Mathematical models of the epidemiological dynamics of soil-borne pathogens

by

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Submitted in partial fulfilment of the requirements for the degree

Magister Scientiae

in the Department of Mathematics and Applied Mathematics,

in the Faculty of Natural & Agricultural Sciences

University of Pretoria

Pretoria

August 2018

Declaration

I, Rebecca Anne Bekker declare that the dissertation, which I hereby submit for the degree Magister Scientiae at the University of Pretoria, is my own work and has not previously been submitted by me for a degree at this or any other tertiary institution.

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Summary

Despite the increase in agricultural crop yield over the last century, the world's food supply is in grave danger, as an estimated 16% of global yield is lost to various pathogens annually. As a result, mathematical epidemiology is regularly used to study the mechanisms of transmission, and to determine possible control strategies. A basic analysis of the SEIR model with linear diffusion on the infective compartment published in Gilligan (1995) is carried out first. When the population size is constant the temporal model admits a disease free equilibrium, which is asymptotically stable when $\mathcal{R}_0 \leq 1$, and locally asymptotically stable when $\mathcal{R}_0 > 1$, as well as a locally asymptotically stable endemic equilibrium which only exists when $\mathcal{R}_0 > 1$. Numerical investigations confirm the existence of travelling wave solutions. Next an SEIR model with non-linear diffusion on the infective compartment is investigated numerically. The behaviour of the two models is consistent, although non-linear diffusion with a small diffusion constant results in travelling waves with significantly lower speed.

The host-pathogen model was developed to circumvent the underlying issues of placing a diffusion operator directly onto the infective compartment. This model consists of susceptible and infected hosts, and free and attached pathogen. Although $\mathcal{R}_0 < 1$ for all parameter values, the model admits either only the pathogen free equilibrium **PFE**, or the **PFE** and two endemic equilibria. The **PFE** is always locally asymptotically stable and the global asymptotic stability is proven using two methods: the application of LaSalle's Invariance Principle, and the construction of a monotone system that approximates the model from above. These methods lead to two sets of *sufficient* conditions for the global stability of the **PFE**. The parameter values satisfying these conditions have some overlap. However there are values that satisfy one set and not the other.

Although the stability properties of the endemic equilibria have not been proven, numerical simulations indicate that the equilibrium with the higher level for free pathogen is asymptotically stable on $\mathcal{R}_+^4$, and the other is unstable, with the possibility of being a saddle point. Conditions for the persistence of the pathogen, and thus the infection, were derived. A local sensitivity analysis is completed, and from this possible control methods have been suggested.

The model was extended to include a spatial component, by the addition of diffusion on the free pathogen sub-population. This inclusion did not result in solutions deviating from the behaviour that had been proven for the temporal model. Indeed, under the conditions for persistence, solutions initiated at the level of the stable endemic equilibrium result in a travelling infection front that joins this equilibrium to the **PFE**. The wave speed was calculated for diffusion constants $\mu \in [10^{-7}, 10^{-1}]$, and an equation of the form $c(\mu) = a\mu^b$ was fitted to data. The obtained value of b, namely $b = 0.4189$ is close to the expected value of 0.5 as for FKPP equations.

Acknowledgements

First and foremost I would like to thank my supervisors Prof Roumen Anguelov and Dr Yves Dumont for their invaluable support, guidance and encouragement throughout this project. Prof Anguelov thank you for working with me throughout my postgraduate studies, and believing in me and my abilities. Yves, thank you for your infectious passion for mathematical results with biological interpretations. It reminded me often of why this work is important, and ignited my imagination regarding how maths can be used in epidemiology.

I would also like to acknowledge the support of the South African National Research Foundation and the French Ministry of Foreign Affairs and International Development in the framework of the PHC PROTEA 2015 call, PHC no 38879RD. Thanks also go to the CIRAD UMR AMAP for hosting me for two weeks in Montpellier (France) in 2016 during which time this research was initiated.

Special thanks go to the DST/NRF SARChI Chair in Mathematical Models and Methods in Bioengineering and Biosciences (M^3B^2) for the weekly Biomath Coffee sessions which exposed me to a wide range of models being developed, and opened my eyes to a variety of methods in which mathematics can be applied.

A large thank you goes out to everyone at the Department of Mathematics and Applied Mathematics. Special thanks to Alta van der Merwe, Lorelle September, Rachel Combrink and Ronel Oosthuizen for all their assistance. Many thanks go to Hanlie Venter who took me under her wing and who I was able to lean on multiple times. Thanks go to Rachid Ouifki as well, for all the coffee and impromptu discussions, some of which led to ideas for future work. I would also like to thank Prof Jacquie van der Waals, her PhD students from the Department of Plant and Soil Sciences at the University of Pretoria and Prof Neal McLaren from the University of the Free State for taking the time to discuss the biological aspect of this project with me.

For the sacrifices all four of my parents have made in getting me to this point, thank you from the bottom of my heart. I truly would not have been able to do this without all of you. Extra thanks to my dad for all the discussions regarding the agricultural side, which always served to remind me of the true meaning of this research. To the Robinsons, thank you for allowing me to be a part of your family, and for the support you've given me. I would also like to thank Shirley MacMahon for giving me a place to stay during the past year, and my grandmother who has been my cheerleader and has reminded me to laugh at the little things.

To Gin and Steph thank you for surviving so much, and having full faith that I'll survive too. To everyone in the PhinisheD community - your support and vibes made this much easier. Special thanks to my cousins Sheree and Stefan for being my companions on the road less travelled. Many thanks go to Dipuo, Jamie, and everyone from First Fridays for the space to let off some steam. Many thanks to Phindile and Michelle for supporting me in different ways, yet believing in me completely.

Lastly, to all the people who have helped, supported and contributed to this research, thank you.

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1 Introduction

1.1 Background on Soil-Borne Pathogens

Although agricultural crop yield has increased over the last century, the global food supply is experiencing tremendous pressure from climate change and ever increasing demand. Another major concern is the impact of pathogen since an estimated 16% of the global crop yield is lost to various pathogens annually [10], [62], [90]. Consequently there has been an increase in research of botanical pathogens and the resulting diseases, with foliar pathogens being the focus of the majority of published work. One important difference between foliar and soil-borne pathogens is the environment wherein each occurs. Foliar pathogens have to contend with external factors such as wind, radiation and varying temperatures. However, the soil environment dampens the effects of such factors, although the inherent opacity of soil poses a number of challenges of its own. Added to these are challenges relating to capturing the direct and indirect influence of the environment on processes such as survival, dispersal and germination of pathogens, tissue growth, spatial distribution and susceptibility of hosts [73]. Each of these challenges needs to be thoroughly understood before they can be overcome, which indicates the need for a better understanding of the soil as an environment.

Soil is a dynamic environment, made up of biotic and abiotic components. The biotic component consists of microbes and micro-organisms, which can exhibit mutualistic or antagonistic behaviour. Mutualistic microbes generally occur in the rhizosphere which is the "the portion of the soil that forms the complex habitat of plant roots, the composition of which is altered by root activity" [39]. Examples of mutualistic relationships are *Rhizobium* and members of the pea family and certain *Azospirillum* species, associated with cereal grasses. These bacteria convert free nitrogen into ammonia, which the host uses for development. The rhizosphere is also where plant exudates act as messengers that initiate certain interactions between roots and a large number of soil-organisms [39]. Unfortunately these exudates are often used by pathogens to identify and locate suitable hosts.

The abiotic soil component consists of all non-living factors such as the soil matrix, water, and even temperature. The soil matrix is the solid component of soil and consists mainly of particles of differing sizes, and "usually contains considerable void space since soil particles are irregular in size and shape" [73]. A larger structure does exist, which results from the collection of individual particles into larger units. Clay particles play a role in the formation of these aggregates, as do fungal mycelium, and the mucilaginous materials that plant roots and soil micro-organisms secrete [73]. The larger spaces between aggregates are where water and air infiltration [73], as well as root growth occur [26]. Although roots are also capable of pushing soil particles aside [73] this ability is limited and root growth will decrease abruptly if faced with sufficient resistance within the matrix [26], [100], [110].

The biotic and abiotic soil components, and the interactions between them

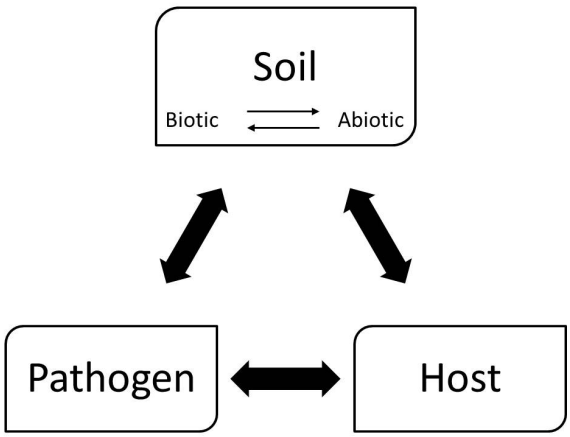

Figure 1.1: The initiation of a disease is dependent on the soil, pathogen and host, and the interactions between them.

influence a pathogens ability to survive in the soil. Soil with a negative impact on a pathogen or its ability to cause disease is said to be suppressive; and can either be generally suppressive, or suppressive to a particular pathogen. Examples of pathogens for which suppressive soils have been found are given in [61],[86], [102], [107], [125]. A pathogen in a suppressive soil either does not persist at all, persists but does not cause disease, or persists and the resulting disease does little to no damage [20]. One would expect that generally suppressive soils therefore be ideal for agricultural crops, and although suppressiveness can be induced by the introduction of certain microbes, the transition from laboratory to field experiments leaves something to be desired [81]. This is largely due to the survival and activity of the 'alien' microbe not being as expected.

In a patch of non-suppressive soil two things are needed for a disease to take hold: a pathogen, and a suitable susceptible host. Soil-borne pathogens can be bacteria, fungi, viruses or nematodes, most of which can survive in the soil in the absence of hosts for some time. This survival can be active or passive, with passive survival achieved by the creation of resting structures, which can lie dormant for various lengths of time before germinating in favourable conditions or in the presence of certain host exudates [59]. The exudates, which are nutrients and microbiological compounds, follow decreasing gradients from the root surface to the soil, and so the influence of the root diminishes with distance [59]. When some pathogens detect exudates of a host, they travel along the gradient to reach the host. However, mycelially-spread fungi, motile spores of fungi, bacteria and nematodes experience undirected movement, which is effectively random. [21].

Once a pathogen comes into contact with a suitable host, entry occurs either via natural openings or specially created structures [39]. If the host plant is immune to the disease minute necrotic flecks appear, as a result of rapid cell death in the vicinity of the invading pathogen [109]. Otherwise, and in favourable conditions, entry leads to infection which is followed by colonization of the host as the pathogen advances.

Infected plants display four main symptoms namely hyperplasia, hypertrophy, hypoplasia, or necrosis [109]. The interaction of symptoms could delay a proper

diagnoses, which increases the impact a pathogen has on the crop. For example, permanent wilt suggests that the up-take and transport of water has been disturbed, although no information regarding the cause or site of the disturbance is revealed. A blockage in the vascular system could be the cause, or general destruction of root tissue. However, it could also be a consequence of excessive water loss due to increased transpiration. Cabbage rot is another example; although the physical symptoms are hypertrophy and hyperplasia of root tissue, the first visible symptom is wilting of the aerial component of the plant [71].

1.2 Historical Background: Modelling Soil-Borne Pathogens

Deterministic epidemiological modelling seems to have started in the 20^{th} century, although Daniel Bernoulli formulated and solved a non-linear differential equation model for smallpox in 1760 [12]. In 1906, in an attempt to understand the recurrence of measles epidemics, Hamer constructed and analysed a discrete time model [42]. This model might have been the first to assume that the number of new cases per unit time depends on the product of the densities of the susecptibles and infectives [44]. Five years later, Ross [97] developed a system of differential equations that highlighted the host-vector nature of malaria to investigate the incidence and control of the disease. Following this, other deterministic epidemiological models were developed and published [4],[27], [28]. A crucial threshold result requiring the density of susceptibles to exceed a critical value in order for an epidemic to occur was also obtained [4], [60], [82].

Literature reviews show the rapid growth of mathematical epidemiology starting in the middle of the 20^{th} century [11], [17], [27], [28], [29], [43], [45], [46], [126]. Although compartmental models were originally applied to human diseases, they can and have been extended to livestock and wildlife diseases, as well as botanical epidemics. A robust theoretical framework for the analysis and control of botanical epidemics has emerged ([34], [35], [77]), developed from pioneering work by van der Plank [116], [117], Waggoner [121], Zadoks [127], [128], Leonard [67], Madden [74] and others.

Although host demographics were often not included in early models ([14], [31], [32],[116]) they are now a feature in most contemporary models ([34] [35], [57], [76], [77]). The inclusion of host dynamics is especially important when the mean duration of a host generation is sufficiently short to have an impact on the outcome of the epidemic [22]. The simplest method of incorporating host growth is to include an exponential function for the net increase rate of the susceptible hosts. While unlimited growth is avoided by density-dependent pathogen-induced host mortality, in the absence of the pathogen biological plausibility collapses. This can be avoided by considering 'natural' host mortality and the growth-decay parameter values can be chosen in such a way that the host population size remains constant. This greatly simplifies the analysis of the model. However, bounded growth functions offer more realism. Two commonly used examples are the monomolecular function which introduces a simple upper bound on host growth, ([32], [37], [55], [75], [106], [114]) and the logistic function which allows for non-linear feedback in the net birth rate of the host ([6], [7], [8], [34], [37], [40],

[41], [94], [95], [112], [123], [124]).

Host roots growing in soils that are highly structured may grow through the channels of old roots [118]. This almost guarantees that new roots come into contact with a high density of pathogen propagules. Along with this, the spread or dispersal of inoculum is itself important to the possible establishment of infection. The extensive porous structure of the soil matrix restricts the dispersion of soil-borne pathogens to such as extent that it is best described as an 'impact filter' [73]. This is presumably overcome by the motility of the pathogen, helping to navigate the soil environment [73]. Linear diffusion has been used to model random undirected movement, with the movement of bacteria ([89], [101]), fungus ([24]) as well as that of nematodes ([30]) having been considered. However, porous or fractal diffusion have also been suggested as more suitable for representing highly structured networks of pores and voids in the soil environment ([30], [70]), although it is possible that such improvements do not lead to any observable differences in model predications, at least for biologically plausible parameter ranges [47].

Gilligan [33] presented an SEIR model with linear diffusion on the infectious subpopulation for the study of soil-borne pathogens, although no biological motivation for this was found during the literature review. This paper presents a refinement of this model by using nonlinear diffusion terms in a host-pathogen model, avoiding the issues that arise from placing a diffusion operator directly onto the infective compartment. The organisation of the thesis is as follows. Chapter 2 introduces the necessary mathematical concepts used in the proceeding analysis of models. In Chapter 3 we discuss Gilligan's model [33] and investigate the effects of changing the diffusion constant. In Chapter 4 we amend the diffusion operator to reflect the assumption that the soil environment is a porous medium. Chapter 5 introduces the temporal host-pathogen model, and, along with the basic properties of the model, presents numerical investigations on the asymptotic behaviour of solutions. We extend the temporal model to include a spatial movement of a sub-population of the pathogen in Chapter 6, and investigate whether this change affects the behaviour of solutions.

2 Theoretical Preliminaries

2.1 Mathematical Preliminaries

2.1.1 Dynamical Systems

We recall some preliminary theorems and definitions that will be used throughout the paper. Let $x(t) \in \mathbb{R}^n$ denote a vector valued function of $t \in \mathbb{R}$. Consider the problem:

$$\dot{x} = f(x) \tag{2.1}$$

where we assume $f \in C^1(\mathbb{R}^n)$

Definition 2.1. Let $\phi_t : I \to \mathbb{R}^n$ be a continuously differentiable function that satisfies (2.1). Then ϕ_t is said to be a solution of the system, and the solution starting at the point $x_0 \in I \subset \mathbb{R}$ is denoted by $\phi_t(x_0)$. ◁

Definition 2.2. A subset $D \subset \mathbb{R}^n$ is said to be positively invariant if

$$\forall x_0 \in D \implies \phi_t(x_0) \in D \quad t \geq 0.$$

Definition 2.3. Equation (2.1), along with an initial condition, are said to define a dynamical system on a subset $E \subseteq \mathbb{R}^n$ if, for every $x_0 \in E$, there exists a unique solution of (2.1) which is defined for all $t \in [0, \infty)$ and remaining in E for all $t \in [0, \infty)$. ◁

Definition 2.4. A dynamical system on $\mathbb{R}^n$ is said to be dissipative if there is a bounded, positively invariant set B with the property that, for any bounded set $E \subseteq \mathbb{R}^n$, there exists $t^* = t^*(B, E) \geq 0$ such that if $x_0 \in E$ then $\phi_t(x_0) \in B$ for all $t > t^*$. The set B is called an absorbing set. ◁

Definition 2.5. If a solution $\phi_t(x)$ is defined for all $t \geq 0$, the positive orbit through the point x is defined as $\gamma^+(x) = \{\phi_t(x) : t \geq 0\}$.

Remark 2.1. If system (2.1) is dissipative, then all forward orbits have compact closure in D.

Definition 2.6. A point $x^* \in \mathbb{R}^n$ such that $f(x^*) = 0$ is called an equilibrium point or steady state of (2.1). ◁

Definition 2.7. An equilibrium point x^* of the dynamical system (2.1) is said to be stable if, for any $\epsilon > 0$, there exists $\delta = \delta(\epsilon) > 0$ such that if $x_0 \in B(x^*, \epsilon)$ for all $t \geq 0$. If, in addition, $\|\phi_t(x_0) - x^*\| \to 0$ as $t \to \infty$ for all $\|x_0 - x^*\|$ is sufficiently small, then the steady state is said to be asymptotically stable. A steady stable which is not stable is said to be unstable. ◁

Theorem 2.1. A steady state x^* of a dynamical system (2.1) is globally asymptotically stable if and only if every neighbourhood centered at the steady state is an absorbing set.

Definition 2.8. Let J_f denote the Jacobian matrix of a function $f : \mathbb{R}^n \to \mathbb{R}^n$ which is defined as

$$J_f = \begin{pmatrix} \frac{\partial f_1}{\partial x_1} & \cdots & \frac{\partial f_1}{\partial x_n} \\ \vdots & \ddots & \vdots \\ \frac{\partial f_n}{\partial x_1} & \cdots & \frac{\partial f_p}{\partial x_n} \end{pmatrix}.$$

Furthermore, the Jacobian can be evaluated at a point u, in which case it is denoted $J_f(u)$.

Theorem 2.2. The Fundamental Existence-Uniqueness Theorem [96]
Let E be an open subset of $\mathbb{R}^n$ containing x_0, and assume that $f \in C^1(E)$. Then there exists a t^* such that the initial value problem has a unique solution $\phi_t(x_0)$ on the interval $[-t^*, t^*]$.

Definition 2.9. An a priori bound is an estimate for the size of a solution of a differential equation or its derivative.

The Latin term translates to 'from before', which refers to the fact that the bound is derived before the solution is known to exist.

Theorem 2.3. [49] If the solution $\phi_t(x_0)$ of (2.1) has an a priori bound A, then the solution exists for all $t \in \mathbb{R}$.

Theorem 2.4. Bony-Brezis Theorem [48]
Let D be closed subset of a C^2 manifold $\mathcal{M}$ and let f be a vector field on $\mathcal{M}$ which is Lipschitz continuous. The following conditions are equivalent:

- Any integral curve of f starting in D remains in D.
- The inner product of f and v is non-positive for any exterior normal vector v at a point m in D. That is, $(f(m), v) \leq 0$.

Theorem 2.5. Gronwall Lemma [108]
Let $z(t)$ satisfy

$$\frac{dz}{dt} \leq az + b, \quad z(0) = z_0,$$

for constants a, b. Then, for $t \geq 0$

$$z(t) \leq e^{at} z_0 + \frac{b}{a}(e^{at} - 1), \quad a \neq 0$$

and

$$z(t) \leq z_0 + bt, \quad a = 0.$$

2.1.2 Matrices and their properties

We present the following definitions for an $n \times n$ matrix A.

Definition 2.10. The scalar ξ is said to be an eigenvalue of A if there is a non-zero vector $u \in \mathbb{R}^n$ such that $Au = \xi u$. The vector u is then the eigenvector corresponding to ξ. The characteristic polynomial of A is $\det(A - \xi I_n) = 0$, and the eigenvalues are the roots of this equation.

Definition 2.11. The set of all eigenvalues of A is denoted $\sigma(A)$, and the stability modulus of A is defined by $s(A) = \max\{Re(\lambda) : \lambda \in \sigma(A)\}$.

Definition 2.12. The matrix A is said to be Metzler if all the off-diagonal entries are non-negative. That is, if $a_{ij} \geq 0$ for all $i \neq j$.

Definition 2.13. A matrix is reducible if and only if it can be placed into block upper-triangular form by simultaneous row/column permutations. A square matrix that is not reducible is said to be irreducible.

2.1.3 Theory of Monotone Systems

Definition 2.14. Let $<_r$ denote any one of the relations $<, \leq$ *or* $\ll$. System (2.1) defines a monotone dynamical system on an ordered metric space D if, for any initial conditions satisfying $x_0 <_r y_0$ the solutions satisfy $\phi_t(x_0) <_r \phi_t(y_0)$.

Definition 2.15. System 2.1 is a cooperative system if $\frac{\partial f_i}{\partial x_j} \geq 0$ for $i \neq j$, $x \in D$.

If f is differentiable, then the system 2.1 is cooperative if and only if the Jacobian matrix J_f is Metzler for each $x \in D$.

Theorem 2.6. [69] Let f be a C^1 vector field in $\mathbb{R}^n$ defined on a convex subset $D \subset \mathbb{R}^n$. Then system (2.1) is monotone if and only if it is cooperative.

Theorem 2.7. [1] Let $\mathbf{a}$, $\mathbf{b} \in D$ such that $\mathbf{a} < \mathbf{b}$, $[\mathbf{a}, \mathbf{b}] \subseteq D$ and $f(\mathbf{b}) \leq \mathbf{0} \leq f(\mathbf{a})$. Then (2.1) defines a (positive) dynamical system on $[\mathbf{a}, \mathbf{b}]$. Moreover, if $[\mathbf{a}, \mathbf{b}]$ contains a unique equilibrium p then p is globally asymptotically stable on $[\mathbf{a}, \mathbf{b}]$.

Theorem 2.8. [104] Let system (2.1) be cooperative in D and let x_0 be an equilibrium. Suppose that the stability modulus of $J_f(x_0)$ is positive, that is $s(J_f(x_0)) > 0$ and there is an eigenvector $v \gg 0$ such that $J_f(x_0)v = sv$. Also, assume that for some $\epsilon > 0$, $\gamma^+(x_r)$ has a compact closure in D for each $x_r \equiv x_0 + rv$, $r \in (0, \epsilon]$. Then there exists $\epsilon_0 \in (0, \epsilon]$, and $e \in E$ such that for each $r \in (0, \epsilon]$, the solution $\phi_t(x_r)$ has the following properties:

1. $x_r \ll \phi_t(x_r) \ll \phi_s(x_r) \ll e$, $0 < t < s$.
2. $\frac{d}{dt}\phi_t(x_r) \gg 0$, $t > 0$.
3. $\phi_t(x_r) \to e$, $t \to \infty$.

If, in addition, $J_f(x_0)$ is irreducible, then there exists y satisfying $x_0 \ll y \ll e$ such that $\frac{d}{dt}\phi_t(y) \gg 0$ for all $t \in \mathbb{R}$, $\phi_t(y) \to e$ as $t \to \infty$ and

4 $\phi_t(y) \to x_0$ as $t \to -\infty$. Furthermore, $\phi_t(y)$ approaches x_0 tangent to the eigenvector v.

Example: The Ross model (Part 1)

One example of a monotone system is the well-known Ross model which can be found in [98] amongst others.

$$\begin{aligned} \dot{x} &= ab_1my(1-x) - \gamma x \\ \dot{y} &= ab_2(1-y)x - \mu y \end{aligned} \tag{2.2}$$

The set $D = \{(x, y) \in \mathbb{R}^2_+ \,|\, 0 \leq x, y \leq 1\}$ can be shown to be positively invariant with respect to system (2.2), and the Jacobian is

$$J_f(x, y) = \begin{pmatrix} -(ab_1my + \gamma) & ab_1m(1-x) \\ ab_2(1-y) & -(ab_2x + \mu) \end{pmatrix}.$$

Thus, since all the off-diagonal entries are non-negative, $J_f(x, y)$ is Metzler on D. Therefore system (2.2) is cooperative.

Example: The Ross model (Part 2)

Clearly $\mathbf{0}$ is an equilibrium of system (2.2). The global asymptotic stability of this *disease free equilibrium* can easily be proven using the properties of monotone systems, namely Theorem 2.7. Based on the definition of D we take $\mathbf{a} = \mathbf{0}$, and $\mathbf{b} = (1,1)^T$. Then,

$$f(\mathbf{0}) = \mathbf{0} \qquad \text{and} \qquad f(\mathbf{b}) = -\begin{pmatrix}\gamma\\ \mu\end{pmatrix} < 0.$$

Since $\mathbf{0}$ is the unique equilibrium in $[\mathbf{0}, \mathbf{b}]$ when $\frac{ma^2b_1b_2}{\mu\gamma} < 1$, it is globally asymptotically stable on $D \equiv [\mathbf{0}, \mathbf{b}]$.

2.1.4 Local Stability Theorems for Non-Linear Systems

The main result of this subsection shows that the non-linear system

$$\dot{x} = f(x) \tag{2.3}$$

has the same qualitative structure near a hyperbolic equilibrium point x^* as the linear system

$$\dot{x} = Ax, \tag{2.4}$$

where $A = J_f(x^*)$. This allows us to circumvent the difficulties that arise when using definition 2.7 to prove the stability properties of system (2.3).

Definition 2.16. An equilibrium point x^* of (2.1) is said to be hyperbolic if all the eigenvalues of $J_f(x^*)$ have non-zero real parts.

Theorem 2.9. Hartman-Grobman
Let E be an open subset of $\mathbb{R}^n$ containing the origin, let $f \in C^1(E)$, and let ϕ_t be the flow of the non-linear system (2.1). Suppose $f(x^*) = 0$ and that $A = J_f(x^*)$ has no eigenvalue with zero real part. Then there exists a homeomorphism H of an open set U containing the origin onto an open set V containing the origin such that for each $x_0 \in U$, there is an open interval $I^* \subset \mathbb{R}$ containing the origin such that for all $x_0 \in U$ and $t \in I^*$

$$H \circ \phi_t(x_0) = e^{At} H(x_0);$$

that is, H maps trajectories of system (2.3) near a hyperbolic equilibrium onto trajectories of system (2.4) near the zero equilibrium and preserves the parameterization by time.

