Plant Pathology (2001) **50**, 154–164

Modelling the progress of light leaf spot (*Pyrenopeziza brassicae*) on winter oilseed rape (*Brassica napus*) in relation to leaf wetness and temperature

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A compartmental model was developed to describe the progress with time of light leaf spot (*Pyrenopeziza brassicae*) on leaves of winter oilseed rape (*Brassica napus*) during the autumn in the UK. Differential equations described the transition between the four compartments: healthy susceptible leaves, infected symptomless leaves, sporulating symptomless leaves and leaves with necrotic light leaf spot lesions, respectively. The model was fitted to data on the progress of light leaf spot on winter oilseed rape at a single site during the autumn of the 1990–1991 season. Model parameters were used to describe rates of leaf appearance, leaf death, infection by airborne ascospores (primary inoculum) and infection by splash-dispersed conidiospores (secondary inoculum). Infection was dependent on sufficient leaf wetness duration. The model also included delay terms for the latent period between infection and sporulation and the incubation period between infection and the appearance of necrotic light leaf spot lesions. This modified *SEIR* model formulation gave a reasonable fit to the experimental data. Sensitivity analysis showed that varying the parameter accounting for the rate of infection by ascospores affected the magnitude of the curves after the start of the epidemic, whilst including a parameter for conidiospore infection improved the fit to the data. Use of ascospore counts from different sites and different years showed variation in spore release patterns sufficient to affect model predictions.

Keywords: ascospores, conidia, disease progress, infectious period, latent period, postinfection period, susceptibility

Introduction

Light leaf spot, caused by the pathogen *Pyrenopeziza brassicae*, is one of the two most important diseases of winter oilseed rape in the UK (Su *et al*., 1998). Yield losses of up to £40 million per annum have been estimated in recent years (Gladders, 1998). Fungicides can be used to control the spread of the disease, but the optimum timing of fungicide application, particularly of the first treatment, is critical and differs between sites and seasons (Sansford *et al*., 1996). This is a consequence of the fact that the severity of light leaf spot epidemics differs between seasons and regions in the UK and between crops within the same region (Fitt *et al*., 1996). Severe epidemics can be controlled only if fungicides are applied in the autumn, before symptoms appear on the plants (Figueroa *et al*., 1994). Understanding the mechanisms of epidemic development early in the season can assist the prediction of the risk of a severe epidemic. Empirical modelling (Welham *et al*., 1999) provides information about light leaf spot risk in relation to weather and crop factors at a regional scale, but cannot provide detailed information about disease progress in individual crops. Little work has been done to model the effects of environmental factors on epidemic progress for individual crops in the UK. Light leaf spot is a polycyclic disease that infects leaves, stems, flowers and pods (McCartney & Lacey, 1990). Two sources of inoculum are involved in the spread of the disease. Epidemics are thought to be initiated in autumn by airborne ascospores of *P. brassicae* produced on debris from crops in the previous season that are deposited on the new crop (Gilles *et al*., 2000b). After primary infection, conidia are produced on leaves and secondary disease spread occurs within and between plants by means of splash-dispersed conidia. Vertical disease spread up the crop canopy in spring may occur both through secondary spore dispersal and through extension of stems with infected meristematic tissue (Paul & Rawlinson,

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Accepted 3 October 2000.

1992). The epidemic can become severe only if suitable weather conditions occur for repeated cycles (generations) of infection, production of conidia and re-infection.

Previous work on light leaf spot has produced information on the effects of temperature and wetness duration on the infection of oilseed rape leaves by *P. brassicae* conidia and the latent period in controlled environments, together with some validation of the results under field conditions (Figueroa *et al*., 1995a, b; Gilles *et al*., 2000a). According to these results, the minimum criterion for infection of leaves at 12°C and 18°C is a wetness duration of at least 16 h. Field experiments showed that the time from infection to sporulation (latent period) generally ranged between 150 and 250 degree-days (accumulated mean daily temperature above zero). This suggested that accumulated temperature might be used to predict the length of the latent period of light leaf spot on winter oilseed rape. Necrotic lesions, which are the symptoms usually visible for assessment in the field, do not appear until much later. The interval from infection to the appearance of necrotic lesions (incubation period) has not been previously determined.

SIR models consist of differential equations describing the rates of change of susceptible (*S*), infected (*I*) and removed (*R*) categories, and have been used by several authors (e.g. Jeger, 1982) to model plant disease epidemics within crops. Gilligan *et al*. (1997) fitted an *SIR* model to stem canker disease of potatoes caused by the soil-borne fungus *Rhizoctonia solani*, but most of the work on leaf and stem diseases has been restricted to a theoretical analysis of the model properties or predictions using given parameter values (e.g. van Oijen, 1992; Jeger *et al*., 1998). Standard four-compartment *SEIR* models define categories for susceptible (*S*), latently infected (*E*), infectious (*I*) and postinfectious (*R*) individuals. This paper describes a modified *SEIR* model, with categories for numbers of susceptible leaves, leaves with latent infection, sporulating leaves (infectious without symptoms), leaves with necrotic light leaf spot lesions (infectious with symptoms) and dead leaves (postinfectious, removed from the epidemic). This framework was used to build a model for the progress of light leaf spot epidemics on individual leaves within a winter oilseed rape crop, based on the life cycle of the pathogen and the influence of environmental factors. The aim of this work was to investigate whether this relatively simple model can predict epidemic progress and to examine, under the assumptions of the model, the relative importance of model inputs and parameter values.

