Regulation of a biennial host plant population by an autoecious, demicyclic rust fungus:
Puccinia hysterium on *Tragopogon pratensis* in the Park Grass Experiment

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Declaration

The work presented in this thesis was conducted at the Wye Campus of Imperial College London, Silwood Park Campus of Imperial College London and Rothamsted Research, Harpenden. This thesis is the result of my own work and any quotation from, or description of the work of others is acknowledged herein by reference to the sources, whether published or unpublished. This thesis is not the same as any that I have submitted for any degree, diploma or other qualification at any other University. No part of this thesis has been or is being concurrently submitted for any such degree, diploma or other qualification. It is less than 50,000 words.

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Abstract

Models developed in continuous-time have been used to study the epidemiology and population dynamics of plant hosts, usually in cultivated systems. Here discrete-time *SIR*–type models are developed which contain parameters representing characteristics of an uncultivated, biennial host plant – systemic, castrating pathogen system. This thesis presents 4 epidemiological model forms representing a generic *SIR* model, a constant pathogen-induced mortality model, a variable pathogen-induced mortality model, and a model which has an additional phase representing a seedbank. Using a range of parameter values it is possible to produce simulation outcomes with population crashes, cycles and steady-state populations. For each of the models a pathogen epidemic criterion is derived as is a term describing population steady-state values. For the pathogen-induced mortality models, the invasion criteria include a pathogenicity term indicating that the pathogen in part regulates the host population dynamics.

The biennial host plant *Tragopogon pratensis* has been recorded in the Park Grass Experiment and has been described as an outbreak species regulated by the autoecious, demicyclic rust fungus, *Puccinia hysterium* (Silvertown *et al.*, 2006). The rust is shown to castrate the host plant by reducing the numbers of seed set and the viability of seeds produced by infected individuals. Further characteristics of this host – pathogen system are identified by using the developed models. This is justified as the recurrence-relationships derived from the models fit the observed data, and that the accuracy of the fit is increased with larger values of pathogen-induced mortality. These models produce simulation outcomes that are similar to the host population dynamics. The models also show that the system is governed by density-dependent factors.

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Abbreviations

PGE: Park Grass Experiment
spp: species (plural)
f. spp.: species form
sp: species (singular)
cm: centimetre
mm: millimetre
ha: hectare
m: metre
mg: milligram
^{o}C: degrees Celsius

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1. Introduction

1.1. General Introduction

Population dynamics of plant communities have been long studied since the inception of the Park Grass Experiment (PGE) at Rothamsted Research in 1856 (e.g. Silvertown *et al.*, 2006). Many of these studies have been related to the influence of fertiliser regimes on plant community dynamics and structure; however the influence of plant pathogens within the PGE has been largely ignored, even though there is evidence that nutrient regimes not only alter plant communities but also their associated organisms (e.g. Graham, 1983, Shaw *et al.*, 2008), in particular plant pathogens. One particular plant species in the PGE affected in its long-term population dynamics is the biennial *Tragopogon pratensis* L. (Dodd *et al.*, 1995), which displays the dynamics of an outbreak species over some 60 year period. The definition of an outbreak species is given by Silvertown *et al*. (2006) as where a "species first increased and decreased again". It is hypothesised by Silvertown *et al.* (2006) that this outbreak dynamic is regulated by a specific pathogen *Puccinia hysterium* (Str.) Röhl, an autoecious, demicyclic rust fungus. This thesis will present the development and investigation of epidemiological models representing the regulation of a host biennial plant, such as *T. pratensis*, *by* a pathogen with characteristics similar to those of *P. hysterium*, thus being a tool in investigating the *T. pratensis – P. hysterium* system in the PGE. This thesis will encompass topics relating to plant ecology with particular emphasis on plant population dynamics, and plant pathology with attention focused on rust pathogens and epidemiological modelling.

1.1.1. Plant ecology

Although there are many encompassing definitions, ecology can be simply described as "the scientific study of the interactions that determine the distribution and abundance of organisms" (Krebs, 2001). Plantecology could be defined as the study of abiotic and biotic factors altering the performance of plant organisms. Plant ecology is rarely studied at the individual level, with most studies being at the population or community level or at the wider ecosystem level, also ecology involves the study of species throughout evolutionary time (Begon *et al.*, 1996). Plant ecologists have looked at many of aspects of ecology, including studies of plant life-cycle evolution and reproduction

(e.g. Rees *et al.*, 2006), plant dispersal ranges (e.g. Pearson and Dawson, 2004), interactions with herbivores (e.g. de Mazencourt and Loreau, 2000), pathogens (e.g. Frantzen, 2002) and pathogen vectors (e.g. Jeger *et al.*, 2004), and population dynamics and community structure (e.g. Dodd *et al.*, 1994).

Much of the work relating to plant populations involve observations of plant adaptations which optimise fitness (Tilman, 1988); whereby plants adapt life history traits in able to cope better with disturbance, competition or environmental stress. Plant pathogens are biotic factors which affect the dynamics and evolution (Gilbert, 2002). The response by plant populations over time can be observed as varying types of population dynamics, such as population cycles, crashes or growth (Dodd *et al.*, 1994) depending on the ability of the plant to respond to the biotic stress. The study of the effects and symptoms of pathogens (as well as abiotic environmental stress) on plants is the remit of plant pathology (Agrios, 2005), both in cultivated and natural plant populations.

1.1.2. Plant pathology and plant pathogens

Plant pathology is the study of plant disease in cultivated and non-cultivated systems and has interested man since the era of hunter-gathering (Agrios, 2005). Plant pathology relates to the symptoms and prevention of diseases as well as the use of diseases as biological control agents of pest plant species such as weeds. Disease can be caused by biotic agents such as viruses (e.g. Jeger *et al.*, 2004), fungi (e.g.Weber *et al.*, 2003), bacteria (e.g. Fininsa, 2003), phytoplasmas (e.g. Hogenhout *et al.*, 2008), parasitic plants (e.g. Wilson and Calvin, 2006), nematodes (e.g. Taylor and Rodríguez-Kábana, 1999) and protozoa (e.g. Dollet, 1984); or by abiotic factors such as stresses or toxic effects of nutrients, water, light soil acidity and air pollution (Johnston and Booth, 1983). Plant pathology encompasses a wide range of study areas including identification of pathogens (e.g. Bruckart *et al.*, 2007), understanding of pathogen life history (e.g. Böllmann, 2006), assessment of economic impact of diseases (e.g. Wittwer *et al.*, 2005), prevention and control of pathogens (e.g. Gilligan, 2008), and epidemiology (e.g. Jeger, 2000).

1.1.3. Host-Pathogen relationships

The impact of pathogens on plant populations has a long history with reports extending back to ancient civilisations and the Homeric era in around 1000 BC (Agrios, 2005). The Romans paid devotion to the God of rust prevention, Robigus, and of blight, Robiga, for some 1700 years (Boyd, 2005). The Bible also speaks of pathogenic plagues of mildew (Deuteronomy, 28:22), blight (Amos, 4:9) and blasts (Haggai, 2:17). Mysticism surrounding plant and host interactions continued beyond the advent of microscopy. Robert Hooke, although observing rose rust microscopically, believed in spontaneous generation within the host. Subsequentially microscopy has allowed detailed relationships between host and pathogen to be investigated (Waller *et al.*, 2002).

The interaction between hosts and their pathogens are dependent on a wide range of factors relating to host abundance, pathogen prevalence and virulence, as well as aspects of local host population and pathogen strain adaptations. In addition to these biological factors, the relationship can be strongly influenced by abiotic factors such as climate (Garcia-Guzman and Morales, 2007) and host condition due to water (Marks and Clay, 2007) and nutrient availability (Snoeijers *et al.*, 2000). Similarly, the impact of a pathogen on its host also varies. Pathogens are able to alter growth patterns within plants, decreasing growth rates (e.g. Burdon, 1993), and sometimes acting by increasing growth rates (e.g. Marks and Clay, 2007), altering morphology (e.g. Roy, 1993) and castrating hosts (e.g. Schurch *et al.*, 2000). Pathogens can either modify host size, fecundity and longevity or have a combination of impacts, affecting competitive ability, reducing net photosynthesis, increasing dark respiration rate, altering patterns of assimilate partitioning, and altering nitrogen metabolism, ion uptake, water relations and metabolism (Burdon *et al.*, 1989). In particular, biotrophic pathogens establish long-term associations and may have profound effects on plant metabolism. Duration of the disease within the population is also important, the longer a disease is present the greater the cumulative effects of the disease are on the host population (Burdon, 1987). Regulation of host dynamics by plant pathogens has also been shown to have impact on the wider biota, as in the case of the powdery mildew (*Podosphaera plantaginis*) – *Plantago lanceolata* system impacting on the butterfly species *Melitaea cinxia*; larvae disperse away from infected individuals and through eating less they develop slower,

causing higher winter mortalities impacting on distribution and population size (Laine, 2004).

1.1.4. Mechanisms of disease action

Much of the effects of pathogens are cosmetic, however a pathogen can often effect a host through reduced growth and increased mortality. This has been shown in detail in studies of the rust *Puccinia lagenophorae* which inhibits leaf expansion in groundsel (*Senecio vulgaris*), so reducing growth and plant dry weight (Paul and Ayres, 1987). Plants were stunted because growth of leaf area was inhibited and photosynthetic capacity reduced (Paul and Ayres, 1984). In winter the rust increased plant vulnerability to environmental conditions, with smaller plants being more vulnerable to the reduced winter temperatures (Paul and Ayres, 1986a). In the summer, senescence of rusted plants occurred earlier and more rapidly than uninfected plants (Paul and Ayres, 1987).

By altering the ability of a host to absorb water and nutrients from the soil and translocate photosynthetic products from leaves, a pathogen is able to affect many plant functions. There are several modes of interfering with water and nutrient translocation by damaging root structure, xylem and phloem integrity, increasing transpiration and diverting resources away from plant material. Root rot caused by *Phytophthora* spp. is a major cause of root damage altering plant function. Such an example is *Phytophthora cinnamomi*, a soil-borne pathogen, which infects a wide range of plants especially woody species (Pryce *et al.*, 2002) and causes water deficiency (Broadbent and Baker, 1974). Pathogens which cause galls, wilts and cankers also increase water stresses by reducing translocation by isolating water movement through the clogging up the xylem or producing pressure on plant material leading to damage. Examples of such pathogens are gall forming nematodes (*Anguina sp.*) on cocksfoot (Southey, 1969), bacteria such as crown gall (*Agrobacterium tumefaciens*) on dicotyledonous plants especially members of the rose family (Sigee, 1992), and fungi such as *Fusarium oxysporum* (Agent green) used as a mycoherbicide agent on *Coca* in South America (Sands *et al.*, 1997) as well as a wide range of weeds (Hajek, 2004). Foliar fungi can also alter the rate of transpiration by damaging the cuticle, thus speeding up water loss (Burdon, 1987). In groundsel, after rust sporulation, transpiration in rusted plants is more rapid than in healthy individuals due to rupturing of the leaf and stem cuticles, leading to a lack of control over water loss (Paul and Ayres, 1984).

Host seed production and viability is also influenced by pathogens, with direct effects due to the pathogen actively affecting the reproductive capability, or indirect effects due to consequences of pathogen interference with host physiology (Burdon 1993). One of the main effects is due to infected individuals often being smaller and less developed so producing only smaller flowering bodies and fewer number of seeds and fruit (Burdon, 1987). For example *Puccinia lagenophorae* has been shown to cause inhibited capitula and to lower the number of florets (Paul and Ayres, 1987). The time of infection has an impact on seed production, infections before flowering reduce both seed number and weight, whilst infection during flowering and subsequent seed development cause reductions only in seed weight (Burdon, 1987). Lower weight seeds are less developed and less likely to be viable, or if they are viable, compete with uninfected seeds. This competitive inferiority leads to lower reproductive fitness in subsequent post-epidemic generations (Jarosz *et al.*, 1989). However, Paul and Ayres (1986c) showed that although seeding time was delayed when *S. vulgaris* was infected with *P. lagenophorae*, the number of florets produced non-significantly different number of seeds. Ericson *et al*. (2002) reported that *Filipendula ulmaria* (meadowsweet) seeds showed high levels of nonviable seeds collected from plants infected with the rust *Triphragmium ulmariae*. Hanley and Groves (2002) report that *Puccinia chondrillina* has a marked effect on host plant (*Chondrilla juncea*) size in non-resistant individuals, this in turn cause individuals to become competitively excluded in natural environments. Another example is *Puccinia abrupta*, which causes increased leaf senescence in *Parthenium hysterophorus* and decreases life-span, yield and growth rate, and more importantly reduces flower and in turn, seed production and viability(Parker *et al.*, 1993).

1.1.5. The impact of plant pathogens

Pathogens influence many plant systems including agricultural crop systems, where plant pathogens cause some 14% loss of worldwide crop production (Agrios, 2005). There are many studies reporting the impacts and range of effects of pathogens in agricultural systems, especially by those caused by the plant pathogen groups: prokaryotes, viruses, fungi, oomycetes and nematodes.

There are approximately 100 known bacterial plant diseases (Mishra 2003) . The most common are the blights and spots caused by the genus *Pseudomonas* and *Xanthomonas*.

Spot and blight diseases cause necrotic tissue to develop causing a loss of photosynthetic material; sometimes the bacteria produce toxins that exacerbate the loss of plant tissue. An example of yield loss due to blight is *Xanthomonas campestris* infection of *Phaseolus vulgaris* which also decreases seed production (Fininsa, 2003)due to reduction of photosynthetic tissue leading to senescence. Similarly, seed production was reduced in winter wheat infected with the bacterial streak *Xanthomonas translucens* (Tillman *et al.*, 1999).

Although plant viruses cause production losses in many agricultural systems, little work is conducted on natural populations infected with plant viruses. One such long-term study indicated that geminivirus-infected *Eupatorium makinoi* individuals had reduced growth, were less competitive and in turn had lower rates of flowering and survival (Funayama *et al.*, 2001). Depending on environmental conditions, populations could become reduced and even extinct after viral infection. A study of *E. chinense* infected by tobacco leaf curl virus indicated that infected individuals had higher mortality, reduced growth and had lower seed production (Yahara and Oyama, 1993). Infection of barley and cereal dwarf virus in wild populations of bunchgrass in California resulted in a lower seedling rate altering host population dynamics (Malmstrong *et al.*, 2006).

Nematodes are responsible for introducing microscopic plant pathogens such as viruses in to plants, however they are also responsible for direct deleterious effects (Agrios, 2005). Much of the damage is produced by feeding from the plant and the production of galls or knots in roots which can cause plant yields to decrease. This is seen in the case of reduced yield of peanut crop infected by the root-knot nematode *Meloidogyne arenaria* (Taylor and Rodríguez-Kábana, 1999).

There are approximately 10,000 plant diseases caused by both obligate and non-obligate fungal pathogens (Agrios, 2005) including rusts, smuts and mildews that can alter host populations in a wide variety of ways. Conventionally but not taxonomically, oomycetes such as *Phytophthora*, *Pythium* and downy mildews are often grouped with fungal diseases.

Rust fungi are in the order Uredinales of the class Basidiomycetes. They are obligate parasites that live on living tissue on a wide range of hosts including ferns, conifers, and

angiosperms. There are over 100 genera containing some 5000 species of rust fungi (Cummins and Hiratsuka, 1983). Rust fungi are usually host specific, heteroecious with two different hosts and have up to five distinct spore phases; however, there are many variations of lifecycles such as where fungi are autoecious (only having one host) or have reduced spore phases. Rusts are categorised according to their life-cycles and host association into five recognised groups: heteromacrocyclic (five spore phases with a telial phase on one host and aecial on an alternate host), automacrocyclic (five spore phases occurring on a single host), heterodemicyclic (lacks a uredinial phase, requires two hosts), autodemicyclic (requires one host to complete its lifecycle, without a uredinial phase) and microcyclic (lacks aecial and telial phase). *Puccinia hysterium* is an autoecious, demicyclic (also known as autodemicyclic) rust as it has a single host (*T. pratensis*) and lacks a urediospore phase.

Rust fungi are important economic pathogens for example *Puccinia graminis* in cereal crops (e.g. Singh *et al.*, 2006), soybean rust (e.g. Del Ponte, 2006), *P. hordei* on barley (e.g. Das *et al.*, 2007) and in less economically important crops such as in rusts of garlic (Anikster *et al.*, 2004) Less attention has been focused on natural systems, with most work coming from the use of rust fungi as mycoherbicides (Butt *et al.*, 2001).

1.1.6. Epidemiology

Epidemiology is defined by Kranz (1974) as: "the science of populations of pathogens in populations of hosts, and the disease resulting therefrom under the influence of the environment and human interference". Epidemic studies often involve the categorising of individuals within the host population to represent the state of the disease presence within the population. The development of epidemiological models is one particular method utilised in the study of epidemics. By producing mathematical models of a biological system it is possible to gain an understanding of the conditions determining the incidence, severity and spread of disease and the resulting population dynamics of hosts exposed to a pathogen. Much of the original work on epidemiological models arose from work in zoological and human systems, and was developed using variations of the Kermack-McKendrick model (Kermack and McKendrick, 1927, 1932, 1933). The theory of underlying mathematical models for describing and addressing disease epidemics was developed by Bailey (1975) and extended to address many ecological applications by Anderson and May (e.g. Anderson and May, 1979, May and Anderson,

1979). Plant-pathogen epidemic models were first devised by Van der Plank (1963), specifically to be consistent biologically with the life-histories of plants and pathogens.

1.2. The host – pathogen system

The host *Tragopogon pratensis* has been observed within the Park Grass Experiment (PGE) at Rothamsted Research, Harpenden, Hertfordshire, UK over a period of around 60 years, which is described by Dodd *et al*. (1995) as exhibiting outbreak population dynamics (Figure 1) and it was hypothesised that the obligate pathogen *Puccinia hysterium* is responsible for the regulation of the host dynamics (Silvertown *et al.*, 2006). There have been no reported accounts of reproducing the life-history of *P. hysterium* in cultured cohorts of *T. pratensis*: some simple experiments on which to base observations relevant to estimation of model parameters are described in Chapter 6.

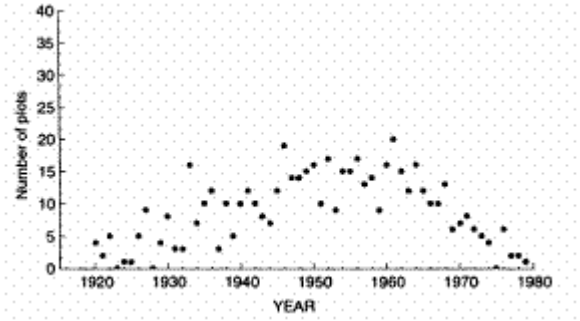


Figure 1. Frequency of plots with recordings of *T. pratensis* within the PGE (Silvertown *et al.*, 2002).

1.2.1. *Tragopogon pratensis* L.

T. pratensis is a dicot of the class Compositae, family Asteraceae (Pico *et al.*, 2003) and is a native plant found across Europe and North Africa. It has since colonised northern states of the USA and areas of British Columbia (Clements *et al.*, 1999). It can form a hybrid species when crossed with *T. dubius*, to produce *T. miscellus* (Clements *et al.*, 1999). Within the UK it flowers between June and July (Clapham *et al.*, 1987) and is insect pollinated. Once pollinated plants demonstrate monocarpic behaviour and die after setting seed (Clements *et al.*, 1999).

The common English name of the plant is Goat's beard because of the shape of the seeding structures (Stace, 1997). It is also known by several other colloquial names including, noon flower, shepherd's clock and jack-go-to-bed-at-noon and when cultivated it is known as meadow salsify. It is described by Rose (1981) as an erect biennial with milky latex, growing 30-100cm tall and with little branching. The lower leaves are 10-30cm long, linear lanceolates (grass-like), keeled lengthwise and are grey-green in colour and are wider and sheathing at the base. The upper leaves are shorter and erect. The flower heads are up to 5cm wide, long stalked, and only open in the morning sunshine and closing before noon, except in cloudy weather where they remain closed. The florets are bright yellow with eight or more equalling bracts which are lanceolate, pointed in a row and are 23-28mm long and 3-5mm wide, and are widest nearer middle, whilst the bracts have red brown borders 1mm wide. The achenes are 15-20mm long, rough, long-beaked, pappus with main rays almost woody and radiating, interwoven across with fine white side hairs. The plant grows from a rhizome and during its vegetative phase, an extensive root system develops, not only for acquiring soil nutrients and water but also acting as carbohydrate storage for below ground over-wintering. Erect rosettes allow plants to emerge through litter or other vegetation, aiding colonisation of areas where plant communities are already established. Size correlates with survival, flowering, fecundity and fitness of a plant, whilst individuals within populations vary with size (Clements *et al.*, 1999).

Within the British Isles, *T. pratensis* is distributed across most of England and some coastal areas of South and North Wales and Eastern Scotland, whilst being sparsely recorded across Ireland (Figure 3); however, although it is distributed across the United Kingdom it is often recorded at low density. It grows in a range of habitats including dry grasslands, hedgerows and disturbed land (Pico *et al.*, 2003).

T. pratensis is a biennial plant which grows between May and August (Rose, 1981). In the first year the plant grows grass-like leaves and stems (Figure 2a) which can be difficult to observe in grassland communities, then dies back and remains in a dormant stage over winter. Upon temperature-related cues between $10^{o}C$ – $30^{o}C$ (Qi & Upadhyaya 1993) the plant re-emerges as an adult individual (Figure 2a), produces flowers and fruits and sets seed, and then dies (Qi and Upadhyaya, 1993, Qi *et al.*, 1996). However, it is also known to act as an annual and as a monocarpic perennial (Clapham *et al.*, 1987).

T. pratensis produces around 6 flower heads containing between 20-130 seeds with individuals producing some 100-850 seeds. The seeds are then dispersed by the wind up to a distance of 250m (Clements *et al.*, 1999). There are two differing morphologically distinct types of seeds; dark, heavy seeds produced in outer florets containing phenols and lighter seeds on inner florets. On average, seven percent of the seeds produced are not-viable. A high proportion of the seedlings emerge in the autumn after seed set and die due to environmental conditions and therefore do not contribute to population recruitment. There is also further mortality through predation and seed decay, creating a seed bank with only some 3% of seeds set one year previously (Haubensak and Smyth, 2002). Seedling mortality has been recorded in *T. pratensis* as 50% within seasons and 88% between seasons (Mahesh *et al.*, 1996). With approximately 12% of seeds set in a season emerging in the autumn of the same year (Roberts, 1986) and with seedling mortality being density-independent (Haubensak and Smyth, 2002), this means that a very small proportion of seeds that are set survive to become second-year, reproducing plants.

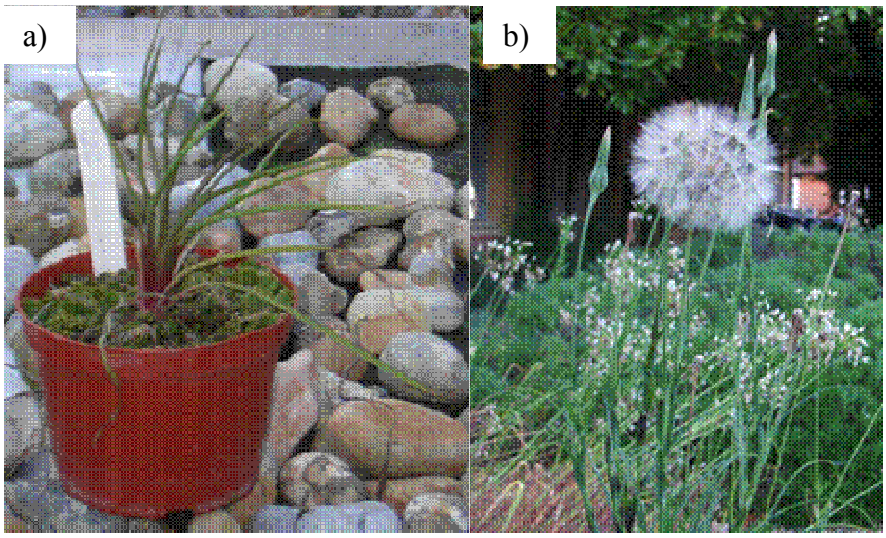


Figure 2. a) The grass-like structure of a first year *T.pratensis*. b) The flowering and seeding second year individual.

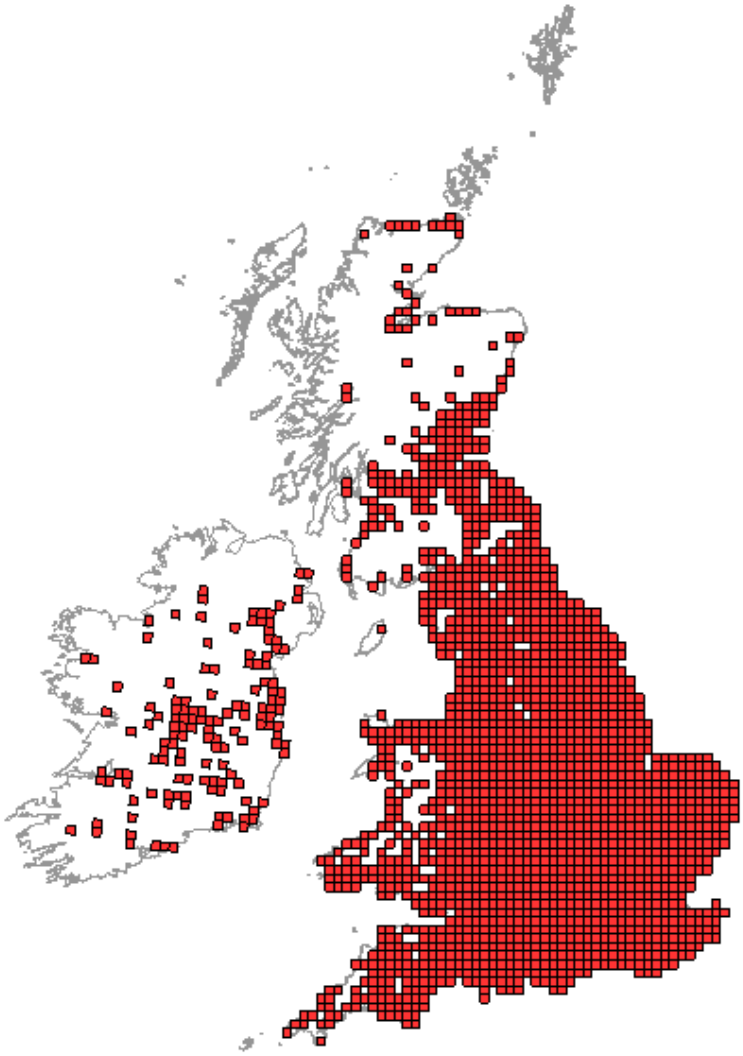


Figure 3. The distribution of *T. pratensis* in UK and Ireland 1987 – 2009. (NBN Gateway, 2009a)

T. pratensis is susceptible to various pathogens, particularly *P. hysterium* (Figure 4) (Parmelee and Malloch, 1972) previously known as *P. tragopogi* (Plowright, 1889). There was a high level of rust infection in *T. pratensis* in the Park Grass Experiment in 1993, a year in which *T. pratensis* was at high density (Silvertown *et al.*, 2002). *T. pratensis* is also reported by Clements *et al*. (1999) to be susceptible to infection by *Erisyphe cichoracearum* (mildew) and *Albugo candida* (white rust) when grown under greenhouse conditions; in addition the smut *Ustilago tragopogonis pratensis* is occasionally found on *T. pratensis*. Other oomycete pathogens such as *Albugo tragopogonis* and *Bremia lactucae* can also be occasionally found on *T. pratensis* (Ellis and Ellis, 1985). These other pathogens have not been recorded within the PGE so it is assumed that any pathogen regulation is due to *T.* p*ratensis*.

T. pratensis has had little work dedicated to it due to its weed-like status. However Uphadhyaya *et al* (1993) reported some herbicidal control methods, concluding that grazing and chemical applications as succeful control mechanisms, but also identify *P hysterium* as a potential biological control agent. Some aspects of the natural history of *T. pratensis* has been reported by Qi *et al* (1993: 1996) relating to conditions for seed germination and seed bank survival, whilst Pico *et al* (2003) investigate inbreeding in *T. pratensis* and report that dispersal ability does not differ between inbred or outcrossed lines indicating the success of isolated, fragmented populations. Jorritsma-Wienk *et al* (2007), expanded on the germination work by identifying germination success of plants derived from varies different populations and habitats indicating that individuals from tall grass habitats grow more slowly than plants from low hight communities . However, plant size did not differ between sites or populations.

1.2.2. *Puccinia hysterium* (Str.) Röhl, 1813

*P. hysterium (*Figure 4) is a rust fungus which predominantly infects, *T. pratensis*, and may be found in North America on *T. buphthalmoides*, *T. dubius*, *T. floccosus*, *T. hybridus*, *T. latifolius*, *T. minor*, *T. orientalis*, *T. palaestinus* and *T. porrifolius* (Parmelee and Malloch, 1972). Due to the difficulty of identifying the rust, its recorded distribution within the British Isles is minimal (Figure 5), and although it is not explicit it is assumed that it will be recorded on *T. pratensis*.

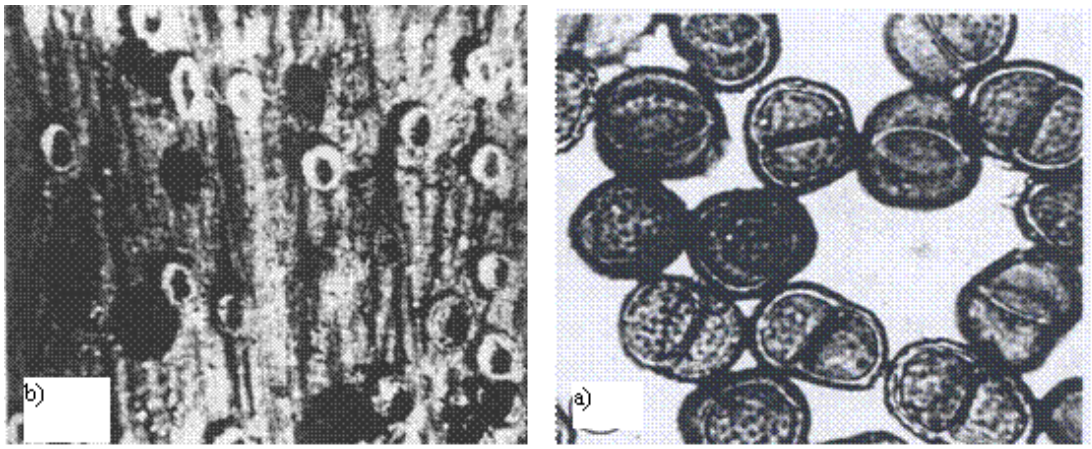


Figure 4. 1) *P. hysterium* aecia and telia on *T. pratensis* (mag. x8) and 2) *P. hysterium* teliospores (mag. x400) (Parmelee and Malloch, 1972)

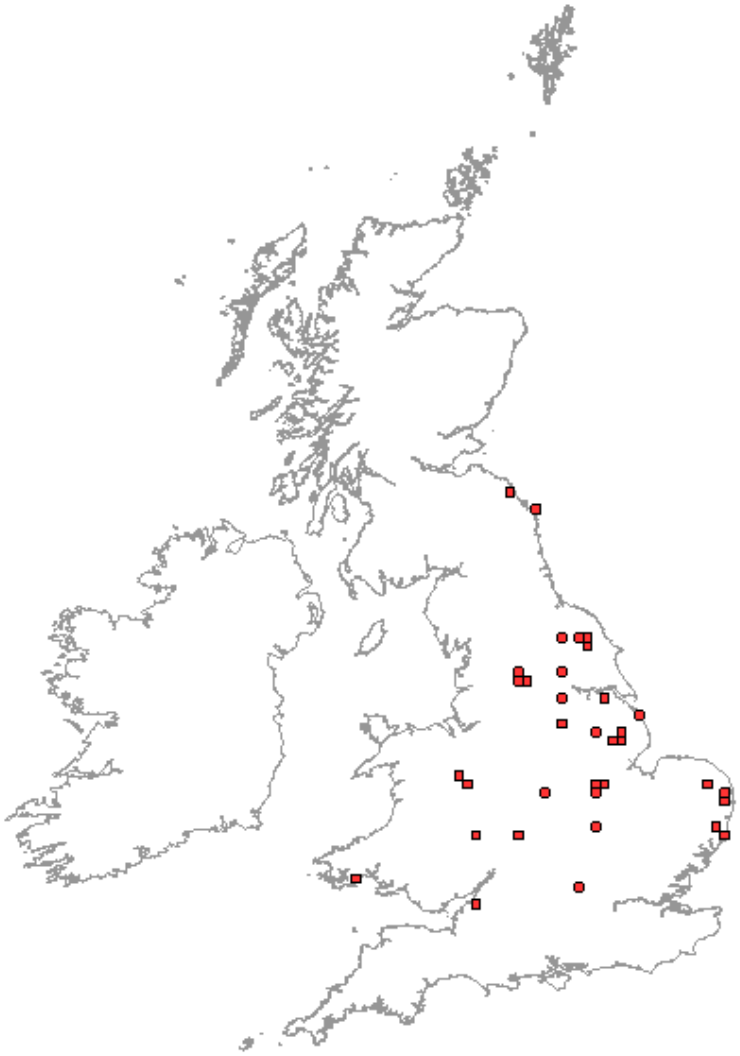


Figure 5. The distribution of *T. pratensis* in UK and Ireland 1900 – 2009. (NBN Gateway, 2009b)

P. hysterium is an autoecious, demicyclic rust fungus that does not have a repeating urediospore-producing phase (Wilson and Henderson, 1966). The life cycle of *P. hysterium* (Figure 6) involves teliospores being wind dispersed from infected second year plants to potential first year plants from June to the end of the season (Parmelee and Malloch, 1972). In hay meadows, such as the PGE, there is only a short period of teliospore dispersal before the hay cut at the end of June. It is unlikely that the teliospores are able to overwinter in the ground as *P. hysterium* is an obligate parasite that has not been cultured. The dispersed teliospores which land on first year plants germinate to develop basidia producing basidiospores; alternatively the basidiospores are directly transported to new hosts. Once on a first year host the basidiospores germinate to produce mycelia which become systemic, produce pycnia and survive the die-back period when the host overwinters below ground. The pycnia produce

pycniospores which allows for a sexual stage through hyphae which then produce more mycelia. In the following season the surviving plants re-emerge and aecia are then formed on plant parts, specifically on the stem producing aeciospores. Aeciospores then germinate on the host tissue, growing germ tubes (which either directly penetrate tissue or grow through natural openings e.g. stomata). After the aeciospores have germinated and grown mycelium within the plant, they produce telia, bearing teliospores on localised mycelium (Wilson and Henderson, 1966, Scott and Chakravorty, 1982, Cummins and Hiratsuka, 1983).

Due to the limited recorded distribution of this species there are very few published studies of the biology of *P. hysterium*, however van der Merwe *et al* (2007) have reported the evolutionary relationship of *P.hysterium* with other *Puccinia* spp.

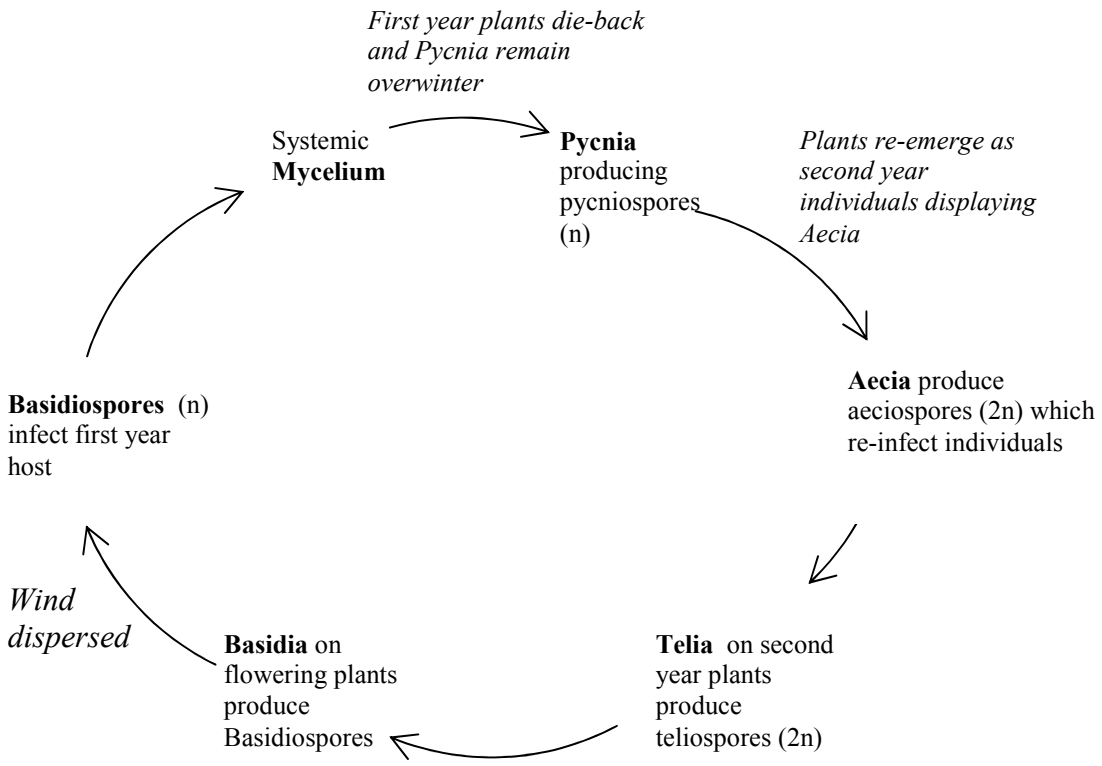


Figure 6. The assumed life-cycle of *P. hysterium* (n=haploid, 2n=diploid).

1.2.3. *Tragopogon pratensis* dynamics in the Park Grass Experiment

T. pratensis is described as outbreak species, shown by changes in frequency with a peak year in 1952 (Figure 1), such an outbreak may be part of longer term changes or cycles as Figure 1 only displays a record of presence of *T. pratensis* in plots over the study period and there is no indication of abundance within each sub-plot. In the PGE, *T. pratensis* usually exists at very low densities (typically <0.1%) on many of the non-acidified plots, but in 1993 the abundance on the limed parts of plot 18 accounted for 18% of the plot biomass. When *T. pratensis* was in higher density plots, there was also a high level of rust infection, thought to be *P. hysterium*, which was described as being host density-dependent (Silvertown *et al.*, 2002); therefore plant density would play a role in the epidemic. Because frequency of the host and abundance of the rust are correlated, such dramatic changes in host abundance would also be likely to affect pathogen distribution within plots (Dodd *et al.*, 1995).

The characteristic dynamics of an outbreak species are that the species increases in population size until an optimum where it then diminishes to a low level. The outbreak nature of *T. pratensis* is also apparent in the increase and decrease (Figure 7) of occurrences in plots treated with lime (Dodd *et al.*, 1995) where *T. pratensis* was not observed in the 5 year mean time-frame in 1930, but then increased in 1945 coverage and also in 1960 recording where it reached a maximum, and then declined to four sub-plots in 1975.

T. pratensis is a selfing outbreak species which, like many outbreak species, tends to demonstrate a higher level of inbreeding than species showing non-outbreak population growth (Silvertown *et al.*, 2002). One consequence of inbreeding is reduced genetic heterozygosity and diversity, constraining the ability of a population to adapt and survive in a mosaic of environments. In the PGE, outbreak species increase when there is a reduction in interspecific competition because these species have higher intrinsic rates of natural increase than other non-outbreak species. Outbreak species eventually decrease in population size due to lacking the genetic variation necessary to adapt and persist under conditions of interspecific competition. Additionally, with competition, outbreak species produce much lower numbers of seeds (Silvertown *et al.*, 2002).

Although there are many issues relating to experimental design with using data collected from the PGE, it provides a long-term data set including *T. pratesnsis* and more recently *P. hysterium* records which are not found elsewhere. Without such records the observed "outbreak" dynamics would not have been detected,

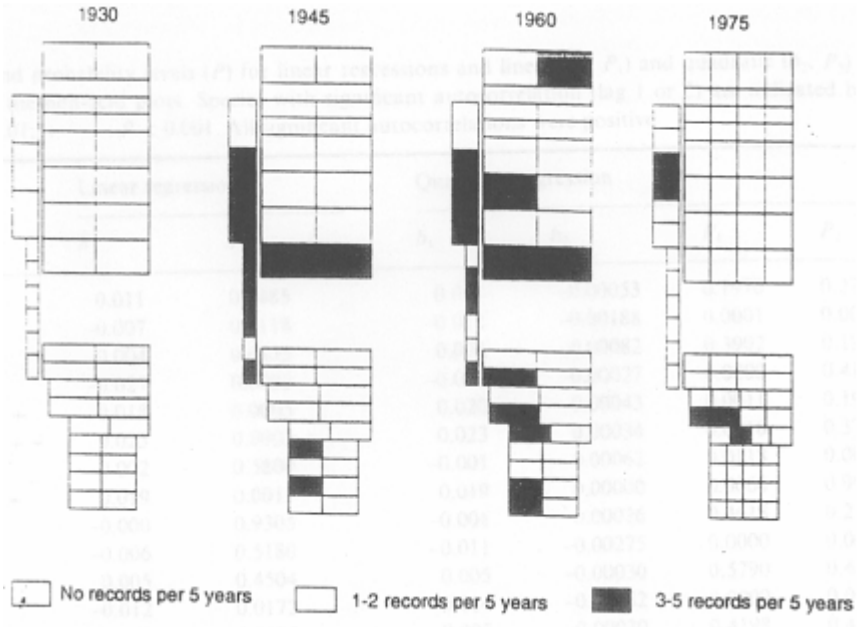


Figure 7. Presence of *T. pratensis* on non-acidified plots through time, using a 5 year running mean (Dodd *et al.*, 1995).

1.3. The biology of T. pratensis

In an attempt to validate the assumptions of the models within this thesis a range of analysis and experimentation were conducted. Attempts to validate some of the parameters however are inconclusive.

1.3.1. *T. pratensis* survey

Vegetative and flowering *T. pratensis* individuals and signs of rust infection were counted by G. Edwards (*unpublished*) just prior to the hay cut at the PGE in late-June from the years 1995 – 1998 and 2000 – 2004 using one 10 x 2 m quadrat placed in the centre of each plot. These counts may be an underestimation due to the difficulty of identification (G. Edwards *pers. comm.*). Between 2005 – 2008 the methods of data collection were modified by the author due to restrictions in place at the PGE relating to access and disturbance of the plots. The modified methods involved using five 1m x 1m

quadrats on the edges of each sub plot laid at random positions identified using a random number generator indicating distance along the edge transect. Within each transect, numbers of flowering and vegetative individuals were recorded and rust incidence noted.

The dynamics of healthy *T. pratensis* and *P. hysterium* infected individuals in the PGE over the period of 1995-2008 were analysed graphically by the author. The recorded densities were converted to estimated population sizes by multiplying the recorded density by plot size. One-way Analysis of Variance was conducted to assess the relationship between rust and *T. pratensis* incidence in relation to pH and fertiliser regime. Additionally, regression analysis was conducted to assess the relationship between host-density and rust-incidence and also rust-incidence on the number of hosts the following season.

The seeds data collected from the PGE were analysed using one-way ANOVA to ascertain the impact of infection on seed set numbers, and seed dimensions; whilst G-tests (Dytham, 2007) were used to assess seed germination success.

The population dynamics of *T. pratensis* in the entire PGE were plotted against year. A polynomial model of the 6th order was used to predict omitted data using Excel which tightly fitted the data for R_t (R^2=0.953) and also for I_t (R^2=0.873) which enabled interpolation of omitted R_t and I_t values. This was fitted to provide estimated populations for years without recorded data (1999, 2000 and 2005). The models presented in Chapters 2 and 3 were compared semi-quantitatively with the PGE data. Based on these comparisons a simplified model was proposed that produces dynamics reminiscent of the dynamics seen in the PGE from 1995 – 2008.

T. pratensis was recorded in all but one plot of the PGE (11/1) between 1995 – 2008, whilst in 15 of the subplots (Table 1) *T. pratensis* was absent.

Table 1. Sub-plots within the PGE where T. pratensis has not been recorded between 1995 – 2008.

Plot	pH treatment	Treatment
		FYM 1856-63, K
2/1c	5	1996
4/1c	5	P
4/1d	Unlimed	P
4/2c	5	N2 P
4/2d	Unlimed	N2 P
9/2d	Unlimed	N2 P K Na Mg
10 (B)	6	N2 P Na Mg
10 (C)	5	N2 P Na Mg
10 (D)	Unlimed	N2 P Na Mg
11/1a	7	N3 P K Na Mg
11/1b	6	N3 P K Na Mg
11/1c	5	N3 P K Na Mg
11/1d	Unlimed	N3 P K Na Mg
11/2c	5	N3 P K Na Mg Si
11/2d	Unlimed	N3 P K Na Mg Si

This data were used to observe if the host dynamics were variable across sub-plots and treatments. The change in the number of sub-pots where *T. pratensis* was recorded between 1995 – 2008 is show in Figure 8. There is an outbreak peak similar to that seen in the long-term datasets however the outbreak cycle is over shorter time series. When plotted in a comparable way to the data of Silvertown *et al.* (2006), where it was shown that in non-acidified plots there was an outbreak pattern, this pattern can also be seen in the recent data (Figure 9).

