HGCA

PROJECT REPORT No. 215

APPROPRIATE FUNGICIDE DOSES FOR WINTER BARLEY VOLUME II: PROGRESSING THE CONCEPT

January 2000

Price £6.00

PROJECT REPORT No. 215

APPROPRIATE FUNGICIDE DOSES FOR WINTER BARLEY

VOLUME II: PROGRESSING THE CONCEPT

By

S J WALE

SAC Aberdeen, Ferguson Building, Craibstone Estate, Bucksburn, Aberdeen AB21 9YA

This is part of the final report of two HGCA-funded research projects. Project no. 1398 (grant of £377,845) started in September 1993 and lasted for 40 months. Project no. 1397 (grant of £177,503) started in October 1996 and lasted for 30 months.

NB. Volume II comprises three parts. Parts 1 and 2 were funded as project no. 1397. Part 3 reports Experiments 1 and 2. These were funded as project no. 1398 for three years, but were then extended by a further two years as project no. 1397.

Contents

Winter barley appropriate fungicide dose project

HGCA WINTER BARLEY APPROPRIATE FUNGICIDE DOSE PROJECT
VOLUME II: PROGRESSING THE CONCEPT

PROJECT Reference 1397 and 1398 (part)

COLLABORATORS

Appropriate Fungicide Doses for Winter Barley

Project leader:

Dr S.J. Wale,
SAC, Crops Division, Ferguson Building, Craibstone Estate,
Bucksburn, Aberdeen AB21 9YA
Tel: 01224 711213
Fax: 01224 711293
email: s.wale@ab.sac.ac.uk

Statistics:

Dr M.J. Hims,
Central Science Laboratory, Sand Hutton, York YO41 1LZ
Mr C.G. Harbron, Dr M.F. Franklin & Dr Carol Reid
BioMathmatics and Statistics Scotland,
Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB

Site managers

Aberdeen
Experiments 1&2
& Physiology Expt

Dr Fiona Murray,
SAC, Crops Division, Ferguson Building, Craibstone Estate,
Bucksburn, Aberdeen AB21 9YA

Morley
Experiments 1&2

Mr R.I. Hindley & Mr D.B. Stevens,
Morley Research Centre, Morley, Wymondham, Norfolk
NR18 9DB

Rosemaund
Experiments 1&2
& Physiology Expt

Dr D.R. Jones,
ADAS Rosemaund, Preston Wynne, Hereford, HR1 3PG

Starcross
Experiments 1&2

Mr K.D. Lockley,
ADAS, Mamhead Castle, Mamhead, Exeter EX6 8HD

Shute
Mitchell
Newquay
Experiment 1

Mr O. Jones,
Westward Arable Centres, 10 Cousens Close, Langstane
Park, Dawlish, Devon, EX7 9TE

Elmdon
Experiment 2

Mr A. du Rieu
Novartis (formerly Ciba Agriculture), Whittlesford, Cambridge, CB2 4QT

Physiology review authors

Dr I Bingham, Dr J Thomson & Dr S Wale
SAC, Crops Division, Ferguson Building, Craibstone Estate,
Bucksburn, Aberdeen AB21 9YA

PART 1
EVALUATION OF BASELINE PHYSIOLOGICAL DATA FOR WINTER BARLEY IN CONTRASTING DISEASE SITUATIONS

S J WALE & F MURRAY & I J BINGHAM

SAC, CROPS DIVISION, FERGUSON BUILDING, CRAIBSTONE ESTATE, BUCKSBURN, ABERDEEN AB21 9YA

SUMMARY

A small pilot physiological study was carried out on replicated winter barley trials at two sites growing different varieties where three fungicide programmes were used to develop different disease epidemics. From GS 30 to GS 75 physiological measurements were made to assess the impact of disease on crop development. Two key findings emerged from the study. Firstly, there was a close relationship between dry matter accumulation and accumulated light interception. This indicates that the impact of disease is largely mediated through the loss of green tissue. This factor needs to be taken into account when determining the appropriate fungicide dose. Secondly, despite large differences in disease levels at the two sites, the impact on green leaf area index was similar. This suggested that the variety at one site was more sensitive to disease than the variety at the other site.

