Eur J Plant Pathol (2007) 118:359–373
DOI 10.1007/s10658-007-9137-5

FULL RESEARCH PAPER

Relating plant and pathogen development to optimise fungicide control of phoma stem canker (*Leptosphaeria maculans*) on winter oilseed rape (*Brassica napus*)

Julie M. Steed · Andreas Baierl · Bruce D. L. Fitt

Received: 16 November 2006 / Accepted: 5 April 2007 / Published online: 25 May 2007

Abstract In winter oilseed rape experiments at Rothamsted in 2000/01 to 2002/03 growing seasons, the severity of phoma stem canker epidemics in summer depended on the timing of phoma leaf spot epidemics in the previous autumn, and hence on the timing of *Leptosphaeria maculans* ascospore release. The first major release of *L. maculans* ascospores was earlier in 2000 (26 September) and 2001 (18 September) than in 2002 (21 October). Consequently, the autumn phoma leaf spot epidemic was also earlier in 2000 and 2001 than in 2002. The resulting stem canker epidemics were severe by harvest (July) in 2001 and 2002 but not in 2003. No correlation was found between the severity or duration of phoma leaf spotting (lesion days or lesion °C-days) and the subsequent severity of phoma stem canker epidemics. Rates of leaf production and loss were similar in the three growing seasons. Out of ca. 25 leaves produced on plants during each season, leaf numbers 10–14 generally remained on plants for the longest. Treatment with flusilazole + carbendazim in autumn decreased the severity of phoma leaf spotting for several weeks after treatment, decreased the severity of stem canker the following summer and increased yield significantly in 2001 and 2002 but not in 2003. The most effective timings for flusilazole + carbendazim application were when leaves 7–11 were present on most plants and at least 10% of plants were affected by phoma leaf spot. Two half-dose applications of fungicide reduced phoma stem canker and increased yield more than a single full dose application when phoma leaf spot epidemics were early (<800 °C-days after sowing).

Keywords Blackleg · *Brassica* diseases · Disease management · Fungicide timing · Host–pathogen interactions · Thermal time

J. M. Steed · A. Baierl · B. D. L. Fitt (✉)
Rothamsted Research, Harpenden, Hertfordshire AL5 2JQ, UK
e-mail: bruce.fitt@bbsrc.ac.uk

Introduction

Phoma stem canker (blackleg), caused by *Leptosphaeria maculans*, is a damaging disease of oilseed rape (*Brassica napus*, rapeseed, canola) worldwide (West et al. 2001; Fitt et al. 2006). In Europe, it is the most serious disease of winter oilseed rape, causing estimated annual losses up to € 118M despite autumn fungicide treatment of 65–85% of crops in the UK (harvest years 2000–2002) and up to € 154M in France (Fitt et al. 2006; www.cropmonitor.co.uk). However, the severity of epidemics varies greatly between seasons, regions and individual farm crops (West et al. 1999). These variations result from interactions between weather factors, crop growth and pathogen development. In the UK, the crucial weather factor affecting phoma stem canker epidemics is rainfall in

August/September, which greatly affects the time when the epidemic cycle starts (Gladders and Symonds 1995; Toscano-Underwood et al. 2003). However, there is little information about how interactions between crop growth and pathogen development affect severity of stem canker epidemics.

In Europe, phoma stem canker is a monocyclic disease, with one epidemic cycle per season (Gladders and Musa 1980; Hammond et al. 1985). During autumn and early winter, air-borne ascospores cause leaf lesions (phoma leaf spot) on early leaves. The pathogen then grows down petioles to the crown of the plant and causes cankers at the stem base in spring and summer. It is generally only the stem base canker phase associated with *L. maculans* that causes yield loss in western Europe as phoma leaf spots on later leaves result in less damaging upper stem lesions associated with the less damaging related species *L. biglobosa* (West et al. 1999, 2001; Fitt et al. 2006). Ascospores are first released in autumn after rain and phoma leaf spots appear 14–25 days later (West et al. 2002). Ascospores are able to infect at temperatures ranging from 5 to 24°C, with leaf wetness durations of between 8 and 72 h (Biddulph et al. 1999; Toscano-Underwood et al. 2001). The probability that a phoma leaf lesion will cause a stem canker increases with increasing time between infection and leaf abscission (Hammond and Lewis 1986).

Temperature affects the rate of pathogen growth along the petiole as well as the rates of leaf development and abscission (Hammond and Lewis 1987). The differential effect of temperature on the rates of pathogen progress and leaf senescence determines whether leaf abscission occurs before the pathogen reaches the stem. Occurrence of phoma leaf spot early in the autumn often results in the most severe stem base cankers (Sun et al. 2000) and in glasshouse experiments leaf spots on leaves after leaf 5 did not produce severe stem cankers (Marcroft et al. 2005), although this pattern may be different in farm crops under natural weather conditions. The thermal time (°C-days) between first leaf spots (autumn) and first cankers (spring), was constant over seasons for a given cultivar but differed between cultivars (Sun et al. 2001). However, correlations between severity of phoma leaf spot in autumn and severity of stem canker in summer vary greatly (Sun et al. 2000). Hammond and Lewis (1986) used field experiments to relate the duration of phoma leaf spot lesions, stages of plant development and ambient temperature to subsequent development of stem canker, but the relationships described were complex and it is not clear whether they can be usefully applied to current cultivars on a crop scale.

The need for fungicide application during the phoma leaf spot phase of the epidemic to decrease subsequent severity of stem canker and maximise yield response is recognised in the UK (Gladders et al. 1998; West et al. 1999). It has been established that, in Europe, autumn fungicide sprays control stem canker more effectively than spring sprays (Brazauskiene and Petraitiene 2004; Kruse and Verreet 2005). Penaud (1995) has suggested that adequate protection of French oilseed rape crops requires at least three autumn fungicide applications, but this is uneconomic. It is likely that fewer applications would suffice, but there is much debate about the optimum timing of fungicide applications in relation to plant and pathogen development (West et al. 2002). Optimal timing may also depend on the product, since different products vary in their efficiency as protectant or eradicant fungicides (Gladders et al. 1998). This paper reports a series of field experiments to investigate plant and pathogen development in relation to fungicide timing.

Materials and methods

The winter oilseed rape experiments at Rothamsted in the 2000/01, 2001/02 and 2002/03 growing seasons were all randomised block experiments with three replicates and 20 m × 3 m plots. In 2000/01 and 2001/02, the untreated plot was duplicated in each block. In all experiments, winter oilseed rape (cv. Apex rated 5 for resistance to *L. maculans* and 5 for resistance to *P. brassicae* in the UK on a 1 (susceptible) to 9 (resistant) scale, www.hgca.com) was sown at 80 or 90 seeds m^{-2} (after cereals) and there were three flusilazole + carbendazim (applied as Punch C at 0.8 l ha^{-1}; 300 g a.i. ha^{-1}) treatment timings in autumn: F1 in October/early November when the incidence of plants with phoma leaf spot was about 10%, and F2 and F3 at ca. monthly intervals thereafter (Table 1). Some treatments were combinations of spray timings, for example two half-dose rate applications. One treatment (routine [R]) involved four or five full-rate applications at monthly

Table 1 Dates of sowing, harvest and fungicide application, and leaf numbers present on 30 untreated, marked plants of winter oilseed rape (cv. Apex) on date of flusilazole + carbendazim treatment, in experiments at Rothamsted in 2000/01, 2001/02 and 2002/03

