A Model for Stripe Rust Growth with Two Fungicidal Effects

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July 25, 2014

Abstract

Stripe rust, also known as yellow rust, is a disease caused by the fungus *Puccinia striiformis* that affects host crops, primarily barley and wheat. It is the most prevalent strain of wheat rust within North America, and can account for a significant amount of yield loss within a harvest. In order to estimate the dynamic yield loss of a wheat crop infected by stripe rust undergoing fungicide application, a single-host, single-pathogen compartmental model is introduced using a system of nonlinear ordinary differential equations. Two fungicidal effects are considered: preventative and anti-sporulant, which reduce susceptibility and inhibit the further spread of an infection, respectively. The stability conditions associated with the disease-free and endemic equilibria are identified. Numerical simulations are then used to examine the time-dependent behavior of fungicide efficacy and approximate yield loss. Finally, sensitivity indices are calculated to study the impact of fungicide efficacy and retention relative to plant growth.

1 Introduction

Pathogens in agriculture have long been studied in order to maximize yield and minimize risks related to disease spread. Conservative estimates of plant disease costs to global yield production are of $220 billion annually solely from plant diseases before harvest [2]. Wheat rust diseases (stem, stripe, and leaf rust) have been affecting wheat since the initial domestication of the crop in the Fertile Crescent [15,21]. *Puccinia striiformis* is a species of fungus that is pathogenic to wheat and other cereal crops such as barley, tricale, and rye. It causes the stripe rust (yellow rust) disease in wheat, which parasitically depletes the plants' nutrients in order to reproduce, ultimately stunting plant growth and limiting crop yield [7].

Annual loss estimates are inaccurate because of the high variability of the epidemic, both in location and in severity. For example, in California during 2003, stripe rust caused a 25% loss of production as well as 10% in both Kansas and Nebraska [6]. The previous year only had a 5% effect on production only in Arkansas.

Due to the mechanism behind the spread, life cycle, and evolution of the fungus, it has become a global epidemic, affecting crops in more than 60 countries [6]. Without intervention, losses caused by stripe rust can cause complete decimation of a plantation [32]; it is estimated to cost billions of dollars annually in yield loss and management worldwide [1,29]. In 2003 alone, stripe rust accounted

for 11,746,401 metric tons of yield loss in the US [6], and over the past 30 years, epidemics have continued to reemerge due to the evolutionary adaptation of the fungus [19]. The pandemic of stripe rust perpetuates as a result of the fungus's life cycle. *P. striiformis* is dispersed by the wind on a continental scale with new strains of the pathogen migrating between countries [12, 18], and is capable of reproducing both asexually and sexually – by utilizing an alternate host as a survival mechanism when climatic conditions become unfavorable. In the absence of a co-host, which is necessary for *P. striiformis* to sexually reproduce, the fungus reproduces asexually. During the asexual process, airborne urediniospores (a certain stage in the fungus's life cycle) land on wheat leaves and colonize the area by producing genetically identical urediniospores, parasitically thriving under the plant tissue, inhibiting growth and permanently damaging wheat leaves [31]; this, in turn, creates pressure underneath the surface of the leaf and eventually results in pustules forming on the surface. After about 7–10 days, after the single spore has generated thousands of clone spores [21], the pustule erupts and the spores are scattered via wind at approximately 0.59 m^2/week [10], reportedly traveling up to 2000 km annually [31].

When weather conditions are not conducive for urediniospore replication, the fungus transitions from uredinia into the telial stage, where it undergoes sexual reproduction; otherwise, if temperatures are above 20 °C or below 3 °C the fungus will not survive [32]. Teliospores are capable of thriving in more severe weather conditions than urediniospores, and have the ability to infect plants other than wheat via wind dispersal. Several grass species [19] as well *Berberis* spp. [17], have been identified as essential alternate hosts in order for the fungus to sexualy reproduce and maintain its spread across seasons [16]. Once infected, the alternate host produces aeciospores, which are capable of infecting the primary host – urediniospores can be formed from aeciospores, as well as by other urediniospores, completing the life cycle of *P. striiformis* [34]. Removal of the alternate host helps to reduce the pathogen levels across seasons. For this purpose, eradication efforts for *berberis* spp. near wheat fields have been established [16]. Although useful, these measures have a limited effect because of the long distances traveled by the spores. Because of many factors, the rapid adaptation of the pathogen is prevalent: its survival across seasons using alternate hosts, a sexual reproduction stage in the life cycle allowing for genetic recombination, and the rapid spread across distances significantly larger than original infection sites. Varieties of wheat with more resistance to rust and other diseases are constantly developed and selected to counteract these effects [6, 8, 22]. However, the protection is only temporary as adaptation of the fungus to resistant varieties of wheat can occur in the period of three to four years [32]. In fact, failure to actively select for more resistant varieties of wheat can result in severe losses of yield. Roland F. Line (2002) outlines a history of the major epidemics and yield losses in the US caused in part by the lack of research activity for selection against stripe rust [19].

Another method for fungi control is the timely application of chemical treatment to protect crop yield from potential losses caused by the pathogen [15]. Fungicides applied at the beginning stages of an infection can stop the further spread of infection more effectively. However, due to the short time span (7-10 days) between infection and sporulation relative to the fungicide application times (applications per month) and crop harvest timespan (six months), monitoring must be continued after applying fungicide to ensure there is no re-emergence of the infection.

Fungicides are usually reapplied during the growth of crops because of natural degradation and weathering, low application coverage, and fungicide resistance. Fungicide resistance mechanisms are not completely understood and to date there is no significant evidence of fungicide resistance developed by stripe rust [5].

Fungicides may be categorized in several ways: according to mobility through the plant tissue, protective role, activity breadth, mode of action, or by chemical composition [24]. Fungicide mobility in the plant is divided into two classes: contact and systemic. Contact fungicides act

on the surface of the leaf and protect only the treated area from further infection. In contrast, systemic fungicides are transported through the plant tissue and are able to have a protective post-infection effect. Fungicides are also categorized by their protective role: they can have preventative, early-infection, eradication, and anti-sporulant effects. Preventative effects act as a barrier to the pathogen, whilst early-infection and eradication effects stop disease development before and after infection occurs, respectively.

When there is no significant level of the disease, fungicide application can result in net monetary losses. Profitability of fungicide application will depend on several factors, including cultivar resistance, efficacy of fungicide application, environmental conditions conducive to disease development, and prices of both fungicide and wheat [38].

In this paper, we consider a fungicide with both a systemic preventative effect as well as a contact anti-sporulant effect. Since complete curative effects are not currently attainable in wheat rusts, we do not consider the eradication role in our model. Early-infection effects are, for simplicity, included in the preventative and systemic effect of such fungicide. Under the assumption that chemical composition of a fungicide can be varied to provide different rates of protective and anti-sporulant treatments, we set out to determine which fungicidal effect has the most impact in containing the disease under the conditions set out by our model.

