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CONTENTS

SUMMARY

In this paper we study the dynamical properties of models for botanical epidemics, especially for soil-borne fungal infection. The models develop several new concepts, involving dual sources of infection, host and inoculum dynamics. Epidemics are modelled with respect to the infection status of whole plants and plant organs (the *G* model) or to lesion density and size (the *SW* model). The infection can originate in two sources, either from the initial inoculum (primary infection) or by a direct transmission between plant tissue (secondary infection). The first term corresponds to the transmission through the free-living stages of macroparasites or an external source of infection in certain medical models, whereas the second term is equivalent to direct transmission between the hosts in microparasitic infections. The models allow for dynamics of host growth and inoculum decay. We show that the two models for root and lesion dynamics can be derived as special cases of a single generic model. Analytical and numerical methods are used to analyse the behaviour of the models for static, unlimited (exponential) and asymptotically limited host growth with and without secondary infection, and with and without decay of initial inoculum. The models are shown to exhibit a range of epidemic behaviour within single seasons that extends from simple monotonic increase with saturation of the host population, through temporary plateaux as the system switches from primary to secondary infection, to effective elimination of the pathogen by the host outgrowing the fungal infection. For certain conditions, the equilibrium values are shown to depend on initial conditions. These results have important consequences for the control of plant disease. They can be applied beyond soil-borne plant pathogens to mycorrhizal fungi and aerial pathogens while the principles of primary and secondary infection with host and inoculum dynamics may be used to link classical models for both microparasitic and macroparasitic infections.

1. INTRODUCTION

Modelling of animal diseases occupies a central role in the theory and ecology of host–parasite and predator–prey relationships (Anderson & May 1991). Plant diseases have so far impinged relatively little on the attention of ecological and epidemiological modellers (Gilligan 1985; Campbell & Madden 1990; Roughgarden *et al.* 1989). Yet the principles for epidemic spread within and between fields, problems of scale in linking infection of an individual to population behaviour and the amenability of plants to experimental analysis imply that botanical epidemiology is both theoretically and experimentally suited to

contribute to broader epidemiological analyses. This is particularly true for soil-borne plant pathogens for which there is currently much interest in biological control (Cook & Baker 1983; Cook 1988; Gilligan 1994; Kleczkowski *et al.* 1996; Gubbins & Gilligan 1996, 1997) and in problems of spatial heterogeneity (Gilligan 1995).

Soil-borne plant pathogens include fungi, nematodes, bacteria, viruses, mycoplasmas and ricketsia-like organisms. Amongst these, fungi are collectively and often individually the principal causes of crop losses. Soil-borne parasitic fungi have two broad types of infection cycle, primary infection from a reservoir of inoculum in soil and secondary infection from infected to uninfected hosts. Compared with more general epidemiological systems, including animal as well as plant hosts, secondary infection corresponds to transmission between susceptibles and infectives, while primary infection is equivalent to introductions from an external source. Within the soil, the reservoir of inoculum is depleted by decay of inoculum due to senescence, parasitism and predation. Inoculum is replenished by the decay and incorporation of infected plant tissue.

Selection of appropriate host, pathogen and disease variables for quantification, modelling and analysis of botanical epidemics depends on the characteristics of the host pathogen system and on the scale and dynamics of interest. Almost all fungal symbionts of plants invade from a localized point of entry. Certain fungi, such as the beneficial endomycorrhizal fungus, *Glomus mossae*, and the common root and stem pathogen, *Rhizoctonia solani*, frequently grow to form an identifiable infection unit or lesion. Subsequent infection leads to a population of lesions or infection units. As the density of lesions increases the proportion of diseased or infected root tissue increases and the epidemic is limited by the availability of susceptible tissue. A second class of fungi, typified by *Gaeumannomyces graminis* which causes the take-all disease of cereals, produce infections and lesions that grow indeterminately so that they effectively occupy an entire root. A third class arises with highly pathogenic fungi which can girdle and kill an entire root from a single lesion.

All three classes of symbiont behaviour, involving discrete, indeterminate or virulent infections, can be modelled by one of two broad approaches. One models the change in status of plants or of plant organs from susceptible to diseased (Brassett & Gilligan 1988). The other models the dynamics of lesions, infections or points of entry, later referred to collectively as lesions, and the relative changes in lengths of diseased and susceptible root tissue (Smith & Walker 1981). Early models dealt with the disease status of whole plants (Van der Plank 1963), but many plants produce large numbers of roots so that classification as diseased or undiseased, depending on the presence of a single lesion on an extensive root system, is unsatisfactorily coarse. Considerable dynamical activity and spatial heterogeneity is evident (Werker & Gilligan 1990) even when most plants are infected in an agricultural crop. For many plants, therefore, especially monocotyledons, main root axes are an appropriate unit of population (Gilligan 1985).

In practice, root-based models are most appropriate for those epidemics where lesions occur singly on roots, where lesions expand to occupy entire roots or when a single lesion is sufficiently virulent to incapacitate an entire root. The ratio of susceptible to infected roots, rather than root tissue, is then an important determinant of epidemic development. Root-based models are also appropriate in dealing with experimental data where there is loss of root tissue on recovery from soil but where roots can still be classified as diseased or healthy. Lesion-based models are best suited to fungi that produce discrete lesions with limited, determinate growth, in which the dynamics of fungal population growth is driven by lesion density and proportion of infected or diseased tissue. Lesion-based models may also be used, however, to examine the fine-scale dynamics of infection and population growth of almost all root-infecting fungi.

In this paper, we present and analyse two general models for the temporal spread of plant infections; one deals with roots as units, the other with lesions. The models allow for dual sources of primary and secondary infection, for inoculum decay and for host growth. The models are motivated for soil-borne epidemics including ectotrophic pathogens, such as *G. graminis*, damping-off fungi, *R. solani* and *Pythium ultimum* and necrotrophic root rotting fungi including *Fusarium* and *Phytophthora* spp. The foregoing include some of the most economically important pathogens in temperate and tropical agriculture. The models can also be applied to non-pathogenic colonization by endomycorrhizae and ectomycorrhizae and to aerial epidemics of plants and animals in which there are two sources of infection, for example, sheath blight of rice caused by *R. solani*.

The dynamical behaviour of the two types of model is used to test practical questions about the influence of host growth and inoculum decay on the dynamics of infection and the equilibrium densities of infection and disease. We identify criteria for equilibrium densities to distinguish between epidemics that saturate the host population, with all roots and root tissue becoming infected, from those in which there is some lower limit to the carrying capacity. We show that the amount of initial inoculum present in soil at the beginning of a season can influence this carrying capacity, thereby perpetuating patchiness in the occurrence of disease. Particular attention is given throughout to the relative importance of primary and secondary infection. The rate of decay of soil-borne inoculum is also shown to affect the switch from primary to secondary infection. We also examine how the distribution of lesions can affect the dynamics of an epidemic by comparing disease trajectories arising from relatively few large lesions with those that derive from many small lesions.

The role of host growth in disease dynamics is analysed by comparing unlimited, indeterminate root growth with limited growth, in which there is a definite carrying capacity, and static host populations

where roots or stems rapidly grow through an inoculum layer and then remain exposed to infection. Here we test whether the host can outgrow infection or if the multiplicative effect of secondary infection enables the epidemic to 'keep up' with host expansion.

Although the models are introduced as separate paradigms for root and lesion dynamics, we show that the two models can be derived as special cases of a single generic model that bridges the biological properties of each model. Finally, the relationships between the general plant models, SIR (Susceptible, Infected and Removed) and micro- and macroparasite (Anderson & May 1979, 1991; May & Anderson 1979) models are discussed.

2. GENERAL MODELS

Depending on the variable of interest, two general models may be distinguished. Brassett & Gilligan (1988), with elaborations by Gilligan (1990), proposed a model for the change in density of infected roots (N_i) in a population of roots (N). The simplest version of the model, with fixed inoculum (P) and host densities, is a convolution of the monomolecular and logistic functions,

$$\text{infected roots:}\quad \frac{\mathrm{d}N_\mathrm{i}}{\mathrm{d}t} = (r_\mathrm{p}P + r_\mathrm{s}N_\mathrm{i})(N - N_\mathrm{i}), \qquad (1)$$

in which r_p is the rate of primary infection and r_s is the rate of secondary infection. Primary infection is therefore driven by contacts between inoculum (P) and uninfected, susceptible roots ($N - N_\mathrm{i}$), while secondary infection depends on contacts between infected and susceptible individuals, familiar in many epidemiological models.

Smith & Walker (1981) and later Walker & Smith (1984) introduced a model to describe the dynamics of lesions or single infections (U), hereafter referred to as lesions, together with the length of infected root (L_i):

$$\text{lesions:}\quad \frac{\mathrm{d}U}{\mathrm{d}t} = r_\mathrm{p}P(L - L_\mathrm{i}), \qquad (2)$$

$$\text{infected root length:}\quad \frac{\mathrm{d}L_\mathrm{i}}{\mathrm{d}t} = r_\mathrm{l}U\left(1 - \frac{L_\mathrm{i}}{L}\right), \qquad (3)$$

in which L is the total root length, r_p is the rate of primary infection, defined in this case as the number of lesions per unit time per unit of inoculum per unit of susceptible tissue, and r_l is the rate of growth of lesions. Lesions are produced solely by contacts with soil-borne inoculum (P) with no secondary production of new lesions from existing lesions. The rate of growth of lesions along roots is restricted by the availability of susceptible tissue ($1-L_\mathrm{i}/L$). The models of Smith & Walker (1981) and of Walker & Smith (1984) were introduced to describe the spread of infections of beneficial, endomycorrhizal fungi on roots of clover plants but the model can also be applied to pathogenic fungi as long as the effects of the fungi on host growth are not explicitly included in the model.

A natural extension of the Smith & Walker model, which we propose here, is to introduce secondary infection as an additional source of lesions, thus,

$$\text{lesions:}\quad \frac{\mathrm{d}U}{\mathrm{d}t} = (r_\mathrm{p}P + r_\mathrm{s}L_\mathrm{i})(L - L_\mathrm{i}), \qquad (4)$$

$$\text{infected root length:}\quad \frac{\mathrm{d}L_\mathrm{i}}{\mathrm{d}t} = r_\mathrm{i}U\left(1 - \frac{L_\mathrm{i}}{L}\right), \qquad (5)$$

in which r_s is the rate of secondary infection. This implies that infected tissue is potentially infectious but otherwise the biological assumptions of the model are identical to those for equations (2) and (2). Note that the parameters, though denoted in the same way as in equation (1), have different meaning and therefore different values. The principal variables and parameters used in the models are summarized in table 1.

(a) *Incorporation of host and inoculum dynamics*

Following Gilligan (1985, 1990) we introduce dynamics for inoculum and host growth, giving, for the root model,

$$\text{infected roots:}\quad \frac{\mathrm{d}N_\mathrm{i}}{\mathrm{d}t} = (r_\mathrm{p}P + r_\mathrm{s}N_\mathrm{i})(N - N_\mathrm{i}), \qquad (6)$$

$$\text{total roots:}\quad \frac{\mathrm{d}N}{\mathrm{d}t} = r_\mathrm{n}f(N), \qquad (7)$$

$$\text{inoculum:}\quad \frac{\mathrm{d}P}{\mathrm{d}t} = -r_\mathrm{d}P, \qquad (8)$$

in which r_n is the rate of root production, r_d is the rate of decay of inoculum and $f(N)$ is a general term that incorporates exponential [$f(N) = N$] and logistic [$f(N) = N(1 - N/N_\mathrm{max})$] growth.