Materials and methods

Experimental data

Data on light leaf spot epidemics were available from experiments at ADAS Bristol, UK, during the autumn of four seasons (1987–1988, 1988–1989, 1989–1990 and 1990–1991). Since full information on disease, weather and ascospore concentrations was available only for 1990–1991, data for the other three years were used only in a limited validation exercise. In each autumn, the percentage of leaves affected by necrotic light leaf spot lesions was recorded on 25 winter oilseed rape plants (cv. Jet Neuf). Single plots (5 m × 5 m) were sown on 26 August 1987, 2 September 1988, 24 August 1989 and 30 August 1990 at a seed rate of 10 kg ha^{-1} with emergence on 7 September for the first two seasons and on 28 August and 6 September for the 1989–1990 and 1990–1991 seasons, respectively. Inoculum of light leaf spot was introduced immediately after sowing by scattering oilseed rape stem debris from a previous crop affected with light leaf spot at a rate of one to three pieces per square metre. Assessments began soon after crop emergence when the first true leaves appeared in mid-September. Each new leaf on each of 25 selected plants was marked as soon as its petiole became visible. The presence or absence of necrotic light leaf spot lesions (Fitt *et al*., 1998) was recorded on each leaf on these plants at approximately weekly intervals during each of the four autumns (until 23 December 1987, 19 December 1988, 4 January 1990 and 19 December 1990). For each of the affected leaves, the percentage leaf area with necrotic lesions was assessed. At the end of each experiment, a complete weekly record of leaves could be constructed for all 25 plants. The data consisted of numbers of 'apparently healthy' leaves (i.e. leaves without necrotic lesions due to light leaf spot infection), numbers of leaves showing necrotic lesions and numbers of dead leaves at each sample date.

Daily meteorological records from the Bristol weather centre, situated approximately 3 km from the experimental site, during the course of the experiments included minimum and maximum temperature (used to estimate mean daily temperature) and total daily rainfall (mm). Periods of rain duration in hours (used as an indication of leaf wetness duration) were also available from Lyneham, approximately 30 km from the experimental site. No closer or more complete records of rain duration have been found. Both rainfall and rain duration were recorded over 24-h intervals calculated from 09·00 hours on the day to which each record refers until 09·00 hours on the following day. However, there were days for which a rainfall record was available but not a corresponding record of its duration, and the mean rain duration value of 10·7 h was used for these days.

In the autumn of the 1990–1991 season, a Burkard volumetric spore sampler was operated at the site of the experiment at ADAS Bristol between 13 September and 19 December to estimate concentrations of airborne *P. brasssicae* ascospores (McCartney & Lacey, 1990). Ascospore concentrations were available from Rothamsted for the seasons 1991–1992, 1992–1993 and 1993–1994. These data were used to investigate the influence of different patterns of spore release on epidemic progress. Data were recorded from 23 September 1991 to 15 July 1992, from 1 October 1992 to 20 July 1993, and from 5 October 1993 to 19 July 1994.

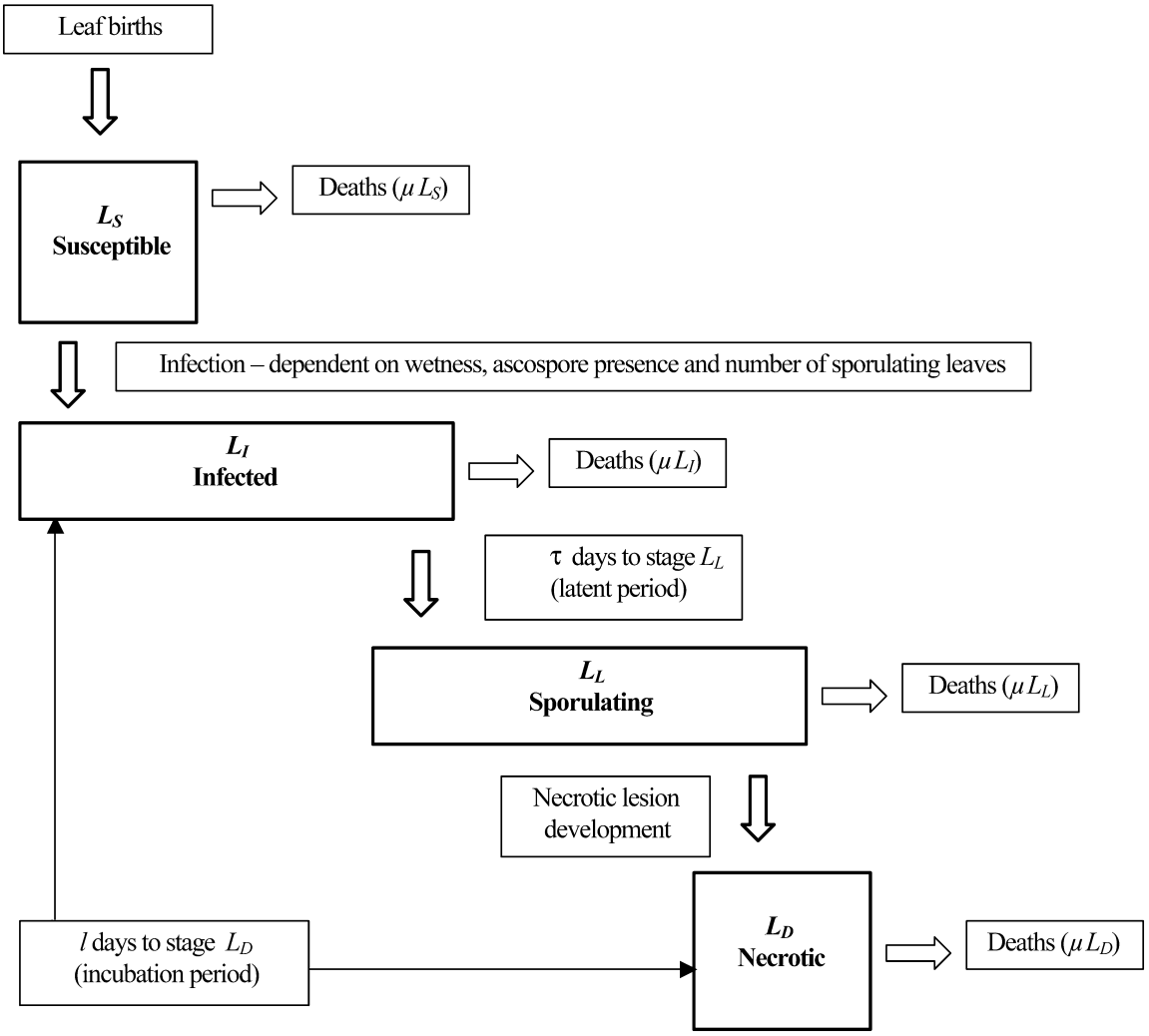

Figure 1 Schematic diagram of light leaf spot model, showing compartments L_S (number of susceptible healthy leaves), L_I (number of infected leaves without sporulation or necrotic light leaf spot lesions), L_L (number of sporulating leaves without necrotic lesions) and L_D (number of sporulating leaves with necrotic lesions) and the transition processes between compartments. Arrows represent input to/output from the compartments.