	Treatment code	2000/01	2001/02	2002/03[e]	
				Large plants	Small plants
Sowing date		23 August	14 August	20 August	
Approx. date of emergence		4 September	24 August	1 September	17 September
Date of fungicide treatment	F1[a]	9 October (47 DAS)[f]	22 October (68 DAS)	4 November (76 DAS)	
	F2[a]	6 November (75 DAS)	19 November (97 DAS)	18 December (120 DAS)	
	F3[a]	14 December (113 DAS)	8 January (147 DAS)	16 January (149 DAS)	
	R[b] (routine)	F1 + F2 + F3 + 30 January + 26 February	F1 + F2 + F3 + 5 March	F1 + F2 + F3 + 14 February	
	T[c]	3 April	5 April	18 March	
Leaf numbers present on date of treatment[d]	F1[a]	2–6	3–9	4–11	2–4
	F2[a]	4–10	6–12	8–17	4–10
	F3[a]	7–15	9–15	10–20	7–13
Harvest date		23 July (334 DAS)	18 July (338 DAS)	14 July (328 DAS)	

[a] F1, F2 and F3 all refer to applications of flusilazole + carbendazim, applied as Punch C at 0.8 l ha^{-1}. F1, F2 and F3 were also applied as all combinations of two half-dose rates (i.e. F1 & F2, F1 & F3 and F2 & F3)

[b] R refers to series of 4 or 5 flusilazole + carbendazim applications, each applied as Punch C at 0.8 l ha^{-1}

[c] T refers to tebuconazole, applied as Folicur at 1.0 l ha^{-1}

[d] Leaf numbers present and living on >50% of 30 untreated, marked plants at time of treatment. Leaves were numbered sequentially from 1 (first true leaf). Leaves were assessed as born when large enough to be numbered with indelible pen on the underside (approx. 3 cm long). Leaves were assessed as dead when approx. <10% tissue remained green

[e] In 2002/03, dry weather resulted in two phases of emergence with plants of different sizes; 30 large and 30 small plants were marked and assessed on this original site. However, the fungicide experiment was moved to a new site on the same field and destructive samples were taken from the new experiment

[f] DAS; days after sowing

intervals to maximise control of diseases. Basal treatments included seed treatment (beta-cyfluthrin + imidacloprid + thiram), fertiliser (600–700 kg ha^{-1} Sulphur Gold [30% N, 7.6% S] split between two or three application dates), pre-emergence (Katamaran, 2 l ha^{-1}) and post-emergence (Laser, 0.75 l ha^{-1} or Kerb, 1.7 kg ha^{-1}) herbicides, autumn molluscicides (Genesis, 5 kg ha^{-1}; Judge, 5 kg ha^{-1}, 2000 only) and insecticides (Hallmark, 50 ml ha^{-1}, post-emergence in autumn 2000 and 2002; flowering in spring 2003).

Before the F1 timing, 25 plants were destructively sampled each week along a transect across the experimental area. After the F1 timing, ten plants/plot were sampled monthly throughout the season. Plants were assessed for the presence of diseases on the day they were sampled, unless light leaf spot (*Pyrenopeziza brassicae*) was thought to be present, when the plants were placed in polyethylene bags and incubated for 3–5 days at 10–15°C to induce *P. brassicae* sporulation before assessment to improve accuracy of the diagnosis (Fitt et al. 1998). To control light leaf spot on pods, tebuconazole (T) was applied as Folicur at 1.0 l ha^{-1} each spring to all plots, except those containing marked plants in 2000/01 and 2002/02. Plots were desiccated with diquat (3 l ha^{-1}) in early July, combine harvested in mid to late July and yields (t ha^{-1}) determined.

In addition to these destructive samples, five plants at 2 m intervals along each long side of each of three untreated plots (i.e. 10 plants/plot; one plot/block) were marked at the beginning of the season and individual leaves monitored at ca. weekly intervals as they were produced (born), developed phoma leaf spot and subsequently lost (died). Leaves were assessed as born when large enough to be numbered on the underside (approximately 3 cm long) and dead when <10% tissue remained green. Leaves were

numbered in sequence on the underside with a permanent pen, and at each assessment the number of phoma leaf spot lesions was recorded for each leaf. Leaf 1 was the first true leaf after emergence. Once branching began, only leaves on the main stem were monitored. In 2002/03, a dry period after sowing caused emergence to occur in two distinct phases, resulting in a mixture of early-emerging, large plants and late-emerging, small plants. The fungicide experiment from which destructive samples were taken was therefore moved to another part of the same field where emergence was more uniform. However, 30 large and 30 small plants from the original experimental site were marked and monitored as seasons with two phases of emergence are not uncommon in the UK (five large and five small plants arranged alternately at 1 m intervals down each side of three plots). In each season, marked plants were also assessed for phoma stem canker at 1–3 week intervals from stem extension to harvest using a 0–4 scale (Zhou et al. 1999); 0, no disease; 1, <50% of the stem circumference girdled by lesions; 2, 51–75% of the stem girdled by lesions; 3, >75% of the stem girdled by lesions; 4, plant dead.

In the summers of 2002 and 2003 [GS (growth stage, Sylvester-Bradley and Makepeace, 1985) 5, 5 to 6, 0], the diameter of marked plant stem bases was measured using Mitutoyo Digimatic callipers. In each season, the number of pods/plant on marked plants was estimated at harvest. A Burkard spore sampler (Burkard Manufacturing Co., Rickmansworth, UK; Lacey and West 2006) was located approximately 1 km from the experiment and surrounded by trays containing stems collected from the previous season's untreated plots (Huang et al. 2005). The sampler was operated each season to monitor timing of *L. maculans* ascospore release. Some ascospores collected were those of *L. biglobosa*, which are visually indistinguishable from those of *L. maculans*; *L. maculans* spores are generally released earlier and are most significant in the development of basal phoma stem canker epidemics (Fitt et al. 2006).

Air temperature (°C) was measured by an on-site weather station. For each leaf with number k = 1, 2, 3..., 26 (26 was the maximum number of leaves affected observed in any season), the presence or absence of that leaf on each of the 30 marked plants was recorded. Then the life of leaf 1–26 denoted by D_1, D_2, ..., D_{26} was estimated as:

$$D_k = \frac{1}{m_k}\sum_{j=1}^{m_k} D_{kj}$$

The mean maximum number of lesions which developed on leaf k = 1,2, ..., 26 (L_k) was estimated as:

$$L_k = \frac{1}{m_k}\sum_{j=1}^{m_k} L_{kj}$$

The mean lesion °C-days (thermal time, assuming a base temperature of 0°C) on leaf number k = 1,2, ..., 26 was estimated as:

$$LD_k = \frac{1}{m_k}\sum_{j=1}^{m_k}\sum_{s=1}^{L_{kj}}\sum_{i=1}^{d_{kjs}} LD_{kjsi}\Delta t$$

where m_k = number of plants sampled with leaf number k present (maximum 30 plants); D_{kj} = number of days from birth to death of leaf number k on plant j; L_{kj} = maximum number of lesions which developed on leaf number k of plant j; d_{kjs} = number of days that lesion s was present on leaf number k of plant j; LD_{kjsi} = mean temperature at day i when lesion s was present on leaf number k of plant j; and Δt = 1 day.

Correlation analysis was used on data from marked plants in each of the three seasons to investigate relationships between different measures of phoma leaf spotting and final stem canker severity and also between number of pods/plant and stem diameter. Analysis of variance was applied to data from marked plants to compare final stem canker severity, between different seasons and between small and large plants in 2002/03. Analysis of variance was also used to compare the effect of different fungicide treatments on the incidence and severity of disease and yield. All data analyses were done using the statistical software package GenStat 5 (Payne et al. 1993).