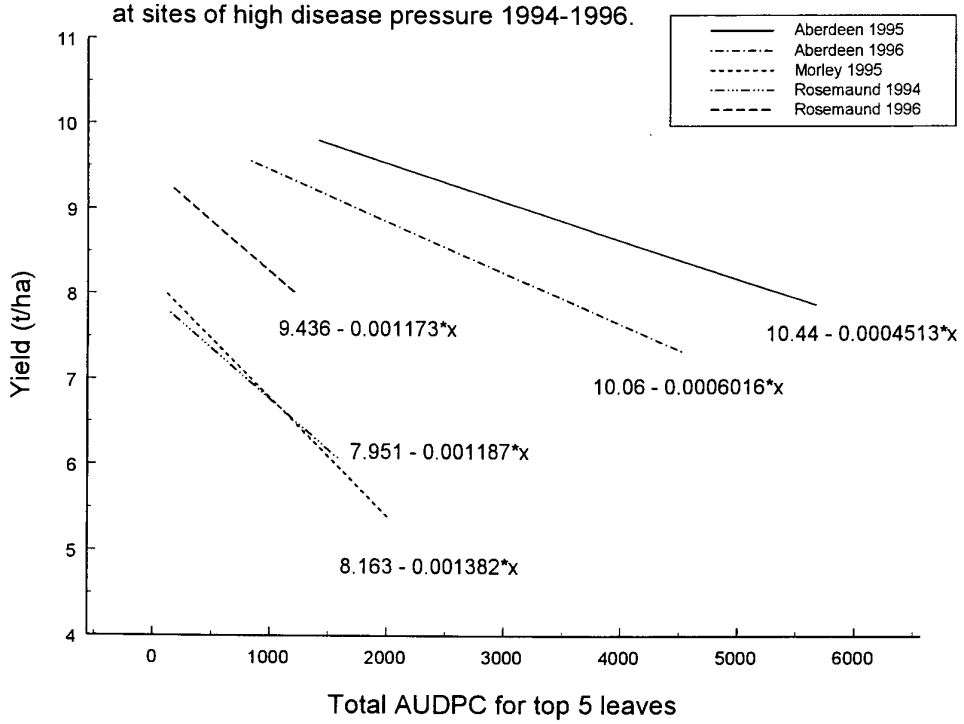
INTRODUCTION

In attempting to identify the appropriate dose for any situation, the assumption in winter barley to date has been that the control of disease on the critical last formed leaves is related to improvement of yield. That there is a direct relationship has been shown in Experiment 3 of the HGCA Appropriate Fungicide Dose project (0052/01/92). Figure 1 shows the relationship for five (out of nine) trials in which moderate to severe disease epidemics occurred in the untreated control.

If the regression lines were all parallel, the relationship between disease and yield would have been consistent from trial to trial. However, the relationship differed markedly across sites. When the trials comprising this series were examined it was evident that those with the steepest gradients were sown with the lowest seed rates (350 - 400 seeds/m^2) and visually appeared to be thinner than those trials with shallower gradients (500 seeds/m^2). This suggested that denser crops with more foliage were more tolerant of disease. Indeed the disease severity at the two Aberdeen sites was the greatest of all trials and yet the yield loss per unit of disease were the lowest.

In the appropriate fungicide dose trial series, disease was measured as % leaf area infected and like many pathology cereal trials scant attention was given to measuring the crop except for yield. Work by Gaunt (1995) and Bryson *et al* (1995) on winter wheat have shown that relationships between disease and yield are more consistent where the green tissue remaining is measured because it is light absorption and dry matter accumulation that are crucial to yield. For winter wheat, a series of benchmarks have been described in a Wheat Growth Guide (Anon., 1997) for the development of the crop. These benchmarks can be used to modify inputs to achieve the optimum crop.