Model structure

The schematic representation of the model in Fig. 1 illustrates the main stages of the light leaf spot epidemic by assigning leaves to one of five categories, which are the model compartments. These compartments are the number of susceptible leaves (L_S) (i.e. healthy uninfected leaves), number of nonsporulating infected leaves (L_I), number of sporulating leaves with no necrotic lesions (L_L), number of sporulating leaves with necrotic lesions (L_D) and number of removed (dead) leaves (L_R). Plant growth was accounted for in the model by including the production of new leaves. The presence of primary inoculum (ascospores, A) was also included by using data for daily ascospore concentrations for the 1990–1991 experiment.

The model structure can be described as follows. All new leaves start as healthy. If ascospores or conidiospores are present and the infection criterion (16 h rain duration) is satisfied, then a proportion of the healthy leaves become infected and move into the second compartment: latent infection (infected leaves without any symptoms). After time τ (the latent period in days), the infected leaves start sporulating and move into the third compartment: sporulating leaves without lesions. From this time until their death, these leaves produce conidia, the spores that cause secondary infection. After l days from infection, surviving infected leaves start to show necrotic light leaf spot lesions and move into the fourth compartment: sporulating leaves with necrotic lesions. Within each compartment, leaves die with a common probability per day μ and are moved into the final compartment: dead (removed) leaves.

Model equations

$$[dL_S(t)]/dt = [BirthRate] - [InfectionRate] - [DeathRate]_S$$

$$[dL_I(t)]/dt = [InfectionRate] - [RateOfSporeAppearance] - [DeathRate]_I$$

$$[dL_L(t)]/dt = [RateOfSporeAppearance] - [RateOfLesionAppearance] - [DeathRate]_L$$

$$[dL_D(t)]/dt = [RateOfLesionAppearance] - [DeathRate]_D$$

$$[dL_R(t)]/dt = [DeathRate]_S + [DeathRate]_I + [DeathRate]_L + [DeathRate]_D$$

Inspection of the rate of leaf production over time in all four seasons (1987–1988, 1988–1989, 1989–1990 and 1990–1991) indicated that it was related to temperature and could be approximately described as a constant rate in thermal time. Smith & Scarisbrick (1990) also reported leaf appearance rates in autumn in terms of thermal time. Leaf appearance rate was therefore written as

$$[BirthRate] = \gamma T(t)$$

where $T(t)$ was the mean daily temperature and γ was the rate at which new leaves appeared per degree-day.

The rate at which susceptible leaves became infected is described in terms of the infection rate α_t and the number of susceptible leaves on day t, $L_S(t)$, as

$$[InfectionRate] = \alpha_t L_S(t)$$

Successful infection, controlled by the rate of infection α_t, depends on favourable conditions for infection given the presence of infective inoculum. Infective inoculum may occur as airborne ascospores or splash-dispersed conidia. Ascospore numbers are represented by A, the smoothed concentration of ascospores on day t. No measurement of conidia was available, so numbers of conidiospores were taken to be equal to the numbers of sporulating leaves ($L_L + L_D$). Given the presence of spores, the rate of infection depends on the chance of deposition on a leaf followed by germination and infection. The parameters p and q were used as the infection rates for ascospores and conidia, respectively. It was assumed that the success of infection depends only on a sufficient period of wetness, W, namely 16 h (Figueroa *et al.*, 1995b), here represented by rain duration. The infection rate α_t can then be written as

$$\alpha_t = [pA(t) + q(L_L(t) + L_D(t))]I[W(t) \geq 16\,\mathrm{h}]$$

where $I[X]$ is a step function that takes the value 1 when the condition X is true, and is zero otherwise.

The removal of leaves from each compartment due to death was given as the product of the probability of death μ and the number of leaves in each compartment. A single parameter μ was used as the death rate since too few diseased leaves died during the period of the experiment for a separate death rate due to disease to be calculated. Therefore,

$$[DeathRate]_j = \mu L_j(t)$$

where $j = S$, I, L or D for the four compartments. Removed leaves made no further contribution to the epidemic as conidia were thought to form only on living tissue and ascospores were not formed on the dead tissue during the time-scale of this experiment (Gilles, 2000).

In the model, the leaves that started sporulating were the leaves that had been infected τ days ago and were still alive (present on the plants). Thus,

$$[RateOfSporeAppearance] = \alpha_{t-\tau} L_S(t - \tau)\exp(-\mu\tau)$$

where the time-delayed product $[\alpha_{t-\tau} L_S(t - \tau)]$ represented infections that occurred time τ ago and $\exp(-\mu\tau)$ (Gurney & Nisbet, 1998) was the probability that a leaf did not die during the latent period τ. Similarly, the necrotic lesions were allowed to appear on infected leaves l days after infection. Therefore, the leaves that moved into compartment L_D were the leaves that were infected l days ago and were still alive (present on the plants). Thus,

$$[RateOfLesionAppearance] = \alpha_{t-l} L_S(t - l)\exp(-\mu l)$$

where the time-delayed product $[\alpha_{t-l} L_S(t - l)]$ represented infections that occurred l days ago and $\exp(-\mu l)$ was the probability that a leaf did not die during the time period l.