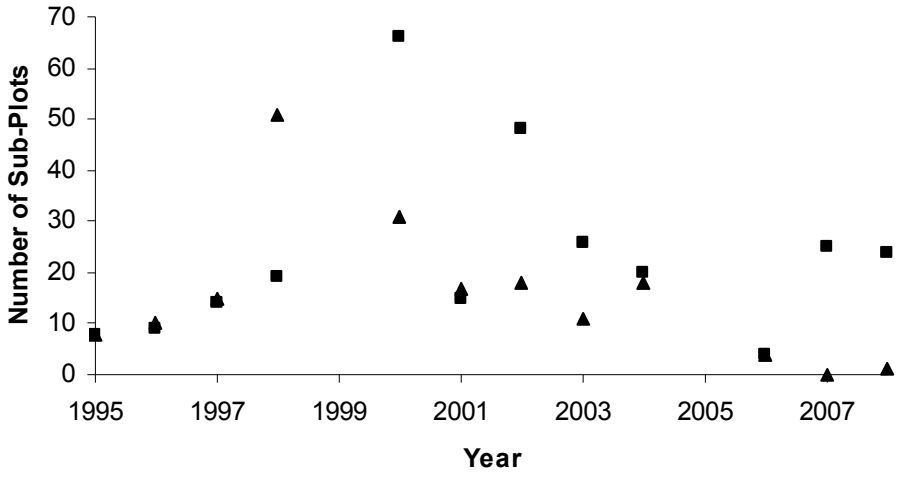


Figure 8. The recorded number of sub-plots containing healthy individuals (■) and rusted individuals (▲): 1995 – 2008.

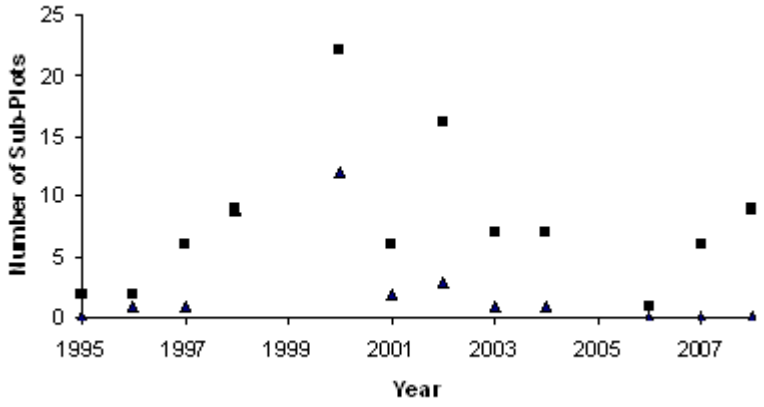


Figure 9. The recorded number of acidified sub-plots containing healthy individuals (■) and rusted individuals (▲): 1995 – 2008

The number of plots with *T. pratensis* recorded, showed dynamics that increased to a maximum in 2000 and then decreased (Figure 8,Figure 9). The same pattern was seen in the number of plots with rusted plants; however in 2007 increase in incidence of healthy *T. pratensis* did not correspond.

The survey allowed estimates of the total number of healthy individuals within the PGE collected between 1995-2004 and 2006-2008 and showed that the overall pattern was reminiscent of the outbreak pattern observed in the long-term data sets (Figure 10). The following polynomial was used to interpolate the missing data points:

$$\text{population count} = 0.0431x^6 - 2.0498x^5 + 37.147x^4 - 315.3x^3 + 1219.6x^2 - 1716.8x + 955.93$$

(R^2 =0.95) where x is years from 1994.

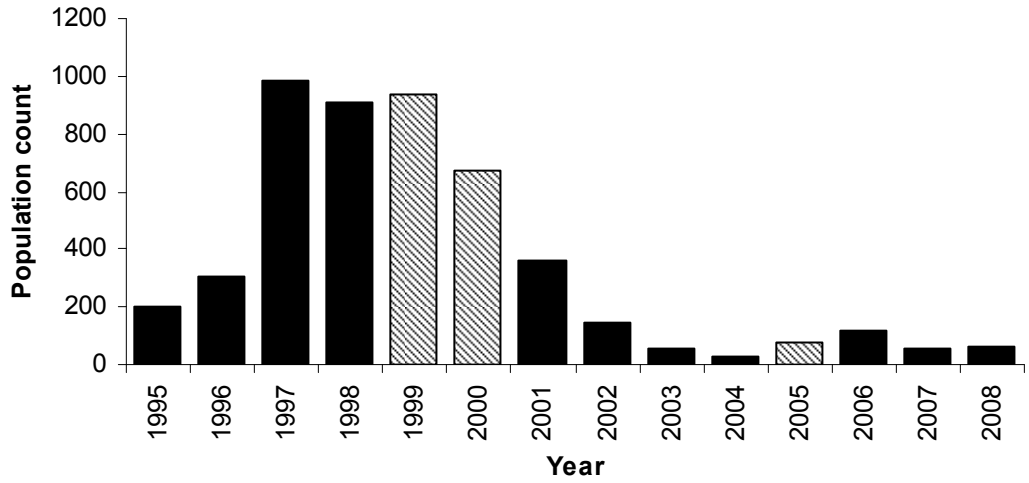


Figure 10. The estimated population sizes of healthy second year *T. pratensis* between the years 1995 – 2008 with interpolated values for years 1999, 2000 and 2005.

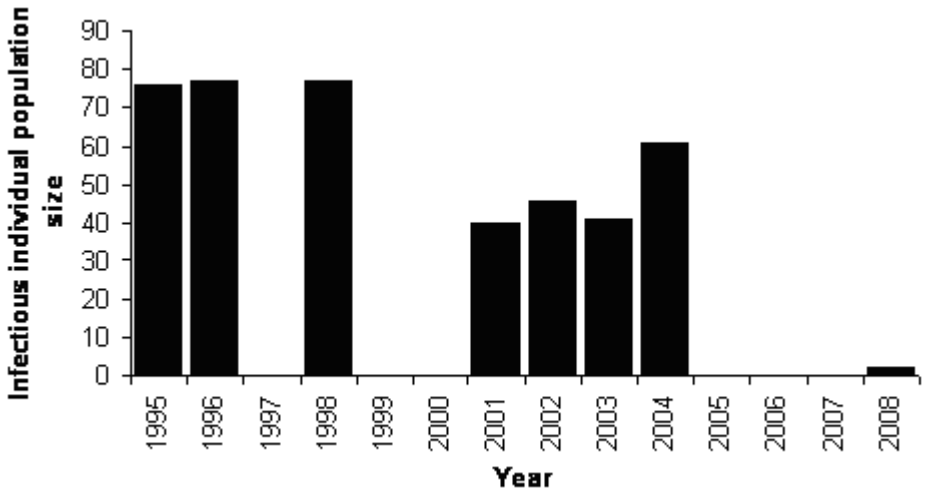


Figure 11. The population counts of infected second year *T. pratensis* between the years 1995 – 2008. No recordings for 1997, 1999, 2000 and 2005. Zero counts in 2006 and 2007

There appeared to be a general reduction in the numbers of infectious individuals over the study period (Figure 11) when comparing the periods 1995-1998 and 2001-2004.

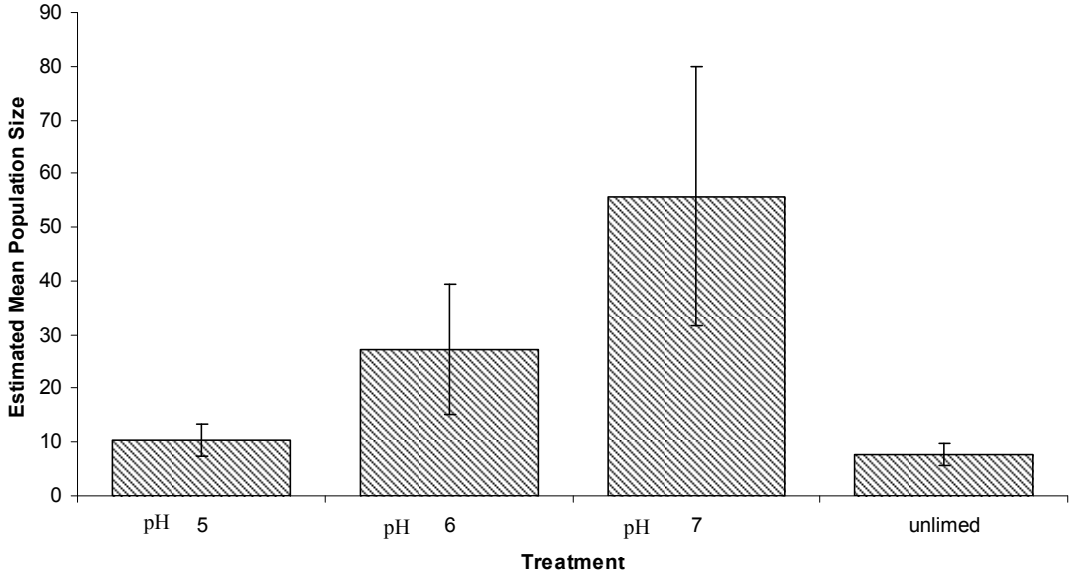


Figure 12. The estimated mean and standard errors of population sizes of healthy second year *T. pratensis* between the years 1995 – 2008 in relation to pH treatment.

There was no significant difference ($F_{3,\ 93}$=2.49) between the pH treatments and the estimated mean number of individuals (Figure 12). Sub-plots were then grouped by fertiliser regime and further analysis performed. There was a significant difference ($F_{29,\ 67}$=8.63***) between the fertiliser treatment and the estimated mean number of individuals surveyed indicating that nutrient conditions influence the abundance of *T. pratensis* (Figure 13).

Plots 18 and 18.2 (treated with 96 kg N ha^{-1}, 225 kg K ha^{-1}, 15 kg Na ha^{-}1, 10 kg Mg ha^{-} 1) had plant counts at a scale above those of the rest of the PGE plots and are shown separately.

T. pratensis was only recorded in a few sub-plots for 6 or more of the 12 year survey. These recorded dynamics for 12 years of observations (Figure 14), 11 years (Figure 15), and the remaining plots with more than 6 years of observations (Figure 16).

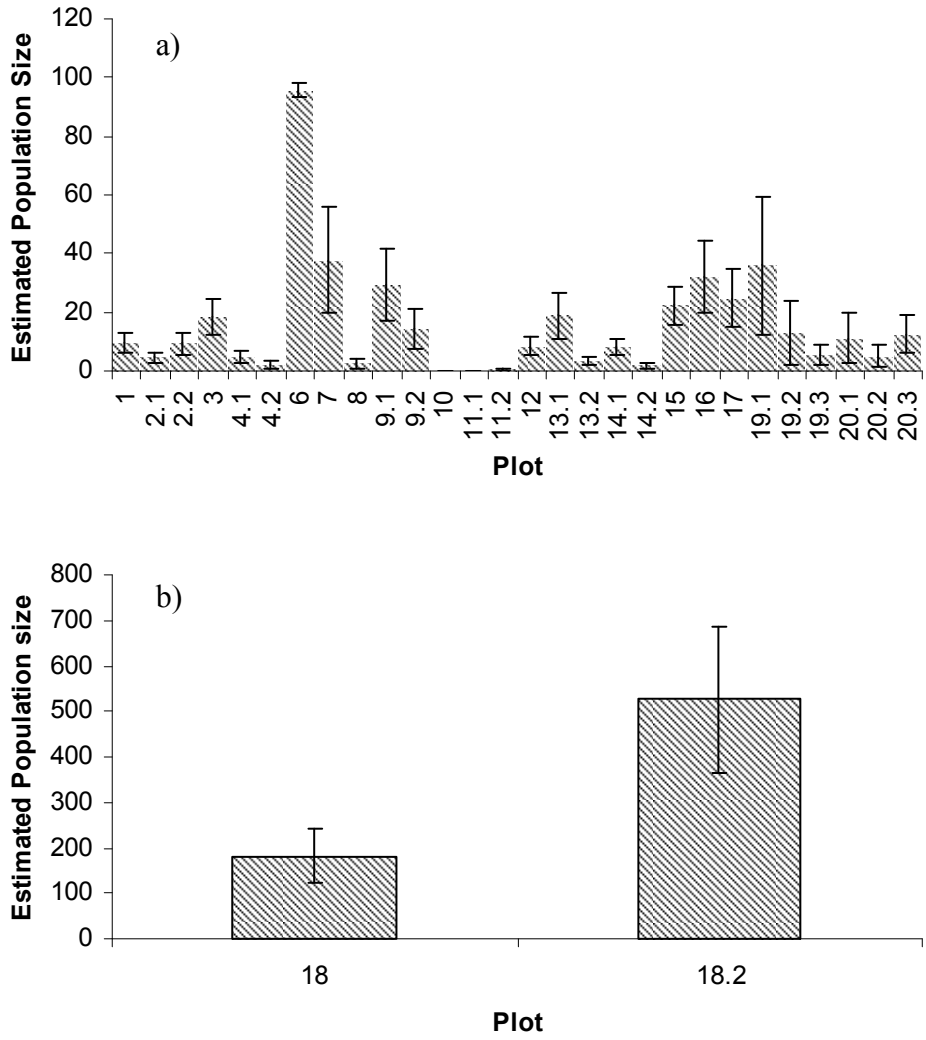


Figure 13. The estimated mean and standard errors of population sizes of healthy second year *T. pratensis* between 1995 – 2008 at the plot level a) indicates the estimated mean population all of the PGE plots except for plots 18 and 18.2 which are described in b).

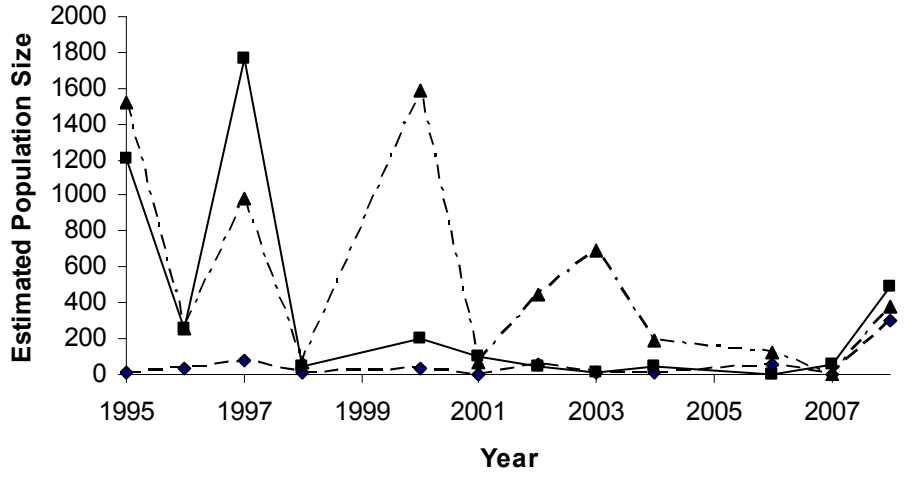


Figure 14. Population dynamics of *T. pratensis* in sub-plots 18c 18a **and** 18.2 **across the details survey between 1995-2008 where plants were recorded in 11 of the years.**

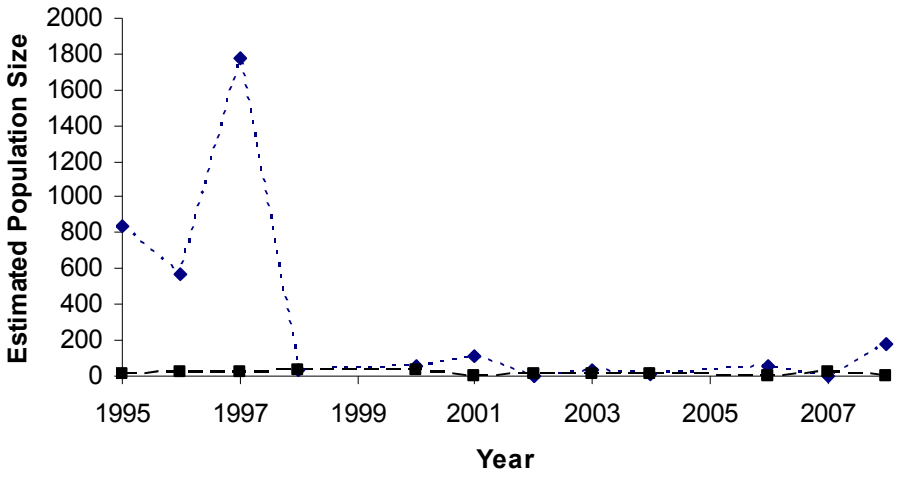


Figure 15. Population dynamics of *T. pratensis* in sub-plots 2.2b and 18b across the details survey between 1995-2008 where plants were recorded in 10 of the years.

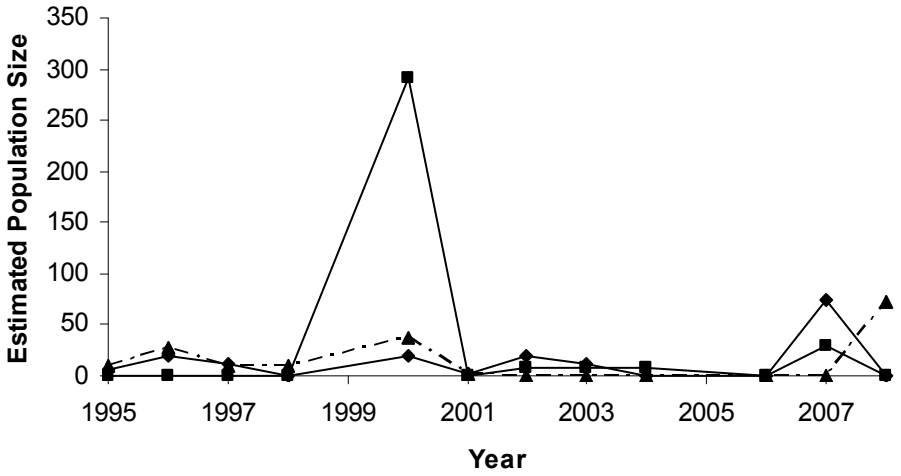


Figure 16. Population dynamics of *T. pratensis* in sub-plots with number of year observations. 2.1b (8 years), 15b (7years) and 1b (8years) between 1995-2008.

Dynamics at the plot level (Figure 17) shows diminishing cycles of *T. pratensis* in plot 18 followed by a period of low incidence, peaks followed by decline followed by an increase is seen in plots 1, 2.1, 2.2, 6, 9.1 and 15. These dynamics are reminiscent of simulated plots under conditions of medium host and high pathogenicity whereby the pathogen causes a crash and populations drop below the epidemic threshold allowing for the host to re-establish to a stable steady-state

Figure 18 shows that there were fluctuations with pH treatment. However, there was a difference in the mean abundance of individuals with higher populations recorded in soils that had been limed to avoid acidic conditions. Similar dynamics are seen in Figure 19 where observations are grouped based on plots treated with farmyard manure and/or fishmeal and those not. There are an increased mean number of individuals observed in plots treated with non-farmyard manure treatments, however this analysis does not take in to account the application rates and type of treatment applied. Similar dynamics are observed for the number of infectious individuals recorded within plots treated with farmyard manure applications and those with alternative treatments (Figure 20). However what is clear is that there is no difference in the incidence between the two treatment types.

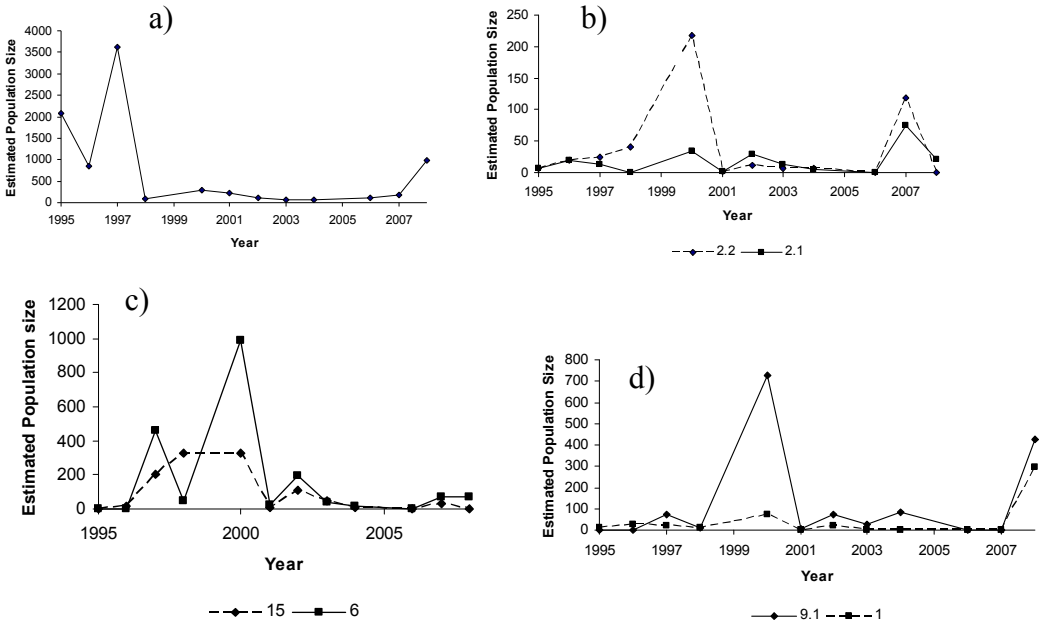


Figure 17. The dynamics of a) plot 18 with 12 years of data, b) plots 2.1 and 2.2 with 10 years of data, c) plot 15 and plot 6 with 9 years data and d) plot 9.1 and plot 1 with 8 years of data.

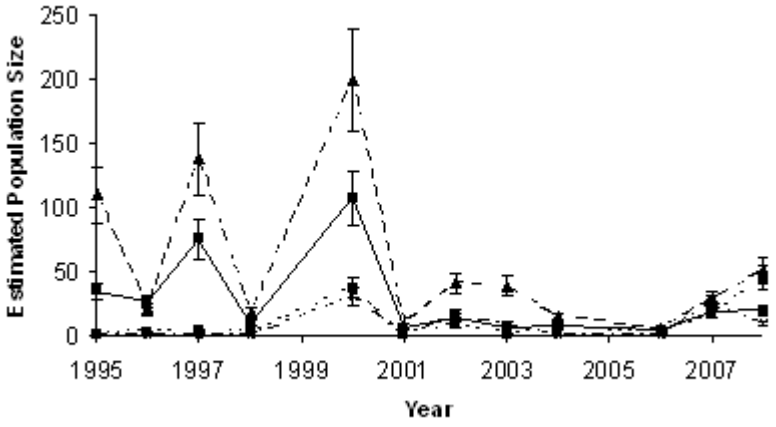


Figure 18. The population dynamics of *T. pratensis* when grouped by soil pH regime (5 , 6 , 7 and unlimed) between 1995 – 2008.

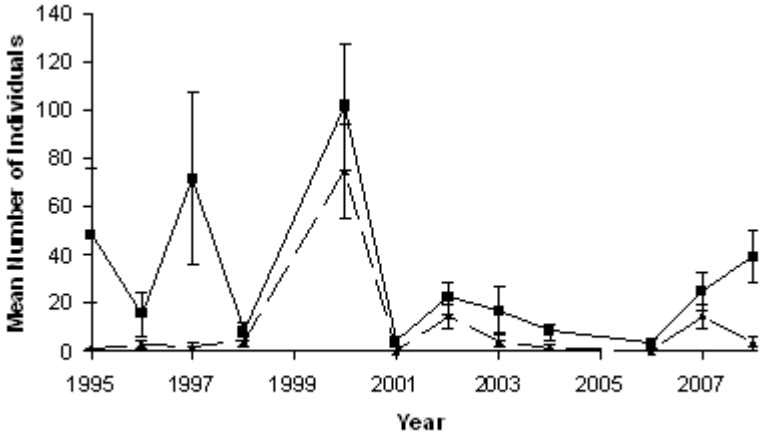


Figure 19. The population dynamics of the mean number of individuals of *T. pratensis* recorded in plots with farmyard manure and/or fishmeal nutrient regimes (–▲–) and chemical fertilisers (—■—) between 1995 – 2008.

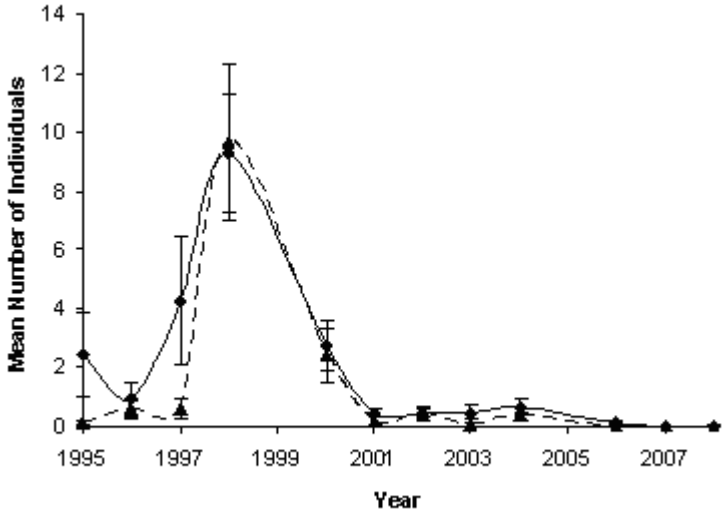


Figure 20. The population dynamics of the mean number of infectious individuals of *T. pratensis* recorded in plots with manure and/or fishmeal nutrient regimes (–▲–) and chemical applications (—■—) between 1995 – 2008.

T. pratensis was first recorded in the PGE in 1862 and has been continually found in a range of plots at levels as low as 0.001% (dry-mass of the hay cut) of the community of plots to approximately 3.8% of the plot community structure. It was noted by Dodds *et al*. (1995) that *T. pratensis* displayed characteristic population dynamics of an outbreak species based upon the numbers of non-acidified subplots where *T. pratensis* presence was recorded. Over some 60 year period, the number of plots with recorded individuals increased to a peak in the mid-1950's and subsequentially decreased to low levels in the 1980's. In recent years *T. pratensis* has been recorded in all but plot 11.1, which had

been treated with $Ca(H_2PO_4)_2$, K_2SO_4, Na_2SO_4, $MgSO_4$ and high levels of $(NH4)_2SO_4$. A possible reason for the absence of *T. pratensis* is due to it being outcompeted by fast-growing species in a nutrient rich plot compared to the status of other plots within the PGE.

Silvertown *et al*. (2002) reported that in years of high *T. pratensis* density there was a high incidence of *P. hysterium*. Here, it is shown that the dynamics demonstrated by *T. pratensis* are not necessarily characteristic of an outbreak species, as originally described. The original data only present the presence of *T. pratensis* without discussion of density, abundance or infectious status of plants (Dodd *et al.*, 1995). Furthermore, the plot data presented are only for those with non-acidified soil conditions. The number of plots with *T. pratensis* present has increased and decreased over a much shorter timescale, reaching a peak in 2000 and subsequentially decreasing to low levels in recent years. However, there seem to be signs that the number of plots showing presence of individuals is again on the increase (Figures 14– 20), potentially reflecting a cyclical pattern of population dynamics. Because the data recorded since 1995 contain detail on infection status, it is also apparent that the numbers of plots with infection in both acidified and non-acidified conditions increase at the same time and subsequentially diminish; however, in 2007 and 2008 the increase in plots with healthy hosts has not been mirrored in the numbers of plots with diseased hosts. This suggests that the plant population decreased to such low levels, below the epidemic threshold so that the rust population reduced to unsustainable levels. Although there are lower abundances in acidified subplots in the numbers of individuals counted, the overall qualitative patterns are the same irrespective of pH treatment applied.

Considering the PGE as a single community, the estimated population sizes of *T. pratensis* has fluctuated; however *P. hysterium* has decreased in an almost linear fashion since 1995, but this may be part of long-term dynamics that can not be observed in short term studies. This suggests that the host population sizes observed since 1995 have been insufficient to support the rust population leading to its crash. This in turn may have led the host population to increase to a stable population size. However, it would be unwise to consider the PGE as a single, uniform habitat. There are particular plots where the mean population densities of *T. pratensis* are significantly different from others. For example plots 18 and 18.2 which have the same fertiliser applications as 11/1, but with lower levels of ammonium sulphate and no $Ca(H_2PO_4)_2$, whilst plot 6 has high densities

with the same treatment as 11/1 but with half the amount of ammonium sulphate and additional $Ca(H_2PO_4)_2$. When considering each plot individually for a range of plot populations there are diminishing cycles, which tend towards low densities (Figure 17), which in recent years have started to increase once more. These same patterns are seen when considering each sub-plot on an individual basis (Figure 14-16). If it is assumed that each pH treatment is a factor determining *T. pratensis* and/or rust incidence the population dynamics demonstrate decreasing population cycles towards low levels which appear to be increasing in recent years (Figure 18). These population cycles that have been observed in recent years are reminiscent of model simulated dynamics under conditions of high plant performance and medium pathogenicity across a range of model structures, however the results form the PGE indicate that *T. pratensis* is a low performing host. In the model simulations, the host population cycles with the number of infectious individuals lagging behind by two years; the host population crashes to low levels, infectious individuals may become extinct within the population, and allow the host population to increase towards a steady population size. This may reflect the situation observed in the PGE. If correct, then it is unlikely that *T. pratensis* can be described as an outbreak species regulated by *P. hysterium*, it would be more accurate to describe the system as fluctuating, influenced in part by the pathogen.

Treatments with farmyard manure and/or fishmeal lead to a reduced population of *T. pratensis* compared to plots where chemical based fertilisers are added. Irrespective of population size and treatment, the host population fluctuates annually in similar patterns. However, the low numbers recorded in recent years have been in plots treated with organic fertilisers, whereas in chemically-treated plots *T. pratensis* is increasing.

1.3.2. Seed Production

Seed was collected by G. Edwards (*unpublished*) in 2000 from 49 infected and 33 non-infected individuals, producing 208 and 976 seeds respectively. Seeds were germinated in control cabinets at 20^{o}C and the dates of germination were recorded for the 143 seeds collected from infected and the 167 from healthy individuals. In 2005, six controlled environment growth chambers were set up for 12 hours at 10^{o}C (representing temperature conditions at night) and 12 hours at either: 10^{o}C, 15^{o}C, 18^{o}C, 20^{o}C, 23^{o}C or 25^{o}C. In each chamber, 20 seeds were germinated on moistened filter papers in Petri

dishes; the seeds were selected at random from a mixed stock of seeds collected from sites at the PGE, Sidelands field at the Wye campus of Imperial College London (Kent), the Royal Horticultural Society (RHS) gardens, Wisley, and Wisley Village (Surrey). The dishes were observed each day for 10 days and the number of new germinating seeds was recorded. Seeds that did not germinate after 10 days were assumed to be non-viable under these experimental conditions.

In order to assess the impact of *P* hysterium on the production of *T. pratensis* seeds, data were recorded by G. Edwards (*unpublished*) in 1997 on the mass (mg), length (mm) of pappus and seeds, and also the numbers of seeds set per individual, for 15 infected individuals and 15 healthy individuals. In 2000, seeds were collected from 49 infected individuals and 33 healthy individuals, number of seeds per individual was counted and the same measurements recorded. A 2-way analysis of variance was conducted in SPSS 10.0. (SPSS Inc. 1999) to compare seed number, mass and lengths between infected and uninfected individuals.

There was a significant difference in seed production ($F_{2,109}$=137.01***) between non-infectious individuals than those that are infected (Figure 21).

Seeds from infected individuals were heavier from the sample in 1997 whilst they are lighter in 2000 (Figure 22), however there was no significant difference in the mean mass of seeds between seeds from healthy and infected plants ($F_{1,1084}$=0.98 NS) Pappus length was significantly ($F_{1,1084}$ =2964.27***) shorter in seeds from infected individuals than those from healthy individuals (Figure 23).

Seeds from healthy individuals also produced significantly ($F_{(1,1084)}$=646.08***) longer seeds than those collected from infected adults (Figure 24)

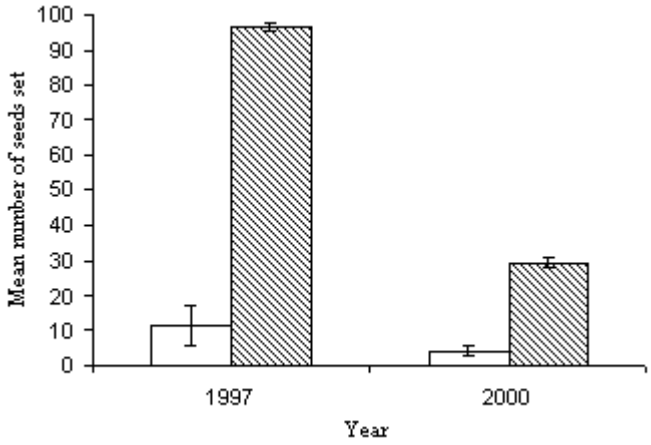


Figure 21. The mean number of seeds set for ☐ infected and ▨ non-infectious individuals and the corresponding standard errors.

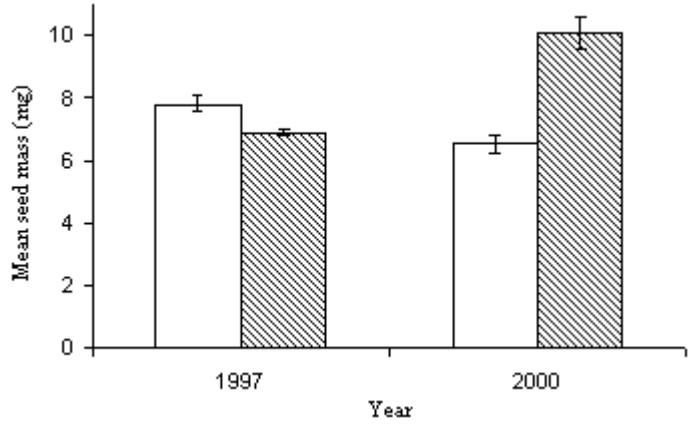


Figure 22. The mean mass of seeds set for ☐ infected and ▨ non-infectious individuals and the corresponding standard errors.

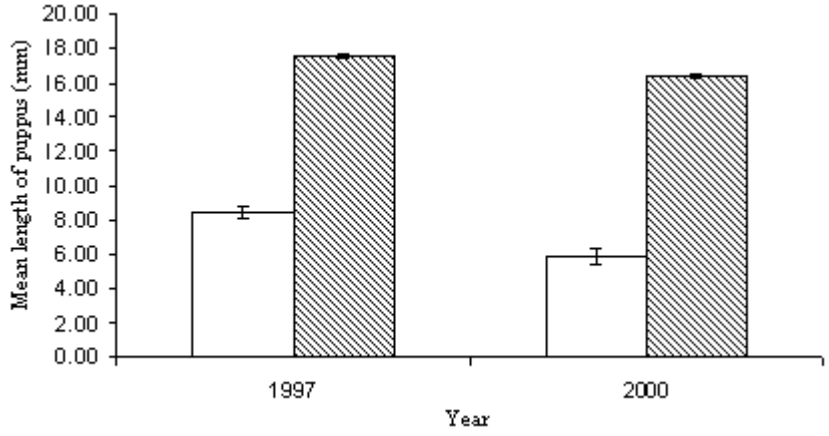


Figure 23. The mean pappus length of seeds set for ☐ infected and ▨ non-infectious individuals and the corresponding standard errors.

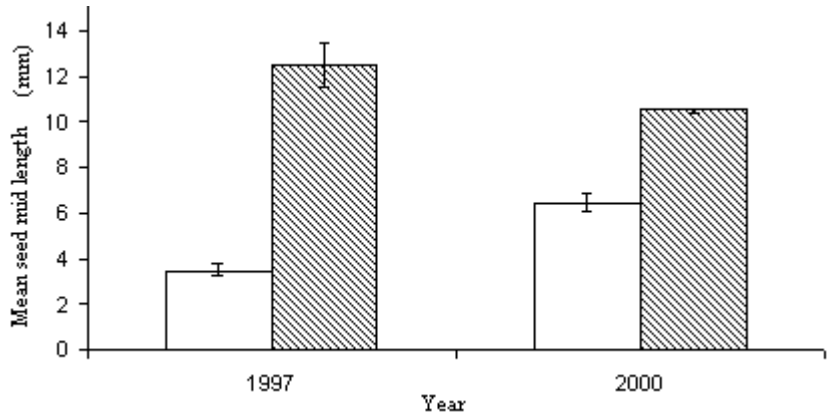


Figure 24. The mean seed mid-length of seeds set for ☐ infected and ▨ non-infectious individuals and the corresponding standard errors.

1.3.3. Seed Germination

In order to consider the viability of seeds produced from infected and non-infected second year plants. Small germination experiments were conducted. The optimal temperature for successful seed germination was found to be 20^{o}C (Table 2) where all but one of the seeds germinated

Table 2. Results for cumulative number of seeds germinated at varying daytime temperatures over a period of 10 days.

Day	Day Temperature °C					
	10	15	18	20	23	25
1	0	0	1	0	0	0
2	0	0	6	1	2	0
3	0	3	8	10	8	8
4	0	4	11	13	12	13
5	1	11	13	17	15	16
6	1	11	13	19	15	16
7	1	11	14	19	15	16
8	3	12	14	19	15	16
9	4	12	14	19	15	16
10	4	13	14	19	15	16

Of the 167 seeds selected for germination set by uninfected plants 124 successfully germinated within 25 days. The median number of days for germination of seeds from an infected adult was 11 days (Figure 25) and also 11 days for seeds from healthy individuals (Figure 26).

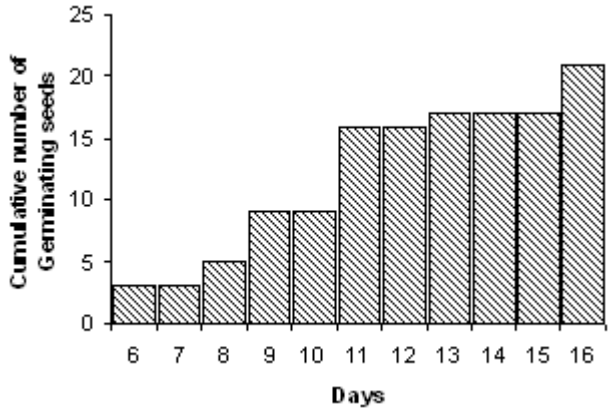


Figure 25. The number of days taken for seeds set from infected individuals to germinate.

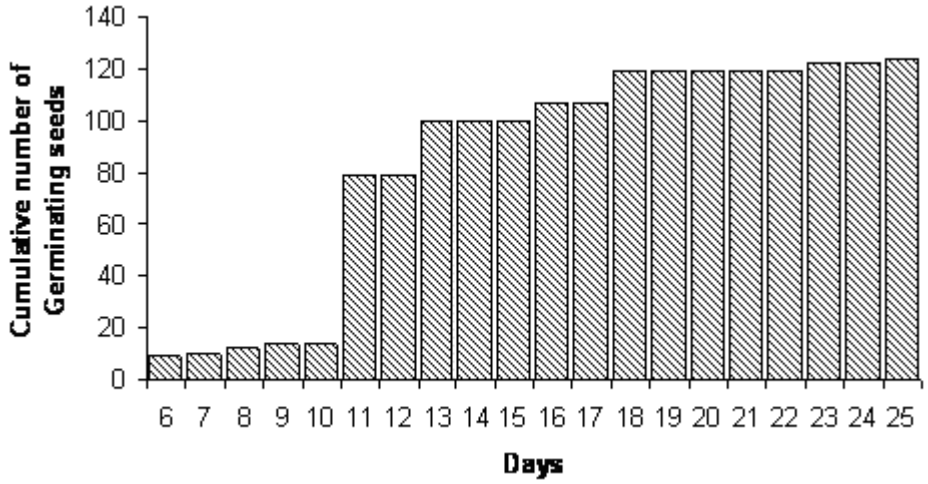


Figure 26. The number of days taken for seeds set from non-infected individuals to germinate.

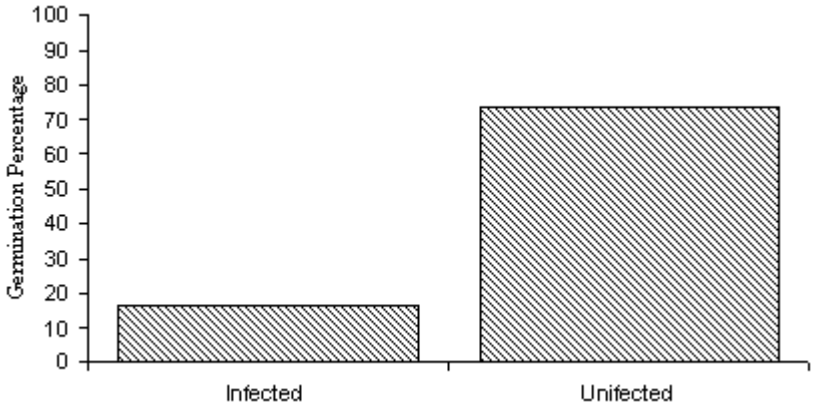


Figure 27. Percentage of seeds that germinate collected from infected and uninfected individuals.

There was a significant ($\chi^2_{(1d.f.)}$=40.46***) difference in the germination percentages between seeds from infected and healthy plants (Figure 27). Using the mean number of seed set in 1997 by infected individuals and the germination success rate, it is possible to conclude that an infected individual will produce a mean of 1.98 successful

germinating seeds, where as a healthy second year will produce a mean of 73.46 successfully germinating seeds. This effectively shows that *P. hysterium* is a castrating pathogen.

Pathogen infection leads to individuals producing fewer seeds, which in turn have a lower germination rate. This is contrary to the findings of Paul and Ayres (1986c) who reported that the similar rust, *Puccinia lagenophorae*, had no impact on production or on viability of seeds collected from inoculated individuals of groundsel (*Senecio vulgaris*). However, subsequentially, Paul and Ayres (1987) reported that seed production was drastically reduced in infected hosts. Therefore it supports the hypothesis that *P. hysterium* acts by castrating the host so impacting on the subsequent population recruitment as opposed to simply altering the survivorship of hosts. In the case of the *P. lagenophorae – S. vulgaris* system, where seed production is related to adult dry mass, senescence occurs earlier in the season and individuals develop flowers later than uninfected individuals, so having less time to develop viable seeds (Paul and Ayres, 1987). This could be the case in the *T. pratensis – P. hysterium* system, however there are no data regarding host dry mass differences between infected and uninfected *T. pratensis*. The impact on seed production could be due to allocation of nutrients in plant defence or active translocation of nutrients by the pathogen, so in turn reducing plant growth, allowing for reduced maturity at the point of setting seed. A reduced seed mass is seen in a range of rust-infected plants such as: *P. coronata* (crown rust) infecting *Lolium multiflorum* (Mattner and Parbery, 2007) and *Hordeum vulgare* infected with *P. hordei* (Das et al., 2007). Additionally, seeds from infected adults have a shorter pappus length and shorter mid-lengths to aid wind-dispersal. It is reported that smaller seeds with smaller pappus lengths disperse further than heavier seeds with large pappus lengths in *T. dubious* (McGinley and Brigham, 1989). This could be a potential evolutionary trade-off, whereby infected adults produce seeds with less mass and pappus length to increase seed dispersal away from diseased regions. There is no evidence to suggest seed infection by the rust.

The optimal condition for seed germination was 20^{o}C. This corresponds to the 20cm below-soil temperatures recorded at Rothamsted in May, where the mean monthly temperature for 2008 was 21.3^{o}C, whereas it was 13.8^{o}C, 23.5^{o}C and 25.3^{o}C in April, June and July respectively. The median germination time was the same for infected and uninfected individuals; suggesting that diseased adults produce a majority of nonviable

seeds, however those few that are viable will germinate in the same time as those from uninfected adults. It is likely that viable seeds from infected plants are not outcompeted by seedlings produced from uninfected individuals. The proportion of seeds germinating from uninfected individuals is within the range reported by Frantzen (2002) for *S. vulgaris*, where between 0.7 – 0.85 of the seeds planted emerged as seedlings.

1.3.4. Mortality and inoculation experiments

In an attempt to parameterise some aspects of the model relating to mortality and pathogen infection probabilities, artificial cohorts were cultivated and attempts to inoculate were made. However, due to the low numbers of individuals inoculated, parameter estimations could not be made with any degree of certainty.

To address mortality, 100 seeds selected at random from collections made in 2005 and 2006 from *T. pratensis* stocks at RHS Wisley, and also from plants within the PGE were germinated, planted in individual pots (diameter 130mm, height 121mm) and observed over the course of their life-cycles to determine cohort survival of healthy individuals.

As the pathogen is an obligate biotrophic parasite, it was not possible to isolate the pathogen and artificially inoculate hosts. First year individuals from seeds collected in 2004 were exposed to the pathogen using a range of methods to identify the optimal procedures for inoculation.

The methods tested involved exposure of first year plants to second year plants bearing aecia and telia obtained from sites at RHS Wisley. First-year plants were exposed to spore-bearing plants: proximity exposure, artificial application at dusk, and artificial application followed by placement in a dew-chamber. In the proximity exposure, four infectious individuals were positioned within 12 first year plants (Figure 28).

