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Mathematical modelling of fungal contamination of citrus produce along the pre-harvest supply chain

by

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Declaration of Authorship

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Abstract

South Africa is one of the major exporter of the citrus produce to sophisticated markets such as the European markets. Unfortunately, its trading position was compromised when *Guignardia citricarpa* invaded and persisted in citrus produce growing regions. The epidemiological development of this pathogen has no harm to humans, but reduce fruits quality and quantity. As a result this negatively affects the farmers' income, diet supplier and the economy in general. Fresh produce has a natural mechanism of effectively protecting themselves against microbial contamination. However, inappropriate manipulation during the planting, harvesting, storage, processing, distribution, preparation or handling and abuse of temperature during storage or transportation increases the risk of microbial contamination of citrus produce along the supply chain. In most cases the quantity of the micro-organism contaminating the citrus produce may be very small but then grows with time in the presence of suitable conditions. In this study, three mathematical models are developed to study the transmission dynamics of *Guignardia citricarpa* at different levels and scales. The first model captures the dynamics of epidemiological aspects of Citrus Black Spot (CBS). The second model is an immunological model which deals with the within-host dynamics and the third model is the fusion of the immunological and epidemiological models. The mathematical properties of all models are studied to determine whether they are mathematically and epidemiologically well-posed. The basic reproduction number R_0 for all models are computed using the generation operator approach and the Jacobian matrix. If $R_0 < 1$, then the disease free equilibrium states of all models are locally and globally asymptotically stable, but if $R_0 > 1$ then the endemic equilibrium state is locally and globally asymptotically stable. Sensitivity analysis is done on the variation of their model parameters and finally the numerical simulation of the models is done to confirm the behaviour of the models with time. In all the models the results show that their reproduction number is more sensitive to changes in the recruitment rate parameters and the transmission probability. Hence, cleaning the orchard floor regularly, spraying susceptible fruits while they are still young and emphasise on the good agriculture practise can slow down the probability transmission rate.

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Contents

Declaration of Authorship i

Abstract ii

Acknowledgements iii

Contents iv

List of Figures viii

1 INTRODUCTION 1

1.1 Background 1

1.2 Life cycle of *Guignardia citricarpa* Kiely 4

1.3 Supply chain of citrus produce 7

1.4 Problem statement 9

1.4.1 Aims 10

1.4.2 Objective 10

1.4.3 Hypothesis 10

1.5 Methodology 10

1.6 Outline of the thesis 11

1.7 Conclusion 12

2 MATHEMATICAL MODELLING FRAMEWORK 13

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Creating Future Leaders

2.1 Introduction 13
2.2 Formulation of mathematical models 15
2.3 Analysis of the model 20
2.3.1 Equilibrium 20
2.3.2 Stability 22
2.3.3 Reproduction number R_0 25
2.3.4 Numerical methods 28
2.4 Modelling in Agriculture. 30
2.5 Modelling fresh produce 32
2.6 Modelling citrus in general 33
2.7 Modelling citrus fungal disease 34
2.8 Modelling Citrus Black Spot 35
2.9 Conclusion 36

3 EPIDEMIOLOGICAL MODEL FOR CITRUS BLACK SPOT DYNAMICS BETWEEN FRUITS 37

3.1 Introduction 37
3.2 Model formulation 38
3.2.1 Fruit population 39
3.2.2 Pathogen population 39
3.3 Basic properties 42
3.3.1 Positivity of solutions 42
3.3.2 Feasible region of the model. 42
3.4 Determination of disease-free equilibrium (DFE) of the model (3.2.4) and stability. . . . 44
3.4.1 Disease free equilibrium (E_0) 44
3.5 Reproduction number of the dynamical system of CBS 45
3.6 Local stability of disease free equilibrium E_0 47
3.7 Global stability of the disease free equilibrium. 49
3.8 The endemic equilibrium state and its stability 50

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3.8.1 Endemic equilibrium 50
3.8.2 The local stability of the endemic equilibrium E_1 52
3.8.3 Global stability of the endemic equilibrium 54
3.9 Numerical simulation 55
3.10 Summary 61

4 IMMUNOLOGICAL MODEL OF *Guignardia citricarpa* 62
4.1 Introduction 62
4.2 Model formulation 63
4.3 Analysis of the model 65
4.4 Basic Properties 67
4.4.1 Positivity of solution 67
4.5 Determination of disease free equilibrium and its stability 68
4.5.1 Reproduction number of the dynamical system of CBS 69
4.5.2 Local stability of disease free equilibrium E_0 71
4.5.3 Global stability of the disease free equilibrium. 72
4.6 The endemic equilibrium state (E_1) and its stability 74
4.6.1 The local stability of the endemic equilibrium (E_1) 75
4.7 Numerical simulation 77
4.8 Conclusion 80

5 A MULTI-SCALE MODEL FOR CITRUS BLACK SPOT 82
5.1 Introduction 82
5.2 Model formulation 83
5.3 Analysis of the model 88
5.4 Basic properties 88
5.5 The disease free equilibrium point and its stability. 90
5.6 Reproduction number of the dynamical system of Citrus Black Spot 91
5.7 Local stability of disease free equilibrium 96
5.8 Global stability of disease free equilibrium. 98

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Creating Future Leaders

5.9 The endemic equilibrium state its stability 100
5.10 Existence and uniqueness endemic equilibrium 102
5.10.1 The Local Stability of the Endemic Equilibrium E_1 103
5.11 Sensitivity analysis 111
5.12 Numerical analysis 114
5.13 Conclusion 119

6 DISCUSSION AND CONCLUSION **121**
6.1 Introduction 121
6.2 Summary 123
6.3 Further research directions 124

Bibliography **125**

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Creating Future Leaders

List of Figures

1.1 *The life cycle of Guignardia citricarpa Adapted from Citrus Research and Education Center* 6
1.2 *Citrus fresh produce supply chain.* 7

2.1 *A conceptual diagram for mathematical modelling process.* 14
2.2 *A schematic representation of the general CBS model with pathogens in the environment* 18
2.3 *Simulation of model (2.2.3) showing the evolution with time of Guignardia citricarpa whenever* $R_0 < 1$ 29
2.4 *Simulation of model (2.2.3) showing the evolution with time of Guignardia citricarpa whenever* $R_0 > 1$ 30

3.1 *The possible environments of where Guignardia citricarpa evolves with time* 39
3.2 *Conceptual diagram of pathogen and fruits.* 40
3.3 *Simulation of model system 3.2.4 showing the evolution with time of susceptible fruits* (S)*, infected fruits* (I)*, Ascomata* (P_0) *and Ascospores* (P_A) *for about 30 days by varying* β*.* 56
3.4 *Simulation of model system 3.2.4 showing the evolution with time of susceptible fruits* (S)*, infected fruits* (I)*, Ascomata* (P_0) *and Ascospores* (P_A) *for about 30 days by varying* ψ*.* 57
3.5 *Simulation of model system 3.2.4 showing the evolution with time of susceptible fruits* (S)*, infected fruits* (I)*, Ascomata* (P_0) *and Ascospores* (P_A) *for about 30 days by varying* α*.* 58
3.6 *Simulation of model system 3.2.4 showing the evolution with time of susceptible fruits* (S)*, infected fruits* (I)*, Ascomata* (P_0) *and Ascospores* (P_A) *for about 30 days by varying* η*.* 59
3.7 *Simulation of model system 3.2.4 showing the evolution with time of susceptible fruits* (S)*, infected fruits* (I)*, Ascomata* (P_0) *and Ascospores* (P_A) *for about 30 days by varying* δ*.* 60

4.1 *Conceptual model of the within host dynamics of Citrus Black spot.* 64
4.2 *Simulation results showing the evolution of fruit and pathogen population behaviour* β . 78
4.3 *Graphs of numerical solution of the model system 4.2.1 showing appressoria, ascospores, infected defence system and susceptible defence system varying* μ 79
4.4 *Graphs of numerical solution of the model system 4.2.1 showing appressoria, ascospores, infected defence system and susceptible defence system varying* ρ 80

5.1 *Conceptual model of linked between-host and within-host transmission dynamics of CBS* 84

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Creating Future Leaders

5.2 *The fruits and The pathogen result of over 30 days.* 115
5.3 *The fruits and The pathogen result of over 30 days about the* ψ. 116
5.4 *Fruit population behaviour over a period of time when varying* β 117
5.5 *Fruit population behaviour over a period of time when varying* μ 118
5.6 *Fruit population behaviour over a period of time when varying* ρ 119

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Chapter 1

INTRODUCTION

1.1 Background

Citrus production has been done for years and is well known for consumption as fresh produce and as processed products like juices and jams. It is the most important fruit crop in the world trade and globally, in terms of production (Paul, 2006). Citrus is primarily produced for export to some of the most sophisticated world markets demanding high-quality standards. The South African citrus industry significantly contributes to its economy and it is the second largest earner of foreign exchange in terms of agriculture exports (Paul, 2006).

Various diseases reduce the farmers' income and employment of the Southern African citrus industry. Intervention strategies for these diseases impact on the profitability and production and may also lead to environmental contamination and undermine the sustainability of production.

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Citrus Black Spot (CBS) is a fungal disease caused by *Guignardia citricarpa* in the sexual stage and *Phyllosticta citricarpa* in the asexual stage (Kotzé, 1981) infecting citrus leaves and fruits causing lesions on the rind of fruits. It is known to have developed in Australia in 1895 on fruits growing areas around Sydney (Benson, 1895). McAlpine recorded anamorphic stage of the causal organism in 1899 and named the pathogenic organism *Phoma* as a new species (*Phoma citricarpa* McAlpine) (Kiely et al., 1948) then later in 1953 the name was changed to *Phyllostictina citricarpa* and was changed again to *Phyllosticta citricarpa* in 1973 by Van der Aa (Warnich et al., 1996). The sexual stage, *Guignardia citricarpa* was then recorded by Kiely (Kiely et al., 1948) and various stages in its life cycle were established, these entails ascomata, ascospores, appressoria, pycnidia and conidia, more details of the life cycle is given in figure 1.2. CBS surfaced in South Africa in 1929 as reported by (Doidge, 1929), in the coastal region of Natal, then spread to major citrus producing inland eastern and northern Transvaal (Kotzé, 1981). It occurs in subtropical regions with summer rainfall in the following countries: Swaziland, Mozambique, China, Indonesia, Argentina, Australia, Brazil, Hong Kong, Kenya, Honduras, Jamaica, Trinidad, Nigeria, Peru, South Africa, Taiwan, Uruguay, Zimbabwe, and Japan (Kotzé, 1981). CBS distribution shows that it is limited to areas where climate favours the occurrence of the pathogen. The risk of CBS transmitting to areas where it does not currently occur depends on climate conditions (Wager and Chase, 1952). Recently, the development of this disease has raised concerns in the citrus industry worldwide.

The symptoms of Citrus Black Spot (CBS) occur in various forms such as hard spots, cracked spots, false melanose and virulent spots, which can be described further in the following manner.

(i) Hard spots normal appear when fruits start maturing and these are the most common lesions and they are small, round, and sunken with gray at the centre and brick red to black margins. The average diameter of hard spot lesions ranges from 3–10mm (Kotzé, 2000). Generally, they are known to be pre-harvest symptoms but can be found in early harvested fruits after distribution. Hard spots normal occurs on the part exposed to sunlight (Kotzé, 1981).

(ii) False melanose are numerous small tan slightly raised lesions. Normally occurs on green fruits and the lesions are much smaller than the hard spot variety with an average diameter of less than

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$1mm$ (Kotzé, 2000). In many cases, false melanose develops into hard spot as the season progress.

(iii) Freckle spot lesions usually occur after fruits have changed colour from green to yellow. Several spots may form one big lesion or a stain similar to melanose, this may result in a virulent spot during storage. Freckle spots occur on both unripe and ripe fruits and are large, slightly raised, dark brown spots (Dewdney et al., 2010).

(v) Virulent spots are small reddish, differently shaped lesions which develop late during the season when fruits are matured and during high temperatures. Virulent spots are also a problem at post-harvest in storage (Dewdney et al., 2010). These spots appear on healthy but latently infected rinds.

Citrus Black Spot development is greatly influenced by three major factors, that is, the inoculum availability, the climatic conditions required for infection and the susceptible host (fruit development stage). In general, ascospores produced on dead leaves on the orchard floor, represent the main source of inoculum. Infectious *Guignardia citricarpa* ascospores' availability depends on the frequency of wetting and drying as well as prevailing temperatures (Kiely et al., 1948). When ascospores mature, the availability of moisture leads to swelling of the ascus which press against the opening of the perithecium and forcibly eject the ascospores which become infectious to susceptible trees (Kotzé, 1981). The ascospores are spread by either air current or water to come in contact with young citrus fruit, germinate and infection takes place (Kiely et al., 1948). Young fruits are only susceptible to ascospores during prolonged rainfall during the first four months after fruits set and then become resistant. Once *Guignardia citricarpa* ascospores invades, it becomes latent until the fruit becomes mature and it may further develop to produce pycnidia (Kiely et al., 1948). Long distance transmission can be facilitated by fruits or leaves that have a latent infection when they are moved to trash. The use of uncertified nurseries can also facilitate transmission through infected stocks of young plants from infected regions.

CBS is currently managed mostly by the use of certified nursery trees, approved pre-harvest fungicides spray programmes, good agriculture practise (GAPs), orchard sanitation and proper fruit handling during

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picking and packing (Snowden, 2008). Trees in poor conditions are most susceptible, therefore, the use of good agriculture practice and other management measures can assist in preventing the wide spread of the disease. This involves controlling primary inoculum caused by ascospores, by removing the orchard floor litter. The secondary inoculum caused by conidia can be managed by clearing all infected matured fruits from the orchard and removing all litter on the orchard floor.

Due to its impact in various sectors of the economy, CBS has become an important subject of investigation in the citrus growing areas. CBS lesions on fruits significantly lower the market value of fruit in the sophisticated international market and result in the product being re-directed to the local insensitive market. This results lower prices because of an oversupply of fruit on the local market. Pre-harvest CBS losses arise when severely affected fruit drop prematurely in the orchard and go to waste (Kotzé, 2000). Furthermore, CBS management programmes are very costly (Kiely, 1969). If CBS is not managed it may cause total loss of the crop and in some regions, citrus production will be impossible. Besides farmers income, CBS has also promoted job losses in the citrus industry. Citrus planting in Southern African is mainly done for exporting to international markets such as European market. These foreign markets demand high-quality standard products and inspection is scrutinised all the way along the post-harvest supply chain. This follows that, if symptoms appear on fruits in a container already packed destined for international markets it has to be unpacked to get it out or redirected the whole package to local markets, which are less sensitive to avoid huge losses associated with products rejection in the international destination (Paul, 2006).

1.2 Life cycle of *Guignardia citricarpa* Kiely

The epidemiology of CBS occurs in two ways, that is, the primary source of infection facilitated by ascospores spread by the air current, found on the orchard floor litter and the secondary source in the form of conidia found in infected fruit. Initially, leaves and fruits on the ground from previous seasons harbour ascomata for a certain period until moist presents itself, then ascomata begin producing ascospores in leaf litter on the orchard floor. Ascospores production is catalysed by a sequential wetting and drying of the leaf litter. The ascospores are ejected from the fruiting bodies of the fungus during rainfall or irrigation and then spread by the air current. When the ascospores land on susceptible tissue, it germinates forming

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appressoria (Truter et al., 2007). After the infection pin along with mycelia settles in the area between the cuticle and epidermal wall, The symptoms do not appear right away because the infection remains latent until the fruit is mellow. The pathological process in leaves remarkably remains inactive until the leaves drop. The leaf pathology commonly gives rise to ascospores, but sometimes develop pycnidia. These pycnidia discharge (conidia) in a jellylike mass (Truter et al., 2007). Whenever there are wet conditions the jelly-like mass dissolves and the spores are dispersed by splashes of water. This splash spread method limits secondary infection to close fruit or leaves of the same tree. Fruit pathological process stays latent until fruit matures. The mycelium grows into the outer rind, also known as a flavedo. Here in the flavedo, circular lesions are formed, which are sometimes accompanied by pycinidia. Plants infected by Citrus Black Spot usually drop fruits early before they fully mature, reducing plant yields and rendering highly blemished fruits which are not marketable even in domestic markets. The diagram shown on figure 1.1 summarise the life cycle of *Guignardia citricarpa* and the dynamics of disease development. This also helps in modelling the pattern formed during the life time of the CBS.

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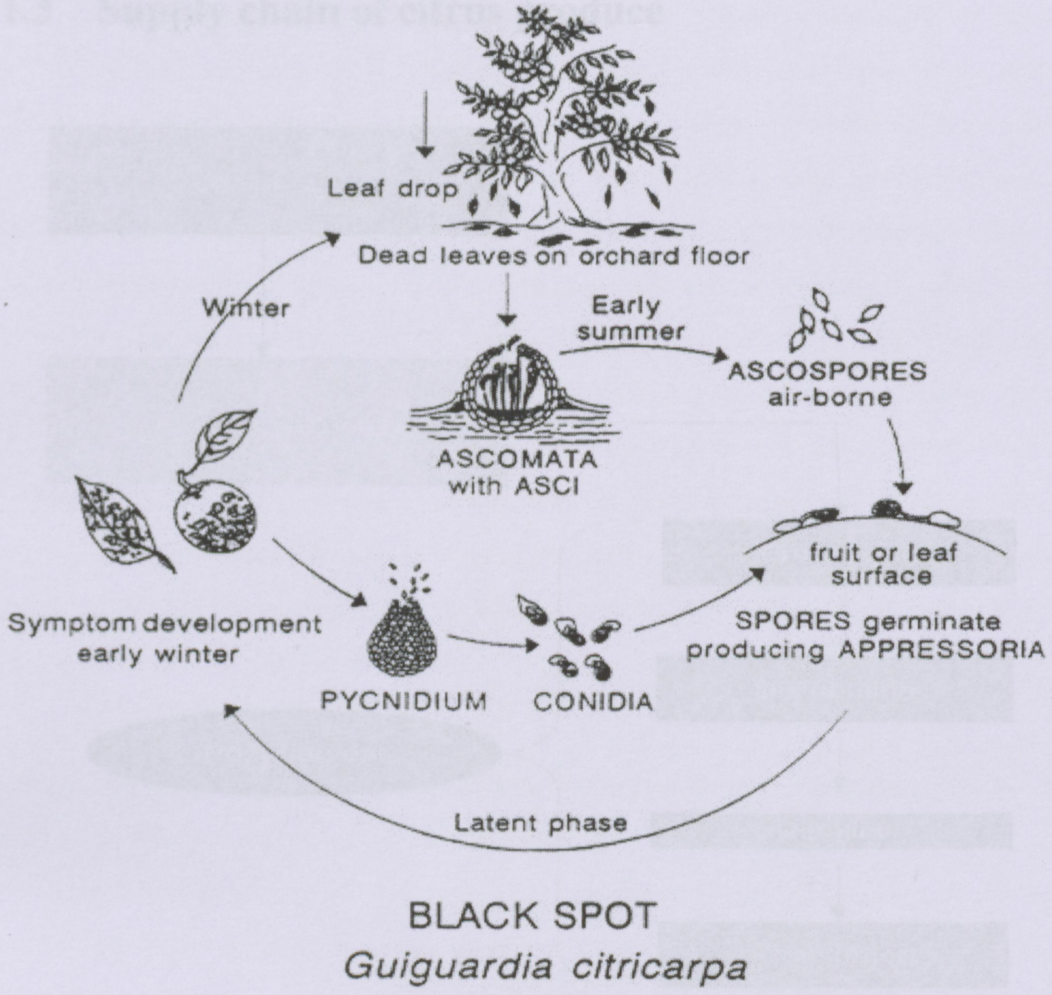

Figure 1.1: *The life cycle of Guignardia citricarpa Adapted from Citrus Research and Education Center*

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1.3 Supply chain of citrus produce

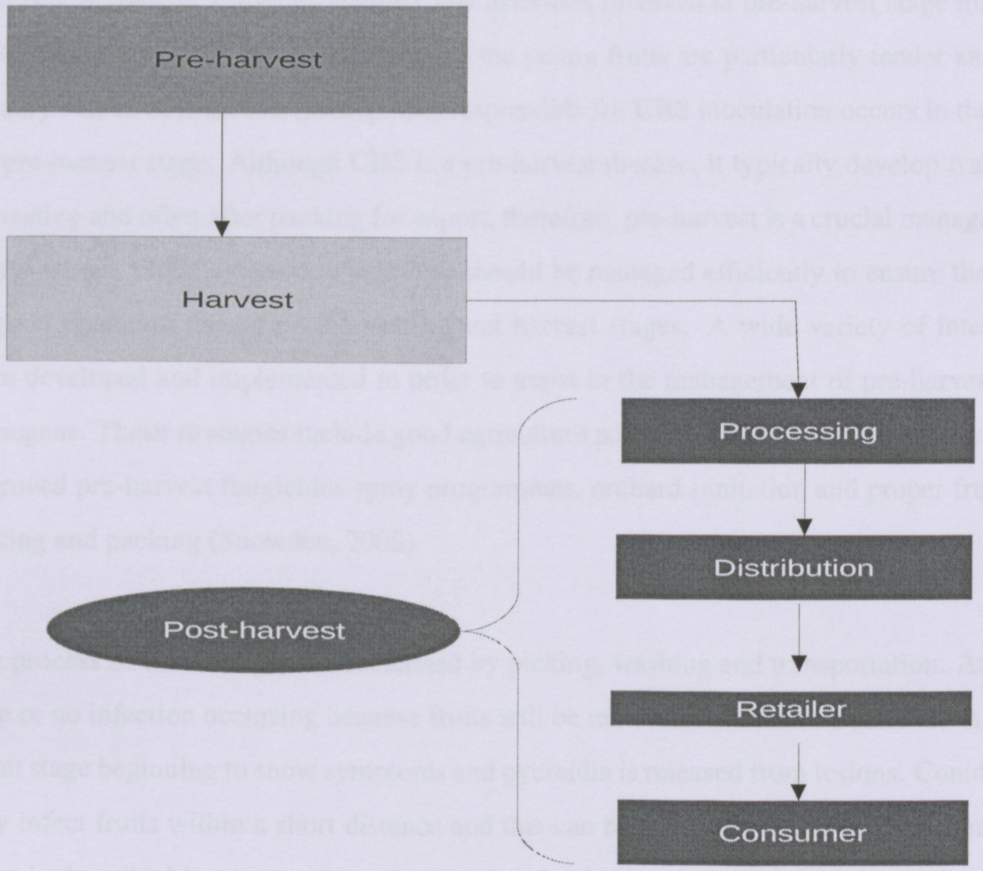

Figure 1.2: *Citrus fresh produce supply chain.*

Citrus produce supply chain provided in figure (1.2) illustrate the distribution of citrus produce from the producer (pre-harvest activities) to the final consumer (post-harvest activities) through various network nodes. The *Guignardia citricarpa* virulence, fruit susceptibility and environmental conditions influence the frequency and intensity of disease occurrence most at the pre-harvest stage, but, however, decay and spoilage can occur at any point in the supply chain. In each and every node of the supply chain, there are activities which may promote fungal diseases affecting quality and shelf life of citrus produce.

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These activities may include workers handling fruits with contaminated hands or machinery, the use of trees from infected nurseries and leaving contaminated litter unattended until the next growing season. To this end, there is a high probability that activities involved at pre-harvest stage may facilitate fungal contamination because it is the time when the young fruits are particularly tender and susceptible. The primary source of infection (ascospores) responsible for CBS inoculation occurs in the orchard, which is the pre-harvest stage. Although CBS is a pre-harvest disease, it typically develop fruits symptoms after harvesting and often after packing for export, therefore, pre-harvest is a crucial management point to prevent wastage. Good agriculture practices should be managed efficiently to ensure that fruits are always in good condition during pre-harvesting and harvest stages. A wide variety of intervention strategies were developed and implemented in order to assist in the management of pre-harvest and post-harvest pathogens. These strategies include good agriculture practices (GAPs), the use of certified nursery trees, approved pre-harvest fungicides spray programmes, orchard sanitation and proper fruit handling during picking and packing (Snowden, 2008).

The process of harvesting is characterised by picking, washing and transportation. At this stage there is little or no infection occurring because fruits will be matured, however, infected fruits which were in the latent stage beginning to show symptoms and pycinidia is released from lesions. Conidia at this stage can only infect fruits within a short distance and this can be spread by water or air current. The processing stage is described by a sequence of events involved in sanitation and packing. During this stage, the fruits are screened, washed with chemicals and packed in branded packages. During this stage, there is a high unlikely chance of infection to occur due to the resistance nature of mature fruits since infection can only occur to tender susceptible young fruits, but however, if pycinidia is virulent enough, infection is likely to occur. Proper washing needs to be done to avoid unnecessary contamination.

After processing, the fruits are taken to various markets. This is the distribution level where handling should be prioritised in order to prevent contamination and the growth of fungal infection. If there is an abuse of temperature during distribution, the tiny blemish can over time develop into a disease that will spoil the fruit.

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CBS spoilage is characterised by changes that renders fruits undesirable and unacceptable for human consumption by reducing the quality and quantity of the fruits but is not harmful to humans and animals. The main focus of this study is on the transmission dynamics of Citrus Black Spot (CBS) diseases along pre-harvest the supply chain.

Modelling of plant diseases is rapidly growing within plant disease epidemics. The first plant disease model was constructed by (Vanderplank, 1963) and have since been spreading like diseases themselves. According to (Kranz and Royle, 1978) modelling is done to serve three purposes: to predict, describe disease pattern and conceptualise in line with the objectives.

1.4 Problem statement

Citrus Black Spot is a difficult disease to address due to its complex elements of infection, which are not yet fully understood. Efforts have been made to manage diseases of this nature by introducing good agricultural practice measures to enable smooth international trading, fair farmers' income, better employment opportunities and diet support. However, the use of fungicides to manage the disease should be limited to avoid disease resistance and other damages associated with its application. The disease is also hard to control where the host is used for various purposes such as, feeding livestock by the rind and pulp of the infected host and preparing compost which will be applied as manure back into the orchard. The compost may be a reservoir for the disease and promotes inoculation in the next session. The major concern is that fungal plant disease reduces the quality and quantity of fresh citrus produce. As a result, the product becomes unmarketable, rendering it less valuable in terms of diet support and monetary. European markets were forced to ban the imports from South Africa due to Citrus Black Spot but then later the ban was lifted due to scientific reasons but there is a high level of scrutiny of the products. Producing citrus fruits free from the black spot is crucial to increase revenue and improve customer satisfaction. A compartmental deterministic model of the transmission of Citrus Black Spot between the host and within the host is constructed. Much attention is given to the dynamics of the pathogen as the disease develops and fruits transit from one class to another.

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1.4.1 Aims

The main aim of the research is to construct a mathematical model that will improve our understanding of *Guignardia citricarpa* dynamics by providing a theoretical framework in which factors affecting the transmission of the *Guignardia citricarpa* can be explicitly considered.

1.4.2 Objective

- To investigate the transmission dynamics of Citrus Black Spot through simplest possible epidemiological models that incorporate major features responsible for the invasion and persistence.
- To improve our understanding of the within-host of Citrus Black Spot dynamics by developing a deterministic theoretical framework.
- To establish a multi-scale model framework for Citrus Black Spot by linking the within-host and epidemiological model.

1.4.3 Hypothesis

Mathematical models are helpful in describing and predicting invasion and persistence, and improving our understanding of plant disease epidemics and should be introduced to economists, plant pathologists and farmers because mathematical models provide an improved theoretical framework where experiments are not possible to conduct.

1.5 Methodology

This research employs the use of three deterministic models which have been proved to be crucial in the study of mechanisms underlying the transmission dynamics of diseases. The disease is studied intensively to provide a base for mathematical modelling and analysis of an epidemiological disease by the construction of state transition models at the host population level. Further extension of the model by

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zooming in the within-host dynamics and link the epidemiological model is done to solve multi-scale problems. Our theoretical framework is numerically solved using python v 2.7.1 software 2014.

1.6 Outline of the thesis

The dissertation is organised as follows: Chapter 1 presents an overview of Citrus Black Spot and the biological background of *Guignardia citricarpa*. It discusses the background of the study and the life cycle of CBS. The aims and objectives of the dissertation are laid out the introductory chapter and is concluded with a description of the structure of the dissertation.

Chapter 2 presents the literature review on mathematical modelling in general and background of mathematical modelling concepts used in epidemiology. The overview includes a bare-bones example used to explain mathematical tools that are used throughout the rest of this thesis. Some definitions, theorem and notation about dynamical systems and stability analysis, and theories that are required to analyse such systems is presented.

Chapter 3 provides the basic model of Citrus Black Spot epidemiological model capturing the transmission dynamics between hosts in the environment.

Chapter 4 provides the within host model that trace the cell scale level dynamics of disease development. Analytical and numerical results of the model were computed.

Chapter 5 present the multi-scale model describing the link between the within host and epidemiological of pathogen-host interactions. The theoretical framework provided basis underlying transmission of CBS. The basic reproduction number is calculated. The two steady states of the model was studied. Furthermore, sensitivity analysis of the basic reproduction number and the endemic equilibrium was carried out.

Chapter 6 provides the conclusion and summary of the main results.

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1.7 Conclusion

This chapter gives an overview of Citrus Black Spot and the biological background of its pathogen termed *Guignardia citricarpa*. The problems associated with the existence of CBS were discussed, giving an account of intervention strategies which were proposed by other researchers. The aims and objectives of the dissertation are laid out the introductory chapter is concluded with a description of the structure of the dissertation. There is an overwhelming evidence that Citrus Black Spot has caused a burden in various aspects of life. This disease has been given little attention, in as far as mathematical modelling is concerned. To this end, there has been no solution to completely eliminate and eradicated the disease. There is a need to use mathematical tools to improve intervention strategies. Establishing the link between epidemiology and within host transmission dynamics will be part of the research. To date, mathematical modelling of CBS has only been done on CBS distribution using Climex models (Yonow et al., 2013) and the prediction of ascospores on set using regression models. However, the focus of this dissertation is on the models that describe the transmission dynamics of CBS.

