Potential benefits of crop-specific models for forecasting light leaf spot severity

A. BAIERL (1), N. EVANS (1) B. D. L. FITT (1) & P. GLADDERS (2)

1. Rothamsted Research, Harpenden, Herts. AL5 2JQ, UK

(2)ADAS Boxworth, Cambridge CB3 8NN, UK

Introduction

Seasonal yield losses caused by light leaf spot (*Pyrenopeziza brassicae*) were estimated to range from £13M to £40M in the UK over harvest years 1987 to 2001 (Fitt *et al.*, 1997; CSL, www.csl.gov.uk), making light leaf spot one of the two major diseases on winter oilseed rape. However, the severity of light leaf spot differs between seasons, between different regions of the UK and between crops within a region (Gilles *et al.*, 2000a). For effective control of light leaf spot, fungicides need to be applied in autumn (Figueroa *et al.*, 1994). An empirical forecasting system for light leaf spot predicts light leaf spot incidence in spring on a regional level, using monthly temperature, rainfall and previous seasons light leaf spot incidence as inputs (http://www3.res.bbsrc.ac.uk/leafspot/). Crop-specific elements have been incorporated into the interactive web-based version of this forecasting scheme so that growers can input information about their own cultivars and sowing date to modify the forecasts (Souter *et al.*, 1999; Evans *et al.*, 2002). Furthermore, the initial forecasts made in September/October are updated in early March by including observed (as opposed to predicted) winter weather (Souter *et al.*, 1999). However, these forecasts cannot be operated in 'real time' because they are based on empirical rather than mechanistic models. Various relationships were investigated recently (Papastamati *et al.*, 2000; Welham *et al.*,2000; Gilles *et al.*, 2000b, 2001c) that describe the progress of light leaf spot at a crop-specific level. This paper describes recently developed crop-specific models, and evaluates their potential benefits for forecasting light leaf spot severity.

Models

In relation to the role of ascospores and conidia of *Pyrenopeziza brassicae* in light leaf spot epidemics (Gilles *et al.*, 2001a), models have been developed to describe specific stages in epidemic development. These include the effects of temperature on the development of apothecia of *Pyrenopeziza brassicae* (Gilles *et al.*, 2001a), the effects of temperature and wetness duration on development of light leaf spot on inoculated plants (Gilles *et al.*, 2000b), and the effects of temperature on the latent period (Gilles *et al.*, 2001c). These models have been combined in work to predict the timing of stages in the development of light leaf spot epidemics as part of a spatio-temporal investigation using detailed assessments on micro-plots at

Rothamsted by Evans *et al.* (2003). For two seasons, in which neighbouring plots were inoculated with debris infected by *P.brassicae*, the progress (cycles) of the light leaf spot epidemics were predicted from meteorological data using models developed previously at Rothamsted (Gilles *et al.*, 2001c). There was no oilseed rape stem debris on the micro-plot experimental area (previous crops: Great Knott I [1998/99], spring barley - 1998, winter oats - 1997; Great Knott II [1999/2000], set-aside [fallow] - 1999, winter peas - 1998). It was assumed that epidemics were initiated by ascospores produced on infected stems scattered on the adjacent large fungicide experiment (i.e. this local inoculum source was more important than any background inoculum from more distant sources). As the debris had been stored dry in a barn until the field was inoculated and meteorological data were available, the time *(t)* from inoculation until 50% of the maximum number of *P. brassicae* -- apothecia matured was estimated using the equation of Gilles *et al*. (2000a):

$t\,(T) = 7.6+55.8(0.839)^{T}$

where T = temperature (recorded hourly) during periods when the debris was wet (estimated with a leaf wetness sensor).
1/(24t (T)) was the contribution to the maturation process of a period of 1 hour at temperature T when the debris was wet. The hourly progress of maturation (depending on T)$T$was added until the sum equalled 1. The time until maturation was then the total number of hours from inoculation needed to reach a value of 1. Ascospore release was considered to take place over a period of 3-4 days following 50% maturation. Wind data (direction and wind run) were analysed to predict the predominant direction of dispersal of the air-borne ascospores.

