
**PROJECT REPORT No. OS51**

# IMPROVING STEM CANKER CONTROL IN WINTER OILSEED RAPE BY ACCURATE TIMING OF FUNGICIDE APPLICATIONS BASED ON DISEASE FORECASTS

JULY 2001

Price: £8.30

**PROJECT REPORT No. OS51**

# IMPROVING STEM CANKER CONTROL IN WINTER OILSEED RAPE BY ACCURATE TIMING OF FUNGICIDE APPLICATIONS BASED ON DISEASE FORECASTS

By

P GLADDERS[1], M R GREEN[2], J M STEED[3], J S WEST[3]
and B D L FITT[3]

[1]ADAS Boxworth, Battlegate Road, Boxworth, Cambs. CB3 8NN
[2]ADAS High Mowthorpe, Duggleby, Malton, North Yorks. YO17 8BP
[3]IACR Rothamsted, Harpenden, Herts. AL5 2JQ

This is the final report of a three year project, which started in April 1998.
This work was funded by a grant of £158,571 from HGCA (project 1866).



**PROJECT REPORT No. OS51E**

**IMPROVING STEM CANKER CONTROL IN WINTER OILSEED RAPE BY ACCURATE TIMING OF FUNGICIDE APPLICATIONS BASED ON DISEASE FORECASTS**

by

**Contents**

**Abstract**

This three year project focused on improving strategies for control of canker (*Leptosphaeria maculans*) in winter oilseed rape. There were three main objectives, which were addressed using historical data and field experiments: a) to quantify yield losses in relation to the development of phoma leaf spot and the severity of stem canker epidemics, b) to identify factors affecting ascospore discharge and hence phoma leaf spot attacks and c) to optimise fungicide timing for stem canker control and yield response.

Canker incidence at harvest was related to the maximum incidence of phoma leaf spot up to 100 days from sowing and maximum incidence of phoma leaf spot up to stem extension, on four cultivars. A second relationship between phoma leaf spot pre-flowering and phoma stem lesions at harvest was also demonstrated, but this was not applicable across cultivars. The development of canker in spring was related to the development of phoma leaf spot in the previous autumn/winter. The thermal time (degree-days) from the first appearance of phoma leaf spot (autumn) to the first appearance of crown canker (spring) varied between cultivars and averaged 1220-1240 degree-days for cvs Lipton and Capitol and 1120-1140 for cv. Apex. The relationships between % yield loss and % plants with different stem canker severity scores at different growth stages showed that the greatest yield losses were associated with the largest severity scores and were also associated with the early development of stem lesions. Air-borne ascospores of *Leptosphaeria maculans* were present from autumn (September/October) to spring (April/May). Ascospores were first detected after 16-23 days with rain from 1 August and this varied by three weeks between years in this project. Phoma leaf spotting appeared in untreated field plots 14-25 days after ascospores were first detected in autumn.

Control of canker and light leaf spot was investigated using sprays of difenoconazole + carbendazim applied to cv. Apex at four main timings with all combinations of one to four applications. Secondary treatments examined the impact of delaying the first or last spray by two or four weeks. Canker was more severe in 2000 than in 1999, reflecting the earlier phoma leaf spot epidemic in autumn and was controlled by various multiple spray programmes. Significant yield responses (16-31%) were recorded in four out of six experiments, the larger responses being associated with control of light leaf spot and canker. Optimum timings for fungicides differed between sites and years and a two spray programme provided more consistent benefits than single sprays. Early phoma leaf spot epidemics are a major cause of yield loss but can be managed economically with fungicides applied in autumn and winter. Conversely, phoma epidemics developing from December onwards may have little effect on yield. The project has improved understanding of when canker causes yield loss and this will lead to better assessment of the need to apply fungicides.

## Summary

Stem canker (*Leptosphaeria maculans*) was the most serious disease on winter oilseed rape in England and Wales in the 1995/96 season, causing losses of £29M (by comparison with £13.3M for light leaf spot (*Pyrenopeziza brassicae)*), despite expenditure of *c.* £5 million on fungicides to control the disease. Furthermore, losses from stem canker were estimated to be an average of £38M per annum for the harvest years 1993-1995 using ADAS/CSL disease survey data and yield loss formulae derived from results of HGCA/MAFF-funded experiments in 1991-1994.

Fungicide timing for control of stem canker needs to be improved. Good disease control has been achieved in ADAS and Rothamsted experiments with multiple spray treatments. These provide a theoretical basis for stem canker control, but there is now a need to develop practical disease management programmes for farms. It is also important to ensure that fungicides are applied only to those crops which need treatment. This project aims to improve strategies for control of stem canker on winter oilseed rape through optimising fungicide timing and choice, based on a clear understanding of the critical factors determining stem canker severity and associated yield losses.

**Objectives:**

a) To quantify yield losses in relation to the development of phoma leaf spot and the severity of stem canker epidemics.

b) To identify factors affecting ascospore discharge and hence phoma leaf spot attacks.

c) To optimise fungicide timing for stem canker control and yield response.


**Relationships between phoma leaf spot, canker and yield loss**

The biological model for phoma development in oilseed rape is considered to be monocyclic, with air-borne spores (ascospores) from infected oilseed rape stubbles producing phoma leaf spots in winter oilseed rape crops during the autumn and winter. Subsequently, the canker fungus grows down within the leaf petiole and invades the stem. After a period of about six months, sunken brown lesions (cankers) appear at the stem base which are capable of causing crop lodging and premature ripening if they girdle the stem. Phoma also causes stem lesions well above the stem base region associated with leaves infected in late winter or spring and these are termed ‘phoma stem lesions’. The development of phoma within the plant has been described, but requires field validation and quantification on current cultivars.

Phoma leaf spot - stem canker relationship

The relationships between the percentage of plants with phoma leaf spot at different growth stages in autumn/winter or early spring and incidence of stem canker (basal canker or stem lesions) in summer on winter oilseed rape in southern England were described by the model: $y_1 = \beta_0 + \beta_1 x_1 + \beta_2(x_2 - x_1)$ if $x_2 > x_1$, and $y_1 = \beta_0 + \beta_1 x_1$ if $x_2 \leq x_1$, in which $y_1$ was the incidence (% plants affected) of basal canker at harvest, $x_1$ was the maximum incidence of phoma leaf spot during the period from sowing to growth stage (GS) 1,6-1,7 (about 100 days after sowing) and $x_2$ was the maximum incidence of phoma leaf spot between GS 1,7 and GS 2,0 (start of stem extension). A second model which described the relationship between phoma leaf spot and phoma stem lesions was identified as $y_2 = \alpha_0 + \alpha_1 x_3 + \alpha_2 x_4$, in which $y_2$ was the incidence of stem lesions at harvest, $x_3$ was the incidence of phoma leaf spot at GS 3,3-3,5 (flower buds visible) and $x_4$ was the incidence of phoma leaf spot at GS 4,5-5,5 (mid flowering). Data from field experiments with four winter oilseed rape cultivars at Boxworth or Rothamsted in the 1992/93, 1993/94, 1996/97, 1997/98 or 1998/99 seasons were used to test the models. The values of $R^2$ for the regression equations testing model 1 for the phoma leaf spot/basal canker relationship were 0.75, 0.93, 0.91 and 0.89 for cultivars Apex, Bristol, Capitol and Envol, respectively. The values of $R^2$ for the regression equations testing the model for the phoma leaf spot/stem lesion relationship were 0.58, 0.57, 0.54 and 0.71 for cultivars Apex, Bristol, Capitol and Envol, respectively. The phoma leaf spot/basal canker model could also be fitted to the combined data set for all four cultivars ($R^2 = 0.65$), whereas the phoma leaf spot/stem lesion relationship model could not. There were strong relationships between the incidence and severity of stem canker and phoma stem lesions ($R^2$ values 0.91 for basal canker and 0.89 for stem lesions).

Thermal time for canker development

Experiments in winter oilseed rape at Rothamsted with cvs Lipton and Capitol in 1997/98, cv. Apex in 1998/99 and cvs Apex, Lipton and Capitol in 1999/2000 were used to relate the development of canker and phoma stem lesions in spring to the development of phoma leaf spot in the previous autumn/winter. The thermal time (degree-days) from the first appearance of phoma leaf spot (autumn) to the first appearance of crown canker (spring) varied between cultivars and averaged 1220-1240 degree-days for cvs Lipton and Capitol and 1120-1140 for cv. Apex. In all three seasons, fungicide treatments generally decreased the proportions of plants at harvest with severe canker (severity scores 3 or 4) and increased the proportions with slight infections. There was a linear increase with accumulated degree-days in the severity of both canker and phoma stem lesions in plots (with or without fungicide treatment) in 1997/98 (cv. Lipton), 1998/99 (cv. Apex) and 1999/2000 (cv. Apex). The severity of canker at harvest in July was related to severity in early June in 1997/98, and in April in 1998/99 and 1999/2000.

Canker and yield loss relationships

Data from four experiments (Rothamsted in 1991/92, Withington in 1992/93, Boxworth in 1993/94 and Rothamsted in 1997/98) were used to define the relationships between yield loss and canker incidence (% plants with stems affected) or severity (mean stem score, 0 - 4 scale). 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. The critical point models for % yield loss on stem canker incidence for three of the four experiments were similar, but differed for Rothamsted in 1991/92. Models of % yield loss on AUDPC of both incidence and severity for these three experiments were also similar. 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 derived from the three experiments were: $L = -0.76 + 0.0075X$ ($R^2$ =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 showed that the greatest yield losses were 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. The % yield loss was related to incidence or severity of both basal cankers and upper stem lesions in experiments at Boxworth in 1993/94 and at Rothamsted in 1997/98.

Effects of canker and light leaf spot epidemics in the same crop on yield loss.

Data from experiments at Rothamsted in 1992/93, 1994/95 and 1995/96 were used in correlation, regression and principal component analyses to define relationships between yield loss and incidence or severity of stem canker and light leaf spot in the same crop of winter oilseed rape. Growth stages (GS) 6.3/6.4 (seed green to green brown) and 4.0/4.5 (early to mid flowering) were identified as the critical points for relating % yield loss to stem canker and light leaf spot (on stems), respectively. The general critical point model relating % yield loss ($L$) to combined incidence of stem canker ($X_1$) at GS 6.3/6.4 and light leaf spot (stems) ($X_2$) at GS 4.0/4.5 was: $L = 0.85 + 0.079X_1 + 0.065X_2$ ($R^2$=43.7%, p<0.001, 92df). A general AUDPC model relating % yield loss ($L$) to the AUDPC of combined incidence of stem canker ($X_3$) from GS 5.7 to GS 6.5 and light leaf spot (stems) ($X_4$) from GS 4.0 to GS 6.3 was constructed using data from the 1992/93 and 1994/95 experiments: $L$=0.07+0.00096$X_3$+ 0.0026$X_4$ ($R^2$ = 43.6%, p<0.001, 68df). These two general yield loss models were tested with data from Rothamsted in 1993/94 and Boxworth in 1992/93. The CP model based on combined incidence of stem canker and light leaf spot (stems) gave better predictive accuracy than the AUDPC model. Yield losses based on predictions based on models for losses from individual diseases overestimated actual losses when both canker and light leaf spot were present.

**Key results**

- There is a strong correlation between the incidence of phoma leaf spot and canker incidence at harvest.
- Records of the maximum incidence of phoma leaf spot up to the 6-leaf stage and from the 6-leaf stage to early stem extension are necessary to predict final levels of canker.
- The phoma leaf spot - canker relationship has been established for a range of cultivars.
- Phoma stem lesions are correlated with phoma leaf spotting at early stem extension and at early-mid flowering, though this relationship appears to be more variable than for canker infection.
- The first appearance of cankers can be predicted from first appearance of phoma leaf spots using thermal time. There is variation between cultivars and this averaged 1130 degree days for Apex and 1230 degree days for Capitol and Lipton.
- Fungicides can delay the appearance of canker.
- Yield loss is mainly associated with cankers which girdle more than half the stem before harvest.
- Cankers appearing at or before flowering are likely to reduce yield.
- Yield loss from canker was greatest when disease severity was high. This was associated with early development of cankers.
- Where canker and light leaf spot are present, yield loss can be estimated from the incidence of light leaf spot on stems at early flowering and canker incidence pre-harvest.
- Using yield-loss models for the individual diseases tends to over-estimate actual yield loss when canker and light leaf spot are both present.

**Factors affecting the production and release of ascospores of *Leptosphaeria maculans***

The dispersal of ascospores of *Leptosphaeria maculans* is essential to generate phoma leaf spot epidemics and subsequent canker development in winter oilseed rape in the UK. The date of onset of phoma leaf spotting shows considerable variation between sites and years, and understanding the factors contributing to this variation is central to improving advice on fungicide use. This part of the project considers maturation of ascospores on rape stubble after harvest, quantification of the intensity and duration of ascospore release and appearance of phoma leaf spot.

Materials and Methods

Field experiments were established at Rothamsted in southern England in the 1997/98, 1998/99 and 1999/2000 seasons with winter oilseed rape cultivars Capitol and Lipton sown in late August. Samples of 100 plants from control plots were assessed for diseases at monthly intervals from late September. A

Burkard spore trap situated 2 km from the field experiment, was used each season to monitor the daily pattern in concentration of air-borne *L. maculans* ascospores. Development of *L. maculans* pseudothecia on the stem debris was investigated by microscopic examination of stem pieces each week from late August onwards. Spore trapping was also carried out at ADAS Boxworth using naturally infected winter oilseed rape stems collected shortly after harvest of farm crops at Boxworth in 1998 and 1999. The stubble was stored outdoors under ambient conditions until late August, when material was arranged in a circle of about 3 m diameter centred on a Burkard spore trap. Daily records of spore catches were made from 26 August 1998 to 6 April 1999 and 24 August 1999 until 1 March 2000. Disease progress was recorded weekly in experimental plots of cv. Apex on the farm.

Results

Ascospore maturation and release

After harvest in July at Rothamsted, mature pseudothecia were first observed on infected stem debris on 26 September 1997, 23 July 1998 (at sites of severe basal cankers only) and 22 September 1999. In August 1999, stem debris that was > 1-year old was found to have predominantly (95%) empty (discharged) pseudothecia with occasional (5%) mature pseudothecia present. By 15 September, new immature pseudothecia were observed on the 1-year old stem debris and the incidence of mature pseudothecia had increased to 30% of those sampled. This was one week before mature pseudothecia were first detected on the stems from crops harvested in July 1999.

There were differences between seasons in the numbers and patterns of ascospores released. At the start of each season, few or no ascospores were detected before mid-September. The first days with > 4 ascospores detected per $m^3$ were 8 October 1997, 27 September 1998 and 14 September 1999. Ascospores continued to be released until late spring in each season. These first releases of ascospores occurred when the daily mean temperatures were <14°C after at least 3 days without rain and were associated with rainfall. The number of rain-days (≥ 0.2 mm of rain per day) from 20 July (harvest) to the date of first spore release each season was 28, 27 and 17, days respectively, for 1997/98, 1998/99 and 1999/2000. There were 23, 21 and 16 rain-days from 1 August to the date of first spore release. Subsequent releases of ascospores were associated with occurrence of rain or dew.

At Boxworth, the first ascospores were detected on 22 September in 1998 and 1 September in 1999. This followed 16 days with rain after 1 August in 1998 and 17 rain days in 1999. In both years, the number of rain days from harvest to first spore detection was 21 days. Large spore catches were only recorded from 6 October 1998 and 21 September 1999. The first release coincided with a fall in temperature to <14°C and followed three dry days in September 1998, but there was no recorded rainfall to trigger spore release. In

1999, the first spores were caught in early September when mean temperatures were 17-18°C after 6 days without rain, but the <14°C criterion coincided with larger spore catches from 21 September onwards. Spore release occurred over the autumn and winter in both years, though peak spore numbers occurred rather earlier in 1999 than in 1998.

Phoma leaf spot development

Phoma leaf spotting was first observed at a low incidence at Rothamsted on 22 October 1997, 22 October 1998 and 28 September 1999 (i.e. 15, 25 and 14 days after the first observed spore release in 1997, 1998 and 1999 respectively). The development of phoma leaf spotting in untreated plots differed between the seasons; in 1997/98 the incidence of affected plants reached 20% in late October. In 1998/99, although a few leaf lesions were visible in late October, the main leaf spotting epidemic began 3 weeks later than in 1997/98, with incidence reaching 20% in mid-November. In contrast, in 1999/2000, the epidemic was early and severe, with incidence reaching 20% in early October.

At Boxworth, the first signs of phoma leaf spot were detected in late October 1998 and experimental plots showed 13.8% plants affected on 3 November, about five weeks after first ascospores were detected (and four weeks after the first large spore catch). In 1999, phoma leaf spot was present on 4% plants on 28 September and this had increased to 48% incidence by 5 October (Fig. 3) - four weeks after the first spore catch and only two weeks after the first large ascospore capture.

**Key results**

- The maturation and release of ascospores of the canker fungus showed seasonal variation, differing in calendar date by three weeks in the autumns of 1998 and 1999.
- Ascospore production was delayed by dry conditions in autumn 1998.
- First spore release was 16-23 rain days after 1 August or 16-28 rain days after harvest and the lower value could be used as an early predictor of spore maturity.

- Phoma leaf spot was detected 2-4 weeks after spores were first caught in spore traps.

- Weekly monitoring of crops at high risk sites in autumn is essential to follow phoma epidemics.

## Optimising fungicide timing for stem canker control and yield response

Three field experiments were completed in both 1998/1999 and 1999/2000 season on cv. Apex grown at ADAS Boxworth, ADAS High Mowthorpe and Rothamsted. A standard fungicide treatment of

difenoconazole + carbendazim was used throughout and was applied on four main dates in a full factorial design with four replicates. A further four secondary dates were used to establish the consequences of delaying the first timing by two or four weeks and similarly delaying the final spray. The first spray treatment was applied when phoma leaf spot was active (at least 20% plants affected) with subsequent timings at 4-6 week intervals. Spray schedules were carried out as planned except for three secondary timings at High Mowthorpe in 198/1999 where weather conditions prevented applications. Disease assessments were made weekly in autumn on untreated plants and full assessments of all plots from the second main treatment application, at flowering and pre-harvest. Plots were combine harvested and yields corrected to 90% dry matter.

1998/1999

At the Boxworth site, high levels of phoma leaf spot were present from late November onwards, reaching 100% plants affected by 19 January and this resulted in 76% plants with canker and 81% plants with phoma stem lesions pre-harvest. There were no significant effects on yield, though 14 out of 19 treatments were higher yielding than the untreated control of 4.34 t/ha (range of increases 0.03- 0.20 t/ha). Treatments were highly successful in controlling canker which was reduced to 0% by four sprays, 5-15% by the best two spray programmes and to 30% by the best single spray (19 January. Phoma stem lesions were rather less well controlled and the best single spray was 9 March which gave 30% plants affected compared with 81% in control plots. At High Mowthorpe, there were no significant yield differences (range 0.07-0.85 t/ha) from the untreated (2.71 t/ha), though later timings had the highest yield. Phoma leaf spotting gave low levels of phoma stem lesions rather than canker. Light leaf spot on stems was reduced from 3% area affected to 1% by spring sprays.

At Rothamsted, both canker and light leaf spot developed in the experiment, affecting 65% plants on 30 June and almost 10% leaf area in the spring, respectively. The phoma leaf spotting was relatively late (as at Boxworth) with a rapid increase during early December. The 16 December spray timing gave the lowest canker (10% plants). A two spray programme applied on 16 December and 25 February gave complete control of canker. Light leaf spot was also controlled by the same sprays which were effective against canker, with suggestions that spring sprays were of greater benefit for light leaf spot control than for canker. There were significant effects on yield with increases up to 0.73 t/ha (16%).

1999/2000

At Boxworth, high levels of phoma leaf spot were present from early October onwards, reaching 100% plants affected by 12 October and this resulted in 99% plants with canker and 95% plants with phoma stem lesions pre-harvest. There were significant yield responses of up to 0.69 t/ha compared with the untreated control of 3.07 t/ha. Treatments were moderately successful in controlling canker, which was reduced by

>50% by some 3 or 4-spray programmes. Phoma stem lesions were also difficult to control and only three of the four spray programmes achieved >50% control.

At High Mowthorpe, there was a later and less severe phoma leaf spot epidemic which affected 64% of plants on 7 December and produced 41% plants with canker and 41% plants with phoma stem lesions pre-harvest. The canker infections were well controlled with multiple sprays and even by a single spray on 9 November. Control of phoma stem lesions required at least two sprays. Light leaf spot was also present at High Mowthorpe and activity from spring onwards resulted in stem (6% area affected) and pod infection (23% area affected) pre-harvest. Good control of light leaf spot required more than one spray. The highest yields at High Mowthorpe (3.32 t/ha untreated) were from 4-spray programmes (yield response range 0.79-1.03 t/ha).

At Rothamsted, the phoma leaf spotting was early (as at Boxworth) with a rapid increase during early October resulting in 100% plant infection by 19 October. Pre-harvest, canker affected 98% plants and phoma stem lesions 75% of stems. Light leaf spot developed in the experiment from mid February and became moderately severe on pods, affecting 18.8% pod area in July. Early November was the most effective time of application, but three or four sprays were usually required to achieve good control of canker, which was reduced to as little as 10% incidence. Phoma stem lesions were more difficult to control and only one four-spray programme gave >80% control. Light leaf spot on pods was also only moderately well controlled and only 6 of the 19 spray regimes gave >50% control. There were significant effects on yield (3.75 t/ha untreated) with yield responses of up to 0.89 t/ha.

A summary of canker and yield data from the six field experiments provides a basis for explaining new yield loss data obtained in the project (Table 1).

**Table 1.** Summary of yield and canker data from experiments 1998-2000.

| Site | Year | Canker index pre-harvest | | Yield (t/ha) | | Light leaf spot severity pre-harvest |
|---|---|---|---|---|---|---|
| | | Untreated | Treated (lowest) | Untreated | Treated (max.) | |
| Boxworth | 1998/1999 | 1.05 | 0.00 | 4.34 | 4.47 | 1% stem area |
| High Mowthorpe | 1998/1999 | 0.00 | 0.00 | 2.71 | 3.56 | 3% stem area |
| Rothamsted | 1998/1999 | 0.78 | 0.00 | 4.64 | 5.37* | 8% stem area<br>16% pod area |

| | | | | | | |
|---|---|---|---|---|---|---|
| Boxworth | 1999/2000 | 2.84 | 0.75 | 3.07 | 3.76* | traces |
| High Mowthorpe | 1999/2000 | 0.74 | 0.00 | 3.32 | 4.35* | 6% stem area<br>23% pod area |
| Rothamsted | 1999/2000 | 2.87 | 0.13 | 3.75 | 4.64* | 18% stem area<br>19% pod area |

* Significant yield increases recorded in these experiments

The canker severity indices show the large variation between years and lower severity in the north compared with sites in eastern region. Good control of canker was achieved in all the affected crops, but this required two or more sprays. The largest yield responses were associated with disease control in crops infected with both light leaf spot and canker or with severe canker alone. Slight canker infection (index <1) appeared to have little effect on yield (eg Boxworth 1999) and is acceptable commercially.

**Key results**

- Early and severe phoma leaf spot epidemics caused severe canker and yield loss of up to 0.7 t/ha on cv. Apex.
- Good control of canker was achieved with multiple spray programmes of fungicides and these also gave control of light leaf spot.
- Large yield losses (0.7-1.0 t/ha) were associated with a combination of phoma and light leaf spot in the north and east of England.
- Late development of phoma leaf spot may not cause significant leaf loss and fungicide treatment may not be cost-effective.
- The optimum spray timing varied between sites and years.
- Single sprays of fungicide gave variable results, but two spray programmes were more consistently cost-effective where canker epidemics were moderate to severe.
- There is some flexibility in timing of sprays, particularly where plants of moderately resistant cultivars are well developed.

**CONCLUSIONS AND IMPLICATIONS FOR LEVY PAYERS**:

Early epidemics of phoma leaf spot pose the greatest threat to yield as these are most likely to produce moderate or severe cankers by harvest. It might now be possible to forecast epidemic onset using the number of rain days after harvest to provide an indication of first spore release and hence first signs of phoma leaf spot. Plan for a two spray programme with early epidemics, as single sprays showed very variable and inconsistent yield responses. A threshold for fungicide treatment of >20% plants affected with phoma leaf spot is suggested for susceptible and moderately resistant varieties. Weather and ground conditions in

autumn 2000 prevented fungicide applications to many crops and alternative strategies such as systemic seed treatments need to be evaluated for early disease control. Fungicides are more likely to be profitable if light leaf spot also develops in autumn and winter. A spring fungicide may also be needed for growth regulation and canopy management to control light leaf spot epidemics on pods. There is some flexibility in spray timing particularly when plants have large leaves.

Factors influencing canker development and therefore risk of yield loss can be summarised as follows:

<u>High risk of yield loss from canker:</u>

Above average rainfall in August and September

Phoma leaf spot appear early (in September or October)

Late sowings which result in small plants

Susceptible or moderately resistant cultivars

<u>Low risk of yield loss from canker</u>:

Few rain days in August and September

Phoma leaf spot does not exceed 20% infection until December or later

Early sowings producing large plants which reach the 6 leaf stage before phoma leaf spot appears

Resistant variety (NIAB 7 or more)

The forecasting of phoma epidemics and optimisation of fungicides using results from this and other projects is being developed further in the PASSWORD project which is supported HGCA and other industry collaborators using MAFF LINK funding.

# TECHNICAL REPORT

## Chapter 1

## Introduction

Stem canker (*Leptosphaeria maculans*) was the most serious disease on winter oilseed rape in England and Wales in the 1995/96 season, causing losses of £29M (by comparison with £13.3M for light leaf spot), despite expenditure of *c.* £5 million on fungicides to control the disease. Furthermore, losses from stem canker were estimated to be an average of £38M *per annum* for the harvest years 1993-1995 (Fitt *et al.*, 1997), using ADAS/CSL disease survey data (e.g. Gladders *et al.,* 1997) and yield loss formulae derived from results of HGCA/MAFF-funded experiments in 1991-1994 (Sansford *et al.,* 1996). However, severe stem canker can be controlled by correctly timed fungicide applications, with yield responses of >1t/ha recorded in these HGCA/MAFF and other ADAS and Rothamsted experiments.

It is important to ensure that control fungicides are applied only to those crops which need treatment. Survey data indicate that there is considerable variation in the severity of stem canker epidemics between seasons, between regions and between crops. For example, losses from stem canker were estimated as only £11.2M in 1991, by comparison with £42.3M in 1993. There is good evidence that stem canker epidemics are most severe in eastern England, southern England and the Midlands, with associated losses of 1.0 - 1.5 t/ha regularly recorded in experiments (e.g. at ADAS Boxworth, Rothamsted, ADAS Rosemaund and at Cereals '96). These high risk areas account for 72% of the winter oilseed rape grown. In the 1995/96 survey, 60% of stems were affected by stem canker in the East/South East ADAS regions (65% of the area) by comparison with 9% in the North/South West regions (28%). Even in low risk areas, survey data indicate that severe stem canker epidemics occur on some crops.

Fungicide timing for control of stem canker also needs to be improved. Good disease control has been achieved in ADAS and Rothamsted experiments with multiple spray treatments. These provide a theoretical basis for stem canker control, but there is now a need to develop practical disease management programmes for farms. Severe losses from stem canker occurred throughout Eastern England in 1994/95 and 1995/96, when 98% of winter oilseed rape crops were sprayed with fungicides (46% with 2 sprays, 24% with 3 sprays). Only 3% of these crops were sprayed in October, the critical time for stem canker control in those seasons. The conclusion that the optimum spray timing to control stem canker is from November and February (Sansford *et al.* 1996) does not apply in all seasons. In recent experiments, control of stem canker has been ineffective if sprays were applied when phoma leaf spot was already present; yield responses have sometimes been halved by delaying applications from October to November. MAFF-funded appropriate

dose experiments at ADAS Boxworth suggest that more than two half-rate sprays are needed to control severe stem canker epidemics. Furthermore, these experiments indicate that the best spray timings and products for control of stem canker are different from those for light leaf spot (*Pyrenopeziza brassicae*).

To ensure that fungicides are timed correctly, it is essential to identify the critical factors which determine the onset and severity of phoma leaf spot epidemics in the autumn. Regression analyses on eastern England survey data suggest that occurrence of rainfall in August and September greatly influences the severity of stem canker epidemics in the following season (Gladders and Symonds, 1995). Work in France suggests that this rainfall may affect development and release of the ascospores of *Leptosphaeria maculans*, which are responsible for initiating stem canker epidemics when they infect oilseed rape leaves in the autumn (Penaud, 1995). However, further work is needed to clearly identify those factors which result in the development of severe stem canker epidemics and to determine the principles for optimising fungicide treatment regimes to achieve effective disease control.

Monitoring of ascospore release would assist the development of early warning systems particularly when linked to improved understanding of phoma leaf spot - canker relationships and canker severity yield loss relationships.

This project aims to improve strategies for control of stem canker on winter oilseed rape through optimising fungicide timing and choice, based on a clear understanding of the critical factors determining stem canker severity and associated yield losses.

**Objectives:**

a) To quantify yield losses in relation to the development of phoma leaf spot and the severity of stem canker epidemics.

b) To identify factors affecting ascospore discharge and hence phoma leaf spot attacks.

c) To optimise fungicide timing for stem canker control and yield response.

## Chapter 2

### Relationships between phoma leaf spot, canker and yield loss

The biological model for phoma development in oilseed rape is considered to be monocyclic, with air-borne spores (ascospores) from infected oilseed rape stubbles producing phoma leaf spots in winter oilseed rape crops during the autumn and winter. Subsequently, the canker fungus grows down within the leaf petiole and invades the stem (Fig 1). After a period of about six months, sunken brown lesions (cankers) appear at the stem base which are capable of causing crop lodging and premature ripening if they girdle the stem. Phoma also causes stem lesions well above the stem base region associated with infected stem leaves and these are termed 'phoma stem lesions'. The development of phoma within the plant has been described by Hammond & Lewis (1986) but requires field validation. Within this project, the biological model is extended to current commercial cultivars and developed quantitatively to improve guidance on risk of yield loss and, hence, the need for fungicides.

