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Phil. Trans. R. Soc. Lond. B 1997 **352**, 353-364
doi: 10.1098/rstb.1997.0026

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Analysis and fitting of an *SIR* model with host response to infection load for a plant disease

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SUMMARY

We reformulate a model for botanical epidemics into an *SIR* form for susceptible (S), infected (I) and removed (R) plant organs, in order to examine the effects of different models for the effect of host responses to the load of infection on the production of susceptible tissue. The new formulation also allows for a decline in host susceptibility with age. The model is analysed and tested for the stem canker disease of potatoes, caused by the soil-borne fungus, *Rhizoctonia solani*. Using a combination of model fitting to field data and analysis of model behaviour, we show that a function for host response to the amount (load) of parasite infection is critical in the description of the temporal dynamics of susceptible and infected stems in epidemics of *R. solani*. Several different types of host response to infection are compared including two that allow for stimulation of the plant to produce more susceptible tissue at low levels of disease and inhibition at higher levels. We show that when the force of infection decays with time, due to increasing resistance of the host, the equilibrium density of susceptible stems depends on the parameters and initial conditions. The models differ in sensitivity to small changes in disease transmission with some showing marked qualitative changes leading to a flush of susceptible stems at low levels of disease transmission. We conclude that there is no evidence to reject an *SIR* model with a simpler linear term for the effect of infection load on the production of healthy tissue, even though biological considerations suggest greater complexity in the relationship between disease and growth. We show that reduction in initial inoculum density, and hence in the force of infection, is effective in controlling disease when the simple model applies.

1. INTRODUCTION

Many epidemics of agricultural crops exhibit monotonic increase within seasons with a seemingly inexorable rise towards an asymptote. This has led to the use of simple classes of nonlinear models to describe and analyse botanical epidemics (Gilligan 1985; Campbell & Madden 1990). Many models take the generic form

$$\frac{dI}{dt} = [\beta_1 f_1(P) + \beta_2 f_2(I)]\,(N - I), \qquad (1)$$

in which I is the density of infected individuals within a population of N individuals per unit area: β_1 and β_2 are the transmission parameters for primary infection from a reservoir of inoculum (P) and for secondary infection between infected and susceptible individuals (Brassett & Gilligan 1988). The carrying capacity for infected individuals (N) is typically treated as a constant when whole plants are used as the units of measurement since the total density of plants within fields is often unaffected by disease. Even so, considerable dynamical activity occurs within plants with the production, infection and death of leaves, roots or other organs. This has important consequences for the economic yield of crops as tissue is lost due to disease.

To model these changes it is necessary to redefine I and N relative to smaller units such as roots, stems or leaves depending on the site of activity of the parasite. The generic model is then expanded to give:

$$\frac{dI}{dt} = [\beta_1 f_1(P) + \beta_2 f_2(I)]\,(N - I), \qquad (2a)$$

$$\frac{dN}{dt} = f_3(N, I), \qquad (2b)$$

where $f_3(N, I)$ encompasses birth and death of susceptible tissue and the effect of the amount of infection on the production of new host tissue. In practice, the growth of the host population is sometimes treated independently of parasite activity, so that the function, f_3, is restricted to N and the equations are uncoupled (see the discussions in Gilligan 1990, 1994). This then yields a variable carrying capacity $N(t)$ for the disease population in equation (1) but it is biologically unrealistic because it ignores the fact that disease affects the dynamics of plant growth. The effect of disease on plant growth may be complicated, however. Root and leaf pruning experiments to simulate disease, together with experiments in controlled environments have shown that small amounts of infection on plants may stimulate growth, while larger amounts become inhibitory.

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In this paper, we reformulate the model in equation (2) into an *SIR* form (Hethcote 1989) for susceptible (S), infected (I) and removed (R) plant organs, in order to examine the effects of different models for the amount of infection on the production of susceptible tissue, hereafter referred to as the host response to infection load. We analyse and test the system for the stem canker disease of potatoes caused by the soil-borne fungus, *Rhizoctonia solani* Kühn. Specifically the objectives of the work are: (i) to examine the *SIR* formulation for stem canker, including the effects of changing host susceptibility; (ii) to introduce functions for the host response to the load of infection and analyse the effects on model behaviour; and (iii) to test by model fitting, whether or not there is evidence for stimulation or inhibition of stem production on infected potatoes. Stem canker is an economically important disease that occurs on potatoes wherever the crop is grown (Hide *et al.* 1985). Infection arises from inoculum in soil (Gilligan *et al.* 1996) or from contaminated seed tubers (James & McKenzie 1972). There is no secondary (stem-to-stem) infection (G. Hide, personal communication). The fungus colonizes emerging stems from the germinating tuber, producing dark brown lesions at the base of the stem. Infection can totally destroy stems so that stems can be classified as susceptible (healthy), infected, or removed (dead). Plants become resistant to infection after stems emerge from the soil (Van Embden 1965).

2. METHODS

(*a*) *Model*

The stem canker system can be modelled by a set of linked differential equations, for a compartmental system to describe the change in status of stems from susceptible (S) but uninfected stems to infected (I) and ultimately to removed (R) or dead stems. The general form of the equations is given by the following:

susceptible stems

$$\frac{\mathrm{d}S}{\mathrm{d}t} = b(\kappa - N) - \lambda(t)S - f(I,S), \tag{3a}$$

infected stems

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \lambda(t)S - dI, \tag{3b}$$

removed stems

$$\frac{\mathrm{d}R}{\mathrm{d}t} = dI, \tag{3c}$$

in which $N = S+I+R$ is the total number of stems, b is the *per capita* rate of production of susceptible stems, d is the *per capita* death rate of infected stems, and κ is the carrying capacity or maximum number of stems per plant. The force of infection $\lambda(t)$ is given by $\lambda = \beta P s(t)$, in which β is the rate of infection of susceptible stems by soil-borne (or plant-borne) inoculum, and P is the (fixed) density of inoculum and $s(t)$ is the host susceptibility. The function $s(t)$ is given by $s(t) = s_0 \exp(-\mu t)$ to allow for the loss of infectivity of inoculum and increased resistance of the host with age. Thus $\lambda(t) = \lambda_0 \exp(-\mu t)$, where $\lambda_0 = \beta P s_0$. The principal variables and parameters are summarized in table 1.