2 Model Description

In order to study the dynamics of the stripe rust fungus *Puccinia striiformis* spreading through a wheat field undergoing fungicidal control, we construct a single-host single-pathogen compartmental model that takes into account the protective and anti-sporulant effects.

In our analysis, we divide our total population into four classes: a class of average susceptible leaf area (S) that has not been infected but is prone to infection, a class of average infected and infectious leaf area (I) that is actively producing spores, a class of leaf area prevented from infection (P) that is temporarily immune to infection, and a class of non-infective class (T) that has been infected but does not contribute to sporulation due to anti-sporulant treatment – refer to Figure 1. We assume that the field has a carrying capacity K which allows for a maximum possible leaf area. We let r be the intrinsic growth rate of healthy area using logistic growth with carrying capacity K. As it replicates, the spores of *Puccinia striiformis* consumes resources from the plant that would otherwise be destined for growth of the crop. For this purpose, we assume that only healthy leaf area (susceptible and protected) contributes to new growth, and growth of the infective classes (infected and treated) is stunted. Finally, we assume that the resources of the field are consumed by every class at the same rate.

Our model is a four-dimensional system of nonlinear ordinary differential equations. Equations (1) – (4) illustrate the dynamics of our model.

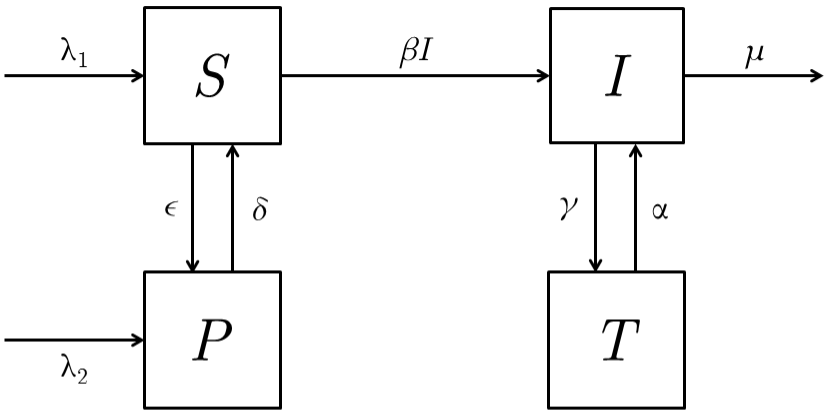
$$\dot{S} = (1-q)\,rP\left(1-\frac{N}{K}\right) + rS\left(1-\frac{N}{K}\right) - \beta SI - \epsilon S + \delta P \tag{1}$$

$$\dot{P} = qrP\left(1-\frac{N}{K}\right) + \epsilon S - \delta P \tag{2}$$

$$\dot{I} = \beta SI - \gamma I + \alpha T - \mu I \tag{3}$$

$$\dot{T} = \gamma I - \alpha T \tag{4}$$

Here, N denotes the sum of the leaf areas in each compartment and is not constant.



$$\lambda_1 = (1-q)rP\left(1-\frac{N}{K}\right) + rS\left(1-\frac{N}{K}\right)$$

$$\lambda_2 = qrP\left(1-\frac{N}{K}\right)$$

Figure 1: This figure illustrates the compartmental model used for determining the epidemiological dynamics of stripe rust and the interactions between susceptible, S, and protected, P, as well as infected, I, and treated, T, due to application of the two fungicidal effects

Since systemic fungicides have the ability of moving inside plant tissue, we assume that a certain proportion (q) of the new growth produced by the protected area will continue to be protected, and the rest $(1-q)$ becomes susceptible. We assume application of the fungicide at some rate ϵ has also a percent efficacy with preventive effect against the fungus. In turn, this protective property has a natural degradation rate of δ. Similarly, γ represents the successful penetration rate of the anti-sporulant property of the fungicide, with a natural degradation rate of α. Table 1 summarizes the explanation of the parameters used in the model, as well as the estimated values used for each parameter. Without considering fungicide effects, leaves in the susceptible class become infected at

Parameter		Units	Estimate	Reference
β	Infection contact rate per leaf area	$\text{days}^{-1}\text{meters}^{-2}$	$[1.5\times10^{-7}, 2.8\times10^{-8}]$	Estimated
q	Proportion of new protected growth	—	0.5	Assumed
r	Per unit area growth rate	days^{-1}	0.074522	[9, 21]
K	Maximum foliage area	meters^2	[1022544, 5317229]	[20, 23, 28]
ϵ	Preventative fungicide application rate	days^{-1}	[0.0111, 0.0222]	[11]
δ	Degradation rate of preventative fungicide	days^{-1}	[0.0333, 0.047619]	[11, 26, 27, 36]
γ	Anti-sporulant fungicide application rate	days^{-1}	[0.0111, 0.0222]	[11]
α	Degradation rate of anti-sporulant fungicide	days^{-1}	[0.0333, 0.047619]	[11, 26, 27, 36]
μ	Natural death rate of infected leaf area	days^{-1}	[0.047619, 0.0714285]	[35]

Table 1: Parameter definitions, units, and values used in our model. A complete description of the derivation of their variability is described in the next section.

a rate β per unit area. Finally, we account for the death rate of infected and infectious areas at rate μ. This death rate is assumed to include both the defense mechanism that the tissue implements to contain the disease, as well as the depletion of nutrients required for its survival due to the stripe rust infection.

Stripe rust spores can travel hundreds of miles in a season [22] carried by wind. As such, spatial effects are not that relevant for relatively small fields used in this model. Instead, we use mass action incidence; the infection of a new susceptible area does not depend significantly on the proximity to an infected area, since the spread of the pathogen is relatively larger than the population density.

3 Estimation of Parameters

3.1 An Assumption for q

The parameter q represents proportion of new wheat growth that remains protected. Throughout leaf growth, cells divide by the biological process of mitosis. During the last stage of mitosis (telophase), cytokinesis, the process by which the cytoplasm of a parent cell is divided to form two daughter cells, occurs simultaneously. We assume that after this, the fungicide in a parent cell will be split into the two daughter cells and thus will protect the new plant cells half as much as it would before. This is equivalent to saying that half of the new cells that grow from the protected class will remain protected, and the other half will be susceptible to infection. Therefore, we assume $q = 0.5$. The actual value of q may change depending on how the effective protection of the systemic fungicide effect changes with concentration of the chemical.