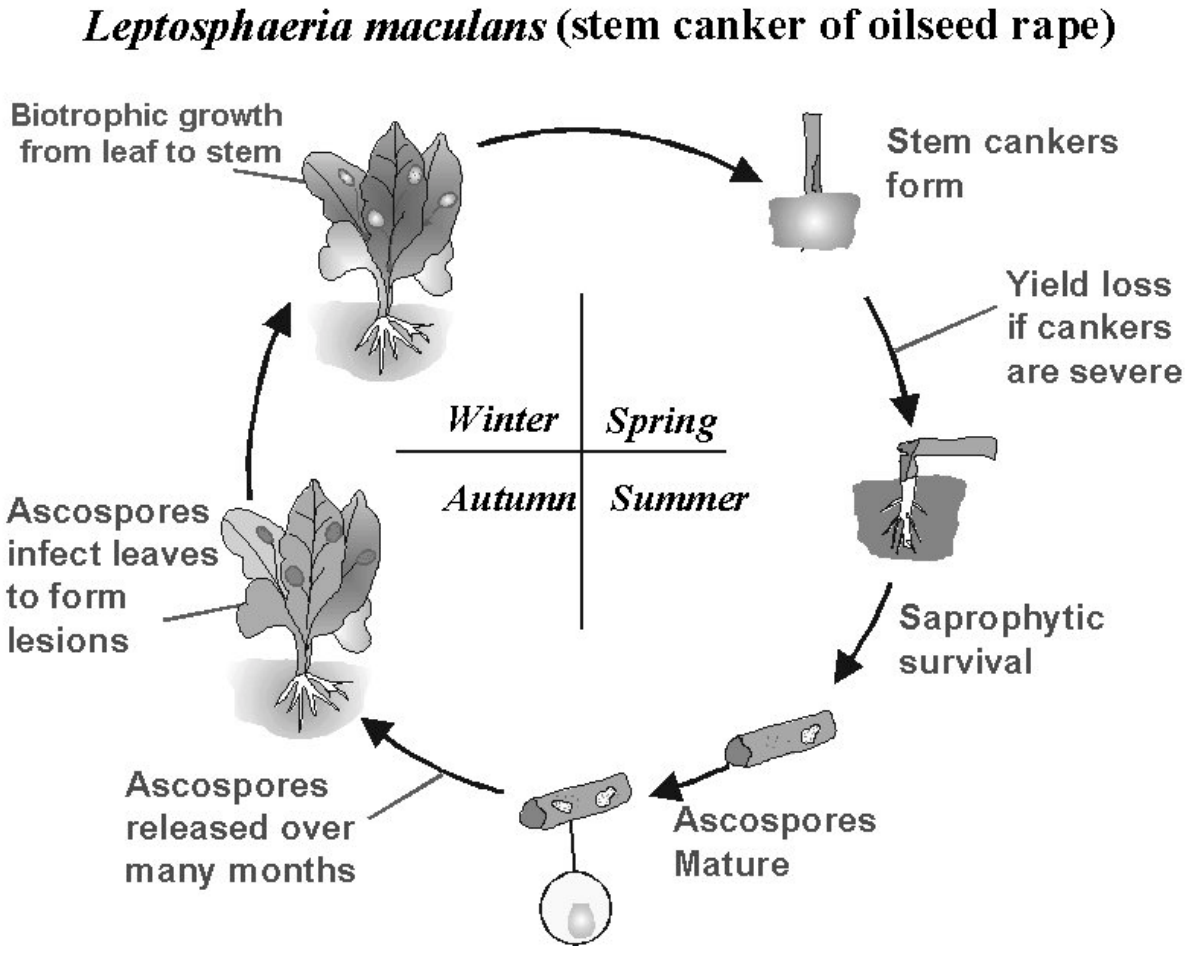



Fig. 1. Disease cycle of canker in winter oilseed rape.

A number of refeered scientific papers have been published during the course of this project and

only abstracts of the component parts of the desk study are presented in this report.

### 2.1. Phoma leaf spot- stem canker relationship

The relationships between the percentage of plants with phoma leaf spot at different growth stages in autumn/winter or early spring and incidence of stem canker (basal canker or stem lesions) in summer on winter oilseed rape in southern England were described by the models:
$y_1 = \beta_0 + \beta_1 x_1 + \beta_2(x_2 - x_1)$ if $x_2 > x_1$, and $y_1 = \beta_0 + \beta_1 x_1$ if $x_2 \leq x_1$, in which $y_1$ was the incidence (% plants affected) of basal canker at harvest, $x_1$ was the maximum incidence of phoma leaf spot during the period from sowing to growth stage (GS) 1,6-1,7 (about 100 days after sowing) and $x_2$ was the maximum incidence of phoma leaf spot between GS 1,7 and GS 2,0 (start of stem extension). A second model which described the relationship between phoma leaf spot and phoma stem lesions was identified as $y_2 = \alpha_0 + \alpha_1 x_3 + \alpha_2 x_4$, in which $y_2$ was the incidence of stem lesions at harvest, $x_3$ was the incidence of phoma leaf spot at GS 3,3-3,5 (flower buds visible) and $x_4$ was the incidence of phoma leaf spot at GS 4,5-5,5 (flower buds opening). Data from field experiments with four winter oilseed rape cultivars at Boxworth or Rothamsted in the 1992/93, 1993/94, 1996/97, 1997/98 or 1998/99 seasons were used to test the models. The values of $R^2$ for the regression equations testing model 1 for the phoma leaf spot/basal canker relationship were 0.75, 0.93, 0.91 and 0.89 for cultivars Apex, Bristol, Capitol and Envol, respectively. The values of $R^2$ for the regression equations testing the model for the phoma leaf spot/stem lesion relationship were 0.58, 0.57, 0.54 and 0.71 for cultivars Apex, Bristol, Capitol and Envol, respectively. The phoma leaf spot/basal canker model could also be fitted to the combined data set for all four cultivars ($R^2$ = 0.65), whereas the phoma leaf spot/stem lesion relationship model could not. There was strong relationships between the incidence and severity of stem canker and phoma stem lesions ($R^2$ values 0.91 for basal canker and 0.89 for stem lesions).

Full details in: 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.

### 2.2. Thermal time for canker development

Experiments in winter oilseed rape at Rothamsted with cvs Lipton and Capitol in 1997/98, cv. Apex in 1998/99 and cvs Apex, Lipton and Capitol in 1999/2000 were used to relate the development of canker and phoma stem lesions in spring to the development of phoma leaf spot in the previous autumn/winter. The thermal time (degree-days) from the first appearance of phoma leaf spot (autumn) to the first appearance of crown canker (spring) varied between cultivars and averaged 1220-1240 degree-days for cvs Lipton and Capitol and 1120-1140 for cv. Apex. A November fungicide treatment delayed crown canker development

in spring in 1999 and 2000, but did not affect the rate of increase in severity. The 1997/98 season was rather different, as fungicide treatments did not delay the appearance of crown canker but still decreased the rate of increase in crown canker severity. In all three seasons, fungicide treatments generally decreased the proportions of plants at harvest with severe canker (severity scores 3 or 4) and increased the proportions with slight infections. There was a linear increase with accumulated degree-days in the severity of both canker and phoma stem lesions in plots (with or without fungicide treatment) in 1997/98 (cv. Lipton), 1998/99 (cv. Apex) and 1999/2000 (cv. Apex). The severity of canker at harvest in July was related to severity in early June in 1997/98, and in April in 1998/99 and 1999/2000.

**This contribution is based on unpublished data from** P SUN, B D L FITT, J M STEED, C T UNDERWOOD and J S WEST.

### 2.3. Canker and yield loss relationships

Data from four experiments (Rothamsted in 1991/92, Withington in 1992/93, Boxworth in 1993/94 and Rothamsted in 1997/98) were used to define the relationships between yield loss and canker incidence (% plants with stems affected) or severity (mean stem score, 0 - 4 scale). 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. Since yield in t $ha^{-1}$ is influenced by 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 for Rothamsted in 1991/92. Models of % yield loss on AUDPC of both incidence and severity for these three experiments were also similar. 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 derived from the three experiments were: $L = -0.76 + 0.0075X$ ($R^2$ =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 showed that the greatest yield losses were 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. The % yield loss was related to incidence or severity of both basal cankers and upper stem lesions in experiments at Boxworth in 1993/94 and at Rothamsted in 1997/98.

The full paper is: 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.

### 2.4. Effects of canker and light leaf spot epidemics in the same crop on yield loss

Data from experiments at Rothamsted in 1992/93, 1994/95 and 1995/96 were used in correlation, regression and principal component analyses to define relationships between yield loss and incidence or severity of stem canker and light leaf spot in the same crop of winter oilseed rape. Growth stages (GS) 6.3/6.4 (seed green to green brown) and 4,0/4,5 (early to mid flowering) were identified as the critical points for relating % yield loss to stem canker and light leaf spot (on stems), respectively. Critical point (CP) and area under disease progress curve (AUDPC) models relating % yield loss to combined incidence or severity of stem canker and light leaf spot (stems) in each experiment were constructed by linear regression. CP models for incidence were similar for all three experiments and the AUDPC models for incidence did not differ for 1992/93 and 1994/95 experiments. The general CP model relating % yield loss ($L$) to combined incidence of stem canker ($X_1$) at GS 6.3/6.4 and light leaf spot (stems) ($X_2$) at GS 4.0/4.5 was: $L = 0.85 + 0.079X_1 + 0.065X_2$ ($R^2$=43.7%, p<0.001, 92df). A general AUDPC model relating % yield loss ($L$) to the AUDPC of combined incidence of stem canker ($X_3$) from GS 5.7 to GS 6.5 and light leaf spot (stems) ($X_4$) from GS 4.0 to GS 6.3 was constructed using data from the 1992/93 and 1994/95 experiments: $L=0.07+0.00096X_3+ 0.0026X_4$ ($R^2$ = 43.6%, p<0.001, 68df). These two general yield loss models were tested with data from Rothamsted in 1993/94 and Boxworth, England in 1992/93. The CP model based on combined incidence of stem canker and light leaf spot (stems) gave better predictive accuracy than the AUDPC model. Yield losses based on predictions based on models for losses from individual diseases overestimated actual losses when both canker and light leaf spot were present.

Full details can be found in: Zhou, Y., Fitt, B.D.L., Welham, S.J., Evans, N. & Gladders, P. (2000). Effects of stem canker (*Leptosphaeria maculans*) and light leaf spot (*Pyrenopeziza brassicae*) on yield of winter oilseed rape (*Brassica napus*) in southern England. *Plant Pathology* **49**, 487-497.

# Chapter 3

## Factors affecting the production and release of ascospores of *Leptosphaeria maculans*

### 3.1 Introduction

The dispersal of ascospores of *Leptosphaeria maculans* is essential to generate phoma leaf spot epidemics and subsequent canker development in winter oilseed rape in the UK. The onset of phoma leaf spotting shows considerable variation between sites and years and understanding the factors contributing to this variation is central to improving advice on fungicide use. A number of epidemiological components are likely to be involved and this part of the project considers maturation of ascospores on rape stubble after harvest, quantification of the intensity and duration of ascospore release and appearance of phoma leaf spot.

### 3.2 Materials and methods

Field experiments were established at Rothamsted in southern England in the 1997/98, 1998/99 and 1999/2000 seasons. Winter oilseed rape cultivars Capitol and Lipton were sown on 26-27 August in four randomised blocks of five main plots, with each main plot split between the two cultivars. The NIAB rating for resistance to stem canker for Capitol was 6 and that for Lipton was 5 (Anon., 1997). Each sub-plot was 15 m x 3 m, including a 9 $m^2$ sampling area and a central area of 12 $m^2$ for harvest yield. Guard rows, 3 m wide, between plots and areas of the field surrounding the experiment were sown with cv. Capitol. Areas adjacent to the experiment were inoculated in early September each season with infected winter oilseed rape stem base debris (*c.* 200 stems per field), which had been collected at the end of the previous season and kept outdoors. Samples of 25 plants from each control plot were assessed for diseases at monthly intervals from late September. More frequent assessments were made at Rothamsted on other field experiments in this project (see Chapter 4).

A Burkard spore trap (surrounded by 8 trays, each 0.5 $m^2$ and containing *c.* 100 of the infected stems that were used to inoculate the field experiment), situated 2 km from the field experiment, was used each season to monitor the daily pattern in concentration of air-borne *L. maculans* ascospores. Development of *L. maculans* pseudothecia on the stem debris was investigated by microscopic examination of 25 pseudothecia, taken from five different stem pieces (five per piece) each week from late August onwards. Pseudothecia were classed as mature when they contained differentiated ascospores visible inside asci, with more than four cells per spore. In autumn 1999, numbers of mature pseudothecia on stems from the crop harvested in 1999 were compared with numbers on stems from crops harvested in 1998 and kept outdoors subsequently.

Meteorological data were collected using an automatic meteorological station situated in the same fields as the experiments in each season.

Spore trapping was also carried out at ADAS Boxworth using naturally infected winter oilseed rape stems collected shortly after harvest of farm crops at Boxworth (harvest dates were 17 July 1998 and 12 July 1999) in 1998 and 1999. The stubble was stored outdoors under ambient conditions until late August, when material was arranged in a circle of about 3 m diameter on short grass centred on a Burkard volumetric spore trap. Daily records of spore catches were made from 26 August 1998 to 6 April 1999 and 24 August 1999 until 1 March 2000. Weather records were collected by an automatic weather station about 100 m from the spore trap. Disease progress was recorded weekly in experimental plots of cv. Apex on the farm, where sources of ascospores are likely to have been surrounding commercial fields of winter oilseed rape, not exclusively that used for spore trapping.

### 3.3 Results

Ascospore maturation and release

After harvest in July at Rothamsted, mature pseudothecia were first observed on infected stem debris on 26 September 1997, 23 July 1998 (at sites of severe basal cankers only) and 22 September 1999. In August 1999, stem debris that was > 1-year old was found to have predominantly (95%) empty (discharged) pseudothecia with occasional (5%) mature pseudothecia present. By 15 September, new immature pseudothecia were observed on the 1-year old stem debris and the incidence of mature pseudothecia had increased to 30% of those sampled. This was one week before mature pseudothecia were first detected on the stems from crops harvested in July 1999.

There were differences between seasons in the numbers and patterns of ascospores released. At the start of each season, few or no ascospores were detected before mid-September. The first days with > 4 ascospores detected per $m^3$ were 8 October 1997, 27 September 1998 and 14 September 1999 and ascospores continued to be released until late spring in each season. These first releases of ascospores occurred when the daily mean temperatures were <14°C after at least 3 days without rain and were associated with rainfall (Fig. 2). For example, in 1997/98 (Fig. 2a) the first spore release on 8 October 1997 was after the temperature had fallen to *c.*12.5°C on the previous two days and rain on 7 October. Small numbers of spores were released on days without rainfall for the next week but none were released during the subsequent 20 days without rainfall. The next rainfall on the 4 November promoted release of many ascospores. The number of rain-days (> 0.2 mm of rain per day) from 20 July (harvest) to the date of first spore release each season was 28, 27 and 17, days respectively, for 1997/98, 1998/99 and 1999/2000. There were 23, 21 and 16 rain-days from

1 August to the date of first spore release. Subsequent releases of ascospores were associated with occurrence of rain or dew. There were maxima of 1465 (19 November 1997), 566 (27 November 1998) and 1222 (23 October 1999) spores per $m^3$.

(Fig. 2)

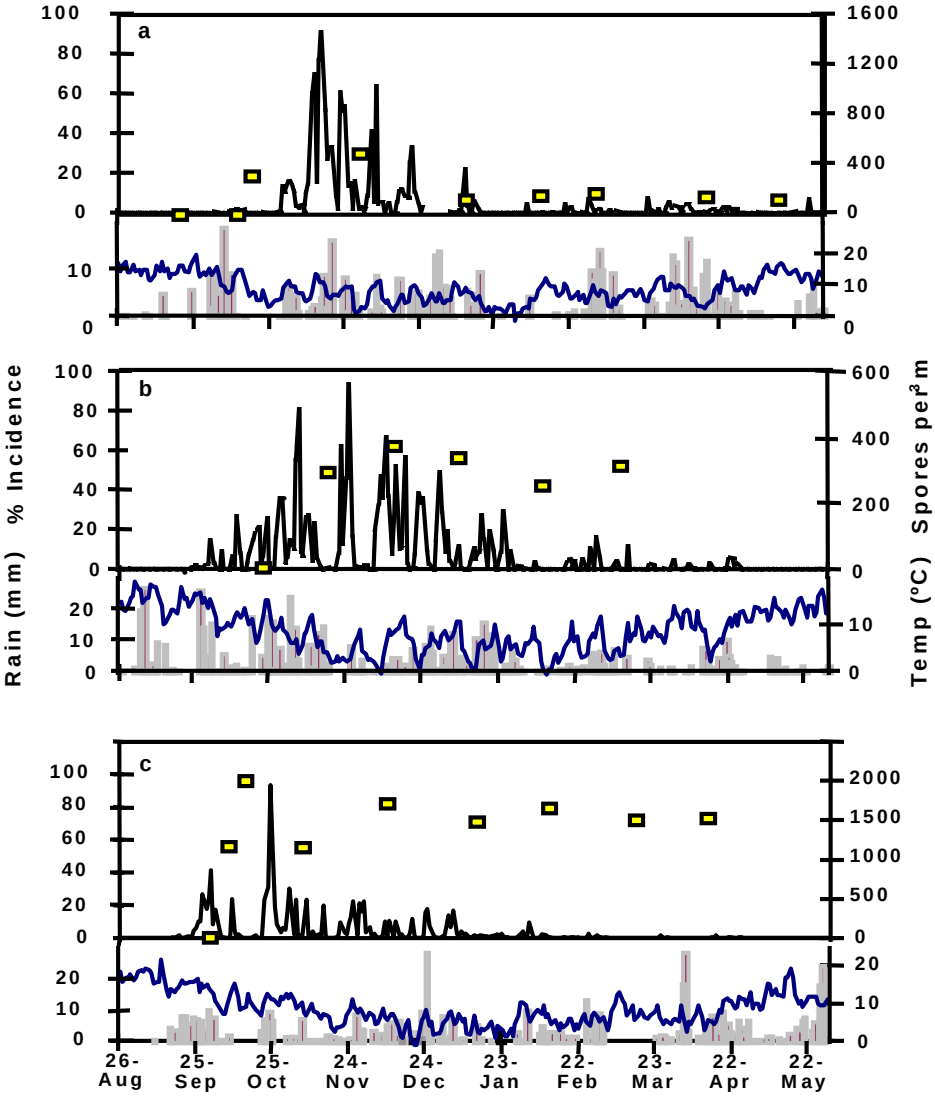
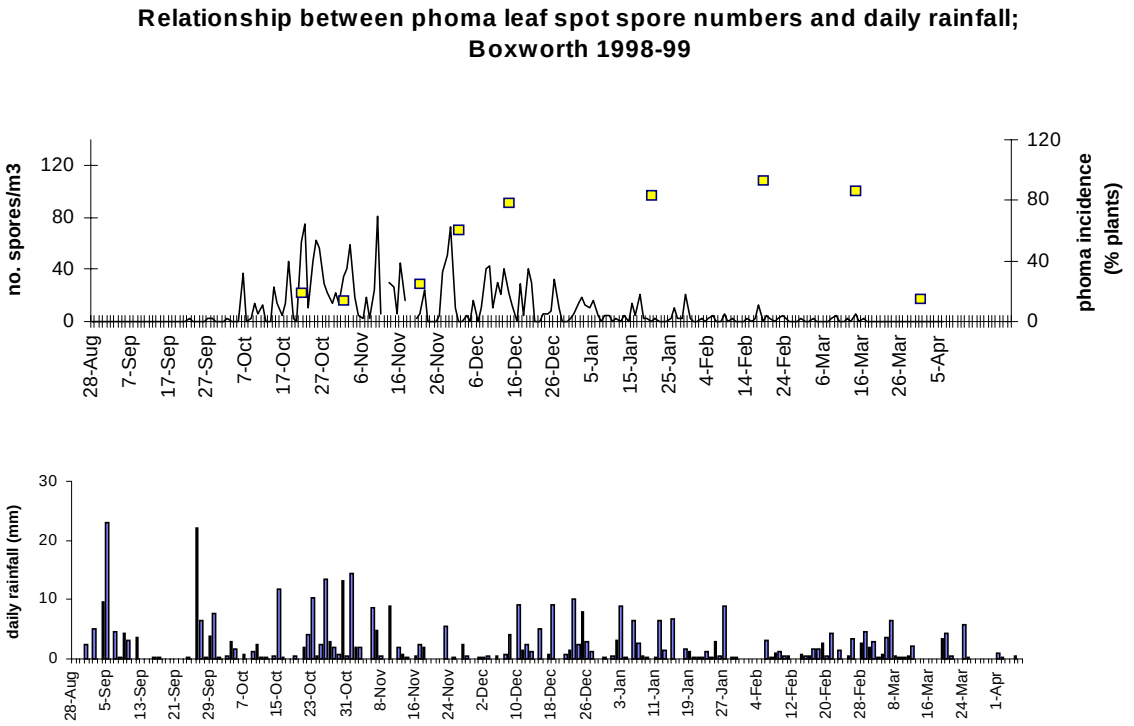



Fig. 2. Changes with time in the numbers of ascospores of *Leptosphaeria maculans* (line on upper axis) in the air and incidence (% plants affected) of phoma leaf spot (squares on upper axis) in untreated winter oilseed rape (cv. Lipton) in relation to daily rainfall (vertical bars on lower axis) and temperature (line on lower axis) at Rothamsted in 1997/98 (a), 1998/99 (b) and 1999/2000 (c).

a)




b)

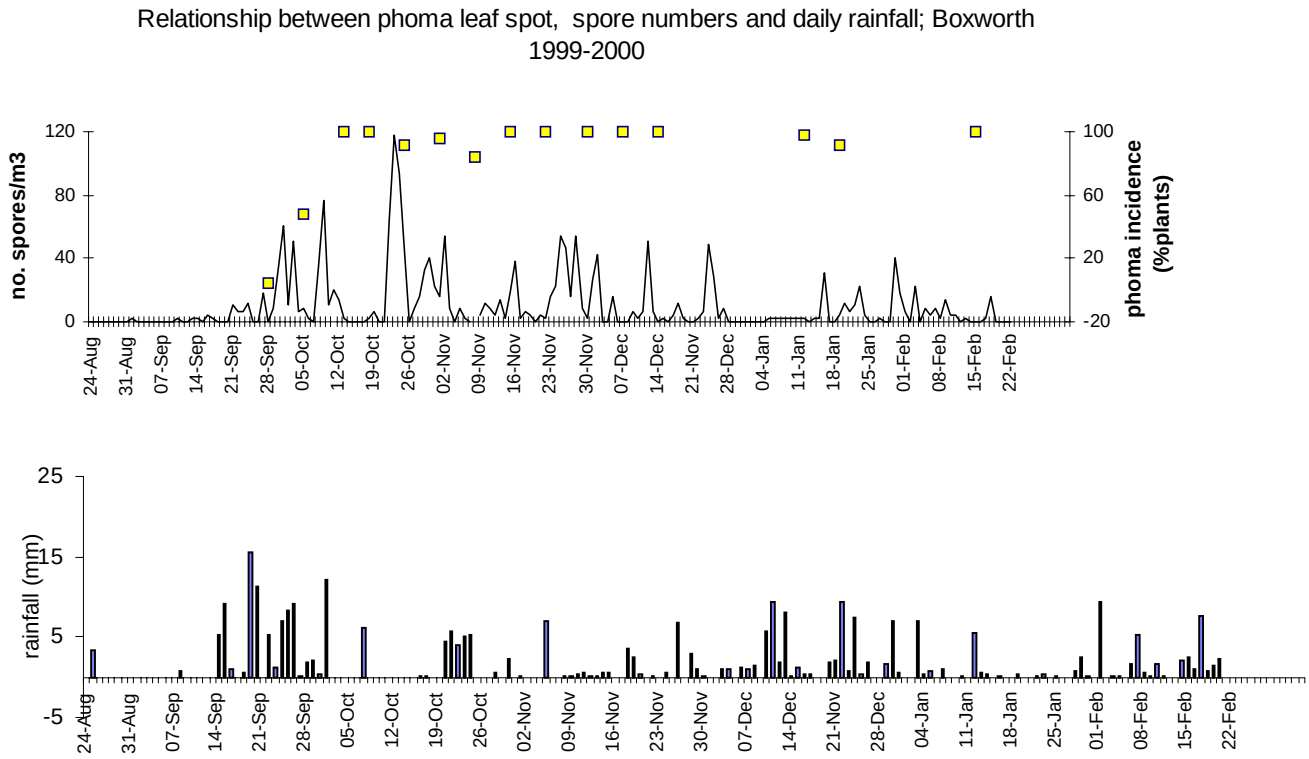



Fig. 3. Numbers of ascospores of *Leptosphaeria maculans* caught each day during autumn and winter a) 1998/99 and b) 1999/2000 and daily rainfall at Boxworth

At Boxworth, the first ascospores were detected on 22 September in 1998 and 1 September in 1999 (Fig. 3). This followed 16 days with rain after 1 August in 1998 and 17 raindays in 1999. In both years, the number of raindays from harvest to first spore detection was 21 days. Large spore catches were recorded only from 6 October 1998 and 21 September 1999. The first release coincided with a fall in temperature to <14°C and followed three dry days in September 1998, but there was no recorded rainfall to trigger spore release. In 1999, the first spores were caught in early September when mean temperatures were 17-18°C after 6 days without rain, but the <14°C criterion coincided with larger spore catches from 21 September onwards. Spore release occurred over the autumn and winter in both years, though peak spore numbers occurred rather earlier in 1999 than in 1998.

Phoma leaf spot development

Phoma leaf spotting was first observed at a low incidence at Rothamsted on 22 October 1997, 22 October 1998 and 28 September 1999 (i.e. 15, 25 and 14 days after the first observed spore release in 1997, 1998 and 1999, respectively). The development of phoma leaf spotting in untreated plots differed between the seasons; in 1997/98 (Fig. 2a) the incidence of affected plants reached 20% in late October and the maximum observed incidence was 32% in untreated plots (cv. Lipton) in mid-December. In 1998/99 (Fig. 2b), although a few leaf lesions were visible in late October, the main leaf spotting epidemic began 3 weeks later than in 1997/98, with incidence reaching 20% in mid-November and a maximum of 63% in mid-December. In contrast, in 1999/2000 (Fig. 1c) the epidemic was early and severe, with incidence reaching 20% in early October and a maximum of 84% in untreated plots (cv. Lipton) in mid-October. In 1999/2000, a period of dry weather immediately after sowing caused two phases of emergence. Approximately 60% of seedlings emerged one week after sowing, followed by the remaining 40% about 3 weeks later. As leaf spotting was relatively early and severe, the 84% of plants with leaf spots on 12 October 1999 included plants with lesions on leaves three, four and five (from the first plants to emerge) and plants with severe lesions on the cotyledons and first two leaves (from the second emergence phase).

At Boxworth, the first signs of phoma leaf spot were detected in late October 1998 and experimental plots showed 13.8% plants affected on 3 November, about five weeks after first ascospores were detected (and four weeks after the first large spore catch). In 1999, phoma leaf spot was present on 4% plants on 28 September and this had increased to 48% incidence by 5 October (Fig. 3) - four weeks after the first spore catch and only two weeks after the first large ascospore capture.

### 3.4 Discussion

Results from this project indicate that the risk of severe basal canker epidemics in southern England are greatest when there is a high incidence of phoma leaf spotting early in the season. The severity of basal canker epidemics and size of related yield losses were greater in 1997/98 and 1999/2000, when incidence of leaf spotting increased in October, than in 1998/1999, when incidence did not increase until November/December. Other studies in southern England (Hammond & Lewis, 1986; Gladders *et al.*, 1998; Zhou *et al.*, 1999; Sun *et al.*, 2000), France (Poisson & Pérès, 1999) and Australia (Barbetti & Khangura, 1997) have also shown that the most severe crown cankers develop from spots on cotyledons and young leaves early in the growing season. Infections of early leaves are likely to allow the pathogen to spread rapidly to the stem due to the small size of early leaves and the relatively warm temperatures of early autumn in southern England. The results from Rothamsted experiments suggest that fungicides are able to protect uninfected leaves for about one month. New lesions continued to appear immediately after a fungicide application in late September 1999, having presumably been initiated by the ascospores released in mid-September, before the fungicide was applied. However, in both the 1999/2000 field and controlled environment experiments, the fungicide was not able to eradicate established infections, but did reduce the growth rate of the fungus isolated from the lesions or infected leaves. Furthermore, fungicides decreased the incidence and severity of basal canker in field experiments (the incidence of basal cankers was 10% at harvest by comparison with 50% of plants with phoma leaf spotting after the treatment (J West, unpublished data). This suggests that the canker fungus can now be prevented from reaching the stem by fungicide applications soon after lesions appear on leaves. Results from this project suggest delays of a few days in making applications are not serious particularly if plants are large.

Since leaf spotting early in the autumn poses the greatest risk and accurate timing of sprays is essential to prevent development of severe basal cankers, a method of predicting early development of leaf spotting would be invaluable (West *et al*., 2001). These results suggest that the first increase in leaf spotting may be related to the first release of ascospores after crop establishment. The observed incubation periods between the initial release of ascospores and the appearance of leaf spotting in the field experiments fitted with results of controlled environment experiments (Biddulph *et al.*, 1999; Toscano-Underwood *et al.*, 2001). It would be impracticable for growers to assess concentrations of air-borne ascospores on their farms (which requires specialist equipment) or to estimate maturation of ascospores in pseudothecia (which is technically difficult and did not relate well to numbers of ascospores released in the Rothamsted experiments, perhaps because sample sizes were too small) (West *et al.*, 1999). However, in France the first release of ascospores has been related to the number of rain-days (16-19) occurring since harvest (Pérès & Poisson, 1997). In these Rothamsted experiments the number of rain-days (> 0.2 mm) between harvest and the first release of ascospores varied from 17 to 28 in the three seasons but the number of rain-days between 1 August and the first ascospore release was more consistent (16-23). At Boxworth, the number of rain days were 16 and 17, which fitted the French model. The temperature criteria favouring ascospore production also merit further

validation as a fall in temperature to <14°C was promising. However, it appears that dew rather than rain may be able to stimulate spore release.

In analyses of survey data on phoma stem canker epidemics in eastern England over a 20-year period, rainfall in August and September was found to be an important indicator of epidemic severity the following summer (Gladders & Symonds, 1995). Thus it might be possible to develop a weather-based scheme for predicting the first release of ascospores, and thus initial development of leaf spotting, for use by growers in southern England (West *et al.*, 1999). Since the timing of sprays is less critical later in the autumn when temperatures are lower, observations on the occurrence of leaf spotting on crops should then be sufficient to guide spray decisions. Further validation of the criteria for the number of rain days required for ascospore maturation and/ or total rainfall should be considered as this would enable forecasts to be made without the need for extensive spore trapping

Results of these and other (e.g. Gladders *et al.*, 1998) winter oilseed rape experiments have shown that fungicides can give good control of severe basal canker epidemics with consequent yield responses and that accurate timing is essential if fungicides are to be effective. If the onset of phoma leaf spotting in the autumn does not occur until November/December (as in the 1998/99 experiment), it seems unlikely that more than one spray can be justified against phoma stem canker. However, if the incidence of leaf spotting increases rapidly in early October (as in 1999/2000), then a single spray is unlikely to be sufficient to control the epidemic and a two-spray programme is needed. However, application of more than two fungicide sprays against phoma stem canker in the period autumn to spring did not increase yield greatly, and is unlikely to be justified in southern England. For many growers in the UK, it will be necessary to assess the risk of severe phoma stem canker epidemics together with risks posed by other diseases such as light leaf spot, as part of an integrated strategy for disease management on winter oilseed rape (Fitt *et al*., 1997; Gladders *et al*., 1998).

# Chapter 4

## Optimising fungicide spray treatments for canker control

### 4.1 Introduction

Stem canker, caused by the fungal pathogen *Leptosphaeria maculans*, is a major disease of winter oilseed rape, causing yield losses of up to 10 – 20% in some years. The disease is monocyclic: ascospores are released in the autumn, causing phoma leaf spot, and the fungus then grows systematically within the plant and appears as stem canker later in the season. The most damaging cankers occur at the base of the stem, although lesions also occur higher up the stem, often at the leaf axils. Since there is no complete cultivar resistance to the disease and cultural practices can only help reduce it, fungicides are needed to give effective control. Timing of fungicide sprays is critical since once the fungus has penetrated the stem, they are less effective. Treatments also need to be cost-effective after allowing for costs of the fungicide and their application. This is most likely to be achieved by controlling the phoma leaf spots which produce moderate or severe cankers before harvest. Fungicide treatment and disease monitoring in this project will define when yield loss is occurring.

