Mathematical modelling has an immense role in the investigation of problems that emerge in our day-to-day life and when applied to experimental data, it can reveal correlation between several observable phenomena [7, 35].

Re-infection of diseases is a great challenge in our country [10]. developed a model on tuberculosis re-infection where only a small percentage of persons who are infected with the disease experience primary disease. Those who survive the original illness may, at some point in their lifetimes, reactivate this concealed infection. Infected individuals are susceptible to re-infection and sickness as a result of fresh exposure.Basic reproduction number was developed by employing next generation matrix. Sensitivity analysis showed that when the disease was widespread, a latent infected individual was likely to contact an infectious individual.In contrast, as the disease incidence declined, the likelihood that a latently infected person would be exposed again depends increasingly on the topology of the contact network.

Wangari, I. M. et al [36] developed a mathematical model with an aim of investigating how re-infection mechanisms influence covid 2019 dynamics in Kenya. The population was divided into infected people who are asymptomatic, symptomatic but only mildly symptomatic, and symptomatic but very severely ill. The outcome showed that the model's infection-free equilibrium is locally asymptotically stable whenever $R_O < 1$ and unstable anytime $R_O > 1$. Sensitivity analysis revealed that non-pharmaceutical intervention techniques including face mask use and keeping one's distance from others were successful in reducing the spread of the COVID 2019 re-infection. However mathematical modelling on re-infection has not been explored in the field of agriculture [36].

Uncertainty ridden agriculture requires reliable and well-timed forecast [13]. Diseases are the prominent causes of reduction in crop yield. To reduce the yield loss, prior knowledge of time and severity of the disease is necessary. Crop modelling provides reliable forecast of crop yield in

advance and also forewarning of crop disease attack so that suitable plant protection measure could be taken up to protect the crops.

Rice (Oryzasativa L.) is a particularly distinctive crop since it thrives well in wet conditions. Nearly 50% of the world's population relies on rice as a steady food source, which accounts for 20% of all of the calories we consume as humans [16]. It is Kenya's third-most significant food crop after maize and wheat. Small-scale farmers grow it mostly for commercial and food purposes [16]. It is heavily consumed by both urban and rural residents. For the best growth, rice needs soil with a vertisol soil type, a high temperature of 28 degrees Celsius, that is found below 1200 meters above sea level, as well as a higher water holding capacity to hold the flooded water [1].

Among the several diseases affecting rice production in Kenya, the most threatening is rice blast [11]. Sheath blight (Thanatephorus cucumeris) and Pyriculariaoryzae are the two organisms that produce rice blast. In farmers' fields in western Kenya, the illness is reported to result in output losses of up to 50% [27]. Infected farms have reportedly suffered losses of more than 70·C80 percent [24]. A more recent epidemic of the disease happened in 2009. It was initially discovered in the Mwea irrigation scheme in 2006. This required action since the disease poses a risk to the security of the food supply.

Entire areas of the shoot, stem, and panicle may develop lesions as a symptom of the disease. All plant portions above the infection site die when nodes become infected, and yield losses are significant. Elongated diamond-shaped white to grey lesions with dark green to brown borders and a yellowish halo appear on the leaves as a result. The leaf collar, stem, and occasionally the internodes are also killed by the disease. Every country where rice is farmed has experienced rice blast, and fungicide applications have been made in particular where the environment is ideal for effective management. If the disease strikes when the plant is heading, it may cause the stem to break at the diseased node or to produce a white panicle. The neck rot, neck blast, or panicle blast stage of the disease is named after this damaging symptom [15]. Early infection prevents the grains from filling and keeps the panicle upright. However, late infection causes the grains to only partially fill, and as a result of the weight, the panicle's base splits and droops.

This study developed a model to study the dynamics of blast re-infections in rice.