3.2 Estimation of β

The model below provides a method for estimating $\hat{r}$ [37]:

$$\dot{x} = \hat{r}x(1 - x) \tag{5}$$

where x represents the proportion of foliage covered by stripe rust in a field experiment, and obtains $\hat{r} = 0.1096$. The author later describes a corrected value (to account for host growth) of $\hat{r} = 0.149$. In order to use this result for obtaining a rough estimate for β, we need to use an SI model with constant population:

$$\dot{S} = -\beta SI \tag{6}$$
$$\dot{I} = \beta SI \tag{7}$$

Let $N = S + I$. From this, we obtain $S = N - I$, which we can substitute into equation (7) and obtain:

$$\dot{I} = \beta I(N - I) \tag{8}$$

Since N is a constant we can divide equation (8) by N and obtain:

$$\frac{\dot{I}}{N} = \beta I(1 - \frac{I}{N}) \tag{9}$$

We can express β in the form $\beta = \hat{\beta}/N$, and by substituting this into equation (9), we get

$$\frac{\dot{I}}{N} = \hat{\beta}\frac{I}{N}\left(1 - \frac{I}{N}\right) \tag{10}$$

We notice that the definition of I/N is the proportion of foliage infected with stripe rust, which is equivalent to the definition of x in equation (5). Furthermore, if we substitute $x = I/N$ and $\hat{r} = \hat{\beta}$, into equation (10) we obtain equation (5). Therefore, a rough estimate for β is:

$$\beta = \frac{\hat{\beta}}{N} = \frac{r}{N}$$

For our model, population is constant when $N = K$ and therefore

$$\beta \approx \frac{\hat{r}}{K} = \frac{0.1096}{K}$$

If we use the corrected $\hat{r}$, we get:

$$\beta \approx \frac{0.149}{K}$$

It should be noted that the estimate for β does not take into account disease mortality.

3.3 Estimation of K

Crop fields across the United States vary in size, providing a range for the carrying capacity K, the maximum possible foliage area. By taking certain aspects into consideration such as farm size, the number of seeds planted, how many planted seeds germinate into plants, how many leaves are produced on a wheat plant, and the approximate area of each leaf, we are able to derive a general form for the carrying capacity:

$$K = \underbrace{\left(\frac{\text{acres}}{\text{farm}}\right)}_{\text{farm acreage}} \underbrace{\left(\frac{\text{seeds}}{\text{acre}}\right)}_{\text{seeding rate}} \underbrace{\left(\frac{9}{10}\right)}_{\text{germination rate}} \underbrace{\left(\frac{\text{leaves}}{\text{seed}}\right)}_{\#\text{ wheat leaves}} \underbrace{\left(\frac{\text{area}}{\text{leaf}}\right)}_{\text{leaf area}}$$

The range is incoprorated by selecting the median and average farm sizes in the US of 45 and 234 acres, respectively. Seeding rate was chosen as 1,578,000 seeds per acre from a study that carried out field tests in order to determine an ideal seeding rate for optimal yield [3]. Since leaf areas range from 0 to 50cm^2, 20cm^2 was chosen from a relative mean of all leaf areas [4]. Further, the number of leaves for fully developed wheat plants is 8 [23, 28]. The germination rate, the number of seeds who germinate in average, for a good seed is above 90%, and as such we will be taking the lower bound for biological relevance.
Incorporating these values, K becomes:

$$K = \left(\frac{45 \text{ acres}}{1 \text{ farm}}\right)\left(\frac{1,578,000 \text{ seeds}}{1 \text{ acre}}\right)\left(\frac{20 \text{ cm}^2}{1 \text{ leaf}}\right)\left(\frac{8 \text{ leaves}}{1 \text{ seed}}\right)\left(\frac{9}{10}\right)$$

$$K = 10,225,440,000\text{cm}^2 = 1,022,544\text{m}^2 \text{ per farm of 45 acres}$$

Using mean farm acreage of 234, K becomes

$$K = \left(\frac{234 \text{ acres}}{1 \text{ farm}}\right)\left(\frac{1,578,000 \text{ seeds}}{1 \text{ acre}}\right)\left(\frac{20 \text{ cm}^2}{1 \text{ leaf}}\right)\left(\frac{8 \text{ leaves}}{1 \text{ seed}}\right)\left(\frac{9}{10}\right)$$

$$K = 53,172,288,000\text{cm}^2 = 5,317,289\text{m}^2 \text{ per farm of 234 acres}$$

3.4 Estimation of r

We will use the value of K associated wth farms maintaining 45 acres of cropland, 1,022,544m^2 [3, 4, 20, 23, 28]. To find t, we look at the time of season just before the head of the wheat begins growing. The leaves of the wheat plant reach their maximum growth at the end of booting, just prior to the beginning of heading, around 18 weeks into the season [9, 21]. The value of S_0 is a variable number since it depends on when the wheat leaves are considered initially susceptible to the disease. We set initial susceptibility at the begining of the tilling phase, around 4 weeks after sowing, when there are about 3 smaller leaves on the plant [9]. Thus, t becomes 14 weeks, or 98 days, to match our other time parameters. For S_0, we assume that leaves at this 4 week stage provide approximately 1/16 of the total area, so in the infection-free setting, S_0 is approximately $K/16$. Finally, we'll consider that at the final time (after the 18th week), the plants will be 99% of the total carrying capacity. With these values considered, the logistic explicit solution turns into

$$.99K = \frac{K\left(\frac{K}{16}\right)e^{98r}}{K + \left(\frac{K}{16}\right)(e^{98r} - 1)}$$

Solving for r, we get that

$$r = .074522\text{m}^2 \text{ per day}$$

An interesting thing to note is that r does not change at all regardless of what value of K is substituted into the equation. The cap of leaf growth should not have any affect on the rate at which the plants grow. There would be a change in r if we changed the overall length of time the plants have to grow, or if the value of S_0 is manipulated. Thus, for our model, despite what K we use, r will maintain the same value.

3.5 Estimation of ϵ and γ

For application rates of the protective, ϵ, and anti-sporulant, γ, fungicide effects, we take into consideration restrictions on the number of applications per season. For this, we selected the fungicide Stratego [11]. In this case, the single fungicide has both effects, causing the application rates to be identical to one another. Varying by location, there exist laws limiting the application of fungicides (i.e. 1–2 times per season, where a season can be approximately 180 days [11]). Thus, we consider ϵ and γ to range between the normal period of application and a more extreme case when the rate of application will be 4 times per 180 period, or [0.0111,0.0222].

3.6 Estimation of δ and α

The estimations for the degradation rates of the preventative effect, δ, and anti-sporulant effect, α, of the fungicide are likely to vary depending on chemical composition and environmental conditions. Factors such as increased humidity, sunlight, temperature will affect the degradation of the fungicide. Under circumstances of heavy rainfall, for example, it is suggested that reapplication of the fungicide is a viable option as most of it will have been washed away [30]. Increased frequency of application may also affect the degradation rate of both effects. In the case of the fungicide Stratego, it has an average degradation rate of 21-30 days. This range is caused by the specific chemicals inside the fungicide that have different rates of decay in efficacy [26, 27], and secondary applications require a minimum of 14 days prior to being resprayed [11, 36]. With these considerations, we consider the values of δ and α to be in the range [0.0333,0.047619].

