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Phil. Trans. R. Soc. Lond. B 1997 **352**, 1935-1949
doi: 10.1098/rstb.1997.0180

Biological control in a disturbed environment

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CONTENTS

SUMMARY

Most ecological and epidemiological models describe systems with continuous uninterrupted interactions between populations. Many systems, though, have ecological disturbances, such as those associated with planting and harvesting of a seasonal crop. In this paper, we introduce host–parasite–hyperparasite systems as models of biological control in a disturbed environment, where the host–parasite interactions are discontinuous. One model is a parasite–hyperparasite system designed to capture the essence of biological control and the other is a host–parasite–hyperparasite system that incorporates many more features of the population dynamics. Two types of discontinuity are included in the models. One corresponds to a pulse of new parasites at harvest and the other reflects the discontinuous presence of the host due to planting and harvesting. Such discontinuities are characteristic of many ecosystems involving parasitism or other interactions with an annual host. The models are tested against data from an experiment investigating the persistent biological control of the fungal plant parasite of lettuce *Sclerotinia minor* by the fungal hyperparasite *Sporidesmium sclerotivorum*, over successive crops. Using a combination of mathematical analysis, model fitting and parameter estimation, the factors that contribute the observed persistence of the parasite are examined. Analytical results show that repeated planting and harvesting of the host allows the parasite to persist by maintaining a quantity of host tissue in the system on which the parasite can reproduce. When the host dynamics are not included explicitly in the model, we demonstrate that homogeneous mixing fails to predict the persistence of the parasite population, while incorporating spatial heterogeneity by allowing for heterogeneous mixing prevents fade-out. Including the host dynamics lessens the effect of heterogeneous mixing on persistence, though the predicted values for the parasite population are closer to the observed values. An alternative hypothesis for persistence involving a stepped change in rates of infection is also tested and model fitting is used to show that changes in some environmental conditions may contribute to parasite persistence. The importance of disturbances and periodic forcing in models for interacting populations is discussed.

1. INTRODUCTION

Most successful programmes of biological control involve systems that allow continuous interactions between host, parasite (the pest) and hyperparasite (the biological control agent) (Hassell 1981; Waage & Greathead 1988). These systems do not have many ecological upheavals, such as those associated with the cultivation of an annual crop. However, the use of biological control against crop diseases has been less successful (Deacon 1988; Adams 1990; Cook 1993). Many examples fail to perform as well in the field as they do in the laboratory. One problem is the failure of empirical experimentation to take into account spatial and temporal heterogeneity in the field.

Temporal heterogeneity arises from the ecological

disturbances associated with the planting and harvesting of a seasonal crop. For such crops, the interactions between the host and parasite are discontinuous with host tissue only available for infection during the growing season. Thus, the parasite population is confronted with pulsed behaviour upon germination and harvest of the crop, a neglected, but ecologically important, problem. In most ecological and epidemiological models, births are assumed to be continuous in time (Murray 1989) or alternatively, the birth rate is allowed to vary seasonally (Anderson & May 1981). Equivalently, a seasonally varying infection term is common in epidemiological models (London & Yorke 1973; Aron & Schwartz 1984; Schwartz 1985) where it has been introduced as a mechanism to produce long-term oscillations. However, the effect of disturbances has received little attention to date (Barlow 1993; Hanski *et al.* 1993; Shaw 1994; Briggs & Godfray 1996; Gubbins & Gilligan 1997*a*, *b*).

Few field experiments in biological control provide time series that include disturbances and data for the population dynamics between growing seasons. One exceptional data set involves a two-year experiment (including five growing seasons) to investigate the use of the mycoparasite *Sporidesmium sclerotivorum* Uecker, Ayers & Adams as a persistent biological control agent of *Sclerotinia minor* Jagger, an economically important fungal parasite of lettuce (Adams & Fravel 1990). The *S. minor*–*S. sclerotivorum* system has been analysed using simple models in two earlier studies. The first analysed the population dynamics of *S. minor* and *S. sclerotivorum* in a closed system in the absence of a host of *S. minor* (Gubbins & Gilligan 1996). The other used a simple parasite–hyperparasite model with disturbances to investigate the interaction between spatial heterogeneity and disturbances and their role in the persistence of *S. minor* (Gubbins & Gilligan 1997*a*).

In this paper, we extend the results of these earlier studies using a combination of mathematical analysis, model fitting and parameter estimation. In particular, we examine the various mechanisms that contribute to the observed persistence of the parasite and specifically we examine the roles played by discontinuities due to planting and harvesting of the lettuce crop, spatial heterogeneity and changes in environmental conditions. Two models are used to test these hypotheses. The first model is a two-species parasite–hyperparasite system that captures the essential features of biological control. The second model is developed from the first by incorporating the host dynamics explicitly, thus producing a three-species host–parasite–hyperparasite system. The models are developed with close reference to the *S. minor*–*S. sclerotivorum* system but their generic structure makes them applicable to a broad spectrum of host–parasite interactions.

2. THE BIOLOGICAL SYSTEM

The models are used to analyse the biological control of an economically important fungal plant parasite *S. minor* by the fungal hyperparasite *S. sclerotivorum*. *Sclerotinia minor* is an ecologically obligate plant pathogen that infects lettuce plants from soil-borne sclerotia, causing the characteristic wilting symptoms of lettuce drop. Sclerotia of *S. minor* are produced on and within the leaves, stem and upper portion of the roots of lettuce plants (Adams 1986) and on previously infected lettuce tissue returned to the soil after harvest (Imolehin & Grogan 1980; Dillard & Grogan 1985*a*). *Sporidesmium sclerotivorum* is a naturally occurring hyperparasite of sclerotia and has been widely used as a potential biological control agent (Adams & Ayers 1982; Adams & Fravel 1990).

Adams & Fravel (1990) carried out a two-year experiment investigating the use of *S. sclerotivorum* as a persistent biological control agent of *S. minor*. In May 1987 field plots measuring 3 m by 3 m were treated with a single preparation of inoculum of *S. sclerotivorum* at rates of 0.2, 2 and 20 kg ha^{-1} (corresponding to levels of 0.08, 0.8 and 8 spores g^{-1} soil). Five successive lettuce crops (comprising three autumn and two spring crops) were grown in the field plots between September 1987 and November 1989. There were five replicates of each treatment. Soil samples were taken at two-week intervals and assayed for the total number of sclerotia of *S. minor*, the proportion of sclerotia of *S. minor* infected by *S. sclerotivorum* and the density of *S. sclerotivorum*. The mean data from this trial are used to test the model.

3. THE MODELS

(*a*) *Hybrid compartmental, parasite–hyperparasite system (two-species model)*

The first model is developed from the classical predator–prey model used by Gubbins & Gilligan (1997*a*) to allow aspects of the dynamics of both the parasite and hyperparasite to be examined. The hyperparasite can only reproduce on infected parasites (Ayers & Adams 1979); infected parasites die more rapidly than uninfected ones, while only uninfected parasites are able to infect a host and reproduce (Adams & Ayers 1982). Accordingly, the prey component is expanded to a compartmental system of susceptible (uninfected) and infected parasites (Gubbins & Gilligan 1996). This yields the generic form for the two-species model,

$$\left.\begin{aligned} \frac{\mathrm{d}S}{\mathrm{d}t} &= (r_1 + r_2\delta(t - T_\mathrm{h}))S\left(1 - \frac{S}{K}\right) \\ &\quad - \mathrm{d}S - Xf(S, X), \\ \frac{\mathrm{d}I}{\mathrm{d}t} &= Xf(S, X) - (d + a)I, \\ \frac{\mathrm{d}X}{\mathrm{d}t} &= gI - hX, \end{aligned}\right\} \tag{1}$$

where S and I are the density of susceptible and infected sclerotia of *S. minor* in the soil, respectively, and X is the density of spores of *S. sclerotivorum* in the soil.

The ability of *S. minor* to reproduce depends on the amount of infected host tissue. Thus, parasite births are density dependent with a carrying capacity, K, that is a measure of the quantity of infected tissue available for infection. The birth rate has two components: (i) the continuous addition (r_1) of sclerotia dropping off lettuce plants (Adams 1986) or being produced on previously infected lettuce tissue in soil (Imolehin & Grogan 1980; Dillard & Grogan 1985*a*); (ii) a pulse (r_2) of sclerotia at harvest ($t = T_\mathrm{h}$) when the infected lettuce plants are disked into the soil (Adams 1986). The pulse is represented in the model by a Dirac delta function, $\delta(t-T_\mathrm{h})$. Parasite death occurs at a constant rate, d, through the natural decay of inoculum. The functional response of the hyperparasite to changes in parasite density, $f(S, X)$, is discussed in § 4; a is the additional parasite death rate due to infection by the hyperparasite, g is the hyperparasite birth rate and h is the hyperparasite death rate.