Results

Development of marked plants

Differences between seasons in the rates of leaf birth and death with time affected the number of leaves/

plant (Fig. 1). In the first 70 days after sowing (DAS), the rate of leaf production was similar in 2000/01, 2001/02 and for large plants in 2002/03, with an average of one new leaf produced every 7 days (Fig. 1a). Thereafter, the rate of leaf production was greatest on large plants in 2002/03 and lowest in 2001/02. The rate of leaf production on small plants was lower than on large plants in 2002/03 or in 2001/02 and 2002/03. Up to ca. 200 DAS, the rate of leaf loss was similar in 2000/01, 2001/02 and on large plants in 2002/03, but was slower on small plants in 2002/03. In 2001/02 and 2002/03, when leaf assessments were continued into spring, a rapid increase in leaf loss was observed ca. 200 DAS and from ca. 235 DAS leaf production on the main stem ceased.

Up to the time when leaf 10 (GS 1,10) was present on the majority of plants (70 DAS in 2000/01, 2001/02 and on large plants in 2002/03; 120 DAS on small plants in 2002/03), leaf production was rapid, and leaf loss was negligible (Fig. 1a). Therefore, the mean number of leaves/plant also increased rapidly (Fig. 1b). From this time until ca. 160 DAS, leaf loss occurred at a similar rate to leaf production and the number of leaves/plant remained relatively constant at ca. 6.5 in 2001/02 and on small plants in 2002/03, 7.5 in 2000/01 and 11.5 on large plants in 2002/03. Although leaf production on small plants in 2002/03 was initially slow, by 90 DAS the mean number of leaves/plant was similar to that in 2000/01 and 2001/02 because leaf loss was less. The leaf production and mean number of leaves/plant increased rapidly between 180 and 220 DAS, when stem extension occurred, in 2001/02 but not in 2002/03. The period of rapid leaf loss after 220 DAS coincided with flowering and pod development. In 2000/01, leaf assessments stopped before stem extension, at the beginning of February, because of frost damage.

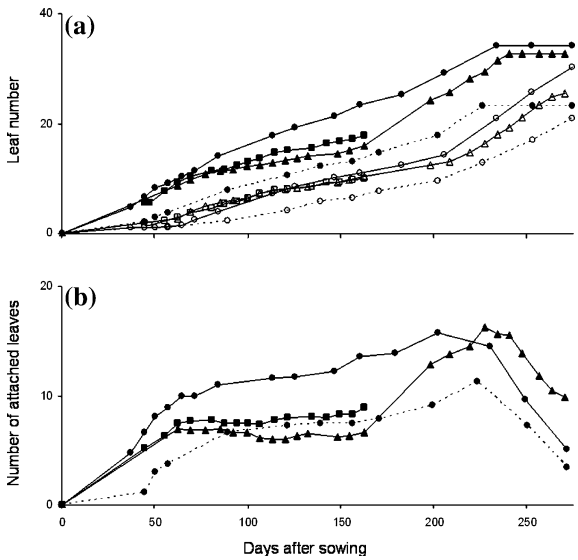

Fig. 1 Leaf development on winter oilseed rape experiments at Rothamsted in 2000/01, 2001/02 and 2002/03. (**a**) Oldest (■▲●) and youngest (□△○) leaf number present in 2000/01 (■□), 2001/02 (▲△) and 2002/03 (●○ (large plants (——), small plants (- - - -)). (b) Number of attached leaves (youngest leaf number − oldest leaf number + 1) in 2000/01 (■), 2001/02 (▲) and 2002/03 (● (large plants (——), small plants (- - - -)). Data are means for 30 untreated, marked plants assessed approximately weekly

As leaf number increased in each season, leaves stayed on the plant for progressively longer periods initially but then for progressively shorter periods towards the end of the season (Table 2). Leaf number 12 (present for 106 days), 14 (for 120 days) and 10 (101 days) were present for the longest times in 2001/02, 2002/03 (large plants) and 2002/03 (small plants), respectively.

In all seasons GS 3,5 (flower buds visible above leaves) was reached ca. 225 DAS (late March/early April). Thereafter, the rate of plant development was similar in 2000/01 and 2001/02, but with pod and seed development occurring approximately 10 days earlier in 2002/03. The growing season (DAS to harvest) was shorter in 2002/03 than 2000/01 or 2001/02 (Table 1). The average number of pods/plant at harvest was 180 (2000/01), 170 (2001/02), 415 (2002/03 large plants) and 150 (2002/03 small plants). The average diameter of stem bases was 1.9 cm in 2001/02 and 2.6 for large plants and 1.4 cm for small plants, respectively, in 2002/03. The number of pods/plant (N) and stem diameter (D) were highly correlated for large ($N = 25.1\ D - 230.0$, $R^2 = 0.72$) and small ($N = 17.0\ D - 88.2$, $R^2 = 0.64$) plants in 2002/03, suggesting that average stem diameter in 2000/01 was similar to that in 2001/02 since pods/plant were not significantly different.

Epidemic development on marked plants

In the autumn of the 2002/03 season, the first major *L. maculans* ascospore release (taken as when >20 spores m^{-3} were collected by the Burkard spore

Table 2 Length of leaf life, maximum number of phoma leaf spot lesions and estimated lesion °C-days for each leaf number from approximately weekly assessments of 30 untreated, marked plants in winter oilseed rape (cv. Apex) experiments at Rothamsted in 2000/01, 2001/02 and 2002/03

Leaf no.[a] (k)	2000/01			2001/02			2002/03[d]					
							Large plants			Small plants		
	Leaf life[b] (D_k)	Max. no. lesions[c] (L_k)	Lesion °C-days [c] (LD_k)	Leaf life[b] (D_k)	Max. no. lesions[c] (L_k)	Lesion °C-days [c] (LD_k)	Leaf life[b] (D_k)	Max. no. lesions[c] (L_k)	Lesion °C-days [c] (LD_k)	Leaf life[b] (D_k)	Max. no. lesions[c] (L_k)	Lesion °C-days[c] (LD_k)
1								0.0	0		0.0	0
2		0.4	37					0.0	0		0.1	15
3		1.0	112		0.6	129		<0.1	9	63	0.4	68
4	44*	2.4	281		1.5	266		0.1	7	62	1.0	152
5	50*	5.5	449		1.8	305		0.4	48	66	1.2	180
6	54*	10.0	1062	60*	1.4	196	62	0.3	43	87	1.6	242
7	64	9.6	1242	64*	1.5	173	71	0.8	112	96	1.4	192
8	76	8.7	1186	75*	1.2	94	79	1.4	183	98	1.6	234
9		7.0	886	89*	1.3	152	87	3.0	379	99	1.1	163
10		4.1	492	102	0.8	80	97	3.2	490	101	1.0	194
11		2.7	238	104	0.3	42	106	4.3	679	94	0.3	70
12		1.1	111	106	0.1	33	115	3.8	713	91	0.6	157
13		1.2	89	97	0.1	13	109	2.8	460	86	0.3	82
14	Frost damage prevented further assessments			88	0.2	15	120	2.3	497	84	0.2	46
15				78	0.1	21	115	2.5	532	73	0.1	10
16				71	<0.1	5	111	1.4	292		0.1	22
17				68	0.2	21	112	1.3	246		0.0	0
18				63	0.1	12	106	0.9	195		<0.1	16
19				64	0.0	0	100	0.9	172		0.0	0
20				68	0.0	0	98	0.3	87		0.0	0
21				70	0.0	0	96	0.3	61		0.0	0
22				73	<0.1	14	98	1.2	222		0.0	0

