Scaling and spatial dynamics in plant–pathogen systems: from individuals to populations

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SUMMARY

Components of transmission for primary infection from soil-borne inoculum and secondary (plant to plant) infection are estimated from experiments involving single plants. The results from these individual-based experiments are used in a probabilistic spatial contact process (cellular automaton) to predict the progress of an epidemic. The model accounts for spatial correlations between infected and susceptible plants due to inhomogeneous mixing caused by restricted movement of the pathogen in soil. It also integrates nonlinearities in infection, including small stochastic differences in primary infection that become amplified by secondary infection. The model predicts both the mean and the variance of the infection dynamics of *R. solani* when compared with replicated epidemics in populations of plants grown in microcosms. The broader consequences of the combination of experimental and modelling approaches for scaling-up from individual to population behaviour are discussed.

1. INTRODUCTION

One of the principal challenges in epidemiological modelling is to predict population dynamics from the behaviour of individuals. In botanical epidemiology, in particular, there is much experimental detail on infection and the growth of individual plants (Sneh *et al.* 1996) and the description of epidemics on a population level (Gilligan 1985; Brasset & Gilligan 1988*a, b*) but there is little knowledge of the link between the two scales. It is, therefore, difficult to assess the effects of changes in local infection dynamics on the behaviour of an epidemic. For example, the incorporation of a biological control agent into soil is known to affect the probability of infection of plants (Bailey & Gilligan 1997). However, to date, the effect has been used only to predict disease progress in a system limited to primary infection caused by transmission from soil-borne inoculum to plants (Bailey & Gilligan 1997). The spread of soil-borne plant infection can also be mediated by secondary infections when the pathogen spreads from plant to plant (Brasset & Gilligan 1988*a, b*; Gilligan 1994*b*). A description of this process is complicated by spatial correlations introduced by localized character of the spread leading to local saturation amongst groups of infected plants.

In this paper we show how to link local pathozone dynamics of an economically important plant pathogen to the interpretation of population behaviour involving both primary and secondary infections. We use a combination of replicated experiments, nonlinear modelling, parameter estimation and a spatially explicit stochastic model (a probabilistic cellular automaton) to analyse and interpret epidemics of damping-off disease on radish seedlings,

Raphanus sativus L., caused by the commonly occurring soil-borne fungal plant pathogen *Rhizoctonia solani* Kühn. Substantial losses result from the infection due to damping-off of seedlings of many crop and other plants, and from infections of mature plants including potatoes, rice and wheat (Sneh *et al.* 1996).

The dynamics of infection progresses from germination of initial soil-borne inoculum, primary infection of a plant, the formation of secondary inoculum and subsequent spread to other plants (Kleczkowski *et al.* 1996; Brasset & Gilligan 1988*a, b*; Gilligan 1994*a, b*). Different phases of the spread are studied in separate experiments, followed by model construction and testing. This involves the following steps: (i) estimation of parameters for temporal dynamics of infection when individual plants are challenged with soil-borne (primary) or plant-borne (secondary) inoculum; (ii) parameters estimated at the individual level are used to calculate transition rates in a probabilistic cellular automaton (PCA); (iii) the PCA is used to predict population trajectories of epidemics; and (iv) the mean and variance of the predicted epidemics is compared with independently collected experimental data for replicated epidemics. We show that the population model, in which all parameters are estimated at the lower (single-plant) level, successfully predicts the mean and variability of the epidemics.

2. EXPERIMENT

Placement experiments are used to quantify the dynamics of infection of individual plants challenged with single propagules of inoculum placed at different distances from the host within the pathozone. The term

pathozone is used, *sensu* Gilligan (1985b), to describe the region surrounding a target organ, in this case a hypocotyl, within which inoculum must lie in order to have some finite probability of infecting the host. Two types of inoculum were used, primary inoculum corresponding to an initial phase of fungal invasion from inoculum that survived an inter-crop period, and secondary inoculum comprising infected plants corresponding to secondary infections. The latent period between infection and production of secondary inoculum and the subsequent transmission of disease to the other plants is estimated by transmission experiments involving a single donor surrounded by recipient plants. The population experiment involved monitoring the dynamics of epidemics with primary and secondary infection in replicated microcosms comprising radish and randomly distributed known amounts of primary inoculum.

