Development and validation of a model to describe the progress of phoma stem canker epidemics in England and France

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Abstract

Leptosphaeria maculans causes phoma stem canker, the most serious disease on winter oilseed rape in Europe. This paper reports the validation of a mechanistic model, designed to describe the progress of phoma stem canker epidemics, as they go through different stages: ascospore production, leaf infection, phoma leaf spot development, growth of the pathogen down the leaf petiole and stem canker development. Severity of epidemics differs between seasons, between regions and between individual farms, depending on weather and agronomic factors. Therefore, a wide range of independent data (not used to construct the model), originating from Rothamsted (2003-2004 and 2004-2005 seasons, incorporating different cultivars) and Boxworth (ADAS, 2003-2004 and 2000-2001 seasons incorporating different seed rates and sowing dates) in the UK and Thiverval-Grignon (2003-2004 season) in France were used to validate the model. From a total of 14 data sets the model fitted well to 9 data sets.

Key words: *Leptosphaeria maculans*, *Phoma lingam*, epidemiology, oilseed rape, Model Maker.

Introduction

Phoma stem canker, caused by *Leptosphaeria maculans*, is the most serious disease on winter oilseed rape in Europe, but the severity of epidemics differs between seasons, between regions and between individual farms, depending on weather and agronomic factors (Fitt et al., 1997; Gladders and Symonds, 1995 ; West et al., 1999; West et al., 2000; West et al., 2002; Zhou et al., 1999). Aspects of different components of the *L. maculans* life cycle, including ascospore release (Gladders and Musa, 1980; Thurwachter et al., 1999; West et al., 2002) and infection conditions (Biddulph et al., 1999; Huang, 2002; Toscano-Underwood et al., 2001), have been reported but little work has been done to model epidemic development. The aim of this work was to develop and validate a mechanistic model of the life cycle, similar to that developed for light leaf spot of winter oilseed rape (Papastamati et al., 2002).

Materials and methods

Model development: The development of phoma leaf spot and phoma stem canker in relation to winter oilseed rape leaf production and death was studied at Rothamsted Research during three consecutive seasons: 2000/01, 2001/02 and 2002/03. During the 2002/03 season, a second wave of seedling emergence meant that the experiment contained small and large plants and these were assessed separately. Data were used to develop a phoma stem canker epidemic model, in the form of a series of delay-differential equations. The "epidemic" is started by the input of data for concentrations of air-borne ascospores, as these are produced on crop debris from the previous season. The model accounts for subsequent production of new leaf area, growth of existing leaf area and senescence of leaf area due to disease and leaf death. The probability that a stem does not get infected by *L. maculans* depended on the rate of phoma growth with time, the probability of survival of the plant tissue during a specific growing period and the probability that the stem was not already infected.

Model fitting: optimised parameters: The model equations were implemented using Model Maker software and Runge-Kutta integration to solve differential equations numerically. The final estimated model parameters were found by fitting the model to data for mean phoma lesion area per plant with time. The Marquardt optimisation algorithm was used to minimise the weighted residual sum of squares, using the standard error of each mean (per plant) phoma lesion area as the weight of each datum (each mean).

Model validation: Individual plant disease progress (leaf tagging) data were collected weekly during the autumn and winter over a number of seasons from 14 sites in England (Rothamsted and Boxworth) or France (Grignon) (Table 1). Data from each site included input data (ascospore numbers and weather) to run the model and output data (disease observations). Data were collated into a database, fitted to the model and assessed for goodness of fit.

Results

Phoma leaf spot progress: *observed data and model fitting for parameter estimation*: The pattern of phoma leaf spot epidemic was very different in the three different seasons (2000/01, 2001/02, 2002/03) (Figure 1). However, model predictions appeared to be acceptable, with the pattern of predicted phoma leaf spotting being similar to that observed in the crops (Figure 1). On average, the greatest phoma lesion area per plant was observed in 2000/01 (Figure 1).