In summary:

$$[BirthRate] = \gamma T(t)$$

$$[InfectionRate] = \alpha_t L_S(t)$$

$$[RateOfSporeAppearance] = \alpha_{t-\tau} L_S(t - \tau)\exp(-\mu\tau)$$

$$[RateOfLesionAppearance] = \alpha_{t-l} L_S(t - l)\exp(-\mu l)$$

$$[DeathRate] = \mu L_j(t), j = S, I, L, D$$

Substituting these terms into the equations gives the full form of the model:

$$[dL_S(t)]/dt = \gamma T(t) - \alpha_t L_S(t) - \mu L_S(t)$$

$$[dL_I(t)]/dt = \alpha_t L_S(t) - \alpha_{t-\tau} L_S(t - \tau)\exp(-\mu\tau) - \mu L_I(t)$$

$$[dL_L(t)]/dt = \alpha_{t-\tau} L_S(t - \tau)\exp(-\mu\tau) - \alpha_{t-l} L_S(t - l)\exp(-\mu l) - \mu L_L(t)$$

$$[dL_D(t)]/dt = \alpha_{t-l} L_S(t - l)\exp(-\mu l) - \mu L_D(t)$$

$$[dL_R(t)]/dt = \mu[L_S(t) + L_I(t) + L_L(t) + L_D(t)]$$

Table 1 Variables and parameters used for modelling the progress of light leaf spot (*Pyrenopeziza brassicae*) on leaves of winter oilseed rape with units and initial values for model parameters as calculated from the data

Variable	Description (units)		
t	Time (day)		
A	Mean daily concentration of ascospores (ascospores m^{-3})		
L_S	Healthy susceptible leaves on day t (number)		
L_I	Infected leaves with no sporulation or lesions (number)		
L_L	Sporulating leaves with no lesions (number)		
L_D	Sporulating leaves with lesions (number)		
T	Mean daily temperature[a] (°C)		
W	Daily rain duration[a] (h)		
Parameter	**Description (units)**	**Initial value**	**(SE)**
γ	Rate of birth of new leaves (number degree-day^{-1})	0·248	(0·042)
μ	Probability per day of leaf death (day^{-1})	0·01	(0·002)
p	Ascospore infection rate (day^{-1} (ascospore $m^{-3})^{-1}$)	0·183	(0·08)
q	Conidiospore infection rate (day^{-1} $number^{-1}$)	0·183	(0.08)
τ	Time from infection to sporulation (latent period) (day)	16	
l	Time from infection to lesion development (incubation period) (day)	60	

[a]Measured between 09·00 hours on one day and 09·00 hours on next day.

Model fitting

The model was fitted to the numbers of leaves with and without necrotic lesions on every plant. The number of apparently healthy leaves, i.e. the number of live leaves without necrotic light leaf spot lesions, corresponds to the total $L_S + L_I + L_L$. The number of leaves with necrotic lesions corresponds to L_D. The numbers of dead leaves were also calculated from the experimental data, but since dead leaf tissue was considered irrelevant to the progress of the epidemic during autumn and winter and the larger numbers meant that dead leaves could dominate the fit, these data were not used in the model fitting.

Model fitting and sensitivity analysis of the fitted model were performed using the software package ModelMaker (Anonymous, 1997) employing the Runge–Kutta method to solve the differential equations numerically. Model parameters were estimated using the Marquardt optimization algorithm to minimize the weighted residual sum of squares. Weights were used to give data points in the disease progress curve 10 times the weight of the other data points. Since the number of all healthy leaves was approximately 10 times the number of diseased leaves, this weighting was intended to give the healthy and diseased data points equal importance in the fitting process.

In order to optimize fitting the model to the data, initial parameter estimates were required. The model had six parameters: γ, μ, p, q, l and τ (Table 1). The parameters γ and μ were calculated using the data provided during the experiment. The numbers of new leaves on each day and the intervals (measured in degree-days) between them were used to derive an initial estimate of the number of leaves appearing per degree-day (γ). The parameter μ was calculated from the data on dead leaves as a probability of leaf death per unit time. A sensible initial estimate of p (rate of infection per ascospore) could not be calculated from the data and an initial value was found by scaling up the rate of new infections until plausible predictions were generated. Since there was no information available on q (conidiospore infection rate), the value of p was used as an initial estimate of q.

The parameters τ and l represented the two time delays in the model (the latent period and the incubation period). These were taken as constant and were not optimized during the model fitting procedure. The latent period (τ) was set to 16 days using results from controlled experiments on conditions for infection of winter oilseed rape leaves by conidia (Gilles *et al.*, 2000a) employing the mean daily temperature for the 1990–1991 season (10·1°C) and the criterion for infection of at least 16 h of wetness; the incubation period (l) was set to 60 days as shorter periods (50 or 55 days) were not consistent with the 1990–1991 data.

Sensitivity analysis and validation

Sensitivity analysis for the model parameters γ, μ, p and q was conducted by varying the values of these parameters (0·1–0·5, 0–0·1, 0–2·0 and 0–0·2, respectively) after optimization and examining the effect on the model. This was done by changing the value of one parameter, whilst keeping the optimized values of the remaining parameters. The possible influence of variability in ascospore release patterns on model predictions was investigated by substituting the three ascospore release patterns from Rothamsted into the Bristol 1990–1991 model, using the optimized parameter values.

Although spore counts were not available for the Bristol 1987–1988, 1988–1989 and 1989–1990 seasons, and so the full model could not be fitted or validated on these data, these data sets were used to investigate plausible values for the incubation period. It has been shown (Figueroa *et al.*, 1995b) that the latent

period is approximately constant in thermal time, so it is possible that the same pattern holds for the incubation period. In each season, for the main increase in disease incidence, all candidate rain events were found, which fell 40–70 days earlier. The number of days and degree-days (accumulated temperature above 0°C) from the rain events to the time of disease increase were calculated. Since the disease increase could have occurred at any time between the previous sample date and the sample date when the increase was found, this gave a range of values. The same calculations were made from the rain events identified as initiating infection in the fitted model for 1990–1991 and the corresponding increase in symptom (necrotic lesion) appearance.