Conditions for infection by *P. brassicae* ascospores were described by an equation developed to predict maximum percentage leaf area with *P. brassicae* sporulation (c) (Gilles *et al.*, 2001c), which depends on temperature (T) band daily hours of leaf wetness (W) at the time of infection:

$$c(T,W)=(3.65+7.02T-0.3T^2)\exp(-\exp(-0.15(W-(55.47-6.08T+0.21T^2)))) \text{ for } W\geq 6 \quad [2]$$

$c(T,W) = 0$ for $W<6$

After infection, visible sporulation was predicted after a latent period described as a function of temperature recorded hourly during this period by the equation (Gilles *et al.*, 2001c):

$$l(T)=48.0-3.87T+0.11T^2 \quad [3]$$

The equations used to calculate infection criteria and latent period were developed from data for light leaf spot infection by conidia (Gilles *et al.*, 2000a, 2001c). However, Karolewski *et al*. (2002) recently demonstrated that the infection criteria and latent period for ascospores were similar to those for conidia at a range of

temperatures (8, 12, 16 and 20◦C) and leaf wetness durations (6, 10, 16, 24, 48 and 72 h). In both seasons, the dates when the first sporulation of *P. brassicae* was expected were predicted from weather data, using equation 1 (first predicted ascospore release), 2 (first dates when infection conditions occurred after ascospore release) and 3 (latent period after first predicted infections).

Rain-splash dispersion of conidia was expected to cause the next "infection events". Rain splash was considered to occur on days with recorded rainfall exceeding 1 mm. Dates when the first secondary sporulation of *P. brassicae* was expected were predicted from predicted first dates for secondary inoculum dispersal (first rainfall after predicted sporulation), using the latent period equation (3). To compare disease development in time and space predicted from meteorological data with observed epidemic progress in micro-plots, maps indicating the amount of light leaf spot on each tagged plant in each plot were drawn for each assessment date in the two seasons. Increase in disease in plots that were already infected was considered to be caused by splash-dispersed conidia. New light leaf spot infections on previously unaffected plots were assumed to be caused either by incoming air-borne ascospores or through splash-dispersed conidia from infected neighbouring plots.

These models have also been incorporated into mechanistic models of the progress of light leaf spot in relation to initial ascospore concentrations and weather factors by Papastamati *et al.* (2000).

Results

By inserting observed hourly temperatures (during periods of wetness) into equation 1, it was predicted that apothecia (ascospores) would mature on stem debris inoculum by 18 December 1998, 42 days after inoculation and by 12 November 1999, 31 days after inoculation in the 1998/99 and 1999/2000 seasons, respectively (Table 1).

Table 1. Comparison between predicted (from weather data, using empirical equations) and observed development of light leaf spot (*P. brassicae*) epidemics on oilseed rape at Rothamsted in 1998/99 and 1999/2000

	1998/99		1999/2000	
	predicted[a]	observed[b]	predicted[a]	observed[b]
Plots inoculated	-	6 Nov 98	-	12 Oct 98
First ascospore release (equ 1)	18 Dec 98	-	12 Nov 99	-
Infection criteria fulfilled (equ 2)	-	after 21 Dec 98	-	after 12 Nov 99
Latent period	28 days	-	24 days	-
First sporulation (conidia)	19 Jan 99	1 Feb 99	6 Dec 99	6 Dec 99
Rain splash events	-	19,20,23,25,26 Jan 99	-	6,8,10 Dec 99
Latent period	30 days	-	37 days	-
First secondary sporulation	23 Feb 99	2 Mar 99	15 Jan 00	17 Jan 00

[a] Predictions were approximate using meteorological data available.

[b] Observations were made every two weeks during the growing season from 7 December 1998 until 15 March 1999 and 18 October 1999 to 16 March 2000.