In some areas, light leaf spot, caused by the fungal pathogen *Pyrenopeziza brassicae*, is also a disease of winter oilseed rape of major economic. In contrast to stem canker, it is a polycyclic disease: conidia, produced throughout the season whenever conditions are favourable, are spread by rain splash to previously healthy leaves, stems or pods. Strategies for canker control also need to be adapted for situations when both light leaf spot and canker are present. Experiments at Rothamsted and ADAS High Mowthorpe are oriented to look at control of both these diseases.

The aim of this experiment was to study the effect of fungicide timing on disease control and yield. The study considered the number and timing of applications of fungicide on cultivar Apex, which has moderate disease resistance. Practical concerns about the effect of weather delaying the first autumn spray or the spring stem extension spray was also addressed in these field experiments.

### 4.2 Objectives

To improve the strategies for control of stem canker and light leaf spot on winter oilseed rape by:

- optimising fungicide timing for stem canker and light leaf spot control and yield response.

- quantifying yield losses in relation to development of phoma leaf spot and severity of stem canker epidemics.

### 4.3 Materials and methods

Field experiments were carried out with a standard design at three sites (ADAS Boxworth, Cambs, ADAS High Mowthorpe, North Yorks and IACR Rothamsted, Herts) for the cropping years 1998/99 and 1999/2000 on winter oilseed rape cv. Apex (NIAB rating of 6 for both canker and light leaf spot resistance in 1998). A randomised block design with four replicates of 19 different fungicide treatments and a double untreated control was used with 3 (Rothamsted) or 4 (ADAS sites) replicates (Table 1). The fungicides difenoconazole (as Plover) at the rate of 0.25 l/ha (product) and carbendazim (as Bavistin DF) at 0.25 l/ha product were used as a tank mixture for all treatments. Treatments were organised in a full factorial design with spray dates of application to provide varying degrees of fungicidal control from disease onset in autumn until the early stem extension stage in spring. In addition, two treatments were used to examine the effects of delaying the first autumn spray by two or four weeks. A comparable pair of treatments in the spring was used to determine the effects of delaying the final spring spray by two or four weeks. Actual treatment dates are provided for all sites in Table 2. Weather conditions hampered spraying at High Mowthorpe in 1998/99 and three of the four supplementary timings were not applied.

Treatments were applied by OPS hand-held spraying equipment. Conditions at spraying and the sprayer details are given in Appendix B.

**Table 1.** Fungicide treatment timings and scheduled dates of application.

| Treatment code | Scheduled application dates | Treatment application dates | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | (2) | Delay (2) 2 wk | Delay (2) 4 wk | (3) | (4) | (5) | Delay (5) 2 wk | Delay (5) 4 wk |
| 1 | Untreated | | | | | | | | |
| 2 | Disease onset/late Oct | + | | | | | | | |
| 3 | 4-6 weeks later than (2) (early Dec) | | | | + | | | | |
| 4 | 4-6 weeks later than (3) (Jan) | | | | | + | | | |
| 5 | 4-6 weeks later than (4) (early March) | | | | | | + | | |
| 6 | 2+3 | + | | | + | | | | |
| 7 | 2+4 | + | | | | + | | | |
| 8 | 2+5 | + | | | | | + | | |
| 9 | 3+4 | | | | + | + | | | |
| 10 | 3+5 | | | | + | | + | | |
| 11 | 4+5 | | | | | + | + | | |
| 12 | 2+3+4 | + | | | + | + | | | |
| 13 | 2+3+5 | + | | | + | | + | | |
| 14 | 3+4+5 | | | | + | + | + | | |
| 15 | 2+4+5 | + | | | | + | + | | |
| 16 | 2+3+4+5 | + | | | + | + | + | | |
| 17 | 2 weeks after 2, then 3+4+5 | | + | | + | + | + | | |
| 18 | 4 weeks after 2, then 3+4+5 | | | + | + | + | + | | |
| 19 | 2+3+4, then 2 weeks after 5 | + | | | + | + | | + | |
| 20 | 2+3+4, then 4 weeks after 5 | + | | | + | + | | | + |

**Table 2.** Dates of fungicide applications at all sites 1998-2000.

| Treatment timing | Schedule of applications | Actual dates of spray applications | | | | | |
|---|---|---|---|---|---|---|---|
| | | Boxworth 1998/99 | High Mowthorpe 1998/99 | Rothamsted 1998/99 | Boxworth 1999/00 | High Mowthorpe 1999/00 | Rothamsted 1999/00 |
| 1 | Untreated | | | | | | |
| 2 | Disease onset/late Oct | 20.10.98 | 02.12.98 | 06.11.98 | 06.10.99 | 09.11.99 | 05.10.99 |
| 3 | 4-6 weeks later than (2) (early Dec) | 03.12.98 | 01.02.99 | 16.12.98 | 15.11.99 | 17.01.00 | 03.11.99 |
| 4 | 4-6 weeks later than (3) (Jan) | 21.01.99 | 24.02.99 | 21.01.99 | 10.01.00 | 22.02.00 | 13.12.00 |
| 5 | 4-6 weeks later than (4) (early March) | 09.03.99 | 16.04.99 | 25.02.99 | 22.02.00 | 31.03.00 | 20.01.00 |
| | 2 weeks after (2) | 04.11.98 | No spray | 19.11.98 | 22.10.99 | 29.11.99 | 25.10.99 |
| | 4 weeks after (2) | 11.11.98 | No spray | 03.12.98 | 03.11.99 | 13.12.99 | 29.10.99 |
| | 2 weeks after (5) | 25.03.99 | 21.04.99 | 10.03.99 | 13.03.00 | 10.04.00 | 03.02.00 |
| | 4 weeks after (5) | 08.04.99 | No spray | 25.03.99 | 20.03.00 | 28.04.00 | 19.02.00 |

**Sampling and assessment**

Disease assessments were carried out to a standard protocol. The incidence (% plants affected) and severity (e.g. % leaf or pod area affected or stem disease index) of each disease was recorded on leaves, stems and pods when these were present. Stem canker, phoma stem lesions, botrytis and sclerotinia stem rots were assessed on a 0 – 4 score (0 = healthy, 1 = less than half stem girdled, 4 = plant dead). Light leaf spot on stems was recorded as percentage stem area affected.

Field samples representative of the experimental area were 25 plants and full trial assessments were 10 plants per plot. To ensure detection of light leaf spot on leaves, samples were kept for 24 hour at ambient temperatures or stored cool for longer periods before assessment. Pre-harvest assessments were made in the field at some sites. After initial field samples of 25 plants, control plots were sampled at or close to each spray application and then full plot assessments made at intervals to coincide with spray applications and six week intervals thereafter until crop maturity. Plant populations were counted after harvest using five quadrats ($0.25m^2$) per plot.

**Statistical analyses**

Data were collated and checked at each site and analysed by GENSTAT at IACR Rothamsted. Tabulated results are presented after analysis of variance with standard errors of the difference between treatment means (SED), residual degrees of freedom (df) and level of significance of the analysis of variance (F test). Where there were no significant treatment effects, this is indicated by 'ns' in tables of results. Data which showed a skewed distribution were subjected to a logistic transform and re-analysed. Transformed data are presented when this enabled variates to be analysed.

**Methods at individual sites**

<u>Boxworth 1998/1999</u>

The experiment was sown on 3 September 1998 and made steady growth in autumn. There was no inoculation of plots at this site, but the crop was close to oilseed rape residues from the previous year (c. 100 m), though separated from these by a narrow belt of trees. Oilseed rape is grown intensively on surrounding farms and disease pressure from phoma is high. Plot size was 21m x 4 m.

Agronomic details and agrochemical usage are given in Appendix A. Fertiliser, molluscicide, insecticide and herbicide inputs were applied to ensure the crop reached a good commercial standard based on crop monitoring and local experience of problems.

Disease assessments were made throughout the life of the crop (Table 3). Spore trapping of ascospores of *Leptosphaeria maculans* was done using a Burkard volumetric spore trap operated within a circle of oilseed rape debris collected after harvest at ADAS Boxworth. The debris was kept outdoors and subject to natural weathering. The spore trap provided daily records of spores/$m^3$ from 26 August 1998 to 6 April 1999. Meteorological records were collected within the crop using a Delta T data logger with sensors for temperature, relative humidity, surface wetness (3 heights after stem extension) and rainfall, which was operated in an adjacent field within an oilseed rape crop. An automatic weather station was also operational at Boxworth.

Plots were harvested on 18 July using a Sampo 2025 plot combine and harvested plot lengths were recorded individually. Harvesting conditions were good with very little lodging. Moisture content was assessed by a Dickey-John GS2000.

**Table 3.** Details of assessments at Boxworth 1998/99.

| Date sampled | Date assessed | Growth stage | Assessment | Assessment of | | |
|---|---|---|---|---|---|---|
| | | | | Leaves | Stems | Pods |
| 14.10.98 | 15.10.98 | 1,04 | 25 plants | + | | |
| 22.10.98 | 23.10.99 | 1,06 | 6 control plots | + | | |
| 03.11.99 | 03.11.99 | 1,09 | 6 control plots | + | | |
| 20.11.99 | 23.11.99 | 1,10 | 6 control plots | + | | |
| 03.12.98 | 7-8.12.99 | 1,10 | All plots | + | | |
| 18.01.99 | 19-20.01.99 | 1,11 | All plots | + | | |
| 08.03.99 | 10-11.03.99 | 2,8, 3,3 | All plots | + | | |
| 14.04.99 | 15.04.99 | 4,1-4,2 | All plots | + | + | |
| 26.05.99 | 27.05.99 | 5,9, 6,2 | All plots | + | + | + |
| 28.06.99 | 28.06.99 | 6,5 | All plots | | + | + |
| 18.07.99 | 18.07.99 | 6,9 | All plots harvested | | | |
| 23.07.99 | 23.07.99 | stubble | Untreated + treatments 19&20 - plant population | | + | |

**High Mowthorpe 1998/1999**

The experiment was sown on 7 September 1998 and the crop grew slowly with an adequate plant population. Plot size was 24m x 1.5 m. Autumn fungicide treatments were delayed by several weeks because of adverse weather conditions and the first spray was applied on 2 December. This coincided with the first significant phoma spotting. There was no inoculation of plots at this site, although oilseed rape is grown on surrounding farms and disease pressure from phoma and light leaf spot was moderate.

Agronomic details and agrochemical usage are given in Appendix A. Fertiliser, molluscicide, insecticide and herbicide were applied to minimise impact of pests, weeds and other factors which might otherwise impair disease control programmes. A summary of the main disease assessments is in Table 4. Plots were harvested on 29 July 1999 using a Sampo 2025 plot combine.

**Table 4.** Details of full assessments at High Mowthorpe in 1998/1999.

| Date | Assessment | Assessment of | | |
|---|---|---|---|---|
| | | Leaves | Stems | Pods |
| 16.12.98 | All plots | + | | |
| 13.01.99 | All plots | + | | |
| 24.02.99 | All plots | + | | |
| 16.04.99 | All plots | + | | |
| 22.04.99 | All plots | + | | |
| 15.07.99 | All plots | | + | + |
| 29.07.99 | All plots harvested | | | |
| 03.08.99 | All plots - plant population | | + | |

**Rothamsted 1998/1999**

Soil conditions were good at sowing on 25 August and frequent autumn rain ensured emergence and growth was rapid and even. There was some slug and pigeon damage in the autumn at one end of the experimental area, but the plants soon recovered. A warmer than average spring ensured rapid development: stem extension began in mid February and by early March flower buds were visible. Nitrogen fertilizer applied on 8 February caused some temporary leaf scorch.

Rape debris from experimental plots harvested in July 1997 was scattered over each plot (0.25 bales/plot) on 6 November to help ensure moderately severe epidemics of phoma and light leaf spot. Phoma leaf spot was first seen on 20 October, but, due to adverse weather, the first spray was not applied until two weeks later. All other sprays were applied according to the schedule. An on-site weather station was assembled on 8 October to record temperature, relative humidity, leaf surface wetness and rainfall.

Fertilizer, molluscicide, insecticide and herbicide were applied as necessary to ensure crop growth was to a good commercial standard. Details are given in Appendix A.

Samples of 25 plants from untreated plots were assessed weekly from mid September to mid December and then monthly until mid-March, to monitor disease progress. The plants were assessed on the same day as they were sampled, but on some occasions, they were re-assessed after incubating in plastic bags for a few days at 5 – $10^0$C to check for light leaf spot sporulation. On six occasions, all plots were assessed (Table 5).

For the first three assessments, the plants were first placed in plastic bags and incubated for between 2 and 5 days at 7 - 9°C to allow light leaf spot symptoms to develop.

**Table 5.** Details of full disease assessments at Rothamsted, 1998/1999.

| Date of sample | Growth stage | Assessment method | | Assessment of | | |
|---|---|---|---|---|---|---|
| | | After incubation | In field | Leaves | Stems | Pods |
| 1.12.98 | 1,9 | + | | + | | |
| 20.1.98 | 1,11 | + | | + | | |
| 18.2.99 | 1,12 | + | | + | | |
| 31.3.99 | 3,6 | | + | + | + | |
| 26.5.99 | 5,9 | | + | | + | + |
| 30.6.99 | 9,5 | | + | | + | + |

Plots were harvested on 17 July using a small plot combine. Harvest conditions were good with very little lodging. Plant numbers/$m^2$ were assessed after harvest in six untreated plots and in three plots which had received four fungicide sprays (treatment 18) by counting stems in 0.25$m^2$ quadrats (5 quadrats/plot).

**Boxworth 1999/2000**

Sowing was completed on 26 August 1999 in a field cropped continuously with oilseed rape since autumn 1987. The site had been ploughed to bury most crop residues; nevertheless it provided high risk conditions for phoma development. Oilseed rape is grown intensively on surrounding farms and disease pressure from phoma was also generally high in the area. Plot size was 24m x 4 m. Plant growth in autumn was very vigorous and the 10-leaf stage was reached by mid October. Fertiliser, molluscicide, insecticide and herbicide were applied to ensure that the crop performance met experimental requirements with no interference from factors other than disease. A Delta T data logger with sensors for temperature, relative humidity, surface wetness (3 heights after stem extension) and rainfall was operated in an adjacent field within an oilseed rape crop. An automatic weather station was also operational at Boxworth.

After an initial field sample of 25 plants, control plots were sampled at or close to each spray application and then full plot assessments (on 10 plants per plot) made at approximately 6 weekly intervals from December until crop maturity (Table 6). The plants were assessed after a short period of incubation except

for the final assessment on 29 June, which was made on the same day as plants were sampled. Plots were harvested on 18 July 2000 using a Sampo 2025 plot combine and harvested plot lengths were recorded individually. Harvesting conditions were good with very little lodging. Moisture content was assessed by a Dickey-John GS2000.

**Table 6.** Details of assessments at Boxworth 1999/2000.

| Date of sample | Date assessed | Growth stage | Assessment | Assessment of | | |
|---|---|---|---|---|---|---|
| | | | | Leaves | Stems | Pods |
| 28.09.99 | 28.09.99 | 1,04 | 25 plants | + | | |
| 05.10.99 | 05.10.99 | 1,05 | 25 plants | + | | |
| 12.10.99 | 12.10.99 | 1,07 | 25 plants | + | | |
| 18.10.99 | 18.10.99 | 1,10 | 8 control plots | + | | |
| 25.10.99 | 25.10.99 | 1,12 | 25 plants | + | | |
| 01.11.99 | 01.11.99 | 1,12 | 25 plants | + | | |
| 08.11.99 | 08.11.99 | 1,13 | 25 plants | + | | |
| 15.11.99 | 15.11.99 | 1,14 | 25 plants | + | | |
| 22.11.99 | 22-23.11.99 | 1,15 | All plots | + | | |
| 30.11.99 | 30.11.99 | 1,16 | 25 plants | + | | |
| 07.12.99 | 07.12.99 | 1,16 | 25 plants | + | | |
| 14.12.99 | 14.12.99 | 1,17 | 25 plants | + | | |
| 11.01.00 | 12-13.01.00 | 1.16-17 | All plots | + | | |
| 19.01.00 | 19.01.00 | 1,18-1,20 | 25 plants | + | | |
| 15.02.00 | 15.02.00 | 2,2-2,3 | 25 plants | + | | |
| 07.03.00 | 08-09.03.00 | 2,8-2,9, 3,1-3,3 | All plots | + | | |
| 28.03.00 | 29-31.03.00 | 3,5; 4,0 | All plots | + | + | |
| 22.05.00 | 23-24.05.00 | 6,2 | All plots | + | + | |
| 29.06.00 | 29.06.00 | 6,5 | All plots | | + | + |
| 18.07.00 | 18.07.00 | 6,9 | All plots harvested | | | + |
| 19.07.00 | 19.07.00 | stubble | All plots - plant population | | + | |

**High Mowthorpe 1999/2000**

The experiment was sown on 31 August 1999, established well and grew steadily in autumn. The first fungicide sprays were applied on 9 November, which coincided with 20% plants showing phoma leaf spot. The next two sprays were delayed by adverse weather, including snow cover in November. The second main treatment scheduled for 21 December was also delayed by adverse weather conditions until 17 January. Agronomic inputs were directed to minimise interference from pests and weeds so that disease control objectives could be met in a crop grown to good commercial standard (Appendix A). Disease assessments on weekly samples in autumn and subsequently on the whole experiment (Table 7). Plots were swathed on 24 July, harvested on 6 August 2000 and plant counts completed on 8 August.

**Table 7**. Details of full experiment assessments at High Mowthorpe, 1999/2000.

| Date of sample | Date assessed | Assessment | Assessment of | | |
|---|---|---|---|---|---|
| | | | Leaves | Stems | Pods |
| 20.01.00 | 20.01.00 | All plots | + | | |
| 01.03.00 | 01.03.00 | All plots | + | | |
| 25.05.00 | 25-30.05.00 | All plots | + | + | |
| 20.07.00 | 20-27.07.00 | All plots | | + | + |
| 06.08.00 | 06.08.00 | All plots harvested | | | |
| 08.08.00 | 08.08.00 | All plots - plant population | | + | |

**Rothamsted 1999/2000**

Plots (20m x 3m) were sown on 27 August 1999 and showed some uneven emergence; by 28 September some plants were at growth stage 1,6, while some were just emerging. However, growing conditions in autumn and spring were good and development was rapid. Five bales of rape debris (cf. 15 bales in 1998) from experimental plots harvested in July 1998 were scattered over the plots on 12 October (6 November in 1998) to help initiate epidemics of phoma and light leaf spot. Untreated plots were sampled weekly from mid September to mid December and then monthly until mid-March, to monitor disease progress. On 11 occasions, all plots where the spray programme had been completed were assessed (Table 8). In addition, on two occasions, treatments 17 and 18 were assessed after only the first spray had been applied. Phoma leaf spot was first seen on 28 September and the first spray was applied on 5 October (one month earlier than in 1998). All other sprays were also applied according to the schedule, except for treatments 17 and 18 when the first sprays were only 4 days apart. Fertilizer, molluscicide, insecticide and herbicide were applied as necessary to produce a crop of good commercial standard. Details are given in Appendix A.

**Table 8.** Assessments of completed spray programmes at Rothamsted 1999/2000.

| Date of sample | Growth stage | Assessment method | | Assessment of | | |
|---|---|---|---|---|---|---|
| | | Before incubation | After incubation | Leaves | Stems | Pods |
| 12.10.99 | 1,9 | + | | + | | |
| 19.10.99 | 1,9 | + | + | + | | |
| 26.10.99 | 1,10 | + | + | + | | |
| 02.11.99 | 1,11 | + | | + | | |
| 09.11.99 | 1,11 | + | + | + | | |
| 23.11.99 | 1,12 | + | | + | | |
| 06.01.00 | 1,13 | + | + | + | | |
| 15.02.00 | 2,1 | | + | + | | |
| 27.03.00 | 3,6 | | + | + | + | |
| 14.06.00 | 6,3 | + | | | + | + |
| 12.07.00 | 9,9 | + | | | + | + |

An on-site weather station was assembled on 17 September to record temperature, relative humidity, leaf surface wetness and rainfall. Plots were desiccated on 12 July and harvested on 20 July using a small plot

combine. Harvest conditions were good. High summer rainfall and low light levels were thought to have contributed to the generally poor yields this season.

### 4.4 Results

**Boxworth 1998/1999**

Initial establishment from 3 September sowing was generally satisfactory though autumn growth was rather slow in October because of cold conditions and a few thinner areas of crop were apparent. The trial area had reasonably uniform plant population and pest damage was limited. After a mild winter, crop development was earlier than average and stem extension began in February and flowering started in early April. The crop ripened very rapidly in late June despite adequate soil moisture.

Disease development on untreated plots

Downy mildew was active on the first leaves in October, but then declined during November before remaining at a high incidence from December until April (Table 9). Phoma leaf spot was first seen in late October, and built up rather slowly until late November/early December, when 65% plants were affected. There was a high incidence of phoma for the rest of the winter and during flowering, and symptoms only declined as lower leaves were lost in late May. The first phoma stem lesions were found in mid April, but significant infection was only found in late May when canker lesions (55% plants affected) were slightly more common than stem lesions (41% plants). The incidence of cankers on untreated plots increased to 76% of plants affected with basal stem canker and 81% with stem lesions by 28 June. However, disease severity remained mainly in the slight category.

Light leaf spot was only seen on the occasional plant on leaves and stems. There were low levels of stem diseases - botrytis, sclerotinia and rhizoctonia (at the stem base) which in the case of sclerotinia caused premature ripening in small patches. There was some disease on pods by 28 June, with alternaria mainly confined to minor spotting and botrytis developing on pods damaged by bladder pod midge (Table 9).

Disease control

The first assessment on 8 December showed that the first two sprays (20 October and 4 November) had given good control of incidence and severity of phoma leaf spot (Table 10). The third spray on 11 November

appeared to be taking effect, though there was no control on an incidence basis. In January, the November + December sprays were most effective and even the October + December programme had limited effect on disease incidence. In late March, only the January spray (of the single spray treatments) gave moderate control, whilst all the two spray treatments except Oct + Dec and the multiple sprays gave some reduction in disease incidence and severity. In April there was a strong effect of the 9 March spray with no additional advantage from multiple earlier sprays. In May, there were no treatment differences.

At the May assessment there were significant differences between untreated and treated plots, with most treatments reducing the incidence and severity of basal cankers and stem lesions by more than 50% (Table 11). Of the single sprays, mid January was the most effective and March appeared to be ineffective. All programmes comprising two or more sprays significantly reduced incidence and severity of the disease with Oct + Mar being rather less effective than many of the other two spray treatments. Programmes of three or four sprays gave similar or only slightly better control than two- spray programmes. Control of stem lesions in late May was slightly less effective than for canker, but January was the most effective single spray. A range of timing combinations gave similar results.

In late June, good control of canker (>80%) was achieved with Dec + Jan and Jan + Mar two spray programmes, whilst Dec + Mar and Oct + Mar were rather poor. All the three and four spray programmes worked well with the exception of Oct + Dec + Jan. Indeed, four sprays at standard timings gave complete control of canker. Phoma stem lesions were less well controlled; a few programmes reduced the untreated incidence of 81% to less than 30%. Programmes with a Jan + Mar component appeared to be most effective. The best single spray timing was Mar, which contrasted with canker where January timing was most effective.

There was negligible light leaf spot in this experiment and no firm conclusions can be drawn about spray timing requirements (Table 12).

There were low levels of alternaria leaf spotting during the winter, though except for December levels, these were too low to identify treatment effects. In June, there was some pod infection, but this was very low and unlikely to have affected yield (Table 12).

The plant population averaged about 60 plants/$m^2$ on six untreated plots and 79 plants/$m^2$ receiving four sprays (treatments 19 and 20). These both represented robust populations with adequate populations for good commercial yields. There is, however, the possibility that treatments reduced plant losses.

This was a high yielding site which averaged 4.4 t/ha. There were no significant effects of fungicide on yield. A separate factorial analysis of yield showed that responses to Jan timing (P = 0.054) and Dec + Jan component (P= 0.051) were close to being significant. Examination of mean responses showed trends of 0.05 t/ha per spray with up to three spray applications (Table 13).

**Table 9.** Disease development on winter oilseed rape plants and leaves in untreated control plots Boxworth 1998/1999.

| Date | Leaves | | | | | | Stems | | | | | Pods | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| assessed | Downy mildew | Phoma | Light leaf spot | Bot | Alter-naria | PM | Phoma * | Light leaf spot | Bot* | Sclero -tinia | Canker* | Downy mildew | Bot | Alter-naria | Light leaf spot | Phoma |
| 15.10.99 | 100 (0.76) | 0 | | | | | | | | | | | | | | |
| 23.10.98 | 100 (0.77) | 0 | 0 | 0 | 1.3 (0.003) | 0 | | | | | | | | | | |
| 03.11.98 | 50 (0.43) | 13.8 (0.03) | 0 | 0 | 0 | 0 | | | | | | | | | | |
| 23.11.98 | 46 (0.14) | 25.0 (0.03) | 0 | 0 | 0 | 0 | | | | | | | | | | |
| 08.12.98 | 94 (0.21) | 65.0 (0.19) | 0 | 0 | 14 (0.003) | 0 | | | | | | | | | | |
| 19.01.99 | 97 (0.28) | 100.0 (0.67) | 0 | 0 | 0 | 0 | | | | | | | | | | |
| 11.03.99 | 99 (0.23) | 90.0 (0.44) | 0 | 0 | 1 (0.001) | 0 | | | | | | | | | | |
| 23.03.99 | 93 (0.25) | 97.0 (0.85) | 0 | 0 | 3 (0.01) | 0 | 0 | 0 | 0 | 0 | 0 | | | | | |
| 15.04.99 | 90 (1.60) | 92..5 (0.84) | 1.2 (0.006) | 0 | 0 | 0 | 1.2 (0.12) | 0 | 0 | 0 | 0 | | | | | |
| 27.05.99 | 37.5 (0.07) | 22.5 (0.09) | 1.2 (0.004) | 92.5 (0.28) | 22.5 (0.03) | 0 | 41.2 (0.41) | 3.7 (0.11) | 1.2* (0.04) | 1.2 (0.04) | 55.0 (0.56) | 0 | 0 | 0 | 4 (0.09) | 0 |
| 28.06.99 | | | | | | | 81.2 (0.95) | 1.2 (0.02) | 0 | 3.7** (0.14) | 76.2 (1.05) | 15 (0.011) | 13.8 (0.22) | 45 (0.04) | 3.7 (0.06) | 2,5 (0.04) |
| | | | | | | | | | | | | | | | | |

Bot = Botrytis PM = Powdery Mildew

* Nil Rhizoctonia in controls but up to 5% in treatments

** Sclerotinia on pods of 1.2% plants (0.01% pods)

**Table 10.** Effect of spray timing on phoma leaf spot incidence (% plants and % leaves affected) and severity (% leaf area affected) Boxworth 1999.

| Treatment code | Treatment | Assessment dates | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 8.12.98 | | 19.1.99 | | 31.3.99 | | 15.4.99 | | 27.5.99 | |
| | | % plants | % leaf area | % *plants | %leaf area | % plants | %leaf area* | % plants | %leaf area* | % plants | % leaf area |
| 1 | Untreated | 78.5 | 0.21 | 98.5 (1.43) | 0.96 | 86.7 | 0.39 (-2.77) | 92.5 | 0.84 (-2.58) | 22.5 | 0.09 (-3.40) |
| 2 (20/10) | Disease onset/late Oct | 21.3 | 0.02 | 100.0 (1.52) | 0.97 | 82.5 | 0.34 (-2.79) | 85.0 | 0.53 (-2.67) | 27.5 | 0.06 (-3.41) |
| 3 (03/12) | 4-6 weeks later than 2 | 67.5 | 0.16 | 87.5 (0.94) | 0.38 | 68.7 | 0.24 (-2.95) | 75.0 | 0.48 (-2.67) | 27.5 | 0.06 (-3.40) |
| 4 (21/1) | 4-6 weeks later than 3 | | | | | 48.7 | 0.13 (-3.19) | 85.0 | 0.42 (-2.68) | 25.0 | 0.05 (-3.43) |
| 5 (09/03) | 4-6 weeks later than 4 | | | | | | | 20.0 | 0.05 (-3.55) | 25.0 | 0.10 (-3.40) |
| 6 | 2+3 | 19.2 | 0.03 | 78.3 (0.68) | 0.30 | 75.0 | 0.36 (-2.83) | 82.5 | 0.68 (-2.54) | 22.5 | 0.06 (-3.46) |
| 7 | 2+4 | | | | | 36.2 | 0.08 (-3.56) | 75.0 | 0.48 (-2.74) | 17.5 | 0.06 (-3.52) |
| 8 | 2+5 | | | | | | | 55.0 | 0.15 (-3.17) | 12.5 | 0.05 (-3.50) |
| 9 | 3+4 | | | | | 42.5 | 0.18 (-3.15) | 75.0 | 0.47 (-2.77) | 12.5 | 0.04 (-3.53) |
| 10 | 3+5 | | | | | | | 30.0 | 0.20 (-3.21) | 22.5 | 0.04 (-3.51) |
| 11 | 4+5 | | | | | | | 32.5 | 0.18 (-3.17) | 12.5 | 0.03 (-3.61) |
| 12 | 2+3+4 | | | | | 38.7 | 0.09 (-3.31) | 77.5 | 0.31 (-2.86) | 17.5 | 0.05 (-3.51) |
| 13 | 2+3+5 | | | | | | | 30.0 | 0.11 (-3.38) | 17.5 | 0.04 (-3.52) |
| 14 | 3+4+5 | | | | | | | 30.0 | 0.11 (-3.33) | 17.5 | 0.03 (-3.61) |
| 15 | 2+4+5 | | | | | | | 30.0 | 0.40 (-3.13) | 22.5 | 0.05 (-3.48) |
| 16 | 2+3+4+5 | | | | | | | 15.0 | 0.03 (-3.58) | 20.0 | 0.05 (-3.49) |
| 17 | 2 weeks after 2, then 3+4+5 | 22.5 | 0.02 | 45.0 (-0.09) | 0.13 | 42.5 | 0.11 (-3.25) | 35.0 | 0.10 (-3.31) | 25.0 | 0.07 (-3.39) |
| 18 | 4 weeks after 2, then 3+4+5 | 60.0 | 0.09 | 37.5 (-0.24) | 0.12 | 30.0 | 0.06 (-3.42) | 22.5 | 0.06 (-3.45) | 12.5 | 0.02 (-3.61) |
| 19 | 2+3+4, then 2 weeks after 5 | | | | | | | 82.5 | 0.38 (-2.78) | 7.5 | 0.01 (-3.68) |
| 20 | 2+3+4, then 4 weeks after 5 | | | | | | | 57.5 | 0.23 (-2.98) | 5.0 | 0.03 (-3.66) |
| Mean | | 45.0 | 0.097 | 0.99 | 0.59 | 56.9 | -3.09 | 56.2 | -3.007 | 18.8 | 0.05 (-3.50) |
| SED (min rep) | | 11.8 | 0.062 | 0.249 | 0.155 | 11.36 | 0.162 | 15.07 | 0.247 | 10.21 | 0.162 |
| df | | 75 | 75 | 75 | 75 | 71 | 71 | 61 | 61 | 61 | 61 |
| F test | | <0.1% | <0.1% skew | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | NS | NS |

* Logit transformed data analysed

**Table 11.** Effect of spray timing on stem canker and stem lesion incidence (I) (% plants affected) and severity (S) (0 – 4 score) Boxworth 1999.