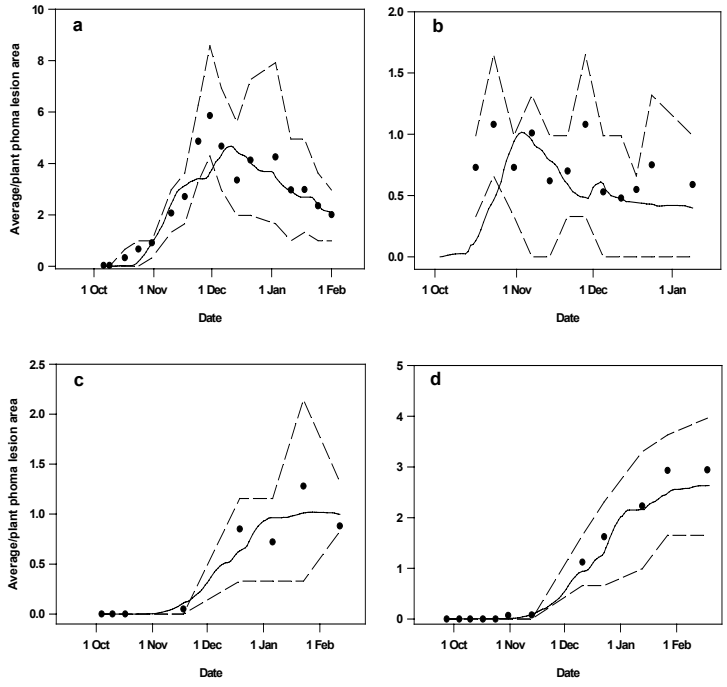

Fig. 1: Average estimated phoma lesion area assessed in winter oilseed rape at Rothamsted during: 2000/01 (a), 2001/02 (b); 2002/03-small plants (c) and 2002/03-large plants (d) with the corresponding 1st and 3rd quartiles (— —). The solid (——) line represents the phoma leaf area for a single oilseed rape plant as estimated by the stem canker model.

Stem canker development: The model predicted the progress of the average phoma stem lesion area per plant satisfactorily as the model estimates followed the observed pattern of lesion area well (Fig. 2). The predicted date that the model suggested that canker should start agreed with the date when cankers of severity score 2 (2000/01 and 2001/02, Fig. 2a and 2b, respectively) or of severity score 1 (2002/03 small and large plants, Fig. 2c and 2d, respectively) were observed in the crop. The greatest value of the estimated probability at the end of each model run agreed best in value with the proportion of plants (stems) with a severity score of at least 4 (2000/01, Fig. 2a), at least 1 or 2 (2001/02, Fig. 2b), 0 (2002/03, small plants, Fig. 2c) and at least 1 (2002/03, large plants, Fig. 2d).

Model validation: Datasets collected over three seasons (2000/01, 2003/04 and 2004/05) at two sites in the UK (Rothamsted and Boxworth) and one in France (Thieverval Grignon) were used to validate the model (Table 1). Goodness of fit of the observed data to that predicted by the model indicated that for 50% of the datasets tested, the goodness of fit had an R^2 value >80%. Examples of the fit of the model to crop data are given in Figure 3.

Discussion

The phoma leaf spot model developed describes epidemic progress with respect to the development of phoma leaf lesions and subsequent stem cankers and provides a useful tool for modelling epidemic progress. Generally, predictions are good and this reflects the fact that the model concentrates on the early infection processes in the autumn when plants are at the rosette stage (Huang et al., 2005; Thurwachter et al., 1999). One disadvantage of the model is that ascopore counts are used as an input to initiate predictions and this requires the need for a spore trap and the expertise to process samples and count spores on a regular basis. However, it may be possible to incorporate, or use output from, an ascospore prediction model. For example, an empirical model for *L. maculans* has been developed for Australian conditions (Salam et al., 2003) and it may be possible to adapt such a model to produce accurate predictions of ascopore release under UK conditions.