(*b*) *Host–parasite–hyperparasite system (three-species model)*

The omission of the host dynamics from the two-species model means the model cannot be used to assess the impact of the hyperparasite on disease incidence. Parasite reproduction also depends on the quantity of host tissue available for infection. For lettuce drop, the whole plant is the unit of population, so the total host density, N, remains constant. Plant to plant spread plays a minimal role in the epidemiology of lettuce drop (Dillard & Grogan 1985*b*), so secondary infection can be neglected. For a larger host, the probability of infection is greater, so the rate of infection of the hosts is proportional to the amount of host tissue. Thus, disease progress is described by a monomolecular equation, with the rate dependent on the susceptible parasite density and the amount of tissue per host. That is,

$$\frac{\mathrm{d}N_\mathrm{i}}{\mathrm{d}t} = r_{N_\mathrm{i}} SL(N - N_\mathrm{i}), \tag{2}$$

where N_i is the density of infected plants and r_{N_i} is the rate of infection of lettuce plants by *S. minor*. The logistic equation is used to describe how the amount of tissue per host, L, varies with time,

$$\frac{\mathrm{d}L}{\mathrm{d}t} = r_L L\left(1 - \frac{L}{K_L}\right), \tag{3}$$

where r_L is the growth rate and K_L is the carrying capacity. In the absence of senescence, the total amount of infected host tissue is $L_\mathrm{i} = eN_\mathrm{i}L$ where e represents the efficiency of the parasite at infecting host tissue. Assuming the rate of senescence to be proportional to the amount of infected tissue implies

$$\frac{\mathrm{d}L_\mathrm{i}}{\mathrm{d}t} = e\frac{\mathrm{d}}{\mathrm{d}t}(N_\mathrm{i}L) - uL_\mathrm{i}, \tag{4}$$

where u is the rate of senescence. Data are not available for N_i, L and L_i, so these variables can be rescaled to reduce the number of parameters in the model. Let,

$$n_\mathrm{i} = \frac{N_\mathrm{i}}{N}, \quad l = \frac{L}{K_L}, \quad l_\mathrm{i} = \frac{L_\mathrm{i}}{eNK_L}.$$

Then equations (2)–(4) become

$$\left.\begin{aligned} \frac{\mathrm{d}n_\mathrm{i}}{\mathrm{d}t} &= r_\mathrm{i} Sl(1 - n_\mathrm{i}), \\ \frac{\mathrm{d}l}{\mathrm{d}t} &= r_L l(1 - l), \\ \frac{\mathrm{d}l_\mathrm{i}}{\mathrm{d}t} &= \frac{\mathrm{d}}{\mathrm{d}t}(n_\mathrm{i} l) - ul_\mathrm{i}, \end{aligned}\right\} \tag{5}$$

where r_i is the rescaled infection rate of lettuce plants by *S. minor*.

Incorporating the host dynamics in the model requires a change to the parasite reproduction term: it is no longer described by the logistic equation, rather births are proportional to the amount of infected host tissue, l_i. When the host dynamics described by (5) are combined with the modified parasite–hyperparasite model, the resulting host–parasite–hyperparasite system includes the necessary features of the system during a growing season. Modelling the system over a number of seasons introduces a further discontinuity as the host is introduced at planting and removed at harvest. This gives

$$\left.\begin{aligned} \frac{\mathrm{d}S}{\mathrm{d}t} &= (r_1 + r_2\delta(t - T_\mathrm{h}))l_\mathrm{i} \\ &\qquad - \mathrm{d}S - Xf(S, X), \\ \frac{\mathrm{d}I}{\mathrm{d}t} &= Xf(S, X) - (d + a)I, \\ \frac{\mathrm{d}X}{\mathrm{d}t} &= gI - hX, \\ \frac{\mathrm{d}n_\mathrm{i}}{\mathrm{d}t} &= \begin{cases} r_\mathrm{i} Sl(1 - n_\mathrm{i}), & \text{host present,} \\ 0, & \text{host absent,} \end{cases} \\ \frac{\mathrm{d}l}{\mathrm{d}t} &= \begin{cases} r_L l(1 - l), & \text{host present,} \\ 0, & \text{host absent,} \end{cases} \\ \frac{\mathrm{d}l_\mathrm{i}}{\mathrm{d}t} &= \frac{\mathrm{d}}{\mathrm{d}t}(n_\mathrm{i} l) - ul_\mathrm{i}. \end{aligned}\right\} \tag{6}$$

The principal variables and parameters for the two- and three-species models are summarized in Appendix 1.

(*c*) *Decay of parasite and hyperparasite inoculum*

Sclerotia of *S. minor* survive well in soil for four to five years (Adams & Ayers 1979). Because this period of survival is long relative to the timescale of the experiment and there is likely to be a lag before substantial decay of inoculum (Dimond & Horsfall 1965), d is taken to be zero when fitting the model to the data. Spores of *S. sclerotivorum* survive well in soil for at least fifteen months (Ayers & Adams 1979), shorter than the duration of the field trial. In addition, *Laterispora brevirama* a known parasite of *S.*

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sclerotivorum was detected during the trial (Adams & Fravel 1990). Thus, h also incorporates per capita death of *S. sclerotivorum* due to general antagonism.

4. THE PARASITE–HYPERPARASITE INFECTION TERM

The infection term, $f(S, X)$, is the functional response of the hyperparasite to changes in parasite density (Hassell 1981; May 1981). We consider three forms here following Gubbins & Gilligan (1996). The simplest form is the Lotka–Volterra type functional response,

$$f(S, X) = bS, \tag{7}$$

which corresponds to mass action transmission. A generalization of the basic functional response supposes that each individual hyperparasite can make contact with $C(P)$ parasites per unit time (Heesterbeek & Roberts 1995), where $P = S + I$. Then the infection term has the form,

$$f(S, X) = bC(P)(S/P), \tag{8}$$

where b represents the probability of infection given contact, $C(P)$ is the contact rate and (S/P) is the proportion of contacts that are susceptible. Following Anderson & May (1991), we take the contact rate, $C(P) = P^m$ where $0 \leqslant m \leqslant 1$. At one extreme, $(m = 1)$, the functional response increases linearly with the total parasite population density; at the other, $(m = 0)$, each hyperparasite has contact with a roughly fixed number of susceptible parasites.

Responses (7) and (8) assume that the populations mix homogeneously, so any individual hyperparasite can infect any susceptible parasite. The functional response can be modified to allow for heterogeneous mixing by allowing the incidence rate to vary as powers of S and X (Liu *et al.* 1986, 1987; Hochberg 1991). Then $f(S, X)$ becomes

$$f(S, X) = bS^m X^{n-1}, \tag{9}$$

where the mixing parameters, m and n, are real and positive. Various mechanisms can account for different values of the mixing parameters (see discussion). In the present study, we are interested primarily in spatial interpretations of m and n, hence we refer to this response as the heterogeneous mixing response. Values of m or n less than one arise when only a proportion of a population can mix; if m or n equals one, the populations mix homogeneously; and values of m or n greater than one occur when the population is aggregated.

5. ENVIRONMENTAL FACTORS

The influence of environmental factors on activity has been studied for *S. minor* (Adams 1987) and *S. sclerotivorum* (Adams & Ayers 1980; Adams 1987). Arguably all the parameters in the models depend on environmental variables, but allowance for the environmental dependence of each parameter complicates the model unnecessarily. After preliminary work, we introduce environmental change selectively, for certain parameters, as a form of periodic forcing and a discrete change in parameters through time.

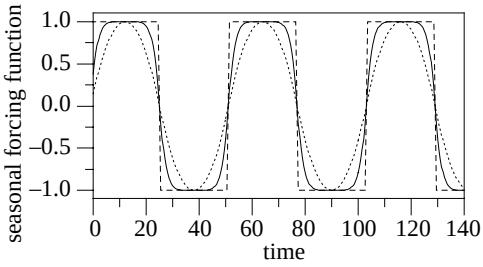

Figure 1. Seasonal forcing function, $w(t)$, described by equation (11) for three different values of q. (*a*) $q = 1$ (dotted curve). (*b*) $q = 3$ (solid curve). (*c*) $q = 100$ (dashed curve). Other parameters are $T = 52$ and $\varphi = 12$.