| Treatment | Treatment | Assess | Assessed 27.5.99 (* logit transform analysed) | | | | Assessed 30.6.99 | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| code | | 15.4.99 | canker | | stem lesions | | canker | | stem lesions | |
| | | Stem I | I | S | I* | S* | I | S | I | S |
| 1 | Untreated | 1.2 | 55.0 (0.10) | 0.56 (-2.55) | 41.2 (-0.17) | 0.41 (-2.72) | 76.2 | 1.05 | 81.2 | 0.95 |
| 2 (20/10) | Disease onset/late Oct | 2.5 | 27.5 (-0.61) | 0.27 (-3.08) | 32.5 (-0.48) | 0.32 (-2.97) | 57.5 | 0.65 | 57.5 | 0.70 |
| 3 (03/12) | 4-6 weeks later than 2 | 2.5 | 25.0 (-0.52) | 0.25 (-2.93) | 25.0 (-0.64) | 0.20 (-3.24) | 72.5 | 0.80 | 67.5 | 0.77 |
| 4 (21/1) | 4-6 weeks later than 3 | 0 | 12.5 (-0.92) | 0.12 (-3.26) | 7.5 (-1.14) | 0.07 (-3.46) | 30.0 | 0.37 | 67.5 | 0.80 |
| 5 (09/03) | 4-6 weeks later than 4 | 2.5 | 42.5 (-0.28) | 0.45 (-2.84) | 20.0 (-0.66) | 0.20 (-3.05) | 52.5 | 0.60 | 35.0 | 0.35 |
| 6 | 2+3 | 0 | 17.5 (-0.81) | 0.17 (-3.19) | 27.5 (-0.51) | 0.27 (-2.95) | 35.0 | 0.37 | 52.5 | 0.52 |
| 7 | 2+4 | 2.5 | 2.5 (-1.37) | 0.02 (-3.66) | 20.0 (-0.76) | 0.12 (-3.16) | 47.5 | 0.57 | 30.0 | 0.30 |
| 8 | 2+5 | 0 | 22.5 (-0.71) | 0.22 (-3.12) | 10.0 (-0.99) | 0.10 (-3.32) | 37.5 | 0.42 | 32.5 | 0.32 |
| 9 | 3+4 | 2.5 | 10.0 (-1.09) | 0.10 (-3.42) | 10.0 (-1.09) | 0.10 (-3.40) | 5.0 | 0.05 | 42.5 | 0.57 |
| 10 | 3+5 | 10.0 | 10.0 (-0.99) | 0.10 (-3.32) | 5.0 (-1.22) | 0.05 (-3.53) | 42.5 | 0.50 | 27.5 | 0.30 |
| 11 | 4+5 | 0 | 5.0 (-1.29) | 0.07 (-3.56) | 12.5 (-0.92) | 0.12 (-3.26) | 15.0 | 0.15 | 22.5 | 0.30 |
| 12 | 2+3+4 | 0 | 5.0 (-1.29) | 0.05 (-3.53) | 0.0 (-1.52) | 0.00 (-3.80) | 32.5 | 0.32 | 47.5 | 0.50 |
| 13 | 2+3+5 | 0 | 5.0 (-1.37) | 0.05 (-3.60) | 10.0 (-0.99) | 0.10 (-3.32) | 15.0 | 0.15 | 27.5 | 0.27 |
| 14 | 3+4+5 | 0 | 10.0 (-0.92 | 0.10 (-3.25) | 12.5 (-0.92) | 0.12 (-3.26) | 12.5 | 0.12 | 17.5 | 0.17 |
| 15 | 2+4+5 | 2.5 | 5.0 (-1.29) | 0.05 (-3.60) | 2.5 (-1.37) | 0.02 (-3.66) | 12.5 | 0.20 | 37.5 | 0.37 |
| 16 | 2+3+4+5 | 0 | 2.5 (-1.37) | 0.02 (-3.66) | 10.0 (-0.99) | 0.10 (-3.32) | 0.0 | 0.00 | 22.5 | 0.22 |
| 17 | 2 wk after T2+ 3+4+5 | 2.5 | 5.0 (-1.22) | 0.05 (-3.53) | 10.0 (-1.19) | 0.10 (-3.53) | 7.5 | 0.07 | 27.5 | 0.27 |
| 18 | 4 wk after T2+ 3+4+5 | 0 | 2.5 (-1.37) | 0.02 (-3.66) | 2.5 (-1.37) | 0.02 (-3.66) | 2.5 | 0.02 | 30.0 | 0.32 |
| 19 | 2+3+4 + 2 wk after T5 | 0 | 2.5 (-1.37) | 0.02 (-3.66) | 7.5 (-1.07) | 0.07 (-3.39) | 10.0 | 0.10 | 37.5 | 0.37 |
| 20 | 2+3+4 + 4 wk after T5 | 0 | 10.0 (-0.99) | 0.10 (-3.32) | 5.0 (-1.22) | 0.05 (-3.53) | 7.5 | 0.07 | 30.0 | 0.30 |
| Grand mean | | 1.4 | 15.8 (-0.93) | 0.16 (-3.30) | 14.9 (-0.92) | 0.15 (-3.30) | 30.8 | 0.36 | 41.7 | 0.46 |
| SED (61degrees of freedom) | | n/d | 0.315 | 0.263 | 0.291 | 0.239 | 9.51 | 0.132 | 13.62 | 0.165 |
| F test | | | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% |

**Table 12**. Effect of spray timing on alternaria leaf spot and light leaf spot incidence (I) (% plants affected) and severity (S) (% leaf area (L) or % pod area (P) affected) Boxworth 1999.

| Treatment code | Treatment | Assessment date | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Alternaria 8.12.98 | | Alternaria 19.1.99 | Alternaria 15.4.99 | Alternaria 27.5.99 | Light leaf spot 15.4.99 | | Light leaf spot 27.5.99 | | Alternaria 28.6.99 | |
| | | I | S (L) | I | I | I | I | S (L) | I | I Stem | I (P) | S (P) |
| 1 | Untreated | 19.5 | 0.006 | 0.0 | 6.2 | 22.5 | 1.2 | 0.006 | 1.2 | 3.7 | 45 | 0.036 |
| 2 (20/10) | Disease onset/late Oct | 5.6 | 0.002 | 0.0 | 0 | 17.5 | 0 | 0 | 0 | 0 | 27 | 0.024 |
| 3 (03/12) | 4-6 weeks later than 2 | 19.4 | 0.004 | 0.0 | 0 | 12.5 | 0 | 0 | 0 | 0 | 27 | 0.009 |
| 4 (21/1) | 4-6 weeks later than 3 | | | | 0 | 25.0 | 0 | 0 | 2.5 | 2.5 | 40 | 0.131 |
| 5 (09/3) | 4-6 weeks later than 4 | | | | 2.5 | 5.0 | 0 | 0 | 0 | 0 | 22 | 0.021 |
| 6 | 2+3 | 9.2 | 0.002 | 0.0 | 0 | 2.5 | 0 | 0 | 0 | 0 | 22 | 0.009 |
| 7 | 2+4 | | | | 0 | 15.0 | 0 | 0 | 0 | 0 | 30 | 0.007 |
| 8 | 2+5 | | | | 0 | 7.5 | 0 | 0 | 0 | 0 | 22 | 0.007 |
| 9 | 3+4 | | | | 0 | 27.5 | 0 | 0 | 0 | 0 | 22 | 0.014 |
| 10 | 3+5 | | | | 0 | 10.0 | 0 | 0 | 0 | 0 | 20 | 0.016 |
| 11 | 4+5 | | | | 0 | 7.5 | 0 | 0 | 0 | 0 | 17 | 0.002 |
| 12 | 2+3+4 | | | | 0 | 10.0 | 0 | 0 | 0 | 0 | 30 | 0.019 |
| 13 | 2+3+5 | | | | 0 | 5.0 | 0 | 0 | 0 | 0 | 17 | 0.033 |
| 14 | 3+4+5 | | | | 0 | 12.5 | 0 | 0 | 0 | 0 | 12 | 0.006 |
| 15 | 2+4+5 | | | | 0 | 7.5 | 0 | 0 | 0 | 0 | 17 | 0.008 |
| 16 | 2+3+4+5 | | | | 0 | 7.5 | 0 | 0 | 0 | 0 | 32 | 0.014 |
| 17 | 2 weeks after 2, then 3+4+5 | 0.0 | 0.000 | 0.0 | 0 | 17.5 | 2.5 | 0.07 | 0 | 0 | 25 | 0.007 |
| 18 | 4 weeks after 2, then 3+4+5 | 10.0 | 0.003 | 2.5 | 0 | 17.5 | 0 | 0 | 0 | 0 | 20 | 0.004 |
| 19 | 2+3+4, then 2 weeks after 5 | | | | 0 | 12.5 | 0 | 0 | 0 | 0 | 32 | 0.024 |
| 20 | 2+3+4, then 4 weeks after 5 | | | | 0 | 2.5 | 0 | 0 | 0 | 0 | 35 | 0.024 |
| Mean | | 12.5 | 0.003 | 0.12 | 0.7 | 12.7 | 0.2 | 0.004 | 0.2 | 0.48 | 27 | 0.022 |
| SED | min rep | 6.83 | 0.0034 | n/d | n/d | 11.65 | n/d | n/d | n/d | n/d | 12.2 | 0.0331 |
| Df | | 75 | 75 | 55 | | 61 | | | 61 | 61 | 61 | 61 |
| F test | | <0.1 % | 5% skew | | | ns skew | | | | | ns | ns |

**Table 13.** Effect of spray timing on yield, Boxworth 1999.

| Treatment code | Treatment | Yield at 90% dry matter (t/ha) |
|---|---|---|
| 1 | Untreated | 4.34 |
| 2 (20/10) | Disease onset/late Oct | 4.37 |
| 3 (03/12) | 4-6 weeks later than 2 (early Dec) | 4.30 |
| 4 (21/1) | 4-6 weeks later than 3 (Jan) | 4.30 |
| 5 (09/03) | 4-6 weeks later than 4 (early March) | 4.43 |
| 6 | 2+3 | 4.31 |
| 7 | 2+4 | 4.40 |
| 8 | 2+5 | 4.37 |
| 9 | 3+4 | 4.54 |
| 10 | 3+5 | 4.30 |
| 11 | 4+5 | 4.40 |
| 12 | 2+3+4 | 4.53 |
| 13 | 2+3+5 | 4.45 |
| 14 | 3+4+5 | 4.46 |
| 15 | 2+4+5 | 4.40 |
| 16 | 2+3+4+5 | 4.39 |
| 17 | 2 weeks after 2, then 3+4+5 | 4.34 |
| 18 | 4 weeks after 2, then 3+4+5 | 4.47 |
| 19 | 2+3+4, then 2 weeks after 5 | 4.45 |
| 20 | 2+3+4, then 4 weeks after 5 | 4.47 |
| Grand mean | | 4.40 |
| Mean of 1 spray | | 4.35 |
| Mean of 2 sprays | | 4.40 |
| Mean of 3 sprays | | 4.46 |
| Mean of 4 sprays | | 4.42 |
| SED (61Df)<br>F test | | 0.100<br>ns |

**High Mowthorpe 1998/1999**

The crop was sown on 7 September 1998 and made slow growth during 1998, reaching the six leaf stage by 1 December. Phoma leaf spot appeared in late October. Due to unfavourable weather conditions in late October and November, the spray programme was delayed by about 5 weeks, and not started until 2 December, which fitted in well with phoma leaf spot development reaching 20% plants affected (Table 14). Subsequent sprays were made as scheduled. Phoma leaf spotting remained fairly constant during the winter and then increased to affect 52% plants in mid February. Light leaf spot developed strongly in April and was not well controlled by fungicides (Table 15) in terms of its incidence, though its severity was reduced by 60-80% by most treatments.

Prior to harvest phoma stem lesions were present, but not controlled by fungicides and canker did not develop. Light leaf spot was present on most stems affecting up to 4.9% stem area and was controlled most effectively by treatments that included a combination of timings 3+4 (22 February + 16 April) (Table 16). Sclerotinia affected 13% plants overall and alternaria affected 0.5% stem area, both with no significant treatment effects.

Yield variation was higher than usual and no significant differences in yield were found between treatments, although the yields of all treated plots were higher than the untreated control by 0.07-0.85 t/ha (Table 17). The greatest margin (yield advantage) over fungicide input would appear to have come from either single spring sprays or treatments which had at least three spray applications.

**Table 14.** Disease development on winter oilseed rape plants and leaves in untreated control plots (mean of 25 plants) High Mowthorpe 1999.

| Date sampled | Date assessed | GS | %plants phoma leaf spot | % area phoma leaf spot | %plants light leaf spot | % area light leaf spot | %plants downy mildew | % area downy mildew | %plants phoma stem lesions | phoma stem index | %plants phoma canker | phoma canker index | %plants stem light leaf spot | %area stem light leaf spot |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | | | |
| 21.09.98 | 21.09.98 | 1.00 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 28.09.98 | 28.09.98 | 1.00 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 06.10.98 | 06.10.98 | 1.01 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 12.10.98 | 12.10.98 | 1.02 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 19.10.98 | 19.10.98 | 1.02 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 26.10.98 | 26.10.98 | 1.03 | 8 | 0.08 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 03.11.98 | 03.11.98 | 1.04 | 8 | 0.008 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 10.11.98 | 10.11.98 | 1.04 | 12 | 0.48 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 17.11.98 | 17.11.98 | 1.05 | 12 | 0.12 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 24.11.98 | 24.11.98 | 1.05 | 20 | 0.52 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 01.12.98 | 01.12.98 | 1.06 | 20 | 0.65 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 08.12.98 | 08.12.98 | 1.06 | 16 | 0.21 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 15.12.98 | 15.12.98 | 1.06 | 20 | 0.68 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 18.01.99 | 18.01.99 | 1.07 | 20 | 0.20 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 16.02.99 | 16.02.99 | 1.10 | 52 | 2.6 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 17.03.99 | 17.03.99 | 2.01 | 40 | 0.33 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 21.03.99 | 21.03.99 | 3.01 | 0 | 0 | 95 | 1.05 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 14.07.99 | 14.07.99 | 5.08 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 52.5 | 26.3 | 95.0 | 3.4 |

**Table 15.** Effect of spray timing on phoma leaf spot and light leaf spot incidence (% plants) and % severity on leaves, High Mowthorpe 1998/1999.

| Treatment code | Treatment | Phoma leaf spot | | | | Phoma leaf spot | | Light leaf spot | |
|---|---|---|---|---|---|---|---|---|---|
| | | 13.1.99 | | 16.4.99 | | 22.4.99 | | 22.4.99 | |
| | | % plants | % severity | % plants | % severity | % plants | % severity | % plants | % severity |
| 1 | Untreated | 0 | 0.00 | 11.3 | 0.11 | 0.0 | 0.00 | 95.0 | 2.44 |
| 2 | Disease onset/late Oct | 0 | 0.00 | 0 | 0.00 | 0.0 | 0.00 | 90.0 | 1.40 |
| 3 | 4-6 weeks later than 2 | 5 | 0.05 | 0 | 0.00 | 2.5 | 0.03 | 97.5 | 1.28 |
| 4 | 4-6 weeks later than 3 | 0 | 0.00 | 2.5 | 0.03 | 5.0 | 0.05 | 95.0 | 1.55 |
| 5 | 4-6 weeks later than 4 | 5 | 0.05 | 2.5 | 0.03 | 2.5 | 0.03 | 97.5 | 1.58 |
| 6 | 2+3 | 0 | 0.00 | 2.5 | 0.03 | 2.5 | 0.03 | 90.0 | 1.00 |
| 7 | 2+4 | 2.5 | 0.03 | 2.5 | 0.03 | 2.5 | 0.03 | 87.5 | 0.88 |
| 8 | 2+5 | 2.5 | 0.03 | 0 | 0.00 | 0.0 | 0.00 | 70.0 | 0.70 |
| 9 | 3+4 | 2.5 | 0.03 | 0 | 0.00 | 2.5 | 0.03 | 92.5 | 0.93 |
| 10 | 3+5 | 0 | 0.00 | 0 | 0.00 | 5.0 | 0.05 | 97.5 | 1.38 |
| 11 | 4+5 | 5 | 0.05 | 2.5 | 0.03 | 7.5 | 0.08 | 97.5 | 1.28 |
| 12 | 2+3+4 | 0 | 0.00 | 2.5 | 0.03 | 2.5 | 0.03 | 90.0 | 0.90 |
| 13 | 2+3+5 | 0 | 0.00 | 5 | 0.05 | 5.0 | 0.05 | 77.5 | 0.88 |
| 14 | 3+4+5 | 2.5 | 0.03 | 2.5 | 0.03 | 2.5 | 0.03 | 82.5 | 1.15 |
| 15 | 2+4+5 | 0 | 0.00 | 5 | 0.05 | 7.5 | 0.08 | 87.5 | 1.28 |
| 16 | 2+3+4+5 | 0 | 0.00 | 2.5 | 0.03 | 0.0 | 0.00 | 75.0 | 0.75 |
| 17 | 3+4+5 | 0 | 0.00 | 0 | 0.00 | 2.5 | 0.03 | 92.5 | 0.93 |
| 18 | 3+4+5 | 2.5 | 0.03 | 0 | 0.00 | 2.5 | 0.03 | 87.5 | 0.88 |
| 19 | 2+3+4, then 2 weeks after 5 | 0 | 0.00 | 2.5 | 0.03 | 2.5 | 0.03 | 77.5 | 0.78 |
| 20 | 2+3+4 | 0 | 0.00 | 5 | 0.05 | 0.0 | 0.00 | 60.0 | 0.60 |
| Mean | | 1.31 | 0.01 | 2.86 | 0.03 | 2.86 | 0.03 | 87.4 | 1.19 |
| SED (61df) min rep | | 2.66 | 0.03 | 3.43 | 0.03 | 3.21 | 0.03 | 10.91 | 0.419 |
| Untr v. treated | | 2.30 | 0.02 | 2.97 | | 2.77 | 0.03 | 9.45 | 0.363 |
| F test | | ns | ns | 5% | 5% | ns | ns | ns | <0.1% |

**Table 16.** Effect of spray timing on light leaf spot, phoma stem lesions, sclerotinia and alternaria incidence and severity pre-harvest. High Mowthorpe 1999

| Treatment code | Treatment | Assessment date | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | 15.7.99 | | | | | | | |
| | | Light leaf spot | | Stem phoma | | Sclerotinia | | Alternaria | |
| | | stem % inc | stem % sev | Index (0-100) | % incidence | Index (0-100) | % incidence | % stem severity | % stem incidence |
| 1 | Untreated | 98.7 | 3.2 | 8.44 | 23.8 | 9.7 | 10.0 | 0.11 | 6.3 |
| 2 | Disease onset/late Oct | 100.0 | 3.7 | 7.5 | 20.0 | 20.0 | 20.0 | 0.43 | 12.5 |
| 3 | 4-6 weeks later than 2 | 92.5 | 4.9 | 13.1 | 35.0 | 5.0 | 5.0 | 0.70 | 20.0 |
| 4 | 4-6 weeks later than 3 | 87.5 | 1.6 | 6.3 | 20.0 | 11.9 | 12.5 | 0.30 | 10.0 |
| 5 | 4-6 weeks later than 4 | 90.0 | 2.6 | 12.5 | 32.5 | 15.0 | 15.0 | 0.00 | 0.0 |
| 6 | 2+3 | 97.5 | 2.6 | 11.3 | 30.0 | 12.5 | 12.5 | 1.03 | 30.0 |
| 7 | 2+4 | 97.5 | 2.3 | 6.3 | 17.5 | 12.5 | 12.5 | 0.18 | 7.5 |
| 8 | 2+5 | 97.5 | 3.3 | 8.1 | 25.0 | 14.4 | 15.0 | 0.20 | 10.0 |
| 9 | 3+4 | 77.5 | 1.0 | 7.5 | 17.5 | 6.9 | 7.5 | 0.25 | 15.0 |
| 10 | 3+5 | 85.0 | 2.2 | 7.5 | 20.0 | 16.9 | 17.5 | 1.23 | 27.5 |
| 11 | 4+5 | 87.5 | 2.0 | 8.8 | 25.0 | 17.5 | 17.5 | 0.85 | 22.5 |
| 12 | 2+3+4 | 65.0 | 1.0 | 6.3 | 17.5 | 10.0 | 10.0 | 0.50 | 10.0 |
| 13 | 2+3+5 | 100.0 | 4.6 | 8.8 | 27.5 | 12.5 | 12.5 | 1.33 | 40.0 |
| 14 | 3+4+5 | 80.0 | 1.3 | 4.4 | 12.5 | 20.0 | 20.0 | 0.10 | 10.0 |
| 15 | 2+4+5 | 87.5 | 1.5 | 5.6 | 20.0 | 7.5 | 7.5 | 0.28 | 7.5 |
| 16 | 2+3+4+5 | 92.5 | 2.0 | 9.4 | 25.0 | 21.9 | 22.5 | 0.55 | 35.0 |
| 17 | 3+4+5 | 87.5 | 2.3 | 8.8 | 25.0 | 20.0 | 20.0 | 0.28 | 7.5 |
| 18 | 3+4+5 | 85.0 | 1.1 | 11.9 | 30.0 | 11.3 | 12.5 | 0.63 | 22.5 |
| 19 | 2+3+4, then 2 weeks after 5 | 65.0 | 1.0 | 6.3 | 22.5 | 11.9 | 12.5 | 0.03 | 2.5 |
| 20 | 2+3+4 | 70.0 | 1.5 | 3.1 | 7.5 | 2.5 | 2.5 | 0.30 | 20.0 |
| Mean | | 87.7 | 2.32 | 8.10 | 22.7 | 12.8 | 13.1 | 0.45 | 15.4 |
| SED (61df) min.rep | | 9.60 | 0.93 | 4.91 | 11.32 | 7.97 | 8.04 | 0.48 | 13.35 |
| Untr v. treated | | 8.31 | 0.80 | 4.25 | 9.81 | 6.94 | 6.96 | 0.42 | 11.56 |
| F test | | 0.1% | <0.1% | ns | ns | ns | ns | ns | ns |

**Table 17**. Effect of spray timing on yield, High Mowthorpe 1999.

| Treatment code | Treatment | Yield at 90% dry matter (t/ha) |
|---|---|---|
| 1 | Untreated | 2.71 |
| 2 | Disease onset/late Oct | 2.78 |
| 3 | 4-6 weeks later than 2 (early Dec) | 2.77 |
| 4 | 4-6 weeks later than 3 (Jan) | 3.12 |
| 5 | 4-6 weeks later than 4 (early March) | 3.15 |
| 6 | 2+3 | 3.02 |
| 7 | 2+4 | 3.00 |
| 8 | 2+5 | 3.10 |
| 9 | 3+4 | 3.22 |
| 10 | 3+5 | 2.80 |
| 11 | 4+5 | 2.97 |
| 12 | 2+3+4 | 3.17 |
| 13 | 2+3+5 | 2.96 |
| 14 | 3+4+5 | 3.35 |
| 15 | 2+4+5 | 3.56 |
| 16 | 2+3+4+5 | 3.19 |
| 17 | 3+4+5 | 3.45 |
| 18 | 3+4+5 | 3.47 |
| 19 | 2+3+4, then 2 weeks after 5 | 3.34 |
| 20 | 2+3+4 | 3.36 |
| Grand Mean | | 3.10 |
| Mean of 1 spray | | 2.96 |
| Mean of 2 sprays | | 3.00 |
| Mean of 3 sprays | excluding 17,18 & 20 | 3.26 |
| Mean of 4 sprays | | 3.27 |
| SED (61df) min rep<br>Untr v. treated | | 0.337<br>0.292 |
| CV (%) | | 15.3 |
| F test | | ns |

**Table 18**. Plant populations at harvest, High Mowthorpe 1999.

| Treatment code | Treatment | Plants/$m^2$ |
|---|---|---|
| 1 | Untreated | 42.4 |
| 2 | Disease onset/late Oct | 44.6 |
| 3 | 4-6 weeks later than 2 (early Dec) | 40.8 |
| 4 | 4-6 weeks later than 3 (Jan) | 44.0 |
| 5 | 4-6 weeks later than 4 (early March) | 43.6 |
| 6 | 2+3 | 42.8 |
| 7 | 2+4 | 45.4 |
| 8 | 2+5 | 49.0 |
| 9 | 3+4 | 46.0 |
| 10 | 3+5 | 36.0 |
| 11 | 4+5 | 42.8 |
| 12 | 2+3+4 | 41.8 |
| 13 | 2+3+5 | 49.0 |
| 14 | 3+4+5 | 43.4 |
| 15 | 2+4+5 | 48.4 |
| 16 | 2+3+4+5 | 47.4 |
| 17 | 3+4+5 | 48.6 |
| 18 | 3+4+5 | 44.8 |
| 19 | 2+3+4, then 2 weeks after 5 | 45.2 |
| 20 | 2+3+4 | 50.6 |
| Grand mean | | 44.7 |
| SED (61df) min. rep<br>Untr v. treated | | 3.73<br>3.23 |
| F test. | | ns |

**Rothamsted 1998/1999**

Disease development on untreated plots

Downy mildew affected the cotyledons and first leaves but had disappeared by late October. Traces of this disease were found again on leaves in March (Table 19).

Phoma leaf spot was first seen in late October (before inoculum was applied to plots), and by mid March, 88% of plants were affected (0.8% leaf area). Most affected leaves were at the base of the plants, and by 31 March many of these had fallen off, so that only 15% plants were affected at this time. The first stem canker was seen on 31 March (single plant on a treated plot). The incidence of stem canker increased to 65% of plants affected with basal stem canker and 62% with stem lesions by 30 June. However, disease severity remained low, with the average severity index for basal canker and stem lesions on untreated plots remaining below 1.0 (Table 20).

Light leaf spot was first seen in late January (8 weeks after inoculation) and by late March, 98% of plants (10.4% leaf area) were affected (Table 19). The disease also affected stems and pods, and by 30 June 16% of pod area was affected (Table 20).

Other diseases (sclerotinia on stems and alternaria and botrytis on leaves and pods) occurred at very low levels.

**Table 19.** Disease development on winter oilseed rape plants and leaves in untreated control plots at Rothamsted 1998/1999.

| Date | Growth stage | Phoma leaf spot | | | Light leaf spot | | | Downy mildew | |
|---|---|---|---|---|---|---|---|---|---|
| | | % plants | % leaves | % leaf area | % plants | % leaves | % leaf area | % plants | % leaf area |
| 15.09.98 | 1.1 | 0 | | | 0 | | | 0 | |
| 23.09.98 | 1.2 | 0 | | | 0 | | | 48 | 5 |
| 29.09.98 | 1.4 | 0 | | | 0 | | | 64 | 5.4 |
| 06.10.98 | 1.6 | 0 | | | 0 | | | 76 | 3.8 |
| 13.10.98 | 1.6 | 0 | | | 0 | | | 80 | 1.4 |
| 20.10.98 | 1.7 | 12 | 1.7 | 0.2 | 0 | | | 20 | 0.2 |
| 27.10.98 | 1.8 | 4 | 0.5 | 0.04 | 0 | | | | |
| 03.11.98 | 1.8 | 8 | 1.0 | 0.024 | 0 | | | | |
| 10.11.98 | 1.8 | 40 | 5.7 | 0.064 | 0 | | | | |
| 17.11.98 | 1.8 | 48 | 11.2 | 0.164 | 0 | | | | |
| 23.11.98 | 1.8 | 68 | 14.8 | 0.58 | 0 | | | | |
| 03.12.98 | 1.9 | 72 | 16.8 | 0.4 | 0 | | | | |
| 08.12.98 | 1.10 | 88 | 27.2 | 1.0 | 0 | | | | |
| 15.12.98 | 1.10 | 56 | 16.0 | 0.8 | 0 | | | | |
| 19.01.99 | 1.10 | 60 | 12.7 | 0.74 | 12 | 1.6 | 0.16 | | |
| 15.02.99 | 1.12 | 84 | 19.0 | 0.7 | 24 | 3.7 | 1.1 | | |
| 18.03.99 | 3.1 | 88 | 16.3 | 0.8 | 88 | 35.5 | 6.4 | | |
| 31.03.99* | 3.6 | 15 | 1.3 | 0.2 | 98 | 49.8 | 10.4 | 15 | 0.03 |

* mean of 60 plants; mean of 25 plants for all other assessments

**Table 20.** Disease development on winter oilseed rape stems and pods in untreated control plots (mean of 60 plants) at Rothamsted 1999.

| Date | Canker | | Stem lesions | | Light leaf spot | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | stems | | Pods (main raceme) | |
| | % plants | Index | % plants | Index | % plants | % area | % plants | % area |
| 31.03.99 | 0 | 0 | 0 | 0 | 41.7 | 1.4 | - | - |
| 26.05.99 | 25.0 | 0.27 | 33.3 | 0.35 | 90.0 | 5.6 | 2.5 | 0.23 |
| 30.06.99 | 65.0 | 0.78 | 61.7 | 0.68 | 100 | 7.6 | 38.8 | 16.0 |

**Table 21.** Effect of spray timing on phoma leaf spot incidence (% plants and % leaves affected) and severity (% leaf area affected) Rothamsted 1998/1999.

| Treatment code | Treatment | Assessment dates | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1.12.98 | | | 20.1.98 | | | 18.2.99 | | | 31.3.99 | | |
| | | % plants | % leaves | % leaf area | % plants | % leaves | %leaf area | % plants | % leaves | % leaf area | % plants | % leaves | % leaf area |
| 1 | Untreated | 60.3 | 13.9 | 0.4 | 83.3 | 18.5 | 0.9 | 93.3 | 30.5 | 2.4 | 15.0 | 1.3 | 0.23 |
| 2 (6/11) | Disease onset/late Oct | 47.7 | 10.2 | 0.2 | 72.5 | 15.7 | 0.8 | 91.7 | 23.6 | 1.5 | 10.0 | 0.7 | 0.10 |
| 3 (16/12) | 4-6 weeks later than 2 | | | | 58.3 | 11.5 | 0.6 | 83.3 | 21.5 | 1.4 | 16.7 | 1.3 | 0.13 |
| 4 (21/1) | 4-6 weeks later than 3 | | | | | | | 86.7 | 24.4 | 1.7 | 20.0 | 1.6 | 0.14 |
| 5 (25/2) | 4-6 weeks later than 4 | | | | | | | | | | 13.3 | 1.4 | 0.10 |
| 6 | 2+3 | | | | 66.7 | 13.2 | 0.7 | 88.3 | 23.0 | 1.6 | 23.3 | 2.2 | 0.14 |
| 7 | 2+4 | | | | | | | 88.3 | 22.7 | 1.9 | 10.0 | 1.2 | 0.02 |
| 8 | 2+5 | | | | | | | | | | 3.3 | 0.3 | 0.03 |
| 9 | 3+4 | | | | | | | 91.7 | 23.6 | 1.7 | 3.3 | 0.3 | 0.03 |
| 10 | 3+5 | | | | | | | | | | 3.3 | 0.3 | 0.003 |
| 11 | 4+5 | | | | | | | | | | 10.0 | 0.8 | 0.07 |
| 12 | 2+3+4 | | | | | | | 85.8 | 23.6 | 1.7 | 13.3 | 1.0 | 0.09 |
| 13 | 2+3+5 | | | | | | | | | | 10.0 | 0.9 | 0.01 |
| 14 | 3+4+5 | | | | | | | | | | 3.3 | 0.3 | 0.003 |
| 15 | 2+4+5 | | | | | | | | | | 16.7 | 1.1 | 0.04 |
| 16 | 2+3+4+5 | | | | | | | | | | 13.3 | 1.0 | 0.10 |
| 17 | 2 weeks after 2, then 3+4+5 | | | | | | | | | | 3.3 | 0.3 | 0.003 |
| 18 | 4 weeks after 2, then 3+4+5 | | | | | | | | | | 10.0 | 0.7 | 0.07 |
| 19 | 2+3+4, then 2 weeks after 5 | | | | | | | | | | 10.0 | 0.8 | 0.04 |
| 20 | 2+3+4, then 4 weeks after 5 | | | | | | | | | | 16.7 | 1.6 | 0.08 |
| Mean | | 54.3 | 12.1 | 0.3 | 70.6 | 14.5 | 0.8 | 87.8 | 24.1 | 1.8 | 11.4 | 0.98 | 0.08 |
| SED | | 10.8 | 3.1 | 0.12 | 10.1 | 3.1 | 0.27 | 7.3 | 4.5 | 0.5 | 8.7 | 0.79 | 0.10 |
| Df | | 58 | 58 | 58 | 55 | 55 | 55 | 51 | 51 | 51 | 41 | 41 | 41 |
| F test | | <5% | <5% | <5% | <1% | <1% | ns | ns | ns | ns | ns | ns | ns |