In 1998/99, on the days immediately after the first predicted ascospore release (18 December), temperatures were below 4ºC and leaves were dry during daytime. Therefore, little disease progress was expected because infection criteria were not fulfilled (see equation 2). From 21 December onwards, conditions were more favourable for infection, with temperatures above 8ºC and leaf wetness during the day. *P. brassicae* sporulation on leaves, the first visible sign of the epidemic, was expected after a latent period (equation 3) of 28 days (19 Jan 1999). In 1999/2000, during the period following the first predicted ascospore release (12 November 2000), temperatures were between 5 and 10ºC and leaf wetness duration was generally short. According to equation 2, low incidences of primary infection were predicted. Sporulation was expected after a latent period of 24 days (6 December 1999). These predictions fitted with dates of the field observations of the first sporulation in both seasons. In 1998/99, light leaf spot was first observed on 1 February 1999 on four different plants. In 1999/2000, the first two plants with light leaf spot were observed on 6 December 1999.

During both seasons, rain events to disperse conidia occurred frequently after the predicted date for sporulation (e.g. 19, 20, 23, 25, 26 January 1999 in 1989/99 and 6, 8, 10 December 1999 in 1999/2000). Thus, it was predicted that secondary

infections would produce new sporulation after further latent periods (equation 3) on about 23 February 1999 and 15 January 2000, respectively. Disease assessments from 2 March 1999 showed there had been a rapid increase in disease incidence, especially on eastern plots. In 1999/2000, no additional plants with sporulation were observed until 4 January 2000. By 17 January 2000, there were increases in disease incidence on plants in close proximity to previously infected plants. Increased incidence of light leaf spot near to sporulating plants was observed in the following assessments (after another latent period). Additionally, the disease had spread to plots that had not already been affected.

These results suggest the relationship between light leaf spot development and weather factors can be evaluated on most fields as local weather stations are usually available. Ideally, hourly readings of temperature, rainfall and leaf wetness (through a wetness sensor) are required. In Figure 2, predicted maximum values for percentage leaf area with *P.brassicae* sporulation are plotted for each day between early October and mid-February in the 1998/99 growing season. These predictions are the expected development of light leaf spot disease, given the temperature/wetness conditions on each day and assuming that inoculum is available. Figure 3 shows for each day the number of potential infection events (predicted percentage leaf area with sporulation greater than 10%) that are not yet visible (e.g. that occurred within one latent period before that day).

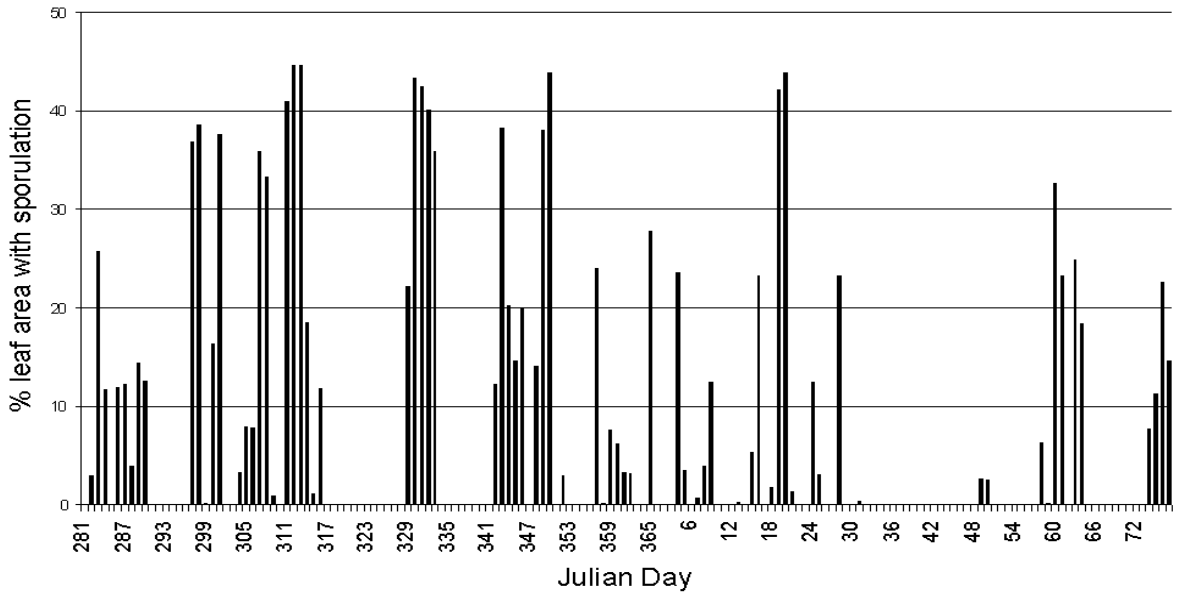

Figure 2: Predicted percentage leaf area with *Pyrenopeziza brassicae* sporulation which results from temperature/wetness conditions on each day, calculated using equation (2) for each day between October 1998 and mid February 1999 at Rothamsted.

