HGCA

PROJECT REPORT No. OS17

OILSEED RAPE: DISEASE DEVELOPMENT, FORECASTING AND YIELD LOSS RELATIONSHIPS

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OILSEED RAPE: DISEASE DEVELOPMENT, FORECASTING AND YIELD LOSS RELATIONSHIPS

by

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ABSTRACT

A series of fourteen replicated plot scale experiments was established between 1991 and 1994 to determine the effect of disease on the yield of winter oilseed rape and the economic threshold and optimum fungicide timings for disease control. The cultivar Envol was selected for use throughout the experiment as it was a popular cultivar and was moderately susceptible to both canker and light leaf spot.

A mixture of fungicides effective against all major and most minor fungal diseases of oilseed rape (iprodione and thiophanate-methyl @ 167g/l each plus prochloraz @ 450g/l at 1.5 and 0.55 l/ha respectively) was applied to plots of oilseed rape at four-weekly intervals from the beginning of October. The experiment design was planned to allow the differential development of a series of epidemics to occur by the sequential application of multiple sprays of the fungicide mixture to the plots. The first series of sprays all began in the autumn and finished progressively later, continuing until harvest; the second series of sprays all finished at harvest and started progressively earlier.

The range of diseases that developed across the experiment sites was extensive, with all of the major, and most of the minor diseases detectable at some or all of the locations. This report is based upon the three major oilseed rape diseases which generated the most data and which appeared to have the greatest effect on yield.

Leaf spot and canker caused by *Leptosphaeria maculans* developed to damaging levels at ten sites. A strong relationship was found between yield and the incidence of canker at pod ripening at seven of the sites such that for every 1 per cent of stems affected by canker at pod ripening 0.01 t/ha yield loss occurred. Yield losses related to canker were mainly associated with sites where the mean leaf area affected by the foliar phase of the disease in untreated plots was $\geq$ 1.0 per cent before or during January. The disease was controlled by sprays applied between November and February. Economic disease control was associated with sites where foliar severity exceeded 1.0 per cent

before or during January with 34.0 per cent of stems affected by canker at pod ripening.

Light leaf spot caused by *Pyrenopeziza brassicae* became damaging at nine sites and appeared to cause twice as much yield loss as canker. For every 1 per cent of stems affected by light leaf spot at pod ripening, 0.019 t/ha yield loss occurred. Yield losses related to light leaf spot infection of the stem were mainly associated with sites where the mean leaf area affected in untreated plots during the season on at least one assessment date was $\geq$ 13.0 per cent. No relationship existed between pod disease and yield. As with canker, sprays applied between November and February gave good disease control. Economic disease control was associated with sites where foliar disease severity exceeded 13.0 per cent during the season with more than 17.0 per cent of stems affected by light leaf spot at pod ripening.

Sclerotinia stem rot caused by *Sclerotinia sclerotiorum* was only damaging at two sites. At one site the disease was particularly damaging causing a high yield loss of 0.016 and 0.032 t/ha respectively for every 1 per cent of stems or plants with racemes affected at pod ripening. At this site the disease was well controlled on the stems and the racemes by sprays applied at late flowering (GS 4.7 and 4.8). At the second site the disease was less severe and caused a yield loss of 0.01 t/ha for every 1 per cent of stems or plants with racemes affected at pod ripening. Symptoms on the main stem were not well controlled at this site, but significant reductions in the incidence of plants with disease on the racemes occurred as a result of the application of post-flowering sprays in June. Of particular interest was the significant increase in disease on the main stems which resulted from treatments that received their final fungicide applications in early spring, just prior to flowering. This was thought to be related to the possible destruction of beneficial organisms pre-flowering, which may otherwise have helped to prevent infection by *S. sclerotiorum*.

This detailed 3-year study into the effect of disease on yield and the economics of disease control has shown that light leaf spot, canker, and sclerotinia stem rot are all damaging diseases reducing seed yield on average by 0.010, 0.019, and 0.016 t/ha respectively for every 1 per cent of stems affected at pod ripening. The optimum timing and number of fungicide sprays to control both light leaf spot and canker has been shown to be between early November and late February with the application of one or two sprays. Although few of the sites were affected by sclerotinia stem rot the data obtained indicated that the disease can be successfully controlled by the application of late or post-flowering sprays. *Sclerotinia* can infect the oilseed rape crop at any time during the flowering period, hence the traditional early to mid-flowering treatment. Judgement on spray timings for this disease should in practice be based on an assessment of risk derived from information on cropping and disease history together with weather data and petal culturing to detect ascospore inoculum. Hence, this unusual result has shown that it is possible to delay treatment for *Sclerotinia* until a late stage in the development of the crop, but this approach should not be normally adopted unless more information is available to support it.

Prior to this study, little or no data were available on the effect of disease on double-low cultivars. With respect to light leaf spot there was no evidence that light leaf spot infection of the stem had any effect on yield, but it is now clear that stem infection can be extremely damaging. Data on canker and yield were inconsistent and were only available for single-low cultivars such as Jet Neuf, as few disease epidemics had occurred in the UK since double-low cultivars were introduced. This study has produced substantial data on both diseases. The disease-yield loss relationships calculated in this experiment may however be specific to the cultivar Envol, or similar cultivars, since oilseed rape cultivars with different NIAB ratings for disease resistance may suffer greater or less loss in yield for the same amount of disease.

It is now clear that the application of fungicides to winter oilseed rape in the autumn can be beneficial for the control of light leaf spot and canker. However, it was clear

that there is insufficient information on which to base a judgement with respect to the threshold level of either disease at the time when the autumn spray should be applied. It is also clear that where a spring treatment is required, the timing should be brought forward from stem extension, which often occurs in March, to a spray applied to the crop by the end of February.

Further work is required to determine more specifically the exact timing, dose and frequency of fungicide application required within the November to February period for the control of light leaf spot and canker, or the flowering period in relation to the development stage of the crop for sclerotinia stem rot, and to forecasting potential disease development and yield loss, in relation to early levels of disease symptoms, or the detection of infection by non-visual means such as diagnostic kits. In conjunction with meteorological data this information will allow specific judgements to be made for disease control in the winter oilseed rape crop.

OILSEED RAPE: DISEASE DEVELOPMENT, FORECASTING AND YIELD LOSS RELATIONSHIPS

OBJECTIVE OF RESEARCH

To determine the effect of disease on the yield and quality of oilseed rape and to ascertain the economic thresholds for their control.

INTRODUCTION

The oilseed rape crop is affected by a number of important diseases which can be detrimental to yield (Hardwick *et al.* 1991). The severity and incidence of diseases varies from season to season and is influenced by geographical location (ADAS/CSL surveys unpublished, 1984 to 1995). Although adequate fungicidal control methods are available for a number of oilseed rape pathogens, the routine application of fungicides is not justified, particularly following the change of subsidy from a payment on seed to an area payment scheme for crops harvested since 1991, effectively halving the price of rapeseed. The yield response required to cover the cost of fungicide inputs therefore doubled.

Previous work in 1990/91 (Sansford, 1992) at six sites evaluated the effect of differently timed single and multiple sprays of prochloraz (Sportak) and iprodione plus thiophanate-methyl (Compass) on disease control and yield of winter oilseed rape. Light leaf spot (*Pyrenopeziza brassicae*) was the most common disease encountered. Control of this disease was significant in the spring with autumn and early spring applications at two sites. *Phoma* leaf spot (*Leptosphaeria maculans*) developed at all except one site. Sprays applied in mid-March gave the best control of canker. There was no obvious relationship between disease control and yield. At individual sites few treatments would have been profitable when rapeseed prices were approximately £130/t. Prior to the change in rapeseed prices rapeseed was sold at £240/t. At this

value the most economic timing (meaned across the sites) would have been the mid-March application.

Hardwick *et al.* (1991) considered that there were three principle diseases that posed the main threat to the UK crop, namely light leaf spot (*Pyrenopeziza brasssicae*), dark leaf and pod spot (*Alternaria brassicae* and *A. brassicicola*) and canker (*Leptosphaeria maculans*) and made recommendations that high priority be given to studying disease/yield loss relationships for these diseases. Relationships between disease and yield were not well-defined. Control of light leaf spot was achieved by fungicides applied in the autumn and early spring. Dark leaf and pod spot was controlled by fungicide sprays applied at pod ripening. Little information was available on the relationship between canker and yield or on the control of the disease.

This experiment, with its wide range of geographical locations, was designed to determine which diseases are important in affecting yield and to identify the key timings for the application of fungicides for their control. It was anticipated that the design of the experiment would generate much data on the development of diseases and their effect on yield on winter oilseed rape in the UK.

Disease/yield loss relationships and optimum spray timings described in the report were obtained by extrapolation from treatments that had received multiple sprays. As such it must be stressed that to validate this work fully, further investigations based on single or double-spray treatments will be required. Also, at some sites more than one major disease occurred. By examining the relationships between yield and disease it was possible to determine which disease was causing the most damage to yield, but it is possible that some lesser effect on yield was being caused by what appeared to be the least damaging disease. These general disclaimers apply throughout the text.

MATERIALS AND METHODS

Sites

Sites were established at four locations in 1991/92, five in 1992/93 and five in 1993/94. These are listed in Table 1.

Table 1. Site locations

Year	Site	County
1991/92	Foveran	Aberdeenshire
	Kington Langley	Wiltshire
	Rosemaund	Herefordshire
	Rothamsted	Hertfordshire
1992/93	Boxworth	Cambridgeshire
	Pettymuick	Aberdeenshire
	Rothamsted	Hertfordshire
	Tarrant Hinton	Dorset
	Withington	Herefordshire
1993/94	Boxworth	Cambridgeshire
	Rosemaund	Herefordshire
	Rothamsted	Hertfordshire
	Thurloxton	Somerset
	Udny Station	Aberdeenshire

Site history and husbandry

Details of site history are listed in Appendix 1. Site husbandry details are listed in Appendix 2.

Design

The experiment was of a randomised block design with two replicate blocks (design modified from Thomas *et al.*, 1989). The size of the plots differed from site to site, but were within the range 56 to 108 m^2.

Husbandry

Plots were located in commercial crops of winter oilseed rape. All treatments other than fungicides were as per farm practice.

Treatments

Details of fungicides are given in Table 2.

Table 2. Fungicides, active ingredients (a.i.) and dose rates

Fungicide product	**a.i.**	**Amount a.i. in product**	**Dose rate product/ha**
Compass	iprodione	167g/l	1.50l
	+		
	thiophanate-methyl	167g/l	
Sportak 45	prochloraz	450g/l	0.55l

Products were used in a tank-mixture equivalent to half the manufacturer's recommended rate of each product.

Treatments were applied under Experiment Permit and the seed from the treated plots was destroyed after harvest.

Target fungicide application dates for each year of the experiment are given in Table 3.

Table 3. Target fungicide application dates

Treatment	Date (week beginning)											
	1991/2	7/10	4/11	2/12	30/12	27/1	24/2	23/3	20/4	18/5	15/6	13/7
	1992/3	5/10	2/11	30/11	28/12	18/1	22/2	22/3	19/4	17/5	14/6	12/7
	1993/4	27/9	25/10	22/11	20/12	17/1	14/2	14/3	11/4	9/5	6/6	4/7
1		-	-	-	-	-	-	-	-	-	-	-
2		X	-	-	-	-	-	-	-	-	-	-
3		X	X	-	-	-	-	-	-	-	-	-
4		X	X	X	-	-	-	-	-	-	-	-
5		X	X	X	X	-	-	-	-	-	-	-
6		X	X	X	X	X	-	-	-	-	-	-
7		X	X	X	X	X	X	-	-	-	-	-
8		X	X	X	X	X	X	X	-	-	-	-
9		X	X	X	X	X	X	X	X	-	-	-
10		X	X	X	X	X	X	X	X	X	-	-
11		X	X	X	X	X	X	X	X	X	X	-
12		X	X	X	X	X	X	X	X	X	X	X
13		-	-	-	-	-	-	-	-	-	-	X
14		-	-	-	-	-	-	-	-	-	X	X
15		-	-	-	-	-	-	-	-	X	X	X
16		-	-	-	-	-	-	-	X	X	X	X
17		-	-	-	-	-	-	X	X	X	X	X
18		-	-	-	-	-	X	X	X	X	X	X
19		-	-	-	-	X	X	X	X	X	X	X
20		-	-	-	X	X	X	X	X	X	X	X
21		-	-	X	X	X	X	X	X	X	X	X
22		-	X	X	X	X	X	X	X	X	X	X

X = Spray application of half-rate Compass plus Sportak

Fungicide application

The actual dates of treatments varied from site to site depending upon the suitability of spray conditions. Details of spray dates and the development stage of the crop at application in relation to treatment number are presented in Appendix 3.

Assessment dates

The target dates for disease assessment are given in Table 4. These were timed to coincide with the target spray dates.

Table 4. Target disease assessment dates

Treatment	Date											
	1991/2	7/10	4/11	2/12	30/12	27/1	24/2	23/3	24/4	18/5	15/6	13/7	3/8
	1992/3	5/10	2/11	30/11	28/12	18/1	22/2	22/3	19/4	17/5	14/6	12/7	2/8
	1993/4	27/9	25/10	22/11	20/12	17/1	14/2	14/3	11/4	9/5	6/6	4/7	25/7
1		A	A	A	A	A	A	A	A	A	A	A	A
2		-	A	A	A	A	A	A	A	A	A	A	A
3		-	-	A	A	A	A	A	A	A	A	A	A
4		-	-	-	A	A	A	A	A	A	A	A	A
5		-	-	-	-	A	A	A	A	A	A	A	A
6		-	-	-	-	-	A	A	A	A	A	A	A
7		-	-	-	-	-	-	A	A	A	A	A	A
8		-	-	-	-	-	-	-	A	A	A	A	A
9		-	-	-	-	-	-	-	-	A	A	A	A
10		-	-	-	-	-	-	-	-	-	A	A	A
11		-	-	-	-	-	-	-	-	-	-	A	A
12		A	A	A	A	A	A	A	A	A	A	A	A
13		-	-	-	-	-	-	-	-	-	-	A	A
14		-	-	-	-	-	-	-	-	-	A	A	A
15		-	-	-	-	-	-	-	-	A	A	A	A
16		-	-	-	-	-	-	-	A	A	A	A	A
17		-	-	-	-	-	-	A	A	A	A	A	A
18		-	-	-	-	-	A	A	A	A	A	A	A
19		-	-	-	-	A	A	A	A	A	A	A	A
20		-	-	-	A	A	A	A	A	A	A	A	A
21		-	-	A	A	A	A	A	A	A	A	A	A
22		-	A	A	A	A	A	A	A	A	A	A	A

A = 10 to 25 plants/plot assessment

Disease assessment

On each assessment date 10 to 25* plants were taken at random from the plots indicated (25* in the first year of the experiment only). Prior to stem extension, plant samples were incubated for 24 hours in a polyethylene bag at room temperature before laboratory assessment, to encourage the development of symptoms of light leaf spot.

Plants were assessed for the percentage area of leaves, stems and pods affected by each disease. An assessment scale was used to score stems for grey mould, sclerotinia stem rot, and canker as indicated below.

Assessment scale

0	=	no disease
1	=	less than half the stem girdled by lesion
2	=	more than half the stem girdled by lesion
3	=	whole stem girdled by lesion
4	=	plant dead

Growth Stages

Growth stages were recorded using the key produced by Sylvester-Bradley (1985).

Harvest

Plots were harvested and yields corrected to 91% dry matter. Assessments of the degree of ripening and lodging were made as appropriate before harvest.

Statistical analysis

Data were subjected to analysis of variance. Data that were skew were transformed using either the logit or angular transformation. Treatment means were separated using Duncan's Multiple Range Test where the variance ratio was significant ($p \leq 0.05$).

Regression analysis was performed on yield (y) versus disease and ripening data (x) and correlation coefficients were calculated.

Data presentations

The volume of data that was generated was substantial. Individual disease assessments have been archived at ADAS Wolverhampton with the most relevant data presented in this report in graphical form. Individual yield data are also presented in graphical form but can be obtained from ADAS Wolverhampton.

Disease development in untreated plots is plotted by month. The percentage of plants affected is described as disease incidence; disease severity is described as the percentage area of leaf, stem, or pod affected by the disease, or the severity score (as described in the disease assessment section). Each month is represented twice on the x-axis where either more than one assessment was made in the same month, or, if the assessment was made very early or very late in the month then disease values are plotted above the first or second repeat of the month's name respectively. Final disease assessments and yields in treated plots are plotted according to the month in which the treatment ended (Treatments 2 to 12) or began (Treatments 22 to 13); untreated values are quoted in the text for comparison.

Yield responses were also calculated for each treatment and these are represented graphically according to the month in which the treatment ended or began. Where treatments consisted of a single fungicide spray only the yield response of the treated plots was calculated by subtracting the mean yield of the untreated plots from the mean yield of the treatment concerned. Where treatments consisted of more than one spray and commenced in the autumn, finishing progressively later (Treatments 3 to 12) then the effect of the final spray in each treatment was calculated by substracting the yield of a treatment with two or more sprays from the yield of a treatment with one additional spray, thus calculating the effect of the additional spray (ie Treatment 4 minus Treatment 3 = effect of additional final spray in Treatment 4). Where treatments consisted of more than one spray and started progressively later (Treatments 22 to 14) then the effect of the initial spray in each treatment was calculated by substracting the

yields as described above (ie Treatment 22 minus Treatment 21 = effect of additional initial spray in Treatment 22).

Treatment numbers referred to in the text of this report relate to individual sites and the exact number and date of application of fungicide sprays can be found in Appendix 3.

RESULTS

All of the major, and most of the minor diseases of oilseed rape were detected at some or all of the site locations. However the data for diseases caused by *Alternaria brassicae*, *Botrytis cinerea*, *Erysiphe cruciferarum* and *Pseudocercosporella capsellae* were limited. Downy mildew (*Peronospora parasitica*) was common but is known to be of minor importance as its effect on yield is known to be limited. *Phoma* leaf spot and canker (*Leptosphaeria maculans*), light leaf spot (*Pyrenopeziza brassicae*) and sclerotinia stem rot (*Sclerotinia sclerotiorum*) were the most common and severe diseases throughout the experiment and, therefore, the development of these diseases at individual sites and their effect on yield are described below. At some sites both canker and light leaf spot were prevalent and at one site both canker and sclerotinia stem rot prevailed; each disease is dealt with separately in the relevant section. It was possible at some sites to determine which disease was having the greatest effect on yield, but it should be remembered that where two major diseases occurred, some indeterminable yield effect could have arisen from what appeared to be the least damaging disease. The term "significant" implies statistical significance at $p \leq 0.05$ throughout the text.

1. Leaf spot and canker

Leaf spot and canker developed at ten sites during the course of the study. Disease development in untreated plots and the effect of fungicide treatment on disease and yield at affected sites is detailed below. Results are summarised in Section 1.1.

(i) Kington Langley 1991/92

Leaf spot and canker and sclerotinia stem rot developed to high levels at this site. Sclerotinia stem rot is discussed in Section 3(i).

The development of the *Phoma* leaf spot phase of canker in untreated plots is illustrated in Figure 1. Leaf spot was first detected at low levels in late October 1991. The incidence of plants affected reached a maximum of 49 per cent on 2 January but the severity of the disease was extremely low throughout the season reaching a maximum of 0.1 per cent leaf area affected in January and February. The percentage of plants affected by the disease declined to 6 per cent in late February with the loss of older leaves, rose to 35 per cent in late March, but declined thereafter reaching zero in late June.

Canker development in untreated plots is illustrated in Figure 1A. Lesions were detected on both the aerial and basal parts of stems on 11 May towards the end of flowering (GS 4.9/5.7). Both types of lesion continued to develop and at the final assessment on 8 July (GS 6.8) 79 per cent of untreated plants were affected by stem disease; 54 per cent had penetrating aerial lesions and 60 per cent had penetrating basal lesions. However, neither type of lesion was severe (severity score ≤ 1.0) (ie less than half the circumference of the stem was girdled by disease symptoms).

No significant effects of fungicide treatment on the leaf spot phase were found. However, on 14 April (GS 3.6) 26 per cent of untreated plants were affected compared to ≤ 4 per cent in Treatments 4 to 7, 12, and 17 to 22 but this result was not significant. All of these treatments received at least one fungicide application between November and March.

Figure 1B illustrates the final incidence of aerial and basal stem lesions in each treatment on 8 July (GS 6.8). Optimum and significant control of penetrating aerial lesions reducing the incidence from 54 to ≤ 10 per cent was achieved by Treatments 6 to 12, and 18 to 22. The first group of treatments all commenced on 8 October and finished on 2 February or later. The second group started between 29 October and 24 February and finished on 25 June. These results appear to show that sprays applied in February were critical for the control of penetrating aerial lesions.

Canker lesions (basal lesions which penetrated the stem) were significantly and optimally reduced from 60 to ≤ 10 per cent by Treatments 6 to 12 and 20 to 22. These were the same treatments that controlled the aerial lesions with the exception of Treatment 18 and 19 which started in February and ended in June. Thus, control of basal canker lesions was optimum where autumn sprays were applied.

All lesion types were significantly reduced from 79 to ≤ 10 per cent by Treatments 7, 9 to 12, and 20 to 22. It therefore appears that combinations of sprays starting in the autumn and including a late February treatment gave optimal control of *Phoma* lesions on the stem.

The untreated yield at this site was 3.42 t/ha. Significant yield increases were only obtained from Treatments 16, 20 and 21 (0.44, 0.47 and 0.70 t/ha respectively). Figure 1C shows the yields obtained from all of the treatments and Figure 1D shows the responses attributable to individual spray timings, obtained by subtraction of yields from related treatments. The largest response of 0.70 t/ha resulted from a nine-spray programme between November and June (Treatment 21). Trends in yield showed that there appeared to be no relationship between leaf spot or canker and yield despite the high incidence of canker. Regression analysis confirmed that no relationship existed between the incidence or severity of canker in July and the yields obtained. This is likely to be due to the low severity of the leaf spot phase during the season but particularly during the winter when cankers usually start to develop. Low foliar disease severity at this time suggests that insufficient fungal inoculum was available to cause disease of sufficient damage to yield.

Trends in the yields however do suggest that yield responses were related to the control of *Sclerotinia* at this site (Section 3).

FIGURE 1 : KINGTON LANGLEY 1991/92
LEAF SPOT DEVELOPMENT

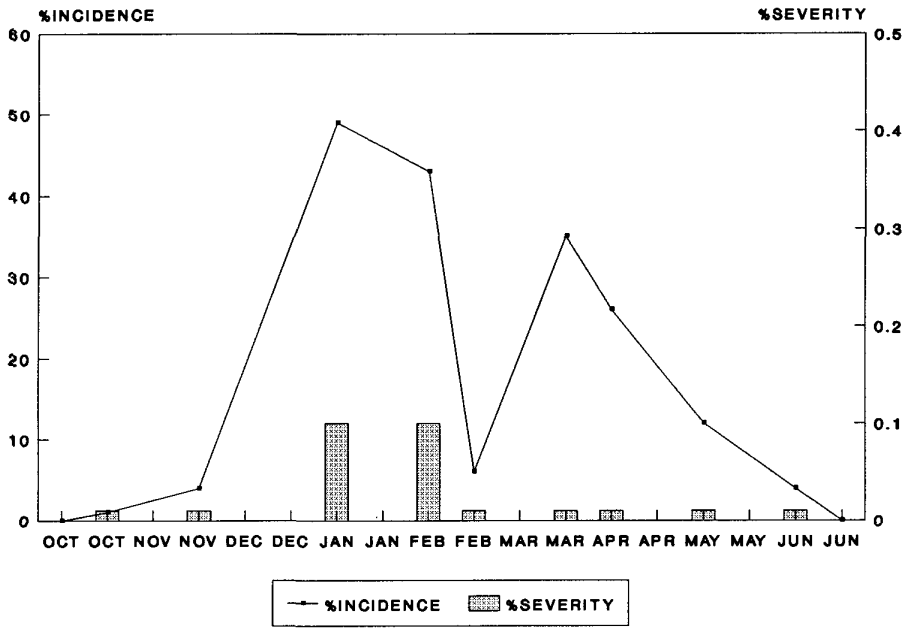


FIGURE 1A : KINGTON LANGLEY 1991/92
CANKER DEVELOPMENT

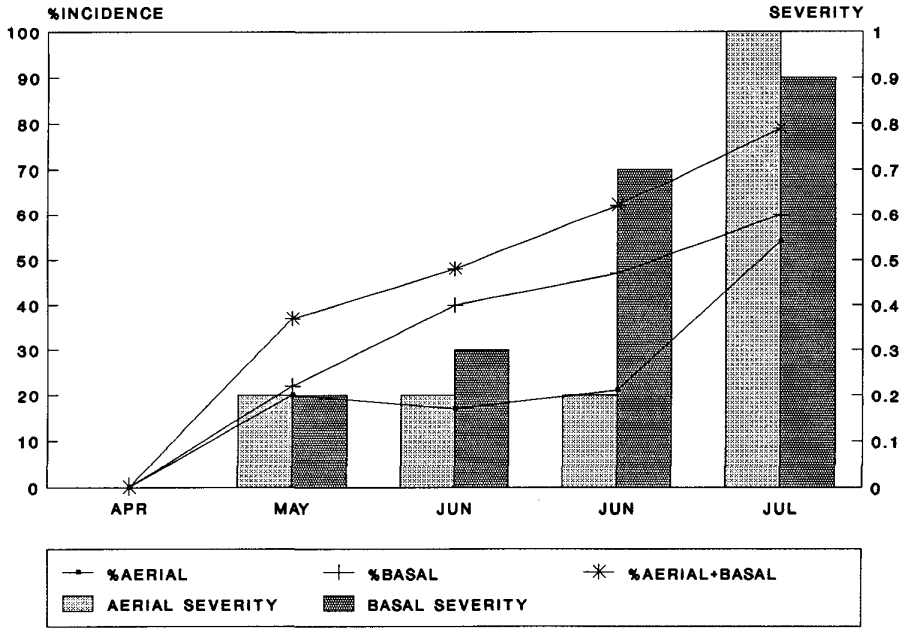


FIGURE 1B : K. LANGLEY CANKER
8 JULY 1992, GS6.8

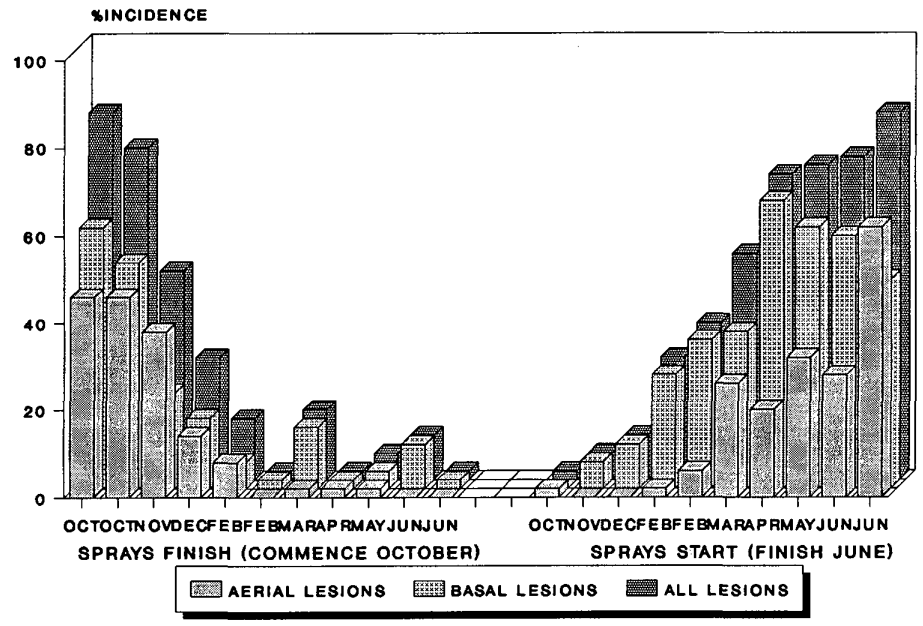
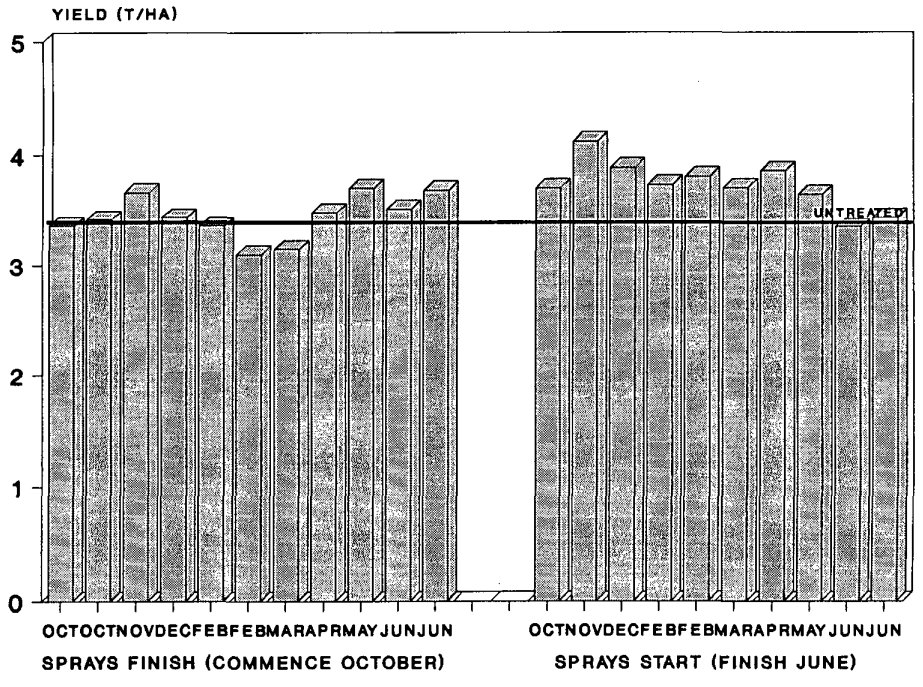


FIGURE 1C : K. LANGLEY YIELD 1992 (T/HA)



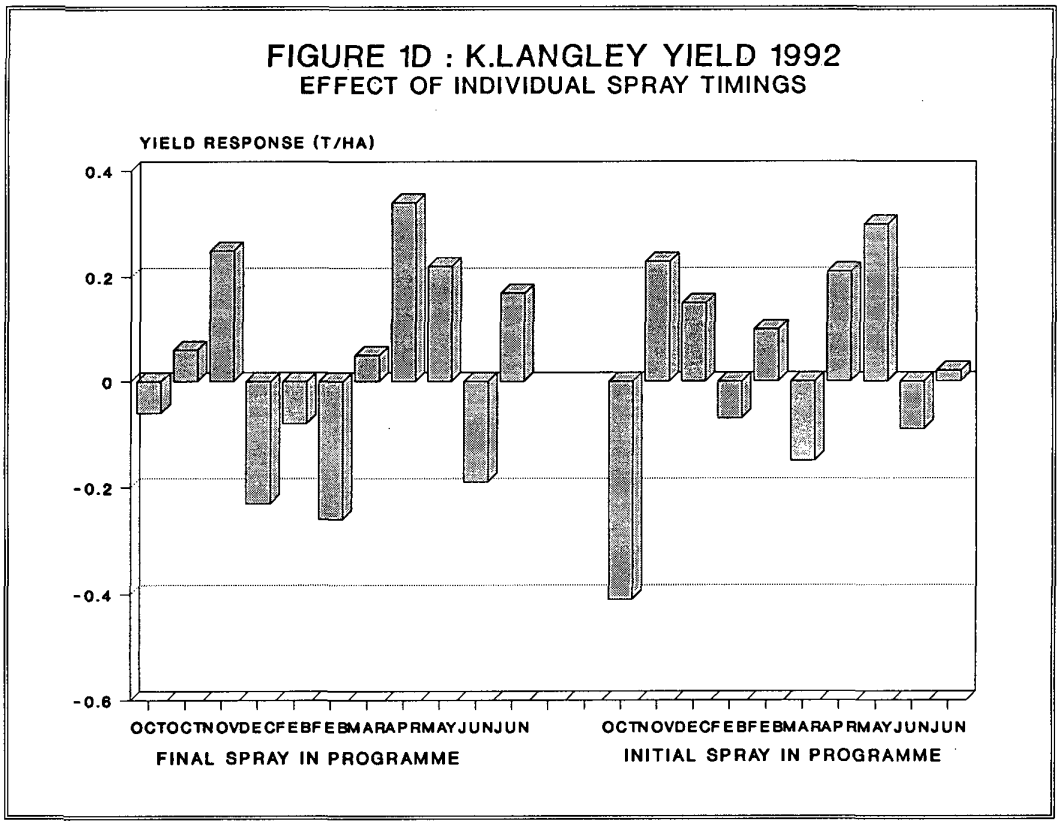


FIGURE 1D : K.LANGLEY YIELD 1992
EFFECT OF INDIVIDUAL SPRAY TIMINGS
YIELD RESPONSE (T/HA)
0.4
0.2
0
-0.2
-0.4
-0.6
OCTOCTNOVDECFEBFEBMARAPRMAYJUNJUN
FINAL SPRAY IN PROGRAMME
OCTNOVDECFEBFEBMARAPRMAYJUNJUN
INITIAL SPRAY IN PROGRAMME

(ii) Rothamsted 1991/92

The development of the *Phoma* leaf spot phase of canker in untreated plots is illustrated in Figure 2. Leaf spot was first detected at low levels in early October 1991. The incidence of plants affected fluctuated, reaching 66 per cent on 6 March, declining thereafter, but rose again finally affecting 96 per cent of plants on 4 June. The disease was not severe, affecting ≤ 0.3 per cent of the leaf area from October until May and 2.5 per cent leaf area on 4 June.

Figure 2A illustrates canker development in untreated plots. Stem symptoms were first seen on 4 June (GS 6.3). By 6 July (GS 6.5), 94 per cent of untreated plants were affected with a mean severity score of 2.5. Figure 2B illustrates the final incidence and severity of canker in each treatment on 6 July (GS 6.5). Significant control of canker was obtained with Treatments 9, 10 and 19 to 22 resulting in reductions to ≤ 42 per cent of plants affected. The best of these (10, 19 to 22) reduced disease incidence to ≤ 30 per cent. Spray timings for these treatments commenced between October and February and finished in June. These were also the most effective treatments to reduce disease severity (≤ 0.42).

The untreated yield at this site was 2.92 t/ha. Figure 2C and 2D show the yields obtained from all of the treatments and the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. None of the treatments resulted in a significant yield response, but of the treatments that commenced in October and finished between January and June (5 to 10) there was a tendency for yields to increase with the addition of further spray timings, such that Treatment 10 (nine sprays) gave the greatest response (0.60 t/ha). Treatment 20 received six sprays between January and June and also led to a response of 0.60 t/ha.

Despite the severity and high incidence of canker at this site there was no obvious relationship between yield and the final levels of disease in July. This was confirmed by regression analysis. As with Kington Langley this was probably related to the low severity of the leaf spot phase throughout the season, especially during the winter.

FIGURE 2 : ROTHAMSTED 1991/92
LEAF SPOT DEVELOPMENT

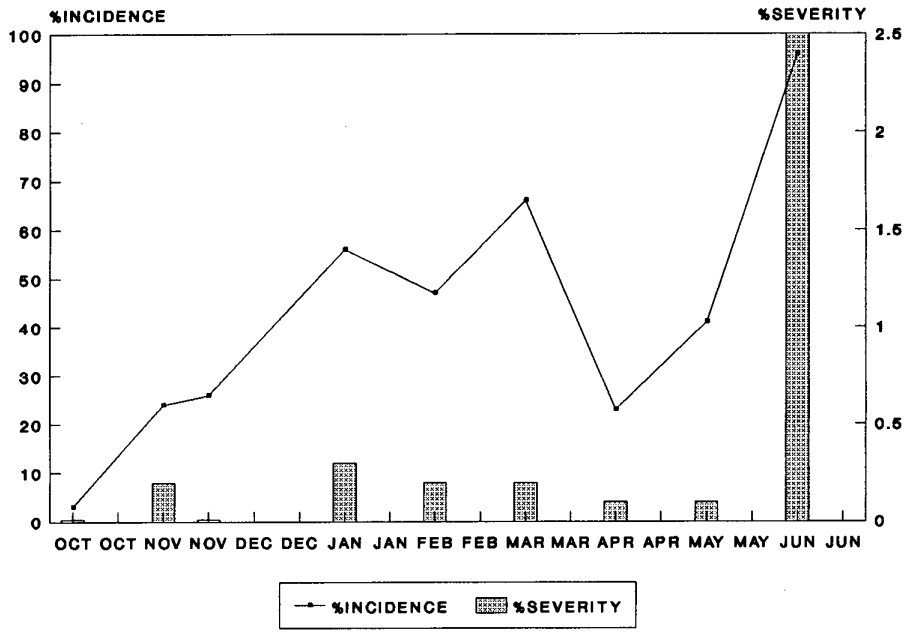


FIGURE 2A : ROTHAMSTED 1991/92
CANKER DEVELOPMENT

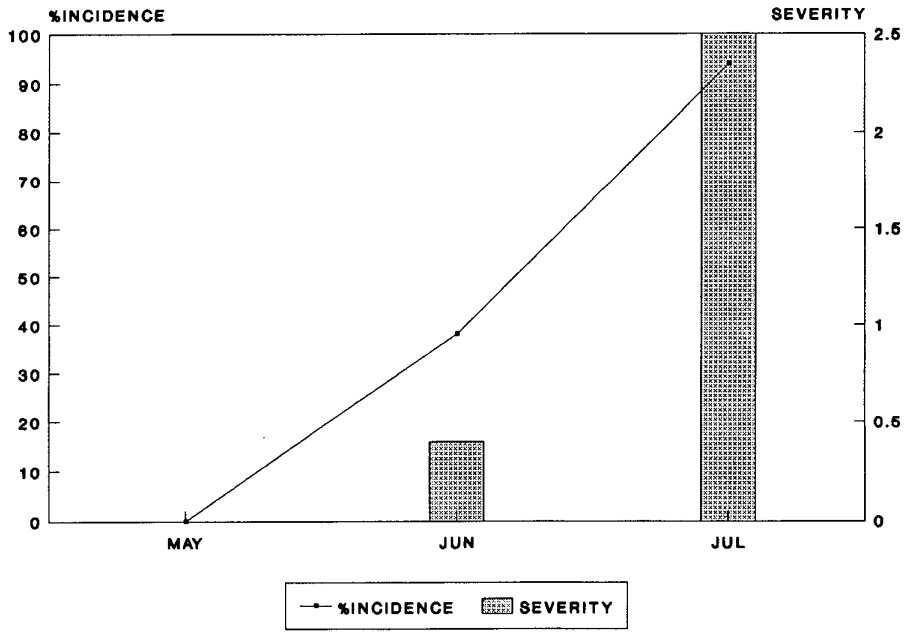


FIGURE 2B : ROTHAMSTED CANKER
6 JULY 1992, GS6.5

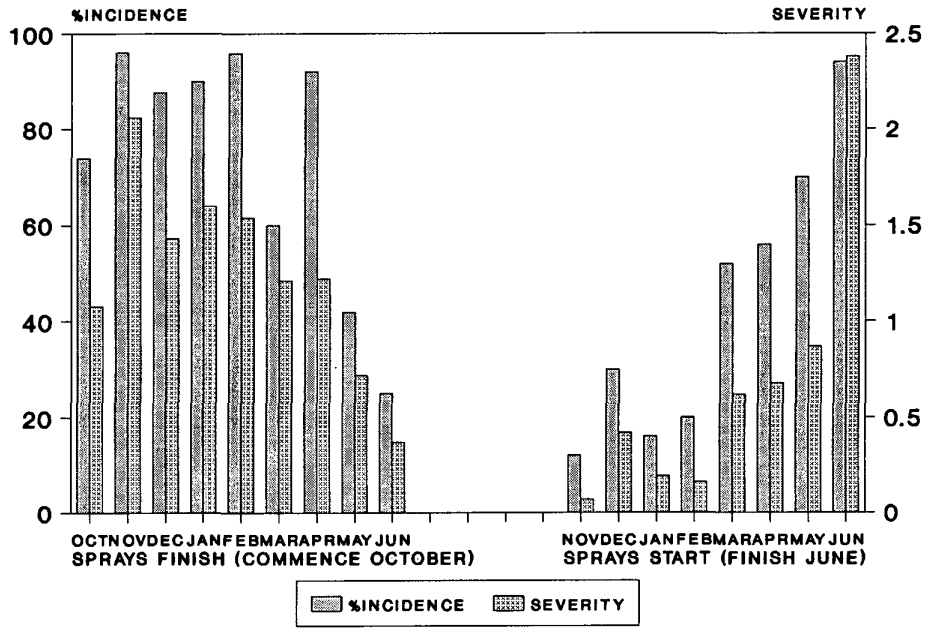


FIGURE 2C : ROTHAMSTED YIELD 1992 (T/HA)

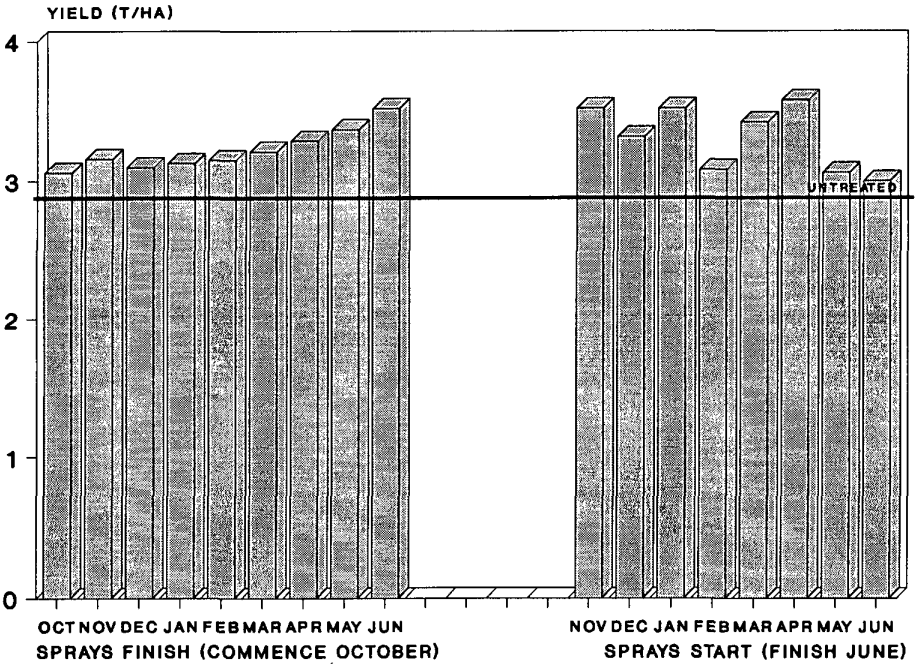
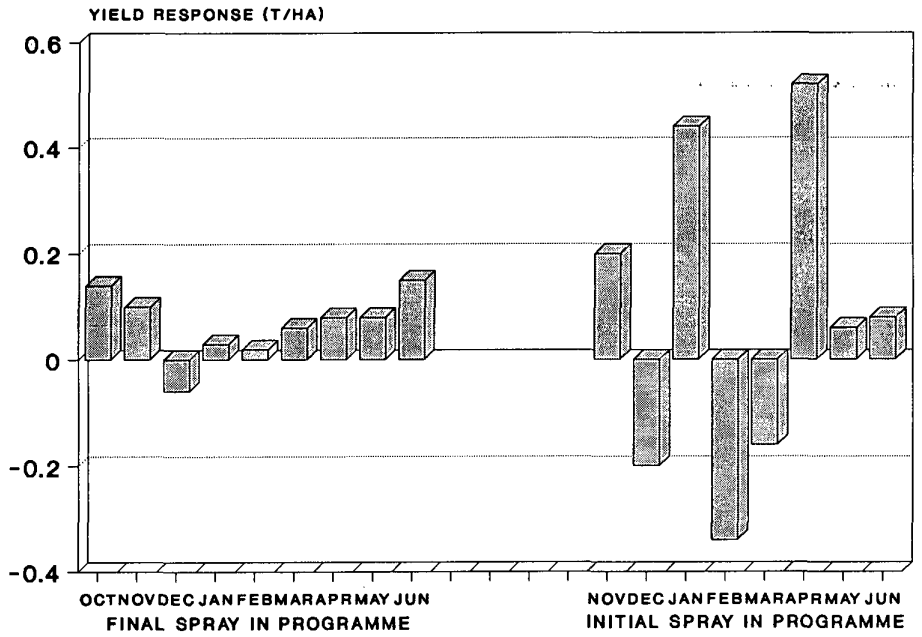


FIGURE 2D : ROTHAMSTED YIELD 1992
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(iii) Boxworth 1992/93

Both *Phoma* leaf spot and light leaf spot developed at this site but the main disease was *Phoma* leaf spot and canker. Light leaf spot is discussed in Section 2(ii).

The development of the *Phoma* leaf spot phase of canker in untreated plots is illustrated in Figure 3. Leaf spot was obvious by mid-October. The incidence and severity of disease reached its maximum by 21 January when 98 per cent of plants were severely affected at 4.8 per cent leaf area. Thereafter there was a gradual decline in the incidence of plants affected, with disease severity dropping to < 1 per cent leaf area affected by 3 February. By 28 June foliar symptoms had declined to zero.

Figure 3A illustrates canker development in untreated plots. Low levels of canker were seen during the winter but the main period of canker development occurred from late flowering onwards (7 May, GS 4.9/5.4). By 28 June (GS 6.4) 70 per cent of plants in the untreated plots were affected by aerial stem lesions and 98 per cent by basal cankers.

Significant effects of fungicide treatment on the leaf spot phase were detected. Of the treatments which received their first spray in October and finished treatment progressively later (2 to 12) significant reductions in the incidence and severity of foliar disease were evident for a limited period of time only. The treatments with the longest-lived effect against the incidence of leaf spotting received their final spray on 10 December (Treatment 4) and 1 February (Treatment 6) respectively and still had significantly less disease than the untreated plants approximately 10 weeks later (14 February, 8 April respectively). Of the treatments which received their first sprays progressively earlier (Treatments 13 to 22) reductions in disease incidence and severity were obtained at intervals throughout the season with the exception of treatments which received their first spray on or after 4 May (GS 4.1) (Treatments 15,14,13). This was too late to have a significant effect on disease when assessed in June.

Figure 3B and 3C illustrate the final incidence and severity of aerial and basal lesions in each treatment on 28 June (GS 6.4). Treatments receiving their first spray in October and finishing progressively later resulted in significant reductions in the incidence of aerial stem lesions from 70 to ≤ 15 per cent where spray applications continued up to or beyond 1 February (GS 1.12, treatments 6, and 8 to 12). Significant reductions in basal stem cankers were also obtained from these treatments with the addition of Treatments 4 and 5 which finished in December and January respectively. However, optimum control to ≤ 30 per cent incidence was only obtained from treatments receiving a February spray. Of the treatments which started progressively later (13 to 22) significant reductions in the incidence of both aerial stem lesions (to ≤ 30 per cent) and basal cankers (to ≤ 70 per cent) were obtained from treatments that had received their initial spray by 11 March (17 to 22, except Treatment 18 which started on 11 February and had 80 per cent basal cankers). Optimum control of both types of lesions however depended upon the application of sprays in November or December (Treatments 22 and 21) leading to reductions to less than 10 and 35 per cent of aerial and basal stem lesions respectively. Disease severity (Figure 3C) was similarly affected by fungicide treatment. It therefore appears that combinations of sprays applied between November and February were giving optimum control of disease.

The untreated yield at this site was 3.81 t/ha. Figure 3D and 3E show the yields obtained from all of the treatments and the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. Yields significantly greater than the untreated were obtained from Treatments 8 and 9 (sprays began in October and finished in March and April, 0.88 and 0.83 t/ha greater than the untreated respectively), and Treatment 20 (sprays began in January and finished in June, 0.65 t/ha yield response). Whilst there were no spray timings exclusive to these treatments that could be identified as associated with these yield increases there was a trend in the responses overall. Treatments which received their first spray on 19 October and finished progressively later showed an increase in yield with the addition of further sprays up until 11 March (final spray, Treatment 8).

Treatments which received their final spray on 24 June and started progressively earlier gave the greatest yield responses where the first spray was applied between 6 November and 1 February.

Yield responses were primarily related to the control of canker at this site. Optimum disease control occurred where the final spray applied in Treatments 2 to 12 occurred on or after 1 February, the contribution of the 1 February timing was 0.25 t/ha (Treatment 6 - Treatment 5). Likewise where the initial spray applied in Treatments 13 to 22 occurred on or before 1 February the control of canker was optimum; the contribution of this spray timing was 0.31 t/ha (Treatment 19 - Treatment 18). The results of regression analyses of the incidence and severity of canker on 28 June (GS 6.4) and the incidence of green stems and lodging scores on 12 July, with yield, are shown in Table 5.

Table 5. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of canker (X), incidence of green stems (X), and lodging (X).

X parameter	Regression equation	Correlation coefficient(r)*
% Basal canker incidence	Y = 4.58 - 0.007X	-0.79
Basal canker severity	Y= 4.45 - 0.373X	-0.75
% Aerial stem lesion incidence	Y= 4.47 - 0.008X	-0.74
Aerial stem lesion severity	Y= 4.45 - 0.632X	-0.74
% Green stem incidence	Y= 3.37 + 0.011X	0.90
Lodging score	Y= 3.87 + 0.514X	0.82

* $p \leq 0.001$ for all values of r

The relationship of the incidence and severity of both basal and aerial cankers with yield was strong with correlation coefficients ranging between -0.74 and -0.79, all of

which were highly significant ($p \leq 0.001$). The value of the slope b in each equation was significantly different from zero at a probability of 99.99 per cent confirming the strength of the relationship between canker and yield. For every 1 per cent increase in the incidence of either type of stem infection there was a loss in yield of 0.008 t/ha.

