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Effects of severity and timing of stem canker (*Leptosphaeria maculans*) symptoms on yield of winter oilseed rape (*Brassica napus*) in the UK

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Abstract

The relationships between yield loss and incidence (% plants with stems affected) or severity (mean stem score, 0–4 scale) of stem canker in winter oilseed rape were analysed using data from experiments at Rothamsted in 1991/92, Withington in 1992/93, Boxworth in 1993/94 and Rothamsted in 1997/98. Critical point models and area under disease progress curve (AUDPC) models were better than multiple point models for describing relationships between yield (t ha^{-1}) and incidence or severity of stem canker for the four experiments. Since yield is influenced by many factors other than disease, % yield loss was calculated and critical point models and AUDPC models relating % yield loss to stem canker were constructed. The critical point models for % yield loss on stem canker incidence for three of the four experiments were similar, but differed from that for Rothamsted in 1991/92. There were also no differences between models of % yield loss on AUDPC of both incidence and severity for these three experiments. Therefore, general models of % yield loss (L) against AUDPC of incidence (X) or severity (S) of stem canker from growth stages 4.8 to 6.4 were derived from the combined data sets for the three experiments: $L = -0.76 + 0.0075X$ ($R^2 = 35\%$, $p < 0.001$), $L = 0.26 + 0.53S$ ($R^2 = 37\%$, $p < 0.001$). The relationships between % yield loss and % plants with different stem canker severity scores at different growth stages were also analysed; the greatest yield losses were generally associated with the largest severity scores, for plants assessed at the same crop growth stage, and were also associated with the early development of stem lesions. Further analyses showed that % yield loss was related to incidence or severity of both basal stem cankers and upper stem lesions in experiments at Boxworth in 1993/94 and at Rothamsted in 1997/98.

Introduction

Stem canker (blackleg), caused by *Leptosphaeria maculans* (Desm.) Ces. & de Not., is a damaging disease of oilseed rape (canola) in the UK, France, Germany, Australia and Canada. The pathogen attacks cotyledons, leaves, stems, roots and pods of oilseed rape to produce leaf spots, basal stem cankers, upper stem lesions and pod spots (Gabrielson, 1983; Petrie, 1979; Paul and Rawlinson, 1992). In the UK, basal stem cankers on winter oilseed rape (*Brassica napus* L. spp. *oleifera* D.C.) originate from phoma leaf spots produced on leaves while the crop is at the rosette stage during the autumn and winter, because the pathogen spreads from these leaves down petioles to reach the stem bases (Hammond et al., 1985). Upper stem lesions originate from leaf spots on leaves produced later in the season (Gladders and Symonds, 1995). The basal stem cankers and upper stem lesions both girdle and weaken stems, and cause lodging and premature ripening of

the pods. In central France, when a severe stem canker epidemic occurred in 1966, the average yield was 40% less than in 1964 (Lacoste et al., 1969; Tiberghien, 1974). In Canada, average yield losses due to stem canker in Saskatchewan were 7.2% in 1984, 5.2% in 1985, and exceeded 30% in some individual crops (Gugel and Petrie, 1992). In Australia, yield losses from stem canker were very widespread in 1972 and 1973 (Barbetti, 1975; Roy, 1978). In the UK, yield losses of up to 50% in susceptible cultivars have been reported when incidence of severe stem canker was high (Gladders and Musa, 1979). In the Netherlands, a yield loss of about 30% was reported for the susceptible cultivars, such as Primor, in a cultivar trial with a high incidence of stem canker (Van der Spek, 1981).

Fungicides are one of most effective means for controlling stem canker in the UK (Gladders, 1988; Gladders et al.,1998; Sansford et al., 1996), but their use must be optimised to achieve the maximum economic response and to avoid unnecessary fungicide applications. Spray timing experiments have shown that stem canker can be effectively controlled only by spray programmes initiated in the autumn, when the pathogen has not yet spread from leaves to the stems. To guide fungicide applications in the autumn, it is necessary not only to predict the risk that severe stem canker epidemics will develop in the spring but also to know relationships between yield loss and severity of stem canker.

Most previous work done in Australia, Canada, France or the UK (Table 1) has indicated that relationships between yield or yield loss and stem canker incidence (% plants with stems affected) or severity (stem severity score) can be described by a linear arithmetic equation (McGee and Emmett, 1977; Rempel et al., 1991; Church and Fitt, 1995; Sansford et al., 1996; Pierre et al., 1982). However, Hall et al. (1993) suggested that the relationship between yield loss and stem canker severity in Canada was best described by a logarithmic equation. These relationships between yield or yield loss and stem canker were all single point models based on assessment of the disease at a single growth stage, although the growth stage and method of assessment differed. They did not attempt to determine the critical point (CP) for estimating yield loss by analysis of the relationship between yield loss and the disease at different growth stages. Thus, these single point models produced relationships which differed from season to season and site to site. The lack of a consistent model over different sites and different seasons implied that the models were specific to individual crops. Furthermore, these models did not differentiate the effects of lesions of differing severity or of basal stem cankers and upper stem lesions. More accurate estimates of relationships between yield loss and stem canker, which may be applicable to different sites and seasons, might be obtained by examining relationships between yield loss and disease epidemic timing using CP, multiple point (MP) or area under the disease progress curve (AUDPC) models (Campbell and Madden, 1990). This paper reports detailed examinations of the effects of severity and timing of stem canker symptoms on yield of winter oilseed rape in the UK, using CP, MP and AUDPC models.

Materials and methods

Field experiments

UK data sets that were used to analyse and establish relationships between yield loss of winter oilseed rape and incidence or severity of stem canker were from field experiments at Rothamsted in 1991/92, Withington in 1993/94, Boxworth in 1993/94 and Rothamsted in 1997/98 (Table 2). In these experiments, light leaf spot and other oilseed rape diseases were absent or present at a low severity. The experiments were arranged in randomised block designs, with two replicate blocks of 22 plots at Rothamsted in 1991/92, Withington in 1993/94 and Boxworth in 1993/94, and with four replicate blocks of five main plots, each split into two subplots (two cultivars) at Rothamsted in 1997/98. The plot areas were 75 m^2 in the 1991/92 experiment, 96 m^2 in 1992/93, 108 m^2 in 1993/94 and 45 m^2 in 1997/98. The winter oilseed rape cultivar Envol was sown at Rothamsted on 28 August 1991 (after oilseed rape), at Withington on 14 September 1992 (after oilseed rape), at Boxworth on 7 September 1993 (after barley) and the cultivars Capitol and Lipton were sown at Rothamsted on 26 August 1997 (after barley). The experiments at Rothamsted in 1991/92 and Withington in 1992/93 were uninoculated, but the experiments at Boxworth in 1993/94 and Rothamsted in 1997/98 were inoculated with infected oilseed rape stem debris in October. Different disease epidemic patterns in different experiment plots were obtained by leaving the plots unsprayed or using fungicide treatment regimes; a mixture of iprodione and thiophanate-methyl (as Compass at $0.25 + 0.25$ kg a.i. ha^{-1}) with prochloraz (as Sportak

Table 1. Equations describing relationships between yield or yield loss of oilseed rape crops and incidence or severity of stem canker that have been established in Australia, Canada and the UK, in experiments using different assessment methods at different growth stages