3.7 Estimation of μ

When the infection under the plant tissue is severe enough, pustules of the spores erupt on wheat leaves, spores are continually produced for a period of 14 to 21 days [35]. If the host tissue remains alive, pustules can continue sporulating even longer. Disregarding fungicide application, sporulation can be partially stopped due to death of plant tissue caused by nutrient restriction as well as a containment mechanism. The average infectious period is $1/\mu$, and this is equivalent to the time interval in which pustules can continue releasing spores. Assuming this period of sporulation is caused by this process of host tissue death, the following estimate is calculated:

$$\mu \in [\frac{1}{21}, \frac{1}{14}]$$

4 Analysis

4.0.1 Stability of Trivial Equilibrium

Evaluating the Jacobian at the first disease free equilibria $E_0 = (0, 0, 0, 0)$, we have that

$$J = \begin{pmatrix} r-\epsilon & r(1-q)+\delta & 0 & 0 \\ \epsilon & rq-\delta & 0 & 0 \\ 0 & 0 & -(\gamma+\mu) & \alpha \\ 0 & 0 & \gamma & -\alpha \end{pmatrix}.$$

The characteristic polynomial of J is

$$\begin{aligned} P_J(t) &= [(-(\gamma+\mu)-t)(-\alpha-t)-\alpha\gamma][(r-\epsilon-t)(rq-\delta-t)-\epsilon(r(1-q)+\delta)] \\ &= (t^2+(\mu+\gamma+\alpha)t+\alpha\mu)(t^2+(\epsilon+\delta-r(q+1))t+r(qr-(\epsilon+\delta))). \end{aligned}$$

By the Routh-Hurwitz Criterion, the quadratic $t^2+(\mu+\gamma+\alpha)t+\alpha\mu = 0$ has both roots in the left-hand side of the complex plane. However, the other quadratic $t^2+(\epsilon+\delta-r(q+1))t+r(qr-(\epsilon+\delta)) = 0$ does not since it requires two conditions to hold: $\epsilon+\delta-r(q+1) > 0$ and $qr-(\epsilon+\delta) > 0$. Therefore the equilibrium $E_0 = (0, 0, 0, 0)$ is unstable.

4.0.2 Stability of Disease-Free Equilibrium

Evaluating the Jacobian at the second disease free equilibrium $E_1 = (\delta K/(\epsilon+\delta), \epsilon K/(\epsilon+\delta), 0, 0)$, we have that

$$J = \begin{pmatrix} -\frac{\epsilon(\delta+\epsilon)+r(\delta-q\epsilon+\epsilon)}{\delta+\epsilon} & \frac{\delta(\delta+\epsilon)-r(\delta-q\epsilon+\epsilon)}{\delta+\epsilon} & -\frac{k\beta\delta+r(\delta-q\epsilon+\epsilon)}{\delta+\epsilon} & -\frac{r(\delta-q\epsilon+\epsilon)}{\delta+\epsilon} \\ \epsilon-\frac{qr\epsilon}{\delta+\epsilon} & -\frac{\delta^2+\epsilon\delta+qr\epsilon}{\delta+\epsilon} & -\frac{qr\epsilon}{\delta+\epsilon} & -\frac{qr\epsilon}{\delta+\epsilon} \\ 0 & 0 & -\gamma-\mu+\frac{k\beta\delta}{\delta+\epsilon} & \alpha \\ 0 & 0 & \gamma & -\alpha \end{pmatrix}$$

The characteristic polynomial of the Jacobian evaluated at the Disease Free Equilibrium splits into two quadratic polynomials. The first quadratic polynomial is

$$\begin{aligned} f(t) &= t^2+[r(1-qP^*/K)+\epsilon+rqP^*/K+\delta]t \\ &\quad +r(1-qP^*/K)\delta+\epsilon rqP^*/K+\epsilon r(1-qP^*/K)+\delta rqP^*. \end{aligned}$$

Since all the coefficients are positive, the roots are on the left side of the complex plane by the Routh-Hurwitz Criterion. Next we examine the roots of the other quadratic polynomial. We have that

$$g(t) \quad = \quad t^2 + (\gamma + \alpha + \mu(1 - R_0)) + \alpha\mu(1 - R_0)$$

Suppose that $R_0 < 1$. Then, by Routh-Hurwitz Criterion, we have that all the roots are to the left side of the complex plane. This implies that the DFE is stable. Now suppose that $R_0 > 1$. Then the leading coefficient and the constant term have different signs which implies that there is a root with positive real part and another root with negative real part. Therefore, the DFE would be unstable in this case.

4.1 The Basic Reproduction Number

We will compute the basic reproduction number by using the next generation operator method. Let

$$\mathscr{F} = \begin{pmatrix} \beta SI \\ 0 \end{pmatrix},$$

and

$$\mathscr{V} = \begin{pmatrix} (\gamma + \mu)I - \alpha T \\ \alpha T - \gamma I \end{pmatrix}.$$

Then

$$F = \begin{pmatrix} \beta\delta K/(\epsilon + \delta) & 0 \\ 0 & 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} (\gamma + \mu) & -\alpha \\ -\gamma & \alpha \end{pmatrix}.$$

Computing FV^{-1}, we have that

$$FV^{-1} = \begin{pmatrix} \beta\delta K/(\mu(\epsilon + \delta)) & \beta\delta K/(\mu(\epsilon + \delta)) \\ 0 & 0 \end{pmatrix}$$

By taking the spectral radius of this matrix, we have that

$$\mathcal{R}_0 = \frac{\beta\delta K}{\mu(\delta + \epsilon)}.$$

This number represents the average amount of secondary infections produced by an infected unit area of leaf introduced in a completely susceptible crop during its total infectious period. If $\mathcal{R}_0 < 1$ then the disease-free equilibrium is stable. If $\mathcal{R}_0 > 1$ the endemic equilibrium is stable.

4.2 Endemic Equilibrium

$$\begin{aligned} S' &= rS(1-N/K) + r(1-q)P(1-N/K) - \beta SI - \epsilon S + \delta P \\ P' &= rqP(1-N/K) + \epsilon S - \delta P \\ I' &= \beta SI + \alpha T - (\gamma + \mu)I \\ T' &= \gamma I - \alpha T \end{aligned}$$

We find the endemic equilibria in the following manner:

Suppose $I^* \neq 0$. Then $T^* = \frac{\gamma}{\alpha} I^*$ and $S^* = \frac{\mu}{\beta}$. Solving for I^* in the equation $S' + P' = 0$ we have that

$$I^* = \frac{(S^* + P^*)(K - S^* - P^*)}{(S^* + P^*)(1 + \frac{\gamma}{\alpha}) + \frac{K\mu}{r}}.$$

Substituting I^* in $P' = 0$, we have that P^* is the solution to the equation

$$\begin{aligned} 0 = & -\left(\frac{rqK\mu}{r} + \delta\left(1 + \frac{\gamma}{\alpha}\right)\right) P^2 \\ & + \left[(rq(K-S) - \delta K)\frac{K\mu}{r} + (\epsilon - \delta)SK\left(1 + \frac{\gamma}{\alpha}\right)\right] P + \epsilon KS\left[\left(1 + \frac{\gamma}{\delta}\right) S + \frac{K\mu}{r}\right] \end{aligned}$$

Since the leading coefficient and the constant term have opposite signs, it is easy to show that there exists a unique endemic equilibrium. The stability is undetermined, but it is suspected to be stable if $R_0 > 1$. Numerical simulations were used to support this hypothesis.