**Table 22.** Effect of spray timing on stem canker and stem lesion incidence (I) (% plants affected) and severity (S) (0 – 4 score) Rothamsted 1999.

| Treatment code | Treatment | Assessment date 26.5.99 | | | | Assessment date 30.6.99 | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Canker | | Stem lesions | | Canker | | Stem lesions | |
| | | I | S | I | S | I | S | I | S |
| 1 | Untreated | 25.0 | 0.27 | 33.3 | 0.35 | 65.0 | 0.78 | 61.7 | 0.68 |
| 2 (6/11) | Disease onset/late Oct | 20.0 | 0.20 | 20.0 | 0.20 | 40.0 | 0.40 | 33.3 | 0.33 |
| 3 (16/12) | 4-6 weeks later than 2 | 23.3 | 0.27 | 16.7 | 0.17 | 10.0 | 0.10 | 43.3 | 0.43 |
| 4 (21/1) | 4-6 weeks later than 3 | 10.0 | 0.10 | 13.3 | 0.13 | 23.3 | 0.23 | 26.7 | 0.27 |
| 5 (25/2) | 4-6 weeks later than 4 | 33.3 | 0.37 | 16.7 | 0.17 | 33.3 | 0.37 | 36.7 | 0.40 |
| 6 | 2+3 | 0 | 0 | 3.3 | 0.03 | 26.7 | 0.33 | 46.7 | 0.47 |
| 7 | 2+4 | 3.3 | 0.03 | 6.7 | 0.07 | 16.7 | 0.20 | 26.7 | 0.28 |
| 8 | 2+5 | 3.3 | 0.03 | 10.0 | 0.10 | 6.7 | 0.07 | 30.0 | 0.30 |
| 9 | 3+4 | 0 | 0 | 6.7 | 0.07 | 13.3 | 0.13 | 23.3 | 0.23 |
| 10 | 3+5 | 5.7 | 0.07 | 3.3 | 0.03 | 0 | 0 | 26.7 | 0.27 |
| 11 | 4+5 | 30.0 | 0.30 | 16.7 | 0.17 | 26.7 | 0.27 | 20.0 | 0.20 |
| 12 | 2+3+4 | 6.7 | 0.07 | 6.7 | 0.07 | 16.7 | 0.17 | 16.7 | 0.17 |
| 13 | 2+3+5 | 6.7 | 0.07 | 10.0 | 0.10 | 6.7 | 0.07 | 10.0 | 0.13 |
| 14 | 3+4+5 | 3.3 | 0.03 | 6.7 | 0.07 | 6.7 | 0.07 | 10.0 | 0.10 |
| 15 | 2+4+5 | 3.3 | 0.03 | 0 | 0 | 10.0 | 0.10 | 23.3 | 0.27 |
| 16 | 2+3+4+5 | 0 | 0 | 10.0 | 0.10 | 6.7 | 0.07 | 13.3 | 0.13 |
| 17 | 2 weeks after 2, then 3+4+5 | 3.3 | 0.03 | 6.7 | 0.07 | 10.0 | 0.10 | 6.7 | 0.10 |
| 18 | 4 weeks after 2, then 3+4+5 | 10.0 | 0.10 | 13.3 | 0.17 | 10.0 | 0.10 | 3.3 | 0.03 |
| 19 | 2+3+4, then 2 weeks after 5 | 0 | 0 | 3.3 | 0.03 | 6.7 | 0.07 | 3.3 | 0.03 |
| 20 | 2+3+4, then 4 weeks after 5 | 10.0 | 0.10 | 10.0 | 0.10 | 10.0 | 0.10 | 6.7 | 0.07 |
| Grand mean | | 10.6 | 0.11 | 11.7 | 0.12 | 19.5 | 0.21 | 25.2 | 0.26 |
| Mean for one spray | | | | | | | 0.27 | | |
| Mean for two sprays | | | | | | | 0.17 | | |
| Mean for three sprays | | | | | | | 0.10 | | |
| Mean for four sprays | | | | | | | 0.09 | | |
| SED (41 df) | | 10.36 | 0.11 | 8.77 | 0.09 | 11.75 | 0.14 | 9.16 | 0.10 |
| F test | | ns | ns | ns | ns | <0.1% | <0.1% | <0.1% | <0.1% |

**Table 23.** Effect of spray timing on light leaf spot incidence (I) (% plants affected) and severity (S) (% leaf area (L) or % pod area (P) affected) Rothamsted 1998/1999.

| Treatmnt code | Treatment | Assessment date | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1.12.98 | | 20.1.98 | | 18.2.99 | | 31.3.99 | | 26.5.99 | | 30.6.99 | |
| | | I | S (L) | I | S (L) | I | S (L) | I | S (L) | I | S (P) | I | S (P) |
| 1 | Untreated | 0 | 0 | 2.7 | 0.03 | 82.2 | 6.39 | 98.3 | 10.4 | 90.0 | 0.23 | 100 | 16.0 |
| 2 (6/11) | Disease onset/late Oct | 0 | 0 | 3.3 | 0.05 | 53.3 | 1.78 | 86.7 | 3.8 | 53.3 | 0.04 | 96.7 | 4.8 |
| 3 (16/12) | 4-6 weeks later than 2 | | | 4.2 | 0.04 | 21.7 | 0.88 | 66.7 | 2.7 | 26.7 | 0.07 | 83.3 | 9.1 |
| 4 (21/1) | 4-6 weeks later than 3 | | | | | 61.7 | 2.51 | 76.7 | 6.5 | 73.3 | 0.83 | 96.7 | 21.7 |
| 5 (25/2) | 4-6 weeks later than 4 | | | | | | | 86.7 | 2.7 | 60.0 | 0.02 | 100 | 8.1 |
| 6 | 2+3 | | | 2.2 | 0.04 | 35.0 | 1.51 | 43.3 | 0.8 | 46.7 | 0.003 | 100 | 6.2 |
| 7 | 2+4 | | | | | 41.7 | 1.03 | 96.7 | 5.6 | 53.3 | 0.02 | 93.3 | 15.1 |
| 8 | 2+5 | | | | | | | 66.7 | 1.9 | 40.0 | 0.003 | 96.7 | 5.4 |
| 9 | 3+4 | | | | | 23.3 | 0.31 | 50.0 | 2.6 | 33.3 | 0 | 76.7 | 6.0 |
| 10 | 3+5 | | | | | | | 46.7 | 0.9 | 20.0 | 0 | 80.0 | 1.6 |
| 11 | 4+5 | | | | | | | 83.3 | 2.9 | 40.0 | 0.07 | 93.3 | 7.3 |
| 12 | 2+3+4 | | | | | 20.8 | 0.28 | 40.0 | 2.8 | 36.7 | 0.03 | 83.3 | 5.3 |
| 13 | 2+3+5 | | | | | | | 60.0 | 1.2 | 23.3 | 0.01 | 86.7 | 2.7 |
| 14 | 3+4+5 | | | | | | | 20.0 | 0.2 | 26.7 | 0 | 80.0 | 3.6 |
| 15 | 2+4+5 | | | | | | | 33.3 | 1.1 | 6.7 | 0 | 93.3 | 6.5 |
| 16 | 2+3+4+5 | | | | | | | 53.3 | 0.9 | 10.0 | 0.05 | 73.3 | 4.4 |
| 17 | 2 weeks after 2, then 3+4+5 | | | | | | | 33.3 | 0.4 | 23.3 | 0.02 | 70.0 | 4.2 |
| 18 | 4 weeks after 2, then 3+4+5 | | | | | | | 20.0 | 0.4 | 40.0 | 0.02 | 73.3 | 5.2 |
| 19 | 2+3+4, then 2 weeks after 5 | | | | | | | 50.0 | 0.9 | 30.0 | 0.04 | 76.7 | 7.2 |
| 20 | 2+3+4, then 4 weeks after 5 | | | | | | | 23.3 | 0.1 | 26.7 | 0.02 | 73.3 | 3.6 |
| Mean | | | | 2.86 | 0.036 | 40.3 | 1.75 | 58.7 | 2.83 | 40.5 | 0.04 | 87.0 | 7.6 |
| SED | | | | 3.36 | 0.059 | 13.15 | 1.35 | 10.11 | 2.24 | 19.33 | 0.07 | 11.18 | 4.36 |
| Df | | | | 55 | 55 | 51 | 51 | 41 | 41 | 41 | 41 | 41 | 41 |
| F test | | | | ns | ns | <0.1% | <0.1% | <0.1% | <0.1% | 5% | ns | ns | 5% |

**Table 24**. Effect of spray timing on seed and oil yield Rothamsted 1999.

| Treatment code | Treatment | Yield at 90% DM (t/ha) | Oil yield (t/ha) |
|---|---|---|---|
| 1 | Untreated | 4.64 | 2.09 |
| 2 (6/11) | Disease onset/late Oct | 4.97 | 2.21 |
| 3 (16/12) | 4-6 weeks later than 2 (early Dec) | 5.27 | 2.37 |
| 4 (21/1) | 4-6 weeks later than 3 (Jan) | 4.88 | 2.21 |
| 5 (25/2) | 4-6 weeks later than 4 (early March) | 4.97 | 2.23 |
| 6 | 2+3 | 4.84 | 2.17 |
| 7 | 2+4 | 5.05 | 2.26 |
| 8 | 2+5 | 5.34 | 2.38 |
| 9 | 3+4 | 5.05 | 2.27 |
| 10 | 3+5 | 4.82 | 2.17 |
| 11 | 4+5 | 5.23 | 2.33 |
| 12 | 2+3+4 | 4.96 | 2.23 |
| 13 | 2+3+5 | 5.14 | 2.29 |
| 14 | 3+4+5 | 5.23 | 2.35 |
| 15 | 2+4+5 | 5.18 | 2.33 |
| 16 | 2+3+4+5 | 5.35 | 2.39 |
| 17 | 2 weeks after 2, then 3+4+5 | 5.16 | 2.32 |
| 18 | 4 weeks after 2, then 3+4+5 | 5.04 | 2.25 |
| 19 | 2+3+4, then 2 weeks after 5 | 4.87 | 2.19 |
| 20 | 2+3+4, then 4 weeks after 5 | 5.37 | 2.39 |
| Grand mean | | 5.05 | 2.26 |
| Mean of 1 spray | | 5.02 | 2.26 |
| Mean of 2 sprays | | 5.05 | 2.26 |
| Mean of 3 sprays | | 5.13 | 2.30 |
| Mean of 4 sprays | | 5.16 | 2.31 |
| SED (41 Df) | | 0.281 | 0.115 |
| F test | | ns | ns |

Disease control

At its peak in February, all spray programmes applied to date had only reduced phoma leaf spot to a small extent, and two or three sprays were no better than one (Table 21). At the June assessment, there were significant differences between untreated and treated plots, with most treatments reducing the incidence and severity of cankers and phoma stem lesions by more than 50% (Table 22). Of the single spray timings, mid December was the most effective, although good reductions were also achieved with a January spray. All programmes comprising 2 or more sprays significantly reduced incidence and severity of canker from 65% to <10% incidence for some treatments. For phoma stem lesions, there was a stronger trend for three and four spray programmes to be more effective than programmes of one or two sprays (Table 22).

In February and March, when light leaf spot was at its peak on leaves, all 2, 3 or 4 spray programmes gave significant control of light leaf spot but, of the single sprays, only the December spray gave significant reductions (Table 23) . Single sprays were not effective in controlling the disease on pods in May and, by June, even multiple spray programmes were not very effective.

The plant population at harvest averaged 57 plants/$m^2$ on both untreated plots and those receiving four sprays. It was therefore concluded that disease had not caused plant deaths and a full assessment of all plots was not carried out.

The untreated control gave a high seed yield of 4.64 t/ha and an oil yield of 2.09 t/ha (Table 24). There were significant responses to fungicide treatment. Single sprays gave an average yield increase of 0.38t/ha. Each additional spray gave a slightly higher yield approaching 0.05 t/ha, but even four sprays only increased yield by 0.14 t/ha compared with a single spray. There were interesting trends from delaying the first spray with autumn treatments declining from 5.35 t/ha to 5.04 t/ha with the four week delay. In spring, a final spray in early Feb gave 0.48-0.50 t/ha than sprays either two weeks earlier or two weeks later.

**Boxworth 1999/2000**

Initial establishment was generally satisfactory after sowing on 26 August 1999, though plant population was low and averaged 23 plants/$m^2$ at harvest with no significant differences between treatments. The mild wet autumn enabled plants to grow strongly and they reached the 10-leaf stage by 18 October and 18-20 leaves by early January. After a mild winter, crop development was earlier than average and stem extension began in February and first flowering started in late April. The crop ripened very rapidly in late June despite adequate soil moisture.

**Table 25**. Disease development on winter oilseed rape plants and leaves in untreated control plots (mean of 25 or 60 plants*) Boxworth 1999/2000.

| Date | Leaves | | | | | Stems | | | | Pods | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| assessed | Downy mildew | Phoma leaf spot | Light leaf spot | Botrytis | Alter-naria | Phoma* | Light leaf spot | Scler-otinia | Canker * | Downy mildew | Alter-naria | Light leaf spot | Powdery mildew |
| 28.09.99 | 28 (0.05) | 4 (0.02) | | | | | | | | | | | |
| 05.10.99 | 24 (0.04) | 48 (0.09) | | | | | | | | | | | |
| 13.10.99 | 40 (0.10) | 100 (0.33) | | | | | | | | | | | |
| 18.10.99 | 18 (0.04) | 100 (0.82) | | | | | | | | | | | |
| 25.10.99[1] | 4 (0.004) | 92 (0.54) | | | | | | | | | | | |
| 01.11.99 | 52 (0.10) | 96 (0.37) | | | | | | | | | | | |
| 08.11.99 | 64 (0.14) | 84 (0.28) | 0 | 0 | 0 | | | | | | | | |
| 15.11.99 | 0 | 100 (0.51) | 0 | 0 | 0 | | | | | | | | |
| 22.11.99 | 45 (0.15) | 100 (1.85) | 0 | 0 | 0 | | | | | | | | |
| 30.11.99 | 28 (0.06) | 100 (0.56) | 0 | 0 | 0 | | | | | | | | |
| 07.12.99 | 60 (0.17) | 100 (0.72) | 0 | 0 | 0 | | | | | | | | |
| 14.12.99 | 52 (0.14) | 100 (0.39) | 0 | 0 | 0 | | | | | | | | |
| 12.01.00 | 48 (0.12) | 98.0 (0.77) | 0 | 0 | 0 | | | | | | | | |
| 19.01.00 | 80 (0.58) | 92 (0.79) | 0 | 0 | 0 | | | | | | | | |
| 15.02.00 | 80 (0.24) | 100 (0.75) | 0 | 0 | 0 | | | | | | | | |
| 08.03.00 | 71 (0.31) | 100 (0.98) | 13.8 (0.55) | 0 | 0 | | | | | | | | |
| 29.03.00 | 94 (1.00) | 96.2 (0.48) | 41.2 (4.40) | 0 | 0 | 8.8 (0.09) | 0 | 0 | 22.5 (0.23) | | | | |
| 23.05.00 | 70 (0.25) | 38.7 (0.15) | 43.7 (0.91) | 73.7 (0.94) | 0 | 40.0 (0.43) | 41.2 (0.72) | 0 | 91.2 (1.38) | | | | |
| 29.06.00 | 37 (0.07) | 22.5 (0.09) | 1.2 (0.004) | 92.5 (0.28) | 22.5 (0.03) | 95.0 (1.25) | 37.5 (1.24) | 2.5 (0.09) | 98.7 (2.84) | 68.7 (0.09) | 1.25 (0.001) | 23.8 (0.47) | 7.5 (0.01) |

[1] Powdery Mildew 4% plants

Disease development

Downy mildew was active on the first leaves in September, and fluctuated until early December before remaining at a high incidence from December until May (Table 25). The severity of downy mildew was very low in autumn and peak severity was at the start of flowering in late March.

Phoma leaf spot was first seen in late September, which was very early. It built up very rapidly in the first half of October to affect 100% of plants. This high incidence was maintained throughout the winter and up to the end of March (Table 25). Phoma leaf symptoms only declined as lower leaves were lost in late May. The severity of phoma leaf spot was quite variable (e.g. increasing from 0.5% to 1.8% leaf area affected within a week and then declining a week later), which was not unexpected given the rapid production of leaves in autumn. Late November was the period of greatest phoma leaf spot activity, but even at that time, week to week variation was large. The first phoma stem lesions were found in late March when 9% stems had lesions. There was further lesion development during flowering, but the main increase in stem lesions took place after the end of flowering. In late March, basal stem canker lesions (23% plants affected) were slightly more common than phoma stem lesions (9% plants). The incidence of basal canker on untreated plots increased to 91% of plants affected at the end of flowering and to 99% plants by 29 June. Canker severity was much higher than in 1999, with most lesions girdling the stem (index 2.84).

Light leaf spot was only seen from early March onwards and it increased sharply from 14% plants affected to 41% plants with leaf symptoms during March. Rather similar levels of light leaf spot were recorded on stems where severity remained low (1.2% area affected). There were low levels of sclerotinia at crop maturity and low levels of light leaf spot, powdery mildew and downy mildew on pods. Petals stuck to leaves in the unsettled weather during flowering and this led to a high incidence of botrytis on leaves, though little stem infection (Table 25).

Disease control

The assessment of phoma leaf spot on 22 November showed that the two delayed sprays (22 October and 3 November) had given good reductions in the severity of phoma leaf spot, but the 6 October was no longer effective (Table 26). The second main application on 15 November was still effective on 11 January as a single spray and in programmes. In early March, there were reductions in the incidence and severity of phoma leaf spot, particularly where January and late February sprays were combined. Three and four spray regimes consistently gave the lowest disease severity. In late March, only the February single spray gave control of phoma leaf spot, but multiple spray treatments showed significantly lower disease incidence.

Phoma leaf spot had declined by the end of flowering and in late May, there were no treatment differences in phoma leaf spot incidence. Phoma severity tended to be higher in fungicide treated plots than in the untreated control plots (Table 26).

At the end of March, there were encouraging signs that there were delays in the appearance of cankers and stem lesion in many treatments (Table 27). Differences between untreated and treated plots were significant by the end of May, when canker incidence was reduced from 91% to as little as 23% with treatments 17 and 18. The same treatments also had the lowest incidence of stem lesions. All programmes comprising 2 or more sprays significantly reduced canker severity pre-harvest from 2.84 to 2.00 or less. Of the single sprays, only the November treatment achieved this level of control (Table 27). Only one three-spray programme and two four-spray programmes reduced the canker index to <1.0. Good control (>50% control) of stem lesions was only demonstrated with four-spray programmes (treatments 17, 18 and 20) in late June.

There was light leaf spot in this experiment from early March onwards and it was consistently more prevalent in the untreated control than in any treatment. All the programmes with two or more sprays gave good reductions in disease incidence on leaves in late March, as did all the single sprays (Table 28). The advantage of programmes over single sprays was pronounced in later assessments on leaves stems and pods. Pre-harvest, the single December spray appeared to be the most effective for control of stem infection.

This was a moderately yielding site which averaged 3.46 t/ha. There were highly significant effects on yield and a yield increase of 0.20 t/ha over the untreated control was a significant treatment difference at the 5% level. All treatments therefore gave yield responses (Table 29). Differences of 0.23 t/ha between treatments were also significant, indicating that some programmes of sprays were better than single spray treatments (e.g. treatments 12,13,16 and 17 gave yields of 3.63-3.76 t/ha). Mean yields for one, two, three or four spray programmes suggest that second and third sprays contributed about 0.1 t/ha and there was no benefit from a fourth spray. Delaying the first or last fungicide had little effect on yield and averaged about 0.05 t/ha loss per two week delay.

**Table 26.** Effect of spray timing on phoma leaf spot incidence (% plants affected) and severity (% leaf area affected) Boxworth 2000.

| Treatment code | Treatment | Assessment dates for phoma leaf spot | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 22.11.99 | | 11.1.00 | | 08.3.00 | | 29.03.00 | | 22.05.00 | |
| | | % plants | % leaf area | % plants | % leaf area | % plants | % leaf area* | % plants | % leaf area | % plants | % leaf area* |
| 1 | Untreated | 100.0 | 1.85 | 98.0 | 0.77 | 100.0 | 0.98 (-2.30) | 96.2 | 0.48 | 38.7 | 0.15 (-3.18) |
| 2 (6/10) | Disease onset/late Oct | 100.0 | 1.17 | 98.1 | 0.75 | 100.0 | 1.56 (-2.09) | 100.0 | 0.55 | 47.5 | 0.46 (-2.88) |
| 3 (15/11) | 4-6 weeks later than 2 | 100.0 | 1.77 | 83.7 | 0.22 | 97.5 | 0.97 (-2.32) | 97.5 | 0.53 | 55.0 | 1.02 (-2.83) |
| 4 (10/01) | 4-6 weeks later than 3 | | | | | 95.0 | 0.39 (-2.76) | 87.5 | 0.37 | 40.0 | 0.16 (-3.13) |
| 5 (22/02) | 4-6 weeks later than 4 | | | | | 95.0 | 0.96 (-2.33) | 65.0 | 0.14 | 55.0 | 0.53 (-2.75) |
| 6 | 2+3 | 100.0 | 1.15 | 66.7 | 0.21 | 82.5 | 0.83 (-2.42) | 90.0 | 0.37 | 40.0 | 0.43 (-2.91) |
| 7 | 2+4 | | | | | 97.5 | 0.21 (-3.02) | 90.0 | 0.29 | 32.5 | 0.09 (-3.36) |
| 8 | 2+5 | | | | | 80.0 | 0.67 (-2.47) | 62.5 | 0.05 | 57.5 | 1.43 (-2.50) |
| 9 | 3+4 | | | | | 100.0 | 0.19 (-3.07) | 82.5 | 0.24 | 52.5 | 1.11 (-2.41) |
| 10 | 3+5 | | | | | 80.0 | 0.50 (-2.61) | 40.0 | 0.07 | 35.0 | 0.13 (-3.25) |
| 11 | 4+5 | | | | | 35.0 | 0.31 (-2.90) | 45.0 | 0.06 | 47.5 | 0.20 (-3.06) |
| 12 | 2+3+4 | | | | | 92.5 | 0.09 (-3.34) | 72.5 | 0.15 | 52.5 | 0.56 (-2.83) |
| 13 | 2+3+5 | | | | | 42.5 | 0.50 (-2.62) | 37.5 | 0.06 | 42.5 | 0.12 (-3.27) |
| 14 | 3+4+5 | | | | | 55.0 | 0.06 (-3.40) | 27.5 | 0.04 | 50.0 | 0.12 (-3.19) |
| 15 | 2+4+5 | | | | | 52.5 | 0.12 (-3.25) | 35.0 | 0.06 | 40.0 | 0.15 (-3.12) |
| 16 | 2+3+4+5 | | | | | 40.0 | 0.08 (-3.35) | 20.0 | 0.02 | 47.5 | 0.26 (-2.97) |
| 17 | 2 weeks after 2, then 3+4+5 | 70.0 | 0.10 | 80.0 | 0.16 | 37.5 | 0.07 (-3.38) | 17.5 | 0.04 | 52.5 | 0.15 (-3.14) |
| 18 | 4 weeks after 2, then 3+4+5 | 90.0 | 0.27 | 27.5 | 0.04 | 30.0 | 0.07 (-3.37) | 17.5 | 0.02 | 40.0 | 0.08 (-3.33) |
| 19 | 2+3+4, then 2 weeks after 5 | | | | | 57.5 | 0.13 (-3.23) | 47.5 | 0.08 | 15.0 | 0.06 (-3.42) |
| 20 | 2+3+4, then 4 weeks after 5 | | | | | | | 75.0 | 0.22 | 25.0 | 0.06 (-3.41) |
| Mean | | 98.1 | 1.35 | 82.1 | 0.44 | 75.8 | 0.47 (-2.85) | 62.0 | 0.21 | 43.1 | 0.35 (-3.05) |
| SED (min rep) | | 1.60 | 0.452 | 9.92 | 0.174 | 11.43 | 0.151 | 10.18 | 0.062 | 14.59 | 0.476 (0.282) |
| Df | | 78 | 78 | 78 | 78 | 65 | 65 | 61 | 61 | 61 | 61 |
| F test | | skew | <0.1% | skew <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | NS | 5% |

Logit transformed data analysed

**Table 27.** Effect of spray timing on canker and phoma stem lesion incidence (I) (% plants affected) and severity (S) (0 – 4 score) Boxworth 2000.

| Treatment code | Treatment | 29.03.00 | | 22.05.00 | | | | 29.6.00 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Canker | | Stem lesions | | Canker | | Stem lesions | |
| | | Canker 1 | Stem lesion I | I | S | I* | S* | I | S | I | S |
| 1 | Untreated | 22.5 | 8.8 | 91.2 | 1.38 | 40.0 | 0.43 | 98.7 | 2.84 | 95.0 | 1.25 |
| 2 (6/10) | Disease onset/late Oct | 10.0 | 15.0 | 72.5 | 0.93 | 30.0 | 0.33 | 100.0 | 2.95 | 90.0 | 1.35 |
| 3 (15/11) | 4-6 weeks later than 2 | 17.5 | 5.0 | 67.5 | 0.80 | 20.0 | 0.20 | 97.5 | 1.83 | 92.5 | 0.80 |
| 4 (10/01) | 4-6 weeks later than 3 | 25.0 | 5.0 | 92.5 | 1.30 | 30.0 | 0.35 | 97.5 | 2.15 | 92.5 | 1.08 |
| 5 (22/02) | 4-6 weeks later than 4 | 10.0 | 5.0 | 90.0 | 1.38 | 40.0 | 0.45 | 95.0 | 2.03 | 90.0 | 1.13 |
| 6 | 2+3 | 5.0 | 7.5 | 75.0 | 0.90 | 17.5 | 0.25 | 92.5 | 1.88 | 92.5 | 1.30 |
| 7 | 2+4 | 12.5 | 7.5 | 70.0 | 0.95 | 25.0 | 0.25 | 92.5 | 2.00 | 85.0 | 0.93 |
| 8 | 2+5 | 30.0 | 2.5 | 82.5 | 1.30 | 30.0 | 0.30 | 92.5 | 1.85 | 72.5 | 0.85 |
| 9 | 3+4 | 12.5 | 5.0 | 50.0 | 0.63 | 20.0 | 0.23 | 90.0 | 1.58 | 92.5 | 1.13 |
| 10 | 3+5 | 0.0 | 2.5 | 60.0 | 0.78 | 25.0 | 0.25 | 87.5 | 1.45 | 65.0 | 0.78 |
| 11 | 4+5 | 12.5 | 0.0 | 67.5 | 0.78 | 22.5 | 0.23 | 90.0 | 1.88 | 70.0 | 0.80 |
| 12 | 2+3+4 | 10.0 | 7.5 | 55.0 | 0.73 | 15.0 | 0.20 | 60.0 | 1.08 | 80.0 | 0.85 |
| 13 | 2+3+5 | 17.5 | 0.0 | 65.0 | 0.80 | 32.5 | 0.33 | 95.0 | 1.68 | 80.0 | 0.90 |
| 14 | 3+4+5 | 15.0 | 7.5 | 45.0 | 0.53 | 22.5 | 0.23 | 55.0 | 0.90 | 52.5 | 0.63 |
| 15 | 2+4+5 | 7.5 | 2.5 | 67.5 | 0.80 | 22.5 | 0.23 | 87.5 | 1.65 | 70.0 | 0.83 |
| 16 | 2+3+4+5 | 10.0 | 2.5 | 67.5 | 0.75 | 17.5 | 0.18 | 72.5 | 1.43 | 67.5 | 0.75 |
| 17 | 2 weeks after 2, then 3+4+5 | 10.0 | 0.0 | 22.5 | 0.28 | 7.5 | 0.08 | 45.0 | 0.75 | 42.5 | 0.50 |
| 18 | 4 weeks after 2, then 3+4+5 | 10.0 | 0.0 | 22.5 | 0.23 | 10.0 | 0.10 | 52.5 | 1.03 | 52.5 | 0.55 |
| 19 | 2+3+4, then 2 weeks after 5 | 2.5 | 2.5 | 25.0 | 0.25 | 25.0 | 0.25 | 67.5 | 1.30 | 55.0 | 0.63 |
| 20 | 2+3+4, then 4 weeks after 5 | 12.5 | 2.5 | 27.5 | 0.30 | 17.5 | 0.18 | 47.5 | 0.93 | 50.0 | 0.50 |
| Grand mean | | 13.1 | 4.6 | 62.3 | 0.82 | 24.3 | 0.26 | 81.7 | 1.71 | 75.4 | 0.89 |
| SED (61df) | | n/d | n/d | 15.29 | 0.241 | 13.35 | 0.141 | 10.20 | 0.301 | 10.75 | 0.185 |
| F test | | | | <0.1% | <0.1% | ns | ns | (8.84 unt v. treated) <0.1% | (0.261 unt v. treated) <0.1% | (9.31 unt v. treated) <0.1% | (0.161 unt v. treated) <0.1% |

* Logit transformed data analysed

**Table 28.** Effect of spray timing on light leaf spot incidence (I) (% plants affected) and severity (S) (% leaf area or stem area affected) Boxworth 2000.