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Chapter 2

MATHEMATICAL MODELLING FRAMEWORK

2.1 Introduction

In this dissertation, mathematical modelling is used to study fungal contamination of citrus produce along the pre-harvest supply chain. The aim of this chapter is to explain crucial concepts of mathematical modelling which are used throughout this dissertation. Mathematical modelling by definition is a theoretical representation of real life situations by simplification of reality by attempting to summarise the dynamical systems, to put forward a hypothesis and to affirm their consequences (Xu, 2006). It is a broad concept that spans in a very wide of fields. These models are common in the natural sciences and engineering disciplines as well as in the social sciences. A model may be helpful to explain a system and to study the effects of different components, and to make predictions about behaviour (Billings, 2013). Furthermore, in epidemiology, mathematical models can predict how infectious diseases develop to show the likely outcome of an epidemic and provide information to improve intervention strategies.

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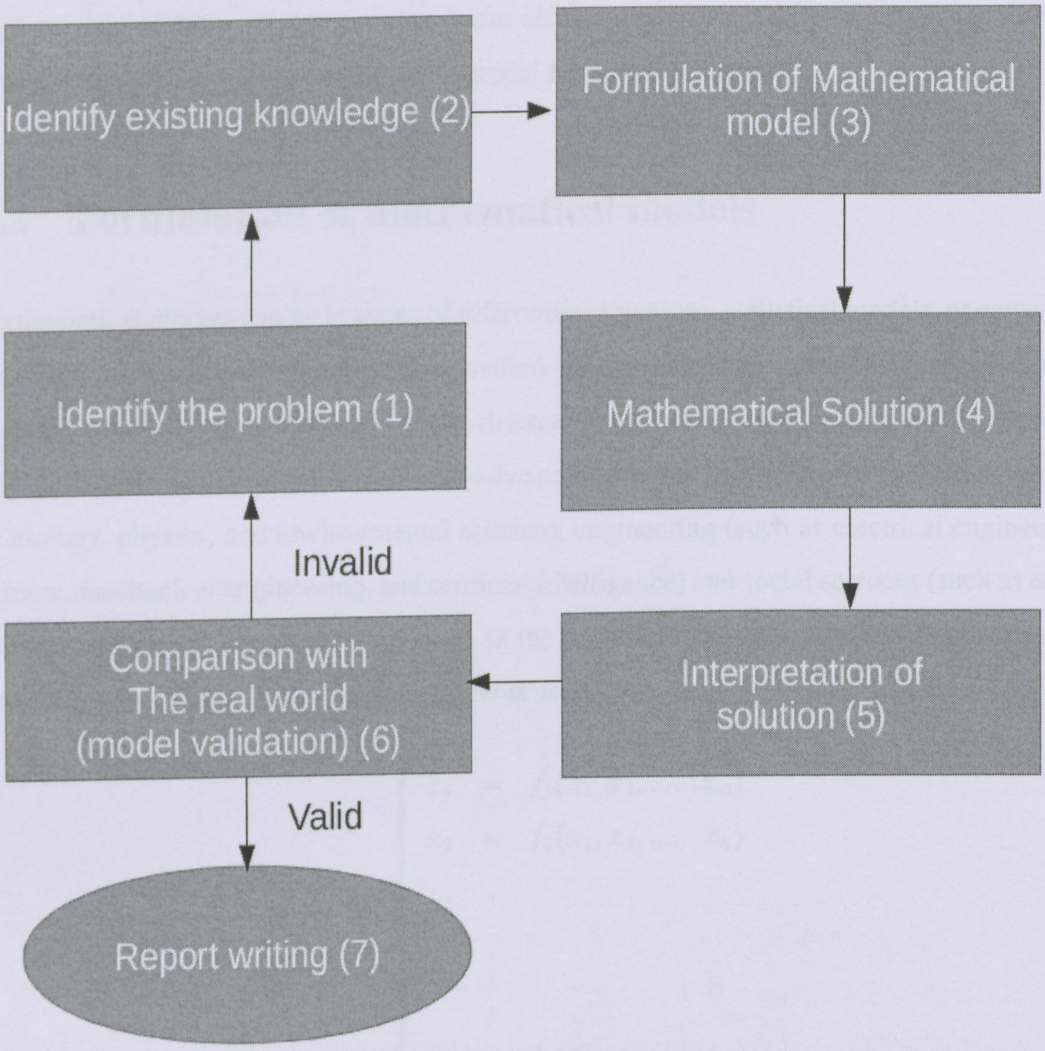

Figure 2.1: *A conceptual diagram for mathematical modelling process.*

The model development process conceptual diagram shown in figure (2.1) illustrate the sequence of events employed when designing a mathematical model. The activities translate a real life problem into mathematics for subsequent analysis of the problems. The first stage is concerned with the identification of the problem, then the relevant literature is reviewed to cover the gap between what has been done and what needs to be done. Sequentially the formulation of the mathematical model is done following literature review. Hence, the mathematical model is solved either analytically or numerically. Thereafter, interpretation of the solution is done before the comparison with the real world situation, at this stage,

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final model validation is done so that it can either be adjusted if invalid, otherwise a report is written. The following section fully explains the model formulation process.

2.2 Formulation of mathematical models

Mathematical models can be in terms of differential equations, statistical models, or game theory models, and many more. Models can also be classified into linear or non-linear; dynamic or statics; deductive, inductive, or floating; explicit or implicit; discrete or continuous; and deterministic or stochastic. Mathematical models have been widely used to solve problems that particularly occurs in natural sciences (such as biology, physics, and environmental science), engineering (such as electrical engineering, computer science, mechanical engineering, and artificial intelligence) and social sciences (such as economics, psychology, sociology, and political science). In the context of ordinary differential equations, mathematical models are represented by dynamical systems as follows:

$$\begin{cases} \dot{x}_1 &= f_1(x_1, x_2, ..., x_n) \\ \dot{x}_2 &= f_2(x_1, x_2, ..., x_n) \\ . \quad & . \\ . \quad & . \\ . \quad & . \\ \dot{x}_n &= f_n(x_1, x_2, ..., x_n) \end{cases} \tag{2.2.1}$$

The above system of equations can be written more concisely in vector notation

$$\dot{\mathbf{x}} = \mathbf{f}(\mathbf{x}) \tag{2.2.2}$$

where **x** and **f** are vectors of $n-$dimension, with components x_i and f_i respectively. Where $i = 1, 2, ..., n$ and is assumed to be smooth enough so that through each point $\bar{x}$ there passes a unique solution of (2.2.1)

In relation to epidemiology, x_i can be any state variable of the model. For instance x_1 can be equated to susceptible class (S), x_2 to infected class (I) and so on, The possible variables for an epidemiological

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model are summarised in Table (2.2)

Description of fundamental compartments of epidemiological models.

Symbol	Names	Explanation
S	Susceptible	These are individuals who are not yet infected, but are susceptible to infection
E	Exposed	Individuals who are infected but unable to infect others
I	Infectious	Infective individuals, they can infect others
P	Pathogens in the environment	infectious pathogen population to susceptible hosts
R	Recovered/Removed	individuals who are recovered from the disease or removed from the population

The choice of the model depends on the characteristics of the particular disease being studied and the main objective of the model (Hethcote, 2000). The table (2.2) defines possible variables that can be used in studying diseases. In this context, various models of combined variables will be explain in relation to force of infection as follows:

- SI models, these are the simplest models in which the transmission of the disease occurs through the interaction of two states, namely susceptible S and infectious I. The assumption is that the route of infection is through direct contact between susceptible individuals and infected individuals and the infective remain infectious for the rest of the disease lifetime. These types of models are mostly applicable to the study of disease that does not confer immunity.
- SIP models are the simplest models in which the transmission of the disease occurs between the susceptible and free-living pathogens in the environment. The force of infection is assumed to be dependent on the population of free-living pathogens.
- SIS model, these models are similar to the SI-models, but the assumption is that infectious confer immunity to recover from the disease and become susceptible again.
- SEI models are the same as the SI-models, however, it considers the exposed or incubation period.

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- $SEIS$ models are the same as to the SIS-models, except that an infected individual has a chance to develop a temporary immunity to recover from the disease and become susceptible again.
- SIR models are similar to the SI models, but an infected individual has a chance to develop a permanent immunity to recover from a disease or removed from the population. In 1927, Kermack and Mckendrick used this type of models to study the spread of Cholera epidemic.
- $SIRS$-models have the same character as that of SIR-models with an added class (S) assuming that an individual who have developed immunity and has a chance to be reinfected again.
- $SEIR$-models are similar to the SIR-models, but the latent or incubation period in which an infected individual cannot transmit the disease before becoming infectious is considered.
- $SEIRS$-models are the same as the $SEIR$ models, except that an individual who has developed immunity has a chance to be reinfected again.

Mathematical models are most useful when directed at a clearly specified and well-defined problem. The nature of the problem to be addressed is likely to determine the most appropriate modelling techniques to use (Van Maanen et al., 2000). After clearly establishing the model objective, the variables are then determined in order to develop the model. We develop a bare bones model to illustrate concepts of epidemics modelling. For brevity, we consider the $Susceptible - Infected - Pathogen$ (SIP) model (2.2.1), in which the total fruit population size N is divided into non-overlapping compartments called susceptible $(S) = x_1$ and infected $(I) = x_2$. Epidemics are a result of the pathogen $(P) = x_3$ introduction into a wholly susceptible population. The model is constructed to demonstrate how to capture the transmission dynamics underlying the interaction between the pathogen and its host within the environment. Emphasis is made on mathematical concepts used in this dissertation

The formulation of the state transition SIP model incorporates features of primary inoculum and free-living pathogen $P(t)$ in the environment. This model consists of a system of three ordinary differential equations which monitors the rate of change of the fruit population and the pathogen population. The total fruit population is denoted by $N(t)$ entails of the susceptible fruit population $S(t)$ and the infected fruit population $I(t)$. The susceptible population is increased by recruitment rate Λ of new fruits, decreased by μ by means of decaying due to natural causes and decreased by λ which happens to be the

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force of infection when the pathogen contact the susceptible class. This force of infection is given by $\lambda = \left(\frac{\beta P}{P_0 + \epsilon P}\right)$ and is assumed to follow the saturation law of infection due to its non-linear nature. The infected host population is simultaneously increased by the same force of infection λ that decrease the susceptible population and it is reduced through disease induced decay characterised by shedding pathogens into the environment at the rate α and natural decay at the rate μ. The pathogen population in the environment is increased by infected fruit population when shedding pathogens into the environment at the rate α and δ denoting a reduction of pathogen population, sometimes known as the diminishing rate of pathogens while β is the transmission rate between the pathogen and susceptible population. The mean length of the infectious period is given by $\frac{1}{\alpha + \mu}$. The Figure (2.2) shows a schematic conceptual model flowchart being used as an example

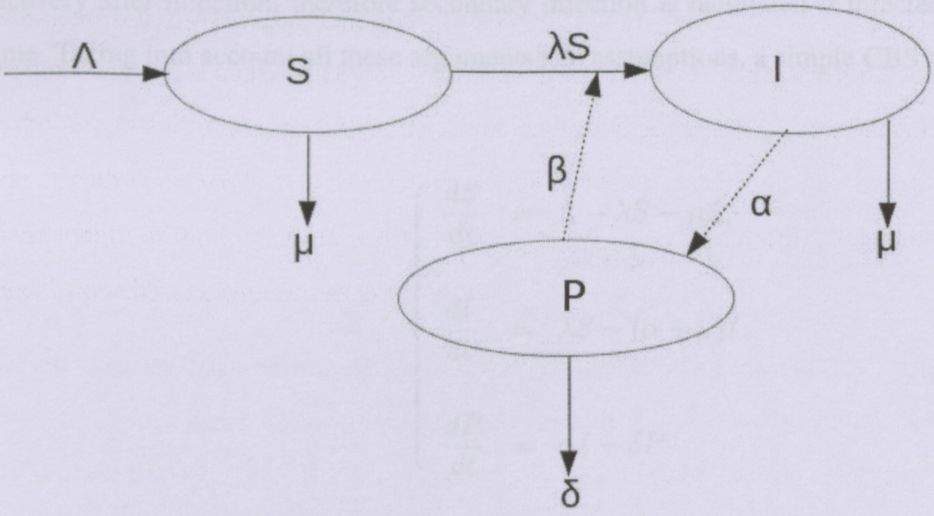

Figure 2.2: *A schematic representation of the general CBS model with pathogens in the environment*

This schematic diagram provides an overview of the transmission dynamics of CBS, which is, then

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further characterised by a set of ordinary differential equations as given in the model (2.2.3). The model state variables are summarised in the table (2.1)

Term	Description
S	Health susceptible fruits
I	Infected fruits
P	pathogen in the environment

Table 2.1: *Description of the variables of the dynamical system in (2.2)*

The population of infected fruits is increased by recruitment of infected plants from outside the orchard as well as infection of susceptible fruits. It is diminished by both natural decay and by disease induced. The population of pathogens is increased by pathogen shed by infected fruits and simultaneously reduced by transmission facilitated through either air current or water. The SIP model indicates that there is no recovery after infection, therefore secondary infection is facilitated if infected trees are not rouged in time. Taking into account all these arguments and assumptions, a simple CBS model is given by

$$\begin{cases} \dfrac{dS}{dt} &= \Lambda - \lambda S - \mu S, \\ \dfrac{dI}{dt} &= \lambda S - (\alpha + \mu) I, \\ \dfrac{dP}{dt} &= \alpha I - \delta P \end{cases} \tag{2.2.3}$$

A non-monotonic saturation incidence rate of the form $\lambda = \left(\dfrac{\beta P}{P_0 + \epsilon P}\right)$ is assumed throughout this dissertation. This represents the inhibition effect of the behavioural change of the susceptible healthy fruits where there is an increase in the number of infective fruits. Where P_0 is the half saturation constant, β is the contact rate of ascospores and susceptible host in the environment, ϵP is the limitation of the growth velocity of primary inoculation with the increase of cases. This model incorporates terms for

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primary infection in the form of functional responses of the pathogen to changes in the host. This model ignores the secondary infection because it's almost insignificant during the first stages of infection.

2.3 Analysis of the model

The aspect of model analysis in the mathematical modelling process is a very crucial step in giving the feasible set of system solutions. Analysing the model involves determining a set of analytical conditions which will ensure the global existence and uniqueness of a solution. Model analysis is done in various ways, we will demonstrate how to analysis a model paying attention to the model stability, equilibrium states, the reproduction number and numerical simulation. To this end, we will be explaining model analysis components with the aid of a detailed epidemiological model (2.2.3). First we define the equilibrium states to show its importance in model analysis.

2.3.1 Equilibrium

Definition 2.3.1. An equilibrium point of a dynamical system generated by an autonomous system of ordinary differential equations (2.2.1) is a solution that does not change with time.

These equilibria are also known as fixed points or steady states and they are characterised by time independent solutions. These equilibria are either disease free (E_0) or endemic (E_e), hence the model system (2.2.3) given as an example, permits the existence of two types of equilibrium states, that is, the disease-free equilibrium and endemic equilibrium. Since the rate of change of the dynamical system is stationary at equilibrium, we equate the left-hand side to zero, when determining equilibrium point. With reference to the general model (2.2.1), the following $n-$ dimensional model is obtained by equating the rate of change to zero, given by

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$$\begin{cases} f_1(\bar{x_1}, ..., \bar{x_n}) = 0 \\ f_2(\bar{x_2}, ..., \bar{x_n}) = 0 \\ . \\ . \\ . \\ f_n(\bar{x_1}, ..., \bar{x_n}) = 0. \end{cases} \tag{2.3.4}$$

and this is a set of values that satisfy the model system (2.3.4) representing the model system state, such that, it remains in the same state for all $t > 0$, in the absence of external perturbation. Now we use the model system (2.2.3) as an example to discuss equilibrium in term of epidemic models. For example for our simple model system (2.2.3) we have

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dP}{dt} = 0$$

This gives the system

$$0 = \Lambda - \lambda S - \mu S, \;\; 0 = \lambda S - (\alpha + \mu) I, \;\; 0 = \alpha I - \delta P$$

which result in at least two equilibrium points: the disease free equilibrium (trivial critical point) given by

$$(S^0, I^0, P^0) = \left(\frac{\Lambda}{\mu}, 0, 0\right)$$

and the endemic equilibrium (non-trivial critical point) given by

$$(S^*, I^*, P^*) = \left(\frac{\Lambda}{\lambda + \mu}, \frac{\lambda\Lambda}{(\lambda + \mu)(\alpha + \mu)}, \frac{\alpha\lambda\Lambda}{\delta(\lambda + \mu)(\alpha + \mu)}\right)$$

Sometimes the disease free equilibrium and endemic equilibrium are denoted by $E_0 = (S^0(t), I^0(t), P^0(t))$ and $E_e = (S^e(t), I^e(t), P^e(t))$ respectively.

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2.3.2 Stability

Stability theory in mathematics is used to addresses the behaviour of solutions of differential equations of dynamical systems under a small perturbation of initial conditions (Arnold, 1973). The effects that these perturbations have on an equilibrium point are important, hence, it is necessary to distinguish between stable and unstable equilibria. For the purpose of a simplified explanation, the determination of stability is illustrated by the use of a two-dimensional model such that the general model system (2.2.1) becomes

$$\begin{cases} \dfrac{dx_1}{dt} = f_1(x_1, x_2), \\ \\ \dfrac{dx_2}{dt} = f_2(x_1, x_2). \end{cases} \tag{2.3.5}$$

if $(x_1(t), x_2(t))$ is a solution of this system for $t \geq 0$ such that $(x_1(0), x_2(0))$ is near an equilibrium $(\bar{x_1}, \bar{x_2})$, this follows that

$$y_1 = x_1(t) - \bar{x_1}, \;\; y_2 = x_2(t) - \bar{x_2} \tag{2.3.6}$$

then

$$\begin{cases} \dfrac{dx_1}{dt} = f_1(x_1 + y_1(t), x_2 + y_2), \\ \\ \dfrac{dx_2}{dt} = f_2(x_1 + y_1(t), x_2 + y_2) \end{cases} \tag{2.3.7}$$

Assuming that the functions f_1 and f_2 are sufficiently differentiable, the expression (2.3.7) can be expanded by Taylor series. $y_1(t)$ and $y_2(t)$ are assumed to be small perturbation from the equilibrium, then disregard the terms of degree two or higher and consider linear part of the equations. Since $(\bar{x_1}, \bar{x_2})$ is an equilibrium state, $f_1(\bar{x_1}, \bar{x_2}) = f_2(\bar{x_1}, \bar{x_2}) = 0$. Therefore, the appropriate linear equations are obtained as follows

$$\begin{cases} \dfrac{dy_1}{dt} = a_{11}y_1 + a_{12}y_2, \\ \\ \dfrac{dy_2}{dt} = a_{21}y_1 + a_{22}y_2. \end{cases} \tag{2.3.8}$$

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where a_{11}, a_{12}, a_{21}, a_{22} are partial derivatives of the function f_i evaluated at equilibrium points $(\bar{x_1}, \bar{x_2})$. The system (2.3.8) can be expressed in the form

$$\frac{d\mathbf{Y}}{dt} = J\mathbf{Y},$$

where $\mathbf{Y} = (y_1, y_2)^T$ and

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial x_1}(\bar{x_1}, \bar{x_2}) & \frac{\partial f_1}{\partial x_2}(\bar{x_1}, \bar{x_2}) \\ \\ \frac{\partial f_2}{\partial x_1}(\bar{x_1}, \bar{x_2}) & \frac{\partial f_2}{\partial x_2}(\bar{x_1}, \bar{x_2}) \end{pmatrix} = \begin{pmatrix} a_{11} & a_{12} \\ \\ a_{21} & a_{22} \end{pmatrix} \tag{2.3.9}$$

is called the Jacobian matrix (J) of the system (2.2.1) evaluated at equilibrium state $(\bar{x_1}, \bar{x_2})$. The behaviour of the solution of the model (2.3.8) is determined by the eigenvalues of J, which are the roots of λ of the equation

$$det(J - \lambda I) = 0 \tag{2.3.10}$$

Every solution of (2.3.7) has the property that $y_1(t) \longrightarrow 0$ and $y_2 \longrightarrow 0$ as $t \longrightarrow +\infty$ if all the roots of (2.3.10) are negative or are complex with negative real parts. In this case, it can be proved that any solution of (2.2.1) with initial value sufficiently close to $(\bar{x_1}, \bar{x_2})$ will tend to $(\bar{x_1}, \bar{x_2})$ as $t \longrightarrow +\infty$. Then $(\bar{x_1}, \bar{x_2})$ is said to be a locally asymptotically stable equilibrium of the model (2.2.1). If one or both eigenvalues are positive or have positive real parts, then most solutions becomes unboundedly large as $t \longrightarrow +\infty$ and consequently most solution of (2.2.1) do not remain near $(\bar{x_1}, \bar{x_2})$, even if they start very close and in this case, $(\bar{x_1}, \bar{x_2})$ is said to unstable.

Now we explain the general stability process given above using the model system (2.2.3) in the same manner in which it was applied on the general model (2.2.1), then expand by Taylor series such that $S(t)$, $I(t)$ and $P(t)$ are assumed to be small perturbation from the equilibrium, then the terms with second order or higher are disregarded while considering linear part of the equations. If $S(t), I(t), P(t)$ is a solution of the model (2.2.3) for $t \geqslant 0$ such that $(S(0), I(0), P(0))$ is near an equilibrium $(\bar{S}, \bar{I}, \bar{P})$, Taking partial derivatives of the set of ordinary differential equations (2.3.1), the system can be expressed

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in the form

$$J = \begin{pmatrix} \frac{\beta P}{P_0 + \epsilon P} - \mu & 0 & \frac{(P_0 + \epsilon P)\beta S - \beta P S \epsilon}{(P_0 + \epsilon P)^2} \\ \frac{\beta P}{P_0 + \epsilon P} & -(\alpha + \mu) & \frac{(P_0 + \epsilon P)\beta S - \beta P S \epsilon}{(P_0 + \epsilon P)^2} \\ 0 & \alpha & -\delta \end{pmatrix} \tag{2.3.11}$$

which is the Jacobian matrix of the model (2.2.3), then evaluating the Jacobian matrix at disease free equilibrium given by $E_0 = (S^0, I^0, P^0) = \left(\frac{\Lambda}{\mu}, 0, 0\right)$, then we have

$$J(E_0) = \begin{pmatrix} -\mu & 0 & \frac{\beta\Lambda}{\mu P_0} \\ 0 & -(\alpha + \mu) & \frac{\beta\Lambda}{\mu P_0} \\ 0 & \alpha & -\delta \end{pmatrix} \tag{2.3.12}$$

The behaviour of solution of the model (2.2.3) is determined by the eigenvalues of J, which are roots of the characteristics equation $det(J - \lambda I)$, where

$$det(J - \lambda I) = [(-a - \lambda)(-b - \lambda)(-f - \lambda)] + [(a - \lambda)(ce)] \tag{2.3.13}$$

$$det(J - \lambda I) = (-a - \lambda)[\lambda^2 + \lambda(b + f) + bf + ce] = 0 \tag{2.3.14}$$

where

$a = -\mu$, $b = -(\alpha + \mu)$, $c = \alpha$, $d = \frac{\beta\Lambda}{\mu P_0}$, $e = \frac{\beta\Lambda}{\mu P_0}$, $f = -\delta$

Therefore the eigenvalues corresponding to the matrix above are as follows:

$\lambda_1 = -\mu$, $\lambda_2 = -(\alpha + \mu)$, $\lambda_3 = -\delta$

Therefore, E_0 is a locally asymptotically stable equilibrium since all the eigenvalues are negative and

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have negative real parts.

2.3.3 Reproduction number R_0

This is a non-dimensional quantity which can be used to discuss model system stability. One can show that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$ and unstable whenever $R_0 > 1$. After demonstrating how to obtain the equilibria, it is essential to illustrate how to compute the model threshold parameter termed the reproduction number (R_0). This is arguably the most powerful tool developed for analysing and interpreting epidemic models. Theoretically, R_0 is defined as the average number of secondary cases generated by one primary inoculum introduced in an entirely susceptible population over a disease infectious period (Castillo-Chávez et al., 2002). The next generation operator approach computes R_0 through the study of eigenvalues of the Jacobian matrix evaluated at the disease-free equilibrium (Castillo-Chávez et al., 2002). A different approach of the next generation operator was developed by (Diekmann et al., 1990) which define R_0 as the spectral radius of the next generation operator. In this section, we are going to follow the same approach. This approach is considered when we define discrete non-homogeneous groups with respect to certain fixed characteristics, that is, in epidemiology, models can be re-arranged in the form

$$\begin{cases} \dfrac{dX}{dt} = f(X,Y,Z), \\ \dfrac{dY}{dt} = g(X,Y,Z), \\ \dfrac{dZ}{dt} = h(X,Y,Z) \end{cases} \tag{2.3.15}$$

Where $X \in \mathbb{R}^r$, $Y \in \mathbb{R}^s$, $Z \in \mathbb{R}^n$, $r,s,n \geq 0$ and $h(X,0,0) = 0$. The components of X denote the number of susceptible and other state variables of non-infected individuals. The components of Y represent the number of infected individuals who are not infectious. The components of Z represent the number of infected and infectious individuals (Castillo-Chávez et al., 2002). Let $U_0 = (X^*,0,0) \in \mathbb{R}^{r+s+n}$ denote the disease free equilibrium, that is, at $U_0 = (X^*,0,0)$, $f(X^*,0,0) = g(X^*,0,0) =$

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$h(X^*, 0, 0) = 0$. Assume that the equilibrium $g(X^*, Y, Z) = 0$ implicitly determines a function $Y = \tilde{g}(X^*, Z)$. Let $A = D_Z h(X^*, \tilde{g}(X^*, 0), 0)$ and further assume that A can be written in the form $A = M - D$, with $M \geq 0$ and $D > 0$, a diagonal matrix. The spectral bound of matrix B is denoted by $m(B) = max\{\mathbb{R}\lambda : \lambda \in \sigma(B)\}$, where $\mathbb{R}\lambda$ means the real part of λ, while $\rho(B) = \lim_{n \longrightarrow \infty} ||B^n||^{\frac{1}{n}}$ denotes the spectral radius of B. The proof of the following theorem involving matrix A is found in (Diekmann et al., 1990): Either

$$m(A) \Longleftrightarrow \rho(MD^{-1}) < 1$$

or

$$m(A) > 0 \Longleftrightarrow \rho(MD^{-1}) > 1$$

The basic reproduction number is defined as the spectral radius (dominant eigenvalue) of the matrix MD^{-1}, that is,

$$R_0 = \rho(MD^{-1})$$

As an example, we use the model system (2.2.3) to compute R_0. First, we consider the model system (2.2.3) defined by fixed characteristics (Castillo-Chávez et al., 2002), that can be re-arranged in the form

$$\begin{cases} \dfrac{dX}{dt} = f(X, Y, Z), \\ \dfrac{dY}{dt} = g(X, Y, Z), \\ \dfrac{dZ}{dt} = h(X, Y, Z) \end{cases} \tag{2.3.16}$$

Where

$X = (S)$ represents the group of non infected host

$Y = (I)$ denotes the infected but not infectious individuals

$Z = (P)$ infectious. Then

$$X \in \mathbb{R}^r, \ Y \in \mathbb{R}^s, \ Z \in \mathbb{R}^n, r, s, n \geq 0$$

and

$$h(X, 0, 0) = 0$$

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Let

$$U_0 = (X, 0, 0) \in \mathbb{R}^{r+s+n}$$

denote the disease free equilibrium state

$$U_0 = (X^*, 0, 0),\ f(X^*, 0, 0) = g(X^*, 0, 0) = h(X^*, 0, 0) = 0 \tag{2.3.17}$$

and assume that

$$\tilde{g}(X^*, Z) = \frac{\alpha\beta X Z}{(\alpha+\mu)(P_0+\epsilon Z) - \delta Z} \tag{2.3.18}$$

We further assume that A matrix

$$A = D_z h(X^*, \tilde{g}(X^*, 0), 0) = \left[\frac{\alpha\beta\Lambda}{\mu(\alpha+\mu)P_0} - \delta\right] \tag{2.3.19}$$

can be presented in the form $A = M - D$ with $M \geq 0$ and $D > 0$, a diagonal matrix, where in this case M is a single entry matrix as follows:

$$M = \frac{\alpha\beta\Lambda}{\mu(\alpha+\mu)P_0} \tag{2.3.20}$$

and

$$D = \delta \tag{2.3.21}$$

The reproduction number is the spectral radius of the matrix $T = MD^{-1}$, given by

$$R_0 = \rho(T)$$

In this case, the reproduction number of the model (2.2.3) is expressed by the following quantity,

$$R_0 = \frac{\alpha\beta\Lambda}{\mu(\alpha+\mu)P_0\delta} \tag{2.3.22}$$

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2.3.4 Numerical methods

There are two techniques that can be used to solve the dynamical systems, that is, analytical and numerical. Analytical techniques refers to the use of calculus and other mathematics techniques to compute the model solution for the purpose of the model behaviour under any circumstances. These analytical methods are exact, for instance, integration, differential and so on. But this seems to work only for simple models. For more complex models, the analysis becomes more complicated, hence the use of numerical methods of solving equations, for instance, the Runge-Kutta method. The numerical methods start with initial values of the variables and then employs the equations to figure out the changes in these variables over a very brief period of time. This numerical method is only an approximation. Numerical methods for differential equations are tools used to find numerical approximations to the dynamic system solution by means of techniques such as plots. Complex dynamic systems cannot be solved analytical therefore the use of a numerical approximation to the solution is often sufficient. Some numerical methods commonly used include linear multistep methods or Runge-Kutta. These methods can be divided into explicit and implicit. For instance, implicit linear multi-step methods include Adams-Moulton methods, and backwards differentiation methods, including Runge-Kutta among others. Explicity methods include, but not limited to, Adam-Bashforth methods (Simo, 1998). However, these methods can be demonstrated by means of plots among other techniques