2. Model Formulation

The model was developed from a system of ODEs .The rice crop population was divided into four classes: S, the susceptible rice crop which represent seedlings from the nursery; I, the seedlings infected by blast for the first time; R; the naturally recovered rice crops; X; the secondarily infected rice crops by the blast. The recruitment rate is g(s) which represents all seedlings transplanted from the nursery, h represents the natural removal rate of all classes.

2.1. Assumptions of Model Development

  i The fungal disease spread through one population
 ii There is a simple density dependent growth of the host up to a carrying capacity;
iii Primary and secondary infection does not occur at the same time

2.2. Model Flow Chart
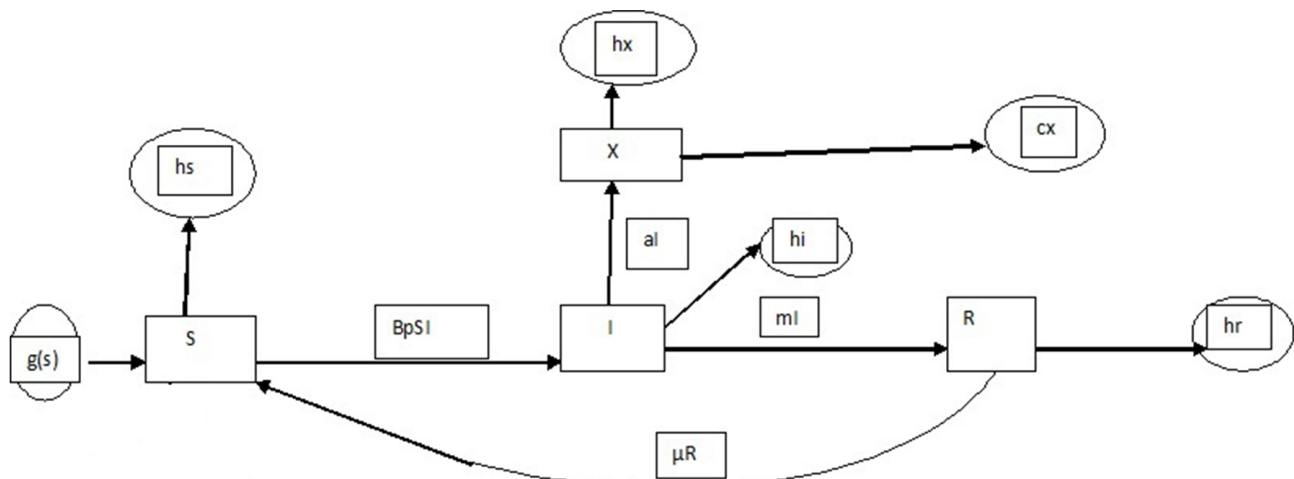
The flow diagram of the system is shown in Figure 1:

Figure 1. Model Flow Chart.

The seedling from the nursery represented by g(s) leads to production of susceptible class, S. Primary infection takes place when there is interaction between the susceptible and the infected leading to the compartment of the infected (I). Some may recover naturally 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 naturally recovered infected host that are susceptible to the disease again (μR). The susceptible class is reduced by natural death rate(hS),the primary infection brought by interaction between susceptible and the infected crops ($\beta_p SI$). 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). Density of recovered host is produced by naturally recovered infected rice crop which may be susceptible to blast again and the number minimized by death rate. 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.

From the above description,we have the following equations:

$$\frac{dS}{dt} = g(s) + \mu R - \beta_p SI - hS$$
$$\frac{dI}{dt} = \beta_p SI - mI - aI - hI$$
$$\frac{dR}{dt} = mI - hR - \mu R \tag{1}$$
$$\frac{dX}{dt} = aI - hX - cX$$

Table 1. *Model parameters and Values.*

Parameter	Definition	Source	Source
μ	co-efficient for R going to S	0.5	Aminiel et al(2015)
β_s	rate of secondary infection	0.075	Angriani(2018)
β_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

3. Positivity and Boundedness of Solution

By demonstrating that the model's solutions are positive and bounded, this study shows the model's well-posedness. 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 [5].

