Article (refereed) - postprint

Salama, N.K.G.; van den Bosch, F.; Edwards, G.R.; **Heard, M.S.**; Jeger, M.J.. 2012 Population dynamics of a non-cultivated biennial plant Tragopogon pratensis infected by the autoecious demicyclic rust fungus Puccinia hysterium. *Fungal Ecology*, 5 (5). 530-542. 10.1016/j.funeco.2011.12.009

Population dynamics of a non-cultivated biennial plant *Tragopogon pratensis* L. infected by the autoecious demicyclic rust fungus *Puccinia hysterium* (Str.) Röhl.

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ABSTRACT

Population dynamics of the biennial plant *Tragopogon pratensis* have been monitored in the Park Grass Experiment at Rothamsted Research, Harpenden, UK, over many years. Observations of diseased *T. pratensis*, systemically infected by the autoecious demicyclic rust *Puccinia hysterium*, were made over the period 1995-2008, and confirmed an outbreak pattern of dynamics, characterised by an increase to a relatively high incidence to low almost indiscernible levels. An epidemiological model was developed taking into account the biennial habit of the host plant, and the systemic nature of infection during the winter period, and the partial sterilisation of infected second year plants. Seedling emergence rate and natural mortality between season and within season were key parameters affecting host performance. The transmission rate between infected second year plants and susceptible first year seedlings, and the probability that the fungus would survive the winter systemically as mycelium producing aecia and telia on emerging second year plants, were key parameters associated with pathogenicity. Furthermore the possibility of pathogen-induced additional mortality was modelled. The model predicted that outbreak dynamics of *T. pratensis* would occur with high pathogenicity and medium or high host performance. In the former case the population dynamics would be cyclical, with in some cases infected plants going to extinction. In the latter case both host and pathogen would go to extinction. The model predicted that the two pathogenicity parameters were critical in determining whether the pathogen would invade a healthy population; whereas pathogen induced mortality had little influence, a result also obtained in some limited potted plant experiments. Fitting the model to the field data indicated that there was little or no density-dependence in seedling emergence rate, and again that pathogen-induced mortality played little role in the observed population dynamics.

INTRODUCTION

Pathogens can play a key ecological role in natural plant communities impacting on species performance, affecting viability and fecundity of individual plants, reducing population size, generating selective forces for genetic change, and altering community structure (Burdon 1982; Burdon *et al*. 1989; Dinoor & Eshed 1990; Thrall

& Burdon 1997; Frantzen & Muller-Scharer 2002; Burdon *et al*. 2006). However, there have been few attempts to model epidemics in natural plant populations in non-cultivated systems, with many studies mostly limited to empirical descriptions that ignore theoretical implications or applications (Dobson & Grenfell 1995). Models have been developed describing the population dynamics of annual plant hosts and soil-borne pathogens (Thrall et al., 1997); dispersal characteristics and disease dynamics in populations (Thrall & Burdon, 1999; Frantzen & van den Bosch, 2000); and especially in recent years, combining spatial statistics with spatially explicit models to estimate rates of spread and effects of regional heterogeneity on co-evolutionary processes (Ovaskainen & Laine 2006; Brooks *et al*. 2008; Soubeyrand *et al*. 2009; Smith *et al*. 2011).

The Park Grass Experiment (PGE) at Rothamsted Research, Harpenden, UK, is the oldest continuous ecological experiment in the UK, with work conducted on long-term plant population dynamics over many years (Silvertown *et al*. 2002). Plant species within the PGE have shown a range of dynamics including: increases (e.g. *Trifolium pratense*), decreases (e.g. *Veronica chamaedrys*), and fluctuations (e.g. *Conopodium majus*) in population size. Other species (e.g. *Tragopogon pratensis*) followed an outbreak dynamic in which the population increased and then declined (Dodd *et al*. 1995). Many of these patterns have been interpreted as consequences of the long-term fertiliser regimes altering the soil nutrient content and pH. However it has been speculated that the outbreak pattern of dynamics of the biennial species *T. pratensis* has been due to an autoecious, demicyclic rust, *Puccinia hysterium* (Silvertown *et al*. 2006). It is also possible that edaphic factors may affect the plant-pathogen interaction directly, as found in other systems studied (Springer, 2009). Unfortunately the long term PGE data set does not include the recorded incidence of rust infection.

Importantly, the rust is systemic during the wintering of the biennial host. This systemic nature is relatively unusual as systemic rusts are more prevalent in arctic and montane ecosystems (Wennström 1999). The example most cited of a systemic rust is *P. punctiformis* on *Cirsium arvense* and other thistle species (Frantzen 1994; Cripps *et*

al. 2009), with other examples noted including *P. pulsatillae, P. pratensis, P. monoica* and *P. thlaspeos* (Jarosz & Davelos 1995). Similarly to *P. hysterium*, *P. thlaspeos* is a systemic rust but does not vertically transmit through seeds (Kropp *et al*. 2002), therefore transmission is solely through spore dispersal. *P. hysterium* suppressed reproduction of *T. pratensis* (Salama *et al.* 2010), whilst in a similar non-cultivated host-pathogen system, the rust *P. lagenophorae* significantly altered plant fitness by reducing seed production of *Senecio vulgaris* (Paul & Ayres 1986a; 1986b). Both *P. lagenophorae* and *P. hysterium* are demicyclic and lack an asexual repeating urediospore phase (Wilson & Henderson 1966).

The scientific literature on *P. hysterium* is sparse. The life history involves teliospores from diseased second year plants infecting first year plants from June to the end of the growing season (Parmelee & Malloch 1972). Symptoms are not normally seen on first year plants. The dispersal period is often interrupted in traditional hay meadows (such as the PGE) by a "hay cut" at the end of June which limits the infective period. Basidia develop from germinated teliospores on first year plants and produce basidiospores which can also be transported to new hosts. The basidiospores germinate to produce systemic mycelia, which enable the pathogen to survive the die-back period during the host's below-ground overwintering phase. Pycnia develop and undertake a sexual stage as pycniospores. The following season, surviving plants re-emerge exhibiting aecia formed mostly on the stem, giving the typical "cluster cup" appearance, which produce aeciospores. Aeciospores germinate on the host tissue and germ tubes either directly penetrate tissue or grow through natural openings such as stomata. Telia, bearing teliospores on localised mycelium are then produced (Wilson & Henderson, 1966) completing the cycle.

This paper investigates the hypothesis presented by Silvertown *et al*. (2006) that *P. hysterium* has a regulatory influence on its host population dynamics and that this relationship is density dependent (Silvertown *et al*., 2002). This assessment is undertaken by presenting data relating to the long term population dynamics of the system in the PGE and attempting to obtain information on pathogen induced mortality through the use of inoculation experiments. Biological characteristics of a

biennial host – systemic pathogen system are then incorporated into an epidemiological model which is used to determine conditions under which population dynamics similar to those observed for *T. pratensis* infected by *P. hysterium* in the PGE are simulated. Specifically the epidemiological model takes into account the biennial nature of the host plant *T. pratensis* and the systemic characteristics of the rust, *P. hysterium*. The model is of *SIR* form, in which host categories are defined as Susceptible, Infectious and Removed (Anderson & May 1979), and models non-continuous host generations using discrete time. Such compartmentalised models of disease dynamics have been used previously to represent plant – fungal pathogen dynamics in continuous time (Gilligan, 2002). The model developed here is an example where a discrete-time approach (Allen 1994; Switkes, 2003) provides a more appropriate representation of the host–pathogen system, given the biennial host characteristics, the unusual yet key systemic nature of the rust pathogen, and the infection process linking second and first year plants. Expanded forms of the discrete *SIR* model have been used to describe gene frequency and disease spread in plant populations (Kesinger *et al.*, 2001).