Table 2 continued

Leaf no.[a] (k)	2000/01			2001/02			2002/03[d]					
							Large plants			Small plants		
	Leaf life[b] (D_k)	Max. no. lesions[c] (L_k)	Lesion °C-days [c] (LD_k)	Leaf life[b] (D_k)	Max. no. lesions[c] (L_k)	Lesion °C-days [c] (LD_k)	Leaf life[b] (D_k)	Max. no. lesions[c] (L_k)	Lesion °C-days [c] (LD_k)	Leaf life[b] (D_k)	Max. no. lesions[c] (L_k)	Lesion °C-days[c] (LD_k)
23				73	0.0	0		0.2	32		0.0	0
24								0.1	24			
25								0.2	36			
26								<0.1	7			

$D_k = \frac{1}{m_k}\sum_{j=1}^{m_k} D_{kj}$ (Mean number of days from birth to death of leaf number k)

$L_k = \frac{1}{m_k}\sum_{j=1}^{m_k} L_{kj}$ (Mean maximum number of lesions which developed on leaf number k)

$LD_k = \frac{1}{m_k}\sum_{j=1}^{m_k}\sum_{s=1}^{L_{kj}}\sum_{i=1}^{d_{kjs}} LD_{kjsi}$ (Mean lesion °C-days on leaf number k)

where m_k = number of plants sampled with leaf number k present (maximum 30 plants); D_{kj} = number of days from birth to death of leaf number k on plant j; L_{kj} = maximum number of lesions which developed on leaf number k of plant j; d_{kjs} = number of days lesion s was present on leaf number k of plant j; LD_{kjsi} = mean temperature at day i when lesion s was present on leaf number k of plant j

[a] Leaves were numbered sequentially from 1 (first true leaf). Leaves were assessed as born when large enough to be numbered with indelible pen on the underside (approximately 3 cm long). Leaves were assessed as dead when approximately <10% tissue remained green

[b] Only those leaf numbers that were observed from birth to death on 30 marked plants are included, except figures marked *, which include estimates of leaf numbers present from 25 destructively sampled plants

[c] Only those leaf numbers for which leaves were observed on all 30 marked plants at least once are included

[d] In 2000/01 and 2001/02 the 30 marked plants were in untreated plots of the fungicide experiment; in 2002/03 the fungicide experiment was moved to another site in the same field with better emergence and marked plants were on the original site. Dry weather had produced two phases of emergence, resulting in plants of different sizes, therefore large plants and small plants were assessed

sampler, which provides an indication of the date when spore concentrations first increased above background concentrations) occurred on 21 October 2002, ca. 30 days later than in the previous seasons (26 September in 2000 and 18 September in 2001) (Fig. 2). At the time of the first major ascospore release, leaf numbers 1–5 were present on >50% of marked plants in 2000/01 and 2001/02, while in 2002/03, leaves 1–10 or 1–4 were present on >50% of large or small marked plants, respectively. In each season, ascospore release continued until at least the end of January with no periods of more than 10 days on which at least 20 spores m^{-3} were not collected. However, the timing of the maximum ascospore release differed between seasons, occurring 58, 85 and 102 DAS in 2000/01, 2001/02 and 2002/03, respectively. The earlier first and maximum ascospore releases in 2000/01 were associated with an early, severe phoma leaf spot epidemic, while the later ascospore releases in 2002/03 were associated with later development of the phoma leaf spot epidemic (Fig. 3). The development of lesions on marked plants (Fig. 3a) was similar to that on plants sampled from plots (Fig. 3b). In 2001/02, the early first ascospore release was associated with an early phoma leaf spot epidemic (October), but the maximum % leaves with phoma leaf spot was less than in the other two seasons. The number of lesions/leaf on marked plants also differed between seasons, with maxima of 2.8 (30 November), 0.5 (12 December),

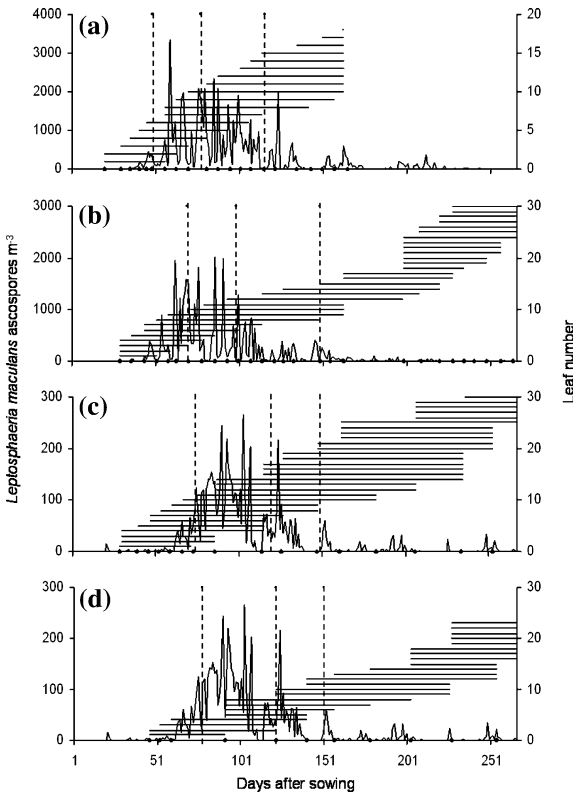

Fig. 2 Daily concentration of *Leptosphaeria maculans* ascospores collected by a Burkard spore sampler surrounded by oilseed rape stems from the previous season's untreated plots and situated approximately 1 km from the field experiment (——); times of assessment of 30 untreated, marked plants (♦) and length of time between assessments when leaf numbers up to 30 were present on ≥15 out of 30 untreated, marked plants (——) from up to 260 DAS, in relation to time of flusilazole + carbendazim applications (¦), in 2000/01 (assessments stopped 160 DAS because of frost damage) (**a**), 2001/02 (**b**) and 2002/03 (two phases of emergence) on large plants (**c**) and small plants (**d**). For clarity, different scales have been used on some primary and secondary y-axes

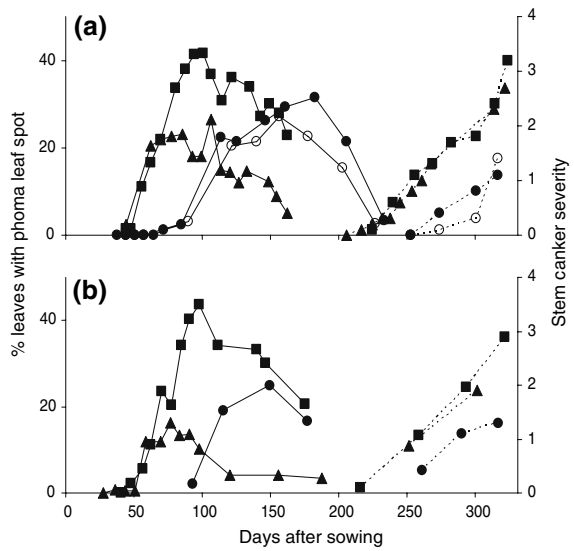

Fig. 3 Development of phoma leaf spot and stem canker on marked (**a**) and destructively sampled (**b**) plants in untreated winter oilseed rape (cv. Apex) experiments at Rothamsted in 2000/01, 2001/02 and 2002/03. (**a**) Shows % leaves affected by phoma leaf spot (——) and stem canker score (0–4 scale) (- - - -) on 30 marked plants in 2000/01 (■), 2001/02 (▲), and 2002/03 (two phases of emergence) large plants (●) and small plants (○). (**b**) Shows % leaves affected by phoma leaf spot (——) and final stem canker score (0–4 scale) (- - - -) on 25 or 30 destructively sampled plants in 2000/01 (■), 2001/02 (▲) and 2002/03 (●). In 2002/03, poor emergence meant that the fungicide experiment was moved to a different site in the same field, so marked and sampled plants were from different untreated plots

0.8 and 0.6 (31 January) in 2000/01, 2001/02 and on large and small plants in 2002/03, respectively.