| Treatmnt code | Treatment | Light leaf spot | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | 8.03.00 | 29.03.00 | 22.05.00 | | 22.05.00 | | 29.06.00 | |
| | | I leaf | I leaf | I leaf | S % leaf area affected | % plants with stem infection | % stem area affected | I (stem) | I (pod) |
| 1 | Untreated | 13.8 | 41.2 | 43.7 | 0.91 | 41.2 | 0.72 | 37.5 | 23.8 |
| 2 (6/10) | Disease onset/late Oct | 7.5 | 10.0 | 27.5 | 0.28 | 10.0 | 0.18 | 37.5 | 17.5 |
| 3 (15/11) | 4-6 weeks later than 2 | 0.0 | 5.0 | 37.5 | 0.81 | 22.5 | 0.57 | 15.0 | 5.0 |
| 4 (10/01) | 4-6 weeks later than 3 | 2.5 | 2.5 | 25.0 | 0.37 | 5.0 | 0.01 | 37.5 | 12.5 |
| 5 (22/02) | 4-6 weeks later than 4 | 0.0 | 10.0 | 27.5 | 0.44 | 35.0 | 0.20 | 15.0 | 2.5 |
| 6 | 2+3 | 2.5 | 0.0 | 0.0 | 0.00 | 0.0 | 0.00 | 5.0 | 2.5 |
| 7 | 2+4 | 0.0 | 2.5 | 10.0 | 0.05 | 2.5 | 0.01 | 10.0 | 7.5 |
| 8 | 2+5 | 0.0 | 5.0 | 12.5 | 0.92 | 10.0 | 0.23 | 0.0 | 5.0 |
| 9 | 3+4 | 2.5 | 0.0 | 12.5 | 0.08 | 2.5 | 0.03 | 10.0 | 0.0 |
| 10 | 3+5 | 2.5 | 0.0 | 2.5 | 0.01 | 2.5 | 0.01 | 7.5 | 5.0 |
| 11 | 4+5 | 0.0 | 0.0 | 5.0 | 0.08 | 2.5 | 0.05 | 10.0 | 12.5 |
| 12 | 2+3+4 | 0.0 | 0.0 | 0.0 | 0.00 | 0.0 | 0.00 | 0.0 | 0.0 |
| 13 | 2+3+5 | 0.0 | 0.0 | 32.5 | 0.37 | 0.0 | 0.00 | 5.0 | 5.0 |
| 14 | 3+4+5 | 0.0 | 0.0 | 10.0 | 0.10 | 5.0 | 0.04 | 0.0 | 0.0 |
| 15 | 2+4+5 | 0.0 | 0.0 | 7.5 | 0.04 | 0.0 | 0.00 | 0.0 | 0.0 |
| 16 | 2+3+4+5 | 0.0 | 0.0 | 5.0 | 0.04 | 0.0 | 0.00 | 2.5 | 0.0 |
| 17 | 2 weeks after 2, then 3+4+5 | 0.0 | 0.0 | 0.0 | 0.00 | 0.0 | 0.00 | 0.0 | 0.0 |
| 18 | 4 weeks after 2, then 3+4+5 | 0.0 | 0.0 | 7.5 | 0.09 | 0.0 | 0.00 | 2.5 | 2.5 |
| 19 | 2+3+4, then 2 weeks after 5 | 0.0 | 2.5 | 10.0 | 0.11 | 0.0 | 0.00 | 0.0 | 7.5 |
| 20 | 2+3+4, then 4 weeks after 5 | | 0.0 | 2.5 | 0.03 | 0.0 | 0.00 | 0.0 | 2.5 |
| Mean | | 2.1 | 5.7 | 15.4 | 0.27 | 8.6 | 0.13 | 11.1 | 6.4 |
| SED | min rep | n/d | n/d | 15.41 | 0.42 | 10.76 | 0.203 | 10.83 (9.38 unt v. treated) | 7.63 (6.60 unt v. treated) |
| Df | | 65 | 65 | 61 | 61 | 61 | 61 | 61 | 61 |
| F test | | Skew | Skew | skew | skew | Skew | Skew | <0.1% | 2% |

**Table 29.** Effect of spray timing on yield, Boxworth 2000.

| Treatment code | Treatment | Yield at 90% dry matter (t/ha) |
|---|---|---|
| 1 | Untreated | 3.07 |
| 2 (6/10) | Disease onset/late Oct | 3.28 |
| 3 (15/11) | 4-6 weeks later than 2 (early Dec) | 3.34 |
| 4 (10/01) | 4-6 weeks later than 3 (Jan) | 3.37 |
| 5 (22/02) | 4-6 weeks later than 4 (early March) | 3.40 |
| 6 | 2+3 | 3.47 |
| 7 | 2+4 | 3.52 |
| 8 | 2+5 | 3.40 |
| 9 | 3+4 | 3.48 |
| 10 | 3+5 | 3.55 |
| 11 | 4+5 | 3.29 |
| 12 | 2+3+4 | 3.65 |
| 13 | 2+3+5 | 3.76 |
| 14 | 3+4+5 | 3.56 |
| 15 | 2+4+5 | 3.48 |
| 16 | 2+3+4+5 | 3.64 |
| 17 | 2 weeks after 2, then 3+4+5 | 3.63 |
| 18 | 4 weeks after 2, then 3+4+5 | 3.57 |
| 19 | 2+3+4, then 2 weeks after 5 | 3.59 |
| 20 | 2+3+4, then 4 weeks after 5 | 3.54 |
| Grand mean | | 3.46 |
| Mean of 1 spray | | 3.35 |
| Mean of 2 sprays | | 3.45 |
| Mean of 3 sprays | | 3.61 |
| Mean of 4 sprays | | 3.59 |
| SED (61Df)<br>F test | | 0.112<br><0.1% |

**High Mowthorpe 1999/2000**

The crop established well after drilling on 31 August 1999 and developed slowly through the autumn to reach the six-leaf stage by late October. Some oilseed rape volunteers were evident across the site. Phoma leaf spot was present at low levels in early autumn and increased sharply after mid-November to affect 60% plants (Table 30). This level of infection was maintained until January and then declined to 20% plants affected in early March.

**Table 30.** Disease development on winter oilseed rape in untreated control plots (mean of 25 plants).High Mowthorpe 1999/2000.

| Date | Growth stage | % phoma leaf spot | | % light leaf spot | |
|---|---|---|---|---|---|
| | | Severity | Incidence | Severity | Incidence |
| 28.09.99 | 1,3 | 0 | 0 | 0 | 0 |
| 05.10.99 | 1.4 | 0 | 0 | 0 | 0 |
| 12.10.99 | 1.4 | 0 | 0 | 0 | 0 |
| 19.10.99 | 1,5 | 0 | 0 | 0 | 0 |
| 26.10.99 | 1.6 | 0 | 0 | 0 | 0 |
| 02.11.99 | 1.6 | 0.08 | 8 | 0 | 0 |
| 09.11.99 | 1,7 | 2.3 | 20 | 0 | 0 |
| 17.11.99 | 1,7 | 0.1 | 20 | 0 | 0 |
| 22.11.99 | 1,7 | 0.76 | 60 | 0 | 0 |
| 07.12.99 | 1,8 | 1.6 | 64 | 0 | 0 |
| 10.12.99 | 1,8 | 1.5 | 60 | 0 | 0 |
| 14.12.99 | 1,8 | 0.4 | 48 | 0 | 0 |
| 18.01.00 | 1,8 | 0.58 | 60 | 0 | 0 |
| 15.02.00 | 1,9 | 0.48 | 32 | 0 | 0 |
| 15.03.00 | 2,3 | 0.2 | 20 | 0 | 0 |

Phoma incidence was generally low (<25% plants affected) on 20 January, significant control was apparent from the 9 November spray with 23 November and 7 December treatments also reducing incidence(Table 31). The 21 December spray was the most effective treatment, giving about 75% reduction in leaf spotting on 1 March assessments.

**Table 31.** Incidence (% plants affected) and severity (% leaf area affected) of phoma leaf spot in relation to fungicide treatment, High Mowthorpe 2000.

| Treatment | Treatment | 20.01.00 | | 01.03.00 | |
|---|---|---|---|---|---|
| | | % phoma Incidence | % phoma Severity | % phoma Incidence | % phoma Severity |
| 1 | Untreated | 22.0 | 0.24 | 21.3 | 0.33 |
| 2 (9/11) | Disease onset/late Oct | 8.8 | 0.09 | 17.5 | 0.18 |
| 3 (21/12) | 4-6 weeks later than 2 (early Dec) | 18.8 | 0.21 | 7.5 | 0.08 |
| 4 (7/02) | 4-6 weeks later than 3 (Jan) | 20.0 | 0.20 | 22.5 | 0.23 |
| 5 (28/3) | 4-6 weeks later than 4 (early March) | | | 22.5 | 0.23 |
| 6 | 2+3 | 9.6 | 0.10 | 7.5 | 0.08 |
| 7 | 2+4 | | | 20.0 | 0.20 |
| 8 | 2+5 | | | 12.5 | 0.13 |
| 9 | 3+4 | | | 7.5 | 0.08 |
| 10 | 3+5 | | | 10.0 | 0.10 |
| 11 | 4+5 | | | 40.0 | 0.40 |
| 12 | 2+3+4 | | | 2.5 | 0.03 |
| 13 | 2+3+5 | | | 10.0 | 0.10 |
| 14 | 3+4+5 | | | 10.0 | 0.10 |
| 15 | 2+4+5 | | | 15.0 | 0.15 |
| 16 | 2+3+4+5 | | | 0.0 | 0.00 |
| 17 | 2 weeks after 2, then 3+4+5 | 12.5 | 0.13 | 0.0 | 0.00 |
| 18 | 4 weeks after 2, then 3+4+5 | 12.5 | 0.13 | 0.0 | 0.00 |
| 19 | 2+3+4, then 2 weeks after 5 | | | 2.5 | 0.03 |
| 20 | 2+3+4, then 4 weeks after 5 | | | 7.5 | 0.08 |
| SED | min rep<br>Untreated v treated | 5.66<br>4.32 | 0.076<br>0.059 | 5.82<br>5.04 | 0.09<br>0.08 |
| 75df | | 60 | 60 | 60 | 60 |
| F test. | | skew | <0.1% | 1% | <0.1% |

**Table 32.** Incidence and severity of leaf and stem diseases at mid flowering, High Mowthorpe 2000.

| Treatment number | Treatment | Phoma on leaves (% incidence) | Phoma on leaves (% severity) | Light leaf spot on leaf (% incidence) | Light leaf spot on leaf (% severity) | Stem canker (% incidence) | Phoma stem lesions (% incidence) | Light leaf spot on stem (% incidence) | Light leaf spot on stem (% severity) |
|---|---|---|---|---|---|---|---|---|---|
| 1 | Untreated | 30.0 | 0.10 | 96.2 | 2.22 (-1.99) | 3.8 | 1.3 | 100 | 4.64 (-1.53) |
| 2 (9/11) | Disease onset/late Oct | 10.0 | 0.02 | 100.0 | 2.02 (-2.01) | 0 | 7.5 | 100 | 4.01 (-1.59) |
| 3 (21/12) | 4-6 weeks later than 2 (early Dec) | 30.0 | 0.07 | 100.0 | 2.21 (-1.98) | 5.0 | 5.0 | 90.0 | 4.28 (-1.69) |
| 4 (7/02) | 4-6 weeks later than 3 (Jan) | 20.0 | 0.03 | 97.5 | 1.21 (-2.21) | 2.5 | 0 | 92.5 | 1.64 (-2.14) |
| 5 (28/3) | 4-6 weeks later than 4 (early March) | 12.5 | 0.06 | 97.5 | 1.86 (-2.03) | 5.0 | 0 | 100.0 | 5.73 (-1.41) |
| 6 | 2+3 | 22.5 | 0.03 | 100.0 | 0.40 (-2.71) | 0 | 2.5 | 60.0 | 0.75 (-2.71) |
| 7 | 2+4 | 27.5 | 0.05 | 100.0 | 0.57 (-2.57) | 0 | 0 | 72.5 | 1.77 (-2.23) |
| 8 | 2+5 | 15.0 | 0.02 | 97.5 | 1.12 (-2.43) | 0 | 0 | 85.0 | 2.34 (-2.12) |
| 9 | 3+4 | 22.5 | 0.03 | 100.0 | 1.67 (-2.12) | 2.5 | 2.5 | 72.5 | 1.47 (-2.26) |
| 10 | 3+5 | 17.5 | 0.09 | 95.0 | 0.52 (-2.61) | 2.5 | 2.5 | 72.5 | 0.88 (-2.58) |
| 11 | 4+5 | 25.0 | 0.05 | 72.5 | 0.93 (-2.61) | 5.0 | 0 | 82.5 | 1.99 (-2.18) |
| 12 | 2+3+4 | 17.5 | 0.02 | 100.0 | 0.81 (-2.39) | 0 | 0 | 15.0 | 0.23 (-3.52) |
| 13 | 2+3+5 | 7.5 | 0.01 | 97.5 | 0.91 (-2.43) | 0 | 2.5 | 45.0 | 1.01 (-2.74) |
| 14 | 3+4+5 | 20.0 | 0.03 | 97.5 | 0.61 (-2.55) | 2.5 | 5.0 | 57.5 | 1.12 (-2.59) |
| 15 | 2+4+5 | 15.0 | 0.11 | 95.0 | 1.10 (-2.37) | 0 | 00 | 52.5 | 0.66 (-2.89) |
| 16 | 2+3+4+5 | 15.0 | 0.03 | 97.5 | 0.37 (-2.78) | 0 | 2.5 | 22.5 | 0.11 (-3.54) |
| 17 | 2 weeks after 2, then 3+4+5 | 15.0 | 0.02 | 95.0 | 0.45 (-2.71) | 0 | 0 | 47.5 | 0.93 (-2.77) |
| 18 | 4 weeks after 2, then 3+4+5 | 17.5 | 0.02 | 100.0 | 0.52 (-2.62) | 0 | 0 | 45.0 | 0.52 (-3.03) |
| 19 | 2+3+4, then 2 weeks after 5 | 10.0 | 0.01 | 87.5 | 0.87 (-2.45) | 0 | 5 | 27.5 | 0.79 (-3.10) |
| 20 | 2+3+4, then 4 weeks after 5 | 10.0 | 0.01 | 97.5 | 0.82 (-2.43) | 0 | 0 | 25.0 | 0.79 (-3.10) |
| SED (61df) | | 10.47 | 0.042 | 8.57 | 0.246 | 3.75 | 2.84 | 14.03 | 0.273 |
| min rep Untr. v treated | | 9.06 | 0.037 | 7.42 | 0.213* | 3.25 | 2.46 | 12.15 | 0.237* |
| F test | | ns | skew | ns | 1% | ns | ns | <0.1% | <0.1% |

* Logit transformed data in parentheses analysed

There were no significant treatment effects on phoma leaf spot at mid flowering on 25 May though all except one treatment had lower phoma leaf spot than the untreated control (Table 32). There were the first signs of canker and phoma stem lesions at this stage in untreated plots and some treatments, but levels were too low for effective statistical analysis. Light leaf spot was the most important disease at this stage affecting leaves and stems of 96-100% plants in control plots.

There was no reduction in the incidence of light leaf spot on the leaves, though the severity of the lesions was reduced from 2.2% area to less than 0.6% by some treatments with two or more sprays. Single sprays did not give significant control of light leaf spot but various combinations of two sprays (e.g. 2+3, 2+4 and 3+5) were as effective as three or four sprays.

Significant differences in both incidence and severity of light leaf spot on the stem was found between spray timings. Severity was reduced from 4.6% stem area to less than 1.0% in the most effective regimes. A single spray on 7 February controlled stem infection whereas earlier or later timings had no effect. There was a strong trend for three and four spray programmes to produce better disease control on stems than two sprays, although timing 2+3 was very effective (Table 32).

**Table 33.** Incidence and severity of stem diseases at crop maturity, High Mowthorpe 2000.

| Treatment number | Treatment | Light leaf spot on stems (% incidence) | Light leaf spot on stems (% severity) | Phoma stem lesions (mean index) | Phoma stem lesions (% incidence) | Canker (mean index) | Canker (% incidence) | Light leaf spot on pods (% incidence) | Light leaf spot on the pod (% severity) |
|---|---|---|---|---|---|---|---|---|---|
| 1 | Untreated | 100 | 6.17 | 0.75 | 41 | 0.74 | 42 | 100 | 23.2 |
| 2 (9/11) | Disease onset/late Oct | 100 | 3.68 | 0.57 | 38 | 0.08 | 6 | 99 | 25.8 |
| 3 (21/12) | 4-6 weeks later than 2 (early Dec) | 100 | 5.51 | 0.57 | 33 | 0.21 | 15 | 99 | 25.6 |
| 4 (7/02) | 4-6 weeks later than 3 (Jan) | 91 | 3.01 | 0.34 | 22 | 0.17 | 10 | 100 | 20.3 |
| 5 (28/3) | 4-6 weeks later than 4 (early March) | 98 | 3.38 | 0.76 | 47 | 0.58 | 29 | 98 | 12.2 |
| 6 | 2+3 | 87 | 2.18 | 0.03 | 5 | 0.02 | 2 | 100 | 16.3 |
| 7 | 2+4 | 95 | 1.90 | 0.08 | 9 | 0.01 | 1 | 100 | 13.3 |
| 8 | 2+5 | 87 | 2.00 | 0.16 | 14 | 0.06 | 5 | 98 | 5.5 |
| 9 | 3+4 | 81 | 1.67 | 0.20 | 14 | 0.14 | 11 | 99 | 8.7 |
| 10 | 3+5 | 88 | 2.33 | 0.14 | 13 | 0.05 | 5 | 99 | 16.1 |
| 11 | 4+5 | 82 | 2.04 | 0.28 | 24 | 0.09 | 8 | 97 | 5.6 |
| 12 | 2+3+4 | 57 | 1.22 | 0.08 | 4 | 0.00 | 0 | 78 | 2.4 |
| 13 | 2+3+5 | 58 | 1.05 | 0.01 | 1 | 0.00 | 0 | 99 | 4.9 |
| 14 | 3+4+5 | 67 | 1.98 | 0.09 | 7 | 0.08 | 5 | 99 | 6.1 |
| 15 | 2+4+5 | 75 | 2.01 | 0.08 | 8 | 0.10 | 9 | 90 | 5.7 |
| 16 | 2+3+4+5 | 19 | 0.94 | 0.01 | 1 | 0.02 | 2 | 93 | 1.8 |
| 17 | 2 weeks after 2, then 3+4+5 | 49 | 1.40 | 0.09 | 5 | 0.07 | 4 | 90 | 3.7 |
| 18 | 4 weeks after 2, then 3+4+5 | 55 | 1.44 | 0.05 | 4 | 0.03 | 3 | 93 | 7.5 |
| 19 | 2+3+4, then 2 weeks after 5 | 52 | 0.91 | 0.02 | 2 | 0.00 | 0 | 99 | 3.4 |
| 20 | 2+3+4, then 4 weeks after 5 | 50 | 0.97 | 0.00 | 0 | 0.00 | 0 | 83 | 3.2 |
| SED (61df)<br>min rep<br>Untr v. treated | | 11.07<br><br>9.59 | 0.911<br><br>0.744 | 0.150<br><br>0.130 | 8.06<br><br>6.98 | 0.128<br><br>0.111 | 7.24<br><br>6.27 | 4.74<br><br>4.11 | 7.03<br><br>6.09 |
| F test | | <0.1% | <0.1%* | <0.1% | <0.1%* | skew | skew | skew | <0.1%* |

* Logit transform unskewed data, untransformed data presented.

Prior to swathing on 20 July, a final disease assessment was made on 25 plants from each plot.The high proportion of disease in the untreated control plots gave a good bench mark for determining the response of the selective spray timing programme. The incidence of light leaf spot on the stem was 100% in the absence of any fungicide and required three or four sprays to reduce incidence levels were reduced by half. The severity of stem lesions reached 6.2% stem area and was reduced by all treatments except the single December spray. Most three and four spray programmes reduced light leaf spot severity to less than 1.0% stem area, but this was not a significant improvement over two-spray treatments (Table 33).

Phoma cankers and stem lesions showed a similar incidence in control plots (42% and 41% plants affected respectively). Control of these two types of stem infection showed a marked contrast, with single sprays (except for 28 March spray) providing good control of canker and no control of stem lesions. Multiple sprays reduced the incidence of both to less than 5% plants affected in many cases and the best two-spray programmes were as good as four sprays (Table 33). Mean canker and phoma stem lesion severity showed an index of 0.74-0.75, which, given 41-42% only incidence, indicates untreated lesions were mainly moderate severity (>50% stem girdled). Fungicide treatments reduced both incidence and severity of lesions and disease indices therefore reflect disease control more effectively than incidence data alone.

Although there were high levels of light leaf spot on the pods, the autumn and winter programmes provided good reductions in disease severity from 23% in the untreated to <7% pod area affected. The final main spray on 28 March narrowly missed giving a significant effect and at least two sprays were needed to give good control (e.g. timing 2+5) (Table 33).

There were significant yield increases (up to 1.03 t/ha) from most three-spray programmes and all the four-spray programmes. Only the Nov + December two spray programme gave a significant yield response (Table 34). There was accumulation of yield with each additional spray up to the fourth treatment of 0.18-0.28 t/ha.

Following harvest, a stubble count made on 8 August revealed significant treatment differences of over 20 plants/$m^2$ . The higher populations were particularly associated with spray programmes which included the first main timing (Table 34).

**Table 34.** Yield and plant population in relation to fungicide treatment, High Mowthorpe 2000.

| Treatment number | Treatment | Yield (t/ha @ 90% DM) | Plants/m2 |
|---|---|---|---|
| 1 | Untreated | 3.32 | 69.1 |
| 2 (9/11) | Disease onset/late Oct | 3.38 | 70.6 |
| 3 (21/12) | 4-6 weeks later than 2 (early Dec) | 3.62 | 62.4 |
| 4 (7/02) | 4-6 weeks later than 3 (Jan) | 3.70 | 67.4 |
| 5 (28/3) | 4-6 weeks later than 4 (early March) | 3.30 | 70.2 |
| 6 | 2+3 | 3.92 | 87.2 |
| 7 | 2+4 | 3.63 | 88.2 |
| 8 | 2+5 | 3.70 | 77.2 |
| 9 | 3+4 | 3.66 | 73.2 |
| 10 | 3+5 | 3.77 | 81.4 |
| 11 | 4+5 | 3.48 | 74.8 |
| 12 | 2+3+4 | 3.73 | 90.0 |
| 13 | 2+3+5 | 3.93 | 86.2 |
| 14 | 3+4+5 | 3.84 | 76.2 |
| 15 | 2+4+5 | 4.25 | 85.0 |
| 16 | 2+3+4+5 | 4.18 | 87.2 |
| 17 | 2 weeks after 2, then 3+4+5 | 4.16 | 81.4 |
| 18 | 4 weeks after 2, then 3+4+5 | 4.11 | 85.6 |
| 19 | 2+3+4, then 2 weeks after 5 | 4.35 | 90.6 |
| 20 | 2+3+4, then 4 weeks after 5 | 4.29 | 99.0 |
| Mean of 1 spray | | 3.50 | |
| Mean of 2 sprays | | 3.69 | |
| Mean of 3 sprays | | 3.94 | |
| Mean of 4 sprays | | 4.22 | |
| SED (61df) min rep<br>Untr v. treated | | 0.244<br><br>0.211 | 8.18<br><br>7.04 |
| F test | | <0.1% | <0.1% |

**Rothamsted 1999/2000**

Disease development on untreated plants

Downy mildew affected the cotyledons and first leaves from late September (0.4% leaf area affected on 5 October) but had disappeared by mid-October.

Phoma leaf spot was first seen on 28 September (one month earlier than in 1998) and, by 19 October, 100% of untreated plants were affected. Older, infected leaves began to fall off in early November and phoma leaf spot incidence and severity therefore decreased. However, phoma leaf spot increased again from mid-November with lesions appearing on younger leaves, and by 23 November 100% plants were affected (0.9% leaf area). Severity continued to increase and 2.8% leaf area was affected by 19 January. The 1999 phoma leaf spot epidemic was therefore earlier, more severe and more prolonged than in 1998 (when it reached a maximum of 88% plants and 1% leaf area affected in early December) (Table 35).

The first stem canker was seen on 7 March (single plant from a treated plot). This was three weeks earlier than in 1999. Incidence of basal stem canker on untreated plots increased to 35% plants by 27 March and 100% plants by 16 May. Stem lesions were found on 72% with on 14 June. Severity was also high, with an average score for basal canker of 2.9 and for stem lesions of 1.2 on 12 July (Table 36). This compares to an average score of less than 1.0 for stem canker in July 1999.

By contrast, light leaf spot developed later than in 1998/99 (first seen in mid-February in 2000 compared to mid-January in 1999) and only a mild epidemic developed on leaves (3.9% leaf area affected in March 2000 compared to 10% in 1999). However, a moderate epidemic developed on stems and pods (18.8% pod area affected in July). There was a very small amount of dark leaf spot (alternaria) on leaves and some stem rot (sclerotinia) on stems. Dark pod spot (alternaria) affected 5% pod area late in the season (Table 36).

Disease control

The first spray timing (5 Oct) significantly reduced early phoma leaf spot (Tables 37 & 38) until 23 November. No other sprays were applied in time to control the early epidemic. The second spray timing (applied 3 November, four weeks after disease onset) gave the most effective control of this later epidemic, clearly evident eight weeks after treatment on 6 January. The third and fourth spray timing (13 December and 20 January) were too late to control either of the two phases of the epidemic. Two or more sprays gave little more control than a single well-timed spray.

The most effective spray timing for canker control was four weeks after the onset of phoma leaf spot (3 November) which, at the last assessment in July, reduced canker incidence from 98 to 70% and the mean severity score from 2.9 to 1.2 (Table 39). As early as 27 March, early suppression of canker was apparent. The 3 November spray appeared to reduce disease severity for longer than the 5 October spray (Table 40). Addition of a later spray (3 December or 20 January) further improved control of canker, but without the 3 November spray, these later sprays, whether applied separately or together, had little effect on the disease. Phoma stem lesions were present on 75% untreated plants as small lesions and were only well controlled by some four-spray programmes (Table 39).

Light leaf spot did not appear until mid-February, 18 weeks after inoculum was applied, and affected 9.5% leaf area in late March and up to 23% pod area. Some multiple spray programmes reduced light leaf spot on pods, but control was unreliable. No spray programmes controlled alternaria on pods (Table 41).

Despite the severe phoma leaf spot and canker epidemics, there were no differences in plant numbers between treatments at harvest.

The maximum yield of 4.64 t/ha (treatment 18) was 0.89 t/ha (19%) higher than untreated yield. Single sprays gave an average yield increase of only 0.02 t/ha, but the best single spray (3 November) gave a yield increase of 0.16 t/ha. Each additional spray gave a slightly higher yield, and, on average, a four-spray programme increased yield by average, 0.77 t/ha (Table 42). Two-spray programmes gave the most reliable yield benefits, increasing yields by an average 0.25t/ha for each spray applied. The best two-spray programme (3 Nov + 13 Dec) increased yields by 0.35 t/ha for each spray applied.

**Table 35.** Disease development on winter oilseed rape plants and leaves in untreated control plots, Rothamsted 1999/2000.

| Date | Sample size (no. of plants | Growth stage | Phoma leaf spot | | | Light leaf spot | | |
|---|---|---|---|---|---|---|---|---|
| | | | % plants | % leaves | % leaf area | % plants | % leaves | % leaf area |
| 21.09.99 | 25 | 1,4 | 0 | 0 | 0 | 0 | | |
| 28.09.99 | 25 | 1,6 | 4 | 0.7 | 0.004 | 0 | | |
| 05.10.99 | 25 | 1,7 | 64 | 13.6 | 0.13 | 0 | | |
| 12.10.99 | 25 | 1,9 | 96 | 26.9 | 0.27 | 0 | | |
| 19.10.99 | 25 | 1,9 | 100 | 27.0 | 0.43 | 0 | | |
| 26-Oct-99 | 30 | 1,10 | 100 | 28.4 | 0.99 | 0 | | |
| 02.11.99 | 30 | 1,11 | 87 | 20.4 | 0.65 | 0 | | |
| 09.11.99 | 30 | 1,11 | 70 | 13.0 | 0.36 | 0 | | |
| 16.11.99 | 30 | 1,11 | 87 | 20.0 | 0.56 | 0 | | |
| 23.11.99 | 30 | 1,12 | 100 | 35.9 | 0.95 | 0 | | |
| 30.11.99 | 30 | 1,12 | 93 | 38.0 | 1.70 | 0 | | |
| 07.12.99 | 30 | 1,12 | 100 | 39.8 | 1.45 | 0 | | |
| 13.12.99 | 30 | 1,13 | 100 | 35.0 | 0.97 | 0 | | |
| 06.01.00 | 60 | 1,13 | 98 | 31.3 | 2.49 | 0 | | |
| 19.01.00 | 30 | 2,0 | 97 | 33.5 | 2.80 | 0 | | |
| 15.02.00 | 60 | 2,1 | 100 | 28.6 | 1.18 | 26.7 | 3.9 | 0.4 |
| 14.03.00 | 30 | 3,3 | 97 | 20.6 | 1.90 | 76.6 | 19.0 | 3.4 |

**Table 36.** Disease development on winter oilseed rape stems and pods in untreated control plots (mean of 30 or 60 plants), Rothamsted 1999/2000.

| Date | Growth stage | Phoma infection | | | | Light leaf spot | | | | Alternaria | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Canker | | Phoma stem lesions | | Stems | | Pods | | Pods | |
| | | % plants | Index | % plants | Index | % plants | % area | % plants | % area | % plants | % area |
| 07.03.00* | 2,5 | 0 | 0 | 0 | 0 | | | | | | |
| 22.03.00* | 3,5 | 16.6 | 0.2 | 0 | 0 | | | | | | |
| 27.03.00 | 3,6 | 35.0 | 0.4 | 0 | 0 | 87 | 0.3 | | | | |
| 06.04.00* | 3,7 | 60.0 | 0.6 | 3.3 | 0.03 | | | | | | |
| 19.04.00* | 4,2 | 86.7 | 0.9 | 0 | 0 | | | | | | |
| 03.05.00* | 4,8 – 5,1 | 93.3 | 1.4 | 10.0 | 0.1 | | | | | | |
| 16.05.00* | 5,7 | 100 | 1.4 | 6.6 | 0.06 | | | | | | |
| 02.06.00* | 6,2 | 100 | 1.6 | 40.0 | 0.4 | | | | | | |
| 14.06.00 | 6,3 | 98.3 | 2.1 | 72.0 | 0.9 | 93 | 6.3 | 93 | 4.7 | 16.7 | 0.08 |
| 19.06.00* | 6,2 – 6,3 | 100 | 1.6 | 50.0 | 0.5 | | | | | | |
| 06.07.00* | | 100 | 1.9 | 66.6 | 0.6 | | | | | | |
| 12.07.00 | 9,9 | 98.3 | 2.9 | 75.0 | 1.2 | 95 | 18.3 | 100 | 18.8 | 82.5 | 5.1 |

* mean of 30 plants assessed by internal examination

**Table 37.** Effect of spray timing on phoma leaf spot (% plants and % leaves affected and % leaf area affected) Rothamsted, autumn 1999.

| Treatment number | Spray timing | Phoma leaf spot assessments | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 12.10. 99 | | | 19.10. 99 | | | 26.10. 99 | | | 02.11.99 | | | 09.11.99 | | |
| | | % plants | % leaves | % leaf area | % plants | % leaves | %leaf area | % plants | % leaves | %leaf area | % plants | % leaves | %leaf area | % plants | % leave | %leaf area |
| 1 | Untreated | 96.0 | 26.9 | 0.27 | 100 | 27.0 | 0.43 | 100 | 28.4 | 1.14 | 86.7 | 20.4 | 0.65 | 70.0 | 13.0 | 0.36 |
| 2 | 5/10 | 96.0 | 20.0 | 0.18 | 84.6 | 19.7 | 0.19 | 73.3 | 14.5 | 0.29 | 73.3 | 13.1 | 0.19 | 53.3 | 9.7 | 0.16 |
| 3 | 3/11 | | | | | | | | | | | | | 90 | 19.7 | 0.68 |
| 2+3 | | | | | | | | | | | | | | 50 | 7.9 | 0.09 |
| 17 (1st 1 or 2 sprays only | 25/10 + 3/11 | | | | | | | *90.0 | *26.7 | *1.0 | *90.0 | *19.0 | *0.56 | **90.0 | **19.0 | **0.65 |
| 18 (1st 1 or 2 sprays only) | 29/10 + 3/11 | | | | | | | | | | *93.3 | *22.5 | *0.66 | **76.7 | **15.2 | **0.48 |
| Mean | | | | | | | | 87.8 | 23.5 | 0.8 | 85.8 | 18.8 | 0.51 | 72 | 14.1 | 0.4 |
| SED | | | | | | | | 5.4 | 1.6 | 0.2 | 5.6 | 3.2 | 0.17 | 10.6 | 2.3 | 0.12 |
| df | | | | | | | | 4 | 4 | 4 | 6 | 6 | 6 | 10 | 10 | 10 |
| F test | | | | | | | | 5% | 1% | 5% | 5% | ns | ns | 5% | 1% | 1% |