4.3 Case $q = 0$

We analyze the sub-case where the preventive effect is not inherited by new growth of protected leaves. When the proportion of protected leaf areas that remain protected, q, is zero, our system reduces to:

$$\begin{aligned} \dot{S} &= r(S+P)\left(1 - \frac{N}{K}\right) - \beta SI - \epsilon S + \delta P \\ \dot{P} &= \epsilon S - \delta P \\ \dot{I} &= \beta SI - \gamma I + \alpha T - \mu I \\ \dot{T} &= \gamma I - \alpha T \end{aligned}$$

Let $I = 0$, then $T = 0$. At equilibrium,

$$\dot{S} + \dot{P} = r(S+P)\left(1 - \frac{(S+P)}{K}\right) = 0$$

where either $S = P = 0$ (trivial) or $S + P = K$ Substituting $P = \frac{\epsilon}{\delta S}$ from $\dot{P} = 0$ and solving for S yields

$$S = \frac{\delta K}{\delta + \epsilon}$$

Therefore, our disease free equilibrium is $E_1 = (\delta K/(\epsilon + \delta), \epsilon K/(\epsilon + \delta), 0, 0)$.

It can easily be shown that the reproductive number is also $R_0 = \frac{\beta\delta K}{\mu(\delta+\epsilon)}$.

To calculate the endemic equilibrium, assume $I \neq 0$. $\dot{T} = 0$ gives $T = \frac{\gamma}{\alpha} I$. Using this result in $\dot{I} = 0$ results in $S = \frac{\mu}{\beta}$. In turn, $\dot{P} = 0$ yields $P = \frac{\epsilon}{\delta} S = \frac{\mu}{\beta}(1 + \frac{\epsilon}{\delta})$. Finally, substituting all of the above in $\dot{S} = 0$ we get

$$\dot{S} = r(S+P)\left(1 - \frac{S + P + (1 + \frac{\gamma}{\alpha})}{K}\right) - \mu I = 0$$

Which we can solve for I as

$$I = \frac{r(S+P)(k - (S+P))}{r(S+P)(1+\frac{\gamma}{\alpha}) + \mu K}$$

From boundedness in appendix B, $K \geq N = S + P + I + T \geq S + P$. Thus, we find the following positive endemic equilibrium:

$$\left(\frac{\mu}{\beta}, \frac{\epsilon\mu}{\delta\beta}, \frac{\alpha r\left(\beta^2\epsilon + \delta\right)\left(\beta\delta K - \mu\left(\beta^2\epsilon + \delta\right)\right)}{\beta\delta\left(\alpha\beta\delta K + r(\alpha+\gamma)\left(\beta^2\epsilon + \delta\right)\right)}, \frac{\gamma r\left(\beta^2\epsilon + \delta\right)\left(\beta\delta K - \mu\left(\beta^2\epsilon + \delta\right)\right)}{\beta\delta\left(\alpha\beta\delta K + r(\alpha+\gamma)\left(\beta^2\epsilon + \delta\right)\right)}\right)$$

5 Simulations

5.1 Lower Bound Parameters

In these simulations we use the lower bound values for the parameters from Table 1. The time scales differ between the different stages of the wheat, from the initial leaf to the booting stage. Each stage was simulated 2 times with different proportions of K.

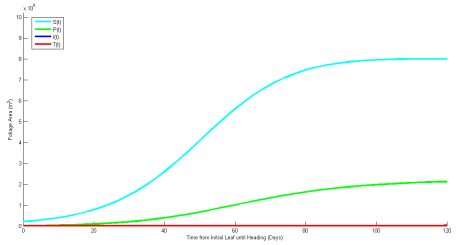


Figure 2: Plot of time against foliage area with $S_0 = \frac{.99K}{48}$, $P_0 = 0$, $I_0 = \frac{.01K}{48}$, $T_0 = 0$

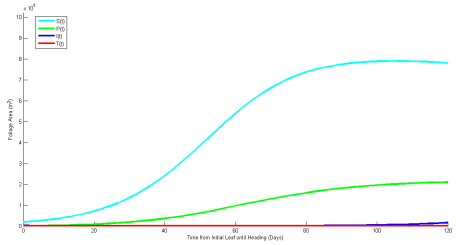


Figure 3: Plot of time against foliage area with $S_0 = \frac{.90K}{48}$, $P_0 = 0$, $I_0 = \frac{.10K}{48}$, $T_0 = 0$

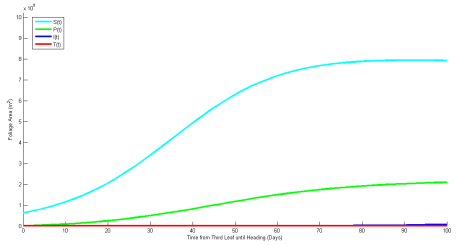


Figure 4: Plot of time against foliage area with $S_0 = \frac{.99K}{16}$, $P_0 = 0$, $I_0 = \frac{.01K}{16}$, $T_0 = 0$

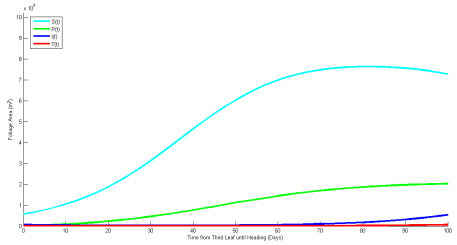


Figure 5: Plot of time against foliage area with $S_0 = \frac{.90K}{16}$, $P_0 = 0$, $I_0 = \frac{.10K}{16}$, $T_0 = 0$

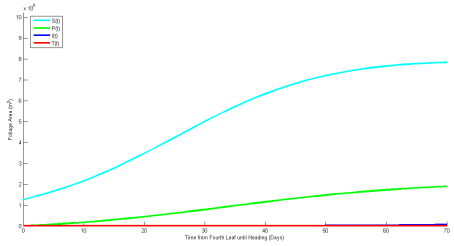


Figure 6: Plot of time against foliage area with $S_0 = \frac{.99K}{8}$, $P_0 = 0$, $I_0 = \frac{.01K}{8}$, $T_0 = 0$

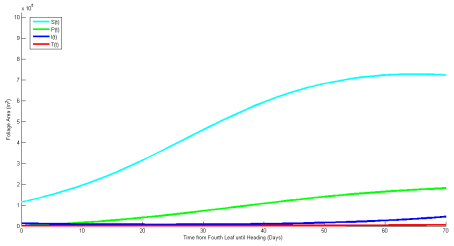


Figure 7: Plot of time against foliage area with $S_0 = \frac{.90K}{8}$, $P_0 = 0$, $I_0 = \frac{.10K}{8}$, $T_0 = 0$