Fig. 1. Linear regression of yield against AUDPC on top five leaves at sites of high disease pressure 1994-1996.



In particular, an optimum green leaf area index was benchmarked. Where a crop produces a GLAI greater than this there is surplus leaf. The impact of disease on this crop might be expected to be less than on a crop with an optimum GLAI. Conversely in a crop with a GLAI below optimum, any loss of green tissue will have an impact on yield. The Wheat Growth Guide clearly highlighted the importance of understanding crop physiology when growing cereal crops. Whilst wheat has become an intensively studied crop, barley, particularly winter barley, has been studied to a much lesser degree (see HGCA Research Review on the Physiology of Responses of Winter Barley to Disease). The study reported below was thus an attempt to provide background data on the winter barley crop and its physiological response to disease as a preliminary to further work.

The study arose out of the winter barley appropriate fungicide dose project and thus, like MAFF funded wheat studies on Integrated Disease Risk (IDR), the physiology of winter barley was viewed from a pathological angle. The specific objective for this study was to determine baseline physiology data for winter barley to identify those parts of disease and growth models developed for winter wheat which might have direct applicability for winter barley.

MATERIALS AND METHODS

At two sites, Tillycorthie Farm, Udny Station, Aberdeenshire (NJ 904228) and ADAS Rosemaund (SO 564486), within the trial evaluating dose responses of winter barley fungicides, three replicates of six plots were established. All six plots were adjacent to each other within the replicate and comprised pairs of plots. One of each pair was used for destructive sampling and the other for yield determination. Three treatments were assigned to the pairs of plots at random. The three treatments were an untreated control, a single fungicide application at GS 31/2 and a programme of fungicide applications at GS 30, GS 31/2 and GS 39/49. The fungicide used was propiconazole (Tilt 250EC, 250 g/litre a.i., 0.5 litres product/ha equivalent to the full

recommended dose) plus fenpropimorph (Aura 750EC, 750 g/litre a.i., 0.75 litres product /ha equivalent to 3/4 of the full recommended dose).

At the Tillycorthie site the variety was Pastoral and at Rosemaund it was Puffin. Plot sizes were 36-40m^2.

Site and husbandry details are given in Appendix 1.

Growth analysis

The plot of each pair for destructive sampling was used for both growth analysis and disease assessment. For growth analysis, sampling was carried out at growth stages 30, 31, 32, 39, 59 and 75. At GS 30 one bulked sample was used from across the trial area.
Plot areas sampled on each occasion were 0.5 - 1.0 m^2. To avoid "local" and systematic bias in selection of samples, they were taken from predetermined areas. At least 20 cm between sample areas and samples taken at least 50 cm from ends and edges of the plots and from tramlines.

At sampling all the above ground material within the sample area, including any dead and dying material, was collected. At GS 30 to GS 32 plants were pulled or dug up and roots cut off. After GS 32 plants were cut off at the soil surface. After sampling care was taken to prevent desiccation of leaves by enclosure in a plastic bag. If growth analysis was delayed, the samples were stored in a cold room at 4-6°C, but analysis was carried out within two days of sampling.

Samples were used to determine total crop dry matter, green canopy projected areas (by layers) and its components, fertile shoot number, ear emergence and development and soluble stem carbohydrates.

a) Analysis in the laboratory

Sub-sampling

If plants were contaminated with soil, they were gently washed under a running tap. Small plants were dried using a domestic lettuce spinner otherwise paper towels or shaking was used to remove all excess water.

The fresh weight of the total sample was recorded. The plant material was spread out into roughly four equal piles. Randomly selected plant material from each pile was taken to give an approximate 10% sub-sample. At and after GS 39, 40 shoots were selected at random to make up the sub-sample. This sub-sample (SS1) was used for the growth analysis. Its fresh weight was recorded and where analysis was delayed it was placed in a cold room at 4-6°C in a plastic bag to prevent dehydration.