Season	Location and country	Type of oilseed rape	Equation[a]	Growth stage and method of assessment	Author
1971	Victoria, Australia	Winter	$Y = 0.19 - 1.49X_s$ ($r = 0.97$, $p < 0.001$) $Y_l = -2.0 + 0.7X_s$ ($r = 0.74$, $p < 0.001$)	Plants recorded as healthy, or with slight or severe cankers[b] at harvest	McGee and Emmett (1977)
—	Ontario, Canada	Spring	$Y_l = -4.0 + 18.7S$ ($R^2 = 0.87$)	Stem canker severity[b] (0–4 scale) at maturity	Rempel et al. (1991)
1986	Ontario, Canada	Winter	$\log_{10}(Y_l + 1) = -0.01 + 0.40S$ ($R^2 = 0.81$)	Basal Stem canker severity (0–4 scale) at maturity	Hall et al. (1993)
1988			$\log_{10}(Y_l + 1) = 0.19 + 0.46S$ ($R^2 = 0.95$)		
1989			$\log_{10}(Y_l + 1) = -0.05 + 0.45S$ ($R^2 = 0.96$)		
1991/92 and 1992/93	Rothamsted, UK	Winter	$Y_l = 0.065 + 0.0071X$ ($R^2 = 0.56$)	% plants with stem canker[b] at GS 6.2	Church and Fitt (1995)

[a]Original symbols and units have been changed to provide consistency, Y = yield (t ha^{-1}); Y_l = % yield loss; X = % plants with stem canker; X_s = % plants with severe stem canker; S = stem canker severity (0–4 scale).
[b]Stem canker assessments did not distinguish basal stem cankers and upper stem lesions.

Table 2. Information about seasons and sites of field experiments providing data used to construct equations relating yield loss of winter oilseed rape to stem canker incidence or severity

Season	Site	Cultivar	Resistance rating[a]	Date of sowing	Date of harvest	Date of disease assessment	Days after sowing	Days before harvest	Growth stage (GS)
1991/92	Rothamsted	Envol	5	28 Aug. 1991	15 Jul. 1992	4 June	281	31	6.2
						6 July	313	9	6.5
1992/93	Withington	Envol	5	14 Sept. 1992	31 Jul. 1993	20 May	248	72	4.9
						14 June	273	47	6.3
						12 July	301	19	6.4
1993/94	Boxworth	Envol	5	7 Sept. 1993	18 Jul. 1994	20 April	225	89	3.7
						13 May	248	66	4.8/5.5
						3 June	269	45	6.2
						28 June	294	20	6.3
1997/98	Rothamsted	Capitol	6	26 Aug. 1997	19 Jul. 1998	16 April	233	94	4.4
		Lipton	5			14 May	261	66	5.5
						4 June	282	45	5.9/6.2
						1 July	309	18	6.4

[a]Using a 0–9 resistance scale; a high figure indicates resistance (Anonymous, 1995, 1998).

45 at 0.3 kg a.i. ha^{-1}) at Rothamsted in 1991/92, Withington in 1992/93 and Boxworth in 1993/94, and difenoconazole plus carbendazim (0.125 kg ha^{-1} and 0.25 kg ha^{-1} a.i., respectively) at Rothamsted in 1997/98 with 10–22 different spray programmes used in each experiment.

Ten plants (Withington in 1992/93; Boxworth in 1993/94) or twenty-five plants (Rothamsted in 1991/92; 1997/98) were sampled at approximately monthly intervals from sampling areas at the edges and ends of plots; it was not always possible to obtain samples from every plot on each occasion. Disease assessment dates and growth stages are shown in Table 2. Incidence (% plants with stems affected) and severity (mean severity score) of stem canker (including basal stem cankers and upper stem lesions) were recorded. The basal stem cankers and upper stem lesions were assessed separately at Boxworth in 1993/94 and Rothamsted in 1997/98. The disease severity assessment used a 0–4 scale (Hardwick et al., 1989; 0 – no disease, 1 – less than half the stem girdled by lesions, 2 – more than half the stem girdled by lesions, 3 – whole stem girdled and weakened by lesions, 4 – plant dead) (Figure 1). Growth stages (GS) of the crop were recorded using the identification key of Sylvester-Bradley and Makepeace (1985). Plots were combine harvested directly and yields adjusted to 90% dry matter.

In the experiment at Rothamsted in 1997/98, the progress of stem canker lesions was also monitored in detail in eight plots (two unsprayed and two each sprayed with three different spray treatments). Twenty-five plants in each plot were selected at random and marked. The basal stem cankers and upper stem lesions on individual marked plants were assessed separately at two-week intervals, and the growth stages were recorded.

Data analyses

Correlation and linear regression analyses were used to examine the relationships between yield or yield loss and stem canker and to establish CP models, MP models and AUDPC models between yield loss and stem canker for the different experiments. AUDPCs were calculated from stem canker assessment data for different dates:

$$\text{AUDPC} = \sum_{i=1}^{i=n-1} 0.5(x_{i+1} + x_i)(t_{i+1} - t_i), \qquad (1)$$

where t_i is day number, x_i is incidence or severity at assessment day i and n is number of assessment dates. Differences between CP models and AUDPC models in different experiments were compared by using linear regression analyses of position and parallelism.

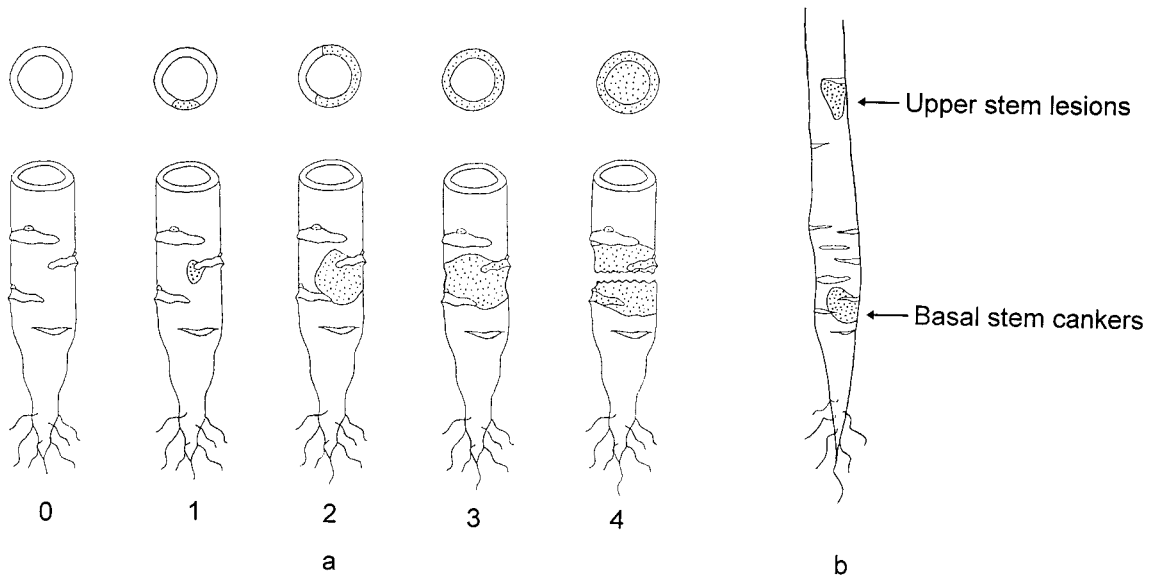

Figure 1. Winter oilseed rape stem bases with stem cankers with different severity scores (0–4 scale) (a) and symptoms of basal stem cankers and upper stem lesions (b).