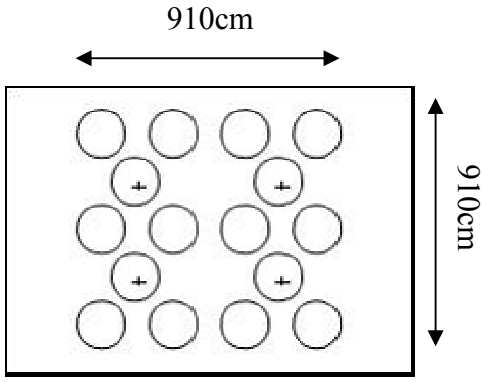


Figure 28. Layout of proximity exposure experiment (+ indicates Infected 2nd year individual, open circle is a 1st year individual).

Artificial inoculations were made by brushing infectious plant material with a camel-hair brush into talc, and then brushed over 12 first year individuals at dusk, so optimising stomatal opening and enclosing the pots in transparent plastic bags to maximise humidity. In the third method, artificial inoculations were made as before but 20 of the exposed plants were then placed in dew-chambers at CABI Biosciences, Silwood Park, for 4 days in August 2005. The size of experiments was restricted due to the number of plants grown from germinated seed and also due to transportation to the facility. This experiment was repeated using dew chambers at Imperial College, Silwood Park Campus, in September 2006 on 48 first year plants grown from the seed stock collected in 2005.

The only method of inoculation that resulted in symptoms of *P. hysterium* infection on second year plants was artificial inoculations followed by the dew chamber method. In 2005 of the 20 first year individuals exposed only three developed aecia in the second year. In 2006 of the 48 exposed individuals nine became infected individuals.

Survivorship probabilities are shown in Figure 29 for the different stages in the lifecycle of *T. pratensis*. Survivorship is indicated for both within and between seasons for each life-stage. The overall trends suggests that survivorship within season was higher than between season. Although survival was high from 2005 to 2006, in subsequent years there was much higher between-season mortality. Although *T. pratensis* is predominantly biennial, it was observed that pot plants can extend life-cycles by having repeated non-flowering first-year stages (*S`*), or healthy second-year stage (*R`*). It must be noted that these are under ideal conditions where plants are removed from biotic and abiotic stresses. Because of the minimal successful inoculations, there were insufficient numbers of infected individuals to ascertain mortality rates.

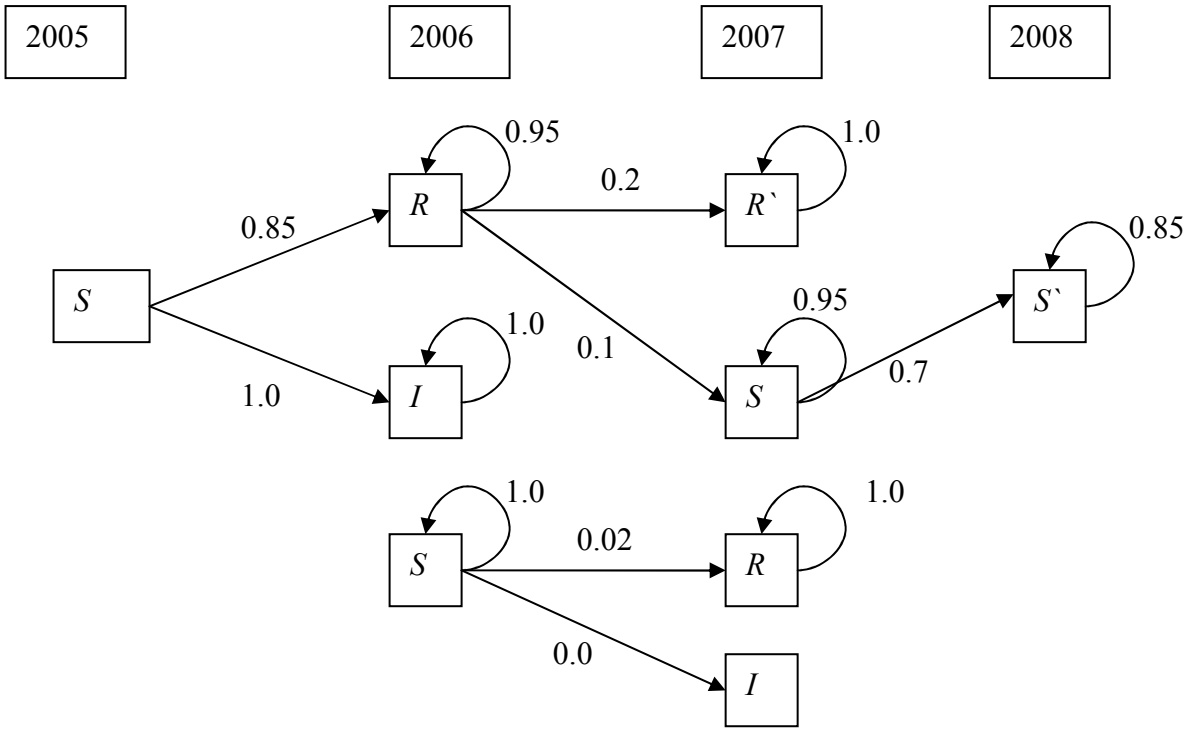


Figure 29. A representation of the survivorship of *T. pratensis* between and within seasons. *S* – first year individuals, *S`* - first year morph-type second years *R* – uninfected second year individuals, *R`* - third year individuals with second year morphology, *I* – infected second year individuals.

The transmission rate *c* is a composite of sporulation, dispersal and infection characteristics. The natural transmission rate is likely to be low as shown by the poor uptake in the proximity experiments. Similarly the probability of an exposed individual become infectious is also low demonstrated by the minimal number of exposed individuals becoming infectious. The pathogen isolates from RHS Wisley used in the proximity and dusting experiments was the same as that in the direct dew chamber experiments where a low level of infection did emerge in second year plants. It is possible that the rust requires higher humidity than was produced outwith of the dew chamber experiments. However, similar low probabilities of infection (0.15-0.35) were reported by Frantzen (2002) when *S. vulgaris* is artificially inoculated with *P. lagenophorae*, under favourable conditions.

Little conclusive information was obtained on host survivorship between and within season as there was variation between the generations. During the four years of the experiment there were populations comprised of: first year seedlings in 2005 grown from seeds collected from the PGE; in 2006 there was a mixed population of newly

grown seedlings and infected and non-infectious second years which were the re-emerged individuals from 2005; in 2007 the small numbers of individuals comprised of second year plants that had re-emerged from the 2006 seedlings, seedlings produced by the 2006 flowering adults, and also some 2006 second year individuals that re-emerged for a third year which resembled second year plants. In 2008 the only plants present in the population were re-emerged 2007 second year plants resembling first year seedlings. The within-season mortality was low for seedlings, infectious individuals and non-infectious second year plants, and also for second year plants emerging with seedling morphology and third year plants emerging with second year morphology. This cohort study allowed approximate estimations of background mortality rates, both between and within season, however the parameter values remains unknown for plants not grown in pot cultures.

1.4. Host – pathogen interactions in non-cultivated populations

The influence of pathogens and diseases on plant populations are covered by Burdon (1987), with several examples drawing on work in natural plant populations. Pathogens may play a key role in natural plant communities, affecting viability and fecundity of individual plants, reducing population size and density, generating selective forces for genetic change, altering community structure and therefore impacting on performance (Burdon, 1982, Burdon *et al.*, 1989, Thrall and Burdon, 1997).

Dobson and Grenfell (1995) argued that studies observing disease of natural plant populations were rarely conducted; the large body of literature often described empirical aspects without relating the implications for linking to theoretical work or applications. Some examples of general descriptive studies are listed in Table 3.

An example of a study in a natural plant – rust system was conducted by Smith *et al*. (2003) recording eleven years of observations at initially 133 sites, the populations of *Filipendula ulmaria* and its associated rust fungus *Triphragmium ulmariae*. Although this study was mainly observing the metapopulation dynamics of the plant, the epidemiology relating to these dynamics was a strong focus of the study. Over the eleven year period, the number of sites, number of infected individuals and uninfected hosts increased, also it was noted that approximately 30% of the sites surveyed always

had the rust present whilst 30% remained rust free. Furthermore, the number of diseased plants in populations and also disease severity increased to a maximum and then fell away in a similar manner to that observed for *T. pratensis* in the PGE.

A study on natural populations of *Plantago lanceolata* and its obligate pathogen, the powdery mildew *Podosphaera plantaginis* (Laine and Hanski, 2006), indicated several factors that can affect the incidence of fungal pathogens. Variation in disease presence could be associated with precipitation levels, with evidence also for variation due to location and microclimate. This study supported previous work indicating that disease incidence is higher in larger natural populations, than small isolated populations (Alexander, 1990). A study in the same system (Ovaskainen and Laine, 2006) showed that the majority of dispersing spores landed within $1m^2$ of the infected host, therefore pathogen spread would be localised during an epidemic.

Table 3. Examples of some plant pathogen studies in natural systems

Host	Pathogen	Source
Hordeum spontaneum	*Erysphe graminis*	Dinoor and Eshed (1990)
Hordeum spontaneum	*Drechslera teres*	Dinoor and Eshed (1990)
Hordeum spontaneum	*Ustilago nigra*	Dinoor and Eshed (1990)
Senecio vulgaris	*Puccinia lagenophorae*	Frantzen and Müller-Schärer (2002)
Silene dioica	*Microbotrym violaceum*	Carlsson and Elmqvist (1992)
Linum marginale	*Melampsora lini*	Jarosz and Burdon (1991)
Trientalis europaea	*Urocystis trientalis*	Piqueras (1999)

Within local regions, pathogens are often adapted to their host population; therefore within a mosaic community of plants, effective dispersal may be interrupted as pathogens may be trapped by non-susceptible plants (Dinoor, 1981, Crute, 1994, Keesing *et al.*, 2006)

There are examples other than *P. hysterium* of autoecious, demicyclic rusts infecting natural plant populations. Agro and Shattock (1999) report studies on *Puccinia smyrii*, a species that is confined to a monocarpic, semelparous (biennial) member of the Apiaceae family, *Smyrnium olusatrum* (horse parsley). As with *P. hysterium, P. smyrnii*

lacks the urediospore phase and infection occurs during both the aeciospore and teliospore phases. This system appears to show local adaptation by the rust and susceptibility of hosts in natural population indicating that regulation is local as opposed to immigration of rust propagules from elsewhere. *P. graminella* is an autoecious, demicyclic rust (Anikster and Wahl, 1979) which infects *Nassella* species in South America and is currently being tested as a potential biological control agent for introduced tussock grasses in Australia (Briese *et al.*, 2000). Weber *et al*. (1998) report that *P. distincta* is an autoecious, demicyclic rust fungus recorded in Britain on daisy (*Bellis perennis*) and was believed to be the cause of the daisy rust epidemic observed during the mid-1990's. *P. distincta* is closely related to *P. lagenophorae* (Weber *et al.*, 2003) which is also an autoecious, demicyclic rust fungus that infects primarily *Senecio* spp (Frantzen, 2002), but has been shown occasionally to infect *B. perennis* (Weber *et al*., 1998). It has been postulated that the two species share a evolved from the macrocyclic, heteroecious species *Puccinia obscura* (Weber *et al.*, 2003).

1.4.1. *Puccinia lagenophorae – Senecio vulgaris*

An example of a system with similar characteristics to the *T. pratensis – P. hysterium* is that of *Senecio vulgaris* infected with *P. lagenophorae* but has been studied in more detail. *P. lagenophorae* is also an autoecious, demicyclic rust fungus lacking the urediospore phase (Van der Merwe *et al.*, 2007) which can infect many host species, but specifically those in the family Asteraceae. The rust is native to Australia and was first observed in the UK in the early 1960's infecting *Senecio vulgaris* (common groundsel) and several other species of *Senecio*, e.g. *S. squalidus* (Whipps and Cooke, 1978); species in the *Compositae, and Erechtites* (Wilson and Henderson, 1966) such as *Calendula officinalis* (marigold), *Pericallis hybrida* (common ragwort) and *Bellis perennis* (daisy); and has recently been recorded on *Emilia* spp (Henricot and Denton, 2005). Because of the similarities with *P. hysterium*, P. *hysterium* is assumed to have similar biological characteristics.

The life-cycle of *P. lagenophorae* (Figure 30) was described by Frantzen and Müller-Schärer (1999) which was adapted from the previous description by Wilson and Henderson (1966).

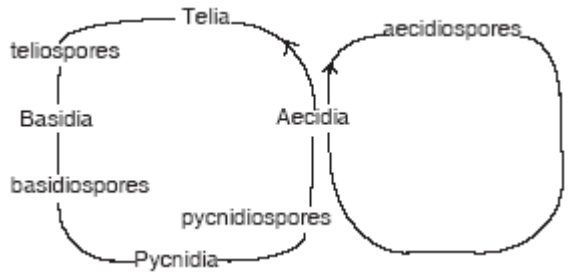


Figure 30. The life cycle of *Puccinia lagenophorae* (Frantzen and Müller-Schärer, 1999)

P. lagenophorae overwinters as mycelium within the host (although mortality is very high), and in the spring aeciospores are produced by aecidia and translocate within the host and are also dispersed to other plants within the local population (Frantzen and Müller-Schärer, 2006).

There has been extensive work on *P. lagenophorae*, mostly relating its use as a biological control agent of *S. vulgaris* (Paul and Ayres, 1986b, Müller-Schärer *et al.*, 2000). The effects of rust lineage (Müller-Schärer, 1996), temperature and epidemic development (Frantzen and Müller-Schärer, 2006), aeciospore production (Kolnaar and Van den Bosch, 2001), insect-mediated dispersal, and soil conditions for optimal rust infection (Tinney *et al.*, 1998) have been investigated to identify the characteristics of *P. lagenophorae*. Experiments investigating the role of *P. lagenophorae* required the collection of aeciospores and inoculations made by spraying hosts with an aeciospore suspension with hosts subjected to high humidity (Frantzen and Müller-Schärer, 1999). As *P. lagenophorae* is an obligate biotroph it has proved difficult to prepare sufficient stocks to use as a mycoherbicide, therefore, epidemics have been artificially generated by introducing infected individuals of *S. vulgaris* into crop systems infested with uninfected *S. vulgaris*, in the spring (Grace and Müller-Schärer 2003).

By understanding the *P. lagenophorae* – *S. vulgaris* system, it may provide insight about the possibilities of nutrient regime affecting performance of rusts such as *P. hysterium*. For example *S. vulgaris* grown in varying nutrient regimes showed that successful infection of *P. lagenophorae* was dependent on the soil phosphate levels (Paul and Ayres, 1988a, Paul and Ayres, 1988b).

1.4.2. Plant pathogens as biological control agents

Many plant pathogens have been investigated as biological herbicides to regulate and eradicate weeds (Charudattan, 2001), however use is less widespread than conventional herbicides and accounts for only a small percentage of total market sales. There are at least 15 commercially produced mycoherbicides marketed since 1980 (Charudattan, 2001). One such is the oomycete, *Phytophthora palmivora* (commercially known as DeVine), was used to regulate *Morrenia odorata* in citrus orchards by rotting the parasitic vine roots. A further commercial fungal herbicide, *Colletotrichum gloeosporioides* (marketed as Collego), regulates *Aeschynomene virginica* in soya and rice fields (Hajek, 2004). One of the most prolific biocontrol agent is *Fusarium oxysporum* f. spp causing wilt in several species of weeds (Hajek, 2004), and also used as a biological weapon against the narcotic trade by being applied to *Coca* (Sands *et al.*, 1997).

The first rust biological control agents were *P. chondrillina* and *P. jaceae* used to control rush skeletonweed (*Chondrilla juncea*) in Australia and California (Supkoff *et al.*, 1988). A highly pathogenic strain was introduced from collections made in Italy and France which caused nearly 90% mortality in rush skeletonweed (Hajek, 2004). There are several other rust fungi herbicides that are also used, such as *P. carduorum* on *Cirsium arvense* (nodding thistle), *Phragmidium violaceum* on blackberry and *Uromycladium tepperianum* on *Acacia saligna* (Hajek, 2004). Another rust biocontrol agent is *P. scanaliculata*, which has been used as a systemic inoculative biocontrol agent against yellow nutsedge (*Cyperus esculentus*), where populations are pre-treated to allow natural development of the rust earlier in the year (Phatack *et al.*, 1983). Further the rust *Puccinia punctiformis,* shown to regulate *Cirsium arvense* has also been identified as a potential biological control agent (Wandeler and Bacher, 2006).

P. lagenophorae significantly alters the growth of groundsel (Paul and Ayres, 1986a) and is a candidate for use as a biological control agent for *S. vulgaris* in tomato and lettuce crops, and also clearing groundsel on disturbed land (Frantzen and Hatcher, 1997). *S. vulgaris* has the potential to become a serious weed pest in carrot (Grace and Müller-Schärer 2003) and research shows the possibilities of using *P. lagenophorae* as a mycoherbicide in carrot crops (Wyss and Müller-Schärer, 1999). However, there are regional strains of the rust and different host populations have differing tolerance to

infection, so integrated approaches are being considered by combining biological control with chemical control. *P. lagenophorae* is believed to be native to Australia and New Zealand (Littlefield *et al.*, 2005) but has been found in wild host populations occurring in Canada (Bruckart *et al.*, 2007), America (Littlefield *et al.*, 2005) Argentina (Berndt, 2002), and in Europe, both in Poland (Piatek, 2003) and the United Kingdom (Henricot and Denton, 2005). Although difficult to isolate, only a small amount of *P. lagenophorae* inoculum is required to alter the growth of *S. vulgaris* (Frantzen and Müller-Schärer, 1999). Once the rust establishes itself, it prevents crop plants from being outcompeted by groundsel (Müller-Schärer and Reiger, 1998). Furthermore, *P. lagenophorae* does not alter the reproduction or cause death of groundsel, only the rate of growth in infected individuals, therefore a further advantage of using *P. lagenophorae* is that ground cover is not reduced allowing other weeds to replace groundsel.

1.5. Modelling epidemics

There are several ways in which disease epidemics have been modelled in host systems, e.g. change in dispersal of a pathogen within and between host populations, disease progression with time or the manner in which host population dynamics are affected by disease presence (Madden *et al.*, 2008). This section will relate to the development of epidemic models which describe the change in healthy and diseased plant populations in time only, and not with spatial models such as the reaction-diffusion gradients demonstrated by Frantzen (2002) in the spread of *P.lagenophorae*.

Models in ecology and epidemiology provide an approximation of a biological system which incorporate general aspects of the system (Kranz, 1974). Defined simply in Madden *et al*. (2008): " a model is a simplification of reality". By producing models it is possible to predict future dynamics, understand aspects of the epidemics, compare dynamics in varying systems and describe aspects of the system (Daley and Gani, 2005). The simplest way to describe an epidemiological model is to subdivide the individuals within a population into categories, most commonly: susceptible (*S*), latent (*E*), infectious (*I*) and removed (*R*) (Madden *et al.*, 2008). A susceptible individual is an individual within the population that has the potential to be in contact with infectious individuals and become infected. Latent is defined as the state of individuals within a population that have been exposed to inoculum, without becoming infectious. Once an

individual produces infectious units, it is re-categorised as an infectious individual. From being infectious, individuals may enter the removed phase of the epidemic where infectious propagules are no longer produced and add no further contribution to the spread of pathogen within the population (Daley and Gani, 2005).

When considering the population dynamics of hosts in relation to pathogen presence most epidemic models take the form of sets of equations as developed by Anderson and May (1979) and May and Anderson (1979) based on the Kermack-McKendrick model (Kermack and McKendrick, 1927). The general form of the model allows for various host categories within an epidemic, these being Susceptible (*S*), Exposed (*E*), Infectious (*I*) and Removed/Resistant (*R*) corresponding to the categories described above. This model involves a set of differential equations indicating the rate of change in the numbers of individuals within the infection states and various formulations are possible including *SI*, *SIS*, *SIR* and *SEIR* according to the pathogen characteristics.

The simple *SIR* model in continuous time takes the general form:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

Where *I* is the number of infected individuals, *S* the number of susceptible individuals, *R* is the number of individuals removed from the pathogen epidemic, γ is the recovery rate and β is the infection rate. The epidemic threshold is defined in terms of R_0, the number of new infections created by a diseased individual in a healthy population (Jeger and Van Den Bosch, 1994) and is obtained when: $R_0 = \frac{\beta S}{\gamma} \geq 1$. If R_0<1 an epidemic will not develop.

SIR models have been used to investigate a variety of systems including human disease epidemics such as measles and HIV (Haubensak and Smyth, 2002), animal disease epidemics such as tick-borne viruses (Laurenson *et al.*, 2003) and a variety of studies in

plant populations involving fungal (Gilligan *et al.*, 1997) and viral diseases (Chan and Jeger, 1994).

By applying Euler's method it is possible to obtain a discrete form of the Kermack-McKendrick model (Switkes, 2003) which can be used to describe an epidemiological model in discrete time. This takes the general form:

$$S_t = S_{t-1} - \beta S_{t-1} I_{t-1}$$

$$I_t = I_{t-1} + \beta S_{t-1} I_{t-1} - \gamma I_{t-1}$$

$$R_t = R_{t-1} + \gamma I_{t-1}$$

Expanded forms of the discrete *SIR* model have been used in a variety of studies such as the descriptions of rabies in rabbit populations (Allen *et al.*, 2002) and gene frequency and pathogen spread in plant populations (Kesinger *et al.*, 2001). By modelling in a discrete time format it is possible to replicate natural systems where there are non-continuous generations.

In modelling pathogen dynamics in this study, a discrete time *SEIR* model is first developed and then reduced to an *SIR* model. This resulting model mirrors in a natural way a demicyclic, autoecious rust and the systemic infection of a biennial plant.

1.6. Thesis aims, objectives and outline

The aim of this thesis is to assess the the hypothesis postulated by Silvertown *et al* (2006) that an autoecious, demicyclic pathogen can regulate the dynamics of a biennial host species based on the longterm recordings of *T. pratensis* and recent observations of *P. hysterium* in the PGE. This thesis assesses the biology and conditions associated with the outbreak population dynamics of a plant species with the characteristics of *T. pratensis*, in particular its biennial habit. As there is no diffinitive information relating to the life-cycle of *P. hysterium,* there is the assumption that it shares a similar life-cycle to the similar pathogen, *P. lagenophorae*. In order to assess the question of host dynamics, it is hypothesised that the host population is regulated by a systemic, obligate, castrating pathogen, which within the original observations is the rust *P.*

hysterium. The development of an appropriate suite of models to describe such a system forms the major part of this thesis. Furthermore, analysis is conducted of data obtained from surveys of the PGE between 1995 – 2008 relating to the population dynamics of *T. pratensis* under the varying pH treatments and fertiliser applications.

Firstly a model is developed to represent the interaction between a biennial host and a pathogen in a closed system without the recruitment to the population by immigration of seeds or losses through emigration of dispersing seeds, which has been shown to stabilise population equilibria in simple population models (e.g. Stone and Hart, 1999). This model is investigated to clarify conditions required to allow: host and pathogen to coexist with stable including cycling dynamics; host and pathogen to go extinct; or host population to grow unchecked by disease. This will be achieved by varying a range of life-history parameters relating to fecundity of the host and pathogenicity. Within this thesis, pathogenicity will be understood to represent the severity of symptoms caused by a pathogen similar to the use of virulence as used in zoological and medical terminology but not in plant pathology (Antonovics, 2005) as opposed to the abilty of a pathogen to cause disease. Virulance is not used to avoid confusion with gene-for-gene interaction as applied to host succeptability/resistance and pathogen virulence/avirulence. Furthermore, this model will enable an estimation of potential long-term population dynamics of a host biennial system when regulated by a pathogen.

The model is then expanded to take into account the assumption that not only does infection lead to castration, but there is an additional cost to the individual. By including additional pathogen-induced mortality in the model it is possible to give an accurate portrait of the population dynamics of *T. pratensis* in the presence of pathogen. In addition, the pathogen-induced mortality term is investigated to assess whether there are trade-offs between this term and other pathogen parameters. Simulations are used to examine outcomes arising from varying host-pathogen traits so altering host population dynamics. A range of assumptions concerning the pathogen-induced mortality term are made which enable an investigation of the role the abundance of infectious individuals in the population on pathogen-induced mortality. This variable pathogen-induced mortality enables a prediction of the optimal conditions for host and pathogen to exist. In order to counter the effects of increasing disease performance, an additional variation to the model is developed by means of adding a seedbank to the system, the rationale for this is that a seedbank enables the host to escape the effects and outlive highly

pathogenic strains. Although *T. pratensis* has a short-lived seedbank, other biennial species may have larger seedbanks, altering its population dynamics and epidemiology, therefore this aspect of the modelling process is required to address the general nature of the epidemiological model.

This General Introduction has provided a foreword to the subject of host-pathogen interactions, including some background to key concepts of plant ecology, pathology and epidemiology; and also more detailed introductions to the host-pathogen system in the Park Grass Experiment, biology of the model host and pathogen, and the models that can be used to investigate population dynamics. In Chapter 2 a basic generic model to describe the epidemiology of host-pathogen populations is developed. In Chapter 3, the basic model is extended by incorporating constant pathogen-induced mortality in the population. The model is further adapted in Chapter 4, to investigate the role a variable pathogen-induced mortality. In Chapter 5 a seedbank stage is introduced into the model. Chapter 6 provides an analysis of data relating to *T. pratensis* in the PGE and also experiments assessing infection of *P. hysterium* on seed production and viability. An overall discussion relating the theoretical models to the long-term data is given in Chapter 6.

2. A generic SIR-type model to describe the population dynamics of a biennial host plant infected by a systemic pathogen

2.1. Summary

A discrete-time *SIR* type model is developed to simulate the population dynamics of a biennial host plant infected by a systemic pathogen, as occurs with *Tragopogon pratensis* infected by the autoecious, demicyclic rust *Puccinia hysterium*. Investigations are conducted to ascertain the parameter conditions required to enable an epidemic to occur and also identify stable steady-state descriptions for the host in the absence of pathogen. Examples are provided of simulations made using default parameter values obtained from long-term field observations and published data referring to the *T. pratensis – P. hysterium* and related systems. The model simulations describe a range of population dynamics that include increases to a stable population, population cycles and extinctions. Steady-state expressions are derived which show that the steady-state of healthy second-year plants are dependent on mortality and density-dependency parameters. An invasion criterion is obtained and found to be independent of the frequency of infectious individuals within the population, whilst being reliant on the pathogenicity parameters of the pathogen.

2.2. Introduction

To produce a descriptive model both characteristics of the host – pathogen must be included. In this case the biennial nature of the plant and the systemic infection by the pathogen such as seen in *T. pratensis* and *P. hysterium*.

Data collected from the park grass experiment (PGE) at Rothamsted Research, UK, has been used to observe a wide range of population and community changes since its inception in 1856 (Silvertown *et al.*, 2006). Dodds *et al*. (1995) describe plant population dynamics in the PGE that increase (e.g. *Trifolium pratense*), decrease (e.g. *Veronica chamaedrys*), fluctuate (e.g. *Conopodium majus*) or follow an outbreak pattern (e.g. *Tragopogon pratensis*). Many of these dynamics have been considered as consequences associated with the long-term fertiliser regimes altering the soil nutrient content and pH. However, it is speculated that the cause of the outbreak dynamics

observed within the population of *T. pratensis* was due to an autoecious, demicyclic rust, *Puccinia hysterium* (Silvertown *et al*., 2006). Observations conducted by G. Edwards (*unpublished*) have indicated that over an 11 year period there has been a two year delay between pathogen incidence and host population size. In natural populations, work on the regulation of *Senecio vulgaris* by the rust *P. lagenophorae* indicates that rust presence can significantly alter host fitness specifically by altering seed production (Paul and Ayres, 1986a, Paul and Ayres, 1986c). Similarly to *P. lagenophorae*, *P. hysterium* lacks a urediospore phase and is believed to be transmitted by wind as teliospores (Wilson and Henderson, 1966) which infect first-year plants, the rust becomes systemic as mycelia which in second-year plants produces aecia and aeciospores once the host has re-emerged.

By relating the host and pathogen dynamics in a model system, it is possible to identify the conditions required for pathogen invasion to occur, and discover what life-history parameters alter the host population dynamics and cause steady-state populations, cycles and rashes. This study provides a generic epidemiological model which could be applied to other biennial host – systemic, castrating pathogen systems which have characteristics similar to *T. pratensis – P. hysterium* system.

The focus of this study has been the development of epidemiological models describing the biennial nature of *T. pratensis* and the systemic characteristics of the rust which produce discrete generations. Here, the Kermack-McKendrick model (Kermack and McKendrick, 1927) is adapted from continuous time, differential equations to discrete time intervals using the Euler method. Compartmentalised models of disease state (i.e. Healthy, Latently infected, Infectious and Recovered) have been used in a variety of examples of plant – pathogen model systems; including fungal infection dynamics (Papastamati *et al.*, 2001) fungal pathogen spread between plant tissue (Gilligan *et al.*, 1997), viral pathogen dynamics (Chan and Jeger, 1994), and viral-vector transmission dynamics (Jeger *et al.*, 1998), but these have been based on continuous time. The model developed is one of the few examples of a system where discrete-time provides a more appropriate portrayal of the host – pathogen system.

This chapter aims to address the hypothesis that a systemic pathogen (such as *P. hysterium*) can regulate a biennial host (such as *T. pratensis*) population and can cause dynamics as observed in the PGE. A mathematical model is used to develop a

framework which will address this question theoretically. By applying a simplified yet biologically-consistent model to this system, it is possible to provide a description of pathogen-regulated dynamics in which criteria for invasion of the pathogen are developed and long-term outcomes in terms of plant population size is determined.

2.3. Methods

2.3.1. The model

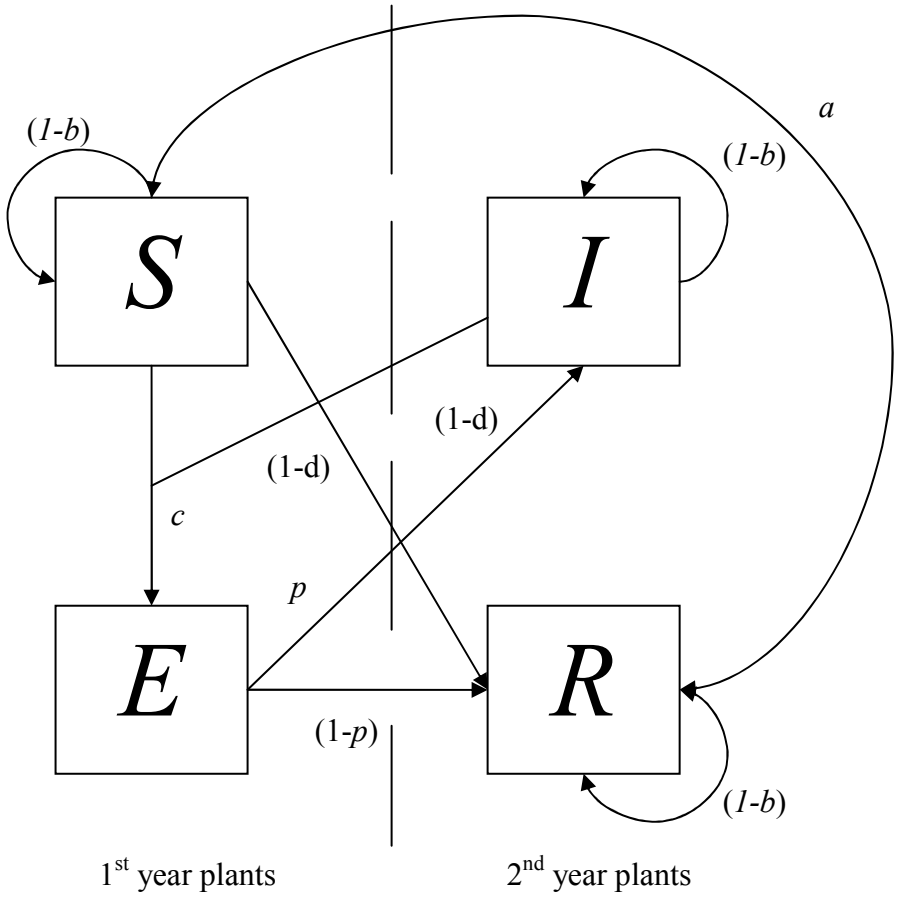
The model that is developed represents the dynamics of an infectious pathogen in a biennial host population with the following assumptions:

1) The system of is closed with no immigration or emigration.
2) Plants are biennial, completing their life-cycle in two seasons where they are vegetative, non-seeding first year individuals during the first season, which over winter below ground, re-emerge the following season and die after setting seed in the second year.
3) The individuals within the host population are classified relating to their infection status: first year plants which are healthy and susceptible to infection (S), or exposed and latently infected (E); second year plants are either infectious (I), or not infected and therefore removed (R) from the epidemic dynamics as they do not contribute to infection of first year plants. A discrete-time model is used to describe the dynamics of each of the categories within the host population.
4) There are basic mortality rates which are constant amongst all categories of individuals, which act both within season (b) and between seasons (d). This basic rate of mortality does not alter between first and second year individuals and is independent of disease status.
5) At the beginning of a growing season (denoted by α) only S, I and R individuals are present. E individuals at the end of the first year become I or R in the second year.
6) During the season S individuals become exposed to the infection with an infection probability c, therefore allowing S, E, I, and R individuals to be present in the population at the end of the season (denoted by ω).
7) The probability of infection of first year S individuals is assumed to be at random and therefore follow a Poisson distribution (Madden *et al.*, 2008).

8) At the end of the season, the infection becomes systemic in *E* individuals. However, not all *E* plants become infectious in the second year. The probability of an *E* individual re-emerging in the second year as an *I* individual is *p*.
9) *S* individuals that do not die between seasons or become infected, re-emerge as *R* individuals in the second year. *E* individuals that do not die between seasons, or in which the systemic infection dies out, re-emerge as *R* individuals in the second year.
10) *S* individuals in the first year are infected by infectious propagules produced by *I* individuals in their second year.
11) *R* individuals are not infected or, if they are, do not produce infectious propagules before they die in the second year.
12) Only *R* individuals set seed with a seedling emergence rate *a*. *I* individuals are castrated.
13) The seedling emergence rate is density-dependent, with the representation proposed by de Wit (1960) specifically for plant populations.
14) There is no seed bank. (Relaxed subsequentially in Chapter 5)

These assumptions lead to the following schematic representation of the system (Figure 31).

a)



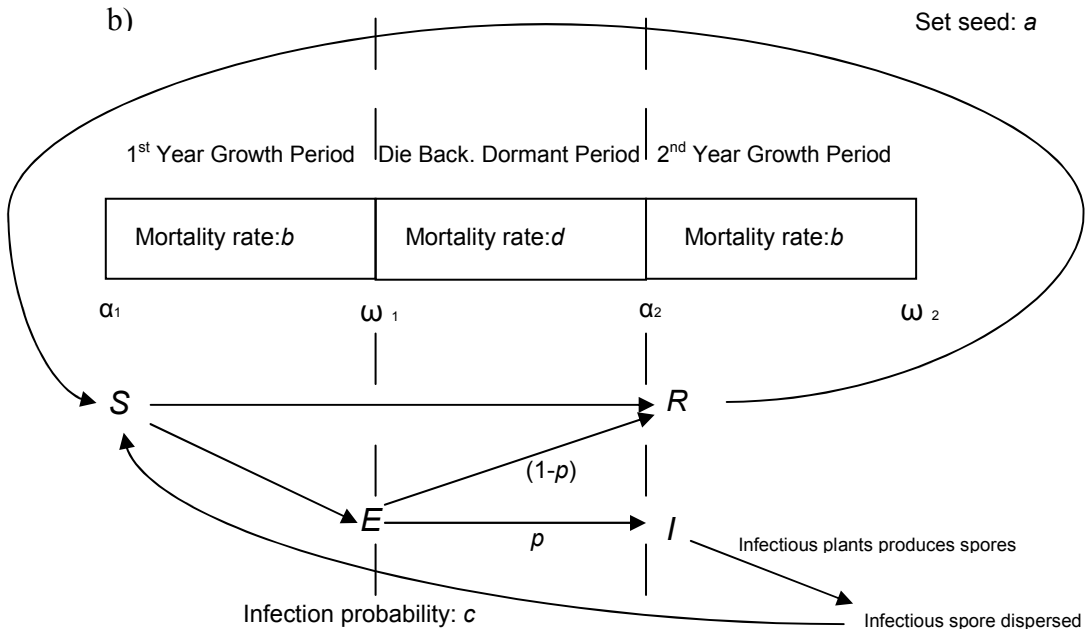


Figure 31. Schematics of the model: α: Beginning of season ω: end of season. In a) the model is presented as a compartmental model; in b) chronologically with the beginning (α) and the end (ω) in the growth periods indicated.

The model is described by the following sets of equations:

$$S_{\alpha,t} = \frac{aR_{\omega,t-1}}{1+\lambda R_{\omega,t-1}} \quad (1)$$

$$S_{\omega,t} = (1-b)S_{\alpha,t}e^{-cI_{\omega,t}} \quad (2)$$

$$E_{\alpha,t} = 0 \quad (3)$$

$$E_{\omega,t} = (1-b)S_{\alpha,t}[1-e^{-cI_{\omega,t}}] \quad (4)$$

$$I_{\alpha,t} = (1-d)pE_{\omega,t-1} \quad (5)$$

$$I_{\omega,t} = (1-b)I_{\alpha,t} \quad (6)$$

$$R_{\alpha,t} = (1-d)[(1-p)E_{\omega,t-1} + S_{\omega,t-1}] \quad (7)$$

$$R_{\omega,t} = (1-b)R_{\alpha,t} \quad (8)$$

Where the subscripts α and ω refer to the beginning of season and end of season respectively, and t is time.

Equations are then derived to describe each of the states of *S*, *I* and *R* at the beginning of the season by substituting terms to reduce the number of equations, rearranging in terms of α and scaling from $t \rightarrow t+1$. This makes it possible to exclude α and ω terminology, and also makes an equation for *E* obsolete as at the beginning of the season they have become either *I* (with probability *p*) or *R* (with probability of 1-*p*) second-year plants.

The equations are:

$$S_{t+1} = \frac{a(1-b)R_t}{1+(1-b)\lambda R_t} \quad \textbf{(9)}$$

$$I_{t+1} = (1-d)(1-b)pS_t\left\{1-e^{-c(1-b)I_t}\right\} \quad \textbf{(10)}$$

$$R_{t+1} = (1-b)(1-d)S_t\left\{1-p[1-e^{-c(1-b)I_t}]\right\} \quad \textbf{(11)}$$

S, *I* and *R* are the categories of susceptible, infectious and non-infectious plants respectively; *a* is the seedling recruitment rate, *b* is the within season mortality, *d* is the between season mortality, *c* is the infection rate, *p* is the probability of a latently infected individual re-emerging in the second year as an infectious individual, λ is the per unit area density of host individuals and *t* is time.

2.3.2. Methods of analysis

Numerical and analytical techniques were implemented to determine the quantitative and qualitative properties of the model (equations 9 – 11). Steady-state equilibria were produced for each population category by setting increments in equations 9, 10 and 11 equal to zero (i.e. $S_{t+1} - S_t = 0$), and obtaining an implicit solution for *I*. Numerical simulations of the model were investigated using MAPLE 10 software (Waterloo Maple Inc. 2005) for varying parameter values. In addition, a epidemic development criterion was derived. Recurrence-relationships were developed describing relationships between population sizes at different discrete time steps.

Numerical data for approximating parameter values were obtained from field observations and greenhouse trials as described in Chapter 6, personal communications and published material relevant for *T. pratensis, P. hysterium* and similar host – pathogen systems

2.4. Results

2.4.1. Simulated Populations

Simulation outcomes were produced showing the trajectory of each of the categories within the host population, starting from varying initial values. Figure 32 represents host dynamics using default parameter values (Table 4). Under the default parameter values the numbers of *S*, *I* and *R* individuals increase to stable steady-state values, and this outcome will be used as a standard of comparison for subsequent simulations.

By altering the initial parameter values within the parameter range, it is possible to create dynamics where host populations cycle, increase in a monotonic fashion towards a stable population, or tend to extinction. Specifically by setting values outlined in Table5 for low, medium and high pathogenicity (*c*, *p*) and also for low, medium and high host performance (*a, b*, *d*) it is possible to obtain a range of simulation outcomes (Figure 33). The central simulation outcome in Figure 33, reflecting medium pathogenicity and host performance, is identical to outcomes for default values shown in Figure 32.

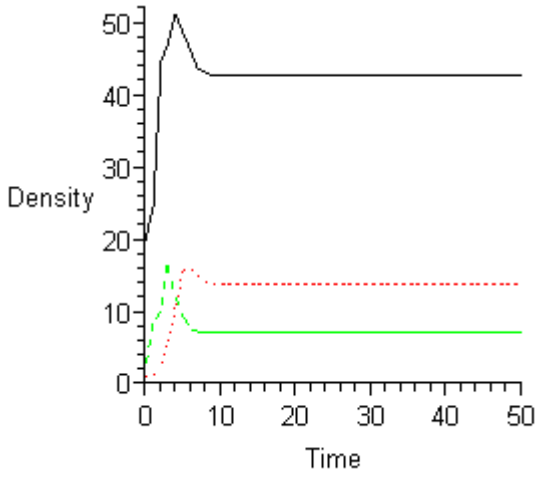


Figure 32. The model simulation outcomes using the default parameter values of medium pathogenicity and medium host performance. (— *S*, — *I*, — *R*). This model depicts stable steady-state population equilibrium.

Table 4. Parameter's and their value ranges used to asses the model system.

Parameter	Explanation	Default value	Source	Range Investigated
S_0	Initial number of 1st year susceptible individuals.	20		
E_0	Initial number of exposed 1st year individuals.	0		
I_0	Initial number of Infectious 2nd year individuals.	1		
R_0	Initial number of non-infectious 2nd year individuals.	2		
a	Seedling recruitment per non-infectious 2nd year individuals	30	*T. pratensis* seed set mean of 26.7 (G. Edwards. *unpublished*)	20 – 60
b	Within-season mortality rate	0.3	0.5 reported for *T. pratensis* (Mahesh *et al.*, 1996)	0.1 – 0.8
d	Between-season mortality rate	0.3	0.88 reported for *T. pratensis* (Mahesh *et al.*, 1996)	0.1 – 0.6
c	Infection rate probability	0.3	Variable depending on temperature for *P. lagenophorae* (Kolnaar and Van den Bosch, 2001)	0.1 – 0.8
p	Probability of exposed individuals becoming infectious	0.7	Greenhouse trials indicate 0.15	0.3 – 1.0
λ	Density-dependent parameter	0.5	Long term PGE data G. Edwards, *unpublished*	0.2 – 1.0

Table5. Parameter ranges for low, medium and high host performance and pathogenicity.

	Low	Medium	High
Pathogen			
c	0.1	0.3	0.8
p	0.3	0.7	1.0
Host			
a	20	30	60
b	0.4	0.3	0.1
d	0.5	0.3	0.1
λ	1.0	0.5	0.2

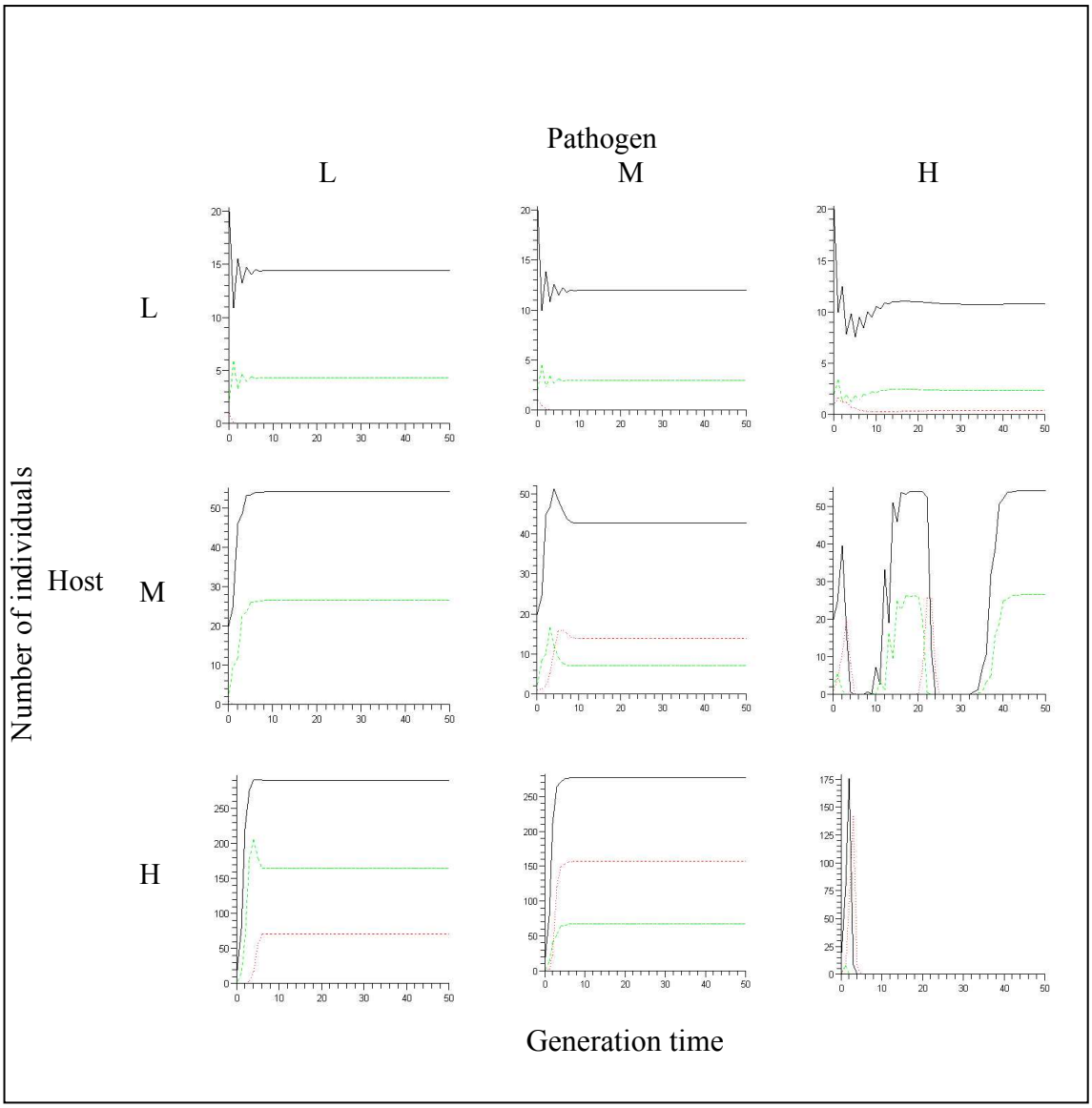


Figure 33. Simulation outcomes produced under conditions of high, medium and low host performance and pathogenicity. Each simulation represents — *S*, — *I*, — *R* dynamics.

