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Summary

1 Soil-borne fungi are a major group of economically important plant pathogens, yet they have rarely been studied in the context of host–pathogen population biology. We develop general models of annual hosts and soil-borne fungal pathogens to explore the conditions for host–pathogen coexistence in both agricultural and natural plant populations. We use empirical data from the literature to parameterize and simulate dynamics with these models.

2 Initially we consider a simple system in which host density is assumed to be constant, as would be appropriate for agricultural systems. Model analysis shows that initial increase of the pathogen population requires that host (crop) density be above a threshold; this threshold decreases with increases in the pathogen's over-winter survival rate and ability to grow saprophytically.

3 A more complex model, in which both host and pathogen populations can vary, is needed for natural populations. Results from this model show that stable coexistence is possible even when the pathogen has a positive intrinsic growth rate (and therefore it is also possible for the pathogen to persist in the complete absence of the host).

4 Model parameter estimates were obtained from the empirical literature for two common and important soil pathogens: *Phytophthora* spp. and *Fusarium oxysporum*; these pathogens differ in several life-history features. Computer simulation showed that for *Fusarium*, there were substantial ranges for which coexistence or loss of the pathogen were predicted, while for *Phytophthora* most parameter estimates resulted in complete extinction when linear disease transmission was assumed; under the assumption of exponential disease transmission, predicted dynamics were most likely to lead to host persistence.

5 For both pathogens, within biologically realistic regions of parameter space, small changes in parameter values could lead to qualitatively different outcomes, including deterministic chaos, suggesting that long-term dynamics may be difficult to predict.

Keywords: *Fusarium*, host–pathogen coexistence, *Phytophthora*, saprophytic growth

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Introduction

After years of relative neglect, studies of the population dynamics of animal hosts and their pathogens in nature have increased dramatically in recent decades (e.g. Anderson & May 1978, 1981; May & Anderson 1978; Dobson 1988; Dobson & Roberts 1994). In contrast, such studies of plant host–pathogen systems are much less common. Although agricultural scientists have quantified within-season disease dynamics, the size of a crop 'population' in one year is largely independent of crop yield in the previous year and thus it is unlikely that these studies can be directly extended to inferences about pathogen effects on multiple year dynamics of unmanaged plant populations. Until recently, work on nonagricultural plant–pathogen interactions (reviewed by Burdon 1987; Alexander 1992; Roy 1994) have rarely explored questions relating to the long-term numerical dynamics of host and pathogen populations.

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Two major exceptions are the pollinator-transmitted floral smut *Ustilago violacea* and its host *Silene alba* (Alexander *et al.* 1996), and the flax rust *Melampsora lini* on the native Australian flax *Linum marginale* (e.g. Burdon & Jarosz 1991, 1992). In the case of the *Silene–Ustilago* system, the integration of empirical and theoretical approaches has resulted in extensive research on the population dynamics of host and pathogen, and the conditions necessary for their coexistence (Alexander & Antonovics 1988; Thrall *et al.* 1993; Thrall *et al.* 1995; Antonovics *et al.* 1994; Thrall & Antonovics 1995). The attributes of such floral smuts (infection causes sterility, and is persistent with relatively small effects on host mortality) have been shown to be very similar to sexually transmitted animal diseases (e.g. syphilis, gonorrhoea; Lockhart *et al.* 1996) but do not permit generalization to other types of plant diseases.

Soil-borne fungi are a major group of plant pathogens which are both ubiquitous in occurrence and cause significant yield loss in agriculture and forestry (Agrios 1988). Most studies of natural systems have ignored soil-borne fungi, although recent work (Augspurger 1983, 1984; Augspurger & Kelly 1984; Van der Putten *et al.* 1993; Bever 1994; Newsham *et al.* 1994) suggests that such organisms may have large effects on plant population size and community composition. This group is phylogenetically diverse and includes such major genera as *Verticillium*, *Phytophthora*, *Sclerotinia*, *Fusarium* and *Rhizoctonia*. Despite their taxonomic diversity, many of the soil-borne fungi share several important attributes which distinguish them from other fungal pathogens. These fungi produce a variety of resting structures (e.g. oospores, sclerotia, rhizomorphs, conidia) that often allow the pathogen population to persist in the soil without the host for many years or even decades (Baker & Snyder 1970; Bruehl 1987). Moreover, many soil fungi have the ability to grow saprophytically on organic debris in the absence of living hosts (Bruehl 1987).

In the present paper, we focus on the dynamics of annual plant host–pathogen systems; we develop general models that incorporate the basic features of both agricultural and natural plant populations. These models are developed with two specific goals. First, we derive analytical conditions for coexistence of host and pathogen populations. Secondly, we use estimates obtained from the plant pathology literature for two common and important soil pathogen systems that differ qualitatively in essential features of their life histories (e.g. the ability to grow saprophytically in the absence of the host), and for which we were able to obtain estimates of some critical model parameters. Through simulation, we evaluate the consequences of different assumptions about the nature of the disease transmission process (e.g. linear vs. exponential functions). We stress that our overall goal is to develop a conceptual basis for population biology studies of soil-borne pathogens and plants, as well as to illustrate the kinds of empirical data needed for dynamic studies of these interactions.

The models

Our models track changes in host and pathogen population density from the beginning of one growing season to the next. In temperate climates, population growth of soil-borne fungi occurs during summer months (through saprophytic growth and spore production, or through infection of new hosts), while during winter months, resting structures (e.g. chlamydiospores) can decay, but no new growth occurs. We assume that infected individuals can reproduce, but that the *per capita* rate of reproduction for infected individuals is less than that for healthy individuals. The equations determining the dynamics of this system are given by:

$$X_{t+1} = X_t[(1-P)(\Phi_h-\zeta_X X_t)+(\Phi_d-\zeta_X X_t)P] \quad (1)$$

$$Y_{t+1} = Y_t\alpha(1+\gamma-\zeta_Y Y_t)+\alpha\varepsilon X_t P \quad (2)$$

where P is the total probability of infection,

$$P = 1-e^{-\beta Y_t} \quad (3)$$

and the transmission parameter, β, is a phenomenological measure of 'transmission efficiency' (Anderson & May 1981). The parameters Φ_h and Φ_d represent the maximum per capita rates of reproduction for healthy and infected hosts, respectively (the assumption that infected hosts have lower fecundity than healthy hosts means $\Phi_d < \Phi_h$); ζ_X and ζ_Y are the strength of density dependent effects on the host and pathogen, respectively. The parameters α and γ are the over-winter survival rate of the pathogen in the soil and the host-independent birth rate ($\gamma > 0$ represents pathogens that can live as saprophytes, and $\gamma \leqslant 0$ represents those for which summer survival is lower than the rate at which new propagules are produced).

To simplify the analyses, we reparameterize the model given by eqns 1 and 2 in the following fashion. Let $\Phi = \Phi_h - 1$, $c = \Phi_h - \Phi_d$ (a measure of the cost of infection), and $\Theta = \alpha + \alpha\gamma - 1$ (the intrinsic rate of growth for the pathogen). If we also redefine $\varepsilon' = \varepsilon\alpha$ (the rate at which infected plants are converted to the pathogen component in the soil, adjusted for over-winter survival of the pathogen), and $\zeta'_Y = \alpha\zeta_Y$ (the effect of density dependence on saprophytic growth rates, adjusted for survival), then eqns 1 and 2 can be rewritten as:

$$X_{t+1} = X_t(1+\Phi-\zeta_X X_t - cP) \quad (4)$$

$$Y_{t+1} = Y_t(1+\Theta-\zeta'_Y Y_t)+\varepsilon' X_t P \quad (5)$$

For convenience, a list of symbols and definitions used in the models is given in Table 1.