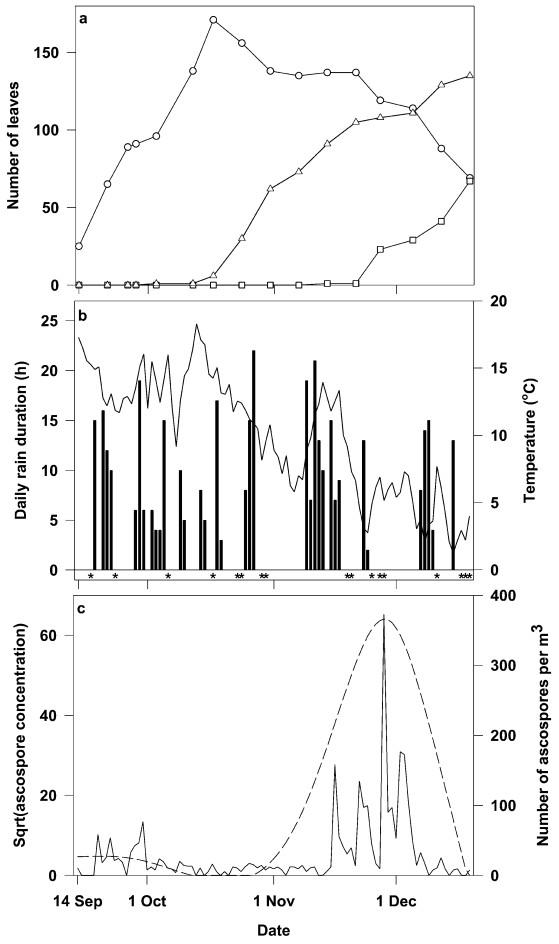

Figure 2 Changes with time in: (a) numbers of winter oilseed rape (cv. Jet Neuf) leaves from 25 plants ('apparently healthy' (○) (healthy susceptible leaves plus infected leaves before appearance of necrotic lesions); showing necrotic light leaf spot lesions (□); dead (△)); (b) daily rain duration (h) and mean daily temperature (°C) observed in Bristol from 14 September (soon after plant emergence) until 19 December 1990 (the asterisks (*) show days for which rainfall was recorded at Bristol but for which there was no corresponding record of rain duration at Lyneham); (c) concentrations of airborne *Pyrenopeziza brassicae* ascospores (full line, number per m^3, square-root-transformed) with cubic spline fitted to the untransformed ascospore concentrations (broken line).

Results

Light leaf spot progress

For 1990–1991, the number of 'apparently healthy' leaves increased until about 45 days after sowing and then started to decrease (Fig. 2a). The decrease was accelerated when necrotic light leaf spot lesions started to appear at around 80 days after sowing. The maximum number of 'apparently healthy' leaves was recorded on 17 October 1990, 48 days after sowing. Only one leaf was found to have necrotic light leaf spot lesions 76 days after sowing and one more was recorded 83 days after sowing (14 and 21 November 1990, respectively). After this, the numbers of leaves with necrotic light leaf spot lesions increased to 23 leaves (16·2% of the total number of live leaves) 89 days after sowing (27 November 1990; Fig. 2a). The mean daily temperature was 17·3°C on 14 September 1990 (date of the first observation) and 4°C on 19 December 1990 (date of last observation). The maximum recorded rainfall was 16·3 mm, which occurred 56 days after sowing, on 25 October 1990. The maximum rain duration of 22 h was recorded 58 days after sowing on 27 October 1990 (Fig. 2b).

Ascospore concentrations

Ascospore concentrations varied from 0 to over 4000 m^{-3}, with only one record being >1000 ascospores m^{-3}. A cubic smoothing spline was fitted to the data (starting from 14 September in order to match the date of the first sample for the 1990–1991 season) to give a representation of the trend of spore release, and this spline was used as input to the model (Fig. 2c). For each set of spore counts from Rothamsted, a section of data was extracted to match the time period of the 1990–1991 season in Bristol (14 September−19 December). Zero was inserted at the start of the series

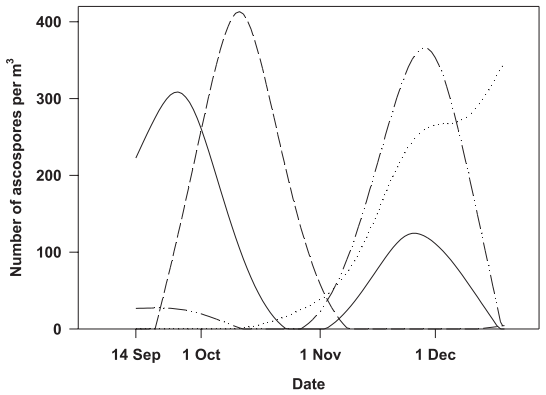

Figure 3 Concentrations of *Pyrenopeziza brassicae* ascospores (number per m^3) fitted by a smoothing spline for Bristol 1990–1991 (–··), Rothamsted 1991–1992 (full line), Rothamsted 1992–1993 (dotted line) and Rothamsted 1993–1994 (broken line).

Table 2 Estimated parameter values obtained by fitting the model for progress of light leaf spot (*Pyrenopeziza brassicae*) on leaves of winter oilseed rape to data for 'apparently healthy' leaves and leaves affected by necrotic light leaf spot lesions, derived from 25 plants of winter oilseed rape, cv. Jet Neuf (Bristol 1990)

Parameter					
γ	μ	p	q	R^2	Degrees of freedom
0·277 (0·03)[a]	0·013 (0·002)	1·239 (0·98)	0·082 (0·087)	0·86	28

[a]Standard error in parentheses.

in order to extend back to 14 September where necessary. A cubic smoothing spline was fitted to each data set to give a representation of the trend of spore release and then scaled to give the same total number of spores as for the 1990–1991 season in Bristol (Fig. 3).