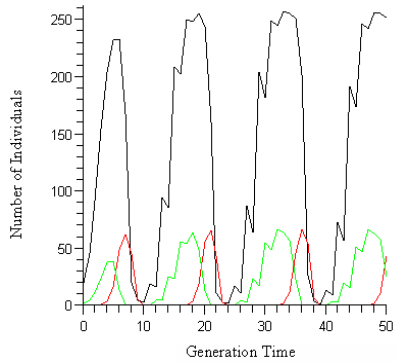


Figure 34. An example simulation of cycling population dynamics, attained by altering the parameter values to include *c*=0.1, *p*=1.0 and *d*=0.7, produces host-population cycles for — *S*, — *I*, — *R* individuals.

Figure 33 shows that the majority of simulations produce outcomes where host populations increase monotonically to a steady-state; however at high pathogenicity the host population becomes extinct when host performance is high; whereas if the host has a medium performance, it is able to buffer the influence of the high pathogenicity through cycles until the host population is at a too low level to sustain pathogen within the system, causing the pathogen to become extinct in the population allowing for the host to tend to a stable population. It is possible to produce continual population cycles with a time period of 25 years (Figure 34) by incorporating high between-season mortality with low values of *c* but very high *p* values. This is not regulation of the host population by the pathogen, but is due to the increased influence of *c* and *p* on a host population with high natural mortality, so enabling the epidemic development criterion to be satisfied at low host frequency. For low pathogenicity it is still possible for the epidemic development criterion to be obtained where there are high values of the host performance parameters.

2.4.2. Steady-state expressions

In the absence of pathogen the host population dynamics are described by the pair of equations:

$$S_{t+1} = \frac{a(1-b)R_t}{1+(1-b)\lambda R_t} \quad \textbf{(12)}$$

$$R_{t+1} = (1-b)(1-d)S_t \quad \textbf{(13)}$$

The steady-state values are:

$$\hat{R} = \frac{a(1-b)^2(1-d)-1}{\lambda(1-b)} \quad \textbf{(14)}$$

$$\hat{S} = \frac{a(1-b)^2(1-d)-1}{(1-b)^2(1-d)\lambda} \quad \textbf{(15)}$$

Provided that $a(1-b)^2(1-d) > 1$

Without pathogen in the system equations 14 and 15 indicate that the host population will lead to a steady-state population size regulated by host density, λ, and host mortality, b and d.

Where pathogen is present it is possible to produce a steady-state expression for I^* however this only gives an implicit value for I^*, as I^* occurs on both sides of the equation:

$$I^* = \frac{p\left[(1-e^{-c(1-b)I^*}\right]}{(1-b)\lambda}\left\{a(1-b)^2(1-d) - \frac{1}{1-p\left[(1-e^{-c(1-b)I^*}\right]}\right\} \quad \textbf{(16)}$$

The solution for I^* is obtained graphically by plotting both sides of equation 16 against I^* and determining the point of intersection.

2.4.3. Epidemic development criterion

For pathogen to persist within the system the basic reproductive number (R_0) must be greater than one.

Within this system the criterion for pathogen to invade a pathogen free system is defined as:

$$I_{t+1} = I_t(1-b)^2(1-d)pc\hat{S}$$
$$\Rightarrow \frac{pc[a(1-b)^2(1-d)-1]}{\lambda} > 1 \quad \textbf{(17)}$$

where for small I, $1-e^{-c(1-b)I_t}$ was approximated by $c(1-b)I_t$ or equivalently:

$$\hat{S}(1-b)^2(1-d)pc > 1 \quad \textbf{(18)}$$

This indicates that the conditions for pathogen to invade is reliant on the steady-state size of the first-year population $(\hat{S})$ the product of the pathogen parameters c and p, and cumulative survival $(1\text{-}b)^2(1\text{-}d)$ across two seasons.

An epidemic occurs with the default parameter values, by altering the values of c, p and S are given in Figure 35.The infection rate (c) and probability of becoming infectious (p) have a strong influence on the epidemic threshold condition. Furthermore, under

default mortality rates and altering of pathogenicity parameters and also the size of the first year population there is an epidemic threshold within the estimated parameter ranges. Therefore pathogen epidemics are reliant on a sufficiently high contact rate and probability of becoming infectious once exposed, in appropriately sized host populations.

By using default parameter values (Table 4) it is possible to produce both epidemic and non-epidemic outcomes with a range of varying parameter values. The product of *pc* in the epidemic development criterion indicates they are of equal importance in determining the value. It is $\hat{S}$ that is the limiting factor in determining epidemic condition, as there is an asymptotic relationship for *c* and *p* values for values greater than zero and less than one. Figure 36 indicates that the product *pc* is required to be relatively low (so that *S* does not reduce to a very low value) but greater than 0.05 to induce an epidemic within a susceptible population. The default *pc* product of 0.21 requires a susceptible population of around 14 susceptible individuals, which is below the parameter range for an epidemic to occur, indicating that with S_0 = 20, epidemic conditions are created from the outset.

By using the corresponding $\hat{S}$ produced by altering one parameter value with the others remaining constant, it is possible to investigate using equation 18 to define the parameter value where R_0>1 is obtained. The region where parameter values lead to the invasion criterion falling below the threshold results in no epidemic (Figure 38). Altering λ obeys the conditions for an epidemic to occur, as does *a* where as when *b*=*d* > 0.516 (3sf) no epidemic occurs. The inference from this is that background mortality of individuals both between and within season is the limiting factor in defining pathogen-induced regulation of this host system.

Altering one parameter value in relation to constant default parameters affects the epidemic outcome. In the case where λ increases between 0.2 and 1.0 as this is the investigated range of values of λ but the other parameters remain at their default values there is a six fold reduction in the value of $\hat{S}$ (Figure 38a). Figure 38b shows an increase in *a* from 20 to 60 which relates the investigated range of parameter *a*. Whilst Figure 38c indicates the effect on $\hat{S}$ of increasing mortality (*b*=*d*), similarly the range of values between 0 to 0.8 correspond to those used in producing simulations.

Figure 37 shows a summary of the steady-state value of infectious second year plants with increasing values of both *p* and *c* calculated by observing implicit values of *I** (equation 16). For low *c* values, *p* is required to be relatively high for a steady-state value of infectious individuals (*I**); similarly low values of *p* require higher *c* values. Increased *c* values give increases in the stable-steady values of infectious second year intervals, whereas increasing *p* (apart from at low *c* values) does not lead to a marked increase in *I**. Figure 39 provides an example of solving values of *I** for increasing values of *c* with the other parameters remaining at default values.

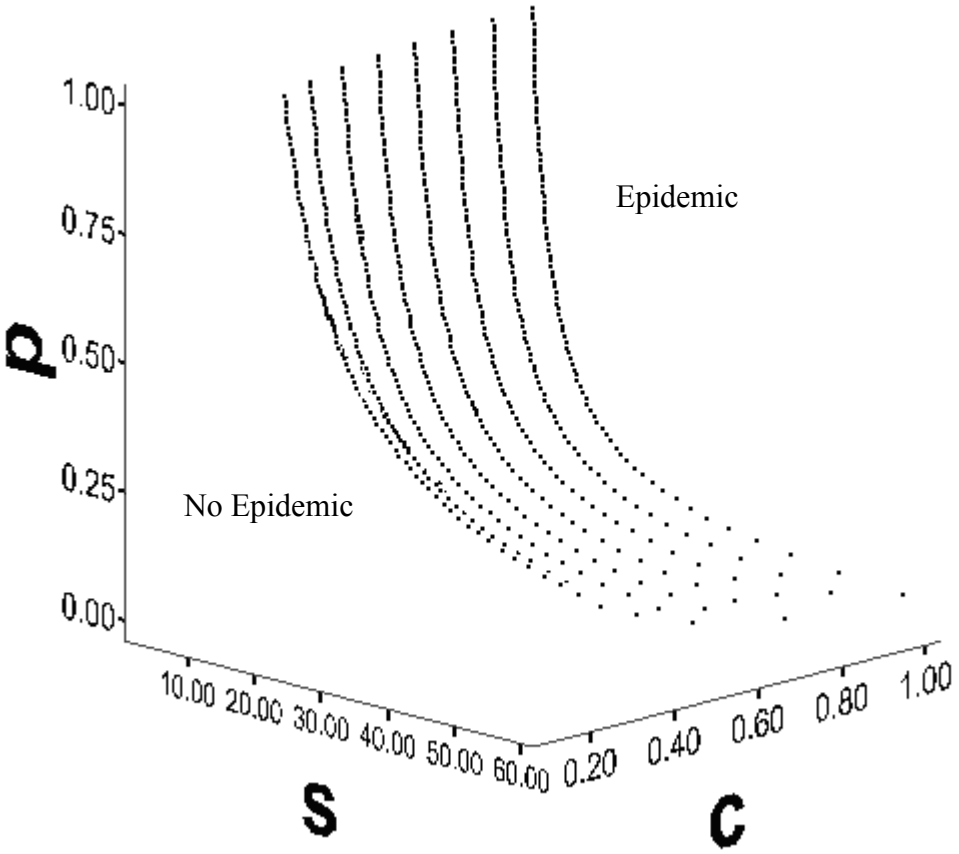


Figure 35. A three dimensional representation of epidemic and non-epidemic regions when using initial default parameter values and altering *c*, *p* and *S*.

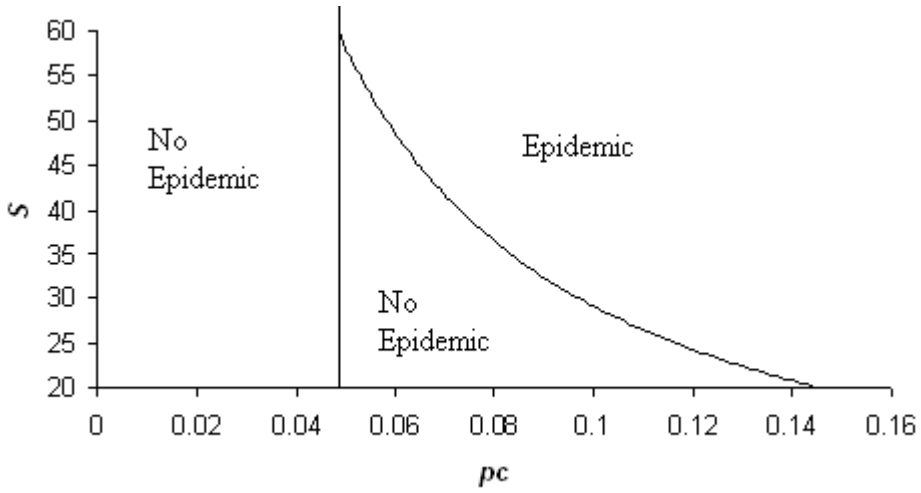


Figure 36. Product of *pc* required to obtain invasion criterion greater than one for values of *S* within the parameter range under the default parameters for *b* and *d*.

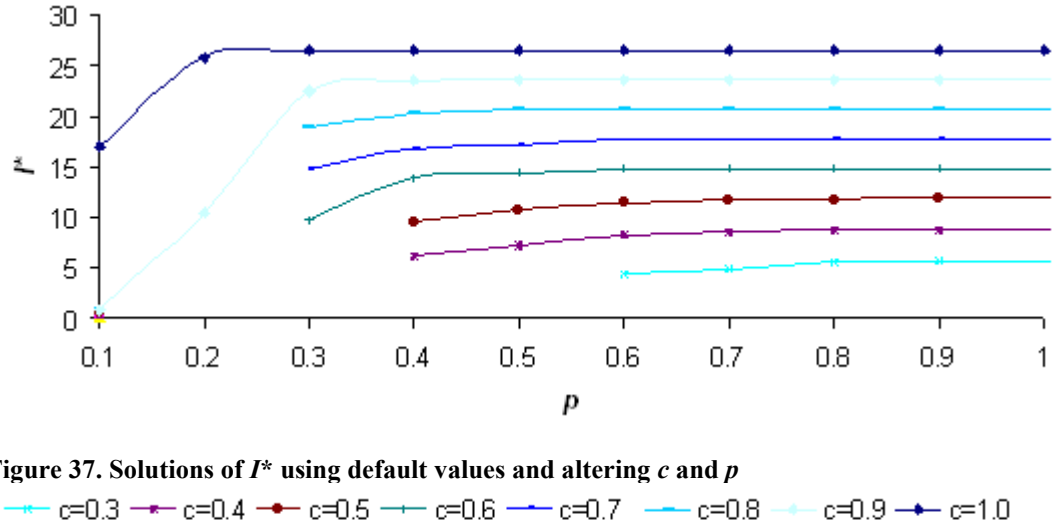


Figure 37. Solutions of *I using default values and altering *c* and *p***

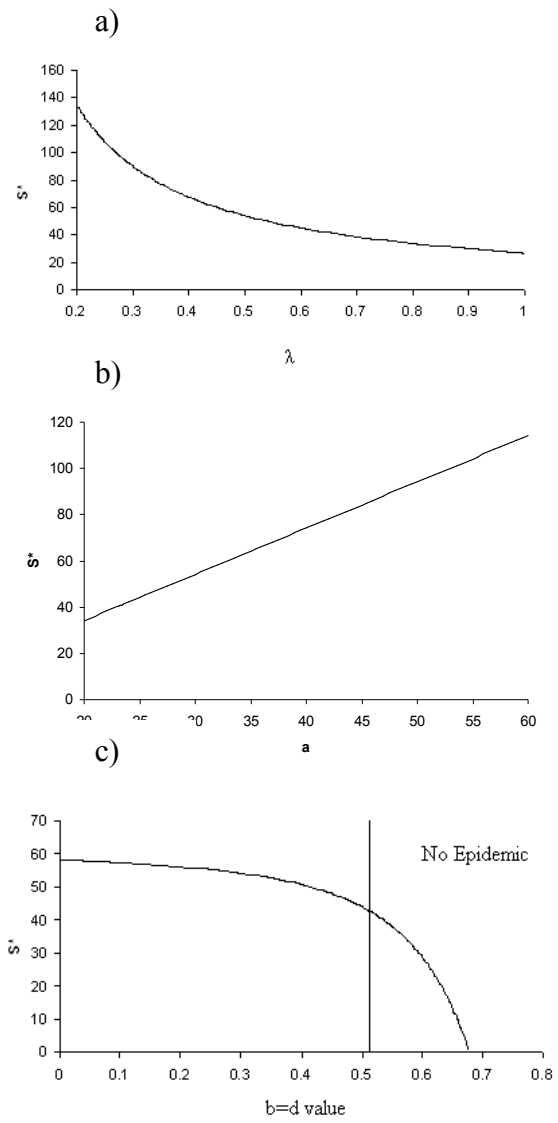


Figure 38. The effect of varying either a) λ, b) a or c) b and d where $b=d$, within the parameter range whilst the other parameters remain at default values.

2.4.4. Recurrence relationships

By rearranging equation 12 for the system without pathogen, a recurrence relationship is obtained that represents the relationship between individuals in sequential years:

$$\frac{1}{S_{t+1}} = \frac{1+\lambda(1-b)R_t}{a(1-b)R_t} \qquad = \frac{\lambda}{a(1-b)} + \frac{1}{a(1-b)R_t} \tag{19}$$

Therefore there is a linear relationship between S_{t+1}^{-1} against R_t^{-1} with a gradient of $\frac{1}{a(1-b)}$ and the intercept $\frac{\lambda}{a(1-b)}$. Rearranging equation 13 to give $R_{t+1} = (1-b)(1-d)S_t$ and substituting in equation 19 gives:

$$\frac{1}{R_{t+2}} = \frac{1}{a(1-b)^2(1-d)R_t} + \frac{\lambda}{a(1-b)^2(1-d)}.$$

In the presence of pathogen from equations 10 and 11.

$$I_{t+1} + R_{t+1} = S_t(1-b)(1-d)$$

Giving:

$$\frac{1}{S_{t+1}} = \frac{(1-b)(1-d)}{I_{t+2} + R_{t+2}} \quad \textbf{(20)}$$

By inserting 20 into 19, a second-order (two time steps) recurrence equation is obtained:

$$\frac{1}{I_{t+2} + R_{t+2}} = \frac{1}{a(1-b)^2(1-d)R_t} + \frac{\lambda}{a(1-b)^2(1-d)} \quad \textbf{(21)}$$

As $a(1\text{-}b)^2(1\text{-}d)$ must be greater than 1 for the host to establish, then the gradient of equation 21 must be less than 1.

The recurrence relationship is independent of both *c* and *p*, therefore there is no pathogen regulation of the number of second-year individuals. Also as the recurrence relationship is of the exactly the same form in the absence of pathogen (I=0) then it can be concluded that pathogen does not regulate host population dynamics in the model developed.

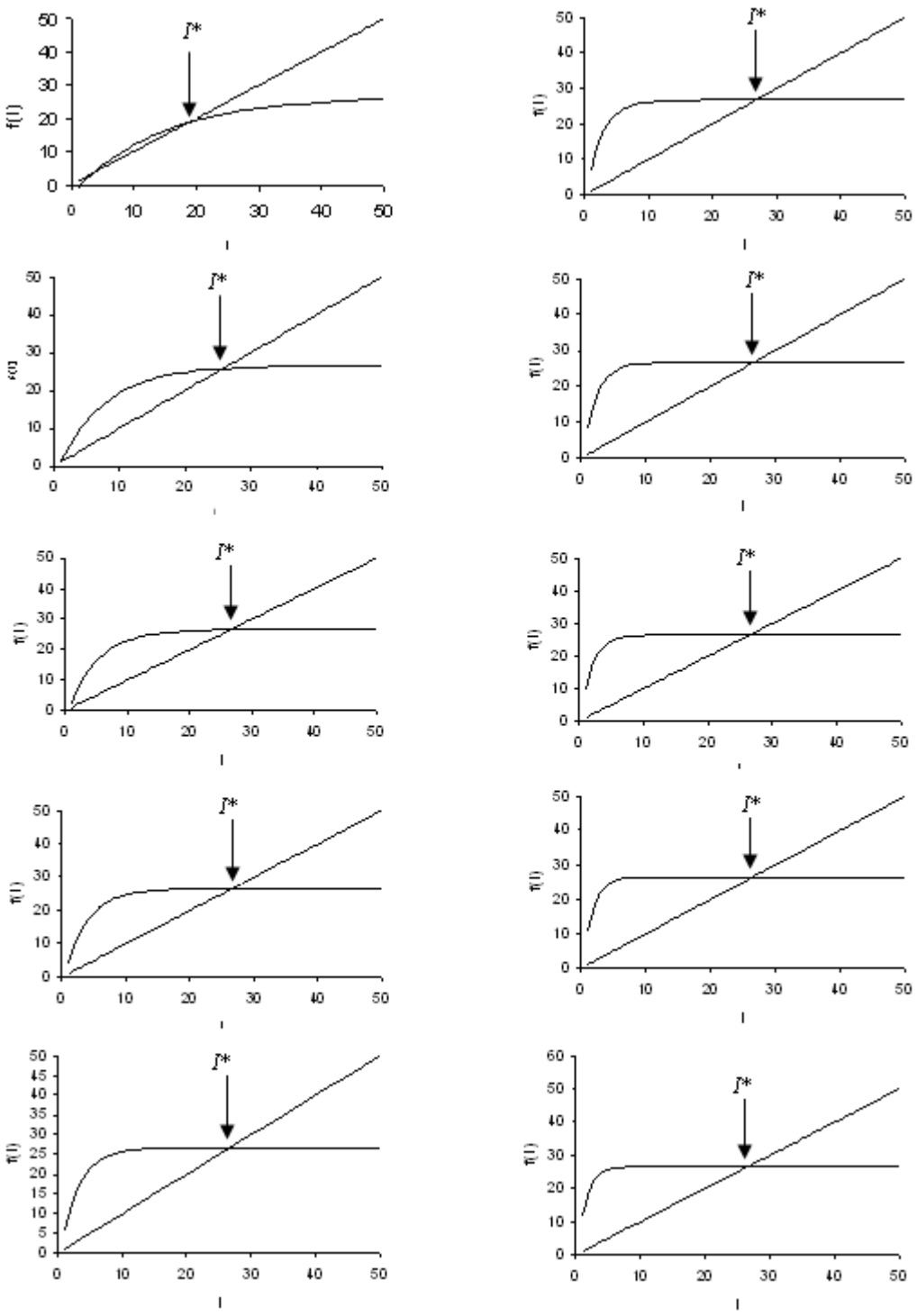


Figure 39. An example of solving stable-state values for *I for default parameters with increasing values of *c* from 0.1-1.0. *I** is defined as the interception between *I* and the f(*I*).**

2.5. Discussion

The model developed relates to a biennial host plant population exposed to a systemic pathogen. The model is a novel, compartmentalised *SIR*- type model in discrete time which reflects the two year-cycle of the host and the systemic nature of the pathogen. The model allows for a simplified description of a generic system involving a monocarpic host population infected by a systemic pathogen without a repeating spore form. The model assumes there is no additional mortality due to being infected. Wennström (1999) claims that in several annual host-systemic pathogen systems, there is very little additional mortality associated with infection as this would not be to the pathogen's advantage; this may also be the case for a biennial or perennials. However vascular annual wilt pathogens cause annual hosts to be killed by disease, the pathogen is able to produce either long-lived spores in the dead plants or produce spores that can be aerially dispersed.

The model highlights that under default parameter values the host population will eventually tend to stabilise at a steady-state population and not undergo cyclical or outbreak dynamics. With high pathogenicity there is some cycling in medium performance host populations, however the pathogen becomes extinct and the healthy host tends to a stable population. High pathogenicity and high host performance causes the host population to become extinct. Long-term data relating to the outbreak dynamics of *T. pratensis* in the PGE (Chapter 1, Figure 1) suggests outbreaks or longer term cycles may occur. This suggests that natural populations of *T. pratensis* may be exposed to strains of *P. hysterium* with high pathogenicity but which have low probability of causing hosts to become infectious in their second year. The natural rate of mortality is reported as 0.5 within season and 0.88 between seasons (Mahesh *et al.*, 1996) and is within the region that will allow for the population cycling to occur. The host population in the PGE is recorded to increase and decrease over longer time periods than produced in the model, but similarly to the detailed PGE observations of G. Edwards over 11 years (*unpublished*, Chapter 6); there is the two year delay between the incidence of susceptible plants and the resulting incidence of infectious individuals within the longer cycles.

The pathogen is eliminated from the system where the epidemic development criterion is not met also the host criterion has also to be met ($a(1-b)^2(1-d)>1$), and can be seen in the simulations where disease individuals are removed from the system allowing healthy individuals to increase towards the steady-state values. As the criterion is based on constant pathogen and host parameters along with the susceptible host density it can be concluded that the threshold is related to the size of the susceptible host population.

This model predicts a two year lag between counts of adult individuals, as would be expected by the biennial nature of the system. However this relationship is independent of the pathogen parameters p and c, indicating that there is no regulation of population size by the pathogen in this model. This form of the recurrence relationship is exactly the same in the presence or absence (I=0). A further factor requiring investigation is the possibility that pathogen causes additional mortality of the host (Chapters 3 and 4) and alters the host population dynamics. Additionally the influence of a seedbank on host population dynamics needs investigating (Chapter 5). It may be possible for a seedbank to replenish the host population without the constraints of the pathogen. Finally the recurrence relationship established in this chapter will be tested against the field data for *T. pratensis – P. hysterium* in the Park Grass Experiment (Chapter 6).

3. The impact of constant pathogen-induced mortality on the population dynamics of a biennial host plant infected by a systemic pathogen

3.1. Summary

An *SIR*-type model developed to describe a biennial host plant infected by a castrating pathogen was extended to include pathogen-induced mortality. A constant pathogen-induced mortality term either acts between seasons, within season or both between and within seasons. Model simulations show outcomes in which the host population cycles, crashes or tends to a stable population. The epidemic development criterion derived for each form of the model gives the same expression where pathogen-induced mortality occurs either between or within season. Steady-state expressions are also derived for the three forms of the model where these exist. All these expressions include the pathogen-induced mortality term indicating this is an important contribution to host plant regulation within the system. A second-order recurrence relationship is derived which relates the numbers of healthy second year plants with the total numbers of second year individuals (healthy and diseased) two years later. It is clear that pathogen-induced mortality is paramount and where it is applied between and both between and within seasons, it is a regulatory factor of the host population dynamics.

3.2. Introduction

Long-term data at the Park Grass experiment at Rothamsted Research has demonstrated outbreak population dynamics of the biennial host plant, *Tragopogon pratensis* (Dodd *et al.*, 1995) and it is hypothesised that it is regulated by the systemic autoecious, demicyclic rust fungus, *Puccinia hysterium* (Silvertown *et al.*, 2006), which castrates infected individuals so altering seedling recruitment the following season. In addition to altering seed production, there is the possibility that infection by *P. hysterium* incurs a cost to the plant in the form of additional mortality, similar to that observed in *Filipendula ulmaria* infected by the rust *Triphragmium ulmariae* (Ericson *et al.*, 2002). Other examples include the impact of *Puccinia lagenophorae* on the survival of its host *Senecio vulgaris* (Frantzen, 2002) by altering the competitive ability of infected plants with non-infected plants (Jarosz, 1992), reducing plant tissue growth, making infected plants more susceptible to changes in environmental conditions (Paul and Ayres, 1987), redistributing nutrients and water towards the pathogen (Paul and Ayres, 1984), and

simply interfering with photosynthesis such as *Puccinia triticina* on wheat (Corinne *et al.*, 2004). Such pathogen-induced mortality is sometimes termed "virulence" in the animal/human epidemiology literature but will be avoided in this thesis as virulence has a different usage in plant pathology (Antonovics, 2005).

Disease-induced mortality of individual plants will have an effect on the population dynamics of the host, not simply by altering the number of seedlings through castration of infectious individuals, but also impacting on the numbers of exposed individuals which become infectious where the additional mortality occurs between-season. Within-season, pathogen-induced mortality will also reduce the number of infectious individuals that can infect susceptible plants.

In Chapter 2 a model was presented in which there was no pathogen-induced mortality. In this chapter the population dynamics of a biennial host plant in the presence of a castrating pathogen which also impacts on individual plant survival within and between seasons is modelled. As in Chapter 2 the criterion for epidemic development is derived along with steady-state expressions. The model is also used to provide simulations of population dynamics with varying levels of host performance and pathogen pathogenicity traits, to observe how life-history characteristics influence the dynamics of the plant population.

Assessment of this system, as in Chapter 2, is conducted through the development of a compartmentalised epidemic model in discrete time similar to the models developed by Bailey (1975) and Anderson and May (1979) where individuals within the population are categorised as susceptible, infectious and non-infectious. Disease-induced mortality has been incorporated in models previously as an added mortality term (e.g. Anderson and May, 1979). Here pathogen-induced mortality is assumed to be constant and independent of infected host population size. Constant pathogen-induced mortality has previously been used in plant epidemic models (e.g. Park *et al.*, 2002). Here, constant pathogen-induced mortality is considered between or within season, or both between and within season.

Disease-induced mortality has been reported in a wide range or host plant species exposed to a range of pathogens causing foliar disease (e.g. *Mycosphaerella laricinia* on Pinaceae) systemic diseases (e.g. *Urocystis trientalis on Trientalis europaea*) as well as

a range of cankers, wilts and butt rots (Gilbert, 2002). Also the model is adapted to observe the impact of pathogen-induced mortality impacts on the abilities of plants to overwinter and to re-emerge the following season. This type of pathogen-induced mortality is reported with late infections of *S. vulgaris* by *P. lagenophorae* late in the season (Frantzen, 2007). Finally pathogen-induced mortality is implemented into the models both within and between season. This has been observed when *S. vulgaris* is infected with *P. lagenophorae* at the beginning of the growing season resulting in infected host plants to have lower survival during the season and also increased mortality in the overwintering phase (Frantzen and Müller-Schärer, 1999).

3.3. The Model

The parameters used in the pathogen-induced mortality are identical to those used in the basic model presented in Chapter 2, with the addition of a term β which represents a constant pathogen-induced mortality rate which can be applied either between or within season, or both within and between season. The reason for the two different times of mortality is related to the mechanism of mortality. Within-season mortality is assumed due to pathogen infection causing the host to be less able to compete for nutriants than uninfected individuals, so are more likely to suffer mortality. Between-season mortality is assumed to occur due to pathogen infection limiting the ability for hosts to re-emerge in the second year.

3.3.1. Within season additional mortality

The assumptions of the model are the same as for the basic model (Chapter 2) with the addition that there is a constant pathogen-induced mortality term β for exposed and infectious individuals within season (Figure 40a)

The equations are derived in the same way as in Chapter 2.

$$S_{t+1} = \frac{a(1-b)R_t}{1+(1-b)\lambda R_t} \quad \textbf{(22)}$$

$$I_{t+1} = (1-d)(1-b)pS_t\left\{1-e^{-c(1-b)(1-\beta)I_t}\right\} \quad \textbf{(23)}$$

$$R_{t+1} = (1-b)(1-d)S_t\left\{1-p[1-e^{-c(1-b)(1-\beta)I_t}]\right\} \quad \textbf{(24)}$$

Note that importantly β appears **within** the exponent in equations 23 and 24.

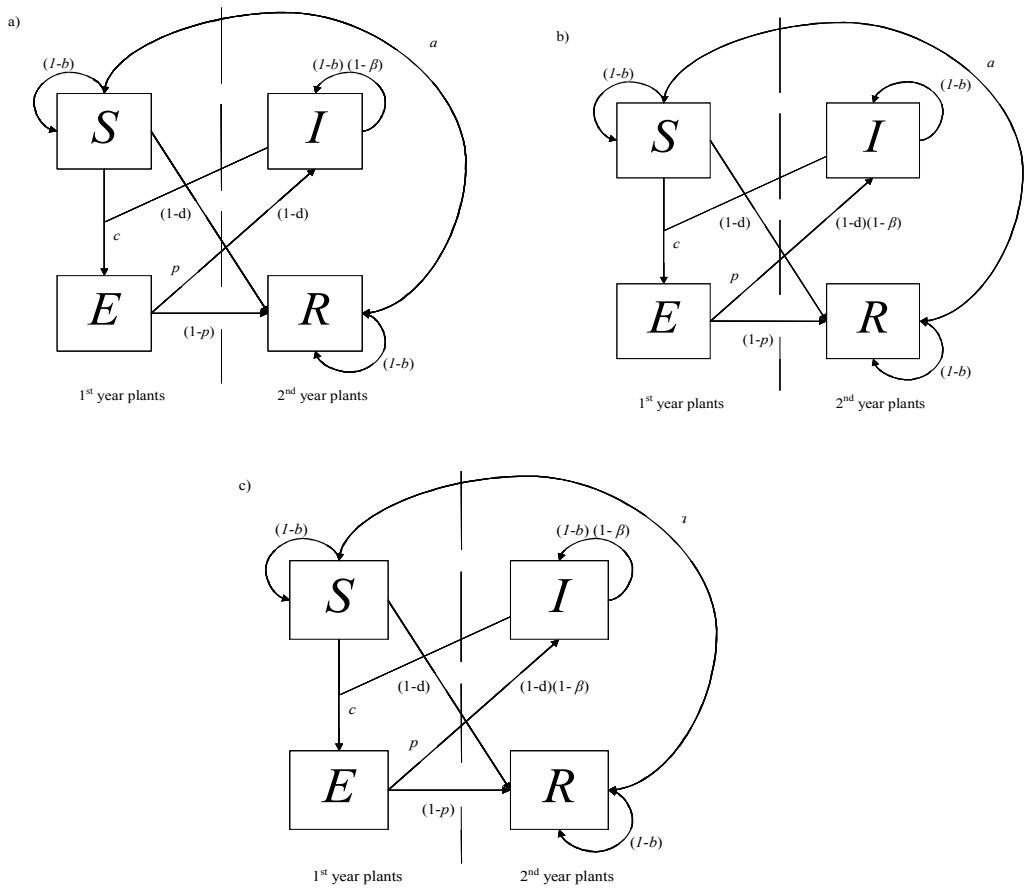


Figure 40 Schematic representations of the compartmentalised model with a) pathogen-induced mortality impacts only within seasons b) pathogen-induced mortality impacts only between seasons and c) pathogen-induced mortality applied between and within season: α: Beginning of season ω: End of Season.

3.3.2. Between season additional mortality

Here pathogen-induced mortality β is applied between-season (Figure 40b).

The derived equations are:

$$I_{t+1} = (1-d)(1-b)(1-\beta)pS_t\left\{1-e^{-c(1-b)I_t}\right\} \quad \textbf{(25)}$$

$$R_{t+1} = (1-b)(1-d)S_t\left\{1-p[1-e^{-c(1-b)I_t}\ ]\right\} \quad \textbf{(26)}$$

In this form of the model, β only appears **outside** the exponent in equations 26 and 27.

3.3.3. Within and between-season additional mortality

Here pathogen-induced mortality β is applied both between and within-season (Figure 40c).

The equations are:

$$I_{t+1} = (1-d)(1-b)(1-\beta)pS_t\left\{1-e^{-c(1-b)(1-\beta)I_t}\right\} \quad \textbf{(27)}$$

$$R_{t+1} = (1-b)(1-d)S_t\left\{1-p[1-e^{-c(1-b)(1-\beta)I_t}]\right\} \quad \textbf{(28)}$$

Where β now appears both **within** and **outside** the exponent in equations 27 and 28.

Note that the term representing succeptible individuals remains as equation 22 across all three models.

3.4. Methods of analysis

In order to observe the properties of this extended model, steady-state equilibria were developed to describe final size values for the population in the absence of pathogen. Implicit steady-state values of I were obtained using the graphical method described in Chapter 2. Simulation outputs were produced using MAPLE 10 (Waterloo Maple Inc. 2005) by varying parameter values representing high, low and medium pathogenicity and host performance traits. The epidemic development criterion was also derived in the same way as in Chapter 2. Additionally, simulations were produced reflecting trade-offs in life-history characteristics of the pathogen by making pathogen-induced mortality (β) inversely related to the pathogen contact rate (c). The parameter range and default values are the same as used for the basic model described in Chapter 2; where used as a default value β was set at 0.2.

3.5. Analysis

3.5.1. Steady-state expressions

In the absence of pathogen for all three models the steady-states are as in Chapter 2:

$$\hat{R} = \frac{a(1-b)^2(1-d)-1}{\lambda(1-b)} \quad \textbf{(29)}$$

$$\hat{S} = \frac{a(1-b)^2(1-d)-1}{\lambda(1-b)^2(1-d)} \quad \textbf{(30)}$$

Steady-state values for I individuals were obtained by solving the relevant implicit equations derived in the same way as described in Chapter 2.

For pathogen-induced mortality acting between and within seasons.

$$I^* = \frac{p(1-\beta)[1-e^{-c(1-b)(1-\beta)I^*}]}{\lambda(1-b)}\left\{a(1-b)^2(1-d)-\frac{1}{1-p[1-e^{-c(1-b)(1-\beta)I^*}]}\right\} \quad \textbf{(31)}$$

For pathogen-induced mortality acting between seasons,

$$I^* = \frac{p(1-\beta)[1-e^{-c(1-b)I^*}]}{\lambda(1-b)}\left\{a(1-b)^2(1-d)-\frac{1}{1-p[1-e^{-c(1-b)I^*}]}\right\} \quad \textbf{(32)}$$

For pathogen-induced mortality implemented within season,

$$I^* = \frac{p[1-e^{-c(1-b)(1-\beta)I^*}]}{\lambda(1-b)}\left\{a(1-b)^2(1-d)-\frac{1}{1-p[1-e^{-c(1-b)(1-\beta)I^*}]}\right\} \quad \textbf{(33)}$$

3.5.2. Epidemic development criteria

With an infinitesimally small amount of infectious material, the term $1-e^{-c(1-b)(1-\beta)I_t}$ can be approximated by $c(1-b)(1-\beta)I_t$.

For an epidemic to occur where pathogen-induced mortality acts both within and between season the following condition must hold:

$$\hat{S}(1-\beta)^2(1-b)^2(1-d)pc > 1 \quad \textbf{(34)}$$

For an epidemic to occur in the model with diseased-induced mortality acting between or within season, the criterion for both forms of the model is:

$$\hat{S}(1-\beta)(1-b)^2(1-d)pc > 1 \quad \textbf{(35)}$$

The pathogen invasion equations indicate that pathogen performance is related to infection probability and pathogen-induced mortality, whilst also being reliant on the background host mortality between and within season and susceptible host density. Where additional pathogen-induced mortality is present both within and between-season then the threshold is determined by $(1-\beta)^2$ rather than $1-\beta$.

The parameter space under which epidemics occur or do not occur, for the form of the model with pathogen-induced mortality **both** within and between seasons (equation 36) under default values, whilst altering the range value of *c*, *p* and $\hat{S}$ is shown Figure 41. The infection and probability terms have a strong influence on the epidemic threshold condition. Furthermore, with default mortality rates and altering of the pathogenicity parameters and also number of susceptible individuals, the epidemic threshold condition holds within the estimated parameter ranges. Therefore it is possible to infer that pathogen epidemics are reliant on sufficiently high infection rate and also high probability of exposed individuals becoming infectious in appropriate sized host populations. By increasing β in relation to the product *cp*, there is no epidemic threshold obtained for $\beta > 0.7$ (Figure 42). As β increases from low to high values the region in which an epidemic can occur reduces, requiring high *cp* values and also high *S* values for the threshold to be achieved. As Figure 35 is equivalent to the model with pathogen-induced mortality within and between-season where $\beta=0$, it is seen that (particularly at low values of *c*) that increased values of β decreases the parameter space of where epidemics occur. At higher β values, higher values of *p* and *S* are required for corresponding *c* values for the threshold to be obtained.

The parameter space for the epidemic threshold where there is pathogen-induced mortality **either** between or within season is shown in Figure 43. Increasing values of *c* and *p* lead to increased regions of epidemic conditions where *S* is lowered. The difference between the two epidemic thresholds terms can be seen comparing Figure 44 (equation 37) and Figure 45 (equation 36) demonstrating that where there is pathogen-induced mortality applied both within and between-seasons the epidemic threshold is crossed at higher *pc* and *S* values, than where pathogen-induced mortality is applied

either between or within season. Similar observations can be seen in Figure 43, where higher β values enable pathogen persistence. Where pathogen-induced mortality acts between or within season increased values of β requires higher values of the product of pc and S for the epidemic threshold to be crossed, however for β>0.9, the threshold is not obtained (Figure 46).

The product of both p and c are required to be relatively low but greater than 0.075 to induce an epidemic within the initial susceptible population where there is pathogen-induced mortality between and within-season (Figure 45) whereas under pathogen-induced mortality between or within season the pc>0.06 for an epidemic to occur (Figure 44). The default p and c product of 0.21 requires a susceptible population of 21.7 for an epidemic to occur, indicating that with S_0 = 20, epidemic conditions are created after the first year of recruitment. Similarly Figure 44 indicates that for the default pc value, the epidemic threshold is obtained with a susceptible population of 17.4.

For the default β value of 0.2, Figure 47 shows that within the range of susceptible individual there are conditions which allow for an epidemic to arise. However, increasing β above 0.3 leads to conditions progressively unsuitable for an epidemic to occur. Figure 48 shows that using the default β value leads to conditions unsuitable for the epidemic to persist with the model where there is pathogen-induced mortality applied both between and within-seasons

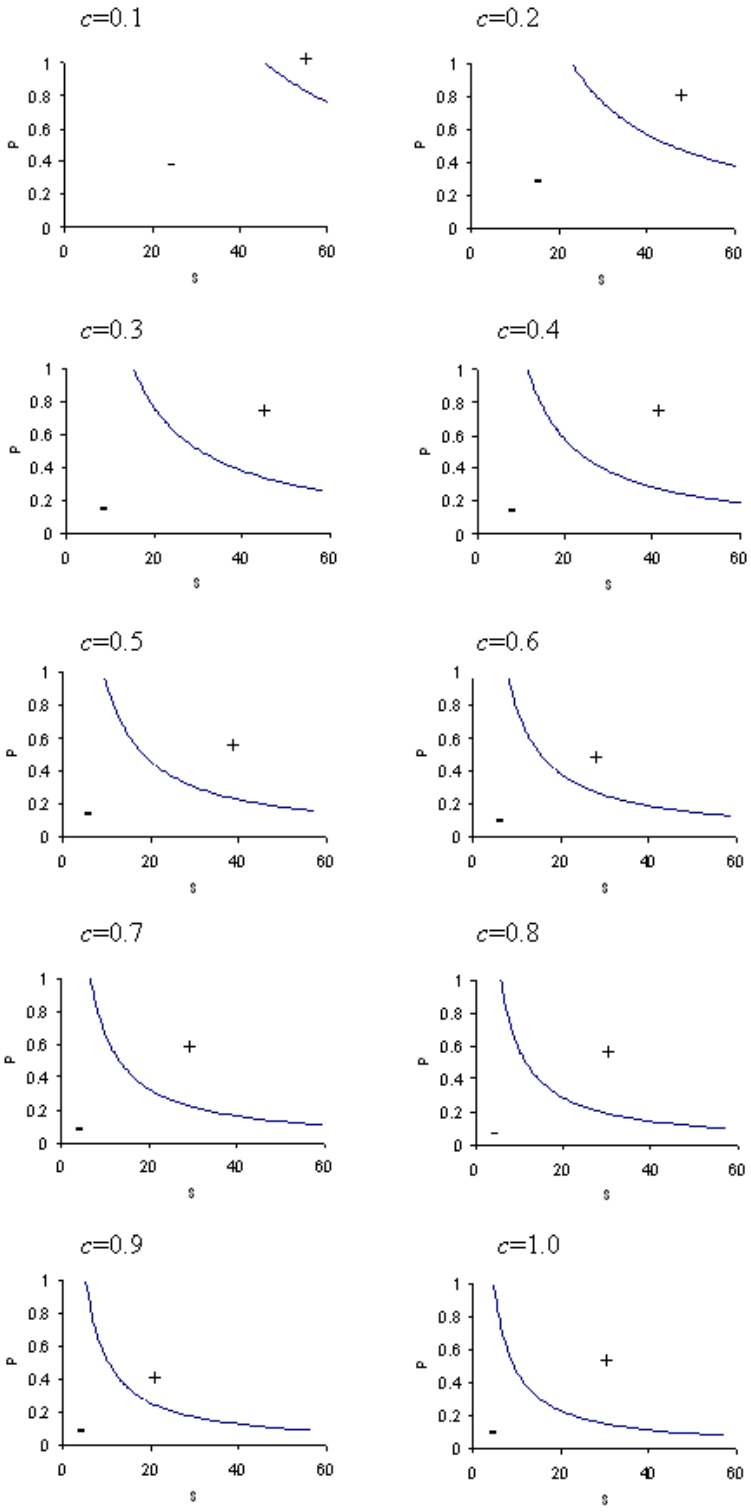


Figure 41. Epidemic development thresholds for varying values of *c* in and *p* relation to varying initial *S* values, for the model with pathogen-induced mortality both within and between seasons. With default values of β =0.2. Epidemic regions indicated with +, whilst non-epidemic regions indicated with -.