The parasitic activity of *S. sclerotivorum* depends on temperature, which affects its ability to control *S. minor* (Adams & Fravel 1990). This temperature dependence is modelled by letting the infection rate of *S. minor* by *S. sclerotivorum*, b, vary seasonally. That is,

$$b = b(t) = b_0(1 + b_1 w(t)), \tag{10}$$

where $0 \leqslant b_1 \leqslant 1$. The forcing function, $w(t)$, is periodic with period T (52 weeks) and phase φ (12 weeks) corresponding approximately to seasonal variation in temperature. We use the following generic form for $w(t)$ (figure 1):

$$w(t) = \begin{cases} 1 - \theta^{2q}, & 0 \leqslant \theta < 1, \\ (\theta - 2)^{2q} - 1, & 1 \leqslant \theta < 3, \\ 1 - (\theta - 4)^{2q}, & 3 \leqslant \theta < 4, \end{cases} \tag{11}$$

where $\theta = (4/T) \mod (t - \varphi, T)$, and q is a positive integer. Function (11) captures a range of shapes from (approximately) sinusoidal ($q = 1$) through to a square wave (q large). After exploratory analysis, the value of the shape parameter, q, was fixed at $q = 3$.

The parameters most likely to be influenced by changes in environmental conditions are the rate of infection of lettuce plants by *S. minor*, r_i, and the rate of infection of *S. minor* by *S. sclerotivorum*, b_0 and b_1 (Adams & Ayers 1980; Adams 1987). Thus, when incorporating a discrete change in parameters through time, three parameters change at time $t = t_\mathrm{sw}$,

$$r_\mathrm{i} \to \frac{r_\mathrm{i}}{c_\mathrm{r}}, \quad b_0 \to \frac{b_0}{c_\mathrm{i}}, \quad b_1 \to \frac{b_1}{c_\mathrm{a}}. \tag{12}$$

The changes, c_r, c_i and c_a, and the time of changing, t_sw, are treated as parameters to be estimated.

6. PARAMETER ESTIMATION

The models were fitted to three data sets from Adams & Fravel (1990). Each has a different initial hyperparasite density designated: low, $X_0 = 0.08$ spores g^{-1} soil; medium, $X_0 = 0.8$ spores g^{-1} soil; high, $X_0 = 8$ spores g^{-1} soil. Because there is

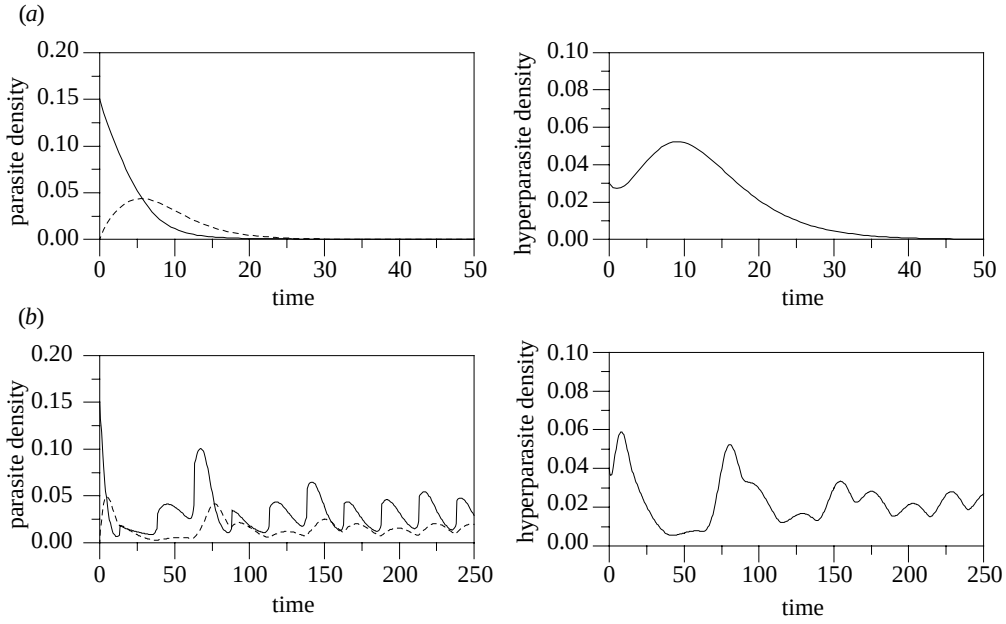

Figure 2. Time course plots for: the susceptible parasite density, S, (solid curve); infected parasite density, I, (dashed curve); and the hyperparasite density, X, for the host–parasite–hyperparasite system, (6), with the basic functional response, (7), and no seasonal forcing. (*a*) System including only within-season dynamics ($r_b = 0.0$). (*b*) System including pulsed inputs and planting and harvesting of host ($r_b = 0.5$). Other parameters are $r_a = 0.2$, $b_0 = 5.3$, $b_1 = 0.0$, $d = 0.04$, $a = 0.2$, $g = 0.3$, $h = 0.2$, $r_i = 1.5$, $r_L = 0.8$ and $u = 0.1$.

an apparent jump from the first data point to the second in the parasite population, the initial population (S_0) was treated as a parameter to be estimated, so that it was varied to obtain the best fit. Data for S, I and X were scaled to give similar magnitudes for fitting, thereby avoiding undue influence of certain components with large absolute deviances. Fitting was by empirical weighted least squares with the reciprocal of the variance for each compartment using FACSIMILE (1995). The goodness of fit was tested by examination of the residual plots and the consistency and convergence of the parameter estimates for small differences in initial estimates (Gubbins & Gilligan 1996, 1997*a*). The consistency of the parameter estimates across the three data sets was also examined since, other things being equal, differences in X_0 would not be expected to affect parameters in the model. The change in deviance for hierarchically related models was also examined (Aitkin *et al.* 1989; Ross 1990) to test for redundant parameters.

7. RESULTS

(*a*) *Planting, harvesting and parasite persistence*

Here we analyse the population dynamics of the two- and three-species models to show the important role planting and harvesting play in the persistence of the parasite. Analysis of the three-species model without disturbances shows that the parasite and hyperparasite always die out (see Appendix 2). This is a consequence of the decay of the infected host tissue: the amount of infected tissue always decays to zero, hence the parasite is unable to reproduce indefinitely and the hyperparasite eradicates the parasite (figure 2*a*). When the discontinuities associated with planting and harvesting are introduced to the three-species model, the parasite is able to persist (figure 2*b*). Repeated planting of the host maintains a quantity of host tissue in the system, enabling the parasite to reproduce and survive.

Equivalently, in the two-species model without disturbances, the parasite is eradicated when the parasite basic reproductive number, r_1/d is less than unity (see Appendix 2). This is equivalent to the host density dropping below a threshold level (Anderson & May 1981, 1991) as occurs in the three-species model. When r_1/d exceeds one in the two-species model, we are implicitly assuming that the host population is maintained above the threshold density. We conclude that for both the two- and three-species models, the disturbances associated with planting and harvesting allow the parasite population to persist.