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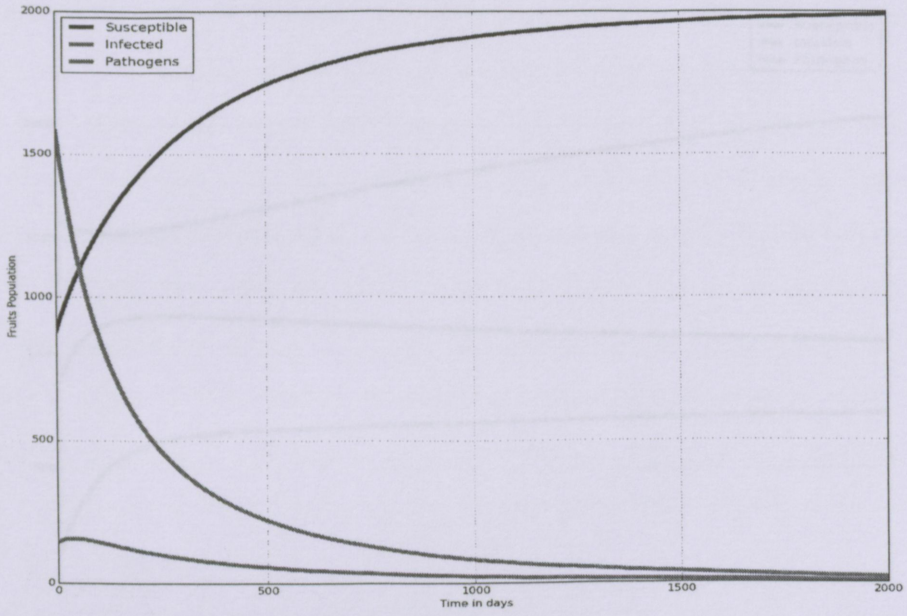

Figure 2.3: *Simulation of model (2.2.3) showing the evolution with time of Guignardia citricarpa whenever* $R_0 < 1$

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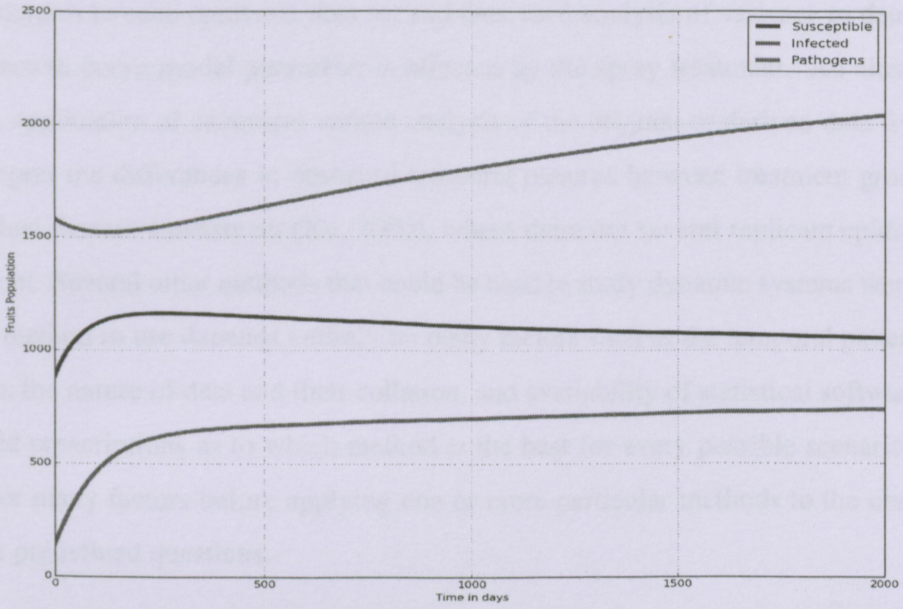

Figure 2.4: *Simulation of model (2.2.3) showing the evolution with time of Guignardia citricarpa whenever* $R_0 > 1$

2.4 Modelling in Agriculture.

Modelling in agriculture plays an important role in various aspects in as far as crop management and their marketing are concerned and the application of models in this field is rapidly growing. A lot of models have been developed in agriculture in the effort to describe the mechanisms underlying various aspects of agricultural systems. The first models of the temporal development of epidemics were developed by (Vanderplank, 1963) where he was describing the quantitative analysis of epidemiological data and also a new way of conceptualising disease increase in populations. His studies contributed a lot to the mainstream of plant disease epidemiology, where he introduced several quantities such as apparent infection rate, infectious and latent periods, demonstrated how differential equations could be used as the basis for quantifying disease progress in plants. Finally, showed the use of model parameter estimates in comparing treatment effects on disease development. (Xu, 2006) showed how temporal dynamics are affected by spraying fungicide, by examining and summarising the pattern of disease progress over time

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for each individual spray programme and compare the patterns. This was done by fitting the growth curve models to each epidemic data set and then used analysis of variance to determine which and how each growth curve model parameter is affected by the spray treatment. An alternative was suggested, that is, application of canonical variate analysis of the original or derived data from which can be used to interpret the differences in observed temporal patterns between treatment groups in relation to each individual disease assessment (Xu, 2006), where there are several replicate epidemic data sets for each treatment. Several other methods that could be used to study dynamic systems were discussed. Precisely, which method to use depends entirely on many factors such as the temporal pattern of the observed epidemics, the nature of data and their collation, and availability of statistical software packages. There are no fixed prescriptions as to which method is the best for every possible scenario. Researchers need to consider many factors before applying one or more particular methods to the observed data in order to answer predefined questions.

Mathematical models have been used as an alternative where data is limited and where experiments are not possible to conduct. But, both modelling and experiments can be done to confirm each others results. (Smith et al., 1988) brought to light that both theoretical and experimental studies revealed that the rates of disease increase were considerably influenced by the number of initial foci. They did an experiment with the southern blight of processing carrot, the rate of disease increase generally increased as the number of initial foci increased. (Madden and Campbell, 1990) extensively reviewed the application and interpretation of simple growth models used to describe temporal disease progress, that is, the monomolecular, Gompertz and logistics models. The objective was to construct disease progress curve shapes and to describe the observed patterns and then use the estimated model parameters for comparing epidemics. (Thornley et al., 2007) established that mathematical models were constructed to improve researchers' understanding and explain agricultural processes, his interest was based on the disciplines of agriculture, such as animal science, plant science and ecology. Furthermore, he showed that the use of applied mathematics can be a tool diagram for improving the accuracy of agricultural research. A lot of scholars have studied and established that the mathematical modelling of agricultural systems and the design and analyses of experimental data and experimental tests has been done to improve understanding of the factors which have an influence on agriculture in general. In decision making and marketing, it was

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shown that its essential to use mathematical modelling as a tool to provide necessary adequate information for proper decision making. (Basanta et al., 2003) established that as agriculture is becoming larger in terms of production activities, the control level of the environment in which the plants grow is also increasing. This control is said to be varying from good agricultural practise to better management of environments. A model of potato early blight epidemics caused by *Alternaria solani* was developed by (Waggoner and Horsfall, 1969) to investigate its transmission dynamics. In (Van Maanen and Xu, 2003) outlined common methodologies that are used to quantify and model spatiotemporal dynamics of plant diseases, with emphasis on developing temporal forecast models and on quantifying spatial patterns. They also established epidemiological models in cereal crops, incorporating the spores, each potentially capable of infection, mycelia and cleistothecia. They found out that passage from one stage to the next can be very fast depending on the environmental condition. The inoculum was divided into primary and secondary, this was found to be very useful when modelling soil-borne pathogens. (Van Maanen et al., 2000) showed the importance of the life cycle stages in the control of fungal dynamics by adopting a patch occupancy model which was constructed by (Caswell and Cohen, 1993), this approach will provide the basis for studying the transmission dynamics of CBS.

2.5 Modelling fresh produce

There are a number of problems solved mathematically, associated with fresh produce, these range from pre-harvesting, harvesting and post-harvest. Most of these issues are characterised by contamination and growth of the pathogen in the pre-harvesting stage along harvesting up until post-harvest. Thus, this is where a lot of plant diseases are developed. (Gilligan, 1985) derived theoretical binomial models for the infection of the hosts by soil-borne fungi and emphasis was given to the phenomena effects of the probability of an infection considering the spatial pattern, displacement of soil by the growth of the host.

During harvest, contamination is likely to occur to due the nature of the activities involved. Under normal circumstances secondary infection occurs during this practise when the infected contact the uninfected. There is not much that has been done on modelling fresh produce during harvest.

Post-harvest stage seems to have a little research and model constructed ranging from prevention to treatment. A time series modelling was done by applying Auto Regression Integrated Moving Average

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(ARIMA) technique to forecast the demand for fresh produce at a disaggregate level (Shukla, 2013). Another interesting aspect which led to modelling is life shelf of a product. Recently (Macedo et al., 2013) studied in modified atmosphere packaging (MAP) in order to extend the shelf life of the fresh produce. They did this by integrating the mathematical modelling depicting product respiration rate and package permeability to simplify the packaging design process. The models developed for fresh produce used temperature and relative humidity data measured throughout the cold chain and quality parameters measured at specific steps needed to be developed to predict the produce quality that can be expected subsequently based on the environmental conditions. Further information was established showing that the development of such models, requires data on physiological and microbial quality changes and on disease development in a wide variety of fresh fruits and vegetables as a function of time, temperature and relative humidity. Unfortunately, the availability of this type of data was relatively limited. Another encounter shows (Russell et al., 2009) demonstrating that the challenges in developing such models were defining the parameters that best describe the overall quality of fresh produce, given that the ideal parameters probably vary considerably depending on the type of produce. Associated with this was the challenge of finding the best tool for measuring the various aspects of horticultural produce quality at any point along the field-to-fork continuum, such systems could be used by growers to identify the optimum market for a particular load of fresh produce. There is a wide range of fresh produce which can be modelled in various ways, but the focus of this thesis is in citrus produce. The next section will discuss modelling in citrus produce.

2.6 Modelling citrus in general

Citrus production comes with its own issues from the pre-harvest stages to post-harvest. This may span from climate conditions, the general environment and even the farmers' activities. The best scenario is to maximise production from the activities undergone during production. A lot of problems arose some of which required mathematical interventions. (Goldschmidt and Monselise, 1977) laid the foundations of the Citrus productivity model to identify and quantify productivity problems, predict yields and devise means for optimisation of Citrus crop production (Bustan et al., 1998). In disease transmission (Chiyaka et al., 2012) developed a mathematical model of the transmission of citrus greening between its psyllid

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vector and citrus host to characterise the dynamics of the vector and disease development, focusing on the spread of the pathogen from flush to flush within a tree. In their model analysis, it was indicated that vector activity is essential for primary infection, but is not necessary for secondary infection because the infection can occur from flush to flush through internal movement in the tree. The vector life cycle was established using systems of non-linear equations and the host population was also modelled using deterministic models. Flush production, within-tree spread, and latent period were discovered to be the most important parameters influencing citrus greening development. (Jacobsen et al., 2013) developed a model of population dynamics for a grove in the presence of citrus greening disease. The model incorporates a control strategy of rouging and replanting infected trees and the parameters were just like what (Chiyaka et al., 2012) used. This section gives a brief background on how to account for some features such as the force of infection, incident law of citrus fungal transmission dynamics.

2.7 Modelling citrus fungal disease

Modelling of citrus fungal disease does not seem to date earlier that 2008. But, plant disease seems to have started a while ago. (Magarey et al., 2005) tried to solve a generic infection for foliar plant pathogens by developing a model that will predict infection periods for pathogens with no extensive epidemiological data. However, a number of researchers in this field expressed some dynamical systems in a different modelling style, such as (Strauss, 2005) who developed a farm-level model which followed a Richardson's approach in decision making. (Khanchouch et al., 2012) proposed a new mathematical model to conduct a rapid and efficient resistance screen test in the case of a causal agent of Mal Secco, *Phoma tracheiphila,* responsible for many important losses in the Citrus crop worldwide. The reaction of many citrus species and cultivars towards the toxin of the parasite and the radiation treatments was investigated to develop new approaches to selection of Citrus lemon resistant cultivars. The approach used in this paper was the polynomial interpolation method and least square approximation. Data was collected and adequate types of regression curves were for fitting curves of the infected cultivar. In (Beresford et al., 2004), developed a new model for assessing variation of a fungal plant disease called apple scab which is caused by *Venturia inaequalis*. The model entailed three components, ascospores

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availability, wetness based infection risk and susceptible leaf area. The model predicted ascospores realise, risk incidence rate. The model was intended to assist in fungicide selection and timing for apple plant diseases. (Meentemeyer et al., 2011), modelled Bahia bark scaling citrus for the cost-effective control of plant disease when epidemiological knowledge is incomplete. In other words, they considered how mathematical models can be used to inform the control of plant disease, even when the identity and biology of the pathogen are not well understood. Analysis of data from an experimental plot concerning the spread of Bahia bark scaling of citrus, an economically-important disease in north-eastern Brazil, by fitting a mathematical model, which also accounts for uncertainty, to disease spread was carried out. The model captures the epidemiological features of the disease, revealing that transmission is localised and that disease spreads gradually slowly. They used a Markov chain Monte Carlo with data augmentation to fit a spatially explicit, stochastic, compartmental $SEIR$ model to represent Bahia bark scaling citrus dynamics at the scale of a grapefruit grove and have estimated the number of key epidemiological parameters. The relevant literature on citrus fungal disease gives the basis on how to quantify fungal pathogen transmission and some parameters, and this will help when developing both epidemiological and within-host models.

2.8 Modelling Citrus Black Spot

The complex dynamic systems of the citrus produce come with various strains, that take time to be identified. A lot of models have been developed to solve various problems associated with CBS. Two Gompertz models were developed to predict the onset of ascospores release and the dynamic of ascospores release in climatically diverse regions (Fourie et al., 2013). Recently (Yonow et al., 2013) developed a CLIMEX model to predict whether European climatic condition is suitable for CBS to establish, however, the models clearly showed that it's not suitable. (Paul, 2006) modelled the distribution of CBS, statistical functions that estimate the probability of the occurrence of a taxon within a climatic space, an approach that has been widely used to predict species distributions. A different approach to modelling was adopted to predict the regions with suitable climatic condition conducive for CBS to develop, however, the focus was on South Africa and Europe but other parts of the world were also considered. The climex and response surface modelling were adopted. This model was actually showing the distribution of CBS

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worldwide. The SI technique has been used in the modelling of citrus fungal disease by (Hattaf et al., 2013). Mathematical modelling in CBS has received little or no attention. In this thesis, a multi-scale model framework to investigate the transmission dynamics of Citrus Black Spot caused by *Guignardia citricarpa* is established to quantify features responsible for the invasion and persistence. Most of the model reviewed had one thing in common, they were all focusing on between-host models only.

2.9 Conclusion

Complex models are hard to understand and, as a result, the intended beneficiaries will not benefit and, on the other hand, a simple model is easy to handle mathematically and to understand and follow but, unfortunately, may not be realistic. Therefore, there is a need to strike a balance between real life complex dynamic systems and simple mathematical models. This chapter was all about introducing theoretical mathematical concepts used in disease modelling, by means an example. The literature was thoroughly reviewed on models used in agriculture, then fresh produce narrowing down to citrus in general and further to citrus fungal disease and final the CBS. Mathematical models have been developed in various fields to serve different purposes, but, however, all these models are classified in several ways, including but not limited to, prediction, description and conceptual, according to their main objective. An SIP model constructed to determine the disease threshold was used as an example to explain the mathematical concepts used in this dissertation. There are other methods that have been developed to solve similar problems, but requiring data that is more intensive than the one outlined above.

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Chapter 3

EPIDEMIOLOGICAL MODEL FOR CITRUS BLACK SPOT DYNAMICS BETWEEN FRUITS

3.1 Introduction

Citrus Black Spot (CBS), caused by *Guignardia citricarpa* Kiely, pose a threat to the citrus industry. The disease was first discovered in Australia by Benson in 1895 and has since spread throughout many of the citrus cultivation areas in South Africa and other areas around the world (Kotzé, 2000). Plants infected by CBS usually drop fruits early before they fully mature, reducing plant yields and rendering highly blemished fruits which are not marketable even in domestic markets (Paul, 2006). The presence of the susceptible citrus trees, conducive environment for disease development and virulence pathogens promotes the development of CBS. Literature has shown that CBS invades and persist in subtropical regions with summer rainfall (Hattingh, 2009). CBS is of great importance due to its impact economically or phytosanitary trade, socially and to citrus farmers. The existence of this pathogen has arisen concerns in the global citrus orchards including South Africa.

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CBS importance has shown several attempts made by researchers to improve intervention strategies. Unfortunately, it has been a difficult practise because of its latency period and other aspects which were not yet fully understood. An SIP model is constructed to capture the mechanism underlying the interaction between the pathogen and its dynamics in the definitive fruits population within the environment. After overwintering in leaf litter, ascomata begin producing ascospores. Ascospores formation is catalysed by sequential wetting and drying of the leaf litter (Truter et al., 2004) and very wet conditions inhibit ascospores development due to leaf decomposition and competition from other fungi and micro-organism (Lee et al., 1973). The ascospores are ejected from the fruiting bodies of the fungus during rainfall or irrigation and then dispersed by wind and water (Kotzé, 1981). Upon landing on susceptible tissue, the ascospores germinate and form appressoria. Different tissues are susceptible to infection at different times. Leaves are susceptible up to 10 months of age. Fruits are susceptible during the 4–5 months after fruit-set (Kotzé, 2000) though trees less than ten years old are only susceptible up to 3 months (Paul, 2006). Soon after, the infection peg along with mycelia colonise the area between the cuticle and epidermal wall. The symptoms are not visible right away due to the fact that the infection remains latent until the fruit is mature (Meentemeyer et al., 2011).

3.2 Model formulation

The biological background information about CBS is now used to formulate the model. This section describes the epidemiological deterministic compartmental model for CBS transmission dynamics by formulating the simple state transition with the use of ordinary differential equation system for the host and pathogen population. The entire dynamical system underlying the transmission mechanism of the disease development is summarised by the Figure (3.1), in which all possible environments are presented.

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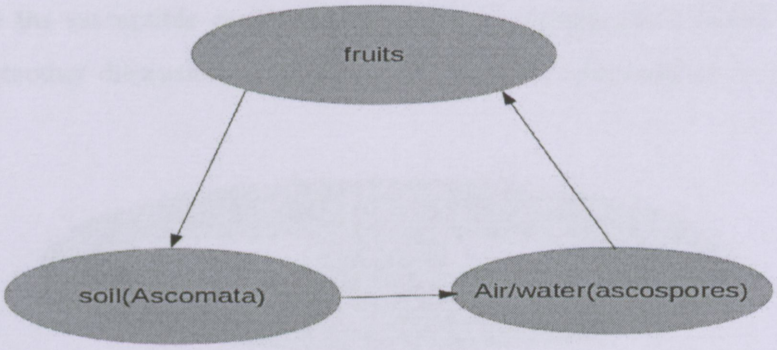

Figure 3.1: *The possible environments of where Guignardia citricarpa evolves with time*

3.2.1 Fruit population

The fruits population N is divided into two subgroups with respect to CBS infection status: susceptible S, Infected and infectious I. It follows that the total host population is given by

$$N(t) = S(t) + I(t) \tag{3.2.1}$$

3.2.2 Pathogen population

The pathogen population P is also divided into two compartments according to their nature: Ascomata P_0 and Ascospores P_A. The total pathogen population is determined by

$$P(t) = P_0(t) + P_A(t). \tag{3.2.2}$$

Ascomata (the fruiting body of ascospores) is found in the dead leaves or fruits on the orchard floor and it is not infectious to susceptible fruits, but it ejects the Ascospores, which are infectious once they come in contact with newly developing fruits or leaf's surface. Spores germinate in fruits to produce appressoria which in turn grows inside the fruit towards the outer layer during the latent phase. Upon maturity the appressoria shoot out of the fruits creating symptoms, thereby producing pycnidia which then releases conidia. Systematically, conidia become the secondary source of infection. When individual fruits are

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infected, they leave the susceptible compartment forming or joining the infected compartment. The figure (3.2) shows another dimension in which we can view the environments in which the pathogen evolves with time.

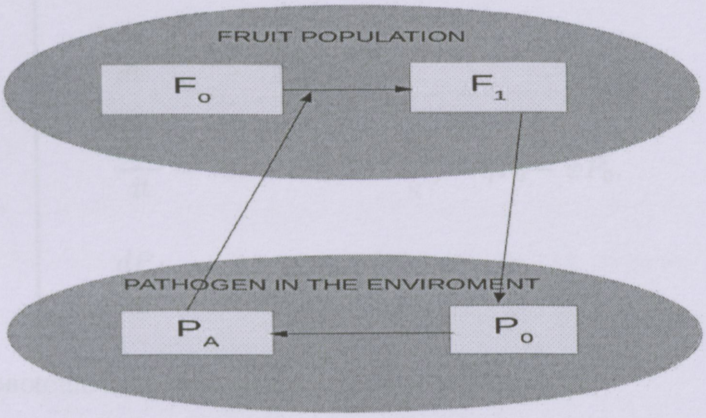

Figure 3.2: *Conceptual diagram of pathogen and fruits.*

Pathogens are acting on independent capacity, meaning pathogen inoculation has no influence on the other pathogen's occurrence to cause infection. However, not all pathogens are able to survive in the environment, some transform into a new stage and some diminish. ψP_0 denote diminishing rate of ascomata while ψP_A denotes the diminishing of ascospores in the air, due to disperse. The pathogen growth is given by a logistic function, where P_E is the pathogen in the environment, λ is the force of infection. κ is the carrying capacity. η is the transformation rate from ascomata to ascospores. α is the decay rate due to disease. An average lifespan of infected fruits is

$$\frac{1}{\alpha + \mu} \tag{3.2.3}$$

Below is the conceptual model which demonstrates the transition of the fruits population in relation to disease density. Contamination made by farm workers and machinery is considered as part of the

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environment to simplify the model

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \lambda S - \mu S, \\ \dfrac{dI}{dt} = \lambda S - (\alpha + \mu) I, \\ \dfrac{dP_0}{dt} = \alpha I + \delta P_0 (1 - \dfrac{P_0}{\kappa}) - \eta P_0 - \psi P_0, \\ \dfrac{dP_A}{dt} = \eta P_0 - \psi P_A. \end{cases} \tag{3.2.4}$$

Considering a non-monotonic saturation incidence rate of the form

$$\lambda = \frac{\beta P_A(t)}{\rho + \epsilon P_A(t)}. \tag{3.2.5}$$

This represents the inhibition effect of the behavioural change of the non-infected fruits where there is an increase in the number of infective fruits. Where ρ is the half saturation constant, β is the contact rate with ascospores in the environment, ϵ is the limitation of the growth velocity of ascomata with the increase of invasion cases. This model incorporates terms for primary inoculum in the form of functional responses of the pathogen to changes in the host. The model state variables are summarized in the table below.

State variable	Description	Initial values
$S(t)$	health susceptible fruits	500000
$I(t)$	infected fruits	0
$P_0(t)$	Ascomata	0
$P_A(t)$	Ascospores	0

Table 3.1: *Description of the state variables of the model system (3.2.4)*

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3.3 Basic properties

In this section analysis of the basic properties of the system of differential equations is done, so that biological aspects are not violated.

3.3.1 Positivity of solutions

The model system (3.2.4) monitors the dynamics of the fruit and pathogen population, thus, we should show that the state variables are non-negative at all time. In other words, there is need to prove that all solutions of the system with positive initial condition will remain positive for all t ¿ 0.

Theorem 3.1. *Let the initial condition be* $S = S^0(0) \geq 0,\ I(0) = I^0(0) \geq 0,\ P_0(0) = P_0^0 \geq 0,\ P_A(0) = P_A^0(0) > 0 \in \Omega$. *Then, the solution of* $S(t), I(t), P_0(t), P_A(t)$ *of the model system (3.2.4) are positive* $\forall\ t > 0$.

Proof. Assume that there exists some time $t_0 = 0$ such that $I(t_0) = 0$. We define

$$\kappa = minimum_{t_0 \leq t \leq t_1}\{\lambda S - (\alpha + \mu)I\}. \tag{3.3.6}$$

Then, $\forall\ t \in [t_0; t_1],\ \ I'(t) \geq \kappa I(t)$. Solving for $I(t)$ by freshman integration we obtain $I(t_0)e^{\kappa t}$. For $t = t_1$, result in $I = I(0)e^{\kappa t_1} > 0$, which is a contradiction. Thus, $I(t) > 0 \quad \forall \quad t > 0$. A similar argument can be used to derive the remaining variables. This shows that the solution of the model system (3.2.4) is such that $min\{S(t), I(t), P_0(t), P_A\} \geq 0$ in its interval of existence Ω (Tchuenche and Chiyaka, 2009). If the initial conditions are positive, then the solution of the system remain non-negative for all $t > 0$ □

3.3.2 Feasible region of the model.

The model system (3.2.4) is capturing monitoring pathogen and fruit population dynamics, therefore, we assume that all parameters of the model are positive. The model system (3.2.4) is analysed in a feasible region as shown below. We first showed that the model system (3.2.4) is bounded in a proper subset of $\Omega \subset \mathbb{R}_+^2 \times \mathbb{R}_+^2$. Letting

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$$S(t), I(t) \in \mathbb{R}^2$$
$$P_0(t), P_A(t) \in \mathbb{R}^2$$

be any solution of the model system (3.2.4), Combining $S(t)$ and $I(t)$ will obtain $N(t)$ then differentiate with respect to time.

$$\begin{cases} \dfrac{dN(t)}{dt} = \dfrac{dS(t)}{dt} + \dfrac{dI(t)}{dt} \\ \dfrac{dN(t)}{dt} = \Lambda - \lambda S - \mu S + \lambda S - (\alpha + \mu)I, \\ \dfrac{dN(t)}{dt} = \Lambda - \mu S - (\alpha + \mu)I. \end{cases} \tag{3.3.7}$$

grouping like terms after operation manipulation

$$\begin{cases} \dfrac{dN(t)}{dt} = \Lambda - \mu(S(t) + I(t)) - \alpha I(t), \\ \dfrac{dN(t)}{dt} = \Lambda - \mu N(t) - \alpha I(t), \\ \dfrac{dN(t)}{dt} \leq \Lambda - \mu N(t) \end{cases} \tag{3.3.8}$$

This implies that

$$\lim_{t \to \infty} sup(N(t)) \leqslant \frac{\Lambda}{\mu} \tag{3.3.9}$$

Similar expression can be derived for the pathogen population using the approach used (in 3.3.9). Therefore, all feasible solutions of the model system (3.2.4) are positive and eventually enter the invariant region

$$\Omega = \left((S, I, P_0, P_A) : 0 \leqslant S + I \leqslant \frac{\Lambda}{\mu}, \ 0 \leqslant P_0 + P_A \leqslant \frac{\Lambda_P}{\psi} \right)$$

where Λ_P is the pathogen recruiment rate. This implies that whenever $\Lambda > \mu$, Ω is positively invariant and attracting region, since all solutions that will start in Ω will remain in $\Omega \ \ \forall \ t \geq 0$. Thus, model

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system (3.2.4) is mathematically and epidemiologically well-posed (Hethcote, 2000) (Garira et al., 2014). Therefore, it is sufficient to consider solutions of the model system (3.2.4) in Ω

3.4 Determination of disease-free equilibrium (DFE) of the model (3.2.4) and stability.

3.4.1 Disease free equilibrium (E_0)

The disease-free equilibrium point (E_0) of the model system (3.2.4) is characterised by the absence of infection, that is, $I = 0$ and it is obtained by equating the rate of change of the model system to zero and solve the remaining function of the state variables. This is a time-independent solution given as

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dP_0}{dt} = \frac{dP_A}{dt} = 0, \tag{3.4.10}$$

such that

$$\begin{cases} 0 = \Lambda - \lambda S - \mu S, \\ 0 = \lambda S - (\alpha + \mu) I, \\ 0 = \alpha I + \delta P_0 (1 - \frac{P_0}{\kappa}) - \eta P_0 - \psi P_0, \\ 0 = \eta P_0 - \psi P_A. \end{cases} \tag{3.4.11}$$

Therefore

$$E_0 = (S(0), I(0), P_0(0), P_A(0)) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right) \tag{3.4.12}$$

In simple terms, the susceptible and possible the ascomata are the only non-zero classes during the absence of the disease.

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3.5 Reproduction number of the dynamical system of CBS

This section deals with the threshold parameter defined as the average number of new invasion generated by one primary infection introduced into an entirely susceptible fruits population over the disease infectious period (Driessche and Watmough, 2002). This is the most arguably crucial aspect in studying infectious diseases. In general, whenever the value of R_0 is less than a unity the disease will die out of the system and the disease-free equilibrium point is locally asymptotically stable (Diekmann et al., 1990), but whenever R_0 is greater than unity the disease will persist in the system and the disease-free equilibrium will be unstable, thus the existence of endemic equilibrium. There are several methods to derive the basic reproductive number of the model system (3.2.4), in this chapter, we are using the next generation method introduced by (Diekmann et al., 1990), here the population is divided into non-overlapping classes. Since we are concerned with the classes that spread the infection, thus we consider modelling the infected I class and ascospores P_A, such that the model system (3.2.4) becomes:

$$\begin{cases} \dfrac{dI(t)}{dt} = \lambda S - (\alpha + \mu)I, \\ \dfrac{dP_A(t)}{dt} = \eta P_0 - \psi P_A. \end{cases} \tag{3.5.13}$$

This follows the decomposition of the model into two categories, that is F and V. Where F is a matrix of the rate of new infection and V is the matrix of net infected class. For this system

$$F = \begin{pmatrix} \lambda S \\ 0 \\ 0 \end{pmatrix} \tag{3.5.14}$$

We take the partial derivatives of the newly infected population evaluated at the disease free equilibrium state. This is the rate at which susceptible fruits transfer into infected given by F

$$F = \begin{pmatrix} 0 & 0 & \dfrac{\beta}{\rho} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{3.5.15}$$

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Now we talk about the rate at which infected fruits and pathogens leave their compartments due to decay caused by natural causes or disease induced. It is given by

$$V = \begin{pmatrix} -(\alpha+\mu) & 0 & 0 \\ \alpha & (\delta-(\eta+\psi)) & 0 \\ 0 & \eta & -\psi \end{pmatrix} \tag{3.5.16}$$

In this case F and V meet the conditions outline by (Diekmann et al., 1990), we can now derive the next generation matrix, since V is non-singular. The determination of V inverse is computed to obtain

$$V^{-1} = \begin{pmatrix} \frac{1}{-(\alpha+\mu)} & 0 & 0 \\ \frac{\alpha}{(\alpha+\mu)(\delta-\eta-\psi)} & \frac{1}{\delta-\eta-\psi} & 0 \\ \frac{\alpha\eta}{(\alpha+\mu)(\delta-\eta-\psi)\psi} & \frac{1}{\delta-\eta-\psi} & \frac{1}{\psi} \end{pmatrix} \tag{3.5.17}$$

Now we find the product of the matrix F and V^{-1}

$$FV^{-1} = \begin{pmatrix} \frac{\alpha\eta\beta}{\rho\psi(\alpha+\mu)(\delta-\eta-\psi)} & \frac{\beta}{\rho(\delta-\eta-\psi)} & \frac{\beta}{\rho\psi} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{3.5.18}$$

Hence, the reproduction number R_0 is given as the spectral radius of the (dominant eigenvalues) of the matrix FV^{-1} as

$$R_0 = \rho_\lambda(FV^{-1}) = \frac{\alpha\eta\beta}{\rho\psi(\alpha+\mu)(\delta-\eta-\psi)} \tag{3.5.19}$$

We observed that the reproduction number is directly proportional to the average lifetime of the ascomata, the average time of staying in the infected class, the average time of staying in the ascospores class and the product of the probability transmission and shedding of the pathogen by the infected class. If R_0 is greater than the unity, then the CBS is likely to invade and persist. Mathematically for disease to die out $\alpha\eta\beta$ should be less than $\rho\psi(\alpha+\mu)(\delta-\eta-\psi)$.