For regression analyses, adjusted R^2 values (% variance accounted for; Payne et al., 1993) were calculated. The statistical package Genstat (Payne et al., 1993) was used for all analyses.

Results

Disease epidemics

The development of stem canker in untreated plots was different in different experiments (Table 3). Stem canker occurred earlier and was more severe at Boxworth in 1993/94 and Rothamsted in 1997/98 than that at Rothamsted in 1991/92 or Withington in 1992/93. Symptoms of stem disease were first seen at Boxworth on 20 April 1993 (GS 3.7) and Rothamsted on 16 April 1997 (GS 4.4); thereafter disease incidence increased quickly to reach 90% on 28 June 1994 (GS 6.3) and 85.5% on 1 July 1998 (GS 6.4), respectively. Stem canker severity scores were greater at Rothamsted in 1997/98 than in other experiments at similar growth stages.

The progress curves from two-weekly monitoring of stem canker at Rothamsted in 1997/98 showed that incidence of both basal stem cankers and upper stem lesions in plots sprayed with fungicide was smaller (Figure 2a) and disease severity was less (Figure 2b) than that in untreated plots throughout the period from 20 May to 15 July. In untreated plots, the incidence of upper stem lesions rapidly increased between 17 June and 1 July, and the severity of upper stem lesions increased between 4 June and 1 July. Incidence of basal stem cankers in untreated plots increased by 10% from 20 May to 4 June, and later remained at 40–45% while severity continued to increase. In the treated plots, both

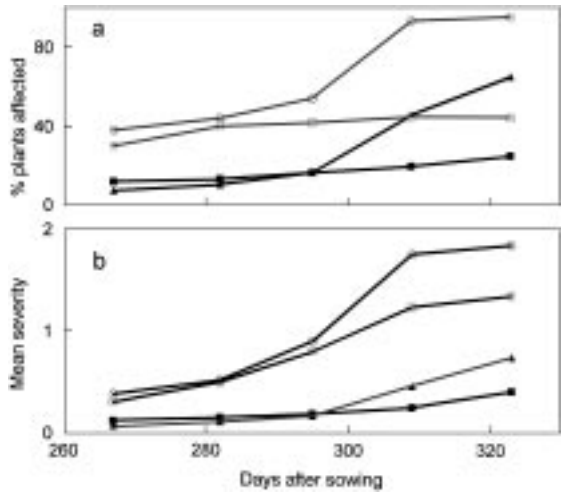

Figure 2. Changes with time in incidence (% plants affected) (a) or severity (0–4 scale) (b) of basal stem cankers (□, ■) and upper stem lesions (○, ▲) in winter oilseed rape plots with (■, ▲) or without (□, ○) fungicide treatment at Rothamsted in 1998. Disease was assessed on 20 May (267 days after sowing), 4 June (282 days after sowing), 17 June (295 days after sowing), 1 July (309 days after sowing) and 15 July (323 days after sowing).

Table 3. Progress of stem canker epidemics on winter oilseed rape in field experiments in different seasons at different sites; incidence and severity data from untreated plots

Season	Site	Date of disease assessment	Incidence (% plants affected)	% plants with different severity scores				Mean severity (0–4 scale)
				1	2	3	4	
1991/92	Rothamsted	4 June	38.0	36.5	1.5	0	0	0.39
		6 July	82.0	28.0	6.0	36.0	12.0	1.96
1992/93	Withington	20 May	10.0	5.0	5.0	0	0	0.15
		14 June	22.5	17.5	5.0	0	0	0.28
		12 July	67.5	12.5	20.0	17.5	17.5	1.75
1993/94	Boxworth[a]	20 April	2.5	2.5	0	0	0	0.03
		13 May	32.5	32.5	0	0	0	0.33
		3 June	62.5	60.0	2.5	0	0	0.65
		28 June	90.0	70.0	20.0	0	0	1.10
1997/98	Rothamsted[a]	16 April	6.5	6.5	0	0	0	0.07
		14 May	52.5	45.0	7.0	0.5	0	0.61
		4 June	76.5	57.0	14.5	4.0	1.0	1.02
		1 July	85.5	38.0	15.0	13.5	19.0	1.85

[a]Experiments for which upper stem lesions and basal stem canker were assessed separately.

incidence and severity of basal stem cankers remained low from 20 May to 15 July, and the incidence and severity of upper stem lesions increased rapidly after 17 June.

In unsprayed plots (Figure 3a and b), only severity score 1 was recorded at Rothamsted on 20 May 1998, for both basal stem cankers and upper stem lesions, and severity scores 2, 3 and 4 were not recorded until 4 June, 17 June and 1 July, respectively. The changes in % plants with different severity scores differed between basal stem cankers and upper stem lesions. The % plants with severity score 1 for basal stem cankers began to decrease gradually when % plants with severity scores 2, 3, 4 increased after 4 June. However, the % plants with severity score 1 for upper stem lesions decreased initially, but increased rapidly again after 17 June to reach 58% on 1 July. These data indicate that basal stem canker lesions with severity score 1 on 20 May became more severe so that they had severity scores of 2, 3 or 4 in June or July, and that not many new basal stem canker lesions (score 1) appeared in June or July. However, many new upper stem lesions (score 1) continued to appear after 17 June. In plots treated with fungicide (Figure 3c and d), no plant with a severity score of 4 had been recorded by 15 July. Basal stem cankers with severity scores of 2 and 3 were recorded on 17 June and 1 July, respectively, then developed slowly. Upper stem lesions with severity scores of 2 and 3 were recorded on 15 July, at incidences of 8% and 4%, respectively. The incidence of basal stem cankers with severity score 1 remained at 12–16% during the period 20 May to 15 July, but that of upper stem lesions increased rapidly after 17 June to reach 53% by 15 July.

Relationships between yield and stem canker at different assessment dates

Coefficients of correlation between crop yield and incidence or severity of stem canker at different assessment dates (Table 4) showed that yield was inversely related to the stem canker incidence or severity from the middle of May (GS 4.8) to early July (GS 6.5), but not in April (GS 3.7). At Boxworth in 1993/94 and Rothamsted in 1997/98, correlation coefficient values were similar on