(*b*) *Testing the models against field data*

We can identify a characteristic pattern of dynamical behaviour in the field data (Adams & Fravel 1990). Initially the susceptible *S. minor* population declines rapidly. When the host is introduced ($t = 18$ weeks), the population density levels off until there is a sharp rise at harvest ($t = 26$ weeks). The population then decays less rapidly than before to a low density ($t = 60$ weeks). The susceptible *S. minor* density remains near this level for the remainder of the experiment with small peaks at harvest ($t = 79$, 110, 133 weeks) (figures 3 and 4). The density of infected *S. minor* is initially zero, but rises sharply when the density of susceptible *S. minor* declines (figures 3 and 4). Planting of the host is marked on

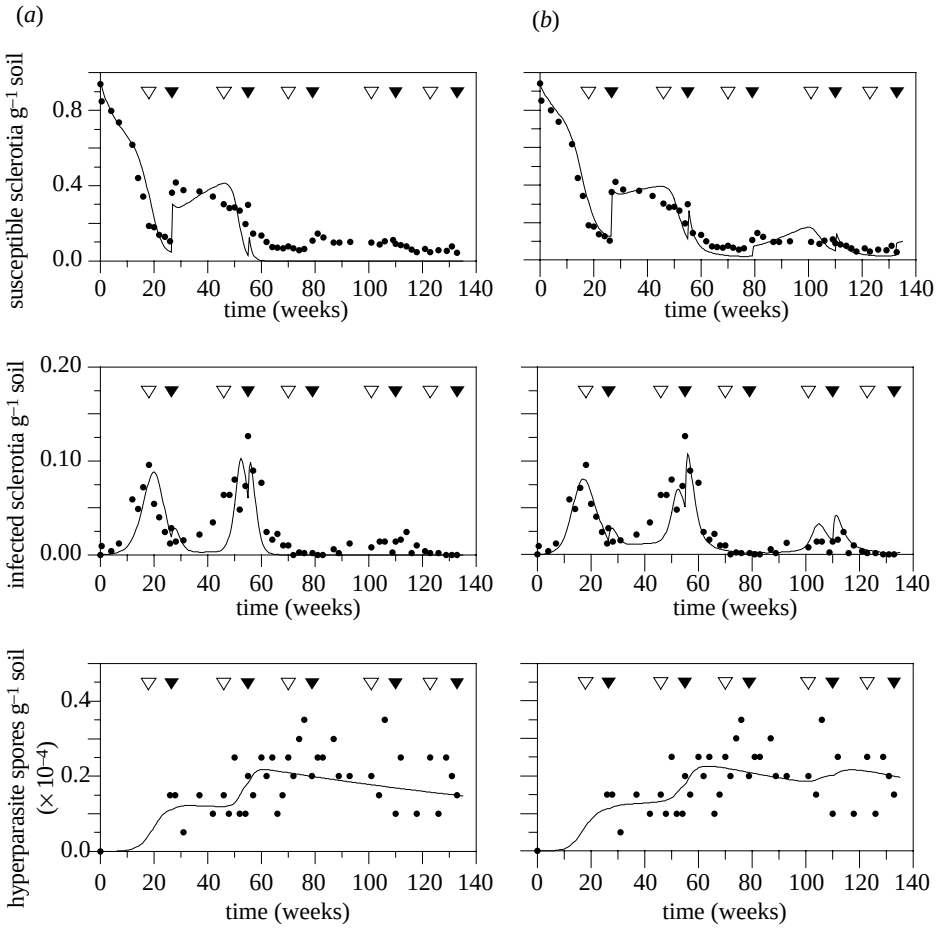

Figure 3. Fit of the hybrid compartmental, parasite–hyperparasite system (two-species model) with pulsed inputs, (1), and a seasonally forced infection term given by (10) and (11) for different functional responses. (*a*) Basic response, (7). (*b*) Heterogeneous mixing response, (9). Fits are shown with medium initial hyperparasite density, $X_0 = 0.8$ spores g^{-1} soil. The solid line is the least-squares fit and the points are the data from Adams & Fravel (1990). Planting of the host is marked by a hollow triangle and harvesting is indicated by a solid triangle.

the figures by a hollow triangle and harvesting is indicated by a solid triangle (figures 3, 4 and 6).

The two-species model (the hybrid compartmental parasite–hyperparasite system) with seasonal forcing, pulsed inputs and functional responses (7) and (9) was fitted to the data (figure 3; table 1). Parameter estimates were consistent across the three data sets (table 1). However, the model with the basic functional response led to fade out of the *S. minor* population (figures 3*a*). This did not occur with the heterogeneous mixing response (figures 3*b*) for which $m > 1$, $n < 1$ (table 1). An approximate F-test (Aitkin *et al.* 1989; Ross 1990) indicated a significant ($\alpha = 0.01$) reduction in deviance for the heterogeneous mixing response over the basic response. The model fitted the data for *S. minor* (separated into susceptible, S, and infected, I, sclerotia) closely, particularly during the early cycles of infection and recovery ($t < 60$ weeks) (figure 3). Peaks in the observed density of infected sclerotia were closely matched by those of the models (figure 3).

The effects of the basic, (7), proportionate mixing, (8) and heterogeneous mixing, (9), responses on the three-species model (the host–parasite–hyperparasite system) with seasonal forcing and pulsed inputs were examined (figure 4; table 2). Because the fits to the data for *S. sclerotivorum* were similar for the two- and three-species models, those for the three-species model are not shown. Parameter estimates were consistent across the data sets for each model (table 2). Approximate F-tests indicated a significant ($\alpha = 0.05$) reduction in deviance only for the proportionate mixing response over the basic response. However, convergence was achieved only for $m \approx 0.95$ (table 2), so that there is little difference between the shape of this response, (8), and the basic response, (7). Predictions from the models using the estimated values for the parameters did not lead to the fade out of susceptible *S. minor*, but tended to very low densities after 105 weeks (figures 4*a*, *b*). Peaks in the density of infected sclerotia were closely matched by the models (figures 4*a*, *b*). Beyond 60 weeks, however, the density of infected sclerotia tended to be over estimated. We conclude that all three models fit the data adequately when the host dynamics are included. However, the predictions for the parasite populations in the troughs between epidemics (figures 4*a*, *b*) are closer to the observed values for the heterogeneous mixing response. We argue that heterogeneous mixing is the most ap-

Table 1. *Parameter estimates for the two-species model, (1), with pulsed inputs and seasonal forcing*

	functional response					
	basic, (7)			heterogeneous mixing, (9)		
parameter	$X_0 = 0.08$	$X_0 = 0.8$	$X_0 = 8$	$X_0 = 0.08$	$X_0 = 0.8$	$X_0 = 8$
S_0	0.957	0.979	1.069	0.877	0.877	0.936
r_1	0.102	0.107	0.216	0.117	0.087	0.099
r_2	1.866	2.835	3.594	1.842	2.012	1.556
K	0.511	0.499	0.645	0.595	0.695	0.849
b_0	2.686	2.270	2.588	2.874	2.459	1.952
b_1	0.903	0.951	0.974	0.767	0.859	0.850
a	0.615	0.657	0.770	0.671	0.772	0.714
m	—	—	—	1.600	1.528	1.470
n	—	—	—	0.909	0.924	0.854
g	0.124	0.132	0.121	0.101	0.116	0.101
h	0.011	0.008	0.006	0.005	0.008	0.008
RSS[a]	148.80	107.74	78.45	74.11	63.95	57.43
d.f.[b]	137	137	140	135	135	138

[a]Residual sum of squares.
[b]Degrees of freedom.

propriate form for the functional response.

The estimated dynamics for the proportion of infected plants (n_i), tissue per plant (l) and total infected tissue (l_i) are shown in figure 5 for the three-species model with heterogeneous mixing, pulsed inputs and seasonal forcing. The discontinuities upon removal of the host crop are evident (figures 5*a*, *b*). The dynamics for the total infected tissue show replenishment during the growing seasons with gradual decline between seasons and reflect the persistence of this source of inoculum (figure 5*c*).

Given the behaviour of the model fit before and after 60 weeks, we allowed the parameters for the rate of infection of lettuce plants by *S. minor*, r_i, and the rate of infection of *S. minor* by *S. sclerotivorum*, b_0 and b_1, to differ before and after approximately 60 weeks (see § 5). To test if changes in environmental conditions alone could account for the persistence of *S. minor*, this model was fitted with the basic functional response (table 2). The temporal change in parameters improved the later fit after 60 weeks (figure 4*c*) with avoidance of low predicted values for susceptible *S. minor*, though the densities of infected *S. minor* still tended to be over estimated (figure 4*c*). We conclude that changes in some environmental conditions may contribute to the persistence of *S. minor*.

(*c*) *The influence of seasonal forcing and pulsed inputs*

Following earlier work (Gubbins & Gilligan 1997*a*), the influence of the delta function pulses and seasonal forcing were separately tested for the two- and three-species models with the basic and heterogeneous mixing responses. For brevity, results are shown only for the three-species model with the heterogeneous mixing response (figure 6). When fitting the two- and three-species model, approximate F-tests indicated a significant ($\alpha = 0.01$) reduction in deviance for the models with seasonal forcing over those without. The absence of seasonal forcing led to severe under estimation of the susceptible sclerotial densities and to systematic error when estimating the density of infected sclerotia (figure 6*b*). We conclude that seasonal forcing is necessary to capture the dynamical features of the data.