The model is based on the following assumptions:

(i) production of susceptible stems is monomolecular, i.e. in the absence of disease the population of stems rises to an asymptote (κ);

(ii) infection occurs from a reservoir of inoculum and there is no secondary infection from infected to susceptible stems ($\beta_2 = 0$ in equations (1) and (2));

(iii) there is no significant death of susceptible stems during the course of the epidemic;

(iv) the susceptibility of the host to inoculum declines exponentially with time;

(v) infected stems follow an exponential 'lifetime'

Table 1. *Summary of principal variables and parameters used in the models*

variable	description	dimensions
S	susceptible stems	[stems]
I	infected stems	[stems]
R	removed (dead) stems	[stems]
N	total number of stems ($N = S+I+R$)	[stems]
parameter	**description**	**dimensions***
b	production rate of susceptible stems	$[\text{time}]^{-1}$
κ	carrying capacity (maximum number of stems per plant)	[stems]
λ_0	force of infection	$[\text{time}]^{-1}$
β	rate of infection of susceptible stems	$[\text{propagule}]^{-1}[\text{time}]^{-1}$
P	density of inoculum (fixed)	[propagule]
μ	decay rate of force of infection	$[\text{time}]^{-1}$
α	rate of inhibition of production of susceptible stems due to infection	$[\text{time}]^{-1}$
α_1	(asymptotic) maximum level of inhibition of stem production	$[\text{stems}][\text{time}]^{-1}$
α_2	controls switch between stimulation and inhibition of stem production (switch occurs at $I = \alpha_2/\alpha_1$)	$[\text{stems}]^2[\text{time}]^{-1}$
α_3	controls maximum level of stimulation of stem production	[stems]
γ	parameter introduced to avoid problem of singularities at low numbers of stems	[stems]
d	death rate of infected stems	$[\text{time}]^{-1}$

* Dimensions for host response parameters are given for Model I (α), Model III ($\alpha_1, \alpha_2, \alpha_3$) and Model V ($\gamma$).

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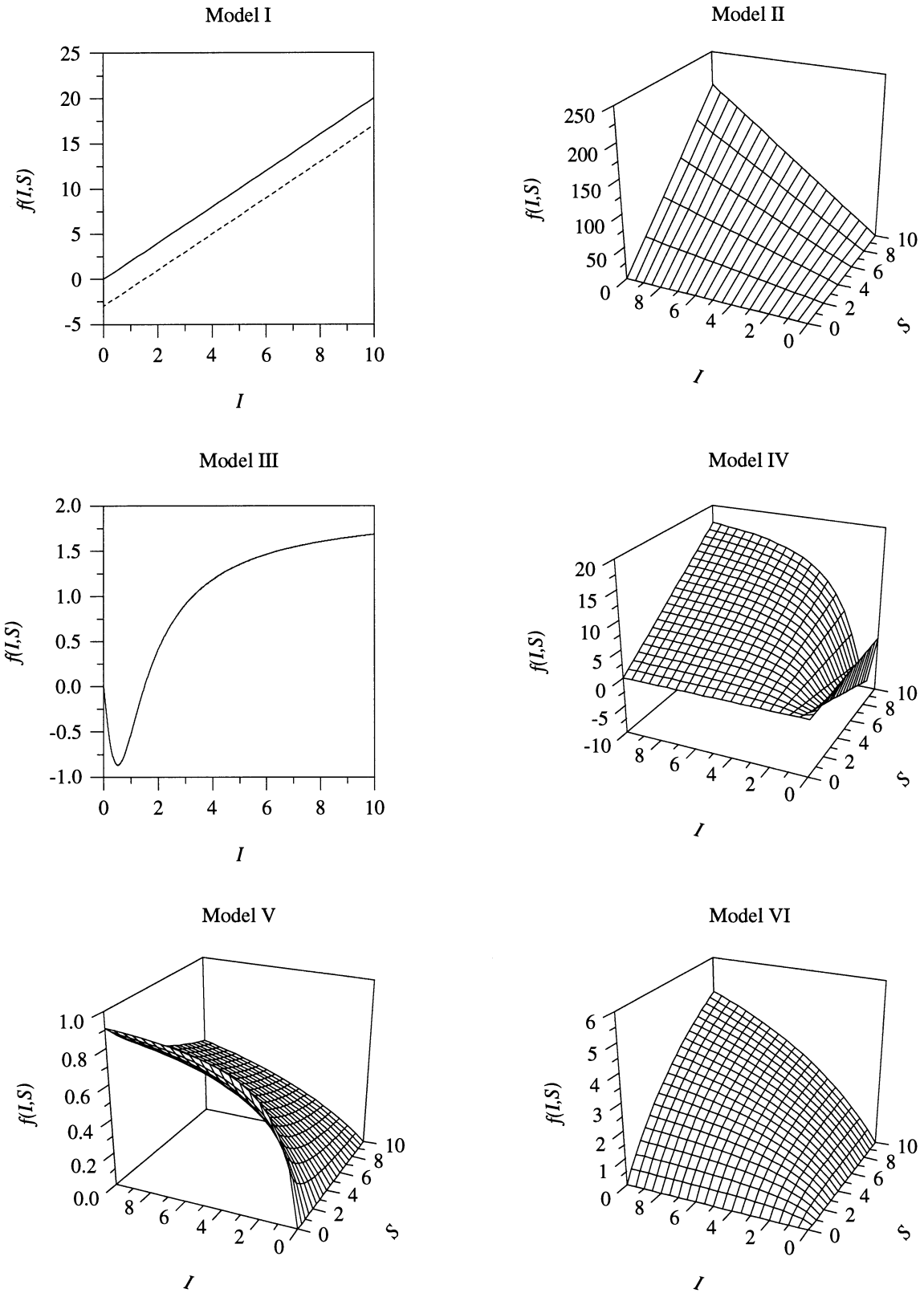

Figure 1. Graphical behaviour of alternative models (see text) for host responses ($f(I,S)$) of the production of susceptible stems to infection load, measured by the number of infected stems per plant (I). Models I, III and V describe absolute effects of I on the rate of production of susceptible stems: Models II, IV and VI show the *per capita* effect of I at different levels of S. The dotted line describes a linear host response, of the form $f(I,S) = \alpha_1 I - \alpha_2$, with stimulation at low and inhibition at higher densities not analysed separately because it collapses into Model I (see Discussion).

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distribution, so that they survive for an average period of d^{-1} and then die;

(vi) the presence of infected stems may affect the production of susceptible stems [$f(I, S)$].

The latter defines the host's response to infection load (see Gilligan 1994).

Seven models (0–VI) are considered, each with different host responses to the load of infected stems on a plant, including a null response:

Model 0

$$f(I, S) = 0,$$

Model I

$$f(I, S) = \alpha I,$$

Model II

$$f(I, S) = \alpha IS,$$

Model III

$$f(I, S) = \frac{\alpha_1 I^2 - \alpha_2 I}{\alpha_3 + I^2},$$

Model IV

$$f(I, S) = \frac{\alpha_1 I^2 - \alpha_2 I}{\alpha_3 + I^2} S,$$

Model V

$$f(I, S) = \frac{\alpha I}{\gamma + I + S},$$

Model VI

$$f(I, S) = \frac{\alpha I}{\gamma + I + S} S.$$

Model 0 describes a characteristic *SIR* form, in which there is no effect of the parasite on the production of susceptible stems. Models I and II describe a linear host response to the amount of infected stems (figures 1*a*, *b*). Both Models III and IV describe stimulation of the plant to produce proportionately more stems [$f(I, S) < 0$] at low disease loads. As the disease load passes a threshold, $f(I, S)$ becomes positive and production of new stems is inhibited (figures 1*c*, *d*). Models V and VI are characterized by proportional responses, with the effect of I on the production of stems being scaled relative to the total stem density per plant (figures 1*e*, *f*). Note that Models II, IV and VI differ from I, III and V, respectively, in having an interaction term for S with $f(I, S)$. The pairs of models do not otherwise differ so that the function, such as αI, represents an absolute reduction in stem production in Model I and a *per capita* reduction in Model II.