From the same four piles plant material was selected from each pile to give a 15-20% sub-sample (SS2). This was used solely to determine total, leaf, stem and ear dry matter content at the time of sampling. This was achieved by weighing fresh, cutting up the plant material into approximately 10 cm lengths either into a tray or labelled paper bag, placing in an oven at 80°C for 48 hours and re-weighing. After ear emergence, the sample was split up into stems and ears, and the fresh and dry weights determined for both.

Growth analysis of SS1 sub-sample

The number of potentially fertile shoots (a shoot was considered emerged when its prophyl or first leaf had emerged from the subtending leaf sheath on the mother shoot) were counted.

Leaf analysis. For each potentially fertile shoot, starting from the top of the shoot each layer was removed in turn and placed in separate trays. In order to identify leaf layers, at each sampling the youngest fully emerged leaves of 10 indicator plants were tagged and after flag leaf emergence, the correct leaf layers in each sample identified retrospectively. The number of leaves still green and dead leaves in each leaf layer were counted. Dead leaves were placed to one side with any other dead material (excluding non-green stem).

For each leaf, green leaf area (%) and disease (%) was estimated. The total projected area of each green leaf was measured using a Delta-T leaf area meter.

For each leaf layer all dead/non-green or diseased leaf material was removed and placed with the dead leaves etc. If dead/non-green or diseased areas of the leaf were patchy, the percentage of leaf area affected was assessed and then that amount removed from the leaf treating that portion as dead leaf material for the purposes of dry weight determination.

Stem analysis. All non-green stem was cut off and collected together. The remaining green stem was cut into approximately 15 cm lengths and their projected green area measured using the Leaf Area Meter. Green stem lengths were collected together.

Ear analysis. If the ear was partially emerged from the flat leaf sheath, the exposed portion was cut off perpendicular to the rachis. If only one side of the ear was visible it was included as part of the stem and leaf sheath portion. The projected areas of all green ears was measured on the Leaf Area Meter. Once ears started senescing only the area of ears with > or = 50% green area were measured, but % green area was assessed.

Dry weight. All the components of SS1 (dead leaf tissue, non-green stem tissue, green leaf tissue by leaf layer, green stem and green ears) were weighed fresh, dried as described above and the dry weight determined

Soluble stem carbohydrates

Additional samples for soluble stem carbohydrate analysis were taken at growth stages 59/61 and 87. These samples were taken between 1100 and 1300 hours after all other field measurements had been made and. From each plot, six potentially fertile shoots were randomly selected and cut off at ground level. Samples were placed in a plastic bag and put into a cool box. Samples were returned to the laboratory as quickly as possible. Immediately on arrival, the fresh weight of the six shoots was recorded. Laminae were removed at the ligule and ears at the collar and the fresh weight of the remaining true stem and sheaths recorded. Stems were immediately placed in a single thin layer on gauze trays in a forced draft oven for flash drying. The stems were dried at 102°C for exactly 2 hours. The time between sampling and oven drying was kept to an absolute minimum to minimise sugar losses due to microbial and plant respiration. Samples were sent to ADAS Wolverhampton for analysis.

b) Harvest analyses

Yield component analysis. Samples of 1.0 m^2 were taken just prior to harvest by cutting off stems at ground level and a full yield component analysis conducted to determine ear no. / m^2 , grain no. / ear, mean grain size, total crop above ground biomass, grain yield, chaff yield, straw yield and biomass harvest index

The fresh weight of the total sample was recorded and all the ears cut off. A random 10-15% sub-sample of straw was removed and the fresh weight, stem number and dry weight (after drying

to constant weight at 80°C) recorded. The number of ears and the fresh weight in the sub-sample was recorded. Ears were threshed and all grain and as much chaff as possible collected. A sample of grain (about 150 g fresh weight) was taken, the fresh weight, dry weight (after drying to constant weight at 80°C) and grain number recorded.