For the lesion model, the equations become

$$\text{lesions:}\quad \frac{\mathrm{d}U}{\mathrm{d}t} = (r_\mathrm{p}P + r_\mathrm{s}N_\mathrm{i})(N - N_\mathrm{i}), \qquad (9)$$

$$\text{infected root length:}\quad \frac{\mathrm{d}L_\mathrm{i}}{\mathrm{d}t} = r_\mathrm{l}U\left(1 - \frac{L_\mathrm{i}}{L}\right), \qquad (10)$$

$$\text{total root length:}\quad \frac{\mathrm{d}L}{\mathrm{d}t} = r_\mathrm{n}f(L), \qquad (11)$$

$$\text{inoculum:}\quad \frac{\mathrm{d}P}{\mathrm{d}t} = -r_\mathrm{d}P, \qquad (12)$$

in which r_n, r_d and $f(L)$ are analogously defined as for the root model above, except that host growth now refers to growth in length rather than in number of roots. We choose an exponential decay of inoculum with constant per capita mortality rate in equations (6) and (9) for simplicity and because it has been shown to apply to many pathogens (Garrett 1970). The rate of host growth is influenced only by the total number of roots (equation (6)) or the total amount of root tissue (equation (9)) without separation of the effects of infection and disease on host growth. We discuss, later, how to incorporate functional responses that allow for the effects of infection on root growth.

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Table 1. *Variables, parameters and functions used in the models.*

symbol	description	units[a] *G* model	units[a] *SW* model
	variables		
N_i	infected roots	numbers	—
N	total roots	numbers	—
P	propagules	numbers	numbers
U	lesions	—	numbers
L_i	infected root length	—	cm
L	total root length	—	cm
	parameters		
N_{max}	maximal root number	numbers	—
L_{max}	maximal root length	—	cm
r_p	primary infection rate	day^{-1}	$(day\ cm)^{-1}$
r_s	secondary infection rate	day^{-1}	$(day\ cm^2)^{-1}$
r_l	lesion growth	—	$cm\ s^{-1}$
r_n	host growth[b]	day^{-1}	day^{-1}
r_d	inoculum death	day^{-1}	day^{-1}
g	infection induced death	day^{-1}	day^{-1}
η	germination effect on inoculum	1	1
ρ	inoculum release	1	cm^{-1}
	functions		
f	functional response	numbers	cm

[a]We assume that all variables are measured in absolute values. Often, however, they are expressed in terms of spatial densities (i.e. numbers or lengths per cm^2) or volume densities (per cm^3). The units will then change accordingly.
[b]Units of r_n depend on the particular choice of f. Here we assume that r_n expresses the *per capita* growth rate.

The models in equations (6)–(6) and (9)–(9) are hereafter referred to as the G and SW models, respectively. The G and SW models with primary and secondary infection are presented here as two paradigms, that differ not only in biological variables but also in mathematical behaviour. We show in Appendix 1 that each can be derived, under certain assumptions, as special cases of a single generic model.

(*b*) *Non-dimensionalization*

It is convenient to introduce dimensionless variables to scale disease relative to the changing host size. Let $n = N_i/N$ be the proportion of infected roots for the G model, then

$$\frac{dn}{dt} = (r_p P + \rho_s(t)n)(1-n) - G(t)n, \tag{13}$$

where

$$\rho_s(t) = r_s N \quad \text{and} \quad G(t) = \frac{1}{N}\frac{dN}{dt} = r_n \frac{f(N)}{N}.$$

For the SW model, let $u = U/L$ be the average number of lesions per unit length of root and $l = L_i/L$ be the proportion of root length infected, then

$$\frac{du}{dt} = (r_p P + \rho_s(t)l)(1-l) - G(t)u, \tag{14}$$

$$\frac{dl}{dt} = r_l u(1-l) - G(t)l, \tag{15}$$

in which

$$\rho_s(t) = r_s L \quad \text{and} \quad G(t) = \frac{1}{L}\frac{dL}{dt} = r_n \frac{f(L)}{L}.$$

In the following analyses, we consider three special cases (expressions for ρ_s are given on the left for the G and right for the SW models).

(i) No host growth:

$$G(t) = 0,$$
$$\rho_s(t) = r_s N = \text{const.}, \qquad \rho_s(t) = r_s L = \text{const.}$$

(ii) Limited host growth:

$$G(t) \to 0 \quad \text{as } t \to \infty,$$
$$\rho_s(t) \to r_s N_{max}, \qquad \rho_s(t) \to r_s L_{max}.$$

(iii) Unlimited (exponential) growth:

$$G(t) = r_n,$$
$$\rho_s(t) = r_s N_0 \exp(r_n t) \to \infty \quad \text{as } t \to \infty, \qquad \rho_s(t) = r_s L_0 \exp(r_n t) \to \infty \quad \text{as } t \to \infty.$$

Populations with no host growth include stems or roots that rapidly grow through an inoculum layer and then remain exposed to infection. Limited host growth represents many plants with a fixed carrying capacity as, for example, main root axes of wheat, while unlimited growth characterizes the early stages of extensive proliferation of lateral roots of many other grasses. An example of limited growth is given by the logistic function, for which

$$\rho_s(t) = \frac{r_s N_{max}}{1 + J\exp(-r_n t)}, \quad G(t) = \frac{r_n J \exp(-r_n t)}{1 + J\exp(-r_n t)},$$

where $J = (N_{max}/N_0 - 1)$.

3. EQUILIBRIUM AND DYNAMICAL BEHAVIOUR

We consider the influence of three dynamical factors, inoculum survival, host growth and secondary infection, on disease trajectories and equilibrium behaviour of epidemics that are initiated by primary infection from a reservoir of inoculum. The survival of primary inoculum is treated simply as an exponential decay that is switched on or off by adjusting r_d. Host growth is treated as being effectively static,

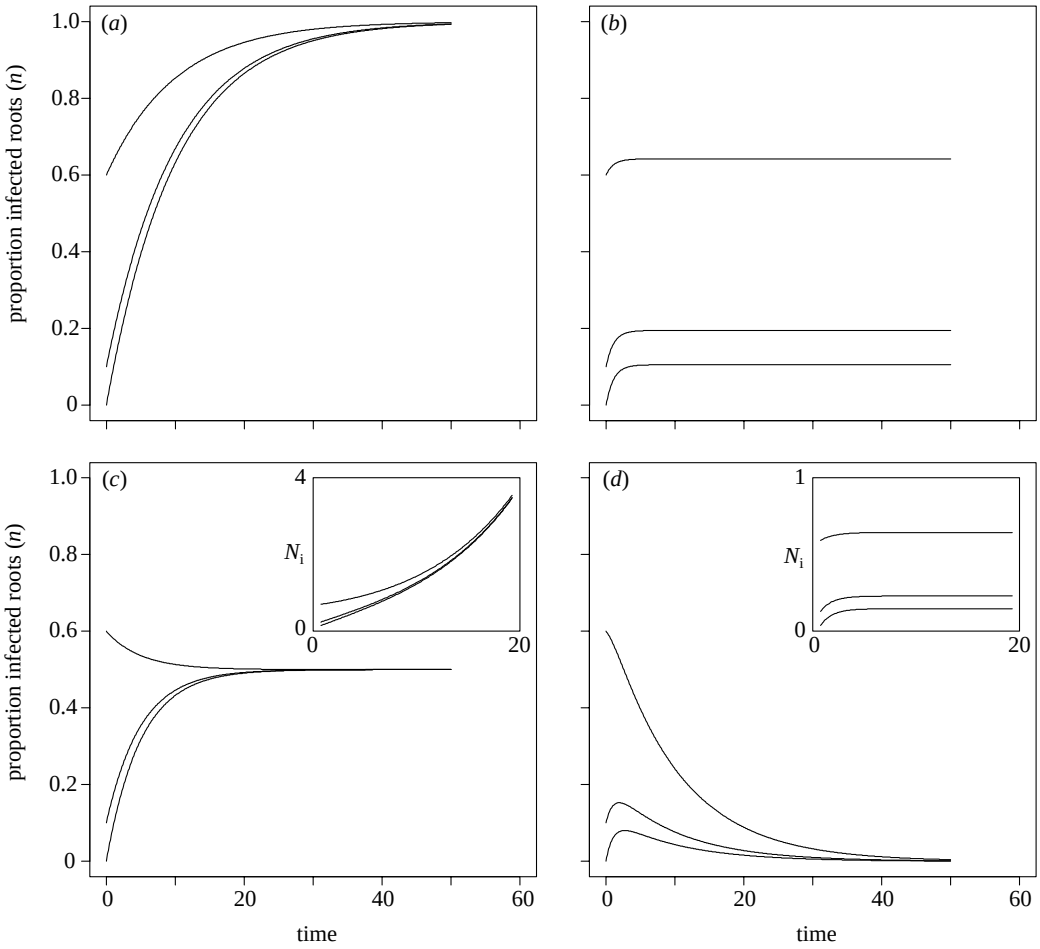

Figure 1. Typical disease progress curves generated by the G model with no secondary infections. The main figures show $n = N_i/N$ as a function of t, whereas the inserts present the absolute numbers of infected roots N_i for the first 20 (arbitrary) time units. Figures are arranged so that r_d changes between columns: (a) and (c) $r_d = 0$; (b) and (d) $r_d = 0.9$, and r_n changes between rows: (a) and (b) $r_n = 0.0$; (c) and (d) $r_n = 0.1$. Other parameters: $r_p = 0.1$, $P(0) = 1$, $N(0) = 1$. Initial conditions: $n(0) = 0$, 0.1 and 0.6.

limited or unlimited, relative to disease dynamics as described above. There is no explicit feedback between disease or infection and host growth (i.e. the functions in f depend only on total host tissue and not diseased tissue). Secondary infection is switched off by setting $r_s = 0$.

Analytical solutions were obtained for certain conditions. These are classified in tables 6 and 7, and are given in Appendix 2. Wherever possible, the dynamical behaviour of the models was examined using these solutions; otherwise, the equations were solved numerically using facsimile (Anon. 1995).

(*a*) *G model*

The equilibrium densities of the proportion of infected roots (n) are summarized in table 2. Secondary infections are very important: the host can outgrow the infection if $r_s = 0$ but cannot if $r_s \neq 0$. If inoculum decays and there are no secondary infections, the equilibrium state depends on the initial conditions of inoculum and the proportion of infected roots (table 2). Since the correlation between initial and equilibrium density is positive, it follows that the dependence on initial conditions may perpetuate patchiness of disease and inoculum in the fields. Thus lower densities of initial inoculum result in less disease which, in turn, produces less inoculum for the next season, while regions of higher initial inoculum give rise to more disease which, in turn, maintains a high level of inoculum.