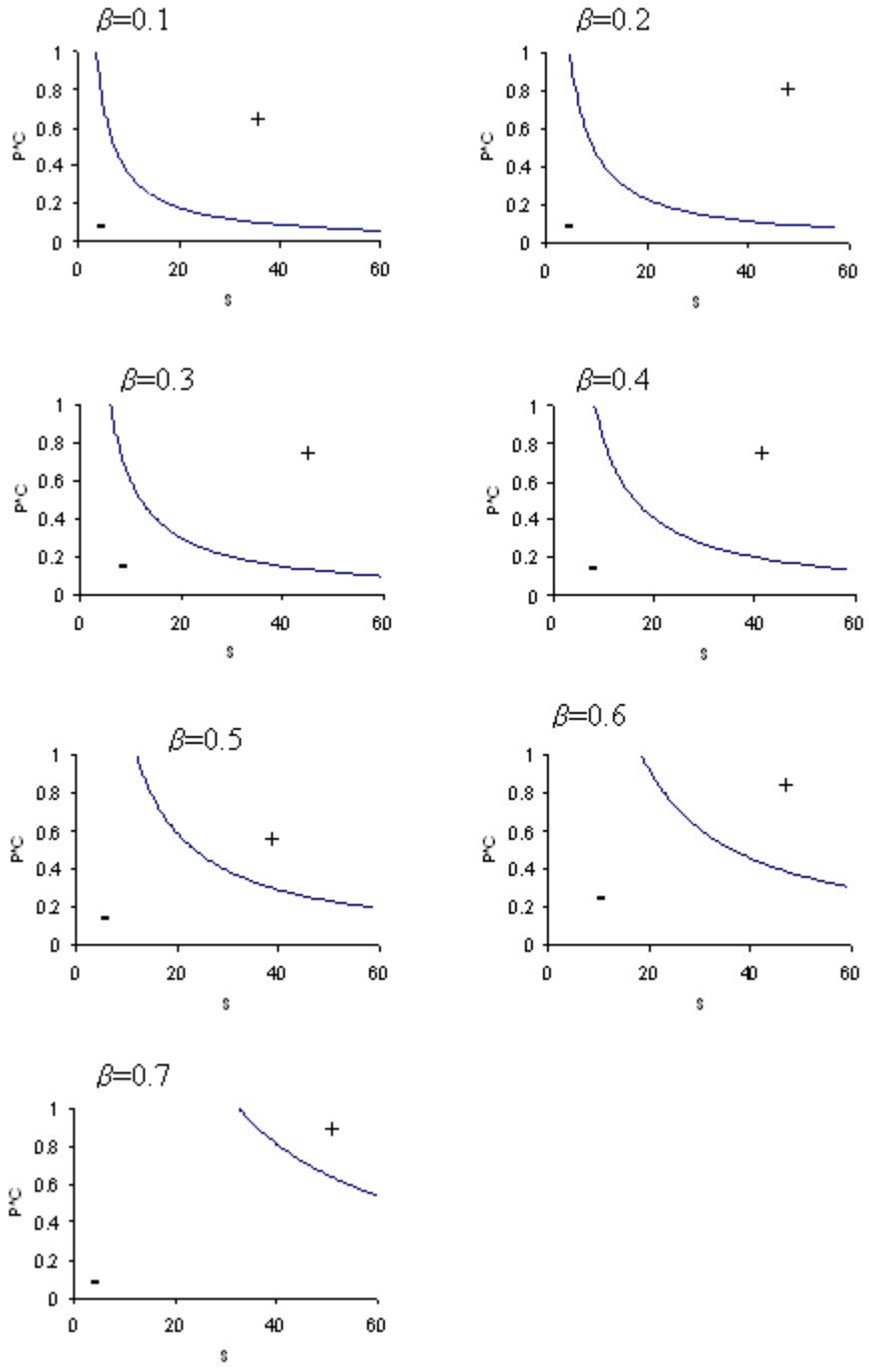


Figure 42. Epidemic development thresholds for varying values of the product of *cp* in relation to varying initial *S* values with increasing values of *β*, for the model with pathogen-induced mortality within and between seasons. Epidemic regions indicated with +, whilst non-epidemic regions indicated with – The pathogen threshold is crossed where *β* is greater than 0.7.

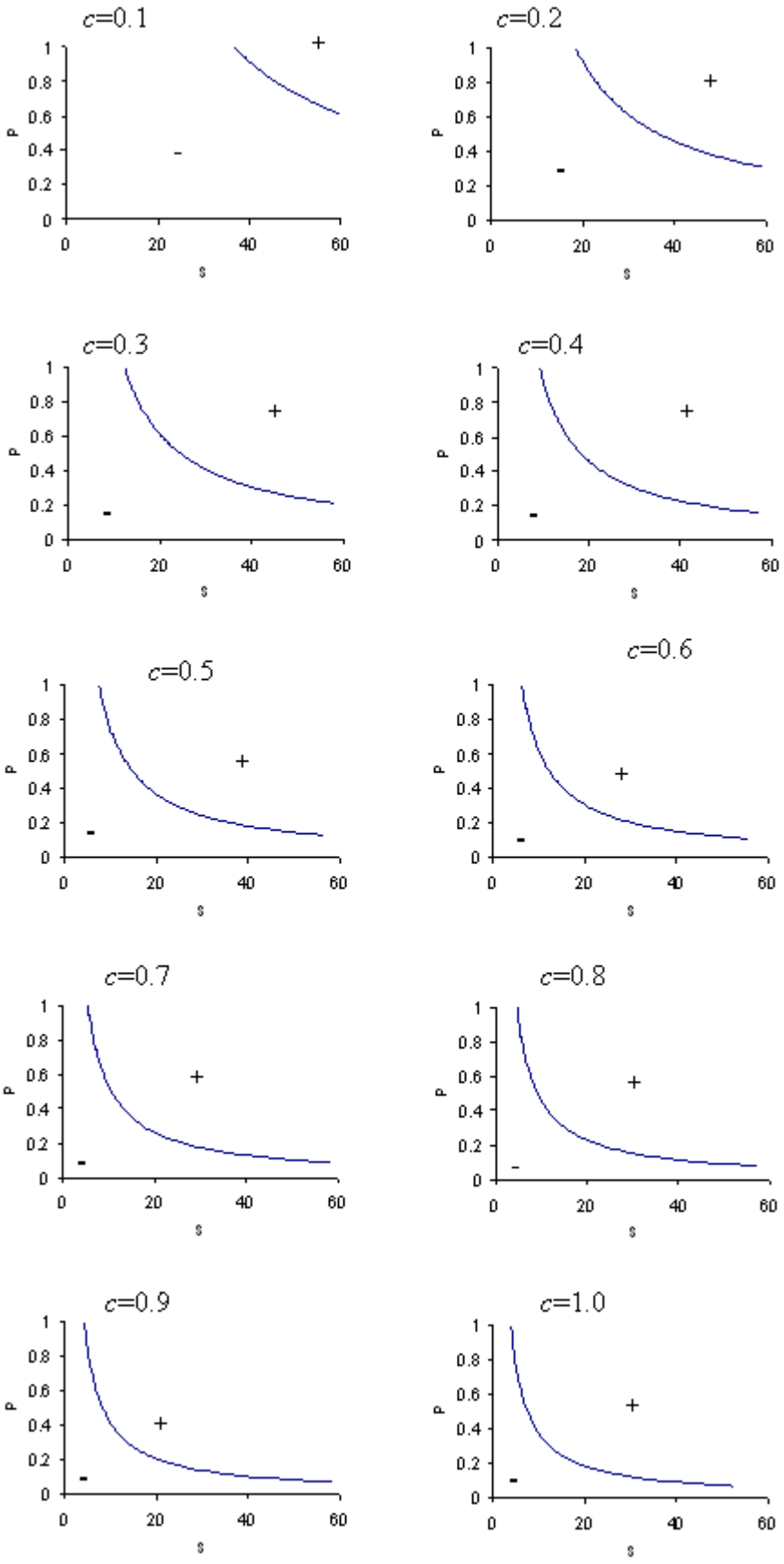


Figure 43. Epidemic development thresholds for varying values of *c* and *p* in relation to varying initial *S* values, for the model with pathogen-induced mortality either within or between seasons. Epidemic regions indicated with +, whilst non-epidemic regions indicated with - .

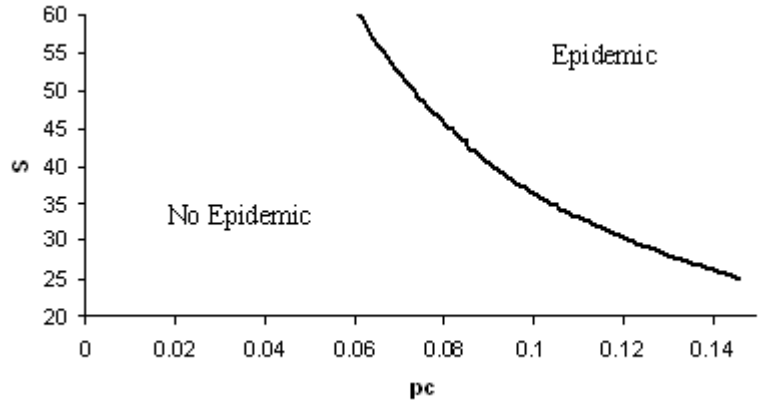


Figure 44. The product of *pc* required to obtain invasion criterion for pathogen-induced mortality acting either between or within season for values of *S* within the parameter range under the default parameters for *b* and *d*.

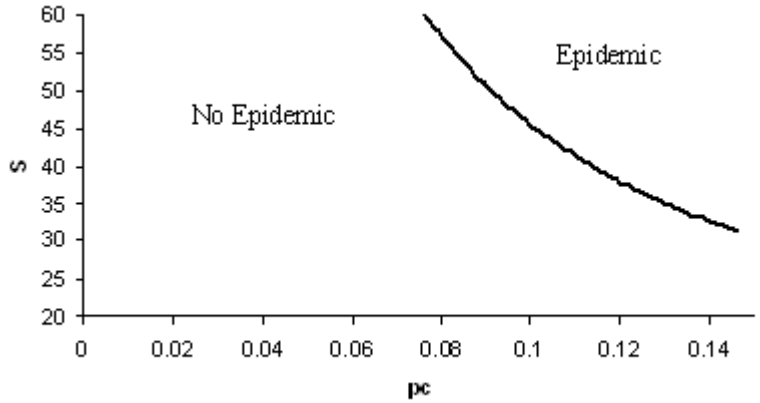


Figure 45. The product of *pc* required to obtain invasion criterion for pathogen-induced mortality acting both between and within season for values of *S* within the parameter range under the default parameters for *b* and *d*.

The effect of altering one parameter value on the steady-state values for the number of susceptible first year individuals is shown in Figure 49. As expected, increasing seedling recruitment increases the number of susceptible individuals (Figure 49b). Also increasing λ (Figure 49a) or mortality rates (Figure 49c) leads to decreased steady-state *S* values. However, with mortality rates greater than 0.46 applied between and within-seasons the conditions do not meet the requirements of the epidemic development criterion, whereas an epidemic is obtained for pathogen-induced mortality between or within season at mortality rates of 0.49. This difference in threshold is due to the additional mortality acting twice on individuals for within and between season model forms, therefore losses due to background mortality rate of the host is required to be lower in order for disease to occur.

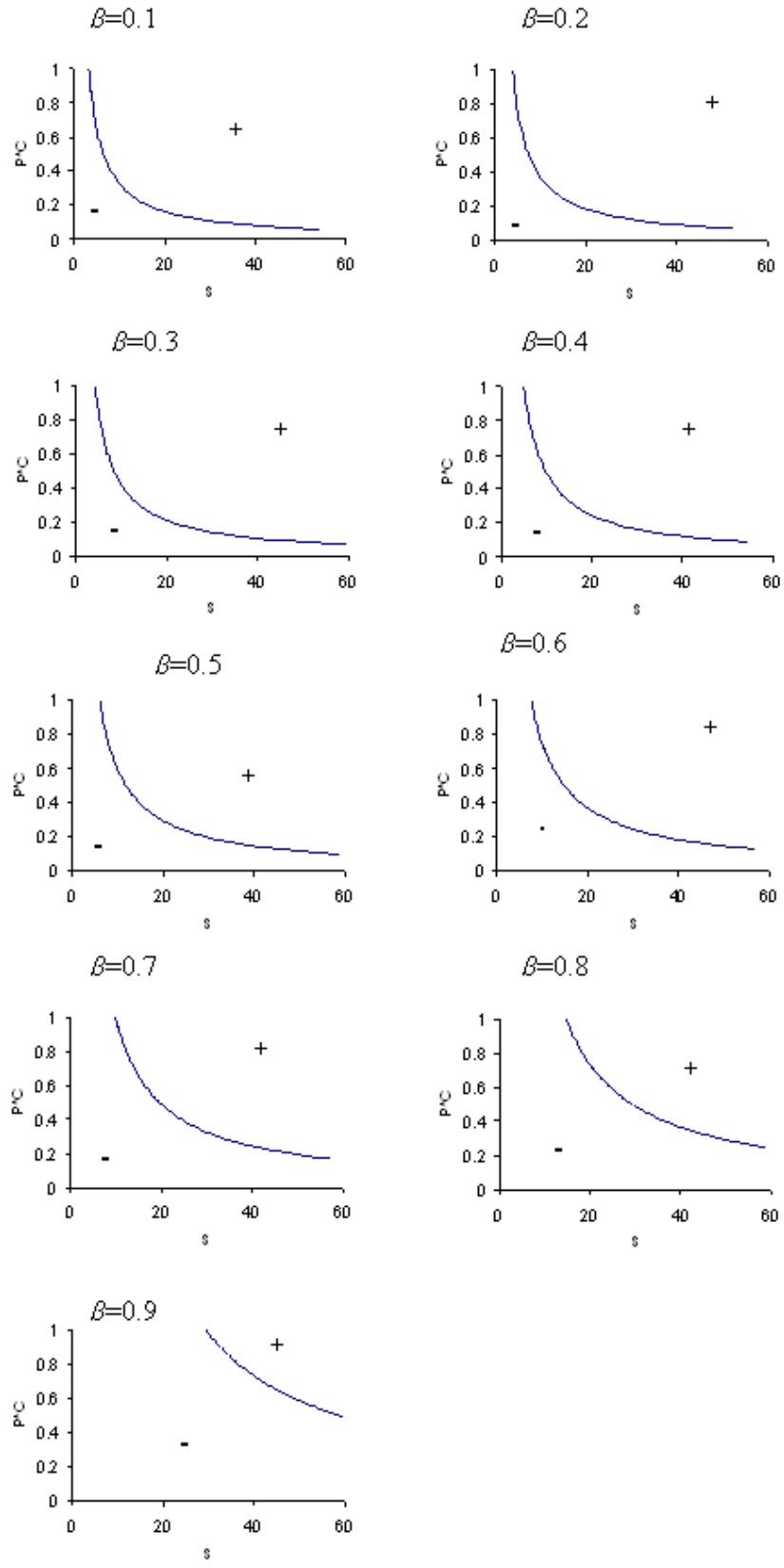


Figure 46. Epidemic development thresholds for varying values of the product of *cp* in relation to varying initial *S* values with increasing values of *β*, for the model with pathogen-induced mortality either within or between seasons. Epidemic regions indicated with +, whilst non-epidemic regions indicated with -. When *β* is greater than 0.9 the threshold is achieved irrespective of *p*, *c* and *S*.

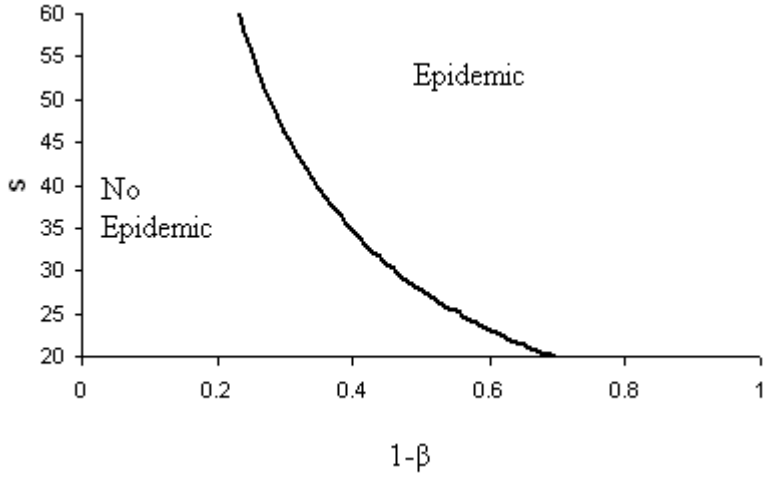


Figure 47. Altering β to obtain the invasion criterion for pathogen-induced mortality either between or within season (equation 37) within the parameter range under the default parameters for *b* and *d*.

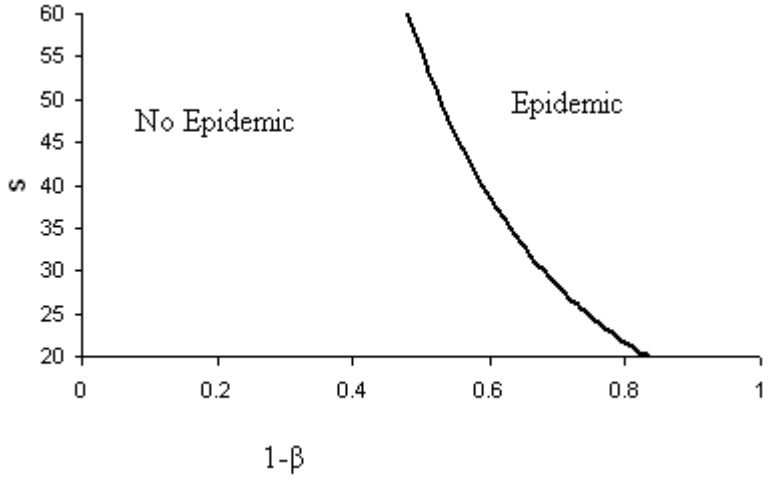


Figure 48. Altering β to obtain the invasion criterion for pathogen-induced mortality both between and within season (equation 35) within the parameter range under the default parameters for *b* and *d*.

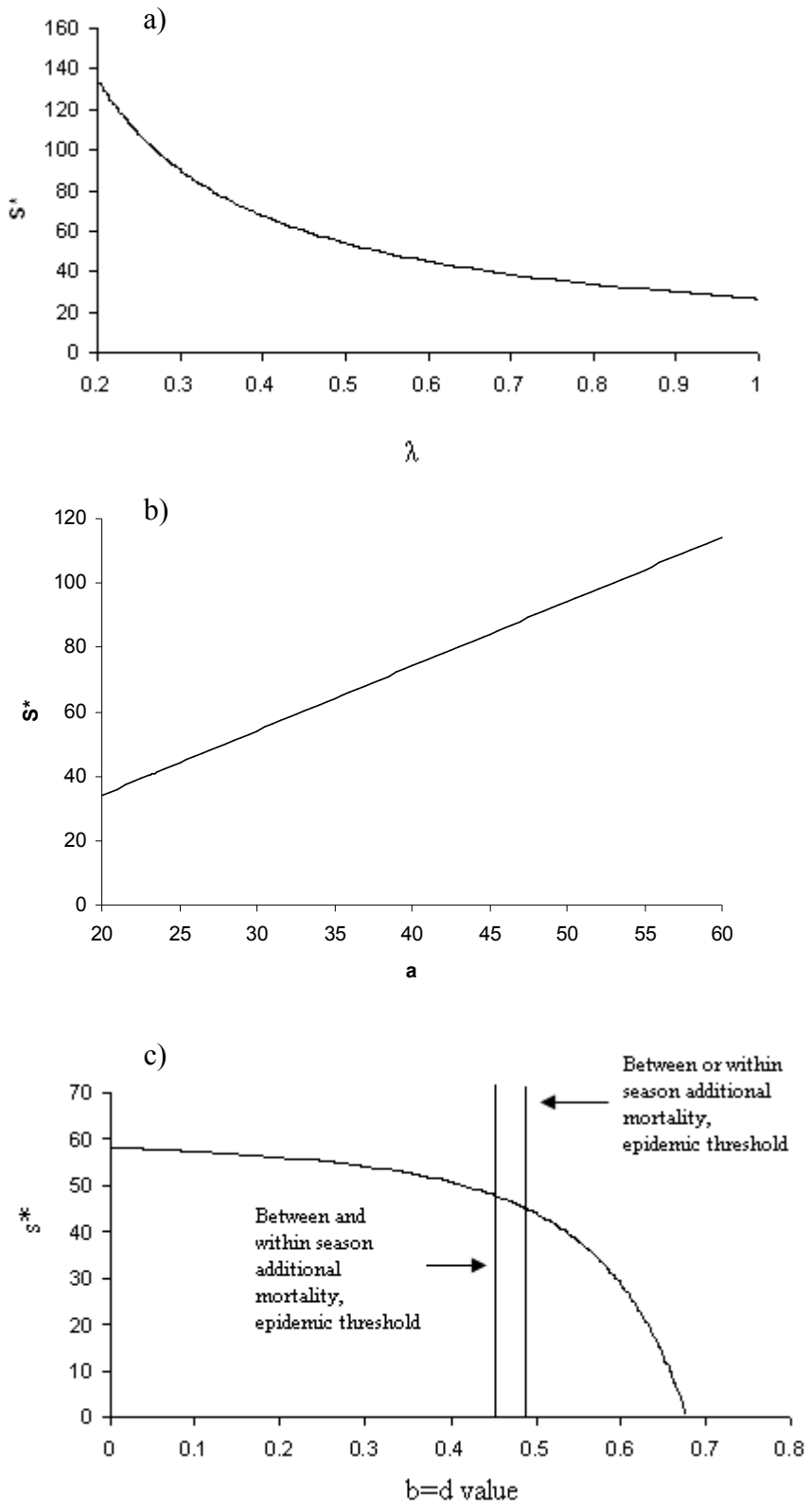


Figure 49. The effect of varying one parameter value within the parameter range for the constant pathogen-induced mortality within and between season model, whilst the other parameters are set at the default value. a) λ , b) *a* and *c*) the mortality rates *b* and *d* where *b=d*. Note that only outcomes for values within the ranges (Table 4).

Using default parameter values and increasing β between 0.1 to 1.0 shows that there is variation in the steady-state I populations (Figure 50 – 33), where the steady-state values of I individuals decrease with increasing β acting within season, between season and both between and within season. Stable populations are produced for β values between 0.1 and 0.3 (1sf) for pathogen-induced mortality between and within season (Figure 50), 0.1 and 0.4 for pathogen-induced mortality acting only between seasons (Figure 51), whereas steady populations are created with values of β applied within season (Figure 52) between 0.1 and 0.6. High β values cause extinction of the pathogen within the population, indicating that high pathogenicity would be selected against. Also shown is that lower stable population values are obtained where pathogen-induced mortality occurs both between and within season, than for corresponding values where β is applied between season only, which in turn has a low steady-state value compared to for where β is applied within season.

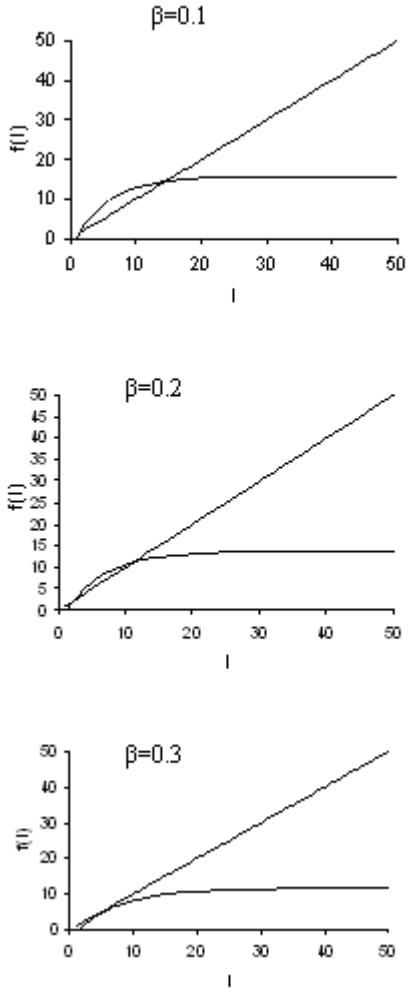


Figure 50. An example of solving stable-state values for *I for pathogen-induced mortality acting between and within season, using default parameters with increasing values of β from 0.1-1.0. *I** is defined as the interception between *I* and the f(*I*). There is no interception for β>0.3.**

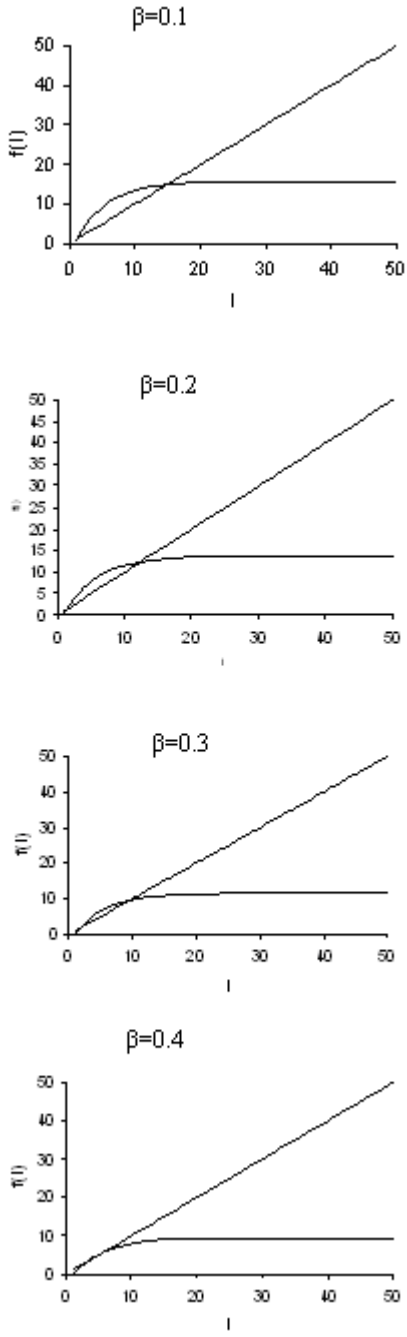


Figure 51. An example of solving stable-state values for *I for the pathogen-induced mortality between season, using default parameters with increasing values of *β* from 0.1-1.0. *I** is defined as the interception between *I* and the f(*I*). There is no interception for *β*>0.4.**

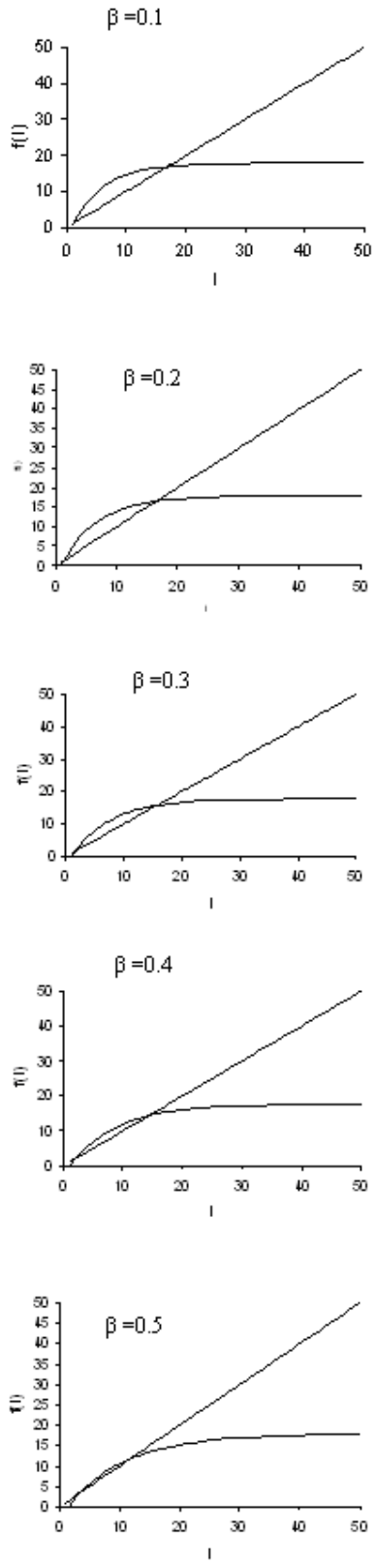


Figure 52. An example of solving stable-state values for *I for the pathogen-induced mortality within season, using default parameters with increasing values of *β* from 0.1-1.0. *I** is defined as the interception between *I* and the f(*I*). There is no interception for *β*>0.5.**

3.5.3. Population simulations

Using the range of parameter values which reflect high, medium and low pathogenicity and host performance traits (Table 6), a number of simulation outcomes are possible (

Figure 53, 54 55). This represents population cycles, populations with steady-state susceptible, infectious and non-infectious second year individuals, steady-state healthy populations with no epidemic, and also host population crashes.

Table 6. Parameter ranges for low, medium and high host and pathogen performance

	Low	Medium	High
Pathogen			
c	0.1	0.3	0.8
p	0.3	0.7	1.0
β	0.1	0.2	0.3
Host			
a	20	30	60
b	0.4	0.3	0.1
d	0.5	0.3	0.1
λ	1.0	0.5	0.2

A evolutionary trade-off representing an augmentation in one pathogen characteristic at the detriment in another can be introduced by assuming an inverse relationship between the infection rate and pathogen-induced mortality (β). Strains with low transmissibility may have a higher pathogen-induced mortality levels and vice-versa (Table 7) producing outcomes shown in Figure 56, 57 and 59.

Table 7. Parameter ranges for low, medium and high host and pathogen performance with pathogen life history trade-off

	Low	Medium	High
Pathogen			
c	0.1	0.3	0.8
p	0.3	0.7	1.0
β	0.3	0.2	0.1
Host			
a	20	30	60
b	0.4	0.3	0.1
d	0.5	0.3	0.1
λ	1.0	0.5	0.2

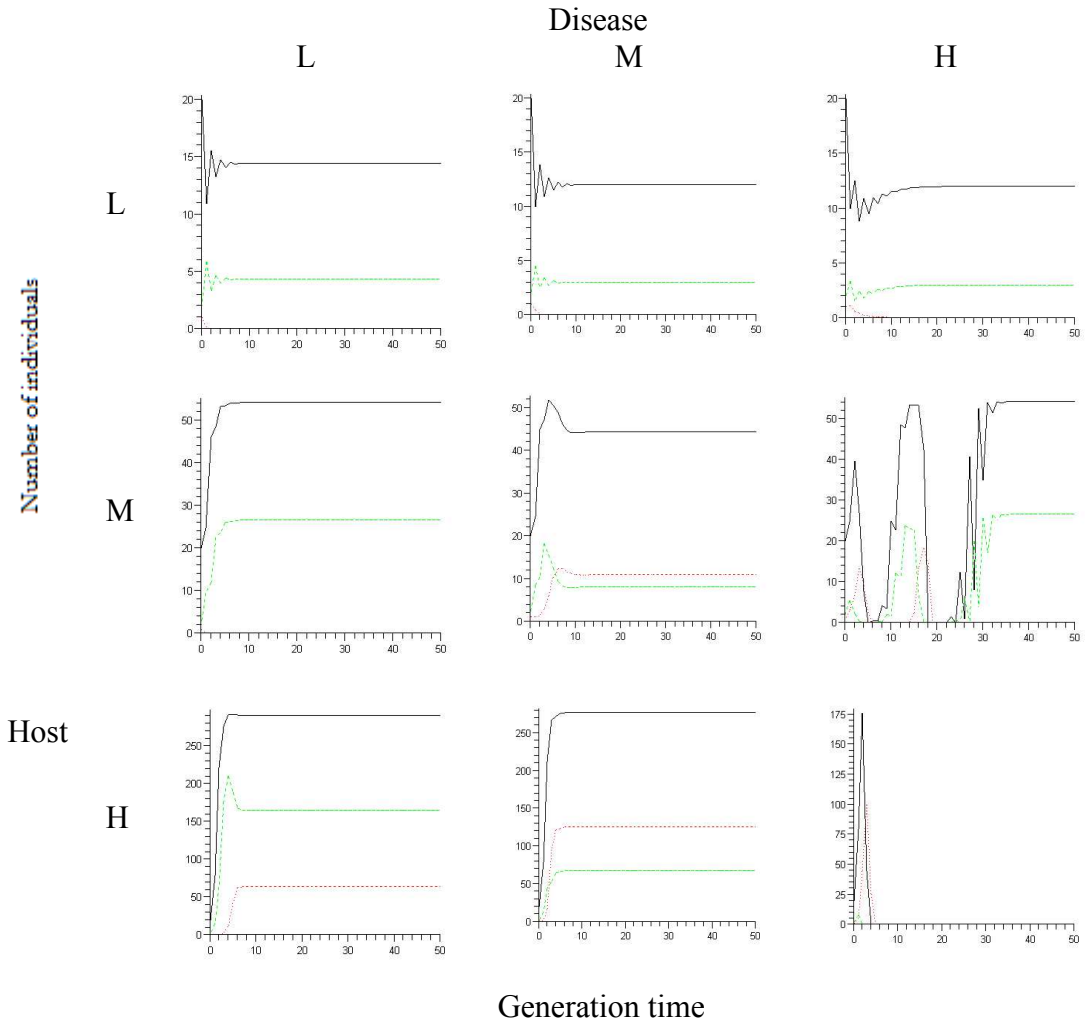


Figure 53. Simulations for pathogen-induced mortality acting between season, produced under conditions of high, medium and low host performance and pathogenicity. Each simulation represents — S, — I, — R dynamics under particular parameter settings.

For low, medium and high pathogenicity with low host performance, pathogen fails to establish in cases where β is applied between-season (

Figure 53), within-season (Figure 54) or both between and within-seasons (

Figure 55). This is also the case for low pathogenicity and medium performance hosts. Disease does establish in the population in all three forms of the model when pathogenicity is either medium or high in medium performance host populations, additionally pathogen is present with low or medium pathogenicity are present in high

performance host populations. In all cases, where there is high pathogenicity and high host performance, the population becomes extinct. Where pathogenicity is high and host performance is medium, cycles occur, however repeating population cycles are only obtained where there is pathogen-induced mortality both between and within-seasons (

Figure 55), whereas with between (

Figure 53) and within (Figure 54) season additional mortality there are *R* individuals with two cycle peaks followed by the extinction of infectious individuals followed by stable populations of *S* and *R* individuals. Additional similarities between the three model forms are that medium pathogenicity in medium or high performance hosts lead to *I* individuals being at higher frequency than *R* individuals; with low pathogenicity in high performance host populations, *R* individuals outnumber infectious individuals.

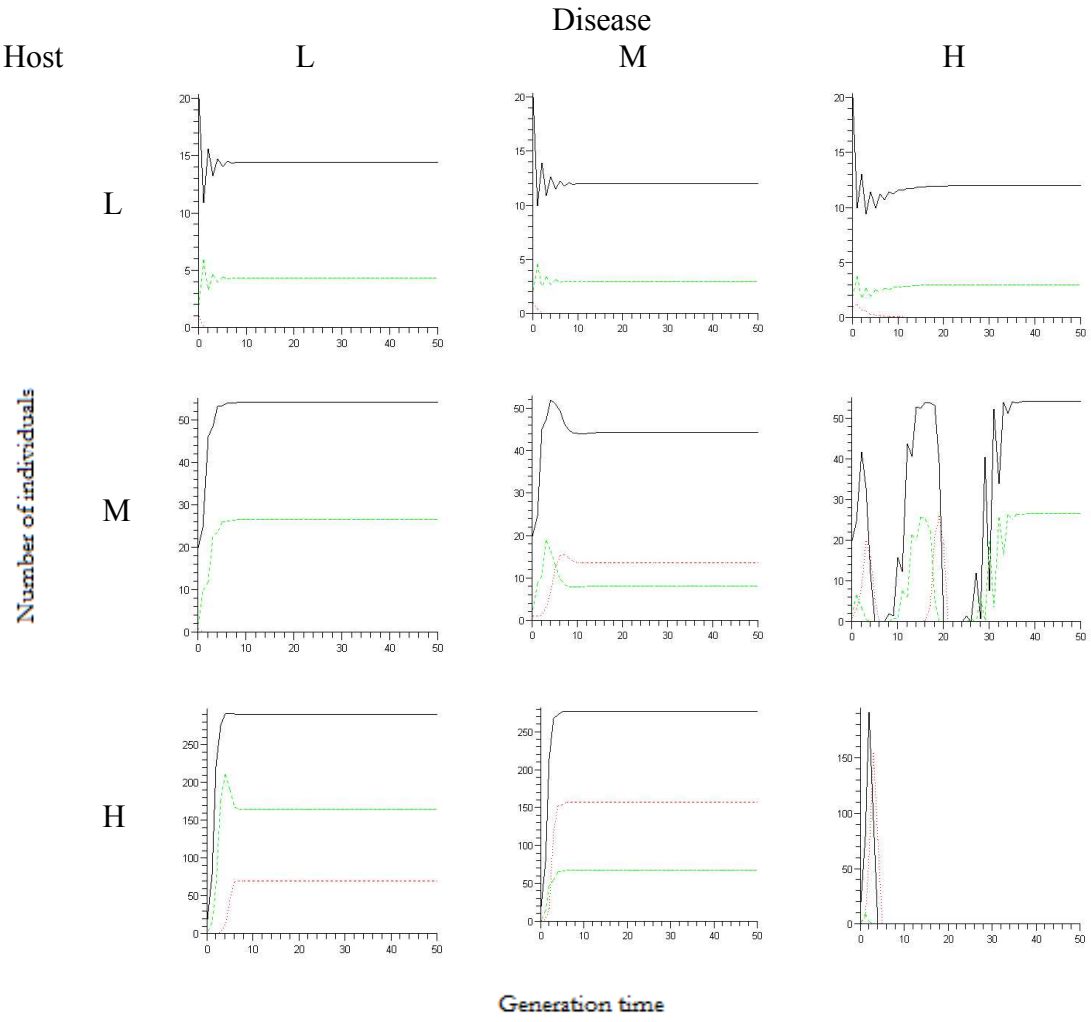


Figure 54. Simulations for pathogen-induced mortality acting within season, produced under conditions of high, medium and low host performance and pathogenicity. Each simulation represents — S, — I, — R dynamics under particular parameter values.

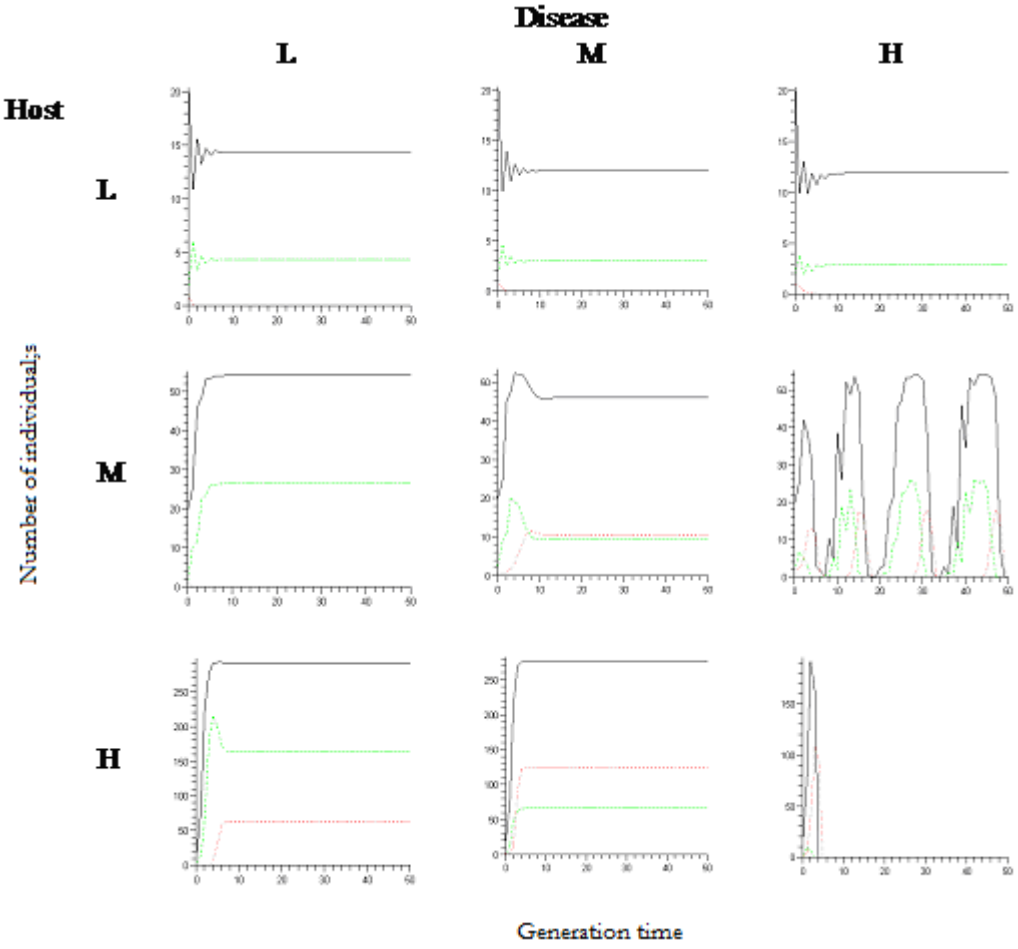


Figure 55. Simulations for pathogen-induced mortality acting between and within season, produced under conditions of high, medium and low host performance and pathogenicity. Each simulation represents — S, — I, — R dynamics under particular parameter values.

Where there is an evolutionary trade-off between c and β, the outputs for pathogen-induced mortality between (Figure 56) and within (Figure 57) produce similar dynamics and outcomes to those without the trade-off (

Figure 53 and 56). However when there is pathogen-induced mortality both between and within-seasons (Figure 58) the recurring population cycles in the high pathogenicity, medium host performance combination do not occur with two cycles followed by pathogen extinction and S and R reaching stable populations.

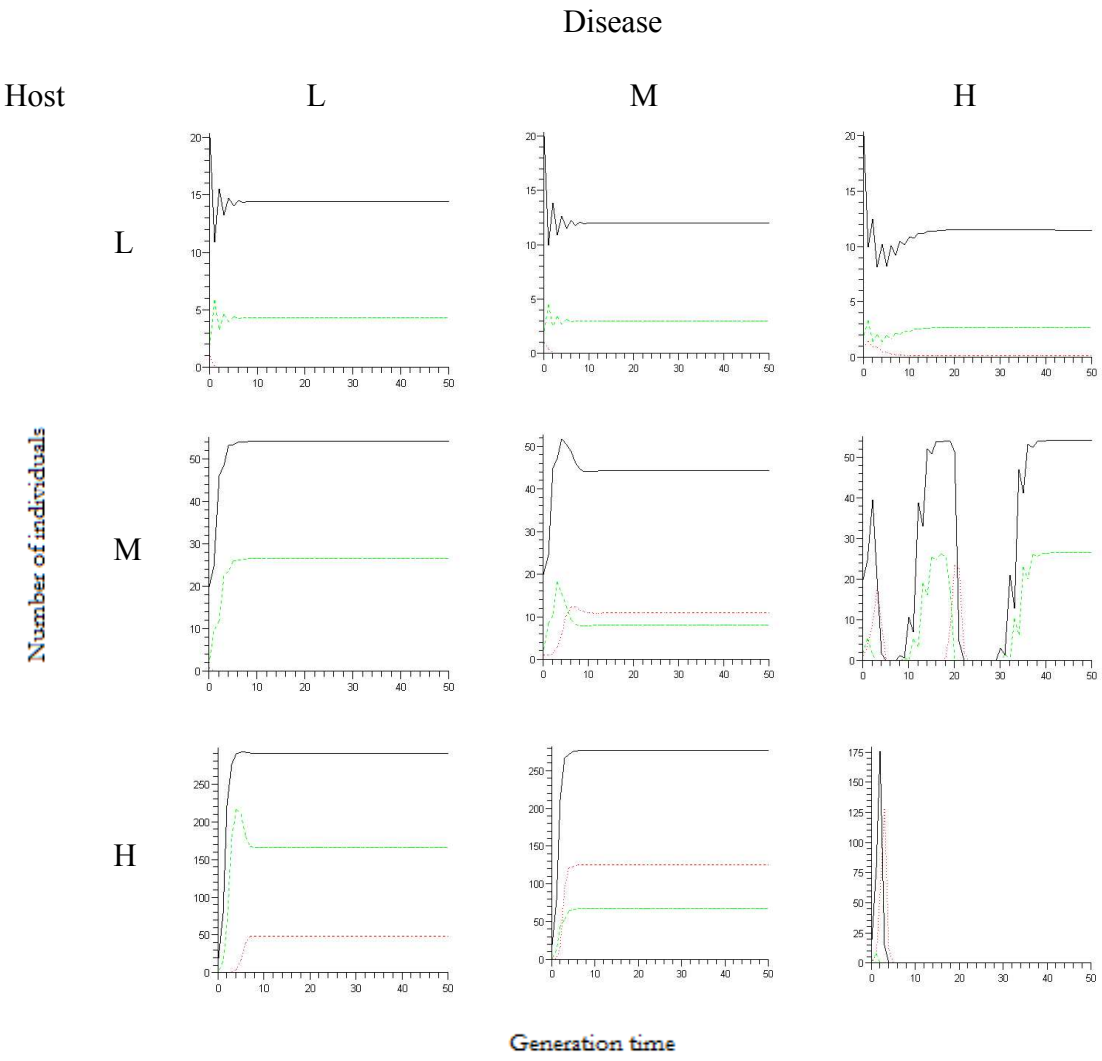


Figure 56. Simulations for pathogen-induced mortality applied between season, produced under conditions of high, medium and low host performance and pathogenicity with a trade-off in pathogen life-history parameters. Each simulation represents — S, — I, — R dynamics under particular parameter values.