(a) Infection of individual plants

Placement experiments involved positioning of individual units of inoculum at precise distances away from the surface of a radish plant grown in sand packs with a moisture content of 10% by weight. The primary inoculum of *R. solani* comprised discs of mycelium measuring 1.0 mm in diameter, removed from the edge of a five-day-old colony of *R. solani* growing on a millipore filter over potato dextrose agar (Gilligan & Bailey 1997). Secondary inoculum was prepared by growing radish seedlings in clear plastic boxes containing an inoculum mixture of chopped potato soil (Benson & Baker 1974) infested with *R. solani* and sand. The plants were grown at 23 °C with a 16 h day length. Infected (damped-off) plants were removed after 7 days and used as a source of infection.

Each distance was replicated 25 times and seedlings were scored daily for the presence (damping-off) or absence of disease upon emergence. Figure 1 shows the relative frequencies of infection as functions of time (t) after infestation and distance (x) between the inoculum and susceptible host for primary and secondary inoculum.

The probability of successful infection was strongly influenced by the distance of inoculum from the host. Transmission of infection is limited to a pathozone extending to 20 mm for primary inoculum and 60 mm for secondary inoculum (figure 1). The drop in the probability of infection for primary inoculum, placed close (less than 2 mm) to the host is due to the disruption of inoculum by germinating radish plants (Gilligan & Bailey 1997).

The delay in the initiation of infection increased with distance for secondary inoculum but changed little within the range over which primary infection occurred. The delay in primary infection is dominated by germination and disease development within the hosts as opposed to time taken to reach a host. It is followed by 2–3 days of high activity and a saturation in the development of the disease on individual host plants, with very little change in the probabilities of infection after 7–8 days. This results from the combined effects of a decline in host susceptibility and infectivity of the inoculum (Deacon 1980; Kleczkowski *et al.* 1996).

The latent period was estimated from an additional experiment involving individual plants (transmission experiment), described later in the paper.

(b) Population dynamics

Clear plastic boxes measuring 100 mm wide, 200 mm long and 100 mm in depth were filled with 1 kg of white

Figure 1. Placement experiment: probabilities of an initially susceptible plant becoming infected at a given time by an inoculum unit located at a certain distance from the host. Points represent experimental data whereas the surfaces are generated by (10) (primary inoculum, (a)) and (11) (secondary inoculum, (b)). No data are available for the time between planting and five days, but there was no detectable activity during this period.

sand (acid washed and sieved to 0.5–1.0 mm in diameter) with a moisture content of 10% by weight. Radish seeds were planted in a grid to a depth of 10 mm with a spacing of 20 mm. In the population experiment ten mycelial discs were placed in randomly selected positions among 50 plants. All other conditions, including host density and genotype, uniformity of inoculum of *R. solani*, water availability, light and temperature were strictly and identically controlled in each of five replicates. The boxes were sealed with clear plastic lids and incubated in a growth chamber at 23 °C with a day length of 16 h. The cumulative numbers of seedlings damped-off were counted daily. The experiment was stopped at day 19.

The mean value (figure 2a) followed a sigmoidal disease progress curve with asymptotic levels of disease less than 100%. There is considerable variation among the individual disease progress curves for each microcosm reflected in the variance (figure 2b).

3. MODELS

(a) Spatially explicit model for population dynamics

Primary infections can be satisfactorily characterized by continuous differential equations describing probabilities of infection (Bailey & Gilligan 1997). However, subsequent spread leads to spatial correlations between infected and susceptible plants and

Figure 2. Spatially explicit model for population dynamics: time dependence of the average values (a) and the variance (b) is shown for the experimental data (points) and for the CA simulations (solid line). The simulations represent the average and variance in 100 replicated runs.

cannot easily be described by simple rate equations (Levin & Durrett 1996). Here we use a spatially explicit model in the form of a PCA with transition probabilities calculated from transmission and latency parameters in individual-based experiments. In the latter, individual plants are involved and, therefore, the spatial correlations are not important. The simple rate equations can then be applied for parameter estimation (see § 3b below).

Each host in the PCA is assumed to be in one of three discrete states, susceptible, latent or infectious, and the proportions of hosts in each of the states are denoted by S , L and I , respectively, ($S + L + I = 1$). No apparent symptomatic difference between latent and infectious plants can be observed in the experiment, and the latent period is introduced here to account for delays in the disease transmission. Latent and infectious hosts are counted as infected plants to compare with observed values of the proportions of infected plants, denoted by $Y = L + I$. The model is based on the following transitions.

(i) *Susceptible to latent host, with primary inoculum as a source of infection*: transition occurs between time t and $t + \Delta t$ with a probability $r_p(x, t)\Delta t$, if a primary inoculum is located at a distance x in a square with length x_d , centred at the susceptible host, where $r_p(x, t)$ is derived from placement experiments.