(i) *No secondary infection*

In the absence of host growth, secondary infection and decay of inoculum, the epidemic saturates the host population (figure 1*a*). The approach to equilibrium is monomolecular (monotonically increasing without inflection, see table 6, Appendix 2). When there is decay of inoculum, the equilibrium levels of infection depend on initial conditions (figure 1*b*, table 2). Host growth, in the form of exponential increase, suppresses the *relative* levels of infection, although absolute levels increase because there is no death or removal of infectious tissue. The dynamical pattern of the disease progress curves is preserved when there is no decay of inoculum (cf. figures 1*a*,*c*).

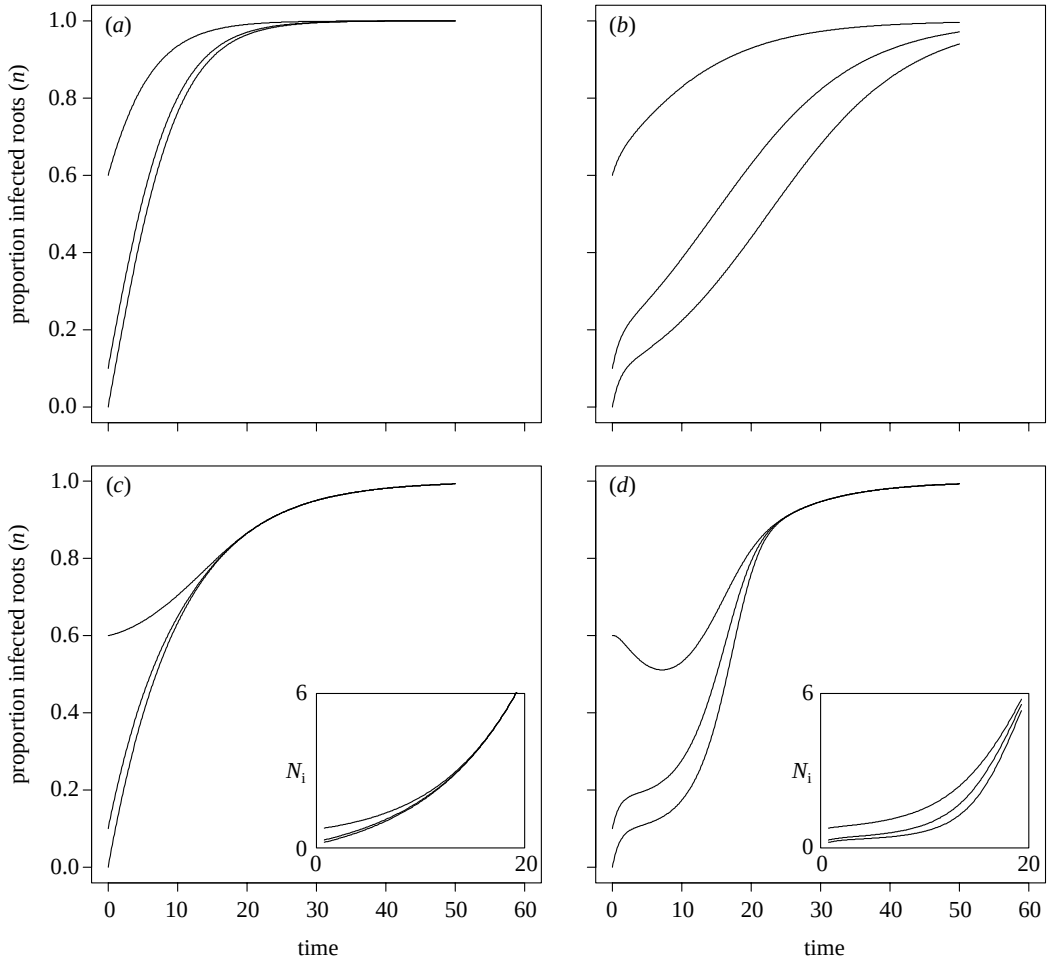

Figure 2. Typical disease progress curves generated by the G model with secondary infections, $r_s = 0.1$. The main figures show $n = N_i/N$ as a function of t, whereas the inserts present the absolute numbers of infected roots N_i for the first 20 (arbitrary) time units. Figures are arranged so that r_d changes between columns: (*a*) and (*c*) $r_d = 0$; (*b*) and (*d*) $r_d = 0.9$, and r_n changes between rows: (*a*) and (*b*) $r_n = 0.0$; (*c*) and (*d*) $r_n = 0.1$. Other parameters: $r_p = 0.1$, $P(0) = 1$, $N(0) = 1$. Initial conditions: $n(0) = 0$, 0.1 and 0.6.

In this case, the relative level of infection at equilibrium ($r_pP/(r_pP + r_n)$) is less than one and depends on the initial amount of primary inoculum, P, (table 2 and figure 1*c*). When, however, inoculum decays, disease equilibria are zero, so the host is able to outgrow infection, since further spread of infection ceases after the initial inoculum is exhausted (figure 1*d*, table 2).

(ii) *Secondary infection*

When secondary infections occur, all roots become infected (figure 2 and table 2). Decay of inoculum and the relative growth of the host combine to affect the dynamics of infection (cf. figures 2*c*,*d*). In particular, after an initial period of increase (driven by primary infection), the proportional amount of infection slows down. The deceleration is caused by decay of inoculum, which reduces the rate of primary infection before secondary infection begins to build up. Rapid growth of the host (figure 2*d*) may dilute the amount of infection so much that the relative amount of infection declines. This is shown more clearly in figure 3 in which several disease trajectories for the same initial condition ($N_i = 0$) are shown for different rates of secondary infection.

In the absence of secondary infection and without decay of inoculum (figures 1*a*,*b* and lowest curves ($r_s = 0$) in figures 3*a*,*b*), disease trajectories are monomolecular. Allowance for secondary infection introduces a point of inflection to the curves (figures 3*a*,*b*). Note that without decay of inoculum but with growth of the host, there will be one point of inflection, or two if separated by a maximum, as long as $r_s > r_n + r_pP + \sqrt{(4r_sr_pP)}$. The stimulatory effect of secondary infection may be delayed by rapid growth of the host (figure 3*d*).

(*b*) *SW* model

The proportion of infected root length reaches saturation ($l_\infty = 1$) for the *SW* model in most cases (table 3). Two exceptions occur depending on whether or not inoculum decays when there is no secondary infection and the host grows exponentially (table 3). Thus when inoculum decays, primary infection is reduced and the host outgrows the epidemic ($l_\infty = 0$).

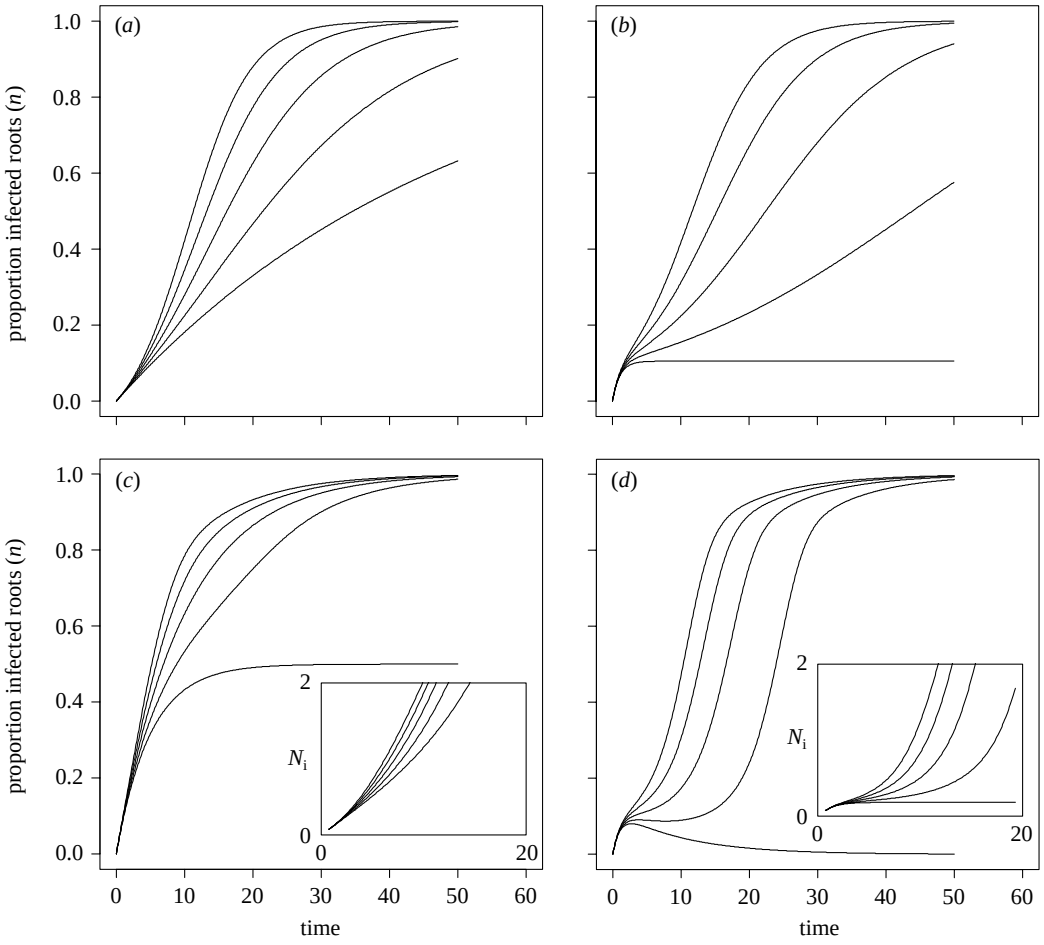

Figure 3. Sensitivity of shapes of the disease progress curves for the G model to the changes in secondary infections. r_d changes between columns: (a) and (c) $r_d = 0$; (b) and (d) $r_d = 0.9$, and r_n changes between rows: (a) and (b) $r_n = 0.0$; (c) and (d) $r_n = 0.1$. Different curves correspond to different values of $r_s = 0$, 0.05, 0.1, 0.15 and 0.2, with initial conditions fixed ($N_i(0) = 0$). Other parameters: $r_p = 0.1$, $P(0) = 1$, $N(0) = 1$, except in (a) where $r_p = 0.02$ for the sake of clarity. Note that the curves for $r_s = 0$ are comparable to figure 1.

If inoculum does not decay, the host does not completely outgrow the epidemic and the proportion of infected root is given by

$$l_\infty = \frac{\sqrt{4r_l r_p P + r_n^2} - r_n}{\sqrt{4r_l r_p P + r_n^2} + r_n}.$$

The density of lesions (number of lesions per unit length) exhibits three types of equilibrium behaviour, depending on the growth dynamics of the host (table 3). When there is no host growth, u_∞ depends on initial conditions $(u(0), l(0))$. For limited host growth, u will approach some finite value, so that no further lesions are produced. When the host grows exponentially and secondary infection occurs, lesions continue to arise on the newly expanded host tissue and therefore u grows exponentially (table 3). Without secondary infections, the density of lesions is either finite or zero depending on the dynamics of inoculum.

Disease trajectories for the SW model are conveniently considered as phase portraits for l versus u, to show concomitant changes in these variables. Trajectories are shown in figure 4 (no secondary infection) and figure 5 (secondary infection) for four sets of initial conditions corresponding to no infection ($u = 0$, $l = 0$), few large lesions (0.01, 0.5), many small lesions (0.5, 0.01) and many large lesions (0.5, 0.5).