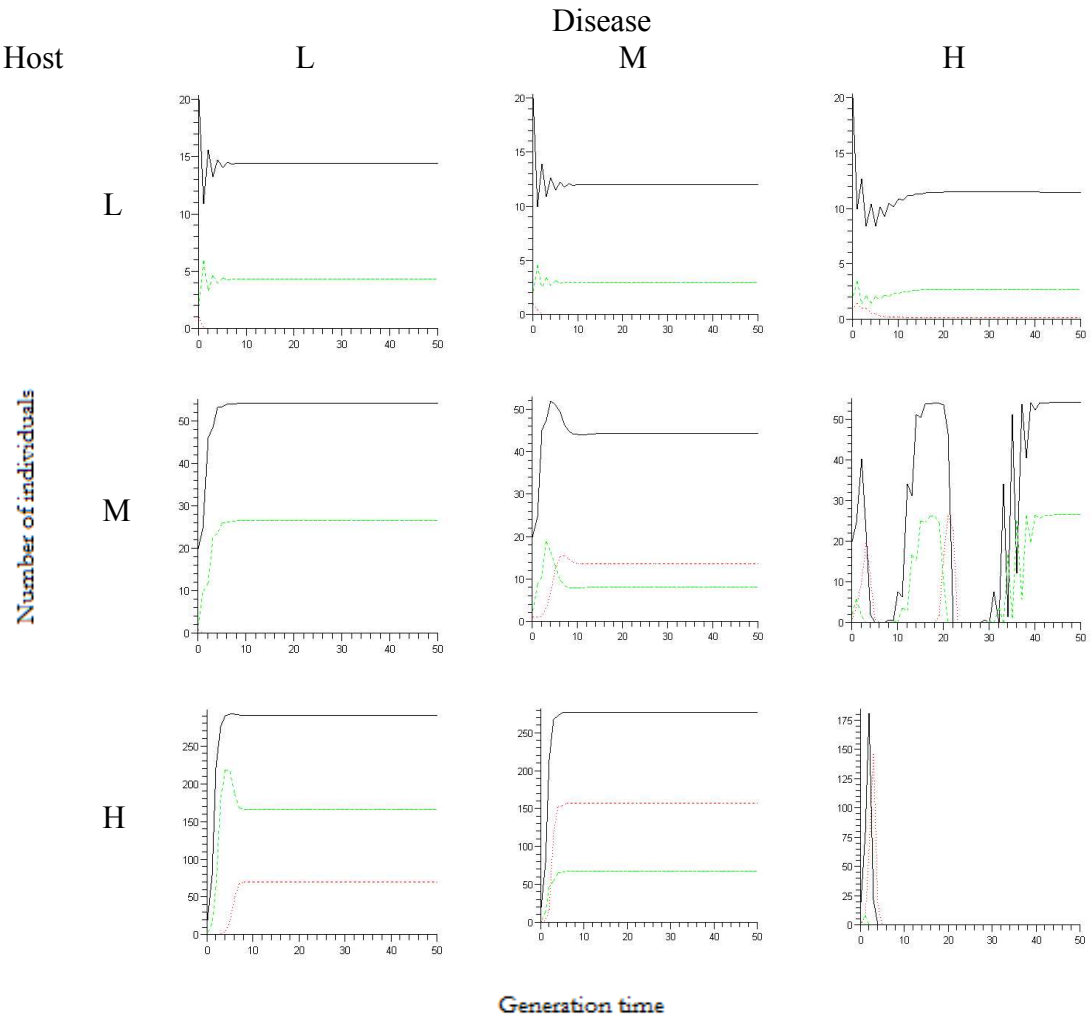


Figure 57. Simulations for pathogen-induced mortality applied within season, produced under conditions of high, medium and low host performance and pathogenicity where there is a trade-off between *c* and *β*. Each simulation represents — S, — I, — R dynamics under particular parameter values.

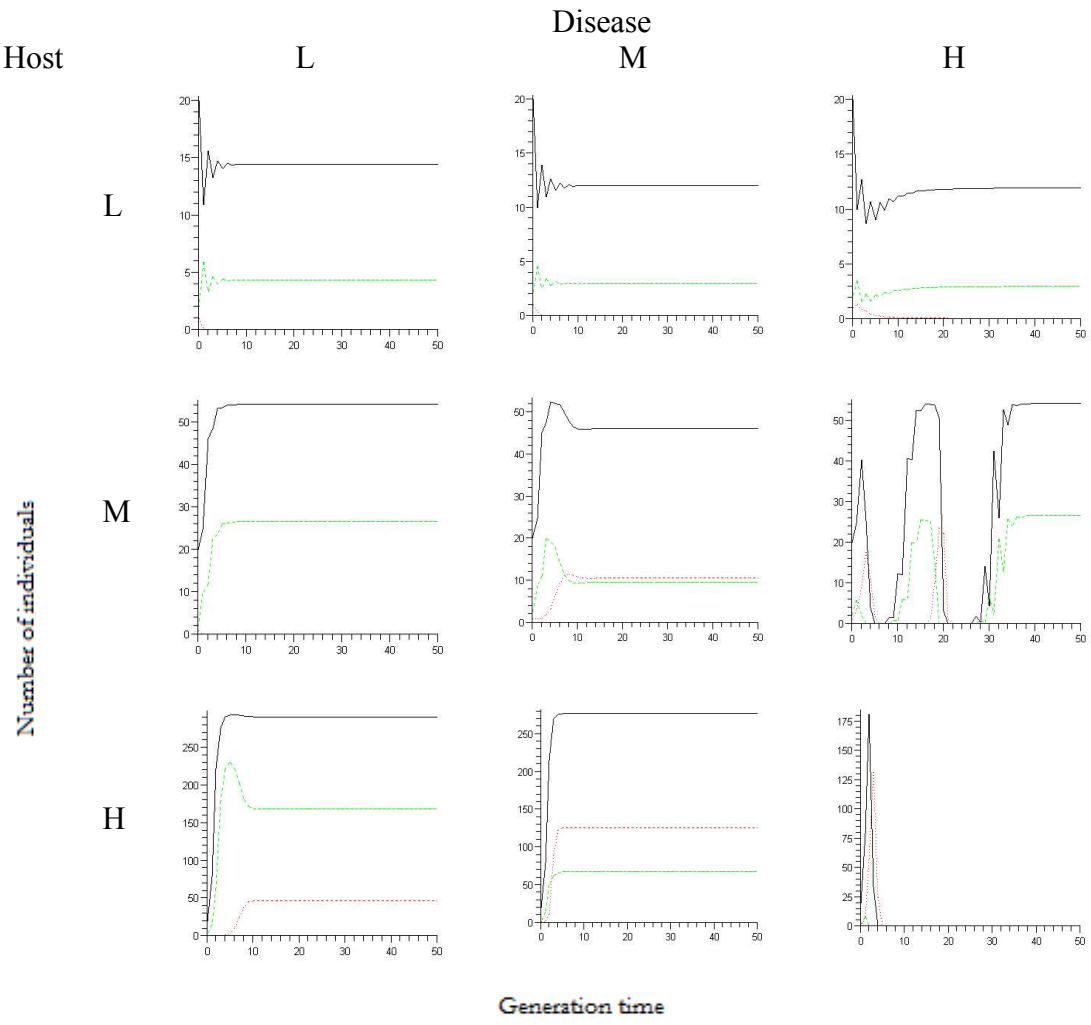


Figure 58. Simulations for pathogen-induced mortality applied between and within season, produced under conditions of high, medium and low host performance and pathogenicity where there is a trade-off in pathogen life-history. Each simulation represents — S, — I, — R dynamics under particular parameter values.

3.5.4. Recurrence relationships

For the system without pathogen, a recurrence relationship is obtained that represents the relationship between individuals in sequential years, the same as given by equations 19 and 20 in Chapter 2.

$$\frac{1}{S_{t+1}} = \frac{1+\lambda(1-b)R_t}{a(1-b)R_t} = \frac{\lambda}{a(1-b)} + \frac{1}{a(1-b)R_t} \quad \textbf{(36)}$$

However, when the pathogen-induced mortality is included in the model, either between seasons or both between and within seasons, a recurrence relationship is derived indicating regulation within the system is by pathogen-induced mortality.

$$\frac{(1-\beta)}{I_{t+2} + (1-\beta)R_{t+2}} = \frac{\lambda}{a(1-b)^2(1-d)} + \frac{1}{a(1-b)^2(1-d)R_t} \quad \textbf{(37)}$$

With β appearing on the left hand side of the equation. When β=0, equation 40 reverts to equation 21. As in Chapter 2, the gradient of equation 40 must be less than 1.

3.6. Discussion

In this chapter, three forms of an extended model are presented describing a biennial host population infected with a systemic, castrating pathogen in which there is also additional pathogen-induced mortality. The three forms of the model reflect the influence of additional mortality acting on the dynamics of the host population between season, within season or both between and within season. The additional mortality parameter β used is constant between and within season and is independent of host or pathogen density and applies to both first and second year individuals.

Three forms of the additional mortality model are developed because there is little evidence as to what stage pathogen-induced mortality influences infected individuals. For example, Paul and Ayres (1987) reported no additional mortality of *Senecio vulgaris* seedlings infected by *Puccinia lagenophorae*, whilst mature individuals

showed increased mortality and decreased growth. This suggests an additional mortality term acting on mature infected individuals within a growing season and not on latently infected first year individuals. However Frantzen and Müller-Schärer (1999) reported that *S. vulgaris* infected with *P. lagenophorae* had reduced survivorship over winter for individuals infected late in the season; whereas early season infected individuals had increased mortality during the growing season. Because of the similarities between the *S. vulgaris – P. lagenophorae* system and the *T. pratensis – P. hysterium* system, it is likely that all three models are valid to describe the system depending on when the host population is exposed to inoculum.

The steady-state expressions for I^* (equations 33-35) under default parameter values for the three models indicate that steady-state values of infectious individuals is highest where mortality acts within season, than where it is applied between season and least of all when it acts between and within season.

The pathogen invasion criteria (equations 36 and 37) also reflect the difference between pathogen-induced mortality acting both within and between season, and where it acts either between or within seasons. What is clear is that the epidemic development criterion is lower where additional mortality acts both between and within season, because of the $(1-\beta)^2$ term. This means there is a necessity for a higher susceptible host population to achieve the epidemic criterion where pathogen-induced mortality applies both within and between seasons than either season alone. This is biologically consistent: a higher proportional loss of infectious individuals will require a greater abundance of healthy susceptible individuals in order for an epidemic to persist.

The simulation outputs predict an optimal life-history strategy for the host population to be not overly performing, as an introduction of a highly pathogenic strain would lead to a population crash. Similarly, the optimal strategy for the pathogen would not to be highly pathogenic as a low performing host population would not fulfill the epidemic development criterion, and similarly, a high performing host population would lead to conditions where the host would go extinct along with the pathogen. This optimal strategy for the pathogen is also supported by the outputs where pathogen-induced mortality is applied within or between seasons as highly pathogenic strains do not tend to a stable population independent of host population size.

The derived recurrence relationships indicate that the system is only regulated by the pathogen where there is pathogen-induced mortality both between and within season or just between seasons (equation 40).

Through the development of three forms of the model representing systems in which there is diseased-induced mortality within season, between season and both within and between season, it is possible to produce differing steady-state expressions and invasion criteria under the various forms. These model forms produce slightly varied population simulations which produce dynamics reminiscent of the PGE data when the host is of medium productivity and high pathogenicity; this is discussed further in Chapter 6.

4. Variable pathogen-induced mortality dependent on the density of diseased individuals, and its impact on the population dynamics of a biennial host plant.

4.1. Summary

In this chapter the pathogen-induced mortality model forms presented in Chapter 3 are extended to include a variable pathogen-induced mortality, dependent upon the numbers of infectious individuals in the previous generation. This density-dependent, pathogen-induced mortality term may act within season, between season and both within and between seasons. Simulation outputs indicate that for most parameter values, both with and without trade-offs in the pathogenicity parameters, the host population tends to steady-state values or to extinction when pathogenicity and host performance parameters are set at high levels. There are some small differences in the cycling behaviour when comparing the high pathogenicity and medium host performance combinations with and without the trade-off. The inclusion of variable pathogen-induced mortality alters the expression for the equilibrium density of infectious individuals; however it does not alter the expression for the epidemic development criterion.

4.2. Introduction

Population dynamics has been of interest to ecologists since the inception of the concept of ecology (Liu *et al.*, 2007) including the role pathogens on host population dynamics (e.g. Bailey, 1975, Anderson and May, 1979, Anderson and May, 1981, Thrall and Jarosz, 1994). However, plant pathogens as drivers of ecological change in natural plant populations have only been considered relatively recently (Harper, 1990, Gilbert, 2002) compared to plant-pathogen dynamics in agricultural systems. Natural host-pathogen systems have been studied using a range of theoretical and experimental approaches (e.g. Burdon *et al.*, 1992, Chan and Jeger, 1994, Alexander *et al.*, 1996, Taylor and Rodríguez-Kábana, 1999, Packer and Clay, 2000, Park *et al.*, 2002). Theoretical investigations have generally involved compartmental *SEIR* epidemiological models in

which the dynamics of *Susceptible*, *Exposed or Latently infected, Infectious*, and *Removed* or *Post-infectious* individuals within a population are described (Madden *et al.*, 2008). Plant pathogens have been reported to alter host dynamics due in a variety of ways due to the pathogen acting as castrators, debilitators or killers (Alexander and Holt, 1998) and occasionally promoting plant growth (Burdon, 1987).

In many ecological systems, density-dependence has been shown to alter population dynamics in a range of plant systems (Watkinson and Harper, 1978, Symonides *et al.*, 1986) where density-dependence effects incorporate biotic interactions including competition, spatial limitations and pathogen epidemics. When density is investigated as a factor determining pathogen persistence in experimental systems, pathogen incidence generally increases with host density (Burdon and Chilvers, 1982, Burdon *et al.*, 1992). However, Pfennig (2001) describes phenotypic variation of pathogens in relation to host density which allows for highly transmissible strains with low pathogenicity. A further possibility is that hosts are more resilient to pathogens at higher densities, with lower mortality. This has been demonstrated in a range of insect host and pathogen systems where individuals in higher density environments have a lower per capita death rate due to pathogen-induced mortality (Cotter *et al.*, 2004). An additional example where plant pathogens reduce in mortality is shown from work by Strong (1992) and Alexander (1992). For populations of *Silene alba* infected by the castrating smut *Ustilago violacea* there is evidence that smaller populations have higher levels of infection and in turn, host pathogen-induced mortality, whereas larger host populations show less signs of infection. Additionally, Alexander and Antonovics (1988) and also Alexander (1990) found that in the same host-pathogen system, smaller populations are less likely to become infected than larger populations. From these observations it could be possible that increased host density has an effect in lowering the pathogen-induced mortality rate, whilst potentially causing a trade-off for highly transmissible strains to develop.

This chapter describes three possible models that represent the addition of variable pathogen-induced mortality in the models described in Chapters 2 and 3. The three forms of the model are: variable pathogen-induced mortality acting both between and within season, variable pathogen-induced mortality within season and constant pathogen-induced mortality between season, and variable pathogen-induced mortality between season and constant pathogen-induced mortality within season. All three forms

of the model are presented in this chapter as it is unclear as to where variable pathogen-induced mortality may apply. In this chapter, the model always contains pathogen-induced mortality.

Liu *et al.* (2007) show that density-dependent, pathogen-induced mortality rates are able to promote cyclical population dynamics. In their case they assume pathogen-induced mortality increases with host density. Here an assumption is made that pathogen-induced mortality varies with the size of the infected host population in an inverse, non-linear fashion. By adapting the existing *SIR*-type model in Chapter 3 to represent this relationship, a range of outcomes can be achieved. Comparisons are made of situations in which variable mortality acts both within and between growing seasons and where variable mortality acts within or between seasons. As in Chapter 3, possible trade-offs between pathogen-induced mortality and transmissibility of pathogen strains are investigated.

4.2.1. Density-dependent mortality

Density-dependent pathogen-induced mortality is assumed to be related to the numbers of infectious individuals according to $\beta' = \beta e^{-I_t}$, where β is the maximum value assumed to be constant in Chapter 3 (Figure 59). In examples where there is constant pathogen-induced mortality (β) there is no indication that there is variable pathogen-induced mortality (β`). This form of density-dependent, pathogen-induced mortality is similar to the density-dependent term derived by Macfadyen (1963) which takes the form e^{-aN}, where aN is some mortality rate of the population N (Bellows, 1981). In this chapter a=1 (for simplification) and $N = I_t$. Here pathogen-induced mortality is related only to the density of infectious individuals in that pathogen strains compete exclusively on infected hosts, as opposed to a situation where pathogen-induced mortality could have been related to the density of the entire population..

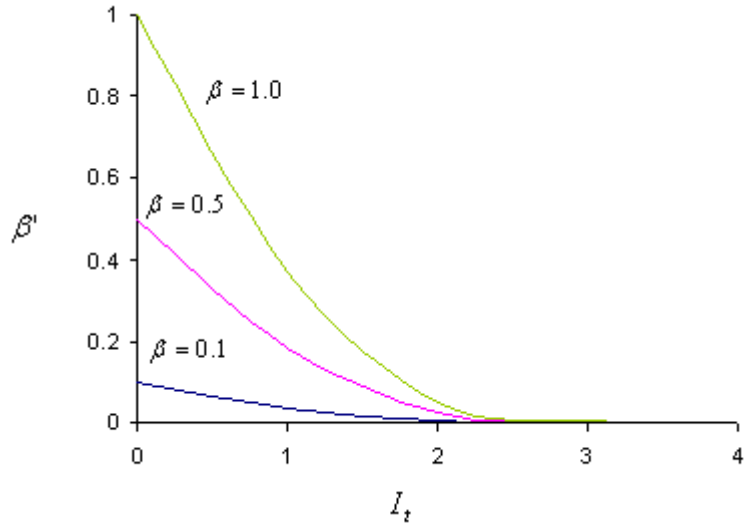


Figure 59. Density-dependent mortality rate (*β*') plotted against the density of infectious individuals (I_t), where the baseline pathogen-induced mortality term is 0.1, 0.5 and 1.0.

4.3. The Models

The models here are developed following the same procedures in Chapter 2 and 3.

The equations that describe the system with variable pathogen-induced mortality between and within season are derived as:

$$S_{t+1} = \frac{a(1-b)R_t}{1+(1-b)\lambda R_t} \quad \textbf{(38)}$$

$$I_{t+1} = (1-d)(1-b)(1-\beta e^{-I_t})pS_t\left\{1-e^{-c(1-b)(1-\beta e^{-I_t})I_t}\right\} \quad \textbf{(39)}$$

$$R_{t+1} = (1-b)(1-d)S_t\left\{1-p[1-e^{-c(1-b)(1-\beta e^{-I_t})I_t}]\right\} \quad \textbf{(40)}$$

Where there is variable pathogen-induced mortality within seasons and constant pathogen-induced mortality between seasons the equations for *I* and *R* are:

$$I_{t+1} = (1-d)(1-b)(1-\beta e^{-I_t})pS_t\left\{1-e^{-c(1-b)(1-\beta)I_t}\right\} \quad \textbf{(41)}$$

$$R_{t+1} = (1-b)(1-d)S_t\left\{1-p[1-e^{-c(1-b)(1-\beta)I_t}]\right\} \quad \textbf{(42)}$$

Note the additional exponent of the exponent in equation 41 compared to equation 39. Likewise the additional exponent of the exponent in equation 40 is abscent in the exponent in equation 42.

Finally where there is variable pathogen-induced mortality between seasons and constant pathogen-induced mortality within season the equations are:

$$I_{t+1} = (1-d)(1-b)(1-\beta)pS_t\left\{1-e^{-c(1-b)(1-\beta e^{-I_t})I_t}\right\} \quad \textbf{(43)}$$

Note that in contrast to previous equations, the moratlity term β exists outside the exponent. Whereas the equation representing R individuals reverts back to equation 40.

The assumptions of these models are the same as in Chapter 3 with the addition that where there is density-dependent pathogen-induced mortality it takes the form $\beta` = \beta e^{-I_t}$ (Figure 59).

4.4. Analysis and Results

The procedures follow the same sequence as in Chapters 2 and 3.

4.4.1. Steady-state expressions

In the absence of pathogen the steady-state equilibria can be defined as

$$\hat{R} = \frac{a(1-b)^2(1-d)-1}{\lambda(1-b)} \quad \textbf{(44)}$$

$$\hat{S} = \frac{a(1-b)^2(1-d)-1}{\lambda(1-b)^2(1-d)} \quad \textbf{(45)}$$

These equilibria are the same for all three models as pathogen-induced mortality only impacts on diseased individuals.

In order to produce steady-state values for *I* individuals when considering the pathogen-induced mortality acting both within and between season is obtained by solving the implicit equation:

$$I^* = \frac{p(1-\beta e^{-I^*})[1-e^{-c(1-b)(1-\beta e^{-I*})I^*}]}{\lambda(1-b)}\left\{a(1-b)^2(1-d) - \frac{1}{1-p[1-e^{-c(1-b)(1-\beta e^{-I^*})I^*}]}\right\} \quad \textbf{(46)}$$

Similarly, steady-state values for *I** for variable pathogen-induced mortality between seasons:

$$I^* = \frac{p(1-\beta e^{-I^*})[1-e^{-c(1-b)I^*}]}{\lambda(1-b)}\left\{a(1-b)^2(1-d) - \frac{1}{1-p[1-e^{-c(1-b)I^*}]}\right\} \quad \textbf{(47)}$$

and for pathogen-induced mortality within season are described as:

$$I^* = \frac{p[1-e^{-c(1-b)(1-\beta e^{-I^*})I^*}]}{\lambda(1-b)}\left\{a(1-b)^2(1-d) - \frac{1}{1-p[1-e^{-c(1-b)(1-\beta e^{-I^*})I^*}]}\right\} \quad \textbf{(48)}$$

4.4.2. Epidemic development criteria

For and epidemic to occur the criterion value must be greater than unity, such that an infectious individual is capable of replacing itself by infecting at least one susceptible individual. In order to derive the criteria for the three forms of the model, the expression $1-e^{-c(1-b)(1-\beta')I_t}$ was approximated by $c(1-b)(1-\beta')I_t$ and similarly $1-\beta e^{-I_t}$ by 1-β, when *I* is infinitesimally small.

For the model with variable pathogen-induced mortality applied both within and between seasons or between or with season the following criterion is obtained:

$$\hat{S}(1-\beta)^2(1-b)^2(1-d)pc > 1 \quad \textbf{(49)}$$

This is the same for each of the model forms, as pathogen-induced mortality occurs both within and between season whether variable or constant.

Using the default parameter values (Chapter 2, Table 4) it is possible to determine the epidemic criterion dependent on varying host density and pathogen parameters (c and p) whilst keeping the baseline mortality rate constant (Figure 60). Figure 61 indicates where altering β alters the region where the epidemic conditions are met.

The manner in which changing one parameter values within the parameter range whilst the remaining parameters stay at default levels is seen in Figure 62. Increasing seedling recruitment increases the number of susceptible individuals. Increasing λ or increasing background mortality rates leads to decreased steady-state S values.

4.4.3. Solving steady-state values for infectious individuals.

Plotting values of I^* against I it is possible to obtain values for steady-state population density of infectious individuals, where they exist, as has been done previously in Chapters 2 and 3.

Steady-state values of infectious individuals decrease with increasing variable pathogen-induced mortality applied within season, between season and both between and within season, only the latter case is shown (Figure 63). Using higher values for β leads to extinction of the pathogen within the population. By comparing the differences between constant β and variable pathogen-induced mortality, the values are the same at corresponding baseline β values for density-dependent, pathogen-induced mortality applied between seasons and where it is applied both between and within season; whilst the steady-state value of I is lower where density-dependent, pathogen-induced mortality is applied within season and constant pathogen-induced mortality is applied between season for corresponding baseline β values such differences are seen in comparing constant both within and between (Figure 50) and variable both within and between (Figure 63).

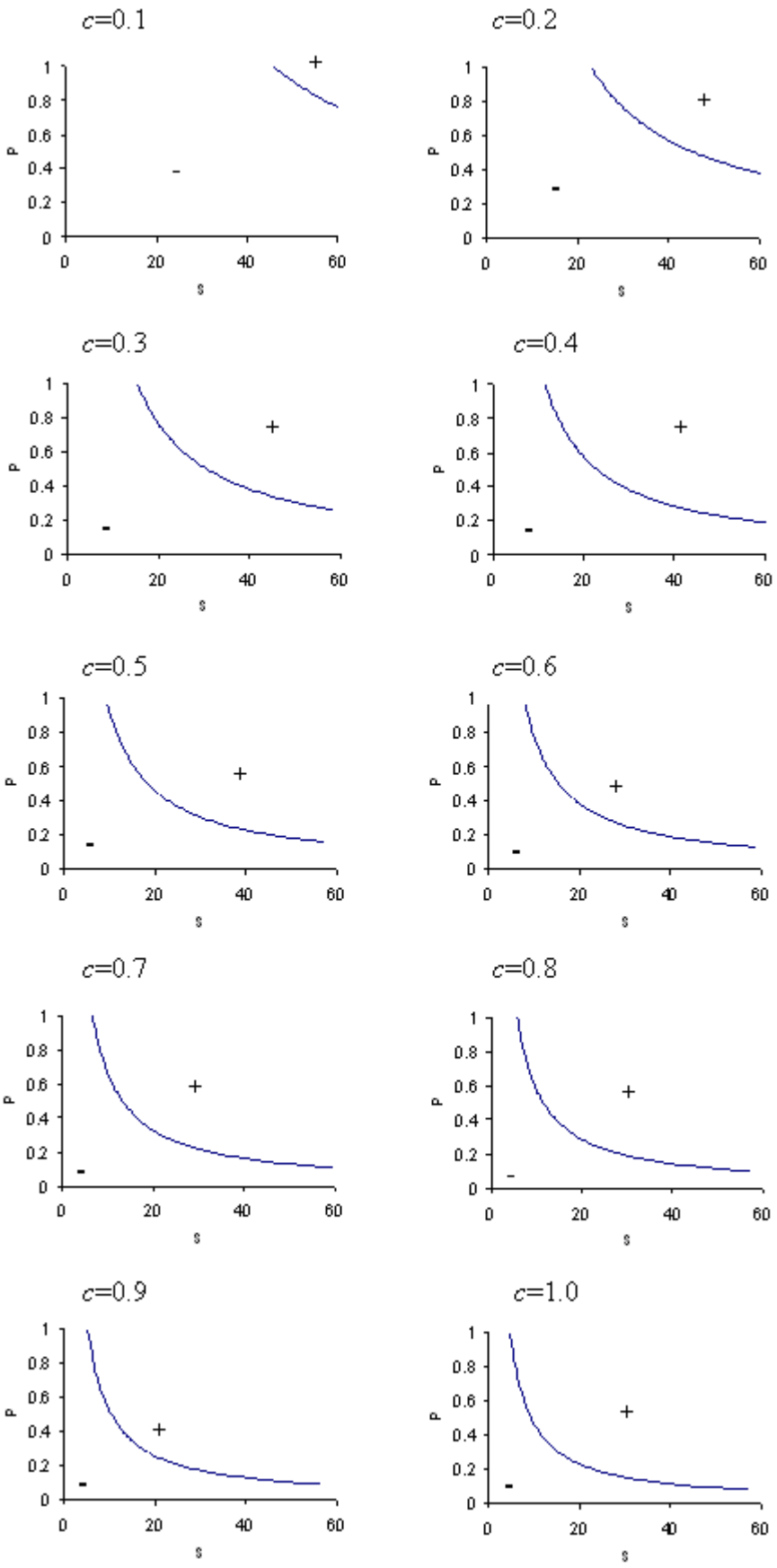


Figure 60. Epidemic development thresholds for varying values of *c* in and *p* relation to varying initial *S* values. Epidemic regions indicated with +, whilst non-epidemic regions indicated with - .

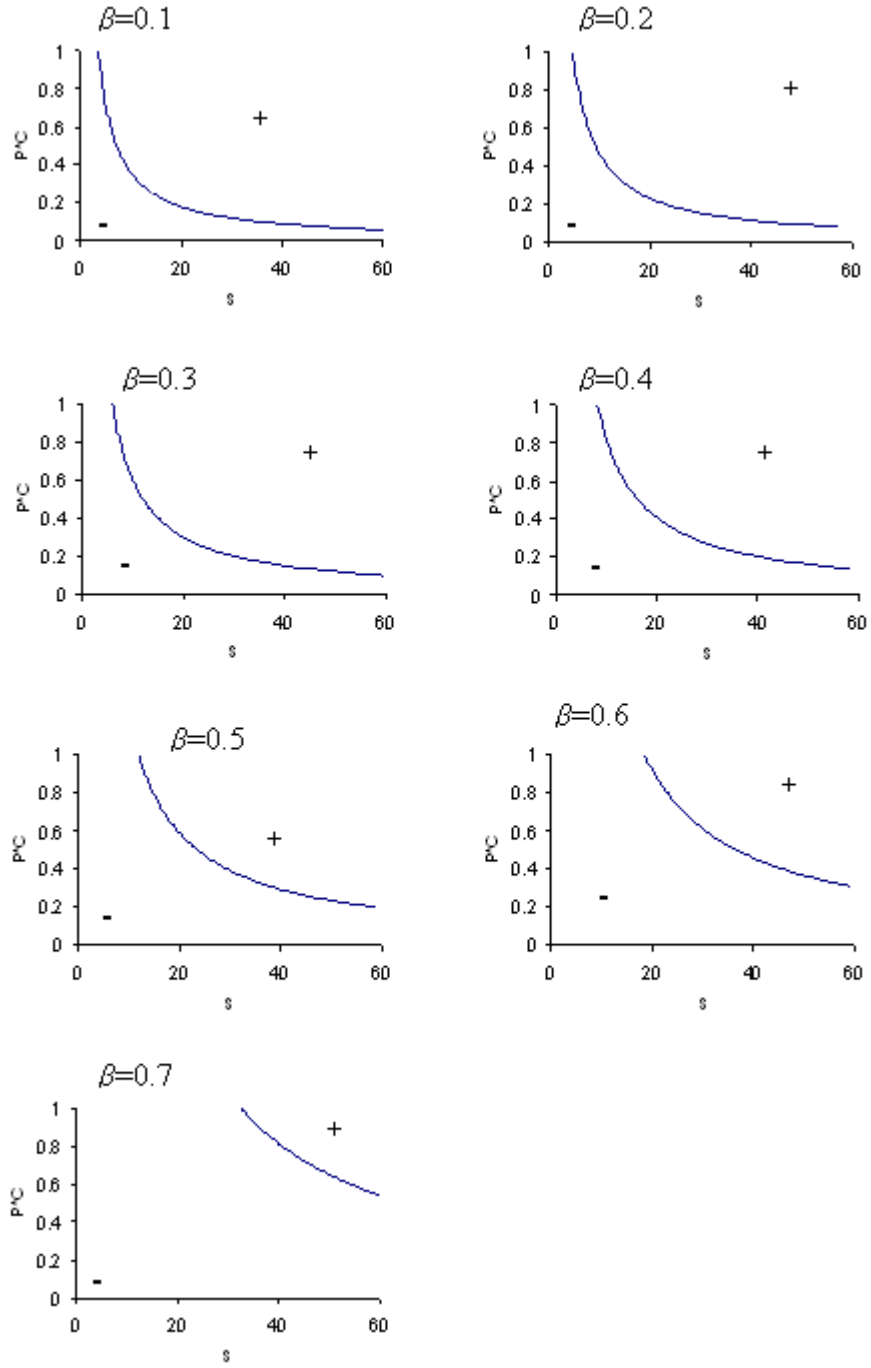


Figure 61. Epidemic development thresholds for varying values of the product of *cp* in relation to varying initial *S* values with increasing values of *β*, for the model with pathogen-induced mortality both within and between seasons. Epidemic regions indicated with +, whilst non-epidemic regions indicated with - .

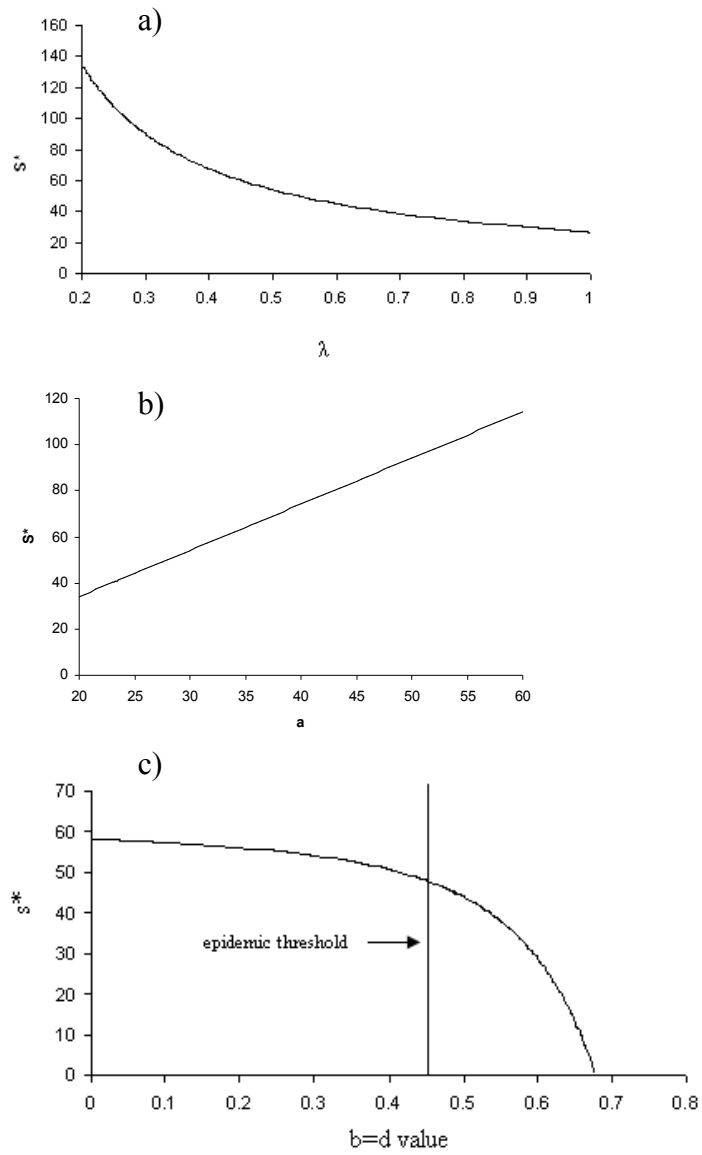


Figure 62. The effect of varying one parameter value within the parameter range for the pathogen-induced mortality within and between season model, whilst the other parameters are set at the default value. a) λ, b) *a* and *c*) the mortality rates *b* and *d* where *b=d*.

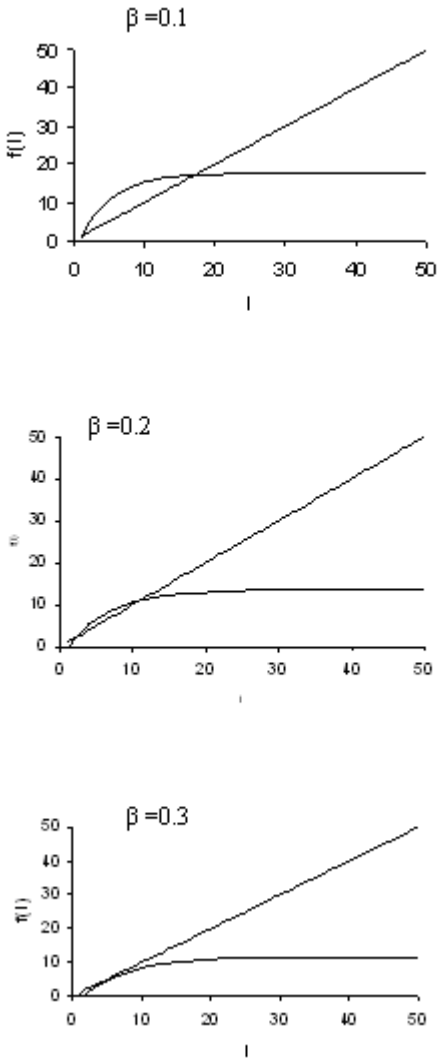


Figure 63. An example of solving stable-state values for *I for variable pathogen-induced mortality between and within season, using default parameters with increasing values of *β* from 0.1-1.0. *I** is defined as the interception between *I* and the f(*I*).**

4.4.4. Population simulations

The parameter values used in Chapter 3 (Table 6) were again used to produce a range of population simulation outcomes using the default values and for simulations where there is a trade-off between pathogen-induced mortality and the transmissibility (Table 7).

There are similarities in the outcomes obtained (Figure 64–66) under the three models. With low host performance parameters, pathogen is not able to invade irrespective of pathogenicity, similarly this is also seen where there is medium host performance and low pathogenicity. At high host performance and high pathogenicity, the population

crashes after an initial outbreak as was observed in Chapter 3. The model outputs also show that *S*, *I*, and *R* individuals tend towards steady-state values when pathogenicity is low and host performance is high, this is also the case when pathogenicity is medium and the host performance is either medium or high. When pathogenicity is low there are more *R* individuals than *I* individuals, whereas with medium pathogenicity, the number of *I* individuals is greater than *R* individuals. Under these conditions variable diseased-induced mortality acting either within season (Figure 65) or between season (Figure 66) leads to two outbreak peaks followed by extinction of pathogen and steady-states for *S* and *R* individuals. However, where variable, there is one outbreak peak followed by *I* extinction and steady-states of *S* and *R* individuals (Figure 64).

The inclusion of a trade-off (Table 7) produces outcomes that are similar to where there is no trade-off, in that three forms of the model produce population crashes at high pathogenicity and high host performance (Figure 67-69). Similarly, low or medium pathogenicity with low host performance, and low pathogenicity with medium host performance lead to no pathogen establishment. As with the model forms without trade-off, the combination of medium pathogenicity and medium or high host performance, and low pathogenicity with high host performance leads to steady-state populations of *S*, *I* and *R* individuals. Again, as with the no trade-off forms of the model, low pathogenicity leads to steady-states, *I* individuals are at lower abundance than *R* individuals and where medium pathogenicity leads to a steady-state, *I* individuals are at higher abundance than *R* individuals.

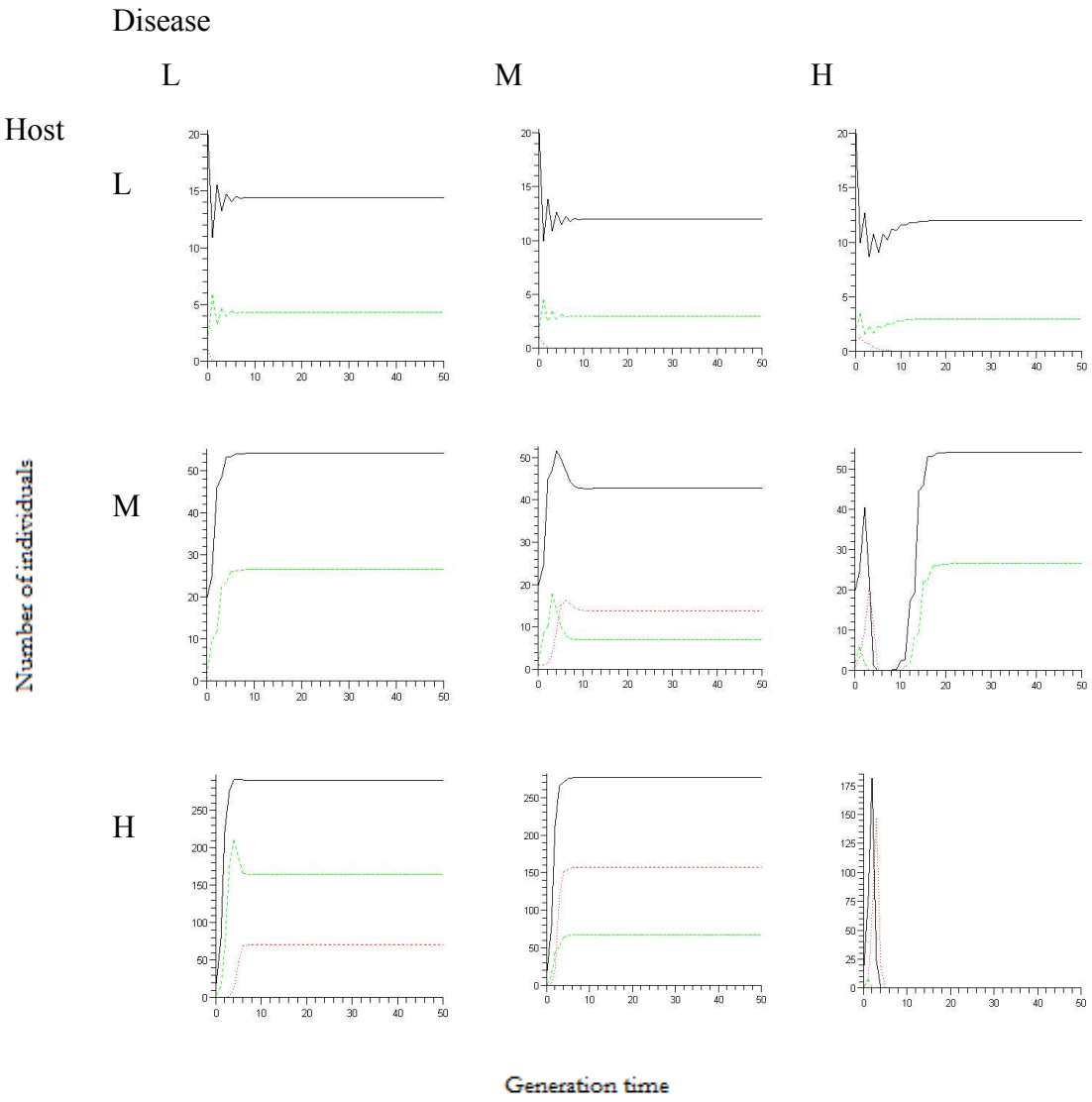


Figure 64. Simulation outcomes for variable pathogen-induced mortality applied between and within season, under high, medium and low host performance and pathogenicity. — *S*, — *I*, — *R*.

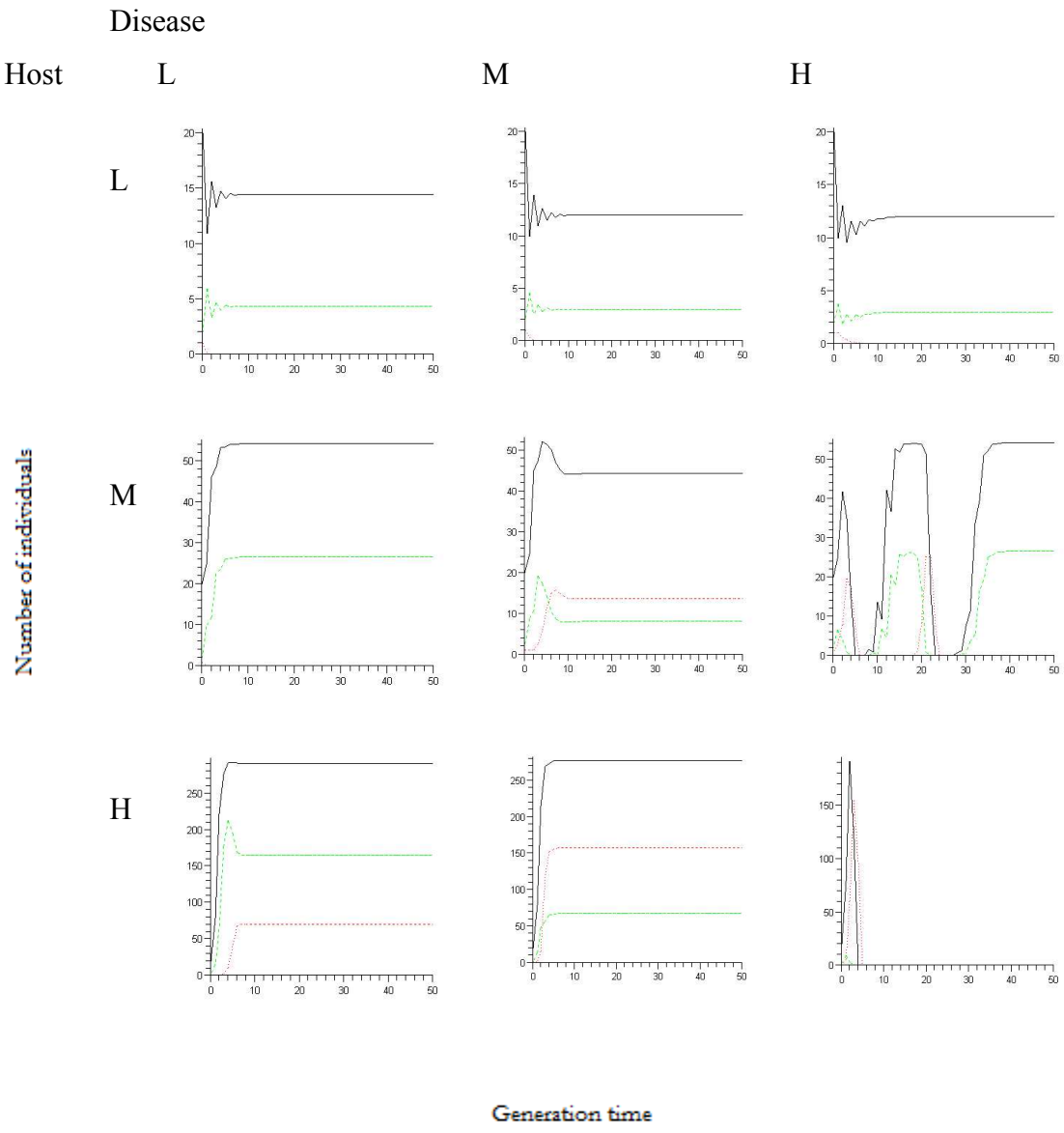


Figure 65. Simulation outcomes for variable pathogen-induced mortality applied within season and constant pathogen-induced mortality acting between season, under high, medium and low host performance and pathogenicity. — *S*, — *I*, — *R*.