Model fitting

The four parameters γ, μ, p and q were estimated concurrently during the optimization procedure. Optimized values are presented in Table 2. The predicted disease progress curve approximately followed the pattern of the data (Fig. 4). The fitted curve for the 'apparently healthy' leaves followed the pattern of the data better after 70 days from sowing than before this time. The optimized values for leaf birth and death rates were close to their initial values (Table 2). The pattern of the fitted disease progress curve could be associated with the patterns of rain events and the presence of inoculum at the start of the incubation period. The three incremental steps in the disease progress curve were related to rain events 60 days before lesion appearance, i.e. 21, 30 and 49 days after sowing. The optimized

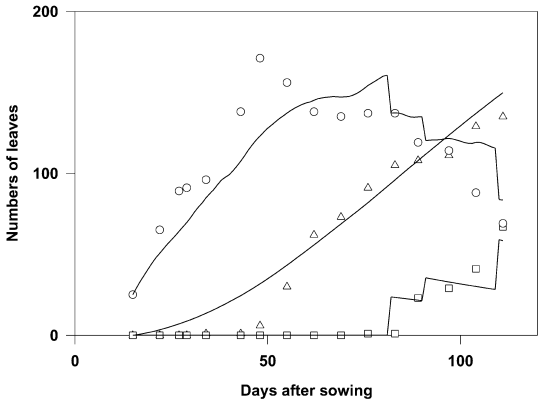

Figure 4 Lines fitted by the delay-differential equation model for the progress of light leaf spot (*Pyrenopeziza brassicae*) on leaves of winter oilseed rape (cv. Jet Neuf) in relation to disease progress observed at ADAS Bristol in autumn 1990. Observed data were the number of symptomless leaves (○, healthy leaves plus infected leaves without necrotic light leaf spot lesions), the number of leaves with necrotic light leaf spot lesions (□) and the number of dead leaves (△).

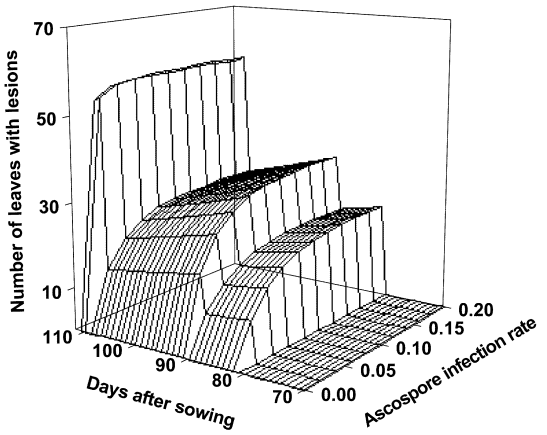

Figure 5 Sensitivity of the fitted model predictions for the number of winter oilseed rape leaves with necrotic light leaf spot (*P. brassicae*) lesions to the ascospore infection rate p when it was varied between 0 and 0·2. Remaining parameter values: $\gamma = 0{\cdot}277$, $\mu = 0{\cdot}013$, $q = 0{\cdot}082$, $\tau = 16$ and $l = 60$.

ascospore infection rate p was 15 times the optimized conidiospore infection rate q. However, since the ascospore infection rate is scaled relative to spore trap estimates and the conidial infection rate is scaled relative to numbers of sporulating leaves (without and with necrotic light leaf spot lesions), these values are not directly comparable. Both estimates are small compared with their standard error. This reflects uncertainty in the estimates, rather than indicating that the parameters can be omitted from the model, as shown in sensitivity analysis later.

Sensitivity analysis

The sensitivity of the model to changes in the parameter values was examined. The number of leaves with necrotic light leaf spot lesions increased linearly as the birth rate of leaves per degree-day (γ) increased, due to the increase in the number of susceptible leaves. Increasing the probability of leaf death (μ) gave an exponential decrease in the number of leaves with necrotic lesions, since the death parameter was common for all leaf categories, leading to a decrease in the number of susceptible leaves and a corresponding greater number of dead leaves with necrotic light leaf spot lesions. In both cases, the proportion of infected leaves was unaffected by changes to the birth and death parameters.

As the ascospore infection rate (p) increased, the number of leaves with necrotic lesions increased exponentially to a maximum when $p = 0{\cdot}2$ (Fig. 5). No further increase occurred if p was increased further as all leaves present at each infection period had become infected. When the conidiospore infection rate (q) was varied (Fig. 6), only the last predicted value of the number of leaves with necrotic lesions changed (111 days after sowing). When $q = 0$, the last observation point

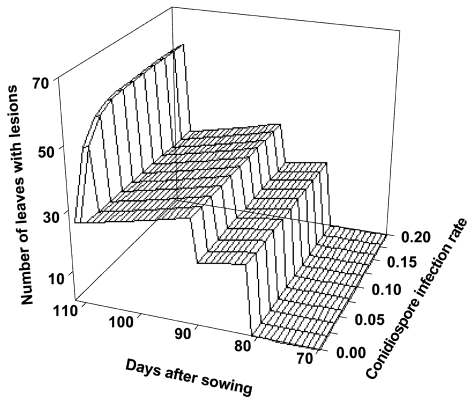

Figure 6 Sensitivity of the fitted model predictions for the number of winter oilseed rape leaves with necrotic light leaf spot (*P. brassicae*) lesions to the conidiospore infection rate q when it was varied between 0 and 0·2. Remaining parameter values: $\gamma = 0{\cdot}277$, $\mu = 0{\cdot}013$, $p = 1{\cdot}239$, $\tau = 16$ and $l = 60$.

was not estimated well as there was no predicted disease increase at this point. The fit of the model to the data was improved when secondary infection was included in the model ($q > 0$), and the number of leaves with lesions at the last sample date increased exponentially as q increased, reaching a maximum around $q = 0{\cdot}10$.

Ascospore release patterns

When the fitted model was run using the ascospore patterns of 1991–1992 and 1993–1994 from Rothamsted, the behaviour of the model did not change. However, when the ascospore data for Rothamsted 1992–1993 were used as input to the model, only the last data point was predicted by the disease progress curve (Fig. 7). This was due to the absence of ascospores at the start of the season (Fig. 3). In all three cases, when no assumption of secondary infection was made (by making the parameter q equal to zero), the last data point of the disease progress curve was still well predicted because ascospores were present 60 days earlier. In each case lesion appearance could be explained in terms of primary infection alone.