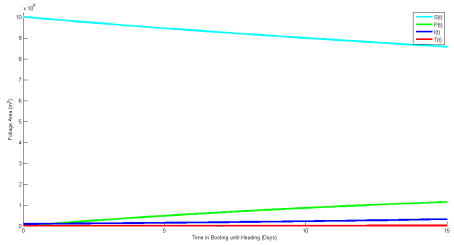


Figure 8: Plot of time against foliage area with $S_0 = (.99)(.99)K$, $P_0 = 0$, $I_0 = (.99)(.01)K$, $T_0 = 0$

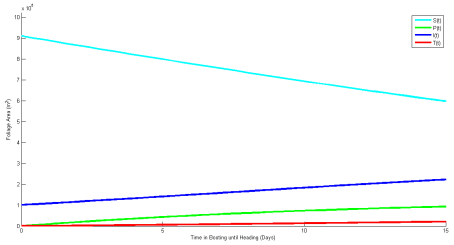


Figure 9: Plot of time against foliage area with $S_0 = (.99)(.90)K$, $P_0 = 0$, $I_0 = (.99)(.10)K$, $T_0 = 0$

The plots of the S and P classes demonstrates the logistic growth of our model. Even while the proportion of K is changing from the initial susceptible and infected at 99% with 1% and 90% with 10%, respectively. From Figures 2 $--$ 7, there is little to no change in the behavior of the model. However in Figures 8 and 9 there is a slight change due to the susceptible population being the total carrying capacity of foliage area. The disease poses no threat under the constant application of the fungicidal effects, showing that the application of fungicide can move or transfer the infectious class to the treated or die out. Since S cannot grow any further than the maximum capacity of foliage area, they die out by transferring into the protected class.

5.2 Upper Bound Parameters

From the values in Table 1, we now take the parameter values on the upper bound for our simulations. The test runs with these parameters at different proportions of K are illustrated as follows:

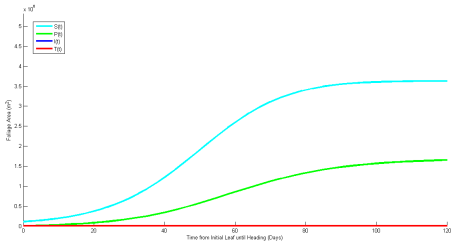


Figure 10: Plot of time against foliage area with $S_0 = \frac{.99K}{48}$, $P_0 = 0$, $I_0 = \frac{.01K}{48}$, $T_0 = 0$

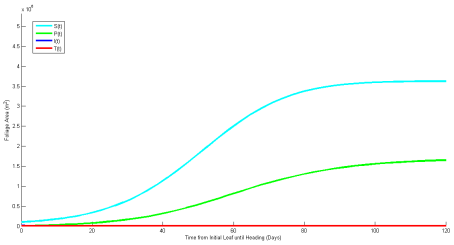


Figure 11: Plot of time against foliage area with $S_0 = \frac{.90K}{48}$, $P_0 = 0$, $I_0 = \frac{.10K}{48}$, $T_0 = 0$

As above in the solutions for the lower bound parameters, the time scale changes for each row

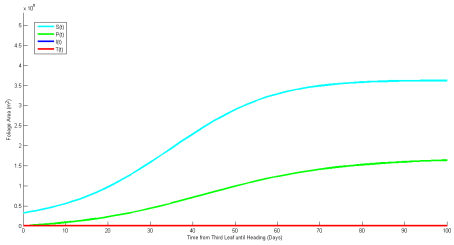


Figure 12: Plot of time against foliage area with $S_0 = \frac{.99K}{16}$, $P_0 = 0$, $I_0 = \frac{.01K}{16}$, $T_0 = 0$

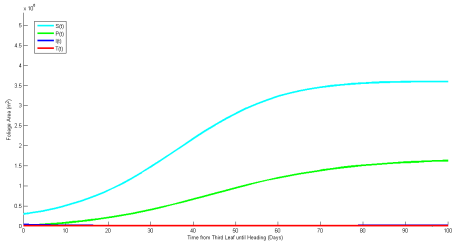


Figure 13: Plot of time against foliage area with $S_0 = \frac{.90K}{16}$, $P_0 = 0$, $I_0 = \frac{.10K}{16}$, $T_0 = 0$

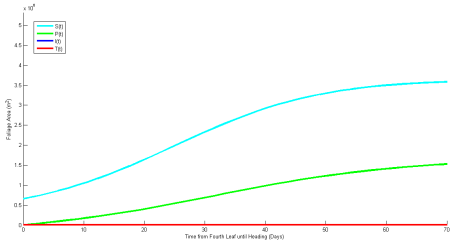


Figure 14: Plot of time against foliage area with $S_0 = \frac{.99K}{8}$, $P_0 = 0$, $I_0 = \frac{.01K}{8}$, $T_0 = 0$

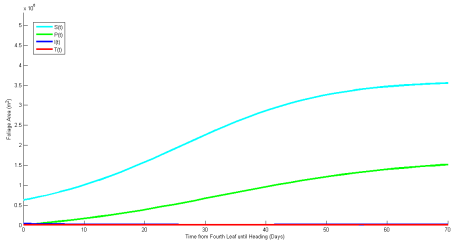


Figure 15: Plot of time against foliage area with $S_0 = \frac{.90K}{8}$, $P_0 = 0$, $I_0 = \frac{.10K}{8}$, $T_0 = 0$

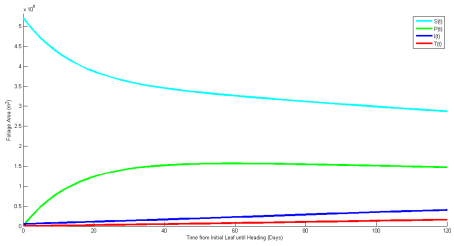


Figure 16: Plot of time against foliage area with $S_0 = (.99)(.99)K$, $P_0 = 0$, $I_0 = (.99)(.01)K$, $T_0 = 0$

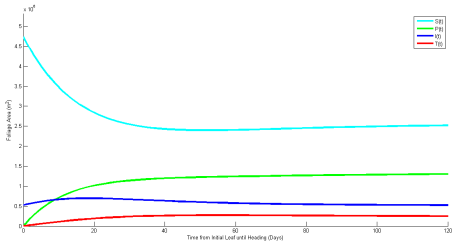


Figure 17: Plot of time against foliage area with $S_0 = (.99)(.90)K$, $P_0 = 0$, $I_0 = (.99)(.10)K$, $T_0 = 0$

of simulations. The proportions of K used in each simulation is kept the same, switching between 99% susceptible with 1% infected and 90% susceptible with 10% infected. In Figures 10 $--$ 15, the behavior of the model is essentially identical. There is no discernable infection, which shows that the consistent application of fungicide is causing those that are initially placed in the infectious class to either die out at the rate μ or transfer to the T class, leaving the S class unaffected and able to transfer into the P class toward the end of each time interval. The plots of S and P in each of these figures also demonstrate a logistic pattern, illustrating the effects of our logistic growth terms in our model. The story changes slightly in Figures 16 and 17, since the initial population of susceptibles is 99% of the total carrying capacity of foliage area. In these two cases, the infectious class acts the same as the prior simulations, but since the S class cannot grow any further the number of susceptibles dies down as they transfer into the protected class.