In 2000/01 and 2001/02, when the timing of leaf development and the start of ascospore release were similar, the relative contributions made by different leaf numbers (layers) to the overall phoma leaf spot epidemic (in lesion °C-days) were also similar (Fig. 4a, b). In these two seasons, leaf numbers 3–10 were most severely affected (Table 2). In 2002/03, when large plants developed quickly and ascospore release was late, early leaves (numbers 1–6) developed little disease with leaf number 12 the most severely affected (Fig. 4c). On small plants in this season, the rate of leaf development was slow and the severity of phoma leaf spotting on different leaf numbers was similar to those in 2000/01 and 2001/02 despite the later ascospore release (Fig. 4d). The relative contribution of each leaf number to the phoma leaf spot epidemics differed between seasons, but later leaves tended to make a larger contribution to the phoma leaf spot epidemic when the contribution was measured as number of lesion °C-days (Table 2).

Development of phoma stem canker on marked plants (Fig. 3a) and destructively sampled plants (Fig. 3b) was similar in all three seasons. The early, severe phoma leaf spot epidemic in 2000/01 was associated with severe stem base canker at harvest. The early, less severe phoma leaf spot epidemic in 2001/02 was also associated with severe stem canker. The mean final stem canker score on marked plants was 3.2 in 2000/01 and 2.7 in 2001/02, and the timing and rate of canker development was similar in the two seasons (Fig. 3a). Stem canker severity in 2002/03, when the phoma leaf spot epidemic was later than in the previous two seasons, was considerably less than in 2000/01 or 2001/02 (Fig. 3). Stem canker severity was not significantly different between large

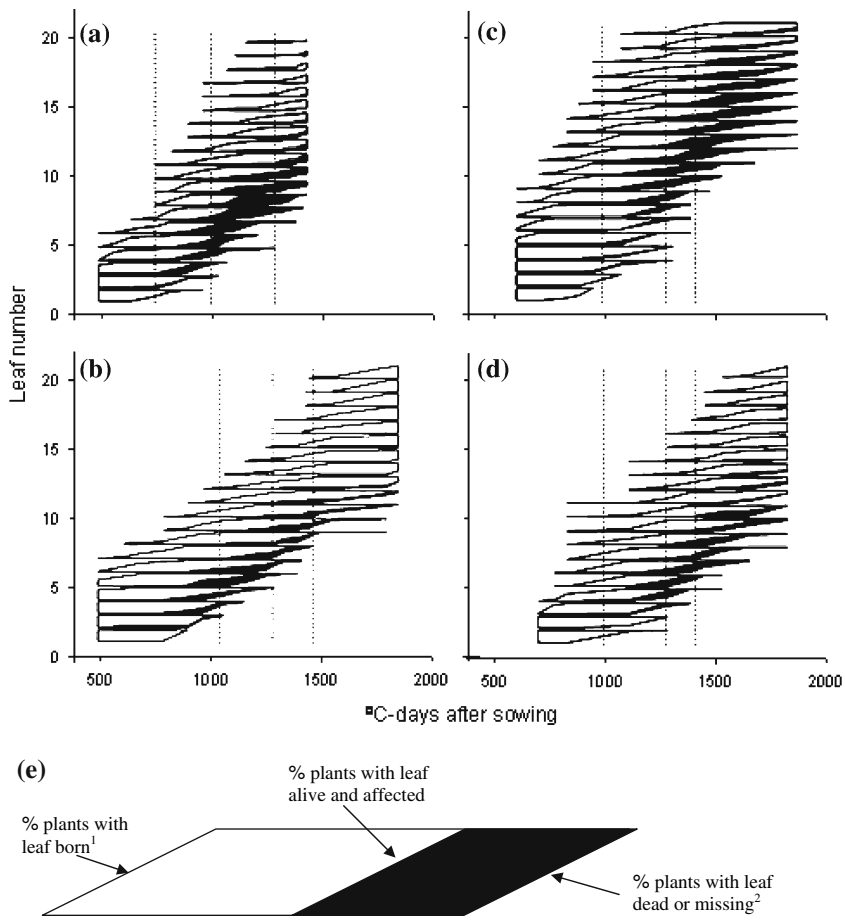

Fig. 4 Thermal time from sowing to birth, development of phoma leaf spot, and death on leaf numbers 1–20 of 30 untreated, marked plants, in relation to time of flusilazole + carbendazium treatments, on winter oilseed rape (cv. Apex) experiments at Rothamsted in 2000/01 (**a**), 2001/02 (**b**) and 2002/03 (two phases of emergence) on large plants (**c**) and small plants (**d**). Vertical lines (¦) indicate timing of fungicide sprays F1, F2 and F3. (**e**) Individual graphs for each leaf number in each season having the components: (1) lines are vertical when some of the leaves were already dead or missing at the first assessment, (2) lines are vertical when some of the leaves were still present 1900 °C-DAS. The area shaded black corresponds to % plants with that leaf (leaf number) multiplied by °C-days that the leaves were affected with phoma spots

and small plants in 2002/03. Analysis of the relationship between different measures of phoma leaf spotting (lesion days, lesion °C-days, and days and °C-days with >1 lesion present per plant) and stem canker severity on marked plants did not demonstrate any significant correlation between severity or duration of phoma leaf spot and severity of stem base canker for any season in these experiments.

Effect of timing of flusilazole + carbendazim spray on phoma leaf spot and stem canker epidemics

In 2000/01, treatment F1 (9 October) was associated with a decrease in phoma leaf spotting at 2 and 5 weeks after application, but there was no difference between treated and untreated plots by January (Table 3). In 2001/02, plots receiving treatment F1 (22 October) had less phoma leaf spotting in November, December and January than untreated plots. In 2002/03, plots receiving treatment F1 (4 November) had less phoma leaf spotting from November to January than the untreated plots but there was no difference by February. In 2000/01, plots receiving treatment F2 (6 November) had fewer leaves affected by phoma leaf spot in the period January to March than untreated plots. In 2001/02, plots receiving treatment F2 (19 November) had <50% the number of leaves affected by phoma leaf spot in December and January than untreated plots, but there was no difference by February. In 2002/03, plots receiving treatment F2 (18 December) did not differ from untreated plots in the % leaves affected. In 2000/01, plots receiving treatment F3 (14 December) had significantly less phoma leaf spot in February and March than untreated plots. In 2001/02, plots receiving treatment F3 (8 January) did not differ from untreated plots in phoma leaf spot incidence. In 2002/03, plots receiving treatment F3 (16 January) did not differ from untreated plots in February, but did in March. In each season, the two half-rate applications, especially those including the F3 timing, decreased severity of phoma leaf spot by more than single full-rate applications from January onwards. Routine fungicide applications greatly decreased the % leaves with phoma leaf spotting in all three seasons.