For the two-species model, approximate F-tests indicated a significant ($\alpha = 0.01$) reduction in deviance for models with pulsed inputs over those without. However, when fitting the three-species model, the absence of pulses did not greatly affect the fit of the model to the *S. minor* data (figure 6*a*). Examination of the deviances shows there was no significant ($\alpha = 0.05$) reduction in deviance for the models with pulses over those without. However, the rise in the susceptible *S. minor* population immediately after harvest is much more gradual without pulsed inputs than is observed in the field or predicted by the three-species model with pulsed inputs (cf. figures 4 and 6). We conclude that pulsed inputs are necessary to capture the dynamical features of the data.

8. DISCUSSION

We introduced and tested two models for biological control in a disturbed environment. The models were developed with close reference to a practical biological problem of controlling an economically impor-

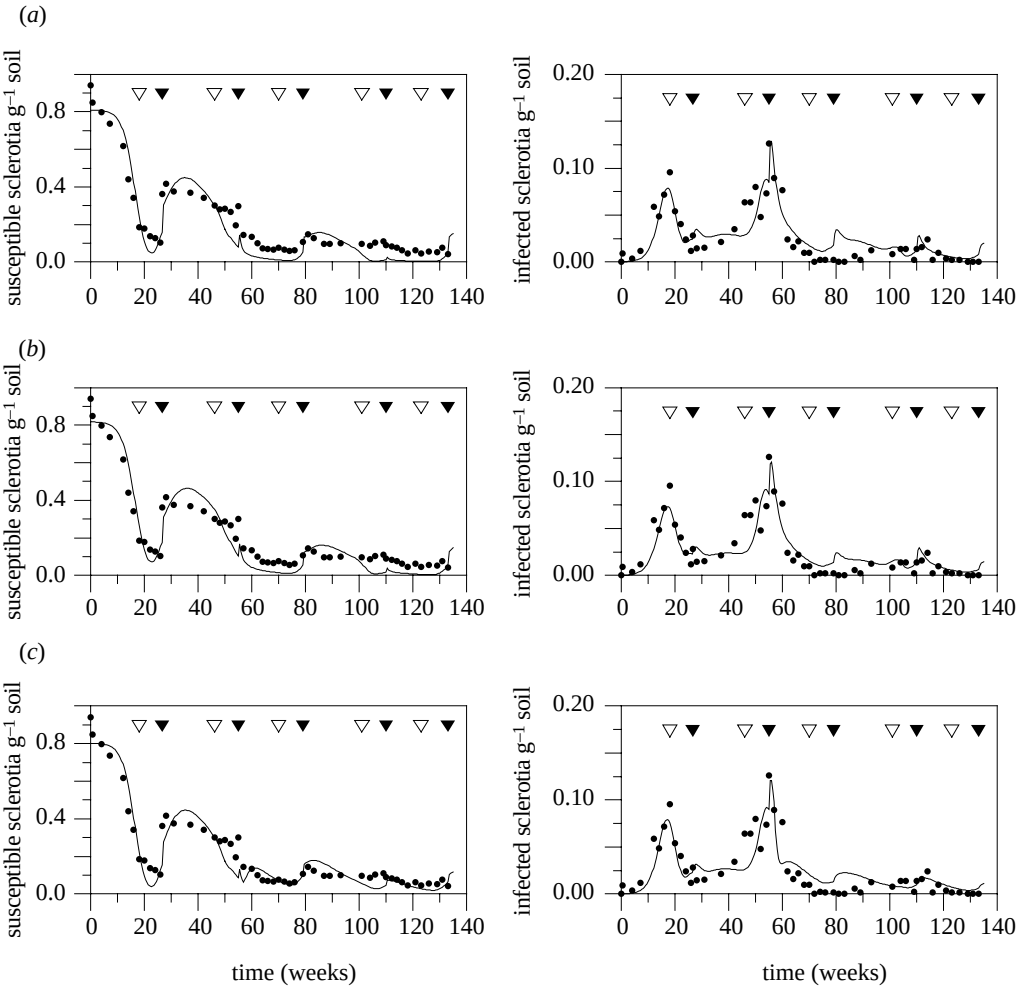

Figure 4. Fit of the host–parasite–hyperparasite system (three-species model) with pulsed inputs, (6), and a seasonally forced infection term given by (10) and (11) for different functional responses. (*a*) Basic response, (7). (*b*) Heterogeneous mixing response, (9). (*c*) Basic response, (7), with a change in the parameters described by (12). Fits are shown for the susceptible and infected *S. minor* populations with medium initial hyperparasite density, $X_0 = 0.8$ spores g^{-1} soil. The solid line is the least-squares fit and the points are the data from Adams & Fravel (1990). Planting of the host is marked by a hollow triangle and harvesting is indicated by a solid triangle.

tant plant disease, but their generic structure makes them applicable to a wide spectrum of host–parasite–hyperparasite systems. The two-species model is an *SI* model with an external source of infection and its structure is similar to Anderson & May's (1981) free-living infective stage model (their model G). With the introduction of secondary infection (host to host spread) (Gilligan & Kleczkowski 1997), the three-species model can be used to analyse the control of other crop diseases (cf. Gilligan 1994). The generic structure of the models also has broad implications for the analysis of ecological systems in disturbed environments. To date, little attention has been given to the analysis of the effect of discontinuities in interacting populations (Barlow 1993; Hanski *et al.* 1993; Shaw 1994; Briggs & Godfray 1996; Gubbins & Gilligan 1997). Most ecological and epidemiological models describe systems that allow continuous interactions between populations, yet discontinuities are an inherent property of many plant, microbial and invertebrate populations.

Analysis and fitting have highlighted several important features of the dynamics of the models which we discuss below. The relationship with other models of biological control is also discussed.

(*a*) *Discontinuities in parasite release and cropping*

Two types of discontinuity are included in the models. One corresponds to a pulse of new parasites at harvest as crop debris is returned to the soil. This is represented by a delta function (two- and three-species models). The other reflects the discontinuous presence of the host (three-species model only). For the two-species model, the pulses are necessary if the model is to describe the data. However, for the three-species model, the omission of pulses did not significantly affect the fit of the model, though it does predict a more gradual increase in the susceptible *S. minor* population immediately after harvest than is observed in the field (figure 6*a*). This suggests that pulsed inputs are necessary if the models are to capture all the dynamical features of the parasite data. The differences between the results for the two-

Table 2. *Parameter estimates for the three-species model, (6), with pulsed inputs and seasonal forcing*

	functional response					
	basic, (7)			proportionate mixing, (8)		
parameter	$X_0 = 0.08$	$X_0 = 0.8$	$X_0 = 8$	$X_0 = 0.08$	$X_0 = 0.8$	$X_0 = 8$
S_0	0.739	0.810	0.839	0.697	0.826	1.115
r_1	0.115	0.115	0.122	0.154	0.148	0.102
r_2	0.202	0.192	2.90	0.202	0.148	0.205
b_0	4.608	4.184	2.674	4.848	3.960	2.541
b_1	0.864	0.815	0.783	0.868	0.840	0.701
a	1.098	1.163	0.881	1.078	1.112	0.957
m	—	—	—	0.968	0.944	0.847
g	0.119	0.111	0.119	0.116	0.116	0.097
h	0.011	0.009	0.012	0.011	0.009	0.008
r_i	5.841	5.864	3.738	5.913	6.056	5.959
r_L	1.176	1.143	1.566	1.059	1.047	1.233
u	0.095	0.101	0.130	0.111	0.116	0.100
RSS[a]	135.06	113.52	97.57	127.40	108.67	151.36
d.f.[b]	135	135	138	134	134	137
	heterogeneous mixing, (9)			change in parameters, (12)		
	$X_0 = 0.08$	$X_0 = 0.8$	$X_0 = 8$	$X_0 = 0.08$	$X_0 = 0.8$	$X_0 = 8$
S_0	0.739	0.817	0.841	0.756	0.801	0.868
r_1	0.109	0.111	0.099	0.109	0.132	0.119
r_2	0.190	0.184	0.234	0.211	0.155	0.227
b_0	4.483	3.717	5.162	4.657	4.067	2.832
b_1	0.861	0.828	0.830	0.865	0.827	0.802
a	0.927	1.157	0.963	1.110	1.116	0.840
m	1.130	1.059	1.239	—	—	—
n	0.990	0.980	1.108	—	—	—
g	0.105	0.109	0.117	0.117	0.117	0.115
h	0.009	0.009	0.010	0.010	0.010	0.011
r_i	6.718	4.165	4.480	5.573	5.713	7.567
r_l	0.972	1.201	1.111	1.195	1.114	1.232
u	0.117	0.106	0.117	0.096	0.117	1.298
t_sw	—	—	—	57.488	57.133	64.994
c_r	—	—	—	3.979	3.731	7.031
c_i	—	—	—	3.998	3.822	3.813
c_a	—	—	—	3.867	2.307	4.033
RSS[a]	133.36	110.96	116.81	142.38	109.69	81.16
d.f.[b]	133	133	136	131	131	134

[a]Residual sum of squares.
[b]Degrees of freedom.

and three-species models imply that the disturbances due to cropping (which are explicitly included in the three-species model) are of even greater importance than the pulsed inputs in the population dynamics.