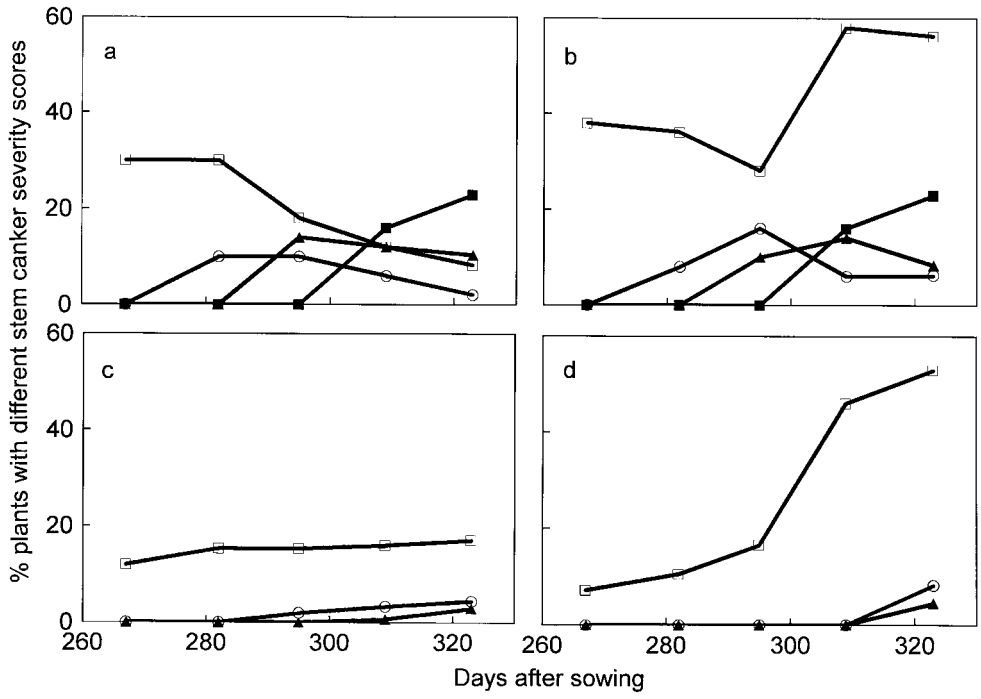

Figure 3. Changes with time in % plants with different severity scores for basal stem cankers (a, c) or upper stem lesions (b, d) [severity 1 (□), severity 2 (○), severity 3 (▲), severity 4(■)] in winter oilseed rape plots with (a, b) or without (c, d) fungicide treatment at Rothamsted in 1998. Disease was assessed on 20 May (267 days after sowing), 4 June (282 days after sowing), 17 June (295 days after sowing), 1 July (309 days after sowing) and 15 July (323 days after sowing).

Table 4. Coefficients of correlation (r) between yield (t ha^{-1}) of winter oilseed rape and incidence or severity of stem canker at different growth stages in different seasons at different sites[a]

Season	Site	Date of disease assessment	Incidence (% plants affected)			Mean severity (0–4 scale)		
		(Growth stage)	r	p	df	r	p	df
1991/92	Rothamsted[b]	6 July (6.5)	−0.40	0.011	38	−0.41	0.010	37
1992/93	Withington	20 May (4.9)	−0.47	0.006	31	−0.46	0.008	31
		14 June (6.3)	−0.39	0.02	34	−0.36	0.033	34
		12 July (6.4)	−0.73	<0.001	38	−0.76	<0.001	38
1993/94	Boxworth	20 April (3.7)	−0.08	0.67	30	−0.08	0.67	30
		13 May (4.8/5.5)	−0.67	<0.001	37	−0.67	<0.001	37
		3 June (6.2)	−0.54	<0.001	40	−0.54	<0.001	40
		28 June (6.3)	−0.72	<0.001	42	−0.68	<0.001	42
1997/98	Rothamsted[b]	14 May (5.5)	−0.49	0.001	38	−0.49	0.001	38
		4 June (5.9/6.2)	−0.47	0.002	38	−0.53	<0.001	38
		1 July (6.4)	−0.50	0.001	38	−0.50	0.001	38

[a]Stem canker data include both basal stem cankers and upper stem lesions.
[b]Stem canker incidence data was insufficient to calculate correlation coefficients for samples on 4 June 1992 and 16 April 1998 at Rothamsted.

assessment dates in May, June and July (GS 4.8–6.4), but at Withington in 1992/93 correlation coefficient values for incidence and severity were greater on 12 July (GS 6.4) than in May or June.

Models of relationships between yield and stem canker

CP models, MP models and AUDPC models relating yield (t ha^{-1}) to stem canker incidence or severity were constructed by simple or multiple linear regression (Table 5). For the data for Rothamsted in 1997/98, all models were first calculated separately for the two cultivars Capitol and Lipton and then compared. The results demonstrated no significant difference between these models for the two cultivars, and therefore CP, AUDPC and MP models were constructed by using combined data for the two cultivars. The linear regressions were statistically significant ($p < 0.05$) for all models. The CP models for the experiment at Rothamsted in 1991/92 accounted for <15% of the variance. The CP, AUDPC and MP models for the experiments at Withington in 1992/93 and Boxworth in 1993/94 all generally accounted for 45–55% of the variance. The CP, AUDPC and MP models for the experiment at Rothamsted in 1997/98 all accounted for 22–27% of the variance. Analyses of model collinearity indicated that there were strong correlations between variables used in MP models. This produced some positive values for slopes in MP models, which are very difficult to interpret biologically, even though the values of negative slopes in MP models did not really represent the contribution of the variables to yield loss. These results demonstrated that CP models and AUDPC models were generally better than MP models.

CP and AUDPC models relating % yield loss to stem canker

Since yield in t ha^{-1} is influenced by many factors specific to the crop other than disease, % yield loss was estimated relative to the mean yield of plots with no disease present. CP models and AUDPC models relating % yield loss to stem canker incidence or severity in different experiments were constructed using linear regression (Figures 4–7). The differences between experiments for these models were compared using analyses of position and parallelism. The results showed that CP models for stem canker incidence at Rothamsted in 1991/92 were significantly different to those for the other experiments, but that there were no differences between models for the other three experiments. Thus a CP model for incidence was based on combined data sets from Withington in 1992/93, Boxworth in 1993/94 and Rothamsted in 1997/98:

$$L = -1.90 + 0.27X \quad (R^2 = 37.7\%,\ p < 0.001,\ \text{df} = 122), \quad (2)$$

where L = % yield loss, X = stem canker incidence at GS 6.3 or 6.4. The fit of the general yield

Table 5. Equations describing relationships between yield (t ha^{-1}) of winter oilseed rape and incidence (% plants affected) or severity (0–4 scale) of stem canker at Rothamsted in 1991/92, 1997/98, Withington in 1992/93 and Boxworth in 1993/94

Season	Site		Equation	R^2 (%)[a]	F[b]	p[c]	df	Parameter definition
1991/92	Rothamsted	CP[d]	$Y = 3.48 - 0.004X$	13.5	7.06	0.011	38	X, S = incidence, severity
			$Y = 3.42 - 0.16S$	14.4	7.42	0.010	37	on 6 July
1992/93	Withington	CP	$Y = 3.16 - 0.0091X_3$	52.1	43.48	<0.001	38	X_1, X_2, X_3 and S_1, S_2, S_3 =
			$Y = 3.11 - 0.32S_3$	56.4	51.53	<0.001	38	incidence and severity on
		AUDPC [e]	$Y = 3.14 - 0.00033X_4$	38.5	21.02	<0.001	31	20 May, 4 June, 12 July;
			$Y = 3.12 - 0.017S_4$	46.6	28.94	<0.001	31	X_4, S_4 = AUDPC of incidence,
		MP[f]	$Y = 3.16 - 0.016X_1 + 0.0011X_2 - 0.0076X_3$	48.6	11.09	<0.001	29	severity from 20 May to
			$Y = 3.11 - 0.61S_1 + 0.15S_2 - 0.30S_3$	49.8	11.59	<0.001	29	12 July
1993/94	Boxworth	CP	$Y = 2.48 - 0.0028X_3$	50.3	44.55	<0.001	42	X_1, X_2, X_3 and S_1, S_2, S_3 =
			$Y = 2.45 - 0.48S_3$	45.0	36.18	<0.001	42	incidence and severity on
		AUDPC	$Y = 2.46 - 0.00017X_4$	51.3	41.08	<0.001	37	13 May, 3 June, 28 June;
			$Y = 2.46 - 0.016S_4$	49.2	37.86	<0.001	37	X_4, S_4 = AUDPC of incidence,
		MP	$Y = 2.46 - 0.0047X_1 - 0.0017X_2 - 0.0026X_3$	52.2	14.81	<0.001	35	severity from 13 May to 28
			$Y = 2.45 - 0.57S_1 - 0.24S_2 - 0.13S_3$	50.1	13.70	<0.001	35	June
1997/98	Rothamsted	CP	$Y = 3.08 - 0.0098X_3$	22.6	12.38	0.001	38	X_1, X_2, X_3 and S_1, S_2, S_3 =
			$Y = 2.86 - 0.36S_3$	23.0	12.16	0.001	38	incidence and severity on
		AUDPC	$Y = 3.02 - 0.00025X_4$	25.8	14.55	<0.001	38	14 May, 4 June and 1 July;
			$Y = 2.90 - 0.014S_4$	27.1	15.40	<0.001	38	X_4, S_4 = AUDPC of incidence,
			$Y = 3.00 - 0.0057X_1 - 0.0016X_2 - 0.0055X_3$	23.4	4.97	0.05	36	severity from 14 May to 1 July
		MP	$Y = 2.92 - 0.032S_1 - 0.51S_2 - 0.15S_3$	23.4	4.97	0.05	36	