3.1. Boundedness of Solution

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

Theorem 1 The solutions of the model are contained in the feasible region γ.

Proof

Adding the system of four equations , we have;

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

$$\frac{dN}{dt} = [g(s) + \mu R - \beta_p SI - hS]$$
$$+ [\beta_p SI - mI - aI - hI]$$
$$+ [mI - hR - \mu R]$$
$$+ [aI - hX - cX]$$

$$\frac{dN}{dt} = g(s) - h(S + I + R + X) - cX$$

$$\frac{dN}{dt} = g(s) - hN - cX$$

$$\frac{dN}{dt} < g(s) - hN$$

$$\frac{dN}{dt} + hN < g(s)$$

Using the integrating factor e^{ht} to solve

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

When $t = 0$

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

Substituting

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

Where N(0) is the beginning population as $t \longrightarrow \infty$
Which means

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

Therefore the solutions are bounded in the invariant region

$$\gamma = \{S(t), I(t), R(t), X(t) \in \mathbb{R}^4_+ :$$
$$S(t) + I(t) + R(t) + X(t) \leq 0\}$$

3.2. 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 $t \geq 0$

Theorem 2 Let the initial conditions be $S(0) \geq 0, I(0) \geq 0, R(0) \geq 0$ and $X(t) \geq 0 \geq 0, \in \gamma$, then the solutions S(t), I(t), R(t) and X(t) of system 1 are positive for all $t \geq 0$

Proof

From the first equation of 1,

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

$$\frac{dS}{dt} \geq -(\beta_p I + h)S$$

By separation of variables and integrating both sides,

$$\frac{\int dS}{dt} \geq \int -(\beta_p I + h)dt$$

$$\ln S(t) \geq -(\beta_p I + h)t + C$$

$$S(t) \geq \exp^-(\beta_p I + h)t \times \exp^c$$

Taking $\exp^c$ to be A

$$S(t) \geq A \exp^{-(\beta_p I + h)t}$$

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

$$S(t) \geq S(0) \exp\left(-(\beta_p I + h)t\right) \qquad (2)$$

Therefore
S(t) ≥ 0 for all $t = 0$ for all $t \geq 0$

From the above it follows that from equations two,three and four respectively that $I(t) \geq 0$, $R(t) \geq 0$ and $X(t) \geq 0$ for all $t \geq 0$

4. Equilibria Analysis

According to [28], an equilibrium point is the constant solution of a model system. Setting the right hand side of differential equations to zero and solving each one to produce a constant solution yields the equilibrium points of a model system.   The disease-free equilibrium and the endemic equilibrium are the two equilibrium points that epidemiological models typically have.   The model's equilibrium points are identified in relation to the fundamental reproduction number, which is obtained using a next-generation matrix technique. To ascertain the circumstances for the spread of blast re-infection, the model's stability analysis is conducted.

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 1 when there is no blast, we get

$$\frac{ds}{dt} = g(S) - h(S)$$
$$\frac{dI}{dt} = 0$$
$$\frac{dR}{dt} = 0 \qquad (3)$$
$$\frac{dX}{dt} = 0$$

Solving the first equation 3

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

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

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

4.2. Basic Reproduction Number

According to [12], the Basic Reproduction Number,(R_0), is the typical number of secondary infections brought on by a single infected person throughout the course of his or her entire life as an infective when introduced into a population that is just susceptible. In this study, blast re-infection is used to describe secondary infection. The rice population's potential for blast re-infection spread is gauged by the fundamental reproduction number [35]. If $R_0 < 1$, the average number of re-infected rice crops produced by each infected crop will be fewer than one, preventing the spread of the rice blast virus. On the other hand, if $R_0 > 1$, each re-infected rice crop will, on average, result in more than one re-infection throughout the course of its lifetime, invading the entire population.In this work, we want to find $R_0 < 1$ in order to limit the spread of blast re-infection in the rice population. This study uses Next Generation Matrix to derive R_0 [35]. Consider the next generation matrix made up of matrices F and V, such that $G = FV^1$, $F = \frac{\delta f_i}{\delta x_j(x_o)}$ and $V = \frac{\delta v_i}{x_j(x_o)}$.   Where x_o is DFE point, f_i is the re-infection matrix(rate of appearance 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 $R_O = \rho F V^{-}1$. The re-infection compartments are I and X. We use second and the fourth equations of 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 $\left(\frac{g}{h}, 0, 0, 0, 0\right)$