The model was developed to be sufficiently flexible to include the impact of pathogen-induced mortality on the ability of first year plants to overwinter and to re-emerge and grow in the second growing season. Pathogen-induced mortality - often termed *virulence* in human and animal epidemiology, see Antonovics (2005) - has been reported for a wide range of plant foliar diseases (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). Thus we incorporated within, as well as between, growing season pathogen-induced mortality. Specifically this type of pathogen-induced mortality has been reported with late infections of *Puccinia,* e.g. *S. vulgaris infection* by *P. lagenophorae*; at the beginning of a growing season, infected host plants have a lower probability of survival, but also increase their chances of mortality in the overwintering phase (Frantzen & Müller-Schärer 1999). Within our model, pathogen-induced mortality of individual plants has an effect on the population dynamics of the host, not simply by altering the number of seedlings through partial sterilisation of infectious individuals, but also by reducing the numbers of infected first year plant individuals which potentially become

infectious when there is additional mortality between seasons. Additionally, within season pathogen-induced mortality reduces the number of second year infectious individuals that can infect first year susceptible plants in the same season. Within the modelling framework we consider both constant and variable pathogen-induced mortality.

METHODS

Field observations

Vegetative and flowering *T. pratensis* individuals and signs of rust infection were recorded in each of the subplots prior to the annual 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 of the 97 subplot from the 24 plots. Each of the plots varied in size and had a range of fertilizer and pH treatments applied (Silvertown *et al*. 2006). These counts may be an underestimation due to the difficulty of identifying first year individuals. During the years 2005 – 2008 the methods of data collection were modified due to plot access restrictions that were imposed at the PGE. This modified method involved using five 1m x 1m quadrats placed at random near the edges of each sub plot. These quadrats are likely to adequately represent the situation in the rest of a subplot because plants are distributed relatively homogeneously throughout a plot and there are sharp boundary characteristics due to minimal lateral nutrient movement between plots (Crawley *et al.* 2005). Within each transect or quadrat, the numbers of flowering and vegetative individuals were recorded and rust incidence assessed using a three point score (uninfected, low infection, high infection); however this scoring was subsequently simplified to rust presence or absence.

Estimations of *P. hysterium* infection, overwintering survival and induced plant mortality

In order to ascertain mortality rates, 100 *T. pratensis* seeds collected from the Royal Horticultural Society (RHS) gardens, Wisley, in 2005 were germinated and grown in pots (diameter 130mm, height 121mm). Half of the cultivated first year individuals

were artificially inoculated with *P. hysterium* using a method of brushing infected plant material, bearing both aecia and telia and also collected from the RHS gardens with a camel hair brush over talc, and then applying the talc/inoculum powder over susceptible individuals at dusk, so optimising stomatal opening, and the other half sprayed with water as a control. The plants were kept in high humidity for four days using dew chambers. Plants were then kept outside under natural conditions, allowed to die back over the winter, and monitored for re-emergence as second year plants in the spring and for the incidence of rust infection. The effects of inoculation on survivorship were quantified (Salama 2009). A second cohort of individuals was grown and inoculated using the same method the following season. For both cohorts the second year plants were kept in pots for a second winter to check on whether they re-emerged for a third year.

Model description

The epidemiological model was developed in discrete time for a closed biennial host – pathogen system (Fig 1) with characteristics similar to the *P. hysterium*/*T. pratensis* system. We assume the biennial host has a vegetative, non-seeding first year when individuals are healthy and susceptible (*S*) to infection. First year plants are exposed to inoculum from infectious second year plants (*I*). The average number of spores deposited on a plant is proportional to the density of infectious second year plants. The probability that no spores are deposited on a healthy plant is given by the first term of the Poisson distribution, $e^{(-cI)}$, which gives the infection frequency as $1-e^{(cI)}$ (Madden *et al*., 2008) where *c* is the effective transmission rate between infectious and susceptible individuals. Individual plants are exposed (*E*) and latently infected at the end of their first growing season, or non-exposed, then die back and overwinter below ground. The plants re-emerge the following year and reproduce (i.e. are monocarpic). A proportion (*p*) of exposed individuals become infectious (*I*) second year plants, a proportion (1–*p*) of the exposed individuals emerge as healthy (*R*) second year plants, as do the non-exposed individuals. Re-emerging healthy second year plants are effectively removed from the epidemic dynamics as they do not contribute to subsequent infection of first year plants, nor do they become infected by *I* plants. Individual plants have mortality rates *b* within season and *d* between seasons, and have a pathogen-induced mortality rate (β) which is assumed to be related to the

numbers of infectious individuals according to the flexible term $\beta e^{-\sigma I_t}$, which can represent different forms of pathogen-induced mortality. For σ = 0, pathogen induced mortality is constant (= β); when σ approaches ∞, there is effectively no pathogen induced mortality; and when $0 < \sigma \leq f$, where f is a finite upper bound for σ, there is a variable form of pathogen induced mortality that decreases with the size of the infected population. The latter possibility may be associated with selection for decreased virulence in the pathogen (or alternatively for increased resistance or tolerance in the host) as the pathogen population increases. *R* individuals set seed with a seedling emergence rate *a*. We assume that *I* individuals are sterilized and do not contribute seed to the following generation, but note that this is an oversimplification as sterilization is incomplete (Salama *et al*., 2010). The seedling emergence rate is also density-dependent, using the λ parameter as proposed by de Wit (1960) specifically for plant populations. The higher is the value of this parameter, the greater the level of density dependence. Although there may be additional intra-specific (from non-seed producing *I* plants) and inter-specific competition from the plant meadow community, these additional complications to the model are not investigated. As noted above there is a natural mortality within (*b*) and between (*d*) seasons.

With these assumptions the host categories are linked in the following system of equations:

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

$$I_{t+1} = (1-d)(1-b)(1-\beta e^{-\sigma I_t})\, pS_t\left(1-e^{-c(1-b)(1-\beta e^{-\sigma I_t})I_t}\right) \quad \textbf{(2)}$$

$$R_{t+1} = (1-b)(1-d)S_t\left[1-p\left(1-e^{-c(1-b)(1-\beta e^{-\sigma I_t})I_t}\right)\right] \quad \textbf{(3)}$$

A full derivation of this *SIR* model from the *SEIR* formulation described above is given in Appendix 1. These equations can also be adapted to cases where there are differential within and between season mortality rates and/or mixed constant and variable mortality rates, but these elaborations are not considered in this paper.

Numerical and analytical techniques were used to determine the quantitative and qualitative properties of the three models representing the three forms of pathogen induced mortality (Equations 1-3 with σ set accordingly). Implicit solutions for the steady-state density of infected individuals I^*, i.e. when there was no change in the size of host categories between time-steps t and $t+1$, were obtained (Appendix 2). Time plots of the model were obtained numerically using *R* 2.7.2 (R development core team) for varying parameter values. In addition, an invasion criterion determining whether the pathogen could establish following introduction was derived. Finally a second-order recurrence equation was derived from the models describing the relationships between population sizes at discrete time intervals (Appendix 3).

Obtaining parameter values

Numerical data for approximating parameter values were obtained from field observations and greenhouse trials (Salama 2009; Salama *et al.* 2010), unpublished survey data by two of the authors (G. Edwards and M. Heard) collected from the PGE, and published material relevant to *T. pratensis, P. hysterium* and similar host – pathogen systems (Table 1). The population dynamics of *T. pratensis* in the entire PGE were plotted against year with polynomial models of the n^{th} order (with $2 \leq n < 6$) used to interpolate missing data using Microsoft Excel (Microsoft Corp. 2003).