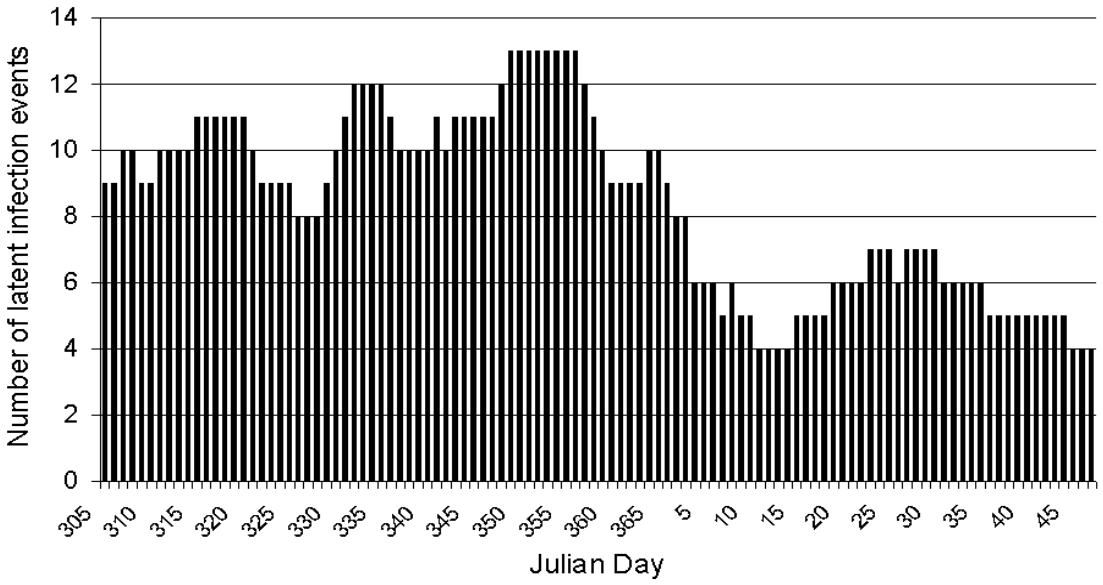

Figure 3: Number of potential infection events (predicted percentage leaf area with sporulation greater than 10%) that occurred within one latent period before the corresponding Julian day.

The location of the inoculum source and corresponding wind conditions greatly influence the amount of initial infection (Evans *et al.* (2003), Figure 4). The individual field situation can be assessed using past records and experience of the grower. Secondly, secondary spread of light leaf spot through splash dispersion of conidia causes significant aggregation of the disease (Evans *et al.*, 2003). This has a consequence for the protocol for sampling crops to estimate disease incidence. To estimate disease incidence with the same accuracy as for randomly distributed disease, larger numbers of small samples are required.