Analysis of the dynamical behaviour of the models demonstrated that repeated planting and harvesting allows the persistence of the parasite. Without disturbances, the equilibrium results show that the parasite is eradicated. However, repeated cropping allows the parasite to persist by maintaining a quantity of host tissue in the system. A more detailed mathematical analysis of the influence of disturbances on persistence is presented elsewhere (Gubbins & Gilligan 1997*b*).

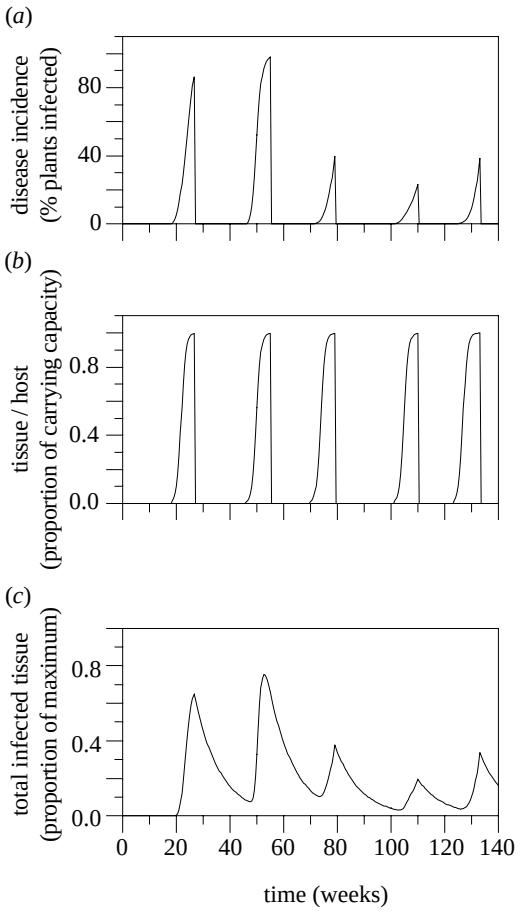

Figure 5. Time course plots for the host variables of the host–parasite–hyperparasite system (three-species model). (*a*) Disease incidence (n_i). (*b*) Tissue per host (l). (*c*) Total infected host tissue (l_i). The plots are for the model with the heterogeneous mixing response, (9), when fitted to the data set with medium initial hyperparasite density, $X_0 = 0.8$ spores g^{-1} soil.

(*b*) *Heterogeneous mixing and fade-out of the parasite population*

The models with the basic functional response, (7), yielded consistent parameter estimates for treatments with different initial hyperparasite densities (tables 1 and 2). The models match the data for the first 60 weeks (three growing seasons), but after that the *S. minor* population fades out (figures 3*a* and 4*a*), with the predicted values well below the observed values. Changing the functional response to the more general proportionate mixing form, (8), does not reduce fade-out (not shown). One possible cause for the discrepancy between the predicted and observed population densities is spatial heterogeneity. Because the models are mean field approximations of a spatial system, spatial effects may account for failure of the biological control agent to eliminate the pathogen. A simple way to allow for spatial effects is to relax the assumption of homogeneous mixing implicit in (7) and (8). This can be done by changing the functional response to the heterogeneous mixing form, (9).

Including heterogeneous mixing, (9), reduces fade-out in the models (figures 3*b* and 4*b*), though the effect is less pronounced in the three-species model. The infection term, bS^mX^{n-1}, increases faster than linearly in S ($m > 1$; tables 1 and 2), implying that when a hyperparasite encounters one parasite, it is likely to encounter another one, that is the parasites are aggregated. This agrees with observations in the field: *S. minor* is found in aggregations in soil after infected lettuce plants are disked into the soil (Adams 1986). Estimates of $n < 1$ (tables 1 and 2) indicate that only a proportion of the hyperparasites are able to infect the parasite. Similar results on the effects of homogeneous and heterogeneous mixing were also obtained in our earlier study of the *S. minor–S. sclerotivorum* system (Gubbins & Gilligan 1997*a*).

Mathematical analyses of other models with heterogenous mixing responses have demonstrated that mixing parameters in the range $m > 1$, $n < 1$ have a stabilizing effect on the population dynamics (Liu *et al.* 1986, 1987; Hochberg 1991). In particular, Liu *et al.* (1986, 1987) proved that there was always a globally stable non-trivial equilibrium (i.e. one at which all populations are non-zero) for $m > 1$, $n < 1$ for several different classes of epidemiological models where the host population is constant. For the models considered in Gubbins & Gilligan (1997*a*) and this paper, we also prove similar results (see Appendix 3). This increase in the stability of the population dynamics accounts for the greater persistence of the parasite with the heterogeneous mixing response.

Fade-out is a phenomenon common in epidemiological models with homogeneous mixing (Anderson & May 1991). This is the case in measles dynamics where spatial effects may account for the observed persistence of infection (Bolker & Grenfell 1993, 1995). Spatial heterogeneity has also been shown to stabilize otherwise unstable insect host–parasitoid systems, thus allowing the populations to persist (Hassell & Pacala 1990; Hassell *et al.* 1991). Spatial heterogeneity was incorporated into our models by allowing for heterogeneous mixing of the populations. For the two-species model, this simple change improves the fit of the model and prevents the fade-out predicted in the homogeneous mixing case. Allowing for heterogeneous mixing in the three-species model does not significantly improve the fit of the model, though the predicted *S. minor* populations are closer to the observed densities (cf. figures 4*a*, *b*).

We have assumed that values of m or n other than one in the nonlinear functional response, (9), arise through heterogeneous mixing of the parasite and hyperparasite populations. Several alternative mechanisms can also account for different values of the mixing parameters (see, for example, Liu *et al.* 1986, 1987; Hochberg 1991). Values of m or n less than one arise through saturation effects in some way analogous to the Holling type II response. Values of m or n greater than one occur when stress through crowding leads to an increase in susceptibility or if the host can survive low levels of infection. However, there is no experimental evidence that suggests any of these mechanisms play a role in the *S. minor–S. sclerotivorum* system. We conclude that any nonlinearities in

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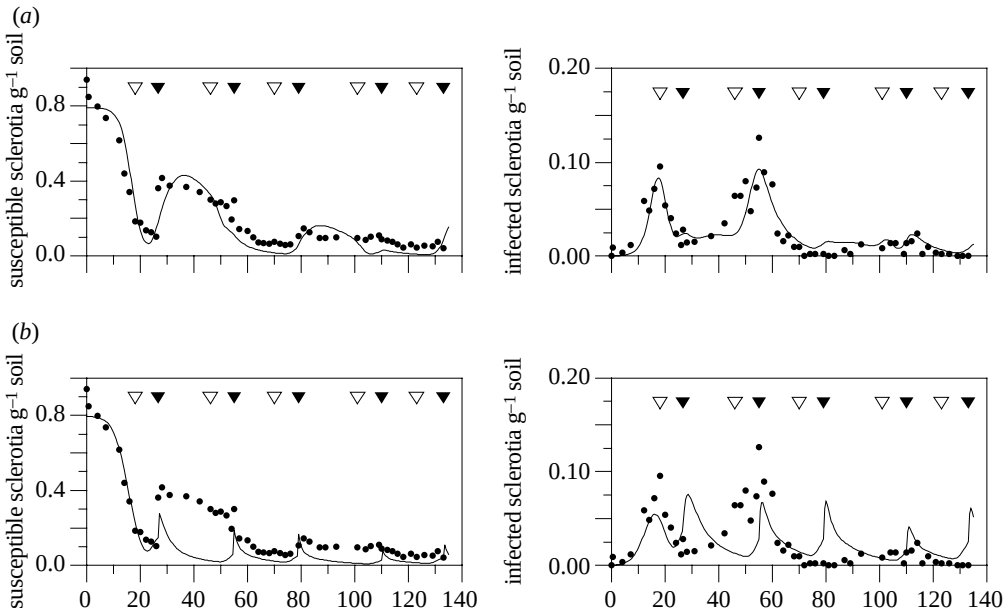

Figure 6. Fit of the host–parasite–hyperparasite system (three-species model), (6), with the heterogeneous mixing response, (9), to the *S. minor* data for the medium initial hyperparasite density, $X_0 = 0.8$ spores g^{-1} soil. (*a*) With seasonal forcing and without the delta function pulse at harvest; (*b*) without seasonal forcing and with the delta function pulse at harvest. The solid line is the least-squares fit and the points are the data from Adams & Fravel (1990). Planting of the host is marked by a hollow triangle and harvesting is indicated by a solid triangle.

transmission arise through heterogeneous mixing of the populations.