Regression analyses of both the incidence of green stems and of lodging on 12 July gave strong positive relationships with yield. Plots which were still green and had lodged tended to be those with the lowest levels of canker.

FIGURE 3 : BOXWORTH 1992/93
LEAF SPOT DEVELOPMENT

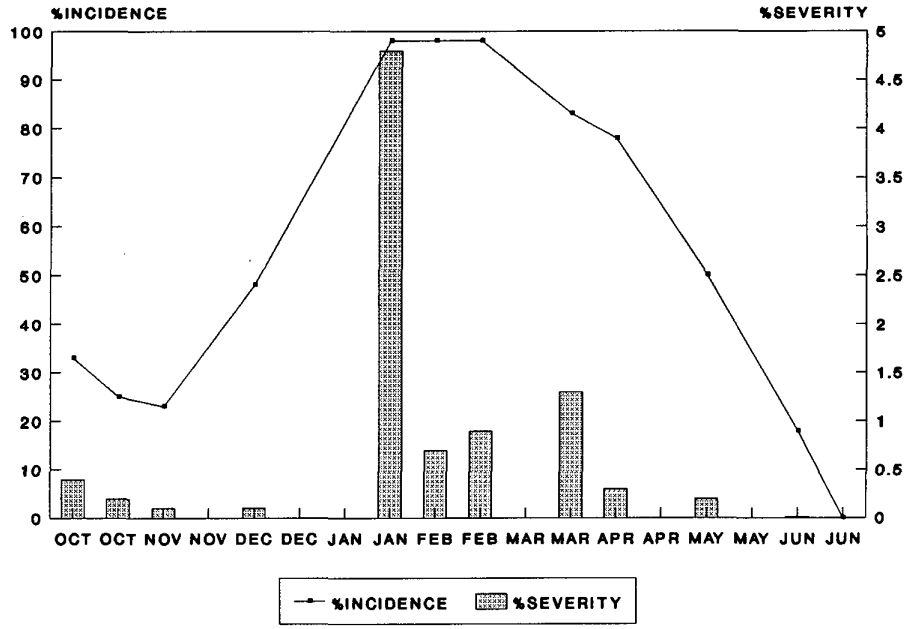


FIGURE 3A : BOXWORTH 1992/93
CANKER DEVELOPMENT

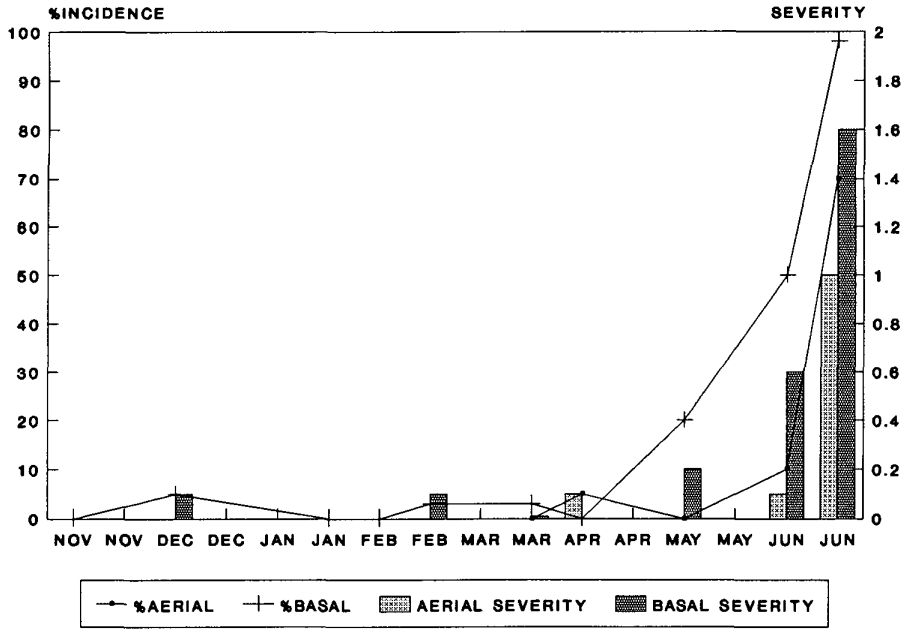


FIGURE 3B : BOXWORTH CANKER
28 JUNE 1993, GS6.4

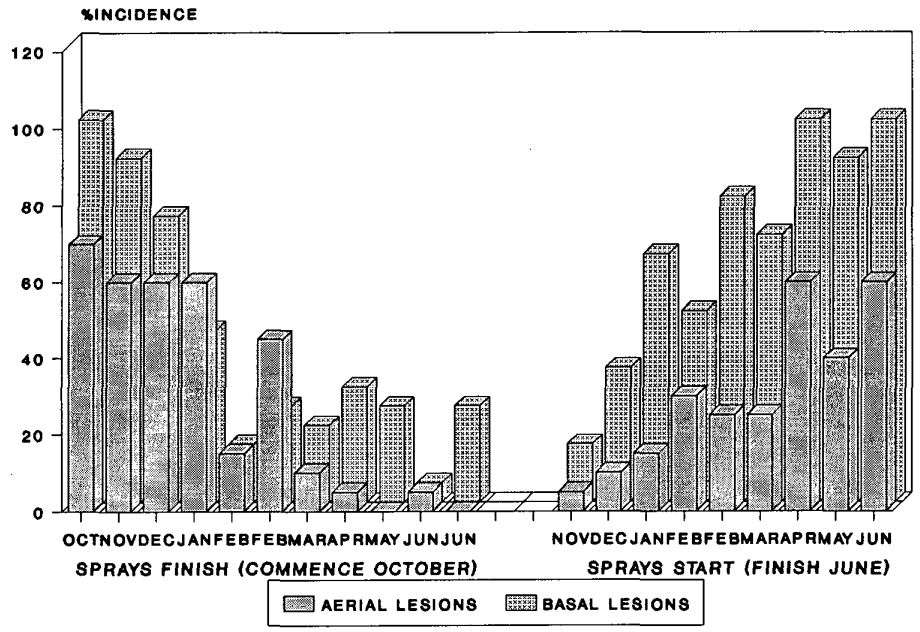


FIGURE 3C : BOXWORTH CANKER
28 JUNE 1993, GS6.4

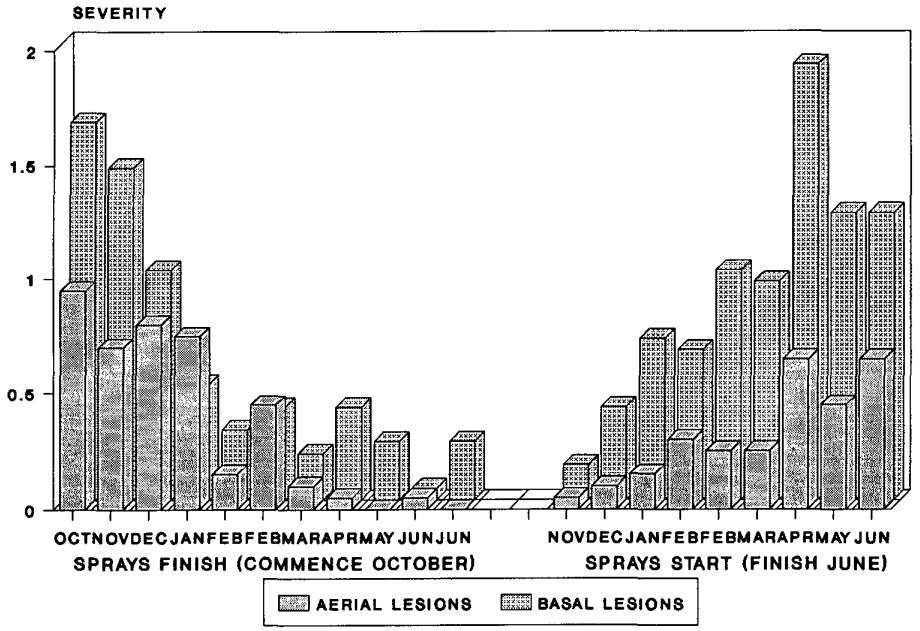


FIGURE 3D : BOXWORTH YIELD 1993 (T/HA)

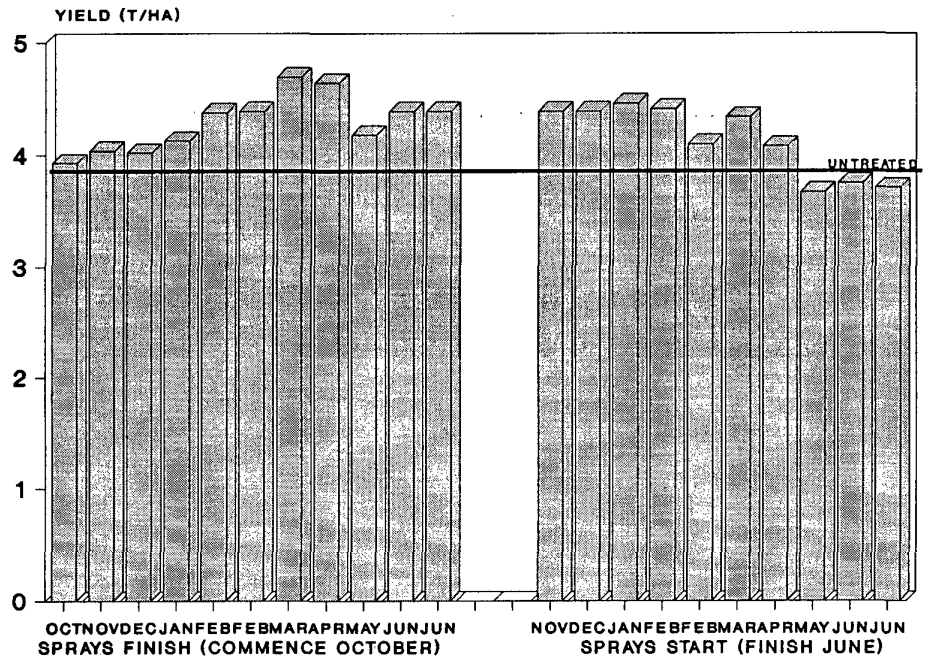
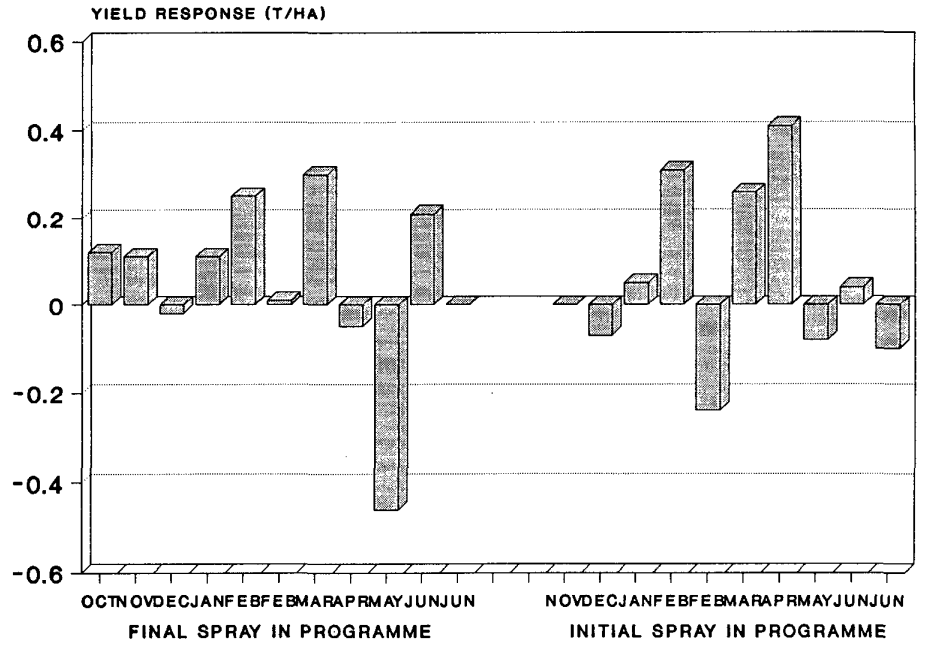


FIGURE 3E : BOXWORTH YIELD 1993
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(iv) Rothamsted 1992/93

Both *Phoma* leaf spot and light leaf spot developed at this site. Each disease was equally important; light leaf spot is discussed in Section 2(iv).

Phoma leaf spot development in untreated plots is illustrated in Figure 4. Symptoms developed early with 33 per cent of plants affected on 1 October (1.0 per cent leaf area). Maximum disease occurred on 29 October, when 95 per cent of plants were affected at 1.9 per cent leaf area. The disease fluctuated thereafter and foliar symptoms were no longer present on 9 June (GS 6.3).

Canker development in untreated plots is illustrated in Figure 4A. Cankers were first detected on 9 June (GS 6.3). By 7 July (GS 6.3) 100 per cent of plants in untreated plots were severely affected (mean severity 2.6).

Significant effects of fungicide treatment on the leaf spot phase were detected on 21 January (GS 1.11/1.13) when 70 per cent of untreated plants were affected. Treatments which included sprays applied on 2 November and 7 December had $\leq$ 25 per cent of plants affected at this time.

Figure 4B illustrates the final incidence and severity of canker on 7 July (GS 6.3) in treated plots. All treatments that included a spray on 7 December (Treatments 4 to 12 and 20 to 22) led to significant reductions in the incidence of canker from 100 to $\leq$ 60 per cent by July. Treatments 3 to 12 and 18 to 22 which had in common sprays applied between 2 November and 23 February led to significant reductions in disease severity from 2.55 to $\leq$ 1.41. Of the treatments that began in October and finished progressively later (Treatments 2 to 12), optimum reductions in disease incidence and severity to $\leq$ 35 per cent and 0.75 respectively were achieved with Treatments 5 to 12. These all included sprays from 14 October until 8 January or beyond. Likewise of the treatments that finished on 13 July and started progressively earlier only those treatments which included an initial spray applied before or on 8 January (Treatments

22, 21, 20) gave similar control to ≤ 25 per cent incidence and ≤ 0.40 severity. Hence the 8 January spray timing was critical for the control of canker at this site.

The untreated yield at this site was 3.31 t/ha. Yield responses significantly greater than the untreated were obtained from all treatments except 13, which received a single spray in July, and 15 which received three sprays between May and July. Significant responses ranged between 0.85 and 1.55 t/ha. Figure 4C shows the yields obtained from all of the treatments and Figure 4D shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments.

Treatments which began on 14 October and ended between 14 October and 13 July (Treatments 1 to 12) gave responses of between 0.91 and 1.55 t/ha. The spray timing common to all of these treatments was 14 October, which by subtraction (Treatment 2-1) gave a response of 0.91 t/ha as the final spray in a treatment (Figure 4D). This was the largest response to any of the timings. The greater the number of sprays applied the larger the yield.

Treatments which ended on 13 July and started progressively earlier (Treatments 13 to 22) gave responses of between 0.46 and 1.54 t/ha. The earlier treatment commenced the greater the yield response.

Yield responses were principally related to the control of canker at pod ripening, since control of this disease was optimum in treatments with the greatest yield (Treatments 5 to 12, and 20 to 22) (see Section 2 (iv) for the effect of light leaf spot on yield).

Regression analyses of yield on the incidence and severity of canker on 7 July are shown in Table 6.

Table 6. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of canker (X) assessed on 7 July (GS6.3)

X parameter	Regression equation	Correlation coefficient(r)*
% Canker incidence	Y = 4.81 - 0.008X	-0.76
Canker severity	Y= 4.77 - 0.363X	-0.80

* $p \leq 0.001$ for both values of r

The relationships between the incidence and severity of canker at pod ripening with yield were relatively strong with correlation coefficients of -0.76 and -0.80 respectively, both of which were significant ($p \leq 0.001$). The value of the slope b in each equation was significantly different from zero at a probability of 99.99 per cent confirming the strength of the relationship between canker and yield. For every 1 per cent increase in disease incidence there appeared to be a loss in yield of 0.008 t/ha. However light leaf spot infection of the stem was also important at this site and a strong relationship between disease control and yield was found (Section 2(iv)). Further work is required to ascertain which disease was most detrimental to yield.

FIGURE 4 : ROTHAMSTED 1992/93
LEAF SPOT DEVELOPMENT

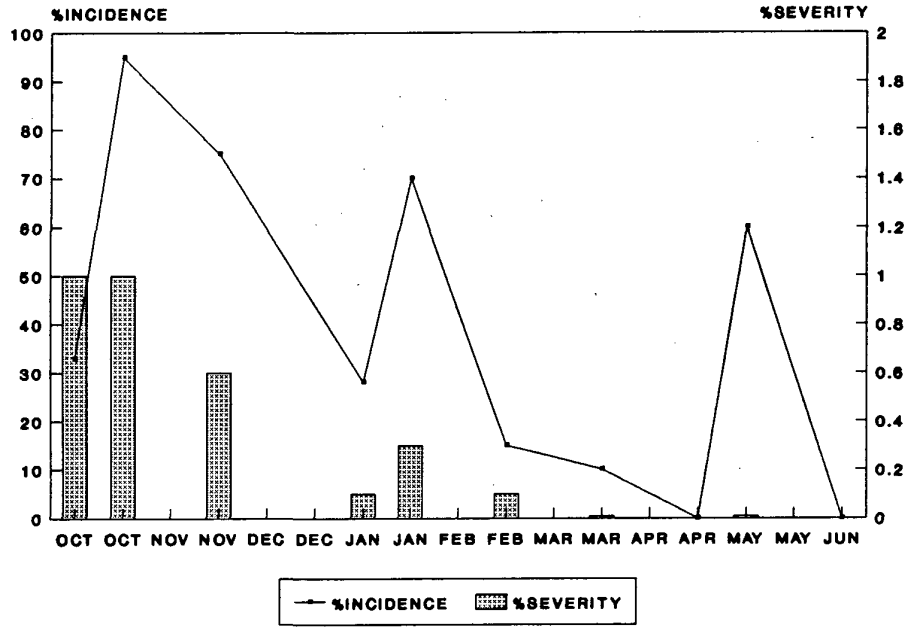


FIGURE 4A : ROTHAMSTED 1992/93
CANKER DEVELOPMENT

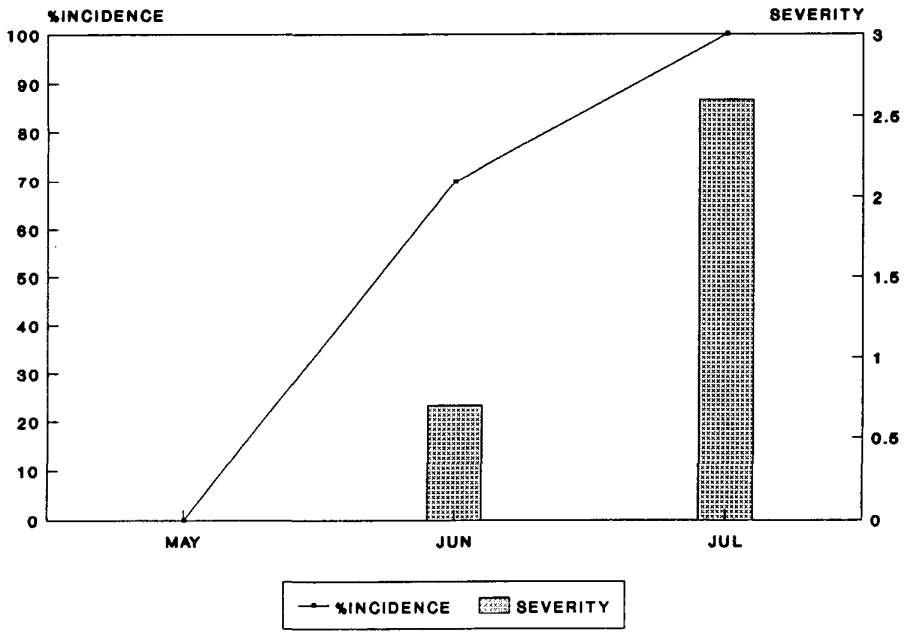


FIGURE 4B : ROTHAMSTED CANKER
7 JULY 1993, GS6.3

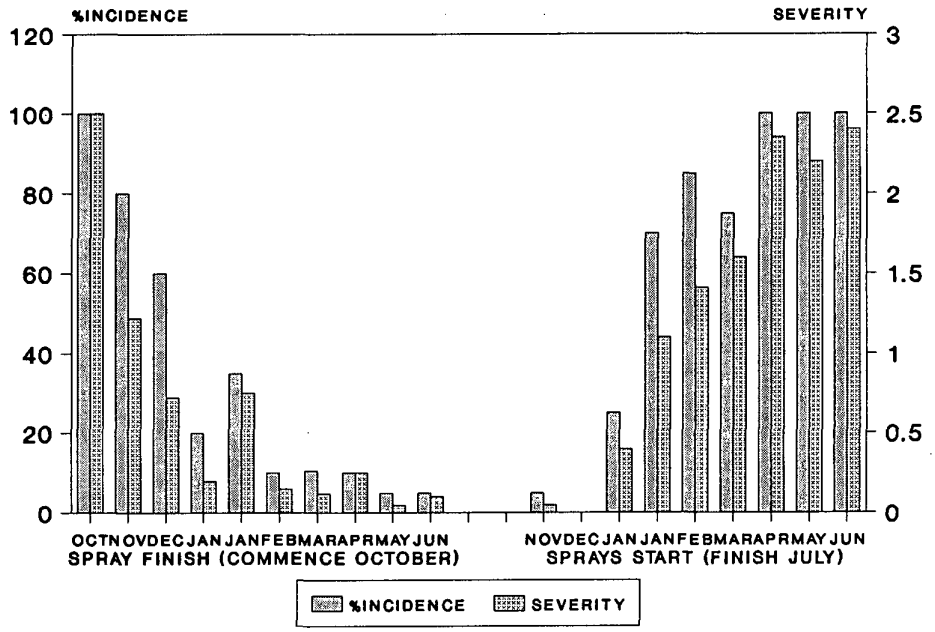


FIGURE 4C : ROTHAMSTED YIELD 1993 (T/HA)

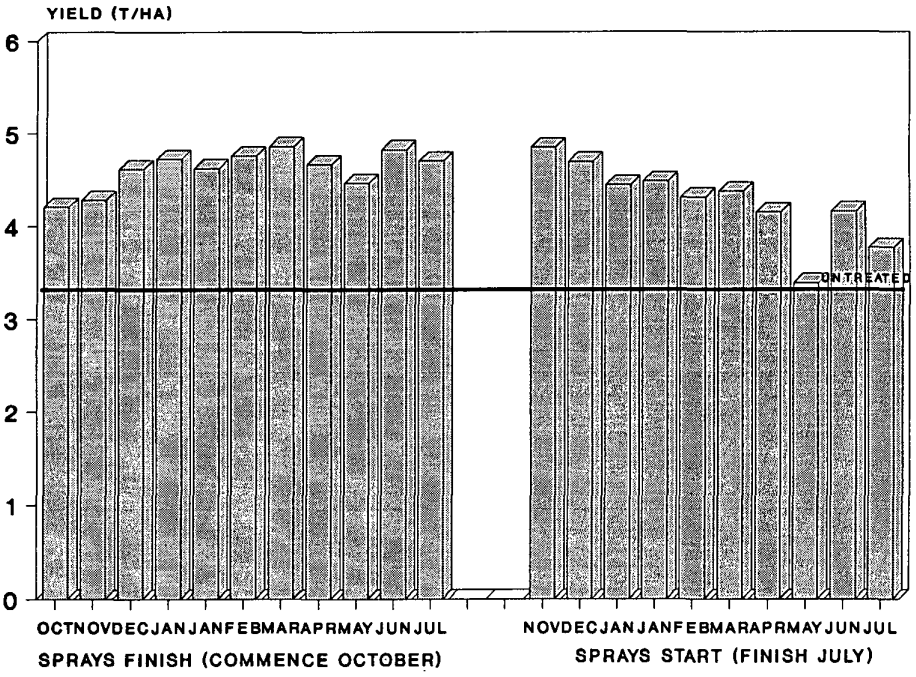
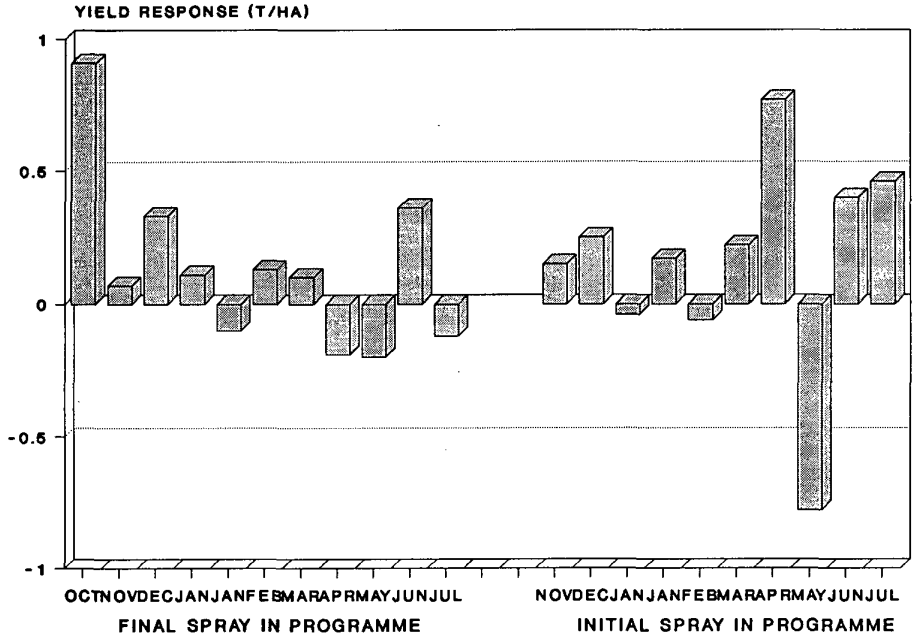


FIGURE 4D : ROTHAMSTED YIELD 1993
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(v) Tarrant Hinton 1992/93

Phoma leaf spot and canker, and light leaf spot both developed at this site. Light leaf spot is discussed in Section 2(v).

Phoma leaf spot development in untreated plots is illustrated in Figure 5. Symptoms were first detected at low levels in early October 1992. The disease was not severe throughout the autumn and winter months, affecting ≤ 0.20 per cent leaf area between 8 October and 29 January. By the end of February (GS 2.06 / 3.1), both the incidence and severity of the disease had increased (60 per cent and 2.0 per cent respectively), reaching a maximum on 19 March (GS 2.09 / 3.6) of 100 per cent incidence and 6.5 per cent severity. Symptoms started to decline in April with the loss of lower leaves, reaching zero on 8 June (GS 6.3).

Canker development in untreated plots is illustrated in Figure 5A. Cankers were first detected on 13 April at early flowering (GS 4.0 / 4.1). By 29 June (GS 6.4) 95 per cent of untreated plants were affected by stem lesions of moderate severity (1.98).

Significant effects of fungicide treatment on the leaf spot phase were detected on 23 February (GS 2.06 / 3.1). Treatments 20 and 21 reduced the incidence of disease from 60 to ≤ 35 per cent; foliar severity was reduced from 2.0 to ≤ 0.9 per cent by Treatments 4, 5, 6, 12 and 19 to 22. Spray timings common to these treatments included 9 December, 4 and 29 January.

Figure 5B illustrates the final incidence and severity of canker on 29 June (GS 6.4) in treated plots. All treatments with the exception of 14, 15 and 16 which received their first spray application on or after 13 April (GS 4.0 to 4.1) led to significant reductions in disease severity from 1.98 to ≤ 1.25. The same treatments, with the exception of Treatment 2 which received only one spray on 8 October (GS 1.04), led to significant reductions in disease incidence to ≤ 60 per cent. The best control came from sprays finishing on 29 January or later (Treatments 6 to 12) or those that had received their

first spray application on or before this date but not later (Treatments 19 to 22). Thus the late January spray appeared to give the greatest reduction in canker.

The untreated yield at this site was 3.05 t/ha. The single early spray (Treatment 2) and the one or two spray treatments applied in June (Treatments 13 and 14) did not significantly increase yield over the untreated. All other treatments (except 7) gave significant yield increases of between 0.25 and 0.63 t/ha. Figure 5C shows the yields obtained from all of the treatments and Figure 5D shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments.

The largest yield increases resulted from treatments that began in December or January (19, 20, 21; 0.50 to 0.63 t/ha). Of the treatments that began in October, Treatment 6 which received five sprays ending on 29 January gave the largest yield increase (0.41 t/ha). The January period coincided with the optimum timing for the control of leaf spot and canker. This suggests that control of this disease was the main factor involved in yield responses at this site. The effect of light leaf spot on yield is discussed in Section 2 (v).

Regression analyses of the incidence and severity of canker on 8 June (GS 6.3) with yield are shown in Table 7.

Table 7. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of canker (X) assessed on 8 June

X parameter	Regression equation	Correlation coefficient(r)*
% Canker incidence	Y = 3.45 - 0.0018X	-0.45
Canker severity	Y= 3.44 - 0.0999X	-0.47

* $p \leq 0.02$ for both values of r

The correlation coefficients (r) for both equations were only moderate but they were significant ($p \leq 0.02$). For every 1 per cent increase in the incidence of canker at pod

ripening there was a loss in yield of only 0.002 t/ha. For every increase of 1 in the stem severity score there was a loss in yield of 0.1 t/ha. This indicates that canker was not particularly damaging at Tarrant Hinton as a maximum severity score of 4 would only result in a yield loss of 0.4 t/ha.

FIGURE 5 : TARRANT HINTON 1992/93
LEAF SPOT DEVELOPMENT

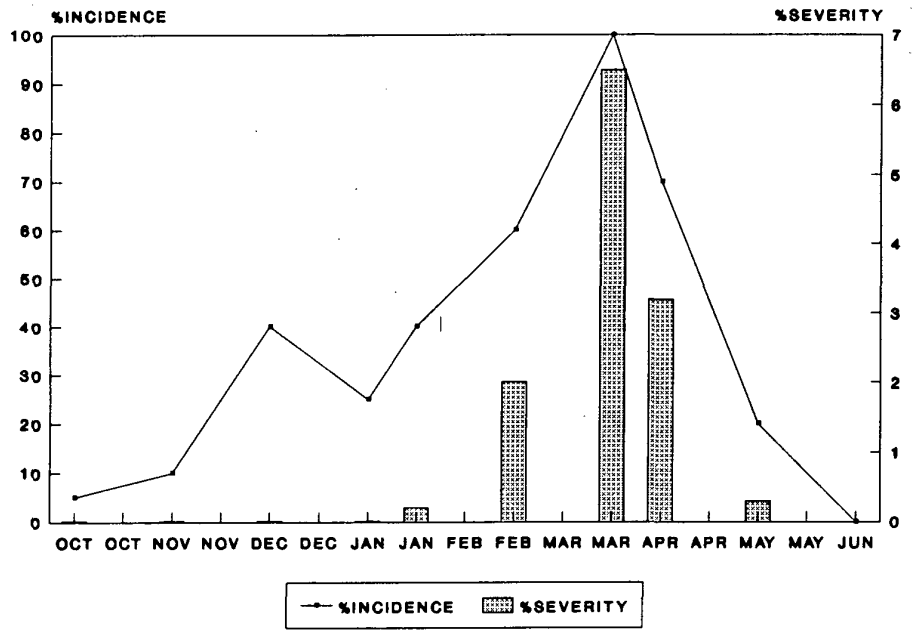


FIGURE 5A : TARRANT HINTON 1992/93
CANKER DEVELOPMENT

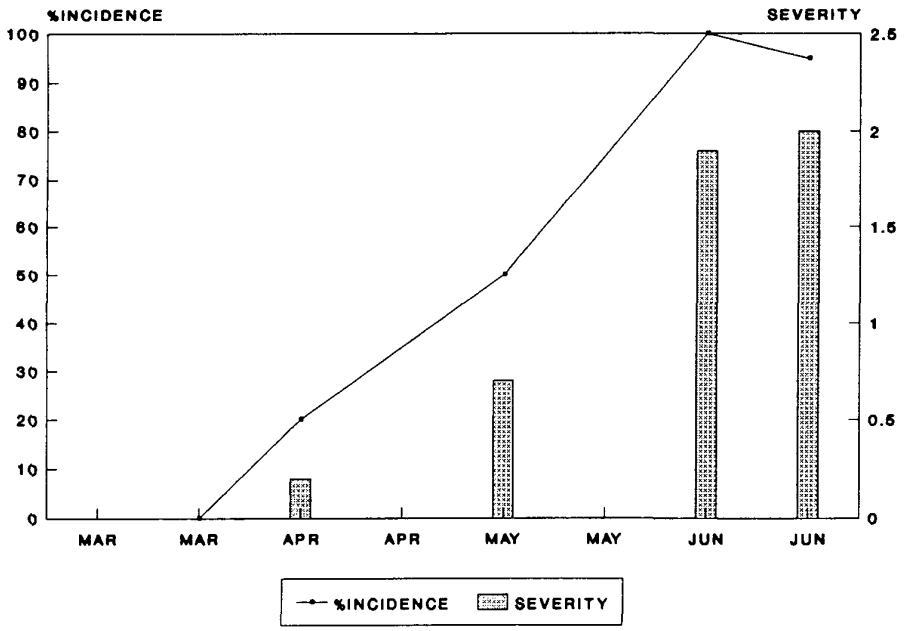


FIGURE 5B : TARRANT HINTON CANKER
29 JUNE 1993, GS6.4

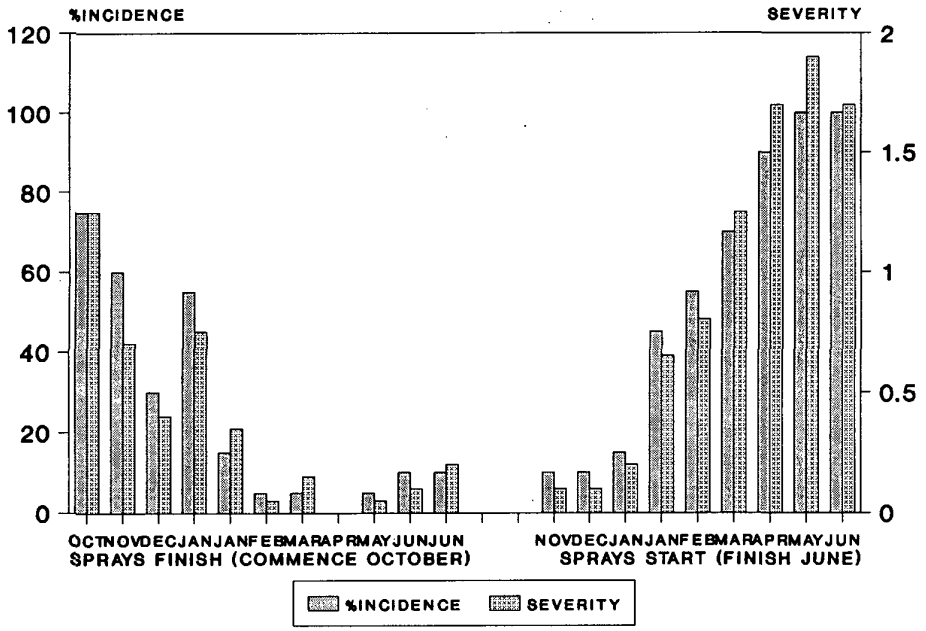


FIGURE 5C : T.HINTON YIELD 1993 (T/HA)

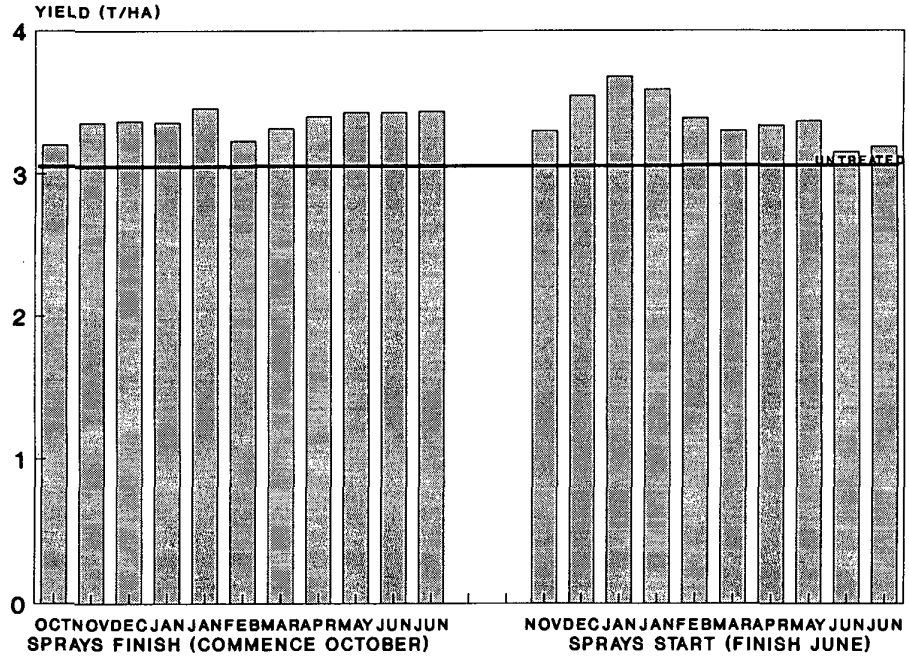
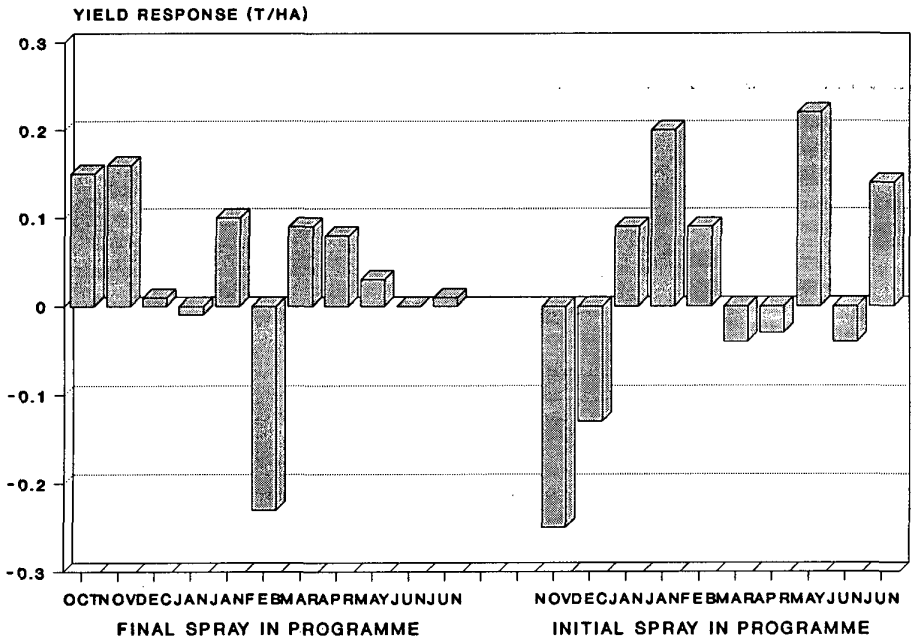


FIGURE 5D : TARRANT HINTON YIELD 1993
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(vi) Withington 1992/93

Phoma leaf spot development in untreated plots is illustrated in Figure 6. Foliar symptoms were found at the beginning of November 1992. Disease incidence and severity increased throughout the winter affecting 60 per cent of plants at severity 2.1 per cent on 29 January (GS 1.05/1.07) and reached a maximum of 75 per cent incidence, 3.9 per cent severity on 25 February. Symptoms declined thereafter with the loss of older leaves.

Canker development in untreated plots as the percentage of plants affected by cankers of severity 1 to 4 is shown in Figure 6A. Cankers were first detected on 19 May (GS 5.3). By 12 July (GS 6.5) 60 per cent of untreated plants were affected by canker of which 28 per cent of plants were dead (severity 4). Forty-three per cent of untreated plants had penetrating lesions.

Significant effects of fungicide treatment on the leaf spot phase were found in some treated plots between 8 January (GS 1.05/1.07) and 25 March (GS 2.0 / 2.05). Treatments having a significant effect on the disease all included at least one fungicide application between 5 December (GS 1.03 / 1.04) and 22 February (GS 1.05).

Figure 6B illustrates the final incidence of all *Phoma* lesions and all penetrating cankers in treated plots. Treatments 3 to 10 which commenced treatment on 4 November and received their final spray on 20 June, and 22 to 19 which commenced treatment between 5 December and 22 February finishing on 20 June, all led to significant reductions in the total incidence of canker (severity 1 to 4) and the incidence of plants with penetrating lesions from 60 and 43 per cent in control plots to $\leq$ 35 and 15 per cent respectively. Spray timings common to all of these treatments were applied between 5 December (GS 1.03) and 22 February (GS 1.05). These were the same timings that controlled the leaf spot phase.

The untreated yield at this site was 2.64 t/ha. Figure 6C shows the yields obtained from all of the treatments and Figure 6D shows the responses attributable to individual

spray timings obtained by subtraction of yields from related treatments. Yields significantly greater than the untreated but not from each other were obtained from three unrelated treatments (7, 19, and 21; 3.16, 3.22, and 3.27 t/ha respectively).

Yield responses appeared to be related to the control of leaf spot and canker since the 5 December spray timing applied as the final spray in Treatment 3 gave a response of 0.45 t/ha (Treatment 3 - Treatment 2) and the 22 February spray timing applied as the initial spray in Treatment 19 gave a response of 0.56 t/ha (Treatment 19 - 18) (Figure 6D). Of the individual spray timings these gave the largest responses; sprays applied on and between these two dates gave the best control of foliar and stem symptoms.

Regression analysis of the incidence of all stem lesions on 12 July (GS 6.5) with yield is shown in Table 8.

Table 8. Regression analysis of yield (t/ha) (Y) versus the incidence of canker (X) assessed on 12 July

X parameter	Regression equation	Correlation coefficient(r)*
% Canker incidence	Y = 3.18 - 0.009X	-0.81

* $p \leq 0.001$

The correlation coefficient (r) was strong and highly significant ($p \leq 0.001$). The value of the slope b in the equation was significantly different from zero confirming the strength of the relationship between canker and yield. For every 1 per cent increase in the incidence of canker at pod ripening there was a loss in yield of approximately 0.01 t/ha.

FIGURE 6 : WITHINGTON 1992/93
LEAF SPOT DEVELOPMENT

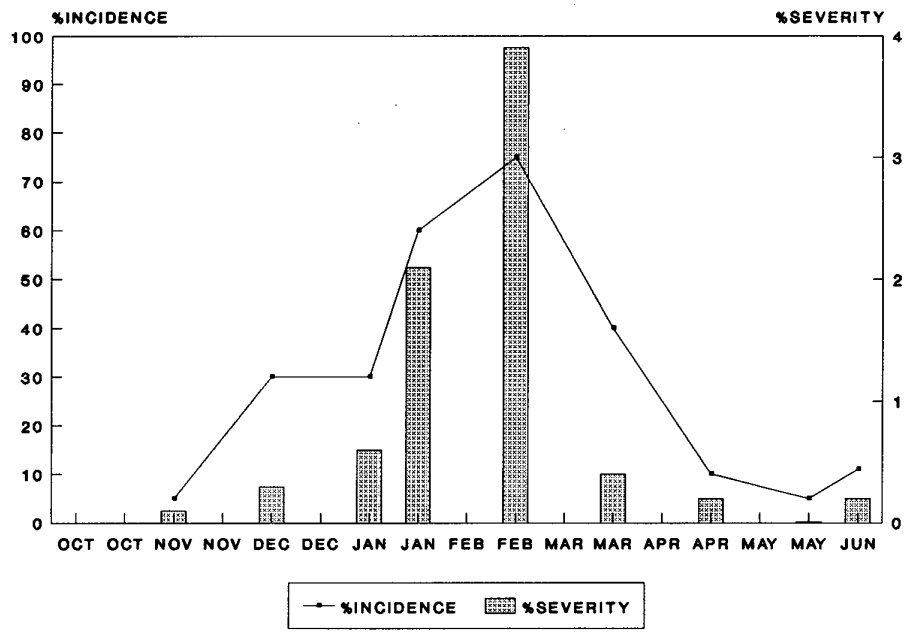


FIGURE 6A : WITHINGTON 1992/93
CANKER DEVELOPMENT

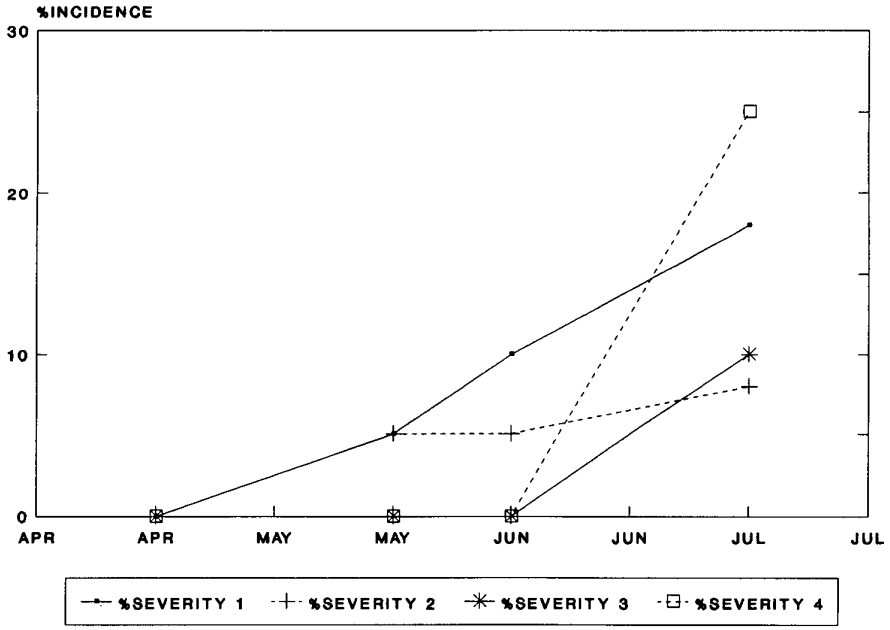


FIGURE 6B : WITHINGTON CANKER
12 JULY 1993, GS6.5

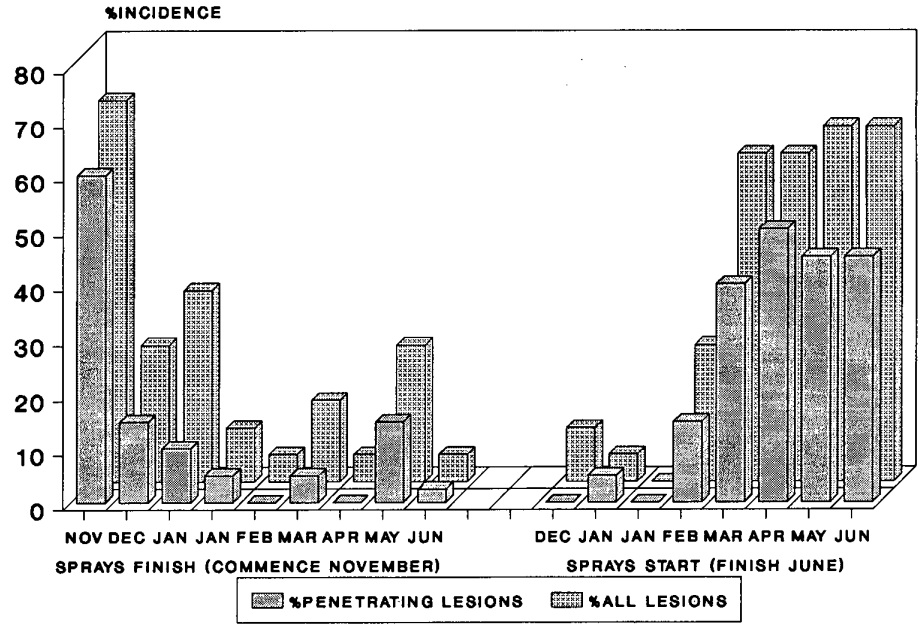


FIGURE 6C : WITHINGTON YIELD 1993 (T/HA)

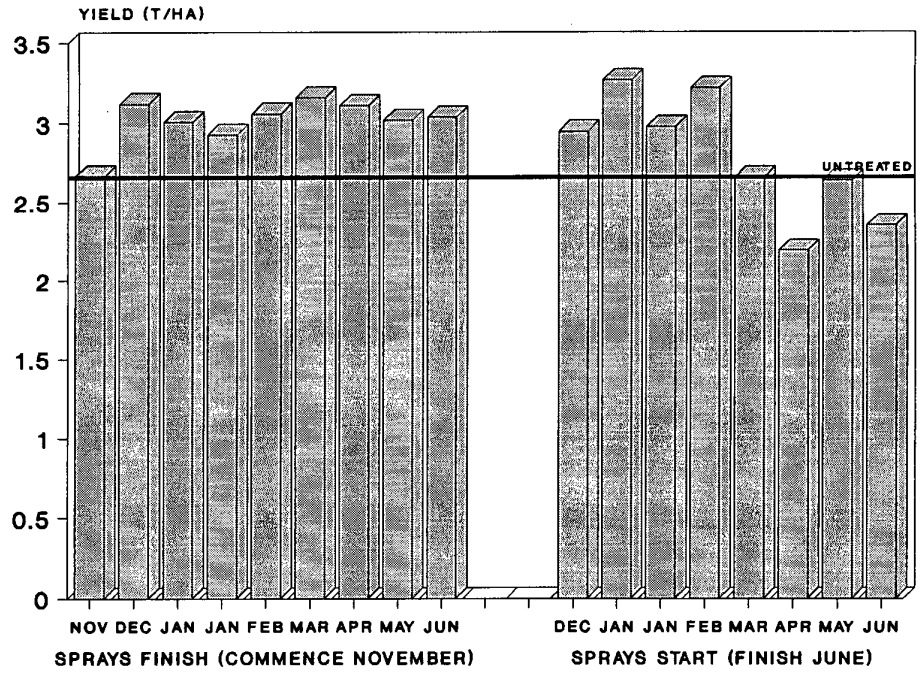
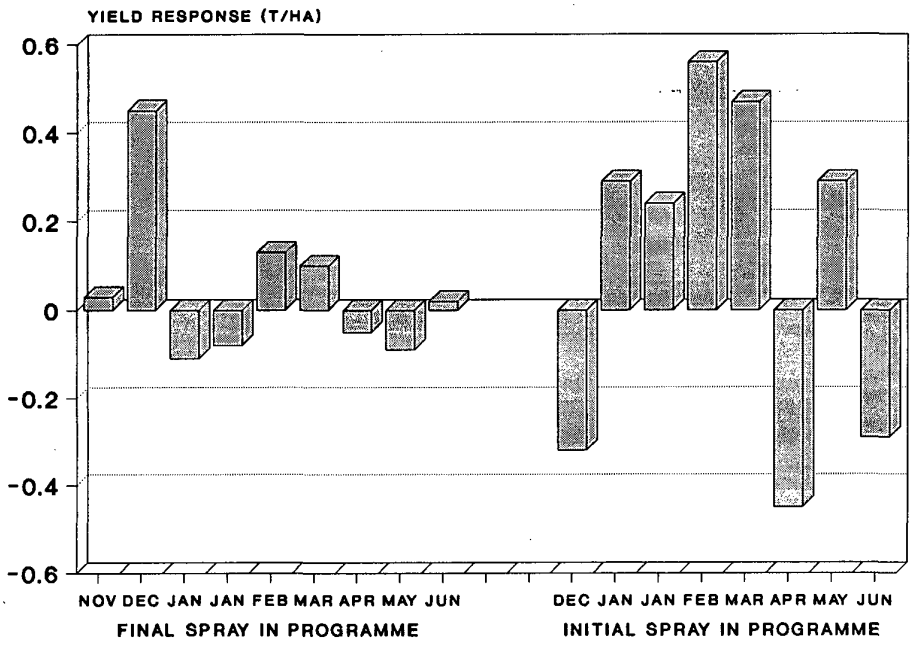


FIGURE 6D : WITHINGTON YIELD 1993
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(vii) Boxworth 1993/94

Phoma leaf spot development in untreated plots is illustrated in Figure 7. Foliar symptoms were first detected on 5 November (GS 1.4). The incidence of disease increased slowly throughout the winter reaching 32 per cent by 27 January (GS 1.8) but rising suddenly to 78 per cent of plants by 18 February (GS 2.1). The highest incidence of plants affected occurred on 13 May (GS 4.8 / 5.5) and declined thereafter reaching zero by 28 June (GS 6.3). The severity of the leaf spot phase remained at less than 1.0 per cent leaf area affected throughout the season.