Due to the above theorem it is sufficient to analyse $J_f(x^*)$ in order to determine the stability properties of the hyperbolic equilibrium x^* of system (2.3).

Theorem 2.10. Let x^* be an equilibrium point of the system (2.3) with f continuously differentiable. Then, x^* is asymptotically stable if and only if $s(J_f(x^*)) < 0$ and unstable if and only if $s(J_f(x^*)) > 0$.

Note that Theorem 2.10 does not allow for the case when there are zero eigenvalues.

2.1.5 Lyapunov Theory and LaSalle's Invariance Principle

Theorem 2.11. Lyapunov Theorem
Let $x^* \in D \subset \mathbb{R}^n$ be an equilibrium point for system (2.1). Let $V : D \to \mathbb{R}$ be a continuously differentiable function such that

1. $V(x^*) = 0$ and $V(x) > 0 \quad \forall x \in D \backslash \{x^*\}$, and
2. $\frac{dV}{dt} = \dot{V}(x) \leq 0 \quad \forall x \in D$

then x^* is stable. If, in addition

$$\dot{V}(x) < 0 \quad \forall x \in D \backslash \{x^*\},$$

then x^* is asymptotically stable.

Theorem 2.12. LaSalle's Invariance Principle [65]
Let $\Omega \subset D$ be a compact set that is positively invariant with respect to system (2.1). Let $V : D \to \mathbb{R}$ be a continuously differentiable function such that $\dot{V}(x) \leq 0$ in Ω. Let E denote the set of all points in Ω such that $\dot{V}(x) = 0$. Let M be the largest invariant set in E. Then every solution starting in Ω approaches M as $t \to \infty$.

Example: The Ross model (Part 3)

The basic reproduction number of model (2.2) is $\mathcal{R}_0 := \frac{ma^2 b_1 b_2}{\mu\gamma}$. This quantity indicates the number of new infectious cases caused by a single infective individual in a population that is wholly susceptible. If $\mathcal{R}_0 < 1$ the model admits only the disease free equilibrium **DFE**, which is asymptotically stable on D. If $\mathcal{R}_0 > 1$ the model admits an endemic equilibrium in addition to the **DFE**.

Theorem (2.7) is only applicable to monotone systems, which not all models are. We present an alternative proof, using Theorem (2.11), of the asymptotic stability of **DFE** in the case $\mathcal{R}_0 < 1$, which does not require models to be monotone. Consider the function

$$V(x, y) = (ab_2 + \mu)x + (ab_1 m + \gamma)y.$$

Clearly $V(\mathbf{DFE}) = 0$, and $V(x, y) > 0 \quad \forall \mathbf{x} \in D \backslash \{\mathbf{0}\}$, hence $V(x, y)$ is a Lyapunov function. The time derivative of $V(x, y)$ is

$$\begin{aligned}
\dot{V} &= (ab_2 + \mu)\dot{x} + (ab_1 m + \gamma)\dot{y} \\
&= (ab_2 + \mu)(ab_1 my(1 - x) - \gamma x) + (ab_1 m + \gamma)(ab_2(1 - y)x - \mu y) \\
&= ((ab_2 + \mu)ab_1 m - (ab_1 m + \gamma)\mu)y + ((ab_1 m + \gamma)ab_2 - (ab_2 + \mu)\gamma)x \\
&- 2a^2 b_1 b_2 mxy - a(b_1 m\mu + b_2\gamma)xy \\
&= \gamma\mu(\mathcal{R}_0 - 1)y + \gamma\mu(\mathcal{R}_0 - 1)x - 2a^2 b_1 b_2 mxy - a(b_1 m\mu + b_2\gamma)xy \\
&< 0 \qquad \text{if } \mathcal{R}_0 < 1.
\end{aligned}$$

Thus **DFE** is asymptotically stable on D by Theorem (2.11).

2.1.6 Theory of Algebraic Equations

We recall some results regarding the roots of algebraic equations, with the aim of obtaining methods to determine how many roots of an equation are contained in a specific interval. This will assist us in subsequent subsections. Consider the algebraic equation with real coefficients:

$$p(x) = a_0x^n + a_1x^{n-1} + \cdots + a_{n-1}x + a_n = 0, \quad a_0 \neq 0 \tag{2.5}$$

Theorem 2.13. Descartes' Rule of Sign
The number of positive real roots of the equation (2.5) is either equal to the number of sign changes in the sequence a_0, a_1,...,a_n of coefficients, where vanishing terms are disregarded, or it is less than this by a positive even integer.

A similar theorem for negative roots can be obtained by applying Theorem 2.13 to $p(-x)$. Although Theorem 2.13 is helpful in determining the number of possible positive roots equation (2.5) has, it is sometimes necessary to determine the exact number of roots in a specific interval without explicitly solving the equation. One method of doing so is *Sturm's method*, is explained in detail in [9], while other methods are also presented in [63].

Proposition 2.1. The Sturm chain, $\mathcal{P}$, of equation (2.5) is a sequence of functions and is constructed as follows:

- $p_0(x) = p(x)$
- $p_1(x) = p'(x)$
- $p_i(x) = -rem(p_{i-2}(x), p_{i-1}(x))$ where $rem(p_{i-2}(x), p_{i-1}(x))$ is the remainder after polynomial long division has been used to divide $p_{i-2}(x)$ by $p_{i-1}(x)$, and $2 \leq i \leq n$.

Note that the number of functions in $\mathcal{P}$ is always $n + 1$, where n is the degree of equation (2.5).

Theorem 2.14. Sturm's Method
Given a real algebraic equation 2.5 without repeated roots, let $\sigma(x)$ be the number of sign changes in the Sturm chain $\mathcal{P}$, once vanishing terms have been disregarded. The number of real roots of equation (2.5) located in the interval of real numbers (a, b) is equal to $\sigma(a) - \sigma(b)$.

If $p(x)$ has repeated roots, $p_n(x)$ will not be a constant and $\sigma(a) - \sigma(b)$ is the number of real roots in the interval (a, b), where each repeated root is counted only once.
It is often helpful to know under which conditions an equation will have roots that are negative or have negative real part. These conditions can be found using the *Routh-Hurwitz* criteria.

Theorem 2.15. The Routh Hurwitz Criteria Given the polynomial with real, constant coefficients

$$p(\xi) = \xi^n + a_1\xi^{n-1} + \cdots + a_{n-1}\xi + a_n,$$

define the n Hurwitz matrices using the coefficients a_i of the characteristic polynomial:

$$H_1 = (a_1), \quad H_2 = \begin{pmatrix} a_1 & 1 \\ a_3 & a_2 \end{pmatrix}, \quad H_3 = \begin{pmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ a_5 & a_4 & a_3 \end{pmatrix},$$

and

$$H_n = \begin{pmatrix} a_1 & 1 & 0 & 0 & \cdots & 0 \\ a_3 & a_2 & a_1 & 1 & \cdots & 0 \\ a_5 & a_4 & a_3 & a_2 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 & 0 & \cdots & a_n \end{pmatrix}$$

where $a_j = 0$ if $j > n$. All of the roots of the polynomial $p(\xi)$ are negative or have negative real part if and only if the determinants of all Hurwitz matrices are positive:

$$\det H_j > 0, \quad j = 1, 2, \ldots, n.$$

Corollary 2.1. For polynomials of degree $n = 2, 3, 4$ the Routh Hurwitz criteria are:

$$\begin{aligned} &n = 2 : a_1 > 0 \text{ and } a_2 > 0 \\ &n = 3 : a_1 > 0, a_3 > 0, \text{ and } a_1a_2 > a_3 \\ &n = 4 : a_1 > 0, a_3 > 0, a_4 > 0, \text{ and } a_1a_2a_3 > a_3^2 + a_1^2a_4. \end{aligned}$$

2.2 Epidemiological Preliminaries

2.2.1 The Basic Reproduction Number, $\mathcal{R}_0$

The basic reproduction number is defined as the number of secondary infections a single infectious individual causes in a completely susceptible population, and is denoted by $\mathcal{R}_0$ [115]. In many deterministic epidemiological models an infection invades only when $\mathcal{R}_0 > 1$, thus the quantity is considered an important threshold. The threshold can be calculated heuristically, or by using the next generation matrix approach outlined in [15] which is based on [115].

In this method the population under consideration is divided into $n + m$ compartments, with n infection and m infection-free compartments. Let the sub-populations in each of these compartments be denoted by $x \in \mathbb{R}^n$ and $y \in \mathbb{R}^m$ respectively, and let $\mathcal{F}_i$ denote the rate of appearance of new infections in infection compartment i, and $\mathcal{V}_i$ denote the rate of transfer of individuals out of infection compartment i by disease progression or recovery. The model can then be written as

$$\begin{aligned} \frac{dx_i}{dt} &= \mathcal{F}_i(x, y) - \mathcal{V}_i(x, y), \quad i = 1, 2, ..., n \\ \frac{dy_j}{dt} &= g_j(x, y) \qquad\qquad\qquad\quad j = 1, 2, ..., m. \end{aligned}$$

The function $g_j(x, y)$ governs the change in the j^{th} disease free compartment and includes the terms for any population dynamics that have been included in the formulation of the model, such as growth or decay.

The decomposition of the dynamics of the model into $\mathcal{F}$ and $\mathcal{V}$ may not be unique, and the various decompositions correspond to different epidemiological interpretations of the model. However, since $\mathcal{R}_0$ is interpreted epidemiologically and not mathematically, there are "incorrect" decompositions, however all the possible $\mathcal{R}_0$ quantities have the same behaviour towards 1 [115]. In other words, if one interpretations $\mathcal{R}_0$ is greater than one, all the different interpretations will be greater than one. We make the following assumptions regarding $\mathcal{F}$ and $\mathcal{V}$:

1. $\mathcal{F}_i(0, y) = 0$ and $\mathcal{V}_i(0, y) = 0 \quad \forall\, y \geq 0, \quad$ for $i = 1, 2, ..., , n$
2. The disease free system has a unique asymptotically stable equilibrium $(0, y_0)$.
3. $\mathcal{F}_i(x, y) \geq 0$ for all non-negative x and y.
4. $\mathcal{V}_i(x, y) \leq 0$ when $x_i = 0$.
5. $\sum_{i=1}^{n} \mathcal{V}_i(x, y) \geq 0$ for all non-negative x and y.

By guaranteeing a disease free invariant set, the first assumption ensures that all new infections are secondary infections arising from infected individuals. Thus any solution with no infected individuals at some point in time will be infection free for all time. Assumption 2 ensures that the disease free equilibrium is also an equilibrium of the full system. The third assumption guarantees that $\mathcal{F}_i(x, y)$ is non-negative, since it represents the new infections. $\mathcal{V}_i(x, y)$ denotes a net outflow

from compartment i, and thus must indicate inflow only when the compartment is empty; while $\sum_{i=1}^{n} \mathcal{V}_i(x, y)$ represents the total outflow from all infective compartments and this can not be negative.

Let $\mathbf{F}$ and $\mathbf{V}$ be the Jacobian matrices, with respect to x, of $\mathcal{F}$ and $\mathcal{V}$. That is, $\mathbf{F} = [\frac{\partial \mathcal{F}_i}{\partial x_j}]$ and $\mathbf{V} = [\frac{\partial \mathcal{V}_i}{\partial x_j}]$. As explained in [15] the (i, j) entry of the matrix $\mathbf{F}$ is the rate at which secondary infections are produced in compartment i by an index case in compartment j. The (i, j) entry of the matrix $\mathbf{V}^{-1}$ can be interpreted as the expected time an individual initially introduced into infection compartment j spends in infection compartment i. The (i, j) entry of the product $\mathbf{FV}^{-1}$ is the expected number of secondary infections in the i^{th} compartment produced by an individual originally introduced into compartment j. The matrix $\mathbf{FV}^{-1}$ is called the *next generation matrix* of the model.

Definition 2.17. The basic reproduction number $\mathcal{R}_0$
$\mathcal{R}_0$ is the spectral radius of the next generation matrix in a fully susceptible population. i.e.

$$\mathcal{R}_0 = \rho\left(\mathbf{FV}^{-1}(0, y_0)\right)$$

The eigenvalue associated with $\mathcal{R}_0$ can be interpreted as the distribution of infected individuals, throughout the population, that produces the greatest number of secondary infections per generation. It is a threshold quantity: in general if $\mathcal{R}_0 > 1$ the disease persists in the population but if $\mathcal{R}_0 < 1$ the disease dies out.

3 Model of Soil Infections using Linear Diffusion

3.1 Introduction

In 1989 Murray published a model for the rabies epidemic that was affecting England's fox population at the time. Rabies drastically changes the behaviour of infected wildlife, resulting in infected foxes, which are normally fiercely territorial, randomly roaming areas of up to 200 km^2 a year [87]. To model this disease-induced physical movement, Murray included a linear diffusion operator on the infected compartment, which accurately showed a travelling disease front across England. Citing Murray's model as inspiration, Gilligan (1995) published an SEIR model which included linear diffusion on the infected compartment. This model was applied to a plant population experiencing disease, and various disease fronts were observed in numerical simulations. Although the traditional interpretation of diffusion – that of undirected random physical movement – makes sense in certain contexts, such as Murray's rabies model, it does not hold up to scrutiny in the context of botanical populations. Instead, we interpret the diffusion as the growth of roots or an increase in root density in the compartment on which the operator acts. The placement of the diffusion operator indicates some kind of disease-induced root growth, which might seem counter-intuitive. One example of precisely such an infection is caused by *Gaeumannomyces graminis* var. *tritici* [8].

The model from [33] is:

$$\begin{aligned}
\frac{\partial S}{\partial t} &= bN - dS - \beta SI \\
\frac{\partial E}{\partial t} &= \beta SI - (d+\alpha)E - \sigma E \\
\frac{\partial I}{\partial t} &= \sigma E - (d+\alpha)I - \gamma I + \mu \Delta I \\
\frac{\partial R}{\partial t} &= \gamma I - (d+\alpha)R
\end{aligned} \tag{3.1}$$

$$\begin{aligned}
&S(x,0) \geq 0, E(x,0) \geq 0, \\
&I(x,0) \geq 0, R(x,0) \geq 0 \\
&\frac{\partial I}{\partial x}(-L,t) = \frac{\partial I}{\partial x}(L,t) = 0
\end{aligned} \tag{3.2}$$

It is assumed that the population's growth is exponential and at a rate of b, and both natural and disease induced mortality occur, at rates d and α respectively. The contact rate β, while σ is the rate of passage through the latent compartment, and γ is the recovery rate.

3.2 Analysis of the Temporal Model

We briefly look at the temporal model based on model (3.1), in order to determine the equilibria and their stability properties. This model is:

$$\begin{aligned}
\frac{dS}{dt} &= bN - dS - \beta SI \\
\frac{dE}{dt} &= \beta SI - (d+\alpha)E - \sigma E \\
\frac{dI}{dt} &= \sigma E - (d+\alpha)I - \gamma I \\
\frac{dR}{dt} &= \gamma I - (d+\alpha)R
\end{aligned} \tag{3.3}$$

By adding the four equations we arrive at a differential equation that governs the entire population:

$$\begin{aligned}
\frac{dN}{dt} &= bN - dN - \alpha(E+I+R) \\
&= rN - \alpha(N-S).
\end{aligned} \tag{3.4}$$

We see that, when $\alpha = 0$, the total population increases exponentially when $r := b - d > 0$, and decreases exponentially when $r < 0$. Equation (3.4) is in equilibrium only when $r = 0$ and $\alpha = 0$. In this case the equilibrium is $N^* = N_0 = N(0)$. For the rest of this chapter we assume $\alpha = 0$.

Since $\frac{dN}{dt} = 0$ we have $R(t) = N_0 - S(t) - E(t) - I(t)$, which allows the decoupling of the fourth equation from model (3.3).

$$\begin{aligned}
\frac{dS}{dt} &= bN_0 - dS - \beta SI \\
\frac{dE}{dt} &= \beta SI - (d+\sigma)E \\
\frac{dI}{dt} &= \sigma E - (d+\gamma)I.
\end{aligned} \tag{3.5}$$

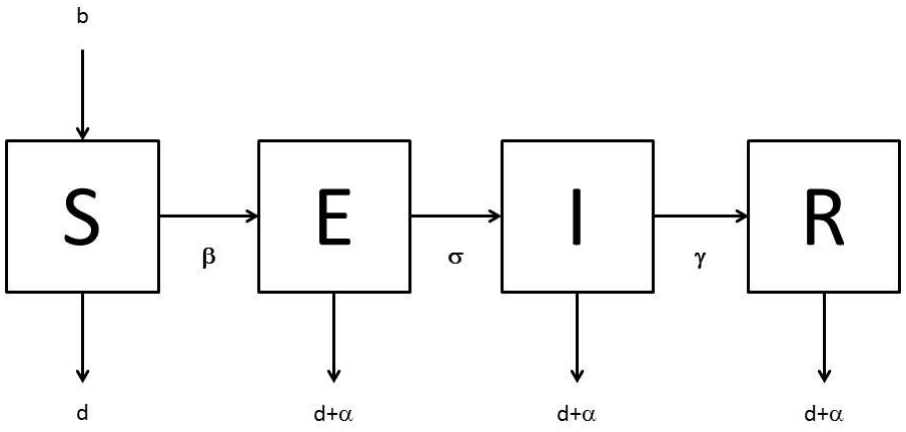

Figure 3.1: Flow chart of the SEIR model.

3.2.1 Basic Reproductive Number

The quantity $\mathcal{R}_0$ is defined as the number of new infections a single infective causes in a completely susceptible population [115]. This is relatively easy to determine for a model with a small number of infection compartments. In unit time the average infective makes βN contacts, all of which are with susceptibles, however, since N is assumed to be constant we have that $N(t) = N_0$ for all $t \geq 0$. The mean infective period is $\frac{1}{(d+\gamma)}$, while the average latency period is $\frac{1}{(d+\sigma)}$, and only the fraction $\frac{\sigma}{(d+\sigma)}$ survives the latency period. Hence,

$$\mathcal{R}_0 = \frac{\sigma\beta N_0}{(d+\gamma)(d+\sigma)}. \tag{3.6}$$

An alternative approach to calculate $\mathcal{R}_0$ is that of the next generation method as described in [15], [115], and listed in section 2.2.1. Model (3.3) has two infection compartments, E and I, and can be decomposed into $\mathcal{F}$ and $\mathcal{V}$ as

$$\mathcal{F} = \begin{pmatrix} \beta SI \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} (d+\sigma)E \\ (d+\gamma)I - \sigma E \end{pmatrix}.$$

The Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ computed to be

$$\mathbf{F} = \begin{pmatrix} 0 & \beta S \\ 0 & 0 \end{pmatrix} \quad \text{and} \quad \mathbf{V} = \begin{pmatrix} d+\sigma & 0 \\ -\sigma & d+\gamma \end{pmatrix}.$$

Next we calculate $\mathbf{V}^{-1}$:

$$\mathbf{V}^{-1} = \begin{pmatrix} \frac{1}{d+\sigma} & 0 \\ \frac{\sigma}{(d+\gamma)(d+\sigma)} & \frac{1}{d+\gamma} \end{pmatrix}.$$

The disease free equilibrium of model (3.3) is $\mathbf{DFE} = (S, E, I, R) = (N_0, 0, 0, 0)$. The product $\mathbf{FV}^{-1}$, evaluated at $\mathbf{DFE}$ is the next generation matrix, namely

$$\mathbf{FV}^{-1}(\mathbf{DFE}) = \begin{pmatrix} \frac{\sigma\beta N_0}{(d+\gamma)(d+\sigma)} & \frac{\beta N_0}{d+\gamma} \\ 0 & 0 \end{pmatrix}.$$

Since $\mathbf{FV}^{-1}(\mathbf{DFE})$ is bidiagonal matrix, the eigenvalues are merely the entries on the main diagonal. Thus,

$$\mathcal{R}_0 = \frac{\sigma\beta N_0}{(d+\gamma)(d+\sigma)}.$$

3.2.2 The Existence of Equilibria

The equilibria are the solutions of the equations obtained by setting the right hand side of system (3.5) equal to zero. That is,

$$\begin{aligned} bN_0 - dS^* - \beta S^* I^* &= 0, \\ \beta S^* I^* - (d+\sigma)E^* &= 0,. \\ \sigma E^* - (d+\gamma)I^* &= 0. \end{aligned} \tag{3.7}$$

The last equation gives the relationship between E^* and I^*, namely

$$E^* = \left(\frac{d+\gamma}{\sigma}\right) I^*,$$

and substituting this into the second equation of (3.7) yields

$$\begin{aligned} \beta S^* I^* - (d+\sigma)\frac{(d+\gamma)}{\sigma} I^* &= 0, \\ \implies S^* = \frac{(d+\sigma)(d+\gamma)}{\beta\sigma} &\text{ or } I^* = 0. \end{aligned}$$

The case $I^* = 0$ results in the disease free equilibrium
DFE $= (S^*, E^*, I^*) = (N_0, 0, 0)$. From the expression $S^* = \frac{(d+\sigma)(d+\gamma)}{\beta\sigma}$ and the first equation in (3.7) we obtain

$$\begin{aligned} bN_0 - \frac{d(d+\sigma)(d+\gamma)}{\beta\sigma} - \beta\frac{(d+\sigma)(d+\gamma)}{\beta\sigma} I &= 0, \\ \implies I = \frac{\beta\sigma bN_0 - d(d+\sigma)(d+\gamma)}{\beta\sigma} &\times \frac{\sigma}{(d+\sigma)(d+\gamma)}, \\ \therefore I^* = \frac{\beta\sigma bN_0 - d(d+\sigma)(d+\gamma)}{\beta(d+\sigma)(d+\gamma)}&. \end{aligned}$$

This is biologically feasible if and only if

$$\beta\sigma bN_0 - d(d+\sigma)(d+\gamma) \geq 0 \quad \iff \mathcal{R}_0 \geq 1.$$

Finally we find E^* to be

$$\begin{aligned} E^* &= \frac{d+\gamma}{\sigma} I^* \\ &= \frac{(d+\gamma)}{\sigma} \frac{\beta\sigma bN_0 - d(d+\sigma)(d+\gamma)}{\beta(d+\sigma)(d+\gamma)}. \end{aligned}$$

The endemic equilibrium of model (3.5) is therefore

$$\begin{aligned} \mathcal{E}_1 = (S^*, E^*, I^*) &= \left(\frac{(d+\sigma)(d+\gamma)}{\beta\sigma}, \frac{d+\gamma}{\sigma} I^*, \frac{\beta\sigma bN_0 - d(d+\sigma)(d+\gamma)}{\beta(d+\sigma)(d+\gamma)}\right) \\ &= \left(\frac{N_0}{\mathcal{R}_0}, \frac{d(d+\gamma)}{\beta\sigma}(\mathcal{R}_0 - 1), \frac{d}{\beta}(\mathcal{R}_0 - 1)\right). \end{aligned}$$

3.2.3 Analysis of the Equilibria

We present a brief local stability analysis of the **DFE** and $\mathcal{EE}_1$. The Jacobian matrix of model (3.3) is

$$J_f = \begin{pmatrix} -d-\beta I & 0 & -\beta S \\ \beta I & -(d+\sigma) & \beta S \\ 0 & \sigma & -(d+\gamma) \end{pmatrix}$$

which, at the **DFE** is

$$J_f(\mathbf{DFE}) = \begin{pmatrix} -d & 0 & -\beta N_0 \\ 0 & -(d+\sigma+\alpha) & \beta N_0 \\ 0 & \sigma & -(d+\gamma) \end{pmatrix}.$$

The characteristic polynomial of $J_f(\mathbf{DFE})$ is

$$q_2(\xi) = -(d+\xi)[(\xi^2 + (2d+\sigma+\gamma)\xi + (d+\sigma)(d+\gamma) - \sigma\beta N_0] = -(d+\xi)q_2^*(\xi).$$

Clearly $\xi = -d$ is a root of $q_2(\xi) = 0$. Consider

$$\begin{aligned} q_2^*(\xi) &= \xi^2 + (2d+\sigma+\gamma)\xi + (d+\sigma)(d+\gamma) - \sigma\beta N_0 \\ &= \frac{1}{\sigma\beta N_0}\left(\sigma\beta N_0\xi^2 + \sigma\beta N_0(2d+\sigma+\gamma)\xi + \frac{1-\mathcal{R}_0}{\mathcal{R}_0}\right). \end{aligned}$$

Since $q_2^*(\xi)$ is quadratic the turning point occurs at

$$\xi = \frac{-(2d+\sigma+\gamma)}{2} < 0,$$

and, if

$$\frac{1}{\sigma\beta N_0}\left(\frac{1-\mathcal{R}_0}{\mathcal{R}_0}\right) > 0 \iff \mathcal{R}_0 < 1,$$

all roots of $q_2^*(\xi) = 0$ will be negative or have negative real part. Thus, the **DFE** is locally asymptotically stable when $\mathcal{R}_0 < 1$. However, if $\mathcal{R}_0 > 1$ the roots of $q_2^*(\xi) = 0$ have different signs and **DFE** is unstable.

The Jacobian matrix of system (3.5) at $\mathcal{EE}_1$ has characteristic equation

$$\begin{aligned} q_3(\xi) = &-\xi^3 - (3d+\sigma+\gamma+\beta I^*)\xi^2 - [(d+\gamma)(d+\sigma) + (2d+\sigma+\gamma)(d+\beta I^*) - \sigma\beta S^*]\xi \\ &- [(d+\gamma)(d+\sigma)(d+\beta I^*) - \sigma\beta d S^*]. \end{aligned}$$

After the simple transformation $q_3^*(\xi) = -q_3(\xi)$ we define

$$\begin{aligned} a_1 &= 3d+\sigma+\gamma+\beta I^*, \\ a_2 &= (d+\gamma)(d+\sigma) + (2d+\sigma+\gamma)(d+\beta I^*) - \sigma\beta S^*, \text{ and} \\ a_3 &= (d+\gamma)(d+\sigma)(d+\beta I^*) - \sigma\beta d S^*. \end{aligned}$$

Theorem 2.1 on page 2.1 guarantees that the roots of $q_3^*(\xi)$ will be negative or have negative real parts if and only if $a_1 > 0$, $a_3 > 0$, and $a_1 a_2 \geq a_3$.