(ii) *Susceptible to latent host, with secondary inoculum as a source*: transition occurs between time t and $t + \Delta t$ with a probability $r_s(x_d, t)$, if an infectious host is located in the immediate neighbourhood (four neighbours in the centre, three at the edges and two at the corners of the microcosm), and where $r_s(x, t)$ is derived from placement experiments.

(iii) *Latent to infective host*: with a probability $1 - \exp(-g\Delta t)$ corresponding to an exponentially distributed latent period with mean $1/g$. The value of g was derived from the transmission experiment.

Changes in host susceptibility and infectivity of the primary and secondary inoculum (Deacon 1980; Kleczkowski *et al.* 1996) are expressed in the time dependence of the rates $r_p(x, t)$ and $r_s(x, t)$, whereas variability in infection efficiency (Gilligan & Bailey 1997) is reflected in the dependence on the distance x .

Hosts are distributed on a five by ten square lattice with a distance $x_d = 20$ mm between the plants to represent conditions in experiments used to test the predictions from the model. Ten primary inoculum units are distributed at random (continuous) positions in a rectangle with dimensions 100 mm by 200 mm containing the lattice. We assume absorbing boundary conditions so that no infections are transmitted outside the boundary. The model is updated synchronously with a time step of $\Delta t = 0.1$ d, and the simulations started at $t = \tau_p$ (accounting for the initial delay in primary infection). The results were insensitive to the choice of Δt ranging from 0.01–0.5 d.

(b) Parameter estimation

The rate at which new infections are produced in the placement experiment involving single plants and inoculum units is proportional to the product of the rates $r_p(x, t)$ or $r_s(x, t)$ and the number of hosts which remain susceptible in the particular experiment. Denoting by $Y_p(x, t)$ and $Y_s(x, t)$ the proportions of infected plants at time t in the placement experiments involving primary and secondary inoculum at a distance x from the host, respectively, we obtain the following equations (in the limit $\Delta t \rightarrow 0$)

$$\frac{dY_p}{dt} = r_p(x, t)(1 - Y_p), \quad (1)$$

$$\frac{dY_s}{dt} = r_s(x, t)(1 - Y_s). \quad (2)$$

We express the rates of infection $r_p(x, t)$ and $r_s(x, t)$ as follows:

$$\text{Rate} = \left(\begin{array}{c} \text{Spatial decay} \\ \text{due to location} \\ \text{of inoculum} \\ \text{away from} \\ \text{host} \end{array} \right) \left(\begin{array}{c} \text{Time decay due} \\ \text{to change in host} \\ \text{susceptibility and} \\ \text{inoculum} \\ \text{infectivity} \end{array} \right) \times \left(\begin{array}{c} \text{Delay in} \\ \text{onset of} \\ \text{infection} \end{array} \right), \quad (3)$$

Table 1. *Parameters of the models, their values, errors and interpretation*

parameter	value	error ^a	interpretation	units
(a) particulate inoculum				
a_p	1.09	0.20	amplitude	1
σ_p	0.277	0.027	spatial decay	mm ⁻¹
d_p	0.55	0.20	temporal decay	d ⁻¹
δ_p^b	—	—	delay	d mm ⁻¹
η_p	0.49	0.15	cec ^c	mm ⁻¹
τ_p	4.898	0.082	time offset	d
(b) infected tissue inoculum				
a_s	14.9	4.0	amplitude	1
σ_s	0.000 99	0.000 22	spatial decay	mm ⁻²
d_s	0.36	0.19	temporal decay	d ⁻¹
δ_s	0.0524	0.0058	delay	d mm ⁻¹
τ_s	3.73	0.15	time offset	d
(c) latent period				
$1/g$	3.89	(3.27 – 4.64)	—	d

^aStandard error or approximate 95% confidence intervals.

^bParameter δ_p was found to be ill-conditioned and the assumption $\delta_p = 0$ improved the fit.

^ccec stands for critical exponential correction.

so that,

$$r_p(x, t) = \Phi_p(x)\Psi_p(t)H(t - \tau_p - \delta_p x), \quad (4)$$

$$r_s(x, t) = \Phi_s(x)\Psi_s(t)H(t - \tau_s - \delta_s x), \quad (5)$$

where the step function $H(t) = 0$, if $t < 0$, and one, if $t > 0$, is used to describe the delay in the onset of infection. The parameters τ_p and τ_s describe the minimum initial delay (cf. figure 1) with δ_p and δ_s allowing for a change in delay with distance. The terms $\delta_s x$ and τ_s , for secondary infection (with $x = x_d$ being a fixed distance between plants), are subsequently subsumed into the latent period.