(i) *No secondary infection*

When there is no host growth and no secondary infection, inoculum decay does not affect the qualitative dynamics of disease (figures 4*a*,*b*). The trajectories settle on equilibria determined by the initial conditions. With the host growing exponentially (figures 4*c*,*d*), the trajectories look initially similar to the case with a static host, at least for small initial loads of infection. With constant inoculum density, the absolute levels of infected root (L_i) and lesions (U) reflect the exponential growth of the host (see insert in figure 4*c*). When inoculum decays, L_i increases more slowly than L and U levels off (see insert in figure 4*d*). The asymptotic level of U depends on initial conditions. It follows that since $u = U/L$, then the trajectories for the *relative* loads of infection (l) and lesions (u) collapse on the single equilibrium (zero

Table 3. *Equilbrium values for the SW model*

(First formula in each column corresponds to l_∞, second to u_∞. The results are independent of the particular form of inoculum decay, apart from case (d) below which assumes an exponential decay with the rate r_d. For limited growth there are no analytical predictions for equilibrium values. Numerical evidence suggests the dependence on initial conditions in this case.)

host growth	$r_\mathrm{s}=0$	$r_\mathrm{s}\neq 0$
no inoculum dynamics		
zero	1, $\sqrt{\dfrac{4r_\mathrm{p}P(1-l(0))+r_\mathrm{l}u^2(0)}{2r_\mathrm{l}}}$ [a]	1, $\sqrt{r_\mathrm{l}r_\mathrm{s}Lu(0)^2+(r_\mathrm{p}P+r_\mathrm{s}L)^2-(r_\mathrm{p}P+r_\mathrm{s}Ll(0))^2}\times(r_\mathrm{l}r_\mathrm{s}L)^{-1/2}$ [b]
limited	1, $\sqrt{r_\mathrm{p}P/r_\mathrm{l}}$	1, $\sqrt{(r_\mathrm{p}P+r_\mathrm{s}L_\infty)/r_\mathrm{l}}$
exponential	$\dfrac{\sqrt{4r_\mathrm{l}r_\mathrm{p}P+r_\mathrm{n}^2}-r_\mathrm{n}}{\sqrt{4r_\mathrm{l}r_\mathrm{p}P+r_\mathrm{n}^2}+r_\mathrm{n}}$, $\dfrac{\sqrt{4r_\mathrm{l}r_\mathrm{p}P+r_\mathrm{n}^2}-r_\mathrm{n}}{2r_\mathrm{l}}$	1, ∞ [c]
inoculum decays to 0		
zero	1, $(1/r_\mathrm{l})[\sqrt{2r_\mathrm{l}r_\mathrm{p}P(0)(1-l(0))+(r_\mathrm{l}u(0)+r_\mathrm{d})^2}-r_\mathrm{d}]$ [d]	1, depends on initial conditions
limited	no analytical results	
exponential	0, 0	1, ∞ [c]

Particular results for $u(0)\to 0$ and $l(0)\to 0$:
[a] $u_\infty=\sqrt{(2r_\mathrm{p}P/r_\mathrm{l})}$.
[b] $u_\infty=\sqrt{((r_\mathrm{s}L+2r_\mathrm{p}P)/r_\mathrm{l})}$.
[c] $u_\infty\sim l_\infty/(1-l_\infty)$ and approaches ∞ with $l(t)$ approaching 1.
[d] $u_\infty=(\sqrt{(2r_\mathrm{l}r_\mathrm{p}P(0)+r_\mathrm{d}^2)}-r_\mathrm{d})/r_\mathrm{l}$.

Table 2. *Equilibrium values for the G model. The table shows the values of* $n=N_\mathrm{i}/N$

(The results are independent of the particular form of inoculum decay, apart from case (a) below which assumes an exponential decay with the rate r_d.)

host growth	$r_\mathrm{s}=0$	$r_\mathrm{s}\neq 0$
no inoculum dynamics		
zero or limited	1	1
exponential	$r_\mathrm{p}P/(r_\mathrm{p}P+r_\mathrm{n})$	1
inoculum decays to 0		
zero or limited	$1-(1-n(0))\times\exp(-r_\mathrm{p}P(0)/r_\mathrm{d})$ [a]	1
exponential	0	1

[a]In particular, if $n(0)=0$, then

$$n_\infty=1-\exp(-r_\mathrm{p}P(0)/r_\mathrm{d}).$$

for the decaying inoculum) instead of going towards the line of $l_\infty=1$ (cf. figures 4*c*,*d* with figures 4*a*,*b*).

(ii) *Secondary infection*

When there is no host growth but secondary infection occurs, the dynamics are essentially similar to those without secondary infections (cf. figures 5*a*,*b* with figures 4*a*,*b*).

The combined effects of secondary infection and exponential host growth lead to quite different behaviour from the case with no secondary infection (cf. figures 5*c*,*d* with figures 4*c*,*d*). The effect of host growth becomes apparent in the later stages of the epidemic when the trajectories converge and then follow a common unbounded trajectory (figures 5*c*,*d*). The underlying dynamics are determined by a faster rate of increase in the *absolute* number of lesions (U) relative to the total amount of host tissue (L).

When the host growth is limited by logistic growth, here with $L_\infty=3$, the dynamics become more complicated. The infection fills all the available tissue even when inoculum decays (cf. figures 6*a*,*b*, where $l_\infty\to 1$, with figures 4*c*,*d*, where $l_\infty<1$). The dynamical behaviour for logistic growth is initially similar to the case for exponential growth but as the host growth slows down, infection prevails and draws the trajectories towards the line $l_\infty=1$. Note that small differences in the values for u_∞ for different initial

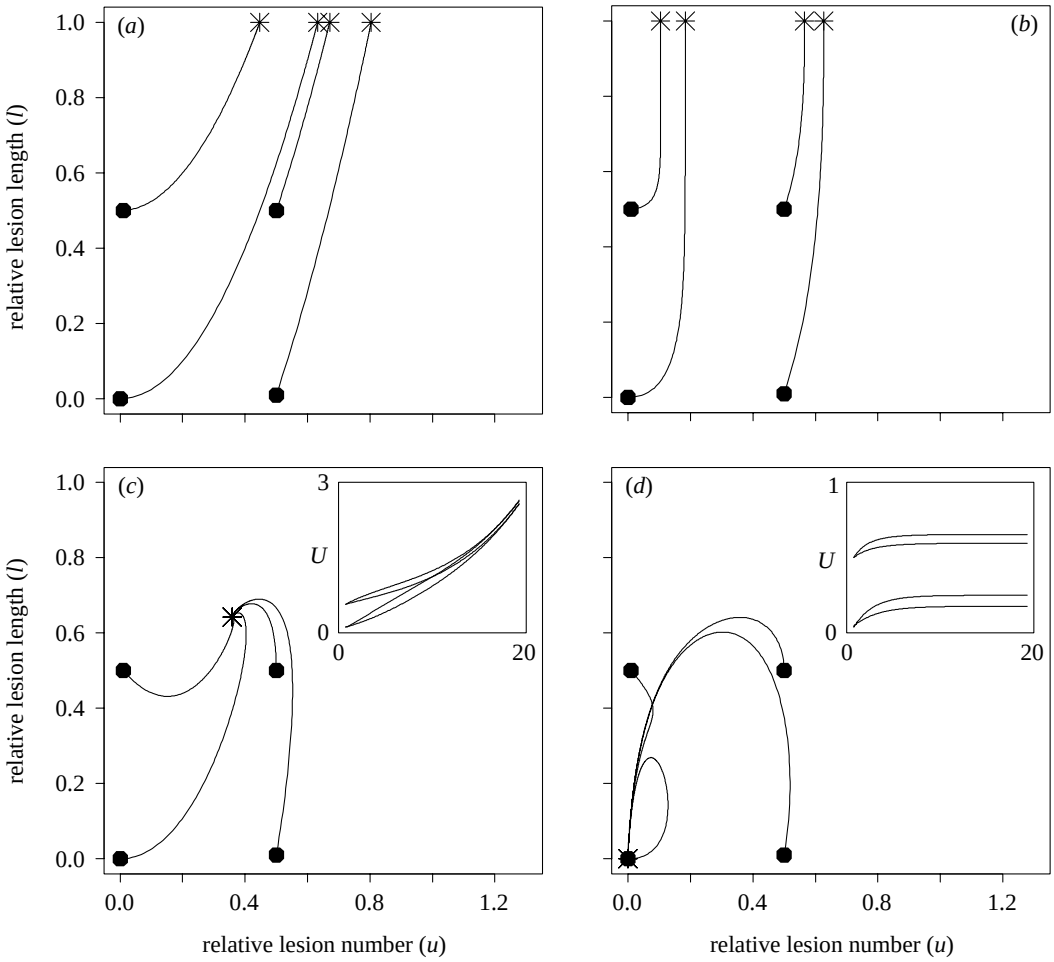

Figure 4. Phase portraits for the *SW* model with no secondary infection, but with inoculum decay and exponential host growth. Main figures show $l(t)$ as a function of $u(t)$, whereas the inserts present the absolute numbers of lesions $U(t)$ as a function of t for the first 20 (arbitrary) time units. Dots indicate the starting points and stars the equilibria. Figures are arranged so that r_{d} changes between columns: (*a*) and (*c*) $r_{\mathrm{d}} = 0$; (*b*) and (*d*) $r_{\mathrm{d}} = 0.5$, and r_{n} changes between rows: (*a*) and (*b*) $r_{\mathrm{n}} = 0.0$; (*c*) and (*d*) $r_{\mathrm{n}} = 0.1$. Other parameters: $r_{\mathrm{p}} = 0.1$, $P(0) = 1$, $r_{\mathrm{l}} = 0.5$, $L(0) = 1$. Initial conditions $(u, l) = (0, 0)$, $(0.01, 0.5)$, $(0.5, 0.01)$ and $(0.5, 0.5)$.

conditions in figures 6*a*,*c*,*d* can be attributed to the numerical round-off error. Further numerical investigation suggests that the different equilibrium values for u in figure 6*b* are real but there is no analytical proof for this.

4. DISCUSSION

We have developed and analysed two models that encapsulate the basic dynamics and commonly measured disease variables of epidemics of soil-borne plant pathogens. The models incorporate primary and secondary infection, inoculum and host dynamics. The *G* model describes the change in disease status of host organs, typically roots, during an epidemic. The *SW* model describes the change in density of lesions or infections and the length of diseased or infected tissue. The *G* model can be simply adjusted to model the status of individual plants. The models are presented for pathological infection but, given the absence of a functional response for the effects of disease on host growth, the results presented here apply also to beneficial colonization by vesicular-arbuscular mycorrhizae, as originally discussed by Smith & Walker (1981), Walker & Smith (1984), as well as Buwalda *et al.* (1982). We shall discuss elsewhere the introduction of functional responses into the models.