Clearly $a_1 \geq 0$, so we focus on the other two conditions.

$$\begin{aligned} a_3 =&(d+\gamma)(d+\sigma)(d+\beta I^*) - \sigma\beta d I^* \\ =&d(d+\gamma)(d+\sigma)\mathcal{R}_0 - \frac{\sigma\beta d N_0}{\mathcal{R}_0} \\ =&\frac{\sigma\beta d N_0}{\mathcal{R}_0}(\mathcal{R}_0 - 1) > 0 \text{ since } \mathcal{E}_1 \text{ only exists when } (\mathcal{R}_0 - 1) > 0. \end{aligned}$$

We simplify a_2 slightly before considering the third condition:

$$\begin{aligned} a_2 =&(d+\gamma)(d+\sigma) + (2d+\sigma+\gamma)(d+\beta I^*) - \sigma\beta S^* \\ =&d(2d+\sigma+\gamma)\mathcal{R}_0 \end{aligned}$$

Now we consider the third condition, $a_1 a_2 > a_3 \iff a_1 a_2 - a_3 > 0$.

$$\begin{aligned} a_1 a_2 - a_3 =&d(3d+\sigma+\gamma+\beta I^*)(2d+\sigma+\gamma)\mathcal{R}_0 - \left(\frac{\sigma\beta d N_0}{\mathcal{R}_0}(\mathcal{R}_0 - 1)\right) \\ =&d(2d+\sigma+\gamma+d\mathcal{R}_0)(2d+\sigma+\gamma)\mathcal{R}_0 - d(d+\gamma)(d+\sigma)(\mathcal{R}_0 - 1) \\ &+ d^2\gamma\mathcal{R}_0^2 - d^3\mathcal{R}_0 - d^2\sigma\mathcal{R}_0 - d^2\gamma\mathcal{R}_0 - \sigma\gamma d\mathcal{R}_0 + d^3 + (\sigma+\gamma)d^2 + \sigma\gamma d \\ =&3d^2\mathcal{R}_0 + 3d^2\sigma\mathcal{R}_0 + d\sigma^2\mathcal{R}_0 + d\sigma\gamma\mathcal{R}_0 + 3d^2\gamma\mathcal{R}_0 + d\gamma^2\mathcal{R}_0 + 2d^3\mathcal{R}_0 \\ &+ d^2(\sigma+\gamma)\mathcal{R}_0 + d^3 + (\sigma+\gamma)d^2 + \sigma\gamma d \\ >&0 \end{aligned}$$

From Theorem 2.1 we conclude that $q_3^*(\xi)$ has only negative roots, or roots with negative real part. Thus $q_3(\xi)$ has only negative roots, or roots with negative real part, and hence $\mathcal{E}\mathcal{E}_1$ is locally asymptotically stable.

Martcheva (2015) proved that if $\mathcal{R}_0 < 1$, the **PFE** of models similar to (3.3) is globally asymptotically stable, by using a Lyapunov function of the form

$$V = \kappa_1 \left(S - N_0 - N_0 \ln\left(\frac{S}{N_0}\right)\right) + \kappa_2 E + \kappa_3 I,$$

where κ_i are appropriately chosen constants. Since the endemic equilibrium is unique and locally asymptotically stable when $\mathcal{R}_0 > 1$, it could also be globally asymptotically stable. This has indeed been proven using a Lyapunov function of the form

$$V(\mathbf{x}) = \sum_{i=1}^{3} \kappa_i \left(x_i - x_i^* - x_i^* \ln\left(\frac{x_i}{x_i^*}\right)\right)$$

where κ_i is a properly chosen constant, x_i is the population of the ith compartment and x_i^* is the equilibrium level of that compartment. The details of these proofs can be found in [64], [79].

3.3 Numerical Investigation of the Spatio-Temporal Model

We investigate model (3.1) numerically, and compare the simulations to those of model (3.3).

If the temporal model exhibits minimal oscillations before converging to the equilibrium - as it does for the parameter values in Table 3.1, the addition of diffusion with a small diffusion constant results in a standing wave of infection moving through the field. This is illustrated in Figures 3.3-3.4. However, if the temporal model experiences significant oscillations before convergence the addition of diffusion with a small diffusion constant leads to the existence of travelling waves of infection through the field. Three waves of progressively smaller amplitudes, but progressively longer wavelengths are visible in Figure 3.6.

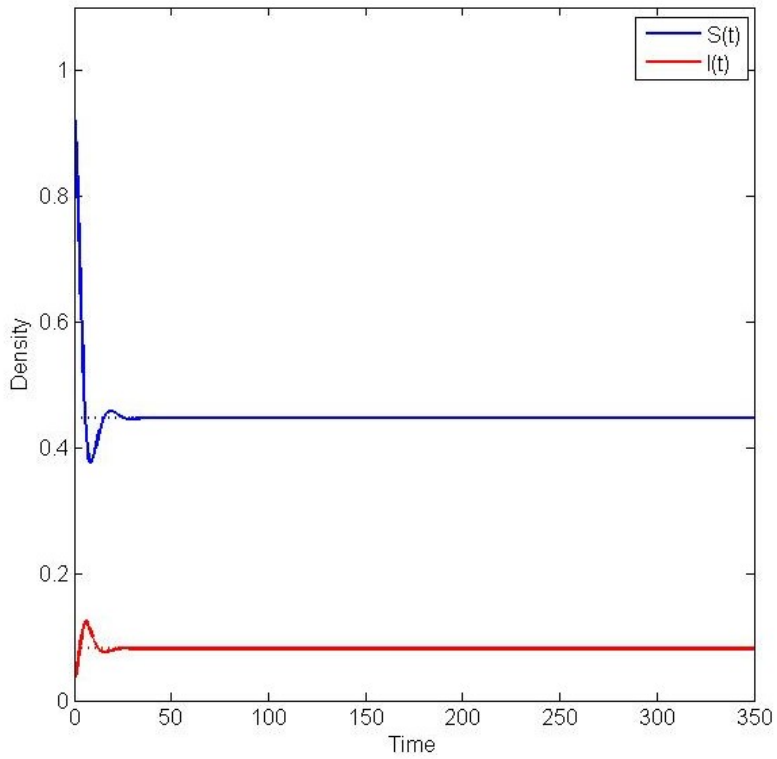

Figure 3.2: The progression of the disease through the compartments S and I over time.

Parameter	Value	Parameter	Value
b	0.20	d	0.20
β	3.00	σ	0.90
γ	0.90	α	0.00

Table 3.1: Parameter values used in Figures 3.2 - 3.4.

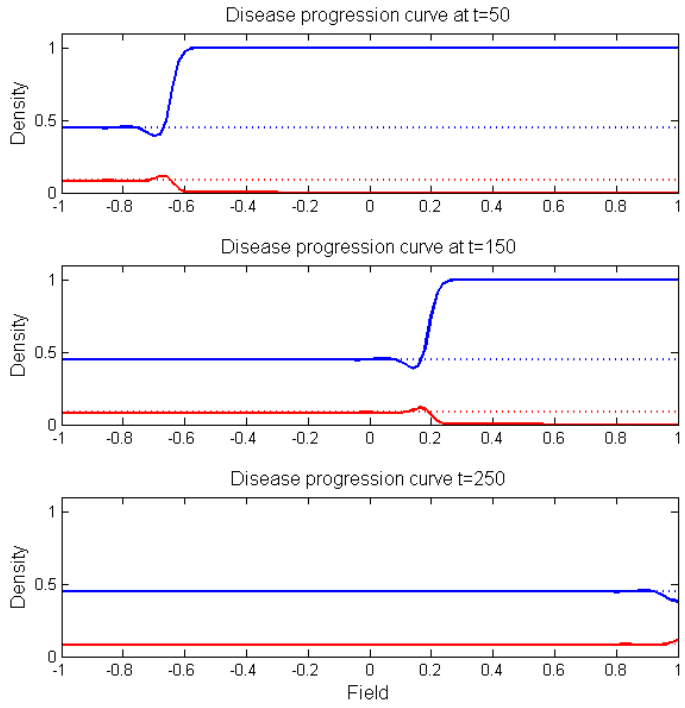

Figure 3.3: Progression of the disease at $t = 50$, $t = 150$ and $t = 250$. The parameter values contained in Table 3.1, and $\mu = 5 \times 10^{-5}$ were used

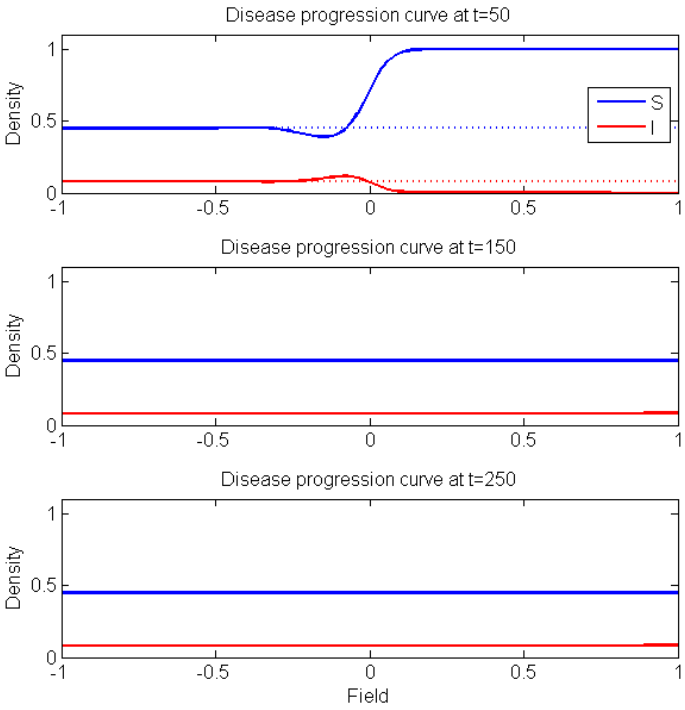

Figure 3.4: Progression of the disease at $t = 50$, $t = 150$ and $t = 250$. The parameter values contained in Table 3.1, and $\mu = 5 \times 10^{-3}$ were used

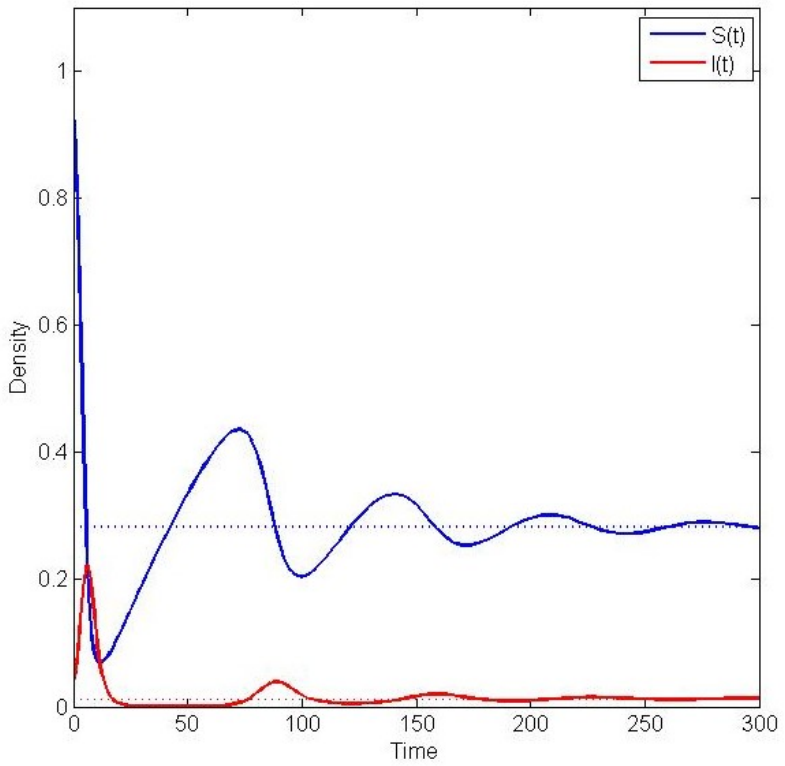

Figure 3.5: The progression of the disease through the compartments S and I over time. The parameter values contained in Table 3.2

Parameter	Value	Parameter	Value
b	0.01	d	0.01
β	2.00	σ	0.85
γ	0.55	α	0.00

Table 3.2: Parameter values used in Figures 3.5 -3.7

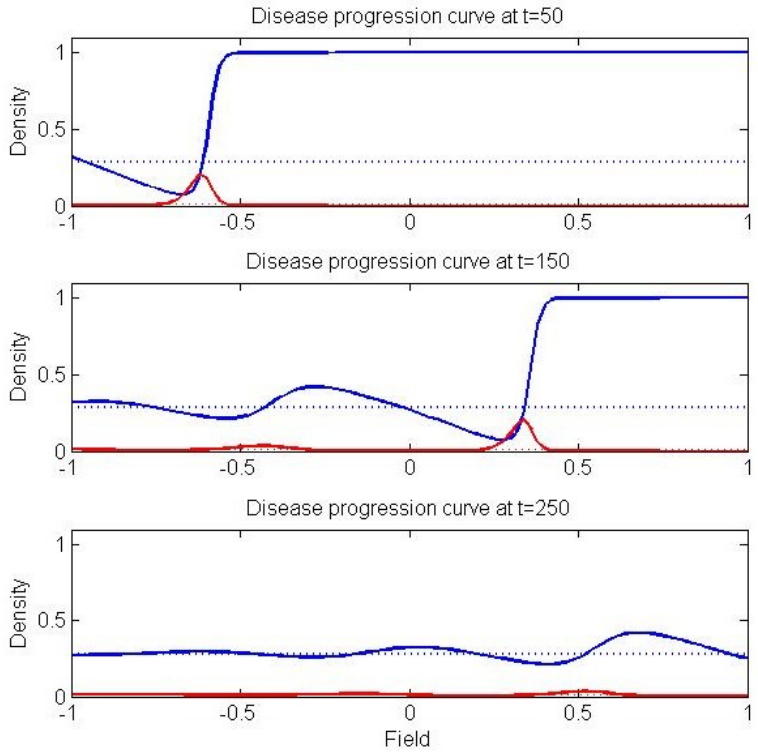

Figure 3.6: Progression of the disease at $t = 50$, $t = 150$ and $t = 250$. The parameter values contained in Table 3.2, and $\mu = 5 \times 10^{-5}$ were used

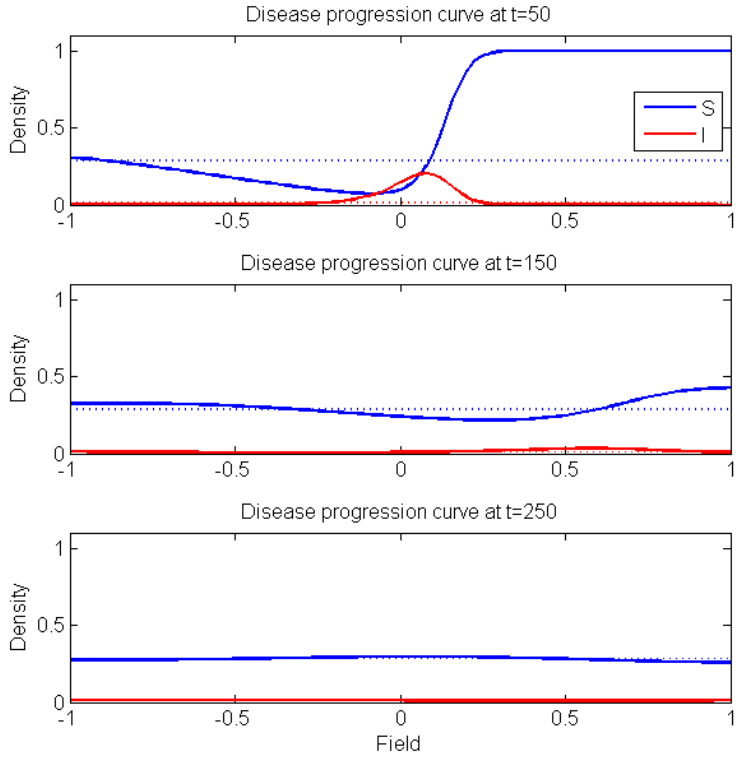

Figure 3.7: Progression of the disease at $t = 50$, $t = 150$ and $t = 250$. The parameter values contained in Table 3.2, and $\mu = 5 \times 10^{-3}$ were used.

4 Model of Soil Infections with Soil as a Porous Medium

4.1 Introduction

In an attempt to improve the realism of the model in section 3 we modify the diffusion term. Instead of assuming random, undirected motion we consider the spread as occurring through a porous medium. This is often used to model the flow of water through soil, and has been suggested to model the motion of fungal mycelium through the soil [70]. Interested readers are referred to [119] for more information regarding this type of movement. The temporal model is identical to model (3.3).

$$\begin{aligned}
\frac{\partial S}{\partial t} &= bN - dS - \beta SI \\
\frac{\partial E}{\partial t} &= \beta SI - (d+\alpha)E - \sigma E \\
\frac{\partial I}{\partial t} &= \sigma E - (d+\alpha)I - \gamma I + \mu\Delta(I^2) \\
\frac{\partial R}{\partial t} &= \gamma I - (d+\alpha)R
\end{aligned} \tag{4.1}$$

$$\begin{aligned}
&S(x,0) \geq 0, \quad E(x,0) \geq 0, \\
&I(x,0) \geq 0, \quad R(x,0) \geq 0 \\
&\frac{\partial I}{\partial x}(-L,t) = \frac{\partial I}{\partial x}(L,t) = 0
\end{aligned} \tag{4.2}$$

4.2 The Numerical Method

The term ΔI^2 is discretized as

$$\Delta I^2 \approx \frac{I_{i+1}^{n+1} I_{i+1}^n - 2I_i^{n+1} I_i^n + I_{i-1}^{n+1} I_{i-1}^n}{h^2},$$

which leads an implicit numerical method that uses non-local approximations of non-linear terms. Each compartment of model 4.1 is approximated by solving systems of the form

$$A_k^n v_k^{n+1} = b_k^n \qquad \text{where } k \in \{S, E, I, R\} \text{ and } n \in \mathbb{N} \tag{4.3}$$

The matrices for the susceptible and infective compartments at time $n+1$ depend on the approximation of I at time n, and a total number of Ns time steps are taken. Let $id(Ns)$ denote the $Ns \times Ns$ identity matrix, then the specific matrices and right hand sides are:

$$\begin{aligned} A_S^n &= (1 + \Delta t(d + \beta v_I^n))id(Ns) \\ b_S^n &= v_S^n + \Delta t b v_N^n \end{aligned}$$

$$\begin{aligned} A_E^n &= (1 + \Delta t(d + \alpha + \sigma))id(Ns) \\ b_E^n &= v_E^n + \Delta t \beta v_S^{n+1} v_I^n \end{aligned}$$

$$\begin{aligned} A_R^n &= (1 + \Delta t(d + \alpha))id(Ns) \\ b_R^n &= v_R^n + \Delta t \gamma v_I^{n+1} \end{aligned}$$

$$b_I^n = v_I^n + \Delta t \sigma v_E^{n+1}$$

The elements of the coefficient matrix A_I depend on non-local approximations and are:

$$\begin{aligned} a_{j,j} &= 1 + \Delta t(d + \alpha + \gamma) + \frac{2\Delta t \mu}{h^2} I_j^n \\ a_{j,j+1} &= -\frac{\Delta t \mu}{h^2} I_{j+1}^n \\ a_{j,j-1} &= -\frac{\Delta t \mu}{h^2} I_{j-1}^n \end{aligned}$$

Take the boundary condition in 4.2 into account we assume $I_0 = I_1$, which results in

$$\begin{aligned} a_{1,1} &= 1 + \Delta t(d + \alpha + \gamma) + \frac{\Delta t \mu}{h^2} I_1^n \\ a_{1,2} &= -\frac{\Delta t \mu}{h^2} I_2^n \end{aligned}$$

Taking the boundary conditions into account on the right means assuming $I_{Ns+1} = I_{Ns}$, and so we obtain

$$\begin{aligned} a_{Ns,Ns} &= 1 + \Delta t(d + \alpha + \gamma) + \frac{2\Delta t \mu}{h^2} I_{Ns}^n \\ a_{Ns,Ns-1} &= -\frac{\Delta t \mu}{h^2} I_{Ns-1}^n. \end{aligned}$$

4.3 Properties of the Numerical Method

Theorem 4.1. The numerical method above preserves the positivity of the model. That is, if $S^0 \geq 0$, $E^0 \geq 0$, $I^0 \geq 0$, and $R^0 \geq 0$ then $S^n \geq 0$, $E^n \geq 0$, $I^n \geq 0$, and $R^n \geq 0$ for all $n \in \mathbb{N}$.

Proof. Considering the structure of the right hand side of the system 4.3, the statement of the theorem clearly holds if $A_S^{-1} \geq 0$,$A_E^{-1} \geq 0$, $A_I^{-1} \geq 0$ and $A_R^{-1} \geq 0$. For A_S, A_E and A_R these inequalities are valid since the matrices are diagonal matrices with positive diagonal entries. It remains to be shown that $A_I^{-1} \geq 0$. We note that the matrix A_I has positive diagonal and nonpositive off-diagonal entries. Using [Theorem 13.9, [83]] together with [Condition 13.10], for $A_I^{-1} \geq 0$ it is enough to show that A_I^T is strictly diagonally dominant.
For $A_I^T = (a_{i,j})$ with $i \in 2, ..., Ns-1$, we have

$$\begin{aligned}\sum_{\substack{i=1\\ i\neq j}}^{Ns} |a_{ij}| &= \left|-\frac{\Delta t\mu}{h^2} I_i^n\right| + \left|-\frac{\Delta t\mu}{h^2} I_i^n\right| \\ &= 2\frac{\Delta t\mu}{h^2} I_i^n \\ &< 1 + \Delta t(d+\alpha+\gamma) + 2\frac{\Delta t\mu}{h^2} I_j^n = a_{i,i}\end{aligned}$$

In a similar way we see that the first and last diagonal entries are also dominant. Indeed,

$$\sum_{i=2}^{Ns} |a_{i1}| = \left|-\frac{\Delta t\mu}{h^2} I_1^n\right| < 1 + \Delta t(d+\alpha+\gamma) + \frac{\Delta t\mu}{h^2} I_1^n = a_{1,1}$$

$$\sum_{i=Ns}^{Ns} |a_{i,Ns}| = \left|-\frac{\Delta t\mu}{h^2} I_{Ns}^n\right| < 1 + \Delta t(d+\alpha+\gamma) + \frac{\Delta t\mu}{h^2} I_{Ns}^n = a_{Ns,Ns}$$

Therefore, A_I^T is strictly diagonally dominant, which completes the proof of the theorem. □

4.4 Numerical Investigation of the Spatio-Temporal Model

The scheme in the previous section was used to investigate the behaviour of solutions of model 4.1. One important question to be answered is whether the inclusion of diffusion through a porous system, rather than linear diffusion changes the behaviour of the solutions significantly. To this end the parameter values in Tables 4.1-4.2 were used, along with either $\mu = 5 \times 10^{-5}$ or $\mu = 5 \times 10^{-3}$.

The parameter values for which the temporal model exhibits minimal oscillations, along with the smaller diffusion coefficient result in a standing wave that travels through the field slowly, invading approximately a third of the field by $t = 250$ (Figure 4.1). Increasing the diffusion coefficient to $\mu = 5 \times 10^{-3}$ results in a wave with greater wavelength that travels at significantly higher speed. In this case the disease infects plants within approximately the first third of the field by $t = 50$, and the entire field is infected by $t = 150$ (Figure 4.2).

As illustrated in Figure 4.3, increasing the diffusion coefficient has a similar effect when the parameter values in Table 4.2 are used. If $\mu = 5 \times 10^{-5}$ the initial wave of infection travels slowly, with successive waves reaching progressively and significantly lower amplitudes. By $t = 250$ this disease has infected less than half of the field. However, if $\mu = 5 \times 10^{-3}$ the wave has travelled to the centre of the field by $t = 50$, and reaches the end of the field before $t = 150$! In this case spatial homogeneity of the disease is almost complete by $t = 250$ (Figure 4.4).

Parameter	Value	Parameter	Value
b	0.20	d	0.20
β	3.00	σ	0.90
γ	0.90	α	0.00

Table 4.1: Parameter values used in Figures 4.1 - 4.2.

Parameter	Value	Parameter	Value
b	0.01	d	0.01
β	2.00	σ	0.85
γ	0.55	α	0.00

Table 4.2: Parameter values used in Figures 4.3 -4.4

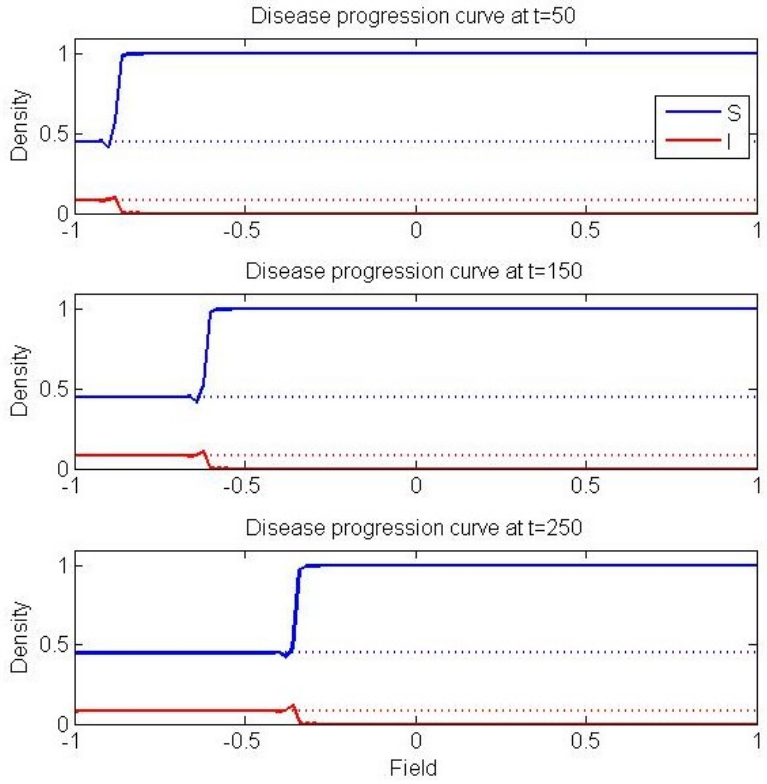

Figure 4.1: Progression of the disease at $t = 50$, $t = 150$ and $t = 250$. The parameter values contained in Table 4.1, and $\mu = 5 \times 10^{-5}$ were used.