Plot yield and grain quality. The plot of each pair not used for destructive sampling was harvested with a small plot combine and the yield at 15% dry matter determined. A 2 kg sample of the grain from each plot was taken, air dried where necessary and kept for grain quality measurements of specific weight and thousand grain weight.

All other assessments on the physiology plots were the same as for Experiment 1 (Part 3 of the Final Report).

RESULTS

ADAS Rosemaund

At this site very little disease developed. Only after flag emergence was any disease recorded and then at very low levels (a total of 8% on the top 3 leaves) on the untreated control. In the 3 spray programme, disease levels remained below a total of 1% on the top 3 leaves, whilst the 1 spray programme was intermediate (c. total of 4% on the top 3 leaves). Mildew was the main disease.

Despite the low levels of disease, significant differences in green leaf area index were recorded from flag leaf emergence onward (Fig. 2). At flag leaf emergence (Julian Date - JD - 122) and ear emergence (JD 139) the untreated control had significantly less GLAI than the fungicide treated plots, but by GS 75 (JD 161) the loss of leaf in the one spray programme approached that of the untreated control and was significantly less than the three spray programme (Fig. 2).

The pattern was similar when the green area index (GAI - lamina area plus area of other green parts) was considered (Fig. 3)

Fig. 2. Changes in green leaf area index with time. Rosemaund 1997

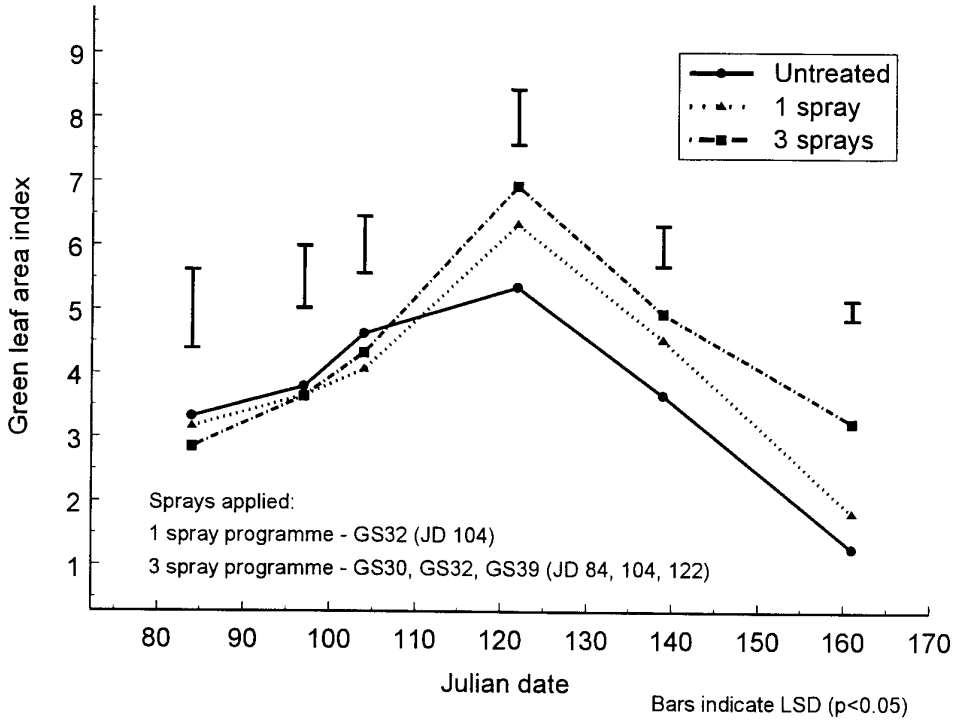
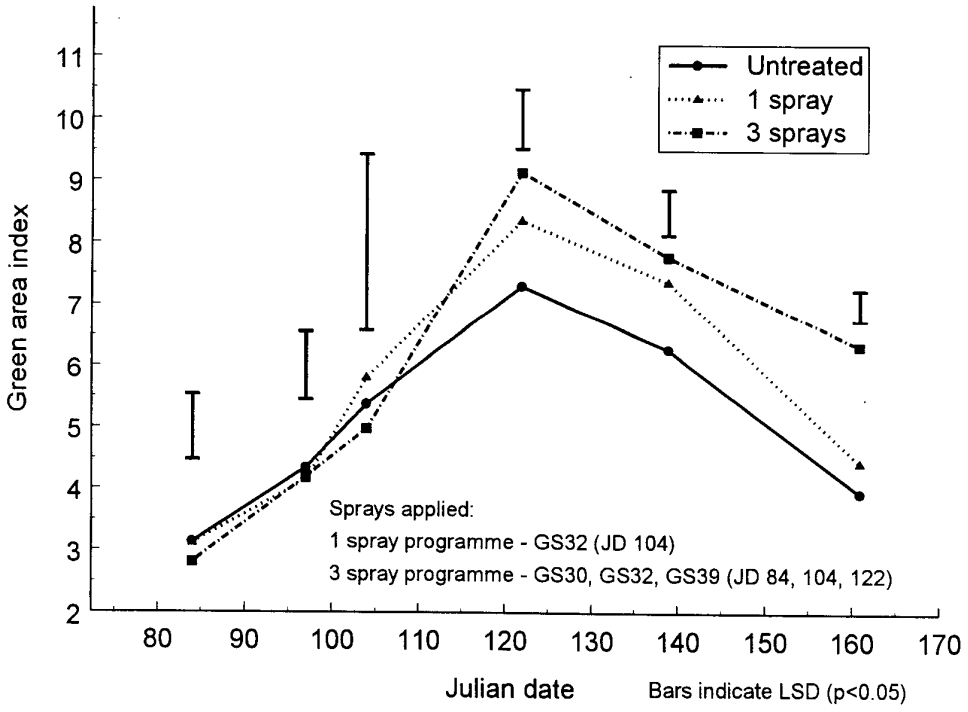
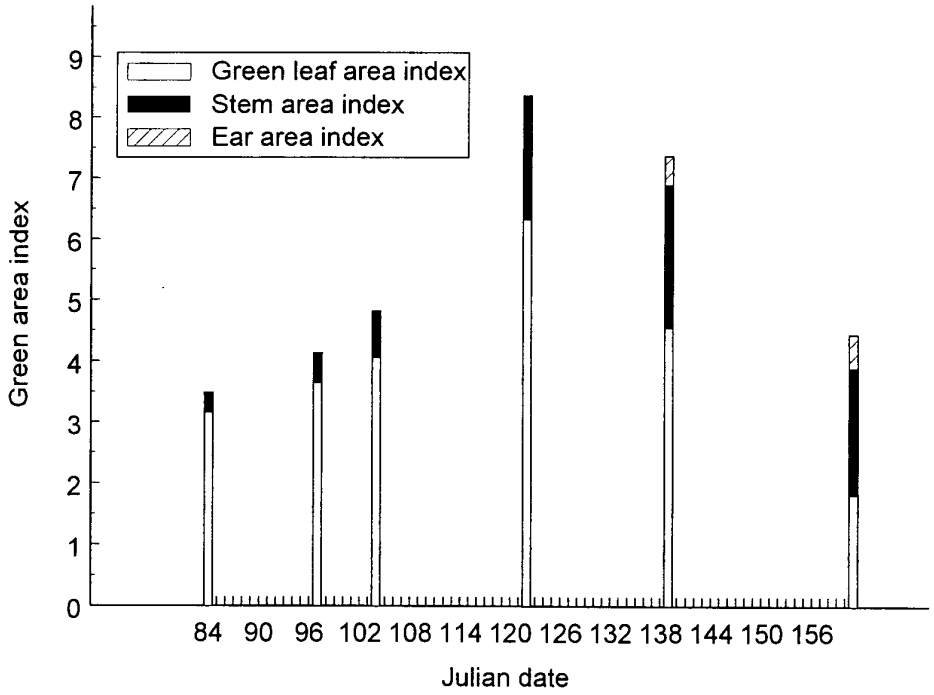