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3.6 Local stability of disease free equilibrium E_0

This section focuses on the determination of the asymptotic behaviour of the model system solutions based on the stability of the model equilibrium states. This model also permits a disease-free equilibrium and, at least, one endemic equilibrium. Hence, the local stability of the disease-free equilibrium is established to determine whether CBS will invade and persisting or just die out. From (Driessche and Watmough, 2002), if the basic reproduction number R_0 is less than unity, then the disease is locally asymptotically stable. The model system (3.2.4) is equated to zero at disease free because there is no infection at this stage, thus, all the entire population will be completely susceptible. At equilibrium state the rate of change is zero $E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$. To determine the stability of disease-free equilibrium, the eigenvalues of the Jacobian matrix evaluated at the equilibrium point are computed. If negative real parts are obtained $R_0 < 1$, then the system is stable at disease free equilibrium, biologically this means that infected individuals introduced to the system would not invade and persist, but if one is positive and the other is negative then the system is unstable. The general Jacobian matrix of the model system (3.2.4) is given by

$$J = \begin{pmatrix} r - \frac{2rS}{\kappa} - \frac{rI}{\kappa} - \frac{\beta P_A}{\rho + \epsilon P_A} - \mu & \frac{rS}{\kappa} & 0 & \frac{\beta S P_A \epsilon - (\rho + \epsilon P_A)\beta S}{(\rho + \epsilon P_A)^2} \\ \frac{\beta P_A}{\rho + \epsilon P_A} & -(\alpha + \mu) & 0 & \frac{\beta S P_A \epsilon - (\rho + \epsilon P_A)\beta S}{(\rho + \epsilon P_A)^2} \\ 0 & \alpha & \delta - \frac{2P_0}{\kappa} - \eta - \psi & 0 \\ 0 & 0 & \eta & -\psi \end{pmatrix} \tag{3.6.20}$$

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The Jacobian matrix is then evaluated at disease free equilibrium and gives,

$$J = \begin{pmatrix} r - \frac{2r\Lambda}{\kappa\mu} - \mu & \frac{r\Lambda}{\kappa\mu} & 0 & \frac{\beta\Lambda}{\rho\mu} \\ 0 & -(\alpha+\mu) & 0 & \frac{\beta\Lambda}{\rho\mu} \\ 0 & \alpha & \delta - \frac{2\Lambda}{\kappa\psi} - \eta - \psi & 0 \\ 0 & 0 & \eta & -\psi \end{pmatrix} \tag{3.6.21}$$

The Jacobian matrix evaluated at DFE is used to assess the local stability by the eigenvalues of the characteristics equation $det(J - \lambda_e I)$, where

$$(a + \lambda_e)(b + \lambda_e)(c + \lambda_e)(d + \lambda_e) = 0 \tag{3.6.22}$$

where

$a = r - \frac{2r\Lambda}{\mu} \quad b = \alpha + \mu, \quad c = \delta - \frac{2\Lambda}{\psi} - \eta - \psi, \quad d = \psi$

Therefore the eigenvalues corresponding to the matrix above are as follows:

$\lambda_{e1} = -\psi, \quad \lambda_{e2} = -(\alpha + \mu), \quad \lambda_{e3} = r - \frac{2r\Lambda}{\kappa\mu} - \mu, \quad \lambda_{e4} = \delta - \frac{2\Lambda}{\kappa\psi} - \eta - \psi$

Clearly all the roots of the characteristic equation (3.6.22) either have negative real parts or are negative (Diekmann et al., 2009). The results are summarised by the following theorem

Theorem 3.2. *The disease-free equilibrium point E_0, of the model system (3.2.4) is locally asymptotically stable whenever $R_0 < 1$ and unstable otherwise*

Now we show that the DFE of the model system (3.2.4) will be globally asymptotically stable when $R_0 < 1$

Proof. An equilibrium is asymptotically stable if all eigenvalues have negative real parts; it is unstable if at least one eigenvalue has positive real part. Since the eigenvalue of $J(E_0)$ are negative except λ_{e3} and λ_{e4}, it follows that the DFE is locally asymptotically stable if $\lambda_{e3} < 0$ and $\lambda_{e4} < 0$ if and only if $R_0 < 1$ □

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3.7 Global stability of the disease free equilibrium.

The next generation matrix operator (Castillo-Chávez et al., 2002) is used to determine the globally asymptotically stability of the disease free equilibrium of the model system (3.2.4). Theorem (3.3) establishes that the disease equilibrium is globally asymptotically stable whenever $R_0 > 1$. Herein, two assumption are listed such that if they are satisfied, the global asymptotically stability of disease free is guaranteed. The model system (3.2.4) is written in this form:

$$\begin{cases} \dfrac{dX}{dt} &= F(X,Z), \\ \dfrac{dZ}{dt} &= G(X,Z). \end{cases} \tag{3.7.23}$$

where,

$X = (S)$ denotes all uninfected compartments.

$Z = (I, P_A, P_0)$ denotes all infected and infectious.

Letting

$$U_0 = (X^*, 0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$$

to denote the DFE of the system. To guarantee global asymptotic stability, the conditions (H1) and (H2) must be satisfied (Castillo-Chávez et al., 2002).

H1. For $\dfrac{dX}{dt} = F(X,0)$, X^* is globally asymptotically stable (g.a.s),

H2. $G(X,Z) = AZ - \widehat{G}(X,Z)$, where $\widehat{G}(X,Z) \geq 0$ for $(X,Z) \in \mathbb{R}^3_+$ and $A = D_Z G(X^*, 0)$ is an M-matrix and $\mathbb{R}^3_+$ is the region where the model makes biological sense (Garira et al., 2014).

In this case, we have

$$F(X,0) = \begin{pmatrix} \Lambda - \mu S \end{pmatrix} \tag{3.7.24}$$

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and matrix A is given by

$$A = \begin{bmatrix} -(\alpha+\mu) & 0 & \frac{\beta\Lambda}{\rho\mu}, \\ \alpha & \delta - \frac{2\Lambda}{\kappa\psi} - \eta - \psi & 0, \\ 0 & \eta & -\psi \end{bmatrix} \tag{3.7.25}$$

and

$$\widehat{G}(X,Z) = \begin{bmatrix} \left(\frac{\Lambda}{\rho\mu} - \frac{S}{\rho+\epsilon P_A}\right)\beta P_A \\ 0 \\ 0 \end{bmatrix} \tag{3.7.26}$$

The condition $\widehat{G}(X,Y) \geq 0$ is satisfied since $\frac{\Lambda}{\rho\mu} \geq \frac{S}{\rho+\epsilon P_A}\ \forall\ (X,Z) \in \mathbb{R}^4_+$
The off diagonal elements of A matrix are non-negative which implies that A is an M-matrix. Therefore, the following theorem summarises the above results.

Theorem 3.3. *The disease free equilibrium* $U_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$ *is a globally asymptotically stable (g.a.s) equilibrium of system (3.2.4) if* $R_0 < 1$ *and the condition (H1) and (H2) are met.*

3.8 The endemic equilibrium state and its stability

3.8.1 Endemic equilibrium

(Castillo-Chávez et al., 2002) is used to determine the globally asymptotically stability of the disease-free equilibrium of the model system (3.2.4). Theorem (3.3) establishes that the disease equilibrium is globally asymptotically stable whenever $R_0 \geq 1$. Herein, two assumptions are listed such that if they are

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satisfied, the global asymptotically stability of disease free is guaranteed.

$$S^* = \kappa - \frac{\kappa(\lambda^* + \mu)}{r} \tag{3.8.27}$$

From the susceptible fruits endemic value (3.8.27), it is noted that susceptible fruits are directly proportional to the carrying capacity of fruits less the product of the average time of staying in the susceptible class and the carrying capacity of fruits and indirectly proportional to the recruitment rate of the newly uninfected rate of susceptible fruits. The recruitment rate in the susceptible class is defined by the development of new fruits (r). Fruits leave this class through either decay due to natural causes or infection, while the infected population is given by

$$I^* = \frac{\lambda^* S^*}{r(\alpha + \mu)} \tag{3.8.28}$$

The infected state variable $I^*$is directly proportional to the average time of staying in the same state variable I^* and the product of its force of infection and the susceptible class. It is also indirectly proportional to the susceptible recruitment rate. The ascomata population is similarly given by

$$P_0^* = \frac{\delta - \eta - \psi}{2} \pm \sqrt{\frac{(\delta - \eta - \psi)^2 - 4\delta(-\mu I)}{2}} \tag{3.8.29}$$

We note that the state variable P_0^* is directly proportional to pathogen shedding into the environment from infected class, its death rate and transformation of ascospores. While its infectious counterpart ascospores population is given by:

$$P_A^* = \frac{\eta P_0^*}{\psi} \tag{3.8.30}$$

We deduce from (3.8.30) that the ascospores population at endemic is directly proportional to the rate at ascomata transform into ascospores and the average time of staying in the ascospores state variable. This relationship suggest that the controlling of ascomata can reduce the infectious ascospores.

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3.8.2 The local stability of the endemic equilibrium E_1

Like any other equilibrium point of any dynamical system, there is no change either in terms of growth of infection or decay, the system can be computed from the model equations 3.2.4 by considering the following reduced model.

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dP_1}{dt} = \frac{dP_A}{dt} = 0$$

and solving for the state variables S, I, P_0 and P_A in terms of the model parameters

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \lambda S - \mu S, \\ \dfrac{dI}{dt} = \lambda S - (\alpha + \mu) I, \\ \dfrac{dP_A}{dt} = \eta P_0 - \psi P_A. \end{cases} \tag{3.8.31}$$

defined in

$$\Phi = \{(S^*, I^*, P_A^*) \in \mathbb{R}_+^3\} \subset \Omega \tag{3.8.32}$$

For existence and uniqueness of endemic equilibrium

$$E_1 = (S^*, I^*, P_0^*, P_A^*), \tag{3.8.33}$$

its state variables should satisfy the condition $S^* > 0, I^* > 0, P_A^* > 0$. From model system 3.2.4 at equilibrium (Tchuenche and Chiyaka, 2009), we have

$$\begin{cases} 0 = \Lambda - \lambda S^* - \mu S^*, \\ 0 = \lambda S^* - (\alpha + \mu) I^*, \\ 0 = \eta P_0^* - \psi P_A^*. \end{cases} \tag{3.8.34}$$

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At the equilibrium states of the reduced model 3.8.31, the Jacobian matrix (J_{E_1}) is given by

$$J = \begin{pmatrix} r - \frac{2rS^*}{\kappa} - \frac{rI^*}{\kappa} - \frac{\beta P_A^*}{\rho + \epsilon P_A^*} - \mu & \frac{rS^*}{\kappa} & 0 & \frac{\beta S^* P_A^* \epsilon - (\rho + \epsilon P_A^*)\beta S^*}{(\rho + \epsilon P_A^*)^2} \\ \frac{\beta P_A^*}{\rho + \epsilon P_A^*} & -(\alpha + \mu) & 0 & \frac{\beta S^* P_A^* \epsilon - (\rho + \epsilon P_A^*)\beta S^*}{(\rho + \epsilon P_A^*)^2} \\ 0 & \alpha & \delta - \frac{2P_0^*}{\kappa} - \eta - \psi & 0 \\ 0 & 0 & \eta & -\psi \end{pmatrix} \tag{3.8.35}$$

Using the additive compound matrices approach by (Li and Muldowney, 1996) (Tchuenche and Chiyaka, 2009), we determine stability of the endemic equilibrium. The second additive compound matrix of the Jacobian matrix $(J_{(E_1)})$is computed. The local stability of the endemic equilibrium point E_1 is given by the following Theorem from (Li and Muldowney, 1996).

Theorem 3.4. *An $n \times n$ matrix A is stable if and only if $A^{[2]}$ is stable and $(-1)^n det(A) > 0$*

Proof. We determine $det(J_{(E_1)})$ of the model system (3.8.34) evaluated at endemic equilibrium, as follows:

$$detJ(E_1) = \begin{vmatrix} M - \frac{\beta P_A^*}{\rho + \epsilon P_A^*} - \mu & \frac{rS^*}{\kappa} & 0 & \frac{\beta S^* P_A^* \epsilon - (\rho + \epsilon P_A^*)\beta S^*}{(\rho + \epsilon P_A^*)^2} \\ \frac{\beta P_A^*}{\rho + \epsilon P_A^*} & -(\alpha + \mu) & 0 & \frac{\beta S^* P_A^* \epsilon - (\rho + \epsilon P_A^*)\beta S^*}{(\rho + \epsilon P_A^*)^2} \\ 0 & \alpha & \delta - \frac{2P_0^*}{\kappa} - \eta - \psi & 0 \\ 0 & 0 & \eta & -\psi \end{vmatrix} \tag{3.8.36}$$

where $M = r - \frac{2rS^*}{\kappa} - \frac{rI^*}{\kappa}$

Clearly, we can see that the $det(J_{(E_1)}) = (-1)^4 det(J_{(E_1)}) > 0$.. For the endemic equilibrium point

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$E_1 = (S^*, I^*, P_0^*, P_A^*)$, let P = diagonal $(1, -1, -1, -1)$ be the diagonal matrix (Li and Muldowney, 1996) (Tchuenche and Chiyaka, 2009). Thus we apply (Okuguchi, 1978) which establishes that if a matrix A has a negative dominant diagonal elements, the, A is stable.

Theorem 3.5. *If an $n \times n$ matrix A is dominant diagonally and the diagonal is composed of negative elements $(a_{ii} < 0 \, \forall \, i = 1, ..., n)$ then, the real parts of all its eigenvalues are negative, that is, A is stable.*

This can be done by studying if the matrix $PJ^{[2]}_{(E_1)}P^{-1}$ is diagonally dominant in rows, since its diagonal elements are negative. Since all diagonal values are less than zero, therefore, all the diagonals are negative, implying that the system has a local stability at the endemic equilibrium point. □

3.8.3 Global stability of the endemic equilibrium

The following theorem by (Tchuenche and Chiyaka, 2009) is used to determine stability of endemic equilibrium E_1

Theorem 3.6. *The model system (3.2.4) has no periodic orbits and its endemic equilibrium is globally asymptotically stable when $R_0 > 1$.*

Proof. The proof herein is based on reducing the model system (3.2.4) into a $2-$dimensional model by eliminating the uninfected and not capable of transmitting the disease classes, that is, S and P_0 to remain with the infected and infectiuos classes:

$$\begin{cases} \dfrac{dI}{dt} = \lambda S - (\alpha + \mu)I, \\ \dfrac{dP_A}{dt} = \eta P_0 - \psi P_A. \end{cases} \tag{3.8.37}$$

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Let $X = \lambda S - (\alpha + \mu)I, \quad Y = \eta P_0 - \psi P_A$, then the Dulac's function is considered (Tchuenche and Chiyaka, 2009) $D = \dfrac{1}{IP_A}$, where $I > 0$ and $P_A > 0$. Therefore

$$\frac{\partial(DX)}{\partial P_A} + \frac{\partial(DY)}{\partial I} = -\left\{\frac{\lambda S + \eta P_0}{(IP_A)^2}\right\} < 0 \tag{3.8.38}$$

Since $(\lambda S + \eta P_0) > ((IP_A)^2)$. Thus, by Dulac's criterion, there are no periodic orbits in $\Omega \mid \Omega^*$, where $\Omega^* = \{(S, I, P_0, P_A) \in \Omega : I = P_0 = P_A = 0\}$. Since Ω is positively invariant, and the endemic equilibrium exists whenever $R_0 > 1$, then it follows from the Poincare-Bendixon Theorem (Dumortier et al., 2000), that all solutions of the limiting system originating in Ω remaining in Ω $\forall t$. Furthermore, the absence of periodic orbits in Ω implies that the unique endemic equilibrium of the special case of the model system (3.2.4) is globally asymptotically stable whenever $R_0 > 1$ (Tchuenche and Chiyaka, 2009) □

3.9 Numerical simulation

This section deals with graphical solutions of the model system (3.2.4) evolution of the various state variables using different parameter values. The plots are constructed using python version 2.7.6 software on Ubuntu 14.0.4. The package has a wide range of modules and among them is odeint function and SciPy (open source library of scientific tools). We observed that focusing intervention strategies on ascospores and ascomata rapidly decrease the level of infection and all state variable classes progress to an epidemic. Some parameters were estimated due to the scarcity of published literature. The graphs showing the evolution of the various classes against time are shown below with the following parameters values.

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Definition	Symbol	Estimate	Source
Host recruitment rate	Λ	20.10	(Bailey and Gilligan, 2004)
Host natural decay rate	μ	0.0035	(Chiyaka et al., 2012)
Host decay due to disease	α	0.00014	(Bailey and Gilligan, 2004)
Probability of transmission	β	0.105	Estimated
Pathogen transformation rate	η	0.295	(Chiyaka et al., 2012)
Limitation of the growth velocity	ϵ	0.989	(Chiyaka et al., 2012)
Half saturation constant	ρ	20000	(Bailey and Gilligan, 2004)
Carrying capacity	κ	5000000	(Bailey and Gilligan, 2004)
Proliferation rate	δ	0.1	Estimated
Ascospores diminishing rate	ψ	0.045	(Bailey and Gilligan, 2004)

Table 3.2: *Fruit host and pathogens parameter values used in simulation*

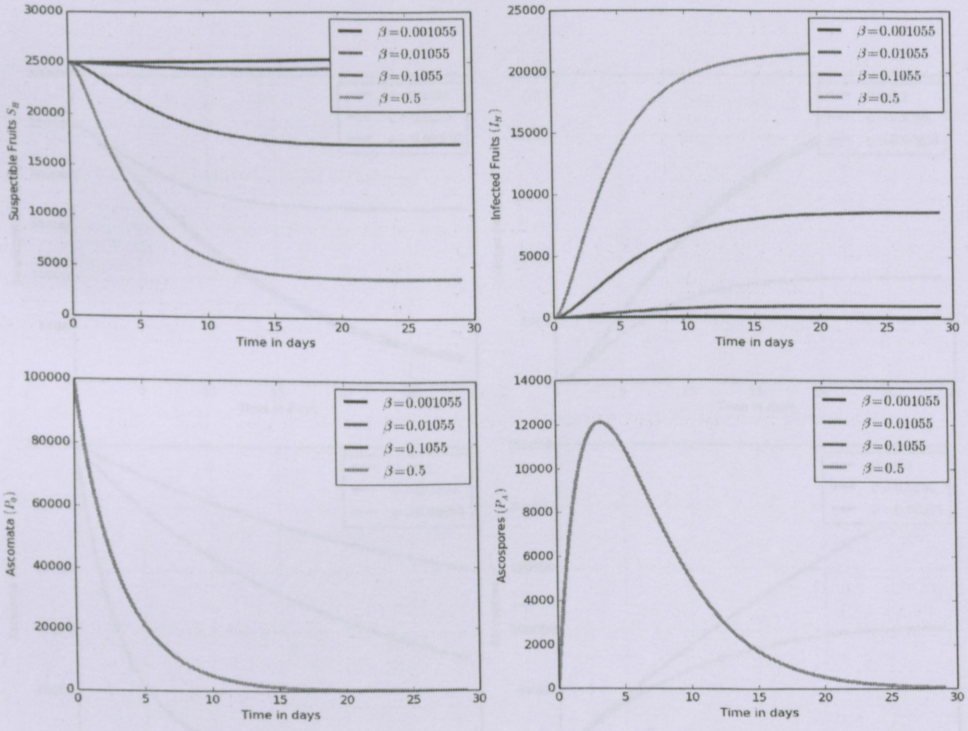

Figure 3.3: *Simulation of model system 3.2.4 showing the evolution with time of susceptible fruits* (S)*, infected fruits* (I)*, Ascomata* (P_0) *and Ascospores* (P_A) *for about 30 days by varying* β*.*

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The simulation on Figure (3.3) show the behaviour of the system of susceptible fruits, infected, ascomata and ascospores evolving in time. When the probability of the transmission β was varying from 0.001055 to 0.5 the observation made agreed with R_0 on the basis that as β increases so does the infective class (I), and inversely affects non-infectious (S) population class, during the initial stages the infected class of the host increased at an increasing rate up to a peak of 20000 and ascospores increased up to about 12000 where it reached its carrying capacity then both gradually decrease. We also observed that as the infectious classes were increasing, non-infectious classes were decreasing. The variation of β does not have an effect on the pathogen population but rather on the host population. Therefore, this means that controlling the transmission probability does not have an influence on the ascomata and ascospores. So the use of intervention strategies aimed at directly controlling transmission probability will only be temporary because the root of the problem remains unattended.

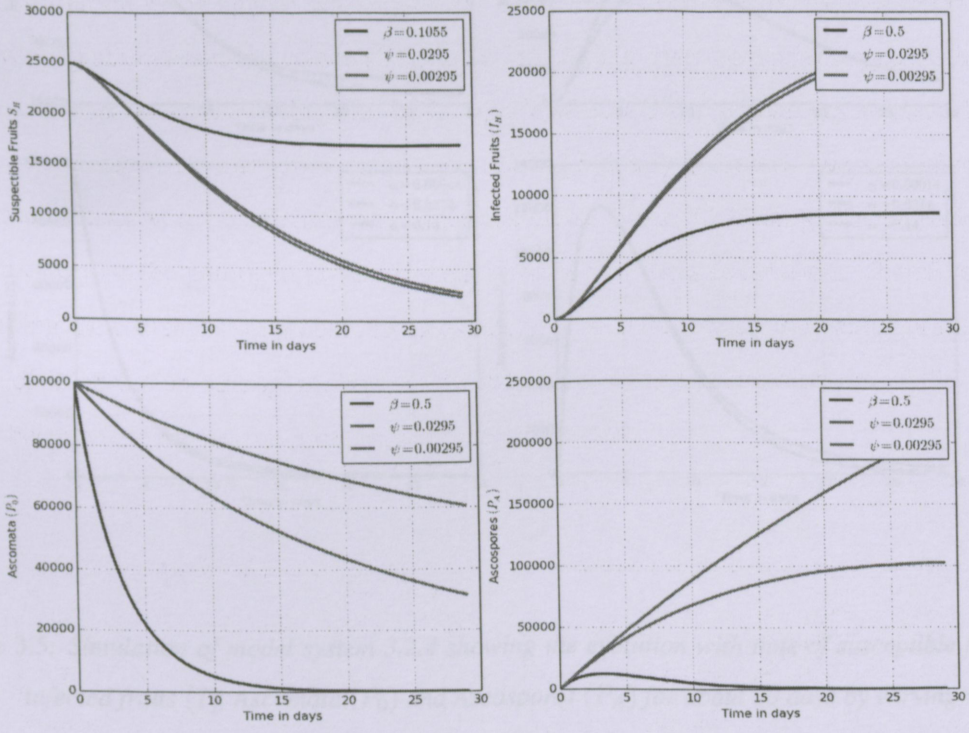

Figure 3.4: *Simulation of model system 3.2.4 showing the evolution with time of susceptible fruits* (S)*, infected fruits* (I)*, Ascomata* (P_0) *and Ascospores* (P_A) *for about 30 days by varying* ψ*.*

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The parameter ψ is the most powerful parameter to be considered when designing intervention strategies because it has an influence on all classes. When the ψ increases it sharply decreases the ascomata and ascospores in the environment within a short period of time. Still, by the same token, susceptible fruits are sharply increasing. Infection is shown to be relatively low as ψ increases. This simulation brings in a new dimension, confirm that controlling strategies should also improve on methods to accelerate ascomata and ascospores diminishing rate. All things being equal, it is sufficient to destroy ascomata and ascospores in order to eliminate and eradicate CBS.

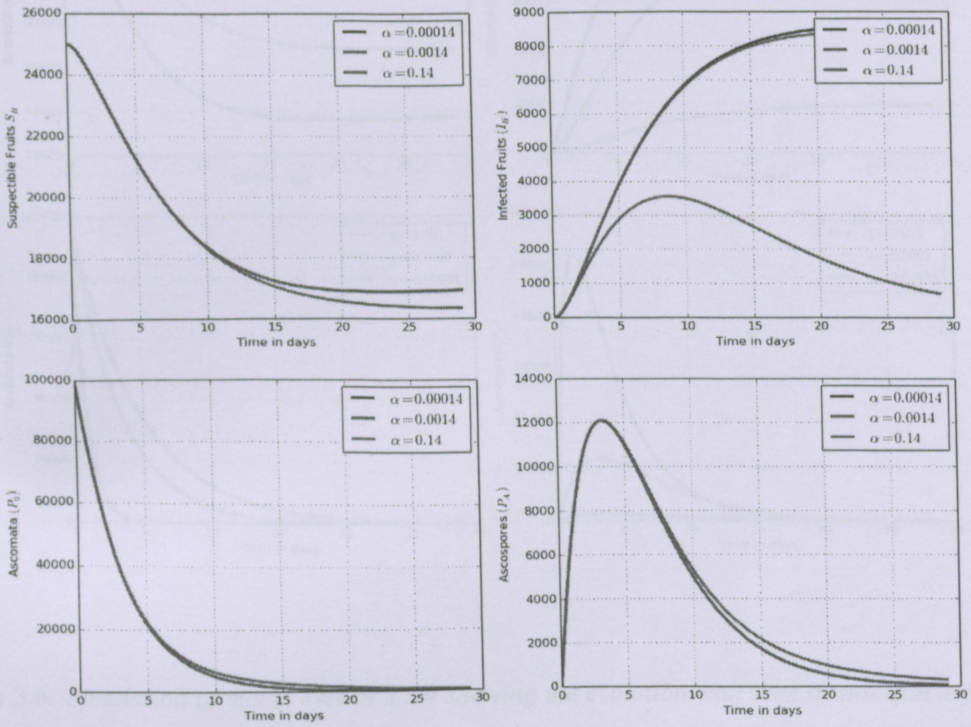

Figure 3.5: *Simulation of model system 3.2.4 showing the evolution with time of susceptible fruits* (S)*, infected fruits* (I)*, Ascomata* (P_0) *and Ascospores* (P_A) *for about 30 days by varying* α*.*

The graphical simulation indicates that the fruits decay due to disease has an impact on all state variables. Observation from the graph suggests that the increase in α result in a huge decrease of infected class and a slight decrease in the susceptible class. Furthermore, the ascomata and the ascospores class increase

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slightly at the same rate as the infected class decreases. This dynamic aspect shows that the more infected fruits available in the environment, the more the pathogens. One way of controlling or breaking the cycle is to make sure no infected fruits are made available in the farming areas before the next fruit onset season.

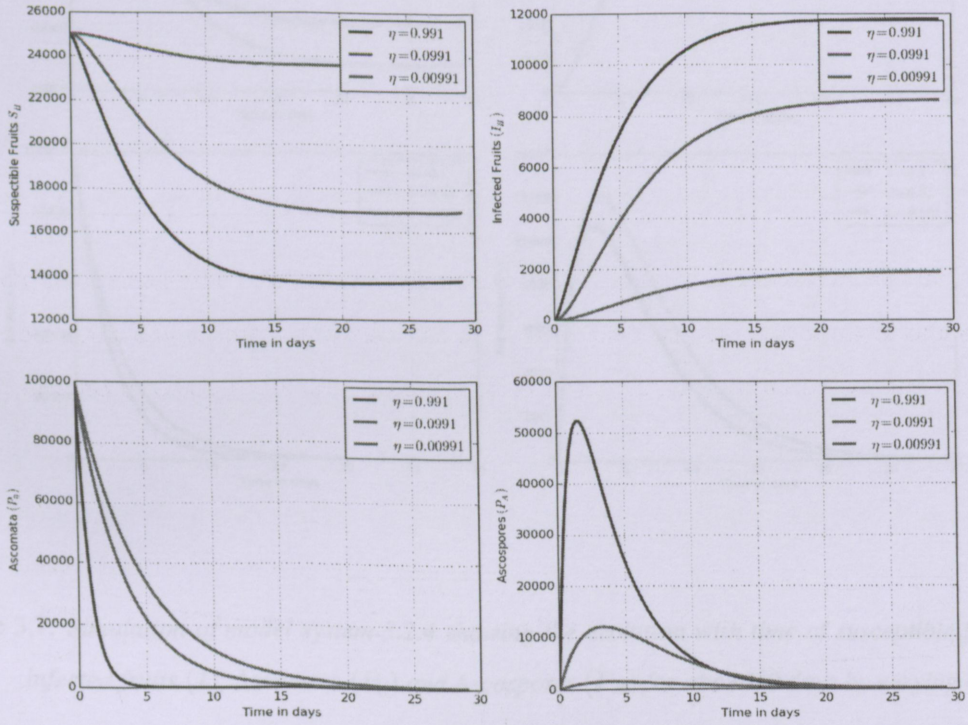

Figure 3.6: *Simulation of model system 3.2.4 showing the evolution with time of susceptible fruits* (S)*, infected fruits* (I)*, Ascomata* (P_0) *and Ascospores* (P_A) *for about 30 days by varying* η*.*

The graphs shows the impact of pathogen transformation rate and it is shown to be playing a major role consider all state variables are affected by its variation. Pathogen transformation rate refers to ascomata ejecting ascospores at the rate η. To successfully control the disease, this parameter needs to be seriously considered. The decrease in the ascospores formation shows a huge decline of the infected class and the ascospores while the susceptible class is showing a huge increase and the ascomata showing a slight increase.