Canker development in untreated plots is shown in Figure 7A. Basal cankers and aerial stem lesions were first detected on 13 May when 5 and 33 per cent of plants were affected respectively. Disease incidence increased rapidly thereafter so that by 28 June (GS 6.3) 65 per cent of plants were affected by basal canker (severity 0.8) and 90 per cent by aerial stem lesions (severity 0.95).

Significant effects of fungicide treatment on the leaf spot phase were first detected on 27 January (GS 1.8) when Treatments 4 and 5 (first sprayed on 15 October, final spray 26 November and 17 December) and 22, 21 and 20 (first sprayed on 2 and 26 November and 17 December respectively) had $\leq$ 9 per cent of plants affected compared to 34 per cent in the untreated plots. Spray timings common to these treatments included 26 November and 17 December. Mid-October and early November sprays were no longer effective at this time. Between 18 February and 13 May significant effects of fungicide treatment continued to be detected. In mid-February, Treatment 4 (sprayed on 15 October, 2 and 26 November) was no longer effective, and Treatment 19 (commenced treatment 24 January) had not controlled leaf spotting, but did so on 23 March (GS 2.07/3.1 to 3.3). This pattern was repeated through late assessments with treatments giving good control for approximately 8 weeks after a spray programme ceased or 8 weeks after the initial spray was applied.

Figure 7B and 7C illustrate the incidence and severity respectively of aerial stem lesions and basal cankers on 28 June (GS 6.3) in treated plots. Aerial lesions were

significantly reduced from 90 per cent incidence and 0.95 severity to ≤ 55 per cent and 0.55 respectively by Treatments 6 to 12 and 22 to 17. Treatments 6 to 12 received sprays commencing on 15 October and finishing on or after 24 January, Treatments 22 to 17 received initial treatment between 2 November and 21 March. Basal cankers were significantly reduced from 65 per cent incidence in untreated plots to ≤ 25 per cent by Treatments 6 to 12 and 22 to 18, the latter treatment group excluded 17, such that initial sprays had to be applied on or before 17 February for significant control to occur. Treatments 6 to 12 and 22 to 19 gave significant reductions in disease severity from 0.80 in untreated plots to ≤ 0.15. The latter group of treatments commenced between 2 November and 24 January. Thus the common group of spray timings that significantly reduced the incidence and severity of both types of lesions were Treatments 6 to 12 and 22 to 19; the spray timing common to all these treatments was 24 January (GS 1.8).

The untreated yield at this site was extremely low at 1.89 t/ha. Figure 7D shows the yield obtained from all of the treatments and Figure 7E shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. Yields significantly greater than the untreated were obtained from Treatments 5, 7 to 12, and 19 to 22. With the exception of Treatment 5 which with Treatment 7 had the lowest of the significant yields (2.29 t/ha) these treatments had the lowest incidence of basal canker and aerial stem lesions at the final assessment on 28 June.

Yield responses appeared to be related to the control of leaf spot and canker at this site. Regression analyses of the incidence and severity of aerial stem lesions and basal cankers on 28 June (GS 6.3) with yield are shown in Table 9.

Table 9. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of canker (X) on 28 June (GS 6.3)

X parameter	Regression equation	Correlation coefficient(r)*
% Basal canker incidence	Y = 2.43 - 0.007X	-0.75
Basal canker severity	Y = 2.44 - 0.616X	-0.80
% Aerial stem lesion incidence	Y = 2.50 - 0.006X	-0.84
Aerial stem severity	Y = 2.50 - 0.621X	-0.86

* $p \leq 0.001$ for all values of r

The relationships between the incidence and severity of aerial and basal cankers at pod ripening with yield were relatively strong with correlation coefficients ranging between -0.75 and -0.86 all of which were significant ($p \leq 0.001$). The value of the slope b in each equation was significantly different from zero at a probability of 99.99 per cent confirming the strength of the relationship between canker and yield. For every 1 per cent increase in the incidence of both aerial and basal cankers there appeared to be a loss in yield of approximately 0.007 t/ha.

FIGURE 7 : BOXWORTH 1993/94
LEAF SPOT DEVELOPMENT

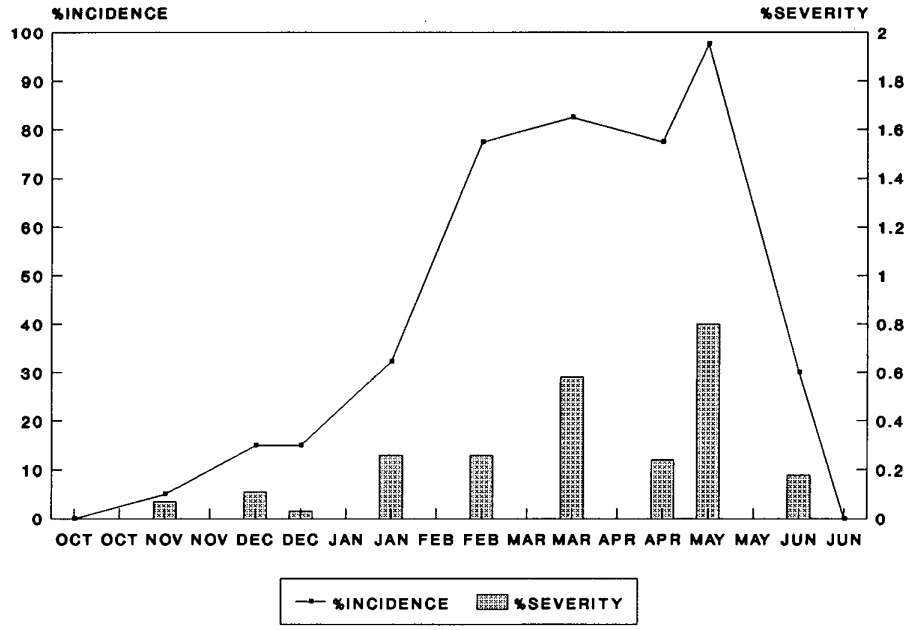


FIGURE 7A : BOXWORTH 1993/94
CANKER DEVELOPMENT

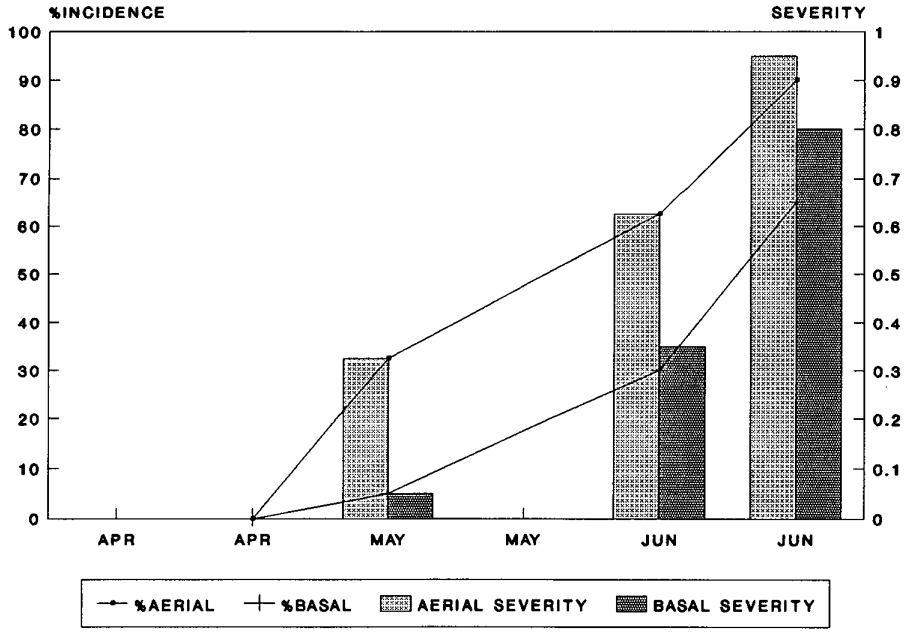


FIGURE 7B : BOXWORTH CANKER
28 JUNE 1994, GS6.3

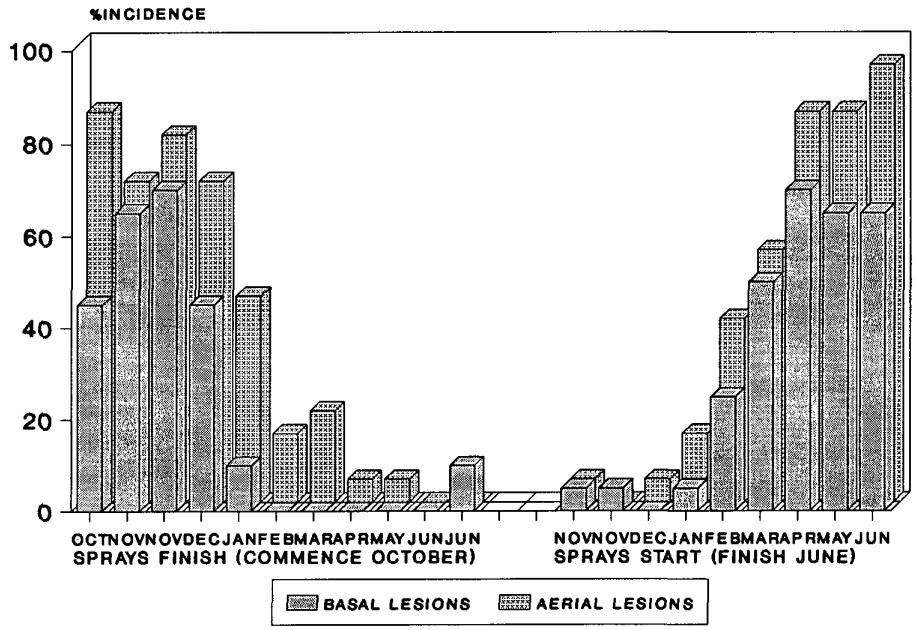


FIGURE 7C : BOXWORTH CANKER 1994
28 JUNE 1994, GS6.3

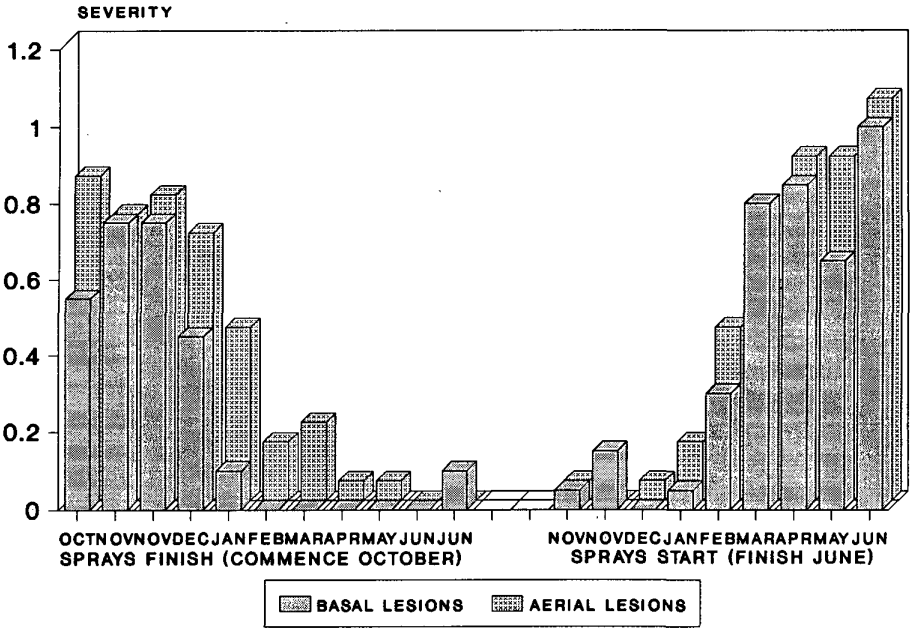


FIGURE 7D : BOXWORTH YIELD 1994 (T/HA)

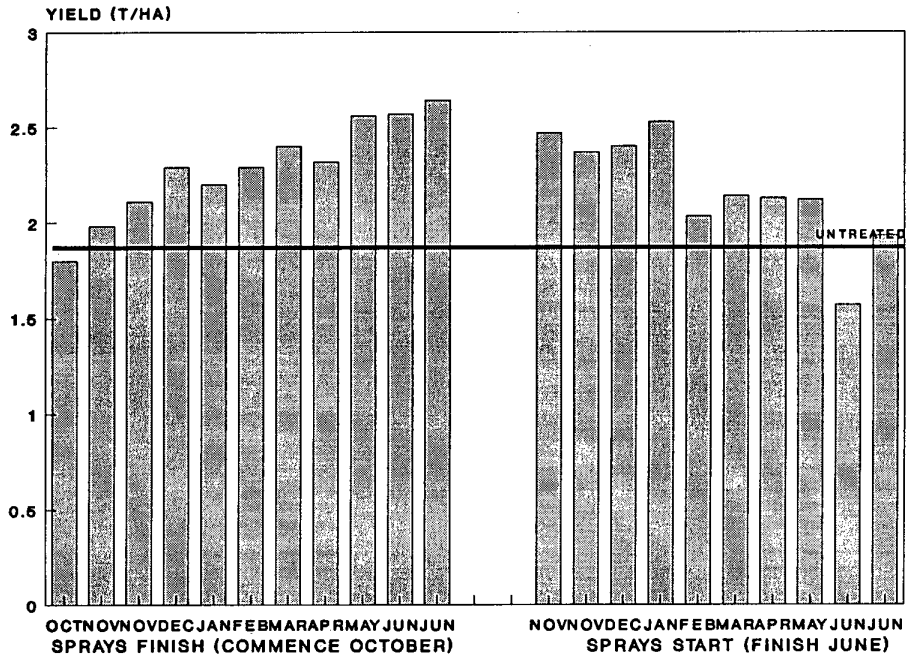
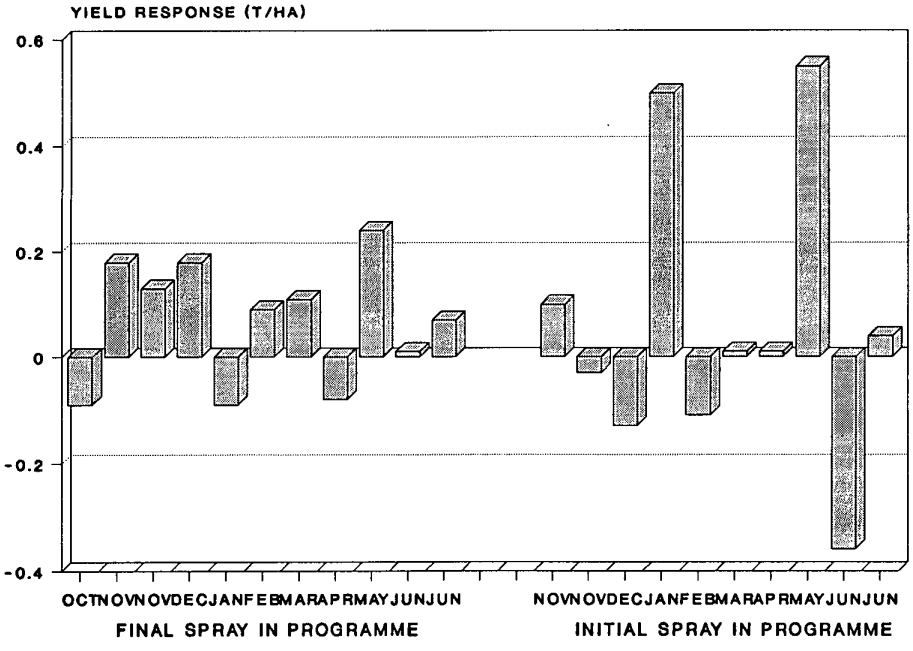


FIGURE 7E : BOXWORTH YIELD 1994
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(viii) Rosemaund 1993/94

Phoma leaf spot and canker and light leaf spot both developed at this site. Light leaf spot is discussed in Section 2(vi).

Phoma leaf spot development in untreated plots is illustrated in Figure 8. Foliar symptoms were first detected on 22 December (GS 1.05), when 25 per cent of plants in untreated plots were affected at low severity (0.17 per cent). Symptoms increased slowly over the winter and reached a maximum on 17 February (GS 1.06/1.08) when 50 per cent of plants were affected at 1.35 per cent leaf area. Symptoms declined thereafter but were still detectable on 7 June (GS 4.8/6.2).

Canker development in untreated plots is shown in Figure 8A. Symptoms of canker were detected late in the season. On 7 June (GS 4.8/6.2), 15 per cent of stems were affected in untreated plots at very low severity (0.15). By 4 July, the disease had developed further and 75 per cent of untreated plants were affected at 1.25 disease severity.

Significant effects of fungicide treatment on the leaf spot phase were first detected on 22 December (GS 1.05) when all treatments except the single 4 October spray (Treatment 2) led to significant reductions in disease incidence from 25 to $\leq$ 10 per cent. Disease severity was also significantly reduced from 0.4 per cent to $\leq$ 0.05 per cent by Treatments 3, 4, 12 and 22. The most effective treatments were those that included the 25 October spray.

On 21 January (GS 1.4) disease severity was very low in untreated plots (0.15 per cent), but 45 per cent of plants were affected by *Phoma* leaf spot and inconsistent effects of fungicide treatment were noted. By 17 February when the disease was at its maximum (50 per cent incidence, 1.4 per cent severity) significant reductions in disease incidence to $\leq$ 20 per cent were achieved with Treatments 3, 6, 12, and 19 to 22. The most effective treatments were 6 and 12 which had received 5 sprays from 4 October until 17 January at this time. This was the maximum number of sprays applied.

Disease severity was significantly reduced to ≤ 0.35 by Treatments 5, 6, 12, 19, 21 and 22. The most effective treatments reducing foliar severity to ≤ 0.05 were also 6 and 12.

Figure 8B illustrates the final incidence and severity of canker in treated plots at this site on 4 July (GS 6.4). The incidence of canker was significantly reduced from 75 per cent in untreated plots to ≤ 45 per cent by all treatments except the two-spray programme in May and June (Treatment 15) and the single spray in June (Treatment 14). The same treatments significantly reduced disease severity from 1.25 to ≤ 0.65. The most effective treatments reduced disease incidence and severity to ≤ 20 per cent and 0.2 respectively. These included Treatments 3 to 12 (initial spray 4 October, final spray on or after 25 October), and 18 to 22 (initial spray on or before 17 February). Thus sprays applied between 25 October and 17 February gave good control of canker at this site.

The untreated yield at this site was 1.92 t/ha. Figure 8C shows the yields obtained from all of the treatments and Figure 8D shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. There were no significant effects of treatment on yield at this site. In general however, the greater the number of sprays applied, the greater the yield. The maximum yield was obtained from treatment 9 which received 8 sprays between 4 October and 11 April (3.29 t/ha).

Regression analyses of the incidence and severity of canker on 4 July (GS 6.4) with yield are shown in Table 10.

Table 10. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of canker (X) on 4 July (GS 6.4)

X parameter	Regression equation	Correlation coefficient(r)*
% Canker incidence	Y = 2.82 - 0.013X	-0.71
Canker severity	Y = 2.78 - 0.780X	-0.71

* $p \leq 0.001$

The relationships between the incidence and severity of canker at pod ripening with yield were relatively strong, both had a correlation coefficient of -0.71 which were significant ($p \leq 0.001$). The value of the slope b in each equation was significantly different from zero at a probability of 99.99 per cent implying a strong relationship between canker and yield at this site. For every 1 per cent increase in the incidence of canker there appeared to be a loss in yield of approximately 0.01 t/ha. However light leaf spot also affected yield at this site (see Section 2(vi)) with stem infection appearing to cause a similar loss in yield. Further work is required to separate the effect of each disease on yield.

FIGURE 8 : ROSEMAUND 1993/94
LEAF SPOT DEVELOPMENT

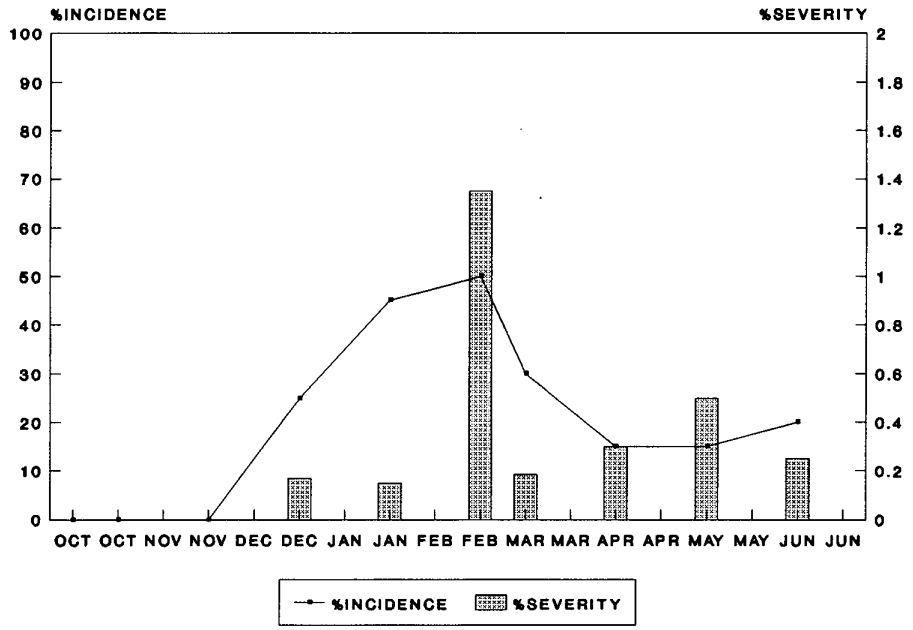


FIGURE 8A : ROSEMAUND 1993/94
CANKER DEVELOPMENT

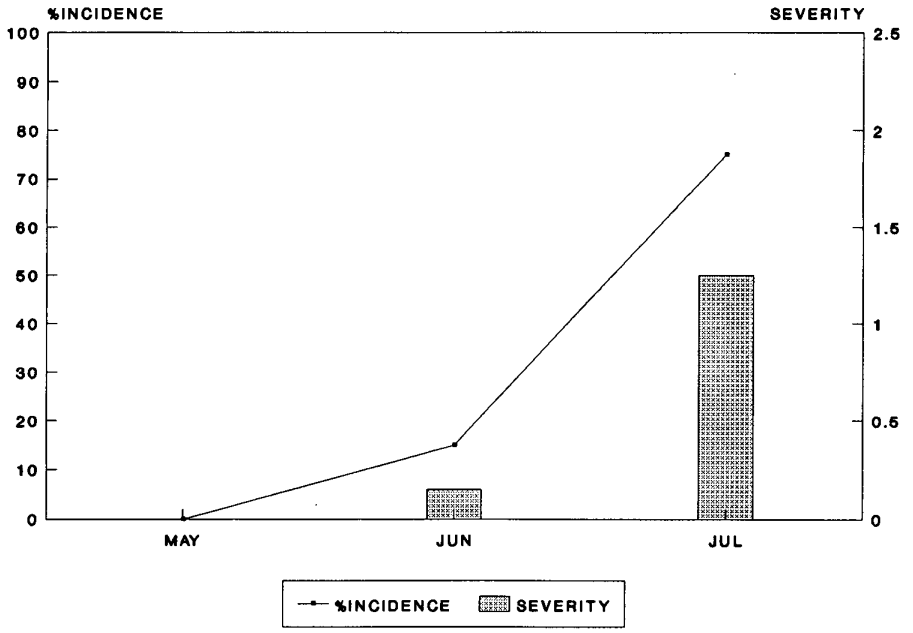


FIGURE 8B : ROSEMAUND CANKER
4 JULY 1994, GS6.4

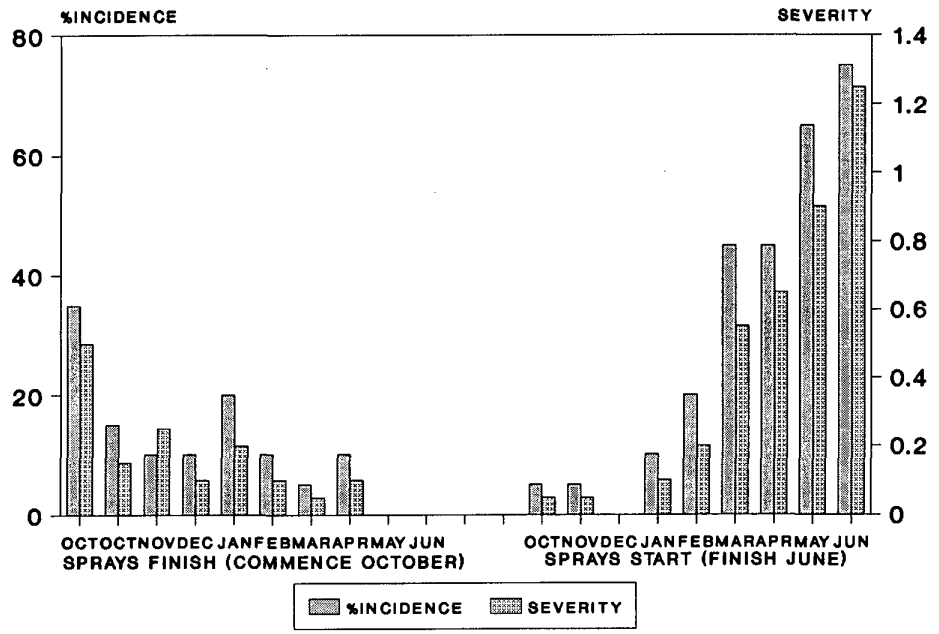


FIGURE 8C : ROSEMAUND YIELD 1994 (T\HA)

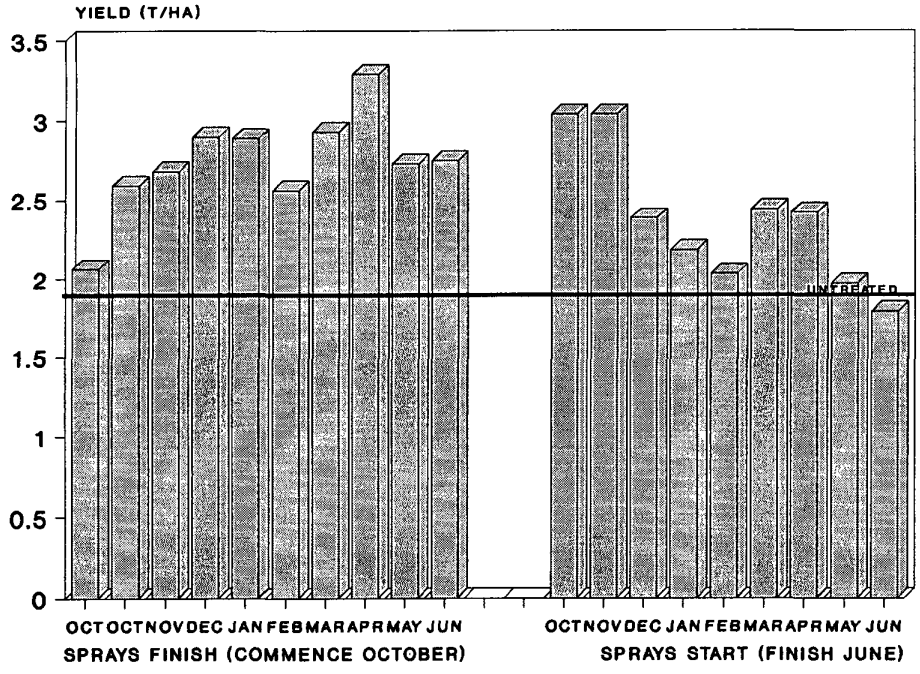
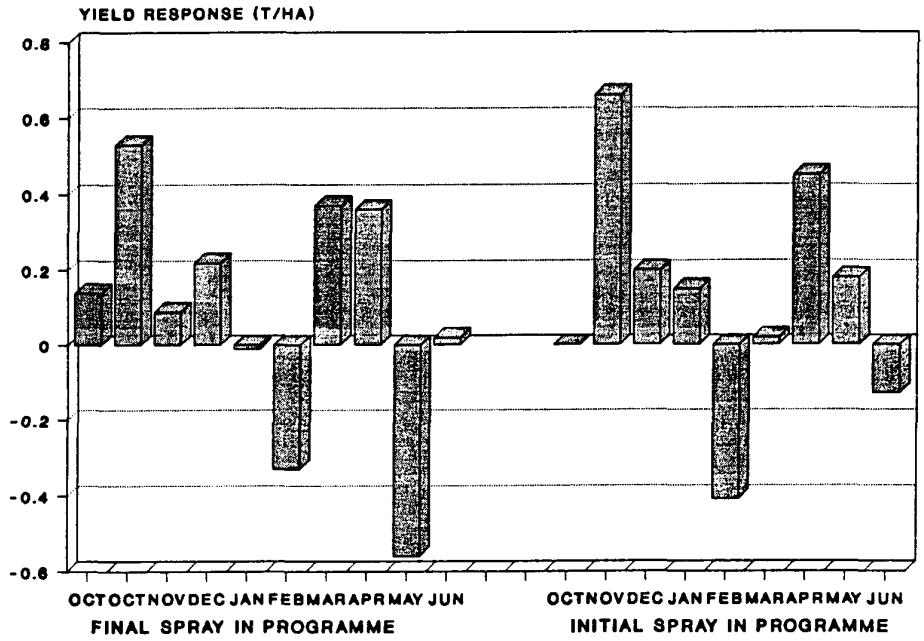


FIGURE 8D : ROSEMAUND YIELD 1994
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(ix) Rothamsted 1993/94

Phoma leaf spot and canker and light leaf spot both developed at this site. Light leaf spot is discussed in Section 2(vii).

Phoma leaf spot development in untreated plots is illustrated in Figure 9. Symptoms were first detected on 18 November (GS 1.04) when 17 per cent of plants were affected but at only 0.1 per cent leaf area. Disease severity remained at less than 1.0 per cent throughout the season. Disease incidence fluctuated but reached a maximum on 4 May (GS 4.3) when 70 per cent of plants showed foliar symptoms.

Symptoms of canker were first observed late in the season on 1 June (GS 6.1) when 55 per cent of plants were affected (severity 0.53). Symptoms increased thereafter reaching a maximum on 20 July when 100 per cent of plants were severely affected at severity 2.95 (Figure 9A).

Significant reductions in the leaf spot phase were detected on 9 March (GS 2.3) when treatments that had received a spray on 19 January had significantly less disease (untreated 25 per cent incidence; Treatments 6, 7, 12, 19, 20, $\leq$ 5 per cent). Significant reductions in foliar severity were detected on 4 May but the untreated plots were not severely affected at this time (0.22 per cent leaf area).

Figure 9B illustrates the incidence and severity of canker in all treated plots on 20 July (GS 6.5). Data relating to disease incidence were skew and could not be transformed to normality. However, significant reductions in disease severity were obtained with Treatments 5 to 12, and 16 to 20, reducing the severity score from 2.95 in untreated plots to $\leq$ 1.65. A single November spray (Treatment 4) was insufficient to control the disease (severity 2.95). Likewise, treatments that began after 19 April (Treatments 15, 14, 13) also gave no significant control. Thus, sprays applied between 17 December and 19 April gave some control of the disease.

Where sprays began in November and finished progressively later, the addition of the 17 February spray in Treatment 7 substantially reduced the disease severity score from 1.65 (Treatment 6, final spray 19 January) to 0.20 (Treatment 7). Likewise where treatments began progressively later, initial sprays applied on or before 17 February had very low canker severity (≤ 0.20, Treatments 18, 19, 20); treatments that began on 30 March or later (Treatments 17 to 13) had disease severity ≥ 0.90. Thus, 17 February appeared to be the key spray timing for disease control at this site.

The untreated yield at this site was average at 3.35 t/ha. Figure 9C shows the yields obtained from all of the treatments and Figure 9D shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. No significant differences in yield were detected. Of the treatments that began in the autumn and finished later (Treatments 4 to 12) there were no consistent trends other than the addition of a final spray in February in Treatment 7 caused an increase in yield of 0.36 t/ha when compared to Treatment 6 which finished in January. Likewise, commencing treatment in February (Treatment 18) was more beneficial than commencing in January (Treatment 19) or March (Treatment 17), (0.55 and 0.41 t/ha greater respectively). Yields therefore appeared to be related to the control of canker since the February spray timing was critical for control of the disease. However light leaf spot on leaves and stems was also controlled by this spray (Section 2 (vii)).

Sprays applied in January and July appeared to particularly damaging to yield (Figure 9D). January temperatures were particularly low and it is possible that the application of fungicides during this time resulted in crop damage.

Regression analysis of the incidence and severity of canker on 20 July (GS 6.5) with yield are shown in Table 11.

Table 11. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of canker (X) on 20 July (GS 6.5)

X parameter	Regression equation	Correlation coefficient(r)*
% Canker incidence	Y = 3.78 - 0.005X	-0.63
Canker severity	Y = 3.70 - 0.195X	-0.57

* $p \leq 0.08$

The relationships between the incidence and severity of canker at pod ripening with yield were not especially strong, with correlation coefficients of -0.63 and -0.57 respectively, however, both were significant ($p \leq 0.008$). The value of the slope b in each equation was significantly different from zero at a probability of 99.99 per cent implying that a relationship did exist between canker and yield at this site. For every 1 per cent increase in the incidence of canker there was a very low yield loss of only 0.005 t/ha. Canker was not especially damaging at this site. Similar yield losses were seen with light leaf spot infection of the stem (see Section 2(vii)) and further work is required to separate the effect of each disease on yield.

FIGURE 9 : ROTHAMSTED 1993/94
LEAF SPOT DEVELOPMENT

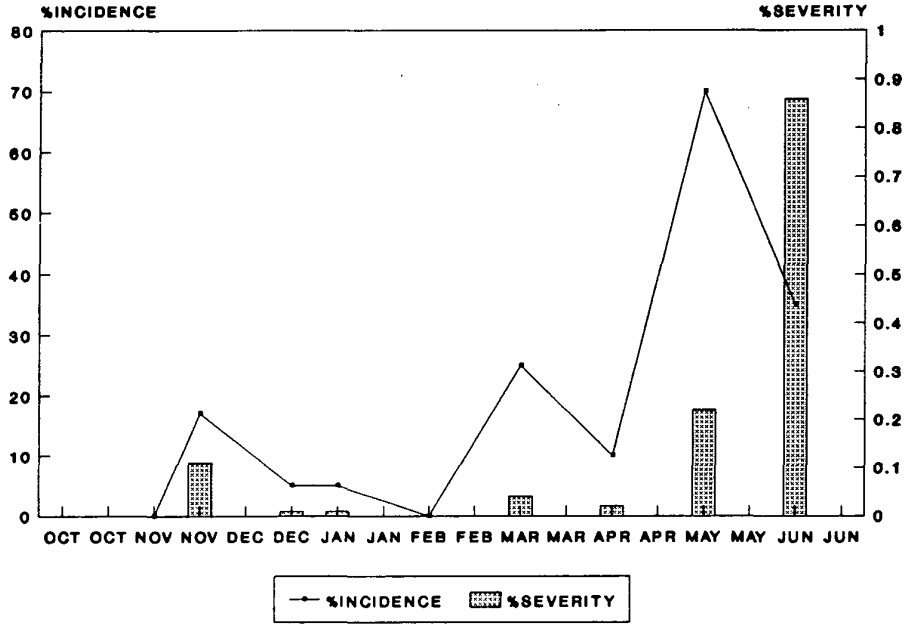


FIGURE 9A : ROTHAMSTED 1993/94
CANKER DEVELOPMENT

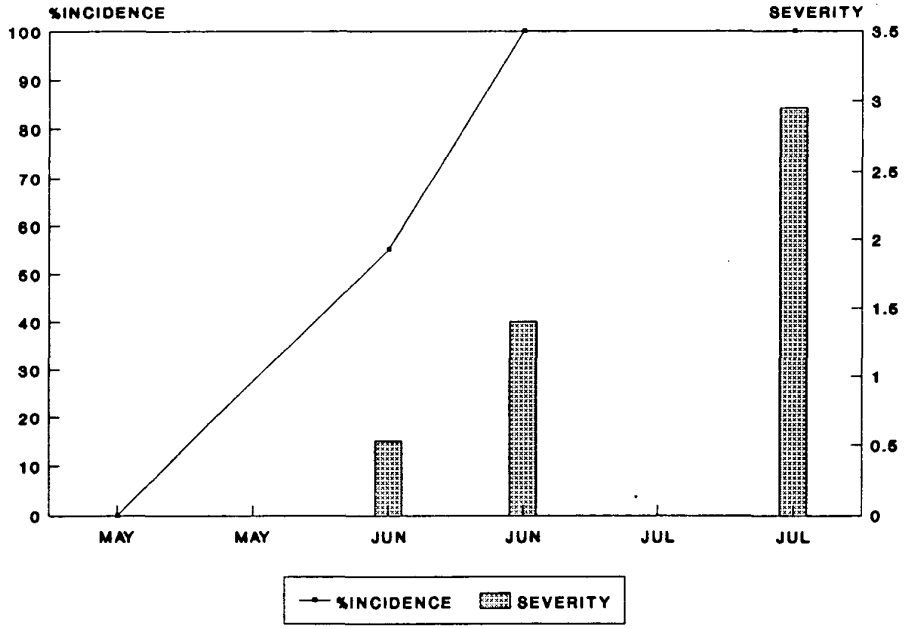


FIGURE 9B : ROTHAMSTED CANKER
20 JULY 1994, GS6.5

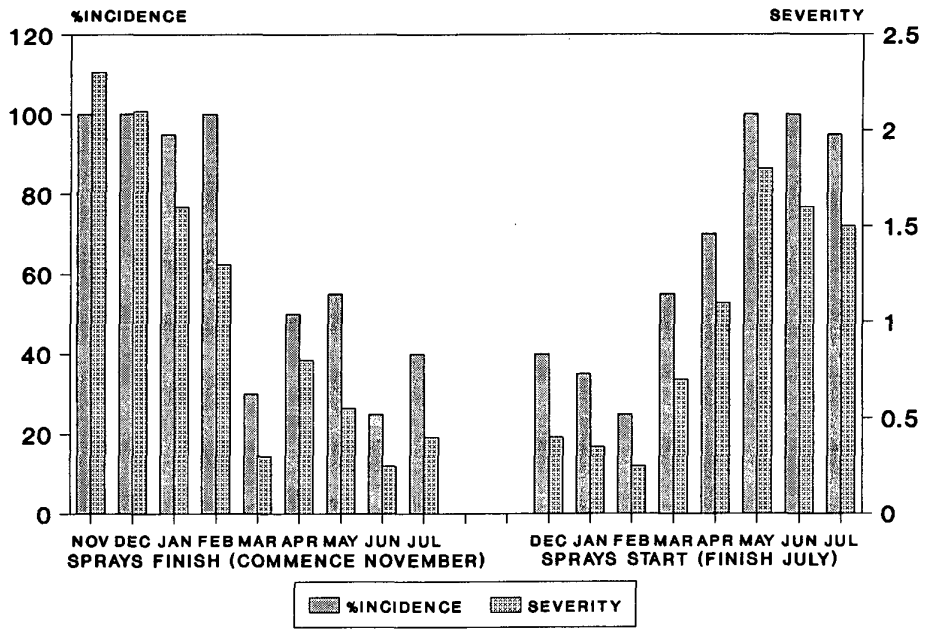


FIGURE 9C : ROTHAMSTED YIELD 1994 (T\HA)

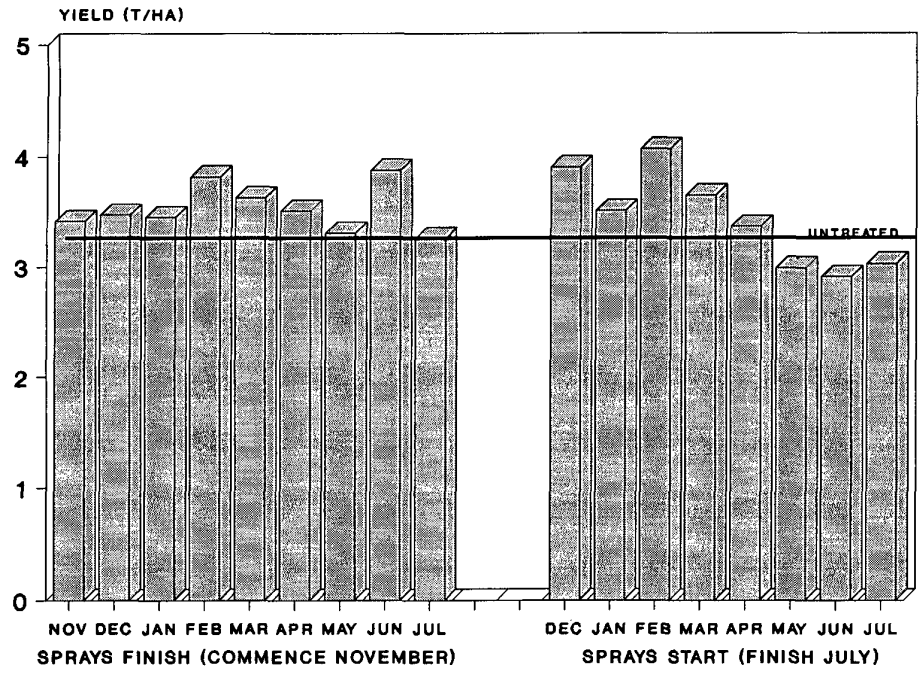
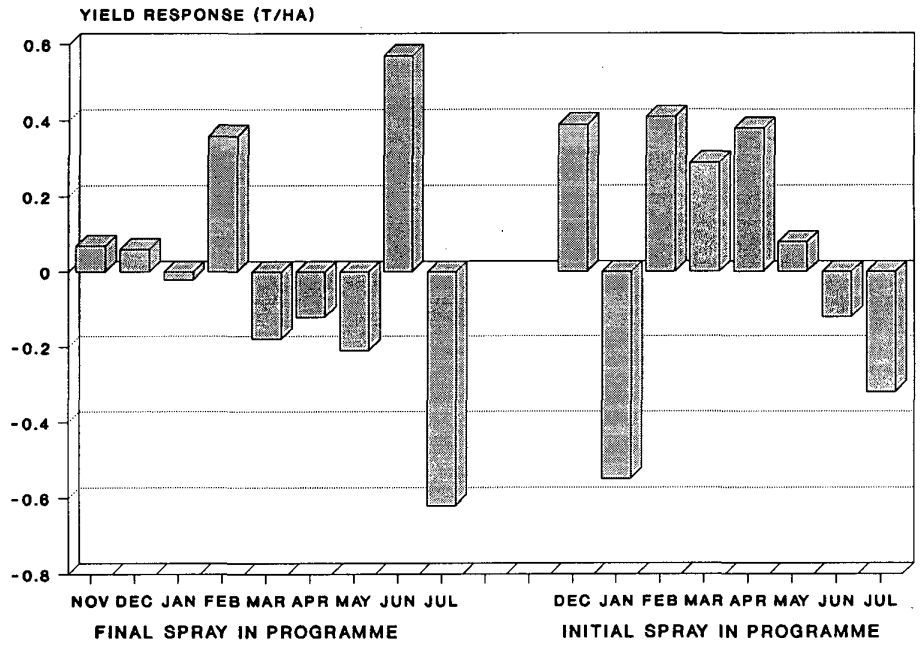


FIGURE 9D : ROTHAMSTED YIELD 1994
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(x) Thurloxton 1993/94

Phoma leaf spot and canker and light leaf spot both developed at this site. Light leaf spot is discussed in Section 2(viii).

Phoma leaf spot development in untreated plots is illustrated in Figure 10. Symptoms were first detected on 26 November (GS 1.05) when 50 per cent of plants were affected at 0.9 per cent leaf area. Symptoms had declined slightly by 7 January but rose to 60 per cent incidence and 2.3 per cent severity by 31 January (GS 1.08). Maximum foliar disease occurred on 21 February (GS 1.11 / 2.00) when 65 per cent of plants were affected at 2.9 per cent leaf area. Thereafter symptoms declined; the disease was no longer detected by 6 June (GS 6.2 / 6.3).

Canker development in untreated plots occurred late in the season. Symptoms were first observed on 6 June when 50 per cent of plants were affected but the disease was not severe (severity 0.5) (Figure 10A).

Significant reductions in the leaf spot phase were detected on 21 February (GS 1.11/2.00) when the disease was at a maximum. Disease severity was significantly reduced from 2.95 to $\leq$ 1.15 percentage leaf area affected by all treatments except 4 (sprayed 28 October and 26 November). The most effective treatments reducing disease severity to $\leq$ 0.30 were those that had received the most sprays (6, 12, and 21). No significant effects on disease incidence were detected at this time.

Figure 10B illustrates the incidence and severity of canker in all treated plots on 6 June (GS 6.3). Significant reductions in both the incidence and severity of disease from 50 per cent and 0.5 respectively were achieved with Treatments 7 to 10, 12, and 21 and 20; less than 10 per cent of plants were affected at a severity score of 0.1. Of the treatments that received their first spray on 28 October (7 to 10, 12) final sprays applied on or after 21 February (GS 1.11/2.00) resulted in significant disease control. Of the treatments that received initial sprays progressively later, the first spray had to be applied on 26 November or 7 January for significant disease control to occur

(Treatments 21 and 20). Spray timings common to all treatments that significantly reduced canker were applied between 7 January (GS 1.06) and 21 February (GS 1.11 / 2.00).

The untreated yield at this site was extremely low at 1.76 t/ha. Figure 10C shows the yields obtained from all of the treatments and Figure 10D shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. Yields significantly greater than the untreated were obtained from Treatment 4 (sprayed 28 October, 26 November; 2.43 t/ha), 8 to 12 (sprayed 28 October to 15 March or later, 2.90 to 3.39 t/ha), and 21 to 17 (initial spray applied between 26 November and 15 March respectively, 2.68 to 3.51 t/ha). With the exception of Treatment 4, all significant yield increases occurred where a spray was applied on 15 March (GS 2.05 / 3.1). As a final spray in a treatment (Treatment 8 - Treatment 7) a yield response of 0.63 t/ha was obtained; likewise as an initial spray (Treatment 18 - Treatment 17) 0.39 t/ha yield response occurred (Figure 10D).

This suggests that disease control was not solely related to the control of canker; light leaf spot was most likely responsible for yield losses at this site (see Section 2(viii)).

Regression analysis of the incidence and severity of canker on 6 June (GS 6.3) with yield is shown in Table 12.