The models have been used to address specific biological questions about the role of primary and secondary infection, host growth and inoculum decay on the dynamics of infection. Although the models represent simplified systems, the existence of analytical solutions for certain situations of host growth, inoculum decay and secondary infection provide insight into some of the possible dynamics that may be expected and tested in the field. Whereas plants may outgrow epidemics, especially when the source of inoculum in soil decays, secondary infection can prevent this, resulting in saturation with infection and disease. Although primary and secondary infection occur simultaneously during at least part of an epidemic, the change from a phase of predominantly primary infection to secondary infection may lead to

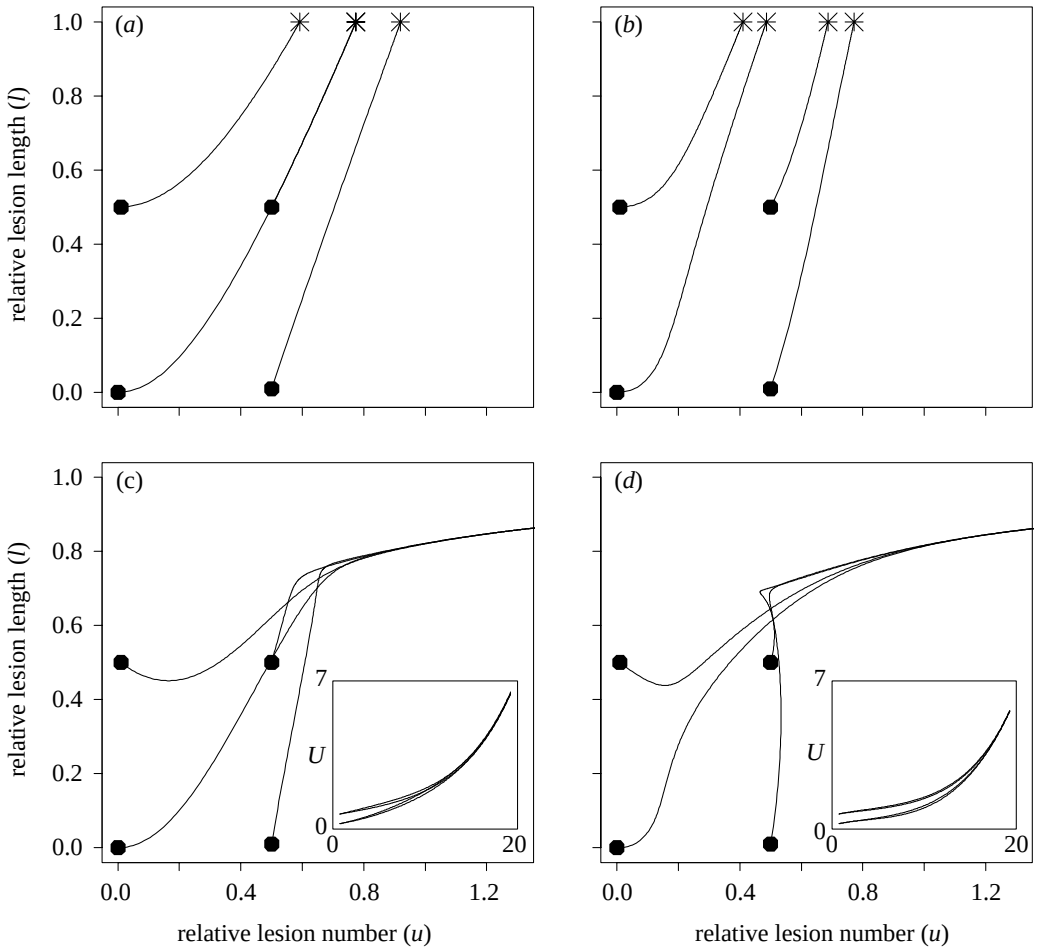

Figure 5. Phase portraits for the *SW* model with secondary infection, $r_s = 0.1$, with inoculum decay and exponential host growth. Main figures show $l(t)$ as a function of $u(t)$, whereas the inserts present the absolute numbers of lesions $U(t)$ as a function of t for the first 20 (arbitrary) time units. Dots indicate the starting points and stars the equilibria. Figures are arranged so that r_d changes between columns: (*a*) and (*c*) $r_d = 0$; (*b*) and (*d*) $r_d = 0.5$, and r_n changes between rows: (*a*) and (*b*) $r_n = 0.0$; (*c*) and (*d*) $r_n = 0.1$. Other parameters: $r_p = 0.1$, $P(0) = 1$, $r_l = 0.5$, $L(0) = 1$. Initial conditions $(u, l) = (0, 0)$, $(0.01, 0.5)$, $(0.5, 0.01)$ and $(0.5, 0.5)$.

a marked change in epidemic rates. This may take the form of a marked acceleration as the multiplicative effect of secondary infection occurs (figure 3*d* for the *G* model). It can also, in certain circumstances, lead to a temporary or even persistent plateau in the period during which most primary infection is completed as the reservoir of soil inoculum decays to zero and a delay occurs before secondary infection accelerates (figure 7).

The sensitivity of the systems to initial conditions (tables 2 and 3, figures 1*c*, 4*a*,*b*, 5*b*) may have important consequences for the occurrence and persistence of disease patches in fields. Thus small differences in the amounts of initial inoculum or of infected tissue in transplanted seedlings could become amplified by the inherent nonlinear dynamics leading to patches with markedly different levels of disease in the same field.

Several other models have been proposed for the dynamics of plant parasitic fungi. Most of these are elaborations of logistic formulations with allowance for host growth. They deal with either primary or secondary infection. They are discussed in Gilligan (1990). We note that Jeger (1987) also proposed a model for primary infection that linked lesion growth and lesion length. The model is given, after some reparameterization, by

$$\text{infected root length:} \quad \frac{\mathrm{d}L_i}{\mathrm{d}t} = (r_i + r_p P)(L - L_i), \tag{16}$$

$$\text{total root length:} \quad \frac{\mathrm{d}L}{\mathrm{d}t} = r_n(L - L_i), \tag{17}$$

$$\text{inoculum:} \quad \frac{\mathrm{d}P}{\mathrm{d}t} = -r_d P. \tag{18}$$

In practice, this model exhibits some of the mathematics of the *G* model and the biology of the *SW* model. Because we wish to keep the mathematical and biological paradigms expressed in equations (6)–(6) and (9)–(9) separate, we do not discuss the Jeger model in detail here. We show in Appendix 1, however, that the model can be derived as a special case of the generic model that encompasses the *G* and *SW* models.

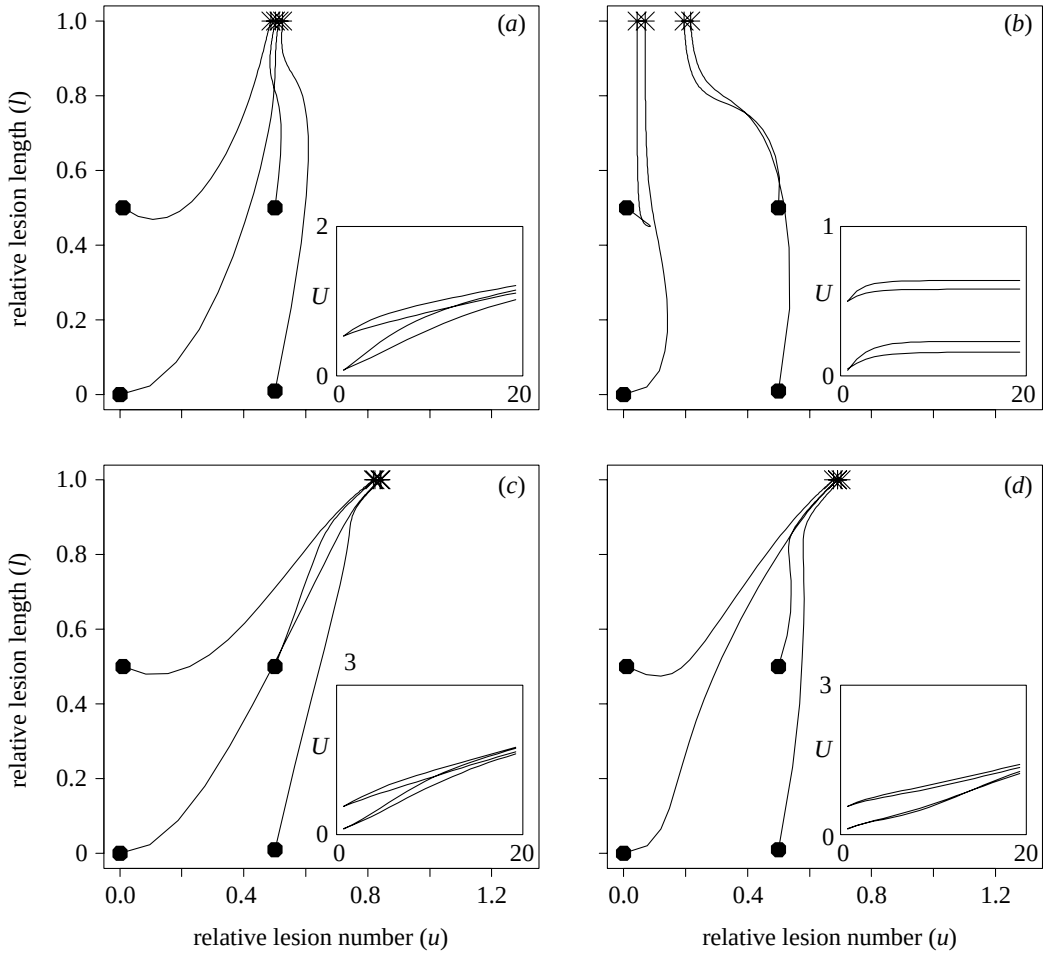

Figure 6. The solution of the SW model for the logistic host growth with the rate $r_{\mathrm{n}} = 0.1$ and the carrying capacity $L_{\max} = 3$. Figures (a) and (b) should be compared with figure 4c,d, and figures (c) and (d) with figure 5c,d. r_{d} changes between columns: (a) and (c) $r_{\mathrm{d}} = 0$; (b) and (d) $r_{\mathrm{d}} = 0.5$. Other parameters: $r_{\mathrm{p}} = 0.1$, $P(0) = 1$, $r_{\mathrm{l}} = 0.5$, $L(0) = 1$. Initial conditions $(u, l) = (0, 0)$, $(0.01, 0.5)$, $(0.5, 0.01)$ and $(0.5, 0.5)$.

The G and SW models presented in equations (6)–(6) and (9)–(9) can be broadly separated into the paradigms of microparasitic and macroparasitic models proposed by Anderson & May (1979) and May & Anderson (1979) for medical and animal epidemics. There are, however, important differences that are dictated by the life cycles of plant parasitic fungi and by the nature of the disease and host variables that can be measured.

Microparasites, which are typified by viruses, bacteria and protozoa amongst animal parasites are characterized by small size with short generation times and extremely high rates of direct reproduction in the host. Infection is usually passed from host to host by direct (binary) contact. The duration of infection is short compared with the lifespan of the host and the development of immunity towards repeated infection is common. Typically, the population of hosts (N) is classified into presence or absence of infection. Infected individuals enter a removed class if they become immune or die. The system is modelled by transfer of individuals between three classes (SIR) representing susceptibles (S), infecteds (I) and removals (R), where $N = S + I + R$, thus

$$\frac{\mathrm{d}S}{\mathrm{d}t} = bN - dS - \beta SI, \tag{19}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - (d + \alpha + \nu)I, \tag{20}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \nu I - dR, \tag{21}$$

in which b is a source of susceptibles, d is the per capita death rate of individuals, α is the enhanced per capita death rate due to infection, β is the transmission rate of infection between infected and susceptible individuals (which may in certain circumstances depend on N (Heesterbeek & Roberts 1995)) and ν is the rate of recovery.