[a]% variance accounted for (coefficient of determination); [b]F value of F test; [c]p value of F test; [d]Critical point model; [e]Area under disease progress curve model; [f]Multiple point model.

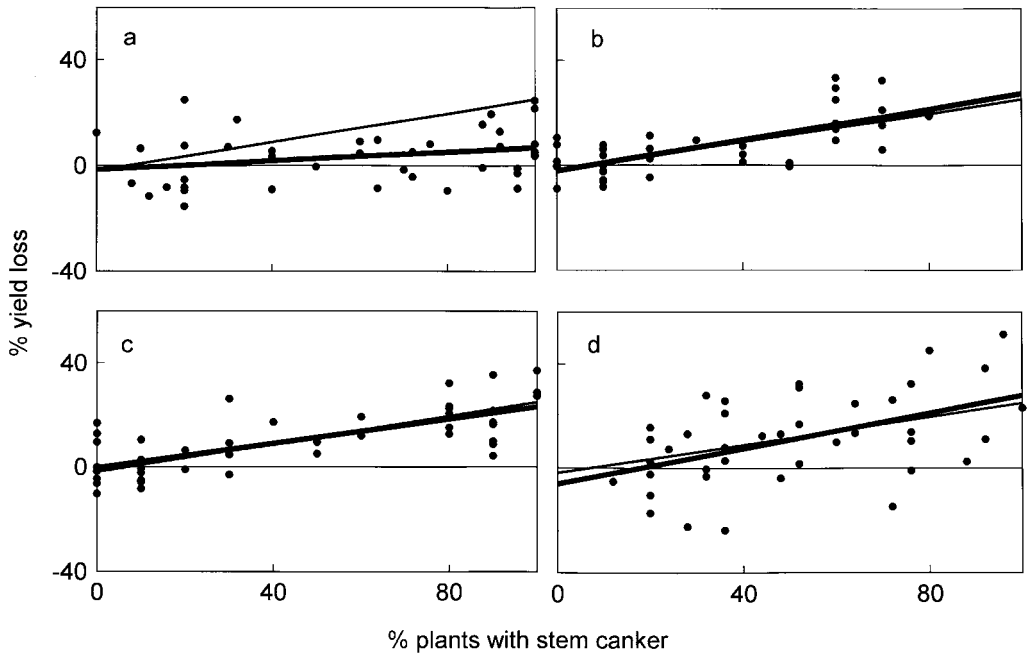

Figure 4. CP models relating % yield loss (L) of winter oilseed rape to incidence (% plants affected) (X) of stem canker at Rothamsted on 6 July 1992 (a: $L = -3.74 + 0.12X$, $R^2 = 13.5\%$, $p = 0.011$), Withington on 12 July 1993 (b: $L = -2.07 + 0.29X$, $R^2 = 52.1\%$, $p < 0.001$), Boxworth on 28 June 1994 (c: $L = -0.36 + 0.24X$, $R^2 = 50.3\%$, $p < 0.001$) and Rothamsted on 1 July 1998 (d: $L = -6.07 + 0.34X$, $R^2 = 22.6\%$, $p = 0.001$) (thick lines), and model based on combined data from three experiments ($L = -1.90 + 0.27X$, $R^2 = 37.7\%$, $p < 0.001$) (thin lines).

loss relationship (Equation (2)) from the three experiments was compared to the fitted yield loss relationship for each experiment. This analysis indicated that there were no significant differences between the line produced by the combined model and the lines produced by regression of observed yield loss measurements on stem canker incidence at Withington in 1992/93 (Figure 4b), Boxworth in 1993/94 (Figure 4c) or Rothamsted in 1997/98 (Figure 4d). However, the slope of the regression line for the combined model was significantly different from that produced by regression of observed yield loss measurements on stem canker incidence in July in the experiment at Rothamsted in 1991/92 (Figure 4a).

The comparison of CP models for severity showed that not only did the model for Rothamsted in 1991/92 (Figure 5a) differ significantly to those for Withington in 1992/93 (Figure 5b), Boxworth in 1993/94 (Figure 5c) and Rothamsted in 1997/98 (Figure 5d), but also there was a significant difference between the models for the experiments at Withington in 1992/93 and Boxworth in 1993/94. Therefore a CP model for stem canker severity using combined data sets from Withington in 1992/93, Boxworth in 1993/94 and Rothamsted in 1997/98 was not constructed.

Since comparison of AUDPC models showed no differences between Withington in 1992/93, Boxworth in 1993/94 and Rothamsted in 1997/98 in models for both incidence and severity, AUDPC models for incidence and severity were derived from the combined data sets for these three experiments:

$$L = -0.76 + 0.0075X \quad (R^2 = 35.3\%,\ p < 0.001,\ \mathrm{df} = 110), \quad (3)$$

$$L = 0.26 + 0.53S \quad (R^2 = 37.2\%,\ p < 0.001,\ \mathrm{df} = 110), \quad (4)$$

where L = % yield loss, X and S = AUDPC of incidence and severity from GS 4.8/5.5 to 6.4, respectively. The combined AUDPC models using data for the three experiments are illustrated in Figures 6 and 7. The fit of the predicted relationships for incidence and severity (Equations (3) and (4)) from combined data sets for the three experiments was also compared to the fitted yield loss relationships for each experiment. The results showed that there were no