Table 1 Definitions of model parameters and symbols

	Definition
X	density of host individuals
Y	density of pathogen in the soil
α	over-winter survival rate of the pathogen in the soil
γ	host-independent 'birth' rate (values > 0 represent soil pathogens that can live as saprophytes)
ζ_X	strength of density-dependent effects on the host birth rate
ζ_Y	strength of density-dependent effects on the pathogen birth rate
β	disease transmission coefficient
ε	rate at which infected plants are converted to the pathogen component in the soil
Φ_h	maximum per capita rate of reproduction for healthy hosts
Φ_d	maximum per capita rate of reproduction for infected hosts
c	measure of the cost of infection ($=\Phi_h-\Phi_d$)
Θ	the intrinsic rate of growth for the pathogen ($=\alpha+\alpha\gamma-1$)
ε'	the rate at which infected plants are converted to the pathogen component in the soil, adjusted for over-winter survival of the pathogen ($=\alpha\varepsilon$)
ζ'_Y	the effect of density-dependence on saprophytic growth rates, adjusted for survival ($=\alpha\zeta_Y$)

MODEL ANALYSIS

As assumed in eqn 3, the probability of infection is likely to be nonlinear (Gregory 1948; van der Plank 1975; Thrall *et al.* 1995). For example, as the proportion of infected individuals increases, the rate at which the remaining susceptible individuals become infected must decrease; this upper limit is set by the general condition that disease prevalence cannot exceed unity. However, as demonstrated by Antonovics *et al.* (1995), when the frequency of transmission events is low (i.e. when disease is rare), this nonlinear form can be approximated by a linear transmission term such that

$$P = \beta Y_y \tag{6}$$

We make this assumption for analytical tractability, and to relate the present study to existing theory on host–pathogen population dynamics (e.g. Anderson & May 1981, 1991; Getz & Pickering 1983).

To link the current study to agricultural plant-pathogen systems, we begin by briefly describing the dynamics of a soil-borne pathogen when the density of plants is 'reset' each year to a constant number, and therefore plant population size at the beginning of the year is independent of pathogen population size in the soil (Model I). This case best describes an annual crop with the density of seeds planted in the spring being independent of yield in the previous harvest. Secondly, we consider the case in which both host and pathogen population sizes are dynamic variables; this is most representative of natural plant host–pathogen systems (Model II). In both cases, we focus on conditions for pathogen increase when rare; this is when the linear approximation to disease transmission will be most accurate.

MODEL I. PATHOGEN DYNAMICS ON AN AGRICULTURAL HOST

If we assume that the number of hosts (X) is constant, but that pathogen density in the soil (Y_t) is a dynamic variable, and further that there can be density-dependent regulation of pathogen growth independent of the host, then the dynamics of the pathogen are as represented by eqn 2. As discussed above, we assume that the probability of infection (P) takes the linear form given by eqn 6.

Increase of the pathogen population when rare (i.e. $Y_t \approx 0$) requires that the host crop density (X) be above a minimum population density; this threshold for initial increase is given by

$$X > \frac{1-\alpha-\alpha\gamma}{\alpha\varepsilon\beta} \tag{7}$$

If over-winter survival rate of the pathogen is high, then even a small amount of saprophytic growth will eliminate the threshold, and the pathogen will always increase. Such saprophytic growth is likely to be regulated by density-dependent factors and in this case, a positive equilibrium (Y^*) is possible:

$$Y^* = \frac{\alpha(1+\gamma+\varepsilon\beta X)-1}{\alpha\zeta_Y} \tag{8}$$

The model represented by eqn 2 is a simple form of the logistic equation, with the intrinsic rate of growth, $r, = \alpha(1+\gamma+\varepsilon\beta X)-1$. As shown by May (1974), there will be nonoscillatory convergence to the equilibrium given by eqn 8 for $0 < r < 1$; when $1 < r < 2$, convergence will be oscillatory, and for progressively higher values of the intrinsic rate of growth, the equilibrium will become unstable and dynamics will consist of limit cycles and then chaos. Stone & Ezrati (1996) recently reviewed the evidence for cyclical and chaotic behaviour in plant populations, and conclude that such dynamics are likely in nature.

It is of particular interest to ask how different agricultural practices might affect the dynamics of soil pathogens. For example, in many areas, fields are allowed to lie fallow for one or more growing seasons in a regular cycle. In the simplest case, we assume that in fallow years there are no suitable hosts available

and there is no density-dependent regulation of the pathogen. Then eqn 1 can be generalized to

$$Y_{t+1} = Y_t[1+\gamma+\varepsilon\beta X_t]\alpha^i(1+\gamma)^{i-1} \quad (9)$$

where the parameter i represents the number of years in the rotational cycle (e.g. i = 3 would mean 3 years from crop to crop, with 2 fallow years between). The condition for initial increase then becomes

$$X > \frac{1-\alpha^i(1+\gamma)^i}{\varepsilon\beta\alpha^i(1+\gamma)^{i-1}} \quad (10)$$

As in eqn 7 above, this threshold density decreases as over-winter survival and host-independent growth rates increase. Not surprisingly, increasing the rotational cycle (i) makes it increasingly difficult for the pathogen to increase. This result would also apply to situations where crop rotation is practiced (i.e. no fallow period), and the pathogen in question is host-specific.

MODEL II. HOST–PATHOGEN DYNAMICS IN NATURAL SYSTEMS

If both host and pathogen abundances can vary, then dynamics are represented by eqns 1 and 2. We again make the simplifying assumption that disease transmission is linear (see above). Increase of the pathogen population when rare requires that

$$\Theta > \frac{-\varepsilon'\beta\Phi}{\zeta_X} \quad (11)$$

If the inequality given by (11) is violated, then the pathogen will decrease and (provided that $0 < \Phi < 2$) the host density will show global convergence to its carrying capacity (Φ/ζ_X). Increase of the host population when rare, requires

$$\Phi > \frac{c\beta\Theta}{\zeta'_Y} \quad (12)$$

In this case, however, it is also possible for the pathogen to persist indefinitely in the complete absence of the host. If inequality 12 is violated, and $0 < \Theta < 2$, the pathogen will converge to its carrying capacity (Θ/ζ'_Y). Because the eigenvalues determining stability are real for both boundary equilibria, whenever convergence occurs, it is always nonoscillatory.

If inequalities 11 and 12 are met, then there will be a single internal equilibrium given by

$$X^* = \frac{\zeta_Y - c\beta\Theta}{\zeta_X\zeta'_Y + c\varepsilon'\beta^2} \quad (13)$$

$$Y^* = \frac{\varepsilon'\beta\Phi + \zeta_X\Theta}{\zeta_X\zeta'_Y + c\varepsilon'\beta^2} \quad (14)$$

Combining inequalities 11 and 12, the overall condition for positivity of the internal equilibrium is

$$\frac{-\varepsilon'\beta\Phi}{\zeta_X} < \Theta < \frac{\zeta'_Y\Phi}{c\beta} \quad (15)$$

In this case, biologically valid values (X^*, $Y^* > 0$) of the internal equilibrium described by eqns 13 and 14 are possible with either positive or negative values of Θ (the above inequality also implies that $\Phi > \Theta$ must be true).

Based on the general criteria for local stability of a two-dimensional system of difference equations (Edelstein-Keshet 1988), analysis of eqns 4 and 5 indicates that there are four conditions determining stability of the internal equilibrium given by eqns 13 and 14; these conditions are complicated, and depend on the relative strength of density dependence in the host and pathogen. The biology of this situation (Model II: density-dependent growth in both host and pathogen) is summarized by the isoclines associated with eqns 4 and 5; these are depicted graphically in Fig. 1.