RESULTS

Survey data

T. pratensis was found in all but one of the plots and 15 subplots between 1995 – 2008, across a range of fertiliser and pH treatments. The survey data demonstrates the outbreak nature of infected second year plants (Fig. 2a) in plots under differing treatment types and of healthy second year plants across all plots in the PGE (Fig. 2b), in both cases followed by a low but stable population size. Although no data were collected in 1999, 2000 and 2005 these data were interpolated using polynomial regression. The intention here was simply to obtain missing values for use in subsequent model investigations, not to model the epidemic. A range of polynomial

orders was used in the regressions, the predicted values were compared with observed values, and goodness of fit tested adjusting for the degrees of freedom in the regressions. Polynomials which predicted negative values were rejected irrespective of the fit obtained. On this basis a 6th order polynomial provided the most consistent estimates for the missing values [adjusted $R^2 = 0.881$; F-value = 13.34 (6,4 d.f.); p = 0.013]. When comparing the observed versus predicted values a good linear fit was obtained for the 6th order polynomial, having a slope not differently different from 1 and a positive intercept close to 0. The missing values obtained in this way for healthy second year plants are shown in Fig. 2(b). A similar procedure was used to estimate missing values for the infected plants and again a 6th order polynomial was chosen as most suitable for the purpose intended - to obtain missing values used in subsequent model investigations. The initial numbers of hosts used in the simulations (Table 1) were set as the lowest non-zero recordings in the PGE.

Inoculation experiments

Because of the difficulty in observing spore dispersal (transmission), disease expression (probability of an exposed first year plant becoming an infected second year plant), and the influence of infection on host survivability in the field, plant inoculations were conducted with potted plants under greenhouse conditions. In 2005 seeds collected from RHS Wisley were germinated, but due to facility restrictions, only 100 of the germinated seeds were grown to form the first cohort of susceptible first year plants; of these, 50 were artificial inoculated and 50 treated as controls. The method of inoculation does not necessarily mean that plants had been exposed to a sufficient infective dose; without detailed histological work (beyond the scope and scale of this study), there was no way of ascertaining until re-growth the following season whether plants had been infected. This made it difficult to estimate the value of p in these inoculation experiments. Furthermore expression of disease in second year plants in the field is a composite of p and c and it is difficult to distinguish the relative contribution of either factor.

281 In 2006, 15 of the control plants did not re-emerge, whereas all the inoculated plants
282 survived the over-wintering phase and three of these plants produced aecia,
283 suggesting a *p* value of 0.06. All re-emerged plants were monitored until July to
284 assess within season mortality. Of the 82 healthy plants (50–15 = 35 from the re-
285 emerged control group, 50–3 = 47 from the re-emerged inoculated group) four
286 suffered mortality whilst all three of the diseased plants survived. These results show
287 no evidence for pathogen-induced mortality. Seeds were collected from healthy and
288 infected plants; additionally these second year plants remained in pots until the
289 following season. In 2007, none of the three diseased plants re-emerged, whereas 16
290 of the 78 healthy plants re-emerged again as healthy second year type individuals,
291 surviving the 2007 season but did not re-emerge in 2008. This demonstrates that pot
292 grown plants have differing characteristics to their biennial nature in the field, most
293 likely due to the managed interventions and conditions which also make the
294 estimation of rates of natural within and between season mortality difficult.

295

296 Of the 100 seeds collected for the second cohort of plants in 2006, 50 were inoculated
297 and nine emerged in 2007 all with aecia, suggesting a *p* value of 0.18. There was also
298 high mortality for the 50 control plants with only five re-emerging as healthy second
299 year plants, suggesting a high natural between-season mortality rate of 0.90; one of
300 the five died in the 2007 season. Again these results show no evidence for pathogen-
301 induced mortality. The remaining plants were left to overwinter again resulting in no
302 re-emergence of the nine infected plants, and re-emergence of three second year type
303 plants from the four remaining healthy plants, of which one died during the 2008
304 season.

305

306 These findings illustrate the difficulties inherent in estimating life-history parameters
307 including natural mortality under pot conditions for a non-cultivated host-pathogen
308 system. The values in Table 1 are thus best estimates based on the sources identified
309 above. The between season mortality rates in these pot-grown plants varied
310 considerably between the two cohorts, and for within-season mortality are likely to be
311 an underestimate due to the managed growing conditions; however within season
312 mortality was in the order of 15-30 %, whilst overwintering mortality was in the order

of at least 30% There was no evidence for pathogen-induced mortality in these results. However, the similar cultivation of *S. vulgaris* (Frantzen 2007) demonstrates that such evidence can be found for a different system. Because of these uncertainties we produce simulations under a range of values for β, c and p.

Modelling investigation

Steady states in the absence of the pathogen

In the absence of the pathogen the equations for healthy first and second year individuals are:

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

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

Giving steady-state expressions in the absence of the pathogen as:

$$\hat{R} = \frac{a(1-b)^2(1-d)-1}{\lambda(1-b)} \quad \textbf{(4)}$$

$$\hat{S} = \frac{a(1-b)^2(1-d)-1}{(1-b)^2(1-d)\lambda} \quad \textbf{(5)}$$

Provided that the condition $a(1-b)^2(1-d)>1$ is met, the plant population will approach a steady-state population size determined by the seedling emergence density-dependence parameter, λ, and plant mortality rates, b and d. This expression is intuitive as it is the product of the seedling rate, mortality within the two growing seasons (thus the squared term), and mortality during the one winter period.

Derivation of the invasion criteria

The conditions for pathogen invasion within the host population were derived for the three forms of pathogen-induced mortality. For the pathogen to persist within the system the basic reproductive number (R_0) must be greater than one (Anderson & May, 1979).

For the case with no disease-induced mortality (equations 1 – 3, β = 0), the criterion for the pathogen to invade was obtained as:

$$\frac{I_{t+1}}{I_t} = (1-b)^2(1-d)pc\hat{S} = \frac{pc[a(1-b)^2(1-d)-1]}{\lambda} > 1 \quad \textbf{(6)}$$

Where for infinitesimally small values of I, $1-e^{-c(1-b)I_t}$ was approximated by $c(1-b)I_t$

or equivalently:

$$\hat{S}(1-b)^2(1-d)pc > 1 \quad \textbf{(7)}$$

The expression on the left hand side has a direct interpretation as the basic reproductive number of the disease. If one infected second year plant is introduced into a susceptible first year population then the number of second year infected plants that will result in the following year is determined by the product pc (the number of successful infections that emerge) corrected for by the survival terms over the two growing seasons and the one between seasons.

Thus, if a pathogen is introduced into a previously healthy plant population then the transmission rate c and the probability of overwintering survival p must be sufficiently high for this criterion to be met and for the pathogen to establish.

Similarly for models with constant pathogen-induced mortality, for infinitesimally small values of I, the term $1-e^{-c(1-b)(1-\beta)I_t}$ can be approximated by $c(1-b)(1-\beta)I_t$. The criterion for pathogen invasion is then:

$$\hat{S}(1-\beta)^2(1-b)^2(1-d)pc > 1 \quad \textbf{(8)}$$

In the case of variable disease-induced mortality, when I is infinitesimally small, the criterion derived is the same as equation 8.

In all three cases the pathogen invasion criteria depends on the transmission rate, the probability that an exposed individual will become infectious in the second year, pathogen induced mortality (where non-zero), as well as the background host mortality between and within season and the susceptible host population size.

Model Simulations

Parameter values (Table 2) were used to generate a range of outputs representing high, medium and low host performance (a, b, d, λ) and similarly high, medium and low pathogenicity (p, c, β). Performance here is used to define the level of host mortality, recruitment and density-dependent seedling emergence; whilst pathogenicity describes the rates of pathogen-induced mortality, and disease expression (following overwintering survival). The ranges in parameter values were considered for the three model formulations presented above. We also applied a transmission-virulence trade-off in the pathogenicity parameters but this aspect is not developed further in this paper due to the absence of genetic information for this host-pathogen system.

In general the models produced similar outcomes, although varying in transient dynamics. The model simulation outputs with medium pathogenicity and medium host performance produced steady-state populations for both healthy and infected plants (Fig. 3a) within the population. Under conditions where low pathogenicity was combined with a low host performance (Fig 3b) infected plants became extinct. Both healthy and infected plant populations became extinct when high pathogenicity and high host performance were combined (Fig 3c).