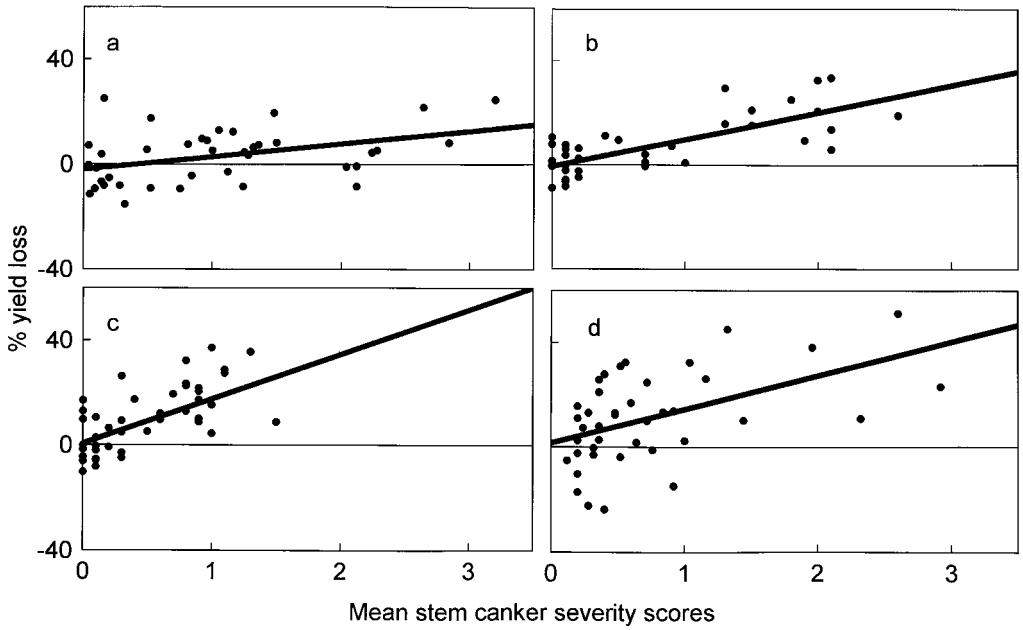

Figure 5. CP models relating % yield loss (L) of winter oilseed rape to severity (0–4 scale) (S) of stem canker at Rothamsted on 6 July 1992 (a: $L = -2.01 + 4.86S$, $R^2 = 14.4\%$, $p = 0.010$), Withington on 12 July 1993 (b: $L = -0.54 + 10.29S$, $R^2 = 56.4\%$, $p < 0.001$), Boxworth on 28 June 1994 (c: $L = 0.57 + 19.51S$, $R^2 = 45.0\%$, $p < 0.001$) and Rothamsted on 1 July 1998 (d: $L = 1.58 + 12.91S$, $R^2 = 23.0\%$, $p = 0.001$).

significant differences between the lines derived from the combined data for the three experiments and the lines derived from data for each individual experiment (Figures 6 and 7).

Relationships between % yield loss and % plants with different severity scores

The relationships between % yield loss and % plants with different severity scores were analysed by linear regression. The values of slopes of regression lines are shown in Table 6. The results demonstrated that the greatest yield losses were generally associated with the largest severity scores for plants assessed at the same crop growth stages. However, there were some differences between experiments. For example, the results suggested that before harvest only severity scores of 3 or 4 at GS 6.5 (6 July in 1991/92; 12 July in 1992/93) and score 4 at GS 6.4 (1 July) in 1997/98 affected yield, but that severity scores of 1 or 2 at GS 6.3 (28 June) in 1993/94 and score 2 at GS 6.4 (12 July) in 1992/93 also affected yield. Nevertheless, yield losses were also associated with severity scores of 1 and 2 in May (GS 4.9–5.5) and June (GS 5.9–6.3), when assessments were made in 1992/93, 1993/94 and 1997/98. Thus, the results suggested that the earlier that lesions with a particular severity score were recorded on plants, the greater was the effect on yield, because lesions with a small severity score in early spring continued to become more severe.

Relationships between % yield loss and incidence or severity for basal stem cankers and upper stem lesions

The relationships between % yield loss and % plants with basal stem cankers or upper stem lesions or between % yield loss and mean severity score for basal stem cankers or upper stem lesions at Boxworth in 1993/94 and Rothamsted in 1997/98 were also analysed by linear regression. The values of slopes of regression lines are given in Table 7. The results showed that yield losses were significantly related to incidence or severity of both basal stem cankers and upper stem lesions in both seasons in May, June and July, except for basal stem cankers at Boxworth on 13 May 1994. The different values of the slopes of the regression lines suggested that effects of basal stem cankers on yield were different from those of upper stem lesions.

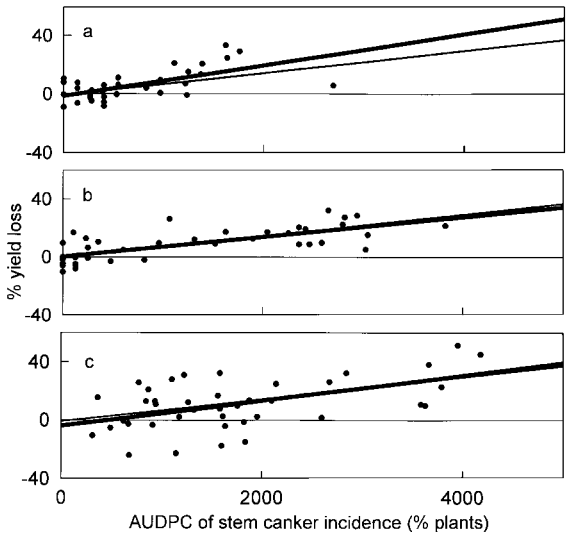

Figure 6. AUDPC models relating % yield loss (L) of winter oilseed rape to incidence (% plants affected) (X) of stem canker at Withington from 20 May to 12 July in 1993 (a: $L = -1.48 + 0.011X$, $R^2 = 38.5\%$, $p < 0.001$), Boxworth from 13 May to 28 June in 1994 (b: $L = -0.23 + 0.0068X$, $R^2 = 51.3\%$, $p < 0.001$) and Rothamsted from 14 May to 1 July in 1998 (c: $L = -3.88 + 0.00859X$, $R^2 = 25.8\%$, $p < 0.001$) (thick lines), and model of combined data for three experiments ($L = -0.76 + 0.0075X$, $R^2 = 35.3\%$, $p < 0.001$) (thin lines).

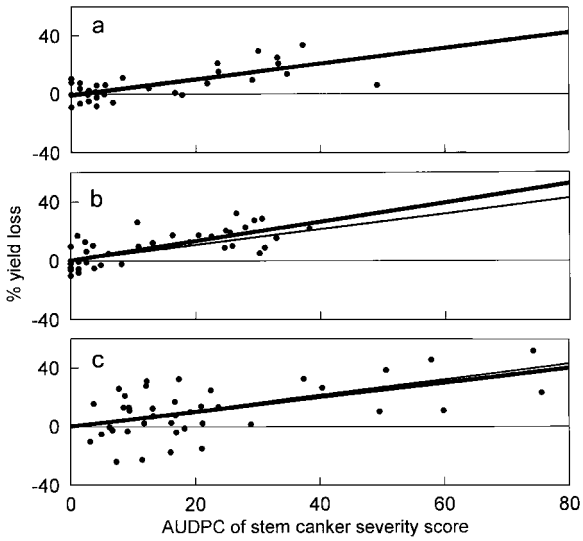

Figure 7. AUDPC models relating % yield loss (L) of winter oilseed rape to severity (0–4 scale) (S) of stem canker at Withington from 20 May to 12 July in 1993 (a: $L = -0.94 + 0.54S$, $R^2 = 46.6\%$, $p < 0.001$), Boxworth from 13 May to 28 June in 1994 (b: $L = 0.38 + 0.65S$, $R^2 = 49.2\%$, $p < 0.001$) and Rothamsted from 14 May to 1 July in 1998 (c: $L = 0.23 + 0.49S$, $R^2 = 27.1\%$, $p < 0.001$) (thick lines), and model using combined data for three experiments ($L = 0.26 + 0.53S$, $R^2 = 37.2\%$, $p < 0.001$) (thin lines).