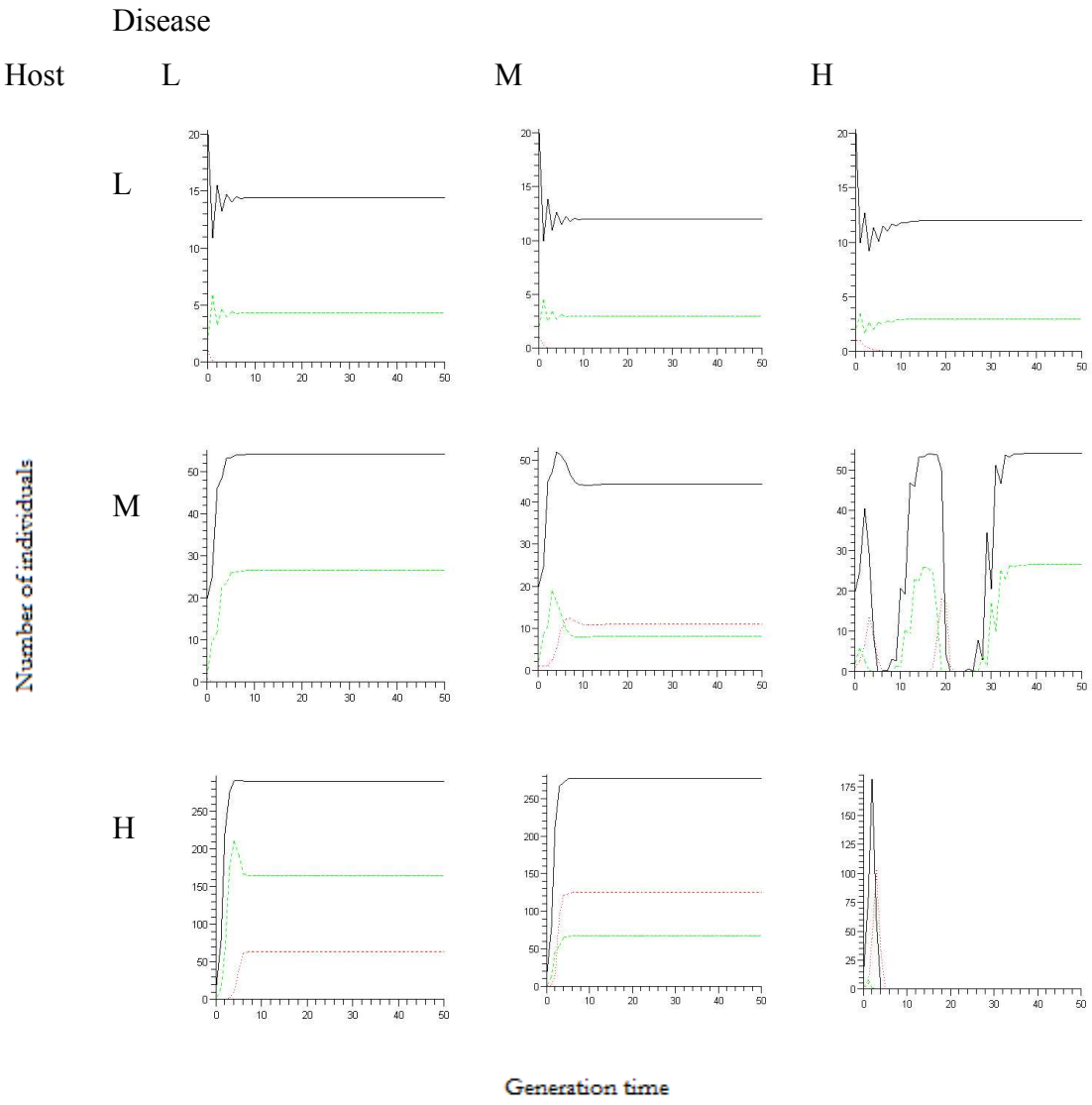


Figure 66. Simulation outcomes for variable pathogen-induced mortality acting between season and constant pathogen-induced mortality acting within season, under conditions of high, medium and low host performance and pathogenicity. — *S*, — *I*, — *R*.

One difference between the trade-off and no trade-off model forms is that all three outcomes have two outbreak peaks followed by infectious individuals becoming extinct for the high pathogenicity – medium host performance combinations. Another difference is that the trade-off forms allow for establishment of low numbers where there is low host performance with high pathogenicity and variable pathogen-induced mortality between season (Figure 69).

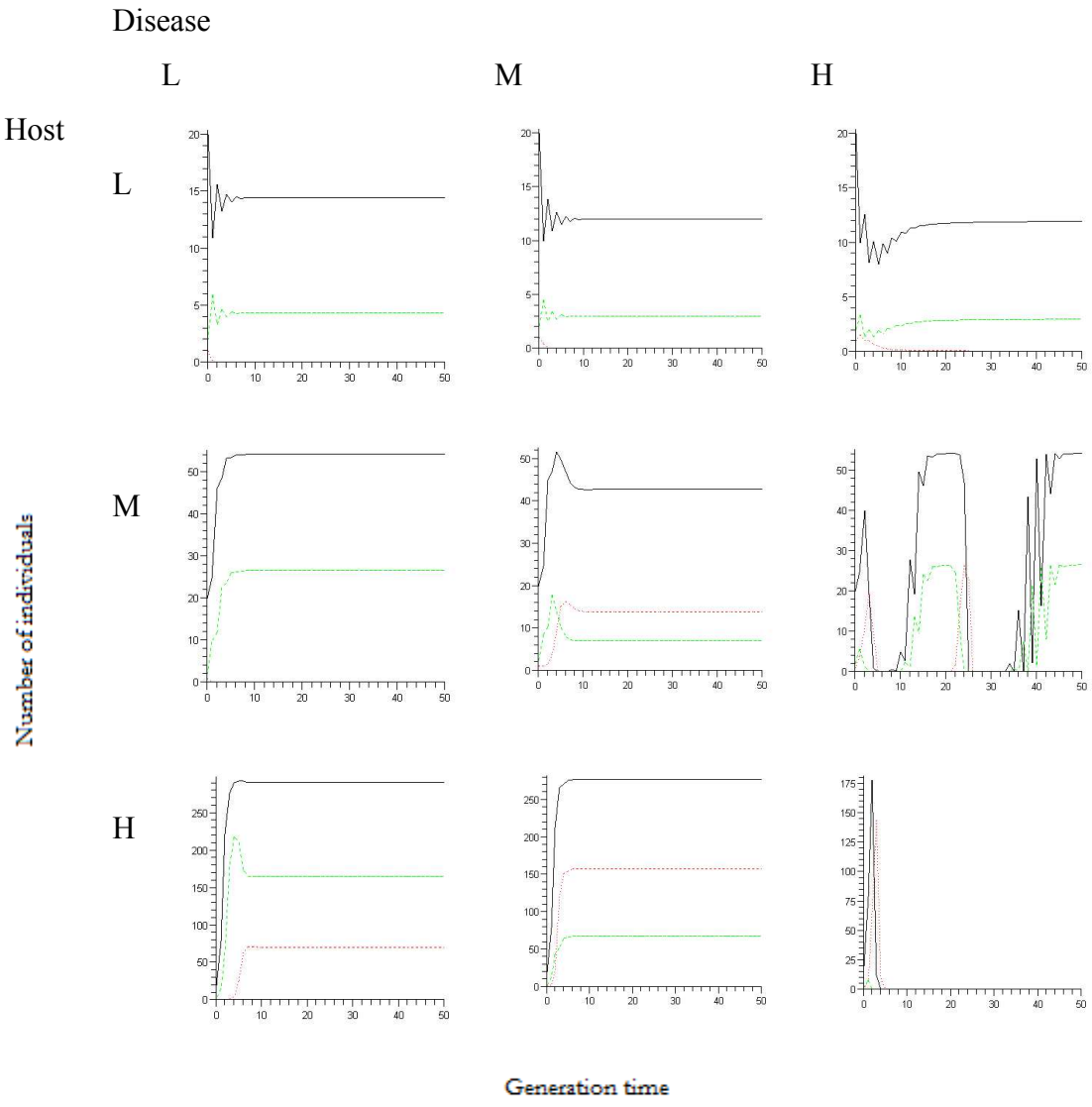


Figure 67. Simulation outcomes for variable pathogen-induced mortality acting between and within season, under high, medium and low host performance and pathogenicity where there is a trade-off in disease life-history. Each simulation represents — *S*, — *I*, — *R* dynamics under the particular parameter values.

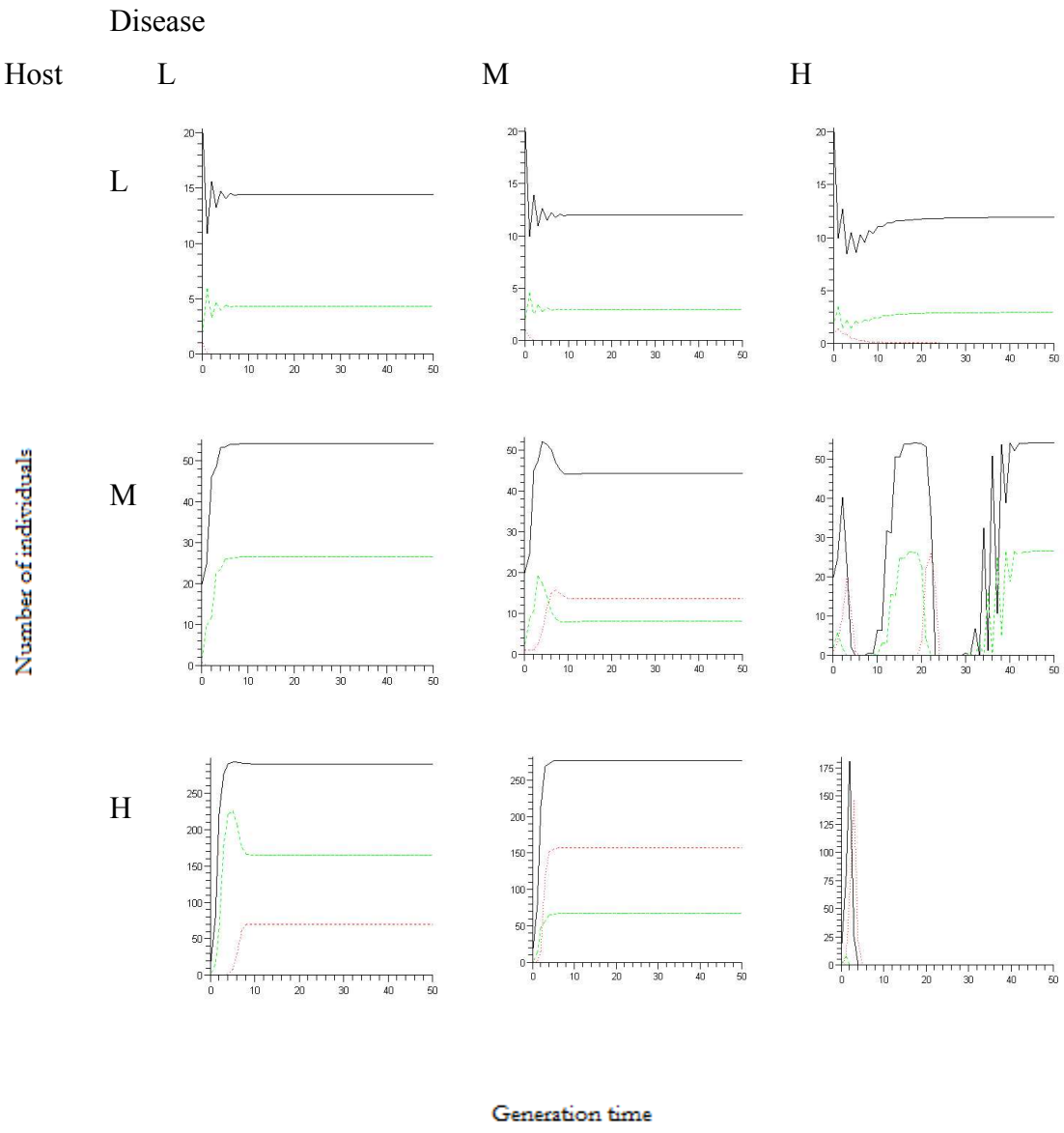


Figure 68. Simulation outcomes for variable pathogen-induced mortality acting within season and constant pathogen-induced mortality acting between season, under high, medium and low host performance and pathogenicity where there is a trade-off between *c* and *β*. Each simulation represents — *S*, — *I*, — *R* dynamics under particular parameter values.

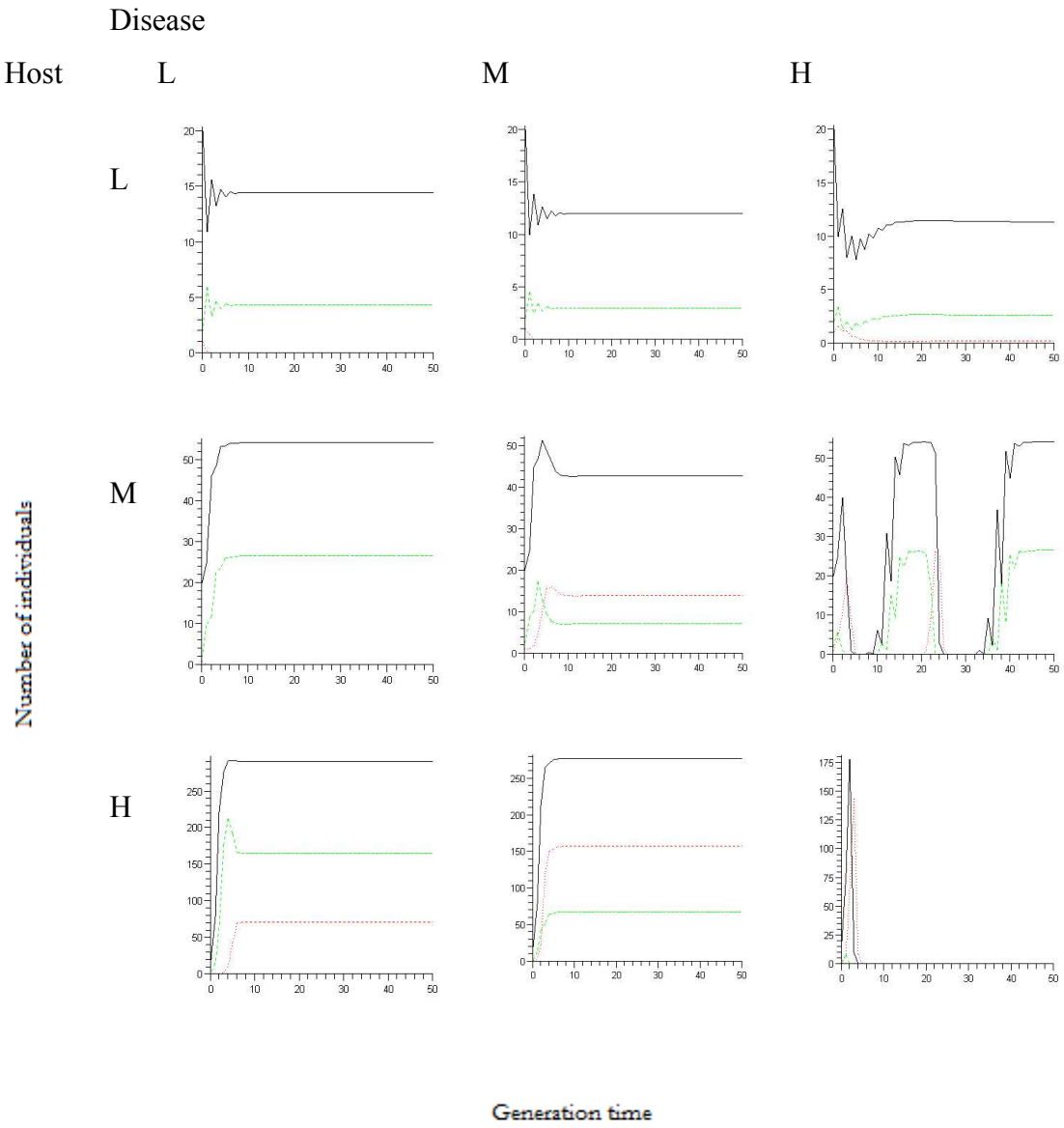


Figure 69. Simulation outcomes for variable pathogen-induced mortality acting between season and constant pathogen-induced mortality acting within season, under conditions of high, medium and low host performance and pathogenicity with a trade-off in pathogen life-history parameters. Each simulation represents — *S*, — *I*, — *R* dynamics under particular parameter values.

4.5. Discussion

The three forms of the models described in this chapter refer to a biennial host plant population which is exposed to a systemic pathogen. The models developed reflect the possibility that pathogen-induced mortality (β) may be density-dependent depending on the size of the infectious population, I, when applied both within and between seasons, or between season or within season. As in Chapter 2 and 3, the model developed is an

SIR-type model in discrete time reflecting the biennial nature of the host and systemic nature of the pathogen, taking into account life history characteristics of the host regarding between and within season background mortality, seedling recruitment and density-dependence. The background host mortality is defined by the constant rates b and d with an additional term $\beta`$, a function of the size of the infected population. This formulation differs from previous work on variable pathogen-induced mortality where the mortality term takes an additive term (Liu et al., 2007) which is of the form $b=b_0+f(N)$ where $f(N)$ is some function relating to the total population density, and b is the total pathogen-induced mortality and b_0 is baseline pathogen-induced mortality (Bellows, 1981). The format of $\beta`$ used here is an extreme form of density, it may be more appropriate to alter the value of $a$ as used in MacFadyen (1963) may lead to less severe variations in the value of $\beta`$, similarly the application of other forms of density-dependence (Bellows, 1981) could have been appropriate.

The simulation results show a range of possible outcomes; the host population increases monotonically, crashes, or there is partial regulation of the host by the pathogen depending on parameter values reflecting high, medium and low host performance and pathogenicity.

The pathogen invasion expression is the same under all three models and the same as that derived for constant pathogen-induced mortality both between and within season this is because it is derived from an initial miniscule amount of pathogen such that $\beta`$ is approximately the same as β.

Where there is variable pathogen-induced mortality, the steady-state values are similar between the three types of the model. However, a noticeable difference is where there is variable mortality applied between and within season causing the steady-state for medium host performance and high pathogenicity to be achieved after one outbreak cycle, but the steady-state value remains the same as where variable mortality is applied either within or between under the same parameter values. An additional difference is that the steady-state of I individuals is lower where variable mortality is applied between season with medium pathogenicity and either medium or high host performance values.

The inclusion of a trade-off in c and β values when using parameters for high pathogenicity and medium host success, allows for two host population cycles with diseased individuals, followed by steady-state populations of S and R individuals irrespective of variable mortality applied within or between or both between and within season.

In summary, the population dynamic simulations tend to steady-state values except where high host performance and high pathogenicity is investigated, which causes crashes. These patterns are also seen where there are trade-offs in pathogen parameter values. The simulations are very similar and correspond to the simulations in earlier chapter where constant pathogen-induced mortality is applied either within or between season, indicating these simulations are possibly due to constant pathogen-induced mortality. This is supported by the observations of where variable pathogen-induced mortality is included both within and between season. The dynamics for high pathogenicity and medium host performance deviate from the majority of simulations, however where there is constant pathogen-induced mortality with medium host performance and medium pathogenicity in earlier chapters, the simulations indicate that there is cycling within the host populations.

5. The impact of a seedbank on the population dynamics of a biennial plant exposed to a systemic plant pathogen.

5.1. Summary

In previous chapters, models were developed to describe plant populations with a single parameter used to denote seedling emergence from the previous generation seed-bearing plants. This chapter aims to extend the discrete time model by incorporating a seedbank, to determine the impact on both plant population dynamics and pathogen epidemiology.

A epidemic development criterion is derived which incorporates parameters representing life-history characteristics of host and pathogen and also incorporates seedbank parameters explicitly, indicating the importance of these in predicting pathogen dynamics. Additionally, steady-state values for the size of the pathogen population indicate that the seedbank parameters are also important factors. Simulation outputs with a range of parameter values tend to produce stable populations. However, high performing hosts with a seedbank exposed to a medium pathogenic strains will give host population cycles. With high pathogenicity and high host performance the host does not become extinct as in the case without a seedbank, but is able to persist due to recruitment from the seedbank. This seedbank recruitment also enables similar dynamics even when mortality within the seedbank is high. Disease is also able to persist when there is high pathogenicity but low host performance, unlike the cases without a seedbank.

5.2. Introduction

Seedbanks have an impact on plant populations by acting as reservoirs of genetic material that transfer across generations so replenishing genes in populations across time (Waples, 2006). For example, the species *Linanthus parryae* has a vast seedbank where seeds remain viable up to six years (Kaj *et al.*, 2001) and *Brassica napus* up to ten years (Pekrun *et al.*, 2005). This in turn can increase population size, reduce changes in genetic variation (Nunney, 2002) and also modify age structure within the population (Evans and Dennehy, 2005). A seedbank allows for the recruitment of individuals irrespective of biotic and abiotic factors which may have affected the previous year's adult stock (Waples, 2006) so potentially altering host population dynamics.

The dormant phase poses problems when attempts are made to predict population dynamics between generations in plant systems. Seeds may remain viable for many years or re-emerge the following season so altering the recruitment of individuals between seasons (Crawley, 1990). There has been a range of theoretical work investigating the role of seedbanks on population dynamics for many plant systems, including monocarpic perennials (e.g. Vidotto *et al.*, 2001) and annuals (e.g. Jarry *et al.*, 1995, Gonzalez-Andujar and Fernandez-Quintanilla, 2004). Models of seedbanks and population dynamics have been predomintly developed as matrix models describing the seedbank age structure (Caswell, 1989). However in this study, the main emphasis is on pathogen dynamics and the impact of plant pathogens in regulating host populations, so the epidemiological model of Chapter 3 is extended to include a seedbank phase.

Tragopogon pratensis (meadow salsify) is a biennial plant that has exhibited dynamics characteristic of an outbreak species (Silvertown *et al.*, 2006) in the Park Grass Experiment, and has been hypothesised to be regulated by the systemic, castrating, rust pathogen *Puccinia hysterium*. The life-cycle of the host is unclear as it is difficult to address the issue of below-ground seedbank structure and survival. However, it has been reported by Haubensak and Smyth (2002) that a seedbank exists for the related species *T. porrifolius* (purple salsify), and there is some evidence that a small seedbank exists for *T. pratensis* (Clements *et al.*, 1999), remaining viable for up to two years (Roberts, 1986). However this seedbank is fragile as anaerobic conditions and temperatures above 25^{o}C cause seeds to become inviable and the seedbank is lost (Roberts, 1986).

This chapter explores the population dynamics of a system with the characteristics of biennial host and a systemic, castrating pathogen which exhibits pathogen-induced mortality where recruitment to the host population is via a seedbank derived from healthy seeding individuals in previous generations. The model is used to simulate population outcomes, derive the steady-state population size in the absence of pathogen and also further derive implicit steady-state population sizes for infected individuals. A epidemic development criterion is derived to describe the conditions required for epidemic to persist.

5.3. The model

The assumptions underlying the model (Figure 70) are the same as the model in Chapter 2 and 3. With the addition of a seedbank, R individuals set seed at the end of the season at a constant rate a_1 with all seeds entering the seedbank with a density-dependence factor relating to the density-dependent term (λ). Within the seedbank there are mortality terms for within season (b_0) and between seasons (d_0) loss of seeds; additionally, the seedbank losses individuals through a seedling recruitment rate (a_0) whereby a proportion of the seedbank become first year plants.

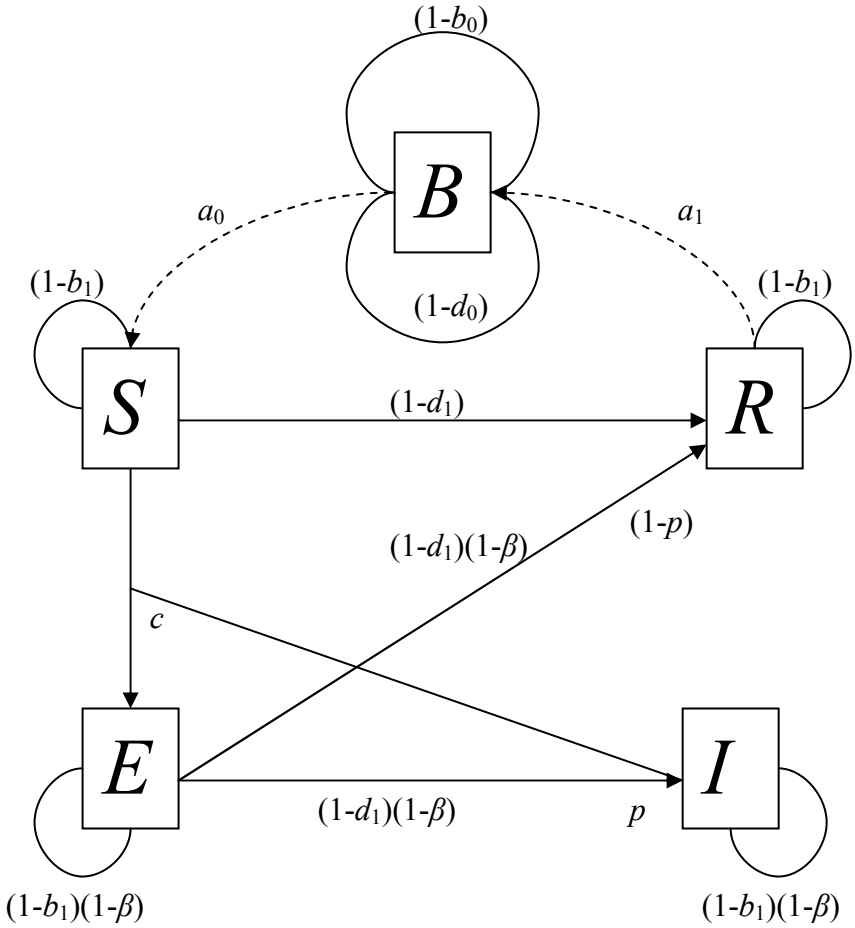


Figure 70. A schematic of the model system with addition of a seedbank phase (B).

Using a similar approach in the model development in chapter 1, the inclusion of a B term results in the substitution of equation 1 with equations 51 – 53. The remaining aspects of the model are the same as 2 – 8 as these terms do not relate directly to the seedbank

$$B_{\alpha,t} = B_{\omega,t-1}\left[(1-d_0)-a_0\right] \quad \textbf{(50)}$$

$$B_{\omega,t} = (1-b_0)B_{\alpha,t} + \frac{a_1 R_{\omega t}}{1+\lambda R_{\omega t-1}} \quad \textbf{(51)}$$

$$S_{\alpha,t} = a_0 B_{\omega,t-1} \quad \textbf{(52)}$$

As described in Chapter 2 for the simplified model, rearranging the equations and re-scaling, a model can be developed to represent the system at the beginning of the season:

$$B_{t+1} = \left[(1-d_0)(1-a_0)\right]\left[(1-b_0)B_t + \frac{a_1(1-b_1)R_t}{1+(1-b_1)\lambda R_t}\right] \quad \textbf{(53)}$$

$$S_{t+1} = a_0\left[(1-b_0)B_t + \frac{a_1(1-b_1)R_t}{1+(1-b_1)\lambda R_t}\right] \quad \textbf{(54)}$$

$$I_{t+1} = (1-b_1)(1-d_1)(1-\beta)\,pS_t\left[1-e^{-c(1-b_1)(1-\beta)I_t}\right] \quad \textbf{(55)}$$

$$R_{t+1} = (1-b_1)(1-d_1)S_t\left\{1-p\left[1-e^{-c(1-b_1)(1-\beta)I_t}\right]\right\} \quad \textbf{(56)}$$

5.4. Analysis

Steady-state population expressions were derived for the system in the absence of pathogen, and steady-state values of *I* derived using an implicit equation. A epidemic development criterion is derived which describes the conditions required for an epidemic to occur. Simulation outputs were produced using MAPLE 10 software (Waterloo Maple Inc. 2005) for parameter values describing high, medium and low pathogenicity and host performance. In addition, as in Chapter 3 and 4 trade-offs in pathogen life-history between transmissibility and pathogen-induced mortality were investigated. Only constant pathogen-induced mortality terms within and between seasons were included in this model.

The parameters and the numerical data for approximating parameter values are the same as used in previous chapters, where a in previous chapters is equal to the product a_0a_1 in this model. The mortality rate d_0 is assumed to be relatively high as described by Clements *et al*. (1999) but minimal within season (b_0).

The initial conditions for the seedbank were set as B_0=0.

5.4.1. Steady-state expressions

In the absence of the pathogen the host population will tend to stable steady-states defined by:

$$\hat{B} = \left(\frac{a_1(1-b_1)(1-d_0)(1-a_0)\hat{R}}{\{1+(1-b_1)\lambda\hat{R}\}\{1-(1-b_1)(1-d_0)(1-a_0)\}} \right) \quad \textbf{(57)}$$

$$\hat{S} = \frac{1}{(1-b_1)^2(1-d_1)\lambda}\left\{\frac{a_0a_1(1-b_1)^2(1-d_1)}{1-(1-d_0)(1-b_0)(1-a_0)} - 1\right\} \quad \textbf{(58)}$$

$$\hat{R} = (1-b_1)(1-d_1)\hat{S} \quad \textbf{(59)}$$

The steady-state equilibrium for the number of infectious individuals is derived by the implicit equation:

$$I^* = (1-b_1)(1-d_1)(1-\beta)p\left[1-e^{-c(1-b_1)(1-\beta)I^*}\right]\left\{\frac{a_0a_1\left\{1-p\left(1-e^{-c(1-b_1)(1-\beta)I^*}\right)\right\}-\left[1-(1-b_0)(1-d_0)(1-a_0)\right]}{\lambda\left\{1-p(1-e^{-c(1-b_1)(1-\beta)I^*}\right\}\left[\left[1-(1-b_0)(1-d_0)(1-a_0)\right]\right]}\right\} \quad \textbf{(60)}$$

This implicit function of I^* is dependent only on the model parameters. Note that for some parameter values the on right hand side of equation 70 can be negative indicating that no steady-state value for pathogen is possible.

5.4.2. Epidemic development criterion

For the diseased to persist the following condition must hold:

$$\frac{pc(1-\beta)^2}{\lambda}\left\{\frac{a_0a_1(1-b_1)^2(1-d_1)}{1-(1-d_0)(1-a_0)(1-b_0)}-1\right\}>1 \qquad \textbf{(61)}$$

Where in deriving equation 70, $1-e^{-c(1-b_1)(1-\beta)I_t}$ is approximated by $c(1-b_1)(1-\beta)I_t$

Thus the epidemic threshold is dependent on all the parameters associated with the model system.

The parameter space in which the epidemic threshold criterion is met is shown in Figure 71 for varying values of pathogen-induced mortality. With the default parameter values of c, p and β it is possible to meet the threshold criteria, indicating that the conditions giving rise to an epidemic are more likely to be met where there is a seedbank (Figure 71) compared to the model without a seedbank (Figure 46).

The epidemic criterion is surpassed using the initial default parameters (Figure 72), indicating that S, I and R individuals will persist within the system. The inclusion of a seedbank allows for steady-states for I^* at low c and p values compared to the model without a seedbank (Figure 37), also at corresponding c and p values, I^* is a higher value.

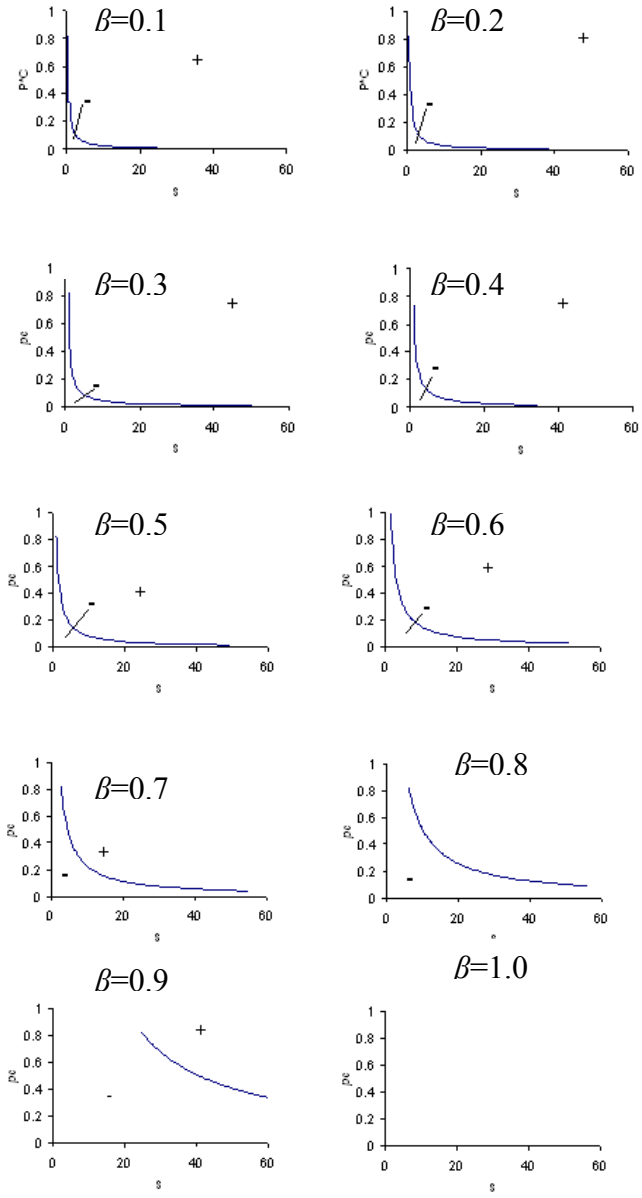


Figure 71. Disease epidemic thresholds for varying values of *cp* and *β* in relation to varying initial *S* values. Epidemic regions indicated with +, whilst non-epidemic regions indicated with - .

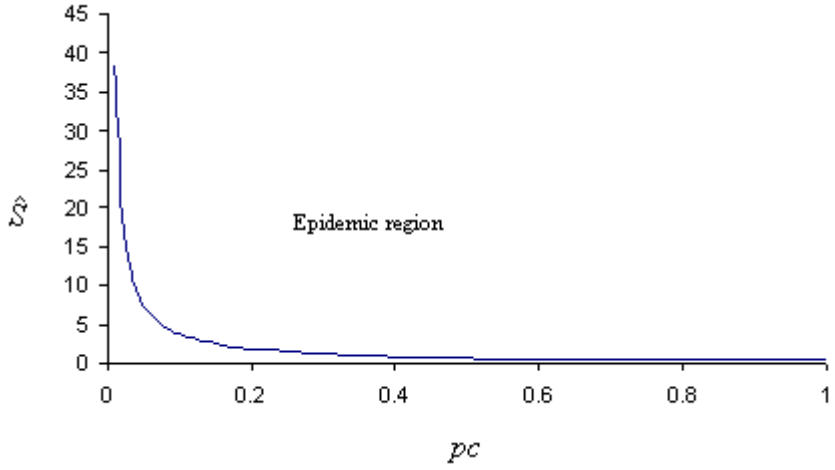


Figure 72. The epidemic region for varying *pc* values and host density.

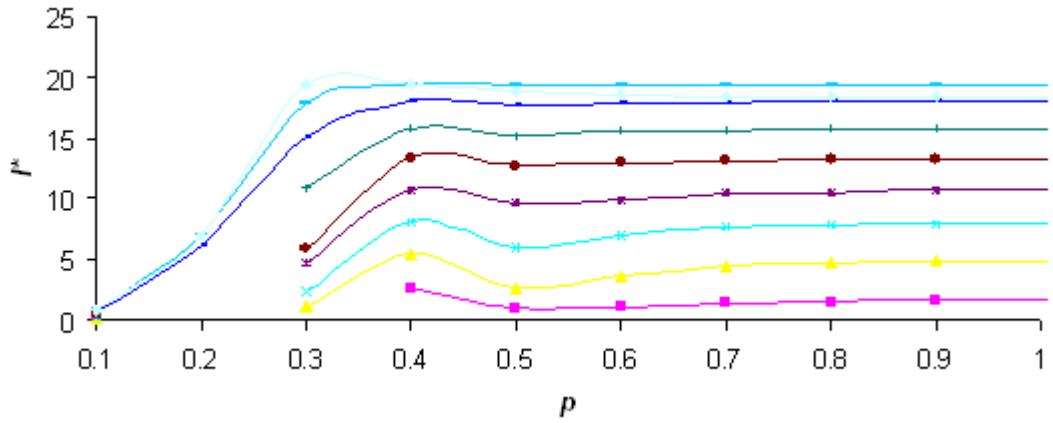


Figure 73. Implicit values of *I with default values and altering *c* and *p***

Steady-state values of infectious second year plants increases with increasing values of both *p* and *c* from estimates of *I** (Figure 73). For low *c* values, *p* is required to be relatively high for a steady-state value of infectious individuals; similarly low values of *p* require higher *c* values. Increased *c* values can be seen to increase the stable-steady-state of infectious second year intervals, where as increasing *p* (apart from at low *c* values) does not lead to a marked increase in *I**. Values of *I** for increasing values of β with the other parameters remaining at default values are shown in Figure 74.

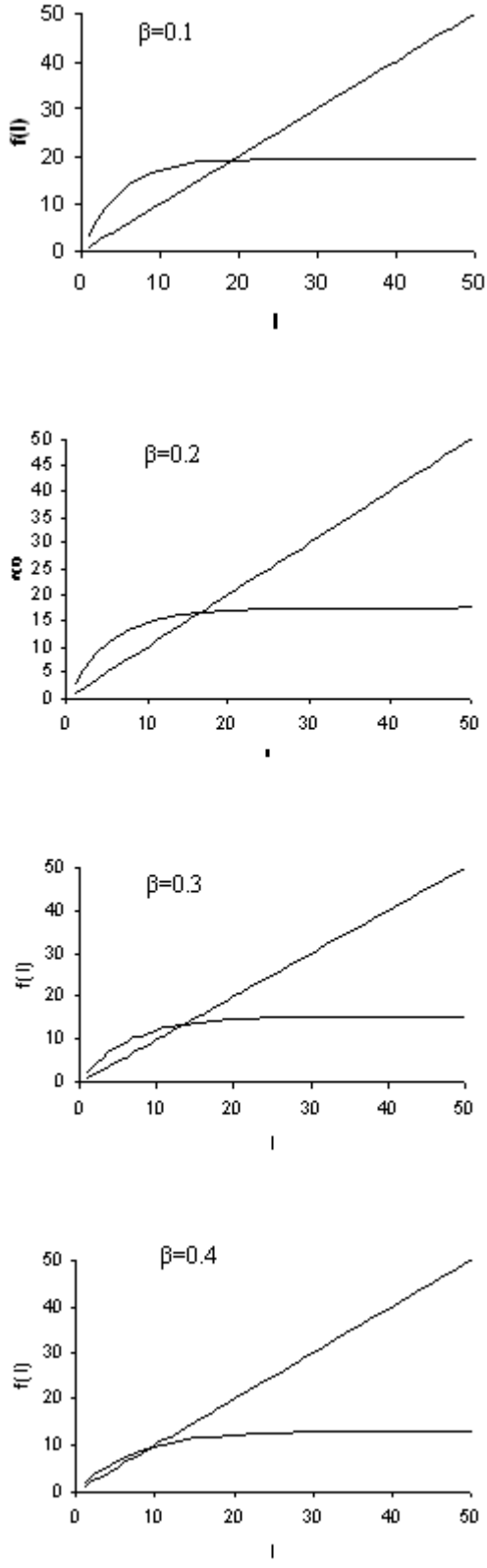


Figure 74. An example of solving stable-state values for *I for default parameters with increasing values of *β* from 0.1-0.4. *I** is defined as the interception between *I* and the f(*I*).**

The dynamics of the S, *I* and *R* individuals within the population were simulated in Maple (Waterloo Maple Inc. 2005). Outputs produced with parameter values representing high, medium and low pathogenicity and host performance (Table 8) are shown in Figure 75. Under the default parameter settings the numbers of *S* and *R* individuals increase to an optimal level, where conditions enable the epidemic development criterion to be fulfilled allowing for the numbers of *I* individuals to increase to a population equilibrium along with the steady-state populations for non-infectious *R* individuals.

Table 8. Parameter ranges for low, medium and high host performance and pathogenicity.

Disease	Low	Medium	High
c	0.1	0.3	0.8
p	0.3	0.7	1.0
β	0.1	0.2	0.3
Host			
a_0	0.2	0.3	0.5
a_1	100	100	120
b_0	0.97	0.7	0.5
b_1	0.4	0.3	0.1
d_0	0.3	0.2	0.1
d_1	0.5	0.3	0.1
λ	1.0	0.5	0.2

Table 9. Parameter ranges for low, medium and high host performance and pathogenicity where there is a trade-off between pathogen-induced mortality and transmissibility.

Disease	Low	Medium	High
c	0.1	0.3	0.8
p	0.3	0.7	1.0
β	0.3	0.2	0.1
Host			
a_0	0.2	0.3	0.5
a_1	100	100	120
b_0	0.97	0.7	0.5
b_1	0.4	0.3	0.1
d_0	0.3	0.2	0.1
d_1	0.5	0.3	0.1
λ	1.0	0.5	0.2

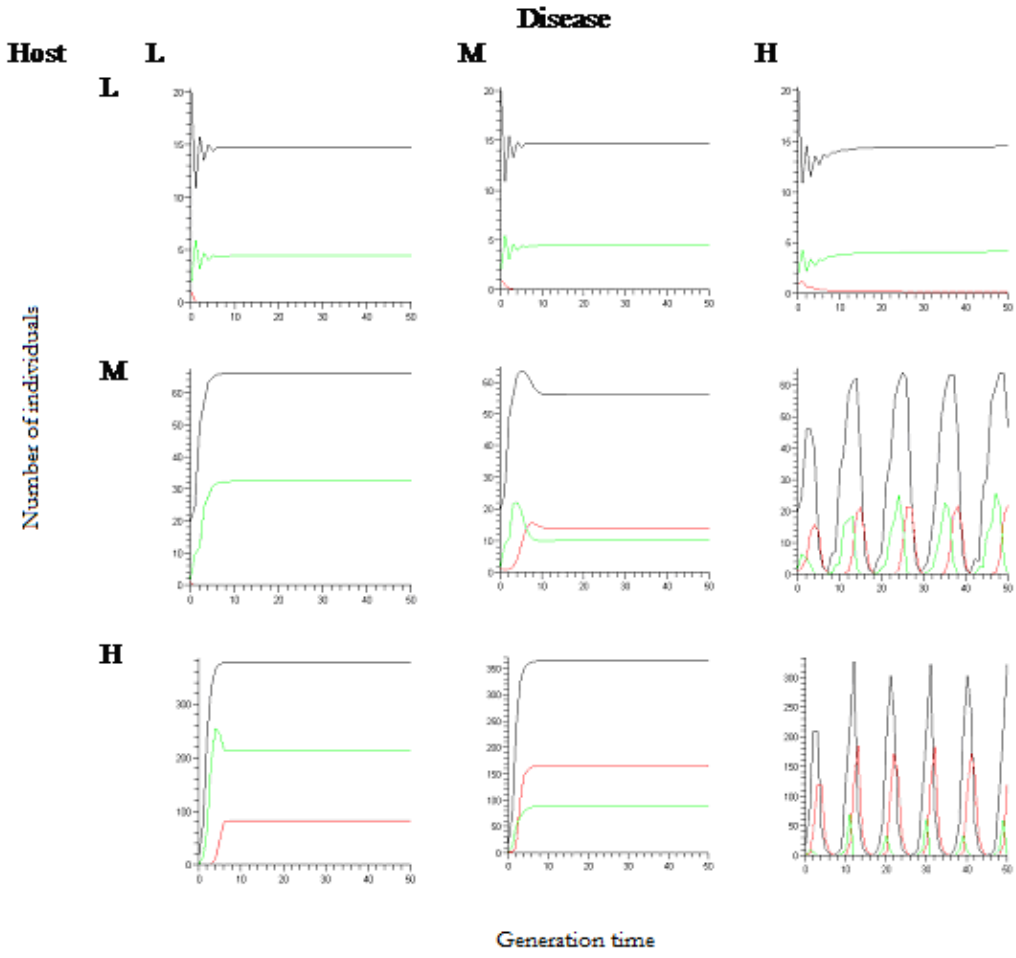


Figure 75. Simulations produced under conditions of high, medium and low host performance and pathogenicity. Each simulation represents — *S*, — *I*, — *R* dynamics.

Steady-state population sizes were achieved for susceptible *S* and healthy second year *R* individuals with low and medium host performance with medium and low pathogenicity and with high host performance when there is low or medium pathogenicity. Population cycles are present when pathogenicity is high and host performance is at medium or high levels (Figure 75) the host population does not extinct under these conditions. Where there is a trade-off between transmissibility and pathogen-induced mortality (Figure 76) these population cycles show outbreaks at a slightly lower frequency. Also at low host performance and high pathogenicity, disease persist (Figure 76).