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

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

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

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

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

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

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

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

$$R_0 = \frac{\beta_p g(s)}{h^2 + ha + hm} \tag{4}$$

5. Stability Analysis

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 7 is developed and is given by:

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

By examining the behavior of JE_O, it is possible to analyze the stability of the Disease Free Equilibrium point. For local stability of Disease Free Equilibrium, each of its eigenvalues must contain negative components. The following eigenvalues were obtained by using the mathematica software.

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

$$\lambda_2 = -h$$

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

$$\lambda_4 = -(h + \mu)$$

Condition
For the root λ_3 to be negative

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

The proof for the local stability of the disease-free equilibrium is provided by the following finding, which incorporates methods from [33].

Theorem 3 In order for disease-free equilibrium to be locally stable $R_0 < 1$

Proof
The proof applies [33] techniques. One of the eigen values of the JE_O is $\lambda 1 = \frac{-ch - h^2}{h}$

By examining the signs of the eigenvalues of the block matrix provided by, we can identify additional eigen values.

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

Now let Tr be Trace of A and K be the determinant of A and consider the linear system $x^{\cdot}(t) = Ax(t)$, 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 $\delta = Tr - 4K \geq 0$, 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 $\delta \geq 0$, 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.

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

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

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

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

$$= c[m + a + h] + h[m + a + h] - [\frac{c\beta_p g(s)}{h} + \beta_p g(s)] \quad (5)$$

For determinant to be greater than one

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

$$\frac{c\beta_p g(s)}{h} + \beta_p g(s) < (c + h)(m + a + h) \quad (6)$$

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

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

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

$$\frac{\beta_p g(s)}{h^2 + ha + hm} < 1 \quad (7)$$

Thus $R_0 < 1$

The block matrix trace is given by

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

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

$$= \frac{\beta_p g(s) - hm - ha - hc - 2h^2}{h} \quad (8)$$

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

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

$$\beta_p g(s) = R_O(h^2 + ha + hm) \quad (9)$$

Substituting 9 and 8 gives

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

For us to have negative eigenvalues, the trace must be negative. Therefore, $R_o < 1$, and 7 must be negative.

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

5.2. Global Stability Analysis of Disease Free Equilibrium (E_O)

We examine the global asymptotic stability of the disease-free state using the Castillo-Chaves theorem [8]. We re-write model system 1 as

$$\frac{dx}{dt} = F(X, Z) \quad (10)$$

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

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

denotes non-infectious compartments and $Z = (I, X) \in R_+^2$ denotes infectious classes. The disease free equilibrium of the system now becomes $E_O = (X^*, 0)$, $X^* = \frac{g(s)}{h}g(S)$. 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 $G(X, Z) = TZ - \hat{G}(X, Z) \geq 0$ 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 1 provided that $R_0 < 1$ and assumptions 1 and 2 are satisfied.

Proof

Considering the model system 1, we have

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

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

Now

$$F(X, 0) = \begin{pmatrix} g(s) + \mu R - hS \\ -hR - \mu R \end{pmatrix}$$

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

Now consider condition 2

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

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

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

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

$$TZ = \begin{pmatrix} \frac{\beta_p I g(s)}{h} - mI - aI - hI & 0 \\ aI - cX - hX \end{pmatrix}$$

5.3. Existence of Endemic Equilibrium Point (E_a^*)

When the basic reproduction number is greater than 1, the population reaches the endemic equilibrium point, which causes blast re-infection to spread. According to this analysis, the endemic equilibrium point is

$E_a^* = (S^*, I^*, X^*)$.To determine this equilibrium, we equate the right side of 1 to zero.