Flusilazole + carbendazim treatments generally decreased the severity of stem canker in June in all three seasons (Table 3). The two half-rate applications were sometimes more effective than single full-rate applications, except in 2001/02, and the routine applications decreased canker severity most. By July, differences in the severity of stem canker between fungicide and control plots were less, especially in 2003 when stem canker was not as severe as in other seasons. Fungicide treatments increased yield significantly in 2000/01 and 2001/02 but not in 2002/03 (Table 3). In 2000/01 and 2001/02, the yield responses from autumn flusilazole + carbendazim plus spring tebuconazole treatments were much greater than from tebuconazole alone, and yields were greatest in plots with routine fungicide application.

Effect of fungicides on light leaf spot

By February or March in each season, light leaf spot (*Pyrenopeziza brassicae*) affected 12–15% leaf area on untreated plants (data not shown). All autumn flusilazole + carbendazim treatments decreased leaf area affected by light leaf spot by more than 50%. Tebuconazole was applied each spring to control epidemics of light leaf spot on pods. Severe light leaf spot epidemics did occur on pods in 2000/01 and 2001/02, and ca. 10% pod area was affected on untreated plants. The spring tebuconazole treatment decreased the % plants with light leaf spot on pods by approximately 50%, whereas the autumn applications of flusilazole + carbendazim had little effect.

Discussion

These experiments suggest that the timing of phoma leaf spotting in autumn has a greater influence on the severity of subsequent phoma stem canker epidemics than either the severity or duration of phoma leaf spot epidemics. Thus, severe stem canker epidemics developed in 2000/01 and 2001/02 when leaf spots appeared in October but not in 2002/03 when they appeared in November. This conclusion is supported by data from the 2003/04 growing season, in which major *L. maculans* ascospore release and associated leaf spotting did not occur until December (Pirie et al.

Table 3 Effect of flusilazole (F) + carbendazim (C), applied at different times in the autumn, and tebuconazole (T), applied in the spring, on phoma leaf spot and stem canker in plants destructively sampled from winter oilseed rape (cv. Apex) experiments at Rothamsted in 2000/01, 2001/02 and 2002/03

Fungicide treatment[a]	% leaves with phoma leaf spot						Stem canker				Yield (t ha^{-1})
							% plants		Severity score (0–4)		
2000/01	24 Oct	13 Nov	8 Jan	13 Feb	26 Mar	8 May	13 June	10 July	13 June	10 July	
Untreated[c]	11.2	33.5	33.3	14.7	15.9						2.91
Unt. + T						1.8	100	98.1	2.0	2.9	4.04
F1 + T	3.1	1.3	33.0	9.1	19.2	2.4	96.7	100	1.3	2.2	4.26
F2 + T		29.5	20.5	8.6	10.6	3.0	70.0	96.7	0.9	2.0	4.72
F3 + T			31.6	4.6	7.8	1.0	80.0	90.0	0.9	1.6	4.73
F1&2 + T	4.2[b]	12.5	28.4	8.2	14.5	2.4	100	96.7	1.7	2.2	4.84
F1&3 + T		13.4[b]	26.4	8.2	6.9	3.5	80.0	80.0	1.1	1.1	4.88
F2&3 + T		34.6[b]	22.6	3.9	6.6	2.3	80.0	76.7	0.9	1.0	4.77
R + T		3.6	19.7	6.4	0.0	2.0	0.0	6.7	0.0	0.1	5.24
SED	1.2	2.8	3.3	3.6	2.8	1.0	9.0	7. 6	0.2	0.4	0.27
df	4	12	14	17	14	14	14	13	14	13	16
2001/02	12 Nov	19 Nov	12 Dec	16 Jan	15 Feb	18 April	11 June		11 June		
Untreated[c]	13.7	10.3	4.1	4.2	3.5	1.7	90.0		2.0		2.82
Unt. + T							90.0		1.9		3.55
F1 + T	8.8	3.1	0.6	1.1	6.2	2.1	56.7		0.6		4.30
F2 + T			1.8	1.1	6.3	2.3	43.3		0.5		4.09
F3 + T				2.4	2.6	1.5	46.7		0.4		4.17
F1&2 + T	9.7[b]	3.5[b]	1.5	0.5	3.8	4.6	53.3		0.6		4.37
F1&3 + T			0.0[b]	1.1	2.4	4.2	50.0		0.5		4.37
F2&3 + T			5.1[b]	1.0	1.6	2.2	30.0		0.3		4.47
R + T			0.0	0.5	0.0	0.0	6.7		0.1		4.83
SED	3.4	2.4	2.4	1.7	1.9	0.9	14.3		0.2		0.22
df	4	4	14	14	14	14	16		16		16
2002/03	20 Nov	13 Dec	16 Jan	12 Feb	24 Mar	7 May	5 June	2 July	5 June	2 July	
Untreated[c]	2.3	19.2	24.9	16.8							
Unt. + T					13.4	0.8	86.7	96.7	1.1	1.3	3.06
F1 + T	0.8	2.2	5.5	14.9	13.1	0.3	60.0	76.7	0.7	0.9	3.05
F2 + T			18.5	10.4	13.1	0.3	40.0	83.3	0.5	0.9	3.31

Table 3 continued

Fungicide treatment[a]	% leaves with phoma leaf spot						Stem canker			Yield (t ha^{-1})
							% plants	Severity score (0–4)		
F3 + T				17.4	4.0	0.0	40.0	73.3	0.4	0.9	3.18
F1&2 + T	1.7[b]		6.5	4.1	13.3	0.5	36.7	70.0	0.5	0.8	3.13
F1&3 + T				6.0	5.2	0.3	43.3	53.3	0.6	0.6	3.33
F2&3 + T				9.3	2.8	0.0	26.7	43.3	0.3	0.6	3.22
R + T				1.8	0.8	1.1	10.0	16.7	0.1	0.2	3.16
SED	1.6	2.2	2.7	3.5	2.1	0.7	12.0	13.7	0.2	0.2	0.13
df	4	2	6	14	14	14	14	14	14	14	14

[a] See Table 1 for dates of fungicide treatments

[b] Only the first of two half-dose rate treatments applied by this date

[c] In 2000/01 and 2001/02 untreated plots were duplicated initially, then one received a tebuconazole treatment in spring; in 2002/03 poor emergence on the original site meant that the fungicide experiment was moved and had only one untreated plot, which received a tebuconazole treatment in spring

2005) and the subsequent stem canker epidemic was less severe nationally (27% plants affected) than in any other season in the previous 10 years (mean 43% plants affected in 1995–2004 [www.cropmonitor.co.uk]). Furthermore, the large differences in severity and duration of phoma leaf spot epidemics between 2000/01 and 2001/02 did not result in large differences in the severity of subsequent stem canker epidemics. Hence a measure of phoma leaf lesion severity/duration, such as lesion °C-days (Hammond and Lewis 1986), was not useful to predict the severity of stem canker epidemics. This provides further evidence that phoma stem canker is a monocyclic disease in Europe, with the severity of epidemics determined by the timing of ascospore release in autumn.