Cyclical dynamics occur when high pathogenicity is combined with medium host performance; however the trajectory of the population cycles differ dependent on the model used. Where there is no pathogen-induced mortality (Fig. 4a) or there is variable pathogen-induced mortality (Fig. 4c) the host population cycles, but then tends to a steady-state population size where infected plants are absent from the

394 population. Continual, repeating population cycles are obtained when constant
395 pathogen-induced mortality is maintained (Fig 4b). Other combinations of host
396 performance and pathogenicity levels tended to stable steady values without cycles.

397

398 *Parameter influence on the disease invasion criterion*

399 Using the derived invasion criterion for the constant pathogen-induced mortality form
400 of the model (Equation 8) it is possible to obtain the parameter space where the
401 criterion conditions are met by altering pathogen parameters and host population size.
402 For the case of constant pathogen-induced mortality, with for simplicity the mortality
403 rates set to $b = d = 0.3$, Fig 5 demonstrates the range of parameter values that satisfy
404 the invasion criterion.

405

406 *Derivation of recurrence relationships*

407 Second-order (two time-step) recurrence equations were derived (Appendix 3) to
408 represent the relationship between population sizes at different discrete time intervals.

409

410 For the model without pathogen induced mortality the following relationship was
411 derived:

412 $$\frac{1}{I_{t+2} + R_{t+2}} = \frac{1}{a(1-b)^2(1-d)R_t} + \frac{\lambda}{a(1-b)^2(1-d)} \quad \textbf{(9)}$$

413 This indicates that infected second year plants are simply replacing healthy second
414 year plants in the overall relationship (the recurrence relationship derived in the
415 absence of disease is identical to equation 9 with $I_{t+2} = 0$). There are no pathogenicity
416 parameters contained on the right hand side of the recurrence relationship.

417

418 However, when pathogen-induced mortality is included in the model, a recurrence
419 relationship is derived in which:

$$\frac{(1-\beta)}{I_{t+2}+(1-\beta)R_{t+2}} = \frac{\lambda}{a(1-b)^2(1-d)} + \frac{1}{a(1-b)^2(1-d)R_t} \quad \textbf{(10)}$$

This indicates that disease-induced mortality would directly affect the population dynamics as the β parameter appears on the left hand side of the equation.

Infected plant steady-states

A necessary condition for an endemic steady-state of diseased plants is that the invasion criterion is met, depending on the form assumed for pathogen-induced mortality (Equations 6-8). As shown in Fig. 4 the transient approach to steady states can show many complexities in the cycling behaviour. Simulations across some 1000 combinations of values of β, p and c, with other parameters held at default values (outlined in Table 1), were made and less than half of these showed single stable steady states corresponding to equation A9 in Appendix 2.The parameter space in which stable endemic steady states occurred are shown in Fig.6. In Fig. 6a-c is shown the values of I^* corresponding to values of p, c and β respectively across the range of the values in the other two parameters. The scatter represents the sensitivity to the tested parameter. Clearly, across the combination of the other parameter default values, there is a clear dependence of I^* on p, the probability that an exposed first year plant becomes an infected second year plant. The scatter only becomes apparent at high values of p. For β and c there is scatter across the range tested and the dependence is conditional on values of the other two parameters.

In the expression for the invasion criterion (Equation 8) the product pc appears, so in Fig. 6d is shown a colour contour plot of I^* against β and pc. The boundary of the steady state region is irregular because of the numerical procedures used in the calculation, but gives a good approximation to the actual parameter space enclosed. As would be expected from the invasion criterion, low values of pc and high values of β do not result in an endemic steady state for disease. The colours for values of I^* indicate a complex response surface but with a reasonably consistent pattern. In general there is an increase in value of I^* above a pc threshold of around 0.4, whereas there appears to be a slight decrease in I^* with increasing values of β.

Relating the models to T. pratensis – P. hysterium in the PGE

Applying equation 9 to the field data and interpolated points, there was a linear relationship between the numbers of healthy flowering plants and the numbers of flowering plants (infected and healthy) two years later (Fig 7). The residuals between the observed data and predicted values based on the recurrence relationship are normally distributed (mean residual is 0.0037 with standard deviations of 0.0019) and do not significantly differ from each other. The regression line describes over half the residual variation in the adult plant population. We recognise that this relationship depends strongly on using interpolated values in the fitting of equation 9; and thus the following interpretation, although informative in terms of estimation, does not refer to statistical significance and should be treated with caution.

In the basic model without pathogen-induced mortality the fitted coefficients (S.E.) gave an intercept $\frac{\lambda}{a(1-b)(1-d)} = 0.002$ (0.002) and gradient $\frac{1}{a(1-b)^2(1-d)} = 0.860$ (0.262). This gradient is less than 1 which means that the host population persists ($a(1-b)^2(1-d) > 1$). Under these conditions, the net reproductive number for the host plant *T. pratensis* is: $R_0 = \frac{1}{0.86} = 1.16$. Fitting the model with pathogen-induced mortality (Equation 10), the greater the value of β the better the fit to observed data although the estimates of the slope barely differ across this range (Table 3). As β approaches 1, the number of diseased plants I approaches 0 and equation 10 reduces to the recurrence relationship obtained in the absence of disease. While acknowledging the caution noted above, the value of $\lambda(1-b)$ can be approximated from the fitted intercepts and gradients. Assuming a default within-season mortality rate (b=0.3) gives a very low λ value of about 0.03 suggesting little influence of seedling emergence density-dependence on the host population dynamics.

INTERPRETATION AND DISCUSSION

The aim of this paper was to examine the hypothesis that *Tragopogon pratensis*, described as an outbreak species in the Park Grass Experiment, is regulated by the autoecious, demicyclic rust pathogen *P. hysterium* (Dodd *et al*. 1995). This was done by reference to field observations for *T. pratensis* and *P. hysterium,* developing a generic epidemiological framework appropriate for the life history characteristics associated with this host and pathogen system, and obtaining parameter values from small scale pot experiments to use in model investigations.

Although several forms of the model are presented, the model without pathogen-induced mortality corresponds to Wennström's (1999) claim that in several annual host-systemic pathogen systems there is little additional mortality associated with disease as this would not be to the pathogen's advantage; this may also be the case for a biennial or perennial host. However, systemic vascular wilt pathogens cause mortality in annual hosts whereby the pathogen is able to produce either long-lived spores in the dead plants or produce spores that can be aerially dispersed. In relating the model outputs to the long-term data describing the outbreak dynamics of *T. pratensis* in the PGE (Dodd *et al*. 1995), there are suggestions of outbreaks or longer term cycles occurring. If this version of pathogen-induced mortality is appropriate for the *T. pratensis* - *P. hysterium* system, it could be inferred that the PGE consists of a medium performing host exposed to highly contagious pathogen but which has a low probability of surviving the overwintering period. The rate of field mortality in *T. pratensis* is reported as 0.5 within season and 0.88 between seasons (Mahesh, Upudhyaya & Turkington 1996), which are within the range that will lead to cycling in the plant population. The host population in the PGE increases and decreases over a 60 year period (Silvertown et al 2002) which is longer than predicted in the basic model; but as with the detailed observations between 1995 – 2008 of *T. pratensis* there is a two year delay in the relationship between the incidence of susceptible plants and the resulting incidence of infectious individuals within the longer cycles. The survey data demonstrates the outbreak nature of both healthy and infected *T. pratensis* and thus indicates that neither population reaches a high endemic steady-state.