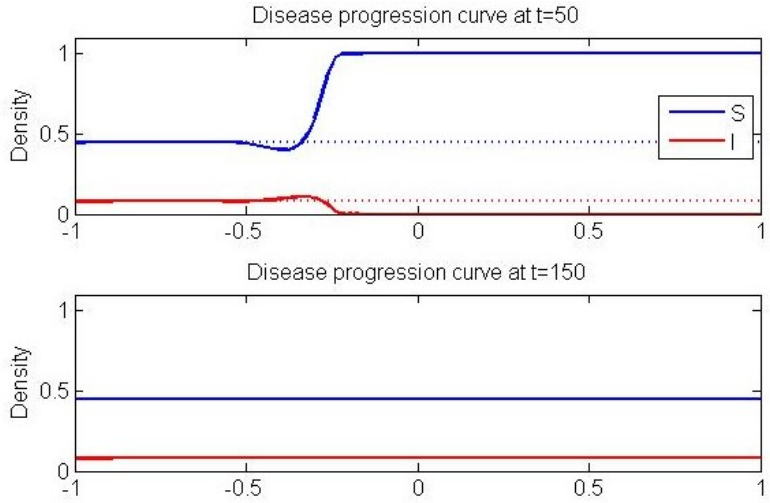

Figure 4.2: Progression of the disease at $t = 50$, $t = 150$ and $t = 250$. The parameter values contained in Table 4.1, and $\mu = 5 \times 10^{-3}$ were used.

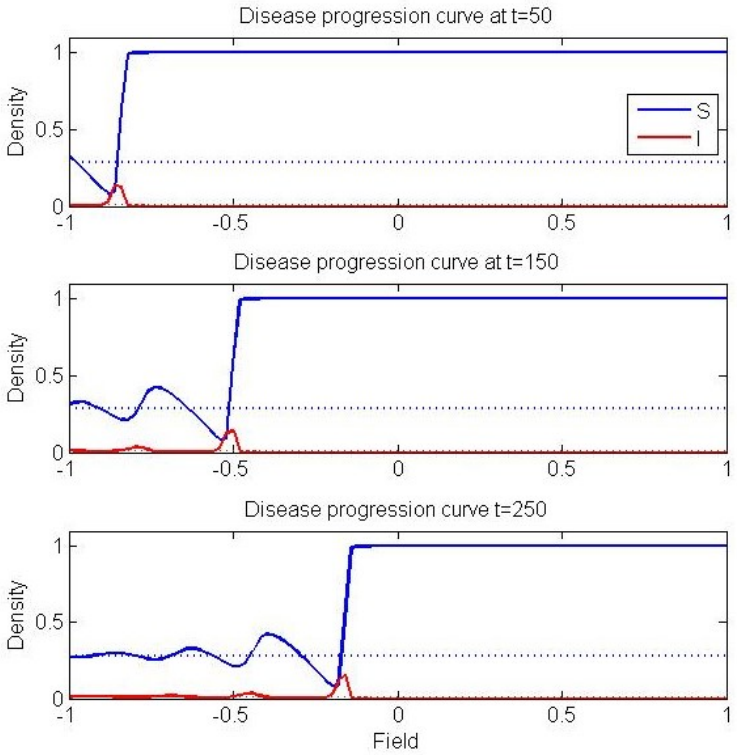

Figure 4.3: Progression of the disease at $t = 50$, $t = 150$ and $t = 250$. The parameter values contained in Table 4.2, and $\mu = 5 \times 10^{-5}$ were used.

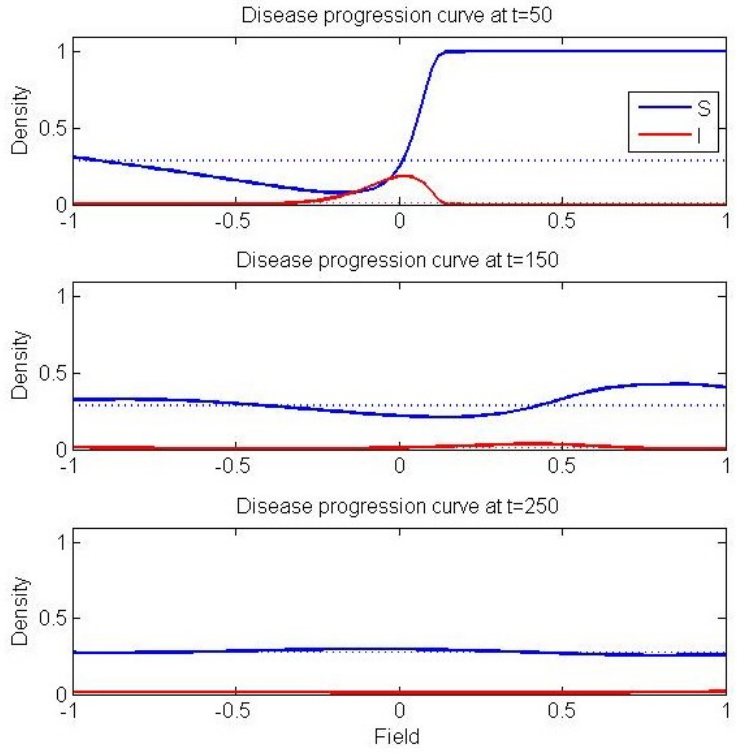

Figure 4.4: Progression of the disease at $t = 50$, $t = 150$ and $t = 250$. The parameter values contained in Table 4.2, and $\mu = 5 \times 10^{-3}$ were used.

5 The Host-Pathogen Model

5.1 Introduction

Consider a population of susceptible host plants with a constant recruitment rate Λ, and a pathogen present in the soil. Assume this pathogen is dependent on its host for nutrients or energy, and as such has an expected off–host death rate δ per time units. After coming into contact with a susceptible host it attaches at rate ρ, and increases in density. The infested host has a limited carrying capacity for the pathogen, which helps regulate the pathogenic increase. These attached pathogen detach from their hosts and go in search of new hosts at a rate of σ per time unit. The natural decay rate of the host is d per time unit, and infected hosts have an addition decay rate of α per time unit. It is assumed that if there is a large initial population of free pathogen, transmission depends solely on β and the level of susceptible hosts present. This type of incidence is called saturation incidence. Using a saturating infestation rate $\frac{\beta F}{M+F}$ is motivated by biological observations that increasing the free pathogen beyond a certain level no longer increases infestation proportionally. From a mathematical point of view, if only mass action principle is applied e.g. βFS, then, since F can potentially be very large, S decreases rapidly, which is unrealistic. For simplicity, the attachment rate is just mass action principle, namely ρFS. However, the growth in the A compartment is limited through the "carrying capacity" $\gamma I = \gamma(N - S)$. Since S cannot decrease unrealistically quickly then A cannot increase unrealistically quickly. The model is presented below.

$$\begin{aligned}
\frac{dA}{dt} &= \lambda A(\gamma I - A) - \sigma A + \rho FS \\
\frac{dF}{dt} &= -\delta F + \sigma A - \rho FS \\
\frac{dS}{dt} &= \Lambda - dS - \frac{\beta F}{M+F}S \\
\frac{dI}{dt} &= \frac{\beta F}{M+F}S - (\alpha + d)I
\end{aligned} \tag{5.1}$$

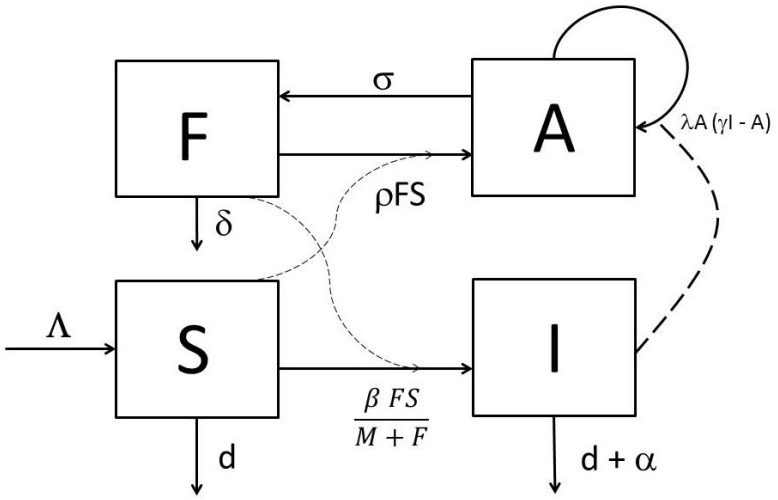

Figure 5.1: Flow chart of the host-pathogen model

5.2 Existence, Uniqueness, Positiveness and Boundedness of Solutions

In this section we prove some basic properties of model (5.1). The notation $x = (A, F, S, I)^T$ allows for the model to be written as

$$\dot{x} = f(x) \quad \text{with} \quad f(x) = \begin{pmatrix} \lambda A(\gamma I - A) - \sigma A + \rho F S \\ -\delta F + \sigma A - \rho F S \\ \Lambda - dS - \frac{\beta F}{M+F} S \\ \frac{\beta F}{M+F} S - (\alpha + d) I \end{pmatrix}.$$

Theorem 5.1. For every $x_0 \in \mathcal{R}^4_+$ there exists t^* such that the system (5.1) has a solution defined on $[0, t^*]$ and satisfying $x(0) = x_0$. Further, the solution is non-negative on its entire interval of existence.

Proof. It is easy to see that the Jacobian of (5.1)

$$J(x) = \begin{pmatrix} \lambda\gamma I - 2\lambda A - \sigma & \rho S & \rho F & \lambda\gamma A \\ \sigma & -\delta - \rho S & -\rho F & 0 \\ 0 & -\frac{\beta M S}{(M+F)^2} & -d - \frac{\beta F}{M+F} & 0 \\ 0 & \frac{\beta M S}{(M+F)^2} & \frac{\beta F}{M+F} & -(\alpha + d) \end{pmatrix}$$

is continuous on an open set E containing $\mathcal{R}^4_+$. In fact, one can take $E = \{x \in \mathcal{R}^4_+ : F > -M\} \supseteq \mathcal{R}^4_+$. It follows from Theorem 2.2 that there exists t^* such that (5.1) has a solution on $[-t^*, t^*]$ satisfying $x(0) = x_0$. It remains to be shown that $\phi_t(x_0) \geq 0$ for $t \geq 0$. For that purpose we apply Theorem 2.4 with $\mathcal{M} = E$ and $D = \mathcal{R}^4_+$. The boundary of D consists of the positive sectors of the coordinate planes, where the second bullet of Theorem 2.4 is easy to verify. For example, on the plane $A = 0$, the exterior normal vector is $v = (-1, 0, 0, 0)^T$ and we have

$$\langle (f(0), F, S, I), v \rangle = -\rho F S \leq 0$$

Then Theorem 2.4 implies that since the solution $\phi_t(x_0)$ has been initiated at $x_0 \in \mathcal{R}^4_+$, it remains in $\mathcal{R}^4_+$ for positive time while it exists. □

Theorem 5.2. The model (5.1) defines a dissipative dynamical system on $\mathcal{R}^4_+$.

Proof. In terms of definition 2.3 we need to show that for every $x_0 \in \mathcal{R}^4_+$ the system (5.1) has a solution on $[0, \infty)$ satisfying $x(0) = x_0$. We obtain these results by applying Theorem 2.3. Let $\phi_t(x_0)$ be a solution of (5.1) initiated in $\mathcal{R}^4_+$. Recall that $\phi_t(x_0) \geq 0$ for $t > 0$ while the solution exists. Therefore the solution is bounded below by $\mathbf{0}$. We obtain an upper bound as follows. The total host population $N = S + I$ satisfies

$$\dot{N} = \Lambda - dN - \alpha I \leq \Lambda - dN$$

Then Theorem 2.5 yields

$$N(t) \leq \frac{\Lambda}{d} + \left(N_0 - \frac{\Lambda}{d} \right) e^{-dt}$$

Hence,

$$\limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{d}, \tag{5.2}$$

which is then also eventually an upper bound for both S and I.
Defining $P = A + I$ as the total pathogen population we find,

$$\begin{aligned}\dot{P} &= \lambda A(\gamma I - A) - \delta F \\ &= \lambda A(\gamma I - A) + \delta A - \delta P \\ &= -\lambda A^2 + (\lambda\gamma I + \delta)A - \delta P\end{aligned}$$

Now, using the fact that the first two terms describe a quadratic function, with maximum value $\frac{\lambda}{4}\left(\gamma I + \frac{\delta}{\lambda}\right)^2$, we obtain that

$$\begin{aligned}\dot{P} &\leq \frac{\lambda}{4}\left(\gamma I + \frac{\delta}{\lambda}\right)^2 - \delta P \\ &\leq \frac{\lambda}{4}\left(\frac{\gamma\Lambda}{d} + \frac{\delta}{\lambda}\right)^2 - \delta P \\ &= \hat{P} - \delta P\end{aligned}$$

After application of Theorem 2.5 we obtain

$$P(t) \leq \hat{P} + (P_0 - \hat{P})e^{-\delta t}$$

Thus,

$$\limsup_{t\to\infty} P(t) \leq \hat{P}, \tag{5.3}$$

and A and F are bounded above. The solution $\phi_t(x_0)$ is then bounded above by $K = (\frac{\Lambda}{d}, \frac{\Lambda}{d}, \hat{P}, \hat{P})^T$, and therefore has an apriori bound. Using Theorem (2.3) we conclude that $\phi_t(x_0)$ can be extended to exist on $[0, \infty)$. According to definition 2.3 model (5.1), along with non-negative initial conditions define a dynamical system.

From above we have that for any $x_0 \in (0, K]$ the solution $\phi_t(x_0) \in [0, K]\ \forall t > 0$. Thus model (5.1) is dissipative according to definition 2.4.

□

5.3 The Basic Reproduction Number $\mathcal{R}_0$

We now know that model (5.1) admits unique positive, bounded solutions, but this tells us little about the disease being modelled. One relatively easy method to glean information abut the infection progression is to calculate the threshold $\mathcal{R}_0$. Called the basic reproduction number, $\mathcal{R}_0$ indicates how many infections are caused by a single infectious individual in a wholly susceptible population. Generally if $\mathcal{R}_0 < 1$ for a disease, contact with more than one infectious individual is required before a susceptible is infected. However, if $\mathcal{R}_0 > 1$ the disease generally persists and becomes endemic to the population. We use the *next generation matrix method* to calculate $\mathcal{R}_0$, which is described in section 2.2.1, and based on [15] and [115]. Assuming that the disease is only in progression in the A and I compartments, and that F is an extension of A, model (5.1) can be decomposed into three infection compartments (A, F, I) and one non-disease compartment S. Let

- $\mathcal{F}_i(x)$ denote the rate of recruitment of new individuals in compartment i, and
- $\mathcal{V}_i(x)$ denote the rate at which the infestation progression, and death decrease the ith compartment.

Model 5.1 can then be written in the form

$$x_i' = \mathcal{F}_i(x) - \mathcal{V}_i(x),$$

with i =1,2,3. As a consequence of the assumption that F is merely an extension of A the vectors $\mathcal{F}$ and $\mathcal{V}$ are

$$\mathcal{F}(x) = \begin{pmatrix} \lambda A(\gamma I - A) + \rho SF \\ 0 \\ \frac{\beta FS}{M+F} \end{pmatrix}, \text{ and } \quad \mathcal{V}(x) = \begin{pmatrix} \sigma A \\ \delta F - \sigma A + \rho FS \\ (\alpha + d)I \end{pmatrix}.$$

The Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$, denoted by $\mathbf{F}$ and $\mathbf{V}$ respectively, at the pathogen free equilibrium $\mathbf{PFE} : (A, F, S, I) = (0, 0, S_0^*, 0)$ with $S_0^* \neq 0$, are computed to be

$$\mathbf{F}(\mathbf{PFE}) = \begin{pmatrix} 0 & \rho S_0^* & 0 \\ 0 & 0 & 0 \\ 0 & \frac{\beta S_0^*}{M} & 0 \end{pmatrix}, \text{ and } \quad \mathbf{V}(\mathbf{PFE}) = \begin{pmatrix} \sigma & 0 & 0 \\ -\sigma & \delta + \rho S_0^* & 0 \\ 0 & 0 & (\alpha + d) \end{pmatrix}.$$

Following definition 2.17 the *next generation matrix* is the product of these matrices, $\mathbf{FV^{-1}}(\mathbf{PFE})$. That is,

$$\mathbf{FV^{-1}}(\mathbf{PFE}) = \frac{1}{M(\delta + \rho S_0^*)} \begin{pmatrix} \rho M S_0^* & \rho M S_0^* & 0 \\ 0 & 0 & 0 \\ \beta S_0^* & \beta S_0^* & 0 \end{pmatrix}, \text{ which has}$$

characteristic equation

$$p(\xi) = \xi^2 \left(\frac{\rho S_0^*}{(\delta + \rho S_0^*)} - \xi \right).$$

Thus

$$\mathcal{R}_0 = \frac{\rho S_0^*}{(\delta + \rho S_0^*)}.$$

It is clear that $\mathcal{R}_0 < 1$ regardless of the expression of S_0^*, and the **PFE** is locally asymptotically stable by the threshold theorem. However, interior equilibria of system (5.1) could exist. We investigate this in the following subsection.

5.4 The Equilibria of the Host Pathogen Model

5.4.1 The PFE, and Endemic Equilibria Equations

We set each equation in model (5.1) equal to zero,

$$\begin{aligned} \lambda A(\gamma I - A) - \sigma A + \rho F S &= 0, \\ -\delta F + \sigma A - \rho F S &= 0, \\ \Lambda - dS - \frac{\beta F}{M+F} S &= 0, \\ \frac{\beta F}{M+F} S - (\alpha + d) I &= 0. \end{aligned} \tag{5.4}$$

From the second equation in (5.4) we obtain an expression for A:

$$A = \sigma^{-1}(\delta + \rho S)F.$$

Similarly, the third equation in (5.4) gives an expression for I in terms of S:

$$\begin{aligned} \frac{\beta F}{M+F} - (\alpha + d)I &= 0, \\ \implies I &= \frac{\beta F S}{(\alpha + d)(M+F)}. \end{aligned}$$

The fourth equation in turns yields an expression for S in terms of F:

$$\begin{aligned} \Lambda - \left(d + \frac{\beta F}{M+F} \right) S &= 0, \\ \implies S &= \frac{\Lambda(M+F)}{(d+\beta)F + dM}. \end{aligned}$$

This expression can be used to obtain an expression for I in terms of F.

$$\begin{aligned} I &= \frac{\beta F}{(\alpha + \beta)(M+F)} \times \frac{\Lambda(M+F)}{(d+\beta)F + dM}, \\ &= \frac{\Lambda \beta F}{(\alpha + d)((d+\beta)F + dM)} \end{aligned}$$

We substitute the expressions found above into the first equation in (5.4) to find an equation in terms of F.

$$\begin{aligned} \lambda A(\gamma I - A) - \sigma A + \rho S F &= 0 \\ \lambda \sigma^{-1}(\delta + \rho S)F(\gamma I - A) - (\delta + \rho S)F + \rho S F &= 0 \\ F[\lambda \sigma^{-1}(\delta + \rho S)(\gamma I - A) - \delta] &= 0 \end{aligned}$$

Thus, either $F = 0$ or $\lambda \sigma^{-1}(\delta + \rho S)(\gamma I - A) - \delta = 0$. If $F = 0$ we have the pathogen free equilibrium

$$\textbf{PFE} : (A, F, S, I) = \left(0, 0, \frac{\Lambda}{d}, 0 \right).$$

Assuming $F \neq 0$, so that

$$\lambda\sigma^{-1}(\delta+\rho S)(\gamma I - A) - \delta = 0,$$
$$\lambda\sigma^{-1}(\delta+\rho S)\gamma I - \lambda\sigma^{-2}(\delta+\rho S)^2 F - \delta = 0.$$

After substituting the expressions for S,I and A we have:

$$\lambda\gamma\sigma^{-1}\left(\delta+\rho\frac{\Lambda(M+F)}{(d+\beta)F+dM)}\right)\frac{\Lambda\beta F}{(\alpha+d)((d+\beta)F+dM)} \\ -\lambda\sigma^{-2}\left(\delta+\rho\frac{\Lambda(M+F)}{(d+\beta)F+dM}\right)^2 F - \delta = 0$$

We multiple this equation by $\sigma^2(\alpha+d)[(d+\beta)F+dM]^2$ to remove the denominators, and obtain

$$\begin{aligned}&\lambda\gamma\sigma\Lambda\beta F[\delta((d+\beta)F+dM)+\rho\Lambda(M+F)]\\&-\lambda(\alpha+d)[\delta((d+\beta)F+dM)+\rho\Lambda(M+F)]^2F\\&-\delta\sigma^2(\alpha+d)[(d+\beta)F+dM]^2=0.\end{aligned}$$

In order to simplify calculations we look at each of the three terms individually, starting with the first term.

$$\begin{aligned}&\lambda\gamma\sigma\Lambda\beta F[\delta((d+\beta)F+dM)+\rho\Lambda(M+F)]\\&=\lambda\gamma\sigma\Lambda\beta(\delta(d+\beta)+\rho\Lambda)F^2+\lambda\gamma\sigma\Lambda\beta(\delta d+\rho\Lambda)MF.\end{aligned} \tag{5.5}$$

The second term can be simplified as follows:

$$\begin{aligned}&-\lambda(\alpha+d)[\delta((d+\beta)F+dM)+\rho\Lambda(M+F)]^2F\\&=-\lambda(\alpha+d)[F(\delta(d+\beta)+\rho\Lambda)+M(\delta d+\rho\Lambda)]^2F\\&=-\lambda(\alpha+d)[\delta(d+\beta)+\rho\Lambda]^2F^3\\&\quad-2\lambda(\alpha+d)[\delta(d+\beta)+\rho\Lambda](\delta d+\rho\Lambda)MF^2\\&\quad-\lambda(\alpha+d)(\delta d+\rho\Lambda)^2M^2F.\end{aligned} \tag{5.6}$$

The third term becomes

$$\begin{aligned}&-\delta\sigma^2(\alpha+d)[(d+\beta)F+dM]^2\\&=-\delta\sigma^2(\alpha+d)(d+\beta)^2F^2\\&\quad-2d\delta\sigma^2(\alpha+d)(d+\beta)MF\\&\quad-\delta\sigma^2(\alpha+d)d^2M^2.\end{aligned} \tag{5.7}$$

We group the terms from expressions (5.5) - (5.7) to find the coefficients of our cubic equations in F. The coefficient of F^3 is

$$-a_1 := -\lambda(\alpha+d)[\delta(d+\beta)+\rho\Lambda]^2 < 0.$$

The coefficient of F^2 is

$$\begin{aligned}a_2^* :=& -2\lambda(\alpha+d)[\delta(d+\beta)+\rho\Lambda](\delta d+\rho\Lambda)M\\&-\delta(\alpha+d)(d+\beta)^2\sigma^2\\&+\lambda\gamma\Lambda\sigma\beta[\delta(d+\beta)+\rho\Lambda],\end{aligned}$$

and the coefficient of F is

$$\begin{aligned} a_3^* :=& \lambda\gamma\sigma\Lambda\beta(\delta d + \rho\Lambda)M \\ & - 2\delta(\alpha + d)(d + \beta)dM\sigma^2 \\ & - \lambda(\alpha + d)(\delta d + \rho\Lambda)^2 M^2. \end{aligned}$$

The constant term of the equation is

$$-a_4 := -\delta(\alpha + d)\sigma^2 d^2 M^2 < 0.$$

The equation for F is then of the form

$$-a_1 F^3 + a_2^* F^2 + a_3^* F - a_4 = 0 \tag{5.8}$$

where the signs of a_2^* and a_3^* are unknown. The signs of these terms are crucial in determining the number of positive real roots of equation (5.8), and by extension the number of endemic equilibria of model (5.1). The following subsections define the threshold quantities $\mathcal{R}_1^*$ and $\mathcal{R}_2^*$, and investigate under which conditions positive roots of equation (5.8) exist.

5.4.2 The Thresholds $\mathcal{R}_1^*$ and $\mathcal{R}_2^*$

As previously mentioned, the signs of terms a_2^* and a_3^* are two factors that determine whether endemic equilibria exist for model (5.1). As a result, these terms are used to define two threshold quantities, namely $\mathcal{R}_1^*$ and $\mathcal{R}_2^*$. Using the definition of a_2^* we see that

$$a_2^* > 0 \iff \frac{\lambda\gamma\Lambda\sigma\beta(\delta(d+\beta)+\rho\Lambda)}{(\alpha+d)[2\lambda(\delta(d+\beta)+\rho\Lambda)(\delta d+\rho\Lambda)M+\delta(d+\beta)\sigma^2]} > 1.$$

As a result we define $\mathcal{R}_1^*$ as

$$\mathcal{R}_1^* := \frac{\lambda\gamma\Lambda\sigma\beta(\delta(d+\beta)+\rho\Lambda)}{(\alpha+d)[2\lambda(\delta(d+\beta)+\rho\Lambda)(\delta d+\rho\Lambda)M+\delta(d+\beta)\sigma^2]}. \tag{5.9}$$

Then,

$$\begin{aligned} a_2^* > 0 &\iff \mathcal{R}_1^* > 1, \\ a_2^* < 0 &\iff \mathcal{R}_1^* < 1, \\ a_2^* = 0 &\iff \mathcal{R}_1^* = 1. \end{aligned}$$

Following this, we can write a_2^* as

$$a_2^* = sgn(\mathcal{R}_1^* - 1)a_2 \quad \text{where } a_2 \geq 0.$$

The term a_3^* is used in the same manner to define $\mathcal{R}_2^*$. Using the definition of this term we see that

$$a_3^* > 0 \iff \frac{\lambda\gamma\sigma\Lambda\beta(\delta d+\rho\Lambda)}{(\alpha+d)[2\delta(d+\beta)d\sigma^2+\lambda(\delta d+\rho\Lambda)^2M]} > 1.$$

Consequently we define $\mathcal{R}_2^*$ as

$$\mathcal{R}_2^* := \frac{\lambda\gamma\sigma\Lambda\beta(\delta d+\rho\Lambda)}{(\alpha+d)[2\delta(d+\beta)d\sigma^2+\lambda(\delta d+\rho\Lambda)^2M]} \tag{5.10}$$

Then

$$\begin{aligned} a_3^* > 0 &\iff \mathcal{R}_2^* > 1 \\ a_3^* < 0 &\iff \mathcal{R}_2^* < 1 \\ a_3^* = 0 &\iff \mathcal{R}_2^* = 1 \end{aligned}$$

This allows us to write

$$a_3^* = sgn(\mathcal{R}_2^* - 1)a_3, \quad \text{where } a_3 \geq 0.$$

The threshold quantities $\mathcal{R}_1^*$ and $\mathcal{R}_2^*$ are used in the next sections to determine conditions under which the model admits interior equilibria.