Additional analyses are possible however, if we make a further assumption that saprophytic growth in the pathogen is not subject to density regulation ($\zeta'_Y = 0$). In nature, this assumption will be most representative for nonsaprophytic soil pathogens (i.e. when $\gamma = 0$, and thus $\Theta < 0$). Note that in this case, the pathogen will be unable to persist in the continued absence of the host. Stability analysis shows that the eigenvalues governing this system are complex, and therefore all solutions are oscillatory. Thus, the question of stability reduces to a single condition on Θ, given by

$$\Theta < 1 - \frac{\varepsilon'\beta\Phi}{\zeta_X} \quad (16)$$

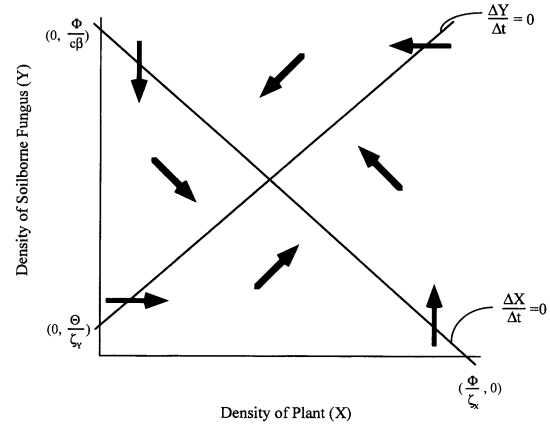

Fig. 1 Interactions between densities of host and pathogen. The X and Y isoclines are the lines on which the rate of change of X and Y are zero [$X^* = (\Phi - c\beta Y)/\zeta_X$, and $Y^* = (\Theta + \varepsilon'\beta X)/\zeta'_Y$ respectively]. The dynamics of the system can be inferred by mapping the isoclines and the direction of change in the host and pathogen population sizes. Beginning in the lower right quadrant, the density of Y is below the X isocline, therefore X increases. When the density of X increases above the Y isocline, Y increases. As the density of Y approaches and then passes the X isocline, the rate of increase of X decreases to a negative value. The dynamics are cyclical, with the amplitude of the cycles decreasing to zero when the equilibrium point is stable. Note that as the intrinsic growth rate of the pathogen (Θ) increases, the equilibrial density of the host decreases, while the equilibrial density of the pathogen increases with increasing intrinsic growth rates of the host (Φ).

If inequality 16 is satisfied, then host and pathogen show oscillatory convergence to the equilibrium given by eqns 13 and 14. If violated, then the internal equilibrium still exists, but is unstable. In conclusion, the internal equilibrium exists and is stable when Θ lies within the range

$$\frac{-\varepsilon'\beta\Phi}{\zeta_X} < \Theta < 1 - \frac{\varepsilon'\beta\Phi}{\zeta_X} \quad (17)$$

The density of the host and pathogen will oscillate out of phase, with the amplitude of oscillations either decreasing or increasing, depending on whether stability conditions are met.

Simulation studies

Although we assume linear disease transmission in the analytical models, we relax this restriction for the simulation studies; we present results for both types of transmission for comparative purposes. We also use simulations of eqns 4 and 5 (Model II) to investigate the effects of variation in life history parameters such as saprophytic ability and costs of infection, on the predicted dynamics.

We focus on two specific genera of fungal pathogens, *Fusarium* and *Phytophthora*. These genera were chosen for several reasons. First, both genera contain species that are common and important pathogens. Secondly, these pathogens differ qualitatively in several aspects of their biology as represented by the models described above. For example, *Fusarium oxysporum* causes wilt disease in a wide variety of host plants. We focus on the species *F. oxysporum* because aerially dispersed spores are of minor importance as primary inoculum (Nelson 1981) and sexual spores are unknown (Nelson *et al.* 1983). In general, *Fusarium* spp. persist as chlamydospores, macroconidia, and/or as mycelium in organic debris in the soil. In contrast, *Phytophthora* spp. cause root and/or stem rot in a wide range of hosts. *Phytophthora* spp. survive in the soil as oospores (the sexual state), as sporangia (where winter temperatures are mild) or as a mycelium in plant debris. Many species of *Fusarium* are able to grow saprophytically (usually defined as the ability to grow through the soil to compete for access to decaying plant material), while many species of *Phytophthora* are comparatively poor saprophytes (Weste 1983b). Finally, for both of these pathogens, sufficient information was available from the published literature to estimate several critical model parameters.

Our literature work was not intended to be exhaustive; the goal was to provide a sampling of values from the literature that would define biologically reasonable numerical ranges of parameter values. In the case of *Fusarium oxysporum* (Table 2), there were insufficient data to provide a range of parameter estimates from any single *forma specialis* (host-specific subgroup); we therefore used all available data, regardless of subspecies (in subsequent discussion, we refer to *F. oxysporum* as *Fusarium*). For *Phytophthora*, we were unable to obtain sufficient information on any one species, and therefore considered the genus as a whole (Table 3). We note that for both species, most of the available data comes from field and/or greenhouse studies using agricultural hosts. Currently, such studies provide the only estimates of critical model parameters; we recognize, however, that estimates derived from natural host–pathogen populations may be quite different.

ESTIMATION OF MODEL PARAMETERS

The inclusion of multiple studies in our literature review made parameter estimation difficult as the units of measurement used were frequently different in different studies (e.g. Weste 1983a used chlamydospores/root system of seedlings, which could not easily be converted to the appropriate units). In all cases, it was important to ensure that model units were consistent for all parameters [we used fungal propagules per gram of soil (ppg)]; estimation of several parameters therefore required additional assumptions in order to convert data into the appropriate units of measure.

Conversion rate of infected plants to pathogen in the soil (ε)

In some cases (e.g. Graham 1995), data were given as propagules per cubic centimetre (in the soil). We estimated the average weight of soil cm^{-3} (laboratory measurements gave a value of $1.6\,g\,cm^{-3}$), and used this to convert such data to get ε estimates in units of ppg. For *Fusarium*, values were typically in terms of ppg roots; we used a value of $0.01\,g$ roots g $soil^{-1}$ (J. Bruhn, personal communication) for purposes of converting estimates into ppg soil. Estimates of ε ranged from 0.46–736.0 for *Fusarium*, with a mean of 106.5 and a median of 14.6. For *Phytophthora*, estimates ranged from 0.18–16.2, with a mean of 7.5, and a median value of 7.25. Values for ε' (the corrected conversion rate) were obtained by multiplying estimates of ε by the estimated values of α (see below).

Overwinter survival rate (α)

There are two general types of literature that pertain to estimation of this parameter. In some studies (e.g. Elmer & Lacey 1987a), a known number of resting structures of a single age were placed into different soil environments, and the rate of degradation of these cohorts was then followed through time. In other studies (e.g. Banihashemi & deZeeuw 1975), the number of resting structures was counted in field plots throughout the growing season and winter months for temperate crops; in both kinds of studies decay rates are calculated as the ratio of propagules present

Table 2 Parameter estimates for *Fusarium oxysporum*. See text for discussion of parameter estimation, and Table 1 for symbol definitions. Note that β_L and β_E represent linear and exponential estimates of the disease transmission parameter, respectively