When the pathogen invasion criterion is not met there will be no endemic steady state for diseased plants and the pathogen will be eliminated from the system; healthy plants would then increase towards steady-state values. Additionally the model replicates the expectation that a biennial host will have a two year lag relationship in counts of second year plants. The derived recurrence relationship (equation 9) is of the same form in the presence or absence of infected second year plants. Where infected plants are present they simply substitute for healthy plants in the recurrence relationship. Therefore it can be inferred that a biennial host population would not be regulated by a systemic pathogen if it does not cause additional mortality of infected hosts. Pathogen regulation of host dynamics may have a limited impact as demonstrated by the absence of pathogen related parameters in the recurrence relationship. This supports the statements of Harper (1990) and Frantzen (2007) that at the population level, pathogen influence on populations may be minimal.

When considering constant pathogen induced mortality in the model ($\sigma = 0$), under the majority of conditions the host population tended rapidly towards a stable population (as in Fig.3a) or the infected or total host population crashed (Fig. 3b,c). The exception was for the combination of medium host performance and high pathogenicity, which leads to initial population cycles (Fig. 4a) which could under certain time-frames be interpreted as repeated outbreaks, rather than the single outbreak pattern seen in the PGE for *T. pratensis* (Fig 2a). Where there is both high host performance and a highly pathogenic strain, the host will become extinct (Fig.3c). However, it is unknown whether our 1995 – 2008 observations in the PGE are leading to a host crash, a trough in continual repeating cycles, or a trough in a population which eventually tends to a stable population. If this form of the model is reflective of *T. pratensis* in the PGE, then the PGE contains a high performance host population that has been exposed to a highly pathogenic strain. However, high pathogenicity would not be of advantage if it leads to extinction of the host plant.

Although constant pathogen induced mortality was assumed within and between seasons, other forms may be more appropriate to describe a natural host-pathogen system depending on when the host population is exposed to inoculum. For example, Paul & Ayres (1987) reported no additional mortality of *Senecio vulgaris* seedlings infected by *P. 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. Our model outputs predict that it would be advantageous for the host population not to be high performing, as an introduction of a highly pathogenic strain would lead to a population crash. Similarly, the most beneficial strategy for the pathogen would not to be highly pathogenic, as in a low performing host population the pathogen invasion criterion would not be met; and similarly, in a high performance host population the host would go extinct along with the pathogen. This 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 independently of host population size.

The third form of the model is for variable pathogen induced mortality. Examples of where there is a reduction in pathogen-induced mortality are given by Strong (1992) and Alexander (1992). *Silene alba* infected by the castrating smut *Ustilago violacea* (also known as *Microbotryum violaceum*) provides evidence that smaller populations have higher relative levels of infection and in turn, host pathogen-induced mortality, whereas larger host populations show less signs of infection. Additionally, Alexander & Antonovics (1988) and Alexander (1990) found that in the same host-pathogen system, smaller populations were less likely to become infected than larger populations. From these observations it could be inferred that increased host density has an effect in lowering the pathogen-induced mortality rate, whilst potentially enabling highly transmissible strains to develop.

Here we apply a reducing factor of the form $e^{-\sigma N}$, where N is a measure of population size (MacFadyen 1963). In our case we take N to be the size of the infected

572 population I and for simplicity σ =1 It could be the case that σ takes other non-zero
573 values; however we have no information on which to apply a different conditional
574 response and this would lead to further model elaboration. Under most parameter
575 combinations the population tends towards a stable population except where there is a
576 strain with high pathogenicity in a high performing host population, in which case the
577 population crashes. The outcome in models with variable disease-induced mortality
578 could be representative of the PGE observations of *T. pratensis* as there are dynamics
579 reminiscent of the recorded observations in all simulations with highly pathogenic
580 strains in medium or high performance host populations; these demonstrate the
581 increase and then decrease in host population numbers as described by Dodd *et al*.
582 (1995). However this prediction is made for all forms of pathogen-induced mortality
583 where there is both high host performance and high pathogenicity.

584

585 In order to assess the model, data collected from the PGE relating to healthy and rust-
586 infected *T. pratensis* were fitted to the derived recurrence relationships.
587 Acknowledging the caution we have already expressed due to the interpolated data
588 points, we note that as the value of β increases towards 1, the better fit to the data.
589 However, although increasing β in the recurrence relationship does improve the fit it
590 only marginally changes the fitted coefficients, suggesting a lack of biological
591 relevance for β as a single parameter, confirming the results of the steady state
592 analysis of the model. The results of the pot-grown *T. pratensis* experiments also
593 provided no evidence for pathogen-induced mortality under those conditions; however
594 the variable results obtained on the other parameters and the managed growing
595 environment mean that direct comparisons with field data should be made with
596 caution.

597

598 Using the estimated coefficients for the recurrence relationships and the approximate
599 parameter values in the model, it was possible to estimate $\lambda(1-b)$. Given there is a low
600 natural death rate of plants within-season (b), and also with those used in the
601 simulations, by inserting the estimates into equation 1, it shows that the seedling
602 emergence rate is not under density dependent regulation. Therefore the seedling

603 emergence density dependent term can be deleted to give $S_{t+1} = a(1-b)R_t$. Where a
604 host population is exposed to a pathogen which is highly transmissible, the outbreak
605 increase and then decrease in host population (Fig 8) similar to that seen for *T*.
606 *pratensis* in the PGE (Fig 2) can be obtained, although the numbers of plants and
607 time-scales are different. Ideally, the model would be validated with alternative
608 datasets; however there are no such long-term dataset of a biennial host plant and an
609 associated systemic, partially sterilising rust pathogen. Although the PGE (and other
610 experiments) provides long-term records for host population dynamics, there is
611 limited or no recording of infection by pathogens which prevents such analysis.
612 However, this simplified density-independent model provides some insight into the
613 dynamics of *T*. *pratensis* within the PGE. The outbreak (Dodd *et al*. 1995) can be
614 interpreted as being partially effected through the reduction of reproductive capacity
615 by association with the partially sterilising rust pathogen, *P*. *hysterium*; however it
616 cannot be concluded that pathogen regulation is the driver of such dynamics without
617 direct evidence for pathogen-induced mortality.
618

619 The model presented in this paper demonstrates the utility of mathematical models in
620 the understanding of disease epidemiology and population dynamics of fungal
621 pathogens in natural plant communities. By developing a flexible model with different
622 forms of pathogen-induced mortality it has been possible to explore the long-term
623 dynamics of a biennial host in the presence of a systemic, partially-sterilising
624 pathogen. For other natural pathosystems sharing some or all of these characteristics it
625 would be theoretically possible to analyse and predict the pathogen's impact on the
626 dynamics of the host population.

627

628 **Acknowledgments**

629 The work presented here was funded by the BBSRC (BBSSA200412470). Particular
630 thanks to Rothamsted Research for providing access to the Park Grass Experiment
631 and its continued upkeep. Further thanks to two anonymous reviewers.

632

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779

780 **APPENDICIES**

781 **Appendix 1. Derivation of the *SIR* model**

782 $$S_{\alpha,t} = \frac{aR_{\omega,t-1}}{1+\lambda R_{\omega,t-1}} \quad \textbf{(A1)}$$

783 $$S_{\omega,t} = (1-b)S_{\alpha,t}e^{-cI_{\omega,t}} \quad \textbf{(A2)}$$

784 $$E_{\alpha,t} = 0 \quad \textbf{(A3)}$$

785 $$E_{\omega,t} = (1-b)S_{\alpha,t}[1-e^{-cI_{\omega,t}}] \quad \textbf{(A4)}$$

786 $$I_{\alpha,t} = (1-d)(1-\beta e^{-\sigma I_{\alpha,t}})pE_{\omega,t-1} \quad \textbf{(A5)}$$

787 $$I_{\omega,t} = (1-b)(1-\beta e^{-\sigma I_{\alpha,t}})I_{\alpha,t} \quad \textbf{(A6)}$$

788 $$R_{\alpha,t} = (1-d)[(1-p)E_{\omega,t-1} + S_{\omega,t-1}] \quad \textbf{(A7)}$$

789 $$R_{\omega,t} = (1-b)R_{\alpha,t} \quad \textbf{(A8)}$$

790

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

By substituting in expressions to reduce the number of equations, and rearranging in terms of α and scaling from $t \rightarrow t+1$, it is possible to exclude α and ω terminology, and also make the equation for E obsolete as (at the beginning of the season second year plants are either I (with probability p) or R (with probability of 1-p).