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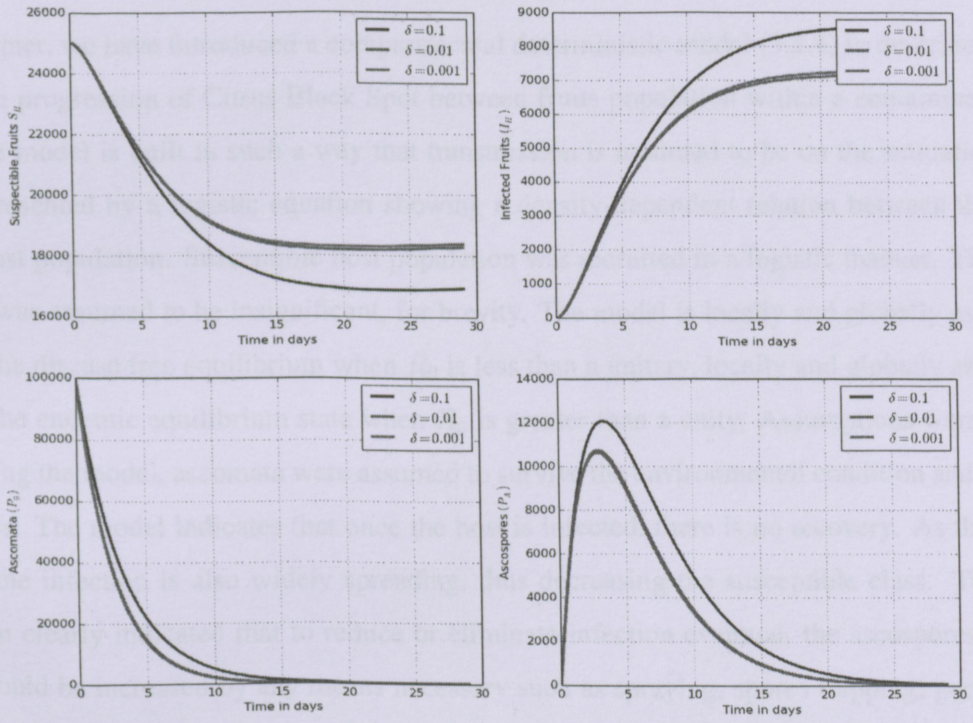

Figure 3.7: *Simulation of model system 3.2.4 showing the evolution with time of susceptible fruits* (S)*, infected fruits* (I)*, Ascomata* (P_0) *and Ascospores* (P_A) *for about 30 days by varying* δ*.*

According to the graph, the effects of ascomata growth within its population δ is directly proportional to the infected class and ascospores class, but indirectly proportional to the susceptible class. This follows that the increase in ascomata growth directly increase the ascospores which result in the increase in the force of infection shown by the increase in the infected class. This brings in an interesting dimension that can help in improving the intervention strategies. Ascomata growth within the orchard floor should be target the most in order to slow down or eradicate CBS.

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3.10 Summary

In this chapter, we have introduced a compartmental deterministic model (3.2.4) to describe and analyse the disease progression of Citrus Black Spot between fruits population within a contaminated environment. The model is built in such a way that transmission is assumed to be on the saturation incidence basis, represented by a logistic equation showing a density-dependent relation between the pathogens and the host population. Susceptible host population was recruited in a logistic manner. The secondary infection was assumed to be insignificant, for brevity. The model is locally and globally asymptotically stable at the disease free equilibrium when R_0 is less than a unitary, locally and globally asymptotically stable at the endemic equilibrium state when R_0 is greater than a unity. Assumptions were made when constructing the model, ascomata were assumed to survive the environmental condition and transform to ascospores. The model indicates that once the host is infected, there is no recovery. As the ascospores increase the infection is also widely spreading, thus decreasing the susceptible class. The numerical simulation clearly indicated that to reduce or eliminate infection eventual, the ascospores diminishing rate ψ should be increased by any means necessary such as spraying, spores trapping, good agriculture practice among other strategies. Realistic parameters were used to validate the model when carrying out numerical simulation.

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Chapter 4

IMMUNOLOGICAL MODEL OF *Guignardia citricarpa*

4.1 Introduction

CBS is one of the most important diseases in the subtropical region and in the world. Initial the fruiting body ascomata eject ascospores and widely spread either by air current or water splash, when it contact the fruit surface it germinates and form appressoria. Appressoria develop a thin tube peg that penetrates the fruit epidemic wall causing infection in the host immune defence system, fruit infection remains latent until fruit maturity, upon maturity the appressoria grows into the outer rind forming lesion accompanied by pycnidia which then burst to produce conidia, which is a secondary infection dispersed by splash of water, enabling it to infect fruits of the same tree due to limited dispersal (Kotzé, 2000). The plant immune defence system is normally replenished within the plant mechanism and infection may invade the mechanism source or the control immune defence system (Jones and Dangl, 2006). These pathogen molecules have been known for depriving nutrients for living immune defence system. When ascospores

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attach itself to plant surfaces, the spores germinate by growing a germ tube and eventually locates a stoma and grow towards a ridge between the epidermal wall cells. Appressoria is produced in the stoma (a structure known for locking the cell walls) and spread around plant cells without invading the cell membrane. An overview of biological dynamic features of the sexual pathogen *Guignardia citricarpa* was considered for successful modelling. It has been noted from the literature gathered that elimination or eradication of this pathogen has never been successful, even to the extent of slowing down the CBS in generally. The aim of this chapter is to determine quantitative features of *Guignardia citricarpa* infection dynamics.

4.2 Model formulation

The simple state transition SIP model for the within-host dynamics is developed to monitor the life of *Guignardia citricarpa* inside the fruit. In this study, similar techniques used in chapter 3 is used to identify the within-host transmission dynamics, these include susceptible host immune defence system, infected host immune defence system and *Guignardia citricarpa* molecules in the form of appressoria and pycnidia proteins activities. The density of pathogen in the form of appressoria within the host is given by P_1, with the increase through the supply rate of λ and the decrease through the transmission rate of β. When pathogens invade the immune defence system, we assume the force of infection follows the law of mass action in which we quantify it by λS_c, with $\lambda = \beta P_1$ being the transmission rate. This implies that the infection is a result of the interaction of the appressoria (P_1) and the susceptible immune defence system (S_c) at rate $\lambda P_1 S_c$. The effect also causes the loss of the susceptible immune defence system at rate $-\lambda P_1 S_c$. The susceptible immune defence system is increased by the source within the host at the rate λ and decrease by the decay due to natural causes at the rate μ_c.

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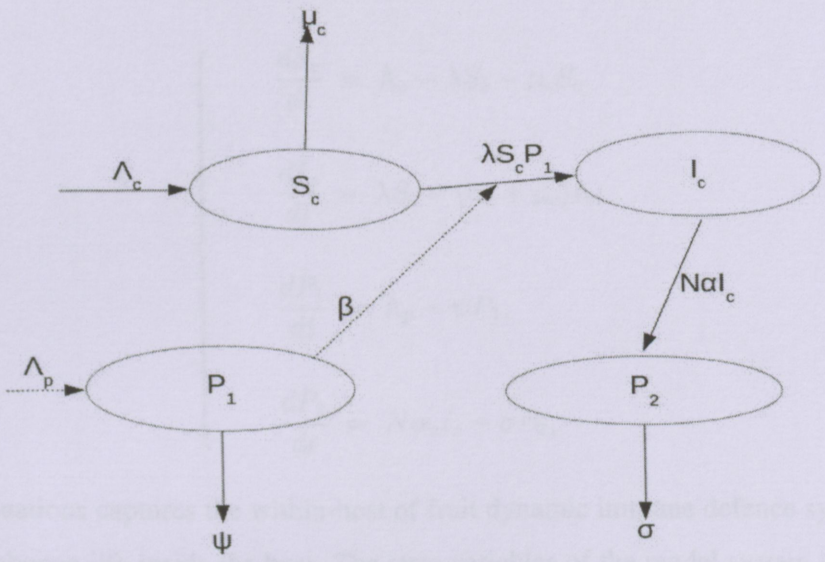

Figure 4.1: *Conceptual model of the within host dynamics of Citrus Black spot.*

Contamination is assumed to occur between the susceptible immune defence system and the appressoria and it is given by (I_c), with a decrease through natural decay μ_c and infection induced decay α. We also assume that the infected state gradually transforms into pycnidia P_2 at the rate α. Thus, the class P_2 is diminished by the rate σ. Focusing on the SIP state transition model of the immune defence system incorporating appressoria and pycnidia disregarding other small stages of the *Guignardia citricarpa*, but within the nature of confines biologically realistic, the model system for the within-host dynamics is

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given by:

$$\begin{cases} \dfrac{dS_c}{dt} = \Lambda_c - \lambda S_c - \mu_c S_c, \\ \dfrac{dI_c}{dt} = \lambda S_c - (\alpha_c + \mu_c) I_c, \\ \dfrac{dP_1}{dt} = \Lambda_p - \psi P_1, \\ \dfrac{dP_2}{dt} = N\alpha_c I_c - \sigma P_2, \end{cases} \tag{4.2.1}$$

The first two equations captures the within-host of fruit dynamic immune defence system. The last two monitors the pathogen life inside the host. The state variables of the model system is given in the table as follows:

Term	Description
S_c	Health Susceptible immune defence system
I_c	Infected immune defence system
P_1	Appressoria
P_2	Pycinidia

Table 4.1: *Description of the state variables of the model system (4.2.1)*

4.3 Analysis of the model

The model system (4.2.1) consists of ordinary differential equations monitoring population dynamics, initial conditions, assumptions on the parameters are also considered. In this section, we determine a set of analytical conditions which will ensure the global existence and uniqueness of a solution (Tchuenche and Chiyaka, 2009). The is analysed in a feasible region as shown below. We assume that parameters and state variables used are positive to avoid violation of biological interest. We use the following lemma established by (Tchuenche and Chiyaka, 2009) to show the feasible region of the model system (4.2.1):

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Lemma 4.1. *The feasible set of model system (4.2.1) is given by* $\Omega = \{(S_c, I_c, P_1, P_2) \in \mathbb{R}^4_+ : S_c + I_c \leqslant \frac{\Lambda_c}{\mu_c}, P_1 + P_2 \leqslant \frac{\Lambda_p}{\sigma_c}\}$

Proof. Adding the fruit population differential equation in the model system (4.2.1) gives

$$\begin{cases} \dfrac{dN_c(t)}{dt} = \dfrac{dS_c(t)}{dt} + \dfrac{dI_c(t)}{dt}, \\ \dfrac{dN_c(t)}{dt} = \Lambda_c - \lambda S_c P_1 + \lambda S_c P_1 - \mu_c S_c - \alpha_c I_c, \\ \dfrac{dN_c(t)}{dt} = \Lambda_c - \mu_c S_c - (\alpha_c + \mu_c) I_c \end{cases} \tag{4.3.2}$$

grouping like terms

$$\begin{cases} \dfrac{dN_c(t)}{dt} = \Lambda_c - \mu_c(S_c(t) + I_c(t)) - \alpha I_c(t), \\ \dfrac{dN_c(t)}{dt} = \Lambda_c - \mu_c N_c(t) - \alpha_c I_c(t), \\ \dfrac{dN_c(t)}{dt} \leq \Lambda_c - \mu_c N_c(t) \end{cases} \tag{4.3.3}$$

From (4.3.3) we deduce that

$$\lim_{t\to\infty} sup(N(t)) \leqslant \frac{\Lambda}{\mu} \tag{4.3.4}$$

Similarly, assigning $P = P_1 + P_2$ and add the last two equation of the model system (4.2.1) we have

$$\frac{dP(t)}{dt} \leq \Lambda_p - \psi_c P \tag{4.3.5}$$

This implies that

$$\lim_{t\to\infty} sup(P(t)) \leqslant \frac{\Lambda_p}{\psi_c} \tag{4.3.6}$$

then, all solutions of the model system (4.2.1) are non-negative and the global attractor is contained in Ω. Therefore the dynamics of the model system is considered in Ω (Tchuenche and Chiyaka, 2009). □

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Lemma 4.2. *The set Ω is positively invariant.*

Proof. In Ω we have

$$\begin{cases} S_c = 0 \Rightarrow & \dfrac{dS_c}{dt} = \Lambda_c, \\ I_c = 0 \Rightarrow & \dfrac{dI_c}{dt} = \lambda S_c, \\ P_1 = 0 \Rightarrow & \dfrac{dP_1}{dt} = \Lambda_p, \\ P_2 = 0 \Rightarrow & \dfrac{dP_2}{dt} = N\alpha_c I_c. \end{cases} \tag{4.3.7}$$

Clearly we can see that all the parameters are positive and any vector field on $\partial\Omega$, the boundary of Ω has inward direction, the region Ω is positively invariant for all solutions of the model system (4.2.1) and remain there for $t \geqslant 1$ □

Lemma 4.3. *A solution of the model system (4.2.1) is feasible in Ω*

Proof. The model system (4.2.1) is improvident, that is, all feasible solutions are uniformly bounded in a subset $\Omega \subset \mathbb{R}^4_+$, then any solution with initial value in Ω attains its supreme and remain there. Thus, Ω is compact and positively invariant □

4.4 Basic Properties

In this section, the analysis of the basic properties of the model system (4.2.1) is done to ensure that biological interests are not violated.

4.4.1 Positivity of solution

The model system (4.2.1) describes the transmission dynamics of a fruit and pathogen population, therefore, there is a need to prove that all state variables are non-negative $\forall\ t > 0$.

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Theorem 4.4. *Let the initial conditions be* $S_c = S_c^0(0) > 0, I_c(0) = I_c^0(0) \geq 0, P_1(0) = P_1^0 \geq 0, P_2(0) = P_2^0(0) \geq 0 \in \Omega$. *Then, the solution of* $S_c(t), I_c(t), P_1(t), P_2(t)$ *of the model system (4.2.1) remains positive* $\forall\ t > 0$.

Proof. For $t \in [0, \bar{S_c}]$, $\frac{dI_c}{dt} = \lambda S_c - (\alpha_c + \mu_c)I_c \geq -(\alpha_c + \mu_c)I_c$, and by freshman integration (Tchuenche and Chiyaka, 2009), we obtain

$$I_c(t) \geq I_c(0)e^{-(\alpha+\mu)} \geq 0, \alpha_c + \mu_c < +\infty$$

The similar argument can be done on the remaining state variable. This indicate that the solution of the model system (4.2.1) is such that $min\{S_c(t), I_c(t), P_1(t), P_2(t)\} \geq 0$ in its feasible region Ω □

4.5 Determination of disease free equilibrium and its stability

The model system (4.2.1) has the disease-free equilibrium state (E_0) and, at least, one endemic equilibrium state. At disease free equilibrium (E_0) there is no ascospore and no conidia and thus, no infection in fruit population. Hence, we let the right hand side of the model system (4.2.1) equal to zero such that:

$$\frac{dS_c}{dt} = \frac{dI_c}{dt} = \frac{dP_1}{dt} = \frac{dP_2}{dt} = 0$$

and

$$\begin{cases} 0 = \Lambda_c - \lambda S_c - \mu_c S \\ 0 = \lambda S_c - (\alpha_c + \mu_c)I_c \\ 0 = \Lambda_p - \psi P_1 \\ 0 = N\alpha_c I_c - \sigma P_2, \end{cases} \tag{4.5.8}$$

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This implies that

$$E_0 = (S_c(0), I_c(0), P_1(0), P_2(0)) = \left(\frac{\Lambda_c}{\mu_c}, 0, 0, 0\right) \tag{4.5.9}$$

4.5.1 Reproduction number of the dynamical system of CBS

The basic reproductive number R_0 is the most crucial parameter in disease modelling and it measures the disease threshold level. We computed the reproduction number to establish whether the disease will die out or persist using the next generating matrix operator approach of (Diekmann et al., 1990). The reproduction number R_0 is defined as the introduction of a primary case into an entire susceptible defence immune system. The study computes eigenvalues of the Jacobian at the disease free equilibrium to determine the spectral radius, that is, R_0. Suppose that $(\bar{X}, \bar{Y}, \bar{Z})$ is the steady state of the model system (4.2.1), then we arrange

$$\begin{cases} \dfrac{dX}{dt} &= f(X,Y,Z), \\ \dfrac{dY}{dt} &= g(X,Y,Z), \\ \dfrac{dZ}{dt} &= z(X,Y,Z), \end{cases} \tag{4.5.10}$$

where

- $X = (S_c)$ represent all non-infected compartments.
- $Y = (I_c)$ represent all infected compartments but not infectious.
- $Z = (P_1, P_2)$ infected and infectious

Let

$$U_0 = (X^*, 0, 0) \tag{4.5.11}$$

be the disease free equilibrium, such that

$$U_0 = (X^*, 0, 0), f(X^*, 0, 0) = g(X^*, 0, 0) = h(X^*, 0, 0) = 0 \tag{4.5.12}$$

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Assume the equation $g(X^*, Y, Z) = 0$ implicitly determines a function $E = \hat{g}(X^*, Z)$. Let $A = D_z h(x^*, \hat{g}(x^*, 0), 0)$ and assume that A can be written in the form $A = M - D$, $M \geq 0$ and $D > 0$, a diagonal matrix (Castillo-Chávez et al., 2002) (Castillo-Chávez et al., 2001). The basic reproduction number is defined as the spectral radius (maximum eigenvalue) of the matrix MD^{-1}, denoted by

$$R_0 = \rho_\lambda(MD^{-1}) \tag{4.5.13}$$

The threshold theorem by (Castillo-Chávez et al., 2002) point out that if $R_0 > 1$ then there will be invasion and persistence but if $R_0 < 1$ the infection will just die out.

Now letting $(X = (S_c), Y = (I_c), Z = (P_1, P_2))$ $U_0 = \left(\frac{\Lambda_c}{\mu_c}, 0, 0, 0\right)$ and $\hat{g}(X*, Z) = \lambda S_c P_1$

$$M = \begin{pmatrix} \frac{\lambda\Lambda_p}{\psi} & 0, \\ 0 & 0 \end{pmatrix} \quad D = \begin{pmatrix} -\psi & 0 \\ 0 & -\alpha_c \end{pmatrix} \tag{4.5.14}$$

and determinant of D is given by

$$D^{-1} = \begin{pmatrix} \frac{1}{\alpha_c} & 0 \\ 0 & \frac{1}{\psi} \end{pmatrix}$$

such that

$$MD^{-1} = \begin{pmatrix} \frac{\lambda\Lambda_p}{\psi\alpha_c} & 0, \\ 0 & 0 \end{pmatrix}$$

$$R_0 = \rho(MD^{-1}) = \frac{\lambda\Lambda_p}{\psi\alpha_c}$$

R_0 is directly proportional to the product of the transmission coefficient λ, susceptible immune defence system recruitment rate, the proportion of the pathogens that survive until infection takes place $\frac{1}{\psi}$ and

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the average infectious period $\frac{1}{\alpha_c}$ of Appressoria. The threshold theorem of (Kermack and McKendrick, 1927) established that whenever $R_0 > 1$ then an outbreak will occur but if $R_0 < 1$ the disease die out.

4.5.2 Local stability of disease free equilibrium E_0

The local stability of the disease-free equilibrium addresses the behaviour of solutions of the model system (4.2.1) near the equilibrium state. At disease free equilibrium there is no infection, thus, the total population is susceptible. This local stability is sometimes known as the linear stability. The model system (4.2.1) is linearised about the disease free equilibrium $E_0 = \left(\frac{\Lambda_c}{\mu_c}, 0, 0, 0\right)$ to transformed into a Jacobian matrix. To determine the stability of disease-free equilibrium, the eigenvalues of the Jacobian matrix are evaluated at the equilibrium points. Using (Van den Driessche and Watmough, 2002)'s theorem If $R_0 < 1$, then the disease-free equilibrium is locally asymptotically stable and the disease will die out. Thus, the following theorem holds

Theorem 4.5. *The disease free equilibrium state of the model system 4.2.1 is locally asymptotically stable whenever* $R_0 < 1$ *and unstable whenever* $R_0 > 1$

Proof. The Jacobian matrix is used to determine stability.

$$J(E_0) = \begin{pmatrix} -\mu & 0 & \frac{-\beta\Lambda_c}{\mu_c} & 0 \\ 0 & -\alpha_c & \frac{\beta\Lambda_c}{\mu_c} & 0 \\ 0 & 0 & -\psi_c & 0 \\ 0 & \frac{\alpha\Lambda_c}{\mu_c} & 0 & -\sigma \end{pmatrix} \tag{4.5.15}$$

The Jacobian matrix evaluated at disease free equilibrium (DFE) is used to determine the local stability by the eigenvalues of the characteristics equation $det(J - \lambda_e I)$, where

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$$det(J-\lambda_e I)=\begin{pmatrix} -\mu-\lambda_1 & 0 & \frac{-\beta\Lambda_c}{\mu_c} & 0 \\ 0 & -\alpha_c-\lambda_2 & \frac{\beta\Lambda_c}{\mu_c}\mu_c & 0 \\ 0 & 0 & -\psi_c-\lambda_3 & 0 \\ 0 & P\alpha & 0 & -\sigma-\lambda_4 \end{pmatrix} \tag{4.5.16}$$

Where $\lambda_1=-\mu,\ \ \lambda_2=-\alpha_c,\ \ \lambda_3=-\psi_c,\ \ \lambda_4=-\sigma$

Since the eigenvalues of $J(E_0)$ are negative, then the DFE is locally asymptotically stable if $R_0<1$ (Van den Driessche and Watmough, 2002). In biological terms, this means the invasion will not be able to persist because the threshold is below the unity. □

4.5.3 Global stability of the disease free equilibrium.

The disease free equilibrium is globally asymptotically stable if $R_0\leq 1$ and it is unstable if $R_0>1$. We follow (Castillo-Chávez et al., 2002)'s approach and express the model in this form:

$$\begin{cases} \dfrac{dX}{dt} &= F(X,Y), \\ \dfrac{dZ}{dt} &= G(X,Z). \end{cases} \tag{4.5.17}$$

where,

$X\ =\ (S_c)$ denotes all uninfected compartments

$Z\ =\ (I_c,P_1,P_2)$ denotes all infected and infectious compartments

Let

$$E_0=(X^*,0)=\left(\frac{\Lambda_c}{\mu_c},0,0,0\right)$$

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to denote the DFE of the system (4.2.1). The conditions (H1) and (H2) must be met for globally asymptomatically stability to exist (Magombedze et al., 2008),

H1. $\frac{dX}{dt} = F(X^*, 0)$, X^* is globally asymptotically stable (g.a.s)

H2. $G(X, Z) = AZ - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0$ for $(X, Z) \in \Omega$

where $A = D_Z G(X^*, 0)$ is an M-matrix and Ω is the region in which the model makes biological sense.

$$F(X, 0) = (\ \Lambda_c - \mu_c S_c,\ ) \tag{4.5.18}$$

and matrix A is given by

$$A = \begin{bmatrix} -(\alpha + \mu) & 0 & \frac{\beta\Lambda}{\rho\mu}, \\ \alpha & \delta - \frac{2\Lambda}{\kappa\psi} - \eta - \psi & 0, \\ 0 & \eta & -\psi \end{bmatrix} \tag{4.5.19}$$

and

$$\hat{G}(X, Z) = \begin{bmatrix} \left(\frac{\Lambda_c}{\mu_c} - S_c\right)\beta P_1 \\ 0 \\ 0 \end{bmatrix} \tag{4.5.20}$$

The condition $\hat{G}(X, Z) \geq 0$ is satisfied since $\frac{\Lambda}{\mu} \geq S_c \ \forall\ (X, Z) \in \mathbb{R}^4_+$. The off diagonal elements of matrix A are non-negative which implies that A is an M-matrix. When this condition holds, it implies that the use of good agriculture practise, proper handling and the right time to apply the certified spray programs will eradicate and eliminate CBS

Theorem 4.6. *(Castillo-Chávez et al., 2002): The disease free equilibrium* $E_0 = \left(\frac{\Lambda_c}{\mu_c}, 0, 0, 0\right)$ *is globally asymptotically stable (GAS) whenever* $R_0 < 1$ *and conditions (H1) and (H2) are satisfied.*

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4.6 The endemic equilibrium state (E_1) and its stability

The endemic state describes the invasion and persistence of the disease, thus the infected class (I_c) is non-zero. In other words, R_0 will be greater than unity. The equilibrium is obtained by setting the right-hand side of the model system 4.2.1 to zero since the state variables does not change with respect to time at equilibrium.

$$\frac{dS_c}{dt} = \frac{dI_c}{dt} = \frac{dP_1}{dt} = \frac{dP_2}{dt} = 0$$

The endemic equilibrium state of the model system 4.2.1 is

$$E_1 = (S_c^*, I_c^*, P_1^*, P_2^*)$$

satisfying

$$\begin{cases} 0 = \Lambda_c - \lambda S_c - \mu_c S, \\ 0 = \lambda S_c - \alpha_c I_c, \\ 0 = \Lambda_p - \psi P_1, \\ 0 = N\alpha_c I_c - \sigma P_2 \end{cases} \tag{4.6.21}$$

for all $\{S_c^*, I_c^*, P_1^*, P_2^*\} > 0$. The following endemic value are obtained after solving for each state variables of the model system (4.2.1): The susceptible immune defence system endemic value is given by

$$S_c^* = \frac{\Lambda_c}{\lambda P_1^* + \mu_c}$$

From the equation (4.6) it is noted that the susceptible immune defence system is directly proportional to the product of the average time of staying in the susceptible class and its recruitment rate. The recruitment rate is expressed as the proportion of the product of the transmission rate and the appressoria, together with the natural death of the susceptible class. The susceptible immune defence system diminishes through either decay due to natural causes or invasion due to inoculation. While the infected immune

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defence system is given by:

$$I_c^* = \frac{\lambda\psi\Lambda_p\Lambda_c}{\alpha_c\psi\lambda\Lambda_p + \alpha_c\psi^2\mu_c}$$

According to the expression (4.6), transmissibility is density dependent, this follows the proof provided by the equation (4.6) above, which supports the law of mass action due to the nature of the incident and is defined by density-dependent interaction. In this disease development cycle within host, the fungal pathogens in terms of the appressoria also play a major role in the invasion of the immune defence system, and is given by:

$$P_1^* = \frac{\Lambda_p}{\psi}$$

Appressoria is a specialised cell used to invade the host directly proportional to appressoria recruitment rate and time of staying in the same class. The pycnidia population is given by:

$$P_2^* = \frac{P_1\lambda\psi\Lambda_p\Lambda_c}{\sigma(\psi\lambda\Lambda_p + \psi^*\mu_c)}$$

The equation 4.6 indicates that the lifetime of pycnidia within the host and the rate at which infected immune defence system transform into conidia in the environment reduce the endemic equilibrium. The use of controlling strategies can break the life cycle of *Guignardia citricarpa*, thus reducing or eliminating CBS transmission.

4.6.1 The local stability of the endemic equilibrium (E_1)

The endemic equilibrium is considered the non-trivial solution. There is no change either in terms of pathogen growth or decay, the stability of the endemic equilibrium can be computed the in a similar manner, by setting the right-hand sides of the model system 4.2.1 to zero as follows:

$$\frac{dS_c}{dt} = 0 \quad \frac{dI_c}{dt} = 0 \quad \frac{dP_1}{dt} = 0 \quad \frac{dP_2}{dt} = 0 \tag{4.6.22}$$

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and solving for the variables S_c^*, I_c^*, P_1^* and P_2^* in terms of the model parameters from the following equations:

$$\begin{cases} 0 = \Lambda_c - \lambda S_c P_1 - \mu_c S, \\ 0 = \lambda S_c P_1 - \alpha_c I_c, \\ 0 = \Lambda_p - \psi P_1, \\ 0 = \alpha_c I_c - \sigma P_2. \end{cases} \tag{4.6.23}$$

Defined in

$\Phi = \{(S_c^*, I_c^*, P_1^*, P_2^*) \in \mathbb{R}_+^4\} \subset \Omega$

For existence and uniqueness of endemic equilibrium $E_1 = (S_c^*, I_c^*, P_1^*, P_2^*)$,

it should be $S_c^* > 0, I_c^* > 0, P_1^* > 0, P_2^* > 0$

At equilibrium state, the Jacobian is given by

$$J_{(E_1)} = \begin{pmatrix} -(\lambda P_1 + \mu) & 0 & -\lambda S_c & 0 \\ -\lambda P_1 & -\alpha_c & \lambda S_c & 0 \\ 0 & 0 & -\psi_c & 0 \\ 0 & N\alpha & 0 & -\sigma \end{pmatrix} \tag{4.6.24}$$

According to (Tchuenche and Chiyaka, 2009) we can use the additive compound matrices approach of (Li and Muldowney, 1996) to determine the stability of the endemic equilibrium.