Macroparasites are larger parasites. They are typified by parasitic helminths. They have longer generation times than macroparasites. Direct multiplication within the host is slow or absent. The infection (inoculum) usually comes from an external source involving free-living stages or secondary hosts with their own dynamics. Immune responses generally depend on the number of parasites in the host and persist for only a short time. The susceptible

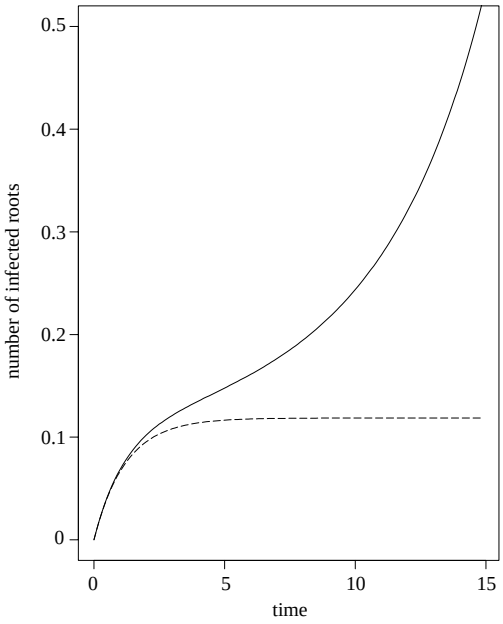

Figure 7. The solution of the G model with secondary infections ($r_s = 0.05$, solid line), exponential host growth ($r_n = 0.1$) and exponential inoculum decay ($r_d = 0.9$) displaying the plateau effect discussed in the text. The solution for $r_s = 0$ is also shown (broken line). Other parameters identical to those in figure 3*d*.

class can again be distinguished among hosts, but the load of infection (i.e. the number of parasites per host) is an important factor in determining the rates of pathogenicity, resistance and removal; thus typical variables describe the number of hosts (N), parasites in the hosts (X) and their free-living stages (w):

$$\frac{dN}{dt} = (a-d)N - \alpha X, \tag{22}$$

$$\frac{dX}{dt} = \beta w N - (\mu + d + \alpha)X - F(X,N), \tag{23}$$

$$\frac{dw}{dt} = \lambda X - cw - \beta w N, \tag{24}$$

in which b, d are per capita birth and death rates for hosts, λ and c are the per capita birth and death rates for free-living parasites, α is the per capita infection-induced death rate, β is the transmission parameter between free-living parasite and hosts, μ is the natural parasite death rate and $F(X,N)$ represents a correction for uneven distribution of macroparasites amongst hosts (May & Anderson 1979).

The G model can be considered as a generalization of the standard microparasitic SIR model by including an external source of infection (inoculum). Roots are classified as infected ($N_i \equiv I$) or susceptible ($N - N_i \equiv S$). Thus the mass action term in a classical SIR model (βSI) is replaced by a term in $\beta S(I+P)$, where P represents the external source of inoculum. This type of SIR model, but with constant and small P, has recently been discussed by Engbert & Drepper (1994) in the context of seasonally driven epidemics. The external source of inoculum is analogous to the free-living stages of the macroparasitic models. Though we have not allowed for recovery or death of roots in the present models, the introduction of a removed class can be incorporated into the G model. We discuss elsewhere the use of SIR models for epidemics of soil-borne pathogens where the force of infection decays as the host population becomes resistant to infection (Gilligan *et al.* 1997).

The SW model is allied to the macroparasitic model, with free-living stages (inoculum) together with measures of host availability and parasite load. However, for the SW model, host availability is represented by the total amount of host tissue rather than a density of discrete hosts. Parasite load is comprised not of density of parasites but two variables, one for lesion density (U) and the other (L_i) for the proportion of infected tissue. The secondary infection term (representing within host multiplication and/or direct transmission) is absent in the macroparasitic models but important for plant systems.

The relationships between variables discussed in this paper and those of equations (19)–(19) and (22)–(22) for microparasitic and macroparasitic models are summarized in tables 4 and 5 from which the hybrid nature of the models can be seen. We conclude that, while it is encouraging to adapt the microparasitic–macroparasitic classification, the differences in the fungal systems introduced here are important since they represent not only the dynamics but also the types of data that can be collected for epidemiological analysis.

Further elaboration of the G and SW models is possible to incorporate functional responses that allow for the effects of infection on root growth (see Gilligan *et al.* 1997), so that the functions f from equations (6)–(6) and (9)–(9) depend not only on N or L (i.e. total root number or root length) but also on the infected portions, N_i and L_i, respectively. In addition, we can also include a term describing death of infected roots. In the case of the G model this results simply in addition of $-gN_i$ to equations (6), (6) for infected and total roots; g defines infection induced death. For the SW model, if a root dies the number of lesions, the length of infected tissue and the total length of tissue are all reduced (cf. equations (28)–(28) below with equations (9)–(9)). The functional responses can be further adjusted to allow for clumping in the manner suggested by Anderson & May (1979).

The dynamics of inoculum in both the models can be elaborated to allow for: (i) a Gaussian or other decay function; (ii) exhaustion of inoculum by germination and infection; and (iii) release of inoculum from dying roots. The models then become (ignoring clumping and assuming exponential decay of inoculum)

$$\frac{dN_i}{dt} = (r_p P + r_s N_i)(N - N_i) - gN_i, \tag{25}$$

$$\frac{dN}{dt} = r_n f(N, N_i), \tag{26}$$

$$\frac{dP}{dt} = -r_d P - \eta r_p P(N - N_i) + \rho g N_i, \tag{27}$$

Table 4. *The variables and parameters for the G model (presented in equations (6)–(6)) compared with the microparasitic model (equations (19)–(19))*

G	microparasite	comments
N	N	
N_i	I	
$N - N_\mathrm{i}$	S	or $S + R$, with R class frequently absent in plants
P	—	no direct equivalence in simple microparasitic models (unless expressed as an external source of infection); dynamical variable for plants
r_n	$b - d$	—
$f(N)$	—	no direct equivalence in simple microparasitic models
—	$\alpha + d$	no direct equivalence in G model but could be incorporated as effect of infected roots on production of new roots, cf. g in equations (25)–(25)
r_s	β	—
r_d	—	no direct equivalence in simple microparasitic models

Table 5. *The variables (and parameters for the SW model (equations (9)–(9)) compared with the macroparasitic model (equations (22)–(22))*

SW	macroparasite	comments
L	N	—
L_i	—	no direct equivalence in simple macroparasitic models but can be given an 'immunological' interpretation, cf. equation (32)
U	X	—
P	w	—
r_n	$a - d$	—
$f(L)$		no direct equivalence in simple macroparasitic models
—	$\alpha + d + \mu$	no direct equivalence in SW model but could be incorporated as effect of infected roots on production of new roots, cf. g in equations (28)–(28)
—	$F(X, N)$	clustering of lesions on dying roots is not considered here
$r_\mathrm{p}(1 - \epsilon U)$	β	$(1 - \epsilon U)$ can be given an 'immunological' interpretation, cf. equation (32) in text
r_s	—	no direct equivalence (within-host multiplication or direct contacts)
—	λ	no direct equivalence in SW model but could be incorporated as release of inoculum from dying roots, cf. ρg in equation (28)
r_d	c	—
r_l	—	no direct equivalence in simple macroparasitic models (but can be given an interpretation of the immunological response)

and

$$\frac{\mathrm{d}U}{\mathrm{d}t} = (r_\mathrm{p}P + r_\mathrm{s}L_\mathrm{i})(L - L_\mathrm{i}) - gU, \tag{28}$$

$$\frac{\mathrm{d}L_\mathrm{i}}{\mathrm{d}t} = r_\mathrm{l}U\left(1 - \frac{L_\mathrm{i}}{L}\right) - gL_\mathrm{i}, \tag{29}$$

$$\frac{\mathrm{d}L}{\mathrm{d}t} = r_\mathrm{n}f(L, L_\mathrm{i}) - gL_\mathrm{i}, \tag{30}$$

$$\frac{\mathrm{d}P}{\mathrm{d}t} = -r_\mathrm{d}P - \eta r_\mathrm{p}P(L - L_\mathrm{i}) + \rho g L_\mathrm{i}, \tag{31}$$

in which η is the per capita reduction of inoculum due to germination and ρ measures inoculum release from infected tissue. We show elsewhere how to elaborate the transmission parameters r_p and r_s in order to analyse small scale effects within the pathozone or rhizosphere on the large scale development of an epidemic (Kleczkowski & Gilligan, unpublished work).

Immunity to fungal infection of plants does not play a significant role in infection. The formalism developed by Anderson & May (1991) for the immunological responses of a host to macroparasites can, however, be used to describe how the probability of lesion production depends on the length of uninfected portions of the root. In the absence of secondary infection, equation (9) for U can be rewritten as

$$\frac{\mathrm{d}U}{\mathrm{d}t} = r_\mathrm{p}PL(1 - L_\mathrm{i}/L) = r_\mathrm{p}PL(1 - \varepsilon U), \tag{32}$$

where the term $1 - \varepsilon U$ can be interpreted as an increase in the 'immunity' of a root to further infection due to the current status of infection, and $\varepsilon = L_\mathrm{i}/LU$. This is a special case of the general formula

$$1 - \varepsilon \int_0^a U(a, t)\exp(\sigma(a - a'))\,\mathrm{d}a',$$

introduced in Anderson & May (1991) to describe acquired immunity against macroparasitic infections, with σ regulating the length of memory and a the age of the host. This, however, leads towards an age-structure model of root infection and exceeds the scope of this paper.

The models presented in this paper continue and extend earlier work on the analysis of botanical epidemics and the spread of root infection (Smith & Walker 1981; Walker & Smith 1984; Gilligan 1985, 1990, 1994; Jeger 1987). The models also form a basis for elaborations incorporating spatial heterogeneity, functional responses for the effects of pathogens on host growth and for scaling from the behaviour of individual propagules to mean field behaviour. Linking of the dynamics of primary infection, which is analogous to an external source of infection in SIR models or to free-living stages in macroparasitic models, and of secondary infection, is likely to find important applications in animal as well as botanical epidemiology.

This work was funded by a grant from the Biotechnology and Biological Sciences Research Council, which we gratefully acknowledge.

APPENDIX 1. GENERAL MODEL

In this appendix we present a general model for the spread of soil-borne pathogens and show how the G and SW models can be obtained as special cases. For brevity, we use the term lesion to include regions of continuous disease (lesion *sensu stricto*) and regions of continuous infection (infection units, *sensu* (Cox & Sanders 1974)). We also show that the general model includes a model for monomolecular primary infection proposed by Jeger (1987).