Forma specialis	Host		Estimate	Reference
apii	celery	ε'	susceptible cultivar: 614 ppg (feeder roots); 1610 ppg (primary roots); moderately resistant cultivar: 419 ppg (feeder roots); 1460 ppg (primary roots);	Elmer & Lacey (1987b)
melonis	melon	ε'	32 000 ppg (fresh weight)	Gordon *et al.* (1989)
spinaciae	spinach	ε'	73 600 ppg	Reyes (1979)
unspecified	red clover	ε'	per gram root: 1000, 6300, 2800; per root: 100, 500 1900	Nan *et al.* (1991)
melonis	melon	α	0.3297	Banihashemi & deZeeuw (1975)
apii	celery	α	0.388, 0.4	Elmer & Lacey (1987a)
melonis	melon	Θ	−0.98	Banihashemi & deZeeuw (1975)
apii	celery	Θ	−0.613	Elmer & Lacey (1987a)
apii	celery	β_L	0.0134 ($r^2 = 0.973$)	Elmer & Lacey (1987a)
		β_E	0.0261 ($r^2 = 0.992$)	
batatas	sweet potato		2 data sets:	Smith & Snyder (1971)
		β_L	0.0002 ($r^2 = 0.945$), 0.0001 ($r^2 = 0.594$)	
		β_E	0.0008 ($r^2 = 0.993$), 0.0028 ($r^2 = 0.993$)	
dianthi	carnation	β_L	3 experiments: 0.0002 ($r^2 = 0.987$), 0.0002 ($r^2 = 0.949$), 0.0001 ($r^2 = 0.810$),	Ben-Yephet *et al.* (1994)
		β_E	0.0003 ($r^2 = 0.980$), 0.0005 ($r^2 = 0.983$), 0.0002 ($r^2 = 0.904$)	
radicis-lycopersici	tomato	β_L	0.0244 ($r^2 = 0.788$)	Marois & Mitchell (1981)
		β_E	0.0744 ($r^2 = 0.987$)	
pisi	pea		2 experiments:	
		β_L	0.0001 ($r^2 = 0.520$), 0.0001 ($r^2 = 0.732$)	Guy & Baker (1977)
		β_E	0.0019 ($r^2 = 0.978$), 0.0006 ($r^2 = 0.981$)	
apii	celery	c/Φ	estimates for several cultivars in two years: 0.652, 0.670, 0.572, 0.648, 0.774, 0.557, 0.418, 0.539, 0.556, 0.422, 0.606, 0.287	Cerkauskas & Chiba (1991)
dianthi	carnation	c/Φ	estimates for 4 experiments: 0.188, 0.234, 0.361, 0.684	Ben-Yephet *et al.* (1994)
melonis	melon	c/Φ	estimates for 2 treatments in two years: 0.757, 0.826, 0.591, 0.578	Gordon *et al.* (1990)

at the beginning of the growing season to the propagules present at the end of the previous growing season. For *Fusarium*, estimates ranged from 0.3297–0.400, with a mean of 0.373 and a median value of 0.388. For *Phytophthora*, values ranged from 0.0014–0.715; the mean was 0.102, and the median was 0.0638.

Pathogen growth rate in the soil (Θ)

Estimates of Θ were obtained from the yearly change in propagule density per unit soil in field plots where host plants were absent. We were unable to obtain such estimates for *Phytophthora*. For *Fusarium*, values ranged from −0.98 to −0.613, with a mean and median of −0.797.

Disease transmission parameter (β)

Our analysis of models I and II assumed that the proportion of newly infected hosts was a linearly increasing function of inoculum density (βY_t). Under this assumption, estimates of β were calculated from linear regressions of untransformed data (regression data was proportion diseased vs. inoculum density, with inoculum density expressed as ppg soil). For estimates of β under nonlinear disease transmission, we assumed that the probability of infection $= 1 - \exp[-\beta Y_t]$. For both linear and nonlinear transmission models, the point (0,0) was included in the regression data. Linear estimates of β: For *Fusarium*, values ranged from 0.0001–0.0244, with a mean of 0.0043 and a median of 0.0002. For *Phytophthora*, values ranged from 0.0076–0.128; the mean was 0.0493, and the median was 0.043. Non-linear estimates of β: For *Fusarium*, values ranged from 0.0002–0.0744, with a mean of 0.012 and a median of 0.0008. For *Phytophthora*, values ranged from 0.051–5.958, with a mean of 1.25 and a median of 0.69. Simulations were run substituting either the linear or nonlinear form for the disease transmission function into eqns (4) and (5).

Table 3 Parameter estimates for *Phytophthora*. See text for discussion of parameter estimation, and Table 1 for symbol definitions. Note that β_L and β_E refer to linear and exponential estimates of disease transmission, respectively

Species	Host		Estimate	Reference
parasitica var. nicotianae	several citrus varieties	ε'	from 2 greenhouse experiments (ppg/root): 2600, 1600, 1600, 3200, 900, 3000, 12300, 1500, 4100 from field experiment: 60, 440, 10, 380, 260, 3390	Graham (1995)
cinnamomi	several tree spp.	α	in forest soil, 3 locations over 2 years: 0, 0.05, 0.091, 0.011, 0.10, 0.23	Weste & Vithanage (1978)
	Fraser fir	α	nursery soil: 0.0014	Kenerley & Bruck (1983)
	clover	α	nursery soil: 0.0168, 0.1091	Kenerley & Bruck (1983)
	rye	α	nursery soil: 0.0278, 0.0776	Kenerley & Bruck (1983)
	fallow	α	nursery soil: 0.0268	Kenerley & Bruck (1983)
parasitica	several citrus varieties	α	sporangia + zoospore cysts: 0.715	Lutz & Menge (1991)
parasitica var. *nicotianae*	tobacco	α	3 varieties, 2 experiments: 0.10, 0.175	Flowers & Hendrix (1972)
capsici	peppers	β_L	4 experiments: 0.0076 ($r^2 = 0.472$), 0.0087 ($r^2 = 0.764$), 0.0087 ($r^2 = 0.730$), 0.0094 ($r^2 = 0.911$),	Bowers & Mitchell (1991)
		β_E	0.421 ($r^2 = 0.999$), 0.098 ($r^2 = 0.991$), 0.109 ($r^2 = 0.999$), 0.051 ($r^2 = 0.999$)	
citrophthora	milkweed vine	β_L	0.0784 ($r^2 = 0.535$)	Mitchell (1978)
		β_E	1.440 ($r^2 = 0.998$)	
palmivora	papaya	β_L	0.0767 ($r^2 = 0.65$)	Mitchell (1978)
		β_E	0.957 ($r^2 = 0.976$)	
palmivora	papaya	β_L	0.0767 ($r^2 = 0.66$)	Ramirez & Mitchell (1975)
		β_E	0.961 ($r^2 = 0.974$)	
parasitica var. *nicotianae*	tobacco	β_L	0.128 ($r^2 = 0.307$)	Kannwischer & Mitchell (1981)
		β_E	5.958 ($r^2 = 0.997$)	
infestans	potato	c/Φ	field expts in Ontario 1953–70: 0.52, 0.42, 0.36, 0.34, 0.33, 0.33, 0.31, 0.26, 0.24	James *et al.* (1972)
megasperma	alfalfa	c/Φ	several cultivars, 2 years: 0.48, 0.69, 0.54, 0.69, 0.67, 0.53, 0.57, 0.43, 0.58	Faris & Sabo (1981)
megasperma f. sp. *glycinea*	soybean	c/Φ	using isolines: −0.098, 0.168, 0.281, −0.081, 0.046, −0.016, 0.065	St. Martin *et al.* (1994)
megasperma f. sp. *glycinea*	soybean	c/Φ	using isolines, mean of 2 years, 2 sites: 0.295, 0.149, 0.081	Moots *et al.* (1988)
megasperma f. sp. *glycinea*	soybean	c/Φ	using near isolines, several years: 0.169, 0.569, 0.4, 0.651, 0.463	Anderson (1986)
megasperma f. sp. *glycinea*	soybean	c/Φ	several cultivars, fungicide trial: −0.251, 0.132, 0.021, 0.019, 0.18, 0.547	Ryley *et al.* 1994
parasitica var. *nicotianae*	tobacco	c/Φ	3 cultivars, 2 years: 0.14, 0.16, 0.10, 0.19, 0.19	Johnson (1991)

Population growth rate of healthy/infected plants (Φ_h, Φ_d)

While it was difficult to obtain direct estimates of growth rates for infected vs. healthy plants, we were able to estimate the proportion of yield loss due to disease (e.g. a tabular value of 0.15 represented a 15% yield loss). Note that these estimates focus only on the harvestable portion of the plant (whatever that might be). For purposes of doing the computer simulations, we assumed that $\Phi_h = 2.0$ (Thrall & Jarosz 1994; Thrall & Antonovics 1995), and then calculated values of Φ_d from estimates of yield loss due to infection. The cost of infection c was then calculated simply as the difference, $\Phi_h - \Phi_d$ (i.e. the difference in growth rate due to infection). For *Fusarium*, the mean percentage reduction ranged from 0.188–0.826, with a mean of 0.546 and a median of 0.575. For *Phytophthora*, values ranged from 0.019–0.69; the mean was 0.320 and the median was 0.332. We note that use of average yield loss to estimate the cost of infection may underestimate such effects because of compensation.