Theorem 4.7. *(Li and Muldowney, 1996): An $n \times n$ matrix A is stable if and only if $A^{[2]}$ is stable and $(-1)^n det(A) > 0$*

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Proof.

$$detJ(E_1) = \begin{vmatrix} -(\lambda P_1 + \mu) & 0 & -\lambda S_c & 0 \\ -\lambda P_1 & -\alpha_c & \lambda S_c & 0 \\ 0 & 0 & -\psi_c & 0 \\ 0 & \alpha & 0 & -\sigma \end{vmatrix} \tag{4.6.25}$$

In this approach, the second additive compound matrices is used to analyse the stability of the endemic. From the second additive compound matrix $J^{[2]}_{(E_1)}$, the stability of $J^{[2]}_{(E_1)}$ is established as follows: For the endemic equilibrium $E_1 = (S^*_c, I^*_c, P^*_1, P^*_2)$, let $P = diag(1, -1, -1, -1)$ be the diagonal matrix (Tchuenche and Chiyaka, 2009). Then the matrix $J^{[2]}_{(E_1)}$ is similar to the matrix given by $PJ^{[2]}_{(E_1)}P^{-1}$. Similarly preserves the eigenvalues, then matrix $J^{[2]}_{(E_1)}$ is stable if and only if the matrix $PJ^{[2]}_{(E_1)}P^{-1}$ is stable. Therefore, we apply the following theorem (McKenzie, 1960) establishing that if the matrix A has a negative dominant diagonal, then, A is stable since $(-1)^4 det(J_{(E_1)}) > 0$.

Theorem 4.8. *If $n \times n$ matrix A is dominant diagonally and the diagonal is composed of negative elements $(a_{ii} < 0 \; \forall \; i = 1, ..., n)$, then the real parts of all its eigenvalues are negative, that is, A is stable (Tchuenche and Chiyaka, 2009) (McKenzie, 1960)*

□

4.7 Numerical simulation

This section graphically present numerical solution of the various state variables as they evolve with time using different parameters values. Simulations are run up to 200 days, since the CBS infection period is about $4 - 5$ months.

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Description	Parameter	values	source
Health immune defence system recruitment	Λ_c	10.1300	(Bailey and Gilligan, 2004)
Natural immune defence system diminish	μ_c	0.00001	(Bailey and Gilligan, 2004)
Infected immune defence system transformation rate	α	0.00034	(Bailey and Gilligan, 2004)
Appressoria recruitment rate	Λ_p	20	(Bailey and Gilligan, 2004)
Appressoria death rate	ψ	0.0295	(Bailey and Gilligan, 2004)
Number of pathogens	N	10000	(Bailey and Gilligan, 2004)
Pycinidia death rate	σ	0.00005	(Bailey and Gilligan, 2004)

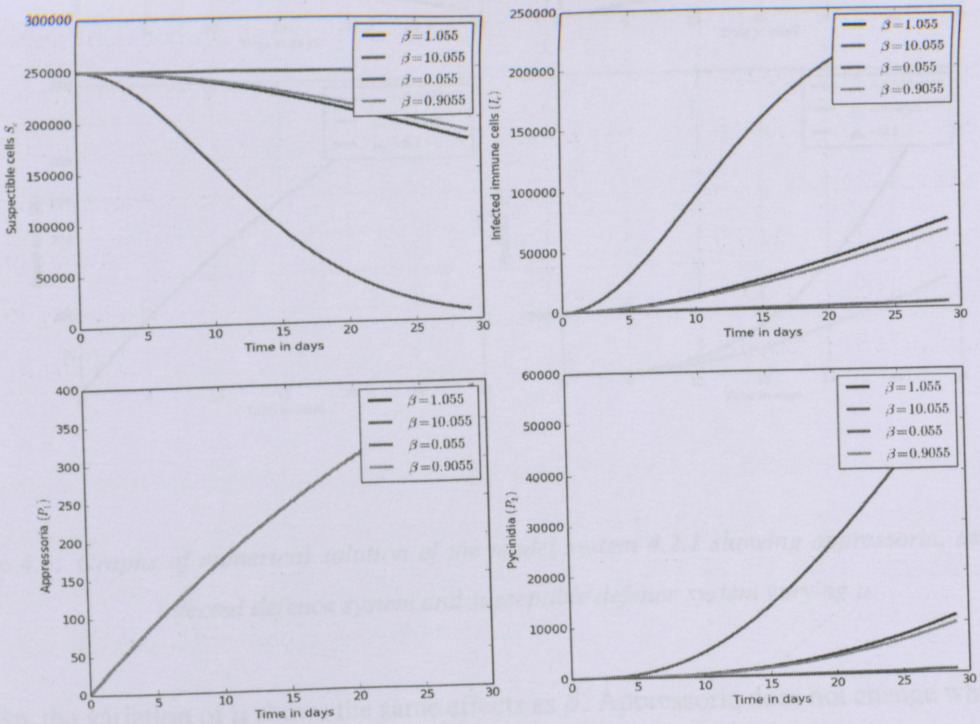

Figure 4.2: *Simulation results showing the evolution of fruit and pathogen population behaviour* β

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The sets of the plot indicate that as β increases so do the infected class and pycnidia while the susceptible immune defence system decrease and appressoria remain unchanged, this implies that controlling of the transmission probability does not have an influence on the appressoria pathogens. Immunologically, controlling strategies should be implemented to target appressoria since it is responsible for invasion.

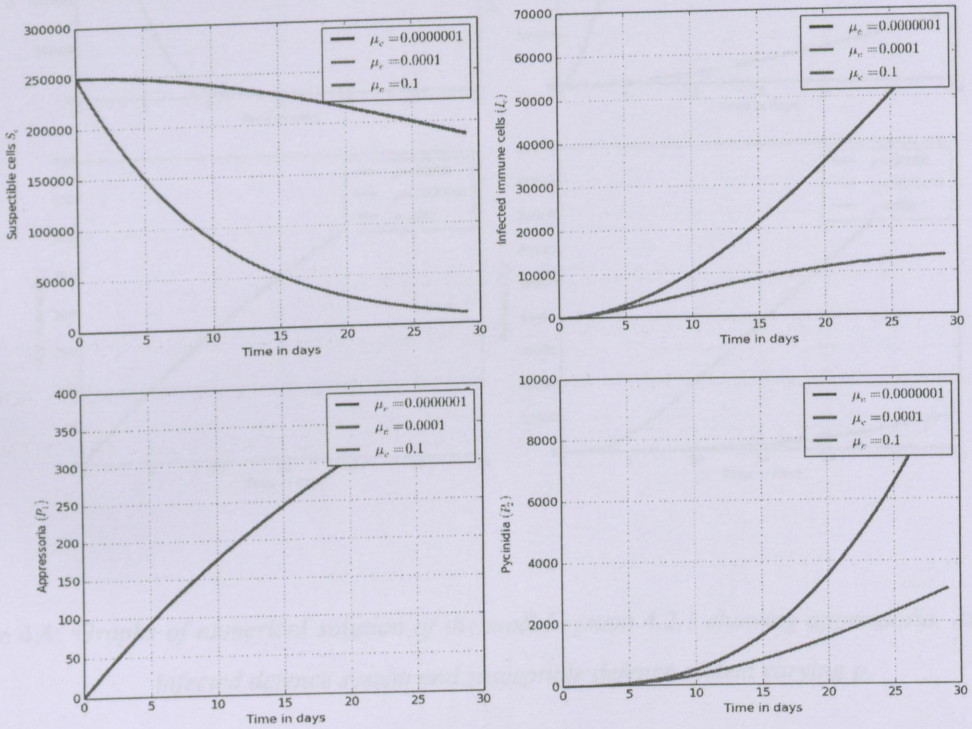

Figure 4.3: *Graphs of numerical solution of the model system 4.2.1 showing appressoria, ascospores, infected defence system and susceptible defence system varying μ*

In this case, the variation of μ shows the same effects as β. Appressoria does not change when varying μ, this shows that the diminishing of pycnidia and the infected defence system does not have an influence on appressoria. An increase in the decay rate of the infected class of immune defence system decreases the pycnidia pathogen pool. This may be useful in explaining a situation whereby infected immune defence system committed suicide before the infection can widely spread.

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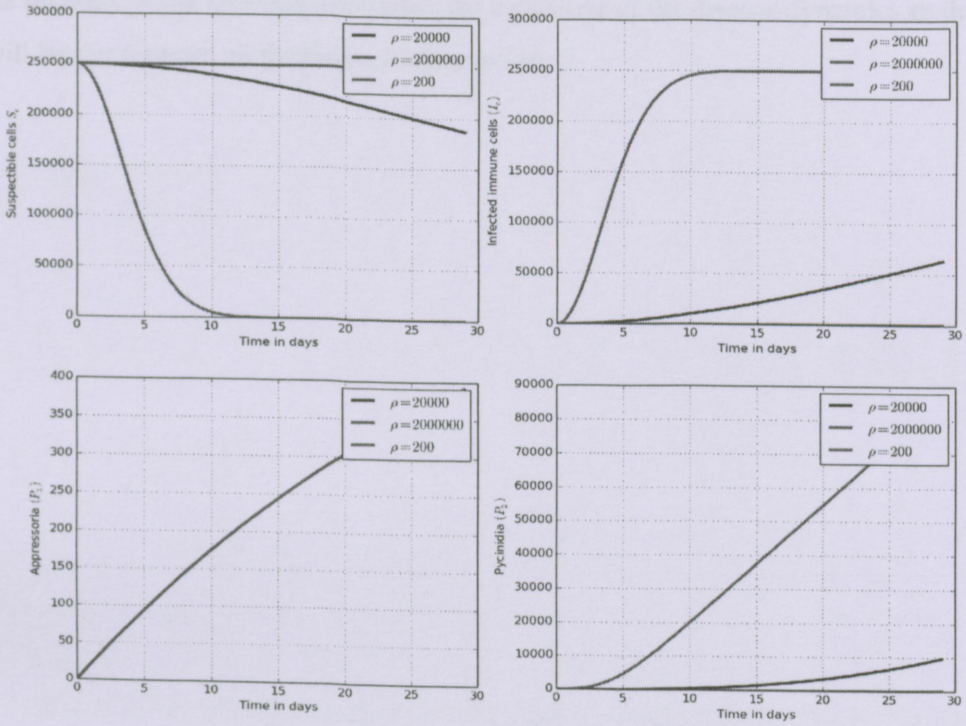

Figure 4.4: *Graphs of numerical solution of the model system 4.2.1 showing appressoria, ascospores, infected defence system and susceptible defence system varying* ρ

The relationship between the susceptible immune defence system and infected immune defence system remains well-posed in all the plots throughout and, however, appressoria shows an insignificant change as ρ varies as the disease evolves with time. The interesting part of the plot is that the more the saturation constant growth the fewer pycnidia and infected class.

4.8 Conclusion

In this chapter, an immunological model was built and analysed to gain insight into the key factors that influence the internal progress development of the CBS disease. The threshold level was identified as a benchmark in which concerned parties will refer to when determining whether the disease will invade

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and persist or just die out. Results from plots bear proofs on the threshold theorem which was computed in previous sections. Plots also help us predict the behaviour of the disease dynamics so that controlling strategy will be put forward on the basis of observation.

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Chapter 5

A MULTI-SCALE MODEL FOR CITRUS BLACK SPOT

5.1 Introduction

Biological dynamics occur at various, different and interlinked scales of complex systems. In this chapter, we look into processes that involve multi-scales by combining the immunological model (4.2.1) consists of an internal plant defence mechanism in the presence of *Guignardia citricarpa* presented in chapter 4 and the epidemiological model (3.2.4) presented in Chapter 3. The objective is to investigate the impact of within-host dynamics on population level quantities such as the prevalence and the basic reproduction number, even pathogen evolution or co-evolution of the pathogen and the host. The inflow of infected class is scaled down to the individual level while the outflow is scaled up to an average population level. The mathematical models are developed to display a theoretical framework which can be used to explicitly identify strong factors influencing disease development both from within and between

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host. Biologically, plants and pathogens co-involved, such that pathogen can recognise the plant by sugars and other molecules it produces. Plants, in turn, can recognise molecules from pathogens. When this happens, plants activate the immune defence system to prevent the wide spread of infection. Some plant molecules bind directly to a plant receptor in the plasma membrane. Animals have a circulating immune system, implying that the immune system can move towards the infected area to defend the host, whereas in plants there is no circulatory system, therefore, immune defence system has to fight own its own. Sometimes when pathogens invade, plants commit suicide to prevent the wide spread of disease and this process is known as hypersensitive reaction (hr).

5.2 Model formulation

The life cycle of *Guignardia citricarpa* inside the host involves different various stages, but in this thesis, for brevity, only the appressoria P_1 and the pycnidia P_A are incorporated in the model. The two major defence mechanism classes to be used in this model are the susceptible immune defence system class S_c and the infected immune defence system class I_c. There are two ways in which the host protects itself, that is, the preformed mechanism and infection-induced response which is taken into consideration but assumed to be insufficient. Thus, the conceptual diagram of the mathematical model of linked between-host and within-host transmission dynamics of *Guignardia citricarpa* is illustrated as follows:

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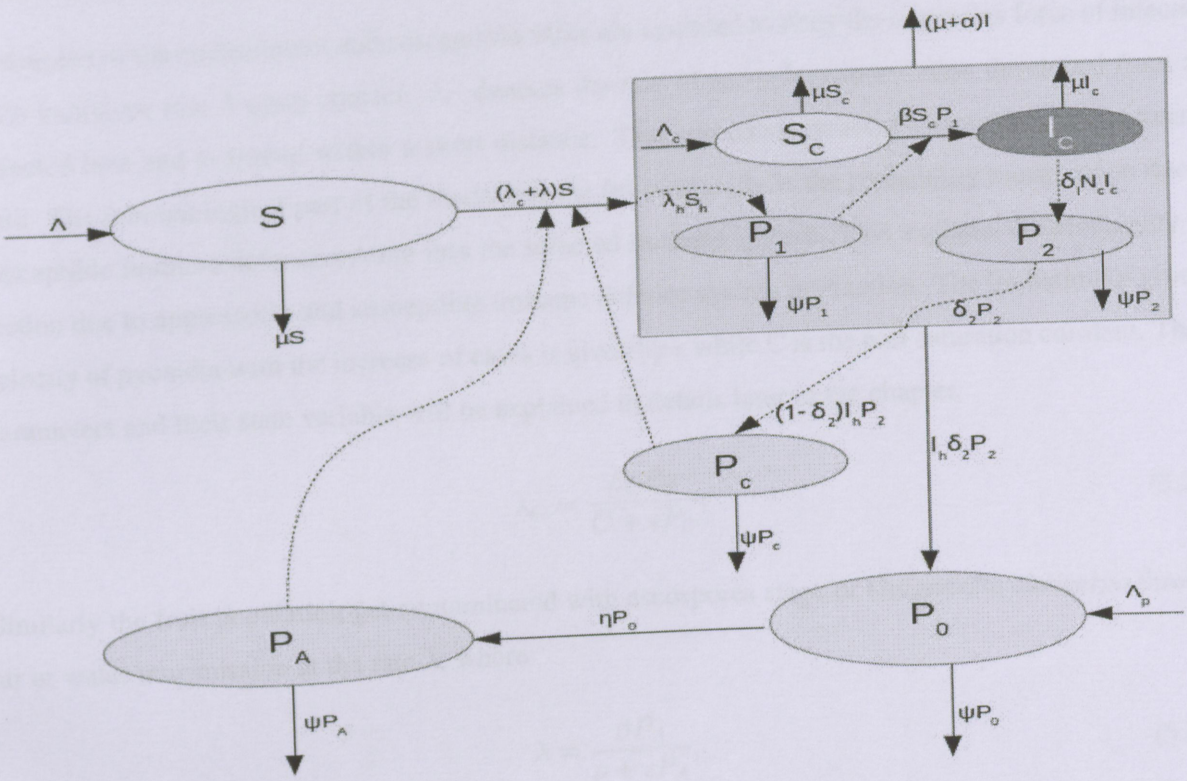

Figure 5.1: *Conceptual model of linked between-host and within-host transmission dynamics of CBS*

This is basically an extension of the CBS epidemiological model in Chapter 3 by incorporating the secondary infection and linking with the within-host transmission dynamics constructed in Chapter 4. The model captures and monitors mechanisms underlying CBS development and it consists of the susceptible fruit population S, made of Λ which is the recruitment rate, μ is the decay rate due to natural causes, λ is the force of infection. The infected fruit population I is made of μ as decay rate due to natural cause, α as decay rate due to disease-induced and λ as the force of infection, increasing the infected class. The conidia class is denoted by P_c consist of the pathogen diminishing rate ψ due to natural causes, the shedding rate δ of the pathogen into the environment. The ascomata class P_0 entails ψ as diminishing rate and η as the rate at which the ascospores are ejected from the fruiting body. The pycnidia P_2 class is reduced by the shedding rate δ and decay rate ψ due to natural causes. However, it is increased by the appressoria population at the rate δ_1. In general, the term $(\lambda_c + \lambda)S$ expresses the average rate of inoculation increasing at a rate proportional to both virulent conidia and ascospores in the presence of susceptible host. The

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pathogens in the environment and susceptible fruits are assumed to obey the saturation force of infection with incidence rate λ upon contact. λ_C denotes the rate of secondary inoculation developed from the infected host and is spread within a short distance. This can either infect the same fruit or the nearby fruit. The immunological part of the model has the following: β_C is the probability transmission rate of susceptible immune defence system into the infected immune system. This explains the probability infection due to appressoria and susceptible immune defence system interaction. The limitation of growth velocity of pycnidia with the increase of cases is given by ϵ while C is the half saturation constant. These parameters and their state variable will be explained in details later in the chapter.

$$\lambda_C = \frac{\beta_C P_C}{C + \epsilon P_C}, \tag{5.2.1}$$

Similarly the fruit population get contaminated with ascospores stage of *Guignardia citricarpa* through air or water transmission at the rate λ, where

$$\lambda = \frac{\beta P_A}{\rho + \epsilon P_A}, \tag{5.2.2}$$

with β being the probability transmission rate, ϵ is the limitation growth velocity of ascospores in the fruiting body on the orchard floor. ρ denotes the half saturation constant. This law shows that the infection is directly proportional to pathogen density when there are favourable climatic conditions. We assume the primary average rate of inoculation of CBS by a single fruit is denoted by the equation below

$$\lambda_g = \frac{\beta_C P_C}{(C + \epsilon P_C) I_g}, \tag{5.2.3}$$

while the secondary inoculation average rate of a single fruit is given by

$$\lambda_h = \frac{\beta P_A}{(\rho + \epsilon P_A) I_h}, \tag{5.2.4}$$

such that

$$\lambda_h S_h = \frac{\beta P_A (S-1)}{(\rho + \epsilon P_A)(I+1)} + \frac{\beta_C P_C (S-1)}{(C + \epsilon P_C)(I+1)}, \tag{5.2.5}$$

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which result in a single infected fruit. We derive such a single transition for primary by

$$(S, I, P_A) \rightarrow (S-1, I+1, P_A), \tag{5.2.6}$$

and for secondary infection is defined by

$$(S-1, I+1, P_A) \rightarrow (S-1, I+1, P_C), \tag{5.2.7}$$

$$\begin{cases} \frac{dS}{dt} = \Lambda - (\lambda + \lambda_C)S - \mu S, \\ \frac{dI}{dt} = (\lambda + \lambda_C)S - (\alpha + \mu)I, \\ \frac{dS_C}{dt} = \Lambda_C - \beta S_C P_1 - \mu_C S_C, \\ \frac{dI_C}{dt} = \beta S_C P_1 - (\delta_1 + \mu_C)I_C, \\ \frac{dP_1}{dt} = \lambda_h S_h - \psi P_1, \\ \frac{dP_2}{dt} = \delta_1 I_C - (\delta_2 + \psi)P_2, \\ \frac{dP_c}{dt} = (1-\delta_2)I_h P_2 - \psi P_C, \\ \frac{dP_0}{dt} = \delta_2 I_h P_2 + \delta P_0(1 - \frac{P_0}{\kappa}) - (\eta + \psi)P_0, \\ \frac{dP_A}{dt} = \eta P_0 - \psi P_A. \end{cases} \tag{5.2.8}$$

The model state variables are summarized in the Table (5.1).

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Term	Description	Initial values
S	Health fruits	500000
I	Infected fruits	0
S_C	Susceptible immune defence system	600000
I_C	Infected immune defence system	0
P_0	Ascomata	0
P_A	Ascospores	0
P_1	Appressoria	0
P_2	Pycinidia	0
P_C	Conidia	0

Table 5.1: *Description of the model state variables of the CBS transmission dynamics*

The first equation models the dynamics of the susceptible host population, showing the primary and secondary force of infections. The recruitment rate of new fruits produced or added external is given by Λ. The preceding term describes the rate at which susceptible individuals form or joins the infected class with λ being the primary, λ_C is the secondary infection and it is assumed to follow a law of saturation incident, and the last term μ denoted natural death rate.

The second equation describes the dynamics of the infected host population. The rate at which infected class increases with respect to the pathogen and susceptible immune defence system collision is given by λ. The infected class population has an outflow due to natural death rate μ and disease induced decay given by α.

Third equation models the citrus susceptible immune defence system dynamics. The source of the new immune defence system in the defence system is given by the rate Λ_C. The outflow rate of infected immune defence system from the susceptible pool of the immune defence system is β while μ is the natural death rate. The ascospores P_A is the density of the initial inoculum.

In the fourth equation, we capture activities of the infected class where an increase in the infected class is shown by the decrease in the susceptible, denoted by β. The presence of the pathogen presents the transmission rate α_C which is assumed to be density dependent and μ_C represents immune defence

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system death rate.

Infected immune defence system eventually transforms to pycnidia at the rate α_C in an infected-pathogen dependent manner. Some pathogens will die due to the host defence mechanism and natural death.

The seventh and eighth equations models the between host pathogens in free living environment where δ_I represents the shedding of pathogen into the environment in the form of conidia from infected hosts, while $\delta P_0(1 - \frac{P_0}{\kappa})$ shows the growth of ascomata in the litter on the ground, which follows a logistic growth. The diminishing of pathogens due to natural causes is denoted by ψ whereas η shows the rate at which ascomata transforms into ascospores. The average lifespan of ascospores is given by $\frac{1}{\psi}$

5.3 Analysis of the model

The model system (5.2.8) is analysed in a feasible region and this model is made up of nine differential equations, the host and the pathogen are shown to have distinct non overlapping life stages. Say;

$$\begin{cases} S(t), I(t) \in \mathbb{R}^2, \\ P_0(t), P_A(t), P_1(t), P_2(t), P_c(t) \in \mathbb{R}^5, \\ S_C(t) I_C(t) \in \mathbb{R}^2 \end{cases} \tag{5.3.9}$$

The total fruit population is given by $N = S + I$ and using the SIP modelling approach, due to the nature of the CBS, that is, it does not confer immunity to re-infection. $N_c(t) = S_C(t) + I_C(t)$ denotes the total average within the host immune defence system. At any time t all classes are greater or equal to zero, this is positive variant, which is mathematical given by $S(t) \geq 0$, $I(t) \geq 0$, $P_0(t) \geq 0$, $P_A(t) \geq 0$, $P_1(t) \geq 0$, $P_2(t) \geq 0$, $P_C(t) \geq 0$.

5.4 Basic properties

In this section, we analysis the basic properties of the model system (5.2.8) and assuming that the state variables and parameters of the model system (5.2.8) are non-negative for all $t > 0$ and further suppose that S is bounded by $\frac{\Lambda}{\mu}$. We can show that all solutions remain bounded. Let $N(t) = S(t) + I(t)$

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represents the total population of the host then $N = S$ at disease free equilibrium. Initial the system has a disease free equilibrium points if $N = \frac{\Lambda}{\mu}$. Considering $N(t) = S(t) + I(t)$. Then $\frac{dN}{dt} = \Lambda - \mu N(t)$ implying that $\lim_{t\to\infty} sup(N(t)) = \frac{\Lambda}{\mu}$. Similarly $N_C = S_C + I_C$, such that

$$\begin{cases} \frac{dN_C}{dt} = \frac{dS_C}{dt} + \frac{dI_C}{dt}, \\ \frac{dN_C}{dt} = \Lambda_C - (\alpha_C + \mu_C)I - \mu_C S_C, \\ \frac{dN_C}{dt} = \Lambda_C - \mu_C(S + I) - \alpha_C I, \\ \frac{dN_C}{dt} \leq \Lambda_C - N\mu_C \end{cases} \tag{5.4.10}$$

Therefore

$$\lim_{t\to\infty} sup(N) \leqslant \frac{\Lambda_c}{\mu_c}$$

The same technique used on (5.4.10) can be applied for pathogen population. The feasible region for the model system (5.2.8) is

$$\Omega_1 = \left\{ \left((S, I) \in \mathbb{R}^2_+ : S + I \leqslant \frac{\Lambda}{\mu} \right), \left((S_c, I_c) \in \mathbb{R}^2_+ : S_c + I_c \leqslant \frac{\Lambda_c}{\mu_c} \right) \right\},$$

$$\Omega_2 = \left\{ \left((P_1, P_2, P_c, P_0, P_A) \in \mathbb{R}^5_+ : P_1 + P_2 + P_c + P_0 + P_A \leqslant \frac{\eta\delta_2}{\eta + \psi} \right) \right\}$$

The dynamics of the model is considered in $\Omega = \Omega_1 + \Omega_2$ since the global attractor of the model system (5.2.8) is contained in Ω.

Lemma 5.1. *The set Ω is positively invariant.*

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Proof. In Ω, we have

$$\begin{cases} S = 0 \Rightarrow \dfrac{dS}{dt} = \Lambda, \\ I = 0 \Rightarrow \dfrac{dI}{dt} = (\lambda + \lambda_C)S, \\ S_c = 0 \Rightarrow \dfrac{dS_c}{dt} = \Lambda_C, \\ I_c = 0 \Rightarrow \dfrac{dI_c}{dt} = \beta S_C P_1, \\ P_1 = 0 \Rightarrow \dfrac{dP_1}{dt} = \lambda_h S_h, \\ P_2 = 0 \Rightarrow \dfrac{dP_2}{dt} = \delta_1 I_C, \\ P_c = 0 \Rightarrow \dfrac{dP_c}{dt} = (1 - \delta_2) I_h P_2, \\ P_0 = 0 \Rightarrow \dfrac{dP_0}{dt} = \delta_2 I_h P_2, \\ P_A = 0 \Rightarrow \dfrac{dP_A}{dt} = \eta P_0. \end{cases} \tag{5.4.11}$$

Hence all the parameters are positive and any vector of field in $\partial\Omega$, the proper boundary of Ω, is tangent, the invariant region Ω of biological interest is positive and all solution of the model system (5.2.8) which enters Ω remains there. □

5.5 The disease free equilibrium point and its stability.

The equilibrium states sometimes referred to as the time independent solutions are either disease free (E_0) or endemic (E_e) in nature. This means at either disease free or endemic the equations are equated to zero. These steady states determine the behaviour of the CBS even when there are disturbed in the

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dynamics. Equating the right hand side to zero allows the results of the disease free or endemic steady state such that $dS = dI = dS_c = dI_C = dP_1 = dP_2 = dP_C = dP_0 = dP_A = 0$. At disease free equilibrium, we obtain

$$E_0 = (S, I, S, I_c, P_1, P_2, P_c, P_0, P_A) = \left(\frac{\Lambda}{\mu}, 0, \frac{\Lambda_C}{\mu_C}, 0, 0, 0, 0, 0, 0\right) \tag{5.5.12}$$

5.6 Reproduction number of the dynamical system of Citrus Black Spot

The reproduction number R_0 sometimes known as the reproductive ratio is commonly used to measure the epidemic size. By definition in this, it is the average number of secondary infections of citrus fruit generated by one primary ascospore introduced into an entirely susceptible fruit population over the CBS infectious period. This helps to determine whether or not the CBS will invade and persist. In general, if the value of $R_0 < 1$, then CBS will die out, but if $R_0 < 1$ then it is hard to control the epidemic (Castillo-Chávez et al., 2002). In this study, the use of the next generation operator approach by (Castillo-Chávez et al., 2002) (Diekmann et al., 2009) is used to determine the reproductive number of the CBS, defined as the spectral radius of the next generation operator. This is possible by classifying heterogeneous fruits into similar subgroups according to infection related characteristics. In this case the equations of the model system 5.2.8 are arranged according to disease status, in the form:

$$\begin{cases} \dfrac{dX}{dt} = f(X, Y, Z), \\ \dfrac{dY}{dt} = g(X, Y, Z), \\ \dfrac{dZ}{dt} = h(X, Y, Z) \end{cases} \tag{5.6.13}$$

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where $X \in (S, S_C, P_0)$ denote the non infectious classes, $Y \in (I, I_C, P_2)$ represents infected but not infectious and $Z \in (P_1, P_C, P_A)$ is the infected and infectious classes and $h(X, 0, 0) = 0$. At disease free state, we let $U_0 = \left(\frac{\Lambda}{\mu}, 0, \frac{\Lambda_C}{\mu_c}, 0, 0, 0, 0, 0\right) \in \mathbb{R}^9$ represents the disease free state (Brauer and Castillo-Chavez, 2001) (Castillo-Chávez et al., 2002) (Driessche and Watmough, 2002). Where $U_0 = (X^*, 0, 0)$, $f(X^*, 0, 0) = g(X^*, 0, 0) = h(X^*, 0, 0) = 0$. Assume that the equation $g(X^*, Y, Z) = 0$ implicitly determines function $Y = \tilde{g}(X^*, Z)$. Let $A = D_Z h(X^*, \tilde{g}(X^*, 0), 0)$ and further assume that A can be written in the form $A = M - D$, where M is the inflow of infected class and D is the outflow of infected class.