(*b*) *Experimental*

Subsamples of potatoes, *cv* Estima, were removed from field plots on six occasions (2, 4, 6, 8, 10 and 14 weeks) after planting and the numbers of healthy (susceptible) and infected stems showing symptoms of stem canker were recorded. The numbers of stems at planting were taken as zero. Sampling times were converted to thermal times as day degrees above 0 °C for consistency with other published work (Simons & Gilligan 1997*a*, *b*) and to allow for different planting dates. There were four factors, density of tuber-borne inoculum (two levels), date of planting (three levels), size of seed tubers (two levels), and pre-emergence irrigation (two levels) arranged in a factorial design consisting of 48 plots in two blocks. The experiment was repeated in two successive years, 1987 and 1988. The models in this paper were fitted to data for all the main treatments. Results are given for the inoculum density treatment in which seed tubers were exposed to artificial inoculum comprising either a high (> 500 colony forming units g^{-1} soil) or low (50 colony forming units g^{-1} soil) inoculum density of *R. solani* prior to planting. Further details of the experimental design are given in Simons & Gilligan (1997*a*).

3. RESULTS

(*a*) *Equilibria and stability*

Examination of a range of empirical data sets for the dynamics of stem canker on potatoes (Simons & Gilligan 1997*a*; cf. also figure 2) shows that the densities of susceptible and infected stems rise to a maximum and then decline to asymptotic values. These equilibria occur because some infected stems die and further disease is inhibited by resistance associated with plant age. Biologically realistic models therefore must have unimodal trajectories for S and I, with stable, non-negative equilibria.

When the force of infection is constant (i.e. $\mu = 0$, so $\lambda = \lambda_0$, and there is no change in susceptibility of the host) and we assume that there is no host response in the absence of infection (i.e. $f(0, S) = 0$), the generic model (equation (3)) has an equilibrium density of susceptible, infected and removed stems, $S_e = 0$, $I_e = 0$, $R_e = \kappa$, which is globally stable.

When the force of infection varies over time (i.e. $\mu \neq 0$), the model can be conveniently turned into an autonomous system by including an equation for λ, so:

susceptible stems

$$\frac{dS}{dt} = b(\kappa - N) - \lambda S - f(I, S), \tag{4a}$$

infected stems

$$\frac{dI}{dt} = \lambda S - dI, \tag{4b}$$

removed stems

$$\frac{dR}{dt} = dI, \tag{4c}$$

force of infection

$$\frac{d\lambda}{dt} = -\mu\lambda, \tag{4d}$$

This system does not have a unique equilibrium, but the solution tends to $(S_e, 0, R_e, 0)$, with $S_e + R_e = \kappa$. Precisely where the equilibrium solution lies depends on the parameters and initial conditions. Interruption by the decaying force of infection ($\lambda(t)$) renders a

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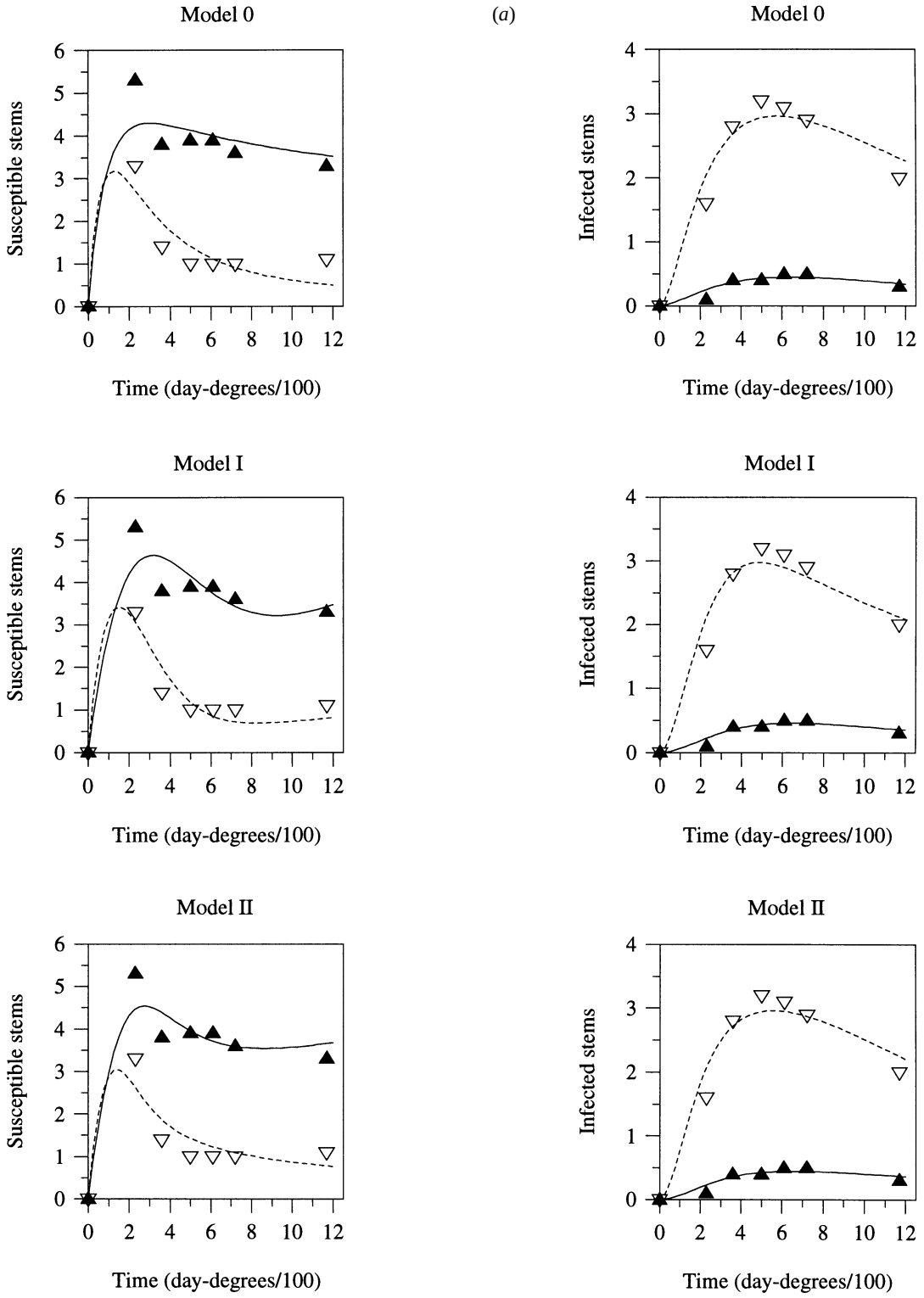

Figure 2(*a*). For legend see p. 359

proportion or previously susceptible stems, resistant to infection. This essentially freezes the epidemic (cf. Kleczkowski *et al*. 1996) as infected stems pass into the removed class with no more new infection occurring.