Appendix 2. Derivation of the implicit expression for stable steady state values

Where the pathogen is able to invade it is possible to obtain implicit steady-state expressions for I^* by setting $I_{t+1}=I_t$ and solving (and dropping time subscripts).

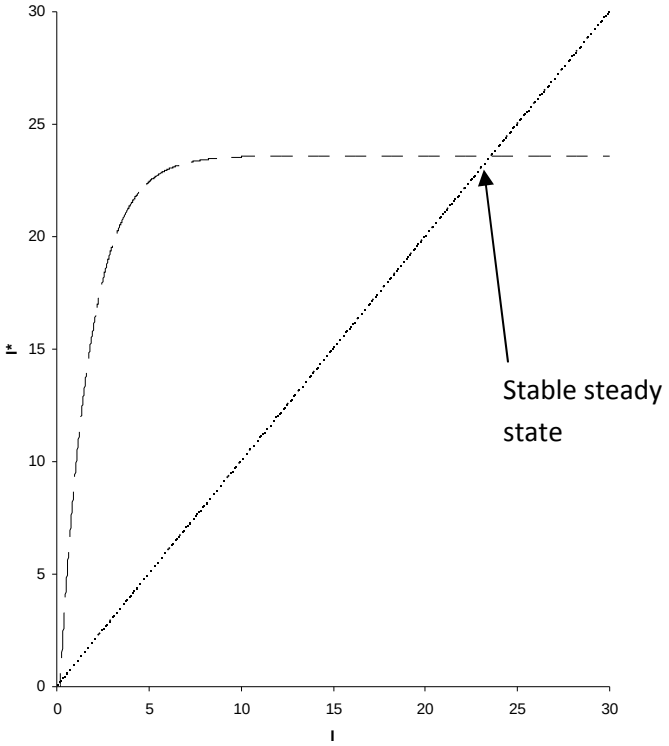
When pathogen-induced mortality is constant between and within seasons ($\sigma = 0$):

$$I^* = \frac{p(1-\beta)[1-e^{-c(1-b)(1-\beta)I^*}]}{\lambda(1-b)}\left\{a(1-b)^2(1-d) - \frac{1}{1-p[1-e^{-c(1-b)(1-\beta)I^*}]}\right\} \qquad \textbf{(A9)}$$

The solutions for I^* are obtained graphically by plotting both sides of the equation for given parameter values and determining the point of intersection.

Expressions for the other two forms of pathogen-induced mortality can be obtained in a similar way.

An example of solving stable-state values for I^* Solutions for constant disease-induced mortality between and within season, a=30, b=0.3, c=0.99, d=0.3, p=0.9, λ=0.5, β=0.1.



Appendix 3 Derivation of the second order recurrence relationship

Rearranging equation 1 for the system without disease, a recurrence relationship is obtained that represents the relationship between individuals in sequential years:

$$\frac{1}{S_{t+1}} = \frac{1+\lambda(1-b)R_t}{a(1-b)R_t} = \frac{\lambda}{a(1-b)} + \frac{1}{a(1-b)R_t} \qquad \textbf{(A10)}$$

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 an intercept $\frac{\lambda}{a(1-b)}$. Rearranging the equation for $R_{t+1} = (1-b)(1-d)S_t$ in terms of S_t, rescaling to t+1, and substituting in equation A10 gives:

$$\frac{1}{R_{t+2}} = \frac{1}{a(1-b)^2(1-d)R_t} + \frac{\lambda}{a(1-b)^2(1-d)}. \qquad \textbf{(A11)}$$

In the presence of disease

$$I_{t+1} + R_{t+1} = S_t(1-b)(1-d) \qquad \textbf{(A12)}$$

822 Re-arranging and re-scaling to t+1 gives:

823 $$\frac{1}{S_{t+1}} = \frac{(1-b)(1-d)}{I_{t+2} + R_{t+2}}$$

824

825 Inserting into equation A10, obtains the second-order recurrence equation:

826 $$\frac{1}{I_{t+2} + R_{t+2}} = \frac{1}{a(1-b)^2(1-d)R_t} + \frac{\lambda}{a(1-b)^2(1-d)} \qquad \textbf{(A13)}$$

827 Following the same procedure a recurrence relationship for the case where there is
828 pathogen-induced mortality is obtained.

829

830

831 **FIGURES**

832 Fig 1 Schematic representations of the compartmentalised SEIR model:

833

834 Fig 2a) The population dynamics of the mean number of infected second year plants
835 of *T. pratensis* recorded in plots with manure and/or fishmeal nutrient regimes (- -▲- -)
836 and chemical applications (—■—) demonstrating the outbreak nature of infected host
837 plants between 1995 – 2008. b) The estimated population sizes of healthy second year
838 *T. pratensis* between the years 1995 – 2008 with interpolated values for years 1999,
839 2000 and 2005.

840

841 Fig 3. Examples of simulation outcomes using the constant pathogen induced
842 mortality model demonstrating: a) healthy S, R and infected (I) plant populations
843 approach steady states monotonically for medium host performance and medium
844 pathogenicity; b) infected plants become extinct from the population for medium host
845 performance and low pathogenicity; and c) the host population becomes extinct for
846 high host performance and high pathogenicity. (—*S,* - - -I, …*R*). Corresponding
847 parameter values for the simulations are given in Table 2.

848

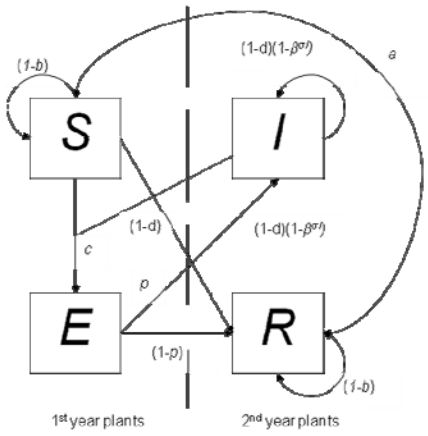
Fig 4. Illustrations of population cycles obtained when using parameters for high pathogenicity and medium host characteristics adapting the model to include: a) no pathogen induced mortality, b) constant pathogen disease induced mortality. c) variable pathogen induced mortality (—*S,* - - -I, …*R*). Corresponding parameter values for the simulations are given in Table 2.

Fig 5. The influence of altering *β, S* and the product *pc* on the invasion criterion. For simplicity *b*=*d* at a default value of 0.3.

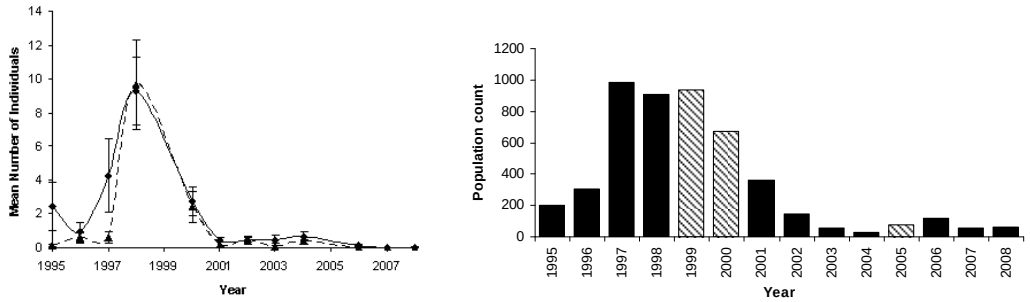
Fig 6. Values of *I** for varying values of a) *p* b) *c* and c) *β*. d) demonstrates a coloured contour plot demonstrating the region of steady-states and values for corresponding *pc* and *β*.