5.4.3 The Influence of $\mathcal{R}_1^*$ and $\mathcal{R}_2^*$ on the Number of Real Roots

Following the notation of the last subsection we rewrite equation (5.8) as

$$-a_1F^3 + sgn(\mathcal{R}_1^* - 1)a_2F^2 + sgn(\mathcal{R}_2^* - 1)a_3F - a_4 = 0 \tag{5.11}$$

Since the values of $sgn(\mathcal{R}_1^* - 1)$ and $sgn(\mathcal{R}_2^* - 1)$, influence the number of real roots equation (5.11) has, we consider all permutations of the possibilities. We first investigate the cases in which a_2 and a_3 are non-zero, followed by the cases that arise when $a_2 = 0$ or $a_3 = 0$. Theorem 2.13 is used to eliminate any cases that do not lead to positive real roots of equation (5.11), and thus do not yield endemic equilibria of model (5.1).

Case I: $sgn(\mathcal{R}_1^* - 1) > 0, sgn(\mathcal{R}_2^* - 1) > 0 \iff \mathcal{R}_1^* > 1, \mathcal{R}_2^* > 1$

There are 2 sign changes, and therefore either 2 or 0 positive real roots. There is 1 sign change when $-F$ is substituted, hence there is exactly 1 negative real root. Consequently there are possibly 2 positive real valued roots of equation (5.11).

Case II: $sgn(\mathcal{R}_1^* - 1) > 0, sgn(\mathcal{R}_2^* - 1) < 0 \iff \mathcal{R}_1^* > 1, \mathcal{R}_2^* < 1$

There are 2 sign changes, and therefore either 2 or 0 positive real roots. There is 1 sign change when $-F$ is substituted, hence there is exactly 1 negative real root. Consequently there are possibly 2 positive real valued roots of equation (5.11).

Case III: $sgn(\mathcal{R}_1^* - 1) < 0, sgn(\mathcal{R}_2^* - 1) > 0 \iff \mathcal{R}_1^* < 1, \mathcal{R}_2^* > 1$

There are 2 sign changes, and therefore either 2 or 0 positive real roots. There is 1 sign change when $-F$ is substituted, consequently there is exactly 1 negative real root. Accordingly there are possibly 2 positive real valued roots of equation (5.11).

Case IV: $sgn(\mathcal{R}_1^* - 1) < 0, sgn(\mathcal{R}_2^* - 1) < 0 \iff \mathcal{R}_1^* < 1, \mathcal{R}_2^* < 1$

In this case there are no sign changes, and hence no positive real roots. Accordingly, in this case there are no endemic equilibria for model (5.1), and we neglect this case for the remainder of the subsection.

Case V: $a_2 = 0$ and $a_3 > 0$

There are 2 sign changes, implying that there are either 0 or 2 positive real roots of equation (5.11).

Case VI: $a_2 = 0$ and $a_3 < 0$

There are no sign changes, indicating that under these conditions equation (5.11) has no positive real roots, and we neglect this case for the remainder of the subsection.

Case VII: $a_2 > 0$ and $a_3 = 0$

There two sign changes, implying that there are either 0 or 2 positive real roots for equation (5.11).

Case VIII: $a_2 < 0$ and $a_3 = 0$

There are no sign changes, indicating that under these conditions equation (5.11) has no positive real roots. We therefore neglect this case for the remainder of the subsection.

Case IX: $a_2 = 0$ and $a_3 = 0$

In this case we obtain the explicit expression for the real valued root F^*:

$$F^* = -\left(\frac{a_4}{a_1}\right)^{\frac{1}{3}} < 0$$

Hence there are no positive real valued solutions of the equation, and therefore no endemic equilibria of model (5.1). We therefore neglect this case for the remainder of the subsection.

We investigate cases I-III, V and VII more thoroughly in the next section. To do this we use proposition 2.1 to construct the Sturm chain for equation (5.11), from which we can determine exactly how many strictly positive roots exist.

5.4.4 Conditions for Existence of Positive Roots

We construct the Sturm chain for equation 5.11 using proposition 2.1. Let equation 5.11 be denoted by $p(F)$. Then the Sturm chain of $p(F)$ is the following sequence of functions:

- $p_0(F) = p(F)$
- $p_1(F) = p'(F)$
- $p_2(F) = -rem(p_0(F), p_1(F))$
- $p_3(F) = -rem(p_1(F), p_2(F))$

Thus, for cases I-III the Sturm chain is

$$\begin{aligned}
p_0(F) &= -a_1F^3 + sgn(\mathcal{R}_1^* - 1)a_2F^2 + sgn(\mathcal{R}_2^* - 1)a_3F - a_4 \\
p_1(F) &= -3a_1F^2 + 2sgn(\mathcal{R}_1^* - 1)a_2F + sgn(\mathcal{R}_2^* - 1)a_3, \\
p_2(F) &= -\frac{2}{3}b_1F - b_2 \\
p_3(F) &= -sgn(\mathcal{R}_2^* - 1)a_3 - \frac{3sgn(\mathcal{R}_1^* - 1)a_2b_2}{b_1} + \frac{27a_1b_2^2}{4b_1^2}
\end{aligned}$$

Here

$$b_1 := \frac{3sgn(\mathcal{R}_2^* - 1)a_1a_3 + a_2^2}{3a_1}$$

and

$$b_2 := \frac{sgn(\mathcal{R}_1^* - 1)sgn(\mathcal{R}_2^* - 1)a_2a_3 - 9a_1a_4}{9a_1}.$$

The signs of these terms are unknown, but depend on $sgn(\mathcal{R}_1^* - 1)$ and $sgn(\mathcal{R}_2^* - 1)$. The Sturm chains for cases V and VII are constructed on pages 47 and 48 respectively.

Since we are only interested in roots of equation (5.11) that yield endemic equilibria, we focus on strictly positive roots, and can use Sturm's theorem to determine the exact number of these roots for each of the cases. In doing so we determine exactly how many endemic equilibria exist for model (5.1). The computations are messy, and the summarized results can be found in Tables(5.1) and (5.2) on page 49.

Case I: $sgn(\mathcal{R}_1^* - 1) > 0, sgn(\mathcal{R}_2^* - 1) > 0 \iff \mathcal{R}_1^* > 1, \mathcal{R}_2^* > 1$

In this case equation (5.11) becomes

$$-a_1 F^3 + a_2 F^2 + a_3^* F - a_4 = 0$$

Our constants b_1 and b_2 are then:

$$b_1 = \frac{a_2^2 + 3a_1 a_3}{3a_1} > 0$$
$$b_2 = \frac{a_2 a_3 - 9a_1 a_4}{9a_1}$$

Subcase A: $b_2 > 0 \iff a_2 a_3 - 9a_1 a_4 > 0$

The signs of the Sturm sequence are:

$$p_0(0) = -a_4 < 0 \qquad p_0(\infty) < 0$$
$$p_1(0) = a_3 > 0 \qquad p_1(\infty) < 0$$
$$p_2(0) = -b_2 < 0 \qquad p_2(\infty) < 0$$

Let $x = \frac{b_2}{b_1} > 0$, and consider p_3:

$$p_3 = -a_3 - 3a_2 x + \frac{27a_1}{4} x^2$$

Hence we have

$$p_3 > 0 \iff \frac{b_2}{b_1} > \frac{2a_2 + 2\sqrt{b_1}}{9a_1}$$
$$p_3 < 0 \iff 0 < \frac{b_2}{b_1} < \frac{2a_2 + 2\sqrt{b_1}}{9a_1}$$

If $p_3 > 0$ the sign changes of the Sturm chains are:

$$p_i(0) : - \quad + \quad - \quad +$$
$$p_i(\infty) : - \quad - \quad - \quad +$$
$$\therefore \ \sigma(0) - \sigma(\infty) = 2$$

Conclusion: equation (5.11) has 2 positive real valued roots under these conditions.

However, if $p_3 < 0$ the sign changes of the Sturm chains are:

$$\begin{aligned} p_i(0) &: - \quad + \quad - \quad - \\ p_i(\infty) &: - \quad - \quad - \quad - \\ \therefore \;\; \sigma(0) - \sigma(\infty) &= 2 \end{aligned}$$

Conclusion: equation (5.11) has exactly 2 positive real valued roots under these conditions.

Subcase B: $b_2 < 0 \iff a_2a_3 - 9a_1a_4 < 0$

The signs of the Sturm sequence are then,

$$\begin{aligned} p_0(0) &= -a_4 < 0 & \qquad p_0(\infty) &< 0 \\ p_1(0) &= a_3 > 0 & \qquad p_1(\infty) &< 0 \\ p_2(0) &= -b_2 > 0 & \qquad p_2(\infty) &< 0 \end{aligned}$$

In this case $x = \frac{b_2}{b_1} < 0$, with $p_3 = -a_3 - 3a_2x + \frac{27a_1}{4}x^2$. Then,

$$\begin{aligned} p_3 > 0 &\iff \frac{b_2}{b_1} < \frac{2a_2 - 2\sqrt{b_1}}{9a_1} < 0 \\ p_3 < 0 &\iff \frac{2a_2 - 2\sqrt{b_1}}{9a_1} < \frac{b_2}{b_1} < 0 \end{aligned}$$

When $p_3 > 0$ the sign changes of the Sturm chains are:

$$\begin{aligned} p_i(0) &: - \quad + \quad + \quad + \\ p_i(\infty) &: - \quad - \quad - \quad + \\ \therefore \;\; \sigma(0) - \sigma(\infty) &= 0 \end{aligned}$$

Conclusion: equation (5.11) has no positive real valued roots under these conditions.

However, if $p_3 < 0$ the sign changes of the Sturm chains are:

$$\begin{aligned} p_i(0) &: - \quad + \quad + \quad - \\ p_i(\infty) &: - \quad - \quad - \quad - \\ \therefore \;\; \sigma(0) - \sigma(\infty) &= 2 \end{aligned}$$

Conclusion: equation (5.11) has exactly 2 positive real valued roots in this subcase.

Case II: $sgn(\mathcal{R}_1^* - 1) > 0, sgn(\mathcal{R}_2^* - 1) < 0 \iff \mathcal{R}_1^* > 1, \mathcal{R}_2^* < 1$

In this case equation (5.11) becomes

$$-a_1F^3 + a_2F^2 - a_3F - a_4 = 0$$

Our constants b_1 and b_2 are then:

$$b_1 = \frac{a_2^2 + 3a_1a_3}{3a_1} > 0$$
$$b_2 = -\frac{a_2a_3 + 9a_1a_4}{3a_1} < 0$$

The signs of the Sturm sequence are then,

$$p_0(0) = -a_4 < 0 \qquad p_0(\infty) < 0$$
$$p_1(0) = -a_3 < 0 \qquad p_1(\infty) < 0$$
$$p_2(0) = -b_2 > 0 \qquad p_2(\infty) < 0$$

The fourth term in our Sturm sequence becomes:

$$p_3 = a_3 - 3a_2\frac{b_2}{b_1} + \frac{27a_1}{4}\frac{b_2^2}{b_1^2}$$

Then, since $x = \frac{b_2}{b_1} < 0$ we have $p_3 > 0$. The signs of the Sturm chains are:

$$p_i(0) : - \quad - \quad + \quad +$$
$$p_i(\infty) : - \quad - \quad - \quad +$$
$$\therefore \ \sigma(0) - \sigma(\infty) = 0$$

Hence, there are no positive real roots of equation (5.11) in this case.

Case III: $sgn(\mathcal{R}_1^* - 1) < 0, sgn(\mathcal{R}_2^* - 1) > 0 \iff \mathcal{R}_1^* < 1, \mathcal{R}_2^* > 1$
In this case equation (5.11) becomes

$$-a_1F^3 - a_2F^2 + a_3F - a_4 = 0$$

Our constants b_1 and b_2 are then:

$$b_1 = \frac{a_2^2 - 3a_1a_3}{3a_1}$$
$$b_2 = -\frac{a_2a_3 + 9a_1a_4}{9a_1} < 0$$

Subcase A: $b_1 > 0$ The signs of the Sturm sequence are:

$$p_0(0) = -a_4 < 0 \qquad p_0(\infty) < 0$$
$$p_1(0) = a_3 > 0 \qquad p_1(\infty) < 0$$
$$p_2(0) = -b_2 > 0 \qquad p_2(\infty) < 0$$

Then $x = \frac{b_2}{b_1} < 0$, and p_3 becomes:

$$p_3 = -a_3 + 3a_2x + \frac{27a_1}{4}x^2$$

where,

$$p_3 > 0 \iff \frac{b_2}{b_1} < \frac{-2a_2 - 2\sqrt{a_2^2 + 3a_1a_3}}{9a_1} < 0$$
$$p_3 < 0 \iff \frac{-2a_2 - 2\sqrt{a_2^2 + 3a_1a_3}}{9a_1} < \frac{b_2}{b_1} < 0$$

If $p_3 > 0$ the sign changes of the Sturm chains are:

$$p_i(0) : - \quad + \quad + \quad +$$
$$p_i(\infty) : - \quad - \quad - \quad +$$
$$\therefore \ \sigma(0) - \sigma(\infty) = 0$$

Conclusion: equation (5.11) has no positive real valued roots under these conditions.

If $p_3 < 0$ the sign changes of the Sturm chains are:

$$p_i(0) : - \quad + \quad + \quad -$$
$$p_i(\infty) : - \quad - \quad - \quad -$$
$$\therefore \ \sigma(0) - \sigma(\infty) = 2$$

Conclusion: equation (5.11) has two positive real valued roots under these conditions.

Subcase B: $b_1 < 0$ The signs of the Sturm sequence are:

$$\begin{aligned} p_0(0) &= -a_4 < 0 & \qquad p_0(\infty) &< 0 \\ p_1(0) &= a_3 > 0 & \qquad p_1(\infty) &< 0 \\ p_2(0) &= -b_2 > 0 & \qquad p_2(\infty) &> 0 \end{aligned}$$

Then $x = \frac{b_2}{b_1} > 0$, and p_3 becomes:

$$p_3 = -a_3 + 3a_2 x + \frac{27a_1}{4}x^2$$

where,

$$\begin{aligned} p_3 > 0 &\iff \frac{b_2}{b_1} > \frac{-2a_2 + 2\sqrt{a_2^2 + 3a_1a_3}}{9a_1} > 0 \\ p_3 < 0 &\iff 0 < \frac{b_2}{b_1} < \frac{-2a_2 + 2\sqrt{a_2^2 + 3a_1a_3}}{9a_1} \end{aligned}$$

If $p_3 > 0$ the sign changes of the Sturm chains are:

$$\begin{aligned} p_i(0) &: - \quad + \quad + \quad + \\ p_i(\infty) &: - \quad - \quad + \quad + \\ \therefore \;\; \sigma(0) &- \sigma(\infty) = 0 \end{aligned}$$

Conclusion: equation (5.11) has no positive real valued roots under these conditions.

If $p_3 < 0$ the sign changes of the Sturm chains are:

$$\begin{aligned} p_i(0) &: - \quad + \quad + \quad - \\ p_i(\infty) &: - \quad - \quad + \quad - \\ \therefore \;\; \sigma(0) &- \sigma(\infty) = 0 \end{aligned}$$

Conclusion: equation (5.11) has no positive real valued roots under these conditions.

Case V: $a_2 = 0$ and $a_3 > 0$ In this case equation (5.11) becomes

$$-a_1F^3 + a_3F - a_4 = 0.$$

The Sturm chain is then

$$\begin{aligned} p_0(F) &= -a_1F^*3 + a_3F - a_4 \\ p_1(F) &= -3a_1F^2 + a_3 \\ p_2(F) &= -\frac{2}{3}a_3F + a_4 \\ p_3(F) &= \frac{27a_1a_4^2}{4a_3^2} - a_3 \end{aligned}$$

Two sub-cases arise when we evaluate $p_i(0)$ and $p_i(\infty)$:

$$\begin{aligned} p_0(0) &= -a_4 < 0 & \qquad p_0(\infty) &< 0 \\ p_1(0) &= a_3 > 0 & \qquad p_1(\infty) &< 0 \\ p_2(0) &= a_4 > 0 & \qquad p_2(\infty) &< 0 \end{aligned}$$

Subcase A: $p_{3,V}(F) < 0$
The sign changes are:

$$\begin{aligned} p_i(0) &: - \quad + \quad + \quad - \\ p_i(\infty) &: - \quad - \quad - \quad - \\ \therefore \quad \sigma(0) &- \sigma(\infty) = 2 \end{aligned}$$

Consequently, there are exactly two strictly positive roots for equation (5.11).
Subcase B: $p_3(F) > 0$

$$\begin{aligned} p_i(0) &: - \quad + \quad + \quad + \\ p_i(\infty) &: - \quad - \quad - \quad + \\ \therefore \quad \sigma(0) &- \sigma(\infty) = 0 \end{aligned}$$

In this case there are no positive roots for equation (5.11)

Case VII: $a_2 > 0$ and $a_3 = 0$

In this case the Sturm chain is:

$$\begin{aligned} p_0(F) &= -a_1F^3 + a_2F^2 - a_4 \\ p_1(F) &= -3a_1F^2 + 2a_2F \\ p_2(F) &= -\frac{2a_2^2}{9a_1}F + a_4 \\ p_{3,VII}(F) &= -\frac{9a_1}{a_2} + \frac{243a_4^2a_1^3}{4a_2^4} \end{aligned}$$

Two sub-cases arise after evaluation of $p_i(0)$ and $p_i(\infty)$:

$$\begin{aligned} p_0(0) &= -a_4 < 0 & \qquad p_0(\infty) &< 0 \\ p_1(0) &= 0 & \qquad p_1(\infty) &< 0 \\ p_2(0) &= a_4 > 0 & \qquad p_2(\infty) &< 0 \end{aligned}$$

Subcase A: $p_{3,VII}(F) < 0$

$$\begin{aligned} p_i(0) &: - \quad 0 \quad + \quad - \\ p_i(\infty) &: - \quad - \quad - \quad - \\ \therefore \ \sigma(0) &- \sigma(\infty) = 2 \end{aligned}$$

Consequently, there are exactly two strictly positive roots for equation (5.11).

Subcase B: $p_{3,VII}(F) > 0$

$$\begin{aligned} p_i(0) &: - \quad 0 \quad + \quad + \\ p_i(\infty) &: - \quad - \quad - \quad + \\ \therefore \ \sigma(0) &- \sigma(\infty) = 0 \end{aligned}$$

From which we conclude there are no strictly positive roots for equation (5.11).

The preceding calculations are summarised in tables (5.1) and (5.2). We see that under certain conditions model (5.1) admits two distinct endemic equilibria. We denote the equilibrium with the lower F^* value by $\mathcal{EE}_1$, and the other by $\mathcal{EE}_2$.

	$b_1 < 0$	$b_1 > 0$
$b_2 < 0$	**Case I** 0 positive roots	**Case I & III:** $p_3 < 0$: 2 positive roots $p_3 > 0$: 0 positive roots **Case II:** $p_3 > 0$: 0 positive roots
$b_2 > 0$	N.A.	**Case I:** $p_3 < 0$: 2 positive roots $p_3 > 0$: 0 positive roots

Table 5.1: The number of positive real roots of equation (5.11) in cases I - III

	$a_2 < 0$	$a_2 = 0$	$a_2 > 0$
$a_3 < 0$	No positive real roots	No positive real roots	No positive real roots
$a_3 = 0$	No positive real roots	No positive real roots	$p_{3,k} < 0$: 2 positive roots $p_{3,k} > 0$: 0 positive roots
$a_3 > 0$	No positive real roots	$p_{3,k} < 0$: 2 positive roots $p_{3,k} > 0$: 0 positive roots	No positive real roots

Table 5.2: The number of positive real roots of equation (5.11) in cases $k \in \{\text{V, VII}\}$.

5.5 The Stability Analysis of Equilibria

We determine the stability properties of the equilibria found in the previous subsection, which is usually done by substituting the equilibria into the Jacobian matrix of the model and determining the eigenvalues. However, since we do not have explicit expressions for our endemic equilibria we analyse the characteristic equation of the Jacobian, keeping the differences between these equilibria in mind.

Recall the Jacobian matrix of model (5.1)

$$J_f = \begin{pmatrix} \lambda\gamma I - 2\lambda A - \sigma & \rho S & \rho F & \lambda\gamma A \\ \sigma & -\delta - \rho S & -\rho F & 0 \\ 0 & -\frac{\beta MS}{(M+F)^2} & -d - \frac{\beta F}{M+F} & 0 \\ 0 & \frac{\beta MS}{(M+F)^2} & \frac{\beta F}{M+F} & -(\alpha + d) \end{pmatrix}$$

Theorem 5.3. The pathogen free equilibrium, **PFE**, is unconditionally locally asymptotically stable.

Proof. We determine the characteristic equation $q(\xi)$ by calculating $\det(J_f(\mathbf{PFE}) - \xi I_4) = 0$. We obtain

$$q(\xi) = (\alpha + d + \xi)(d + \xi)\left(\xi^2 + \left(\sigma + \delta + \frac{\rho\Lambda}{d}\right)\xi + \sigma\delta\right),$$

Since all three coefficients in the quadratic factor are strictly positive, and applying Corollary (2.1), $q(\xi)$ has two complex conjugate roots with negative real parts. This, along with the fact that the remaining eigenvalues are negative, leads to the conclusion that the **PFE** is locally asymptotically stable by Theorem 2.10. □

In all the numerical simulations of model (5.1) we observe two qualitatively different cases and the transition (bifurcation) from one to the other. The first case is when the model has two positive equilibria. As seen in Figure 5.2, one is stable and attracting while the other is unstable (saddle point). Table 5.3 contains the parameter values used for Figure 5.2. The solutions that are initiated below $\mathcal{EE}_1$ converge to the **PFE**. Solutions ϕ_1 and ϕ_2 are initiated at $\mathcal{EE}_1$ with different initial densities for the susceptible compartment, namely $S_0 = 2$ and $S_0 = 20$ respectively. These values are below and above the equilibrium value, and we observe that ϕ_1 converges to the **PFE** while ϕ_2 increases and will eventually converge to $\mathcal{EE}_2$. The unstable equilibrium is typically very close to the **PFE**, so that the basin of attraction of the **PFE** is small. When there are no positive equilibria we observe that the **PFE** is globally asymptotically stable on $\mathbb{R}^4_+$, see Figure 5.6. We could not obtain a general result for these observed properties of the positive equilibria, or alternatively the global asymptotic stability of the **PFE**. However, we supply sufficient conditions for two practically important properties. We use both LaSalle's Invariance Principle and the theory of monotone systems to derive two different sufficient conditions for the global asymptotic stability of the **PFE** in Sections 5.5.2 and 5.6.1. Further, in Section 5.6.2 we show sufficient conditions for persistence of the pathogen.

Parameter	Value	Parameter	Value
Λ	1.0000	λ	0.5000
γ	0.9000	β	5.0000
σ	0.4000	ρ	0.1000
δ	0.1000	M	100.000
α	0.0450	d	0.1000

Table 5.3: Parameter values used in Figures 5.2 and 5.3.

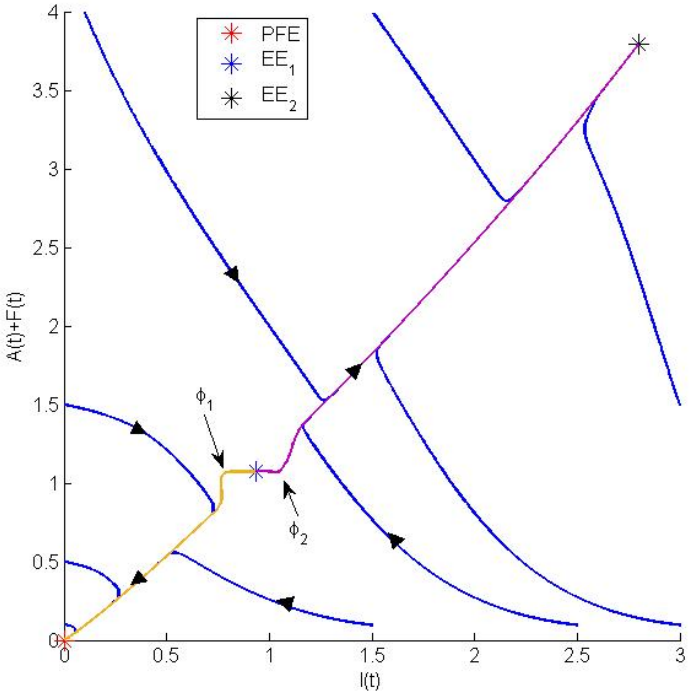

Figure 5.2: If solutions of model 5.1 are initiated 'close' to the **PFE** convergence to this equilibrium occurs. Solutions that are initiated from the 'smaller' endemic equilibrium converge to either the **PFE** or the 'larger' equilibrium, dependent on the initial density of the susceptible compartment.

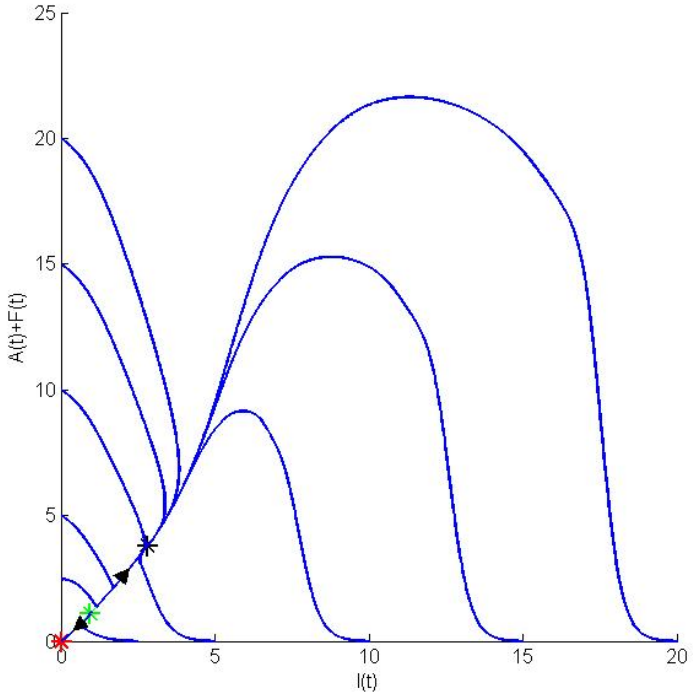

Figure 5.3: Solutions that model the scenario of large free pathogen populations invading wholly susceptible host populations converge to the second equilibrium. The basin of attraction of the **PFE** therefore does not extend far in the direction of the $A(t) + F(t)$ axis. In fact, for the parameter values in Table 5.3 solutions with $F(0) \geq 1.5$ converge to the second endemic equilibrium.