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

$$\beta_p S^* I^* - mI^* - aI^* - hI^* = 0 \qquad (11)$$

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

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

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

$$I^* = 0$$

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

Additive compound matrix approach [23] is used to analyse the local stability of the endemic equilibrium given by $E_a^* = (S^*, I^*, X^*)$ in γ. Local stability of endemic equilibrium is determined by variation matrix $J(E_a^*)$ of the non linear system.

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

where: $Q^* = -(\beta_p I^* + h)$ and $Q^{**} = -(\beta_p S^* + m + a + h)$

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 5. If $R_0 > 1$, the endemic equilibrium E_a^* of the model 1 is locally asymptotically stable in γ

Proof

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

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

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 by equation 12:

$$J(E_a^*) = \begin{pmatrix} Q_4 & -\beta_p S^* & 0 \\ \beta_p I^* & Q_5 & 0 \\ 0 & a & Q_6 \end{pmatrix} \qquad (12)$$

Where $Q_4 = -(\beta_p I^* + h) + K$, $Q_5 = -(\beta_p S^* + m + a + h) + K$ and $Q_6 = -(h + c) + K$

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

$$\begin{pmatrix} -(\beta_p I^* + h) + K & -\beta_p S^* \\ \beta_p I^* & Q_1 \end{pmatrix}$$

Where $Q_1 = -(\beta_p S^* + m + a + h) + K$ The co-efficient of K in the determinant of this matrix is

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

and thus the (1,1) entry of $U_i j$ is

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

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

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

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)

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

The co-efficient of K is

a

Entry (2,2)

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

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)

$$\begin{pmatrix} M_1 & 0 \\ a & -(h + c) + K \end{pmatrix}$$

$M_1 = -(\beta_p S^* + m + a + h) + K$ 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

$$\begin{pmatrix} Q_3 & 0 & 0 \\ a & -h - c & -\beta_p S \\ 0 & \beta_p I & Q_2 \end{pmatrix}$$

Where $Q_2 = -\beta_p S - m - a - c - 2h$, and $Q_3 = -\beta_p I^* - \beta_p S - a - m - 2h$

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

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

5.4. 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 figures 2 and 3.
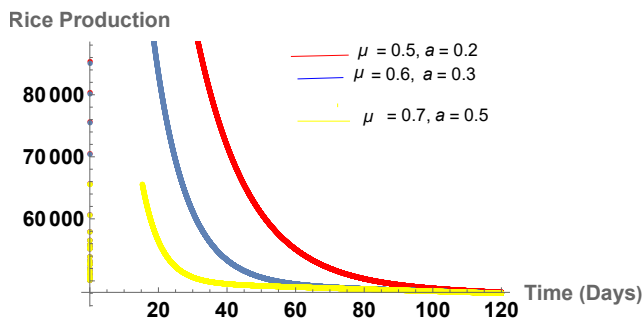

Figure 2. Analysis of $R_O < 1$.

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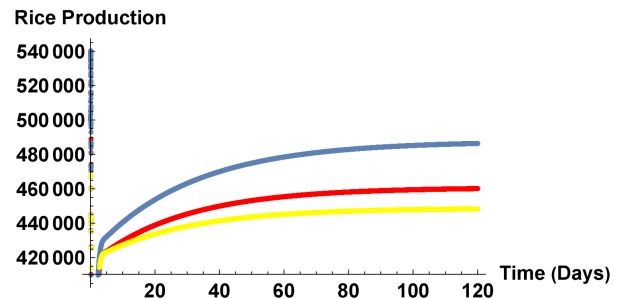

Figure 3. Numerical solutions when $R_O > 0$.