In these experiments, phoma leaf spotting on middle leaves in autumn appeared to make the most important contribution to the development of stem base canker epidemics in the following spring/summer. Infections on the earliest leaves may not contribute much to stem base canker epidemics if these leaves senesce before the pathogen has grown into the stem (Hammond and Lewis 1986), while infections on the latest leaves do not result in stem base cankers (West et al. 2001; Marcroft et al. 2005). Severe basal stem cankers are associated only with phoma leaf spotting on basal leaves whilst leaf spots on later leaves taken up the plant during stem extension result in less damaging upper stem lesions (Sun et al. 2000). Thus there would have been no need to apply autumn fungicides to control phoma stem canker in the 2003/04 growing season, when phoma leaf spotting appeared even later than in 2002/03 (Pirie et al. 2005).

These results suggest that the effectiveness of fungicide sprays to control phoma stem canker on winter oilseed rape depends on the timing of application in relation to plant and disease development, assessed in thermal time (°C-days) after sowing (Fig. 5). They suggest that a single autumn spray of flusilazole + carbendazim can provide effective control of phoma stem canker in growing seasons with short phoma leaf spot epidemics which start in mid-autumn (800–1300 °C-DAS). They suggest that the optimal timing for a single fungicide application is when plants have reached at least GS 1,7 to 1,11 with at least 10% of plants affected by phoma leaf spot (Fig. 5). In 2000/01, the 6 November (F2) and 14

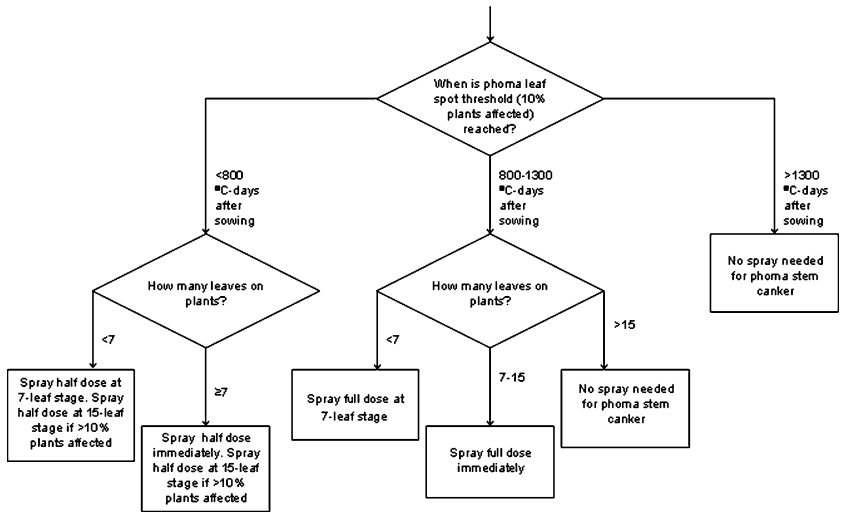

Fig. 5 Scheme showing optimal timings for autumn fungicide applications to control phoma stem canker in winter oilseed rape (cv. Apex), depending on the timing of phoma leaf spot epidemic and crop growth stage (number of leaves present). For farm crops where the data necessary to calculate °C-days are lacking (i.e. no local records), approximate thresholds for times after sowing are: 800 °C-days ≈ 50–60 days; 1300 °C-days ≈ 100–135 days

December (F3) sprays both gave yield responses and in 2001/02 the optimal date for spraying was 22 October (F1). In both seasons, these sprays controlled the disease on leaves 7–11 (Fig. 4a, b) at a time when the incidence of phoma leaf spotting was increasing rapidly (Fig. 2). By the time this application ceases to be effective, new leaf infections are unlikely to produce severe basal stem cankers (Sun et al. 2000). By contrast, in 2000/01 the 9 October (F1) application was too early to give optimal control of stem canker because there was little leaf spotting at that time (Fig. 3).

These experiments provide evidence that a series of sprays of fluzilazole + carbendazin gives the best control of stem canker and hence the greatest yield response in seasons with early phoma leaf spot epidemics (<800 °C-DAS). In both 2000/01 and 2001/02, the greatest yield responses resulted from the routine treatment (Table 3), which included at least four autumn/winter applications of flusilazole + carbendazim (Table 1). Similarly, West et al. (2002) found that the decrease in the severity of phoma stem canker epidemics was greatest when five sprays of difenoconazole + carbendazim were applied, with reduced control as the number of sprays decreased. However, the additional yield increase relative to programmes with only one or two sprays is unlikely to be economic. Nevertheless, a split dose two-spray application programme is optimal when phoma leaf spot epidemics persist too long for all the necessary leaf layers to be protected by a single spray. Since the duration of a phoma leaf spot epidemic cannot be predicted early in the season, two half-dose fungicide applications are advisable whenever the threshold level of leaf spotting (>10% plants affected) is reached earlier than 800 °C-DAS (Fig. 5). In this study the duration of the phoma leaf spot epidemic was longest in 2000/01 (Fig. 3). In this season, the split October/December and November/December spray programmes gave better control of stem canker and greater yield responses than any of the single applications of flusilazole + carbendazim (Table 3). There was also a yield response to the two-spray programmes in 2001/02, when the duration of the phoma leaf spot epidemic was shorter (Fig. 3), but this may have been due to the control of diseases other than phoma stem canker, since the severity of stem canker was not decreased further by a second application (Table 3).

These results suggest that in growing seasons with very late phoma leaf spot epidemics (starting >1300 °C-DAS), the application of fungicides to control stem canker is unnecessary (Fig. 5). Flusilazole + carbendazim applications gave little yield increase when the phoma leaf spot epidemic was late (2002/03) by contrast with the two seasons when it was early (2000/01 and 2001/02) resulting in severe stem canker in untreated plots (Table 3). A similar pattern has been observed with difenoconazole + carbendazim (West et al. 2002). These results also suggest that fungicide application before the appearance of leaf spotting does not control phoma stem canker.

The 4 November (F1) application in 2002/03, applied before leaf spotting appeared, had no effect on yield, whereas the later sprays had a small effect (Table 3). This is in contrast to the control of light leaf spot in winter oilseed rape, whereby effective control requires autumn spraying before symptoms are apparent, as the disease has a long symptomless phase (Gilles et al. 2000).

Other diseases, particularly light leaf spot, and the growth regulatory effects of fungicides are also important considerations in optimising the timing of fungicide application on UK winter oilseed rape crops. In this study, a spring application of tebuconazole to control light leaf spot on pods gave substantial yield responses in two seasons. Spring applications of tebuconazole also slow stem extension and hence reduce lodging, which had as great an effect on yield as controlling phoma stem canker in Germany (Kruse and Verreet 2005). Since the timing of pathogen ascospore release from oilseed rape debris to initiate phoma stem canker and light leaf spot epidemics are both largely controlled by temperature and rainfall (Gilles et al. 2000; Toscano-Underwood et al. 2003), there is likely to be a correlation between the timings of the two disease epidemics. However, given that the optimal fungicide timing in relation to development of these two diseases is different, growers should prioritise the control of the dominant disease in their area.

Acknowledgements This work was supported by the Department for Environment, Food and Rural Affairs (DEFRA) and the Home-Grown Cereals Authority (HGCA) as a Sustainable Arable LINK project (PASSWORD), and the Biotechnology and Biological Sciences Research Council (BBSRC). We thank Yong-Ju Huang, Neal Evans and Maria Eckert for assistance with disease assessments, Alan Todd for some of the statistical analyses and Susan Roques for assistance with preparing the paper.

References

Biddulph, J. E., Fitt, B. D. L, Leech, P. K., Welham, S. J., & Gladders, P. (1999). Effects of temperature and wetness duration on infection of oilseed rape leaves by ascospores of *Leptosphaeria maculans* (stem canker). *European Journal of Plant Pathology, 105*, 769–781.