Table 12. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of canker (X) on 6 June (GS 6.3)

X parameter	Regression equation	Correlation coefficient(r)*
% Canker incidence	Y = 3.18 - 0.020X	-0.75
Canker severity	Y = 3.16 - 1.81X	-0.72

*$p \leq 0.001$

Despite the apparent lack of a relationship between spray timings for the control of canker and those spray timings that increased yield at this site, strong and significant relationships between canker incidence and severity and yield were found. For every 1 per cent increase in canker incidence 0.02 t/ha was lost. However light leaf spot had a greater effect on yield at Thurloxton and this is discussed in Section 2(viii).

FIGURE 10 : THURLOXTON 1993/94
LEAF SPOT DEVELOPMENT

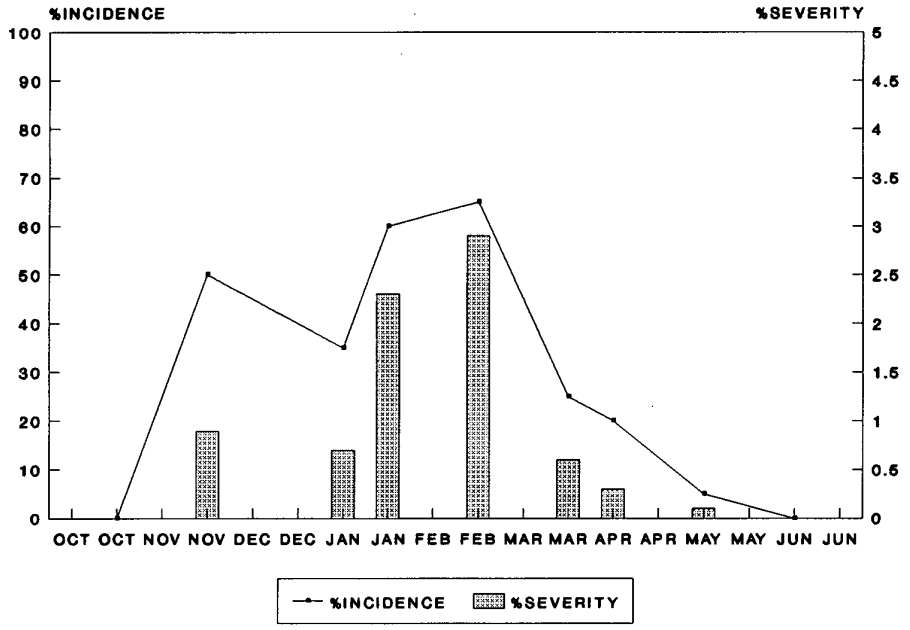


FIGURE 10A : THURLOXTON 1993/94
CANKER DEVELOPMENT

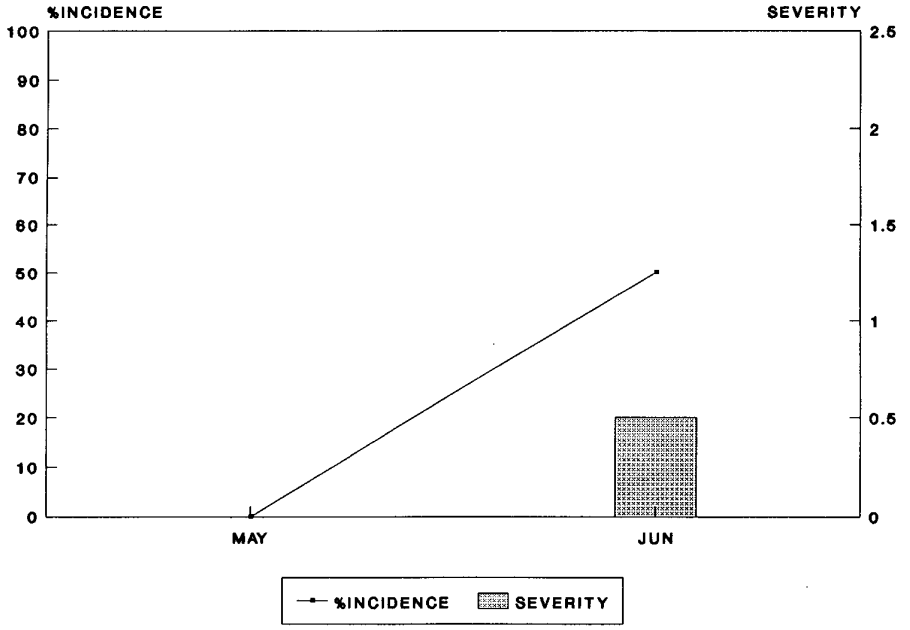


FIGURE 10B : THURLOXTON CANKER
6 JUNE 1994, GS6.3

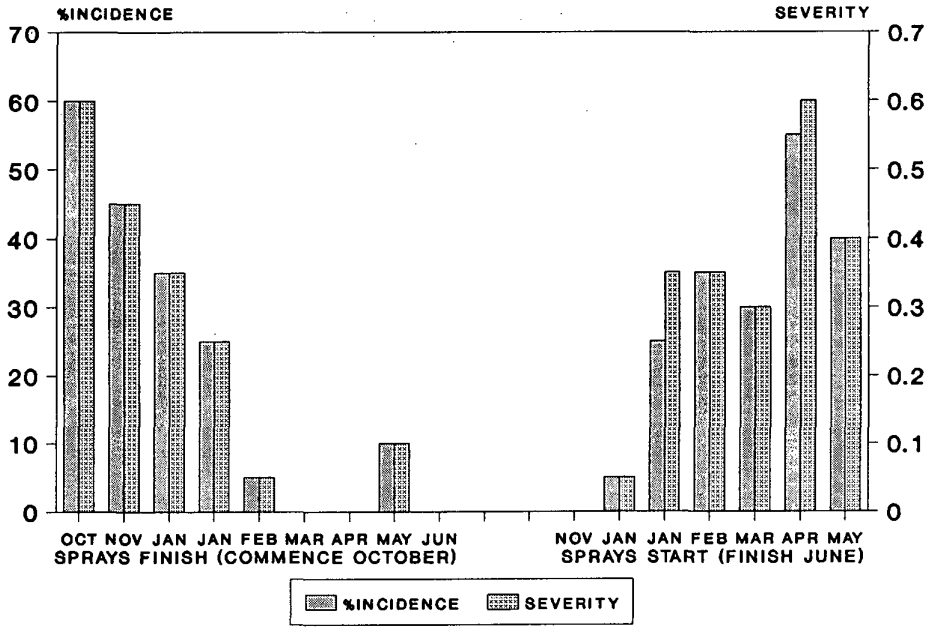


FIGURE 10C :THURLOXTON YIELD 1994 (T/HA)

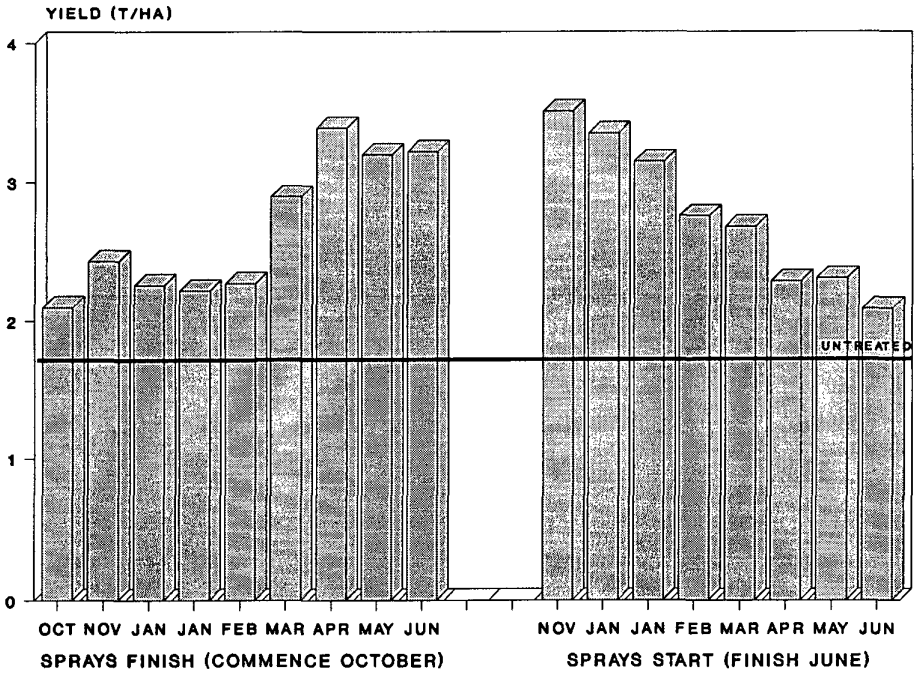
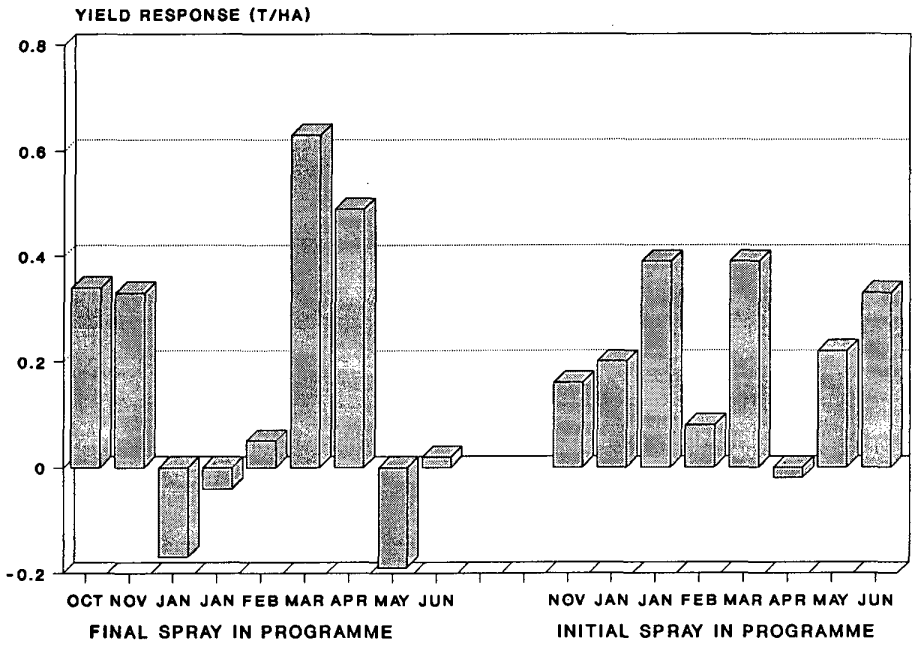


FIGURE 10D : THURLOXTON YIELD 1994
EFFECT OF INDIVIDUAL SPRAY TIMINGS



1.1 Canker summary

Data from the sites where *Phoma* leaf spot and canker developed are summarised in Tables 1.1 a, b, c and d.

The development and control of the leaf spot phase of the disease is summarised in Table 1.1 a. *Phoma* leaf spot developed at ten of the fourteen experiment sites between 1991 and 1994. At six of these sites light leaf spot was also present at significant levels. First symptoms of the leaf spot phase of canker were seen at five sites in October, four sites in November, and at one site (Rosemaund 1993/94) in December.

Foliar disease in untreated plots exceeded 1.0 per cent leaf area affected before or during January at Boxworth, Rothamsted, and Withington (1992/93), and Thurloxton (1993/94). At two of the remaining six sites, disease severity exceeded 1.0 per cent by February at Tarrant Hinton (1992/93) and Rosemaund (1993/94). At Kington Langley and Rothamsted (1991/92) disease severity reached its maximum during January, but was not severe at 0.1 and 0.3 per cent respectively. At Rothamsted (1993/94) disease severity never exceeded 0.1 per cent; this level of disease was recorded during November. At Boxworth (1993/94) disease severity reached a maximum of 0.8 per cent during May, however the percentage of plants affected was high at all sites at certain times during the season.

Foliar disease was controlled at all sites except Kington Langley and Rothamsted (1991/92). The best control was achieved with one or two sprays applied mainly between November and February, although October sprays had some effect at Rosemaund and Thurloxton in 1993/94.

The development and control of canker is summarised in Table 1.1 b. Canker developed at all of the sites where the leaf spot phase had been detected. The earliest

time that canker was observed was during December 1992 at Boxworth. However, the principal development of canker at this and all other sites was during May and June coinciding with the end of flowering and start of pod development and seed expansion. The incidence of canker in untreated plots at all sites at the final disease assessment exceeded 50 per cent of plants affected; the lowest incidence (50 per cent) occurred at Thurloxton (1993/94), the highest at Rothamsted in 1992/93 and 1993/94, when 100 per cent of plants were affected by canker. Disease severity was also at its lowest at Thurloxton (0.50) and at its highest at Rothamsted (2.60 (1992/93) and 2.95 (1993/94)). Canker was controlled at all sites by one or two fungicide sprays applied mainly between November and February, although October, March, and April sprays had some effect at some sites.

The relationship between canker and yield expressed as regression equations is summarised in Table 1.1 c. The disease did not affect yield at two sites (Kington Langley and Rothamsted, 1991/92). Yield losses were related to the incidence and severity of canker at the remaining eight sites including those sites where light leaf spot developed. At four of the six sites where light leaf spot developed, light leaf spot also affected yield, and further work is required to separate the effect of the presence of both diseases on yield.

Table 1.1 d summarises the yield losses associated with canker. By extrapolation of the regression equations, it was possible to determine the effect of canker on yield at each site when 100 per cent of plants were affected by stem disease; also yield losses associated with a severity score of 1 are quoted. At Tarrant Hinton (1992/93) and Rothamsted (1993/94) the relationship between the incidence and severity of canker and yield expressed by the correlation coefficient (r) was weak and yield losses associated with disease were the lowest found. Aerial and basal cankers appeared to cause similar losses at Boxworth in 1992/93 and 1993/94 when comparing disease incidence. However, in 1992/93 aerial cankers caused a much greater loss in yield compared to basal cankers when comparing the effect of disease severity (0.63 t/ha

lost per severity score of 1 compared to 0.37 t/ha for basal cankers). At Rothamsted in 1992/93, 0.8 t/ha was lost per 100 per cent stems affected and the effect of disease severity was similar to that for basal cankers at Boxworth in 1992/93, (0.363 t/ha lost per severity score of 1).

At Rosemaund and Thurloxton (1993/94) the disease caused the most damage to yield. By extrapolation 1.3 and 2.0 t/ha respectively were lost per 100 per cent stems affected, and 0.78 and 1.81 t/ha for a mean severity score of 1.

In general the greatest yield losses were associated with sites where the mean foliar severity in untreated plots was $\geq$ 1.0 per cent area affected before or during January; the exception was Boxworth in 1993/94 where the maximum disease severity before or during January was 0.3, and only reached a maximum during the season of 0.8 during May.

Regression analysis of the relationship between the yield loss associated per 1 per cent stem incidence (Y) versus the yield loss associated per severity score of 1 (X) was extremely strong and significant. The equation is shown below:-

$$Y = 0.268 + 0.961 X$$

The relationship between Y and X was strong, with a correlation coefficient r of 0.94 which was highly significant ($p < 0.001$). The value of the slope b in the equation was significantly different from zero at a probability of 99.99 per cent confirming the strength of the relationship between canker incidence and severity.

At the four sites where canker developed but light leaf spot did not, canker did not affect yield at Kington Langley and Rothamsted (1991/92), but between 0.6 and 0.9 t/ha was lost per 100 per cent plants affected at Boxworth (1993/94) and Withington

(1992/93) respectively. At the six sites where both diseases developed, canker affected yield at all sites and light leaf spot at four (see Section 2.1).

At the eight sites where yield was related to the incidence or severity of canker, light leaf spot was present at six of the sites and affected yield at four (Table 1.1c). At these four sites the maximum percentage yield increase in response to treatment and control of both diseases ranged between 21 per cent (Rothamsted, 1993/94), and 99 per cent (Thurloxton 1993/94), with 47 and 71 per cent response in yield at Rothamsted (1992/93) and Rosemaund (1993/94). At the two sites where light leaf spot developed but did not affect yield the maximum yield increase in response to treatment was 21 and 23 per cent (Tarrant Hinton and Boxworth, 1992/93). At the two sites where only canker developed the maximum yield increase in response to treatment was 24 and 40 per cent (Withington 1992/93 and Boxworth 1993/94).

Table 1.1a Summary of *Phoma* leaf spot incidence and severity on untreated plots and the timing of fungicide for control

Site	Year	*Phoma* leaf spot					
		First symptoms	Maximum leaf severity†			Treatments to control disease	
		Month	%I*	%S**	Month	Control Y/N	Timings Month(s)
Kington Langley	1991/92	October	49	0.1	January	No	-
Rothamsted	1991/92	October	56	0.3	January	No	-
Boxworth[d]	1992/93	October	98	4.8	January	Yes	December-February
Rothamsted[de]	1992/93	October	95	1.9	October	Yes	November-December
Tarrant Hinton[d]	1992/93	October	60	2.0 (0.2 Jan)	February	Yes	December-January
Withington	1992/93	November	75	3.9 (2.1 Jan)	February	Yes	December-February
Boxworth	1993/94	November	98	0.8 (0.3 Jan)	May	Yes	November-January
Rosemaund[de]	1993/94	December	50	1.4	February	Yes	October
Rothamsted[de]	1993/94	November	17	0.1	November	Yes	January
Thurloxton[de]	1993/94	November	65	2.9 (2.3 Jan)	February	Yes	October-January

† Untreated plots

* % Incidence of plants affected during the month with maximum severity

** Maximum % leaf area affected; where disease severity reached its maximum after January, the maximum severity between October and January is shown in brackets.

[d] = Sites with light leaf spot

[de] = Sites where light leaf spot affected yield

Table 1.1b Summary of canker incidence and severity on untreated plots and the timing of fungicide for control

Site	Year	Canker				
		First Symptoms		Pod ripening disease †		Treatment to control disease
		Month	GS	%I	S	Month
Kington Langley	1991/92	May	4.9/5.7	79	-	February (Oct-Feb)
Rothamsted	1991/92	June	6.3	94	2.50	October-February
Boxworth[d]	1992/93	December	1.09	98[b] 70[a]	1.60[b] 0.95[a]	November-February
Rothamsted[de]	1992/93	June	6.3	100	2.60	January (Oct-Jan)
Tarrant Hinton[d]	1992/93	April	4.0/4.1	95	1.98	January (Oct-Jan)
Withington	1992/93	May	5.3	60	1.60[c]	December-February
Boxworth	1993/94	May	4.8/5.5	65[b] 90[a]	0.80[b] 0.95[a]	January (Nov-Jan)
Rosemaund[de]	1993/94	June	4.8/6.2	75	1.25	October-February
Rothamsted[de]	1993/94	June	6.1	100	2.95	February (Dec-April)
Thurloxton[de]	1993/94	June	6.3	50	0.50	January-February

† = Untreated plots
[a] = Aerial stem lesions
[b] = Basal cankers
[c] = Penetrating lesions
[d] = Sites with light leaf spot
[de] = Sites where light leaf spot affected yield

Table 1.1c Summary of canker and its relationship with yield

Site	Year		Yield and canker*		Maximum % yield response **
		Disease related Y/N	Regression equation Y = Yield (t/ha) X = % Incidence	Regression equation Y = Yield (t/ha) X = Severity	
Kington Langley	1991/92	N	-	-	-
Rothamsted	1991/92	N	-	-	-
Boxworth[d]	1992/93	Y	$Y = 4.58 - 0.007 X^b$ (-0.79) $Y = 4.47 - 0.008X^a$ (-0.74)	$Y = 4.45 - 0.373X^b$ (-0.75) $Y = 4.45 - 0.632X^a$ (-0.74)	23
Rothamsted[de]	1992/93	Y	Y = 4.81 - 0.008X (-0.76)	Y = 4.77 - 0.363X (-0.80)	47
Tarrant Hinton[d]	1992/93	Y	Y = 3.45 - 0.002X (-0.45)	Y = 3.44 - 0.010X (-0.47)	21
Withington	1992/93	Y	Y = 3.18 - 0.009X (-0.81)	-	24
Boxworth	1993/94	Y	$Y = 2.43 - 0.007 X^b$ (-0.75) $Y = 2.50 - 0.006X^a$ (-0.84)	$Y = 2.44 - 0.616X^b$ (-0.80) $Y = 2.50 - 0.621X^a$ (-0.86)	40
Rosemaund[de]	1993/94	Y	Y = 2.82 - 0.013X (-0.71)	Y = 2.78 - 0.780X (-0.71)	71
Rothamsted[de]	1993/94	Y	Y = 3.78 - 0.005X (-0.63)	Y = 3.70 - 0.195X (-0.57)	21
Thurloxton[de]	1993/94	Y	Y = 3.18 - 0.020X (-0.75)	Y = 3.16 - 1.81X (-0.72)	99

* Values of r follow equation (r)

** = (Maximum yield - Untreated yield/Untreated yield) x 100

a = Aerial stem lesions

b = Basal cankers

c = Penetrating lesions

d = Sites with light leaf spot

de = Sites where light leaf spot also affected yield

Table 1.1d Summary of yield losses associated with canker and the relationship with *Phoma* leaf spot severity during the winter

Site	Year	Maximum *Phoma* leaf spot severity before/during January	Yield loss (t/ha)/100% canker (r)*	Yield loss (t/ha)/severity score(r) *
Kington Langley	1991/92	0.1	-	-
Rothamsted	1991/92	0.3	-	-
Tarrant Hinton [d]	1992/93	0.2	0.2 (-0.45)	0.010 (-0.47)
Rothamsted [de]	1993/94	0.1	0.5 (-0.63)	0.195 (-0.57)
Boxworth	1993/94	0.3	0.7[b] (-0.75) 0.6[a] (-0.84)	0.616[b] (-0.80) 0.621[a] (-0.86)
Boxworth [d]	1992/93	4.8	0.7[b] (-0.79) 0.8[a] (-0.74)	0.373[b] (-0.75) 0.632[a] (-0.74)
Rothamsted [de]	1992/93	1.9	0.8 (-0.76)	0.363 (-0.80)
Withington	1992/93	2.1	0.9 (-0.81)	**
Rosemaund [de]	1993/94	1.4	1.3 (-0.71)	0.780 (-0.71)
Thurloxton [de]	1993/94	2.3	2.0 (-0.75)	1.810 (-0.72)

- \- No relationship
- * Value of the correlation coefficient (r) follows yield loss
- ** No values, but cankers were mainly penetrating at Withington

a = Aerial stem lesions
b = Basal stem lesions
d = Sites with light leaf spot
e = Sites where light leaf spot affected yield

2. Light leaf spot

Light leaf spot developed at nine sites during the course of the experiment. Disease development in untreated plots and the effect of fungicide treatment on disease and yield at affected sites is detailed below.

(i) Foveran 1991/92

The development of foliar light leaf spot in untreated plots is illustrated in Figure 11. The disease was first detected at low levels on 30 December. Symptoms increased during the winter reaching 76 per cent plants affected on 28 March (1.7 per cent leaf area); by 24 April (GS 4.0) 70 per cent of plants were affected at 2.2 per cent leaf area. Thereafter light leaf spot symptoms declined.

Light leaf spot development in untreated plots on stems and pods is illustrated in Figure 11A. Stem infection was first seen on 24 April when 30 per cent of plants were affected; the disease affected 0.9 per cent of the stem area. Symptoms developed and reached a maximum on 3 August (GS 6.4/6.5) when 100 per cent plants were severely affected at 20.4 per cent stem area. Pod symptoms were first detected on 6 July when 60 per cent of plants were affected at 1.1 per cent stem area. An increase in sooty moulds on prematurely ripened pods occurred so that by 3 August (GS 6.4 / 6.5) only 27 per cent of untreated plants were affected by light leaf spot (4.2 per cent pod area) compared to 72 per cent affected by sooty moulds (65.2 per cent pod area). This reduction in light leaf spot incidence was probably due to an increase in sooty mould incidence which may have masked light leaf spot symptoms.

Significant reductions in the incidence and severity of foliar light leaf spot were detected on 24 April (GS 4.0). Of the treatments that received their first spray on 9 October (2 to 12) and finished progressively later, significant reductions in disease incidence from 70 per cent to $\leq$ 15 per cent occurred where the final treatment occurred on or after 26 November (Treatments 3 to 12); the single October spray (Treatment 2, 9 October) was not effective. Similar control occurred where the initial

spray in treatments started between 7 November and 26 February (Treatments 23 to 18); Treatment 17 received an initial spray on 28 March and was not effective by 24 April.

Figure 11B and 11C illustrate the incidence and severity respectively of light leaf spot infection on stems and pods on 3 August (GS 6.5) in treated plots. Significant reductions in the incidence of stem disease occurred with Treatments 4, 6 to 11, and 17 to 22. Where treatments began in the autumn (9 October) finishing progressively later, the most effective (reducing the incidence to ≤ 31 per cent) were those that continued to be sprayed up until 26 February (GS 1.9/1.11) or later (Treatments 7 to 11). Of the treatments that began progressively later only those that began in November or December reduced disease incidence to ≤ 30 per cent (22 to 20). Significant reductions in stem disease severity were obtained from the same set of treatments as those that affected incidence with the addition of Treatments 3 and 5. The most effective treatment was Treatment 8 which received monthly sprays from October until April; only 5 per cent of plants were affected by stem disease in August with a severity of 0.1 per cent.

Twenty-seven per cent of plants were affected by light leaf spot on the pods in the untreated plots on 3 August (GS 6.5) with a severity of 4.2 per cent. All of the treated plots had a greater incidence of light leaf spot on the pods than the untreated and this was significant for Treatments 4, 6, 7, 8, 14, 15 and 16. Disease severity was also enhanced by some treatments but whilst there were significant differences between treatments there was no significant difference between untreated and treated plots. Premature ripening of the pods was significantly reduced from 85.2 per cent to ≤ 58.6 per cent by all treatments except for those receiving single sprays only (2 and 14). The most effective treatments were 6 to 11, and 17 to 22, reducing premature ripening to ≤ 25 per cent pods affected. These were the same treatments that resulted in a significant reduction in the incidence of light leaf spot on the stem.

The untreated yield at this site was 3.36 t/ha. Figure 11D and 11E show the yields obtained from all of the treatments and the responses attributable to individual spray

timings obtained by subtraction of yields from related treatments. Yield responses were obtained from all of the treatments with the exception of the single spray applied at pod ripening (Treatment 14). However, significant differences in yield were not obtained (yield data were significant at $p = 0.09$).

The largest overall response in yield came from Treatment 8 (1.37 t/ha greater than the untreated) which received seven sprays between 9 October (GS 1.01 / 1.08) and 28 March (GS 3.3 / 3.5). This treatment had the lowest incidence of light leaf spot on the stems (5 per cent).

The greatest contribution to yield from individual timings came from the 7 November spray when applied as the final treatment in a two-spray programme (0.73 t/ha, Treatment 3, Figure 11E). The incidence and severity of light leaf spot on the stems in this treatment was 25 and 8.1 per cent lower respectively compared to the single treatment on 11 October (Treatment 2). These differences in disease were not significant.

The greatest difference in disease incidence by the addition of one spray to a treatment was obtained with the 26 February timing (Treatment 7) which had 45 per cent less plants affected by light leaf spot on the stems than Treatment 6. This was not reflected in yield however (0.32 t/ha less than Treatment 6; both these differences were significant).

Regression analyses of the incidence and severity of light leaf spot on the stem on 3 August (GS 6.4 / 6.5) and other factors in relation to yield are presented in Table 13.

Table 13. Regression analyses of yield (t/ha) (Y) versus disease and physical factors (X) on 3 August (GS 6.4/6.5)

X parameter	Regression equation	Correlation coefficient(r)*
% Stem light leaf spot incidence	Y = 4.61 - 0.00944X	-0.78
% Stem light leaf spot severity	Y = 4.39 - 0.0622X	-0.88
Logit % Stem light leaf spot severity**	Y = 3.33 - 0.0360X	-0.78
% Pod premature ripening	Y = 4.46 - 0.0114X	-0.79
% Plant premature ripening	Y = 5.19 - 0.135X	-0.53
Plant height (cm)	Y = 1.54 + 0.0342X	+0.54

* All correlation coefficients significant at $p \leq 0.02$

** Data skew, but restored to normality by logit transformation

The relationship between yield and disease incidence and severity on the stem at the final assessment was strong and highly significant ($p \leq 0.001$ for both values of r). In addition the relationship between yield and premature ripening of the pods was also strong and significant, though that between yield and plant premature ripening or height was not so strong (r = -0.53 and + 0.54 respectively).

For every 1 per cent of stems affected at pod ripening, a loss in yield of 0.009 t/ha occurred. This was similar to yield losses associated with canker (Section 1).

FIGURE 11 : FOVERAN 1991/92
LIGHT LEAF SPOT DEVELOPMENT

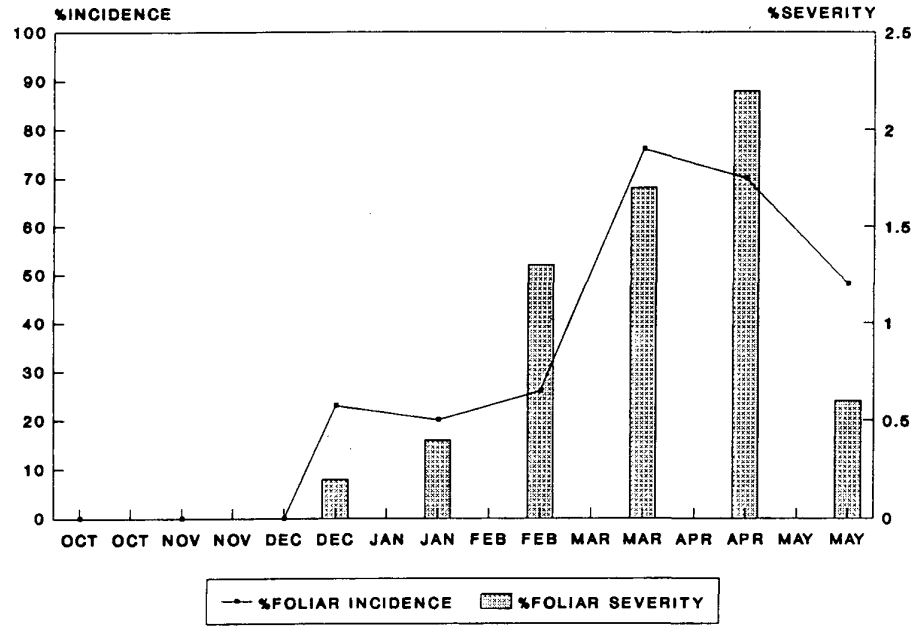


FIGURE 11A : FOVERAN 1991/92
STEM AND POD LIGHT LEAF SPOT

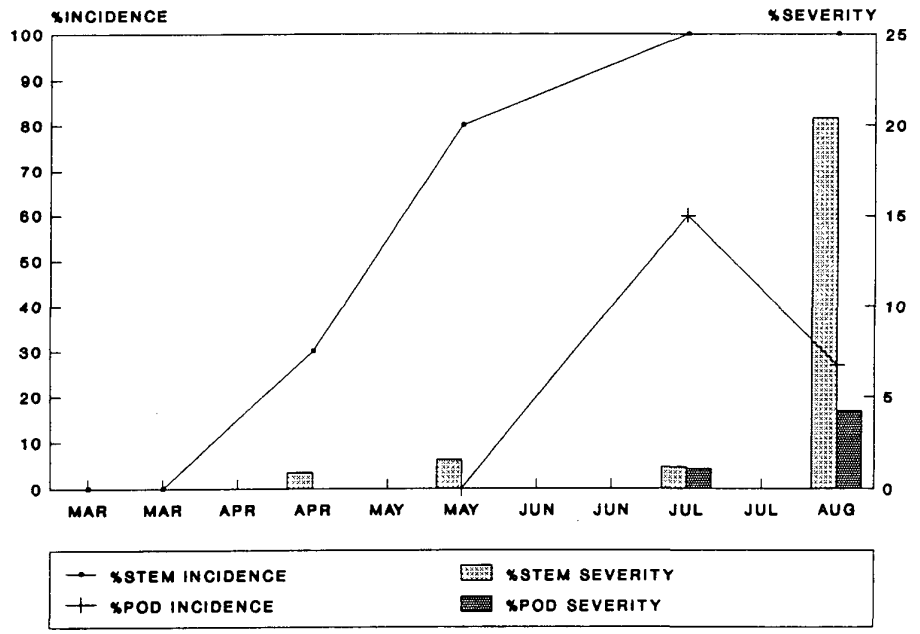


FIGURE 11B : FOVERAN LIGHT LEAF SPOT
3 AUGUST 1992, GS6.5

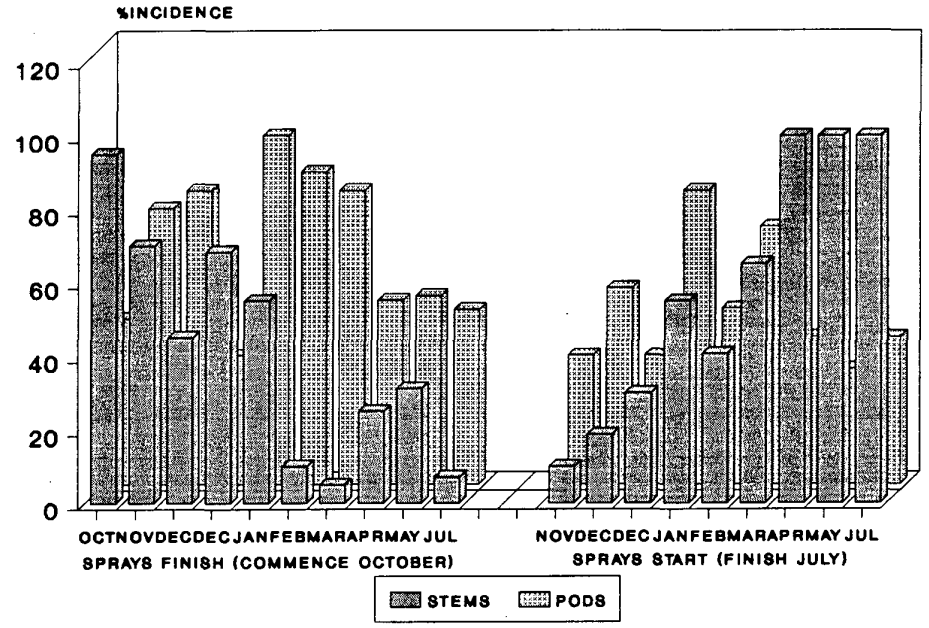


FIGURE 11C : FOVERAN LIGHT LEAF SPOT
3 AUGUST 1992, GS6.5

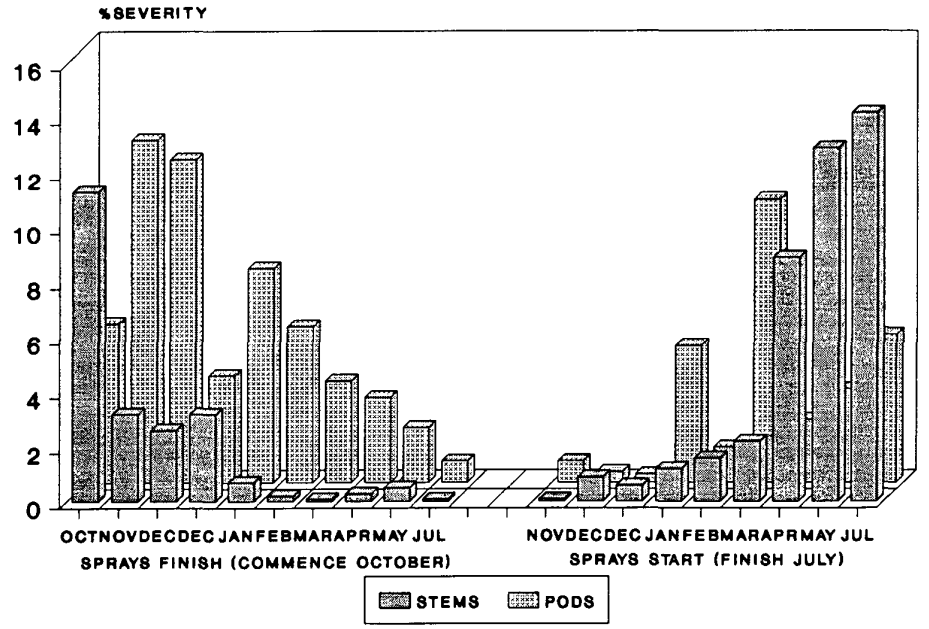


FIGURE 11D : FOVERAN YIELD 1992 (T/HA)

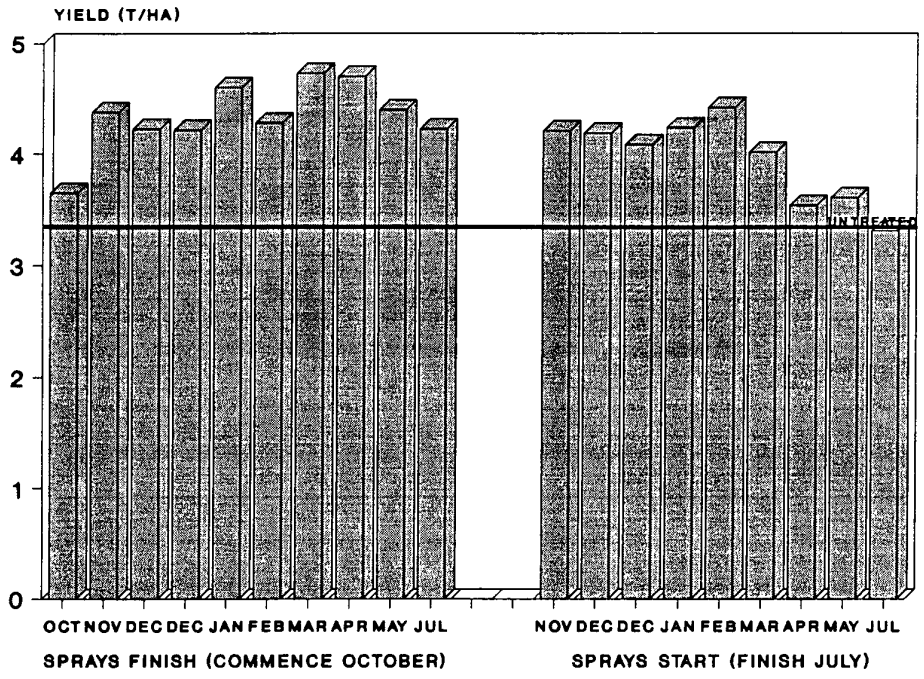
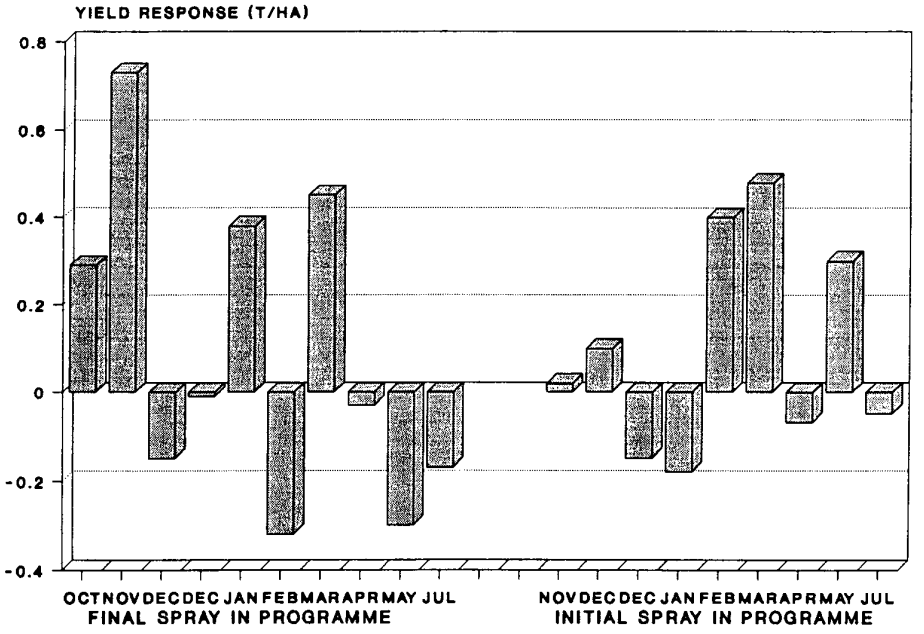


FIGURE 11E : FOVERAN YIELD 1992
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(ii) Boxworth 1992/93

Canker was the main disease at this site (Section 1(iii)) but light leaf spot also developed. Figure 12 illustrates the development of foliar light leaf spot in untreated plots. The disease was first detected on 21 January (GS 1.12) when 15 per cent of the untreated plants were affected, the disease was not severe at this time affecting only 0.14 per cent of the leaf area. Light leaf spot developed in the spring, 73 per cent of untreated plants were affected on 17 March (GS 2.05/3.3) when the disease was at its most severe (8 per cent leaf area). By 8 April (GS 3.7) disease incidence was at its highest at 83 per cent of untreated plants but disease severity had declined to 4 per cent leaf area.

Light leaf spot developed on the stems (Figure 12A) but not the pods. Stem symptoms were first seen on 8 April (GS 3.7) when 3 per cent of untreated plants were affected but the disease was not severe (0.6 per cent stem area). The disease increased steadily; by 28 June (GS 6.4) 65 per cent of stems were affected in untreated plots but symptoms were still slight (3.6 per cent stem area).

The majority of data pertaining to foliar light leaf spot was skew and could not be transformed to normality; however, significant reductions in foliar disease incidence were detected on 8 April (GS 3.7) when disease incidence in untreated plots was at its highest (83 per cent) whilst disease severity had declined to 4 per cent. Treatments 2 to 8 which had received an initial spray on 19 October and further sprays finishing between 19 October and 11 March (GS 2.05 / 3.3) and Treatments 22, 21 and 20 which received initial sprays on 6 November, 10 December, and 18 January respectively all had significantly less disease ($\leq$ 35 per cent incidence) at this time. Treatments 19, 18, and 17 which began treatment on or after 1 February were not significantly different from the untreated ($\geq$ 55 per cent incidence). Thus sprays applied between 19 October and 18 January were significantly effective against foliar light leaf spot on 8 April.

Figure 12B illustrates the incidence and severity of light spot on the stems in treated plots on 28 June (GS 6.4). Sixty-five per cent of untreated plants were affected but symptoms were not severe (3.6 per cent stem area). Data relating to this assessment were skew and remained so after transformation, however, Treatments 2 to 12 (initial spray in October, finishing progressively later) and 22, 21 and 20 (initial spray November, December, January) appeared to lead to reductions in incidence and severity to $\leq$ 20 and 0.2 per cent respectively. Thus sprays applied between October and January were most effective against both foliar and stem light leaf spot.

The untreated yield at this site was 3.81 t/ha. Figure 12C and 12D illustrate the yields obtained from all of the treatments and the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. Yields were primarily related to the control of canker (Section 1(iii)) and not light leaf spot.

FIGURE 12 : BOXWORTH 1992/93
LIGHT LEAF SPOT DEVELOPMENT

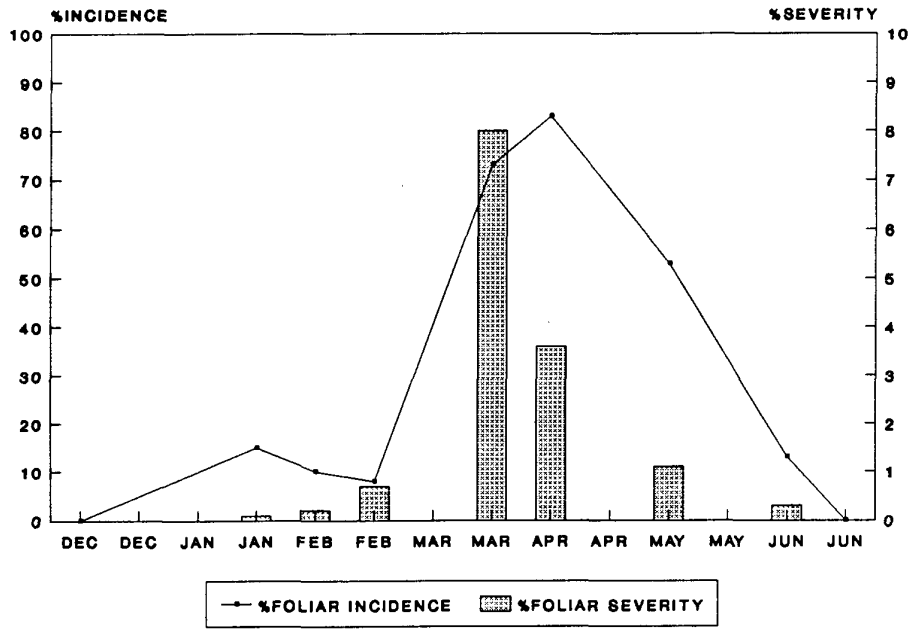


FIGURE 12A : BOXWORTH 1992/93
STEM LIGHT LEAF SPOT

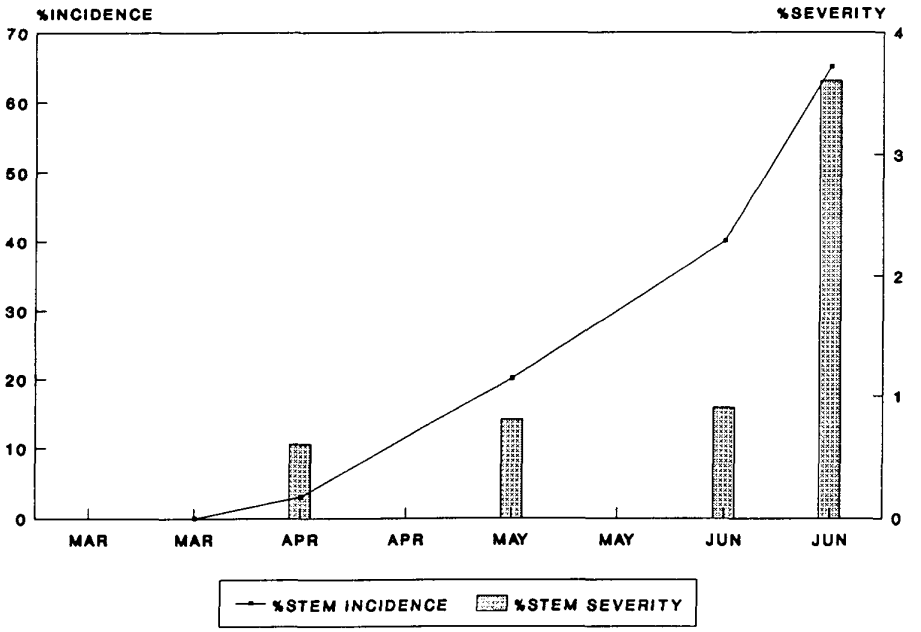


FIGURE 12B : BOXWORTH LIGHT LEAF SPOT
STEM DISEASE, 28 JUNE, GS6.4

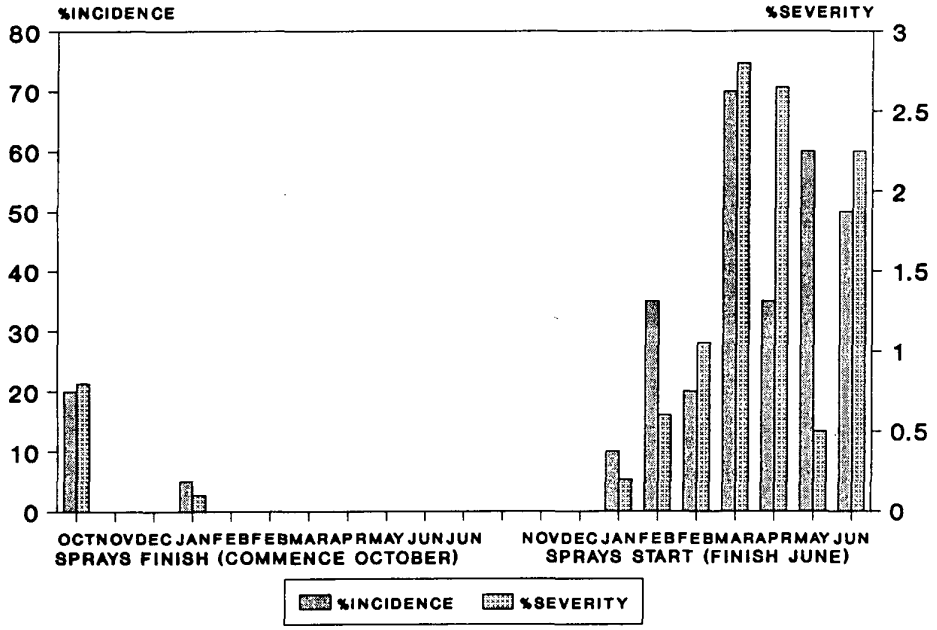


FIGURE 12C : BOXWORTH YIELD 1993 (T/HA)

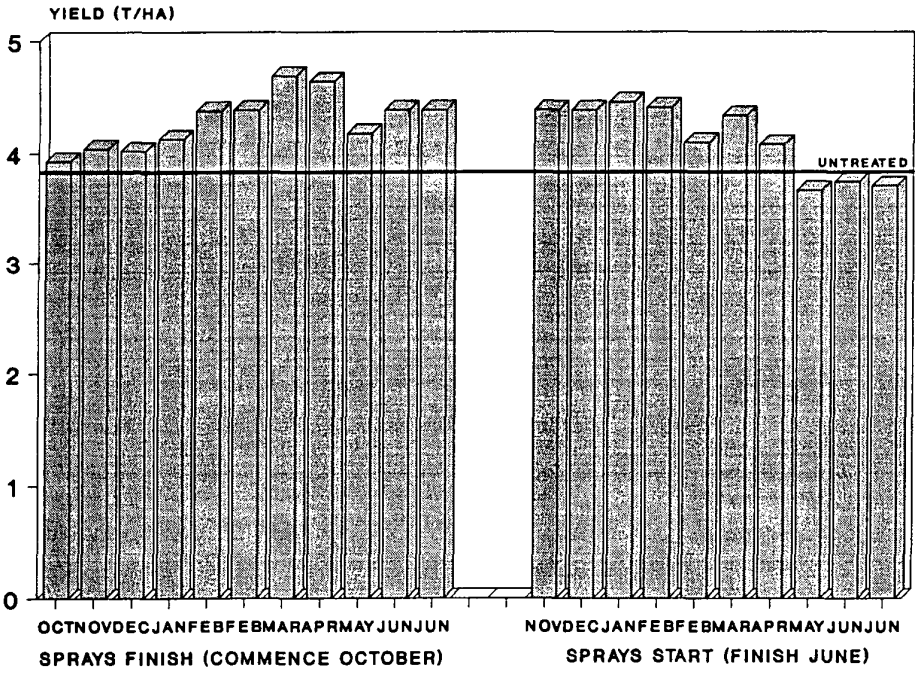
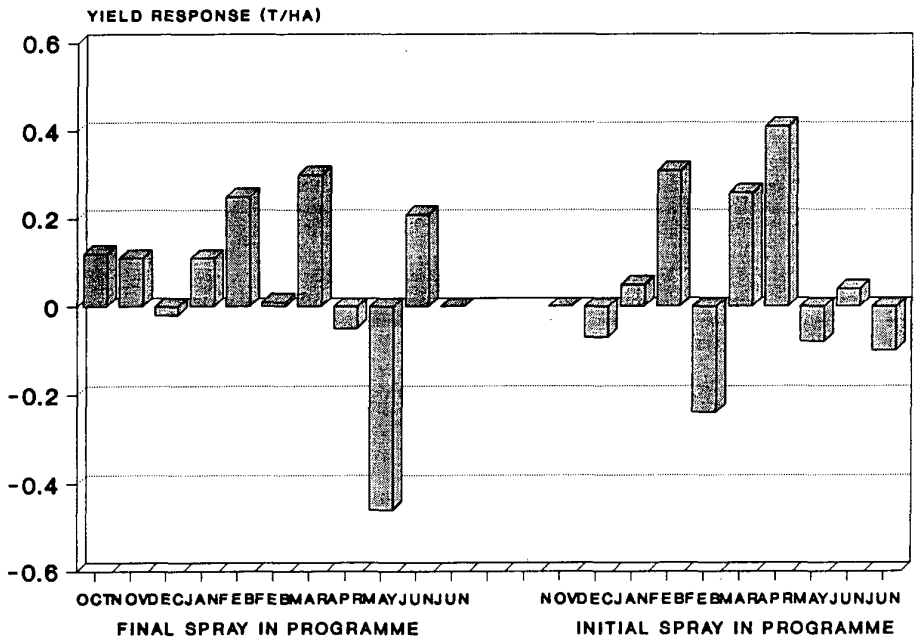


FIGURE 12D : BOXWORTH YIELD 1993
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(iii) Pettymuick 1992/93

The development of foliar light leaf spot in untreated plots is illustrated in Figure 13. The disease was first detected on 8 December (GS 1.08 / 1.09) when 50 per cent of untreated plants were affected (0.76 per cent leaf area). By 21 January, 95 per cent of untreated plants were severely affected at 7.7 per cent leaf area. The disease reached its maximum at the end of March (GS 3.3) when 100 per cent of untreated plants were affected at 21.2 per cent leaf area. Thereafter symptoms decreased.