Density dependence (ζ_X, ζ'_Y)

For the simulations, we assumed that $\zeta_X = 0.004\,m^2$ plant^{-1} (P. H. Thrall & J. Antonovics, unpublished data for *Silene alba*). This value, in combination with our estimate of Φ_h, results in a disease-free carrying capacity of 500 plants. No data were available for estimates of the strength of density dependence in either *Fusarium* or *Phytophthora*; therefore, the value of Φ'_Y used in the simulations was calculated as $\alpha\zeta_X$ (see Model section).

SIMULATION RESULTS

In the computer simulations we assumed that pathogen growth rate in the soil could be either negative (nonsaprophytic: $= -0.75$) or positive (saprophytic: $= 0.75$). Note that for Θ, the only estimates we were able to obtain were for *Fusarium* and these were negative; positive growth in the soil seems likely given the capacity for saprophytic growth (at least for *F. oxysporum*). We also assumed that the cost of infection c could either be low ($c = 0.5$) or high ($c = 1.5$) (broadly corresponding to two pathogen strategies: 'debilitators' vs. 'killers' *sensu* Burdon 1991, 1993). These values were within estimated ranges for *Fusarium* and *Phytophthora*. We chose to use two contrasting values for these parameters as we had the least information about actual ranges from empirical data, and the values we used represented qualitatively different life histories. Maps of dynamical outcomes were produced by systematically varying the transmission parameter (β) along one axis, and the adjusted conversion rate (ε') along the other, and assuming one of the four fixed parameter combinations for Θ and c. For both β and ε', the values investigated ranged from an order of magnitude below to an order of magnitude above the estimated median value (we used the median rather than the mean as it was less likely to be affected by outlying values). We chose to map the dynamics in $\beta - \varepsilon'$ space as these parameters jointly determine the pathogen population growth rate through infection of the host.

For each combination of parameter values, eqns 4 and 5 were iterated until equilibrium (or 1000 generations), and points plotted by colour, depending on the dynamical outcome (Figs 2–5). All simulations assumed an initial value of 100 for the numbers of hosts, and 1 for the number of pathogens; these initial values correspond to our analytical investigation of the conditions for initial increase of the pathogen population.

Overall, the simulations showed that within the ranges of parameter values considered, several dynamical outcomes were possible: extinction of both host and pathogen, persistence of host only, persistence of pathogen only, stable coexistence of host and pathogen, and cyclical or chaotic dynamics. The predicted dynamics were complicated in that small changes in parameter estimates often resulted in qualitatively different dynamics (e.g. Figs 2 and 4). Moreover, the boundaries between dynamic regions were often complex, and of an apparently fractal nature (magnification of these regions showed similar structure, in that smaller regions appeared where other outcomes were possible; Gleick 1987).

Under the assumption of exponential disease transmission, for *Fusarium*, there were substantial parameter ranges for which either coexistence or loss of the pathogen were predicted (Fig. 2), while for *Phytophthora* most parameter values resulted in coexistence of host and pathogen (Fig. 3). For positive values of Θ and high costs of infection, there were small regions for *Phytophthora* and *Fusarium* where the pathogen was predicted to persist even after host extinction (Figs 2b and 3b). Oscillatory or chaotic dynamics predominated with negative values of Θ and high costs of infection for *Phytophthora* (Fig. 3a,b). In general, the dynamics appeared more sensitive to changes in costs of infection than changes in the pathogen saprophytic ability.

While in some cases the general structure of the dynamical maps were unchanged by the assumption of linear disease transmission (compare Fig. 2b,d with Fig. 4b,d), there were often dramatic differences in the outcomes predicted for specific parameter combinations (compare Figs 3 and 5). For example, in contrast to the broad range of conditions in which host persistence was predicted under exponential disease transmission, under the assumption of linear disease transmission, the host was often predicted to go extinct. This contrast was particularly evident for *Phytophthora*. Overall, for both *Fusarium* and *Phytophthora*, going from the exponential to the linear transmission model, there was a decrease in the size of regions where the pathogen was predicted to be lost (as for *Fusarium*) or where coexistence of both host and pathogen occurred (as for *Phytophthora*).

We did not systematically examine the effects of changing either the strength of density dependence (ζ_X, ζ'_Y), or the growth rate for healthy plants (Φ). However, in simulations where we varied these parameters, smaller values of density dependence generally resulted in a compression of the dynamical maps shown in Figs 2–5 down and to the left (regions of extinction increased relative to regions of coexistence); larger values compressed the maps up and to the right (regions of extinction decreased relative to regions of coexistence). Therefore, changing the strength of density dependence in the pathogen relative to the host changes the range within which coexistence is predicted. There seemed to be only small quantitative, rather than qualitative differences, in the predicted outcomes when host growth rate was varied (even for relatively large changes, e.g. $\Phi = 2.5$, rather than the generally assumed value of 2.0).

Discussion

A number of recent studies have shown the importance of soil communities for the dynamics of plant communities and vice-versa (e.g. Augspurger 1983, 1984; Bever 1994; Bever *et al.* 1996; Van der Putten *et al.* 1993). Further, many classic examples of important forest diseases are caused by soil-borne fungi (e.g. *Phytophthora cinnamomi* causing jarrah dieback in western Australia: Weste 1974; Weste *et al.* 1976; *Armillaria* spp. causing root rot: Shaw & Kile 1991; *Ceratocystis fagacearum* causing oak-wilt: Appel & Billings 1995). Clearly, the importance of soil patho-