(*b*) *Fitting to data*

Models 0–VI were fitted by least squares, using FACSIMILE (Anon. 1995), to a range of disease progress curves from Simons & Gilligan (1997*a*) under the assumption of normal errors due to measurement error. Results are presented here for the fits to two treatments having high and low initial inoculum densities (figure 2; table 2) to illustrate model behaviour over a range of densities for I (0–0.5 for the low density treatment and 0–3.3 for high density treatment). Convergence of the models was checked for a range of starting values for the parameters. All parameters in Models 0 and I were concurrently estimated. Sequential fitting was required for Models

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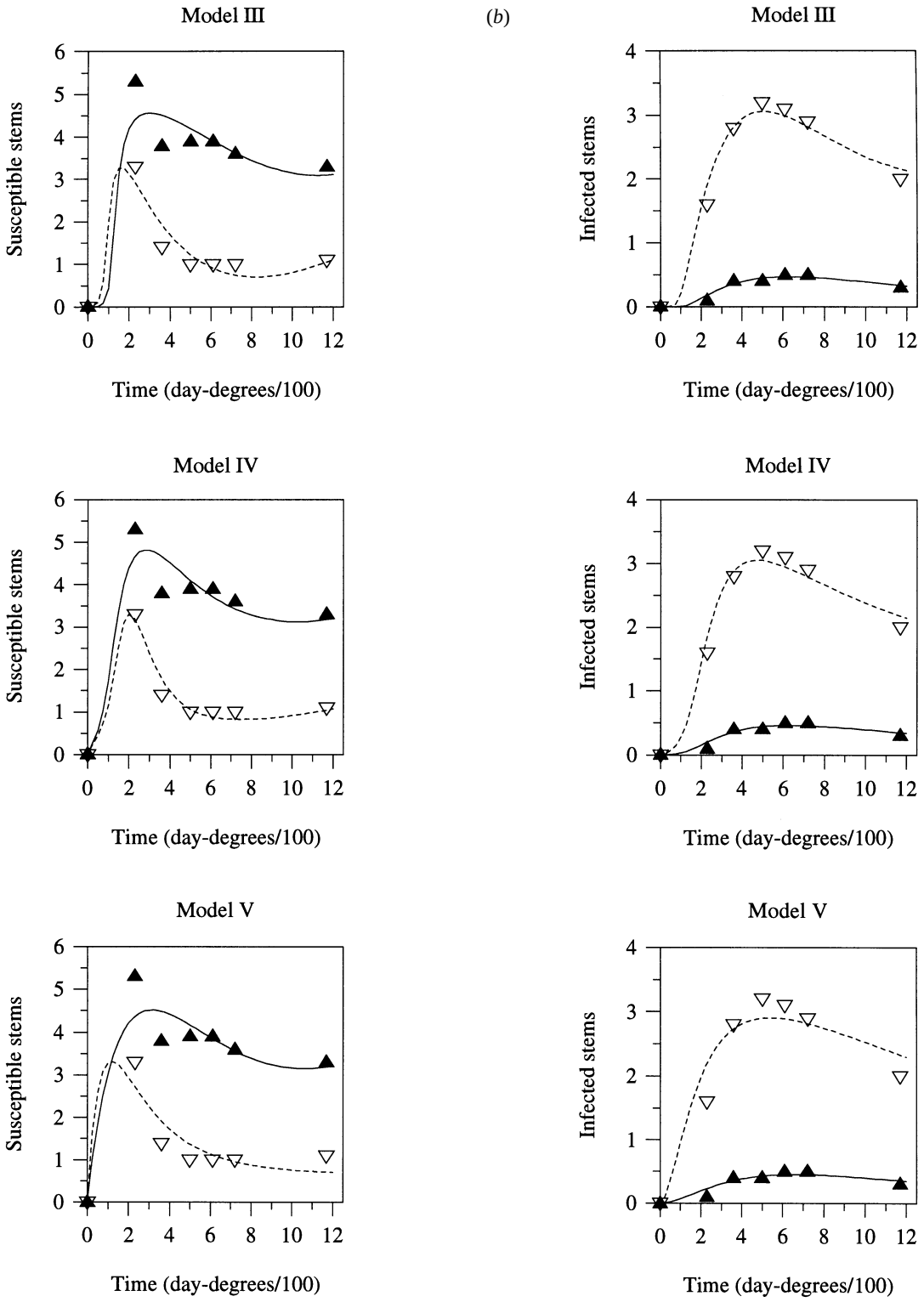

Figure 2(*b*). For legend see opposite.

II–VI, in which certain parameters were fixed after initial exploration of the sensitivity of the residual deviance to small changes in parameter values. Most typically, the parameters fixed were the rate parameter, μ, together with either b (production rate of susceptible stems) or κ (the carrying capacity).

The models fitted rather similarly the data for different initial levels of infection (figure 2; table 2) as well as other disease progress curves from the same experiment not shown here. Fitted curves reflected the rise and fall of the empirical disease progress curves, especially at the higher inoculum density. There was little to distinguish amongst the models on examination of the time course plots for fits and residuals. This reflects the paucity of data in the dynamically critical ascending portions of the curves.

Estimates of the carrying capacity for stems (κ) ranged from 4.9 to 11.9, compared with the commonly found range of 6–8 stems in Estima. Underestimation of stem density was associated with the model (0) with

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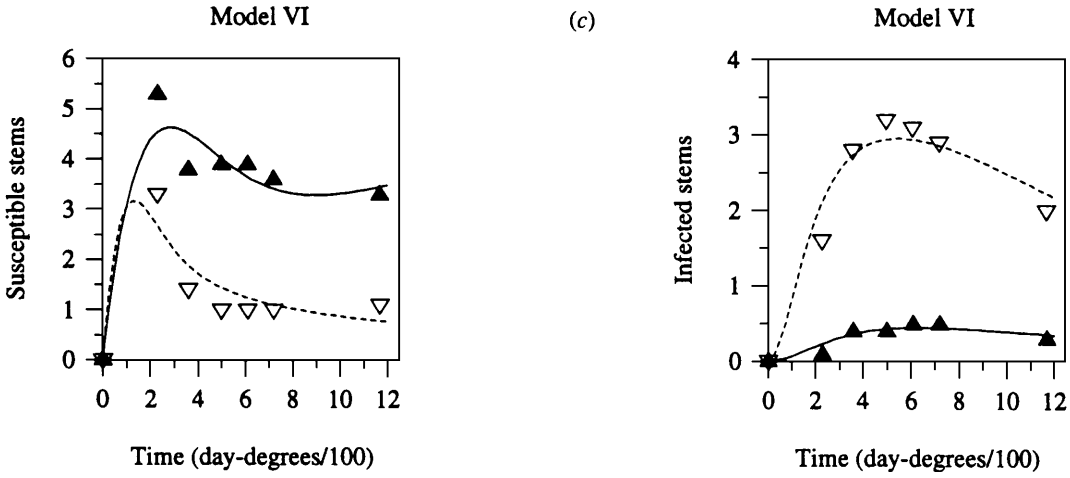

Figure 2. Goodness of fit of *SIR* models with different host responses for infection ($f(I,S)$) to changes in numbers of susceptible and infected stems per plant when potatoes were exposed to relatively high (– –) or low (——) densities of inoculum of *R. solani* in soil. The models differed in the host response terms: Model I, $f(I,S) = \alpha I$; Model II, $f(I,S) = \alpha IS$; Model III, $f(I,S) = (\alpha_1 I^2 - \alpha_2 I)/(\alpha_3 + I^2)$; Model IV, $f(I,S) = (\alpha_1 I^2 - \alpha_2 I)S/(\alpha_3 + I^2)$; Model V, $f(I,S) = \alpha I/(\gamma + I + S)$; Model VI, $f(I,S)(\alpha I/(\gamma + I + S))S$.