* 1st spray only * 1st two sprays only

**Table 38.** Effect of spray timing on phoma leaf spot (% plants and % leaves affected and % leaf area affected), Rothamsted, November – March 1999/2000.

| Treatment number | Spray timing | Phoma leaf spot assessments | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 23.11.99 | | | 06.01.00 | | | 15.02.00 | | | 27.03.00 | | |
| | | % plants | % leaves | % lf area | % plants | % leaves | %leaf area | % plants | % leaves | %leaf area | % plants | % leaves | %leaf area |
| 1 | Untreated | 100 | 35.88 | 0.96 | 98.3 | 31.34 | 2.49 | 100 | 28.6 | 1.18 | 80.0 | 12.09 | 0.49 |
| 2 | Disease onset (5/10) | 90.0 | 26.94 | 0.34 | 96.7 | 30.55 | 2.71 | 100 | 26.8 | 1.00 | 70.0 | 9.98 | 0.26 |
| 3 | 4-6 weeks later than 2 (3/11) | 90.0 | 23.51 | 0.54 | 90.0 | 21.50 | 0.50 | 100 | 27.3 | 1.03 | 53.3 | 7.03 | 0.17 |
| 4 | 4-6 weeks later than 3 (13/12) | | | | 100 | 33.42 | 2.51 | 96.7 | 30.1 | 1.07 | 60.0 | 8.94 | 0.21 |
| 5 | 4-6 weeks later than 4 (20/01) | | | | | | | 100 | 26.1 | 1.10 | 56.7 | 7.63 | 0.30 |
| 6 | 2+3 | 66.7 | 13.83 | 0.10 | 93.3 | 23.09 | 0.84 | 100 | 28.3 | 0.96 | 73.3 | 8.98 | 0.27 |
| 7 | 2+4 | | | | 100 | 32.92 | 2.23 | 100 | 25.6 | 1.33 | 66.7 | 9.43 | 0.44 |
| 8 | 2+5 | | | | | | | 100 | 28.1 | 0.85 | 53.3 | 6.45 | 0.16 |
| 9 | 3+4 | | | | 96.7 | 21.61 | 0.71 | 100 | 28.9 | 1.05 | 53.3 | 15.00 | 0.49 |
| 10 | 3+5 | | | | | | | 100 | 28.9 | 0.76 | 63.3 | 8.56 | 0.18 |
| 11 | 4+5 | | | | | | | 100 | 25.3 | 0.87 | 46.7 | 9.28 | 0.34 |
| 12 | 2+3+4 | | | | 90.0 | 21.35 | 0.66 | 100 | 28.1 | 0.93 | 66.7 | 6.44 | 0.16 |
| 13 | 2+3+5 | | | | | | | 100 | 31.4 | 1.18 | 90.0 | 8.37 | 0.22 |
| 14 | 3+4+5 | | | | | | | 100 | 26.0 | 0.74 | 56.7 | 10.65 | 0.34 |
| 15 | 2+4+5 | | | | | | | 100 | 28.8 | 1.35 | 80.0 | 4.58 | 0.12 |
| 16 | 2+3+4+5 | | | | | | | 100 | 31.0 | 0.84 | 60.0 | 10.43 | 0.27 |
| 17 | 25/10 +3+4+5 | 80.0* | 14.63* | 0.36* | | | | 100 | 26.3 | 0.61 | 76.7 | 11.73 | 0.32 |
| 18 | 29/10 +3+4+5 | 83.3* | 17.53* | 0.48* | | | | 100 | 26.9 | 0.76 | 70.0 | 9.04 | 0.27 |
| 19 | 2+3+4+ 3/02 | | | | | | | 96.7 | 24.7 | 0.59 | 63.3 | 9.34 | 0.23 |
| 20 | 2+3+4+ 18/02 | | | | | | | **96.7 | **25.50 | **0.72 | 73.3 | 7.83 | 0.19 |
| Mean | | 85.0 | 22.0 | 0.46 | 95.9 | 27.5 | 1.7 | 99.5 | 27.7 | 0.9 | 66.3 | 9.2 | 0.3 |
| SED | | 8.6 | 3.4 | 0.12 | 5.5 | 2.9 | 0.3 | | 2.7 | 0.2 | 17.6 | 2.8 | 0.1 |
| df | | 10 | 10 | 10 | 17 | 17 | 17 | | 41 | 41 | 41 | 41 | 41 |
| F test | | 5% | <0.1% | <0.1% | ns | <0.1% | <0.1% | ns | ns | ns | ns | ns | ns |

* 1st two sprays only ** 1st three sprays only

**Table 39.** Effect of spray timing on stem canker and stem lesion incidence (I) (% plants affected) and severity (S) (0 – 4 score), Rothamsted 1999/2000.

| Treatment number | Spray timing | Assessment dates | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 27.03.00 | | 14.06.00 | | | | 12.07.00 | | | |
| | | Canker | | Canker | | Phoma stem lesions | | Canker | | Phoma stem lesions | |
| | | I | S | I | S | I | S | I | S | I | S |
| 1 | Untreated | 35.0 | 0.38 | 98.3 | 2.07 | 71.7 | 0.87 | 98.3 | 2.87 | 75 | 1.18 |
| 2 | Disease onset (5/10t) | 40.0 | 0.43 | 93.3 | 1.47 | 63.3 | 0.67 | 100 | 2.73 | 100 | 1.7 |
| 3 | 4-6 weeks later than 2 (3/11) | 13.3 | 0.13 | 80.0 | 0.9 | 63.3 | 0.67 | 70.0 | 1.17 | 66.7 | 0.8 |
| 4 | 4-6 weeks later than 3 (13/12) | 23.3 | 0.23 | 96.7 | 1.37 | 63.3 | 0.67 | 90.0 | 1.9 | 76.7 | 1.4 |
| 5 | 4-6 weeks later than 4 (20/01) | 36.7 | 0.37 | 93.3 | 1.6 | 60.0 | 0.6 | 100 | 2.3 | 63.3 | 0.67 |
| 6 | 2+3 | 0.0 | 0.00 | 50.0 | 0.57 | 53.3 | 0.6 | 70.0 | 1.13 | 80.0 | 1.07 |
| 7 | 2+4 | 10.0 | 0.10 | 83.3 | 1.17 | 50.0 | 0.5 | 86.7 | 1.47 | 83.3 | 1.03 |
| 8 | 2+5 | 10.0 | 0.10 | 76.7 | 1.2 | 53.3 | 0.53 | 76.7 | 1.6 | 83.3 | 1.07 |
| 9 | 3+4 | 3.3 | 0.03 | 56.7 | 0.6 | 46.7 | 0.47 | 73.3 | 0.9 | 63.3 | 0.83 |
| 10 | 3+5 | 16.7 | 0.17 | 23.3 | 0.23 | 16.7 | 0.17 | 43.3 | 0.63 | 50.0 | 0.5 |
| 11 | 4+5 | 13.3 | 0.17 | 66.7 | 0.93 | 16.7 | 0.17 | 86.7 | 1.6 | 80.0 | 1.0 |
| 12 | 2+3+4 | 10.0 | 0.10 | 40.0 | 0.5 | 26.7 | 0.27 | 43.3 | 0.53 | 66.7 | 0.67 |
| 13 | 2+3+5 | 6.7 | 0.07 | 30.0 | 0.33 | 33.3 | 0.33 | 26.7 | 0.33 | 56.7 | 0.67 |
| 14 | 3+4+5 | 6.7 | 0.07 | 33.0 | 0.37 | 16.7 | 0.17 | 26.7 | 0.4 | 56.7 | 0.77 |
| 15 | 2+4+5 | 16.7 | 0.17 | 56.7 | 0.73 | 50.0 | 0.5 | 66.7 | 1.3 | 46.7 | 0.6 |
| 16 | 2+3+4+5 | 6.7 | 0.07 | 23.3 | 0.23 | 3.3 | 0.03 | 16.7 | 0.17 | 36.7 | 0.37 |
| 17 | 25/10 +3+4+5 | 0.0 | 0.00 | 20.0 | 0.2 | 46.7 | 0.47 | 13.3 | 0.23 | 63.3 | 0.83 |
| 18 | 29/10 +3+4+5 | 3.3 | 0.03 | 10.0 | 0.1 | 23.3 | 0.3 | 10.0 | 0.13 | 20.0 | 0.2 |
| 19 | 2+3+4+ 3/02 | 3.3 | 0.03 | 16.7 | 0.17 | 20.0 | 0.2 | 36.7 | 0.37 | 26.7 | 0.27 |
| 20 | 2+3+4+ 18/02 | 3.3 | 0.03 | 10.0 | 0.1 | 16.7 | 0.17 | 21.1 | 0.21 | 26.7 | 0.33 |
| Grand mean | | 14.0 | 0.15 | 55.1 | 0.8 | 41.3 | 0.44 | 59.7 | 1.18 | 61.7 | 0.82 |
| SED (41 degrees of freedom) | | 7.3 | 0.08 | 11.6 | 0.19 | 12.3 | 0.14 | 11.4 | 0.23 | 12.1 | 0.21 |
| F test | | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% | <0.1% |

**Table 40.** Effect of earliest fungicide timings on development of canker lesions (30 plants assessed by internal examination), Rothamsted 2000.

| Assessment date | Canker development (0-4 index) | | |
|---|---|---|---|
| | Fungicide timing | | |
| | Untreated | Disease onset (5.10.99) | Four weeks after disease onset (3.11.99) |
| 07.03.00 | 0.00 | 0.03 | 0.00 |
| 22.03.00 | 0.40 | 0.23 | 0.10 |
| 06.04.00 | 0.60 | 0.47 | 0.07 |
| 19.04.00 | 0.83 | 0.50 | 0.30 |
| 03.05.00 | 1.37 | 1.10 | 0.40 |
| 16.05.00 | 1.43 | 1.33 | 0.63 |
| 02.06.00 | 1.63 | 1.50 | 0.83 |
| 19.06.00 | 1.63 | 1.83 | 0.80 |
| 06.07.00 | 1.87 | 1.83 | 1.47 |

**Table 41.** Effect of fungicide timing on light leaf spot (LLS) and Alternaria incidence (I) (% plants affected) and severity (S) (% leaf area (L) or % pod area (P) affected) Rothamsted 2000.

| Treatment | Spray timing | Assessment date | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 15.02.00 | | 27.03.00 | | 14.06.00 | | | | 12.07.00 | | | |
| | | LLS | | LLS | | LLS | | Alternaria | | LLS | | Alternaria | |
| | | I | S(L) | I | S(L) | I | S(P) | I | S(P) | I | S(P) | I | S(P) |
| 1 | Untreated | 26.7 | 0.39 | 85.0 | 9.55 | 100 | 4.72 | 16.7 | 0.08 | 100 | 18.80 | 82.5 | 5.1 |
| 2 | Disease onset (5/10) | 30.0 | 0.32 | 80.0 | 12.23 | 100 | 3.11 | 20.0 | 0.05 | 100 | 23.87 | 93.3 | 3.9 |
| 3 | 4-6 weeks later than 2 (3/11) | 33.3 | 0.26 | 60.0 | 5.68 | 86.7 | 3.83 | 20.0 | 0.04 | 100 | 11.90 | 86.7 | 3.3 |
| 4 | 4-6 weeks later than 3 (13/12) | 33.3 | 0.61 | 70.0 | 9.34 | 100 | 4.17 | 23.3 | 0.04 | 100 | 14.80 | 86.7 | 4.2 |
| 5 | 4-6 weeks later than 4 (20/01) | 30.0 | 0.54 | 63.3 | 4.59 | 100 | 2.63 | 16.7 | 0.02 | 100 | 11.97 | 86.7 | 3.4 |
| 6 | 2+3 | 33.3 | 0.63 | 80.0 | 6.97 | 96.7 | 3.95 | 13.3 | 0.01 | 100 | 11.50 | 93.3 | 2.4 |
| 7 | 2+4 | 13.3 | 0.07 | 90.0 | 17.93 | 93.3 | 1.06 | 13.3 | 0.05 | 100 | 12.07 | 93.3 | 3.2 |
| 8 | 2+5 | 36.7 | 0.28 | 76.7 | 11.33 | 90.0 | 2.05 | 23.3 | 0.07 | 100 | 16.53 | 90.0 | 3.8 |
| 9 | 3+4 | 36.7 | 0.66 | 36.7 | 1.67 | 96.7 | 3.33 | 6.7 | 0.07 | 100 | 10.60 | 93.3 | 3.4 |
| 10 | 3+5 | 16.7 | 0.13 | 40.0 | 4.61 | 96.7 | 1.31 | 20.0 | 0.06 | 100 | 5.00 | 86.7 | 2.2 |
| 11 | 4+5 | 10.0 | 0.07 | 73.3 | 6.68 | 100 | 1.97 | 33.3 | 0.06 | 100 | 12.73 | 93.3 | 3.9 |
| 12 | 2+3+4 | 20.0 | 0.15 | 56.7 | 3.57 | 90.0 | 2.74 | 3.3 | 0.00 | 100 | 13.80 | 93.3 | 7.4 |
| 13 | 2+3+5 | 10.0 | 0.06 | 40.0 | 1.47 | 93.3 | 1.86 | 16.7 | 0.05 | 100 | 10.28 | 93.3 | 2.9 |
| 14 | 3+4+5 | 20.0 | 0.25 | 20.0 | 0.32 | 50.0 | 1.17 | 36.7 | 0.06 | 100 | 8.90 | 93.3 | 2.7 |
| 15 | 2+4+5 | 33.3 | 1.03 | 76.7 | 11.00 | 100 | 2.74 | 20.0 | 0.06 | 100 | 10.77 | 96.3 | 4.8 |
| 16 | 2+3+4+5 | 23.3 | 0.48 | 56.7 | 3.79 | 86.7 | 2.33 | 10.0 | 0.00 | 100 | 7.12 | 93.3 | 3.1 |
| 17 | 25/10 +3+4+5 | 33.3 | 0.85 | 23.3 | 0.22 | 63.3 | 0.29 | 10.0 | 0.00 | 100 | 10.40 | 83.3 | 3.9 |
| 18 | 29/10 +3+4+5 | 13.3 | 0.06 | 56.7 | 3.16 | 93.3 | 0.67 | 16.7 | 0.02 | 93.3 | 6.66 | 90.0 | 4.2 |
| 19 | 2+3+4+ 3/02 | 16.7 | 0.10 | 33.3 | 1.68 | 76.7 | 0.91 | 30.0 | 0.05 | 100 | 6.59 | 100 | 3.4 |
| 20 | 2+3+4+ 18/02 | 23.3* | 0.12* | 26.7 | 0.33 | 86.7 | 1.65 | 33.3 | 0.14 | 100 | 8.10 | 89.6 | 3.5 |
| Grand mean | | 24.8 | 0.36 | 58.6 | 6.0 | 90.5 | 2.4 | 19.0 | 0.05 | 99.7 | 12.0 | 90.5 | 3.8 |
| SED (41 df) | | 12.2 | 0.28 | 15.4 | 4.9 | 10.0 | 1.3 | 9.2 | 0.06 | | 3.4 | 7.6 | 1.6 |
| F test | | ns | ns | <0.1 % | ns | 1% | ns | ns | ns | ns | 1% | ns | ns |

* 1st three sprays only

**Table 42.** Effect of fungicide timing on yield, Rothamsted 2000.

| Treatment code | Treatment | Yield at 90%DM (t/ha) | Oil yield (t/ha) |
|---|---|---|---|
| 1 | Untreated | 3.75 | 1.53 |
| 2 | Disease onset (5/11) | 3.65 | 1.49 |
| 3 | 4-6 weeks later than 2 (3/11) | 3.91 | 1.59 |
| 4 | 4-6 weeks later than 3 (13/12) | 3.67 | 1.50 |
| 5 | 4-6 weeks later than 4 (20/1) | 3.85 | 1.57 |
| 6 | 2+3 | 4.22 | 1.71 |
| 7 | 2+4 | 3.96 | 1.62 |
| 8 | 2+5 | 4.12 | 1.70 |
| 9 | 3+4 | 4.46 | 1.82 |
| 10 | 3+5 | 4.40 | 1.80 |
| 11 | 4+5 | 4.41 | 1.82 |
| 12 | 2+3+4 | 4.51 | 1.85 |
| 13 | 2+3+5 | 4.35 | 1.78 |
| 14 | 3+4+5 | 4.54 | 1.87 |
| 15 | 2+4+5 | 3.99 | 1.62 |
| 16 | 2+3+4+5 | 4.57 | 1.91 |
| 17 | 25/10/99 +3+4+5 | 4.59 | 1.90 |
| 18 | 29/10/99 +3+4+5 | 4.64 | 1.91 |
| 19 | 2+3+4+ 3/2/00 | 4.45 | 1.83 |
| 20 | 2+3+4+ 18/2/00 | 4.34 | 1.81 |
| Grand mean | | 4.20 | 1.72 |
| Mean of 1 spray | | 3.77 | 1.54 |
| Mean of 2 sprays | | 4.26 | 1.74 |
| Mean of 3 sprays | | 4.35 | 1.78 |
| Mean of 4 sprays | | 4.52 | 1.87 |
| SED (41df) | | 0.19 | 0.085 |
| F test | | <0.1% | <0.1% |

## 4.5 Discussion

### Boxworth 1998/1999

A moderate to severe phoma leaf spot epidemic produced a high incidence of stem canker, but with a low severity score. This would be accounted for by the late November onset of the main epidemic and the favourable growing conditions in autumn, which resulted in large plants able to withstand disease. By late October, leaves were 14-20 cm long and this increased to 20-25 cm by early November at GS 1,09. Plants matured early and quickly in late June and there was less time than usual for cankers to develop into moderate or severe lesions and affect yield. There were only low levels of other diseases at this site and there were no significant effects on yield.

The first fungicide was not applied until 20 October, which was just prior to onset of phoma leaf spotting in assessed samples (note : phoma leaf spot symptoms had started to appear in other crops at Boxworth in late October 1998). Subsequent build up of phoma spotting was protracted and the October spray was clearly effective in early December against phoma leaf spot. The key element for yield response is canker control rather than leaf spot control and it appears that application in January was still able to control canker, presumably by stopping the canker fungus reaching the stem rather than stopping leaf spots appearing. Such late applications may be a risky strategy if conditions do not allow sprays to be applied. In this case, there was large leaf size and moderate disease resistance (in Apex), which appear to give greater flexibility on spray timing than previous work on susceptible varieties might suggest. There were clear benefits in disease control from multiple applications of fungicide and this implies additive effects - each spray giving control for a limited period and single sprays only occasionally giving worthwhile effects. Phoma stem lesions were not so well controlled as canker lesions and in this case the best single spray was in March, later than for canker (with its January optimum, see Table 5).

A yield increase of 0.2 t/ha is needed to cover the cost of a single fungicide application (based on seed price of £100 t/ha and fungicide costs of £10/ha per spray at 1999 prices). In this crop, not spraying was almost certainly the best option.

This experiment provides new information on the effect of late epidemics on yield loss in moderately resistant varieties. New biological data on disease control, leaf spot-canker relationships and disease-yield loss have been obtained. Important messages for the industry can be formulated so that late epidemics can be managed with only single fungicide sprays.

**High Mowthorpe 1998/1999**

The phoma leaf spot epidemic developed from November onwards and reached 20% plants affected when plants were at the seven leaf stage. Previous work in France had indicated that plants were less susceptible to phoma after the six leaf stage. This may be a useful criterion to include in decision making as phoma stem infections were in the slight category at this site. A further factor to consider is that the peak incidence of phoma leaf spot was only 60% plants affected at this site. Canker did not develop but phoma stem lesions were common at harvest. Future modelling work will need to consider not only timing of epidemics but also the likely incidence of plant infection.

This was a moderate yielding site, which averaged 3.10 t/ha overall and this performance is thought to reflect poor conditions for seed filling. Trends in the yield data suggested spring sprays were providing greater benefits than autumn sprays and this is likely to be due to effects on light leaf spot rather than phoma. The level of light leaf spot control in April was disappointing, given the use of four sprays in some treatments.

**Rothamsted 1998/1999**

A moderate phoma leaf spot epidemic produced a high incidence of stem canker, but with a low severity score. This was probably due to the favourable growing conditions early in the season producing large, robust plants, able to withstand disease, and also to the hot, dry conditions in July which caused the plants to mature quickly and be harvested early, so that there was insufficient time for severe cankers to develop. A moderate epidemic of light leaf spot was largely induced by inoculation, since an adjacent trial, which was not inoculated, had approximately one tenth the quantity of light leaf spot.

The first fungicide was not applied until 20 October; two weeks after phoma leaf spot was first seen. Perhaps because they were applied relatively late, fungicides gave poor control of phoma leaf spot. However, they did give effective control of stem canker and stem lesions in the summer. This suggests that although fungicides were unable to protect the leaf from phoma leaf spot, they were able to inhibit spread of the disease down the petiole. A single spray applied in mid December or late January significantly reduced the incidence and severity of stem canker and additional sprays gave progressively better control.

A single spray applied in mid December gave good control of light leaf spot, but two sprays were needed to give reliable control on leaves. Not even a four-spray programme was able to prevent the disease spreading to the pods.

A yield increase of 0.2 t/ha is needed to cover the cost of a single fungicide application. The average yield benefit from a single fungicide treatment was 0.38 t/ha and was therefore economically justified. Yield increases from additional treatments were very small and not economically justified. In this season therefore, where moderate disease epidemics coincided with excellent growing and early harvesting conditions, benefits from fungicide treatments were small.

**Boxworth 1999/2000**

A particularly early and severe phoma leaf spot epidemic produced a high incidence of stem canker, with a high severity score. This was in contrast to 1998/99 season which had a late epidemic. The highest yielding treatment showed a yield response of 0.69 t/ha despite moderate canker control. The impact of canker on yield was perhaps less than might have been expected because plants were larger than usual and had reached the 7-leaf stage by early October when phoma increased rapidly. In mid October, the larger leaves were up to 25cm long and by 22 November leaf length was 30-35cm. This reflects good growing conditions and the low plant population. Subsequently, plants had large thick stems and these appeared to tolerate numerous small canker lesions without showing premature ripening. The cultivar Apex has moderate resistance to canker and this may well have reduced the effect of canker on yield compared with more susceptible cultivars.

The first fungicide was applied on 6 October, which was just as phoma leaf spotting increased rapidly. It failed to persist effectively up to 22 November when both 22 October and 3 November treatments were very effective at reducing the severity of phoma leaf spot. This suggests that the early October spray was effective until about early November when heavy spore release must have taken place to generate the numerous lesions which appeared on 22 November. With large plants and high disease pressure, it proved impossible to keep plants free from phoma and this explains the relatively high incidence of stem infections.

All the single sprays produced a yield response, which suggests yield loss occurred over a long period with no clear optimum. The variation between best and worst single sprays was only 0.12 t/ha whereas for two spray treatments it was 0.23 t/ha and for three sprays 0.28 t/ha. The timing of two and three spray treatments therefore requires critical guidance. In this case, treatments 2 and 3 (Oct/Nov) contributed more consistently to yields than treatments 4 and 5 (Jan/Mar). This may not be the case where light leaf spot is more significant.

A yield increase of 0.2 t/ha is needed to cover the cost of a single fungicide application . In this crop, a two spray programme in autumn would have been a sound approach, although a single November spray would have given a lower yield and a better margin over fungicide cost.

This experiment provided new information on the effect of very early epidemics on yield loss in moderately resistant varieties. This contrasts with 1998/99 season which had a relatively late epidemic.

**Rothamsted 1999/2000**

An early, prolonged and severe phoma epidemic developed into a severe stem canker epidemic. Fungicide application at disease onset (5 October) reduced the early phoma epidemic, but had little effect on phoma beyond November, nor on stem canker later in the season. In contrast, fungicide applied four weeks after disease onset (on 3 November) gave good control of the later phoma epidemic and of stem canker. This suggests that, either the early phase of the phoma epidemic was less important in developing stem canker, or that the fungicide treatment had curative as well as protective activity and was able to inhibit spread of the disease down the petiole. Fungicide application in December or January was too late to control the phoma epidemic and had little effect against stem canker. However, these later sprays were more effective than the earliest spray when used in combination with the optimum November spray.

Although phoma leaf spot and stem canker were severe this season, and some fungicide treatments gave good control, there were no differences in plant numbers between treatments. This suggests that plants survived the phoma leaf spot epidemic and that stem canker did not kill plants until close to harvest.

Inoculum was applied earlier than in 1998, but less was applied. In previous experiments at Rothamsted, light leaf spot appeared 4 - 8 weeks after inoculum was applied. However, in this season, light leaf spot did not appear until mid-February, 18 weeks after inoculum was applied. This suggests that the inoculum did not initiate a light leaf spot epidemic and that the epidemic that began in February was caused by natural infection. Despite the late appearance of the disease, a moderate light leaf spot epidemic developed on pods, but fungicide treatment gave generally poor and unreliable control. Most spray programmes were completed by 20 January and only treatments 19 and 20 received a spray in February. Most sprays were therefore applied too early to give effective control of the leaf, or subsequent pod epidemics, although four-spray programmes did reduce severity of the pod epidemic by approximately 50%.

Although fungicide treatment gave generally poor control of light leaf spot, there was some correlation between severity of the pod epidemic and yield ($R^2 = 0.52$). However, the correlation between stem canker severity in July and yield was higher ($R^2 = 0.77$) which suggests that yield differences between fungicide treatments can be attributed largely to control of stem canker. Results show that timing of fungicide application for phoma and stem canker control is critical. In this season, the optimum time was one month after the onset of phoma leaf spot. Although a single, well-timed fungicide application was effective against

the disease, two-spray programmes gave more reliable economic benefits, provided the optimum spray was included.

In 1998/99 phoma leaf spot and stem canker were less severe and fungicide programmes had little effect on phoma leaf spot, although they did give some control of stem canker. In contrast, light leaf spot developed earlier in 1998/99 and became more severe on leaves and pods. Fungicides were applied one month later in 19998/99 and therefore coincided more closely with the light leaf spot leaf epidemic, resulting in more effective control of the disease on leaves and pods. In line with the national trend for lower yields in 1999/2000, yield from this season's experiment was approximately 1 t/ha lower than in 1999/98. Despite these differences in disease epidemics and yield, there are similarities between the two seasons. In both seasons application four weeks after disease onset gave most effective control of stem canker, and two-spray programmes gave more reliable control of disease epidemics and better yield responses than a single spray, while 3 or 4-spray programmes gave little additional disease control or yield benefit. However, whereas in 1998/99 a single well-timed spray gave the best economic return, in 1999/2000 two-spray programmes gave better returns.

Thermal time models

The field experiments provided an opportunity to test the thermal time relationships between the appearance of phoma leaf spots and the appearance of cankers. This was estimated to be 1120-1140 degree-days in previous Rothamsted experiments. Calculations were made using the closest comparable incidence of leaf spot and canker from disease assessments as shown in Table 43.

Given the infrequent assessments in the late spring when cankers are likely to appear, the field experiments provide general support for the thermal time model of 1120-1140 degree-days for cankers to appear on Apex. The higher values of 1289 degree-days at Boxworth and 1389 degree-days at Rothamsted can be explained by the relatively high daily temperatures in May so that an over-estimate of 150 degree-days represents an error of less than two weeks in real time. The phoma leaf spot epidemic was late at High Mowthorpe in 1998/1999 and it seems likely that the activity in February was too late to produce cankers pre-harvest. The thermal time model is most useful for predicting early canker epidemics and hence potential for yield loss.

**Table 43.** Estimated thermal time for canker development on cv. Apex after the appearance of phoma leaf spots.

| Site | Year | Date phoma leaf spot recorded | Date canker recorded | Thermal time for canker (degree-days) |
|---|---|---|---|---|
| Boxworth | 1998/1999 | 08.12.98 | 27.05.99 | 1289 |
| High Mowthorpe | 1998/1999 | 16.02.99 | 14.07.99 (no canker) | 1430* |
| Rothamsted | 1998/1999 | 10.11.98 | 26.05.99 | 1389 |
| Boxworth | 1999/2000 | 5.10.99 | 29.03.00 | 1134 |
| High Mowthorpe | 1999/2000 | 02.11.99 | 25.05.00 | 1195 |
| Rothamsted | 1999/2000 | 12.10.99 | 19.04.00 | 1211 |

* No canker detected at this site, calculation shows thermal time available.

Canker severity and yield loss

A summary of canker and yield data from the six field experiments provides a basis for explaining new yield loss data obtained in the project (Table 44).

**Table 44.** Summary of yield and canker data from experiments 1998-2000.

| Site | Year | Canker index (pre-harvest) | | Yield (t/ha) | | Light leaf spot severity |
|---|---|---|---|---|---|---|
| | | Untreated | Treated (lowest) | Untreated | Treated (max.) | |
| Boxworth | 1998/1999 | 1.05 | 0.00 | 4.34 | 4.47 | 1% stem area |
| High Mowthorpe | 1998/1999 | 0.00 | 0.00 | 2.71 | 3.56 | 3% stem area |
| Rothamsted | 1998/1999 | 0.78 | 0.00 | 4.64 | 5.37* | 8% stem area<br>16% pod area |
| Boxworth | 1999/2000 | 2.84 | 0.75 | 3.07 | 3.76* | traces |
| High Mowthorpe | 1999/2000 | 0.74 | 0.00 | 3.32 | 4.35* | 6% stem area<br>23% pod area |
| Rothamsted | 1999/2000 | 2.87 | 0.13 | 3.75 | 4.64* | 18% stem area<br>19% pod area |

* Significant yield increases recorded in these experiments

The canker severity indices show the large variation between years and also the lower severity at High Mowthorpe compared with sites in eastern England. Good control of canker was achieved in all the affected crops, but this required two or more sprays. The largest yield responses were associated with disease control in crops infected with both light leaf spot and canker or with severe canker alone. Slight canker infection (index <1) appeared to have little effect on yield (eg Boxworth 1999) and is acceptable commercially.

# Chapter 5

## General Discussion

Two contrasting seasons were encountered in the years with field experiments and this provided key insights into epidemic development and control. The early phoma epidemic in 1999/2000 gave severe canker at harvest and caused significant yield loss (0.7 t/ha). The late epidemic in 1998/99 gave slight canker infection (index 1) by harvest and had little effect on yield. Monitoring epidemic progress in autumn is therefore a key part of decision making as late phoma epidemics may not justify fungicide inputs. However, even in years with late phoma epidemics, light leaf spot can still cause yield loss and decisions will be led by early diagnosis or risk assessment of light leaf spot. The light leaf spot forecast available under the PASSWORD project on the Internet provides an assessment of risk each autumn.

Early warning of phoma leaf spot is now more realistic following progress made in this and related projects. Maturation of spores on stubble is linked to the number of rain days after harvest or from 1 August - in simple terms wet conditions in August and early September will favour early spore maturation; dry conditions will delay spore production. A fall in temperature to <14°C also appears to be a useful parameter to predict spore release and if combined with daily rain records, could form the basis of local forecasts without the need for spore traps. Further refinement of these criteria should be considered in future work.