5.5.1 The long term behaviour of $S(t)$ and $I(t)$

Conventional methods of stability analysis might not yield much information about the behaviour of solutions near the endemic equilibria due to a lack of explicit expressions. To formulate some idea of the long term behaviour of solutions we turn our attention to the phase diagram of the host population in model (5.1). In particular we calculate:

$$\begin{aligned}\frac{dI}{dS} &= \frac{\frac{dI}{dt}}{\frac{dS}{dt}} \\ &= \frac{\frac{\beta FS}{M+F} - (\alpha+d)I}{\Lambda - dS - \frac{\beta FS}{M+F}} \\ &= \frac{\frac{\beta FS}{M+F} - (\alpha+d)(N-S)}{\Lambda - dS - \frac{\beta FS}{M+F}} \\ &= \frac{\frac{\beta FS}{M+F} - d(N-S)}{\Lambda - dS - \frac{\beta FS}{M+F}} - \frac{\alpha(N-S)}{\Lambda - dS - \frac{\beta FS}{M+F}}\end{aligned}$$

Now, assuming $S(0) + I(0) = N(0) = \frac{\Lambda}{d}$, and since $\alpha \geq 0$, we have

$$\begin{aligned}\frac{dI}{dS} &= \frac{\frac{\beta FS}{M+F} - d(N-S)}{-(\frac{\beta FS}{M+F} + dS - \Lambda)} - \frac{\alpha(N-S)}{\Lambda - dS - \frac{\beta FS}{M+F}} \\ &\leq -1 - \frac{\alpha I}{\Lambda - dS - \frac{\beta FS}{M+F}}.\end{aligned}$$

This explains the local near linearity shown in Figures 5.4 and 5.5. Figure 5.4 shows the trajectories for the host population of model (5.1) with $\alpha = 0.001$, while Figure 5.5 shows the trajectories for $\alpha = 0$. Notice that solutions stay in the neighbourhood of the initial condition for some time before increasing rapidly towards an epidemic equilibrium. The value of α has some influence on both the slope of the trajectories as well as the endemic equilibrium these trajectories converge to.

Theorem 5.4. The solutions for the host population are eventually contained in two intervals, in particular

$$\lim_{t\to\infty} S(t) \in \left[\frac{\Lambda}{d+\beta}, \frac{\Lambda}{d}\right], \text{ and } \lim_{t\to\infty} I(t) \in \left[0, \frac{\beta\Lambda}{d(d+\beta)}\right]$$

Proof. Clearly

$$\Lambda - (d+\beta)S \leq \frac{dS}{dt} \leq \Lambda - dS.$$

After application of Theorem 2.5 this becomes

$$\frac{\Lambda}{d+\beta} + \left(S_0 - \frac{\Lambda}{d+\beta}\right)e^{-(d+\beta)t} \leq S(t) \leq \frac{\Lambda}{d} + \left(S_0 - \frac{\Lambda}{d}\right)e^{-dt}.$$

Taking the limit as $t \to \infty$ we obtain

$$\frac{\Lambda}{d+\beta} \leq \lim_{t\to\infty} S(t) \leq \frac{\Lambda}{d}.$$

That is,

$$\lim_{t\to\infty} S(t) \in \left[\frac{\Lambda}{d+\beta}, \frac{\Lambda}{d}\right].$$

Using this, and the fact that S and I have a negative relationship we find that

$$\lim_{t\to\infty} I(t) \in \left[0, \frac{\beta\Lambda}{d(d+\beta)}\right].$$

□

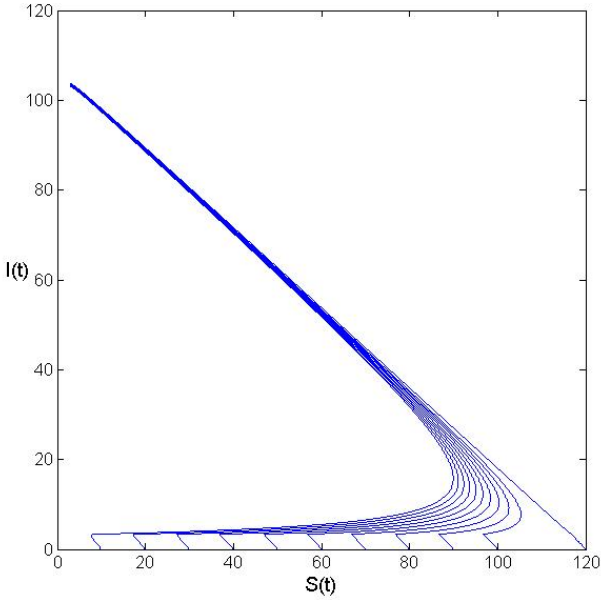

Figure 5.4: The phase diagram of the host population, with $\alpha \neq 0$

5.5.2 Sufficient conditions for global asymptotic stability of PFE

We prove the global asymptotic stability of **PFE** using LaSalle's Invariance Principle (Theorem 2.12).

Theorem 5.5. The **PFE** of model (5.1) is globally asymptotically stable on $\mathbb{R}^+_4\backslash\{\mathbf{0}\}$ if

$$\frac{\gamma(\gamma\lambda\Lambda + \delta d)^2}{4(\alpha+d)d^2} \leq 1 \text{ and } \frac{\beta\Lambda}{\delta M d} \leq 1. \tag{5.12}$$

Proof. We apply Theorem 2.12 to system (5.1) using the function

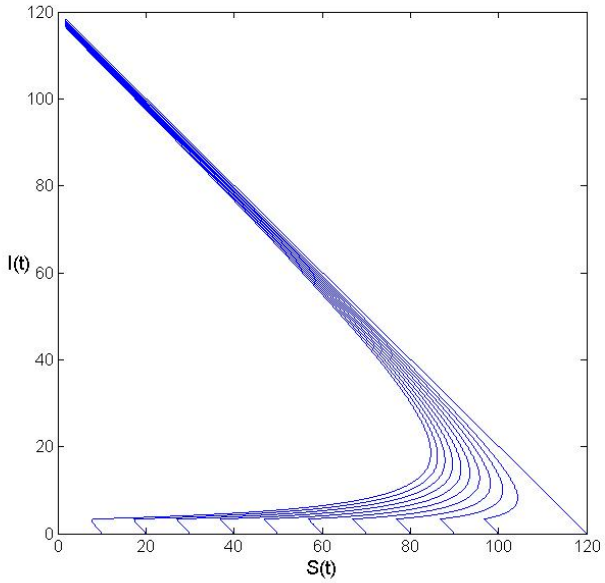

Figure 5.5: The phase diagram of the host population, with $\alpha = 0$

$V(X) = A + F + I$ with $X = (A, F, S, I)$. We have

$$\begin{aligned}
\dot{V}(X) &= \dot{A} + \dot{F} + \dot{I} \\
&= \lambda A(\gamma I - A) - \sigma A + \rho FS - \delta F + \sigma A - \rho FS + \frac{\beta F}{M+F} S - (\alpha + d)I \\
&= \lambda A(\gamma I - A) - \delta F + \frac{\beta F}{M+F} S - (\alpha + d)I \\
&= (\lambda\gamma A - (\alpha + d))\, I - \lambda A^2 + \left(\frac{\beta S}{M+F} - \delta \right) F \\
&\leq \left(\lambda\gamma \left(\frac{\lambda(\gamma\lambda\Lambda + \delta d)^2}{4d^2\lambda^2} \right) - (\alpha + d) \right) I - \lambda A^2 + \left(\frac{\beta S}{M} - \delta \right) F.
\end{aligned}$$

From Theorem (5.1) we have

$$A \leq \frac{\lambda(\gamma\lambda\Lambda + \delta d)^2}{4d^2\lambda^2} \text{ and } S \leq \frac{\Lambda}{d}$$

thus,

$$\dot{V}(X) \leq \left(\lambda\gamma \left(\frac{\lambda(\gamma\lambda\Lambda + \delta d)^2}{4d^2\lambda^2} \right) - (\alpha + d) \right) I - \lambda A^2 + \left(\frac{\beta\Lambda}{Md} - \delta \right) F.$$

Then, given

$$\frac{\gamma(\gamma\lambda\Lambda + \delta d)^2}{4(\alpha + d)d^2} \leq 1 \text{ and } \frac{\beta\Lambda}{\delta dM} \leq 1$$

we have

$$\dot{V}(X) \leq 0 \text{ for all } X \in \mathbb{R}^4_+.$$

The set $E = \{X \in \mathbb{R}^4_+ : \dot{V}(X) = 0\}$ is the nonnegative part of the $S-$axis. Therefore, the set M in Theorem 2.12 is $M = \{\mathbf{0}, \mathbf{PFE}\}$. Since $\mathbf{0}$ is unstable and repelling, every solution initiated at a point other than zero converges to the **PFE**. □

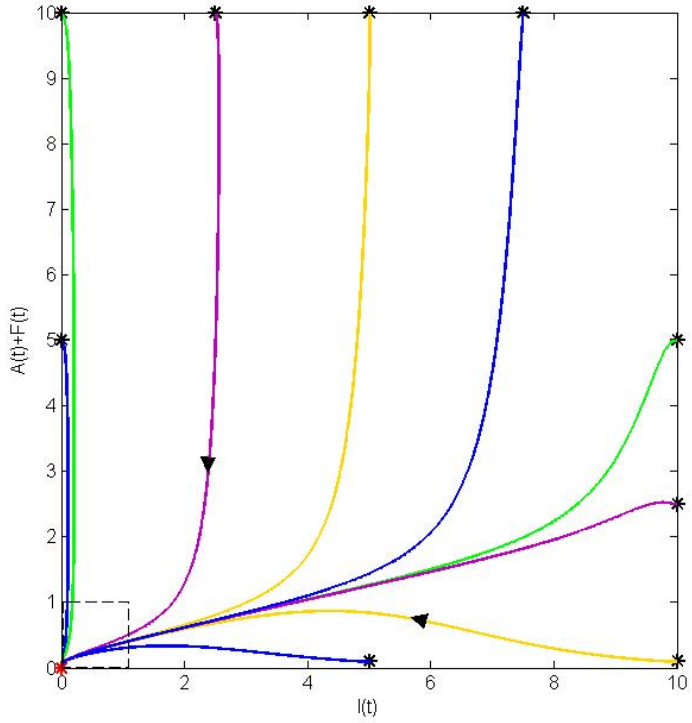

Figure 5.6: Solutions of model 5.1 clearly converge to the **PFE**, when the parameter values satisfy condition 5.12. This is proven theoretically in Theorem 5.5. The parameter values can be found in Table 5.4.

Figure 5.6 shows the global stability of the **PFE**, when the model parameters satisfy the conditions of Theorem 5.7:

$$\frac{\gamma(\gamma\lambda\Lambda + \delta d)^2}{4(\alpha + d)d^2} = 0.3361 \leq 1 \text{ and } \frac{\beta\Lambda}{\delta d M} = 1.$$

The parameter values can be found in Table 5.4. Initial conditions with varying levels of density for each compartment were chosen, although it is not biologically possible for permutations containing $I_0 \neq 0$ and $A_0 = 0$. For this reason no solutions originate from the I axis. Note the sharp decline in pathogen density in cases involving a high pathogen to infective density ratio.

Parameter	Value	Parameter	Value
Λ	1.0000	λ	0.4000
γ	0.2000	β	1.0000
σ	0.0100	ρ	0.4000
δ	0.1000	M	100.000
α	0.0205	d	0.1000

Table 5.4: Parameter values used in Figures 5.6 and 5.7

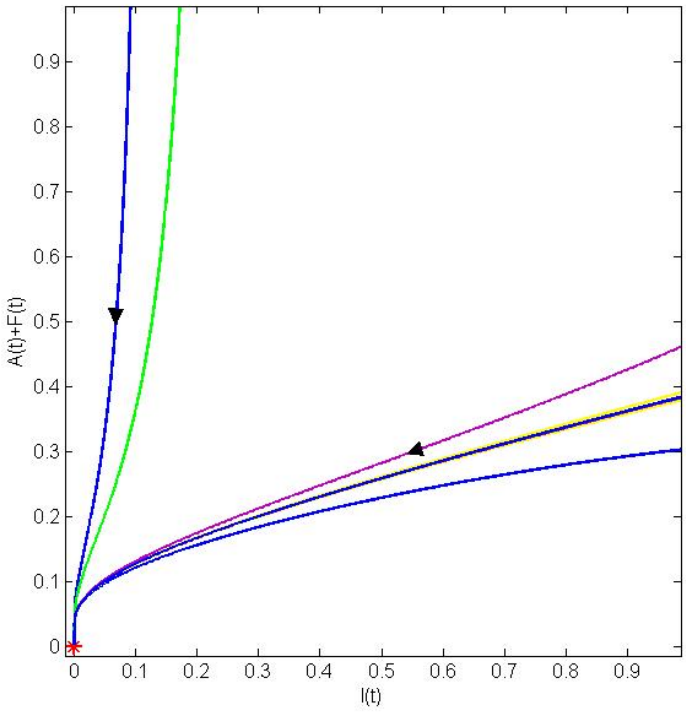

Figure 5.7: Enlargement of the $[0,1]\times[0,1]$ block of Figure 5.6, with the convergence to the **PFE** clearly displayed.

5.6 Analysis via Monotone Systems

In the previous section we used LaSalle's Invariance Principle to prove the stability properties of **PFE**; as an alternative we discuss analysis via monotone systems. We construct and analyse two systems, one which approximates the host pathogen model from above and the other which approximates it from below. The so-called 'upper' system is constructed in such a way that this solution is always above the solution of model (5.1), while the 'lower' system is constructed so that the solution of the host pathogen model is always above the solution to this system. Analysis of the 'lower' system also yields conditions for the persistence of the host and pathogen populations.

5.6.1 Upper Approximation for the Host-Pathogen Model

The system that approximates model (5.1) from above is

$$\frac{dw}{dt} = g(y) = g(A,F,I) = \begin{pmatrix} \lambda A(\gamma I - A) - \sigma A + \frac{\rho\Lambda}{d}F \\ -\delta F + \sigma A - \frac{\rho\Lambda}{\beta+d}F \\ \frac{\beta F}{M+F}\frac{\Lambda}{d} - (\alpha+d)I \end{pmatrix} \tag{5.13}$$

with the following equilibrium expressions for A^* and I^*:

$$I^* = \frac{\Lambda}{d(\alpha+d)}\frac{\beta F^*}{M+F^*}, \text{ and}$$
$$A^* = \frac{F^*}{\sigma}\left(\delta + \frac{\rho\Lambda}{\beta+d}\right).$$

Assuming $F^* \neq 0$, these expressions can be used to find a quadratic equation in F^*:

$$\frac{-\lambda}{\sigma^2}\left(\delta+\frac{\rho\Lambda}{\beta+d}\right)^2 F^{*2}+\left(\frac{-\lambda M}{\sigma^2}\left(\delta+\frac{\rho\Lambda}{\beta+d}\right)^2+\frac{\lambda\gamma\Lambda\beta}{\sigma d(\alpha+d)}\left(\delta+\frac{\rho\Lambda}{\beta+d}\right)-\left(\delta-\frac{\rho\Lambda\beta}{d(\beta+d)}\right)\right)F^* - \left(\delta-\frac{\rho\Lambda\beta}{d(\beta+d)}\right)M=0. \tag{5.14}$$

Equation (5.14) has no positive real roots under the following conditions:

1. Necessary and sufficient condition:

   $$\Delta_1 < 0,$$

   where Δ_1 is the determinant of equation (5.14). In this case equation 5.14 has no real roots.

2. Sufficient conditions, the coefficient of the second term and the constant term are negative:

   $$\frac{\lambda\gamma\Lambda\beta}{\sigma d(\alpha+d)} < \frac{\lambda M}{\sigma^2}\left(\delta+\frac{\rho\Lambda}{\beta+d}\right)+\left(\frac{\delta d(\beta+d)-\rho\beta\Lambda}{\delta d(\beta+d)+\rho d\Lambda}\right), \text{ and}$$
   $$-\left(\delta-\frac{\rho\Lambda\beta}{d(\beta+d)}\right)<0 \tag{5.15}$$

   If the above is true, Theorem 2.13 ensures that no positive real roots exist for equation 5.14

3. However, if $\Delta_1 \geq 0$, and if the F^*-value at which the maximum occurs is non-positive, and the constant term is also non-positive, equation 5.14 will have no positive roots.

Although these conditions ensure no positive equilibrium exists for system (5.13), $\mathbf{0}$ is clearly an equilibrium of the system. The Jacobian of this system is

$$J_g=\begin{pmatrix}\lambda\gamma I-2\lambda A-\sigma & \frac{\rho\Lambda}{d} & \lambda\gamma A\\ \sigma & -\delta-\frac{\rho\Lambda}{\beta+d} & 0\\ 0 & \frac{\beta M\Lambda}{d(M+F)^2} & -(\alpha+d)\end{pmatrix},$$

and,

$$J_g(\mathbf{0})=\begin{pmatrix}-\sigma & \frac{\rho\Lambda}{d} & 0\\ \sigma & -\delta-\frac{\rho\Lambda}{\beta+d} & 0\\ 0 & \frac{\beta\Lambda}{dM} & -(\alpha+d)\end{pmatrix}.$$

Then, all the eigenvalues of $J_g(\mathbf{0})$ have negative real parts if

$$\delta+\frac{\rho\Lambda}{\beta+d}>\frac{\rho\Lambda}{d} \iff \delta>\frac{\rho\beta\Lambda}{d(\beta+d)}.$$

Thus, if $\mathbf{0}$ is the only equilibrium, it is locally asymptotically stable.

Theorem 5.6. Under condition 1,2, or 3 system 5.13 has $\mathbf{0}$ as a globally asymptotically stable equilibrium on $\mathbb{R}^3_+$.

Proof. Consider the third equation of system (5.13).

$$\begin{aligned}\frac{dI}{dt} &= \frac{\beta F}{M+F}\frac{\Lambda}{d} - (\alpha+d)I \\ &\leq \frac{\beta\Lambda}{d} - (\alpha+d)I,\end{aligned}$$

thus $I(t) \leq \max\{I(0), \frac{\beta\Lambda}{d(\alpha+d)}\}$. Let $n \geq 0$ and $I_n \geq n \geq \frac{\beta\Lambda}{d(\alpha+d)}$, then

$$\frac{\beta\Lambda}{d} \leq (\alpha+d)I_n \text{ and } \frac{dI}{dt} < 0. \tag{5.16}$$

Define $F_n = \frac{\sigma}{\delta+\frac{\rho\Lambda}{\beta+d}}A_n$, $A_n = \gamma I_n$ and $\mathbf{b_n} = (A_n, F_n, I_n)\prime$. Then,

$$\begin{aligned}g_1(\mathbf{b_n}) &= \lambda\gamma I_n(\gamma I_n - \gamma I_n) - \sigma\gamma I_m + \frac{\rho\Lambda}{d}\frac{\sigma\gamma}{\delta+\frac{\rho\Lambda}{\beta+d}}I_n \\ &= \gamma I_n\left(-\sigma + \frac{\rho\Lambda}{d}\frac{\sigma}{\delta+\frac{\rho\Lambda}{\beta+d}}\right) \\ &< 0 \qquad \Longleftrightarrow \ \frac{\rho\beta\Lambda}{d(d+\beta)} < \delta \qquad \text{(condition (2))}. \\ g_2(\mathbf{b_n}) &= -\left(\delta + \frac{\rho\Lambda}{d(d+\beta)}\right)\frac{\sigma}{\delta+\frac{\rho\Lambda}{\beta+d}}A_n + \sigma A_n = 0\end{aligned}$$

From (5.16) we have that $g_3(\mathbf{b_n}) < 0$. So, $g(\mathbf{b_n}) < 0$. It follows from Theorem 2.7 that $\mathbf{0}$ is globally asymptotically stable on $[\mathbf{0}, \mathbf{b_n}]$ for any $n > 0$. Hence it is globally asymptotically stable on $\mathbb{R}^3_+$. □

Theorem 5.7. If condition (1),(2), or (3) hold then **PFE** is a globally asymptotic equilibrium of the host-pathogen model (5.1).

Proof. Considering the equation for $\frac{dS}{dt}$, the interval $\frac{\Lambda}{\beta+d} \leq S \leq \frac{\Lambda}{d}$ is a compact attractor. By Theorem 2.12, we need to consider only $S \in \left[\frac{\Lambda}{\beta+d}, \frac{\Lambda}{d}\right]$. Let $(A(t), F(t), S(t), I(t))$ be any solution of 5.1 such that $S \in \left[\frac{\Lambda}{\beta+d}, \frac{\Lambda}{d}\right]$. Denote $x(t) = (A(t), F(t), I(t))$. Then, we can verify that

$$\frac{dx(t)}{dt} \leq g(x(t)).$$

Denote by $\hat{y}(t)$ the solution of 5.13 with $\hat{y}(0) = x(0)$. Then, since g is quasi-monotone, using [[122], Section 12.X] we obtain the inequality

$$\leq x(t) \leq \hat{y}(t), \quad t > 0.$$

Therefore,

$$\lim_{t\to\infty} x(t) = 0.$$

By implication,

$$\lim_{t\to\infty} S(t) = \frac{\Lambda}{d}.$$

□

Parameter	Value	Parameter	Value
Λ	1.0000	λ	0.7000
γ	0.9000	β	1.0000
σ	$0.188\dot{2}$	ρ	0.2000
δ	$0.841\dot{6}$	M	100.00
α	0.0100	d	0.2000

Table 5.5: Parameter values used in Figure 5.8

Theorem 5.7 is illustrated in Figure 5.8 on page 61. In order to maintain biological feasibility initial conditions with $A_0 = 0$ and $I_0 \neq 0$ were not used, instead small values were chosen for A_0. The parameter values can be found in Table 5.5, and we verify that these do indeed satisfy condition (2):

2.1 The coefficient of the second term is negative:

$$\frac{\lambda\gamma\Lambda\beta}{\sigma d(\alpha+d)} - \frac{\lambda M}{\sigma^2}\left(\delta + \frac{\rho\Lambda}{\beta+d}\right) + \left(\frac{\delta d(\beta+d) - \rho\beta\Lambda}{\delta d(\beta+d) + \rho d\Lambda}\right) \approx -1.9126 \times 10^3 < 0$$

2.2 The constant term is also negative:

$$-\left(\delta - \frac{\rho\Lambda\beta}{d(\beta+d)}\right) = -0.008\dot{3} < 0$$

The values used in Figures 5.6 and 5.8 do not satisfy the other conditions. However, there are values that do satisfy both conditions. This indicates that the set of parameter values of one case is not completely contained in the set of the other case although there is some overlap. In fact, the values in Table 5.6 satisfy both conditions. This interesting occurrence indicates that if a specific set of conditions is relatively simple to satisfy, we can focus on the controls indicated by those conditions, rather than those suggested by the other case. It is easy to verify that the values in Table 5.6 satisfy both conditions.

1. Both quantities in the conditions for asymptotic stability via LaSalle's Invariance Principle must be less than or equal to unity:

   (a)
   $$\frac{\gamma(\gamma\lambda\Lambda + \delta d)^2}{4(\alpha+d)d^2} \approx 0.1691 \leq 1$$

   (b)
   $$\frac{\beta\Lambda}{\delta d M} \approx 0.0594 \leq 1$$

2. The parameter values satisfy condition 2 for global asymptotic stability of the **PFE** via monotone systems:

   2.1 The coefficient of the second term is negative:
   $$\frac{\lambda\gamma\Lambda\beta}{\sigma d(\alpha+d)} - \frac{\lambda M}{\sigma^2}\left(\delta + \frac{\rho\Lambda}{\beta+d}\right) + \left(\frac{\delta d(\beta+d) - \rho\beta\Lambda}{\delta d(\beta+d) + \rho d\Lambda}\right) \approx -389.6021 < 0$$

   2.2 The constant term is also negative:
   $$-\left(\delta - \frac{\rho\Lambda\beta}{d(\beta+d)}\right) = -0.008\dot{3} < 0$$

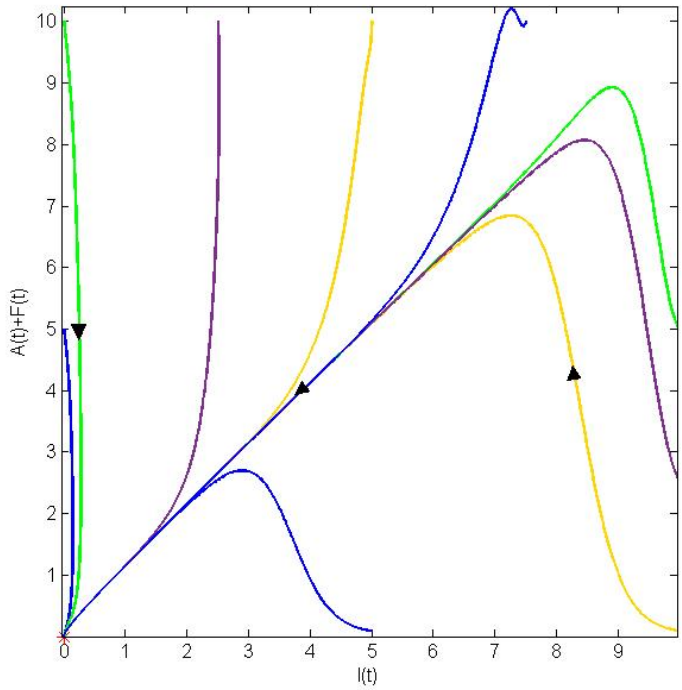

Figure 5.8: Solutions of model 5.1 clearly converge to the **PFE**, when the parameter values satisfy either condition (1),(2) or (3). This illustrates Theorem 5.7. The parameter values in Table 5.5 were used.