(a) *General model*

(i) *Formation of new lesions*

. We follow the convention of the SW model and consider first a single root (or a single organ of a plant). Assume that the rate at which new lesions are formed is proportional to the length of uninfected portions of the root $L - L_\mathrm{i}$ multiplied by a force of infection λ:

$$\frac{\mathrm{d}U}{\mathrm{d}t} = \lambda(L - L_\mathrm{i}), \tag{33}$$

where λ is a sum of two sources of infection, $r_\mathrm{p}P$ (primary infection, caused by initial inoculum) and $r_\mathrm{s}L_\mathrm{i}$ (secondary, caused by the infected tissue):

$$\lambda = r_\mathrm{p}P + r_\mathrm{s}L_\mathrm{i}. \tag{34}$$

If individual lesions rather than the whole tissue form a source of secondary inoculum, the last term should be replaced by $r_\mathrm{s}U$.

(ii) *Lesion growth*

Let c be the average size of a lesion, then

$$c = \frac{L_\mathrm{i}}{U},$$

$$L_\mathrm{i} = Uc,$$
$$\frac{\mathrm{d}L_\mathrm{i}}{\mathrm{d}t} = c\frac{\mathrm{d}U}{\mathrm{d}t} + U\frac{\mathrm{d}c}{\mathrm{d}t}. \tag{35}$$

(iii) *Multiroot system*

Equations (33)–(35) can be used to describe infection dynamics in a multiroot system. Denoting by a superscript (j) a jth root, we obtain

$$\frac{\mathrm{d}U^{(j)}}{\mathrm{d}t} = (r_\mathrm{p}P + r_\mathrm{s}\sum_k L_\mathrm{i}^{(k)})(\hat{L} - L_\mathrm{i}^{(j)}), \tag{36}$$

$$\frac{\mathrm{d}L_\mathrm{i}^{(j)}}{\mathrm{d}t} = \frac{L_\mathrm{i}^{(j)}}{U^{(j)}}\frac{\mathrm{d}U^{(j)}}{\mathrm{d}t} + U^{(j)}\frac{\mathrm{d}c^{(j)}}{\mathrm{d}t}, \tag{37}$$

where $\hat{L}$ is an average length of each root and $c^{(j)}$ is an average lesion size in the jth root. The summation with respect to $k = 1, 2, \ldots$ in (36) includes the same root, because we can have secondary infections between different segments of the same root.

(b) *Special cases*

(i) *SW model*

The SW model does not distinguish between different roots, but rather describes the infection in terms of tissue length $L_\mathrm{i} = \sum L_\mathrm{i}^{(j)}$. The indices in (36) can be omitted and $\hat{L}$ can be assumed to be equal to the total length (or density) of the roots. Additionally, the model assumes the following.

(1) c follows a monomolecular equation, with the carrying capacity determined by an average inter-lesion distance, i.e. $c_\mathrm{max} = L/U$,

$$\mathrm{d}c/\mathrm{d}t = r_l(1 - (c/c_\mathrm{max})). \tag{38}$$

(2) The relative rate of growth of lesions is faster than the rate at which new lesions are produced, thus

$$\frac{1}{U}\frac{\mathrm{d}U}{\mathrm{d}t} \ll \frac{1}{c}\frac{\mathrm{d}c}{\mathrm{d}t}.$$

This leads to the SW model

$$\frac{\mathrm{d}U}{\mathrm{d}t} = (r_\mathrm{p}P + r_\mathrm{s}L_\mathrm{i})(L - L_\mathrm{i}), \tag{39}$$

$$\frac{\mathrm{d}L_\mathrm{i}}{\mathrm{d}t} = r_\mathrm{l}U\left(1 - \frac{L_\mathrm{i}}{L}\right). \tag{40}$$

(ii) *G model*

The G model can be obtained from equations (36) and (36) by assuming that the lesions grow very fast so that any root is either uninfected ($U^{(j)} = 0$, $L_\mathrm{i}^{(j)} = 0$) or infected ($U^{(j)} = 1$, $L_\mathrm{i}^{(j)} = \hat{L}$). Then

$$\sum_j U^{(j)}L_\mathrm{i}^{(j)} = \sum_k L_\mathrm{i}^{(k)},$$

$$\frac{\mathrm{d}U}{\mathrm{d}t} = (r_\mathrm{p}P + r_\mathrm{s}L_\mathrm{i})(L - L_\mathrm{i}),$$

$$\frac{\mathrm{d}N_\mathrm{i}}{\mathrm{d}t} = (r_\mathrm{p}P + r_\mathrm{s}N_\mathrm{i})(N - N_\mathrm{i}),$$

where $L = N\hat{L}$, $\sum_j U^{(j)} = U \equiv N_\mathrm{i}$ and $\sum_j L_\mathrm{i}^{(j)} = L_\mathrm{i} = N_\mathrm{i}\hat{L}$.

(iii) *Jeger model*

A model proposed by Jeger (1987) can be obtained from equations (33) and (35) but with a different form of the monomolecular growth equation for the average lesion size c, cf. (38),

$$\frac{\mathrm{d}c}{\mathrm{d}t} = r_\mathrm{l}(c_\mathrm{max} - c), \tag{41}$$

where $c_\mathrm{max} = L/U$ is a maximal colony size. Then

$$\frac{\mathrm{d}c}{\mathrm{d}t} = r_\mathrm{l}\left(\frac{L}{U} - c\right),$$

$$\frac{\mathrm{d}L_\mathrm{i}}{\mathrm{d}t} = c\frac{\mathrm{d}U}{\mathrm{d}t} + r_l\,(L - L_\mathrm{i}) = \frac{L_\mathrm{i}}{U}\frac{\mathrm{d}U}{\mathrm{d}t} + r_\mathrm{l}\,(L - L_\mathrm{i})\,.$$

Substituting the value for $\mathrm{d}U/\mathrm{d}t$ we arrive at the formula,

$$\frac{\mathrm{d}L_\mathrm{i}}{\mathrm{d}t} = \left(r_\mathrm{l} + \frac{r_\mathrm{s}L_\mathrm{i}^2}{U} + \frac{r_\mathrm{p}P}{U}L_\mathrm{i}\right)(L - L_\mathrm{i})$$

(generalizing the results of Jeger (1987)). Jeger (1987) further simplifies the equation by assuming that the rate at which new lesions are formed decreases with lesion size, thus $r_\mathrm{p} = kc_0/c = kc_0U/L_\mathrm{i}$ (c_0 can be interpreted as a 'minimal' lesion size and k is an 'effective' infection rate). Making a similar assumption about r_s ($r_\mathrm{s} = k'c_0/c$ with another parameter k'), we obtain

$$\frac{\mathrm{d}L_\mathrm{i}}{\mathrm{d}t} = (r_\mathrm{l} + k'c_0L_\mathrm{i} + kc_0P)\,(L - L_\mathrm{i}), \tag{42}$$

where $r_\mathrm{s} = k'c_0/c = k'c_0U/L_\mathrm{i}$. Note that the equation does not contain U.

APPENDIX 2. ANALYTICAL SOLUTIONS

This appendix lists analytical solutions and some general types of behaviour of the solutions of the G and SW models. The principal results are summarized in tables 6 and 7.

(*a*) *G model*

General types of the solutions summarized in table 6 are given below for the G model. For the notation, refer to table 6.

(i) *G.1*

No host growth, no inoculum decay, no secondary infection:

$$N_\mathrm{i}(t) = N_\mathrm{i}(0)\exp(-r_\mathrm{p}Pt) + N(1 - \exp(-r_\mathrm{p}Pt)).$$

(ii) *G.2*

The case for no host growth, no inoculum decay, with secondary infection is shown in equation (43) (for the notation, see table 6).

$$\begin{aligned} n &= y - \chi \\ &= \frac{\nu(\chi + n(0))}{(\chi + n(0))(1 - \exp(-Dt)) + \nu\exp(-Dt)} \\ &\quad - \frac{\chi((\chi + n(0))(1 - \exp(-Dt)) + \nu\exp(-Dt))}{(\chi + n(0))(1 - \exp(-Dt)) + \nu\exp(-Dt)} \\ &= \frac{n(0)(1 + \chi\exp(-Dt)) + \chi(1 - \exp(-Dt))}{n(0)\chi(1 - \exp(-Dt)) + \chi + \exp(-Dt)}, \\ N_\mathrm{i} &= Nn. \end{aligned} \tag{43}$$

(iii) *G.3*

Monomolecular equation with time-dependent carrying capacity κ and constant rate r (for the notation, see table 6):

$$\frac{\mathrm{d}y}{\mathrm{d}t} = r(\kappa(t) - y),$$

$$y(t) = y(0)\exp(-rt) + \int_0^t \exp(r(t - t'))\kappa(t')\,\mathrm{d}t'.$$

(iv) *G.4*

Logistic equation with time-dependent carrying capacity κ and constant rate r (for the notation, see table 6):

$$\frac{\mathrm{d}y}{\mathrm{d}t} = ry(\kappa(t) - y), \quad y(t) = \frac{y(0)F(t)}{1 + ry(0)I(t)},$$

$$F(t) = \exp\left(r\int_0^t \kappa(t')\,\mathrm{d}t'\right), \quad I(t) = \int_0^t F(t')\,\mathrm{d}t'.$$

Note that various authors including Nisbet & Gurney (1976) and Coleman (1979), and especially Ferrandino (in Waggoner 1986), considered a related equation of the form $\mathrm{d}y/\mathrm{d}t = ry(1 - y/\kappa(t))$ in which the carrying capacity κ depends on time.

(v) *G.5*

Exponential host growth, no inoculum decay, no secondary infection:

$$N_\mathrm{i} = N(0)\frac{r_\mathrm{p}P}{r_\mathrm{n} + r_\mathrm{p}P}\exp(r_\mathrm{n}t) + \left(N_\mathrm{i}(0) - N_0\frac{r_\mathrm{p}P}{r_\mathrm{n} + r_\mathrm{p}P}\right)\exp(-r_\mathrm{p}Pt).$$

(vi) *G.6*

No host growth, exponential inoculum decay, no secondary infection:

$$N_\mathrm{i}(t) = N - (N - N_\mathrm{i}(0))\exp(r_\mathrm{p}P_0/r_\mathrm{d}) \times \exp(-\exp(-r_\mathrm{d}(t - \theta))),$$

with $\theta = r_\mathrm{d}^{-1}\ln(r_\mathrm{p}P_0/r_\mathrm{d})$.

(*b*) *SW model*

General types of the solutions summarized in table 7 are given below for the SW model. For the notation, refer to table 7.