(*c*) *Seasonal forcing and change in environmental conditions*

Adams & Fravel's (1990) experiments took place over two years and it is likely that variation in environmental variables occurred over the course of the experiment. These variables influence the activity of the parasite, *S. minor* (Adams 1987) and the hyperparasite, *S. sclerotivorum* (Adams & Ayers 1980; Adams 1987). The variation in the levels of hyperparasite activity was allowed for by seasonally forcing the infection rate. If the transmission term is not seasonally forced, then the three-species model fails to fit the data (figure 6*b*). Given the behaviour of the model fit before and after 60 weeks (figure 4*a*, *b*), we allowed for changes in environmental variables by including a stepped change in rate of infection of the host by the parasite and the infection rate of the parasite by the hyperparasite at some time (approximately 57 weeks) selected by optimization. This improved the fit and noticeably reduced fade-out (figure 4*c*). Although such large changes as the ones given by the fit are unlikely, the improvement shows that changes in some environmental conditions may contribute to the persistence of *S. minor*.

(*d*) *Introduction of the host dynamics*

Although the three-species model includes variables to describe the dynamics of disease and host growth, data were not available to test the assumptions in the model for host growth. However, the fit of the model to the S, I and X data does at least indicate that the mechanisms are plausible. Examination of the n_i plot (figure 5*a*) shows the reduction of disease incidence as the *S. minor* population is controlled. The predicted values for the end of season levels of incidence are not the same as the field trial (Adams & Fravel 1990), but do have the same downward trend. The time courses for the other two host variables, l and l_i, show that hosts grow to their carrying capacity each season (figure 5*b*) and the total amount of infected tissue, l_i, increases during the growing season and decays exponentially between crops (figure 5*c*). Data for the level of disease incidence can be measured experimentally, and is the host variable of most interest to a grower. The amount of infected host tissue is very difficult to quantify, especially after it has been disked into the soil, but this is the most important host variable in controlling the dynamics of infection and biological control.

(*e*) *Relationship with other models of biological control*

In this paper, we have used models to analyse biological control in a disturbed environment. Recently, other biocontrol programmes have been investigated by modellers (Hochberg & Waage 1991; Barlow & Goldson 1993; Lonsdale *et al.* 1995). Theoretical studies have been undertaken to predict the efficacy of control programmes (Kakehashi *et al.* 1984; May & Hassell 1988; Thomas *et al.* 1995), to identify the necessary characteristics for potential biocontrol agents (Beddington *et al.* 1978; Hochberg 1989; Godfray & Briggs 1995; Briggs & Godfray 1996) or to select between different candidate species of biocontrol agent (Godfray & Waage 1991). These studies ex-

amined the control of insect pests using parasitoids and pathogens or the control of weeds using insect herbivores.

Many of these models used parameters estimated from life-history tables. Several studies also used model fitting, especially to estimate transmission parameters (Hochberg & Waage 1991; Thomas *et al.* 1995). In particular, Hochberg & Waage (1991) analysed an age-structured mass-action transmission model of a baculovirus in Rhinoceros beetles. They used model fitting to determine the relative importance of the different transmission pathways and found their results agreed with experimental evidence. Hochberg & Waage (1991) also found that their model under estimated the pest population. They suggested that spatial effects could account for the discrepancy, but did not test this hypothesis.

Barlow & Goldson (1993), Lonsdale *et al.* (1995), Thomas *et al.* (1995) and Briggs & Godfray (1996) used maps to model the change in populations from the end of one season to the beginning of the next in multiseasonal biological control programmes where it is assumed that the pest and control agent do not interact between seasons. In principle, it is possible to describe the between-season dynamics of *S. minor* and *S. sclerotivorum* using a simple map, but this obscures critical dynamics of the lettuce drop system where the parasite and hyperparasite populations interact during inter-crop periods (Adams & Fravel 1990).

The models for biological control discussed above are all essentially parasite–hyperparasite systems (with the exception of Lonsdale *et al.* 1995) that do not include the host explicitly. In part, this is because the parasite–hyperparasite dynamics are of central importance in determining the success of a biological control programme (May & Hassell 1988). Such simple models are useful in theoretical treatments of biological control, such as identifying general characteristics for a potential biocontrol agent. However, their use in practical applications is limited because they do not explicitly include the host dynamics and so cannot be used to predict the impact of control on the host population. This is of importance when assessing the economic benefits of instigating a control programme for an agricultural crop.

Model fitting and parameter estimation using experimental data are important techniques for selecting between various alternative models. We have used only elementary methods in model fitting here with an assumption of independent errors due to measurement variability. Further work is under way to explore the effects of refined techniques that take account of auto-correlation, non-normal errors and unequal variances, as well as the goodness of fit to the replicate data.

9. CONCLUSIONS

Examination of the data showed discontinuities in the population dynamics, with disturbances due to cropping and a pulsed input to the *S. minor* population at harvest. We have developed models with a broad generic structure that capture the features of the lettuce drop system. The data were successfully described by a parasite–hyperparasite system, the two-species model. This model separated the parasite population into a susceptible class that cause disease and an infected class that support the reproduction of the hyperparasite. Because the two-species model is a parasite–hyperparasite system, it does not explicitly include the host dynamics. In the three-species model, we constructed a plausible model for the host dynamics that adequately predicted the observed behaviour and gave biologically reasonable and consistent parameter estimates.

The experimental data show that *S. minor* persists in the field. Repeated planting of the host maintains a quantity of host tissue in the system enabling the parasite to reproduce and survive. Fade-out was prevented in the model by allowing for heterogeneous mixing of the populations. This is not a unique solution: changes in environmental conditions may also prevent fade-out. Further experimental and theoretical work is needed to distinguish between these effects.

We thank P. B. Adams and D. R. Fravel for providing us with their data. This work was funded by the Biotechnology and Biological Sciences Research Council (BBSRC), which we gratefully acknowledge. We also thank A. Kleczkowski, J. E. Truscott and other members of the Botanical Epidemiology group for helpful comments on the work.

APPENDIX 1. SUMMARY OF PRINCIPAL VARIABLES AND PARAMETERS

Table 3 shows a summary of the principal variables and parameters in the current paper.

APPENDIX 2. PARASITE PERSISTENCE WITHOUT DISTURBANCES

Here we present the mathematical results discussed in § 7 *a*. The equation for the rate of change in susceptible sclerotia of the two-species model, (1), without pulses can be rewritten as

$$\frac{\mathrm{d}S}{\mathrm{d}t} = (r_1 - d)S\left(1 - \frac{r_1 S}{(r_1 - d)K}\right) - Xf(S, X). \tag{13}$$

When $r_1/d < 1$ (i.e. $r_1 < d$), the two-species model has a unique equilibrium at the origin. The Lyapunov function,

$$V = S + I + \left(\frac{d + a}{g}\right)X, \tag{14}$$

satisfies $V = 0$, $\mathrm{d}V/\mathrm{d}t = 0$ at $(0, 0, 0)$ and $V > 0$, $\mathrm{d}V/\mathrm{d}t < 0$ everywhere else. Hence the origin is globally asymptotically stable (see, for example, Glendinning 1994). When $r_1/d > 1$, the origin remains a fixed point, but local stability analysis shows it is a saddle point. Thus, we can identify r_1/d as the basic