Table 2. *Parameter estimates obtained by fitting Models 0–VI to data for disease progress of* R. solani *in field plots infested with low and high inoculum densities*

model	data set	parameter									RSS[a]	df[b]
		b	κ	λ_0	μ	α, α_1	α_2	α_3	γ	d		
0	Low	1.177	4.876	0.051	0.096	—	—	—	—	0.246	19.19	9
	High	1.479	5.132	0.434	0.130	—	—	—	—	0.084	14.80	9
I	Low	0.391	9.145	0.048	0.128[e]	4.570	—	—	—	0.167	16.02[e]	9[e]
	High	0.731	7.963	0.438	0.119	0.743	—	—	—	0.101	9.80	8
II	Low	0.455[e]	8.475	0.047	0.105[e]	1.144	—	—	—	0.193	16.61[e]	10[e]
	High	0.697	7.685	0.466	0.073[e]	0.277	—	—	—	0.138	13.04[e]	9[e]
III	Low	0.0005[e]	11.725[e]	0.064	0.140[e]	0.721	0.261	0.0003	—	0.215	10.73[e]	9[e]
	High	0.005[e]	10.650[e]	0.554	0.087[e]	0.750	2.419	0.026	—	0.139	6.58[e]	9[e]
IV	Low	0.175[e]	6.160	0.528	0.121[e]	0.349	0.097	0.0008[e]	—	0.194	12.35[e]	9[e]
	High	0.082	11.909	0.617	0.097	1.122	2.221	0.232[e]	—	0.145	3.59[e]	7[e]
V	Low	0.694	6.467	0.047	0.116[e]	14.294	—	—	0.017[e]	0.178	16.14[e]	9[e]
	High	1.467	5.743	0.499	0.247	2.106	—	—	0.026[e]	0.058	14.10[e]	9[e]
VI	Low	0.490	8.372	0.047	0.111[e]	5.473	—	—	0.098[e]	0.184	16.12[e]	9[e]
	High	0.710	8.281	0.451	0.067[e]	1.979	—	—	0.090[e]	0.141	13.02[e]	9[e]

[a] Residual sum of squares; [b] degrees of freedom; [e] sequential fitting required, marked parameters were fixed after initial exploration of parameter space.

no host response (table 2). There was a ten-fold difference between treatments for all models in estimates of the force of infection (λ_0) (table 2). This reflects the difference in inoculum densities between the treatments. None of the other parameters was expected *a priori* to differ between the inoculum density, but there was some evidence for correlation in parameter estimates for α's with inoculum density (table 2).

Approximate *F*-tests (Aitkin *et al.* 1989; Ross 1990) for the change in deviance due to addition of parameters for host response yielded evidence ($p \leqslant 0.1$) of improved fits for Model I over Model 0 in four out of five disease progress curves for which convergence was achieved without sequential fitting. These included the high density treatment in 1988 (table 2) and planting time in 1987 and 1988. Model I also yielded more plausible estimates than Model 0 for the carrying capacity of stems (table 2). There was no evidence for significant improvement in the fit due to any of the other models nor of significant differences in the goodness-of-fit of the *per capita* over the absolute forms.

Analysis of the host responses (figure 3) for the models using estimated parameters (table 2) for the treatment involving high inoculum density shows marked differences between the linear (I) and non-linear (II–VI) host responses and between absolute (I, III and V) and *per capita* (Models II, IV and VI) forms. The *per capita* responses tend to lag behind the absolute responses (i.e. phase curves are deflected to the right), for Models I–IV, because of the early limiting effects of the susceptibles. For Models V and VI, in which S occurs in the denominator, the reverse is true. The *per capita* formulation of the host response leads to a change in shape of the phase portraits for the linear (II) and proportional (VI) models (figure 3). It

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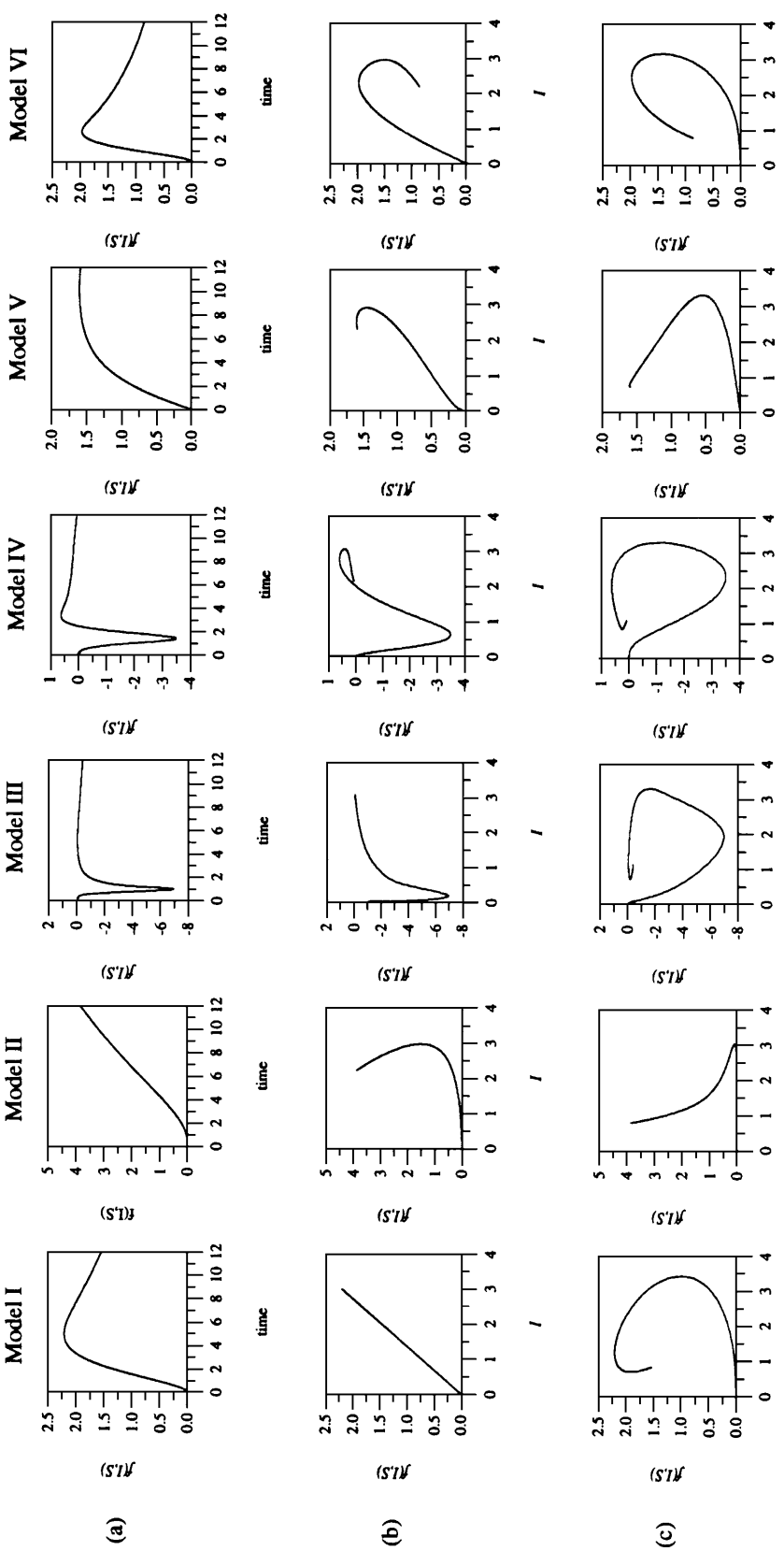