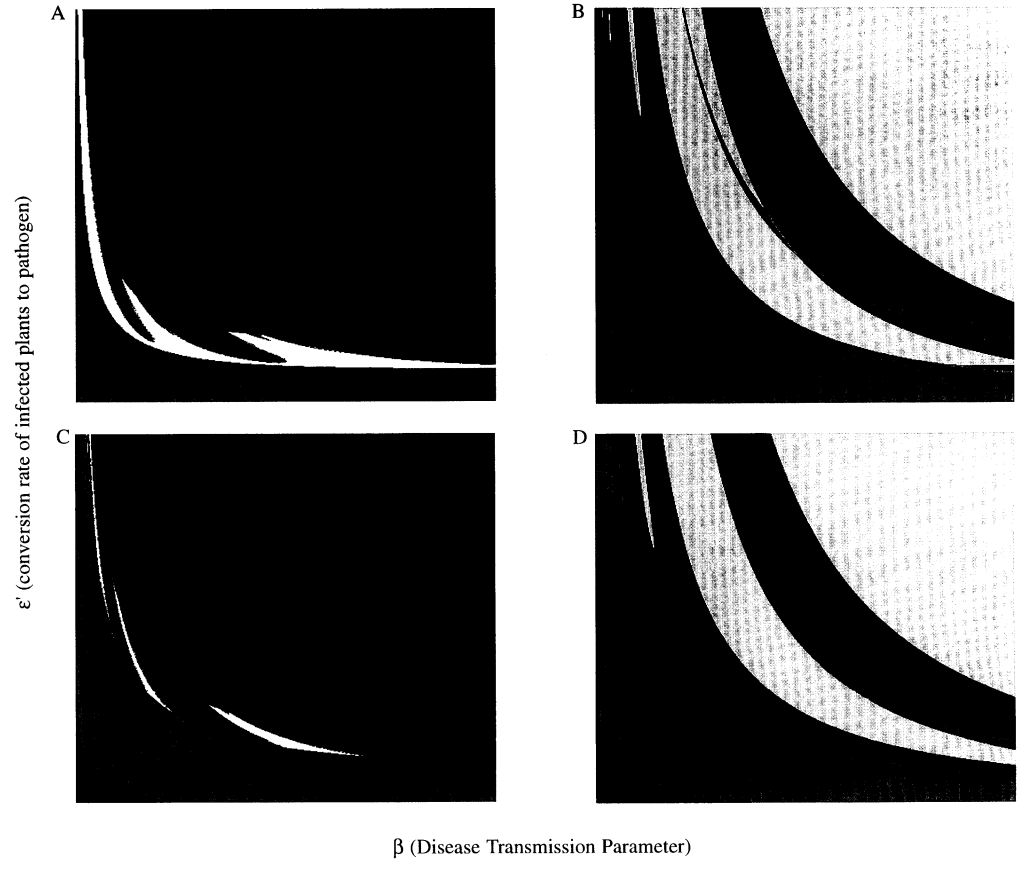

Fig. 2 Predicted dynamics for *Fusarium oxysporum* under the assumption of exponential disease transmission, and for fixed values of the cost of infection (c) and the pathogen growth rate (Θ). For all dynamical maps, values of the transmission parameter, β, are plotted on the x-axis, and values of the conversion parameter, ε', are plotted on the y-axis. For both β and ε' the range of values explored included 1 order of magnitude above and below the medians estimated from the literature (β values ranged from 0.00008 to 0.008; ε' values ranged from 0.5 to 50.0). With respect to the maps, the cost of infection increases from the left pair of maps to the right pair (c = 0.5, 1.5 respectively), and the host-independent pathogen growth rate increases from the bottom pair of graphs to the top pair ($\Theta = -0.75$, 0.75 respectively). On all maps, the grey-scale shades are: medium grey = stable coexistence of host and pathogen, white = stable limit cycles or chaos, dark grey = persistence of host only, black = persistence of pathogen only, light grey = extinction of both host and pathogen.

gens in agricultural situations is also broadly recognized; longstanding agricultural practices such as crop rotation and letting fields lie fallow are, in part, due to an implicit recognition by farmers of the need to control the buildup of pathogens in the soil. However, in general, perhaps because of the difficulty of studying the below-ground component of such systems, studies of the long-term dynamics of plant-soil pathogen interactions have been rare. Moreover, because the predominant theoretical framework in plant pathology focuses on host–pathogen dynamics within a single year (e.g. Gilligan 1983, 1985; Jeger 1987; Campbell & Madden 1990; Jeger & van den Bosch 1993a,b), critical parameters for predicting the long-term dynamics of soil-borne pathogens and their plant hosts have not been identified. Providing a more inclusive conceptual framework may therefore help to guide empirical studies of soil-borne pathogens, and to focus attention on critical parameters for which measurements are lacking. As far as we know, the present study is the first attempt at modelling the between generation population dynamics of plants and soil-borne pathogens.

In Model I, which reflects agricultural situations, the question of coexistence has little meaning, and we therefore focus on pathogen persistence in the soil. As has been shown for animal host–pathogen models where disease transmission is assumed to be density-dependent (e.g. Anderson & May 1981), pathogen persistence requires that the crop density be above a threshold. If crop rotation is practiced, or if fields are allowed to lie fallow for some period, then this threshold is increased relative to the situation where the same crop is planted every year, assuming that the pathogen is unable to increase in the absence of the host. However, this result would not apply to a generalist pathogen that could infect multiple host species. Ultimately, strategies for 'pathogen management' in agricultural situations (e.g. crop rotation vs. letting fields lie fallow) may depend on whether the pathogen in question is specialized on a single host species, or is a generalist (and what other crops are susceptible). It is worth noting that whether or not no-till agriculture is practiced may also have consequences for the persistence of soil pathogens (Sumner *et al.* 1981; Schuh 1990). With no-till agriculture, large amounts

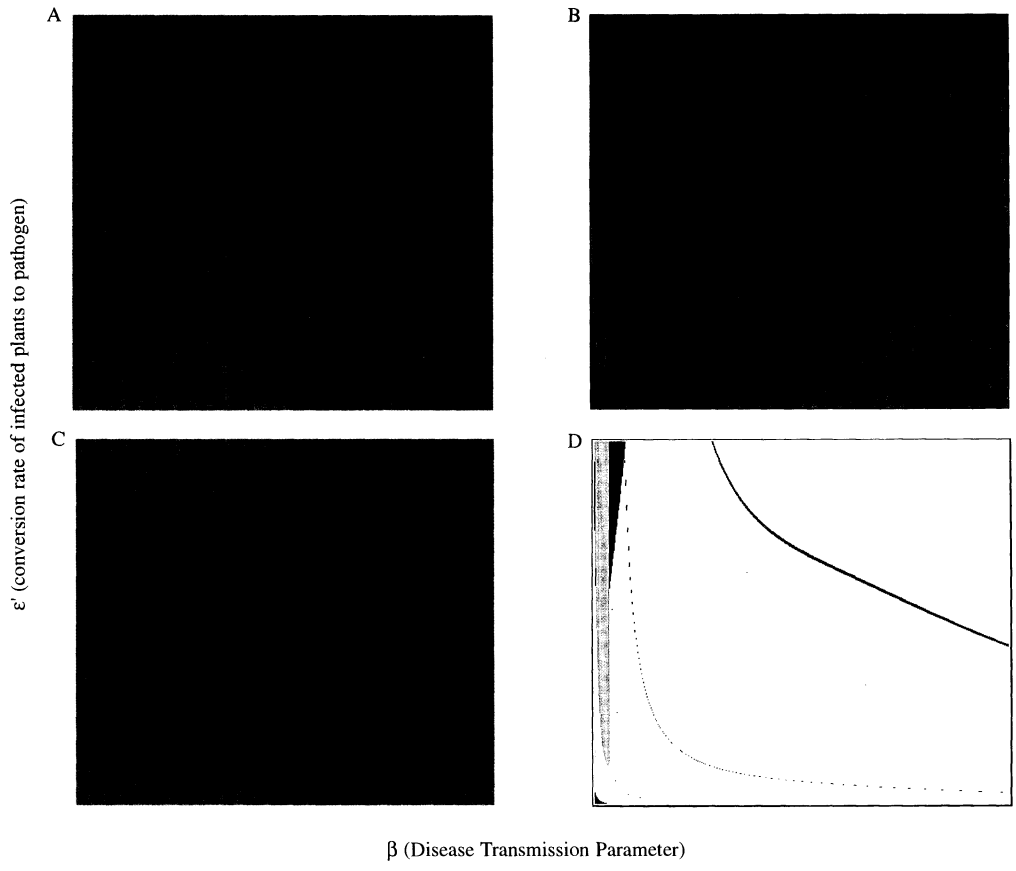

Fig. 3 Predicted dynamics for *Phytophthora* spp. under the assumption of exponential disease transmission, and for fixed values of the cost of infection (c) and the pathogen growth rate (Θ). β values ranged from 0.06 to 6.0; ε' values ranged from 0.08 to 8.0). With respect to the maps, the cost of infection increases from the left pair of maps to the right pair ($c = 0.5, 1.5$ respectively), and the host-independent pathogen growth rate increases from the bottom pair of graphs to the top pair ($\Theta = -0.75, 0.75$ respectively). Grey-scale shadings are as for Fig. 2.

of organic material are left in the top few inches of the soil, providing ample plant material for saprophytic growth of pathogenic fungi, thus potentially increasing disease problems. However, the resulting dynamics will depend upon the trade-off between saprophytic and pathogenic ability and competition with other micro-organisms and may lead to pathogen suppression with increased organic matter (Cook 1991).