Parameter	Value	Parameter	Value
Λ	1.0000	λ	0.7000
γ	0.1000	β	1.0000
σ	0.4235	ρ	0.2000
δ	$0.841\dot{6}$	M	100.00
α	0.0100	d	0.2000

Table 5.6: Parameter values used in Figure 5.9

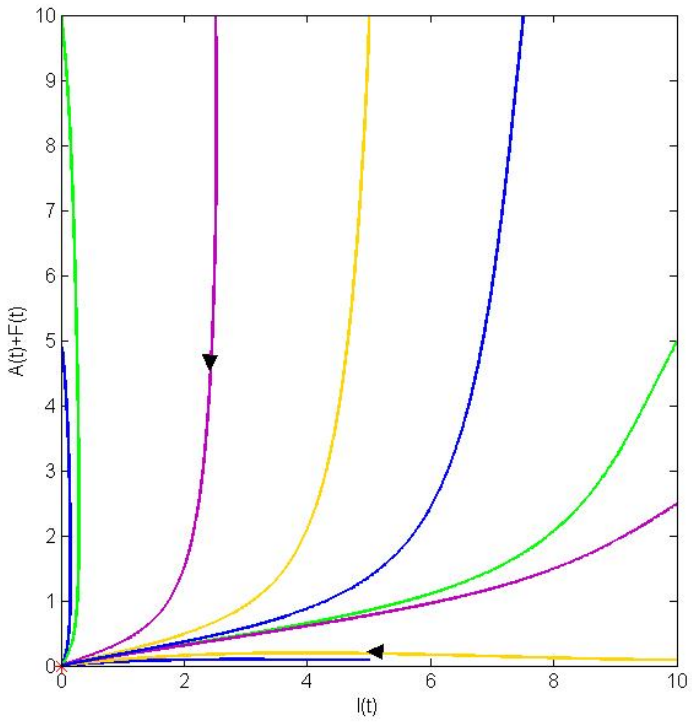

Figure 5.9: Convergence to the **PFE** when the conditions for stability via LaSalle's Invariance Principle (condition (5.12) on page 54) and condition (2) are satisfied is similar to convergence under only the conditions for stability via LaSalle's Invariance Principle. These parameter values can be found in Table 5.6.

Parameter	Value	Parameter	Value
Λ	1.0000	λ	0.7000
γ	0.4000	β	1.0000
σ	0.1000	ρ	0.2000
δ	0.2000	M	100.00
α	0.0100	d	0.2000

Table 5.7: Parameter values used in Figure 5.10

The conditions (5.12) on page 54 and the conditions (1)-(3) are sufficient but not necessary for the global asymptotic stability of the **PFE**. Indeed, the parameter values in Table 5.7 satisfy neither condition, yet result in extinction of the pathogen and resultant infection. We verify that the conditions are not satisfied:

1. Both quantities in the conditions for asymptotic stability via LaSalle's Invariance Principle must be less than or equal to unity:

   (a)
   $$\frac{\gamma(\gamma\lambda\Lambda + \delta d)^2}{4(\alpha + d)d^2} = 1.219047619047618 \not\leq 1$$

   (b)
   $$\frac{\beta\Lambda}{\delta d M} = 0.25 < 1$$

2. The conditions for asymptotic stability via monotone systems are not satisfied by these parameter values either.

   1.
   $$\Delta_1 \approx 8.3914 \times 10^5 \not\leq 0 \tag{5.17}$$

   Condition (1) is not satisfied.

   2.
   $$\frac{\lambda\gamma\Lambda\beta}{\sigma d(\alpha + d)} - \frac{\lambda M}{\sigma^2}\left(\delta + \frac{\rho\Lambda}{\beta + d}\right) + \left(\frac{\delta d(\beta + d) - \rho\beta\Lambda}{\delta d(\beta + d) + \rho d\Lambda}\right) \approx -2.5017 \times 10^3 < 0$$
   $$-\left(\delta - \frac{\rho\Lambda\beta}{d(\beta + d)}\right) = 0.633\dot{3} \not\leq 0 \tag{5.18}$$

   Thus condition (2) is not satisfied.

   3. The discriminant is positive, the F^*-value at which the maximum occurs is approximately $-48.6677 \not\geq 0$, and the constant term is $0.633\dot{3} \not\leq 0$. Thus condition (3) is not satisfied either.

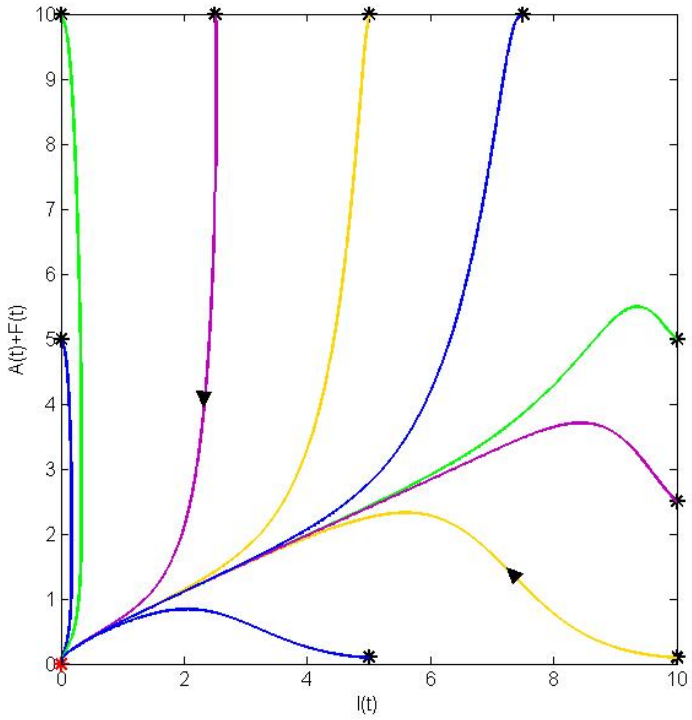

Figure 5.10: Convergence to the **PFE** can occur when neither the conditions for stability via LaSalle's Invariance Principle (equation (5.12) on page 54) nor those derived via the constructed monotone system (conditions (1)-(3)) are satisfied.. This illustrates that the conditions found above are sufficient but not necessary for **PFE** to be globally asymptotically stable.

5.6.2 Lower Approximation for the Host-Pathogen Model

The lower approximating system is

$$\frac{dz}{dt} = h(y) = h(A, F, I) = \begin{pmatrix} \lambda A(\gamma I - A) - \sigma A \\ -\delta F + \sigma A - \frac{\rho\Lambda}{d} F \\ \frac{\beta F}{M+F}\frac{\Lambda}{\beta+d} - (\alpha + d)I \end{pmatrix}. \tag{5.19}$$

Expressions for the equilibrium values for I^* and A^* can be found in terms of F^*:

$$I^* = \frac{\Lambda}{(\beta + d)(\alpha + d)} \frac{\beta F^*}{M + F^*}.$$

Setting the first equation in this system to zero gives

$$\begin{aligned} A^* &= \frac{1}{\sigma}\left(\delta + \frac{\rho\Lambda}{d}\right) F^* \\ \implies A^* &= 0 \text{ or } I^* = \frac{\lambda A^* + \sigma}{\gamma\lambda}. \end{aligned}$$

Observe that if $A^* = 0$ then $F^* = 0$ and $I^* = 0$. However, if $I^* = \frac{\lambda A^* + \sigma}{\gamma\lambda}$ then F^* satisfies the following quadratic equation:

$$\frac{1}{\gamma\sigma}\left(\delta + \frac{\rho\Lambda}{d}\right) F^{*2} + \left(\frac{M}{\gamma\sigma}\left(\delta + \frac{\rho\Lambda}{d}\right) + \frac{\sigma}{\gamma\lambda} - \frac{\beta\Lambda}{(\beta + d)(\alpha + d)}\right) F^* + \frac{M\sigma}{\gamma\lambda} = 0. \tag{5.20}$$

If the parameters satisfy condition (5.21) then equation (5.20) has no positive real roots.

$$\frac{M}{\gamma\sigma}\left(\delta + \frac{\rho\Lambda}{d}\right) + \frac{\sigma}{\gamma\lambda} - \frac{\beta\Lambda}{(\beta + d)(\alpha + d)} > 0 \tag{5.21}$$

On the other hand, equation (5.20) has two positive real roots if and only if

1. $$\Delta_2 > 0, \tag{5.22}$$

   where Δ_2 is the determinant of equation (5.20),

2. and
$$\frac{M}{\gamma\sigma}\left(\delta + \frac{\rho\Lambda}{d}\right) + \frac{\sigma}{\gamma\lambda} - \frac{\beta\Lambda}{(\beta + d)(\alpha + d)} < 0 \tag{5.23}$$

In this case equation (5.20) has two positive roots, denoted F_1 and F_2 such that $0 < F_1 < F_2$. The corresponding equilibria for system (5.19) are $\mathcal{E}_1^L$ and $\mathcal{E}_2^L$.
The Jacobian matrix of system (5.19) is:

$$J_h = \begin{pmatrix} \lambda\gamma I - 2\lambda A - \sigma & 0 & \lambda\gamma A \\ \sigma & -\delta - \frac{\rho\Lambda}{d} & 0 \\ 0 & \frac{\beta M\Lambda}{(\beta+d)(M+F)^2} & -(\alpha + d) \end{pmatrix}.$$

It is easy to see that the equilibrium $\mathbf{0}$ is asymptotically stable, indeed the eigenvalues of $J_h(\mathbf{0})$, $\xi_1 = -\sigma$, $\xi_2 = -\left(\delta + \frac{\rho\Lambda}{d}\right)$ and $\xi_3 = -(\alpha + d)$ are all negative. After using one of the equations for I^* in the simplification we find that $J_h(\mathcal{E}_1^L)$ is:

$$J_h(\mathcal{E}_1^L) = \begin{pmatrix} -\lambda A^* & 0 & \lambda\gamma A^* \\ \sigma & -\delta - \frac{\rho\Lambda}{d} & 0 \\ 0 & \frac{\beta M\Lambda}{(\beta+d)(M+F^*)^2} & -(\alpha+d) \end{pmatrix},$$

which has the characteristic polynomial

$$\begin{aligned} q(\xi) = &-\xi^3 - \xi^2\left(\delta + \frac{\rho\Lambda}{d} + \alpha + d + \lambda A^*\right) \\ &- \xi\left((\alpha+d)\left(\delta+\frac{\rho\Lambda}{d}\right) + \lambda A^*\left(\delta + \frac{\rho\Lambda}{d} + \alpha + d\right)\right). \\ &+ \left(\frac{\sigma\lambda\gamma\beta\Lambda M}{(\beta+d)(M+F_1)^2} - \lambda\frac{(\alpha+d)(\delta d+\rho\Lambda)}{d}\right)A^*. \end{aligned}$$

We observe that $q(\xi) = 0$ admits a single positive eigenvalue if and only if

$$(\alpha+d)(\delta d+\rho\Lambda) < \frac{\sigma\gamma\beta\Lambda M d}{(\beta+d)(M+F_1)^2} \tag{5.24}$$

Theorem 5.8. If conditions (5.22) and (5.23), as well as condition (5.24) hold then for any initial conditions $(A_0, F_0, I_0) \geq \mathcal{E}_1^L$ the infection modelled by (5.1) persists. More precisely, we have

$$\lim_{t\to\infty}(A(t), F(t), I(t)) \geq \mathcal{E}_2^L$$

Proof. System (5.19) defines a dissipative monotone system. When conditions (5.22)-(5.23) are satisfied it admits two interior equilibria, denoted $\mathcal{E}_1^L$ and $\mathcal{E}_2^L$, which satisfy $F_1 \leq F_2$. The irreducible matrix $J_h(\mathcal{E}_1^L)$ has a single positive eigenvalue if and only if condition (5.24) is satisfied, this eigenvalue has corresponding eigenvector u, with $u \gg 0$. Applying Theorem 2.8 we conclude that there exist solutions $y_r(t)$, originating from points $y_r = \mathcal{E}_1^L + ru$ where $r \in (0, \epsilon],$ and $\epsilon > 0$, such that $y_r(t) \to \mathcal{E}_2^L$ as $t \to \infty$.

Consider any solution $(A(t), F(t), S(t), I(t))$ of model (5.1), and denote $\hat{x}(t) = (A(t), F(t), I(t))$ such that $\frac{d\hat{x}}{dt} \geq h(\hat{x})$. Let $\hat{y}(t)$ be any solution of system (5.19), which satisfies $\hat{y}(0) = \hat{x}(0)$. Then, since h is quasi-monotone, using [[122], Section 12.X] we obtain the inequality

$$\hat{y}(t) \leq \hat{x}(t) \quad \forall t > 0.$$

Therefore, under condition (5.24) we have

$$0 < \mathcal{E}_2^L = \lim_{t\to\infty}\hat{y}(t) \leq \lim_{t\to\infty}\inf\hat{x}(t).$$

That is, the infection persists, at least at the level $\mathcal{E}_2^L$. □

Theorem 5.8 is illustrated numerically by Figure 5.11 on page 67. The initial conditions of the solutions in this figure were chosen specifically so that $(A_0, F_0, I_0) \geq \mathcal{E}_1^L$. Clearly any solution initiated at or above the level of $\mathcal{E}_1^L \approx (3.4733, 1.4638, 0.1849)$ persists at a non-zero level above $\mathcal{E}_2^L$ for all time; and in fact converges to an equilibrium of model (5.1), at least for the parameter values in Table 5.8. We verify that the data in Table 5.8 satisfies conditions (5.22)-(5.24) of Theorem 5.8.

We have

1. $\Delta_2 \approx 0.0338 > 0$, thus condition (5.22) is satisfied.

2. $$\frac{M}{\gamma\sigma}\left(\delta + \frac{\rho\Lambda}{d}\right) + \frac{\sigma}{\gamma\lambda} - \frac{\beta\Lambda}{(\beta+d)(\alpha+d)} \approx -0.3968 < 0,$$
   thus condition (5.23) is satisfied.

3. $$(\alpha+d)(\delta d + \rho\Lambda) < \frac{\sigma\gamma\beta\Lambda M d}{(\beta+d)(M+F_1)^2} \approx -8.4206 < 0,$$
   where $F_1 \approx 1.4638$, and thus condition (5.24) is satisfied.

Parameter	Value	Parameter	Value
Λ	0.9000	λ	1.0000
γ	23.52536	β	5.0000
σ	1.0000	ρ	0.9000
δ	0.9000	M	10.000
α	0.0010	d	0.5500

Table 5.8: Parameter values used in Figure 5.11.

Our numerical investigations of model (5.1) have revealed that in addition to the **PFE** which is always locally attracting, there exists an equilibrium close the **PFE**

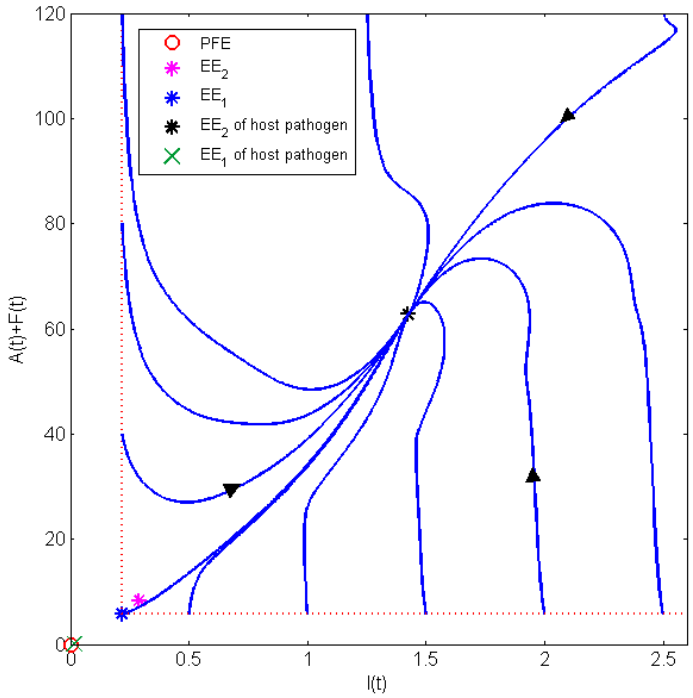

Figure 5.11: Illustration of persistence of the infection as proven in Theorem 5.8 on page 66. Observe that solutions of model (5.1) originating at the level of $\mathcal{E}_1^L$ remain non-zero for all time.

which is repelling, and an attracting equilibrium which is removed from the **PFE**. Although we have not proven this mathematically, the important properties of the model, namely extinction and persistence, have been proven under certain conditions.

5.7 Local Sensitivity Analysis: Finding possible methods of control

As a result of the relative ease of obtaining results from epidemiological models, mathematicians are frequently tasked with identifying which parameters should be focussed on in order to decrease the impact a disease has. Often this is done by investigating which parameters influence the stability properties of equilibria, or even the number of equilibria the model admits.

Consider the case in which persistence of the pathogen occurs (see Subsection 5.6.2). To save a field from infection certain control measures are taken, and a specific method is applied only because it negatively affects the progression of the infection. There is regularly more than option open to farmers, and often a combination of controls is applied to a field.

This section investigates possible control methods in situations where the host-pathogen model is applicable through a local sensitivity analysis of the thresholds $\mathcal{R}_1^*$ and $\mathcal{R}_2^*$. By decreasing these thresholds below unity we ensure that no endemic equilibria exist, and the infection will in theory be eradicated. A local sensitivity analysis of a quantity is done by calculating the sensitivity index of that quantity with respect to the parameters on which it depends.

Definition 5.1. The *normalized forward sensitivity index* of a quantity, Q, that depends differentiably on a parameter, p, is defined as:

$$\Upsilon_p^Q := \frac{\partial Q}{\partial p} \cdot \frac{p}{Q}$$

◁

We calculate the sensitivity indices of the threshold parameters $\mathcal{R}_1^*$ and $\mathcal{R}_2^*$, with respect to all ten of our parameters. Most of the expressions for the indices are complex, but can be found on page 72. These are evaluated at the parameter values used in Section 5.6.2 and are summarised in Table 5.9. We see that β, d and γ are important parameters for both thresholds, along with M for $\mathcal{R}_1^*$ and δ for $\mathcal{R}_2^*$. Since $\Upsilon_\beta^{\mathcal{R}_1^*} = 1.61430$, a 10% increase in β leads to a 16.143% increase in $\mathcal{R}_1^*$. Similarly, since $\Upsilon_d^{\mathcal{R}_2^*} = -1.43638$ an *increase* of 10% in d results in a *decrease* of 14.3638% in $\mathcal{R}_2^*$. All parameters influence at least threshold relatively well, except α.

From Table 5.9 the following are possible control methods:

1. Lowering the constant recruitment rate, Λ, so that the pool of susceptible hosts decreases to the level of the **PFE**, ensuring extinction of the infection.

2. By decreasing the amount of attached pathogen an infective root can support, γ, the carrying capacity is reached sooner, and decay increases as nutrients are depleted.

3. By decreasing the probability of transmission β the number of host-pathogen contacts needed before infection occurs is increased, which effectively increases the probability of death during the search for a new host.

4. If the rate at which pathogen de-attach from hosts, σ, is decreased the carrying capacity of the root is reached sooner, which in turn causes the pathogen to die as a result of competition for nutrients.

5. Increasing the natural host death rate, d, causes the host population to decrease, which in turn leads to more prolific decay of attached pathogen.

6. A counter intuitive result is that increasing the attachment rate, ρ, results in a decrease of both thresholds. By increasing this rate, the carrying capacity of the host is reached in less time, which results in an increase of the decay of attached pathogen.

7. By increasing the decay rate of the free pathogen, δ, the pathogen has a greater chance of dying before finding a new host.

8. Decreasing M leads to the maximum transmission rate being reached quicker. This allows for high levels of infected plants, which results in higher pathogen decay rates both on and off host.

The options that theoretically offer the largest impact, might not be feasible in practise. This could lead to a combination of the 'less' effective strategies being used. For example, the combination (3), (4), (6), (7) might be the most effective cost wise, and control wise.

Parameter	Value	$\mathcal{R}_1^*$	$\mathcal{R}_2^*$
Λ	0.9000	+0.49690	+0.68212
λ	1.0000	$+0,15467$	+0.44351
γ	23.52536	+1.00000	+1.00000
β	5.0000	+1.61430	+0.78024
σ	1.0000	+0.69066	+0.51214
ρ	0.9000	−0.50310	−0.31788
δ	0.9000	−0.34222	−0.80319
M	10.000	−0.84533	−0.07561
α	0.0010	−0.00181	−0.00181
d	0.5500	−1.33970	−1.43638

Table 5.9: Sensitivity indices of $\mathcal{R}_1^*$ and $\mathcal{R}_2^*$ to parameters for the host-pathogen model, evaluated at the given parameter values

The expressions for each sensitivity index are presented on the following pages, although to simplify notation we define

$$f := \delta\sigma^2(d+\beta)^2 + 2\lambda M(\delta(\beta+d)+\rho\Lambda)(\delta d+\rho\Lambda) > 0 \text{ and}$$

$$g := 2\delta(d+\beta)d\sigma^2 + \lambda(\delta d+\rho\Lambda)^2 M > 0.$$

Recall that:

$$\begin{aligned}
\mathcal{R}_1^* &= \frac{\lambda\gamma\sigma\Lambda\beta[\delta(\beta+d)+\rho\Lambda]}{(\alpha+d)[2\lambda M[\delta(\beta+d)+\rho\Lambda](\delta d+\rho\Lambda)+\delta\sigma^2(d+\beta)^2]} \\
&= \frac{\lambda\gamma\sigma\Lambda\beta[\delta(\beta+d)+\rho\Lambda]}{(\alpha+d)f} \\
\mathcal{R}_2^* &= \frac{\lambda\gamma\sigma\Lambda\beta(\delta d+\rho\Lambda)}{(\alpha+d)[2\delta(d+\beta)d\sigma^2+\lambda(\delta d+\rho\Lambda)^2 M]} \\
&= \frac{\lambda\gamma\sigma\Lambda\beta(\delta d+\rho\Lambda)}{(\alpha+d)g}
\end{aligned}$$

$$\Upsilon_{\Lambda}^{\mathcal{R}_1^*} = \frac{\partial \mathcal{R}_1^*}{\partial \Lambda} \cdot \frac{\Lambda}{\mathcal{R}_1^*} = 1 + \frac{\rho\Lambda}{\delta(\beta+d)+\rho\Lambda} - \frac{2\Lambda\rho\lambda M[2(\delta d+\rho\Lambda)+\delta\beta]}{f}$$

$$\Upsilon_{\lambda}^{\mathcal{R}_1^*} = \frac{\partial \mathcal{R}_1^*}{\partial \lambda} \cdot \frac{\lambda}{\mathcal{R}_1^*} = \frac{\delta\sigma^2(d+\beta)^2}{f} > 0$$

$$\Upsilon_{\gamma}^{\mathcal{R}_1^*} = \frac{\partial \mathcal{R}_1^*}{\partial \gamma} \cdot \frac{\gamma}{\mathcal{R}_1^*} = 1$$

$$\Upsilon_{\beta}^{\mathcal{R}_1^*} = \frac{\partial \mathcal{R}_1^*}{\partial \beta} \cdot \frac{\beta}{\mathcal{R}_1^*} = 1 + \frac{\delta\beta}{\delta(\beta+d)+\rho\Lambda} - \frac{2\beta\delta[\lambda M(\delta d+\rho\Lambda)+\sigma^2(d+\beta)]}{[\delta(\beta+d)+\rho\Lambda]f}$$

$$\Upsilon_{\sigma}^{\mathcal{R}_1^*} = \frac{\partial \mathcal{R}_1^*}{\partial \sigma} \cdot \frac{\sigma}{\mathcal{R}_1^*} = 1 - \frac{2\delta\sigma^2(d+\beta)^2}{f}$$

$$\Upsilon_{\rho}^{\mathcal{R}_1^*} = \frac{\partial \mathcal{R}_1^*}{\partial \rho} \cdot \frac{\rho}{\mathcal{R}_1^*} = \frac{\rho\Lambda}{\delta(\beta+d)+\rho\Lambda} - \frac{2\rho\Lambda\lambda M(2(\delta d+\rho\Lambda)+\delta\beta)}{f}$$

$$\Upsilon_{\delta}^{\mathcal{R}_1^*} = \frac{\partial \mathcal{R}_1^*}{\partial \delta} \cdot \frac{\delta}{\mathcal{R}_1^*} = \frac{\delta(\beta+d)}{\delta(\beta+d)+\rho\Lambda} - \frac{2\lambda M\delta(2d\delta(\beta+d)+\rho\Lambda(\beta+2d))}{f} - \frac{\delta\sigma^2(\beta+d)^2}{f}$$

$$\Upsilon_{M}^{\mathcal{R}_1^*} = \frac{\partial \mathcal{R}_1^*}{\partial M} \cdot \frac{M}{\mathcal{R}_1^*} = \frac{-2\lambda M(\delta d+\rho\Lambda)(\delta(\beta+d)+\rho\Lambda)}{f} \leq 0$$

$$\Upsilon_{\alpha}^{\mathcal{R}_1^*} = \frac{\partial \mathcal{R}_1^*}{\partial \alpha} \cdot \frac{\alpha}{\mathcal{R}_1^*} = \frac{-\alpha}{(\alpha+d)} \leq 0$$

$$\Upsilon_{d}^{\mathcal{R}_1^*} = \frac{\partial \mathcal{R}_1^*}{\partial d} \cdot \frac{d}{\mathcal{R}_1^*} = \frac{d\delta}{\delta(\beta+d)+\rho\Lambda} - \frac{d}{(\alpha+d)} - \frac{2d\delta\lambda M(2(\delta d+\rho\Lambda)+\delta\beta)+2d\sigma^2(\beta+d)}{f}$$

$$\Upsilon_{\Lambda}^{\mathcal{R}_2^*} = \frac{\partial \mathcal{R}_2^*}{\partial \Lambda} \cdot \frac{\Lambda}{\mathcal{R}_2^*} = 1 + \mathcal{R}_0 - \frac{2\lambda M\rho\Lambda(\delta d+\rho\Lambda)}{g}$$

$$\Upsilon_{\lambda}^{\mathcal{R}_2^*} = \frac{\partial \mathcal{R}_2^*}{\partial \lambda} \cdot \frac{\lambda}{\mathcal{R}_2^*} = \frac{2\delta(d+\beta)}{g}$$

$$\Upsilon_{\gamma}^{\mathcal{R}_2^*} = \frac{\partial \mathcal{R}_2^*}{\partial \gamma} \cdot \frac{\gamma}{\mathcal{R}_2^*} = 1$$

$$\Upsilon_{\beta}^{\mathcal{R}_2^*} = \frac{\partial \mathcal{R}_2^*}{\partial \beta} \cdot \frac{\beta}{\mathcal{R}_2^*} = 1 - \frac{2\delta d\sigma^2\beta}{g}$$

$$\Upsilon_{\sigma}^{\mathcal{R}_2^*} = \frac{\partial \mathcal{R}_2^*}{\partial \sigma} \cdot \frac{\sigma}{\mathcal{R}_2^*} = 1 - \frac{4\delta d\sigma(d+\beta)}{g}$$

$$\Upsilon_{\rho}^{\mathcal{R}_2^*} = \frac{\partial \mathcal{R}_2^*}{\partial \rho} \cdot \frac{\rho}{\mathcal{R}_2^*} = \mathcal{R}_0 - \frac{2\rho\Lambda\lambda M(\delta d+\rho\Lambda)}{g}$$

$$\Upsilon_{\delta}^{\mathcal{R}_2^*} = \frac{\partial \mathcal{R}_2^*}{\partial \delta} \cdot \frac{\delta}{\mathcal{R}_2^*} = \frac{\delta d}{\delta d+\rho\Lambda} - \frac{2\delta(\delta\lambda M(\delta d+\rho\Lambda)+(d+\beta)d\sigma^2)}{g}$$

$$\Upsilon_{M}^{\mathcal{R}_2^*} = \frac{\partial \mathcal{R}_2^*}{\partial M} \cdot \frac{M}{\mathcal{R}_2^*} = \frac{-\lambda(\delta d+\rho\Lambda)^2}{g} < 0$$

$$\Upsilon_{\alpha}^{\mathcal{R}_2^*} = \frac{\partial \mathcal{R}_2^*}{\partial \alpha} \cdot \frac{\alpha}{\mathcal{R}_2^*} = \frac{-\alpha}{(\alpha+d)} < 0$$

$$\Upsilon_{d}^{\mathcal{R}_2^*} = \frac{\partial \mathcal{R}_2^*}{\partial d} \cdot \frac{d}{\mathcal{R}_2^*} = \frac{\delta d}{(\delta d+\rho\Lambda)} - \frac{d}{\alpha+d} - \frac{2\delta d(\lambda M(\delta d+\rho\Lambda)+(d+\beta)\sigma^2)}{g}$$

6 The Spatio-Temporal Host Pathogen Model

6.1 Introduction

The model in Section 5 looks only at the temporal progression of an infection, and although this approach is acceptable under certain assumptions (such as a pathogen entering an entire field in a uniform manner), the model can be modified slightly to accommodate the spatial movement of pathogens through the field.