Brazauskiene, I., & Petraitiene, E. (2004). Disease incidence and severity of phoma stem canker (*Phoma lingam*) on winter oilseed rape (*Brassica napus* L.) in Lithuania as affected by different prochloraz and tebuconazole application times. *Journal of Plant Diseases and Protection, 111*, 439–450.

Fitt, B. D. L., Brun, H., Barbetti, M. J., & Rimmer, S. R. (2006). Worldwide importance of phoma stem canker (*Leptosphaeria maculans* and *L. biglobosa*) on oilseed rape (*Brassica napus*). *European Journal of Plant Pathology, 114*, 3–15.

Fitt, B. D. L., Doughty, K. J., Gladders, P., Steed, J. M., & Sutherland, K. G. (1998). Diagnosis of light leaf spot (*Pyrenopeziza brassicae*) on winter oilseed rape (*Brassica napus*) in the UK. *Annals of Applied Biology, 133*, 155–166.

Gilles, T., Evans, N., Fitt, B. D. L., & Jeger, M. J. (2000). Epidemiology in relation to methods for forecasting light leaf spot (*Pyrenopeziza brassicae*) severity on winter oilseed rape (*Brassica napus*) in the UK. *European Journal of Plant Pathology, 106*, 593–605.

Gladders, P., & Musa, T. M. (1980). Observations on the epidemiology of *L. maculans* stem canker in winter oilseed rape. *Plant Pathology, 29*, 28–37.

Gladders, P., & Symonds, B. V. (1995). Occurrence of canker (*Leptosphaeria maculans*) in winter oilseed rape in eastern England 1977–1993. *International Organization for Biological Control Bulletin, 18*, 1–11.

Gladders, P., Symonds, B. V., Hardwick, N. V., & Sansford, C. E. (1998). Opportunities to control canker (*Leptosphaeria maculans*) in winter oilseed rape by improved spray timing. *International Organization for Biological Control Bulletin, 21*, 111–120.

Hammond, K. E., & Lewis, B. G. (1986). The timing and sequence of events leading to stem canker disease in populations of *Brassica napus* var. *oleifera* in the field. *Plant Pathology, 35*, 551–564.

Hammond, K. E., & Lewis, B. G. (1987). Variation in stem infections caused by aggressive and non-aggressive isolates of *Leptosphaeria maculans* on *Brassica napus* var. *oleifera*. *Plant Pathology, 36*, 53–65.

Hammond, K. E., Lewis, B. G., & Musa, T. M. (1985). A systemic pathway for the infection of oilseed rape plants by *Leptosphaeria maculans*. *Plant Pathology, 34*, 557–565.

Huang, Y. J., Fitt, B. D. L., Jedryczka, M., Dakowska, S., West, J. S., Gladders, P., Steed, J. M., & Li, Z. Q. (2005). Patterns of ascospore release in relation to phoma stem canker epidemiology in England (*Leptosphaeria maculans*) and Poland (*Leptosphaeria biglobosa*). *European Journal of Plant Pathology, 111*, 263–277.

Kruse, T., & Verreet, J. A. (2005). Epidemiological studies on winter oilseed rape (*Brassica napus* L. var. *napus*) infected by *Phoma lingam* (teleomorph *Leptosphaeria maculans*) and the effects of different fungicide applications with Folicur® (tebuconazole). *Journal of Plant Diseases and Protection, 112*, 17–41.

Lacey, M. E., & West, J. S. (2006). *The air spora: A manual for catching and identifying biological particles*. Dordrecht, The Netherlands: Springer.

Marcroft, S. J., Sosnowski, M. R., Scott, E. S., Ramsey, M. D., Salisbury, P. A., & Howlett, B. J. (2005). *Brassica napus* plants infected by *Leptosphaeria maculans* after the third to fifth leaf growth stage in south-eastern Australia do not

develop blackleg stem canker. *European Journal of Plant Pathology, 112*, 289–292.

Payne, R. W., Lane, P. W., Digby, P. G. N., Harding, S. A., Leech, P. K., Morgan, G. W., Todd, A. D., Thompson, R., Tunnicliffe, W. G., Welham, S. J., & White, R. P. (1993). *Genstat 5, release 3, reference manual*. Oxford, UK: Clarendon Press.

Penaud, A. (1995). Study on the optimum timing to control rapeseed blackleg caused by *Leptosphaeria maculans* with fungicides. *International Organization for Biological Control Bulletin, 18*, 56–61.

Pirie, E. J., Huang, Y. J., Evans, N., & Fitt, B. D. L. (2005). Resistance to *Leptosphaeria maculans* in *Brassica napus* (oilseed rape) leaves (phoma leaf spot) and stems (canker). *Proceedings of BCPC International Congress – Crop Science and Technology, 2005*, 471–474.

Sun, P., Fitt, B. D. L., Gladders, P., & Welham, S. J. (2000). Relationships between phoma leaf spot and development of stem canker (*Leptosphaeria maculans*) on winter oilseed rape (*Brassica napus*) in southern England. *Annals of Applied Biology, 137*, 113–125.

Sun, P., Fitt, B. D. L., Steed, J. M., Underwood, C. T., & West, J. S. (2001). Factors affecting development of phoma canker (*Leptosphaeria maculans*) on stems of winter oilseed rape (*Brassica napus*) in southern England. *Annals of Applied Biology, 139*, 227–242.

Sylvester-Bradley, R., & Makepeace, R. J. (1985). Revision of a code for stages of development in oilseed rape (*Brassica napus*). *Aspects of Applied Biology, 10*, 395–400.

Toscano-Underwood, C., Huang, Y. J., Fitt, B. D. L., & Hall, A. M. (2003). Effects of temperature on maturation of pseudothecia of *Leptosphaeria maculans* and *L. biglobosa* on oilseed rape stem debris. *Plant Pathology, 52*, 726–736.

Toscano-Underwood, C., West, J. S., Fitt, B. D. L., Todd, A. D., & Jedryczka, M. (2001). Development of phoma lesions on oilseed rape leaves inoculated with ascospores of A-group or B-group *Leptosphaeria maculans* (stem canker) at different temperatures and wetness durations. *Plant Pathology, 50*, 28–41.

West, J. S., Biddulph, J. E., Fitt, B. D. L., & Gladders, P. (1999). Epidemiology of *Leptosphaeria maculans* in relation to forecasting stem canker severity on winter oilseed rape in the UK. *Annals of Applied Biology, 135*, 535–546.

West, J. S., Fitt, B. D. L., Leech, P. K., Biddulph, J. E., Huang, Y. J., & Balesdent, M. H. (2002). Effects of timing of *Leptosphaeria maculans* ascospore release and fungicide regime on phoma leaf spot and phoma stem canker development on winter oilseed rape (*Brassica napus*) in southern England. *Plant Pathology, 51*, 454–463.

West, J. S., Kharbanda, P., Barbetti, M. J., & Fitt, B. D. L. (2001). Epidemiology and management of *Leptosphaeria maculans* (phoma stem canker) in Australia, Canada and Europe. *Plant Pathology, 50*, 10–27.

Zhou, Y., Fitt, B. D. L., Welham, S. J., Gladders, P., Sansford, C. E., & West, J. S. (1999). Effects of severity and timing of stem canker (*Leptosphaeria maculans*) symptoms on yield of winter oilseed rape (*Brassica napus*) in the UK. *European Journal of Plant Pathology, 105*, 715–728.