In Model II, used for most of the study, when both host and pathogen dynamics are considered as variables (as in natural or 'semimanaged' systems), the analytical and simulation results show that coexistence of both host and pathogen is readily possible. Our models show that the potential for saprophytic growth by the pathogen, a feature perhaps most common in soil-borne fungal pathogens, has important consequences for host–pathogen dynamics. If there is density-dependence in only the host population, then coexistence requires the pathogen growth rate in the soil to be negative. However, if there is density dependence acting on both host and pathogen, then coexistence is possible, even if pathogen growth rates are positive. While in general, increased rates of saprophytic growth reduce the likelihood of coexistence, because some soil pathogens can grow independently of the host, a new equilibrium state is possible; namely that the pathogen may reach a carrying capacity in the absence of any host.

Perhaps because of the historical and sociological divisions between basic and applied ecology, predator-prey and plant-herbivore systems have been generally perceived to 'fit' within the realm of ecology/evolutionary biology, while plant-pathogen systems have been primarily studied in the context of plant pathology; useful comparisons between models of all of these systems have not often been made (but see Hassell & Anderson 1989). Nevertheless, we can gain a great deal of insight from such comparisons; much of the theory that has been developed in the context of predator-prey dynamics (e.g. Nicholson & Bailey 1935; Hassell 1978) can also be applied to plant–pathogen models.

One qualitative feature that unites predator–prey, plant–herbivore and host–pathogen systems is that one species uses the other species in the interaction as a 'resource', and thus consumes it. Models incorporating this type of interaction generally show cyclical behaviour (Hassell & Anderson 1989). In fact, the models we present are analogous to simple models of predator–prey abundance (Roughgarden 1979) and, if there is no density dependence, the internal equilibrium is exactly equivalent. If we explicitly com-

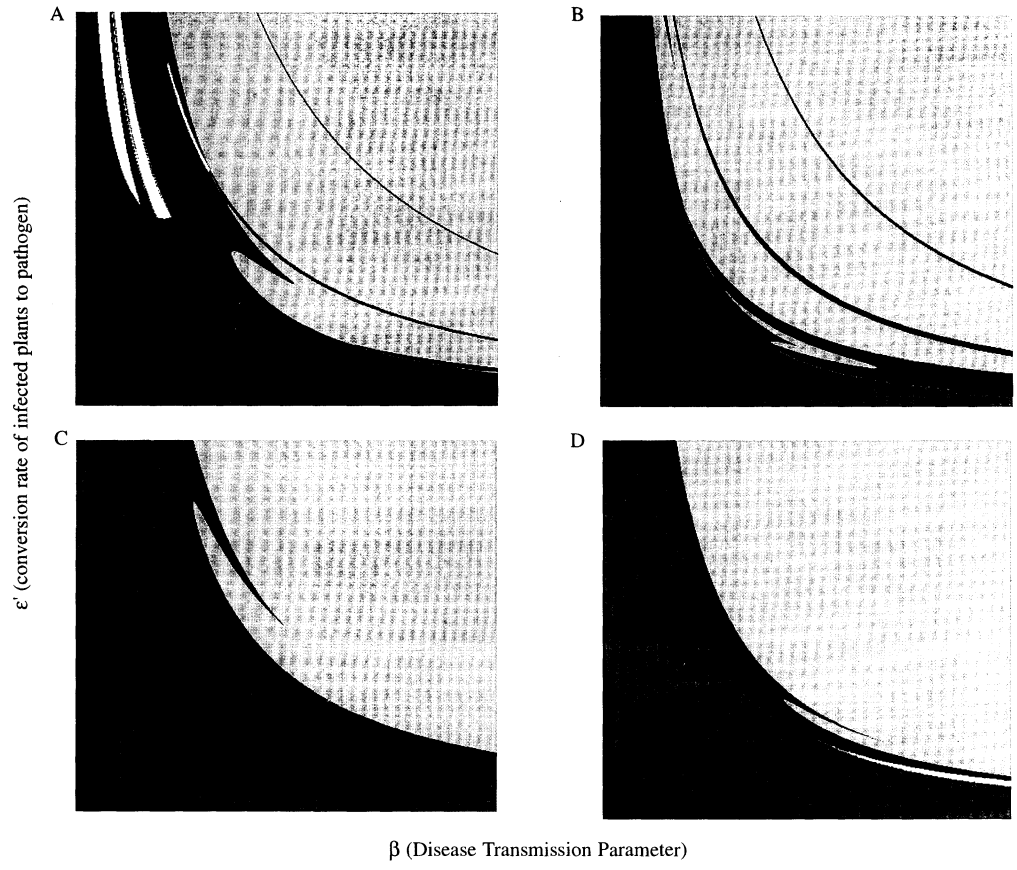

Fig. 4 Predicted dynamics for *Fusarium oxysporum* under the assumption of linear disease transmission, and for fixed values of the cost of infection (c) and the pathogen growth rate (Θ). β values ranged from 0.00002 to 0.002; values of ε' were as in Fig. 2. With respect to the maps, the cost of infection increases from the left pair of maps to the right pair ($c = 0.5, 1.5$ respectively), and the host-independent pathogen growth rate increases from the bottom pair of graphs to the top pair ($\Theta = -0.75, 0.75$ respectively). Grey-scale shadings are as for Fig. 2.

pare results from our models with those from models of predator–prey interactions, we note that, as in the latter (where prey are converted into predators), increasing values of the conversion rate of healthy hosts to pathogen propagules in the soil, ε', decreases host abundance, but leaves pathogen abundance unaffected. Interestingly, a similar effect occurs with respect to the cost of infection, c, where increasing the cost of infection reduces pathogen abundance but does not affect host equilibrium population size (see also Shaw & Peters 1994). This implies that the introduction of tolerant plant genotypes may reduce the average cost of infection (c), and actually result in an increase in pathogen density in the soil.

However, there are also some qualitative differences in the biology (and the resulting dynamics) of predator–prey and plant host–pathogen systems. Host–pathogen models generally assume (as do predator–prey models) that pathogen growth rate is simply equivalent to prey/host loss. An interesting attribute of explicit models of interactions between plant hosts and soil pathogens is that separate disease transmission and pathogen growth terms must be included in the models; this allows the identification of interesting features of the biology of such systems. For example, the fact that, in eqns 4 and 5, the transmission parameter (β) always appears with either the conversion rate (ε') or the cost of infection (c), suggests that these parameters are good candidates for the investigation of trade-offs in these parameters. In fact, it can be shown that as long as the products $\beta'\varepsilon$ and βc remain constant, then the dynamics [and consequently the equilibria given by inequalities 16 and 17] are unchanged. This result was verified by computer simulation; in fact the curvilinear nature of the dynamical maps shown in Figs 2–5 demonstrates the trade-off between β and ε'. Moreover, estimates of these parameters taken from the empirical literature also suggest that this trade-off exists. For illustrative purposes, a comparison of β and ε estimates for *Fusarium oxysporum* on celery vs. other hosts shows that the magnitude of β on celery is two orders of magnitude larger than on other hosts, while the estimate of ε is two orders of magnitude smaller than on other hosts (this is also evident from a comparison between the median values for *Fusarium* and *Phyophthora*).