Fig, 7. The recurrence relationship fitted to observed data with the interpolated points (◇) for 1999 and 2000 indicating a linear inverse relationship between the number of healthy second year individuals (R_t) and the number of healthy and infected second year individuals two years later (R_{t+2}+I_{t+2})

Fig, 8. Simulation outcomes under high pathogenicity and low host performance without pathogen-induced mortality or seedling emergence density-dependency, producing an outcome similar to an "outbreak" dynamic (—*S,* - - -I, …*R*)

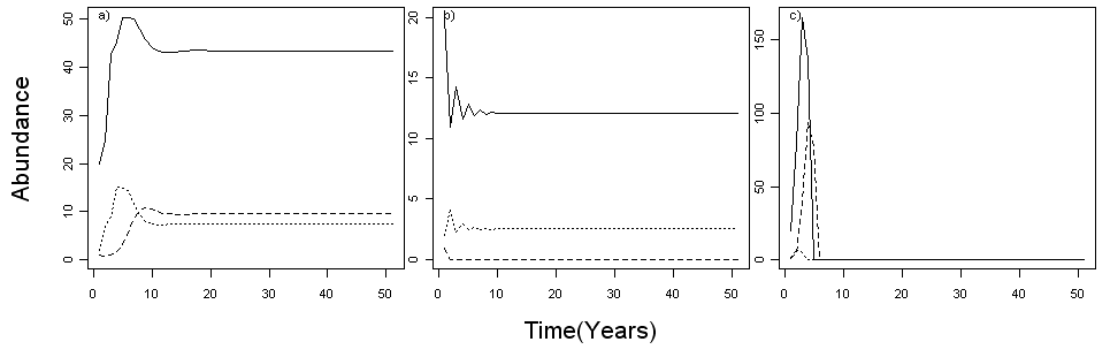


872 **Fig. 1.**

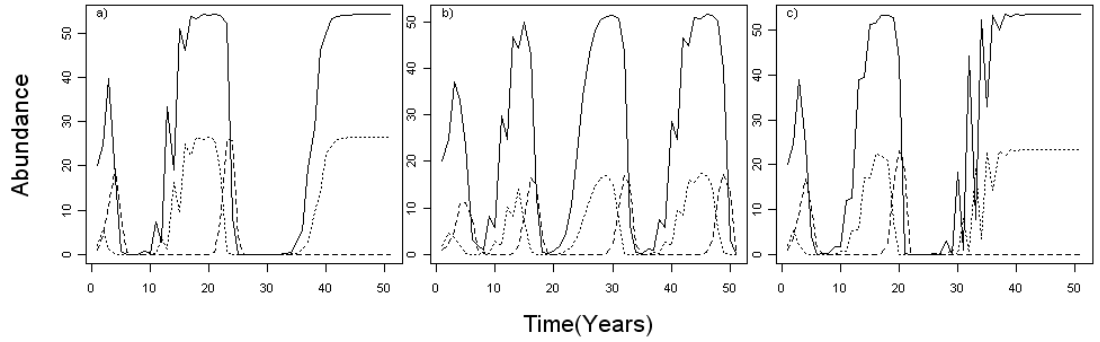


873

874 **Fig. 2**

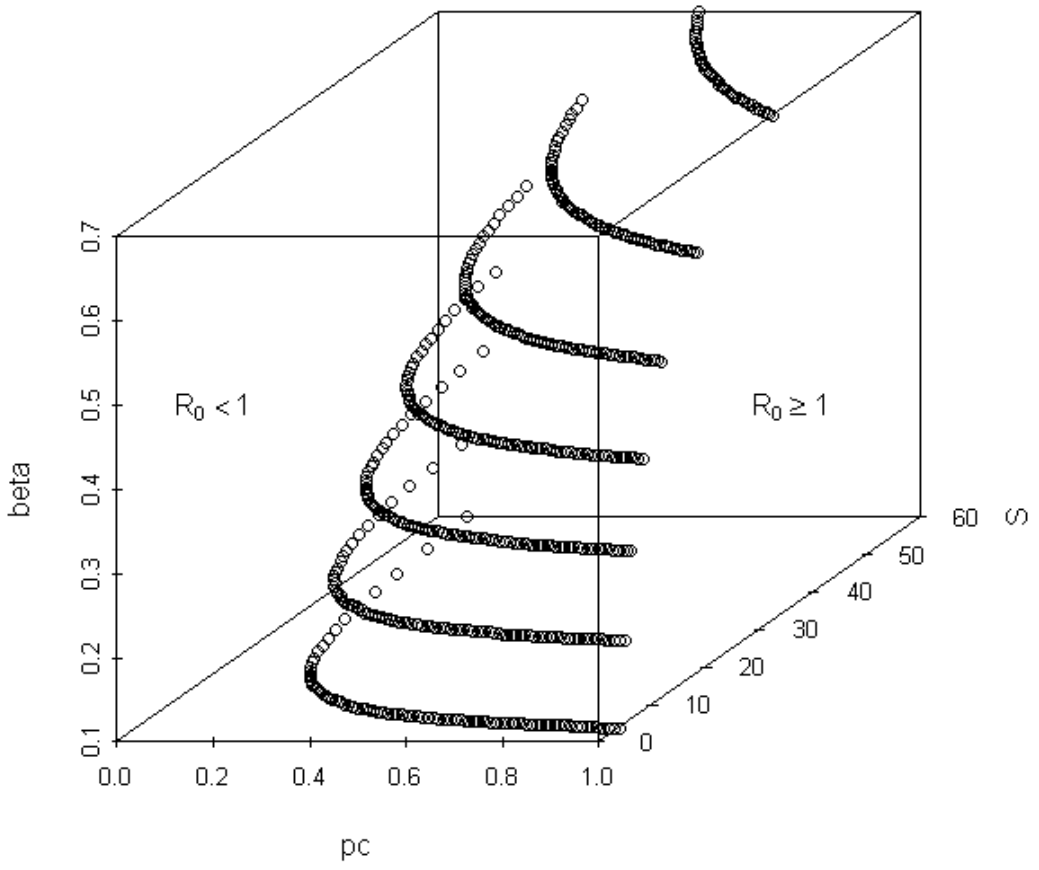


875
876 **Fig. 3**

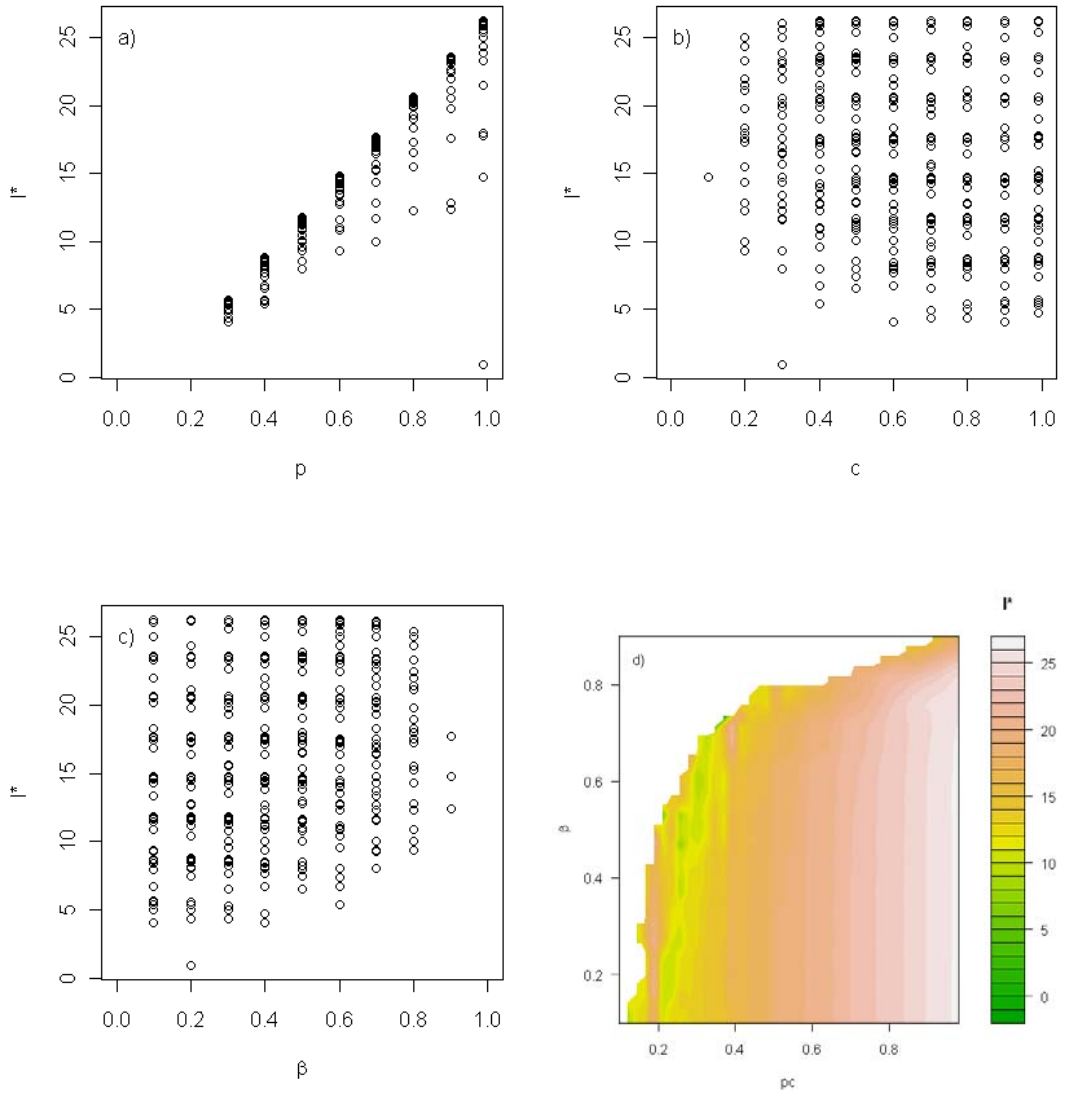


877

878 **Fig. 4**


beta
pc
S
$R_0 < 1$
$R_0 \geq 1$
0.1
0.2
0.3
0.4
0.5
0.6
0.7
0.0
0.2
0.4
0.6
0.8
1.0
0
10
20
30
40
50
60

879
880 **Fig. 5**



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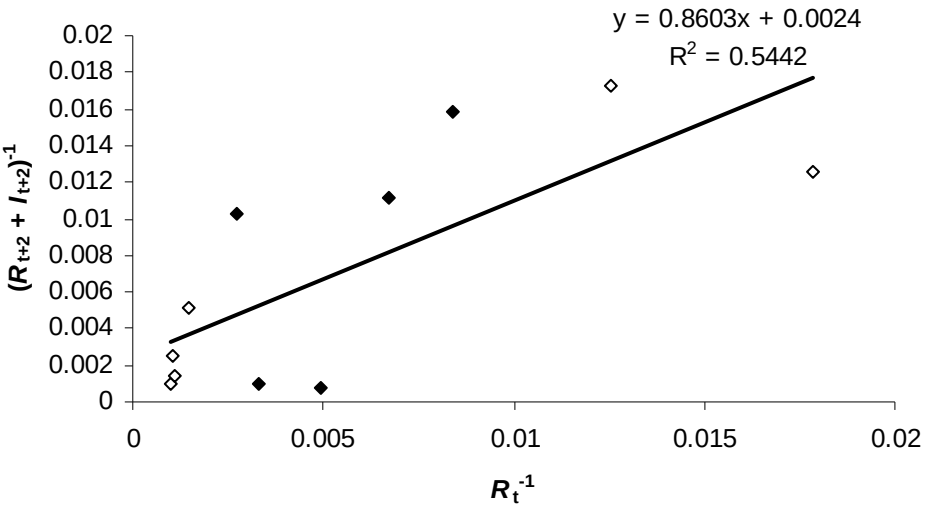
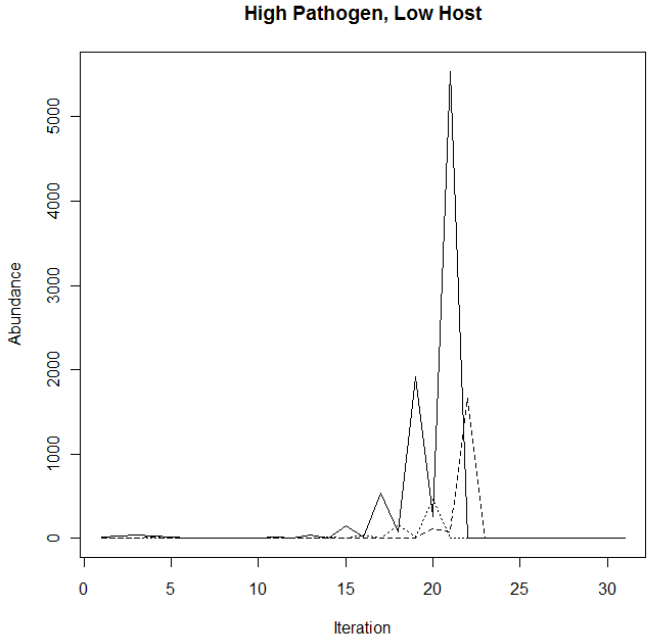
883 **Fig. 6**



884

885 **Fig. 7**

886



887

888 **Fig. 8**

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893 **TABLES**

894 **Table 1. Parameters and their value ranges used to assess the model system**

Parameter	Explanation	Default value	Source	Range Investigated
S_0	Initial number of 1st year susceptible individuals.	20	PGE survey	
E_0	Initial number of exposed 1st year individuals.	0	PGE survey	
I_0	Initial number of Infectious 2nd year individuals.	1	PGE survey	