We use two different shapes for the spatial part of the rate, $\Phi(x)$, a critical exponential function for the primary inoculum (Bailey & Gilligan 1997)

$$\Phi_p(x) = C_p(1 + \eta_p x) \exp(-\sigma_p x) \quad (6)$$

and a Gaussian function for the secondary inoculum

$$\Phi_s(x) = C_s \exp(-\sigma_s x^2). \quad (7)$$

We assume that the time decay is exponential, so that we have,

$$\Psi_p(t) = \exp(-d_p t), \quad (8)$$

$$\Psi_s(t) = \exp(-d_s t). \quad (9)$$

The choice is motivated by Kleczkowski *et al.* (1996), but here we assume different decay rates for primary and secondary infections.

The equations (1) and (2) can be solved in order to obtain

$$Y_p(x, t) = 1 - \exp\{-a_p(1 + \eta_p x) \exp(-\sigma_p x) \times [\exp(-d_p \delta_p x) - \exp(-d_p(t - \tau_p))] \times H(t - \tau_p - \delta_p x)\}, \quad (10)$$

$$Y_s(x, t) = 1 - \exp\{-a_s \exp(-\sigma_s x^2) \times [\exp(-d_s \delta_s x) - \exp(-d_s(t - \tau_s))] \times H(t - \tau_s - \delta_s x)\}, \quad (11)$$

where for the convenience of calculations the constants C_p and C_s are parameterized as $a_p d_p \exp(d_p \tau_p)$ and $a_s d_s \exp(d_s \tau_s)$, respectively.

The functions (10) and (11) capture the essential features of the profiles shown in figure 1, notably the delay in the onset of the disease for secondary infections and the dependence of asymptotic values for long t on the distance from the host x , $Y_p(x, \infty) = 1 - \exp\{-a_p(1 + \eta_p x) \exp[-(\sigma_p + d_p \delta_p)x]\}$ and $Y_s(x, \infty) = 1 - \exp\{-a_s \exp[-(\sigma_s x + d_s \delta_s)x]\}$.

The parameters in (10) and (11) were estimated (table 1) by nonlinear least squares fitting to the profiles in figure 1.

The estimate for the latent period (table 1) was obtained by least squares from a separate experiment which followed the first steps in a chain of infection transmission from the soil inoculum to a donor plant and subsequently to the surrounding plants. A single unit of primary inoculum was placed close (at a distance of 3 mm and at a depth of 5 mm) to a central (donor) radish plant surrounded by four plants each 20 mm from the donor. The equations (1) and (2) were modified so that Y_p represented the infection of central donor plants and Y_s the recipient plants in the replicated experiment. The exponentially distributed latent period was estimated as the reciprocal of the per capita transition rate, g , (table 1) of a compartmental model with continuous transition from latent and infectious states, given by

$$\frac{dY_p}{dt} = r_p(x_p, t)P(1 - Y_p), \quad (12)$$

$$\frac{dL}{dt} = r_s(x_d, t)Y_p[1 - (I + L)] - gL, \quad (13)$$

$$\frac{dI}{dt} = gL, \quad (14)$$

$$Y_s = I + L. \quad (15)$$

4. MODEL PREDICTIONS

The probability of infection transmission from different sources of inoculum to the host located at different distances (placement experiment) is described well by the model, both for primary (equation (10) and figure 1*a*, RSE 0.069 on 25 df) and secondary (equation (11) and figure 1*b*, RSE 0.054 on 25 df) inoculum. The model captures the essential features of the transmission, namely the decay of the rates with time and distance (σ_p , σ_s , d_p and d_s all significantly different from zero, table 1).

The spatial model with the parameters listed in table 1 was run 100 times with different random initial conditions and different realizations of random dynamics. Figure 2 compares the average values and variances of the disease progress curves of the model with the data from the microcosms. The model predicts the data well. The agreement is remarkable, especially in the light of the high degree of variability amongst microcosms (compare figures 2*a, b*). The model predicts a sigmoidal shape for the average infection load (figure 2*a*), with the infection saturating at asymptotic values less than 100% for the population experiment (figure 2*a*). This is caused by the temporal decay in the rates of infection spread (Kleczkowski *et al.* 1996). The variance (figure 2*b*) also follows a sigmoidal shape, with a sharp increase after eight days. The rise can be associated with an onset of secondary infections, occurring approximately four days after the first primary infections. The delay is compatible with an interpretation in terms of the latent period of $1/g \simeq 3.98$ d (table 1).