Laboratory studies have defined conditions for infection by ascospores (Toscano-Underwood *et al*., 2001) and it should now be possible to link this to spore release predictions and forecast the appearance of phoma leaf spots. Considerable variation (14-28 days) was observed between first spore catches and first signs of phoma leaf spot which might reflect the sensitivity of spore traps or test crops. However, the contribution of temperature and wetness factors to this variation could now be examined and developed into a crop-based forecast.

The results from field experiments are consistent with the sequence of events from phoma leaf spotting to canker development described by Hammond & Lewis (1986). The appearance of canker can be estimated using thermal time for Apex and data for other cultivars may prove to be a useful measure of their resistance to canker. Once cankers appear, they become progressively more severe over time. Early appearance of cankers at or before flowering usually results in moderate or severe cankers by harvest and these cankers result in yield loss. Decisions to control canker therefore need to be made when leaf spotting likely to produce damaging cankers is developing. Fungicide treatments tend to delay the development of canker rather than affect its subsequent rate of development. Reducing canker severity index to <1 has resulted in yield increases, but it may not be economically justified to reduce canker severity indices further.

Examination of crops at the seed green-brown stage should provide a good indication of the effectiveness of canker control strategies. The presence of canker lesions of index >2 (more than half the stem circumference affected) indicates that some yield has been lost and programmes could be improved.

Yield loss was related to the severity of canker pre-harvest plus light leaf spot severity at some sites. The examples from 1999 and 2000 form good reference epidemics against which future spray decisions might be judged. There is, however, a need to consider both the growth stage and the size of the plants at which phoma develops. In autumn 1999, plants at Boxworth were very large by late autumn (leaves 25-30 cm long) and this would delay stem colonisation and canker development because the canker fungus had a long distance to grow down the petiole. This may well explain why the best single timings for yield were relatively late (January or February) when phoma leaf spotting started in early October. At Rothamsted, a December spray was most effective though this is confounded by the presence of light leaf spot in the crop.

Fungicide programmes gave good control of canker but this required multiple applications were required to achieve this. Single sprays gave variable responses and two or more sprays gave much more consistent yield responses. This is exemplified by Rothamsted results which showed mean responses of 0.02 t/ha to single sprays and 0.51 t/ha to two-spray treatments in 2000. Spray timing is still an issue as differences between two-spray programmes at Rothamsted in 2000 varied by 0.50 t/ha. Single sprays varied by 0.32 t/ha (0.06-0.38) at High Mowthorpe in 2000 and such differences clearly have a large impact on profitability.

Arguably the control of light leaf spot was less than might have been expected from programmes with up to four sprays. There is growing concern that repeated use of azole fungicides could lead to a build up of resistant strains of the fungal pathogens causing light leaf spot and canker. Monitoring of the current situation in light leaf spot has started in Scotland with HGCA funding in 2001 and this may need to be extended to England if insensitive isolates are recorded.

There were only small effects on yield from delaying the first or last spray treatment. Some caution is needed over the interpretation of the delayed sprays because all were used in conjunction with three other treatments. Data from the single spray timings(discussed above) clearly indicates that delaying sprays can affect yield response. It appears that first sprays do not need to be timed closely to the onset of epidemics. Thresholds for optimal response, however, require further investigation as the contribution of light leaf spot control was a major factor and may have masked the optimum response to phoma control. Timing is also likely to be influenced by temperature, plant growth stage, leaf size and cultivar resistance and such factors should be integrated in future.

There have been major problems applying fungicides in autumn and winter 2000/2001 when ground conditions in addition to wind, rain, frost and snow prevented access to crops. Under these conditions, the value of resistant cultivars should be greater than usual particularly as light leaf spot has increased significantly since spring 2000. An alternative strategy may be the use of systemic seed treatments, which protect young plants against early phoma and light leaf spot. Further work on seed treatments should be considered to establish the degree of protection which can be provided.

Yield loss from canker reached 0.7 t/ha at Boxworth and this provides a reasonable estimate for potential losses on moderately resistant cultivars at high risk sites. This is comparable to previous estimates on cv. Envol (Sansford *et al.,* 1996). If phoma leaf spot develops when plants are well grown from November onwards, yield loss is likely to be much smaller (0.1-0.2 t/ha). The largest yield responses were recorded in crops with both canker and light leaf spot and here potential losses are > 1 t/ha. Estimates of potential yield loss are required to develop a budget for fungicide use and for all except late phoma epidemics, a two-spray approach using autumn and winter treatments is likely to be cost-effective. Further work is required to refine inputs on cultivars with high disease resistance.

The data from this project will be used in the development of Decision Support System for pests and diseases of oilseed rape using MAFF SAPPIO LINK funding. The project, known as PASSWORD, aims to develop forecasting of phoma leaf spot and canker and improved guidelines for optimising fungicidal control. Canker and light leaf spot are currently included in PASSWORD and further research will be required to validate the system and to make it comprehensive for other diseases.

**CONCLUSIONS AND IMPLICATIONS FOR LEVY PAYERS**:

The project has generated a considerable number of new insights into phoma leaf spot and canker epidemics and their control. The data were generated from a small number of sites in only two years of field experimentation. The findings now need to be validated against a wider range of sites, cultivars and seasons. It is clear that variation in the timing of disease epidemics has a major effect on the risk of yield loss and therefore the inputs which can be justified. The major factors to consider are as follows:

Early epidemics of phoma leaf spot pose the greatest threat to yield as these are most likely to produce moderate or severe cankers by harvest.

The number of rain days after harvest provides an indication of first spore release and thereby first signs of phoma leaf spot.

Late epidemics (from late November onwards) of phoma leaf spot may have a small effect on yield on moderately resistant varieties and their control may require only a single fungicide spray.

When phoma develops early in autumn, single sprays showed very variable and inconsistent yield responses and two spray programmes appear to be more reliable and hence more cost-effective.

Phoma leaf spot epidemics are extremely variable from crop to crop and from year to year and weekly monitoring in autumn is required to establish the pattern each year.

Fungicide use should be mainly targeted at the leaf spotting in autumn, leaving spring timings for growth regulation, canopy management and light leaf spot on pods.

There is some flexibility in spray timing particularly when plants have large leaves and some resistance to canker.

In the north, controlling the combination of light leaf spot and canker is worthwhile, despite the lower disease pressure from canker.

Winter oilseed rape crops should be inspected at least weekly in autumn to determine the proportion of plants with phoma leaf spot.

A threshold for fungicide treatment of >20% plants affected with phoma leaf spot is suggested for susceptible and moderately resistant varieties.

**Publications related to OSCOR project**

Scientific papers and Conference proceedings


Biddulph, J.E., Fitt, B.D.L. & Welham, S.J. (1998). Effects of temperature and wetness duration on infection of oilseed rape leaves by ascospores of *Leptosphaeria maculans* (stem canker). Abstract 2.5.13 , 7th International Congress of Plant Pathology, Edinburgh, August 1998.

Biddulph, J.E., Fitt, B.D.L., Gladders, P., Jedryczka, M., West, J.S. & Welham, S.J. (1999). Effects of temperature and wetness duration on infection of oilseed rape by ascospores of A-group or B-group *Leptosphaeria maculans* (stem canker). *Proceedings of the 10th International Rapeseed Congress September 26-29 Canberra, Australia [CD-ROM]*. 5pp. *IRC Organising Committee, Wagga Wagga Agricultural Institute, Wagga Wagga, Australia.*

Biddulph, J.E., Fitt, B.D.L., Gladders, P., Jedryczka, M., West, J.S. & Welham, S.J. (1999). Conditions for infection of oilseed rape leaves by ascospores of UK (A group) and Polish (B group) *Leptosphaeria maculans* (stem canker). *Groupe Consultatif International de Recherche sur le Colza Bulletin* **16**, 82-83.

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.

Fitt, B.D.L., Gladders, P., Sutherland, K.G., Turner, J.A. & Welham, S.J. (1999). Epidemiology, forecasting and management of winter oilseed rape diseases in the UK. *Proceedings of the 10th International Rapeseed Congress, September 26-29 Canberra, Australia [CD-ROM]*. 10pp. *IRC Organising Committee, Wagga Wagga Agricultural Institute, Wagga Wagga, Australia.*

Gladders, P., Fitt, B.D.L. & Turner, J.A. (1999). Improving strategies to control stem canker in the UK. *Proceedings of the 10th International Rapeseed Congress, September 26-29 Canberra, Australia [CD-ROM]*. 5pp. *IRC Organising Committee, Wagga Wagga Agricultural Institute, Wagga Wagga, Australia.*

Jedryczka, M., Dakowska, S., West, J.S. & Fitt, B.D.L. (1999) The influence of wetness and temperature on the release of ascospores of *Leptosphaeria maculans* (blackleg) from oilseed rape debris. Materia y z Sympozjum Naukowego Polskiego Towarzystwa Fitopatologicznego "Bioroznorodnosc w fitopatologii europejskiej na przelomie wiekow", Poznan, 7-9 wrzesien 1999: 75.

Jedryczka, M., Fitt, B.D.L., Kachlicki, P., Lewartowska, E., Balesdent, M.H., Rouxel, T. (1999). A comparison between Polish and UK isolates of *Leptosphaeria maculans*, cause of stem canker of winter oilseed rape. *Zeitshrift fur Pflanzenkrankheiten und Pflanzenschutz* **106**, 608-617.

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.

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., Leech, P. & Fitt, B.D.L. (1998). The effects of rainfall on numbers of air-borne ascospores of *Leptosphaeria maculans* and the development of phoma leaf lesions on winter oilseed rape. Abstract 2.1.7 , 7th International Congress of Plant Pathology, Edinburgh, August 1998.

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., 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., Evans, N., Leech, P.K., Fitt, B.D.L., Welham, S.J., Jedryczka, M. & Penaud, A. (2000). Predicting leaf infection by *Leptosphaeria maculans* on winter oilseed rape. *International Organisation for Biological Control Bulletin* **23,** 23-27.

West, J.S., Evans, N, Leech, P.K., & Fitt, B.D.L. (1999) The effect of climate on the release of ascospores of *Leptosphaeria maculans* (Stem Canker of Oilseed Rape). *Proceedings of the IX Conference "Microscopic Fungi – Plant Pathogens and their Metabolites" 29 April, 1999, Poznan, Poland* (Abstract).

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.

West, J.S., Leech, P.K., Fitt, B.D.L., Welham, S.J., Penaud, A. Peres, A. & Poisson, B. (1999). Optimising the use of fungicides to control stem canker of oilseed rape. *Proceedings of the 10th International Rapeseed Congress, September 26-29 Canberra, Australia [CD-ROM]*. 5pp. *IRC Organising Committee, Wagga Wagga Agricultural Institute, Wagga Wagga, Australia.*

Williams, R.H. & Fitt, B.D.L. (1999). Differentiating A. and B. groups of *Leptosphaeria maculans*, causal agent of stem canker of winter oilseed rape in the UK – a review. *Plant Pathology* **46**, 161-175.

Williams, R.H., & Fitt, B.D.L. (1998). Application of molecular methods to the fore casting of severe stem canker epidemics in winter oilseed rape in the UK. Abstract 3.3.34, 7th International Congress of Plant Pathology, Edinburgh, August 1998.

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.

Zhou, Y., Fitt, B.D.L., Welham, S.J., Evans, N. & Gladders, P. (2000). Effects of stem canker (*Leptosphaeria maculans*) and light leaf spot (*Pyrenopeziza brassicae*) on yield of winter oilseed rape *Brassica napus*) in southern England. *Plant Pathology* **49**, 487-497.

Zhou, Y., Fitt, B.D.L., Welham, S.J., Gladders, P., Sansford, C.E. & West, J.S. (1999). Yield loss of winter oilseed rape in relation to severity of stem canker (*Leptosphaeria maculans* ) in the UK. *Proceedings of the 10th International Rapeseed Congress, September 26-29 Canberra, Australia [CD-ROM]*. 5pp. *IRC Organising Committee, Wagga Wagga Agricultural Institute, Wagga Wagga, Australia.*

## Press articles and other publicity

Demonstration plots (treated and untreated comparison) were included in the HGCA area for Cereals 99 at Wendy, near Royston and the project was promoted at the event , 16-17 June 1999.

Publicity during the project was given via ADAS Crop Action Notes, Technical updates to ADAS Advisers and Crop Centre members as the season progresses. These provide information for Farmers Weekly Arable reports and other press features.

References to phoma leaf spot control were included in HGCA ' Crop Management for Oilseeds'

Talks were presented at three HGCA Roadshows (at Wye, Chelmsford and Newbury) in October 1999 and a summary paper was prepared for the Roadshow Results Booklet.

Canker control has been given a high profile through the Agricultural media, ADAS Crop Action notes and at farmer meetings and technical briefings. This has been supported by editorial and advertorial coverage by Industry partners especially DuPont and Syngenta (formerly Novartis) in autumn 1999 and 2000.

## Acknowledgements

The support and technical contributions from Mr A Selley, DuPont (UK) Ltd and Mr C Mills of Novartis Crop Protection UK Ltd (now Syngenta) is gratefully acknowledged. We thank colleagues particularly S McDonough, A Naik and G Denton for their contributions to the field experiments and A D Todd for all the statistical analyses.

## References


Barbetti, M.J. & Khangura, R.K. (1997). Developments for better management of blackleg disease in Western Austalia. *Proceedings of the 11th Australian research assembly on brassicas*, Perth, Western Australia, 1997, pp.11-14.

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.

Fitt, B.D.L., Gladders, P., Turner, J.A., Sutherland, K.G., Welham, S.J. & Davies, J.M.Ll. (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* **48**, Optimising pesticide applications, 135-142.

Gladders, P. & Musa, T.M. (1980). Observations on the epidemiology of *Leptosphaeria maculans* stem canker in 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. *IOBC/WPRS Bulletin* **18**, 1-11.

Gladders, P., Turner, J.A., Walters, K.F.A. & Wright, D.M. (1997). Winter oilseed rape: survey of pests and diseases, 1995/96. ADAS/CSL Research and Development. 42pp.

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.

Penaud, A. (1995). Studies on the optimum timing to control rapeseed blackleg caused by *Leptosphaeria maculans* with fungicides. *IOBC/WPRS Bulletin* **18**, 56-61.

Pérès, A. & Poisson, B. (1997). Phoma du colza: avancées en épidémilogie. *CETIOM - Oléoscope* **40**, 37-40.

Poisson, B. & Pérès, A. (1999). Study of rapeseed susceptibility to primary contamination of *Leptosphaeria maculans* in relation to plant vegetative stage. Proceedings of the 10th International Rapeseed Congress, September 26-29, 1999, Canberra, Australia.

Sansford, C.E., Fitt, B.D.L., Gladders, P., Lockley, K.D. & Sutherland, K.G. (1996). Oilseed rape: disease development, forecasting and yield loss relationships. HGCA Project Report OS17. 185pp.

Su, H., Fitt, B.D.L., Gladders, P., Hardwick, N.V., Lockley, K.D., Sansford, C.E., Sutherland, K.G. & Welham, S.J. (1997). Estimating yield loss from light leaf spot (*Pyrenopeziza brassicae*) on winter oilseed rape. *Group Consultative International de Recherche sur le Colza Bulletin* **13**, 72-74.

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.

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., 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.

## Appendices

### Appendix A. Site details and agronomic inputs of experimental crops

**Site details, ADAS Boxworth 1998/99**

| | | | |
|---|---|---|---|
| Field name: | Side Hill | | |
| Soil texture: | Clay | | |
| Drainage: | Good | | |
| Soil analysis: (1.99) | pH | : | 8.2 |
| | P mg/l (index) | : | 21 (2) |
| | K mg/l (index) | : | 283 (3) |
| | Mg mg/l (index) | : | 111 (3) |
| | OM% | : | n/d |
| Previous cropping: | 1995 Winter Wheat | | |
| | 1996 Spring Beans | | |
| | 1997 Winter Wheat | | |
| | 1998 Winter Wheat | | |
| Previous cultivation: | 23/8/98 Disced | | |
| | 26/8/98 Flat-lifted | | |
| | 26/8/98 Rolled | | |
| | 29/8/98 Power harrowed | | |
| | 1/9/98 Roll | | |
| | Cultivar | : | Apex |
| | Sowing date | : | 3/9/98 (farm crop sown 2/9/98) |
| | Seed rate (kg/ha) | : | 7.3 (target 70 plants/$m^2$, seeds sown 100/$m^2$) |
| | Fertiliser (kg/ha) | : | 6/11/98 40 kg N/ha (Nitram 34.5% 116 kg/ha |
| | | | 15/3/98 27 kg N/ha (“ 80 kg/ha |
| | | | 19/3/98 27 kg N/ha ( “ 80 kg/ha |
| | | | 07/4/98 17 kg N/ha (“ 50 kg/ha |
| Herbicides: | 15/10/98 Flusilade (0.25 l/ha) + Butisan S (2.0 l/ha) + Comulin (1.0 l/ha) | | |
| | 23/10/98 Benazalox (0.75 l/ha) | | |
| | 24/2/99 Fortrol (0.75 l/ha) | | |
| | 25/2/99 Benazalox 1 kg/ha | | |
| Fungicides: | Only treatment fungicides applied. Farm crop received Punch C (0.4 l/ha) on 23/10/98 and 15/3/99 | | |
| Molluscicides | 26/9/98 Mini slug pellets (7.5 kg/ha) | | |
| Insecticides: | 15/190/98 Cypermethrin (0.25 l/ha) tank mix with herbicides | | |
| Desiccant: | 09/7/99 Diquat (+ Non-ionic wetter 1l/ha) 3.0 l/ha in 1000l water/ha | | |
| Harvest date: | 18 July 1999 | | |

**Appendix A.**

**Site details, High Mowthorpe 1998/99.**

| | | |
|---|---|---|
| Field name: | Elbow North | |
| Soil texture: | SZL | |
| Soil Series: | Panholes | |
| Drainage: | Good | |
| Soil analysis: (1.99) | pH | : n/a |
| | P mg/l (index) | : n/a |
| | K mg/l (index) | : n/a |
| | Mg mg/l (index) | : n/a |
| Previous cropping: | 1995 Winter Barley | |
| | 1996 Grass/Linseed | |
| | 1997 Grass | |
| | 1998 Grass | |
| | Cultivar | Apex |
| | Sowing date | 7/9/98 |
| | Fertiliser (kg/ha) | 30kg/ha N (86.9kg/ha Extran Product) on 10/11/98 |
| | | 85kg/ha N (246kg/ha Extran Product) on 22/2/99 |
| | | 80kg/ha N (232kg/ha Extran Product) on 24/3/99 |
| Herbicides: | Katamaran @ 2.0l/ha | 19/10/98 |
| Fungicides: | as per trial protocol requirements | |
| Molluscicides | Draza @ 2.75kg/ha on 2/10/98 | |
| | Draza @ 5.5kg/ha on 21/9/98 | |
| Insecticides: | Decis @ 0.25l/ha on 26/9/99 | |
| Desiccant: | Gramoxone 100 @ 3.0l/ha on 27/8/98 | |
| Harvest date: | 29 July 1999 | |

**Appendix A.**
**Site details, Rothamsted 1998/99**

| | |
|---|---|
| Field name: | Great Knott I |
| Previous cropping: | 1996 Spring beans<br>1997 Winter oats<br>1998 Spring barley<br>1999 Winter oilseed rape |
| Previous cultivation: | 13/8/98 Ploughed and furrow pressed<br>21/8/98 Rolled (after sowing) |
| | Cultivar<br>Sowing date : 25/8/98 (seed treated with Lindex Plus FS)<br>Seed rate (seeds/$m^2$) : 120 |
| Fertiliser (kg/ha) | :24/9/98 5%N 120kg/ha)<br>8/2/99 5%N (300kg/ha) |
| Herbicides: | 17/9/98 Katarmaran (2.0 l/ha) post sowing<br>6/11/98 Laser (0.5l/ha) |
| Fungicides: | No fungicides applied to farm crop, experimental treatments applied to plots |
| Molluscicides | 29/8/98 Doff Agricultural Slug Killer (7.5kg/ha)<br>16/9/98 Hardy (7.5kg/ha) |
| Insecticides: | 13/11/98 Steffes Cypermethrin 2 (250ml/ha)<br>30/4/99 Hallmark (100ml/ha) |
| Desiccant: | 9/7/99 Glyphosate 3.0 l/ha in 400l water/ha |
| Harvest date: | 17 July 1999 |

**Appendix A.**
**Site details : ADAS Boxworth 1999/2000**

| | | | |
|---|---|---|---|
| Field name: | Whitepits | | |
| Soil texture: | Clay | | |
| Drainage: | Good | | |
| Soil analysis: (2.00) | pH | : | 8.0 |
| | P mg/l (index) | : | 26 (3) |
| | K mg/l (index) | : | 297 (3) |
| | Mg mg/l (index) | : | 107 (3) |
| Previous cropping: | 1996 Winter Oilseed rape | | |
| | 1997 Winter Oilseed rape | | |
| | 1998 Winter Oilseed rape | | |
| | 1999 Winter Oilseed rape | | |
| Previous cultivation: | 29/7/99 Ploughed | | |
| | 31/7/99 Heavy disc Flat-lifted | | |
| | 3/8/99 Power harrowed | | |
| | 26/8/99 Power harrowed and rolled | | |
| | Cultivar | : | Apex |
| | Sowing date | : | 26/8/99 (farm crop sown 26/8/99) |
| | Seed rate (kg/ha) | : | 6.5 |
| | Fertiliser (kg/ha) | : | 12/2/00 40 kg N/ha (Nitram 34.5%) |
| | | | 29/3/00 60 kg N/ha (“ ) |
| Herbicides: | 26/8/99 Pdq (2.0 l/ha) | | |
| | 26/8/99 Katarmaran (2.0 l/ha) post sowing | | |
| | 12/10/99 Falcon (0.3 l/ha) + Sprayprover (1.0 l/ha) | | |
| Fungicides: | No fungicides applied to farm crop, experimental treatments applied to plots | | |
| Molluscicides | 10/9/98 Lynx slug pellets (10.0 kg/ha) | | |
| Insecticides: | 27/1/00 Toppel (0.25 l/ha) | | |
| Desiccant: | 03/7/99 Glyphosate 3.0 l/ha in 1000l water/ha | | |
| Harvest date: | 18 July 2000 | | |

**Appendix A.**

**Site details: ADAS High Mowthorpe 1999/2000**

Field name: Wether Plain

Soil series: Andover

Soil type: SZL

Drainage: Excellent

Soil analysis: 21/10/99

| | mg/l | ADAS Index |
|---|---|---|
| pH | 7.2 | |
| P | 17 | 2 |
| K | 120 | 1 |
| Mg | 52 | 2 |

Previous cropping: 1999 Winter Barley
1998 Winter Wheat
1997 SOSR/Potatoes

Cultivations

Cultivar: Apex
Sowing date: 31/8/99

Fertiliser: Top dressing (kg/ha) Extran 65 kg/ha N 3/3/00
30 kg/ha N 16/3/00
70 kg/ha $SO_3$ 16/3/00
120 kg/ha N 29/3/00

| | Product | Rate | Date applied |
|---|---|---|---|
| Fungicide (overall) | n/a | | |
| Trace elements: | n/a | | |
| Herbicide: | Katamaran | 2 l/ha | 12/9/99 |
| | Falcon | 0.5 l/ha | 8/11/99 |
| Insecticide: | Decis | 0.25 l/ha | 12/9/99 |
| | Hallmark | 75ml/ha | 20/5/00 |
| Molluscicide: | Draza | 15 kg/ha | 9/9/00 |
| Plant growth regulator: | n/a | | |
| Harvest method | Swathed | | |
| Desiccant (rate/volume water) | n/a | | |
| Harvest date: | 6/8/00 | | |

**Appendix A.**
**Site details, Rothamsted 1999/2000**

| | |
|---|---|
| Field name: | Great Knott II |
| Previous cropping: | 1996 winter oilseed rape<br>1997 winter wheat<br>1998 peas<br>1999 set aside |
| Previous cultivation: | 19/7/99 Ploughed and furrow pressed<br>26/7/99 Rolled<br>5/8/99 Rotary harrowed<br>28/8/99 Rolled (after sowing) |
| Cultivar: | Apex |
| Sowing date: | 27/8/99 (seed treated with Lindex Plus FS) |
| Seed rate (seeds/$m^2$): | 120 |
| Fertiliser (kg/ha): | 20/10/99 5%N (87 kg/ha)<br>8/2/99 5%N (300kg/ha)<br>9/2/00 Sulphan 305N, 7.6%S (433kg/ha)<br>15/3/00 Sulphan 305N, 7.6%S (433kg/ha) |
| Herbicides: | 29/8/99 Scythe (2l/ha)<br>29/8/99 Alpha Trifluralin 48 EC (2l/ha)<br>29/8/99 Katamaran ( 2l/ha) |
| Fungicides: | No fungicides applied to farm crop, experimental treatments applied to plots |
| Molluscicides | 30/8/99 PBI slug pellets (8kg/ha)<br>8/10/99 Genesis (5kg/ha) |
| Insecticides: | 14/10/99 Hallmark (100ml/ha)<br>15/5/00 Hallmark Zeon (75ml/ha) |
| Desiccant: | 12/7/00 Glyphosate 3.0 l/ha in 400l water/ha |
| Harvest date: | 20 July 2000 |

**Appendix B Sprayer and spray application details**

**Boxworth 1998/1999**

| Date treated | Growth stage | Temperature ( $^0$C ) | Wind ( kph ) | Crop condition |
|---|---|---|---|---|
| 20.10.98 | 1,05 | 8, RH 90%<br>sunny, cool | 0.5 | dry |
| 04. 11.98 | 1,06 | 3.3, RH 90%<br>cool, overcast | 1.2-2.0 | wet, after frost |
| 11.11.98 | 1,06 | 5.5, RH 98%<br>cool, damp | <1.2 | damp |
| 03.12.98 | 1,06 | 1.3, RH 94%<br>cool, overcast | <1.2 | damp |
| 21.01.99 | 1, | 1, RH 100%<br>cool, damp, sunny | <1.2 | wet |
| 09.03.99 | 2,01, 3,01 | 3.9, RH 94%<br>cool, overcast | 1.2-2.0 | wet |
| 25.03.99 | 3,03-3,05<br>(30 cm tall) | 11, RH 82%<br>sunny, warm | 1.2-2.0 | damp |
| 08.04.99 | 4,01<br>(60-70 cm tall) | 13, RH 71%<br>sunny, warm | 1.2-2.0 | dry |

* Treatments were applied using OPS sprayer in 225 l/ha with 03-F110 nozzles operated at 2.2 bars

**ADAS High Mowthorpe 1998/1999**

| Spray Timing | Date treated | Growth stage | Temperature ( $^0$C ) | Wind ( kph ) | Crop condition |
|---|---|---|---|---|---|
| 1st Timing | 02.12.98 | 1,4 | 1.7 min 4.7 max | n/d | Damp |
| 2nd Timing | 01.02.99 | 1,10 | 5.4 min 8.3 max | n/d | Damp |
| 3rd Timing | 24.02.99 | 1,14 | -1.7 min 4.7 max | n/d | Wet |
| 4th Timing | 16.04.99 | 2,4 | 0.7 min 5.9 max | n/d | Damp |
| 2 weeks after | 21.04.99 | 3.01 | 5.1 min 13.7 max | n/d | Damp |

Treatments were applied using OPS sprayer in 200 l/ha with 03-F110 nozzles operated at 200 kPa

**Appendix B Sprayer and spray application details**

**Rothamsted 1998/1999**

| Date treated | Temperature (°C) | Relative humidity (%) | Wind (kph) |
|---|---|---|---|
| 6.11.98 | 7.5 | 80 | 4.8 |
| 19.11.98 | 5.7 | 80 | <2 |
| 3.12.98 | 1.0 | 80 | <2 |
| 16.12.98 | 7.0 | 96 | 2.5 |
| 21.1.99 | 2.6 | 95 | <2 |
| 25.2.99 | 0.1 | 82 | 4.8 |
| 10.3.99 | 1.8 | 94 | 4.8 |
| 25.3.99 | No record | No record | 4.8 |

Treatments were applied with a hand-held sprayer in 220 l/ha water

**Boxworth 1999/2000**

| Date treated | Growth stage | Temperature ( $^0$C ) | Wind ( kph ) | Crop condition |
|---|---|---|---|---|
| 06.10.99 | 1,05 | 14, RH 90%<br>sunny, cool | 0.5 | dry |
| 22. 10.99 | 1,10 | 12, RH 90%<br>cool and damp,overcast | 0 | wet |
| 03.11.99 | 1,12-13 | 14, RH 90%<br>cool, overcast | 2-4 | damp |
| 15.11.99 | 1,14 | 9, RH 94%<br>cool and damp, overcast | <1.2 | damp |
| 10.01.00 | 1,16-17 | 10, RH 90%<br>cool and thawing after frost, sunny | 2-4 | wet |
| 22.02.00 | 2,2-2,3 | 8, RH 94%<br>cool and damp, sunny | 1.2-2.0 | damp |
| 13.03.00 | 3,1-3,03 | 14, RH 85%<br>cool, dry, overcast | 1.2-2.0 | dry |
| 20.03.00 | 3,6<br>(70-100 cm tall) | 13, RH 75%<br>cool, dry, sunny | 0 | dry |

* Treatments were applied using OPS sprayer in 225 l/ha with 03-F110 nozzles operated at 220 kPA

**Appendix B Sprayer and spray application details**

**ADAS High Mowthorpe 1999/2000**

| Spray application | Spray date | Growth stage | Temperature (°C) | Wind (kph) | Daily rain (mm) |
|---|---|---|---|---|---|
| 1st Timing | 09.11.99 | 1,7 | 5.5 min, 8.8 max | 0-1 | 0 |
| 2 weeks after T1 | 29.11.99 | 1,7 | 4.4 min 10.0 max | 0-4 | 0.1 |
| 4 weeks after T1 | 13.12.99 | 1,8 | 4.9 min 6.8 max | 0-3 | 2.7 |
| 2nd Timing | 17.01.00 | 1,8 | -4.1 min 2.5 max | 0-1 | 6 |
| 3rd Timing | 22.02.00 | 1,9 | 3.3 min 8.7 max | 1-2 | 3.5 |
| 4th Timing | 31.03.00 | 2,3 | 1.2 min 5.6 max | 0-1 | 0 |
| 2 weeks after T4 | 10.04.00 | 3,3 | 2.3 min 6.4 max | 0-2 | 0.6 |
| 4 weeks after T4 | 28.04.00 | 3,6 | 7.0 min 12.7 max | 1-3 | 0.1 |

**Spray application equipment**
Sprayer: OPS
Nozzles: 03F110
Water volume: 200 litres
Pressure: 200 kPa

**Rothamsted 1999/2000**

| Date treated | Temperature (°C) | Relative humidity (%) | Wind (kph) |
|---|---|---|---|
| 06.10.99 | 4.1 | 89 | <2 |
| 25.10.99 | 8.4 | 96 | <2 |
| 29.10.99 | 13.3 | 89 | 2.5 |
| 03.11.99 | 12.9 | 80 | 2.5 |
| 13.12.99 | Not recorded | Not recorded | 2.5 |
| 20.01.00 | 0.7 | 92 | <2 |
| 03.02.00 | 4.3 | 78 | <2 |
| 18.02.00 | 6.7 | 77 | 2.5 |

Treatments were applied with a hand held sprayer in 220 l/ha water