Light leaf spot development on stems and pods in untreated plots is illustrated in Figure 13A. Stem symptoms were first seen on 1 May (GS 3.5 / 4.0) when 95 per cent of untreated plants were affected (3.2 per cent stem area). By 27 July (GS 6.1) 97 per cent of stems were affected; disease severity had increased to 8 per cent stem area in untreated plots. Pod symptoms were only detected on 27 July (GS 6.1) when 95 per cent of pods were affected at a severity of 2.5 per cent pod area.

Significant effects of treatments on foliar disease were first observed on 18 January (GS 1.08 / 1.10). Treatments 2, 3, 4, 10, 21 and 22 had less than 36 per cent of plants affected by light leaf spot compared to 95 per cent in untreated plots. The treated plots had all received at least one spray application between October and early December. Similarly in February Treatments 2 to 5, 10, and 20 to 22 had significantly less plants affected by the disease when compared to untreated plots. The most effective treatments (4, 5, and 10) controlled the disease completely at this time and had received monthly sprays between October and December or January. By the end of March however when the disease had reached maximum levels (100 per cent incidence, 21.2 per cent severity in untreated plots) the effect of the early treatments had declined with only Treatments 6 and 10 having a significant effect on disease incidence. These treatments had received five monthly sprays between October and February. Reductions in disease severity were also found. On 29 March (GS 3.3) Treatments 6, 10, and 20 to 22 had significantly reduced symptoms from 21.2 per cent leaf area to $\leq$ 3 per cent. These treatments had all received at least one spray between

17 November and 18 January. By 1 May (GS 3.5 to 4.0) none of the treatments remained significantly effective against disease incidence but all were effective against disease severity. The best treatments (6, 7, 10, 21, and 22) reduced the disease to ≤ 2.6 per cent leaf area compared to 12.6 per cent in the control plots.

Figure 13B and 13C illustrate the incidence and severity respectively of light leaf spot infection on stems and pods on 27 July, (GS 6.1) in treated plots. Ninety-seven per cent of untreated plants were affected by light leaf spot infection of the stem at 8 per cent stem area. Of the treatments that received their first spray applications in October finishing progressively later, significant reductions in the incidence of stem disease were obtained from Treatments, 7, 9, and 10 (≤ 50 per cent of stems affected). All of these treatments had received their final spray on or after 25 March (GS 3.3). Treatments which finished on 14 July (GS 6.1) and started progressively earlier had significantly lower disease incidence (≤ 45 per cent stems affected) compared to untreated plots, where the initial spray application was made on 17 November (GS 1.07/1.09, Treatment 22) or 18 January (GS 1.08/1.10, Treatment 20). Treatments starting later than 18 January were not effective in reducing the incidence of stem infection.

Significant reductions in stem disease severity were obtained from most of the treatments that received their first spray application on 9 October (GS 1.04) finishing on 17 November (GS 1.07/1.09) or later. Severity was reduced from 8 to 2.4 per cent or less when assessed on 14 July (GS 6.1). Of the treatments that started progressively later only those that commenced between 17 November and 18 January (Treatments 22 to 19) led to significant reductions to ≤ 1.5 per cent stem area on 14 July.

Ninety-five per cent of pods were affected by light leaf spot in untreated plots with a severity of 2.5 per cent pod area. Treatments 9 and 10 (commenced application on 9 October and finished on 8 June or 14 July), 20 and 22 (commenced 18 January or 17 November and finished on 14 July) led to significant reductions in the incidence of pod

light leaf spot from 95 per cent to ≤ 45 per cent. The same treatments led to significant reductions in the severity of infection with the addition of Treatments 3, 7, and 8, to ≤ 0.85 per cent.

The untreated yield at this site was 2.22 t/ha. Figure 13D and 13E illustrate the yields obtained from all of the treatments and the responses attributable to individual spray timings obtained by subtraction of yields from related treatments.

Yield responses significantly greater than the untreated were obtained from Treatments 4, 6 to 10, and 18 to 22.

Treatments which began on 9 October and ended between 9 October and 14 July (Treatments 2 to 10) gave responses of between 0.36 and 1.68 t/ha. The best responses occurred where the final sprays were applied in February or later (Treatments 6 to 10) ranging from 1.32 to 1.68 t/ha. The spray timing common to all of these treatments was 11 February, which, by subtraction (Treatment 6 - 5) gave a response of 1.08 t/ha as the final spray in a treatment (Figure 13E).

Treatments which ended on 14 July and started progressively earlier (Treatments 16 to 22) gave responses of between 0.31 and 1.88 t/ha. The best responses occurred where the initial spray was applied between November and March (Treatments 22 to 18) ranging between 1.10 and 1.88 t/ha. The spray timing common to all of these treatments was 25 March, which, by subtraction (Treatment 19 - 18) gave a response of 0.79 t/ha as the initial spray in a treatment (Figure 13E).

Both final and initial sprays made in January appeared (by subtraction) to be damaging to yield (-0.40 and -0.15 t/ha respectively) (Figure 13E) possibly because the ambient air temperatures at the time of spraying were quite low (3.9 and 4.2^{0}C). However, in general sprays applied between October and March were most likely to lead to a positive response in yield.

Yield responses were, in general, related to the control of light leaf spot since treatments which included sprays applied up to at least 11 February (GS 1.12) (Treatments 6 to 10) or with the first application up to or including 18 January (GS 1.12) (Treatments 20 to 22) gave the best reductions in both the incidence and severity of stem infection and the best yield responses. The relationship between yield and the incidence of pod infection was fairly similar. The best reductions in the incidence of pod infection were obtained from treatments that began on 9 October and finished on 25 March or later (Treatments 7 to 10) or from treatments that received their initial spray between 17 November and 18 January (Treatments 20 to 22) but no later. However, the effect of treatment on the severity of pod symptoms was less clear cut. Whilst the best reductions in symptoms also came from Treatments 7 to 10 and 20 to 22 an additional treatment (3) which received a spray in October and November only, also led to significant reductions in disease severity but gave a non-significant yield response of 0.57 t/ha.

The results of regression analyses of the incidence and severity of stem and pod light leaf spot infection in July with yield are shown in Table 14.

Table 14. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of stem or pod light leaf spot (X) assessed in July (GS 6.1)

X parameter	Regression equation	Correlation coefficient(r)*
% Stem incidence	Y = 4.28 - 0.015X	-0.67
% Stem area	Y = 3.50 - 0.12X	-0.64
% Pod incidence	Y = 4.33 - 0.017X	-0.67
% Pod area	Y = 3.83 - 0.449X	-0.67

* $p \leq 0.001$ for all values of r .

The relationship of the incidence and severity of both stem and pod light leaf spot infection with yield was relatively strong with correlation coefficients ranging between -0.64 and -0.67, all of which were highly significant ($p \leq 0.001$). The value of the slope b in each equation was significantly different from zero at a probability of 99.99 per cent confirming the strength of the relationship between light leaf spot and yield. For every 1 per cent increase in the incidence of either stem or pod infection there was a loss in yield of approximately 0.016 t/ha. For every 1 per cent increase in stem or pod area affected there was a loss in yield of 0.12 or 0.45 t/ha respectively.

FIGURE 13 : PETTYMUICK 1992/93
LIGHT LEAF SPOT DEVELOPMENT

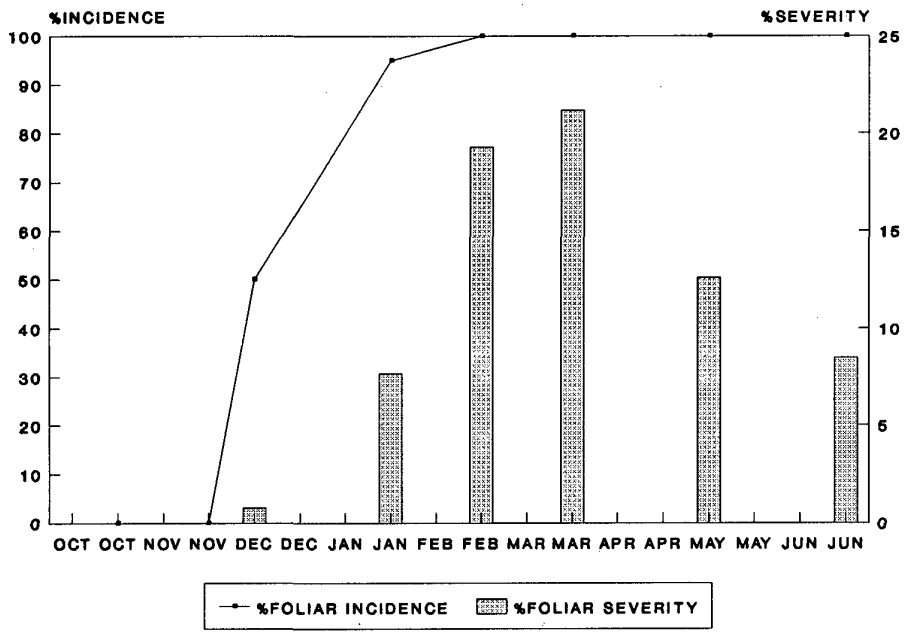


FIGURE 13A : PETTYMUICK 1992/93
STEM AND POD LIGHT LEAF SPOT

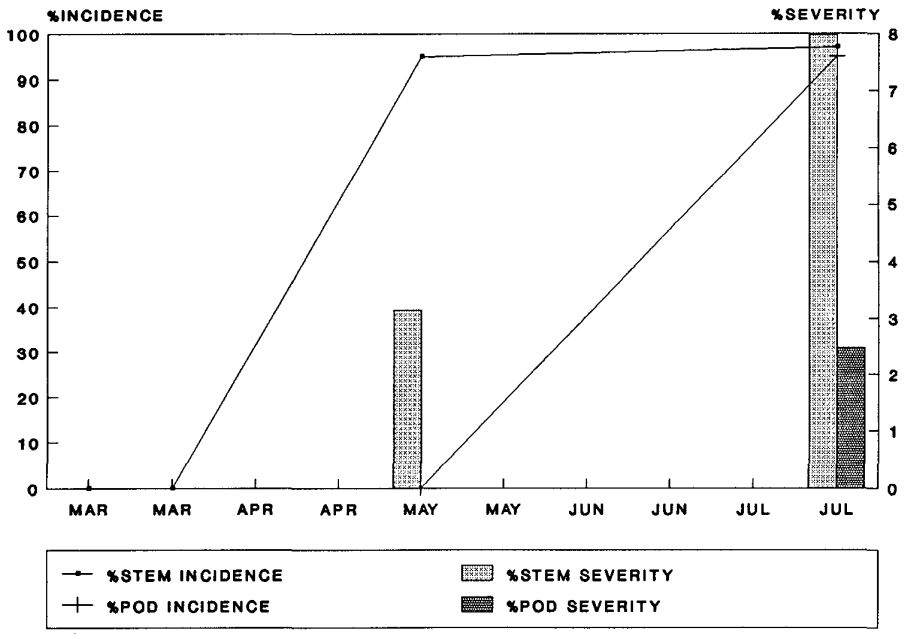


FIGURE 13B : PETTYMUICK LIGHT LEAF SPOT
27 JULY 1993, GS6.1

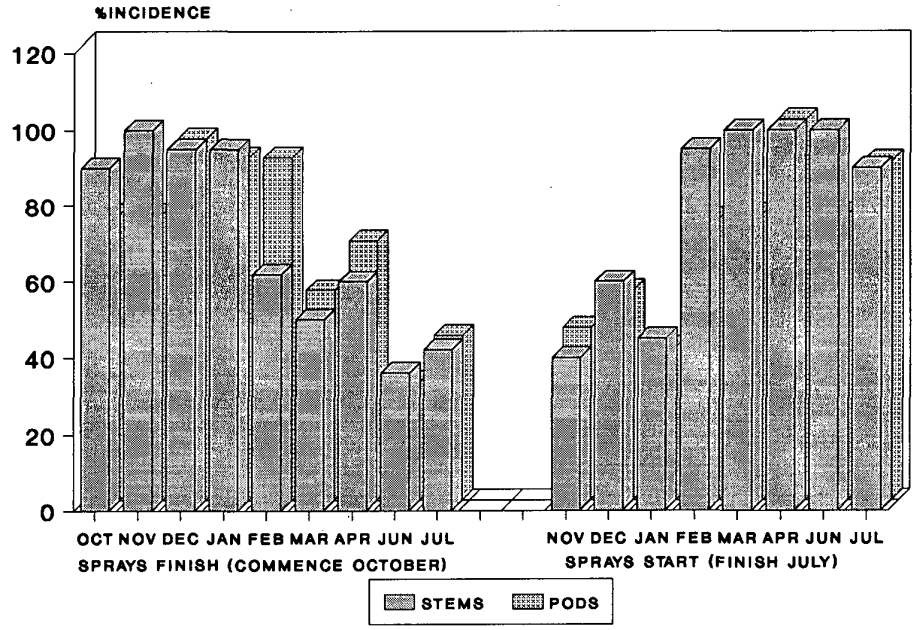


FIGURE 13C : PETTYMUICK LIGHT LEAF SPOT
27 JULY 1993, GS6.1

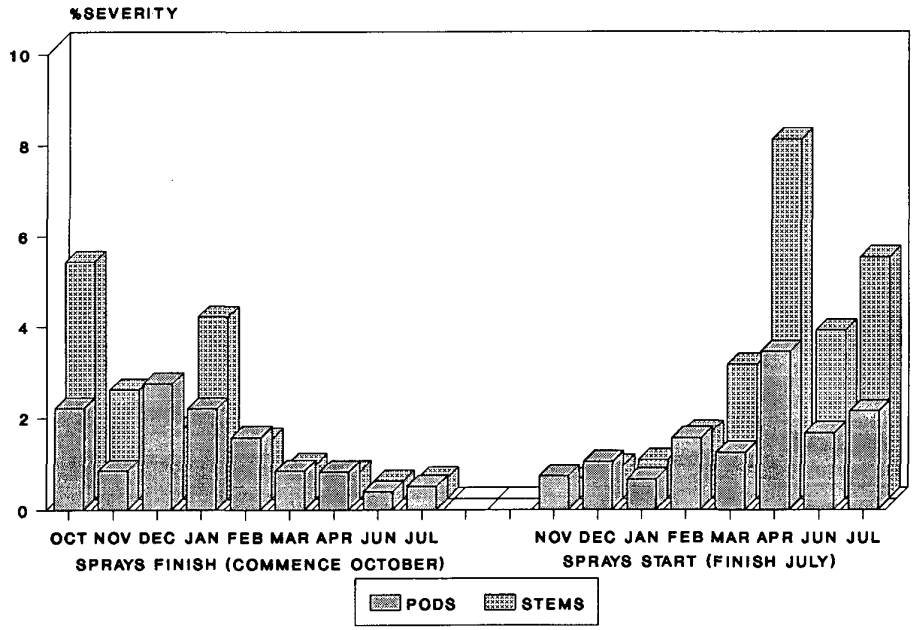


FIGURE 13D :PETTYMUICK YIELD 1993 (T/HA)

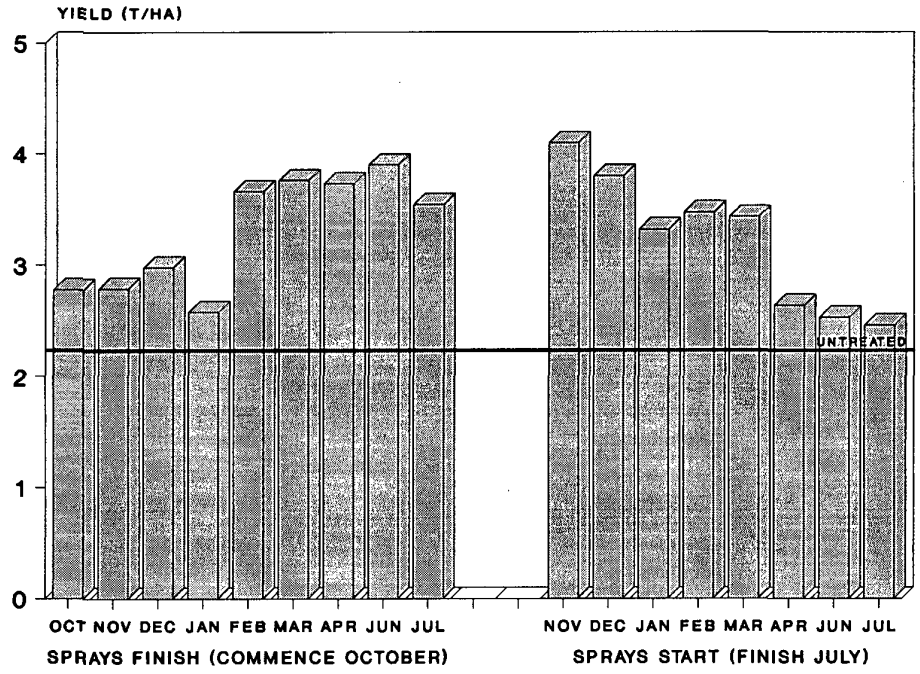
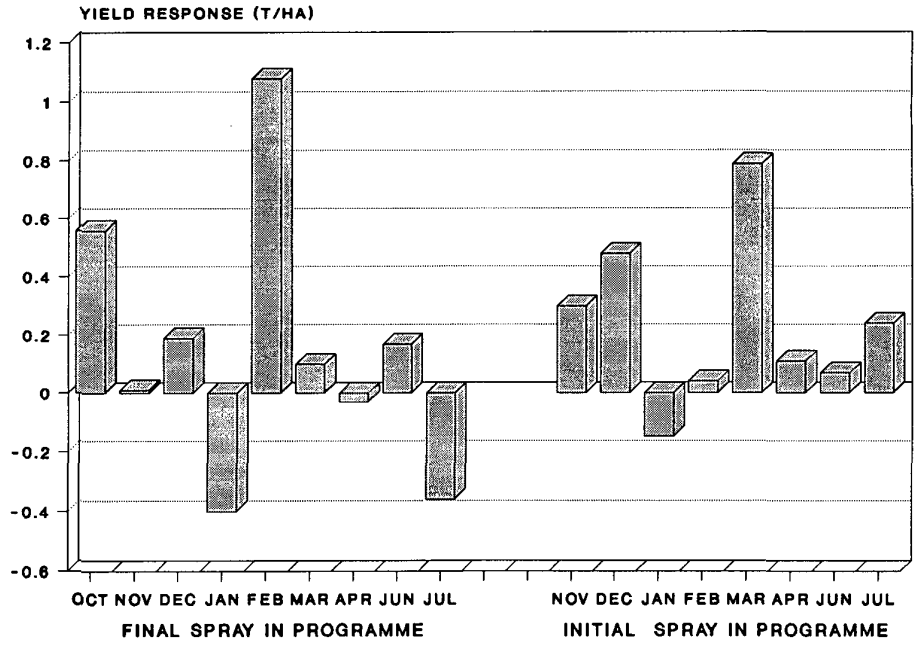


FIGURE 13E : PETTYMUICK YIELD 1993
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(iv) Rothamsted 1992/93

Phoma leaf spot and canker and light leaf spot were both equally important at this site. The former disease is discussed in Section 1(iv). The development of foliar light leaf spot in untreated plots is illustrated in Figure 14. The disease was first detected early in the season on 27 November (GS 1.09) affecting 30 per cent of plants (0.3 per cent leaf area). By 7 January virtually 100 per cent of plants had foliar symptoms and the disease remained at this level throughout the season. Disease severity was relatively high peaking at 13.7 per cent leaf area on 21 January and only showing a substantial decline by 15 April (GS 4.0) when 4.6 per cent leaf area was affected.

Light leaf spot development on stems and pods in untreated plots is illustrated in Figure 14A. Stem symptoms were first detected on 17 March (GS 3.5) affecting 60 per cent of plants at 0.7 per cent stem area. By 15 April (GS 4.0) the disease had reached a maximum affecting 100 per cent of untreated plants and 2.4 per cent of the stem area. The disease declined slowly until 7 July (GS 6.3) when at the final assessment 53 per cent of stems were affected but at relatively low severity (0.5 per cent stem area). Pod symptoms were first detected on 9 June (GS 6.3) affecting 85 per cent of plants at 0.1 per cent pod area; by 7 July (GS 6.3) 95 per cent of plants were affected (3.2 per cent pod area).

Significant effects of treatment on foliar light leaf spot were seen from 7 January (GS 1.09/1.12) onwards. All treatments except the single October spray (Treatment 2) led to reductions in disease severity on 7 January from 9.92 per cent leaf area to $\leq$ 1.29 per cent (Treatments 3, 4, 12, 21 and 22). The effect of treatments varied thereafter but in general, the greater the number of sprays applied, (provided a winter spray was included (2 November to 29 January)), the more effective the treatment in reducing disease severity and/or incidence.

Figure 14B and 14C illustrate the incidence and severity respectively of light leaf spot infection of stem and pods on 7 July (GS 6.3) in treated plots. Stem symptoms had declined by this time to affect 53 per cent of untreated plants (0.46 per cent stem area). Treatments 7, 8, 9, 10, 12 (first sprayed 14 October, final sprays applied on or after 23 February) and 18 and 19 (initially sprayed on 23 February and 29 January respectively) significantly reduced the incidence of light leaf spot on the stems to ≤ 5 per cent. Disease severity on the stems was significantly reduced from 0.5 to ≤ 0.03 per cent by virtually the same treatments. This result showed that sprays applied on 29 January and 23 February had the greatest effect on controlling light leaf spot infection of the stem. The incidence of plants affected by pod light leaf spot was significantly reduced from 95 per cent in untreated plots to ≤ 45 per cent by Treatments 6, 9, 10, 12, and 20 to 22. There was no obvious relationship between spray timings and control of pod disease except that the more sprays applied the greater the reduction in disease incidence. The severity of light leaf spot on the pods was significantly reduced from 3.2 to ≤ 1.10 per cent by all Treatments except 2, 3, 4 (first sprayed 14 October, final spray 14 October, 2 November, 7 December) and 14 (single spray 13 July) indicating that very early (October to December) and very late (June) sprays were ineffective against this phase of the disease.

The untreated yield at this site was 3.31 t/ha. Figure 14D shows the yields obtained from all of the treatments and Figure 14E shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments.

Yield responses significantly greater than the untreated were obtained from all treatments except 13, which received a single spray in July, and 15 which received three sprays between May and July. Significant responses ranged between 0.85 and 1.54 t/ha.

Treatments which began on 14 October and ended between 14 October and 13 July (Treatments 2 to 12) gave responses of between 0.91 and 1.55 t/ha. The spray timing

common to all these treatments was 14 October, which by subtraction (Treatment 2-1) gave a response of 0.91 t/ha as the final spray in a treatment (Figure 14E). This was the largest response to any of the timings. The greater the number of sprays applied the larger the yield.

Treatments which ended on 13 July and started progressively earlier (Treatments 13 to 22) gave responses of between 0.46 and 1.54 t/ha. The earlier treatment commenced the greater the yield response.

Yield responses were related both to the control of light leaf spot infection of the stem and stem canker at pod ripening, since disease control for both diseases was optimum in treatments with the greatest yield. Stem canker was the main disease to affect yield at this site since the greatest control of this disease was associated with the largest yield responses (Treatments 5 to 12, and 20 to 22) (see Section 1(iv)).

The results of regression analyses of the incidence and severity of light leaf spot infection of the stem on 7 July (GS 6.3) are shown in Table 15.

Table 15. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of light leaf spot (X) assessed on 7 July (GS 6.3)

X parameter	Regression equation	Correlation coefficient(r)*
% Incidence stem light leaf spot	Y = 4.71 - 0.009X	-0.65
% Severity stem light leaf spot	Y = 4.68 - 0.982X	-0.66

* $p \leq 0.001$ for all values of r

The relationship between the incidence of light leaf spot infection of the stem at pod ripening with yield was relatively strong with a correlation coefficient of -0.65 which was significant. The value of r was less than that associated with the incidence of

canker and yield however, but the value of the slope b in the equation was significantly different from zero at a probability of 99.99 per cent confirming the strength of the relationship between light leaf spot on the stem and yield. For every 1 per cent increase in the disease there appeared to be a loss in yield of approximately 0.01 t/ha. Further statistical work is required to ascertain which disease was most detrimental to yield, however with two important diseases present at pod ripening, simple linear regression may be insufficient to determine yield losses relative to each disease.

FIGURE 14 : ROTHAMSTED 1992/93
LIGHT LEAF SPOT DEVELOPMENT

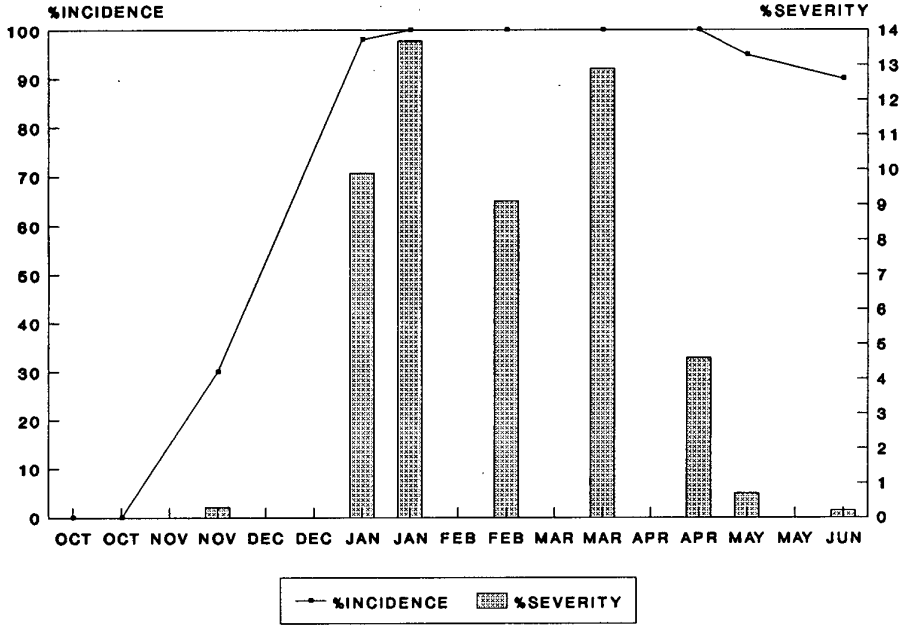


FIGURE 14A : ROTHAMSTED 1992/93
STEM AND POD LIGHT LEAF SPOT

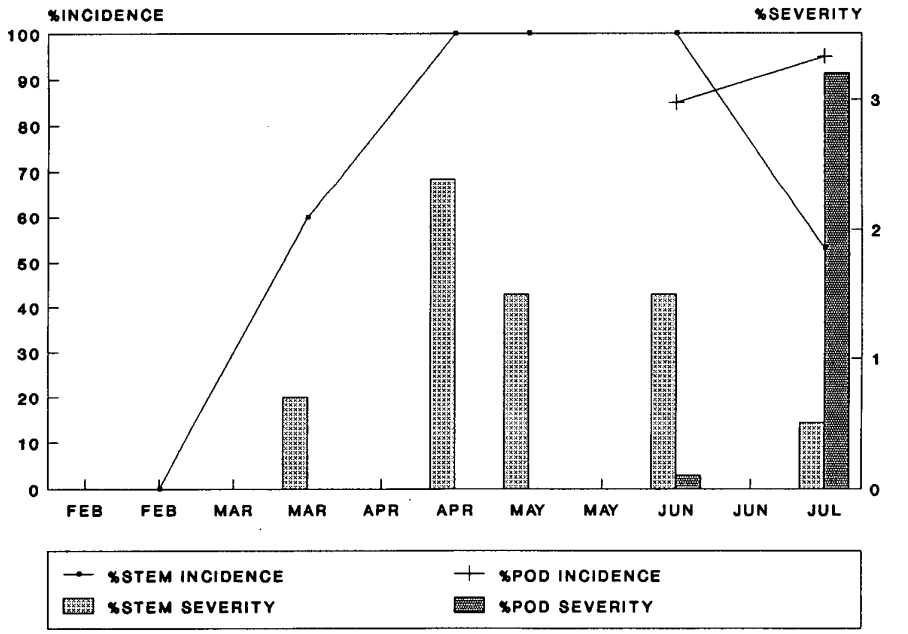


FIGURE 14B : ROTHAMSTED LIGHT LEAF SPOT
7 JULY 1993, GS6.3

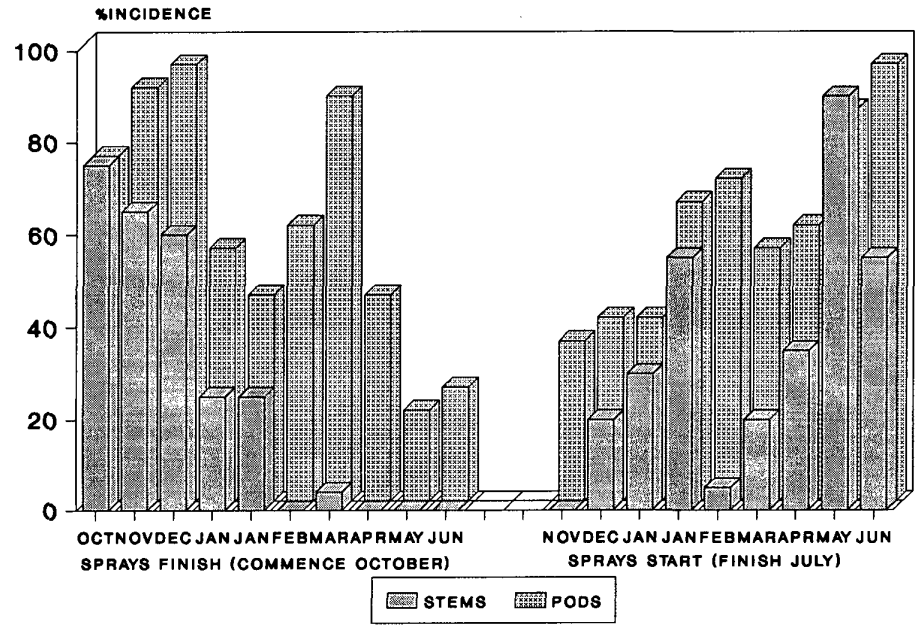


FIGURE 14C : ROTHAMSTED LIGHT LEAF SPOT
7 JULY 1993, GS6.3

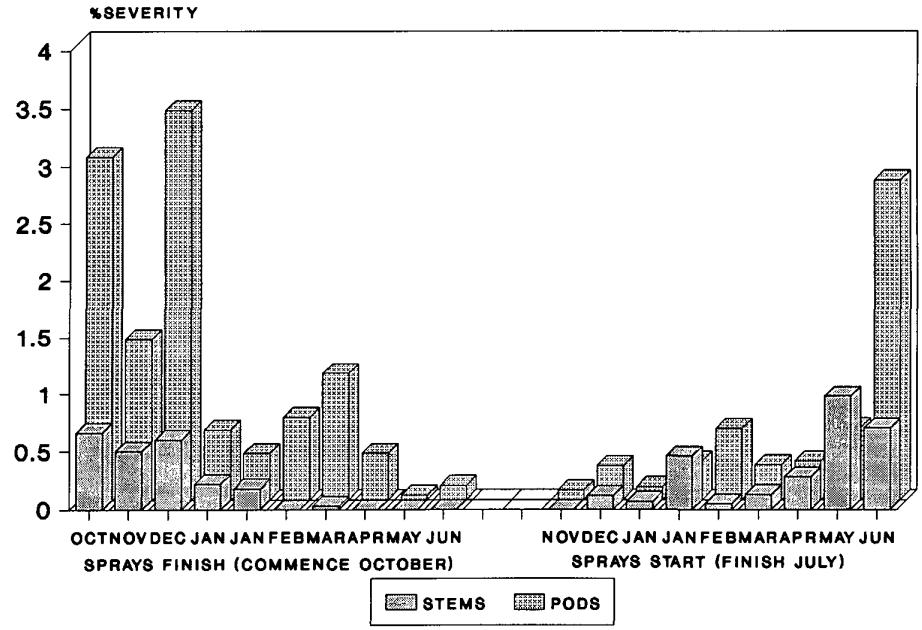


FIGURE 14D :ROTHAMSTED YIELD 1993 (T/HA)

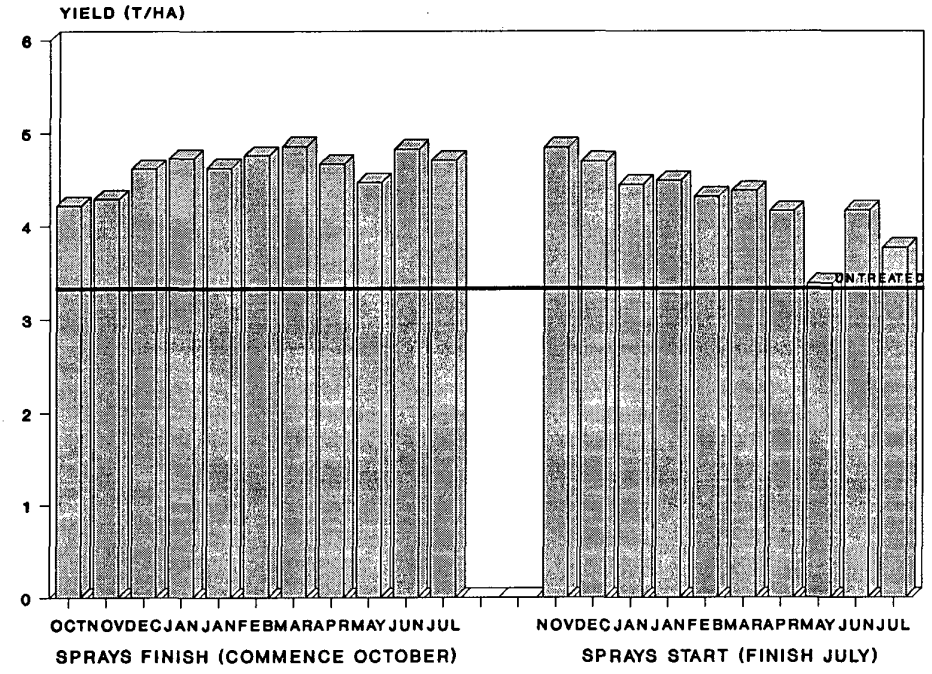
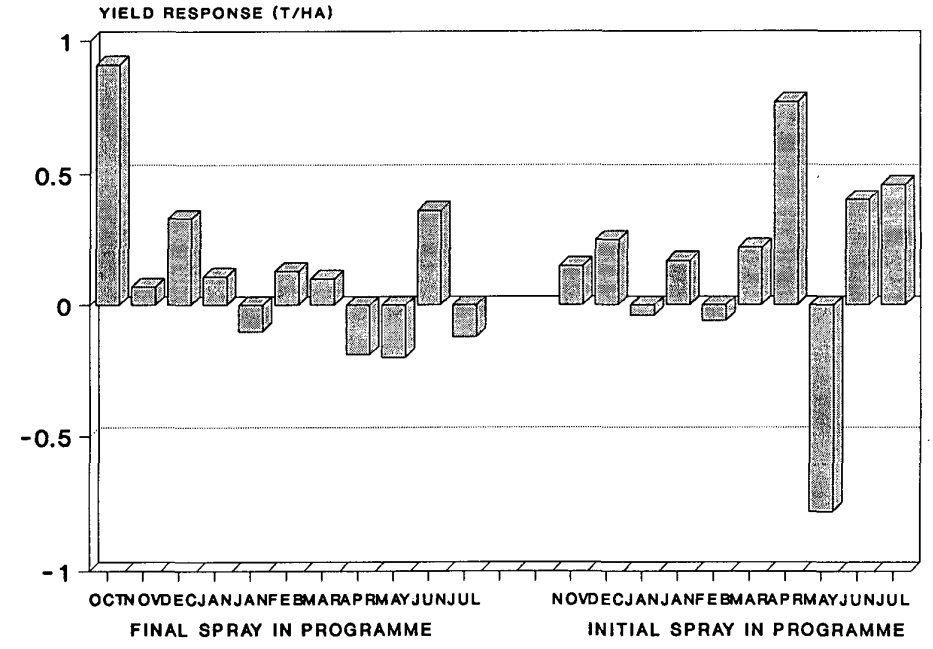


FIGURE 14E : ROTHAMSTED YIELD 1993
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(v) Tarrant Hinton 1992/93

Phoma leaf spot and canker and light leaf spot both developed at this site. *Phoma* leaf spot and canker is discussed in Section 1(v).

The development of foliar light leaf spot in untreated plots is illustrated in Figure 15. Symptoms developed late; on 23 February (GS 2.06 to 3.1) as stem extension began 25 per cent of plants had 0.5 per cent leaf area affected by light leaf spot; affected plants were stunted at this time. Symptoms were most severe on 19 March (GS 2.09 to 3.6) when 90 per cent of untreated plants had 6.6 per cent leaf area affected. Foliar symptoms declined thereafter due to the loss of the lower leaves.

Light leaf spot development on stems and pods in untreated plots is illustrated in Figure 15A. Stem symptoms were first detected on 19 March (GS 2.09 to 3.6) when 65 per cent of stems were affected (1.1 per cent stem area). The disease continued to develop reaching a maximum on 8 June (GS 6.3) when 100 per cent of untreated plants were affected (13 per cent stem area). Symptoms declined thereafter and on 29 June (GS 6.4) 90 per cent of untreated plants were affected at 3.5 per cent stem area.

Pod light leaf spot was first detected on 8 June (GS 6.3) when 80 per cent of untreated plants were affected (5.4 per cent pod area). By 29 June (GS 6.4) 53 per cent of plants were affected (5.3 per cent pod area, Figure 15A).

The effect of fungicide treatment on foliar light leaf spot was difficult to demonstrate statistically since the majority of data were skew and could not be restored to normality. However, on 19 March when the disease had reached a maximum (90 per cent incidence, 6.6 per cent leaf area, untreated plots) all of the treated plots had considerably less disease than the untreated plots ($\leq$ 25 per cent incidence, 0.7 per cent severity).

Figure 15B and 15C illustrate the incidence and severity respectively of light leaf spot infection of stems and pods on 29 June (GS 6.4) in treated plots. All plots included the untreated were equally affected by similar levels of stem disease at this time (2 to 4 per

cent severity, 85 to 100 per cent incidence). Light leaf spot infection of the pods affected 53 per cent of untreated plants at 5.3 per cent pod area on 29 June (GS 6.4). Data were skew and could not be restored to normality, thus disallowing a statistical demonstration of the effect of treatment on pod light leaf spot. However the incidence and severity of pod disease were greatly reduced by treatments that were sprayed from October to December and beyond (Treatments 4 to 12) and by those that began up to and including May (Treatments 15 to 22) (disease incidence $\leq$ 10 per cent, disease severity $\leq$ 0.35 per cent).

The untreated yield at this site was 3.05 t/ha. Figure 15D shows the yields obtained from all of the treatments and Figure 15E shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. The single early spray (Treatment 2) and the one or two-spray treatments applied in June (Treatments 13 and 14) did not significantly increase yield over the untreated. All other treatments (except 7) gave significant yield increases between 0.25 and 0.63 t/ha.

The largest yield increases resulted from treatments that began in December or January (19, 20, 21; 0.50 to 0.63 t/ha). Of the treatments that began in October, Treatment 6 which received five sprays ending on 29 January, gave the largest yield increase (0.41 t/ha). The January period coincided with the optimum timing for the control of *Phoma* leaf spot and canker. This suggests that control of this particular disease was the main factor involved in yield responses. Light leaf spot did not appear to affect yield at this site as regression analysis showed that no significant relationship existed between the two factors.

FIGURE 15 : TARRANT HINTON 1992/93
LIGHT LEAF SPOT DEVELOPMENT

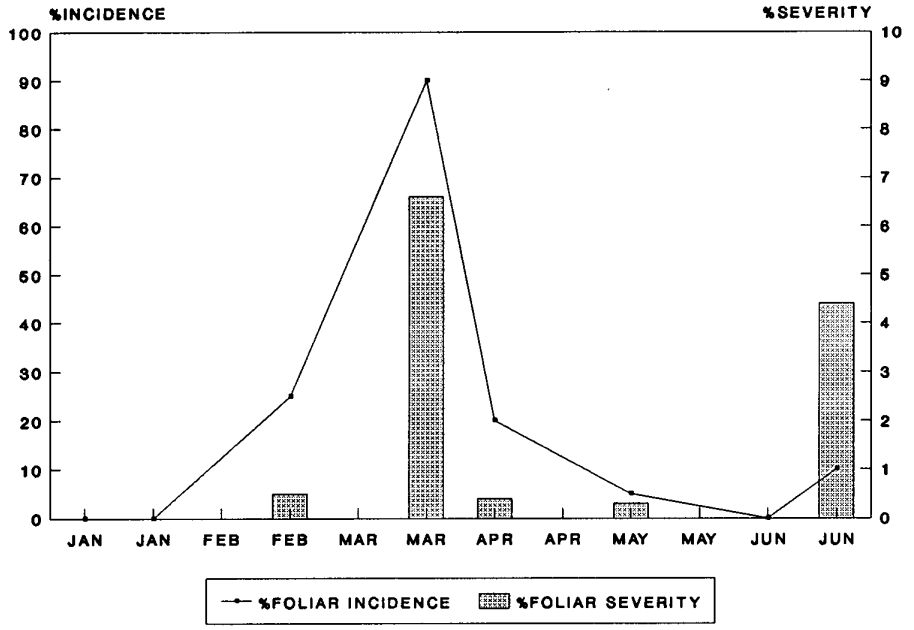


FIGURE 15A : TARRANT HINTON 1992/93
STEM AND POD LIGHT LEAF SPOT

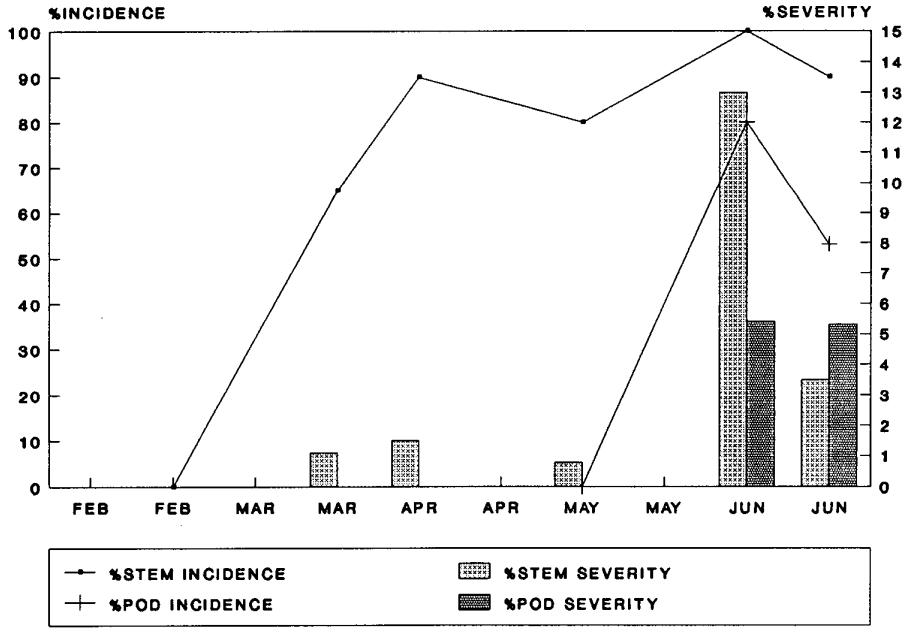


FIGURE 15B : T.HINTON LIGHT LEAF SPOT
29 JUNE 1993, GS6.4

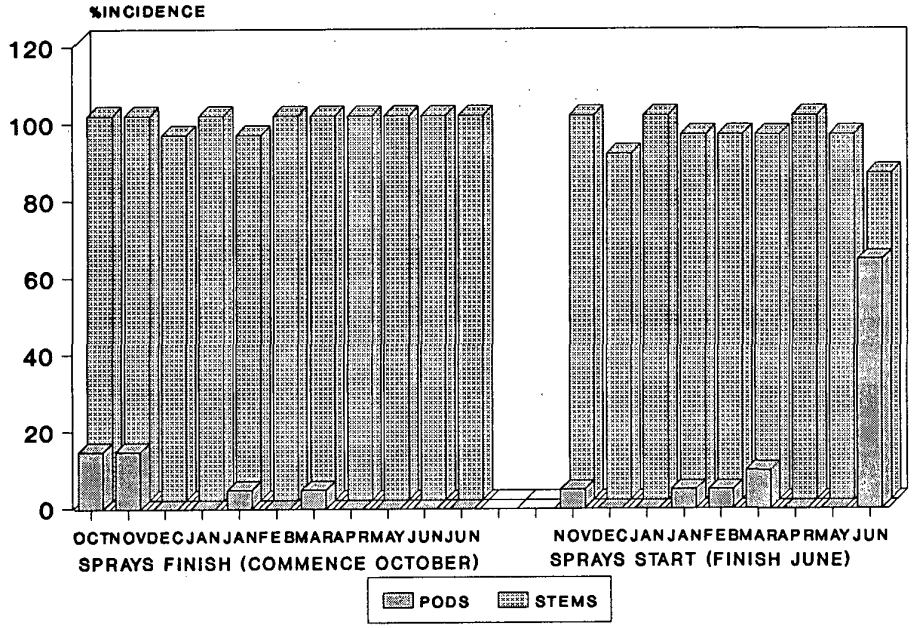


FIGURE 15C : T.HINTON LIGHT LEAF SPOT
29 JUNE 1993, GS6.4

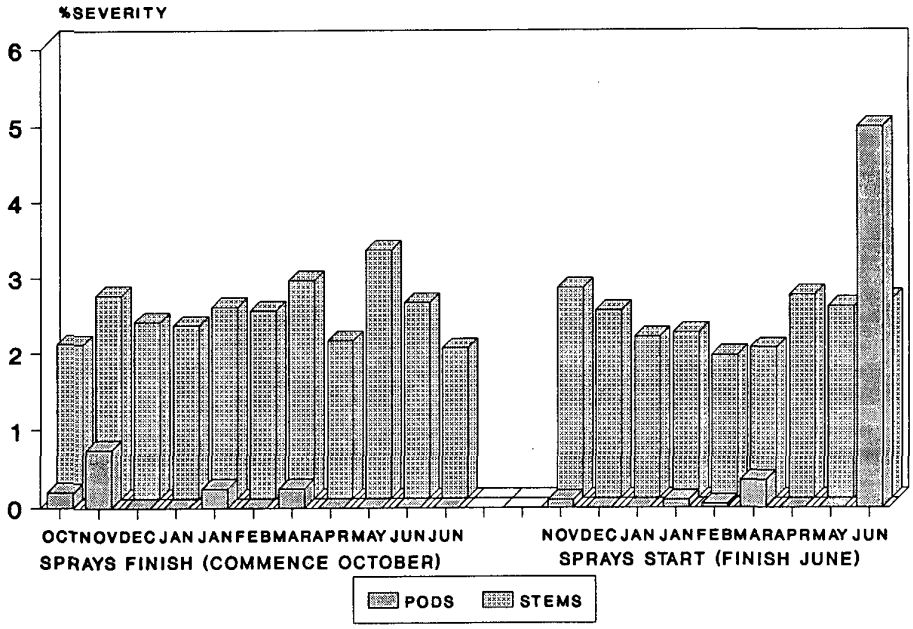


FIGURE 15D : T.HINTON YIELD 1993 (T/HA)

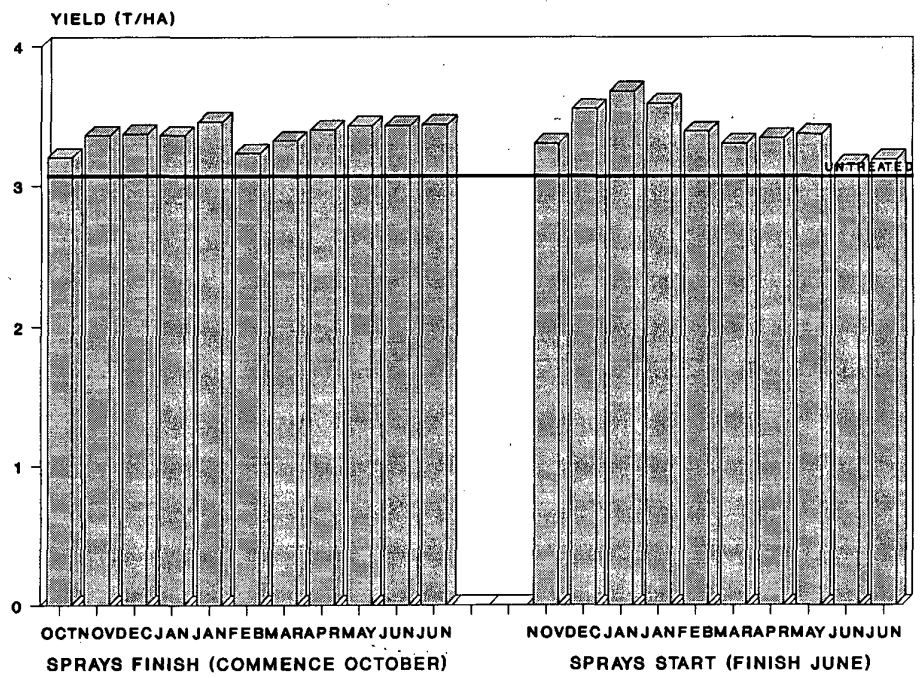
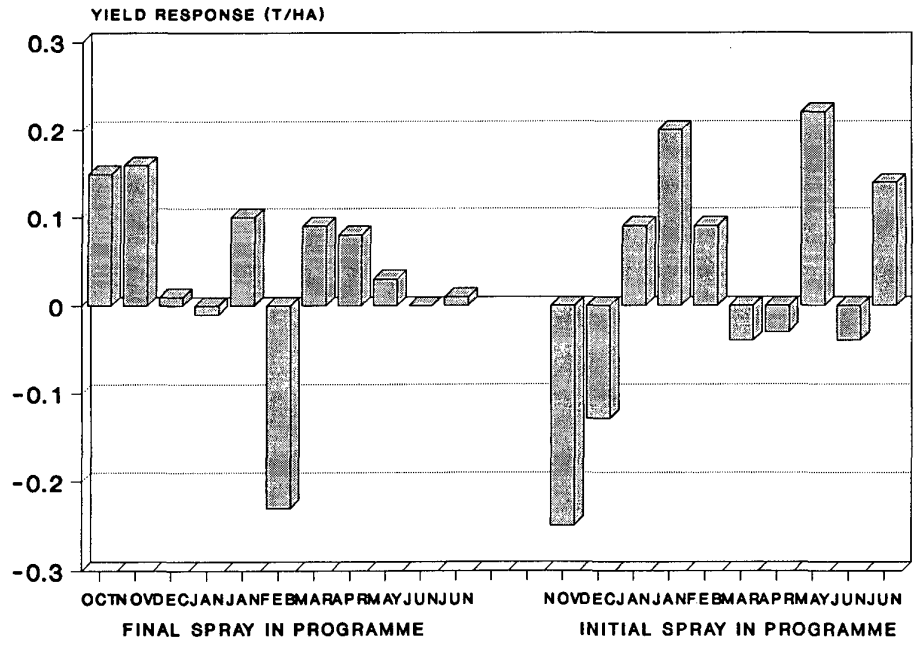


FIGURE 15E : TARRANT HINTON YIELD 1993
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(vi) Rosemaund 1993/94

Phoma leaf spot and canker and light leaf spot both developed at this site. *Phoma* leaf spot and canker are discussed in Section 1(viii). The development of foliar light leaf spot in untreated plots is illustrated in Figure 16. Light leaf spot symptoms were first detected at very low levels on 17 February (GS 1.06/1.08). The disease rapidly increased, and by 11 April (GS 3.3/3.7) 90 per cent of plants were affected at maximum severity (15.5 per cent leaf area). Disease incidence increased to 100 per cent by 10 May (GS 4.8, severity 11.5 per cent). Thereafter symptoms began to decline but even on 7 June (GS 4.8/6.2) 95 per cent of plants were affected at 6.7 per cent leaf area.