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

6. Sensitivity Analysis of Model Parameters

In the effort needed to remove a disease, the basic reproduction number is crucial. To determine the relative impact of each parameter on the disease, we do a sensitivity analysis of the basic reproduction number with regard to the model parameters. By doing so, we will be able to decide which intervention technique is most successful in preventing blast re-infection. Sensitivity indices are computed using the normalized forward sensitivity index. The basic reproduction number's normalized forward sensitivity index with regard to parameter A is defined as follows,

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

Therefore , the sensitivity index of R_0 on parameter β_p is given by:

$$S_{\beta p}^{R_O} = \frac{\delta R_0}{\delta \beta_p} \times \frac{\beta_p}{R_0}$$

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

$$= 1 > 0$$

The sensitivity index of g(s) is similarly given by,

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

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

$$= 1 > 0$$

The sensitivity index of a is similarly generated by,

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

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

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

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

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

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

Substituting parameters values in table 1

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

$$= -0.18178$$

The sensitivity index of m is similarly generated by,

$$S_m^{R_O} = \frac{\delta R_O}{\delta m} \times \frac{m}{R_O}$$

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

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

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

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

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

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

Substituting the parameter values in table 1,

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

$$= -0.8180$$

The sensitivity index of h is similarly generated by,

$$S_h^{R_O} = \frac{\delta R_O}{\delta h} \times \frac{h}{R_O}$$

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

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

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

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

Substituting the parameter values in table 1,

$$\frac{-1.1005}{0.0002750625} = -4000.91$$

Table 2. *Sensitivity indices of model parameters to R_0.*

Parameter	Index
β_p	+1.000
g(s)	+1.000
a	-0.18178
m	-0.8180
h	-4000.91

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.
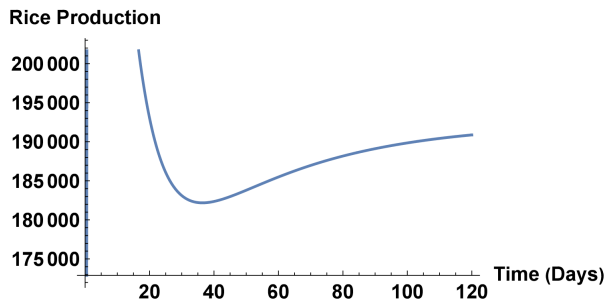

Figure 4. *Effect of parameters with negative indices on re-infection of blast on rice population when their values are high. (a=0.2, m= 0.9 , h= 0.025).*

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.
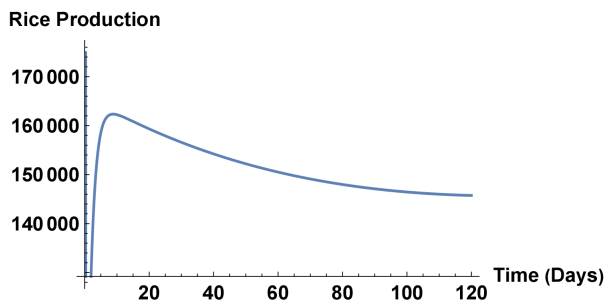

Figure 5. *Effect of parameters with negative indices on blast re-infection on rice production when their values are low.(a= 0.01, m= 0.1 , h= 0.00025).*

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.
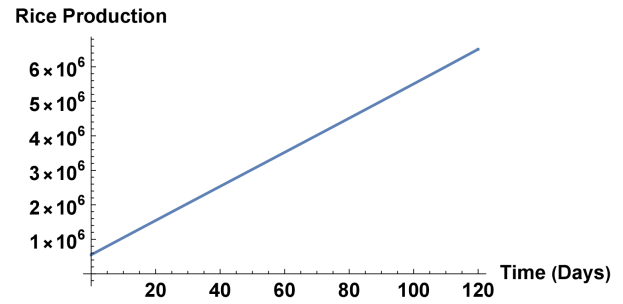

Figure 6. *Effect of parameters with positive indices on rice production when their values are low. ($\beta_p = 0.0000005$, $g(s) = 0.1$).*