For both *Phytophthora* and *Fusarium*, computer simulation of Model II showed that, within the range of parameter values we investigated, several dynamic

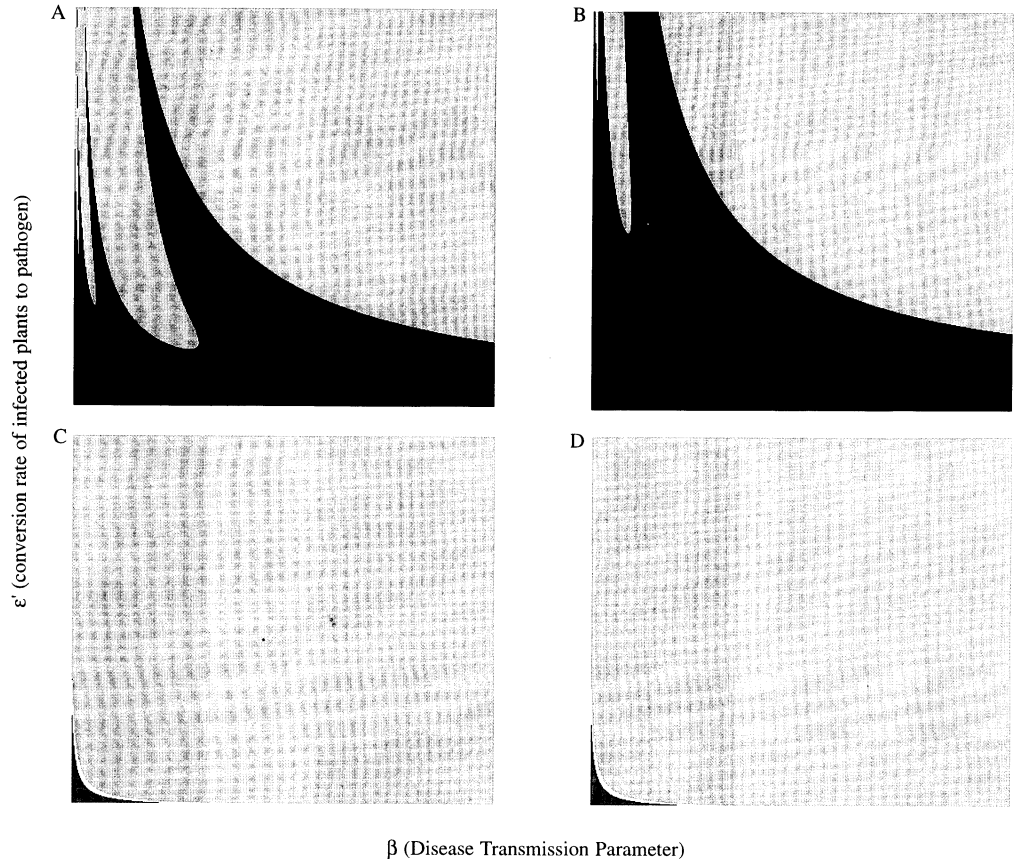

Fig. 5 Predicted dynamics for *Phytophthora* spp. under the assumption of linear disease transmission, and for fixed values of the cost of infection (c) and the pathogen growth rate (Θ). β values ranged from 0.004 to 0.4; ε' values were the same as in Fig. 3. With respect to the maps, the cost of infection increases from the left pair of maps to the right pair ($c = 0.5$, 1.5 respectively), and the host-independent pathogen growth rate increases from the bottom pair of graphs to the top pair ($\Theta = -0.75$, 0.75 respectively). Grey-scale shadings are as for Fig. 2.

outcomes were possible. However, the dynamic maps shown by Figs 2–5 were complex, indicating that small changes in parameter values could often lead to qualitatively different outcomes. This result suggests that long-term dynamics may be difficult to predict under some conditions, and therefore more precise estimates of parameters may be necessary to distinguish between different dynamic outcomes. Moreover, the simulations also showed that the nature of the predicted dynamics were critically dependent on assumptions about the disease transmission process itself.

The exponential transmission model generally provided a better fit to the observed data than the linear transmission model, with r^2 values often being higher for the exponential model. This was particularly evident for estimates from disease transmission data in *Phytophthora* (Table 3). Moreover, the dynamic predictions for *Phytophthora* were radically different for the two transmission models. Alternatively, for *Fusarium*, the r^2 values were generally more similar under the two alternative transmission models (Table 2), and the predicted dynamics were also much more similar. Generally speaking, a simple consideration of the nature of the transmission process makes it clear that the probability of infection [if one considers the entire range of possible disease prevalences] must assume a nonlinear form (Gregory 1948; van der Plank 1975; Thrall *et al.* 1995). Our results suggest that, for *Phytophthora*, dynamics are indeed in the range where transmission is better described by a nonlinear than a linear model, and thus we are more likely to believe dynamical predictions based on the nonlinear transmission function. It is noteworthy, however, that over low to intermediate ranges of pathogen prevalence, a linear transmission model may provide an equally good fit to the observed data. This appears to be the case for *Fusarium*, where the similarity of estimates for both linear and exponential models suggests that dynamics may be more often in the range where a linear model provides a good approximation of the transmission process (Table 2). This is further supported by the fact that the predicted dynamics for the two transmission models also correspond much more closely than for *Phytophthora*.

An important benefit of heuristic models such as those presented here, is in identifying gaps in our empirical knowledge, as well as to identify important components of host and pathogen life histories with regard to population dynamics. While our survey of the literature for *Fusarium* and *Phytophthora* revealed abundant information on some components of fungal life history (e.g. transmission, over-winter survival),

there was a paucity of data on other model parameters such as density dependence and saprophytic ability of the pathogen. However, without an explicit theoretical framework, it is difficult to know *a priori* under what conditions dynamic outcomes will depend on the precision of estimates for these parameters. For example, within estimated ranges of β and ε' for *Phytophthora*, the dynamics were not very sensitive to the cost of infection (c). In contrast, for *Fusarium*, under nonlinear transmission, qualitatively different outcomes were predicted for low vs. high costs of infection.

In recent years, increasing attention has been focused on the importance of spatial structure for host-parasite and predator–prey dynamics and evolution (Levins 1969; Caswell 1978; Hanski & Gilpin 1991). For example, previous work in other systems has shown that the addition of spatial structure will permit coexistence of host and pathogen under conditions in which extinction would have otherwise been predicted (Thrall & Antonovics 1995; Burdon & Jarosz 1991, 1992). This is an issue that has not been addressed with respect to soil-borne pathogens, but seems particularly relevant in this case as the spatial spread of soil-borne pathogens is likely to be slower and more localized than that of their hosts. Thus, even though our models often predict local extinction, it seems likely that coexistence will be possible on broader regional scales. In fact, preliminary results from spatially explicit simulations of hosts and soil-borne pathogens suggest that this is indeed the case.

While our models focus on interactions between a single host and pathogen, the presence of other microorganisms in the soil community and other plant species are likely to have large effects on the dynamics of plants and pathogens (Baker & Snyder 1970; Toussoun *et al.* 1970; Bruehl 1987). Other soil microorganisms have been shown to have large effects on disease transmission, saprophytic ability, the decay rates of resting structures and the cost to the host (e.g. suppressive soils in watermelon: Larkin *et al.* 1993a,b). For example, mycorrhizal fungi have been shown to compete with fungal pathogens for access to plant roots and thereby alleviate the deleterious effects of disease (Newsham *et al.* 1995). Further, many soil pathogens may infect multiple host species in plant communities (Agrios 1988), and therefore coexistence may not depend exclusively on a single host being present.

One value of our modelling approach (Alexander *et al.* 1996) is that it increases our ability to identify parameters that are likely to be of critical importance for determining host–pathogen dynamics and coexistence (i.e. identification of what parameters are important to measure empirically in field experiments). This use of models is very different from much of the modelling in applied plant pathology, where the goal is to mimic closely a particular disease phenomenon or pathosystem (e.g. Fry 1982; Gilligan 1985; Vincelli & Lorbeer 1989; Turkington *et al.* 1991; Jeger & van den Bosch 1993a,b). The more general models presented here are not intended to provide predictions for specific applications, but such heuristic models can be valuable tools for identifying gaps in our empirical knowledge, and in focusing our attention on critical processes for the year to year dynamics in natural systems.

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