The development of light leaf spot on stems and pods is illustrated in Figure 16A. On 7 June (GS 4.8/6.2) 85 per cent of plants were affected at low severity (4.3 per cent stem area). By 4 July (GS 6.4) the disease had increased slightly; 95 per cent of plants were affected at 5.7 per cent stem area. Pod symptoms were first detected on 7 June (GS 4.8/6.2) when only 5 per cent of pods were affected at 0.3 per cent pod area. By 4 July (GS 6.4) pod symptoms had increased and 65 per cent of plants were affected at 3.2 per cent pod area.

Most data related to foliar light leaf spot were skew and could not be restored to normality by transformation. However, by 11 April when the disease was at its most severe (15.5 per cent leaf area; 90 per cent incidence) significant effects of fungicide treatment were detected. Disease incidence was significantly reduced to $\leq$ 45 per cent by all treatments except for 17 which had only received a single spray on 19 March at this time. The most effective treatments reducing disease incidence to $\leq$ 20 per cent were 3 to 8, 12, and 18 to 22. The single October treatment (2), was the least effective of the significant treatments (45 per cent incidence). Thus sprays applied between 25 October and 17 February were most effective against foliar light leaf spot.

Figure 16B and 16C illustrate the final incidence and severity respectively of light leaf spot on stems and pods on 4 July (GS 6.4). Ninety-five per cent of untreated plants

were affected at 5.7 per cent stem area. Significant reductions in disease incidence to ≤ 40 per cent were obtained with Treatments 6, 8, 9, 10, 12, and 18 to 22. The most effective treatments reducing disease incidence to ≤ 10 per cent were Treatments 8, 9, 10, and 12 which all received sprays from 4 October, ending on or after 19 March. Disease severity was significantly reduced from 5.6 per cent to ≤ 2.65 by all treatments except the single October spray (Treatment 2), and the single June spray (Treatment 14). The most effective treatments reducing disease severity to ≤ 0.6 per cent were Treatments 6, 8, 9, 10, and 12, and 19, 20, and 21. This implies that of the treatments that began in the autumn and finished progressively later, final treatments had to include a January spray (Treatment 6 to 12). Of the treatments that began progressively later, with the exception of Treatment 22 which began on 25 October, initial sprays had to be applied between November and January to be effective. Thus the January spray timing appeared to be important for the control of stem disease.

Sixty-five per cent of untreated plants were affected by light leaf spot infection of the pods at 3.2 per cent severity. Significant reductions in disease incidence to ≤ 30 per cent were obtained with Treatments 8, 9, 10, 12 (commenced 4 October, finished on or after 19 March), and 16 to 22 (commenced on or before 11 April). The most effective treatments reducing disease incidence to ≤ 15 per cent included all significant treatments except 16 which received its first spray on 11 April. Thus sprays applied on 19 March appeared critical for reductions in the incidence of light leaf spot on the pods. Significant reductions in disease severity to ≤ 1.1 per cent were achieved with Treatments 3, 5, 7, 8, 9, 10, 12, and 15 to 22. Of the treatments that began in the autumn and finished progressively later, the most effective Treatments were 8, 9, 10 and 12 reducing disease severity to ≤ 0.15 per cent. Of the treatments that began progressively later, the most effective treatments reducing disease severity of ≤ 0.20 per cent were Treatments 17 to 22 which began treatment on or before 19 March. The March spray therefore was effective against both pod disease incidence and severity.

The untreated yield at this site was 1.92 t/ha. Figure 16D shows the yields obtained from all of the treatments and Figure 16E shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. There were no significant effects of treatment on yield at this site. In general however, the greater the number of sprays applied, the greater the yield. The maximum yield (3.29 t/ha) was obtained from Treatment 9 which received 8 sprays between 4 October and 11 April.

Regression analyses of the incidence and severity of light leaf spot on stems and pods on 4 July (GS 6.4) with yield are shown in Table 16.

Table 16. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of light leaf spot on stems and pods (X) assessed on 4 July (GS 6.4)

X parameter	Regression equation	Correlation coefficient(r)*
% Incidence stem light leaf spot	Y = 2.97 - 0.010X	-0.63
% Severity stem light leaf spot	Y = 2.81 - 0.173X	-0.67
% Incidence pod light leaf spot	Y = 2.75 - 0.008X	-0.42
% Severity pod light leaf spot	Y = 2.70 - 0.163X	-0.47

* $p \leq 0.02$

The relationships between the incidence and severity of light leaf spot infection of the stem at this site were moderately strong and significant ($p \leq 0.02$) whereas the relationships between pod infection (incidence, severity) and yield were weak. The value of the slope b in each equation was significantly different from zero at a probability of 99.99 per cent confirming a strong relationship between light leaf spot infection of the stem and yield. For every 1 per cent increase in the incidence of light

leaf spot infection of the stem there appeared to be a loss in yield of approximately 0.01 t/ha. However, canker also affected yield at this site (see Section 1(viii)) with stem infection appearing to cause similar losses in yield. Further work is required to separate the effect of each disease on yield.

FIGURE 16 : ROSEMAUND 1993/94
LIGHT LEAF SPOT DEVELOPMENT

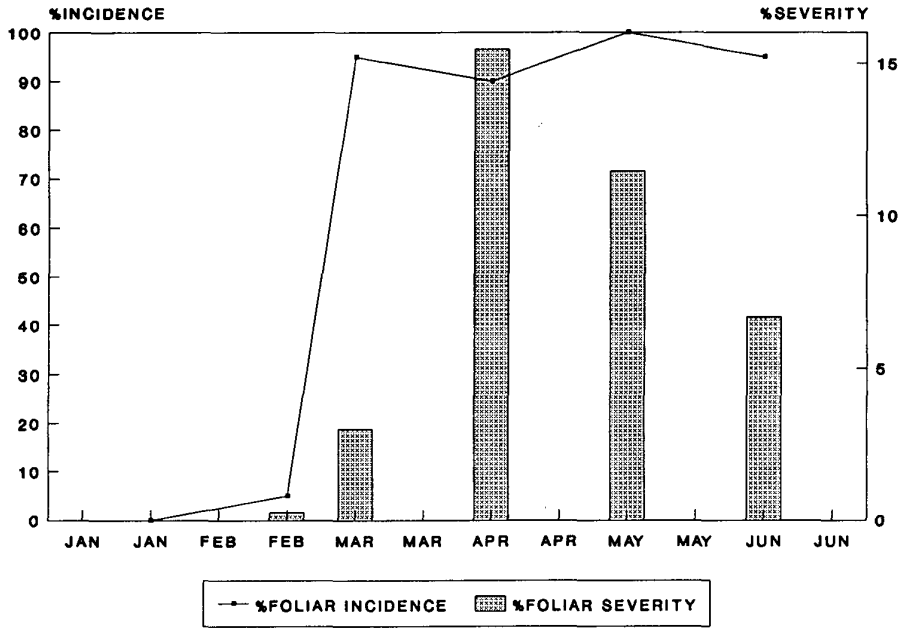


FIGURE 16A : ROSEMAUND 1993/94
STEM AND POD LIGHT LEAF SPOT

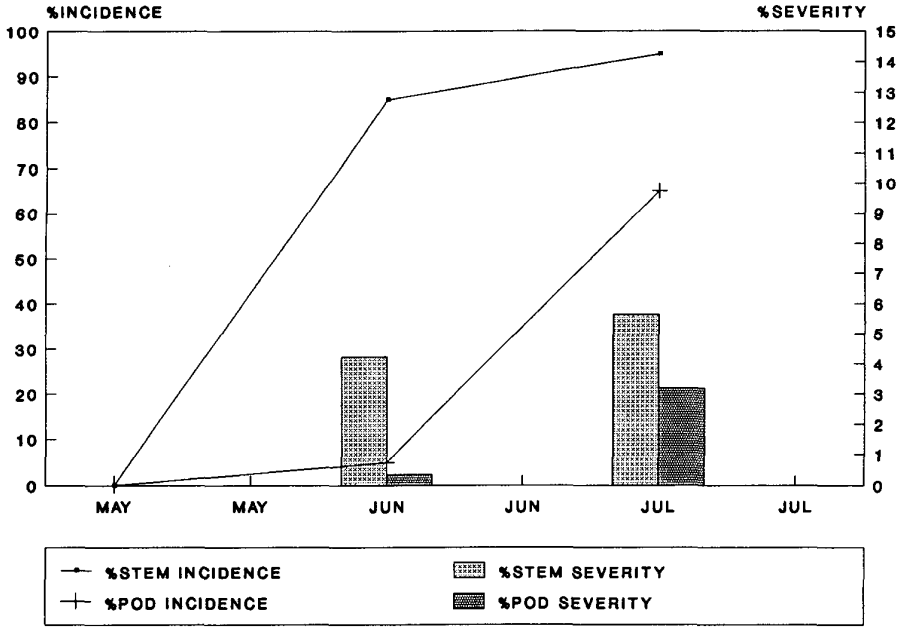


FIGURE 16B : ROSEMAUND LIGHT LEAF SPOT
4 JULY 1994, GS6.4

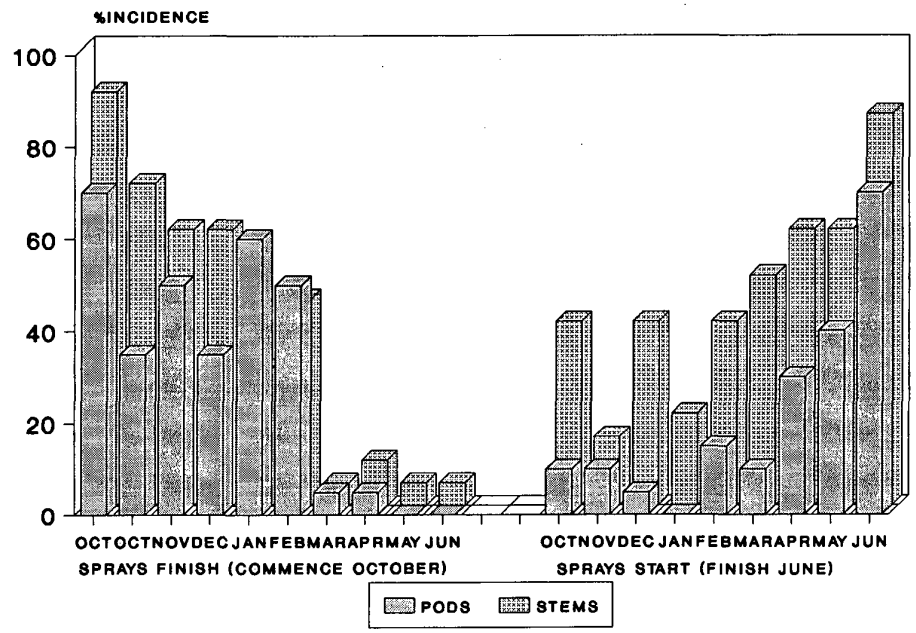


FIGURE 16C : ROSEMAUND LIGHT LEAF SPOT
4 JULY 1994, GS6.4

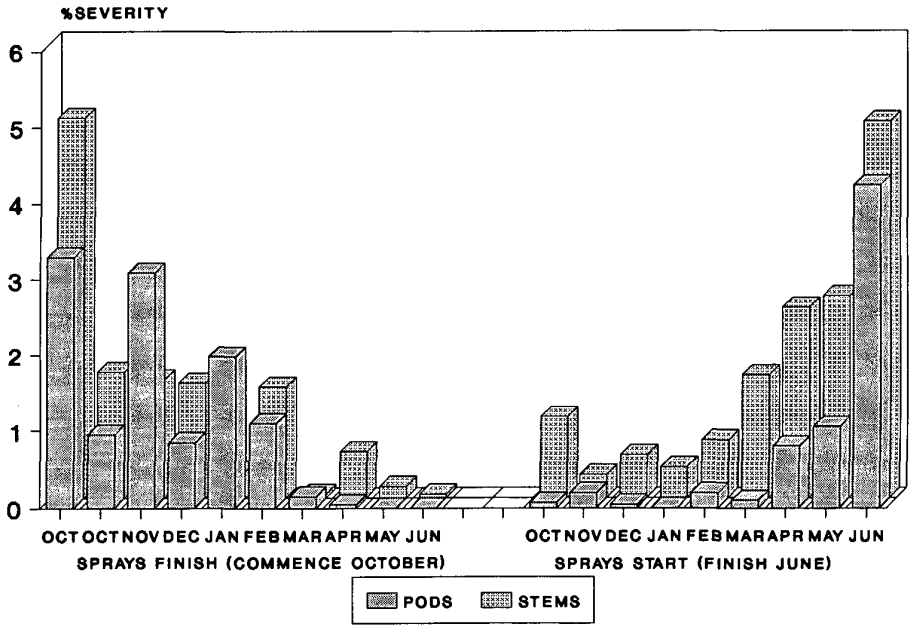


FIGURE 16D : ROSEMAUND YIELD 1994 (T\HA)

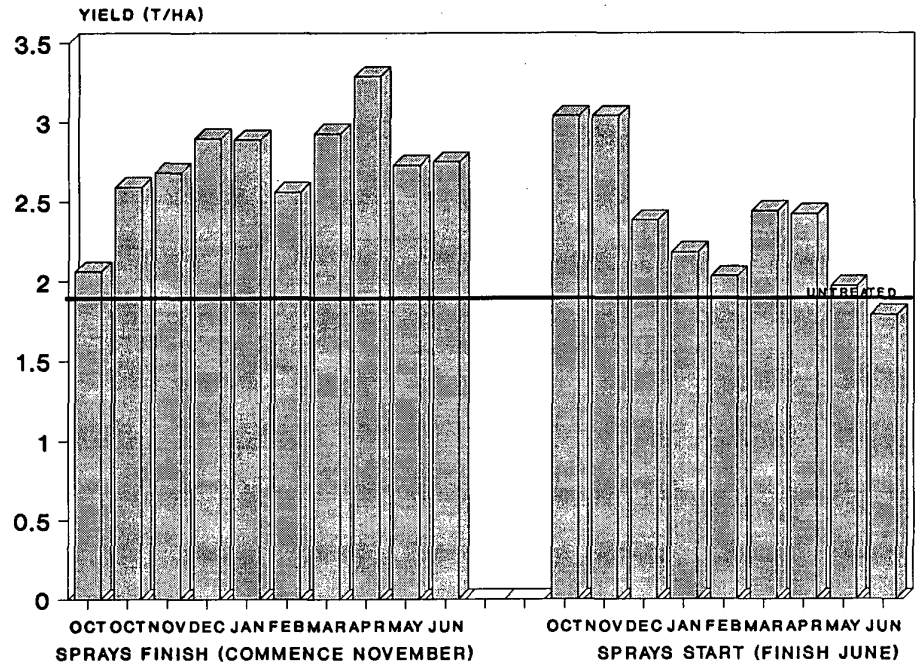
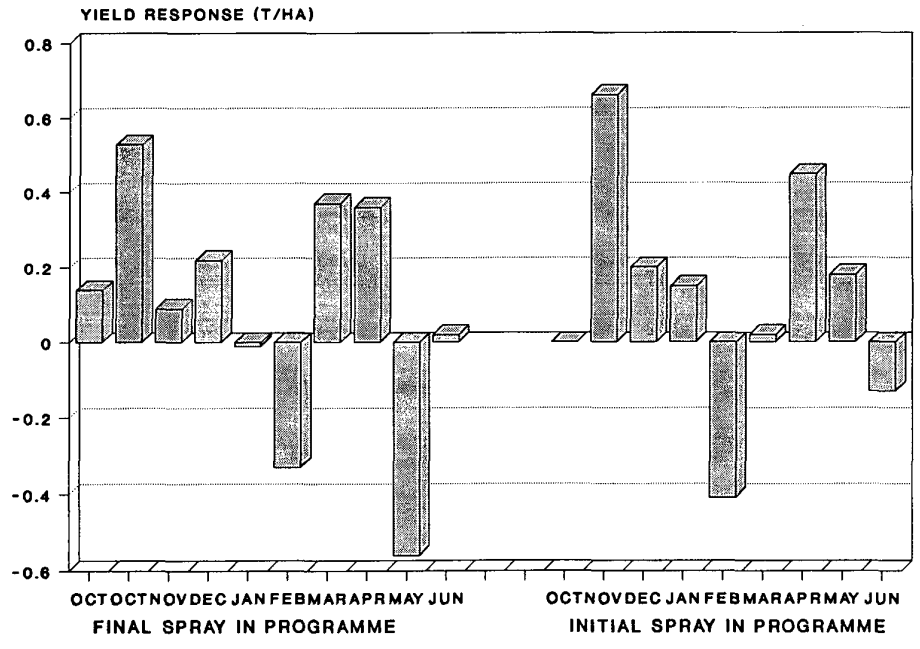


FIGURE 16E : ROSEMAUND YIELD 1994
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(vii) Rothamsted 1993/94

Light leaf spot and *Phoma* leaf spot and canker developed at this site. *Phoma* leaf spot is discussed in Section 1(ix).

The development of light leaf spot on the leaves in untreated plots is illustrated in Figure 17. Symptoms were first detected on 9 February (GS 1.10) when 65 per cent of plants were affected at 1.2 per cent leaf area. The disease increased steadily thereafter reaching a maximum on 4 May (GS 4.3) when 100 per cent of plants were affected at 6.1 per cent leaf area. Symptoms declined slightly thereafter and on 1 June (GS 6.1) 80 per cent of plants were affected at 5.0 per cent leaf area.

The development of light leaf spot on stems and pods is illustrated in Figure 17A. Stem symptoms were first detected on 4 May (GS 4.3) when 55 per cent of plants had very low levels of disease (0.04 per cent stem area affected). The incidence of stem disease reached 100 per cent by 1 June (GS 6.1) and remained at that level. However, while disease severity increased, with time, the maximum severity detected occurred on 20 July (GS 6.5) when only 2.3 per cent stem area was affected.

Pod symptoms were first seen on 1 June (GS 6.1) when 90 per cent of pods were affected (1.5 per cent pod area). Symptoms increased and by 29 June (GS 6.3) 100 per cent of pods were affected at 4.7 per cent pod area. Thereafter symptoms declined; on 20 July (GS 6.5) 15 per cent of pods were affected at 0.15 per cent pod area.

Significant effects of fungicide treatment on foliar light leaf spot were first detected on 9 March (GS 2.3) and continued to be observed throughout the season until 1 June (GS 6.1). Sprays applied between 17 December and 19 April led to significant reductions in disease incidence and/or severity at selected assessments. In March, significant and effective treatments reducing disease incidence and severity from 95 and 1.3 per cent in untreated plots to $\leq$ 5 and 0.01 per cent respectively were those treatments that received their final spray on or after 17 December (5, 6, 7, 12) or

commenced treatment on 17 December (Treatment 20). By 8 April (GS 3.1) significant control to ≤ 40 per cent incidence, 0.2 per cent severity was achieved with Treatments 5, 6, 7, 8, and 12 and 18, 19, 20 compared to 100 per cent incidence and 3.9 per cent severity in untreated plots. Particularly effective treatments reducing disease to ≤ 5 per cent incidence, 0.01 per cent severity were Treatments 7, 8, 12 (initial spray 23 November, final spray on or after 17 February), and 19 and 20 (initial sprays on 19 January and 17 December respectively).

The disease reached a maximum on 4 May (100 per cent incidence, 6.1 per cent severity). At this time significant control to ≤ 20 per cent incidence was achieved with Treatments 7, 8, 9, 12 (initial spray 23 November, final spray on or after 17 February) and 18, 19, 20 (initial spray on or before 17 February). Disease severity in these treatments was the lowest at ≤ 0.13 per cent. At the final foliar assessment on 1 June (GS 6.1) significant reductions in foliar light leaf spot from 80 per cent incidence, 5 per cent severity to ≤ 40 and 1.1 per cent respectively were achieved with Treatments 7 to 12 (final spray on or after 17 February) and 16 to 20 (initial spray on or before 16 April).

Figure 17B illustrates the final incidence and severity of light leaf spot infection of the stem in treated plots at Rothamsted on 20 July (GS 6.5). One hundred per cent of untreated plants were affected but the severity was low (2.3 per cent stem area). Significant reductions in disease severity to ≤ 1.3 per cent were achieved with Treatments 6 to 12, (initial spray 23 November, final spray on or after 19 January) and 16 to 20 (initial spray on or before 19 April). Where sprays began in the autumn and finished progressively later, the most effective treatments included the 17 February spray (Treatments 7 to 12, ≤ 0.8 per cent severity), likewise, where treatments began on or before 17 February (Treatments 18, 19, 20) disease severity was well controlled to ≤ 0.4 per cent. Disease incidence data were skew and could not be restored to normality by transformation but similar trends occurred (Figure 17B). Thus, as with *Phoma* leaf spot and canker (see Section 1(ix)) the 17 February spray appeared critical for control of both foliar and stem symptoms of light leaf spot.

Pod disease was at maximum levels on 29 June (GS 6.3) (100 per cent incidence, 4.7 per cent severity, untreated), and all except the single November spray (Treatment 4) led to significant reductions to ≤ 60 and 2.7 per cent respectively. The most effective treatments were those that had received the most sprays (10, 11, 12, 18, 19, 20); pod disease affected ≤ 5 per cent incidence and 0.05 per cent severity in these plots.

By 20 July (GS 6.5) pod disease had declined; only 15 per cent of untreated plants had pod symptoms at 0.2 per cent severity; treated plots were similarly affected.

The untreated yield at this site was average at 3.35 t/ha. Figure 17C shows the yields obtained from all of the treatments and Figure 17D shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. No significant differences in yield were detected. Of the treatments that began in the autumn and finished later (Treatments 4 to 12) there were no consistent trends other than the addition of a final spray in February in Treatment 7 caused an increase in yield of 0.36 t/ha when compared to Treatment 6 which finished in January. Likewise, commencing treatment in February (Treatment 18) was more beneficial than commencing in January (Treatment 19) or March (Treatment 17), (0.55 and 0.41 t/ha greater respectively).

Yields appeared to be related to the control of foliar and stem light leaf spot at this site since the February spray timing was critical for control of this disease. However canker was also controlled by this spray (Section 1(ix)).

Sprays applied in January and July appeared to be particularly damaging to yield (Figure 17D). January temperatures were particularly low and it is possible that fungicide applications during this time could have resulted in crop damage.

Regression analysis of the incidence and severity of light leaf spot on the stems on 20 July (GS 6.5) with yield are shown in Table 17.

Table 17. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of light leaf spot on the stems (X) on 20 July (GS 6.5)

X parameter	Regression equation	Correlation coefficient(r)*
% Incidence stem light leaf spot	Y = 3.82 - 0.005X	-0.52
% Severity stem light leaf spot	Y = 3.70 - 0.255X	-0.52

*$p \leq 0.015$

The relationships between the incidence and severity of stem infection by light leaf spot and yield were not especially strong, each with a correlation coefficient of -0.52. However, both values of r were significant ($p \leq 0.008$). The value of the slope b in each equation was significantly different from zero at a probability of 99.99 per cent implying that a relationship did exist between light leaf spot and yield at this site. For every 1 per cent increase in the incidence of light leaf spot infection of the stem there was a very low yield loss of only 0.005 t/ha. Light leaf spot was not especially damaging at this site. Similar yield losses were seen with canker (see Section 1(ix)) and further work is required to separate the effect of each disease on yield.

FIGURE 17 : ROTHAMSTED 1993/94
LIGHT LEAF SPOT DEVELOPMENT

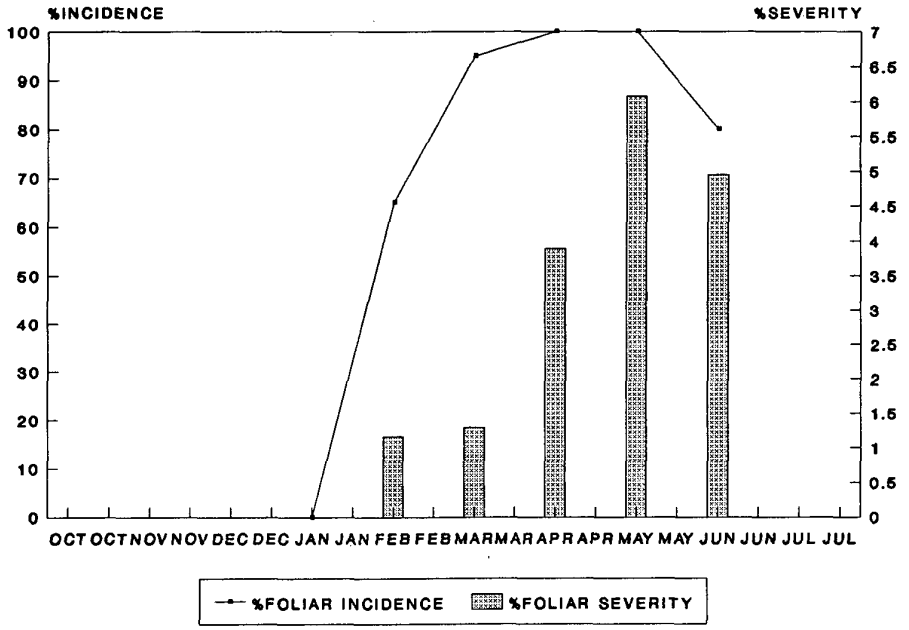


FIGURE 17A : ROTHAMSTED 1993/94
STEM AND POD LIGHT LEAF SPOT

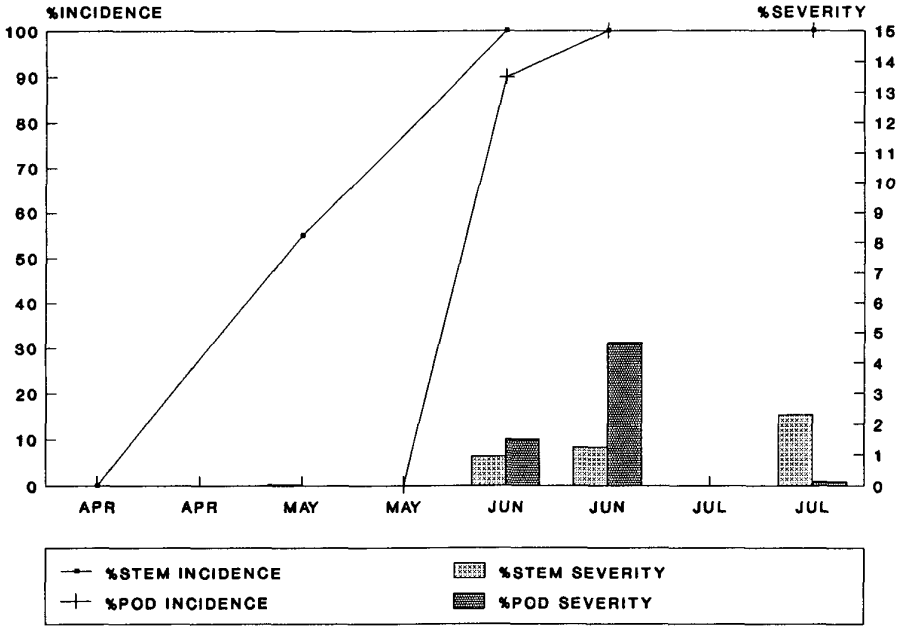


FIGURE 17B :ROTHAMSTED LIGHT LEAF SPOT
STEM DISEASE, 20 JULY 1994, GS6.5

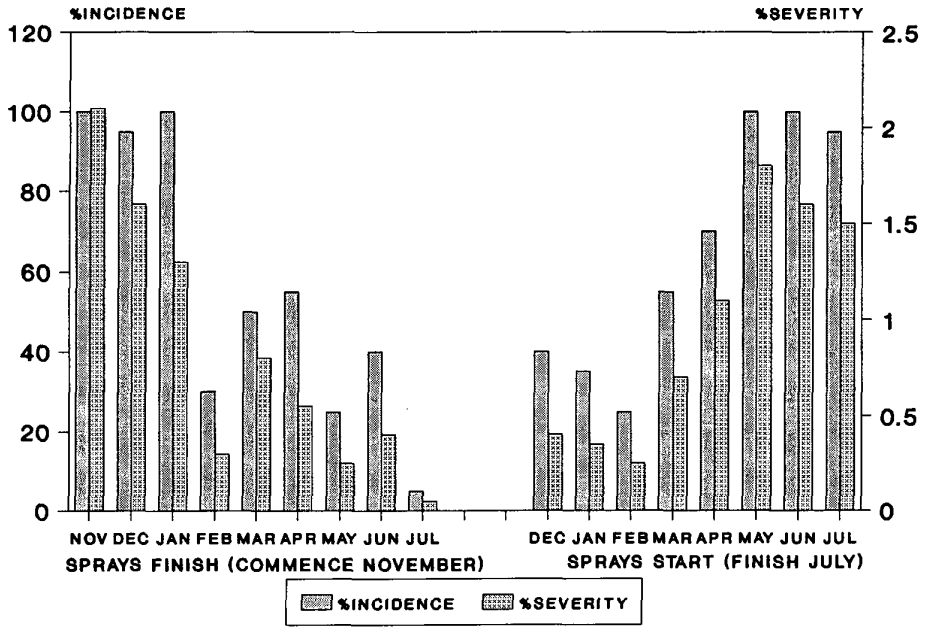


FIGURE 17C :ROTHAMSTED YIELD 1994 (T\HA)

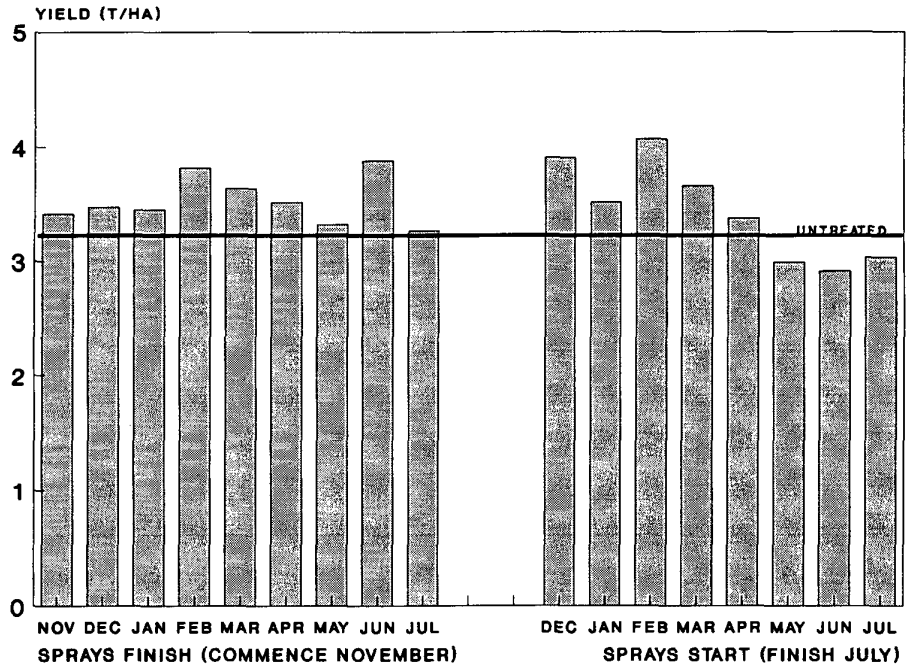
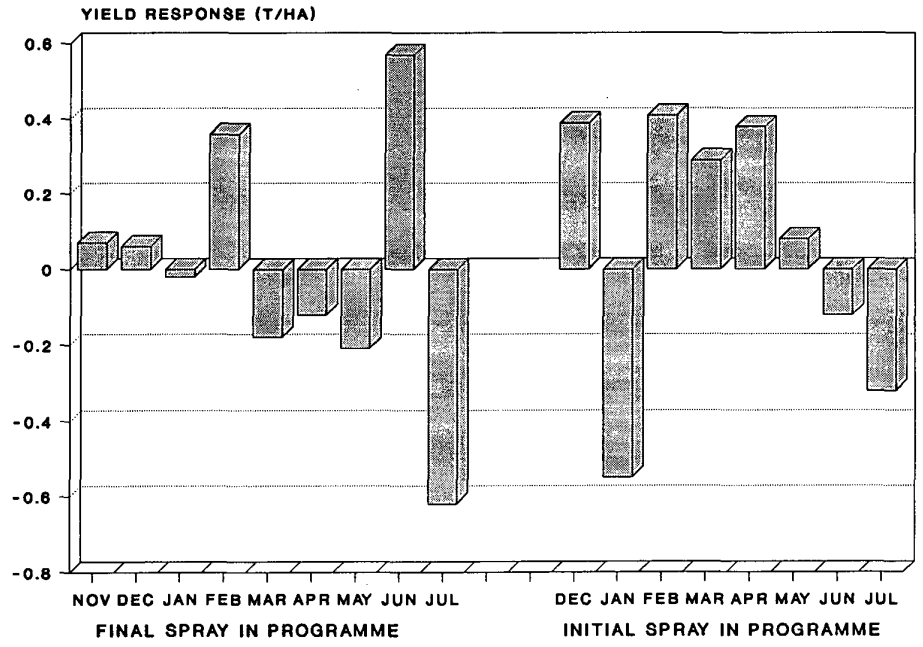


FIGURE 17D : ROTHAMSTED YIELD 1994
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(viii) Thurloxton 1993/94

Phoma leaf spot and canker and light leaf spot both developed at this site. *Phoma* leaf spot and canker is discussed in Section 1(x).

Figure 18 illustrates the development of foliar light leaf spot in untreated plots. Symptoms were first seen on 31 January (GS 1.08) affecting 70 per cent of untreated plants; the disease was already severe affecting 9.4 per cent of the leaf area. Within 3 weeks 100 per cent of plants were affected (22.9 per cent leaf area). Severity increased between 21 February and 11 April when exceedingly high levels of disease were detected. One hundred per cent of plants were affected at the highest foliar severity observed in the whole series of experiments; 63.9 per cent of the leaf area showed symptoms of light leaf spot. Plants were extremely stunted and leaves were badly distorted.

Light leaf spot development on stems and pods in untreated plots is illustrated in Figure 18A. Symptoms were first observed on 11 April (GS 2.08 / 3.7) when 85 per cent of stems were affected at 5.5 per cent leaf area. The disease continued to increase and by 11 July (GS 6.5 / 6.6) 100 per cent of plants had extremely severe lesions affecting 55 per cent of the stem area. Pod symptoms were first seen on 9 May (GS 4.7 / 5.0) affecting 60 per cent of plants at 3.1 per cent pod area. The disease did not develop further on the pods as both the incidence and severity of symptoms was the same on 6 June (GS 6.2/6.3).

Significant control of the foliar stage of the disease was demonstrated on 6 June (GS 6.2 / 6.3) when Treatments 6 to 12 (initial spray 28 October, final spray on or after 31 January (GS 1.08)) and Treatments 21 to 17 (initial spray between 26 November and 15 March (GS 2.05 / 3.1)) had $\leq$ 35 per cent of plants affected compared to 75 per cent in untreated plots. Particularly effective treatments reducing disease incidence to $\leq$ 10 per cent included Treatments 8 to 12, which finished treatment on or after 15 March (GS 2.05 / 3.1) and 21 to 17 which all commenced treatment between 26 November and 15 March. Hence the 15 March spray was particularly effective against

foliar disease. Disease severity data were skew at this time and could not be restored to normality.

On 6 June significant control of stem light leaf spot was also observed. Disease incidence data were skew and were not transformable but significant differences in disease severity occurred. All treatments except 15 and 16 (which did not receive any treatment until after 15 March) resulted in significant reductions in disease severity from 10.7 per cent stem area in untreated plots to ≤ 8.1 per cent where treatments received sprays on or before 15 March. The most effective treatments reducing disease severity to ≤ 3.1 per cent were the same treatments that gave optimal control of foliar disease (8 to 12 and 21 to 17) with the addition of Treatments 5, 6, 7 which were initially sprayed on 28 October and finished treatment between 7 January and 21 February.

Figure 18B illustrates the final incidence and severity of light leaf spot infection of the stem in treated plots at Thurloxton. Disease incidence was high in all of the plots (data skew and untransformable) with even the fully-sprayed treatment (12) highly affected at 78 per cent incidence. Significant reductions in disease severity from 55 per cent to ≤ 28 per cent were achieved with treatments that began on 28 October provided they continued to receive treatment up until 7 January (Treatment 5) or beyond. Of the treatments that began progressively later all treatments that received their initial spray on or before 11 April (GS 2.08 / 3.7) resulted in significant reductions in disease (Treatments 16 to 21). Particularly effective treatments included 8 to 12, and 21 to 17 which all received the 15 March spray and all had ≤ 10 per cent disease severity on 11 July. These were the same treatments to give optimal control of foliar disease.

No significant effects of treatment on pod light leaf spot were found.

The untreated yield at this site was 1.76 t/ha. Figure 18C shows the yield obtained from all of the treatments and Figure 18D shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments.

All treatments increased yield between 0.34 t/ha (single spray on 28 October, Treatment 3) and 1.75 t/ha (8 sprays between 26 November and 6 June, Treatment 21). With one exception, yields significantly greater than the untreated were obtained from all of the treatments where foliar light leaf spot (incidence) and stem light leaf spot (severity) were reduced to less than 10 and 3.1 per cent respectively on 6 June (GS 6.2/6.3) (Treatments 8 to 12, and 21 to 17; responses of 1.0 to 1.75 t/ha greater than the untreated). In addition Treatment 4 (28 October plus 26 November sprays) also resulted in a significant yield of 2.43 t/ha (0.67 t/ha greater than the untreated).

The control of light leaf spot on leaves and stems was related to yield at Thurloxton. The 15 March spray as a final application in Treatment 8 led to an increase of 0.63 t/ha greater than Treatment 7 which received its final spray on 21 February (Figure 18D). This spray timing appeared to be critical for the control of light leaf spot.

The results of regression analyses of the incidence and severity of light leaf spot infection of the stem on 11 July (GS 6.5 / 6.6) are shown in Table 18.

Table 18. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of light leaf spot (X) assessed on 11 July (GS 6.5/6.6)

X parameter	Regression equation	Correlation coefficient(r)*
% Incidence stem light leaf spot	Y = 6.80 - 0.044X	-0.82
% Severity stem light leaf spot	Y = 3.26 - 0.029X	-0.83

* $p \leq 0.001$

The relationships between the incidence and severity of stem infection by light leaf spot and yield were strong with a correlation coefficient of -0.82 and -0.83 respectively. Both values of r were significant ($p \leq 0.001$). The value of the slope b in each equation was significantly different from zero at a probability of 99.99 per cent implying a strong relationship between light leaf spot infection of the stem and yield at

this site. For every 1 per cent increase in the incidence of light leaf spot infection of the stem there was a large yield loss of 0.044 t/ha. Light leaf spot was especially damaging at this site. Canker also developed at this site but apparently caused half as much loss in yield (0.02 t/ha) for a 1 per cent increase in disease incidence. Further work is required to separate the effect of each disease on yield.