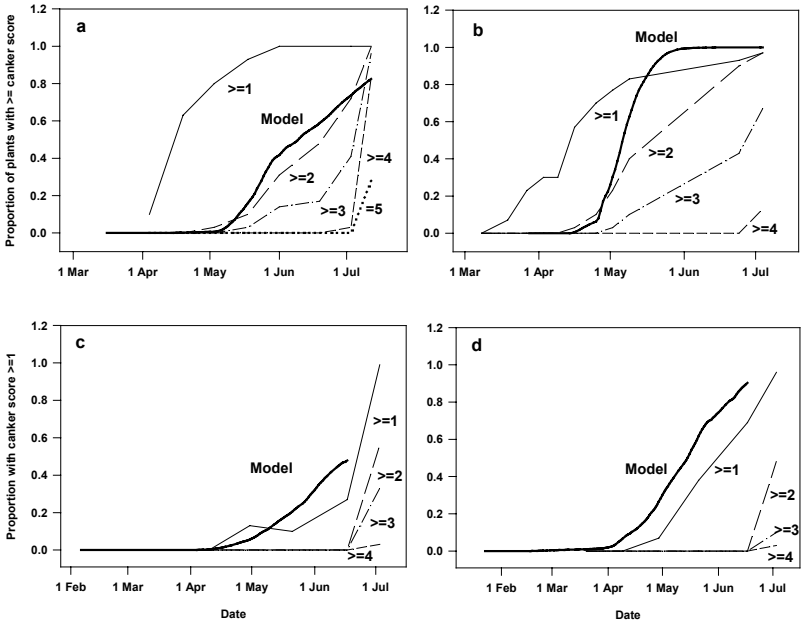

Fig. 2: Proportion of winter oilseed rape plants with stem canker severity scores >=1 (—), >=2 (— —), >=3(— · —), >=4 (---) or =5 (·····), observed in the field during three seasons at Rothamsted: 2000/01 (a), 2001/02 (b), 2002/03-small plants (c) and 2002/03-large plants (d). The model estimate of the probability that the stem developed canker is represented by the thick solid line (—)

Table 1. Details of model parameters fitted and model goodness of fit for winter oilseed rape crop data collected at Rothamsted (UK), Boxworth (UK) and Thiverval Grignon (France).

Site	Year	Cultivar	Sowing date/Seed rate	Parameters		R^2(%)
				Ascospore infectivity[c]	*Probability per unit of lesion formation per d-d*[d, e]	
Boxworth	2000/01	Pronto	21/8/2000/L	1.0×10^{-4}	6.0×10^{-3}	80
		Pronto	21/8/2000/H	1.6×10^{-4}	9.0×10^{-3}	82
		Pronto	11/9/2000/L	6.0×10^{-5}	1.1×10^{-2}	86
		Pronto	11/9/2000/H	1.3×10^{-5}	1.0×10^{-2}	78
Boxworth	2003/04	Apex	3/9/2003	1.1×10^{-4}	8.0×10^{-3}	33
Rothamsted	2003/04	Eurol	17/9/2003	2.0×10^{-7}	6.0×10^{-3}	83
		Darmor	17/9/2003	3.5×10^{-5}	2.7×10^{-2}	82
		Canberra	17/9/2003	8.0×10^{-5}	1.0×10^{-2}	81
Rothamsted[a]	2004/05	Canberra	20/8/2004	2.8×10^{-5}	8.0×10^{-3}	25
		Courage	20/8/2004	5.4×10^{-5}	9.0×10^{-3}	-
Rothamsted[b]	2004/05	Canberra	3/9/2004	3.4×10^{-4}	2.7×10^{-2}	11
		Eurol	3/9/2004	1.0×10^{-5}	1.1×10^{-2}	-
		Darmor	3/9/2004	1.2×10^{-5}	1.0×10^{-2}	91
Thiverval-Grignon	2003/04	Pinochet	5/9/2003	8.0×10^{-4}	5.0×10^{-3}	66

[a] Bones Close field, [b] Little Knott 1 field, [c] Probability that each ascospore per unit area sampled (m^3) infects per unit of time (day), [d] *d-d* = °C-day, [e] Probability per unit leaf area (cm^2) of lesion formation per unit time (day). L – low seed rate H - high seed rate

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