Figure 3. Host responses for Models I–VI: (*a*) change in $f(I,S)$ with time; (*b*) phase portrait: parametric plot of $f(I,S)$ and infected stems (I); (*c*) phase portrait: parametric plot of $f(I,S)$ and susceptible stems (S).

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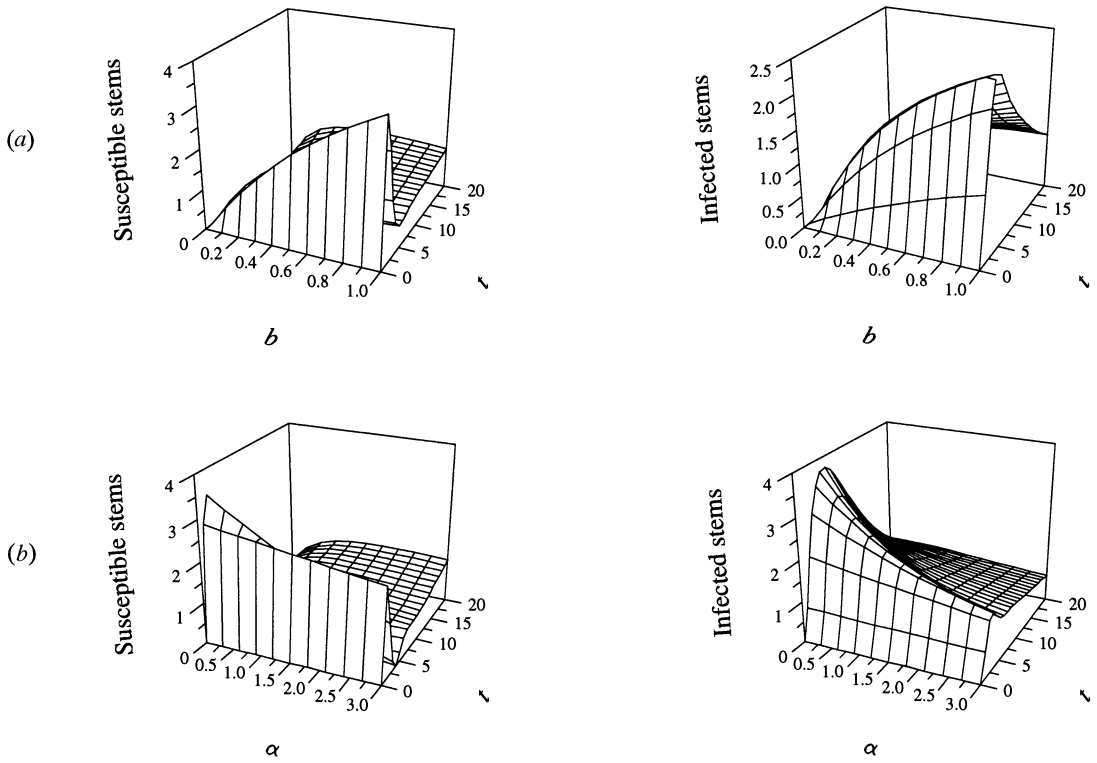

Figure 4(*a*) and (*b*). For legend see page 362.

has no effect, however, on the stimulatory-inhibitory model (IV) in which the contribution of S is dominated by the terms involving I.

The linear response, Model I, has predictably a steadily increasing effect during the early stages of the epidemic (figure 3*a*), before dropping slowly towards zero. Strikingly, the stimulatory-inhibitory host response (III) was dominated by a pulse during the initial phase of the epidemic. It remained negative throughout the epidemic indicating stimulation rather than inhibition of stem production (figure 3*a*). A similar response was obtained for the analogous *per capita* model (IV) except that stimulation occurred later with a less marked pulse than for III, followed by inhibition.

(*c*) *Sensitivity analysis and disease control*

Sensitivity of the models to changing parameter values is examined for the simplest inhibitory model (I) and for the stimulatory-inhibitory model (III) in figures 4 and 5. Increasing the birth parameter (b) and decreasing the magnitude of the infection load (α) for Model I each led (initially for α) to the production of more stems. The advantage to the host, however, is severely offset by an increase in the density of infected stems (figures 4*a* and *b*). The parameters, λ_0, μ and d show inversely correlated changes in infected and susceptible stems (figure 4*c–e*). The most effective control of disease occurs by reduction in the amount of inoculum and hence in the force of infection (λ_0) but the level of reduction in inoculum or transmission needs to be substantial, of the order of 10%.

The effects of changing the parameters λ_0, μ and d for Model III are qualitatively similar to those for Model I except that small changes in λ_0 above zero result in a sharp rise in the density of susceptible stems (figure 5*a*). This occurs because of the marked stimulatory effect of small densities of infected stems. Increasing the decay rate (μ) leads to a rapid cessation of infection, resulting in a low level of infection (figure 5*b*). This low level of infection stimulates a flush of susceptible stems which, ultimately, settle on realistic equilibrium values. Changing the remaining parameters (b, α_1, α_2 and α_3) each produced correlated changes in S and I (not shown).

4. DISCUSSION

We have used a combination of mathematical analysis and goodness-of-fit to experimental data to test model behaviour for an economically important plant pathogen. The models are based on an *SIR* form because this enables identification of the effect of disease on the production of new susceptible tissue. Most models in botanical epidemiology have considered the effects of disease on the total amount of tissue ($N = S+I+R$), which includes both infected and susceptible tissue (Waggoner 1986). Since all new stems, and indeed all new organs on a plant, are usually uninfected, we argue that the *SIR* formulation is more appropriate than the more common *NI* formulation.