FIGURE 18 : THURLOXTON 1993/94
LIGHT LEAF SPOT DEVELOPMENT

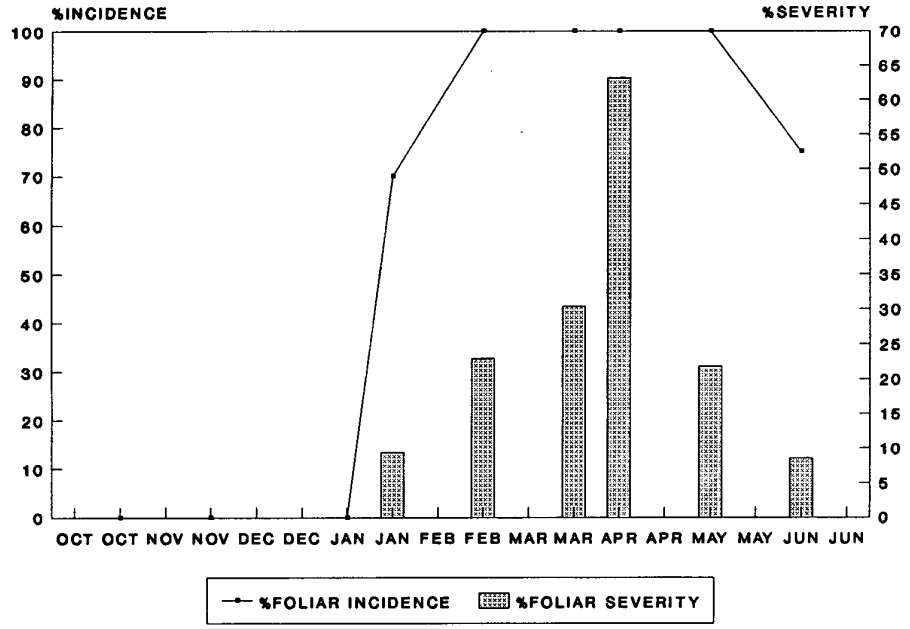


FIGURE 18A : THURLOXTON 1993/94
STEM AND POD LIGHT LEAF SPOT

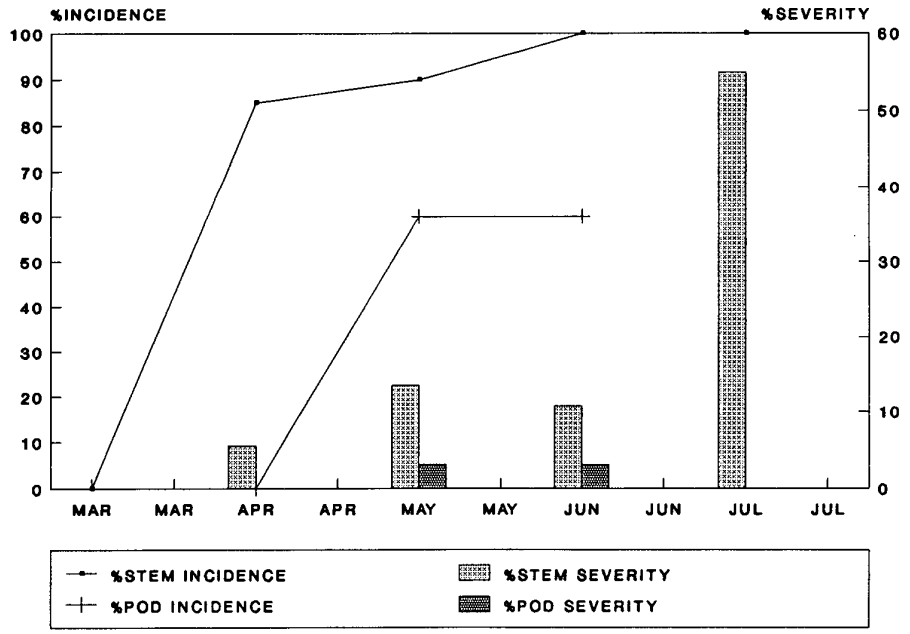


FIGURE 18B : THURLOXTON LIGHT LEAF SPOT
STEM DISEASE, 11 JULY 1994, GS6.5

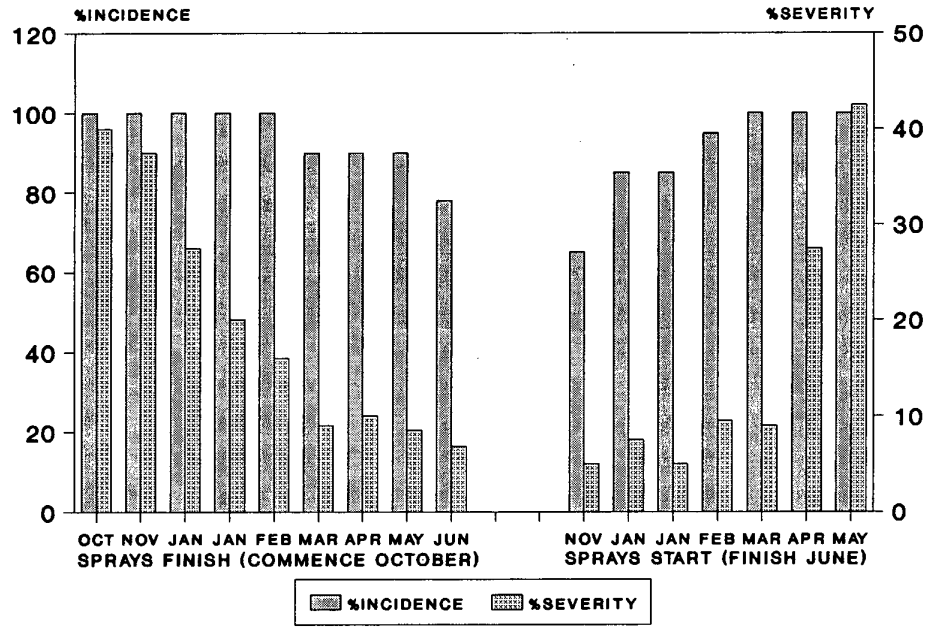


FIGURE 18C : THURLOXTON YIELD 1994(T\HA)

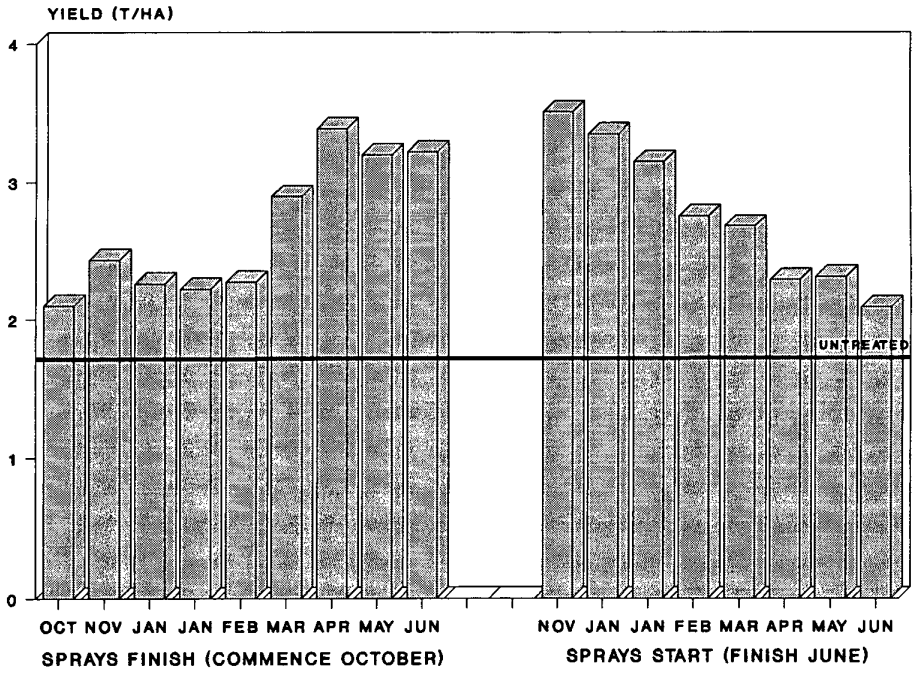
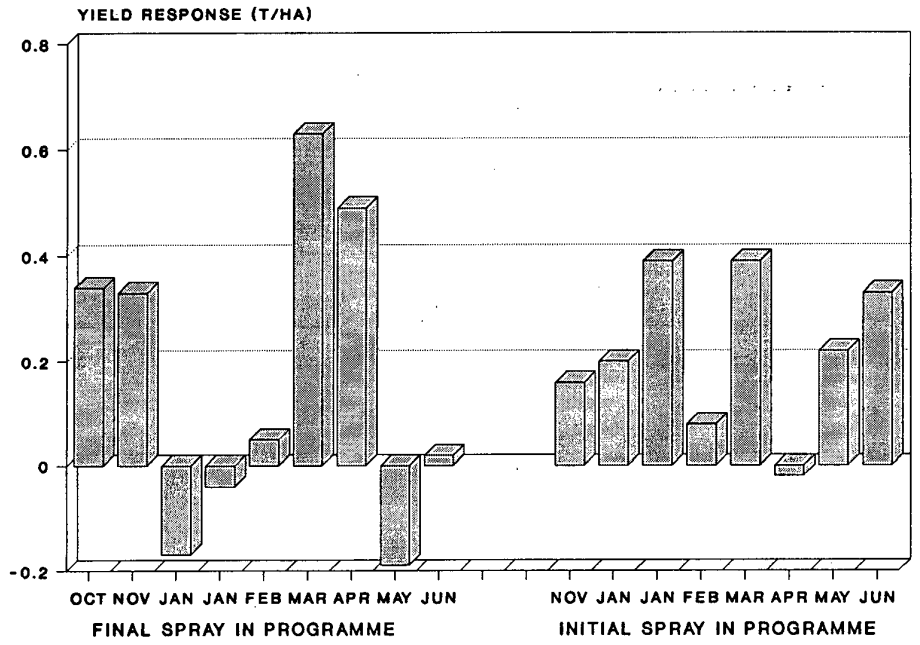


FIGURE 18D : THURLOXTON YIELD 1994
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(ix) Udny Station 1993/94

The main disease at this site was light leaf spot. Figure 19 illustrates the development of foliar disease in untreated plots. Symptoms were first seen on 24 January when 50 per cent of untreated plants were affected at 5.3 per cent severity. The disease disappeared in early March, most likely the result of older infected leaves dropping off, but it returned to a maximum of 80 per cent incidence, 5.7 per cent severity on 30 March (GS 3.1 / 3.3). Symptoms declined thereafter but were still present in June (GS 5.7).

Light leaf spot developed on the stems but not the pods. Figure 19A illustrates the development of stem symptoms. Low levels of disease were first seen on 17 June (GS 5.7) when 45 per cent of plants were affected but the disease was not severe (0.38 per cent stem area). The disease developed to affect 65 per cent of stems by 25 July (GS 6.3) at 5.4 per cent severity.

At the time of maximum foliar symptoms in late March, all fungicide treatments applied significantly reduced disease incidence from 80 to $\leq$ 45 per cent plants affected and disease severity from 5.7 to $\leq$ 2.6 per cent leaf area. All treatments that included a 30 November spray gave the best reductions in disease with $\leq$ 20 per cent incidence and 0.35 per cent severity.

The effect of fungicide treatment on the incidence and severity of light leaf spot symptoms on the stems on 25 July (GS 6.3) is illustrated in Figure 19B. Whilst there were significant differences in disease incidence between treatments there were no significant differences between treated and untreated plots. However, Treatment 6 (6 sprays, 29 October to 3 March), and 22 to 20 (initial spray between 30 November and 24 January) had the lowest disease incidence ($\leq$ 20 per cent). Disease severity was significantly reduced from 5.4 per cent leaf area affected in untreated plots to $\leq$ 1.05 per cent in Treatments 5, 6, 7, 8, 10 (initial spray on 29 October, final spray on or after

24 January, latest final spray = 14 July in Treatment 10), and 20 to 22 (initial spray on or before 24 January).

The untreated yield at this site was 3.93 t/ha. Figure 19C shows the yields obtained from all of the treatments and Figure 19D shows the responses attributable to individual spray timings obtained by subtraction of yields from related treatments. Yields significantly greater than the untreated were obtained from several unrelated treatments although all treated plots had greater yields than the untreated, ranging between 3.95 and 4.92 t/ha (0.02 to 0.99 t/ha greater than the untreated plots). Treatments 3, 5, 8, 9, 10, and 21 and 22 all had significantly higher yields than the untreated. Treatment 8 (7 monthly sprays, October to May) gave the highest yield of 4.92 t/ha.

A single spray in October (Treatment 2) did not significantly increase yield over the untreated (3.95 t/ha). There was a significant benefit of 0.58 t/ha from the addition of a November spray to the October treatment (Treatment 3) (Figure 19D). The May spray in Treatment 8 gave the greatest yield response as a final spray in a treatment, it yielded 0.64 t/ha when compared to Treatment 7 (Figure 19D). The pattern of yield benefit from additional sprays applied December to May was not clear. Delaying the start of spraying until later in the season did not have as obvious an effect on yield as stopping sprays early. However, in general the greater the number of sprays applied the larger the yield.

The results of regression analyses of the incidence and severity of light leaf spot infection of the stem on 25 July (GS 6.3) are shown in Table 19.

Table 19. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of light leaf spot (X) assessed on 25 July (GS 6.3)

X parameter	Regression equation	Correlation coefficient(r)*
% Incidence stem light leaf spot	Y = 4.59 - 0.007X	-0.42
% Severity stem light leaf spot	Y = 4.53 - 0.071X	-0.60

*$p \leq 0.05$

The relationship between the incidence of stem infection by light leaf spot and yield was weak with a correlation coefficient of -0.42. The relationship between the severity of stem infection and yield was slightly stronger at -0.60. Both values of r were significant however ($p \leq 0.05$) and the value of the slope b in each equation was significantly different from zero at a probability of 99.99 per cent. For every 1 per cent increase in the incidence of light leaf spot infection of the stem there was a loss in yield of 0.007 t/ha.

FIGURE 19 : UDNY STATION 1993/94
LIGHT LEAF SPOT DEVELOPMENT

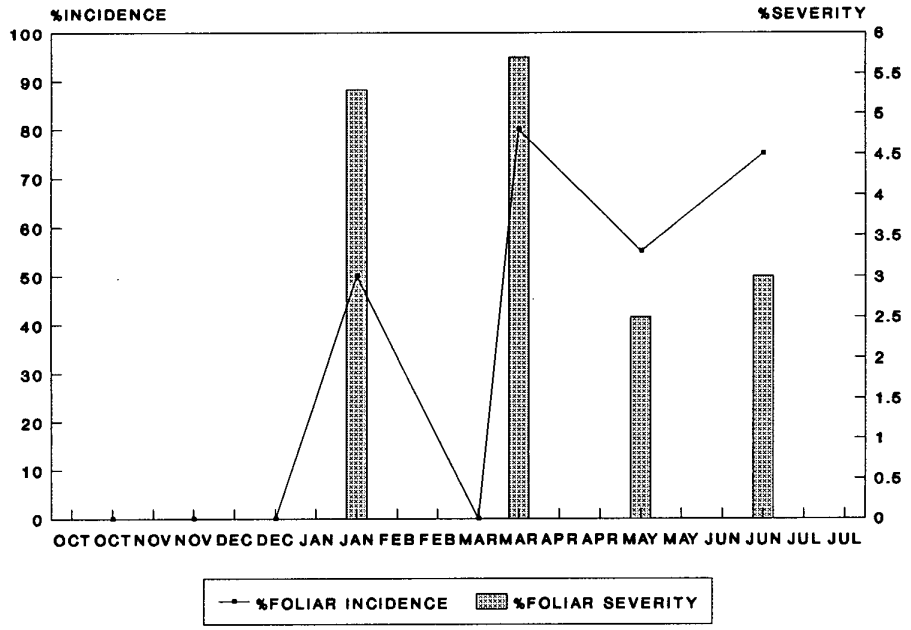


FIGURE 19A : UDNY STATION 1993/94
STEM LIGHT LEAF SPOT

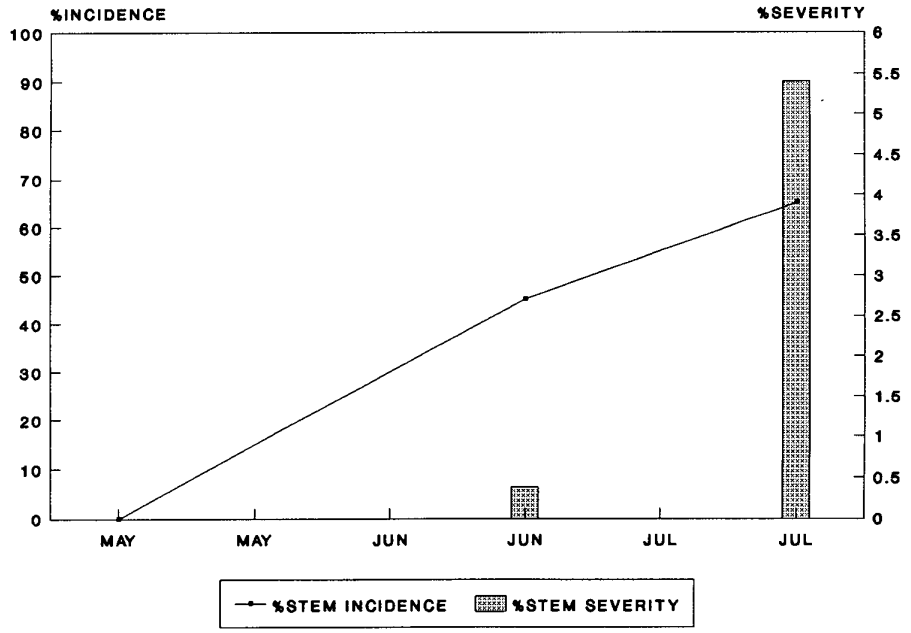


FIGURE 19B :UDNY STATION LIGHT LEAF SPOT
STEM DISEASE, 25 JULY 1994, GS6.3

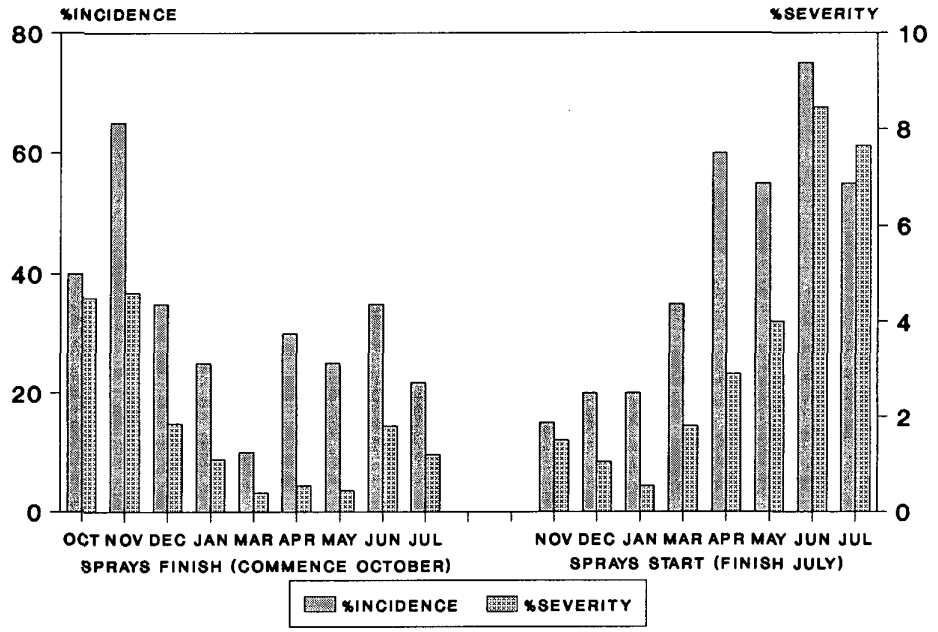


FIGURE 19C : U.STATION YIELD 1994 (T/HA)

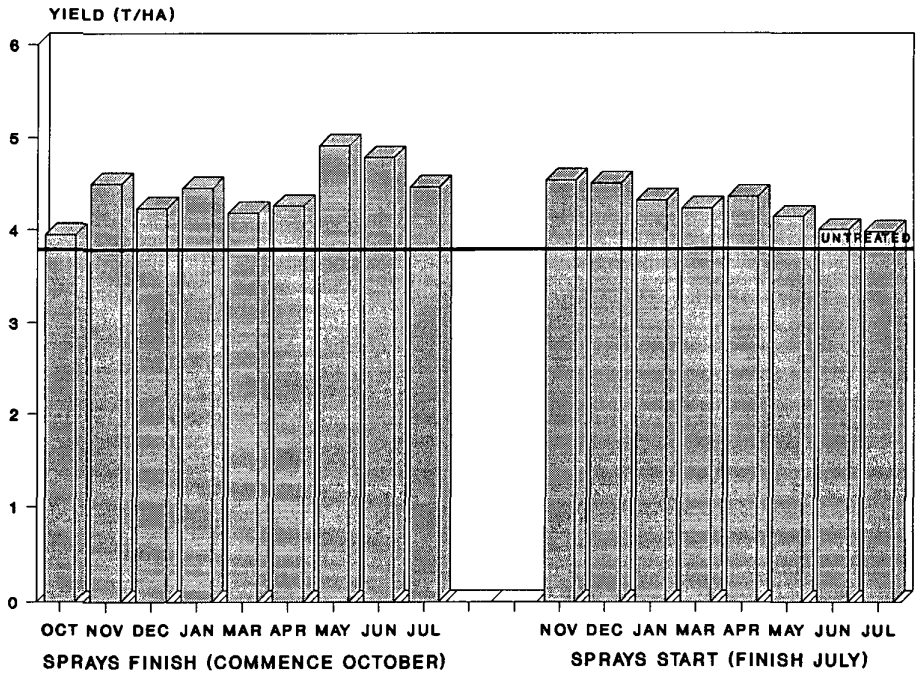
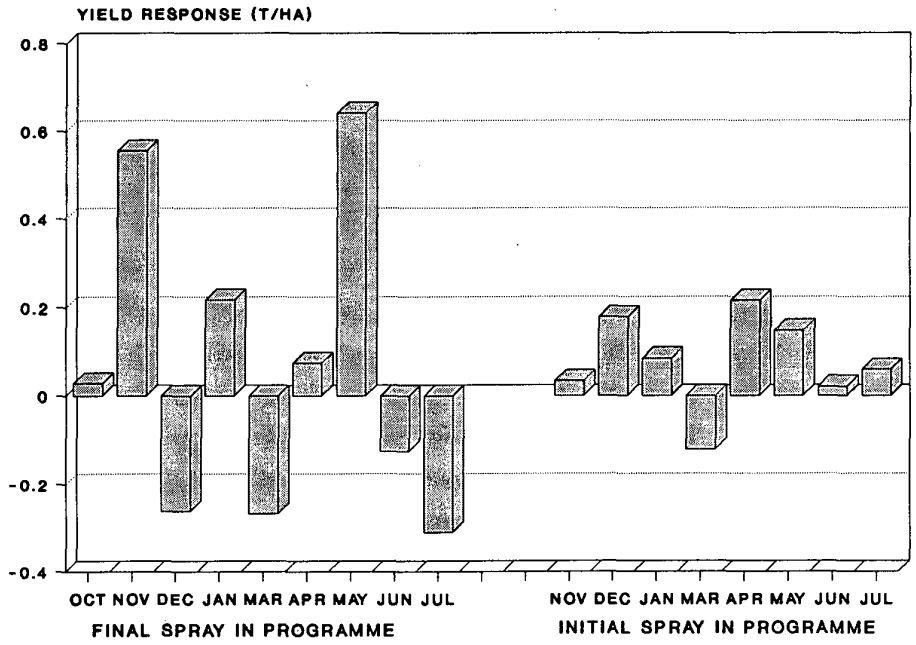


FIGURE 19D : U.STATION YIELD 1994
EFFECT OF INDIVIDUAL SPRAY TIMINGS



2.1 Light leaf spot summary

Data from sites where light leaf spot developed are summarised in Tables 2.1 a, b, c and d.

The leaf spot phase of the disease is summarised in Table 2.1 a. Light leaf spot developed at nine of the fourteen experiment sites between 1991 and 1994. At six of these sites *Phoma* leaf spot also developed. First symptoms of light leaf spot were seen at Rothamsted (1992/93) during November, at two of the Scottish sites during December, at three sites during January, and at three sites during February. Foliar disease severity in untreated plots exceeded 1.0 per cent leaf area affected before or during January at Rothamsted and Pettymuick (1992/93) and Udny Station and Thurloxton (1993/94), affecting 13.7, 7.7, 5.3 and 9.4 per cent leaf area respectively. Maximum foliar severity was in general attained later than January. At Foveron (1991/92) the lowest maximum severity of all the sites was recorded during April (2.2 per cent). At Tarrant Hinton and Boxworth (1992/93) and Rothamsted (1993/94) where disease during January was only present at one site (Boxworth, 0.1 per cent severity), the maximum foliar severity occurred during March and May (Rothamsted), affecting 6.6, 8.0 and 6.1 per cent leaf area respectively. At Udny Station (1993/94) the maximum foliar severity occurred during March also (5.7 per cent). Foliar disease was far more severe at Rothamsted (1992/93; 13.7 per cent, January), Rosemaund (1993/94; 15.5 per cent, April), Pettymuick (1992/93; 21.2 per cent, March) and at the worst affected site Thurloxton (1993/94) 63.9 per cent leaf area was affected during April.

The development and control of stem and pod infection by light leaf spot is summarised in Table 2.1 b. Light leaf spot developed on the stems at all of the sites, and on the pods at all but two of the sites (Boxworth 1992/93, Udny Station 1993/94). First stem symptoms of light leaf spot were detected between March (Rothamsted and Tarrant Hinton 1992/93) and June (Rosemaund and Udny Station 1993/94).

The incidence of light leaf spot on the stems in untreated plots exceeded 53 per cent of plants affected at the final disease assessment. The lowest disease incidence (53 per cent) occurred at Rothamsted (1992/93), the highest at Foveran in 1991/92, and Rothamsted and Thurloxton in 1993/94 when all assessed plants were affected by light leaf spot on the stems. Stem disease severity was also at its lowest at Rothamsted (1992/93, 0.50 per cent area affected), and at its most severe at Thurloxton (1993/94) when an exceedingly high level of disease was recorded (55 per cent stem area affected). Stem infection was controlled at all sites except Tarrant Hinton (1992/93) by one or two fungicide sprays applied mainly between November and February, although October and March sprays had some effect at some sites.

The final incidence of light leaf spot infection on the pods in untreated plots at the seven affected sites ranged between 15 per cent (Rothamsted 1993/94) and 95 per cent (Pettymuick and Rothamsted 1992/93). Pod disease severity at the affected sites was also at its lowest (0.2 per cent) at Rothamsted (1993/94), but the highest severity occurred at Tarrant Hinton (1992/93) where 5.3 per cent of the pod area showed symptoms. Pod infection was controlled by various treatments at all affected sites except Rothamsted and Thurloxton (1993/94). No specific spray timing for disease control could be identified.

Regression analyses of maximum foliar severity during the season (Y) versus stem and pod severity and incidence at pod ripening (X) were performed. Only one moderately strong and significant relationship was found to exist; leaf severity was related to final stem severity as shown in the regression equation below.

$$Y = 7.57 + 0.457\ X$$

The value of r was 0.69 which was significant at $p = 0.025$. The value of the slope b was significantly different from zero at 95 per cent supporting the strength of the

relationship. This equation showed that for every 1 per cent maximum foliar severity that occurred there was an associated 8 per cent stem area likely to be affected at pod ripening. Regression analyses of final pod incidence and severity with final stem incidence and severity showed that no significant relationships existed between stem and pod light leaf spot.

The relationship between light leaf spot and yield expressed as regression equations is summarised in Table 2.1 c. The disease did not affect yield at two sites (Boxworth and Tarrant Hinton, 1992/93). Pod infection only affected yield at two of the seven sites where pod disease developed (Pettymuick 1992/93, Rosemaund 1993/94). At all of the sites where canker developed, this disease also affected yield and further work is required to separate the effect of the presence of both diseases on yield.

Table 2.1 d summarises the yield losses associated with light leaf spot infection. By extrapolation, it was possible to determine the effect of the disease on yield at each site when 100 per cent of plants were affected by disease; also yield losses associated with 1 per cent stem or pod area affected are presented. At Rothamsted and Udny Station (1993/94) the relationship between the incidence and severity of light leaf spot on the stems expressed by the correlation coefficient (r) was weak and yield losses associated with 100 per cent disease incidence were the lowest found (0.5 and 0.7 t/ha respectively). At Foveran (1991/92 and Rothamsted (1992/93) 0.9 t/ha was lost by extrapolation for 100 per cent disease incidence (stems). At Rosemaund (1993/94) and Pettymuick (1992/93) 1.0 and 1.5 t/ha were lost for 100 per cent disease incidence (stems). At these two sites pod disease also affected yield and 0.8 and 1.7 t/ha were lost respectively by extrapolation for 100 per cent disease incidence.

The most dramatic effect of stem disease on yield occurred at Thurloxton (1993/94) where by extrapolation 4.4 t/ha would be lost if 100 per cent of plants were affected by stem disease at pod ripening.

The strongest correlation coefficients between disease incidence and severity versus yield occurred at Foveron (1991/92) and Thurloxton (1993/94) ($r \geq 0.78$).

There was no consistent relationship between disease incidence and severity and their effect on yield at all of the sites. Regression analysis of the relationship between yield loss associated with stem disease incidence and stem disease severity confirmed that there was no correlation between the two factors.

In general (with the exception of Foveran 1991/92), light leaf spot had the greatest effect on yield where foliar disease severity exceeded 13.0 per cent during the season. With the exception of Rosemaund (1993/94) where the values of r were not particularly high (≤ 0.67) and no disease was detected during or before January, and Foveran (1991/92) the disease affected yield only at those sites where foliar disease during January exceeded 5 per cent of the leaf area.

At the seven sites where yield was related to the incidence or severity of light leaf spot, canker was present at four of the sites and also affected yield. At these four sites the maximum percentage yield increase in response to treatment and control of both diseases ranged between 21 per cent (Rothamsted, 1993/94), and 99 per cent (Thurloxton, 1993/94), with 47 and 71 per cent response in yield at Rothamsted (1992/93) and Rosemaund (1993/94). At the remaining three sites canker was not present and the maximum percentage yield increase in response to treatment and control of light leaf spot only was 25, 30 and 85 per cent at Foveran (1991/92), Udny Station (1993/94) and Pettymuick (1992/93) respectively.

Table 2.1a Summary of foliar light leaf spot incidence and severity on untreated plots and the timing of fungicide for control.

Site	Year	Foliar light leaf spot					
		First Symptoms	Maximum leaf severity†			Treatments to control disease	
		Month	%I*	%S**	Month	Control Y/N	Timings Month(s)
Foveran	1991/92	December	70	2.2 (0.4)	April	Yes	November-February
Boxworth[ab]	1992/93	January	73	8.0 (0.1)	March	Yes	October-January
Pettymuick	1992/93	December	100	21.2 (7.7)	March	Yes	October-February
Rothamsted[ab]	1992/93	November	100	13.7	January	Yes	November-January
Tarrant Hinton[ab]	1992/93	February	90	6.6 (0.0)	March	Yes	October-February
Rosemaund[ab]	1993/94	February	90	15.5 (0.0)	April	Yes	October-February
Rothamsted[ab]	1993/94	February	100	6.1 (0.0)	May	Yes	December-April
Thurloxton[ab]	1993/94	January	100	63.9 (9.4)	April	Yes	November-March
Udny Station	1993/94	January	80	5.7 (5.3)	March	Yes	November

† Untreated plots

* % Incidence of plants affected during the month with maximum severity

** Maximum % leaf area affected; where disease severity reached its maximum after January the maximum severity between October and January is shown in brackets.

[ab] = Sites with *Phoma* leaf spot and canker; *Phoma* affected yield at all of these sites

Table 2.1b Summary of stem and pod light leaf spot incidence and severity on untreated plots and the timing of fungicide for control

Site	Year	Stem light leaf spot					Pod light leaf spot		
		First symptoms		Pod ripening disease†		Treatments to control disease	Final disease†		Treatments to control disease
		Month	GS	%I	%S	Month	% I	% S	Month
Foveran	1991/92	April	4.0	100	20.4	November-February	27	4.2	Various
Boxworth[ab]	1992/93	April	3.7	65	3.6	October-January	0	0.0	-
Pettymuick	1992/93	May	3.5/4.0	97	8.0	October-March	95	2.5	Various
Rothamsted[ab]	1992/93	March	3.5	53	0.5	January-February	95	3.2	Various
Tarrant Hinton[ab]	1992/93	March	2.09/3.6	90	3.5	None	53	5.3	Various
Rosemaund[ab]	1993/94	June	4.8/6.2	95	5.7	January (Nov-Jan)	65	3.2	March
Rothamsted[ab]	1993/94	May	4.3	100	2.3	February (Nov-Feb)	15	0.2	None
Thurloxton[ab]	1993/94	April	2.08/3.7	100	55.0	November-March	60	3.1	None
Udny Station	1993/94	June	5.7	65	5.4	October-January	0	0.0	-

† Untreated plots

[ab] = Sites with *Phoma* leaf spot and canker; *Phoma* affected yield at all of these sites.

Table 2.1c Summary of light leaf spot and its relationship with yield

Site	Year	Yield and light leaf spot*				Maximum % yield response**
		Disease related (Y/N)		Regression equation Y = Yield (t/ha) X = % Incidence	Regression equation Y = Yield (t/ha) X = % Severity	
		Pod	Stem			
Foveran	1991/92	N	Y	Y = 4.61 - 0.009X (-0.78)	Y = 4.39 - 0.062X (-0.88)	30
Boxworth[ab]	1992/93	N	N	-	-	-
Pettymuick	1992/93	Y	Y	Y = 4.28 - 0.015X[c] (-0.67) Y = 4.33 - 0.017X[d] (-0.67)	Y = 3.50 - 0.12X[c] (-0.64) Y = 3.83 - 0.45X[d] (-0.67)	85
Rothamsted[ab]	1992/93	N	Y	Y = 4.71 - 0.009X (r = -0.65)	Y = 4.68 - 0.982X (r = -0.66)	47
Tarrant Hinton[ab]	1992/93	N	N	-	-	-
Rosemaund[ab]	1993/94	Y	Y	Y = 2.97 - 0.010X[c] (-0.63) Y = 2.75 - 0.008X[d] (-0.42)	Y = 2.81 - 0.173X[c] (-0.67) Y = 2.70 - 0.163X[d] (-0.47)	71
Rothamsted[ab]	1993/94	N	Y	Y = 3.82 - 0.005X (-0.52)	Y = 3.70 - 0.255X (-0.52)	21
Thurloxton[ab]	1993/94	N	Y	Y = 6.80 - 0.044X (-0.82)	Y = 3.26 - 0.029X (-0.83)	99
Udny Station	1993/94	N	Y	Y = 4.59 - 0.007X (-0.42)	Y = 4.53 - 0.071X (-0.60)	25

* Values of r follow equation (r)

** = (Maximum yield - Untreated yield/Untreated yield) x 100

[ab] = Sites with *Phoma* leaf spot and canker; *Phoma* also affected yield at all of these sites

[c] = Stems

[d] = Pods

Table 2.1d Summary of yield losses associated with light leaf spot and the relationship with foliar light leaf spot severity during the season

Site	Year	Maximum foliar severity (%)		Yield loss (t/ha) per 100% incidence*		Yield loss per 1% severity*	
		October - July	October - January	Pod	Stem	Pod	Stem
Tarrant Hinton [ab]	1992/93	6.6 (March)	0.0 (January)	-	-	-	-
Boxworth [ab]	1992/93	8.0 (March)	0.1 (January)	X	-	X	-
Rothamsted [ab]	1993/94	6.1 (May)	0.0 (January)	-	0.5 (-0.52)	-	0.255 (-0.52)
Udny Station	1993/94	5.7 (March)	5.3 (January)	X	0.7 (-0.42)	X	0.071 (-0.60)
Foveran	1991/92	2.2 (April)	0.4 (January)	-	0.9 (-0.78)	-	0.062 (-0.88)
Rothamsted [ab]	1992/93	13.7 (January)	13.7 (January)	-	0.9 (-0.65)	-	0.982 (-0.66)
Rosemaund [ab]	1993/94	15.5 (April)	0.0 (January)	0.8 (-0.42)	1.0 (-0.63)	0.163 (-0.47)	0.173 (-0.67)
Pettymuick	1992/93	21.2 (March)	7.7 (January)	1.7 (-0.67)	1.5 (-0.67)	0.45 (-0.67)	0.120 (-0.64)
Thurloxton [ab]	1993/94	63.9 (April)	9.4 (January)	-	4.4 (-0.82	-	0.029 (-0.83)

* Value of the correlation coefficient (r) follows yield loss
X = No pod disease
\- = No relationship between disease and yield
ab = Sites with *Phoma* leaf spot and canker; *Phoma* affected yield at all of these sites.

3. Sclerotinia stem rot

Sclerotinia stem rot was detected at several sites during the course of the study but only developed to damaging levels at Kington Langley, Wiltshire and at Rosemaund, Herefordshire during the summer of 1992. Disease development in untreated plots and the effect of fungicide treatment on disease and yield is detailed below.

(i) Kington Langley 1991/92

Both canker and sclerotinia stem rot developed at this site, the former is discussed in Section 1(i). The development of *Sclerotinia* symptoms on the main stem and racemes in untreated plots is shown in Figure 20. The disease was first detected during pod ripening on 8 June (GS 6.2) when 1 per cent of main stems were affected. Raceme infection was not detected until 28 June (GS 6.4) when 14 per cent of plants showed symptoms at severity 0.4, and 19 per cent of plants had main stem symptoms at a severity score of 0.5. By 8 July (GS 6.8) main stem symptoms had not increased (18 per cent incidence, 0.6 severity) whereas symptoms on the racemes had increased to affect 27 per cent of plants at a severity score of 1.0.

The effect of fungicide treatment on the incidence of *Sclerotinia* on main stems and racemes on 8 July (GS 6.8) is illustrated in Figure 20A. None of the treatments resulted in a significant reduction in disease on the main stem but the most effective treatments were those that incorporated post-flowering sprays in early June (Treatments 11 to 21). Treatments that finished during the early spring (6, 7 and 9) resulted in significant increases in disease. The worst affected (7) had 66 per cent of stems with symptoms.

Sclerotinia infection of racemes was found on 27 per cent of untreated plants at pod ripening. As with infection of the main stem the best control came from treatments that included sprays in June. Significant reductions in disease incidence occurred where treatments commenced between 24 February and 5 June, with final sprays on 25 June (Treatments 14 to 18, ≤6 per cent).

Overall, 43 per cent of plants were affected by *Sclerotinia* either on the main stem, the racemes, or both. Treatment 17 (received 5 sprays between 17 March and 25 June) showed the lowest (significant) incidence of disease overall (4 per cent) but Treatment 16 which was initiated as the first flowers were seen in mid-April was also very effective (6 per cent). No sprays were applied at the early petal fall stage and the results provide some indication that post-flowering sprays can give reductions in the incidence of sclerotinia stem rot.

However it does seem possible that fungicide treatment can in some circumstances enhance disease symptoms. This may be related to the destruction of naturally-occurring fungal competitors, antagonists or biological control agents by the fungicide applied just prior to ascospores of *S. sclerotiorum* attempting to infect plant tissue.

The untreated yield at Kington Langley was 3.42 t/ha. Figure 20B shows the yields obtained for individual treatments and Figure 20C the responses attributable to individual spray timings obtained by subtraction of yields from related treatments.

Significant increases in yield were obtained from Treatments 16, 20 and 21 (0.44, 0.47, and 0.70 t/ha). The maximum response to treatment (0.70 t/ha) amounted to 21 per cent of the untreated yield. Trends in the yields suggest that yield responses were related to the control of *Sclerotinia*. Treatments which were completed in early spring appeared to increase the incidence of *Sclerotinia* and gave a non-significant yield reduction. The worst affected (Treatment 7) had 66 per cent of stems affected (untreated 18 per cent) with a yield 0.32 t/ha less than the control.

Regression analyses of the incidence of *Sclerotinia* on the main stems, racemes, or both in relation to yield are presented in Table 20.

Table 20. Regression analyses of yield (t/ha) (Y) versus the incidence of *Sclerotinia* on main stems, racemes, or both (X) assessed on 8 July (GS 6.8)

X parameter	**Regression equation**	**Correlation coefficient (r)***
% Main stems	Y = 3.78 - 0.0098X	-0.65
% Racemes	Y = 3.80 - 0.0109	-0.57
% Main stems plus racemes	Y = 3.84 - 0.071	-0.68

*p ≤0. 0.006 for all values of r

The relationships between yield and the incidence of *Sclerotinia* in July as measured by the correlation coefficients r were highly significant (p ≤0. 0.006) with moderately strong values of r ranging from -0.57 to -0.68 depending upon the variable measured. For all analyses the value of the slope b in the regression equations was significantly different from zero at a probability of 99.99 per cent indicating a strong relationship between yield and the incidence of *Sclerotinia*.

Regression analysis also showed that for every 1 per cent of main stems or racemes affected by *Sclerotinia* approximately 0.01 t/ha yield was lost.

Yield did not appear to be affected by canker at this site (Section 1(i)).

FIGURE 20 : KINGTON LANGLEY 1991/92
SCLEROTINIA DEVELOPMENT

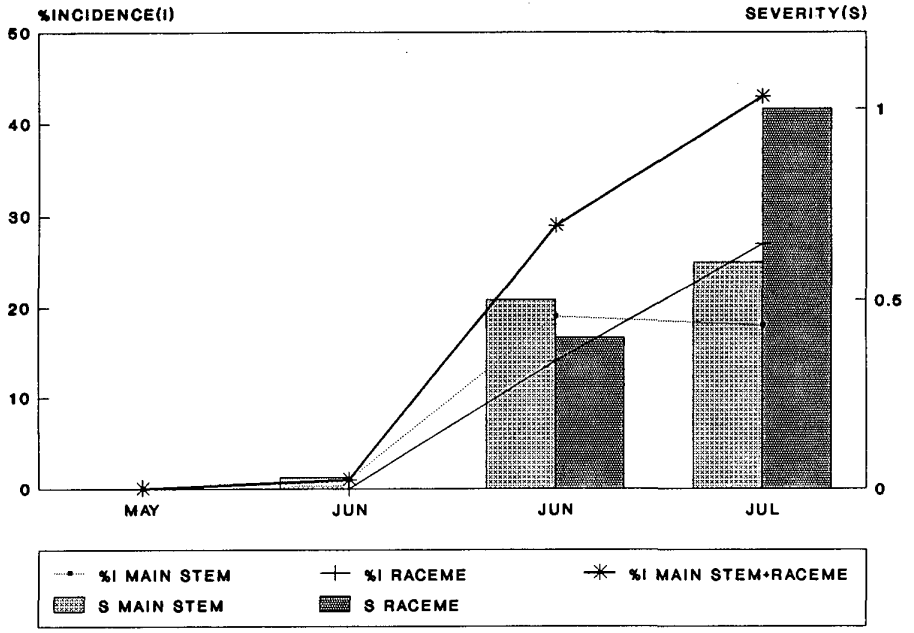


FIGURE 20A : K.LANGLEY SCLEROTINIA
8 JULY 1992, GS6.8

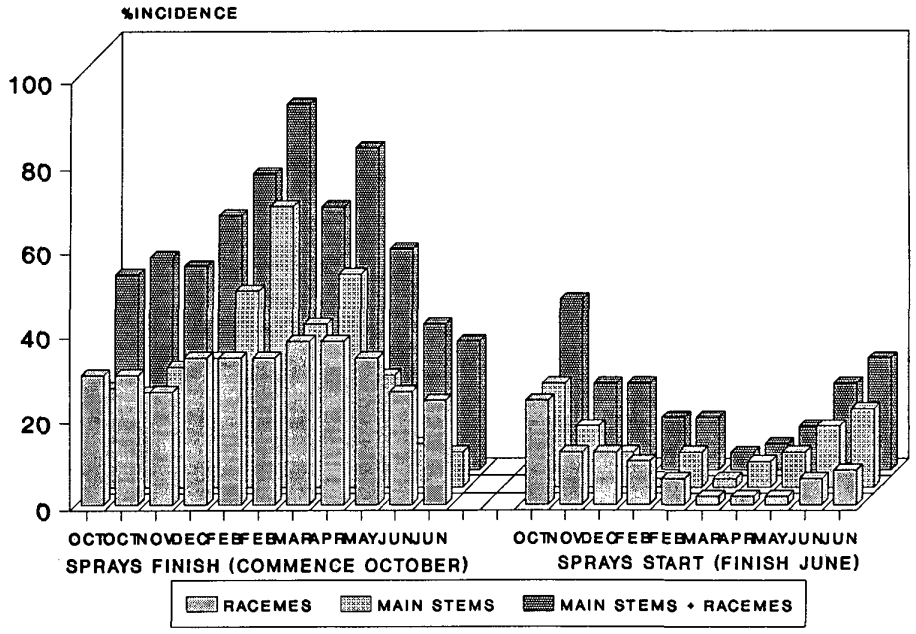


FIGURE 20B : K.LANGLEY YIELD 1992 (T\HA)

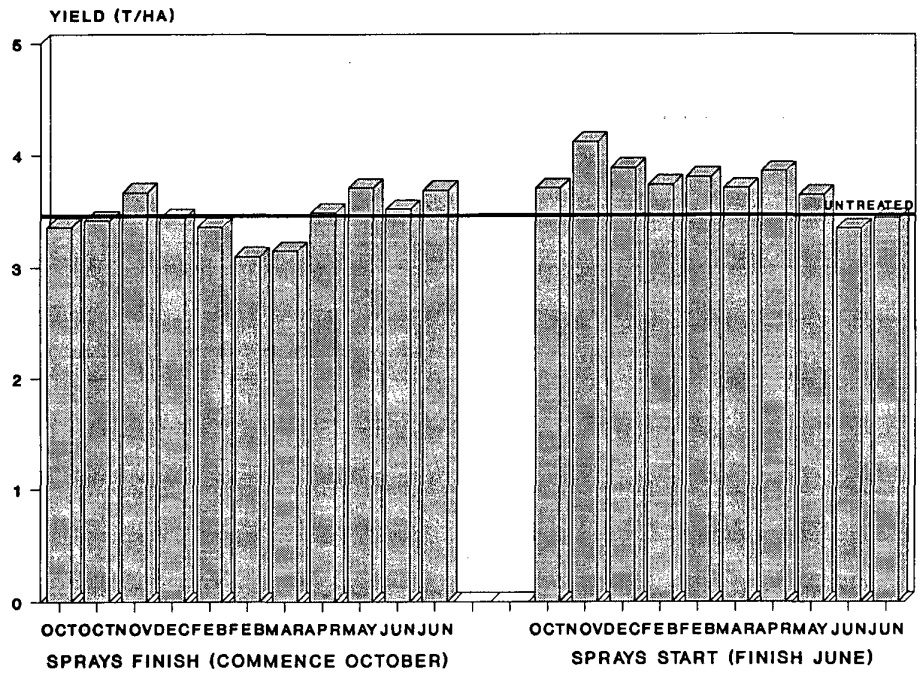
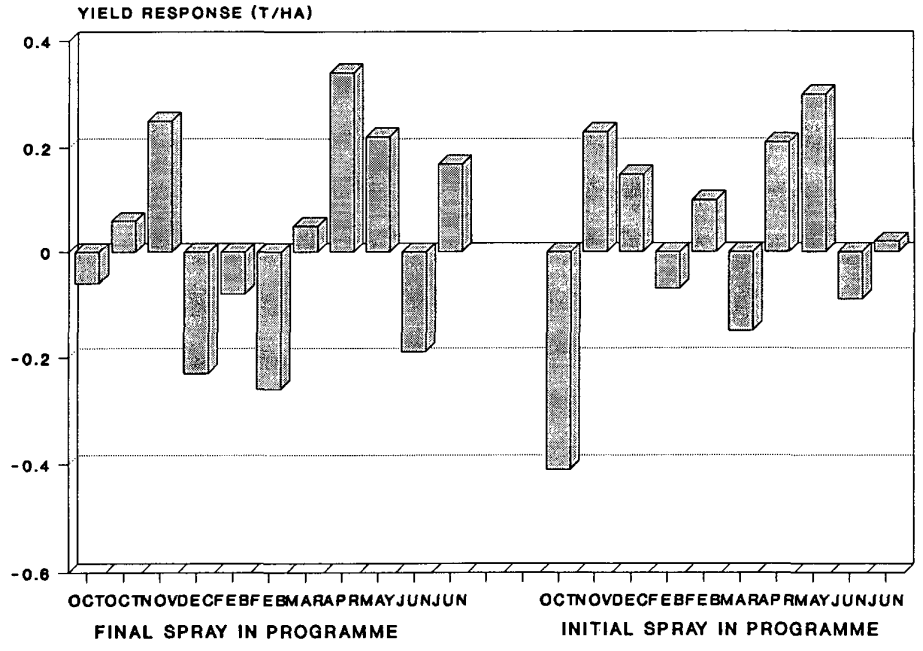


FIGURE 20C : K.LANGLEY YIELD 1992
EFFECT OF INDIVIDUAL SPRAY TIMINGS



(ii) Rosemaund 1991/92

The development of symptoms on the main stem and racemes in untreated plots is shown in Figure 21. The disease was first detected during pod ripening; on 16 June (GS 6.3) 66 per cent of main stems were affected at severity 1.5, raceme infection was not detected at this time. By 14 July (GS 6.5) 58 per cent of stems were affected at a severity score of 2.1. In addition 34 per cent of racemes were affected at severity score 1.2.

The effect of fungicide treatment on the incidence and severity respectively on main stems and racemes on 14 July is illustrated in Figure 21A and 21B. All of the treatments that included a late flowering spray on 18 May (GS 4.8) (Treatments 10, 11, and 15 to 22) resulted in a significant reduction in both the incidence (≤ 18 per cent) and severity (≤ 0.6) of *Sclerotinia* on the main stems.

All fungicide treatments that included both of the flowering sprays (29 April, GS 4.7; 18 May, GS 4.8) led to a significant reduction in the incidence and severity of disease to ≤ 10 and ≤ 0.4 per cent respectively (Treatments 10, 11, and 16 to 22).

The incidence of *Sclerotinia* on the main stems combined with the racemes was significantly reduced from 84 per cent in untreated plots to ≤ 36 per cent by all of the treatments that included the late-flowering spray (18 May, GS 4.8) (10, 11, and 15 to 22).

The pods were severely affected by sooty moulds on 14 July. Seventy per cent of the area of untreated pods was affected. Significant reductions in the severity of symptoms to ≤ 46.8 per cent pod area occurred with the same treatments that controlled S*clerotinia*. This was most likely due to a delay in pod ripening attributable to the control of *Sclerotinia*.

The untreated yield at Rosemaund was 2.69 t/ha. Figure 21C shows the yields obtained for individual treatments, and Figure 21D the responses attributable to individual spray timings obtained by subtraction of yields from related treatments.

Yields significantly greater than the untreated, but not from each other were obtained from all of the treatments that included both of the late flowering sprays in combination (GS 4.7 and 4.8, 29 April and 18 May) (Treatments 10, 11, 16 to 22, except Treatment 19 which gave the largest yield response of the non-significant yields (0.73 t/ha)). The largest overall response came from Treatment 10 (1.43 t/ha) which received all except the final spray in June. This amounted to 53 per cent of the untreated yield. These yield responses were directly related to the control of *Sclerotinia* on the main stems and racemes.

The major contribution to yield was from the spray applied on 18 May (GS 4.8). As a final spray (Treatment 10) it produced 1.25 t/ha more than when sprays finished on 29 April (Treatment 9). Likewise as an initial spray (Treatment 15) it produced 0.81 t/ha more than when sprays commenced on 17 June (Treatment 14). (Figure 21D).

Regression analyses of the incidence and severity of *Sclerotinia* on the racemes and stems on 14 July in relation to yield are presented in Table 21.

Table 21. Regression analyses of yield (t/ha) (Y) versus the incidence and severity of *Sclerotinia* (X) assessed on 14 July (GS 6.5)

X parameter	Regression equation	Correlation coefficient(r)*
% Main stems	Y = 3.82 - 0.0160X	-0.88
% Racemes	Y = 3.82 - 0.0319X	-0.82
% Main stems plus racemes	Y = 3.89 - 0.0128X	-0.90
Main stem severity	Y = 3.80 - 0.423X	-0.87
Raceme severity	Y = 3.82 - 0.838X	-0.82

* $p \leq 0.001$ for all values of r

The relationships between yield and *Sclerotinia* in July as measured by the correlation coefficient r were strong and highly significant ($p \leq 0.001$) with values of r ranging from -0.82 to -0.90 depending upon the variable measured. For all analyses the value of the slope b in the regression equations was significantly different from zero at a probability of 99.99 per cent indicating a strong relationship between yield and the incidence or severity of *Sclerotinia.*

Regression analysis also showed that for every 1 per cent of main stems affected, 0.016 t/ha was lost, twice as much yield was lost when racemes were affected (0.032 t/ha). The disease therefore appears to be twice as damaging to yield when it occurs on the racemes.