Diffusion has been used to model spatial spread in theoretical ecology since the latter half of the twentieth century [47], [92], with its use for modelling fungal growth being justified by the "observation that tip growth occurs to fill space and to capture nutrients" [21]. Davidson (1998) also noted that fungal growth "is, in the main, directed from areas of high hyphal density to areas of low hyphal density", and included diffusion in his model with the warning 'that this flux should not be viewed as the movement of existing biomass, but rather the propensity of new biomass to grow away from high density areas'. We reiterate this warning, and include diffusion to model the spatial growth of off-host pathogen in search of new hosts, with μ denoting the diffusion constant.

$$\begin{aligned}
\frac{\partial A}{\partial t} &= \lambda A(\gamma I - A) - \sigma A + \rho F S \\
\frac{\partial F}{\partial t} &= -\delta F + \sigma A - \rho F S + \mu \Delta F \\
\frac{\partial S}{\partial t} &= \Lambda - dS - \frac{\beta F}{M+F} S \\
\frac{\partial I}{\partial t} &= \frac{\beta F}{M+F} S - (\alpha + d) I
\end{aligned} \tag{6.1}$$

$$\begin{aligned}
&A(x,0) \geq 0, \quad F(x,0) \geq 0, \\
&S(x,0) \geq 0, \quad I(x,0) \geq 0, \\
&\frac{\partial F}{\partial x}(-L,t) = 0 = \frac{\partial F}{\partial x}(L,t).
\end{aligned}$$

If the initial condition is spatially uniform, and taking the boundary conditions into account, then the solution is also spatially uniform, reducing it to a solution of the corresponding temporal system. The properties and long-term behaviour of the temporal model have been theoretically proven in Section 5, and this chapter devotes itself to numerical investigation of the behaviour of solutions of system (6.1). Our interest is mainly in the practically relevant case where the pathogen is introduced in one location, and studying the dynamics of its propagation.

6.2 Numerical Investigations

6.2.1 Under the conditions for asymptotic stability obtained by application of LaSalle's Invariance Principle

The parameter values given in Table 6.1 satisfy the conditions (5.12) which ensure the **PFE** of the temporal model is globally asymptotically stable. The details are given in Section 5.5.2. We investigate whether the solutions of the spatio-temporal model behave in a similar fashion, using the diffusion constant $\mu = 0.01$. Indeed, although convergence occurs over a long time period, the addition of diffusion does not result in observable change in the asymptotic properties of the steady state.

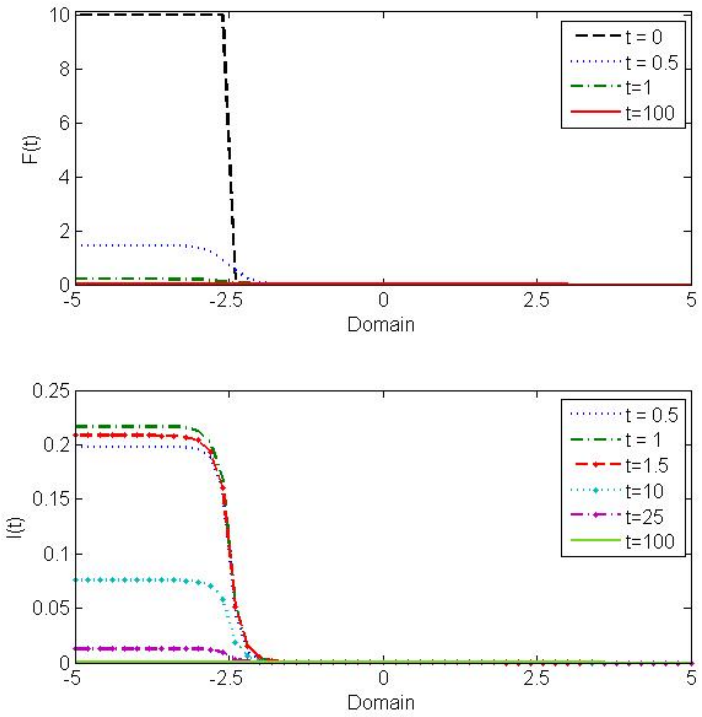

Figure 6.1: The spread of pathogen and disease through the field at different times, using the parameter values that satisfy the conditions for stability of the **PFE** that were obtained by the application of LaSalle's Invariance Principle. It is assumed that there is an initial population of free pathogen over a quarter of the field, not yet having resulted in infection.

Parameter	Value	Parameter	Value
Λ	1.0000	λ	0.4000
γ	0.2000	β	1.0000
σ	0.0100	ρ	0.4000
δ	0.1000	M	100.000
α	0.0205	d	0.1000

Table 6.1: Parameter values used in Figure 6.1

6.2.2 Parameter values for global asymptotic stability of the PFE of the temporal model via monotone systems

An alternative proof of global asymptotic stability of the **PFE** is via an upper approximating monotone system, see Section 5.6.1. The parameter values in Table 6.2 satisfy the conditions of this case, and addition of linear diffusion using $\mu = 0.01$ does not change the properties of the steady state. Indeed convergence occurs by $t = 100$.

The same initial conditions were used for Figures 6.1- 6.2 and we observe that although the initial disease progression occurs quicker in the second case, levels of infected hosts decrease sooner as well. In Figure 6.1 $I(-5, 10) \approx 0.075$ while $I(-5, 10) \approx 0.055$ in Figure 6.2. This suggests that the infection is more virulent in this section, though it runs its course quicker than the infection in the previous section.

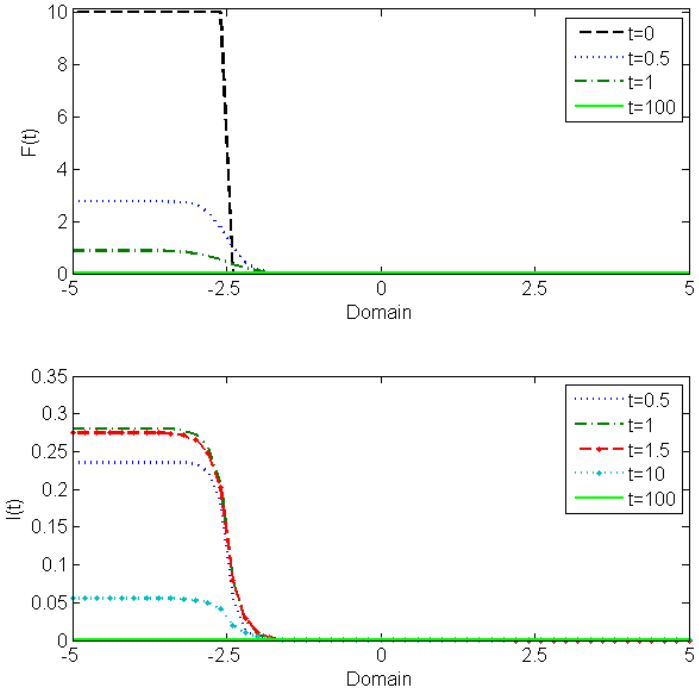

Figure 6.2: The spread of pathogen and disease through the field at different times, using the parameter values that satisfy the conditions for which the **PFE** has been proved to be globally asymptotically stable via the study of monotone systems. The values are given in Table 6.2 on page 76.

When the parameter values satisfy both sets conditions for global stability of the **PFE** the progression is similar to that in Figure 6.2.

Parameter	Value	Parameter	Value
Λ	1.0000	λ	0.7000
γ	0.9000	β	1.0000
σ	$0.188\dot{2}$	ρ	0.2000
δ	$0.841\dot{6}$	M	100.00
α	0.0100	d	0.2000

Table 6.2: Parameter values used in figure 6.2

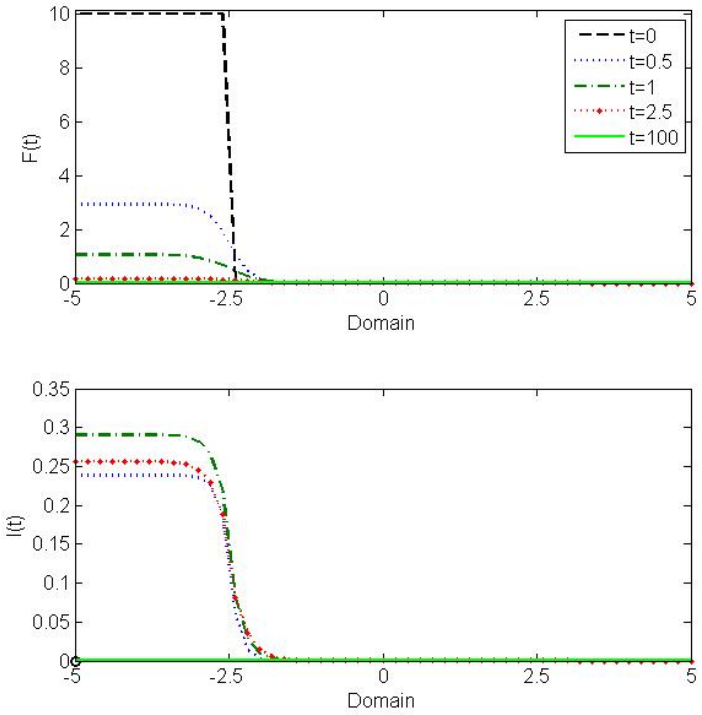

Figure 6.3: The parameter values in Table 6.3 satisfy the conditions for global asymptotic stability found by the application of LaSalle's Invariance Principle and via monotone systems.

6.2.3 Parameter values for persistence of the infection

A monotone system, constructed to approximate the temporal host pathogen model from below was proven to admit two interior equilibrium in Section 5.6.2, denoted $\mathcal{E}_1^L$ and $\mathcal{E}_2^L$, with $F_1^L < F_2^L$. Additional conditions were derived, under which the pathogen persists. Indeed, it was found that solutions initiated at or above $\mathcal{E}_1^L$ and satisfying conditions (5.22)-(5.24) remain non-zero for all $t \geq 0$.

The equilibrium, $\mathcal{E}_1^L$ of the lower approximating system is:
$\mathcal{E}_1^L \approx (3.4733, 1.4638, 0.1849)$. The persistence of pathogen is illustrated in Figure 6.5. In fact, solutions converge to $\mathcal{EE}_2$ (equilibrium of the host-pathogen model with the largest F^* value), although the stability properties of $\mathcal{EE}_1$ and $\mathcal{EE}_2$ have not been proven. How does the inclusion of diffusion affect this phenomenon?

Parameter	Value	Parameter	Value
Λ	1.0000	λ	0.7000
γ	0.1000	β	1.0000
σ	0.4235	ρ	0.2000
δ	$0.8416\dot{6}$	M	100.00
α	0.0100	d	0.2000

Table 6.3: Parameter values used in Figure 6.3

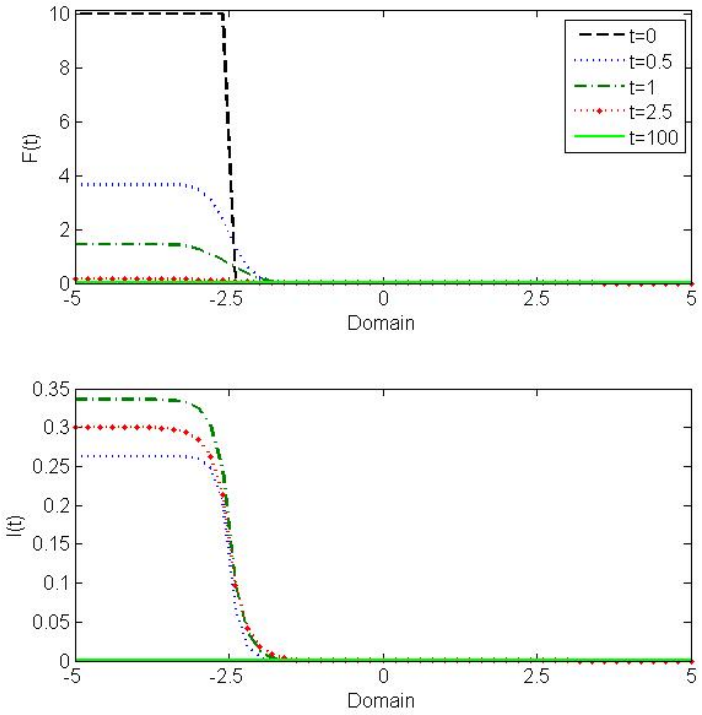

Figure 6.4: The parameter values in Table 6.4 do not satisfy either condition for asymptotic stability of the **PFE**, however convergence is obvious. This indicates that the conditions found in section 5 are merely sufficient rather than necessary for the asymptotic stability of the **PFE**.

Parameter	Value	Parameter	Value
Λ	1.0000	λ	0.7000
γ	0.4000	β	1.0000
σ	0.1000	ρ	0.2000
δ	0.2000	M	100.00
α	0.0100	d	0.2000

Table 6.4: Parameter values used in Figure 6.4

Solutions initiated at the level of $\mathcal{EE}_2$ at the boundary exhibit a travelling infection front, the movement of which is driven by the increase in attached pathogen and infested hosts by the diffusion of the free pathogen (Figure 6.5). This behaviour suggests a possible control strategy: if the speed of the front can be sufficiently decreased, a percentage of the field would be saved from disease.

To this end, we investigate the relationship between μ and the wave speed c. The parameter values in Table 6.5 were again used, and a solution with (A_0, F_0, I_0) taking the value of $\mathcal{EE}_2$ on the left boundary was considered. The diffusion constant μ was taken to be in the interval $[10^{-7}, 10^{-1}]$, which results in $c \in (0, 4.5 \times 10^{-3}]$. An equation of the form $c(\mu) = a\mu^b$ was fitted to the data in Figure 6.6, and the fitting process reveals $a \in (0.010770, 0.011)$ and $b \in (0.416, 0.4218)$ with 95% confidence. In fact, $a = 0.01088$ and $b = 0.4189$. Literature indicates that the value of b should be higher, with Gilligan [33] and Metz, Mollison and van den Bosch [85] finding the wave speed to be proportional to the square root of the diffusion constant; that is $c \propto \sqrt{\mu}$. Although $b < 0.5$ the equation fits the data well, and since $SSE = 8.59 \times 10^{-6}$ its use in making predictions would be justified. The coefficient of determination $r^2 = 0.9933$ indicating that 99.33% of the variance of the data is explained by the equation.

Parameter	Value	Parameter	Value
Λ	0.9000	λ	1.0000
γ	23.52536	β	5.0000
σ	1.0000	ρ	0.9000
δ	0.9000	M	10.000
α	0.0010	d	0.5500

Table 6.5: Parameter values used in Figure 6.5.

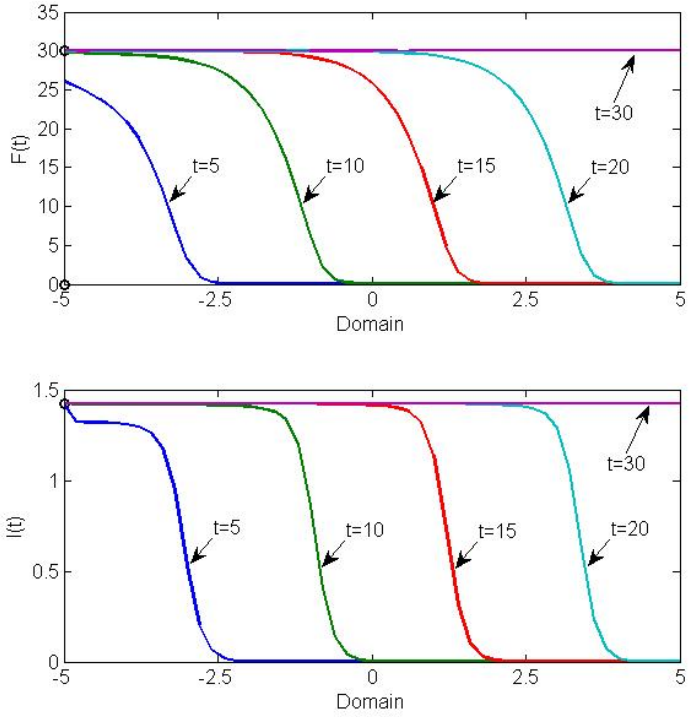

Figure 6.5: When solutions of model 6.1 are initiated with pathogen and infectious hosts at the level of $\mathcal{EE}_2$, on the left boundary, a field of completely susceptible hosts will experience a travelling infection front. This front connects $\mathcal{EE}_2$ and the **PFE**.

6.3 Numerical Scheme

In order to solve model 6.1 numerically, we use forward-time and second order central-space discretisations, subjected to Neumann boundary conditions. The equations are:

$$\begin{aligned}
\frac{A_i^{n+1}-A_i^n}{\Delta t} &= \lambda A_i^n\left(\gamma I_i^n - A_i^{n+1}\right) - \sigma A_i^{n+1} + \rho F_i^{n+1} S_i^n \\
\frac{F_i^{n+1}-F_i^n}{\Delta t} &= -\delta F_i^{n+1} + \sigma A_i^{n+1} - \rho F_i^{n+1} S_i^n + D\left(\frac{F_{i+1}^n - 2F_i^n + F_{i-1}^n}{(\Delta x)^2}\right) \\
\frac{S_i^{n+1}-S_i^n}{\Delta t} &= \Lambda - dS_i^{n+1} - \frac{\beta F_i^{n+1}}{M+F_i^{n+1}} S_i^{n+1} \\
\frac{I_i^{n+1}-I_i^n}{\Delta t} &= \frac{\beta F_i^{n+1}}{M+F_i^{n+1}} S_i^{n+1} - (\alpha+d) I_i^{n+1}
\end{aligned}$$

On the left and right boundary backwards and forwards discretizations are used to properly take the boundary conditions into account. That is, at the left boundary

$$\frac{F_0^n - F_{-1}^n}{\Delta t} = 0 \quad \Longrightarrow \quad F_0^n = F_{-1}^n.$$

This in turn changes the diffusion operator on the boundary to

$$\frac{F_1^n - 2F_0^n + F_{-1}^n}{(\Delta x)^2} = \frac{F_1^n - F_0^n}{(\Delta x)^2}.$$

The right boundary is treated in a similar fashion.

For each time step these equations can be collected into a system of matrices, namely

$$BX^{n+1} = CX^n + D.$$

The matrices B and C must be rebuilt after every time step, although D remains constant.

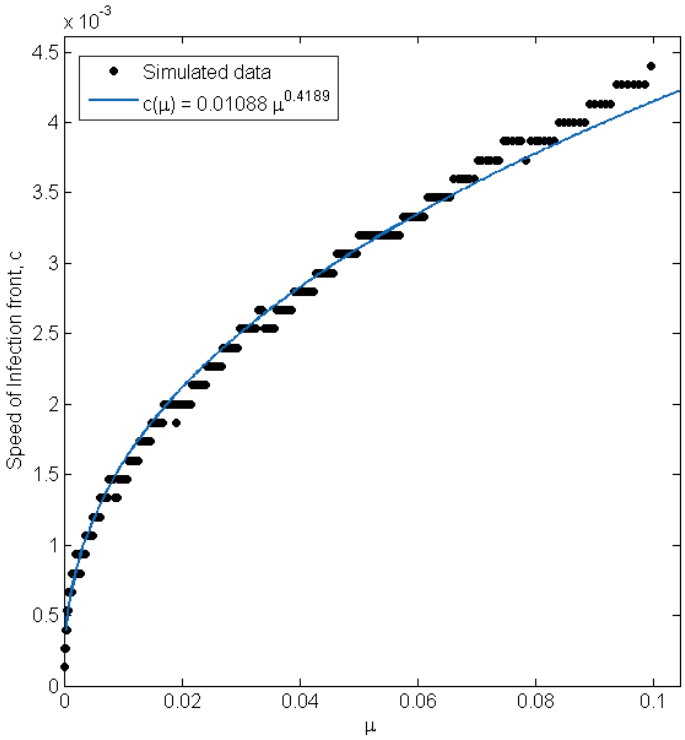

Figure 6.6: The speed of the infection front for different values of μ, for solutions of model 6.1 initiated with levels of inoculum and disease at the level of $\mathcal{EE}_2$ on the left boundary. The parameter values in Table 6.5 were used. Clearly the equation $c(\mu) = 0.01088\mu^{0.4189}$ fits the data well.

7 Discussion and Future work

7.1 Discussion

The spread of disease in agricultural crops places immense strain on the global crop yield which ensures that the study of botanical diseases is of vital importance. The use of mathematical models improves our understanding of the underlying mechanisms for certain biological phenomena and often reveal possible methods to control disease.

By comparing the underlying assumptions of the model in chapter 3 with the biological processes that occur, we found room for improvement. This model was first published in Gilligan (1995) and is an SEIR model with linear diffusion on the infective compartment. When the population size is constant the temporal model admits the disease free equilibrium, which is globally asymptotically stable when $\mathcal{R}_0 \leq 1$, and locally asymptotically stable when $\mathcal{R}_0 > 1$, as well as a locally asymptotically stable endemic equilibrium which only exists when $\mathcal{R}_0 > 1$ [79]. Gilligan's model admits travelling wave solutions: minimal oscillations during convergence to the endemic equilibrium in the temporal model result in a single infection front travelling through the field. If, on the other hand, solutions of the temporal model experience significant oscillations during convergence, a travelling wave solution exists for the spatial-temporal model [33].

In an attempt to improve the realism of the model, an additional assumption was made: that the soil environment is a porous medium. This assumption resulted in replacing the linear diffusion in chapter 3 with a non-linear diffusion operator. It was shown numerically that for large diffusion constants this change does not significantly alter the asymptotic behaviour of solutions; however, for slow diffusion the speed of the infection front decreases considerably.

The host-pathogen model was developed to avoid the underlying issues of placing a diffusion operator directly onto the infective compartment in chapters 3 and 4 – namely that plants generally do not experience physical movement. Instead we considered a model of two populations, the host which could either be susceptible or infective, and the pathogen which could be attached to a host or in search of a new one. Although $\mathcal{R}_0 < 1$ for all parameter values, the model admits either only the pathogen free equilibrium **PFE**, or the **PFE** and two endemic equilibria. The **PFE** is always locally asymptotically stable and the global asymptotic stability was proven using two methods: a Lyapunov function, and the construction of a monotone system that approximates the model from above. These methods lead to two sets of *sufficient* conditions for the global stability. The parameter values that satisfy these conditions have some overlap, but there are values that satisfy only one but not the other. This is a useful results, as it offers two possible sets of control strategies for the eradication of the disease.

Although the stability properties of the endemic equilibria were not proven, numerical simulations indicate that the equilibrium with the higher level for free pathogen is asymptotically stable, and the other is unstable, with the possibility of being a saddle point. It was proven that under certain conditions the pathogen persists for all time.

The model was then extended to include a spatial component, by the addition of diffusion on the free pathogen sub-population. This agrees with the behaviour of pathogens such as fungi searching randomly for new hosts. This inclusion did not result in solutions deviating from the behaviour that had been proven for the temporal model. Indeed, under the conditions for persistence, solutions initiated at the level of the stable endemic equilibrium result in a travelling infection front that joins this equilibrium to the **PFE**. Thus, if effective control strategies are applied, a fraction of the crop could be saved from disease. Some such control strategies include (A) decreasing the probability of transmission β, which increases the number of host-pathogen contacts needed before infection occurs. This effectively increases the probability of death during the search for a new host. (B) Lowering the rate at which pathogen detach from hosts, σ, ensures that the carrying capacity of the root is reached sooner, which in turn causes the pathogen to die as a result of competition for nutrients. (C) Although counter intuitive at first glance, increasing the attachment rate results in the carrying capacity of the host being reached in less time. This leads to an increase of the decay of attached pathogen. (D) By decreasing M the maximum transmission rate is reached sooner, this allows for a quicker infection, and the pathogenic carrying capacity of host roots to be reached in less time. Suggested control strategies (B)-(D) all indirectly cause the carrying capacity of infected roots to be reached sooner. While affecting γ directly might not be feasible, the combination of (B)-(D) give an alternative.

7.2 Future work

In future the host-pathogen model could be extended to include two spatial directions - in order to realistically approximate agricultural fields. This extension could be used to properly investigate host spacing and the affect it has on the propagation of infection, such as in [13], and [66]. Another interesting extension of this research is to consider fields that use rotational farming, a common agricultural practise that helps with disease control. Some pathogen are able to form resting structures in the absence of hosts, or even to colonise sub-optimal hosts, so modelling the availability of hosts as impulses should shed light on the re-occurrence of infections.

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