(i) *SW.1*

No host growth, no inoculum decay, no secondary infection:

$$\mathrm{d}u/\mathrm{d}t = r_\mathrm{p}P(1 - l), \quad \mathrm{d}l/\mathrm{d}t = r_\mathrm{l}u(1 - l).$$

The second equation can be rewritten by change of variable, $v = 1 - l$, and the result substituted into the

Table 6. *Analytical solutions for the G model (c.c. denotes carrying capacity). The notes (G.1)–(G.6) refer to the text*

host growth	no secondary infection	with secondary infection
	no inoculum dynamics	
zero	monomolecular growth, $\mathrm{d}N_\mathrm{i}/\mathrm{d}t = r_\mathrm{p}P(N - N_\mathrm{i})$, cf. (G.1)	$y = \chi + n$ with $\chi = (r_\mathrm{p}P)/(r_\mathrm{s}N)$ transforms the equation into $\mathrm{d}y/\mathrm{d}t = Dy(1 - y/\nu)$, with $D = r_\mathrm{p}P + r_\mathrm{s}N$, $\nu = 1 + \chi$, the solution matches monomolecular when $\chi = 0$ and logistic when $\chi \to \infty$; cf. (G.2)
time-dependent, including limited growth	monomolecular equation with time-dependent c.c. $\mathrm{d}N_\mathrm{i}/\mathrm{d}t = r_\mathrm{p}P(N(t) - N_\mathrm{i})$, cf. (G.3) with $y = N_\mathrm{i}$, $r = r_\mathrm{p}P$ and $\kappa(t) = N(t)$	$y = \alpha P + N_\mathrm{i}$ with $\alpha = r_\mathrm{p}/r_\mathrm{s}$ transforms the equation into $\mathrm{d}y/\mathrm{d}t = ry(\kappa(t) - y)$, with $r = r_s$ and $\kappa(t) = N(t) + \alpha P$, the logistic equation with time varying c.c., cf. (G.4)
exponential	$\mathrm{d}N_\mathrm{i}/\mathrm{d}t = r_\mathrm{p}P(N(0)\exp(r_\mathrm{n}t) - N_\mathrm{i})$, the solution is a difference of exponentials (G.5)	no closed analytical solution
	inoculum decays to 0	
zero	$\mathrm{d}N_\mathrm{i}/\mathrm{d}t = r_\mathrm{p}P(0)\exp(-r_\mathrm{d}t)(N - N_\mathrm{i})$, monomolecular equation with time-varying rate leading to a Gompertz-like solution, (G.6)	$y = N - N_\mathrm{i}$ leads to $\mathrm{d}y/\mathrm{d}t = ry(\kappa(t) - y)$, with $r = -r_\mathrm{s}$, $\kappa(t) = N + \alpha P(t)$ and $\alpha = r_\mathrm{p}/r_\mathrm{s}$, logistic with time-dependent c.c., cf. (G.3)
time-dependent	change of the *independent* variable from t to P leads to a monomolecular equation with varying c.c. $\mathrm{d}N_\mathrm{i}/\mathrm{d}P = -r_\mathrm{p}/r_\mathrm{d}(N(t(P)) - N_\mathrm{i})$, with $t(P) = \ln(P/P(0))/r_\mathrm{d}$, cf. (G.4)	full Riccati equation with time-dependent coefficients (no analytical solutions)

first equation after the latter is differentiated with respect to time. Then

$$\frac{\mathrm{d}u}{\mathrm{d}t} = r_\mathrm{p}Pv, \quad \frac{\mathrm{d}v}{\mathrm{d}t} = -r_\mathrm{l}uv,$$
$$0 = \frac{\mathrm{d}^2v}{\mathrm{d}t^2} - r_\mathrm{p}P\frac{\mathrm{d}v}{\mathrm{d}t} = \frac{\mathrm{d}^2v}{\mathrm{d}t^2} + r_\mathrm{p}Pr_\mathrm{l}uv$$
$$= \frac{\mathrm{d}^2v}{\mathrm{d}t^2} + r_\mathrm{l}u\frac{\mathrm{d}u}{\mathrm{d}t} = \frac{\mathrm{d}}{\mathrm{d}t}\left(\frac{\mathrm{d}u}{\mathrm{d}t} + \tfrac{1}{2}r_\mathrm{l}u^2\right).$$

Thus

$$\frac{\mathrm{d}u}{\mathrm{d}t} + \tfrac{1}{2}r_\mathrm{l}u^2 = K_1,$$

with K_1 being an integration constant. This is a Riccati equation with constant coefficients and one special solution can be easily found, namely $u(t) = \sqrt{(2K_1/r_\mathrm{l})} = u_\infty$. The transformation of the dependent variable u into $u - u_\infty$ transforms the Riccati equation into the solvable Bernoulli equation. For the general properties of Riccati equation see Zwillinger (1992). Note that $K_1 > 0$ as $u > 0$, $l < 1$, and $\mathrm{d}u/\mathrm{d}t > 0$. The integration constant K_1 can be calculated from $l(0)$:

$$r_\mathrm{p}P(1 - l(0)) = \frac{\mathrm{d}u}{\mathrm{d}t} = K_1 - \tfrac{1}{2}r_\mathrm{l}u^2(0),$$

$$K_1 = r_\mathrm{p}P(1 - l(0)) + \tfrac{1}{2}r_\mathrm{l}u^2(0).$$

The equation can be solved in order to obtain

$$u(t) = \sqrt{\frac{2K_1}{r_\mathrm{l}}} \times \tanh\left(\sqrt{\tfrac{1}{2}r_\mathrm{l}K_1}t + \tfrac{1}{2}\ln\left(\frac{\sqrt{2K_1/r_\mathrm{l}} + u(0)}{\sqrt{2K_1/r_\mathrm{l}} - u(0)}\right)\right)$$

and

$$v(t) = 1 - l(0) = \frac{K_1}{r_\mathrm{p}P}\left(1 - \tanh^2\left(\sqrt{\tfrac{1}{2}r_\mathrm{l}K_1}t + \tfrac{1}{2}\ln\left(\frac{\sqrt{2K_1/r_\mathrm{l}} + u(0)}{\sqrt{2K_1/r_\mathrm{l}} - u(0)}.\right)\right)\right)$$

Table 7. *Analytical solutions for the SW model (i.e. denotes initial conditions). The solution for L_i (but not necessarily l) always displays an inflection point for small $u(0)$ including the case of $u(0) = 0$. The notes (SW.1)–(SW.3) refer to the text*

host growth	no secondary infection	with secondary infection
	no inoculum dynamics	
zero	$\mathrm{d}u/\mathrm{d}t + \frac{1}{2}r_l u^2 = K_1$, with an integration constant $K_1 = r_p P(1 - l(0))$, cf. (SW.1)	$\mathrm{d}v/\mathrm{d}t = -Av\sqrt{K_2 + (D - r_s L v)^2}$, with $A = \sqrt{r_l L/r_s}$, $v = 1 - l$, $D = r_p P + r_s L$ and an integration constant $K_2 = r_l r_s L u^2(0) - (D - r_s L v(0))$, cf. (SW.2)
time-dependent	no closed analytical solution found	
	inoculum decays to 0	
zero	$(\mathrm{d}u/\mathrm{d}t) + (1/2r_l)(r_l u + r_d)^2 = K_3$, with an integration constant $K_3 = r_p P(0)(1 - l(0)) + 1/(2r_l)(r_l u(0) + r_d)^2$, cf. (SW.3)	no closed analytical solution found
time-dependent	no closed analytical solution found	

(ii) *SW.2*

No host growth, no inoculum decay, with secondary infection:

$$\frac{\mathrm{d}u}{\mathrm{d}t} = (r_p P + r_s L l)(1 - l), \quad \frac{\mathrm{d}l}{\mathrm{d}t} = r_l u(1 - l).$$

After changing the variable l to $v = 1 - l$, we get

$$\frac{\mathrm{d}u}{\mathrm{d}t} = (r_p P + r_s L - r_s L v)v \equiv (D - r_s L v)v, \quad (44)$$

$$\frac{\mathrm{d}v}{\mathrm{d}t} = -r_l u v, \quad (45)$$

where $D = r_p P + r_s L$. Multiplying equation (44) by $-r_l u$ gives

$$-r_l u \frac{\mathrm{d}u}{\mathrm{d}t} = (D - r_s L v)(-r_l u v) = (D - r_s L v)\frac{\mathrm{d}v}{\mathrm{d}t},$$

$$\frac{\mathrm{d}}{\mathrm{d}t}(\tfrac{1}{2}r_l u^2) = \frac{\mathrm{d}}{\mathrm{d}t}\left(\frac{(D - r_s L v)^2}{2r_s L}\right).$$

Thus,

$$r_l r_s L u^2 = (D - r_s L v)^2 + K_2,$$

$$u = \sqrt{\frac{K_2 + (D - r_s L v)^2}{r_l r_s L}},$$

where K_2 is again an integration constant, which can be found from initial conditions

$$K_2 = r_l r_s L u(0)^2 - (D - r_s L v(0))^2$$

and only a positive solution for u is valid. Note that $D > 0$ and $D^2 + K_2 \geqslant 0$. Then

$$\frac{\mathrm{d}v}{\mathrm{d}t} = -\sqrt{\frac{r_l}{r_s L}}\sqrt{K_2 + (D - r_s L v)^2}\, v.$$

This equation can be easily solved in order to get the formulae shown in equation (46).

$$v(t) = \frac{4F(t)(K_2 + D^2)}{F(t)^2 + 4Dr_s L F(t) - 4r_s L^2 K_2},$$

$$F(t) \equiv \exp\left(\sqrt{\frac{r_l(K_2 + D^2)}{r_s L}}\, t\right) f(v(0)),$$

$$f(v) \equiv \frac{2(K_2 + D^2) - 2Dr_s L v}{v} + \frac{2\sqrt{(K_2 + D^2)(K_2 + (D - r_s L v)^2)}}{v}. \quad (46)$$

(iii) *SW.3*

No host growth, exponential inoculum decay, no secondary infection:

$$\frac{\mathrm{d}u}{\mathrm{d}t} = r_p P v, \quad \frac{\mathrm{d}v}{\mathrm{d}t} = -r_l u v, \quad \frac{\mathrm{d}P}{\mathrm{d}t} = -r_d P.$$

Multiplying the second equation by $r_p P$ and expanding,

$$\frac{\mathrm{d}(Pv)}{\mathrm{d}t} = \frac{\mathrm{d}P}{\mathrm{d}t}v + P\frac{\mathrm{d}v}{\mathrm{d}t} = -r_d P v + P\frac{\mathrm{d}v}{\mathrm{d}t} = -\frac{r_d}{r_p}\frac{\mathrm{d}u}{\mathrm{d}t} + P\frac{\mathrm{d}v}{\mathrm{d}t},$$

from which we get

$$\frac{\mathrm{d}(r_p P v)}{\mathrm{d}t} = -(r_l u + r_d)\frac{\mathrm{d}u}{\mathrm{d}t} = -\frac{1}{2r_l}\frac{\mathrm{d}}{\mathrm{d}t}(r_l u + r_d)^2.$$

Thus,

$$r_p P v + \frac{1}{2r_l}(r_l u + r_d)^2 = K_3$$

or, alternatively,

$$\frac{du}{dt} + \frac{1}{2r_l}(r_l u + r_d)^2 = K_3.$$

The first of the above formulae can be used to find the value of the constant K_3:

$$K_3 = r_p P(0)(1 - l(0)) + \frac{1}{2r_l}(r_l u(0) + r_d)^2.$$

This is again a Riccati equation and one particular solution is known, i.e. $u(t) = \text{const.} = u_\infty$ (cf. above):

$$u_\infty = \frac{1}{r_l}[\sqrt{2r_l K_3} - r_d].$$

The equation can be solved in a similar way as for *SW.1*.

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