The decline in susceptibility of stems as the host becomes more resistant to infection by *R. solani* is represented by a decay term for the force of infection. In the absence of a decay term all the stems become infected and die. The decay term essentially 'freezes' the dynamics of the transient behaviour of the disease progress curves so that the equilibrium density of

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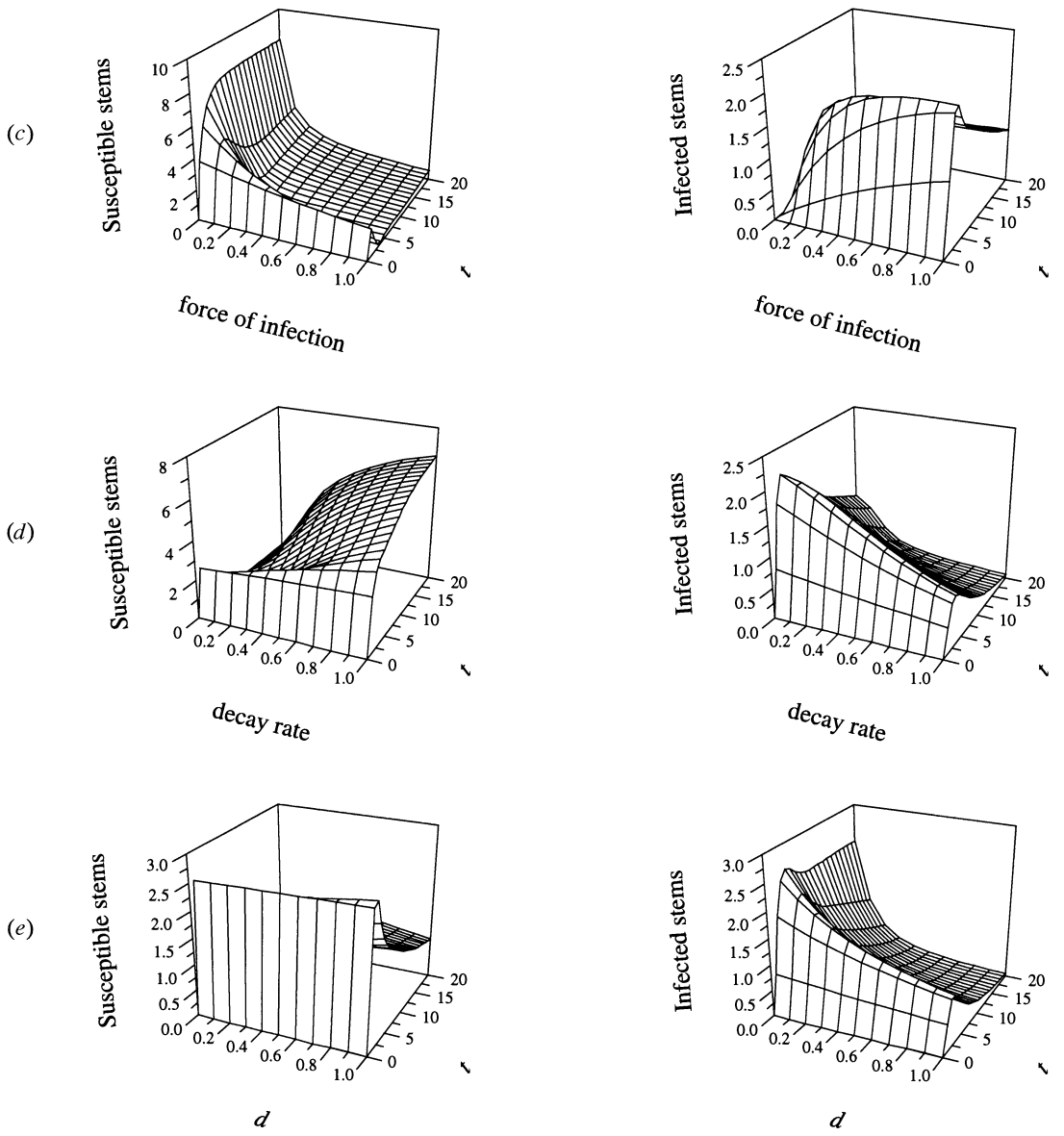

Figure 4. Sensitivity of predictions for the numbers of susceptible and infected stems per plant over time to changes in parameters for *SIR* model with Model I, host response: (*a*) *per capita* production of susceptible stems (*b*); (*b*) infection load (α); (*c*) force of infection (λ_0); (*d*) decay rate of force of infection (μ); (*e*) death rate of infected stems (*d*). Default parameters: $b = 0.6$, $\lambda_0 = 0.5$, $\alpha = 1.5$, $\kappa = 8$, $\mu = 0.1$, $d = 0.15$.

susceptible stems depends on the initial conditions and parameters (Kleczkowski *et al.* 1996). Not all stems become infected: infected stems pass into the removed class as they die and $S_e + R_e = \kappa$.

Three broad classes of host response for the effect of the amount of infection on the production of susceptible stems were each compared with the absence of a host response (Model 0). These included linear inhibition, stimulation followed by inhibition and a proportional response. Each type of response was considered as an absolute effect on dS/dt (Models I, III and V) or as a *per capita* effect on dS/dt (Models II, IV and VI). Thus Models II, IV and VI differ from I, III and IV in having an interaction term for S with $f(I, S)$. An absolute host response implies that the inhibitory effect, say, of infected stems on a potato is mediated via the total reserves of nutrients in the plant and is not directly influenced by the current density of susceptible stems. A *per capita* response, however, implies that the drain on reserves caused by infected stems is directly proportional to the current density of susceptible stems. Alternatively, if there is stimulation, the stimulation acts to release more nutrients for the production of new stems either independently (absolute response) or in proportion (*per capita* response) to the current density of susceptible stems per tuber. We did not find any convincing evidence to favour the *per capita* over the absolute response. While we do not reject the *per capita* models as plausible descriptions of stem canker, we confine further discussion to the absolute forms of the host responses.

The linear model (Model I) for host response implies that the reduction in the production of new stems (dS/dt) changes in direct proportion to the amount of infected stems on a plant. Nonlinear loads were introduced to allow for a period of stimulation by low levels of disease, followed by inhibition (Model III). In practice, parameter estimation from the field data