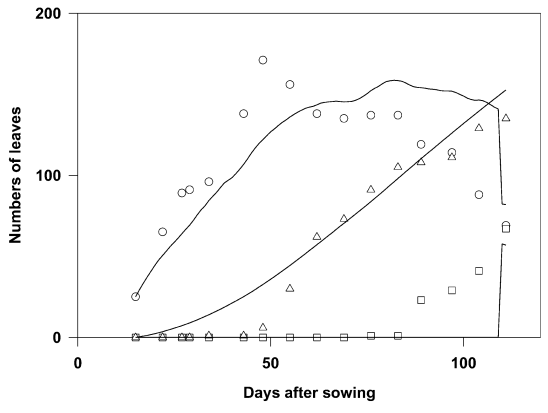

Figure 7 Lines produced by the fitted delay-differential equation model for the progress of light leaf spot (*Pyrenopeziza brassicae*) on leaves of winter oilseed rape (cv. Jet Neuf) in relation to disease progress observed at ADAS Bristol in autumn 1990 and with the ascospore concentration pattern from the Rothamsted 1992–1993 season. Observed data were the number of symptomless leaves (○, healthy leaves plus infected leaves, with or without sporulation, without necrotic light leaf spot lesions), the number of leaves with necrotic light leaf spot lesions (□) and the number of dead leaves (△).

Investigation of the incubation period using other data sets

Disease progress curves from ADAS Bristol in 1987–1988, 1988–1989 and 1989–1990 are shown with mean daily temperature and rainfall in Fig. 8. The first samples indicating a significant increase in diseased leaves (i.e. with necrotic light leaf spot lesions) were 91, 73 and 91 days after sowing in seasons 1987–1988, 1988–1989 and 1989–1990, respectively. Calculation of the number of days and accumulated temperature (degree-days above zero) between candidate rain events (where infection might have occurred) and date of disease increase gave a wide range of values. In 1988–1989, the presence of only one rain event suggested an incubation period between 550 and 650 degree-days or 47–53 days (Table 3). However, for the 1990–1991 season, incubation periods shorter than 60 days did not give a good fit to the data, and calculation of the accumulated temperature between the time of the rain events that led to infection (21 and 30 days after sowing, infection due to ascospores) and the time of the corresponding incremental steps in the disease progress curve 60 days later (Fig. 4) suggested an incubation period between 650 and 750 degree-days (Table 3). The figures in Table 3 suggest that there was no single value for the incubation period measured in time or thermal time that would be consistent with the data from all seasons as long as rain events are used as the only indicators of infection periods.

Discussion

It was possible to describe the progress of a light leaf spot (*P. brassicae*) epidemic on winter oilseed rape using a set of differential and delay-differential equations based on an *SEIR* form. Standard four-compartment *SEIR* models define categories for susceptible (S), latently infected (E), infectious (I) and postinfectious (R) individuals. In this model, it was necessary to split the infectious category into two components: sporulating symptomless leaves (L_L) and sporulating leaves with necrotic lesions (L_I). The removed compartment (postinfectious leaves) contained the dead leaves (L_R). It was assumed that, once a leaf had started sporulating, it would continue to sporulate until it died and then stop, since sporulation was not expected on dead tissue. It was also assumed that the time was insufficient for the production of ascospores on dead tissue (Gilles, 2000).

The majority of *SEIR* models used in plant pathology

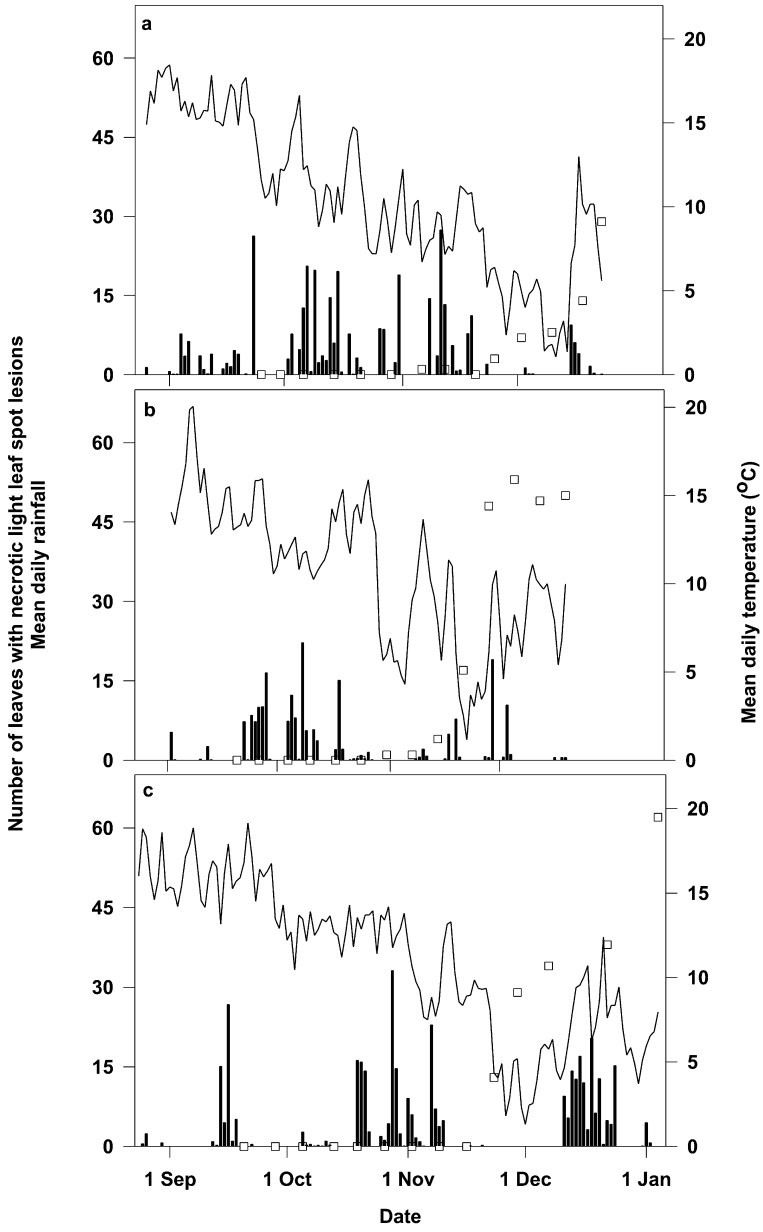

Figure 8 Daily rainfall (mm), mean daily temperature (°C) and number of leaves with necrotic light leaf spot lesions (□) observed at ADAS Bristol (a) from 26 August until 23 December 1987, (b) from 2 September until 19 December 1988, and (c) from 24 August 1989 until 4 January 1990, during the seasons 1987–1988, 1988–1989 and 1989–1990, respectively.