R_0	Initial number of non-infectious 2nd year individuals.	2	PGE survey	
a	Seedling recruitment per non-infectious 2nd year individuals	30	*T. pratensis* seed set mean of 26.7 (Salama *et al.* 2010)	20 – 60
b	Within-season mortality rate	0.3	0.5 reported for *T. pratensis* (Mahesh, Upudhyaya & Turkington, 1996)	0.1 – 0.8
d	Between-season mortality rate	0.3	0.88 reported for *T. pratensis* (Mahesh, Upudhyaya & Turkington,	0.1 – 0.6

			1996)	
But c	Transmission rate probability	0.3	Variable depending on temperature for *P. lagenophorae* (Kolnaar & Van den Bosch, 2001)	0.1 – 0.8
p	Probability of exposed individuals becoming infectious	0.7	Greenhouse trials indicate a maximum of 18% of inoculated plants become infectious, however note there is no evidence that inoculated plants had sufficient exposure for pathogen uptake.	0.3 – 1.0
λ	Density-dependent parameter	0.5	PGE survey	0.2 – 1.0

Table 2. Parameter ranges for low, medium and high host performance and pathogenicity parameters. The pathogenicty parameters relate to the biology of the pathogen and its relationship with the host, whilst host performance is life-history parameters that are independent of pathogen presence

	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

Table 3. 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,)

β	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9
R^2	0.56	0.58	0.59	0.61	0.62	0.64	0.66	0.68	0.71
AIC	-56.3	-56.4	-56.5	-56.6	-56.7	-56.9	-57.0	-57.2	-57.5
m	0.86	0.87	0.87	0.88	0.89	0.90	0.91	0.93	0.95