6 Sensitivity Analysis

6.1 Yield Functional

Let E be the average expected yield from a healthy amount of leaf area. Let $0 \leq a \leq b < 1$, where a represents the contribution of the infected leaf area to the yield, and b the contribution of the treated class to yield. By applying the fundamental theorem of calculus and noting that the initial amount of leaf area is negligible, we arrive to the following:

$$Y = \frac{E}{K}\left(\int_{t_0}^{t_f} \dot{S} + a\int_{t_0}^{t_f} \dot{I} + \int_{t_0}^{t_f} \dot{P} + b\int_{t_0}^{t_f} \dot{T}\right) = E\frac{S(t_f) + P(t_f) + aI(t_f) + bT(t_f)}{K}$$

This allows us to calculate the normalized sensitivity indices below, which represent the ammount of change in yield respective to a 1% change in the parameter in consideration: $\frac{\epsilon}{Y}\frac{\partial Y}{\partial \epsilon}$ and $\frac{\gamma}{Y}\frac{\partial Y}{\partial \gamma}$.

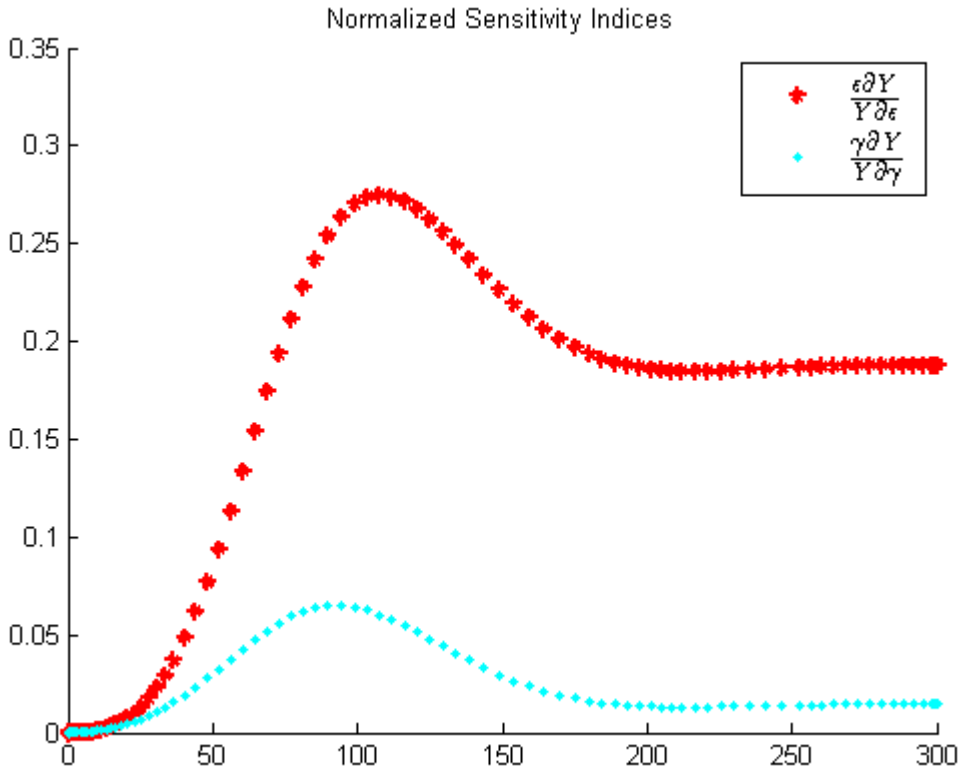


Figure 18: Plots of normalized sensitivity of yield with respect to parameters.

6.2 Estimation of a

In susceptible wheat varieties, stripe rust can potentially cause 75% yield loss [25]. We assume that this loss is due to a complete coverage of the foliage area by the time of harvest, and that during this period the total foliage area is near its carrying capacity. We also assume for simulations that both infected classes (I and T) contribute the same reduced portion to final yield. Using the information

above, we can substitute $Y = (1 - 0.75)E$, $S(t_f) = 0$, $P(t_f) = 0$, $T(t_f) = 0$, and $I(t_f) = K$ and thus obtaining:

$$(1 - 0.75)E = \frac{E}{K} aK$$

Simple algebraic manipulations give us $a = 0.25$

7 Discussion

The parameters explored were those in which we can have some relative control with respect to the fungicide composition. When considering costs related to the fungicide effects, normalized sensitivity indices help us determine which strategy could be most cost-efficient for a continuous application. We observed during simulations that preventive treatment can have a more significant effect than anti-sporulant for large susceptible leaf area and small amount of infectious leaf areas. Preventive treatment was shown to be more cost effective at the beginning of an epidemic, while anti-sporulant effects can have better returns if the application is cheaper and in a later stage of infection. Simulations agree with the common strategy of applying fungicide as early as possible, as often as possible, within monetary and legal restrictions. This practice, however, has been shown to result in monetary losses whenever there is no infection. Similarly, errors in application and effectiveness of the fungicide introduce stochasticity into our model which are not currently accounted for. This model could be extended into a more general framework in which every fungicide effect is explicitly considered. Note that the application of contact fungicide and its effect on the susceptible class was assumed to be negligible. This may not be the case for situations in which there exists homogeneous coverage, with an effect similar to the partial protection models for fungicide previously shown in the literature. It is important to note that this model is a simplified abstraction of the interdependent processes at play. There are numerous factors that were not included in our model in order to be able to conduct analysis. For example, environmental conditions such as wind, humidity and temperature have a significant effect in many of the model parameters and are stochastic in nature. Temperatures and humidity can affect the efficacy and decay of fungicide, as well as the coverage at time of application. Weather has also an important effect in the stages of the rust fungus life cycle over recurring seasons [14] as well as the growth speed of the wheat crop. Assumptions on the homogeneity of the spore distribution in the field do not take into account how an infected plant can act as a source of infection and may not be the best choice when modeling an epidemic where the infection is introduced from a localized source. Spatial spread considerations have been also considered by other studies in which field sizes were large enough to account for spatial effects [33]. Furthermore, the infection dynamics happening within and between the plant occur at different time scales but depend on each other. New and more complex dynamics have been shown to emerge from coupling both systems into the same model [13]. Finally, Although the rust fungi do not develop a significant known resistance [5], the proposed model could be applied to other pathogens in which this mechanism would need to be incorporated into the model.

8 Acknowledgements

We would like to thank Dr. Carlos Castillo-Chavez, Executive Director of the Mathematical and Theoretical Biology Institute (MTBI), for giving us this opportunity to participate in this research program. We would also like to thank Co-Executive Summer Directors Dr. Omayra Ortega and Dr. Baojun Song for their efforts in planning and executing the day-to-day activities of MTBI. We want to extend special thanks to Dr. Anuj Mubayi for helping in modifying our model, Dr. R.P.