$$\tilde{g}_1(X^*, Z) = \frac{\left(\frac{\beta P_A}{\rho + \epsilon P_A} + \frac{\beta_C P_C}{C + \epsilon P_C}\right)\Lambda}{(\mu + \alpha)\mu},$$

$$\tilde{g}_2(X^*, Z) = \frac{\beta P_1 \Lambda_c}{(\delta_1 + \mu_c)\mu_c},$$

$$\tilde{g}_3(X^*, Z) = \frac{\delta_1 \beta \Lambda_C P_1}{(\delta_2 + \psi)(\delta_1 + \mu_C)\mu_C},$$

$$h_1(X^*, \tilde{g}(X^*, Z), Z) = \frac{\beta P_A (S - 1)}{(\rho + \epsilon P_A)(I + 1)} + \frac{\beta_c P_c (S - 1)}{(C + \epsilon P_c)(I + 1)} - \psi P_1,$$

$$h_2(X^*, \tilde{g}(X^*, Z), Z) = (1 - \delta_2) I_h P_2 - \psi P_c,$$

$$h_3(X^*, \tilde{g}(X^*, Z), Z) = \eta \left(\frac{-b + \sqrt{b^2 + 4ac}}{2a\psi}\right) - \psi P_A,$$

where

$$\begin{cases} a = \dfrac{\delta}{\kappa}, \\ \\ b = \eta + \psi - \delta, \\ \\ c = \delta_2 I_h P_2 \ = \ -\delta_2 \cdot \dfrac{\left(\frac{\beta P_A}{\rho + \epsilon P_A} + \frac{\beta_C P_C}{C + \epsilon P_C}\right)\Lambda + (\mu + \alpha)\mu}{(\mu + \alpha)\mu} \cdot \dfrac{\delta_1 \beta \Lambda_C P_1}{(\delta_2 + \psi)(\delta_1 + \mu_C)\mu_C}, \end{cases} \tag{5.6.14}$$

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$$\begin{cases} \dfrac{\partial h_1}{\partial P_1} = -\psi, & \dfrac{\partial h_1}{\partial P_c} = \dfrac{\beta_c(\Lambda - 2\mu)}{2C\mu}, & \dfrac{\partial h_1}{\partial P_A} = \dfrac{\beta(\Lambda - \mu)}{\rho\mu}, \\ \dfrac{\partial h_2}{\partial P_1} = \dfrac{(1-\delta_2)\delta_1\beta\Lambda_c}{(\delta_2+\psi)(\delta_1+\mu_c)\mu_c}, & \dfrac{\partial h_2}{\partial P_c} = -\psi, & \dfrac{\partial h_2}{\partial P_A} = 0, \\ \dfrac{\partial h_3}{\partial P_1} = \dfrac{\eta\delta_2\delta_1\beta\Lambda_c}{\psi(\delta_1+\mu_c)\mu_c(\eta+\psi-\delta)}, & \dfrac{\partial h_3}{\partial P_c} = 0, & \dfrac{\partial h_3}{\partial P_A} = 0. \end{cases} \tag{5.6.15}$$

Let

$$A = D_Z h(X^*, \tilde{g}(X^*, 0), 0) \tag{5.6.16}$$

where A can be composed by M and D, as shown below

$$A = M - D \tag{5.6.17}$$

Therefore A is defined by the following matrix

$$A = \begin{pmatrix} -\psi & \dfrac{\beta_c(\Lambda-\mu)}{C\mu} & \dfrac{\beta(\Lambda-\mu)}{\mu\rho} \\ \dfrac{(1-\delta_2)\delta_1\beta\Lambda_c}{(\delta_2+\psi)(\delta_1+\mu_c)\mu_c} & -\psi & 0 \\ \dfrac{\eta\delta_2\delta_1\beta\Lambda_c}{\mu_c(\delta_1+\mu_c)(\eta+\psi-\delta)} & 0 & -\psi \end{pmatrix} \tag{5.6.18}$$

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where

$$M = \begin{pmatrix} 0 & \frac{\beta_c(\Lambda-\mu)}{C\mu} & \frac{\beta(\Lambda-\mu)}{\mu\rho} \\ \frac{(1-\delta_2)\delta_1\beta\Lambda_c}{(\delta_2+\psi)(\delta_1+\mu_c)\mu_c} & 0 & 0 \\ \frac{\eta\delta_2\delta_1\beta\Lambda_c}{\mu_c(\delta_1+\mu_c)(\eta+\psi-\delta)} & 0 & 0 \end{pmatrix} \tag{5.6.19}$$

$$D = \begin{pmatrix} \psi & 0 & 0 \\ 0 & \psi & 0 \\ 0 & 0 & \psi \end{pmatrix} \tag{5.6.20}$$

The basic reproduction number is the dominant eigenvalue of the matrix $T = MD^{-1}$, denoted by

$$R_0 = \rho(T) \tag{5.6.21}$$

Therefore, the computation of the threshold requires the evaluation of the product of M and D^{-1}

$$D^{-1} = \begin{bmatrix} \frac{1}{\psi} & 0 & 0, \\ 0 & \frac{1}{\psi} & 0 \\ 0 & 0 & \frac{1}{\psi} \end{bmatrix} \tag{5.6.22}$$

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such that

$$MD^{-1} = \begin{bmatrix} 0 & \frac{\beta_c(\Lambda-\mu)}{C\mu\psi} & \frac{\beta(\Lambda-\mu)}{\mu\rho\psi} \\ \frac{(1-\delta_2)\delta_1\beta\Lambda_c}{(\delta_2+\psi)(\delta_1+\mu_c)\mu_c\psi} & 0 & 0 \\ \frac{\eta\delta_2\delta_1\beta\Lambda_c}{\mu_c(\delta_1+\mu_c)(\eta+\psi-\delta)\psi} & 0 & 0 \end{bmatrix} \tag{5.6.23}$$

The matrix (5.6.23) allows us to draw the following characteristic equation to determine the maximum eigenvalue which represents the threshold value.

$$|T - \lambda I| \tag{5.6.24}$$

where I is the identity matrix, so that

$$\begin{vmatrix} -\lambda & \frac{\beta_c(\Lambda-\mu)}{C\mu\psi} & \frac{\beta(\Lambda-\mu)}{\mu\rho\psi} \\ \frac{(1-\delta_2)\delta_1\beta\Lambda_c}{(\delta_2+\psi)(\delta_1+\mu_c)\mu_c\psi} & -\lambda & 0 \\ \frac{\eta\delta_2\delta_1\beta\Lambda_c}{\mu_c(\delta_1+\mu_c)(\eta+\psi-\delta)\psi} & 0 & -\lambda \end{vmatrix} = 0 \tag{5.6.25}$$

Considering the cubic depression approach introduced by (Ebstein, 1923) to solve cubic polynomial obtained from the characteristic equation, as follows

$$\lambda^3 - \lambda\left[\left(\frac{C}{\psi}\times\frac{F}{\psi}\right) + \left(\frac{B}{\psi}+\frac{D}{\psi}\right)\right] = 0 \tag{5.6.26}$$

solving for λ results in

$$\lambda = 0 \quad or \quad \lambda = \sqrt{\left[\left(\frac{C}{\psi}\times\frac{F}{\psi}\right) + \left(\frac{B}{\psi}+\frac{D}{\psi}\right)\right]} \tag{5.6.27}$$

where

$$B = \frac{\beta_c(\Lambda-2\mu)}{2C\mu}$$

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$$C = \frac{\beta(\Lambda - \mu)}{\mu\rho}$$

$$D = \frac{(1-\delta_2)\delta_1\beta\Lambda_c}{(\delta_2+\psi)(\delta_1+\mu_c)\mu_c}$$

$$F = \frac{\eta\delta_2\delta_1\beta\Lambda_c}{\mu_c(\delta_1+\mu_c)(\eta+\psi-\delta)}$$

Hence, the basic reproduction number is given by

$$R_0 = \sqrt{\left(\frac{\beta(\Lambda-\mu)}{\mu\rho\psi}\cdot\frac{\eta\delta_2\delta_1\beta\Lambda_c}{\mu_c(\delta_1+\mu_c)(\eta+\psi-\delta)\psi}\right)+\left(\frac{\beta_c(\Lambda-\mu)}{C\mu\psi}\cdot\frac{(1-\delta_2)\delta_1\beta\Lambda_c}{(\delta_2+\psi)(\delta_1+\mu_c)\mu_c\psi}\right)} \quad (5.6.28)$$

The efforts of such computation are to bring invasion and persistence to control by means of finding parameter conditions responsible for transmission and adjust the mechanism to promote the existence of disease-free equilibrium. The epidemiological parameters all contribute to the CBS development. On that note, we strongly recommend, diminishing the ascomata in the environment and reduce susceptibility through using preventative measures such as spraying before fruit onset. Educating farm workers on good agricultural practise can also help reduce the basic reproduction number. The immunological parameter has influence over epidemiological transmission dynamics. The transformation of appressoria to conidia through pycnidia seem to be significantly changing all classes both immunologically and epidemiologically

5.7 Local stability of disease free equilibrium

This is one of the crucial aspects when analysing epidemiological models in determining the asymptotic behaviour of the model system (5.2.8) solutions based on stability of its equilibria (Tchuenche and Chiyaka, 2009). Epidemiological models commonly subsist of a disease free equilibrium or at least one endemic equilibrium. At disease free equilibrium all classes are assumed to be empty except the susceptible one, this simple describes the absence of infection where the susceptible class is nearly equal to the total population of fruits. The local stability of the disease free equilibrium is determined is based on the

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basic reproduction number R_0. The Jacobian matrix at disease equilibrium free E_0 is

$$J(E_0) = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 & \frac{\beta_c \Lambda}{C\mu} & 0 & \frac{\beta\Lambda}{\rho\mu} \\ 0 & -(\alpha+\mu) & 0 & 0 & 0 & 0 & \frac{\beta_c \Lambda}{C\mu} & 0 & \frac{\beta\Lambda}{\rho\mu} \\ 0 & 0 & -\mu_c & 0 & \frac{\beta\Lambda_c}{\mu_c} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\delta_1+\mu_c) & \frac{\beta\Lambda_c}{\mu_c} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\psi & 0 & \frac{\beta_c(\Lambda-2\mu)}{2C\mu} & 0 & \frac{\beta(\Lambda-\mu)}{\rho\mu} \\ 0 & 0 & 0 & \delta_1 & 0 & -(\delta_2+\psi) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & (1-\delta_2) & -\psi & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \delta_2 & 0 & \delta-\eta-\psi & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \eta & -\psi \end{pmatrix} \tag{5.7.29}$$

Using row elementary operation, gives the eigenvalues of J_{E_0} are: $\lambda_1 = -\mu$, $\lambda_2 = -(\alpha+\mu)$, $\lambda_3 = -\mu_c$, $\lambda_4 = -(\delta_1+\mu_c)$, $\lambda_5 = -\psi$, $\lambda_6 = -(\delta_2+\psi)$, $\lambda_7 = -\psi$, $\lambda_8 = \delta-(\eta+\psi)$, $\lambda_9 = -\psi$ for $\lambda_8 = \delta-(\eta+\psi)$ to be negative $(\eta+\psi) > \delta$. It is clear that all of the eigenvalues are negative and some have negative real parts. Thus we have established the following result

Lemma 5.2. *The DFE of the model system (5.2.8) is locally asymptotically stable if* $R_0 < 1$ *and if* $R_0 > 1$ *then it is unstable.*

Proof. An equilibrium is asymptotically stable if all eigenvalues are negative or have negative real parts; it is unstable if at least one eigenvalue has a positive real part. Since the eigenvalues of the Jacobian

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matrix evaluated at disease free equilibrium are negative. It follows that the DFE is locally asymptotically stable whenever $R_0 < 1$ □

5.8 Global stability of disease free equilibrium.

According to (Castillo-Chávez et al., 2002) the disease-free equilibrium is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. Following the same approach, we are going to establish the global stable for Citrus Black Spot at disease free equilibrium. The model can be written in the form

$$\begin{cases} \dfrac{dX}{dt} = F(X, Z), \\ \\ \dfrac{dZ}{dt} = G(X, Z) \end{cases} \tag{5.8.30}$$

with $G(X, 0) = 0$, where $X \in \mathbb{R}^3$ denotes the classes of non infected individuals while $Z \in \mathbb{R}^6$ shows the classes of infected and infectious.

Let

$$U_0 = (X^*, 0) = \left(\frac{\Lambda}{\mu}, 0, \frac{\Lambda_c}{\mu_c}, 0, 0, 0, 0, 0, 0\right)$$

be the disease free equilibrium of the model system (5.2.8). The conditions (**H1**) and (**H2**) must be satisfied in order to guarantee the globally asymptotically stable.

(**H1**) For $\dfrac{dX}{dt} = F(X^*, 0), X^*$ is globally asymptotically stable (g.a.s)

(**H2**) $G(X, Z) = AZ - \tilde{G}(X, Z), \tilde{G}(X, Z) \geq 0$ for $(X, Z) \in \mathbb{R}^9_+$ where $A = D_z G(X^*, 0)$ is an M-matrix in which the off diagonal elements are not negative and $\mathbb{R}^9_+$ is the region where the model makes biological sense. The following theorem holds if these condition are met.

Theorem 5.3. *The fixed point U_0 is a globally stable equilibrium of (5.2.8) whenever $R_0 < 1$ and provided that conditions (H1) and (H2) are satisfied.*

$$F(X, 0) = \begin{bmatrix} \Lambda - \mu S, \\ \\ \Lambda_c - \mu_c S_c \end{bmatrix} \tag{5.8.31}$$

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Matrix A is given by

$$A = \begin{pmatrix} -(\alpha+\mu) & 0 & 0 & 0 & \frac{\beta_c\Lambda}{C\mu} & \frac{\beta\Lambda}{\rho\mu} \\ 0 & -(\delta_1+\mu_c) & \frac{\beta\Lambda_c}{\mu_c} & 0 & 0 & 0 \\ 0 & 0 & -\psi & 0 & \frac{\beta_c(\Lambda-2\mu)}{2C\mu} & \frac{\beta(\Lambda-\mu)}{\rho\mu} \\ 0 & \delta_1 & 0 & -(\delta_2+\psi) & 0 & 0 \\ 0 & 0 & 0 & (1-\delta_2) & -\psi & 0 \\ 0 & 0 & 0 & 0 & 0 & -\psi \end{pmatrix} \tag{5.8.32}$$

and

$$\begin{aligned} \tilde{G}(X,Z) &= AZ - G(X,Z) \\ &= \begin{bmatrix} \left(\frac{\Lambda}{\mu\rho} - \frac{S}{\rho+\epsilon P_A}\right)\beta P_A \\ \left(\frac{\Lambda-\mu}{\rho\mu} - \frac{S-1}{\rho+\epsilon P_A}\right)\beta P_A \\ 0 \\ 0 \\ 0 \end{bmatrix} \end{aligned}$$

Therefore if $\tilde{G}(X,Z) \geq 0$ for all $(X,Z) \in \mathbb{R}^8_+$. $\frac{\Lambda}{\mu\rho} \geq \frac{S}{\rho+\epsilon P_A}, \frac{\Lambda-\mu}{\rho\mu} \geq \frac{S-1}{\rho+\epsilon P_A}$ whenever $\Lambda \geq \mu$ and $\Lambda_c \geq \mu_c$, for all (X,Z) It is shown that A is an M-matrix, since the off diagonal elements of A are

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non-negative. The result is summarised by the following theorem

Theorem 5.4. *The disease free equilibrium point of the model system 5.2.8 is globally asymptotically stable whenever* $R_0 < 1$ *and assumption (H1) and (H2) are satisfied.*

This implies that good agriculture practise will either slow down or eradicate and contain CBS. If the assumption is not satisfied.

5.9 The endemic equilibrium state its stability

The model system (5.2.8) has an endemic equilibrium point (EE), where the infected classes I, I_c are non-zero, the citrus produce is contaminated by CBS and it is given by

$$\hat{E}_1 = (S^*, I^*, S_c^*, I_c^*, P_1^*, P_2^*, P_0^*, P_A^*) \tag{5.9.33}$$

we give these endemic points in details and explain their expressions. The susceptible endemic equilibrium value is given by

$$S^* = \frac{\Lambda}{\lambda + \lambda_c + \mu} \tag{5.9.34}$$

The susceptible endemic value given in (5.4.11) is the proportion of the entire population of fruits that remains uncontaminated throughout the CBS lifetime no matter how severe and transmissibility the pathogen may be. The susceptible class endemic value is directly proportional to its recruitment rate and the CBS average time of staying in the susceptible class. Contaminated fruit leaves the susceptible class either through decay or contamination and this value is given by:

$$I^* = \frac{(\lambda + \lambda_C)S^*}{\mu + \alpha} \tag{5.9.35}$$

The equation (5.9.35) implies that at equilibrium the infected immune defence system is directly proportion to the product of average time of staying in the infected class, the rate of infection of susceptible both primary and secondary infection and the number of susceptible. The preceding variable denotes the

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susceptible immune defence system within the host during the disease life time.

$$S_c^* = \frac{\Lambda_c}{\beta P_1^* + \mu_c} \tag{5.9.36}$$

The equation (5.9.36) denotes that the susceptible immune defence system is inversely proportional to the probability of transmission, which is density dependent on appressoria and directly proportional to the average of stay in the susceptible class. This equation also relates the epidemiological model and the immunological model with respect to immuno-epidemiology mechanisms underlying transmission. The infected within-host immune system is expressed as given below:

$$I_c^* = \frac{\beta P_1^* S_c^*}{(\delta_1 + \mu_C)} \tag{5.9.37}$$

The infected immune defence system within the host at endemic equilibrium state is influenced by the average life span of pycnidia and the force of infection due to appressoria. The infected immune defence system is also directly proportional to the product of susceptible immune defence system and its force of infection. The appressoria population endemic value is given by:

$$P_1^* = \frac{(\lambda_h(S^* - 1) + \lambda_g(S^* - 2))}{\psi} \tag{5.9.38}$$

From the equation (5.9.38) it is clear that there is a dependent relationship between appressoria and susceptible immune defence system which promote invasion and persistence to occur within the host. In other terms, the value of appressoria is directly proportional to the average time of staying in the of infected fruits and the constant saturation of the pathogen. A direct relationship that exists between appressoria and pycnidia is shown below;

$$P_2^* = \frac{\alpha_c I_c^*}{(\delta_2 + \psi)} \tag{5.9.39}$$

The endemic equilibrium value for pycinidia shown on (5.9.39) implies that it is directly proportional to the average time of staying in conidia and transformation of *Guignardia citricarpa* from appressoria to

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pycinidia. The endemic value of ascomata in the environment is given by,

$$P_0^* = \frac{(\delta - \eta - \psi) + \sqrt{(\delta - \eta - \psi)^2 + 4\frac{\delta}{\kappa}\alpha_c I_h^*}}{\frac{2\delta}{\kappa}} \tag{5.9.40}$$

The ascomata is directly proportional to the product of the average time of staying in the ascomata class, the pathogen shedding of infected host and the life span of ascomata propagules. Ascospores which is the density of initial inoculum can be shown using the following:

$$P_A = \frac{\eta P_0^*}{\psi} \tag{5.9.41}$$

The ascospores endemic value is directly proportional to the product of its average life span and the ascomata in the environment.

5.10 Existence and uniqueness endemic equilibrium

This section shows that for existence and uniqueness of endemic equilibrium $E_1 = (S^*, I^*, S_c^*, I_c^*, P_A^*, P_1^*, P_2^*, P_c^*,)$. The model system 5.2.8 has no periodic orbits and its unique endemic equilibrium of the model which is both locally and globally asymptotically stable whenever $R_0 > 1$. To prove the existence of periodic solutions, Using the Poincare-Bendixon theorem (Ilin and Kozma, 2006), we reduce the model system 5.2.8 into a 2 dimensional, by eliminating all other variables and remain with the infected I and the ascospores P_A classes as follows:

$$\begin{cases} \dfrac{dI}{dt} = (\lambda + \lambda_C)S - (\alpha + \mu)I, \\ \dfrac{dP_A}{dt} = \eta P_0 - \psi P_A. \end{cases} \tag{5.10.42}$$

Let

$$\begin{cases} X = (\lambda + \lambda_C)S - (\alpha + \mu)I \\ Y = \eta P_0 - \psi P_A. \end{cases} \tag{5.10.43}$$

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Consider the Dulc's function $D = \frac{1}{IP_A}$ where $I > 0, P_A > 0$. Then,

$$\frac{\partial(DX)}{\partial I} + \frac{\partial(DY)}{\partial P_A} = \frac{M - N}{\Lambda\kappa(\lambda + \lambda_C)^2(\eta(b) + (b)^2 + 4))} + \frac{ADQ}{(\lambda + \lambda_c)(\rho + \epsilon P_A)^2} \tag{5.10.44}$$

where $M = \Lambda\kappa(\eta(b+(b+4ac)^2))(\delta\psi\mu(\mu+\alpha)\mu(\mu+\alpha))(\lambda+\lambda_c)$, $N = \psi\delta\mu(\mu+\alpha)((\lambda+\lambda_C)\Lambda-\mu(\mu+\alpha)I)(\lambda+\lambda_c)(b+b^{\frac{1}{2}})$, $b = (\delta-\eta-\psi)$, $a = \frac{\delta}{\psi}$, $Q = ((\rho+\epsilon P_A)\beta - \beta P_A\epsilon)$ and $c = \alpha_c(I-1)$. Since $\frac{\Lambda\kappa(\eta(b+(b+4ac)^2))(\delta\psi\mu(\mu+\alpha)\mu(\mu+\alpha))(\lambda+\lambda_c)}{\psi\delta\mu(\mu+\alpha)((\lambda+\lambda_C)\Lambda-\mu(\mu+\alpha)I)(\lambda+\lambda_c)(b+b^{\frac{1}{2}})} > 1$ by Dulac's criterion, There are no periodic orbits in $\Omega \mid \Omega*$, where $\Omega* = \{(S, I, S_c, I_c, P_1, P_2, P_c, P_0, P_A) \in \Omega\}$. Since Ω is positively invariant and the endemic equilibrium exists whenever $R_0 > 1$. The absence of periodic orbits in Ω implies that the unique endemic equilibrium of the special case of the model system is globally asymptotically stable whenever $R_0 > 1$

5.10.1 The Local Stability of the Endemic Equilibrium E_1

At equilibrium state, there is no change with respect to time of any dynamical system. The model system (5.2.8)'s endemic equilibrium is computed by using the Centre Manifold Theory, in which state variables are changed as follows:$S = x_1$, $I = x_2$, $S_c = x_3$, $I_c = x_4$, $P_1 = x_5$, $P_2 = x_6$, $P_c = x_7$, $P_0 = x_8$, and $P_A = x_9$ such that $N = S + I$, $N_c = S_c + I_c$ and $P = P_1 + P_2 + P_c + P_0 + P_A$. Where $\mathbf{x} = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8, x_9)^T$, therefore, as a result the model is given by $\frac{d\mathbf{x}}{dt} = F(\mathbf{x})$, where

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$F(\mathbf{x}) = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8, f_9,)$ and

$$\begin{cases} \frac{dx_1}{dt} = \Lambda - (\lambda + \lambda_C)x_1 - \mu x_1 \\ \frac{dx_2}{dt} = (\lambda + \lambda_C)x_2 - (\alpha + \mu)x_2 \\ \frac{dx_3}{dt} = \Lambda_C - \beta x_3 x_5 - \mu_C x_3 \\ \frac{dx_4}{dt} = \beta x_3 x_5 - (\delta_1 + \mu_C)x_4 \\ \frac{dx_5}{dt} = \lambda_h(x_1 - 1) + \lambda_g(x_1 - 2) - \psi x_5 \\ \frac{dx_6}{dt} = \delta_1 x_4 - (\delta_2 + \psi)x_6 \\ \frac{dx_7}{dt} = (1 - \delta_2)(x_2 + 1)x_6 - \psi x_7 \\ \frac{dx_8}{dt} = \delta_2(x_2 + 1)x_6 + \delta x_8(1 - \frac{x_8}{\kappa}) - (\eta + \psi)x_8 \\ \frac{dx_9}{dt} = \eta x_8 - \psi x_9 \end{cases} \tag{5.10.45}$$

Where,

$$\lambda = \frac{\beta x_9}{\rho + \epsilon x_9}, \quad \lambda_c = \frac{\beta_c x_7}{C + \epsilon x_7} \tag{5.10.46}$$

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The model has to be evaluated at disease free equilibrium E_0 denoted by $J(E_0)$ given by

$$J(E_0) = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 & \frac{\beta_c \Lambda}{C\mu} & 0 & \frac{\beta\Lambda}{\rho\mu} \\ 0 & -(\alpha+\mu) & 0 & 0 & 0 & 0 & \frac{\beta_c \Lambda}{C\mu} & 0 & \frac{\beta\Lambda}{\rho\mu} \\ 0 & 0 & -\mu_c & 0 & \frac{\beta\Lambda_c}{\mu_c} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\delta_1+\mu_c) & \frac{\beta\Lambda_c}{\mu_c} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\psi & 0 & \frac{\beta_c(\Lambda-2\mu)}{2C\mu} & 0 & \frac{\beta(\Lambda-\mu)}{\rho\mu} \\ 0 & 0 & 0 & \delta_1 & 0 & -(\delta_2+\psi) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & (1-\delta_2) & -\psi & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \delta_2 & 0 & \delta-\eta-\psi & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \eta & -\psi \end{pmatrix} \tag{5.10.47}$$

The Jacobian matrix is transformed with bifurcation point β^* which has a simple zero eigenvalue. Hence an extension for Lyapunov that is the centre manifold theory is suitable in this case near $\beta = \beta^*$. (Garira et al., 2014) used the same theory to determine the local stability of the endemic equilibrium point, which is assumed to be the same as the endemic equilibrium point of the original model for the transmission rate.

Theorem 5.5. *Consider the following general system of ordinary differential equations with parameter* ϕ*:*

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$$\frac{dx}{dt} = f(x, \phi), f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}, f : \mathbb{C}^2(\mathbb{R}^2 \times \mathbb{R})$$

where 0 is an equilibrium of the system such that $f(0, \phi) = 0$ *for all* ϕ*, and assume that*

(A1) $A = D_x f(0,0) = ((\frac{\partial f_i}{\partial x_j})(0,0))$ *is linearisation of system around the equilibrium 0 with* ϕ *evaluated at 0; Zero is a simple eigenvalue of A, and other eigenvalues of A have negative real parts;*

(A2) *Matrix A has a right eigenvector u and a left eigenvector v corresponding to the zero eigenvalues.*

Let f_k *be the* k^{th} *component of* f *and*

$$a = \sum_{k,i,j=1}^{n} u_k v_i v_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$

$$b = \sum_{k,i=1}^{n} u_k v_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0)$$

The local dynamics of the system model around are totally limited by a and b (Garira et al., 2014).

i $a > 0, b > 0$. *When* $\phi < 0$ *with* $|\phi| \leq 1$, 0 *is locally asymptotically stable, and there exists a positive unstable equilibrium; when* $0 < \phi \leq 1$, 0 *is unstable and there exists a negative and locally asymptotically stable equilibrium.*

ii $a < 0; b < 0$. *When* $\phi < 0$ *with* $|\phi| \leq 1$, 0 *is unstable; when* $0 < \phi \leq 1$, 0 *is locally asymptotically stable, and there exists a positive unstable equilibrium;*

iii $a > 0; b < 0$. *When* $\phi < 0$ *with* $|\phi| \leq 1$, 0 *is unstable, and there exists a locally asymptotically stable negative equilibrium; when* $0 < \phi \leq 1$, 0 *is stable and a positive unstable equilibrium appears;*

iv $a < 0; b > 0$. *When* ϕ *changes from negative to positive,* 0 *changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.*

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$$R_0 = \sqrt{\left(\frac{\beta(\Lambda-\mu)}{\mu\rho\psi}\cdot\frac{\eta\delta_2\delta_1\beta\Lambda_c}{\mu_c(\delta_1+\mu_c)(\eta+\psi-\delta)\psi}\right)+\left(\frac{\beta_c(\Lambda-2\mu)}{2C\mu\psi}\cdot\frac{(1-\delta_2)\delta_1\beta\Lambda_c}{(\delta_2+\psi)(\delta_1+\mu_c)\mu_c\psi}\right)} \tag{5.10.48}$$

Since β^* is the bifurcation parameter and considering $R_0 = 1$, gives

$$\beta^* = \sqrt{\left(\frac{\mu\rho\psi}{(\Lambda-\mu)}\cdot\frac{\mu_c(\delta_1+\mu_c)(\eta+\psi-\delta)\psi}{\eta\delta_2\delta_1\Lambda_c}\right)+\left(\frac{2kC\mu\psi}{(\Lambda-2\mu)}\cdot\frac{(\delta_2+\psi)(\delta_1+\mu_c)\mu_c\psi}{(1-\delta_2)\delta_1\Lambda_c}\right)} \tag{5.10.49}$$

then the above theorem is applied, letting $\beta = \beta^*$ such that the eigenvectors of the Jacobian evaluated at β^* are associated with the zero eigenvalue given by

$u = [u_1, u_2, u_3, u_4, u_5, u_6, u_7, u_8, u_9]^T$, where

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$$
\begin{cases}
-\mu u_1 - \dfrac{\beta_c^* \Lambda_c}{C\mu_c} \cdot u_7 - \dfrac{\beta_F^* \Lambda}{\rho\mu} \cdot u_9 = 0, \\
-(\alpha+\mu)u_2 + \dfrac{\beta_c \Lambda}{C\mu_c} \cdot u_7 + \dfrac{\beta\Lambda}{\rho\mu} \cdot u_9, \\
-\mu_c u_3 - \dfrac{\beta\Lambda_c}{\mu_c} \cdot u_5 = 0, \\
-(\delta_1+\mu_c)u_4 + \dfrac{\beta\Lambda_c}{\mu_c} \cdot u_5 = 0, \\
-\psi \cdot u_5 + \dfrac{\beta_c(\Lambda-2\mu)}{2C\mu} \cdot u_7 + \dfrac{\beta(\Lambda-\mu)}{\rho\mu} \cdot u_9 = 0, \\
\delta_1 \cdot u_4 - (\delta_3+\psi) \cdot u_6 = 0, \\
(1-\delta_3) \cdot u_6 - \psi \cdot u_7 = 0, \\
\delta_2 \cdot u_6 + (\delta - \eta - \psi) \cdot u_8 = 0, \\
\eta \cdot u_8 - \psi \cdot u_9 = 0
\end{cases}
\tag{5.10.50}
$$

such that

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when

$$\begin{cases} u_5 = 1 \\ u_3 = \dfrac{\beta^* \Lambda_c}{\mu_c^2} \\ u_4 = \dfrac{\beta^* \Lambda_c}{-(\delta_2 + \mu_c)\mu_c} \\ u_6 = \dfrac{\beta^* \delta_1 \Lambda_c}{(\delta_2 + \psi)(\delta_1 + \mu_c)\mu_c} \\ u_7 = \dfrac{(1-\delta_2)(\beta^* \delta_1 \Lambda_c)}{(\delta_2 + \psi)(\delta_1 + \mu_c)\mu_c \psi} \\ u_8 = \dfrac{\delta_2 \beta^* \delta_1 \Lambda_c}{-(\delta - \eta - \psi)(\delta_1 + \mu_c)(\delta_2 + \psi)\mu_c} \\ u_9 = \dfrac{\eta \delta_2 \beta^* \delta_1 \Lambda_c}{-(\delta - \eta - \psi)(\delta_1 + \mu_c)(\delta_2 + \psi)\mu_c} \end{cases} \tag{5.10.51}$$

and the right hand eigenvector is given by:

$\mathbf{v} = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9)$ where

$$\begin{cases} v_1 = 0, \\ v_2 = 0, \\ v_3 = 0 \end{cases} \tag{5.10.52}$$

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letting:

$$\begin{cases} v_4 = 1 \\ v_5 = \dfrac{\beta^* \Lambda_c}{\psi \mu_c} \\ v_6 = \dfrac{\delta_1 + \mu_c}{\delta_1} \\ v_7 = \dfrac{\beta_c(\Lambda - \mu)}{\psi C \mu} \cdot \dfrac{\beta^* \Lambda_c}{\psi \mu_c} \\ v_8 = \dfrac{\delta_2 + \psi}{\delta_2} \cdot \dfrac{\delta_1 + \mu_c}{\delta_1} - \dfrac{(1 - \delta_2)}{\delta_2} \cdot \dfrac{\beta_c^2(\Lambda - \mu)\Lambda_c}{2\psi^2 C \mu_c^2} \\ v_9 = \dfrac{\beta(\Lambda - \mu)}{\psi \rho \mu} \cdot \dfrac{\beta \Lambda_c}{\psi \mu_c} \end{cases} \tag{5.10.53}$$

The sign of a is associated with the following expression after taking partial derivatives twice on F:

$$\begin{cases} \dfrac{\partial^2 f_2}{\partial x_2 \partial x_9} = \dfrac{\beta}{\rho} \\ \dfrac{\partial^2 f_2}{\partial x_9 \partial x_7} = \dfrac{\beta_c}{C} \\ \dfrac{\partial^2 f_4}{\partial x_4 \partial x_5} = \beta \\ \dfrac{\partial^2 f_2}{\partial x_9^2} = \dfrac{2\epsilon\beta}{\rho^2} \\ \dfrac{\partial^2 f_2}{\partial x_7^2} = \dfrac{2\epsilon\beta_c}{C^2} \\ \dfrac{\partial^2 f_8}{\partial x_8^2} = \dfrac{2\delta}{\kappa}. \end{cases} \tag{5.10.54}$$

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Now we substitute the equation 5.10.54 into equation 5.5, we obtain

$$a = \left(\frac{\beta\Lambda_c}{\mu_c^2}\cdot\left(\frac{\beta(\Lambda-\mu)}{\psi\rho\mu}\right)^2\cdot\frac{\beta\Lambda_c}{\psi\mu_c}\cdot\frac{2\epsilon\beta}{C^2}\right)+\left(\beta\cdot-\frac{\beta^*\Lambda_c}{(\delta_2+\mu_c)\mu_c}\cdot\frac{\beta^*\Lambda_c}{\psi\mu_c}\cdot\frac{\delta_1+\mu_c}{\delta_1}\cdot\right)$$

$$+\left(\left(\frac{\delta_2+\psi}{\delta_2}\cdot\frac{\delta_1+\mu_c}{\delta_1}-\frac{(1-\delta_2)}{\delta_2}\cdot\frac{\beta_c^2(\Lambda-\mu)\Lambda_c}{2\psi^2C\mu_c^2}\right)^2\left(\cdot-\frac{\delta_2\beta^*\delta_1\Lambda_c}{(\delta-\eta-\psi)(\delta_1+\mu_c)(\delta_2+\psi)\mu_c}\cdot\frac{2\delta}{\kappa}\right)\right) \quad (5.10.55)$$

$$\left(\frac{\beta_c(\Lambda-\mu)}{\psi C\mu}\cdot\frac{\beta^*\Lambda_c}{\psi\mu_c}\cdot\frac{\beta_c(\Lambda-\mu)}{\psi C\mu}\cdot\frac{\beta^*\Lambda_c}{\psi\mu_c}\cdot\frac{2\epsilon\beta}{C^2}\right)<0$$

We assume $\Lambda > \mu$. The non-zero partial derivative used to determine the sign of b is given by

$$\begin{cases} \dfrac{\partial^2 f_2}{\partial x_9\partial\beta^*} = -\dfrac{\Lambda}{\mu\rho} \\ \dfrac{\partial^2 f_2}{\partial x_7\partial\beta^*} = \dfrac{\Lambda_c-\mu_c}{\mu_c C} \end{cases} \quad (5.10.56)$$

From the equations 5.10.56 it follows that $b = \dfrac{2R_0}{\beta^*} > 0$. Therefore $a < 0$ and $b > 0$, from the theorem 5.5, we established the following result which holds only for $R_0 > 1$

Theorem 5.6. *(Garira et al., 2014) The endemic equilibrium guaranteed by theorem 5.5 is locally asymptotically stable for $R_0 > 1$*

5.11 Sensitivity analysis

Sensitivity analysis is the study of how the uncertainty in the output of a mathematical model can be allocated to different sources of uncertainty in its inputs. In epidemiology, sensitivity analysis is done to evaluate the relative change in basic reproduction number. This section deals with quantification and extension of uncertainty. Ideally, this is carried out to identify parameters that have a huge impact on the reproduction number so that they can be controlled whenever possible. When optimal intervention strategies are to be implemented, variation in the reproductive number is done through sensitivity analysis. The normalised sensitivity forward index of the reproduction number to all the parameters in the

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model is used. Parameters with negative sensitivity indexes decreases the values of the reproductive number as they increase, while the ones with positive sensitivity indexes increase the reproduction number as they increase. Ideally, establishing the normalized forward sensitivity index of the reproduction number R_0 with respect to the decay μ of host is considered as follows:

$$\Upsilon_u^{R_0} = \frac{\partial R_0}{\partial u} \times \frac{u}{R_0} \tag{5.11.57}$$

In some cases, considering the sensitivity index of R_0 with respect to the host infection rate λ given by

$$\Upsilon_\lambda^{R_0} = \frac{\partial R_0}{\partial \lambda} \times \frac{\lambda}{R_0} \tag{5.11.58}$$

Shows that the sensitivity index of R_0 with respect λ does not depend on any of the parameter values. The indices of immune system death rate and pathogen death rate are respectively given by,

$$\Upsilon_{\mu_c}^{R_0} = -\frac{1}{2}\frac{\mu_c}{(\mu_c + \delta_1)} \tag{5.11.59}$$

$$\Upsilon_{\psi}^{R_0} = -\frac{1}{2}\frac{(2\eta + \psi)}{(\delta_2 + \psi)} \tag{5.11.60}$$

The same procedure was done to all other parameters in the model using ipython, paying attention to the sign changes of each and every parameter with the intention to learn the effects on R_0. However parameters with the most significant influence on R_0 were identified as β , β , δ_1 , ψ , η , . This strongly suggest that reducing the litter on the orchard floor and good agriculture practise can improve in the efforts to eradicate or eliminate CBS. The computation of the above equations shows that whenever $R_0 > 1$, $a < 0$ and $b > 0$, thus locally stable. Therefore, the following theorem holds:

Theorem 5.7. *The CBS endemic equilibrium point is locally asymptotically stable if* $R_0 > 1$.

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Parameter	Description	Sensitivity index
Λ	Fruits recruitment rate	+0.45
β	Fruits primary infection rate	+0.501110
μ	Fruit natural decay rate	-0.4998
Λ_c	Immune defence system supply rate	+0.4998
β_c	Fruits primary infection rate	+0.4998
μ_c	Natural decay rate of immune system in the host	-0.00891
δ_1	Induced decay rate of immune system in the host	-0.00954
ρ	Ascomata half saturated constant	-0.4998
ψ	Natural decay rate of *Guignardia citricarpa* in the environment	-0.155
η	Transforming rate of ascomata to ascospores	+0.135
δ_2	Shedding rate of pycinidia in the environment	+0.4998
C	Secondary saturation constant	-0.5

Table 5.2: *Sensitivity Indices of model system (5.2.8) reproduction number R_0 to parameter for the dynamic system of CBS, evaluated at the parameters values in the Table above*

The table 5.2 clearly indicate that the reproductive number R_0 is sensitive to the changes of parameters β, Λ, β, Λ_c, δ_2, α_1, μ_c, ψ, α, ρ and C. This assists in selecting the most elasticity parameter in relation to the reproduction number R_0 when proposing effective intervention strategies. Since $\Upsilon_{\mu_c}^{R_0} = -0.895$, then an increasing or decrease of μ_c by 20% result in an increase or decrease of the reproduction number by 20.1%. Therefore, targeting the appressoria and pycinidia within the fruits by using altered time-scaled means of chemical application, will gradually reduce the transmission of CBS disease. Similarly as $\Upsilon_{\beta}^{R_0} = 0.4899$ and $\Upsilon_{\delta_2}^{R_0} = 0.4899$, decreasing fruits invasion rate β proportional decreases R_0 significant or pycinidia in the environment δ_2 by a significant proportion reduces R_0 directly. As a result, enhancing farmers knowledge about development conditions of the CBS will eliminate or slow the progress of the disease without much of chemical spraying program.

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5.12 Numerical analysis

Numerical simulations are provided in graphical form showing the solution behaviour of state variables evolves with time. Observation from the model indicate that to find means to reduce ascomata on the orchard floor and emphasises of good agriculture practice importance to lower the level of endemicity. The computational of numerical analysis is done using ipython, an open source in ubuntu 14.0.4 for running model simulation through the use of text editors

Definition	Symbol	Estimate	Source
Fruits recruitment rate	Λ	20.10	(Jacobsen et al., 2013)
Fruits natural decay rate	μ	0.0035	(Chiyaka et al., 2012)
Fruits decay due to disease	α	0.00014	(Bailey and Gilligan, 2004)
Probability of transmission	β	0.105	(Jacobsen et al., 2013)
Pathogen transformation rate	η	0.295	(Chiyaka et al., 2012)
Limitation of the growth velocity	ϵ	0.989	(Chiyaka et al., 2012)
Half saturation constant	ρ	20000	(Bailey and Gilligan, 2004)
Carrying capacity	κ	5000000	(Bailey and Gilligan, 2004)
Health immune defence system recruitment	Λ_c	10.1300	(Bailey and Gilligan, 2004)
Natural immune defence system diminish	μ_c	0.00001	(Bailey and Gilligan, 2004)
Infected immune defence system transformation rate	α	0.00034	(Bailey and Gilligan, 2004)
Appressoria recruitment rate	Λ_p	20	(Bailey and Gilligan, 2004)
Appressoria death rate	ψ	0.0295	(Bailey and Gilligan, 2004)
Number of pathogens	N	10000	(Bailey and Gilligan, 2004)
Pycinidia death rate	σ	0.00005	(Bailey and Gilligan, 2004)

Table 5.3: *Parameters used in the model simulation*

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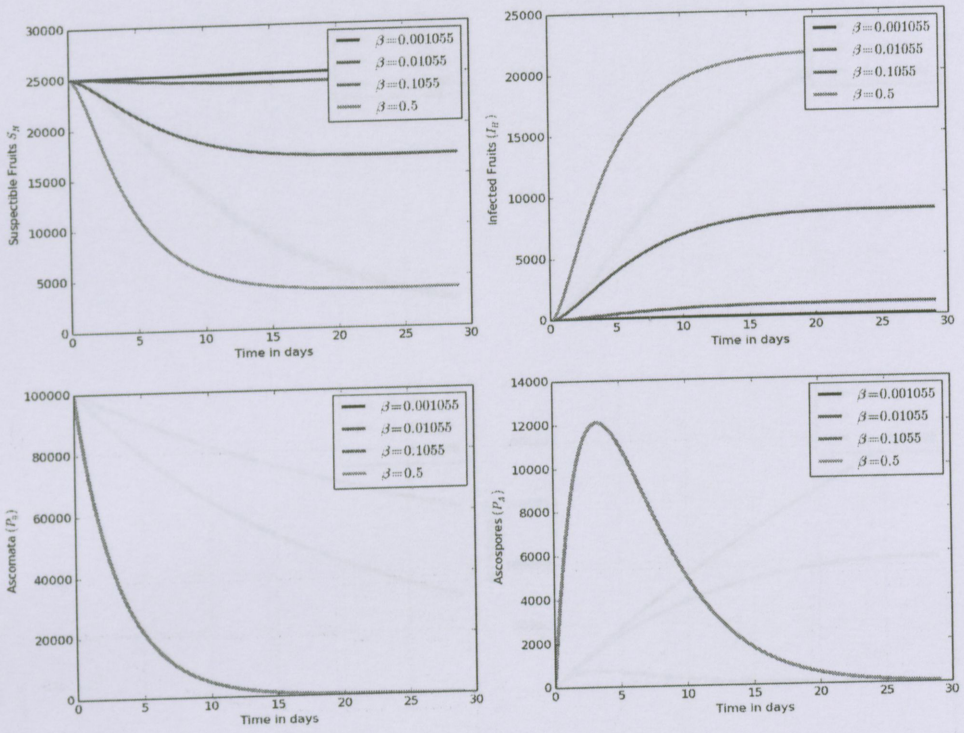

Figure 5.2: *The fruits and The pathogen result of over 30 days.*

The simulation in 5.2 show the behaviour of the system of ordinary differential equation with respect to time when β was varying from 0.001055 to 0.5 the illustrated observation agree with R_0 on the basis that as β increases so does the infective classes and inversely affects non-infectious population classes. During the initial stages, the infected class of the fruits increases rapidly up to a peak level of 20000 then gradually increase at a decreasing rate whereas the susceptible gradually decrease at a steady rate until 5000 level is reached. The variation of β does not have an effect on the pathogen but rather the fruits population.

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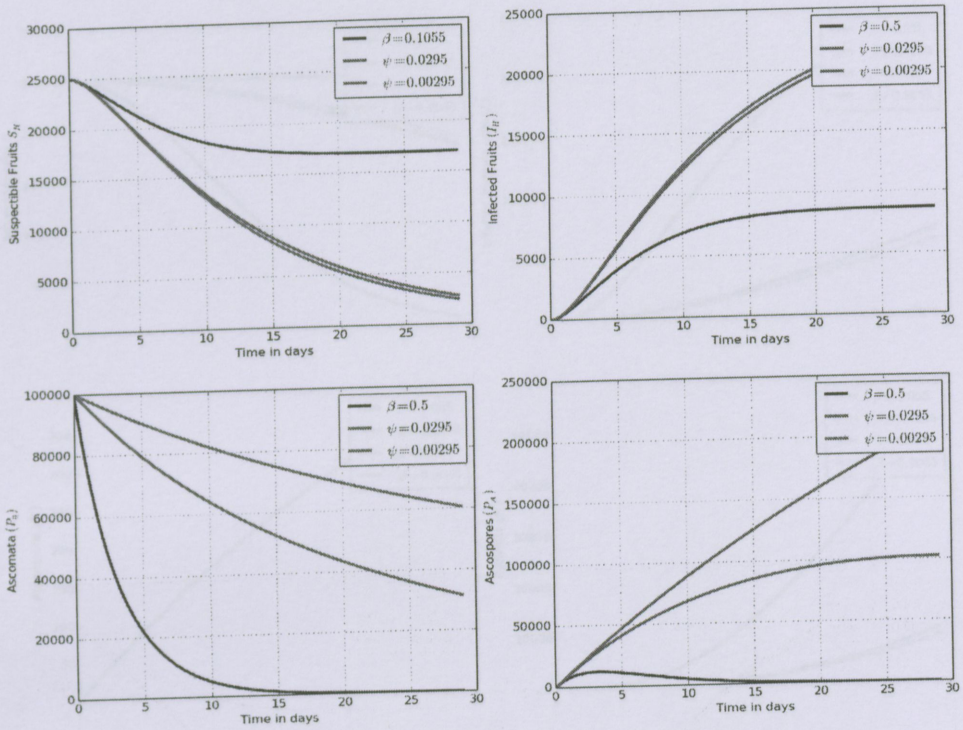

Figure 5.3: *The fruits and The pathogen result of over 30 days about the ψ.*

When the diminishing rate ψ of the pathogen increases, the ascomata in the environment decreases quickly during the first 10 days of infection, then gradually decrease at a steady rate and it also promotes or increase healthy susceptible fruits. Infection is shown to be relatively decreasing as ψ increases and the ascospores population is inversely proportionally to the diminishing rate ψ. Thus the higher the decay rate of ascomata and ascospores, then the lower the disease progression. This follows the result obtain from the sensitivity analysis, which reflected that ψ is the most sensitivity parameter that can be targeted to reduce or eradicate CBS

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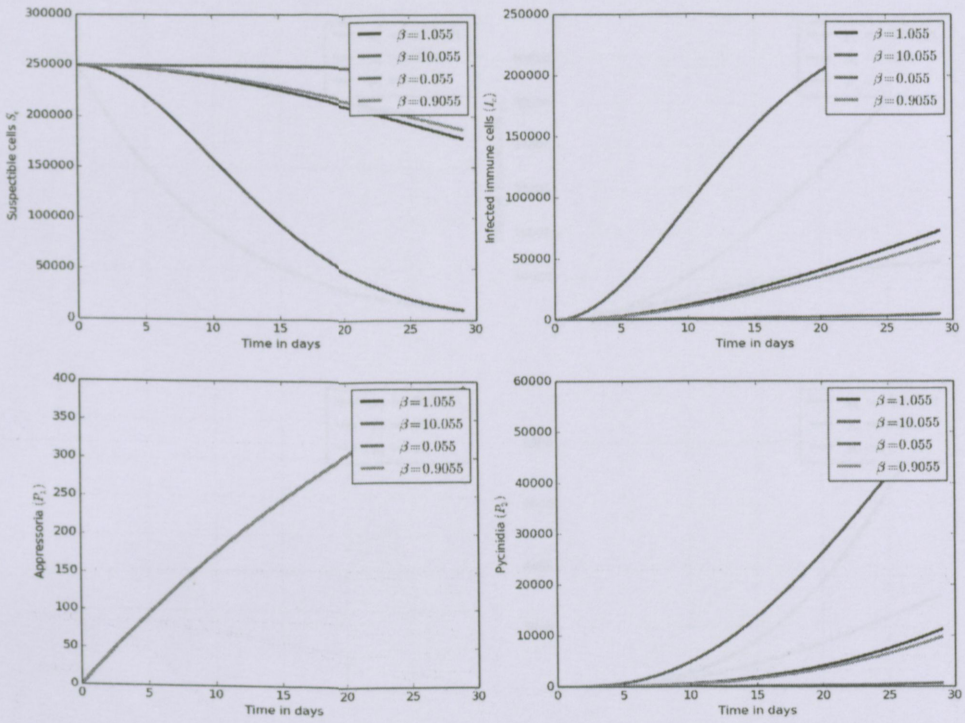

Figure 5.4: *Fruit population behaviour over a period of time when varying* β

The simulation in 5.4 indicate that as β increases so does the infected class and pycnidia while the appressoria remain unchanged, this implies that controlling of the transmission probability does not have influence on the appressoria pathogen population. A positive change was shown on the pycnidia population and the infected immune defence system as the transmission probability increases. Susceptible class of the immune defence system was shown to respond adversely on the β. Immunologically, controlling strategies should be implemented to control appressoria since it has a direct influence on infected immune defence system in term of disease invasion and persistence.

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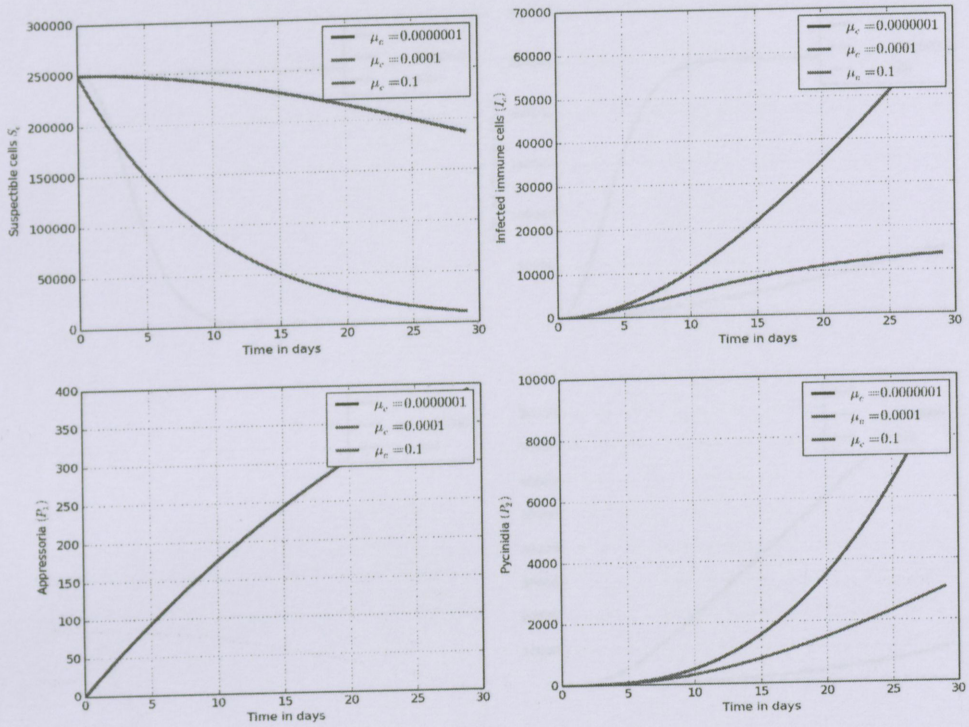

Figure 5.5: *Fruit population behaviour over a period of time when varying* μ

From 5.5 the variation of μ shows the same effects as β particular on appressoria. An increase in the decay rate of infected class of immune defence system decreases the pycinidia population. This may explain a situation whereby an infected immune defence system committed suicide before the infection can spread widely.

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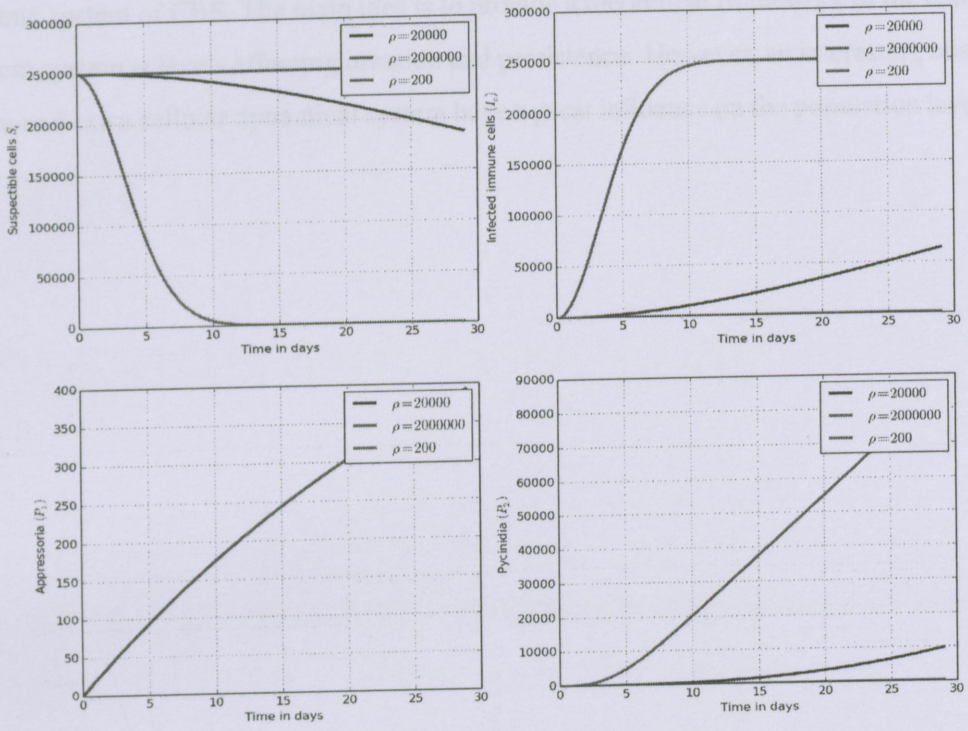

Figure 5.6: *Fruit population behaviour over a period of time when varying* ρ

The relationship between the susceptible immune defence system and infected immune defence system remains well posed in all the plots throughout, however appressoria shows an insignificant change as ρ varies as the disease evolves with time. The interesting part of the plot is that the more the saturation constant growth the less pycinidia and infected class.

5.13 Conclusion

This chapter was dealing with the interlinking of the epidemiological characteristics and the immunological characteristics and investigate the influence on each other. The formulation of a mathematical

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model framework of CBS at different levels and on different scales enable us to comprehend the complex dynamic system of CBS. The main idea is to provide a theoretical framework of the complex disease development system at levels affecting invasion and persistence. However, an interesting observation was made, intra and extra cellular dynamical system have a great influence on the population level dynamics.

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Chapter 6

DISCUSSION AND CONCLUSION

6.1 Introduction

Although efforts have been made to manage CBS caused by *Guignardia citricarpa*, but there are still some difficulties posed by the disease especially in citrus growing industry. A more related study was done by (Chiyaka et al., 2012), but unfortunately it was a vector born disease. To date, CBS is still a disease of interest in the citrus growing industry. Furthermore, there are no multi-scale mathematical models developed to study the CBS disease transmission dynamics at different scales. In this study, mathematical model frameworks for CBS are constructed to characterise the dynamics of the disease development immunological and epidemiological, paying attention to the *Guignardia citricarpa* life cycle at different scale levels. Observations made from the model analysis indicated that the initial inoculum by ascospores is very important for disease development but thereafter ascospores will be no longer necessary due to conidia, an infectious form responsible for secondary infection. Most importantly the model indicated that ascospores and conidia are very important parameters in developing CBS. Numerical simulations of the models were done to explain the theoretical findings. A deterministic model for

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CBS within citrus fruits was developed to trace the average transmission dynamics of *Guignardia Citricarpa* within the fruits, incorporating standard incidence rates due to direct transmission, infection rate and disease induced decay rate. The chief aim of the model was to capture the interaction between the plant defence system and the *Guignardia citricarpa* population. The basic reproduction number was computed and its relationship to the disease progress was shown that the disease will persist for a very long time unless if some controlling measures take place. The theoretical findings were shown through the use of numerical simulation.

The presentation of a compartmental deterministic model which entails internal plant defence mechanism and pathogen interaction was developed in chapter 4, then the model was nested into an epidemiological model in chapter 3. The objective was to improve our understanding on the transmission dynamics effects of within-host on between host of *Guignardia citricarpa* evolution and vice-versa. The inflow of infected class was scaled down to the individual level while the outflow was scale up to average population level.

The conceptual and mathematical model was constructed to display a theoretical framework which can be used for predicting or identify disease patterns. In summary, the following are the findings made through sensitivity analysis and numerical simulation:

(i) Reducing the ascomata on the orchard floors by any meaning possible such as good agriculture practice (GAPs) , altering the time-scaled by using chemical application, will sufficiently reduce the transmission dynamics of CBS disease.

(ii) Taking stain measures during cultivation like treating the general surrounding to prevent contact between ascospores and young tender susceptible fruits is very efficacy to control CBS.

(iii) Once the disease exist in the fruits population, ascospores will be no longer necessary for invasion, therefore conidia will be the point of focus.

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6.2 Summary

The system of differential equations constructed in chapter 3 to describe and analyse the disease progression of CBS between fruits population within a contaminated environment. The model was developed following the saturation incident law of infection represented by a logistic equation showing density-dependant relation between the pathogens and the fruits. Susceptible fruits population was assumed to be recruited in a logistic manner. The model is locally and globally asymptotically stable at disease free equilibrium when R_0 is less than unity and locally and globally asymptotically stable at the endemic equilibrium state when R_0 is greater than unity. The following assumptions were made when constructing the models, ascomata were assumed to survive the environmental condition and transform to ascospores.

The models indicated that once the fruits is infected there is no recovery, thus, they decay eventually. As the ascospores increase the infection is also spreading, decreasing the susceptible and the ascomata. The numerical simulation clearly indicated that to reduce infection ψ should be increased by any means necessary like spray, good agricultural practise among other strategies. The removal of infected leaves and fruits is not sufficient to completely eradicate or eliminate CBS because of the presence of conidia in fruit and dead twigs. Therefore, the reduction of conidia sources should also be considered in CBS management.

An immunology model was built and analysis to gain insight on the key factors that influence the internal progress development of the CBS disease. The threshold level was identified as a benchmark in which concerned parties will refer to when determining whether the disease will invade and persist or just die out. Results from plots bear proofs on the threshold theorem which was computed in previous sections. Plots also helps us predict the behaviour of the disease dynamics so that controlling strategies will be put forward on the basis of informed decisions.

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6.3 Further research directions

This work may require additional information to improve on the findings for future research directions. Human, Plant and animal models can be used to address similar problems. However, some aspects of plant disease are very different and this promotes difficulties particularly in modelling plant disease. It would be important to consider other aspects, but not limited to, the following:

(i) It is recommended that further refinements may be made to the multi-scale model presented in chapter 5. For instance, the effect of fluctuating climatic conditions may be incorporated into the model. Models should consist of sufficient epidemiological aspects in order to analyse the effects of disease and host dynamics on yield.

(ii) One of the interesting aspect about Citrus Black Spot is that it has a long latent period which can be incorporate when modelling its transmission dynamics to improve the model efficacy. Moreover, *Guignardia citricarpa* life-cycle and the host life-cycle can result in time-varying patterns of infectivity.

(iii) To some extent there is a need to quantify the proportion of the total soil population of ascomata to improve our multi-scale modelling efficiency.

(iv) Furthermore, multi-scale modelling of CBS should be extended to account for the combined effects of the host, pathogens, health interventions and the environmental interactions.

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