Table 3. *Summary of principal variables and parameters*

variable	description	dimensions and units
S	susceptible parasite density	[sclerotia g^{-1} soil]
I	infected parasite density	[sclerotia g^{-1} soil]
X	hyperparasite density	[spores g^{-1} soil]
n_i	proportion of hosts infected[a]	(dimensionless)
l	tissue per host (proportion of maximum)[a]	(dimensionless)
l_i	total infected host tissue (proportion of maximum)[a]	(dimensionless)
parameter	**description**	**dimensions and units**
r_1	parasite birth rate (continuous)[b]	$[\text{time}]^{-1}$
r_2	parasite birth rate (pulsed)[b]	$[\text{time}]^{-1}$
d	natural parasite death rate	$[\text{time}]^{-1}$
a	additional parasite death rate due to infection	$[\text{time}]^{-1}$
b_0	infection rate of parasite by hyperparasite (forced)[c]	[spores g^{-1} soil]$^{-1}$ $[\text{time}]^{-1}$
b_1	amplitude of seasonal forcing	(dimensionless)
w	seasonal forcing function	(dimensionless)
q	shape parameter for forcing function	(dimensionless)
m	parasite mixing parameter	(dimensionless)
n	hyperparasite mixing parameter	(dimensionless)
g	hyperparasite birth rate	[spores g^{-1} soil] [sclerotia g^{-1} soil]$^{-1}$ $[\text{time}]^{-1}$
h	hyperparasite death rate	$[\text{time}]^{-1}$
r_i	infection rate of host by parasite[a]	[sclerotia g^{-1} soil]$^{-1}$ $[\text{time}]^{-1}$
r_L	rate of host growth	$[\text{time}]^{-1}$
u	rate of senescence of infected host tissue	$[\text{time}]^{-1}$

[a]Rescaled variable or parameter (see § 3).
[b]Dimensions for r_1 and r_2 are given for the two-species model.
[c]Dimensions for b_0 are given for the basic functional response, (7).

reproductive number for the parasite (Anderson & May 1991).

At a fixed point of the three-species model, (6), without disturbances, $\mathrm{d}n_i/\mathrm{d}t = 0$ and $\mathrm{d}l/\mathrm{d}t = 0$, thus the $\mathrm{d}l_i/\mathrm{d}t$ equation implies that $l_i = 0$ at equilibrium. The equations for $\mathrm{d}S/\mathrm{d}t$, $\mathrm{d}I/\mathrm{d}t$ and $\mathrm{d}X/\mathrm{d}t$ have a unique solution $S = I = X = 0$. The solutions of the $\mathrm{d}l/\mathrm{d}t$ equation are $l = 0$ or $l = 1$. The equilibrium value for $n_i = n_i^*$ is not determined uniquely by the parameters but depends also on the initial conditions. Thus, the system has two equilibria at $(S, I, X, n_i, l, l_i) = (0, 0, 0, n_i^*, 0, 0)$ and $(S, I, X, n_i, l, l_i) = (0, 0, 0, n_i^*, 1, 0)$.

Local stability of an equilibrium is determined by the Jacobian of the system. Note that

$$\frac{\partial}{\partial l}\left(\frac{\mathrm{d}l}{\mathrm{d}t}\right) = r_L(1 - 2l), \tag{15}$$

and all other derivatives of the l equation are zero (see equations (5) and (6)). The row of the Jacobian, J, for the $\mathrm{d}l/\mathrm{d}t$ equation has zero entries except for the l derivative given by (15). Thus, one root of the characteristic polynomial, $\det(J - \lambda I) = 0$, is $\lambda = r_L(1 - 2l)$. At the equilibrium with $l = 0$, one eigenvalue is $\lambda = r_L > 0$, hence it is unstable. A Lyapunov function,

$$V = S + I + \frac{a}{g}X + \frac{r_1}{u}((n_i^* - n_i) + (1 - l) + l_i), \tag{16}$$

shows that $(0, 0, 0, n_i^*, 1, 0)$ is globally stable. Because $n_i(t)$ is monotonic increasing, $0 \leqslant n_i(t) \leqslant n_i^*$ and $n_i^* - n_i \geqslant 0$. Similarly, $l(t)$ is monotonic increasing, so $0 \leqslant l \leqslant 1$ and $1 - l \geqslant 0$. Thus $V = 0$, $\mathrm{d}V/\mathrm{d}t = 0$ at $(0, 0, 0, n_i^*, 1, 0)$ and $V > 0$, $\mathrm{d}V/\mathrm{d}t < 0$ everywhere else. Hence $(0, 0, 0, n_i^*, 1, 0)$ is globally asymptotically stable (see, for example, Glendinning 1994).

APPENDIX 3. HETEROGENEOUS MIXING AND STABILITY

When fitting the models with a heterogeneous mixing response to the *S. minor–S. sclerotivorum* data, we have obtained estimates for the mixing parameters, $m < 1$ and $n > 1$ (tables 1 and 2; see also Gubbins & Gilligan 1997*a*, their table 1). In this appendix, we prove the results on the influence these values for mixing parameters have on the stability of the population dynamics, as discussed in § 8 *b*.

(a) *The Gubbins & Gilligan (1997a) model*

The Gubbins & Gilligan model (1997*a*) is a simple host–parasite model,

$$\left.\begin{aligned}\frac{\mathrm{d}P}{\mathrm{d}t} &= rP\left(1-\frac{P}{\kappa}\right)-bP^mX^n,\\ \frac{\mathrm{d}X}{\mathrm{d}t} &= gbP^mX^n-hX,\end{aligned}\right\} \tag{17}$$

where P is the parasite density and X is the hyperparasite density.

This host–parasite system, (17), always has an extinction equilibrium at $(0,0)$ and a parasite-free equilibrium at $(\kappa,0)$. Local stability of these equilibria is determined by examining the trace and determinant of the Jacobian of the system, J, at the equilibrium. However, for $m>1$ and $n<1$, there is a singularity in J for $X=0$. We can remove this singularity by transforming the system of equations, (17). Let $Y=X^{1-n}$, then system, (17), is transformed to

$$\left.\begin{aligned}\frac{\mathrm{d}P}{\mathrm{d}t} &= rP\left(1-\frac{P}{\kappa}\right)-bP^mY^{n/(1-n)},\\ \frac{\mathrm{d}Y}{\mathrm{d}t} &= (1-n)(bgP^m-hY).\end{aligned}\right\} \tag{18}$$

Examination of the Jacobian at the origin for the transformed system, (18), shows that the origin is a saddle point. Hence, we conclude that the origin is a saddle point in the original system, (17). Note that the parasite-free equilibrium, $(\kappa,0)$ is not a fixed point of the transformed system. Thus, we conclude it must be unstable. This conclusion is supported by numerical simulations of the model.

The system, (17), always has a unique non-trivial equilibrium (i.e. one at which populations are all non-zero) when $m>1$, $n<1$ and local stability analysis proves that the equilibrium is always stable. Moreover, applying Dulac's criterion (see, for example, Glendinning 1994) with weighting $1/P$ to the transformed system, (18), shows there can be no periodic solutions in the positive quadrant. Thus we conclude the non-trivial equilibrium is globally stable for $n<1$ and $m>1$.

(b) *Two-species model*

Provided the parasite basic reproductive number, r_1/d exceeds unity, the two-species model (given as equation (1) in the main body of the text) always has an extinction equilibrium at $(0,0,0)$ and a parasite-free equilibrium at $((r_1-d)\kappa/d,0,0)$. The Lyapunov function, (14), constructed in Appendix 2 can be used to show that the origin is unstable: we can always find a point in the neighbourhood of the origin such that $\mathrm{d}V/\mathrm{d}t>0$; thus, solutions always move away from the origin. Similarly, the Lyapunov function, $V=I+(d+a)X/g$ shows that the parasite-free equilibrium is unstable: there is always a point in the neighbourhood of $((r_1-d)\kappa/d,0,0)$ such that $\mathrm{d}V/\mathrm{d}t>0$; hence, solutions always move away from the parasite-free equilibrium.

Analysis shows that for $m>1$, $n<1$, the two-species model always has a unique non-trivial equilibrium and local stability analysis proves that this equilibrium is always stable. Unfortunately, there are no techniques analogous to Dulac's criterion for proving the non-existence of periodic solutions in third or higher order systems of differential equations. However, we believe that the non-trivial equilibrium is globally asymptotically stable, though we have not proved this. Numerical simulations of the model support the conjecture of global stability.

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Received 12 November 1996; accepted 5 February 1997

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