Table 6. Values of slopes (b) of linear regression lines estimating relationships between % yield loss and % plants with different stem canker severity scores in field experiments in different seasons at different sites. Regressions of the form $y = a + bx$

Season	Site	Date of disease assessment	b for % plants with different severity scores (R^2 (%), p)			
			1	2	3	4
1991/92	Rothamsted	6 July	0.11 (3.3, 0.14)	−0.050 (−, 0.69)[b]	0.22 (9.0, 0.033)	0.42 (10.2, 0.025)
1992/93	Withington	20 May	0.87 (12.7, 0.025)	0.97[a] (1.7, 0.23)		
		14 June	0.41 (13.1, 0.017)	0.40[a] (0.1, 0.32)		
		12 July	0.083 (−, 0.61)[b]	0.70 (36.6, <0.001)	0.89 (31.1, <0.001)	0.48 (38.2, <0.001)
1993/94	Boxworth	13 May	0.30 (33.8, <0.001)			
		3 June	0.24 (40.5, <0.001)	1.01 (7.3, 0.046)		
		28 June	0.25 (47.3, <0.001)	0.61 (28.1, <0.001)	0.59[a] (1.1, 0.23)	
1997/98	Rothamsted	14 May	0.47 (22.8, 0.001)	1.51 (8.0, 0.043)	10.34[a] (11.9, 0.017)	
		4 June	0.30 (9.4, 0.03)	1.37 (26.6, <0.001)	2.44 (9.3, 0.031)	9.80[a] (22.7, 0.001)
		1 July	0.22 (1.9, 0.19)	0.69 (6.1, 0.067)	0.69 (5.7, 0.075)	0.71 (18.2, 0.004)

[a]Few plots had plants with this severity score on this date.
[b]R^2 values were not estimated because residual variance exceeded variance of response variate.

Discussion

Correlation analyses indicate that yield losses from stem canker were related best to disease incidence or severity at GS 6.3 or 6.4, which is the seed development stage (Sylvester-Bradley, 1985) and a key stage for oilseed rape yield production; thus it was concluded that GS 6.3–6.4 was the CP for relating stem canker to yield loss. In previous work on stem canker yield loss models (Table 1), there have been no analyses of the relationships between yield loss and stem canker at different growth stages to determine the CP

Table 7. Values of slopes (b) of linear regression lines estimating relationships between % yield loss and % plants with basal stem cankers or upper stem lesions, or mean severity score for basal stem cankers or upper stem lesions in field experiments at Boxworth in 1993/94 and Rothamsted in 1997/98. Regressions of the form $y = a + bx$

Season	Site	Date of disease assessment	b for % plants affected (R^2 (%), p)		b for mean severity (R^2 (%), p)	
			Basal stem cankers	Upper stem lesions	Basal stem cankers	Upper stem lesions
1993/94	Boxworth	13 May	0.83 (6.7, 0.07)	0.37 (42.3, <0.001)	83.30 (6.7, 0.07)	37.16 (42.3, <0.001)
		3 June	0.63 (39.6, <0.001)	0.21 (29.9, <0.001)	49.48 (36.3, <0.001)	20.72 (29.9, <0.001)
		28 June	0.23 (33.0, <0.001)	0.25 (56.4, <0.001)	17.78 (33.4, <0.001)	23.66 (57.5, <0.001)
1997/98	Rothamsted	14 May	0.43 (21.0, 0.002)	0.91 (16.2, 0.006)	36.50 (21.0, 0.002)	91.10 (16.2, 0.006)
		4 June	0.38 (23.9, <0.001)	0.40 (24.0, <0.001)	29.62 (27.4, <0.001)	36.45 (26.5, <0.001)
		1 July	0.25 (12.6, 0.014)	0.34 (19.7, 0.002)	11.69 (15.8, 0.006)	14.74 (20.4, 0.002)

in epidemic development (McGee and Emmett, 1977; Rempel et al., 1991; Church and Fitt, 1995; Sansford et al., 1996; Pierre et al., 1982; Hall et al., 1993). Generally disease incidence or disease severity near harvest was used as the explanatory variable or predictor, but the models were based on disease assessment at different growth stages and used different methods of assessment. Therefore, it is not surprising that these models differed from season to season and site to site.

CP models for stem canker incidence or severity for Rothamsted in 1991/92 were significantly different from those for the other three experiments. The main reason for this was probably that the CP selected for these models (GS 6.5) was later than those for other experiments (GS 6.3–6.4). It is difficult to clearly assess stem canker symptoms at GS 6.5 because most plants have senesced by then and *L. maculans* rapidly colonises the senescent tissues. Further research on the CP for relating stem canker to yield loss could analyse more experimental data sets, or use other statistical methods such as path analysis and principal component analysis to clarify which growth stage is the CP.

Comparison of CP, AUDPC and MP models for three experiments at Withington in 1992/93, Boxworth in 1993/94 and Rothamsted in 1997/98 suggested that precision of yield loss prediction by CP models and AUDPC models was better than that of MP models. In practice the CP model is the most convenient for users to apply because disease data at only one growth stage is required. However, with CP models it is important to use the correct growth stage and some single point data do not accurately represent effects of disease epidemics on yield during the whole season. Furthermore, the CP model for Rothamsted in 1991/92 differed from those for other three experiments, suggesting the general CP model based on combined data set cannot be applied to all sites and situations. The results of analyses also showed the AUDPC model for incidence based on the combined data from three experiments was no better than the CP model for incidence based on same combined data set. The reason may have been its assumption that, for assessing the relationship between stem canker and yield loss, the weighting of points at different growth stages was same. If stem canker occurring at different growth stages caused different amounts of yield loss, yield loss prediction by AUDPC models would have been less accurate because they did not distinguish between early and late disease that produced the same AUDPC (James, 1974). Theoretically, the limitations of CP and AUDPC models can be overcome by MP model because MP data can be incorporated as different variables, and parameters for each variable can represent the contribution of the variables to yield loss. Unfortunately results of analyses for the three experiments showed that collinearity existed between stem canker variables; thus parameters for each variable in the MP models did not represent its contribution to yield loss. Similar problems have been observed in MP models for some other diseases, such as late blight of potato, wheat stem rust, barley leaf rust and barley powdery mildew (James et al., 1972; Burleigh et al., 1972; Lim and Gaunt, 1986).

Analyses showed that the predictive precision of models for stem canker severity was generally very similar to that for models for stem canker incidence because there was a good relationship between stem canker incidence and severity. For example, R^2 values for the AUDPC models for the combined data sets for three experiments for stem canker incidence and severity were 35.3% and 37.2%, respectively. Generally models based on incidence data are better for practical yield loss prediction because it is simpler and easier to

obtain incidence data from crops and there is less error associated with incidence data than the more subjectively assessed severity data. The models described in this paper were all derived from the experiments where stem canker was the only important disease, although light leaf spot or other oilseed rape diseases commonly occur together with stem canker on crops and also affect yield of oilseed rape. Therefore, it is necessary to investigate interactions between effects of stem canker and light leaf spot or other diseases on yield and to construct combined yield loss model for multiple disease situations.