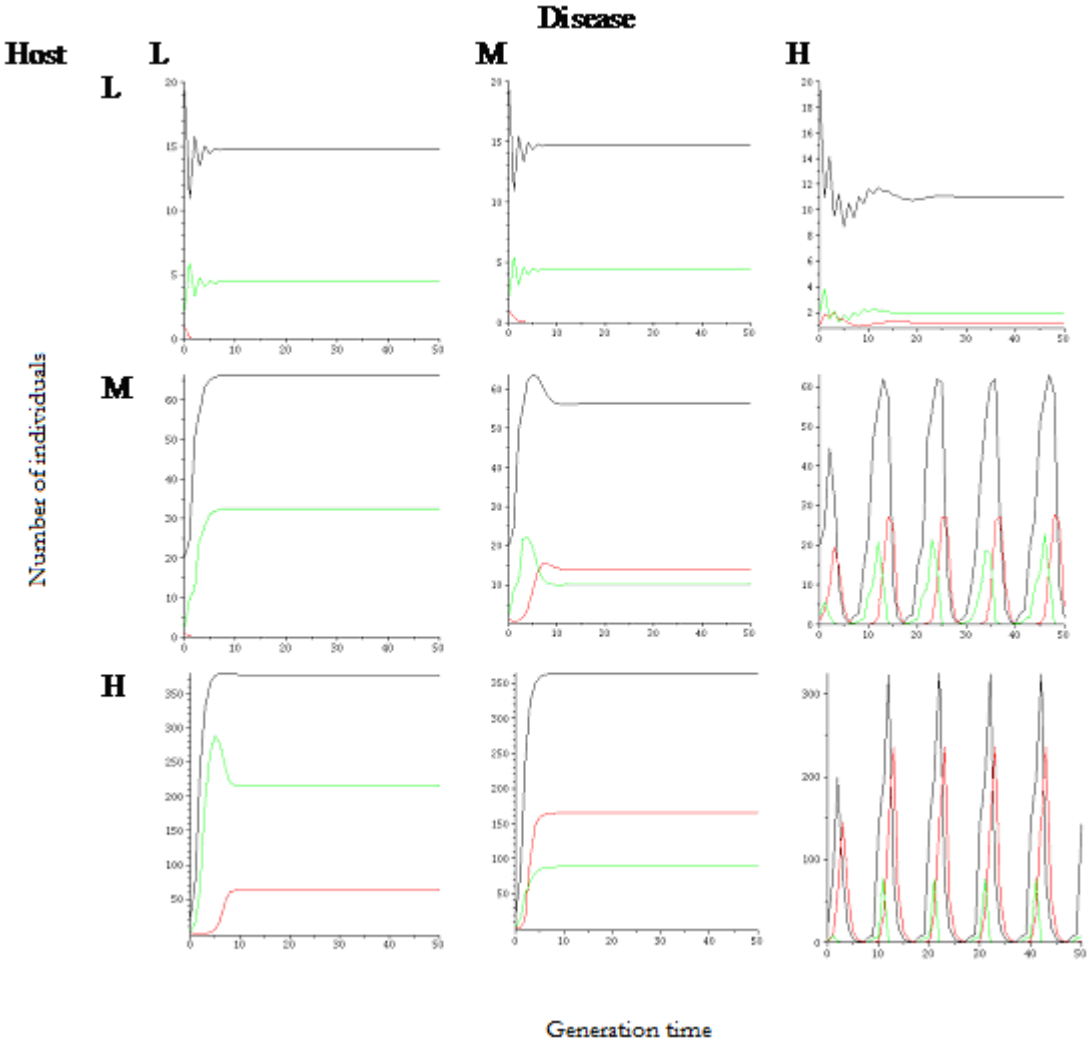


Figure 76. Simulations representing the range of parameter settings indicating, high, medium and low plant performance and pathogenicity where there is a trade-off between transmissibility and pathogen-induced mortality. Each simulation represents — *S*, — *I*, — *R* dynamics.

For a seedbank with low survival, the following simulations demonstrate the impact on the population dynamics for high, medium and low pathogenicity and host performance but with b_0=0.97 being applied to all host performances (Figure 77), and similarly for where there is a trade-off between transmissibility and pathogen-induced mortality (Figure 78). In the former case, repeat population cycles are obtained with the combination of high pathogenicity with a medium or high performance host. In the latter case no such cycles occur. What is distinctive compared to models without a seedbank is the absence of a crash for the high pathogenicity – high host performance combination, lacking any outbreak cycle. Where there is a trade-off in the seedbank model, the repeated cycles are removed and the pathogen eventually crashes.

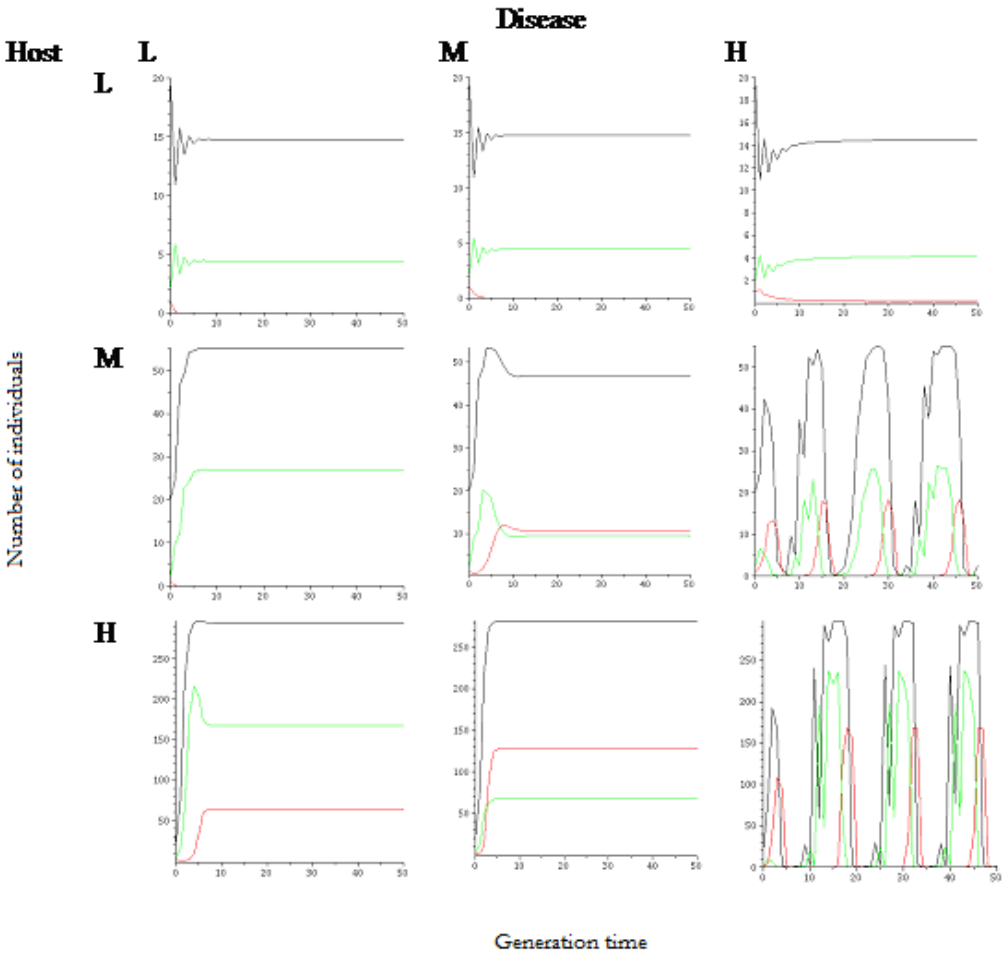


Figure 77. Simulations representing, high, medium and low host performance and pathogenicity where seedbank mortality is high (b_0=0.97). Each simulation represents — *S*, — *I*, — *R* dynamics.

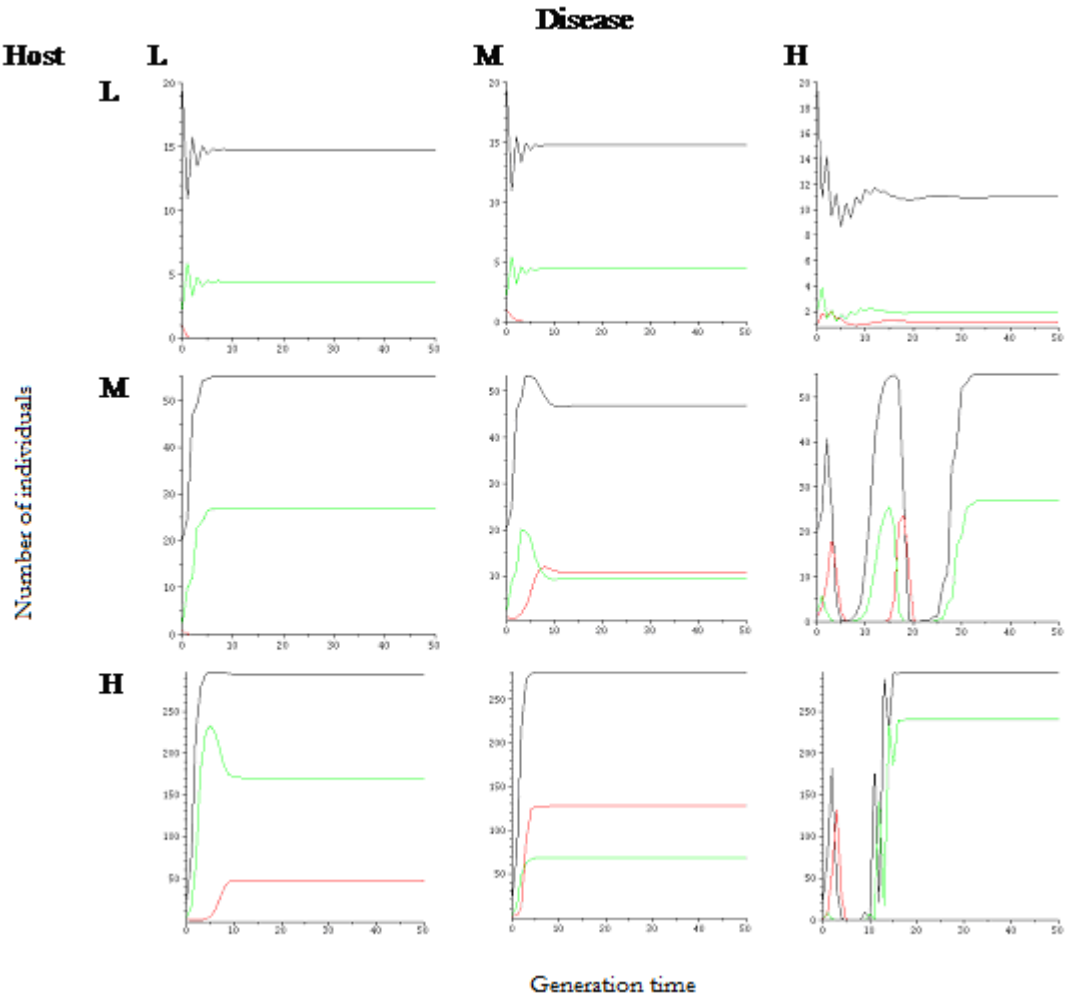


Figure 78. Simulations representing, high, medium and low host performance and pathogenicity where there is a trade-off between transmissibility and pathogen-induced mortality where mortality in the seedbank is high (b_0=0.97). Each simulation represents — *S*, — *I*, — *R* dynamics.

5.4.3. Relationship to previous models

Setting the parameter values such that $a_0a_1=a$ and that there is complete seed mortality within the seedbank (b_0 = 1.0 and d_0=1.0), so that recruitment is direct from seed set from the previous generation it is possible to retrieve a simulation outputs (Figure 79) identical to the outputs (

Figure 55) of the model in Chapter 3 where there is no seedbank with constant pathogen-induced mortality. The parameters a_0 and a_1 are set to the default values of 0.3 and 100 respectively such that a_0a_1=30 (which is the default seedling rate in previous models).

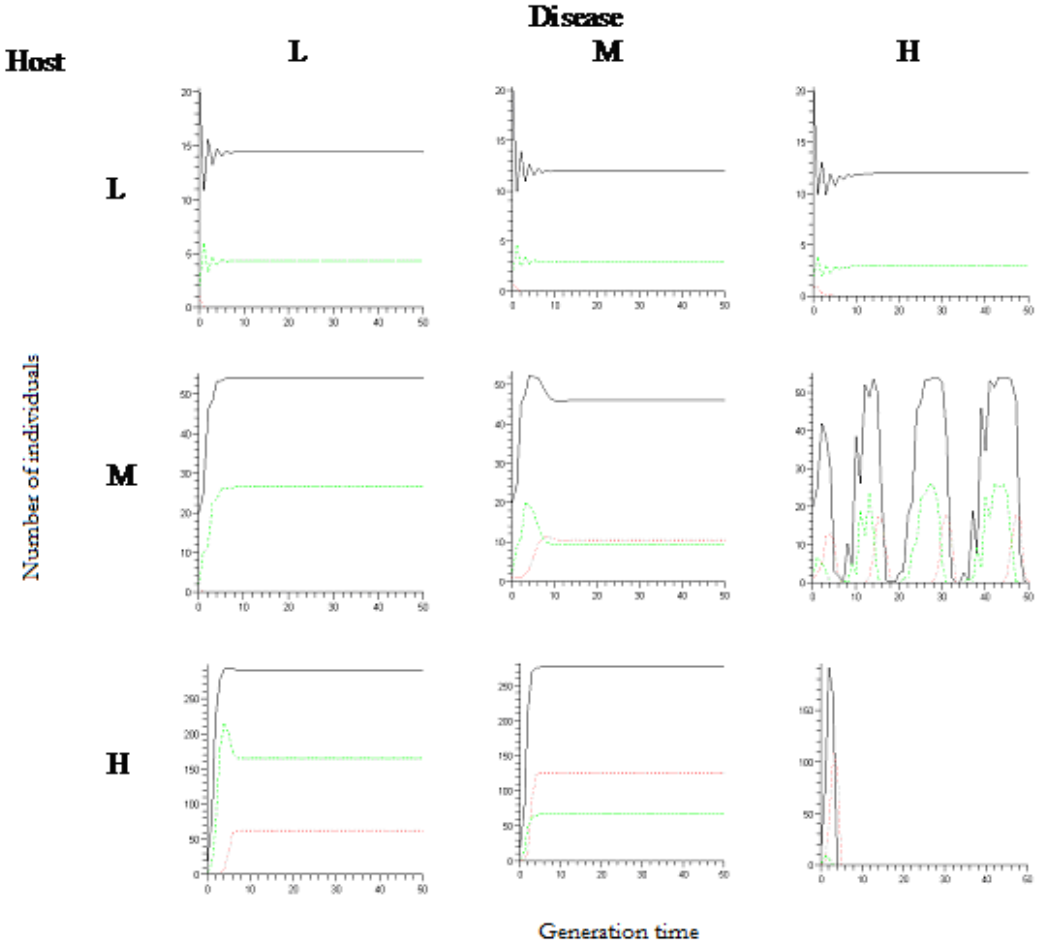


Figure 79. Simulations for the seedbank model where d_0 =1.0 and b_0=1.0, produced under conditions of high, medium and low host performance and pathogenicity. Each simulation represents — S, — I, — R dynamics under particular parameter settings.

The noticeable impact of the seedbank is that it enables the host population to persist where there is high pathogenicity and high host performance. In models without a seedbank (Chapters 2 – 4), under these conditions the host becomes extinct. To some extent the seedbank allows a high level of pathogen to persist where there is high pathogenicity and low host performance.

Therefore where there is complete mortality within the seedbank, the original second order recurrence relationship with pathogen-induced mortality (equation 39) is retrieved with $a = a_0 a_1$.

$$\frac{(1-\beta)}{I_{t+2} + (1-\beta)R_{t+2}} = \frac{\lambda}{a_0 a_1 (1-b_1)^2 (1-d_1)} + \frac{1}{a_0 a_1 (1-b_1)^2 (1-d_1) R_t} \qquad \textbf{(62)}$$

5.5. Discussion

The model presented here describes the population dynamics of a biennial host plant population with a seedbank which is infected by a systemic, castrating pathogen which causes pathogen-induced mortality.

Much of the previous work on the population dynamics of plant species has either ignored a seedbank stage by simply including a parameter to describe the probability of a seed germinating the following year (e.g. Crawley and May, 1987, Damgaard, 2004) or describing the seedbank in an age-structured matrix model (e.g. Jarry *et al.*, 1995). However, if the seedbank is short lived as in *T. pratensis* it is possible to disregard any such age-structure within the seedbank. As the model was used as a tool to study a host-pathogen system, these more complex approaches were disregarded and a seedbank phase was introduced directly into the epidemiological model. The importance of such extension to the *SIR* type model is indicated by the prevention of host population crashes. Even where seedbank mortality is high, inclusion of a seedbank can alter the qualitative outcomes seen for some combinations of pathogenicity and host performance characteristics.

As with the previous models without a seedbanks, this model highlights that under default conditions the host population tends to a stable state which includes diseased plants. It has previously been reported that recorded plant populations tend towards stability (e.g. Rees and Crawley, 1989, Freckleton and Watkinson, 2002). Similarly to the work of Bauer *et al*. (2002), it is possible to produce host population cycles. The simulations show that population cycles are possible for a biennial host in the presence of a castrating, systemic pathogen. The addition of a seedbank allows for outputs that differ from models without seedbanks, as *I* individuals are able to persist in the host population where there is high pathogenicity and high host performance, and also where both there is low host performance and high pathogenicity. This differs from previous models where pathogen does not establish at high pathogenicity and low host performance, and where the host and pathogen go extinct at high parameter values. Where a plant population sets seed which form a seedbank, under no pathogen conditions does the host plant population become extinct. This demonstrates that plants producing a seedbank benefit not only the host population by avoiding extinctions, but

also benefit the pathogen by providing additional situations for pathogen invasion where the host is of low or high performance irrespective of pathogenicity.

Terms from the seedbank are included in the expressions for the steady-state expressions of all host categories of the system demonstrating that the seedbank has an impact on the size of the host population. Altering the parameter values for mortality within the seedbank and recruitment from the seedbank will consequentially alter the host population size and pathogen steady-state value. Similarly, seedbank parameters are included in the epidemic development criterion, indicating that these parameters are important in determining epidemic conditions.

Where there is a trade-off in pathogen parameters there is little difference to the model form without a trade-off; the outputs are consistent including the cycles at medium and high success host populations. However, the cycles are less frequent with a trade-off. This suggests that the dynamics of the system are less driven by the pathogen parameters but by the seedbank parameters.

When applying complete mortality in the seedbank, the model resorts to being identical to the model in Chapter 3 indicating that this model is simply an expansion on the earlier models and is comparable. Therefore, should a seedbank fail then a host population could become extinct when pathogenicity is high. This could suggest that there may be some selection for pathogen to be of medium pathogenicity as a fragile seedbank could fail resulting in the extinction of hosts which in turn removes pathogen. Outputs for simulations with high pathogenicity and medium or high host performance with low seedbank survival show significant differences between models with pathogen parameter trade-offs; steady-state host populations are produced without pathogen persistence where there is a trade-off, whilst repeating population cycles including diseased plants occur without a trade-off.

6. Relating the PGE data to the model

6.1. Introduction

Tragopogon pratensis has been recorded in the Park Grass Experiment as part of the long-term study in the impact of fertiliser treatment on plant community structure (Williams, 1978). However, there have been few observations on the biology of *T. pratensis* and its relationship with the autoecious, demicyclic rust fungus *Puccinia hysterium*. It has been stated previously that in years where *T. pratensis* is of high abundance, there is a high incidence of infection with *P. hysterium* (Silvertown *et al.*, 2006) and that host density is negatively correlated with rust incidence the previous year. A review of the associated literature concerning *T. pratensis* and *P. hysterium* is presented in Chapter 1.

In Chapters 2 – 5 a range of epidemiological models have been developed to represent a biennial host plant infected with a systemic, castrating pathogen. The models are generic but in many respects mirror the life-histories of *T. pratensis* and *P. hysterium*. In this chapter unpublished data collected by G. Edwards between 1995 – 1998 and 2002 – 2004 and by the author between 2005 – 2008. These data was used to assess fitness of infected plants and by inoculating cohorts of plants to estimate parameters values for the models developed in Chapter 2 – 5. Finally, in this Chapter the developed models are related to the PGE observations.

The recurrence relations derived in Chapter 2 predicts a relationship between healthy second year plants at time t and the number of healthy and infected individuals at time t+2 according to:

$$\frac{1}{I_{t+2}+R_{t+2}} = \frac{1}{a(1-b)^2(1-d)R_t} + \frac{\lambda}{a(1-b)^2(1-d)} \qquad \textbf{(63)}$$

With this relationship it is possible to plot the PGE data based upon the surveys of G. Edwards (*unpublished data*) as well as the data collected between 2005-2008 to observe if this theoretical relationship holds in the field using linear regression where there are two years between observations.

Similarly in Chapter 3 a recurrence relationship was derived for the models where there is pathogen-induced mortality between and within season or simply where pathogen-induced mortality is between season. This recurrence relationship takes the form:

$$\frac{(1-\beta)}{I_{t+2}+(1-\beta)R_{t+2}} = \frac{1}{a(1-b)^2(1-d)R_t} + \frac{\lambda}{a(1-b)^2(1-d)} \qquad \textbf{(64)}$$

There was a significant ($F_{(1,9)}$=10.745**) relationship between the numbers of healthy flowering plants and the numbers of flowering plants (infected and healthy) two years later (Figure 80). Although there is much scatter in the recorded data (R^2=0.54), overall the linear relationship indicates that the model can be used to predict the relationship between non-infectious individuals observed in one year and the total number of second year plants two years later and that there is a biological basis to linking the theoretical model with the observed data. In the basic model without pathogen-induced mortality the intercept $\frac{\lambda}{a(1-b)(1-d)}$ = 0.024 and gradient $\frac{1}{a(1-b)^2(1-d)}$ = 0.86. The gradient is less than 1 as required as for the host population to persist it is necessary that $a(1-b)^2(1-d)>1$ as demonstrated in Chapter 2.

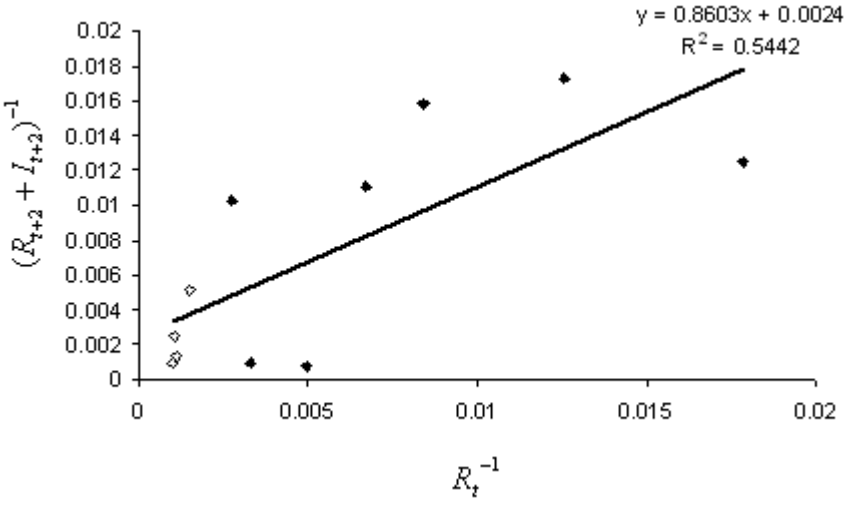


Figure 80. The recurrence relationship fitted to observed data with the interpolated points (◇) for 1999 and 2000 indicating a linear relationship.

Using the recurrence relationship (equation 39, Chapter 3) derived from the model forms with pathogen-induced mortality acting between and within season or only between season, the greater the value of β the better the fit to observed data (Table 10).

Table 10. Summary of regression analysis of the recurrence relationship derived from the pathogen-induced mortality between and within season or only between season model forms with varying β values. (AIC: Akaike's information criterion, *m*: gradient, **: 0.01>p>0.001, ***:p<0.001)

β	0.01	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	0.99
R^2	0.55	0.56	0.58	0.59	0.61	0.62	0.64	0.66	0.68	0.71	0.74
AIC	-56.18	-56.27	-56.38	-56.49	-56.61	-56.74	-56.87	-57.03	-57.21	-57.48	-58.09
m	0.86	0.86	0.87	0.87	0.88	0.89	0.90	0.91	0.93	0.95	0.94
$F_{(1,9)}$	10.81 **	11.43 **	12.19 **	13.03 **	13.96 **	14.96 **	16.04 **	17.24 **	18.77 **	21.73 ***	25.22 ***

As the level of pathogen-induced mortality increases, the better the fit of the recurrence-relationship to the observed data. It is possible that mortality of *T. pratensis* infected by *P. hysterium*, either between season or both between and within season is a significant factor in regulating population dynamics. However, as yet there is no direct evidence for *P. hysterium* causing pathogen-induced mortality within infected *T. pratensis*.

Using the coefficients established in the recurrence relationships and the approximate parameter values in the model, it is possible to estimate $\lambda(1\text{-}b)$. Given there is a low natural death rate of plants within-season (b), and also used in the simulations, Estimates of λ (Table 11) for parameter representing high, medium and low host performance shows that where there is low host mortality, the λ value is high indicating that this system is not under density-dependent regulation.

Table 11. Estimates of λ using parameters representing low, medium and high host performance.

	Low	Medium	High
λ estimate	0.09	0.25	1.05

6.1.1.1. Relationship between a simplified model and the recorded data

Simulation outputs which reflect the outbreak type dynamics with annual fluctuations seen in the PGE result from the basic model (Chapter 2) when the density-dependent impact on seedling recruitment is omitted, such omission is justified by the estimates for $\lambda(1\text{-}b)$. The equations are:

$$S_{t+1} = a(1-b)R_t \quad \textbf{(65)}$$

$$I_{t+1} = (1-b)(1-d)pS_t[1-e^{-c(1-b)I_t}] \quad \textbf{(66)}$$

$$R_{t+1} = (1-b)(1-d)S_t\left[1-p[1-e^{-c(1-b)I_t}]\right] \quad \textbf{(67)}$$

Where there is a low host performance it is possible to produce the outbreak dynamics with medium and high pathogenicity; with other combinations of performance and pathogenicity the host numbers increase without bound or lead to the host and pathogen populations both crashing.

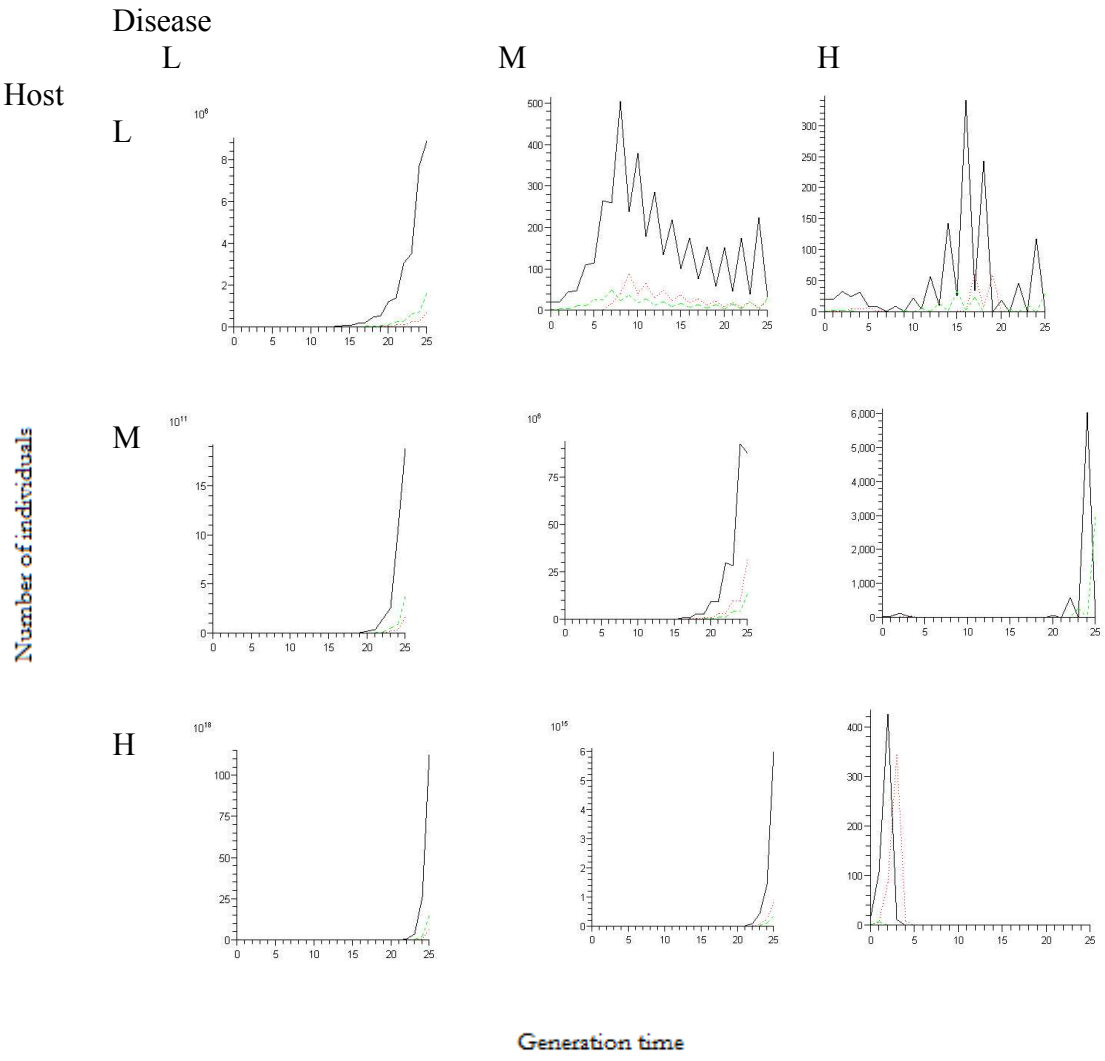


Figure 81. The range of model simulated populations using the basic model where there are no density-dependence factors on the seedling recruitment. — *S*, — *I*, — *R*

6.2. Discussion

6.2.1. Density-dependence

It could be possible that the system is under conditions which do not involve density-dependent factors. From recorded data, *T. pratensis* is at low densities within the PGE, so it is unlikely that the population will be influenced by such factors as seedling recruitment regulated by density-dependency. In the model simulations without density-dependence there is some resemblance between low performance host populations exposed to high and medium pathogenicity rust and the outbreak records observed between 1995 – 2008 (Figure 81). Further support for this interpretation is the low counts of *T. pratensis* throughout the survey. The models in Chapters 2 – 5 show a delayed tracking by the number of infectious individuals to the healthy second-years suggesting that the pathogen has a regulatory impact on host population size, just as is recorded in the Park Grass Experiment.

The good-fit in the recurrence relationship between number of healthy individuals and the total number of second year individuals two years later show that the model can be related to the observational data. The value of the gradient being below the required value of 1, and the high values of λ (e.g. 1.05 under high host performance parameters) with low host mortality terms support the view that density-dependence is not involved in the *T. pratensis – P. hysterium* system.

Ideally it would have been preferable to use an alternate dataset to validate the model, however no such database containing counts of *T. pratensis* and *P.hysterium* exist. The National Biodiversity Network database (http://data.nbn.org.uk/) simply contains presence/absence data for the host with few recordings of the rust. It could have been possible to validate the model using a dataset of a similar species from the PGE, however as stated previously, only plant and not pathogen scores are recorded.

6.2.2. Conclusion

Characteristics of the *T. pratensis – P. hysterium* system within the PGE indicate that over the period of the study there have been fluctuations in the population dynamics. These dynamics can be attributed to the effective castration of flowering individuals by the rust causing a marked reduction in seed production and viability so altering recruitment and therefore the reproductive capacity the following season. Furthermore, the theoretical models indicate that regulation of host plant population dynamics is observed when the pathogen causes additional pathogen-induced mortality. Whether this occurs in the PGE requires further investigation. Applying the recurrence relationships derived from the theoretical models demonstrates the value of epidemiological models to describe a biennial host plant and pathogen system with the characteristics of the *T. pratensis – P. hysterium* system.

7. General Discussion

The principle aim of the research described in this thesis was to develop a generic theoretical model that could be used to examine the hypothesis that the population dynamics of a biennial host plant can be regulated by a systemic, castrating pathogen. The secondary aim was to use the model to examine whether *Tragopogon pratensis*, described as an outbreak species in the Park Grass Experiment, is regulated by the autoecious, demicyclic rust pathogen *Puccinia hysterium* (Silvertown *et al.*, 2006). The model was developed in a general discrete time epidemiological framework which described the relationship between a biennial host and a biotrophic, obligate pathogen. The relationship of host and pathogen within the model was derived from the characteristics associated with *T. pratensis* and *P. hysterium*.

The purpose of Chapter 1 was to provide a literature review of the subject areas for a study of this nature. This chapter strongly indicates that the study must involve and integrate disciplines of plant ecology and plant epidemiology. This chapter also presents the biology of the model system under investigation and discusses the background to the epidemiological modelling which underlies this research.

Chapter 2 presents the development of a basic compartment *SIR*-type epidemiological model in discrete time, appropriate for the biennial nature of the host and the systemic nature of the pathogen. Through analysis and investigation of this model it was possible to observe the way in which biological characteristics of both the host and pathogen interact in order to explain epidemic development criteria and host steady-states. Simulations are produced which describe the host dynamics under a range of host and pathogen parameter values. With default values, the host population will eventually tend to stabilise at a steady-state population density and not undergo cyclical or outbreak dynamics. With high pathogenicity there is some cycling in medium performance host populations, however the pathogen becomes extinct and the healthy host tends to a stable population. High pathogenicity values in high performance host populations causes extinctions in all host categories. The recurrence-relationship derived from the basic model contains no terms relating to the pathogen parameters indicating that the pathogen has no role in host regulation

Many plant pathogens not only impact on the fecundity of hosts, but also cause an additional cost to the survival of the host through pathogen-induced mortality. This additional cost and its impact on the population dynamics of the host is the focus of research in Chapter 3. This chapter extends the basic model by incorporating a constant term that describes pathogen-induced mortality which can affect the emergence of second year plants between season, or mortality within season. By assuming this to be constant this parameter is effectively independent of host density, indicating a lack of adaptation by the pathogen to the host dynamics, and vice-versa. Because of lack of information regarding the additional mortality, the model is developed in three forms, with pathogen-induced mortality applied within season, or between season, or both between and within season. Analysis of the three forms of the additional mortality model was conducted to ascertain what impact there would be on the dynamics of the host. Further investigations were made to observe trade-offs in the life-history parameters of the pathogen, where highly transmissible strains cause less pathogen-induced mortality within the host population. This trade-off allows for observations of different strategies of pathogen strains and the impact on the population simulations produced. The model produces a range of outcomes showing that it is possible to produce cyclical dynamics when applying pathogen-induced mortality within and between seasons where there is high pathogenicity and medium host performance. Simulation outcomes from the basic model in Chapter 2 and the pathogen-induced mortality model in Chapter 3 show the populations tend to steady-state values with pathogen-induced mortality applied either between or within-season, or both between and within season with a trade-off in pathogen parameters. Where there is high pathogenicity and high host performance the population crashes. The noticeable qualitative differences between the models are when pathogen-induced mortality acts within and between season with high pathogenicity and medium host performance without a trade-off leading to repeating population cycles. The recurrence-relationship derived from the constant pathogen-induced mortality model contains the pathogen-induced mortality term β, indicating that pathogen has a regulatory impact on the host population dynamics.

Chapter 4 expands on the concept of pathogen-induced mortality by allowing the mortality term to be dependent on the size of the infected population, reflecting changes

in the characteristic as pathogen presence increase. The model involves variable pathogen-induced mortality occurring both within and between season, between season with constant mortality within season, and between seasons with constant mortality within season. This allows for models representing systems where pathogen-induced mortality throughout the life-span of an individual is related to the number of infectious individuals (the pathogen pool) in the previous generation. This adaptive strategy would allow for lower individual pathogen-induced mortality when there are is a high abundance of infected hosts and vice-versa. The model is assessed to observe if this adaptation allows for a strategy which alters the population dynamics whilst allowing the pathogen to remain within the population. Similarly, the trade-off within the pathogen life-history is explored. The model forms in Chapter 4 show that under most parameter values the host population will tend towards a steady-state population, occasionally harbouring the pathogen.

The final model adaptation is presented in Chapter 5, which incorporates a seedbank. The same discrete time approach is utilised which describes the seedbank as an intermediary stage between seeds set from flowering plants and seedling recruitment. The seedbank stage is developed directly into the model framework to assess the importance of a small proportion of seeds remaining viable after being set, and whether this seedbank model has any impact on the status of the dynamics of the host population and the persistence of pathogen.

The seedbank model with high host performance and medium or high pathogenicity show similar outcomes to the dynamics in the PGE, but this is inconsistent with the indications that there is low performing *T. pratensis* in the PGE. This is also seen when there is a trade-off in the pathogen life-history parameters suggesting that such dynamics could be obtained with a highly transmissible pathogen in a high performance host biennial population with a seedbank. Simulation outcomes demonstrate population cycles, even when the mortality is very high. A high performing host population can survive high pathogenic strains. When host performance is either medium or high and there is a trade-off in the pathogen parameters the pathogen becomes extinct, without the trade-off the infected individuals remain within the population. This explains how a small seedbank allows the host population to survive when exposed to highly infectious pathogen. The steady-state indicates that host and pathogen can exist at stable levels when pathogenicity is low and host performance is either medium or high. However, it

is unlikely to be the case for *T. pratensis*, as *P. hysterium* has been seen to have low transmissibility in pot cultured experiments. Furthermore the slope of the derived recurrence relationship suggests *T. pratensis* is a low performance host. If *T. pratensis* in the PGE has a seedbank it would need to be of either medium or high performance and exposed to a high pathogenic strain of *P. hysterium* to give the patterns observed. Additionally, Roberts (1986) reported that the seedbank of *T. pratensis* is rendered inviable in soils above 25^{o}C, and the mean July temperature of the soil in the PGE is above this temperature.

Chapter 6 presents some analyses relating to the biology of *T. pratensis* to provide insight in to the system under investigation. By using long-term data, the dynamics of the host within the PGE under varying nutrient regimes is highlighted. Furthermore, assessment is made of the impact of infection on the production and viability of seeds. Finally in this chapter the relationship between the developed models and the recent detailed survey data of the *T. pratensis – P. hysterium* is assessed to see if the models have any relationship with the biological observations. By applying the model in Chapter 3 to the PGE data, it suggests that *T. pratensis* is exposed to high pathogenic rust inoculum throughout the season allowing for mortality to impact both within and between seasons, however due to the hay cut *T. pratensis* will only be exposed at the beginning of the season, suggesting a form of the model with pathogen-induced mortality applied within season. The recurrence relationship shows a more accurate fit to the data with increased values of β suggesting that infected *T. pratensis* have high mortality rates in addition to being castrated. By relating the model to the PGE data, these simulations suggest that the PGE comprises a population of medium productive *T. pratensis* and a highly pathogenic *P. hysterium* strain. However, if the host population is in continual cycle containing infected individuals, then pathogen-induced mortality acts both within and between season and that the pathogen is both highly transmissible and causes high levels of pathogen-induced mortality, and shows no sign of a trade-off between transmissibility and pathogen-induced mortality. The dynamics between 1995 – 2008 show some cycling, with recent years having little or no rust presents suggesting that fluctuations are followed by stable host population without pathogen similar to simulations with pathogen-induced mortality acting either within or between-season, or both within and between-season with a trade-off between pathogen-induced mortality and transmissibility. It is inconclusive if there is a tendency for the pathogen to have a trade-off between pathogen-induced mortality and transmissibility; however where there

is low productivity there is a tendency for pathogen strains with a trade-off to remain at low levels within the host population.

There are discrepancies between the model in Chapter 4 and the datasets, but this could be due to the sampling variation and reporting of the PGE as the original evidence for the outbreak dynamics was based on numbers of plots with *T. pratensis* present, Furthermore, the PGE data does not have infection recorded. When considering the trade-offs in pathogen parameters, the population dynamics change but not to the outbreak cycles seen in the PGE. A possible adaptation to the model is to investigate the trade-off being density-dependent on either susceptible hosts or infectious hosts. They may also be a trade-off with contact rate which would identify which life-history trait is most influential in leading to epidemics.

Data analysis of surveys of the PGE indicate that irrespective of nutrient regime there are levels of population cycling, which slightly alter in intensity depending on fertiliser conditions. Analysis of seed productivity and viability collected from infected flowering individuals confirms that the *P. hysterium* does have a castrating effect on infected individuals. Also by relating the models to the collected data it can be seen that there is a good fit of the relationship with increasing levels of pathogen-induced mortality, indicating that it is possible to use this model as an explanatory tool in describing the population dynamics of this host – pathogen system. From this relationship it is also possible to ascertain that there is little evidence of density dependent regulation within the system, indicating that the model can be developed without including such terms to accurately replicate the simulation. By removing density dependence from the simulations it is noted that there are population cycles similar to those within the long-term PGE datasets where there are low productivity host population and medium or high pathogenic strains. Therefore it is likely that *T. pratensis* in the PGE is not under density-dependence effects, but are regulated by highly pathogenic rust which causes pathogen-induced mortality between seasons.

This models presented in this thesis demonstrates the importance of mathematical models in the understanding the epidemiology and population of pathogen in natural plant communities. By developing a range of models it has been possible to simulate the long-term dynamics of a biennial-host – systemic, castrating pathogen system, which can be used to identify conditions and characteristics of the *T. pratensis – P. hysterium*

system in the PGE. Although the model is a simplification of natural systems, such methods are useful and applicable as fitting the model to observed data shows a close relationship. Through the development and analysis of these models it is possible to conclude that *T. pratensis* population within the PGE is not under density-dependence factors and that it is likely that *P. hysterium* is not only a castrating pathogen, but also causes pathogen-induced mortality, which in part regulates the host population dynamics.

7.1. Future work and considerations

Two main areas of additional work required, are: apply the model to long-term datasets of another natural biennial host – systemic pathogen system to compare accuracy of the generic nature of the models, and obtain accurate parameter values for the model. Although the Park Grass Experiment is the longest running ecological experiment recording plant population and community dynamics, there are no recordings of infection status of plants In order to fit the epidemiological models to additional populations, long-term recordings of disease status will be required to be conducted.

Although the models developed are useful in replicating patterns of host population dynamics and isolating key features of the system, it would be beneficial to parameterise the model for more accurate simulations. The critical areas that need assessing are the diseased induced mortality rates in field populations. This would require isolating and tracking the survivorship of infected and exposed individuals in the fields and recording mortality. This would require the development of a screening technique to identify exposed first year individuals. Similarly required is defining the natural mortality rates of first year plants and infected and healthy second year plants to ascertain the impact this has on the recruitment and overall population dynamics. An additional requirement is to determine the transmissibility (c) and subsequent infection probability (p) of *P. hysterium*; this would require screening of individuals in the field and recording the proportion of seedlings that become exposed to inoculum, furthermore individuals that are exposed are required to be monitored to establish the probability of become infected if exposed.

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Appendices

Appendix 1 Plot nutrient applications within the PGE adapted from Williams (1978)

Plot	Treatment	Supply
1	N- Sulphate of ammonia	48 kg N ha^{-1}
2	Farmyard manure (bullocks) (1856-63)	35 t ha^{-1}
3	None	
4/1	P- granular superphosphate	35 kg P ha^{-1}
4/2	N- Sulphate of ammonia	96 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
5	-	
6 (a, b)	N- Sulphate of ammonia	48 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
6 (c, d)	-	
7	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
8	P- granular superphosphate	35 kg P ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
9/1	N- Sulphate of ammonia (last applied 1989)	96 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
9/2	N- Sulphate of ammonia	96 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
10	N- Sulphate of ammonia (last applied 1989)	96 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
	N- Sulphate of ammonia (last applied 1989)	144 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
11/1	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
11/2	N- Sulphate of ammonia (last applied 1989)	144 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
	Si- Silicate of soda	450 kg ha^{-1}
12	-	
13	Farmyard manure	35 t ha^{-1*}
	Fishmeal	63 kg N ha^{-1}
14/1	N- Nitrate of soda (last applied 1989)	96 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
14/2	N- Nitrate of soda	96 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}

15	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
16	N- Nitrate of soda	48 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
17	N- Nitrate of soda	48 kg N ha^{-1}
18a	N- Sulphate of ammonia	96 kg N ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
18b	N- Sulphate of ammonia	96 kg N ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
18/2	N- Sulphate of ammonia	96 kg N ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
18c	N- Sulphate of ammonia	96 kg N ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
18d	N- Sulphate of ammonia	96 kg N ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
	Na- sulphate of soda	15 kg Na ha^{-1}
	Mg- Sulphate of magnesia	10 kg Mg ha^{-1}
19/1	Farmyard manure	35 t ha^{-1*}
19/2	Farmyard manure	35 t ha^{-1*}
19/3	Farmyard manure	35 t ha^{-1*}
20/1	N- Nitrate of soda	48 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
20/2	N- Nitrate of soda	48 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}
20/3	N- Nitrate of soda	48 kg N ha^{-1}
	P- granular superphosphate	35 kg P ha^{-1}
	K- Sulphate of potash	225 kg K ha^{-1}