Figure 6 shows that when parameter values of β_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.
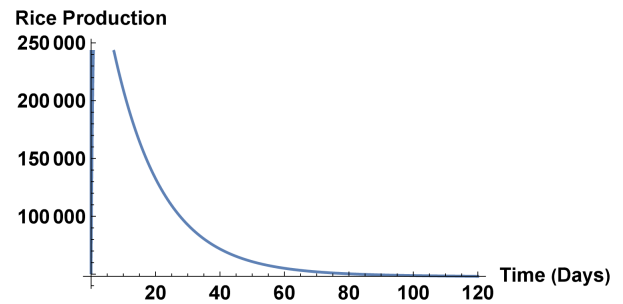

Figure 7. *Effects of parameters with positive indices on rice production when their values are high. ($\beta_p = 0.5$, $g(s) = 0.95$).*

Figure 7 shows that when the parameter values of β_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.

7. 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.
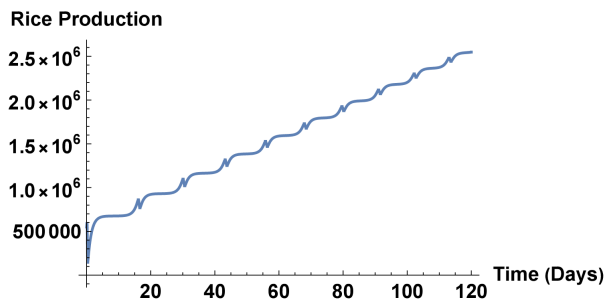
Rice Production

Figure 8. *Rice production in the absence of rice blast re-infection ($\mu = 0$, $a = 0$).*

Figure 8 shows that in the absence rice blast re-infection, rice production increases with time since blast interfere with rice growth and thus production

Figure 9 shows that in the presence of blast re-infection, the production of rice goes down with time since the blast affects plants¡¯ growth and development.
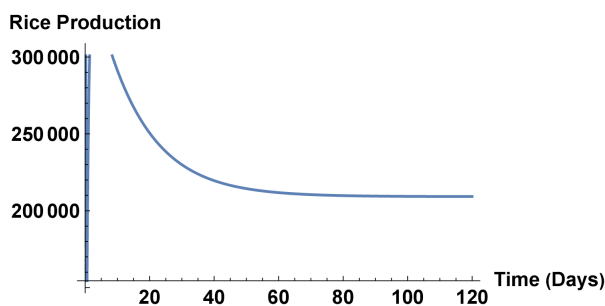
Rice Production

Figure 9. *Rice production in the presence of both secondary infection and recovery class going back to susceptible.(a= 0.2 and μ= 0.3).*

8. Conclusion

In order to investigate the impact of rice blast re-infection on rice production, a mathematical model built on a system of ordinary differential equations and including a study of blast re-infection has been developed and studied. The next generation matrix approach was used to compute the basic reproduction number R_0, which serves as the threshold parameter. It was discovered that if $R_0 < 1$, the blast re-infection does not occur in the population of rice crops, and that if $R_0 > 1$, it does and spreads throughout the entire rice population. The disease-free equilibrium is both locally and globally asymptotically stable when the basic reproduction is less than one, according to the stability analysis of the rice blast re-infection model. This suggests that one method of preventing rice blast re-infection is to maintain R_0 below one. Analysis of endemic equilibrium demonstrates that it exists and is asymptotically stable when $R_0 > 1$.

This demonstrates that the rice blast re-infection survives and spreads throughout the entire population when $R_o > 1$. 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, according to the model's sensitivity analysis, in order to lower the basic reproduction number and, as a result, reduce the risk of rice blast re-infection for the entire rice population. To decrease the basic reproduction number, the parameters $beta_p$ (rate of primary infection) and g(s) (production of susceptible host) should be increased.Both the stability of endemic equilibrium and the disease-free equilibrium are supported by numerical analysis of the model. It also demonstrates how a decrease in rice blast re-infection significantly raises rice productivity.

Acknowledgments

Conflicts of Interest

There are no conflicts to declare.

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