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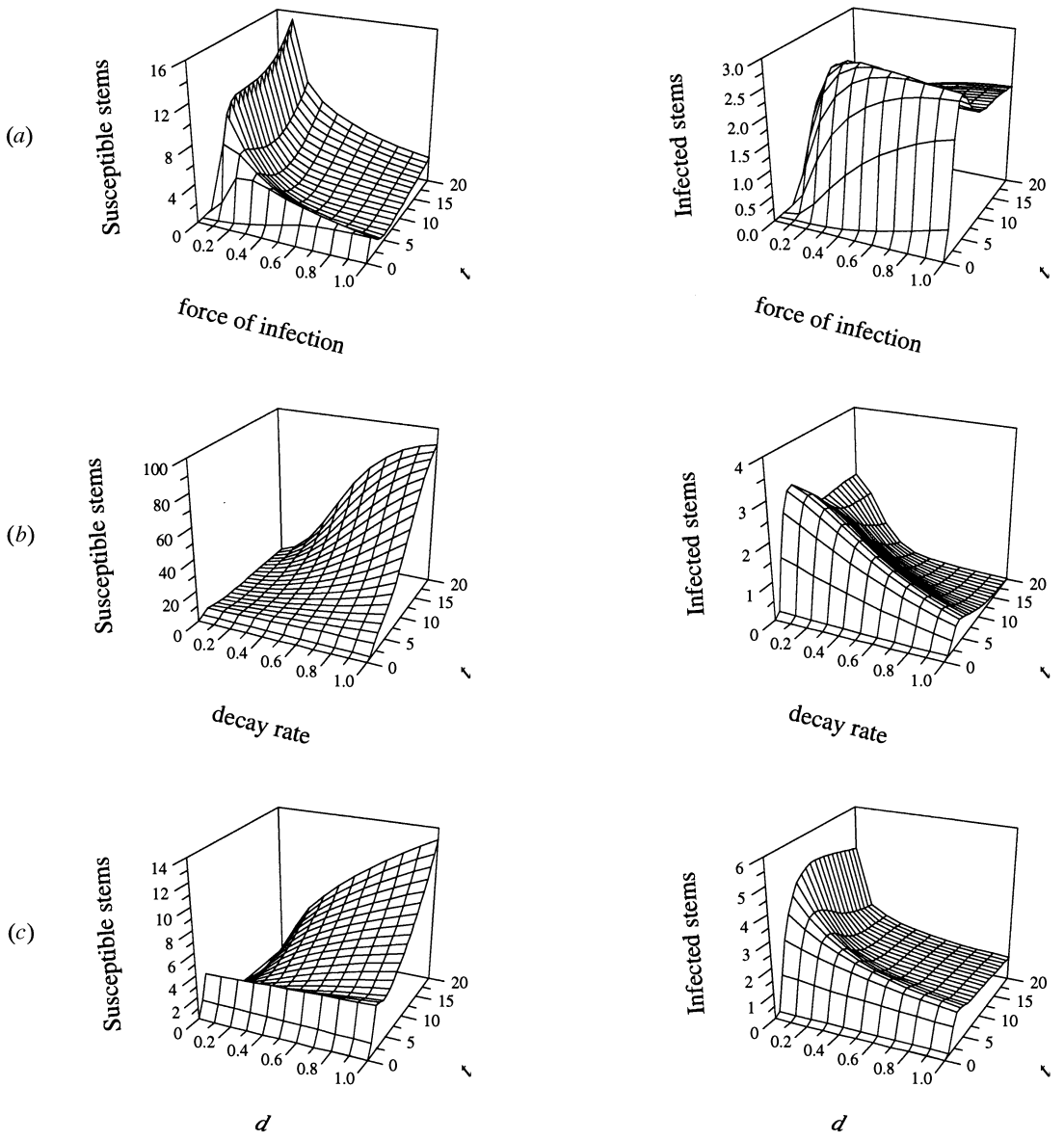

Figure 5. Sensitivity of predictions for the numbers of susceptible and infected stems per plant over time to changes in parameters for *SIR* model with Model III, host response: (*a*) force of infection (λ_0); (*b*) decay rate of force of infection (μ); (*c*) death rate of infected stems (d). Default parameters: $b = 0.005$, $\lambda_0 = 0.5$, $\mu = 0.1$, $\alpha_1 = 0.7$, $\alpha_2 = 2.5$, $\alpha_3 = 0.03$, $\kappa = 10$, $d = 0.2$.

yielded negative (stimulatory) host responses for most of the period of susceptibility of the host (figure 4). Few experiments have been done to analyse the effects of stem canker on the production of healthy stems but stimulation at low levels of infection followed by inhibition is consistent with data of Hide & Read (1990: see their figure 3), while Cother & Cullis (1985) noted a threshold of infection above which inhibition occurs. It is possible that an inhibitory effect of infected stems may diminish as the density of susceptible stems increases (Model V, figure 1) and further experimental work is necessary to distinguish between the models. We conclude, simply on the basis of obtaining biologically consistent parameter estimates and goodness of fits, that there is no evidence to reject the most parsimonious model (Model I) as a plausible description of this *Rhizoctonia*–potato system. Biological intuition, however, suggests that the feedback mechanism is likely to be more complicated as, for example, in Model III. Moreover, the models differ in dynamics, especially in the effects of changing the force of infection (cf. figures 4 and 5). We note, however, that changes in the force of infection (via λ_0 or μ) in Model III may lead to implausibly large flushes of susceptible stems. The simple linear model (I) can be adapted to give $f(I, S) = \alpha_1 I - \alpha_2$, in which there is stimulation below $I = \alpha_2/\alpha_1$ and inhibition above. Although it is arguably plausible, the parameterization is mathematically redundant because the additional constant (α_2) is subsumed into a single constant term along with $b\kappa$ in equation (3*a*), and the model is indistinguishable from Model I. Moreover, the model also implies some stimulation of growth in the absence of infection and it was, therefore, not considered further in the present study.

One criterion for assessing model fits was the

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consistency of parameter estimates across treatments. We have shown consistent changes in λ_0 with inoculum density. None of the other parameters was expected to differ with inoculum density but there was some evidence for correlation in parameter estimates for αs with inoculum density (table 2). Reparameterization of the host responses in Models I and III, scaling I relative to inoculum density (P), with I/P or IP as appropriate, gave similar estimates for the αs across treatments. This implies that the load of inoculum, as well as load of infection, influences host growth. This may be associated with inoculum giving rise to direct infection of tubers (known as black scurf) in addition to the stem canker phase of the pathogen.

We have used a combination of model fitting and analysis of model behaviour to distinguish amongst a number of alternative formulations for the dynamics of host response to infection load. Simple methods of least-squares estimation were used to fit the models. This is based on an assumption that the main source of uncertainty in the measurements derives from the sampling and measurement procedure itself rather than from the stochastic nature of the underlying processes. This is a reasonable starting point in confronting models with data. We have considered alternative error distributions and weightings but the problems of fitting are not trivial, convergence is difficult to achieve, and there are conflicts in correcting for possible correlated errors and for non-normality.

The elaboration we propose to the compartmental *SIR* models involves the introduction of an interaction term of the form $f(I, S)$ or $f(I, S)S$ between S and I that is *independent of disease transmission*. Hence the host response for the effect of infection load on the production of healthy tissue occupies a hybrid position between compartmental models, in which the interaction term relates to the transmission of disease (cf. Hethcote 1989), and predator–prey models, in which the host response defines the loss of predator to prey (cf. May 1981). The concept of parasite load has been discussed in models for macroparasitic infection of animals, where it is defined as the parasite-induced host death rate or equivalently depression of the birth rate (May & Anderson 1979). We suggest that host responses are important in linking the effects of infection and disease by plant pathogens on their hosts with population dynamics of the host. More work is needed, however, to separate the effects of total dynamics (intrinsic birth and death rates for plant organs, with and without density dependence) from parasite-induced changes. This can be approached by the use of controlled inoculation or amputation of plant organs (stems, leaves, roots) to simulate infection, but it may be difficult to uncouple changes due to disease at different growth stages from inherent ageing of the host.

Financial support from the BBSRC (C.A.G., S.G.) and Potato Marketing Board (S.A.S.) is gratefully acknowledged. We thank Adam Kleczkowski, Jonathan Swinton, James Truscott and other members of the Botanical Epidemiology Laboratory for helpful discussion of the work. We also thank one anonymous referee for helpful comments on the model structure.

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Received 26 July 1996; accepted 26 September 1996

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