have assumed that the host population remains constant during the epidemic, although Gilligan *et al.* (1997) allowed for the production of new stems when modelling a stem canker epidemic in potatoes. This model for light leaf spot allowed for both production of new oilseed rape leaves and death of leaves due to natural senescence. The inclusion of leaf birth and leaf death in a model for light leaf spot epidemics on oilseed rape is important because, in many epidemics, the observed disease incidence decreases over time due to the changing population of leaves (e.g. figs 1 and 2 in Figueroa *et al.*, 1994); this might lead to poor estimates of disease risk if not properly accounted for in the model.

The model predicts satisfactorily the general trends in all leaf categories. It predicts well the timing of the start of the epidemic and, by including both primary and secondary infections, it gives a reasonable prediction of the times when disease incidence increased greatly. For the 1990–1991 data, the model could not predict the final increase in diseased leaves without including a secondary infection cycle. However, because secondary infection started relatively late in the experimental period, further data sets are required to investigate the relative importance of the primary and secondary infections. Data sets over periods of more than 3 months are also required to assess whether this model can adequately describe light leaf spot progress during a winter oilseed rape growing season. The sets of ascospore counts from different seasons show that some variation exists in the timing of ascospore release in the autumn. Clearly, if the assumption in the model is

Table 3 Accumulated time (days) or thermal time (degree-days above zero) between observed increase in leaves with necrotic light leaf spot lesions and earlier candidate rain events for infection for the 1987–1988, 1988–1989 and 1989–1990 seasons. Maximum value is accumulated time/thermal time until observed increase, minimum value is accumulated time/thermal time until first day after previous sample.

Season	Candidate rain events (days after sowing)	Degree-days to necrotic lesion appearance Min	Max	Days to necrotic lesion appearance Min	Max
1987–1988					
	23	694	738	62	68
	28	612	656	57	63
	37	504	548	48	54
1988–1989					
	20	550	615	47	53
1989–1990					
	23	805	855	62	68
	25	771	822	60	66
	42	517	567	43	49
1990–1991					
	21	738[a]		60[a]	
	30	673[a]		60[a]	

[a]Values for 1990–1991 season are accumulated time/thermal time between date of infection and date of disease increase as predicted by the model

correct, that ascospore infection is the critical factor driving epidemic progress, then ascospore counts (or a good model of ascospore counts) will be required to accurately predict the timing of epidemics.

Many simplifying assumptions were made in the construction of the model. The assumption of a common death rate for diseased and healthy leaves could not be tested on this short data set, but could be an important factor influencing epidemic progress in data sets over longer periods of time. Another assumption was that temperature did not affect the rate of infection, latent or incubation periods. Figueroa *et al*. (1995b) showed that the latent period was approximately constant in terms of thermal time, with a distribution approximately symmetrical about the mean latent period. Blythe & Anderson (1988) have shown how to incorporate different distributions for delays in these models, which might be expected to give smoother increases in disease than the discrete jumps predicted by the current model. Gilles *et al*. (2000a) have recently shown that the infection criteria for *P. brassicae* conidia in terms of leaf wetness duration change with temperature and that the latent period may also be dependent on both factors. These results could be used to develop more appropriate forms for the rate of infection and latent period. However, no further information has become available on the length of the incubation period (time from infection to appearance of necrotic lesions). There is some uncertainty about the possible length of the incubation period in the seasons before 1990–1991 due to the lack of data on ascospore numbers. Even taking this into account, there appears to be some inconsistency in the possible incubation periods calculated in the different seasons, whether considered in time or thermal time. It is possible that other infection periods associated with dew rather than rain duration occurred, or that the incubation period depends on factors other than temperature, or that the time of appearance of necrotic light leaf spot lesions depends on other factors, such as frost or plant vigour. This critical stage of the epidemic clearly requires further investigation.

The use of rain duration as an indicator of leaf wetness duration was unsatisfactory, as it is likely to underestimate leaf wetness duration and is not readily available from standard sources. Only an incomplete record could be obtained for these experiments. Over the autumn and winter periods, dew accounts for many hours of leaf wetness in oilseed rape crops and, in combination with rain duration, might give a better estimate of leaf wetness duration. However, these data are also not readily available from standard sources and were not available for these experiments. Given sufficient leaf wetness, rain should not be necessary for ascospore or conidial infection once the spore has landed on a leaf. However, rain is required for the splash dispersal of conidia from sporulating leaves to cause new infection and these mechanisms should be reflected in the model.

Another assumption in the model was that infection criteria were the same for airborne *P. brassicae* ascospores and for splash-dispersed conidiospores. The model can easily be extended to take account of more specific information on the influence of wetness and temperature on infection criteria and rates as it becomes available. There is now some evidence that ascospores are considerably more infective than conidiospores (Gilles & Fitt, 1999). The efficacy of splash dispersal of conidia will depend on the characteristics of rain events. Previous work on splash-dispersed cereal diseases (Shaw, 1991; Lovell *et al*., 1997, 1999) indicates methods for incorporating this information into models.

The present model provides a reasonably realistic description of the light leaf spot epidemic as it is

currently understood, and can be modified to take account of further biological information as it becomes available. This gives a framework within which the influence of different factors affecting the epidemic progress can be investigated.

Acknowledgements

We thank the Biotechnology and Biological Sciences Research Council and the UK Ministry of Agriculture, Fisheries and Food for financial support, Frank van den Bosch and Robin Thompson for their insight and guidance of the work, Lauriano Figueroa and Gill Murray for the Rothamsted ascospore data and Alastair McCartney for help with the interpretation of the results.

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