The results of analyses of relationships between % yield loss and % plants with different stem canker severity scores indicated that early stem canker lesions in May and June, even when the disease score was slight, affected yield more than later stem canker because lesions with a small severity score in early spring became more severe later. Further analysis showed that incidences of both basal stem canker and upper stem lesions were related to yield loss of oilseed rape. However, the timing of the first appearance of lesions and the severity of basal stem cankers and upper stem lesions differed between seasons and sites. These results suggested that it is important for farmers to control early stem canker, and that both basal stem cankers and upper stem lesions need to be controlled. The optimum period for control of stem canker with fungicides was reported to be from November to February (Sansford et al.,1996; Gladders et al., 1998). This optimum period may be related to the epidemiology of stem canker (Fitt et al.,1997). In autumn, fungicide sprays may be needed to control the leaf spot phase of stem canker and prevent the spread of the pathogen from leaves into stem bases, and in late winter or early spring a further spray can control the phoma leaf spots on later leaves to prevent upper stem lesions. Ultimately it is necessary to estimate risks that early, severe basal stem cankers or upper stem lesions will develop to guide decision making for stem canker control during the period from autumn to early spring.

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References

Anonymous (1995,1998) Oilseed variety handbook. National Institute of Agricultural Botany, Cambridge

Barbetti MJ (1975) Effects of temperature on development and progression in rape of crown canker caused by *Leptosphaeria maculans*. Australian Journal of Experimental Agriculture and Animal Husbandry 15: 705–708

Burleigh JR, Roelfs AP and Eversmeyer MG (1972) Estimating damage to wheat caused by *Puccinia recondita tritici*. Phytopathology 62: 944–946

Campbell L and Madden LV (1990) Introduction to Plant Disease Epidemiology. John Wiley and Sons Inc, New York

Church VJ and Fitt BDL (1995) Incidence and effects of diseases on seven winter oilseed rape cultivars in 1991/92 and 1992/93. International Organization for Biological Control Bulletin 18: 62–68

Fitt BDL, Gladders P, Turner JA, Sutherland KG, Welham SJ and Davies JML (1997) Prospects for developing a forecasting scheme to optimise use of fungicides for disease control on winter oilseed rape in the UK. Aspects of Applied Biology, Optimising Pesticide Applications 48: 135–142

Gabrielson RL (1983) Blackleg disease of crucifers caused by *Leptosphaeria maculans* (*Phoma lingam*) and its control. Seed Science and Technology 11: 749–780

Gladders P (1988) The contributions and value of pesticides to disease control in combinable break crops. In: Clifford BC and Lester E (ed) Control of Plant Diseases: Cost and Benefits (pp 29–50). Blackwell Scientific Publications, Oxford

Gladders P and Musa TM (1979) The development of *Leptosphaeria maculans* in winter oilseed rape and its implications for disease control. In: Proceedings 1979 British Crop Protection Conference-Pests and Diseases (pp 129–136)

Gladders P and Symonds BV (1995) Occurrence of canker (*Leptosphaeria maculans*) in winter oilseed rape in eastern England 1977–1993. International Organisation for Biological Control Bulletin 18: 1–11

Gladders P, Symonds BV, Hardwick NV and Sansford CE (1998) Opportunities to control canker (*Leptosphaeria maculans*) in winter oilseed rape by improved spray timing. International Organization for Biological Control Bulletin 21: 111–120

Gugel RK and Petrie GA (1992) History, occurrence, impact, and control of blackleg of rapeseed. Canadian Journal of Plant Pathology 14: 36–45

Hall R, Peters RD and Assabgui RA (1993) Occurrence and impact of blackleg on oilseed rape in Ontario. Canadian Journal of Plant Pathology 15: 305–313

Hammond KE, Lewis BG and Musa TM (1985) A systemic pathway in the infection of oilseed rape plants by *Leptosphaeria maculans*. Plant Pathology 34: 557–565

Hardwick NV, Culshaw FA, Davies JML, Gladders P, Hawkins JH and Slawson DD (1989) Incidence and severity of fungal diseases of winter oilseed rape in England and Wales, 1986–1988. Aspects of Applied Biology, Production and Protection of Oilseed Rape and other Brassica Crops, 23: 383–392

James WC (1974) Assessment of plant diseases and losses. Annual Review of Phytopathology 12: 27–48

James WC, Shih CS, Hodgson WA and Callbeck LC (1972) The quantitative relationship between late blight of potato and loss in tuber yield. Phytopathology 62: 92–96

Lacoste L, Louvet J, Anselme C, Alabouvette C, Brunin B and Pierre JG (1969) Rôle de *Phoma lingam* (Tode) Desm. et de sa forme parfaite, *Leptosphaeria maculans* (Desm.) Ces. et de Not. dans les épidémies de nécrose du collet de colza (*Brassica napus* L. var. *oleifera* Metzer). Comptes Rendus de l'Academie d'Agriculture de France 55: 981–987

Lim LG and Gaunt RE (1986) The effect of powdery mildew (*Erysiphe graminis* f. sp. *hordei*) and leaf rust (*Puccinia hordei*) on spring barley in New Zealand. I. Epidemic development, green leaf area and yield. Plant Pathology 35: 44–53

McGee DC and Emmett RW (1977) Blackleg (*Leptosphaeria maculans* (Desm.) Ces. et de Not.) of rapeseed in Victoria: crop losses and factors which affect disease severity. Australian Journal of Agricultural Research 28: 47–51

Paul VH and Rawlinson CJ (1992) Diseases and pests of rape. Verlag Theodore Mann, Gelsenkirchen-Buer, Germany

Payne RW, Lane PW, Baird DB, Gilmour AR, Harding SA, Morgan GW, Murray DA, Thompson R, Todd AD, Tunnicliffe Wilson G, Webster R and Welham SJ (1993) Genstat 5, Release 3, Reference Manual. Clarendon Press, Oxford

Petrie GA (1979) Blackleg of rape. Canadian Agriculture 24: 22–25

Pierre JG, Regnault Y, Peres A, Penot P, Bonnet T, Thoral N and Tiberghien G (1982) Contribution à la mise au point d'une méthode de plein champ destinée à mesurer la sensibilité des variétés de colza au *Phoma*. Informations Techniques, Centre Technique Interprofessionnel des Oléagineux Métropolitains 81: 3–18

Rempel CB, Lisieczko Z and Hall R (1991) Epidemiological aspects of resistance in rapeseed to blackleg. In: Proceedings of the Eighth International Rapeseed Congress, Vol 2 (pp 460–464) Saskatoon, Saskatchewan

Roy N (1978) Wesreo – a blackleg resistant rape seed. Journal of Agriculture, Western Australia 19: 42

Sansford CE, Fitt BDL, Gladders P, Lockley KD and Sutherland KG (1996) Oilseed rape: disease development, forecasting and yield loss relationships. Home-Grown Cereals Authority Project Report OS17. 185 pp

Sylvester-Bradley R and Makepeace RJ (1985) Revision of a code for stages of development in oilseed rape (*Brassica napus* L.). Aspects of Applied Biology, Field Methods and Data Handling, 10: 395–400

Tiberghien G (1974) Étude de l'efficacité de quelques fongicides contre *Leptosphaeria maculans* (Desm.) Ces. et de Not.: agent de la nécrose du collet du colza. Mémoire de fin d'études, Institute Supérieur d'Agriculture de Lille, Centre Technique Interprofessionnel des Oléagineux Métropolitains. 68 pp

Van der Spek J (1981) Blackleg of oilseed rape in Netherlands. Medelelingen van de Faculteit Landbouwwetenschappen Rijksuniversiteit Gent 46: 813–822