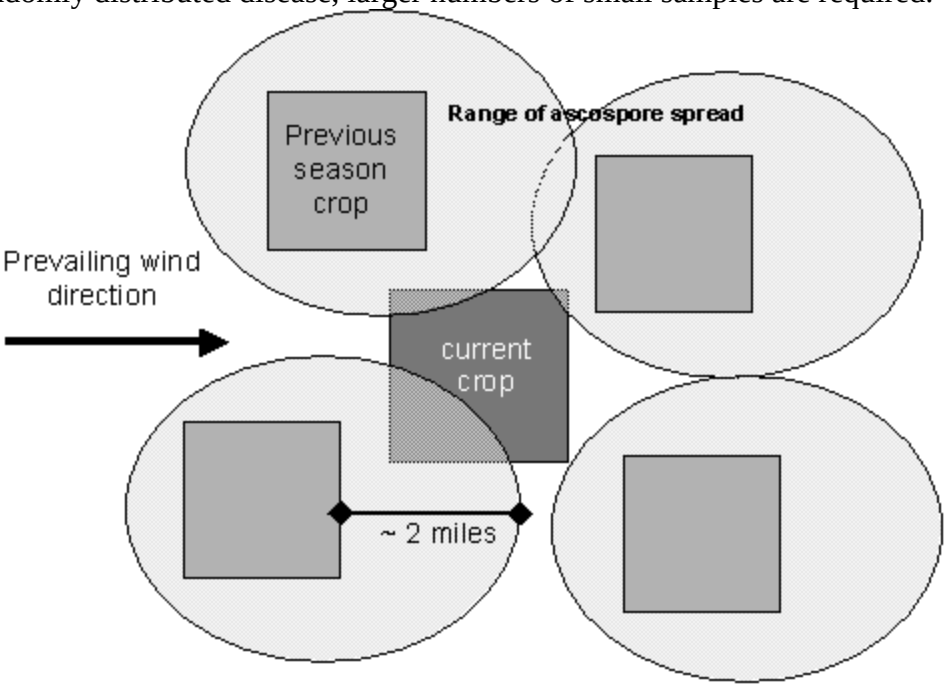

Figure 4: Impact of the location of the inoculum source and corresponding wind conditions on the amount of initial infection from ascospores of *Pyrenopeziza brassicae* (light leaf spot) on winter oilseed rape (schematic representation).

Discussion

The continuous output from the mechanistic crop-specific model means that it is more flexible than the empirical model, with its discrete output. The empirical model gives a point estimate of light leaf spot incidence in spring (GS 2,0-3,3), whereas the mechanistic crop-specific model predicts the disease progress curve through the whole time period. This continuous input and output means that interactions between variables can be dealt with explicitly, whereas in the empirical model, interactions between variables and the timing of events are difficult to include. For example, the interaction between rainfall and numbers of conidia affects the amount of secondary disease spread and is modelled directly by the mechanistic model. However, although the empirical model uses both winter rainfall and the results of the autumn survey, these summary values cannot reflect the interaction that takes place. Similarly, whilst the empirical model modifies its prediction in response to autumn fungicide use in high risk districts, it cannot take any account of the fungicide timing. However, the mechanistic crop-specific model can be extended to model the effect of fungicide application on the disease progress curve, as a reduction in infection efficiency, and effects of different fungicide timings on the epidemic can be compared.

The mechanistic crop-specific model requires daily measurements of mean temperature, rainfall duration and ascospore numbers. Daily measurements of mean temperature could be provided either by an on-site meteorological station, or from a local weather station, but rainfall duration is not a standard measurement. However, Gilles *et al*. (2000) showed that wetness duration has a major influence on the number of lesions established by conidial infections, so it is unlikely that this can be satisfactorily substituted by more standard measurements, such as total rainfall, which do not give a good approximation to rainfall duration. Models for the development of apothecia require a defined starting point for the maturation process, which, under experimental conditions, is provided by the date sources of inoculum are put out on the crop field. For a "commercial" crop, this date is not obvious. One possibility would be to use the harvest date of the previous crop, but because of crop rotation and differences in harvest dates between crops this cannot be applied straight forwardly. The crop-specific model (Papastamati *et al.*, 2000) requirement for regular ascospore counts in autumn is a problem. There are considerable local differences in inoculum sources, so measurements made at regional centres (e.g. ADAS sites) are unlikely to provide accurate information about inoculum on a specific crop.

Thus the two models offer different but complementary approaches to assessment of disease risk. The empirical model can give an initial assessment of regional and crop risk, which can be fed into the simulation model to indicate likely ascospore patterns. If only summary weather information is available, the empirical model can still provide a prediction of spring light leaf spot incidence, together with an indication of uncertainty in the estimate. In both cases, because of high local

variation in crop risk, crop sampling is advised to establish local disease incidence. If detailed weather data are available, the mechanistic model can be run and compared with disease data to establish the likely ascospore input. It can then be run into the future to predict risk of severe epidemics for typical weather scenarios, or to investigate the effects of fungicide application and timing. A combination of the two modelling approaches can be used to provide users with as much information as possible to guide their decision-making.

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