FIGURE 21 : ROSEMAUND 1991/92
SCLEROTINIA DEVELOPMENT

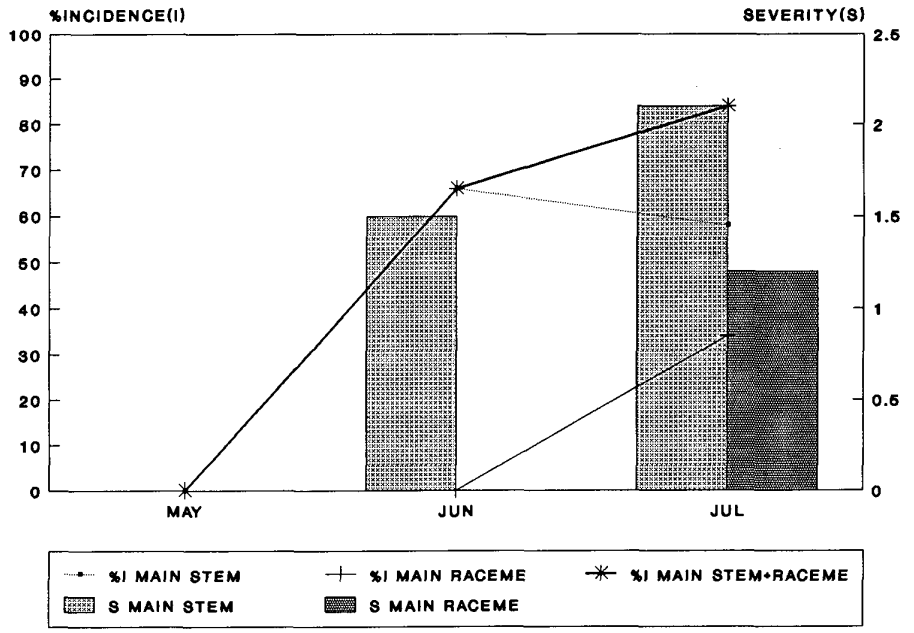


FIGURE 21A : ROSEMAUND SCLEROTINIA
14 JULY 1992, GS6.4-6.9

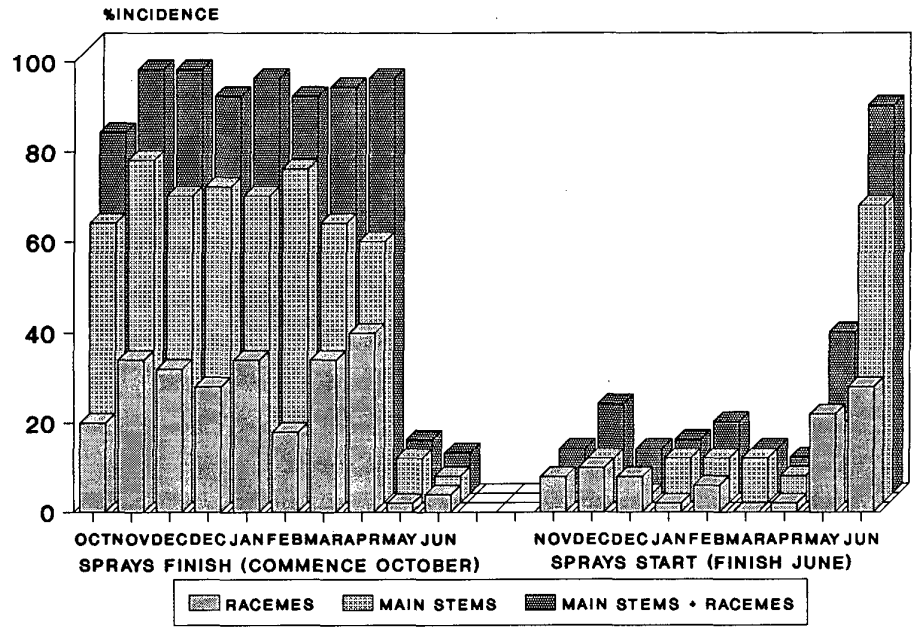


FIGURE 21B : ROSEMAUND SCLEROTINIA
14 JULY 1992, GS6.4-6.9

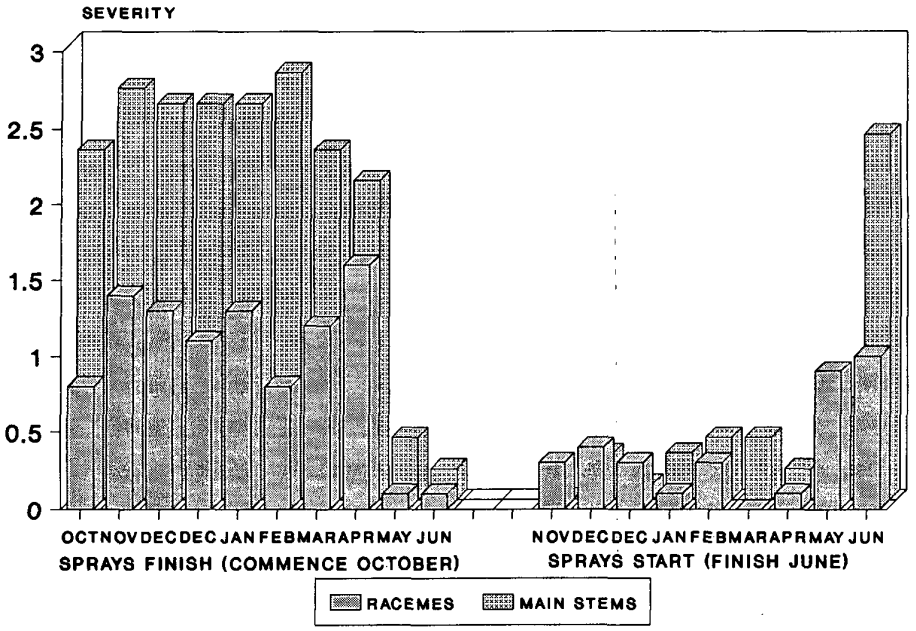


FIGURE 21C : ROSEMAUND YIELD (T\HA)

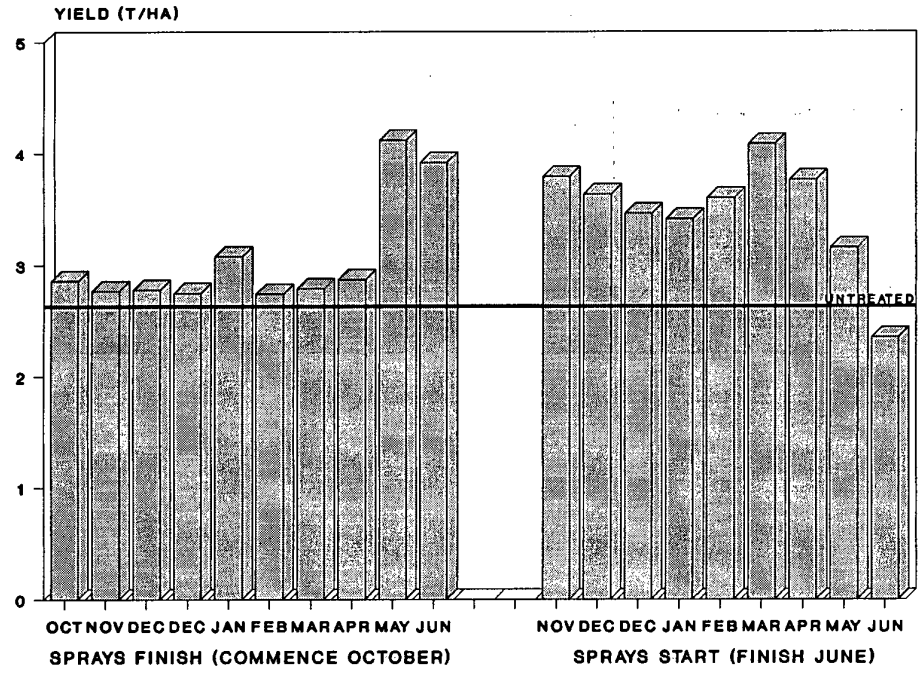
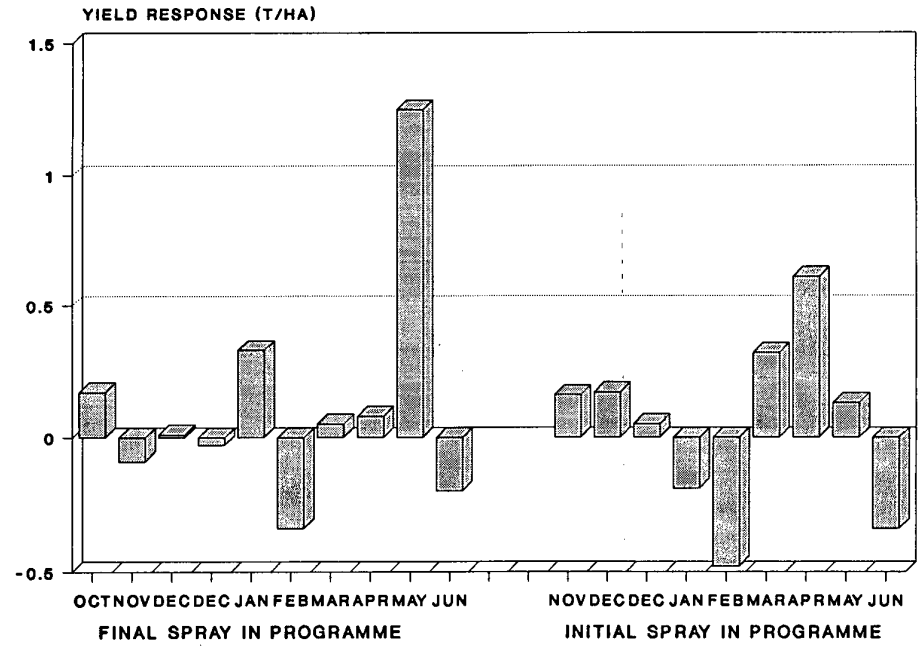


FIGURE 21D : ROSEMAUND YIELD 1992
EFFECT OF INDIVIDUAL SPRAY TIMINGS



3.1 *Sclerotinia* summary

Sclerotinia was present at significant levels at two sites only during the course of this study. Symptoms of infection were detected on the main stem and racemes at both sites during June and July of 1992, at the pod ripening stage. In untreated plots at the final assessment in July, 18 and 27 per cent of main stems and racemes were affected respectively at Kington Langley, with the worst disease at Rosemaund, affecting 58 and 34 per cent of main stems and racemes respectively.

At Kington Langley no significant control of disease occurred on the main stems, but the most effective treatments were those that incorporated post-flowering sprays in early June. Treatments that finished in the early spring resulted in significant increases in disease incidence from 18 per cent in the untreated plots to a maximum of 66 per cent, caused by one of the treatments that finished in the early spring, just prior to flowering. This effect could be related to the destruction of beneficial organisms caused by the high number of fungicide sprays received pre-flowering, thus allowing *S. sclerotiorum* to infect the crop. Significant control of the disease on the racemes was obtained by treatments that included sprays in June. No significant increase in symptoms on the racemes was detected at this site.

At Rosemaund significant control of the disease on the main stems and racemes was obtained from late flowering sprays in late April and mid-May.

The disease affected yield at both sites. At Kington Langley approximately 0.01 t/ha yield was lost for every 1 per cent of main stems affected, or for every 1 per cent of plants with disease on the racemes. At Rosemaund higher yield losses occurred possibly because the disease was slightly more severe at this site. For every 1 per cent of main stems affected 0.016 t/ha yield loss occurred, whereas raceme infection was twice as damaging to yield with 0.032 t/ha yield loss occurring for every 1 per cent of plants affected by disease on the racemes.

DISCUSSION

The objective of this study was to determine disease/yield loss relationships in oilseed rape. This objective was fulfilled with respect to the three most important diseases of winter oilseed rape; *Phoma* leaf spot and canker, light leaf spot and sclerotinia stem rot which all developed to damaging levels. Key timings for the control of all three diseases were also identified.

The relationships discussed below were derived by extrapolation from treatments that had received multiple sprays; to validate these relationships fully, further work would be required to examine the effect of single and multiple sprays applied within the periods identified as critical for the control of each disease.

Canker

The leaf spot phase of canker developed at ten of the fourteen sites during the course of the experiment. The incidence of foliar disease was high at all sites but its severity was not particularly high, the maximum occurring at Boxworth (1992/93) when 4.8 per cent leaf area was affected during January. Canker developed at all sites; the lowest incidence of disease was 50 per cent plants affected in late June or early July. There was no statistically demonstrable relationship between the incidence of leaf spotting in November and the incidence of canker at late flowering, or in June, or at harvest (Sansford, 1995a). This is in contrast to the findings of Gladders & Musa (1980) who found a good correlation between the incidence of leaf spotting in November and the incidence of canker at late flowering (June) or severe canker in July on susceptible single low cultivars. However, in this study there was a strong and statistically significant relationship between yield and the incidence of canker at pod ripening at Boxworth, Rothamsted and Withington in 1992/93, and Boxworth, Rosemaund, Rothamsted and Thurloxton in 1993/94. Regression analyses showed that for every 1 per cent increase in disease incidence there was a loss in yield of between 0.005 and 0.02 t/ha. McGee & Emmett (1977) described a strong relationship between yield loss and canker severity, but their calculation could not be

related to these data. At sites where a strong relationship existed between canker incidence and yield the average loss in yield was approximately 0.01 t/ha for every 1 per cent of stems affected by canker. At some sites both canker and light leaf spot developed but at the sites where canker was the only disease and where it affected yield, the maximum response to fungicide treatment was 40 per cent of the untreated yield (Boxworth 1993/94). Data relating yield losses to canker in double-low cultivars were scarce, but in this study it has proven to be particularly damaging to yield. An extremely strong relationship existed between the yield losses associated with the incidence of canker at pod ripening versus the yield losses associated with unit disease severity at pod ripening. It is possible to calculate the likely loss in yield associated with a given incidence and severity of canker at pod ripening from the regression equation. In general yield losses were associated with sites where the mean foliar severity in untreated plots was $\geq$ 1.0 per cent before or during January (Sansford 1995a).

The leaf spot phase of canker was controlled at all except two of the sites; at Kington Langley and Rothamsted no foliar disease control was detected but no effects on yield were detected either. The best foliar disease control was achieved with one or two sprays mainly applied between November and February. These sprays also controlled canker. However, it is not possible to be certain of the economic necessity for disease control in the autumn since the level of disease to trigger a spray ($\geq$1.0 per cent foliar severity before or during January) may only be attained in January, but the first fungicide application is required in November. At current rapeseed prices (£180/t) it is estimated that the cost of a single fungicide application is equivalent to 0.17 t/ha of rapeseed. On current information, the use of two fungicide sprays is likely to be needed to control canker and this would be financially justified at sites where in addition to satisfying the foliar severity criteria predisposing the crop to an economic loss, a minimum of 34 per cent of stems also developed canker by pod ripening, since on average 0.01 t/ha yield loss occurred for every 1 per cent of stems affected by canker at pod ripening (Sansford , 1995a). There is therefore a need for a forecasting system that could predict the risk of this occurring.

Light leaf spot

Light leaf spot on the leaves developed at nine of the fourteen sites during the course of the experiment. The incidence of foliar disease was high at all sites. Foliar disease severity developed to moderate levels at three sites reaching between 13.7 and 21.2 per cent leaf area during the season. Light leaf spot was particularly severe at Thurloxton in April 1994 when 63.9 per cent of the leaf area was affected; this was the most severe level of disease recorded during the study.

Light leaf spot affected the stems at all of the sites affected by foliar light leaf spot with levels at pod ripening ranging between 53 and 100 per cent stems affected, and disease severity ranging between 0.5 and 55 per cent stem area affected. Pod disease was detected at all but two of the sites, ranging between 15 and 95 per cent plants affected and 0.2 and 5.3 per cent pod area affected. The relationships between leaf, stem and pod disease were tested and it was found that a reasonably strong relationship between the maximum foliar severity of light leaf spot on the leaves at any assessment date during the season and the final disease severity on the stems existed. For every 1 per cent leaf area affected there was an associated stem area affected of 8 per cent. There was however no relationship between disease severity on leaves and final area of pods affected, or maximum foliar severity during the season and final pod or stem incidence. Neither were there any relationships between the final incidence or severity of pod and stem disease.

Regression analyses showed that there was a strong and statistically significant relationship between yield and the incidence of light leaf spot infection on the stem at five sites with yield losses ranging between 0.009 and 0.044 t/ha for every 1 per cent of stems affected. At sites where a strong relationship existed between the incidence of light leaf spot infection of the stem and yield the average loss in yield was approximately 0.019 t/ha for every 1 per cent of stems affected by disease. This was nearly twice the loss associated with canker. The severity of stem infection was also strongly related to yield loss at six sites with yield losses ranging between 0.029 and 0.982 t/ha for a severity of 1 per cent. At some sites both canker and light leaf spot

developed, but at the sites where light leaf spot was the only disease and where it affected yield, the maximum response to fungicide treatment was 85 per cent (1.88 t/ha) of the untreated yield (Pettymuick, 1992/93). Little data have been published on the potential of light leaf spot to affect yield in double-low cultivars, but Rawlinson *et al.* (1989) reported that in combination with high levels of *Alternaria* spp, control of both diseases by fungicides increased yield by between 0.96 and 1.58 t/ha compared with 0.16 t/ha in the single-low cultivar Bienvenu. These results have shown that light leaf spot can be extremely damaging when it affects leaves and stems. No previous reports of the effect of stem symptoms on yield have been found. Not surprisingly no relationship existed between the yield losses associated with the incidence of stem infection at pod ripening versus the yield losses associated with unit stem disease severity. Unlike canker it is therefore not possible to calculate the likely loss in yield associated with a given incidence and severity of stem infection at pod ripening. Sutherland *et al.* (1995) found that over the three years that the study was conducted in Scotland the disease did not consistently affect yield in the same way. This was borne-out by data from the English sites.

Light leaf spot affected the pods at seven of the nine sites but yield losses associated with pod infection were only found at two of the sites with no consistent effect being detected.

In general yield losses were associated with sites where the mean foliar severity in untreated plots exceeded 13.0 per cent during the season.

Foliar light leaf spot was controlled at all of the sites by one or two fungicide sprays mainly applied between November and February. Stem disease was also controlled by the same sprays, but no specific spray timings could be identified for the control of pod disease. It is not possible from these data to determine disease levels before or during November that would trigger a decision to spray and achieve economic disease control. At current rapeseed prices (£180/t) it is estimated that the cost of a single fungicide application is equivalent to 0.17 t/ha of rapeseed. By extrapolation, the use of two fungicide sprays to control light leaf spot infection of the stem would be

economically justified at sites where only 17 per cent of stems developed symptoms of stem infection by pod ripening since nearly twice as much yield loss occurred with light leaf spot compared to canker when relating disease incidence to yield.

At sites where both light leaf spot and canker developed to high levels in combination and where both diseases affected yield, maximum responses to fungicide treatment of between 0.72 t/ha (21 per cent of the untreated yield, Rothamsted 1993/94) and 1.75 t/ha (99 per cent, Thurloxton 1993/94) were detected. In combination and in isolation, both of these disease have the potential to cause major losses in the winter oilseed rape crop.

Sclerotinia stem rot

Sclerotinia stem rot only developed to damaging levels at two sites; Kington Langley and Rosemaund, in the summer of 1992. The disease developed on the stems and the racemes at both sites. Post-flowering sprays in June gave some reduction of raceme infection at Kington Langley, but there was no significant reduction of main stem symptoms. Sprays applied just before flowering resulted in significant and large increases in main stem symptoms at this site, which is thought may have been due to a reduction in non-pathogenic organisms which otherwise may have prevented infection by *S. sclerotiorum* (Sansford, 1989). The maximum response to fungicide treatment at this site was 0.72 t/ha (21 per cent of the untreated yield). At Rosemaund, late flowering sprays gave good control of the disease on both the stems and racemes. At Kington Langley there was no direct relationship between disease control and yield responses as some fungicide treatments increased disease and lowered yield. There were however direct relationships between disease incidence and yield with 0.01 t/ha yield lost for every 1 per cent of stems or plants with raceme infection at pod ripening. The maximum response to fungicide treatment at this site was 0.72 t/ha (21 per cent of the untreated yield). At Rosemaund there was a strong relationship between the control of the disease and the resulting yield. The disease was more severe at Rosemaund and as a result 0.016 and 0.032 t/ha were lost respectively for every 1 per cent of stems or plants with raceme infection at pod ripening (Sansford, 1995b). The

maximum response to fungicide treatment at this site was 1.43 t/ha (53 per cent of the untreated yield). Relationships between the incidence of stem rot and yield have been reported (Krüger and Stoltenburg, 1983) but no regression equations were given and no reference to raceme infection was made. Timing of spray applications to control this disease are normally aimed at mid-flowering to ensure fungicide cover on as many petals as possible (Hardwick *et al.* 1991). In this experiment late and post-flowering sprays were effective. It is possible therefore that where disease control is required but has to be delayed, late flowering sprays may have the potential to control the disease. However, more detailed information on apothecial germination (Sansford & Hardwick, 1994) and colonisation of petals (Davies, 1995) could be incorporated into a forecasting scheme aimed at predicting the requirement for and timing of fungicide application to control *Sclerotinia*.

Further work is required to determine more specifically the exact timing and dose of fungicide required within the November to February period, and the flowering period in relation to the development stage of the crop, and to forecasting in relation to precise levels of disease supported by detailed meteorological information.

SUMMARY OF CONCLUSIONS

Canker

1. *Phoma* leaf spot and canker developed at ten of the fourteen sites.

2. The incidence of *Phoma* leaf spot in untreated plots was high at all affected sites but foliar disease severity never exceeded 5 per cent leaf area affected.

3. The incidence of canker in untreated plots at all affected sites was high but the canker severity score varied between low (0.50) and moderate (2.60) (4 = maximum severity).

4. In contrast to work done in the 1980s it was found that no relationships existed between the incidence of leaf spotting by *Phoma* in November and the final incidence and severity of canker at pod ripening.

5. Yield losses due to canker were mainly associated with sites where the mean foliar severity in untreated plots reached $\geq$1.0 per cent before or during January.

6. Individual regression analyses showed that a strong and significant relationship existed between yield and the final incidence of canker at seven of the ten affected sites .

7. Individual regression analyses showed that for every 1 per cent increase in the incidence of canker at pod ripening, there was an associated loss in yield of between 0.005 and 0.02 t/ha, averaging at approximately 0.01 t/ha.

8. Cross-site regression analysis showed that an extremely strong and significant relationship also existed between the yield losses associated with canker incidence and those associated with canker severity, allowing the likely yield losses to be calculated for a given mean incidence and severity of disease.

9. Optimum control of both *Phoma* leaf spot and canker was obtained by the application of one or more likely two fungicide sprays between November and February. Fungicides must however still be applied before it is known that economic control will be achieved, since foliar disease severity of untreated plants must be ≥1.0 per cent before or during January for yield responses to earlier sprays to be economic.

10. For disease control to be economic and rapeseed at £180/t, based on a two-spray programme, 34 per cent of stems would need to develop canker by pod ripening in association with a foliar severity of ≥ 1.0 per cent leaf area affected before or during January.

Light leaf spot

11. Light leaf spot affected leaves and stems at nine of the fourteen sites, pod symptoms were detected at seven sites.

12. The incidence of foliar disease in untreated plots was high at all affected sites, the maximum severity ranging between 2.2 and 63.9 per cent leaf area affected

13. The incidence of light leaf spot on stems in untreated plots at pod ripening ranged between 53 and 100 per cent stems affected, disease severity ranged between 0.5 and 55.0 per stem area affected.

14. The incidence of pod disease in untreated plots at pod ripening ranged between 15 and 95 per cent plants affected but disease severity was low with only 0.2 to 5.3 per cent pod area affected.

15. Cross-site regression analyses showed that a relatively strong relationship was found between the maximum foliar severity of light leaf spot and the final disease severity on the stems.

16. For every 1 per cent maximum leaf area affected by light leaf spot during the season there was an associated 8 per cent stem area affected at pod ripening.

17. Cross-site regression analyses showed that no relationship existed between either leaf and pod or pod and stem disease incidence or severity.

18. Individual regression analyses showed that for every 1 per cent increase in the number of stems affected by light leaf spot at pod ripening there was an associated loss in yield of between 0.009 and 0.044 t/ha, averaging at 0.019 t/ha. This was twice the yield loss associated with canker.

19. Individual regression analyses showed that the effect of the severity of stem light leaf spot on yield was extremely variable with 0.029 to 0.982 t/ha lost per 1 per cent stem area affected.

20. Individual regression analyses showed that, unlike canker, no relationship existed between the yield losses associated with the incidence of light leaf spot on the stems and those associated with the stem area affected by the disease.

21. Cross-site regression analyses showed that it was not possible to calculate the likely loss in yield associated with a given incidence and severity of stem light leaf spot.

22. Cross-site regression analyses showed that no relationship was found between yield and the incidence and severity of light leaf spot on the pods.

23. Yield losses due to light leaf spot were mainly associated with sites where the mean foliar disease severity in untreated plots exceeded 13.0 per cent leaf area affected during the season.

24. Optimum control of light leaf spot on leaves and stems was achieved with one or two fungicide sprays applied in November and February, as was the case with *Phoma* leaf spot and canker.

25. For disease control to be economic and rapeseed at £180/t, based on a two-spray programme, 17 per cent of stems would need to develop light leaf spot by pod

ripening in association with a foliar severity of $\geq$ 13 per cent leaf area affected during the season.

26. As both light leaf spot and *Phoma* leaf spot and stem canker developed together at six of the experiment sites, the effect of each disease on yield should be treated with caution. Further analysis will be required to separate the individual effects of each disease.

Sclerotinia stem rot

27. Sclerotinia stem rot developed to damaging levels at two sites only.

28. At Rosemaund the disease was severe and it was found that for every 1 per cent of main stems affected by *Sclerotinia* at pod ripening 0.016 t/ha was lost.

29. At Rosemaund for every 1 per cent of plants with racemes affected by *Sclerotinia* at pod ripening 0.032 t/ha was lost.

30. Raceme infection was therefore twice as damaging to yield when compared to main stem infection at Rosemaund.

31. At Kington Langley the disease was less severe than at Rosemaund and for every 1 per cent of main stems or plants with racemes affected by *Sclerotinia* at pod ripening, 0.01 t/ha was lost.

32. Selected treatments which received their final spray in the spring just prior to flowering resulted in an increase in disease incidence at Kington Langley.

33. Late and post-flowering fungicide sprays were the most effective treatments to control both main stem and raceme infection by *Sclerotinia* at both of these sites.

34. Normal grower practice is to apply a fungicide to control *Sclerotinia* no later than mid-flowering. Although later sprays can be effective as shown in this study, this is likely to be the case only where infection by *Sclerotinia* takes place towards the end of flowering.

35. All conclusions were derived by extrapolation from treatments that had received multiple fungicide applications. Further work examining the effect of single or double sprays applied within the critical period identified in this work for disease control would fully validate these derived relationships.

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APPENDIX 1

Table 1. Site history 1991/92

Details	Foveran	Rosemaund	Rothamsted	K Langley
Soil Series:	Blackhouse	Bromyard	Batcombe	Curtisden
Soil texture:	Clay loam - clay till	Silty clay/loam	Silty clay/loam over clay with flints	Sandy loam
Drainage:	Imperfect	Good	Good	Good
Soil analysis:				
pH	-	6.9	7.5	6.4
P index	-	2	2	2
K index	-	2	2	1
Mg index	-	2	3	3
Organic matter (%)	-	-	2.4	5.4
Previous cropping:				
1991	W. barley	WOSR	WOSR	W. barley
1990	W. wheat	W. barley	W. barley	W. barley
1989	WOSR	W. wheat	W. wheat	-
Previous crop residues:	Straw baled and carted	Chopped	Chopped	Straw baled and carted
Pre-sowing cultivations:	Ploughed, one pass Accord drill	Rotavated x 1, ploughed; Maschio x 1, drilled	Flexitined x 2, disced, rotary harrowed	Ploughed and power harrowed

- Not analysed

Table 2. Site history 1992/3

Details	Boxworth	Pettymuick	Rothamsted	Tarrant Hinton	Withington
Soil Series:	Hanslope	Pitmedden	Batcombe	Andover 1	Bromyard
Soil texture:	Silty clay loam	Organic sandy clay loam	Silty clay loam	Silty loam	Silty clay loam
Drainage:	Moderately good	Poor	Good	Good	Good
Soil analysis:					
pH	8.1	5.8	7.0	8.0	6.1
P index	0	3	3	3	4
K index	2	1	3	2	2
Mg index	3	5	2	1	2
Organic matter (%)	-	12.0	2.4	-	2.5
Previous cropping:					
1992	W. wheat	W. barley	W. wheat	W. barley	WOSR
1991	W. wheat	W. barley	W. wheat	W. wheat	W. wheat
1990	W. wheat	WOSR	-	W. wheat	-
Previous crop residues:	Baled Stubble Burnt	Baled and removed	Chopped, baled and removed. Stubble ploughed in	Chopped	Chopped
Pre-sowing cultivations:	Cultivated twice, drilled and rolled	Ploughed	Dynadrived, ploughed, and furrow pressed	Ploughed, cultivated, drilled	Ploughed and crumbled

APPENDIX 1

Table 3. Site history 1993/94

Details	Boxworth	Rosemaund	Rothamsted	Thurloxton	Udny Station
Soil Series:	Hanslope	Bromyard	Batcombe	Milford	Pitmedden
Soil texture:	Clay loam	Silty clay loam	Silty clay loam with flints	Silty clay loam	Sandy clay loam
Drainage:	Moderately good	Good	Good	Good	Poor
Soil analysis:					
pH	8.1	6.8	7.9	7.0	5.8
P index	2	3	3	2	0
K index	3	2	3	2	2
Mg index	2	3	2	2	2
Organic matter (%)	3	3.3	1.7	5.6	-
Previous cropping:					
1993	W. wheat	WOSR	Set-aside	W. wheat	WOSR
1992	S. beans	WOSR	W. wheat	Vining peas	S. barley
1991	W. wheat	WOSR	-	W. barley	SOSR S. barley
Previous crop residues:	Baled and carted	Chopped and ploughed	-	Baled and removed	Chopped, baled and removed
Pre-sowing cultivations:	Disced, power harrowed, drilled and rolled	Ploughed, power harrowed, crumbled, rolled	Ploughed, furrow pressed, rotary harrowed and rolled	Ploughed, rolled, combination drilled	Ploughed, power harrowed, drilled and rolled

APPENDIX 2

Table 1. Crop husbandry details 1991/92

Details	Foveran	Rosemaund	Rothamsted	K Langley
Cultivar:	Envol	Envol	Envol	Envol
Plot size:	60 m2	96 m2	75 m2	72 m2
Sowing date:	23/8/91	6/9/91	28/8/91	1/9/91
Seed rate (kg/ha):	6.60	5.28	5.66	7.28
Seed treatment:	Lindex-Plus FS	Lindex-Plus FS	Lindex-Plus FS	Lindex-Plus FS
Nitrogen (kg/ha):	26 (20/9) 56 (19/2) 56 (25/3)	108 (6/3) 127 (27/3)	76 (19/2) 76 (20/3)	40 (31/1) 43 (28/2) 85 (15/3)
Herbicides:	1.41 Butisan (pre-emergence)	2.3 kg Benazolox (29/11)	2.01 Gramoxone (21/8) 1.51 Butisan (30/8)	1.51 Butisan (pre-emergence)
Fungicides*:	Nil	3.01 Compass (6/5)	Nil	Nil
Insecticides:	Nil	250 ml Decis (30/9) 200 ml Decis (11/4) Nil	Nil	Nil
Molluscicides:	Nil	Nil	Nil	Nil
Harvest:	Desiccated (7/8)	Direct	Desiccated (9/6)	Direct
Harvest date:	14 August	30 July	15 July	28 July

Footnote: All rates shown are per hectare
Dates are shown in brackets

*** To surrounding crop**

APPENDIX 2

Table 2 Crop husbandry details 1992/93

Details	Boxworth	Pettymuick	Rothamsted	Tarrant Hinton	Withington
Cultivar:	Envol	Envol	Envol	Envol	Envol
Plot size:	-	80 m2	-	81 m2	96 m2
Sowing date:	3/9/92	20/8/92	29/8/92	4/9/92	14/9/92**
Seed rate (kg/ha):	6.8	3.5	5.6	6.6	4.0
Seed treatment:	Lindex-Plus FS	Lindex-Plus FS	Lindex-Plus FS	Lindex-Plus FS	Vitavax
Nitrogen (kg/ha):	41 (1/10) 80 (15/2) 96 (12/3)	56 (Sept) 86 (Feb) 60 (Mar)	60 (17/2) 127 (23/3)	32 (28/8) 75 (24/2) 87 (17/3)	30 (7/10) 69 (9/2) 72 (19/3)
Herbicides:	0.75l Fusilade (2/10) 1.0 kg Kerb (10/12)	0.75l Butisan S (22/8) 2.31 Treflan (22/8)	0.075l Pilot (19/10) 1.63 kg Matrikerb (19/1)	0.22kg Matrikerb (23/10) 0.83kg Kerb (25/10) 0.57l Fortrol (25/10) 1.01 Fortrol (10/12)	2.51 Butisan S (3/11) 1.01 Laser (3/11) 2.3kg Benazolox (17/2)
Fungicides:*	3.01 Compass (30/4) 2.01 Rovral Flo (4/6)	0.31 Folicur (Feb) 0.41 Folicur (1/4) 2.01 Compass (27/5)	None	None	1.01 Ronilan FL (8/5) 0.251 Decis (19/9)
Insecticides:	None	0.11 Fastac (27/5)	None	0.221 Decis (23/10)	0.251 Decis (19/9)
Molluscicides:	None	None	5.5kg Draza	None	None
Harvest:	Desiccated (6/7)	Desiccated (10/8)	Desiccated (17/7)	Direct	Direct
Harvest date:	20 July	28 August	28 July	31 July	31 July

Footnote: **All rates shown are per hectare, dates are shown in brackets**

* **To surrounding crop**

** **Crop poorly established after drilling on 30 August, therefore redrilled**

APPENDIX 2

Table 3. Crop husbandry details 1993/94

Details	Boxworth	Rosemaund	Rothamsted	Thurloxton	Udny Station
Cultivar:	Envol	Envol	Envol	Envol	Envol
Plot size:	108 m2	72 m2	75 m2	56 m2	80 to 100 m2
Sowing date:	7/9/93	4/9/93	18/9/93	12/9/93	31/8/93
Seed rate (kg/ha):	6.8	7.0	5.6	7.0	6.0
Seed treatment:	Lindex-Plus FS	Lindex-Plus FS	Lindex-Plus FS	Lindex-Plus FS	Vitavax
Nitrogen (kg/ha):	31 (4/9) 99 (9/2) 60 (21/3)	35 (3/3) 137 (23/3)	56 (7/3)	88 (2/3) 94 (29/3)	19 Seedbed 90 (9/3) 90 (8/4)
Herbicides:	1.0l Fusilade (1/11) 1.4 kg Kerb (24/1)	2.3kg Benazolox (9/3)	3.0kg Carbetamex (28/2) 1.0kg Benazolox (28/2)	None	1.5l Butisan S Pre-emergence
Fungicides*:	3.0l Compass (19/5)	1.5l Ronilan (5/5)	None	None	None
Insecticides:	None	0.2l Fastac (29/4)	None	None	None
Molluscicides:	5.5kg Draza (5/11)	2.3kg Draza (21/9)	5.5kg Draza (30/8 + 18/1)	None	None
Harvest:	Desiccated (11/7)	Desiccated (8/7)	Direct	Direct	Swathed (15/8)
Harvest date:	18 July	3 August	1 August	29 July	22 August

Footnote: All rates are shown per hectare, dates are shown in brackets
*** To surrounding crop**

Table 1. Fungicide application dates: Foveran 1991/92

Treatment	Date and growth stage									
	9.10 1.01 - 1.08	7.11 1.07 - 1.08	2.12 1.06 - 1.11	30.12 1.07 - 1.11	31.1 1.09 - 1.11	26.2 1.13- 1.17	28.3 3.3 - 3.5	28.4 4.0	28.5 4.5	2.7 6.3
1*	-	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-	-
7	X	X	X	X	X	X	-	-	-	-
8	X	X	X	X	X	X	X	-	-	-
9	X	X	X	X	X	X	X	X	-	-
10	X	X	X	X	X	X	X	X	X	-
11**	X	X	X	X	X	X	X	X	X	X
14	-	-	-	-	-	-	-	-	-	X
15		-	-	-	-	-	-	-	X	X
16	-	-	-	-	-	-	-	X	X	X
17	-	-	-	-	-	-	X	X	X	X
18	-	-	-	-	-	X	X	X	X	X
19	-	-	-	-	X	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X	X

* 4 replicates - treatment 13 unsprayed

** 6 replicates - treatment 12 and additional treatment 23 fully sprayed

Table 2. Fungicide application dates: Kington Langley 1991/92

Treatment	Date and growth stage										
	2.10 1.04	29.10 1.06	26.11 1.09/ 1.10	30.12 1.10	2.2 1.12/ 1.14	24.2 2.01	17.3 2.09/ 3.2	16.4 3.6	8.5 4.8/5.7	5.6 6.2	25.6 6.4
1*	-	-	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-	-	-
7	X	X	X	X	X	X	-	-	-	-	-
8	X	X	X	X	X	X	X	-	-	-	-
9	X	X	X	X	X	X	X	X	-	-	-
10	X	X	X	X	X	X	X	X	X	-	-
11	X	X	X	X	X	X	X	X	X	X	-
12	X	X	X	X	X	X	X	X	X	X	X
13	-	-	-	-	-	-	-	-	-	-	X
14	-	-	-	-	-	-	-	-	-	X	X
15	-	-	-	-	-	-	-	-	X	X	X
16	-	-	-	-	-	-	-	X	X	X	X
17	-	-	-	-	-	-	X	X	X	X	X
18	-	-	-	-	-	X	X	X	X	X	X
19	-	-	-	-	X	X	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X	X	X

* 4 replicates - additional treatment 23 unsprayed

Table 3. Fungicide application dates: Rosemaund 1991/92

Treatment	Date and growth stage									
	8.10 1.02	5.11 1.03	2.12 1.05	30.12 1.06	29.1 1.08	24.2 1.10	25.3 2.04	29.4 4.7	18.5 4.8	17.6 6.3
1*	-	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-	-
7	X	X	X	X	X	X	-	-	-	-
8	X	X	X	X	X	X	X	-	-	-
9	X	X	X	X	X	X	X	X	-	-
10	X	X	X	X	X	X	X	X	X	-
11**	X	X	X	X	X	X	X	X	X	X
14	-	-	-	-	-	-	-	-	-	X
15	-	-	-	-	-	-	-	-	X	X
16	-	-	-	-	-	-	-	X	X	X
17	-	-	-	-	-	-	X	X	X	X
18	-	-	-	-	-	X	X	X	X	X
19	-	-	-	-	X	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X	X
22	X	X	X	X	X	X	X	X	X	X

* 4 replicates - treatment 13 unsprayed

** 4 replicates - treatment 12 received same number of sprays

Table 4. Fungicide application dates: Rothamsted 1991/92

Treatment	Date and growth stage								
	12.10 1.06	6.11 1.07	2.12 1.08	14.1 1.10	11.2 1.10 - 1.11	16.3 2.05	21.4 3.4	14.5 4.9 - 5.4	9.6 6.3
1*	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-
7	X	X	X	X	X	X	-	-	-
8	X	X	X	X	X	X	X	-	-
9	X	X	X	X	X	X	X	X	-
10**	X	X	X	X	X	X	X	X	X
15	-	-	-	-	-	-	-	-	X
16	-	-	-	-	-	-	-	X	X
17	-	-	-	-	-	-	X	X	X
18	-	-	-	-	-	X	X	X	X
19	-	-	-	-	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X

* 4 replicates - treatment 14 unsprayed

** 4 replicates - treatment 11 received same number of sprays

Table 5. Fungicide application dates: Boxworth 1992/93

Treatment	Date and growth stage										
	19.10 1.05	6.11 1.06	10.12 1.09	18.1 1.12	1.2 1.12	11.2 1.12/ 2.01	11.3 2.05/ 3.3	7.4 3.7	4.5 4.1	3.6 6.2	24.6 6.4
1*	-	-	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-	-	-
7	X	X	X	X	X	X	-	-	-	-	-
8	X	X	X	X	X	X	X	-	-	-	-
9	X	X	X	X	X	X	X	X	-	-	-
10	X	X	X	X	X	X	X	X	X	-	-
11	X	X	X	X	X	X	X	X	X	X	-
12	X	X	X	X	X	X	X	X	X	X	X
13	-	-	-	-	-	-	-	-	-	-	X
14	-	-	-	-	-	-	-	-	-	X	X
15	-	-	-	-	-	-	-	-	X	X	X
16	-	-	-	-	-	-	-	X	X	X	X
17	-	-	-	-	-	-	X	X	X	X	X
18	-	-	-	-	-	X	X	X	X	X	X
19	-	-	-	-	X	X	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X	X	X

* 4 replicates - additional treatment 23 unsprayed

Table 6. Fungicide application dates: Pettymuick 1992/93

Treatment	Date and growth stage								
	9.10 1.04	17.11 1.07/ 1.09	9.12 1.08/ 1.09	18.1 1.08/ 1.09	11.2 1.11/ 1.12	25.3 3.3	29.4 3.5/ 4.0	8.6 4.8	14.7 6.1
1*	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-
7	X	X	X	X	X	X	-	-	-
8	X	X	X	X	X	X	X	-	-
9	X	X	X	X	X	X	X	X	-
10**	X	X	X	X	X	X	X	X	X
15	-	-	-	-	-	-	-	-	X
16	-	-	-	-	-	-	-	X	X
17	-	-	-	-	-	-	X	X	X
18	-	-	-	-	-	X	X	X	X
19	-	-	-	-	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X

* 6 replicates - treatments 13 and 14 unsprayed

** 6 replicates - treatment 11 and 12 received same number of sprays

Table 7. Fungicide application dates: Rothamsted 1992/93

Treatment	Date										
	14.10	2.11	7.12	8.1	29.1	23.2	24.3	19.4	18.5	22.6	13.7
1	-	-	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-	-	-
7	X	X	X	X	X	X	-	-	-	-	-
8	X	X	X	X	X	X	X	-	-	-	-
9	X	X	X	X	X	X	X	X	-	-	-
10	X	X	X	X	X	X	X	X	X	-	-
11	X	X	X	X	X	X	X	X	X	X	-
12	X	X	X	X	X	X	X	X	X	X	X
13	-	-	-	-	-	-	-	-	-	-	X
14	-	-	-	-	-	-	-	-	-	X	X
15	-	-	-	-	-	-	-	-	X	X	X
16	-	-	-	-	-	-	-	X	X	X	X
17	-	-	-	-	-	-	X	X	X	X	X
18	-	-	-	-	-	X	X	X	X	X	X
19	-	-	-	-	X	X	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X	X	X

Table 8. Fungicide application dates: Tarrant Hinton 1992/93

Treatment	Date and growth stage										
	8.10 1.04	5.11 1.07	9.12 1.12	4.1 1.13	29.1 1.14	23.2 2.06/ 3.1	19.3 2.09/ 3.6	13.4 4.0/ 4.1	10.5 4.9	8.6 6.3	29.6 6.4
1	-	-	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-	-	-
7	X	X	X	X	X	X	-	-	-	-	-
8	X	X	X	X	X	X	X	-	-	-	-
9	X	X	X	X	X	X	X	X	-	-	-
10	X	X	X	X	X	X	X	X	X	-	-
11	X	X	X	X	X	X	X	X	X	X	-
12	X	X	X	X	X	X	X	X	X	X	X
13	-	-	-	-	-	-	-	-	-	-	X
14	-	-	-	-	-	-	-	-	-	X	X
15	-	-	-	-	-	-	-	-	X	X	X
16	-	-	-	-	-	-	-	X	X	X	X
17	-	-	-	-	-	-	X	X	X	X	X
18	-	-	-	-	-	X	X	X	X	X	X
19	-	-	-	-	X	X	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X	X	X

Table 9. Fungicide application dates: Withington 1992/93

Treatment	Date and growth stage								
	4.11 1.02/ 1.03	5.12 1.03/ 1.04	4.1 1.04/ 1.05	25.1 1.05	22.2 1.05	24.3 3.0/ 4.2	19.4 3.3/ 4.2	18.5 4.9	20.6 6.3
1*	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-
7	X	X	X	X	X	X	-	-	-
8	X	X	X	X	X	X	X	-	-
9	X	X	X	X	X	X	X	X	-
10**	X	X	X	X	X	X	X	X	X
15	-	-	-	-	-	-	-	-	X
16	-	-	-	-	-	-	-	X	X
17	-	-	-	-	-	-	X	X	X
18	-	-	-	-	-	X	X	X	X
19	-	-	-	-	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X

* 4 replicates - treatment 14 unsprayed

** 4 replicates - treatment 12 received same number of sprays

Table 10. Fungicide application dates: Boxworth 1993/94

Treatment	Date and growth stage										
	15.10 1.02	2.11 1.04	26.11 1.05	17.12 1.06	24.01 1.08	17.02 2.01	21.03 2.07 - 3.1/ 3.3	18.04 3.7	12.05 4.8 - 5.5	6.06 6.2	27.06 6.3
1*	-	-	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-	-	-
7	X	X	X	X	X	X	-	-	-	-	-
8	X	X	X	X	X	X	X	-	-	-	-
9	X	X	X	X	X	X	X	X	-	-	-
10	X	X	X	X	X	X	X	X	X	-	-
11	X	X	X	X	X	X	X	X	X	X	-
12	X	X	X	X	X	X	X	X	X	X	X
13	-	-	-	-	-	-	-	-	-	-	X
14	-	-	-	-	-	-	-	-	-	X	X
15	-	-	-	-	-	-	-	-	X	X	X
16	-	-	-	-	-	-	-	X	X	X	X
17	-	-	-	-	-	-	X	X	X	X	X
18	-	-	-	-	-	X	X	X	X	X	X
19	-	-	-	-	X	X	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X	X	X

* 4 replicates

Table 11. Fungicide application dates: Rosemaund 1993/94

Treatment	Date									
	4.10	25.10	22.11	21.12	17.1	17.2	19.3	11.4	9.5	10.6
1	-	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-	-
7	X	X	X	X	X	X	-	-	-	-
8	X	X	X	X	X	X	X	-	-	-
9	X	X	X	X	X	X	X	X	-	-
10	X	X	X	X	X	X	X	X	X	-
12	X	X	X	X	X	X	X	X	X	X
14	-	-	-	-	-	-	-	-	-	X
15	-	-	-	-	-	-	-	-	X	X
16	-	-	-	-	-	-	-	X	X	X
17	-	-	-	-	-	-	X	X	X	X
18	-	-	-	-	-	X	X	X	X	X
19	-	-	-	-	X	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X	X

Table 12. Fungicide application dates: Rothamsted 1993/94

Treatment	Date								
	23.11	17.12	19.1	17.2	30.3	19.4	9.5	13.6	4.7
1	-	-	-	-	-	-	-	-	-
4	X	-	-	-	-	-	-	-	-
5	X	X	-	-	-	-	-	-	-
6	X	X	X	-	-	-	-	-	-
7	X	X	X	X	-	-	-	-	-
8	X	X	X	X	X	-	-	-	-
9	X	X	X	X	X	X	-	-	-
10	X	X	X	X	X	X	X	-	-
11	X	X	X	X	X	X	X	X	-
12	X	X	X	X	X	X	X	X	X
13	-	-	-	-	-	-	-	-	X
14	-	-	-	-	-	-	-	X	X
15	-	-	-	-	-	-	X	X	X
16	-	-	-	-	-	X	X	X	X
17	-	-	-	-	X	X	X	X	X
18	-	-	-	X	X	X	X	X	X
19	-	-	X	X	X	X	X	X	X
20	-	X	X	X	X	X	X	X	X

Table 13. Fungicide application dates: Thurloxton 1993/94

Treatment	Date and growth stage								
	28.10 1.03	26.11 1.05	7.1 1.06-1.07	31.1 1.08	21.2 1.11-2.00	15.3 2.05-3.1	11.4 2.08-3.7	9.5 4.7-5.0	6.6 6.2-6.3
1	-	-	-	-	-	-	-	-	-
3	X	-	-	-	-	-	-	-	-
4	X	X	-	-	-	-	-	-	-
5	X	X	X	-	-	-	-	-	-
6	X	X	X	X	-	-	-	-	-
7	X	X	X	X	X	-	-	-	-
8	X	X	X	X	X	X	-	-	-
9	X	X	X	X	X	X	X	-	-
10	X	X	X	X	X	X	X	X	-
12*	X	X	X	X	X	X	X	X	X
14	-	-	-	-	-	-	-	-	X
15	-	-	-	-	-	-	-	X	X
16	-	-	-	-	-	-	X	X	X
17	-	-	-	-	-	X	X	X	X
18	-	-	-	-	X	X	X	X	X
19	-	-	-	X	X	X	X	X	X
22	-	-	X	X	X	X	X	X	X
21	-	X	X	X	X	X	X	X	X

* 4 replicates - treatment 12 and 22 fully sprayed

Table 14. Fungicide application dates: Udny Station 1993/94

Treatment	Date and growth stage								
	29.10 1.03-1.06	30.11 1.04-1.06	21.12 1.05-1.07	24.1 1.05-1.08	3.3 1.08-1.10	5.4 3.1-3.3	11.5 3.5-4.0	13.6 5.7	14.7 6.3
1*	-	-	-	-	-	-	-	-	-
2	X	-	-	-	-	-	-	-	-
3	X	X	-	-	-	-	-	-	-
4	X	X	X	-	-	-	-	-	-
5	X	X	X	X	-	-	-	-	-
6	X	X	X	X	X	-	-	-	-
7	X	X	X	X	X	X	-	-	-
8	X	X	X	X	X	X	X	-	-
9	X	X	X	X	X	X	X	X	-
10**	X	X	X	X	X	X	X	X	X
15	-	-	-	-	-	-	-	-	X
16	-	-	-	-	-	-	-	X	X
17	-	-	-	-	-	-	X	X	X
18	-	-	-	-	-	X	X	X	X
19	-	-	-	-	X	X	X	X	X
20	-	-	-	X	X	X	X	X	X
21	-	-	X	X	X	X	X	X	X
22	-	X	X	X	X	X	X	X	X

* 6 replicates - treatment 13 and 14 unsprayed

** 6 replicates - treatment 11 and 12 received same number of sprays