Singh for his constant communication to offer his knowledge and expertise in the topic, and all faculty, staff, and students of MTBI.

This research was conducted in MTBI at the Simon A. Levin Mathematical, Computational and Modeling Sciences Center (SAL MCMSC) at Arizona State University (ASU). This project has been partially supported by grants from the National Science Foundation (DMS-1263374 and DUE-1101782), the National Security Agency (H98230-14-1-0157), the Office of the President of ASU, and the Office of the Provost of ASU.

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Appendices

A Positivity

Consider the system of ODEs described by Equations (1)–(4),

$$\begin{aligned}
\dot{S} &= (1-q)\,rP\left(1-\frac{N}{K}\right)+rS\left(1-\frac{N}{K}\right)-\beta SI-\epsilon S+\delta P\\
\dot{P} &= qrP\left(1-\frac{N}{K}\right)+\epsilon S-\delta P\\
\dot{I} &= \beta SI-\gamma I+\alpha T-\mu I\\
\dot{T} &= \gamma I-\alpha T
\end{aligned}$$

where $N = T + I + P + S$. We can see that this model has positivity:

$$\begin{aligned}
\dot{S}\big|_{S=0} &= (1-q)\,rP\left(1-\frac{N}{K}\right)+\delta P \geq 0,\\
\dot{P}\big|_{P=0} &= \epsilon S \geq 0,\\
\dot{I}\big|_{I=0} &= \alpha T \geq 0,\\
\dot{T}\big|_{T=0} &= \gamma I \geq 0.
\end{aligned}$$

B Boundedness

To show the solutions are bounded, we consider the total population. Adding all components of N we get that the population size changes as a function of growth minus death.

$$\begin{aligned}
\dot{N} &= r(S+P)\left(1-\frac{N}{K}\right)-\mu I\\
&\leq rN\left(1-\frac{N}{K}\right)
\end{aligned}$$

Where

$$\dot{x} = rx\left(1-\frac{x}{K}\right)$$

implies

$$\lim x = K$$

Thus, by comparison theorem,

$$\limsup N \leq K$$

C Boundary Equilibria

Suppose that $I = 0$. Then $T = 0$. Adding S' and P' together we have that

$$S' + P' = r\,(S+P)\left(1-\frac{S+P}{K}\right) = 0$$

Possible solutions are $S = P = 0$ or $S + P = K$. Let $S = K - P$ and using $P' = 0$, we have that $\epsilon(K - P) - \delta P = 0$. Solving for P, we have that $P = \frac{\epsilon K}{\epsilon+\delta}$ and $S = \frac{\delta K}{\epsilon+\delta}$.
Thus, we have two disease free equilibria, $E_0 = (0, 0, 0, 0)$ and $E_1 = \left(\frac{\delta K}{\epsilon+\delta}, \frac{\epsilon K}{\epsilon+\delta}, 0, 0\right)$.

D Forward Sensitivity Equations

D.1 Forward Sensitivity Equations for ϵ

$$\begin{aligned}
\frac{d}{dt}\frac{\partial S}{\partial \epsilon} &= r(1-(S+P+I+T)/K)((1-q)\frac{\partial P}{\partial \epsilon}+\frac{\partial S}{\partial \epsilon}) \\
&\quad -r((\frac{\partial S}{\partial \epsilon}+\frac{\partial P}{\partial \epsilon}+\frac{\partial I}{\partial \epsilon}+\frac{\partial T}{\partial \epsilon})/K)((1-q)P+S) \\
&\quad -\beta\frac{\partial S}{\partial \epsilon}I-\beta S\frac{\partial I}{\partial \epsilon}-\epsilon\frac{\partial S}{\partial \epsilon}-S+\delta\frac{\partial P}{\partial \epsilon} \\
\frac{d}{dt}\frac{\partial P}{\partial \epsilon} &= qr(-((\frac{\partial S}{\partial \epsilon}+\frac{\partial P}{\partial \epsilon}+\frac{\partial I}{\partial \epsilon}+\frac{\partial T}{\partial \epsilon})/K))P \\
&\quad +qr(1-(S+P+I+T)/K)\frac{\partial P}{\partial \epsilon}+\epsilon\frac{\partial S}{\partial \epsilon}+S-\delta\frac{\partial P}{\partial \epsilon} \\
\frac{d}{dt}\frac{\partial I}{\partial \epsilon} &= \beta\frac{\partial S}{\partial \epsilon}I+\beta S\frac{\partial I}{\partial \epsilon}-\gamma\frac{\partial I}{\partial \epsilon}+\alpha\frac{\partial T}{\partial \epsilon}-\mu\frac{\partial I}{\partial \epsilon} \\
\frac{d}{dt}\frac{\partial T}{\partial \epsilon} &= \gamma\frac{\partial I}{\partial \epsilon}-\alpha\frac{\partial T}{\partial \epsilon}
\end{aligned}$$

D.2 Forward Sensitivity Equations for γ

$$\begin{aligned}
\frac{d}{dt}\frac{\partial S}{\partial \gamma} &= r(1-((S+P+I+T)/K))((1-q)\frac{\partial P}{\partial \gamma}+\frac{\partial S}{\partial \gamma}) \\
&\quad -r((\frac{\partial S}{\partial \gamma}+\frac{\partial P}{\partial \gamma}+\frac{\partial I}{\partial \gamma}+\frac{\partial T}{\partial \gamma})/K)((1-q)P+S) \\
&\quad -\beta\frac{\partial S}{\partial \gamma}I-\beta S\frac{\partial I}{\partial \gamma}-\epsilon\frac{\partial S}{\partial \gamma}+\delta\frac{\partial P}{\partial \gamma} \\
\frac{d}{dt}\frac{\partial P}{\partial \gamma} &= -qr((\frac{\partial S}{\partial \gamma}+\frac{\partial P}{\partial \gamma}+\frac{\partial I}{\partial \gamma}+\frac{\partial T}{\partial \gamma})/K)P \\
&\quad +qr(1-((S+P+I+T)/K))\frac{\partial P}{\partial \gamma}+\epsilon\frac{\partial S}{\partial \gamma}-\delta\frac{\partial P}{\partial \gamma} \\
\frac{d}{dt}\frac{\partial I}{\partial \gamma} &= \beta\frac{\partial S}{\partial \gamma}I+\beta S\frac{\partial I}{\partial \gamma}-\gamma\frac{\partial I}{\partial \gamma}-I+\alpha\frac{\partial T}{\partial \gamma}-\mu\frac{\partial I}{\partial \gamma} \\
\frac{d}{dt}\frac{\partial T}{\partial \gamma} &= \gamma\frac{\partial I}{\partial \gamma}+I-\alpha\frac{\partial T}{\partial \gamma}
\end{aligned}$$