5. DISCUSSION

Scaling-up from individual to population behaviour requires integration of the inherent nonlinearities, spatial correlations and stochastic processes that occur in the interactions between organisms. The problem in relating different levels of description of biological populations has been addressed recently in ecology (Pacala & Tilman 1994; Holt *et al.* 1995; Pacala *et al.* 1996) and in stochastic simulation and mean field models for the spread of plant pathogens (Gilligan 1994*a*; Bailey & Gilligan 1997). Previously, we have used a deterministic model to predict the behaviour of epidemics from the dynamics of infection and disease on individual plants (Bailey & Gilligan 1997). It was restricted, however, to epidemics involving primary infection and did not, therefore, address the problems associated with spatial correlation created by secondary infection.

In this paper we have shown that it is possible to scale up the behaviour of infections of individual plants in order to predict the mean and variance of an epidemic that includes primary and secondary infections. Spatial aspects of the epidemic spread are described here in terms of a probabilistic cellular automaton. This approach has an extensive history in biology, epidemiology and ecology (Mollison 1977; Boccara & Cheong 1992; Ermentrout & Edelstein-Keshet 1993; Rand 1994; Durrett & Levin

1994*b*; Levin & Durrett 1996), but to date relatively little has been done on estimating parameters from experiments and testing the results of scaling up on independent data sets (Holt *et al.* 1995).

In addition to predicting average disease trajectories, the PCA also successfully predicted variability amongst replicate epidemics of the *R. solani*-radish system. This reflects in part the stochastic nature of the model but also the nonlinearity of the biological system in which small differences in initial infection are amplified by secondary infection. Differences in primary infection come about due to the geometrical arrangement and germinability of the primary inoculum. The decay of the rate of primary infection with distance leads to a nonlinear relation between the position of the inoculum and the probability of initial infection. In those replicates where, by chance, primary infections occur later, secondary infections are delayed, causing a large scatter in the transient behaviour amongst replicate epidemics. The decline in the host susceptibility essentially 'freezes' the dynamics, and the variability in the transient behaviour, initiated by stochastic variation in primary infection, is reflected in the final state. Previous work has analysed this dynamically generated variability using a mean field model in which the underlying mechanisms are subsumed into simplified transmission parameters (Kleczkowski *et al.* 1996). Here we have reproduced and tested the effect of interrupted transients using a spatially explicit model in which the mechanisms responsible for the scatter, at the level of the individual plant, are formally included in the transmission parameters.

Interruption of disease progress is typical for plant diseases that attack the host at specific stages of development, such as damping-off or stem diseases (Deacon 1980; Kleczkowski *et al.* 1996; Sneh *et al.* 1996; Gilligan *et al.* 1997). It may also occur due to an environmental switch caused by temperature or humidity, or can be caused by harvesting (Gubbins & Gilligan 1997). Dynamically generated variability is an important factor in the design and interpretation of ecological experiments (Kareiva 1995) since it affects the evolution of the probability distribution for variability amongst replicates. This in turn affects estimation and model fitting (Gilligan 1990; Holt *et al.* 1995) as well as hypothesis testing by influencing the numbers of replicates required to distinguish amongst different treatments for the control of the disease.

The stochastic model presented here assumes that primary infections affect the nearest host only and that the secondary spread is limited to the four nearest neighbours on the square lattice. We do not allow for synergistic effects amongst propagules in the pathozone or multiple infections on a single plant on the probability of infection (Gilligan 1985*b*) or on latency. The description of the decay in pathogen infectivity and host susceptibility as well as the latency and delays due to a separation of the hosts is also simplistic and requires further experimental and model studies. For comparison, we modified equations (12) and (13) to account for a fixed rather than

an exponentially distributed latent period. The results showed little difference between the models as compared with the variability in the experimental data.

The combination of experiments, nonlinear modelling and spatial simulations presented here can be generalized to other systems. The *R. solani*-radish system offers an amenable and highly repeatable experimental tool that can be readily extended to three-species interactions with the introduction of a biological control agent, for example *Trichoderma* spp. (Kleczkowski *et al.* 1996). The system allows experimental testing of hypotheses about spatially-implicit mean-field approximations (Kleczkowski *et al.* 1996; Rand 1994; Durrett & Levin 1994a; Levin & Durrett 1996; Gubbins & Gilligan 1997) as well as the consequences of spatial and temporal variability in contact processes on persistence and invasion of a parasite, patchiness and the percolation of infection through a host population.

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