Fig. 3. Changes in green area index with time. Rosemaund 1997



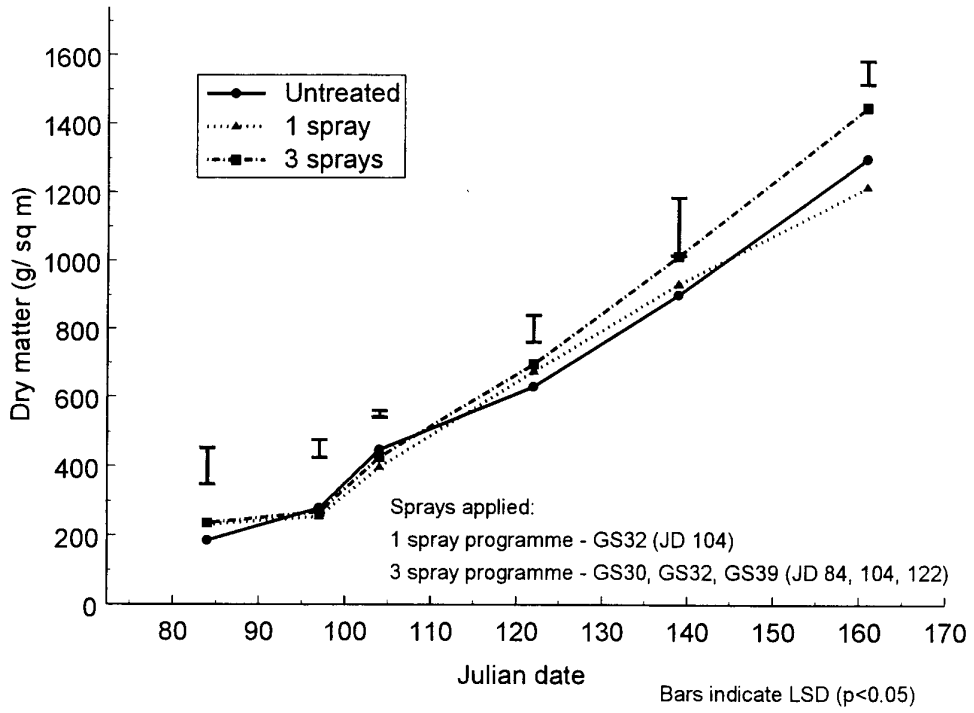
The relative contribution of green area provided by the leaves, stem and ear to GAI is shown in Fig. 4. The leaves consistently provided the greatest green area until GS 75 (JD 161).

Fig. 4. Green area index of leaves, stem and ear with time for one spray fungicide programme. Rosemaund 1997



Dry matter increase was slow between GS 30 (JD 84) and GS 31 (97) but thereafter the increase was uniform up to GS 75. There were no significant differences between GS 30 and GS 59 (Julian days 84 to 139) but after GS 59, the three spray programme had a significantly greater dry matter composition than the other two treatments (Fig. 5).

Fig. 5. Changes in dry matter with time. Rosemaund 1997



The daily incident radiation was recorded at a meteorological station c. 3 miles north of ADAS Rosemaund. The daily green leaf area index was calculated for each replicate of each treatment by assuming a linear increase from one sampling date to the next. Daily light interception was estimated from the GLAI and the total daily incident radiation using a derivation of Beer's Law

$$f = 1 - e^{(-kL)}$$

where f = fraction of light intercepted
k = extinction coefficient
L = GLAI (Bryson et al, 1997)

The accumulated light interception was related to the increases in dry matter from the first sampling date, GS 30 (JD 84). A single errant data point for one replicate of the one spray programme was omitted because it was at extreme variance from all the other data points which exhibited a close relationship between these two factors (Fig. 6). The correlation coefficient was 0.982 when all data points were used.

When a snapshot of dry matter accumulation was taken at GS 75 and related to disease at the same growth stage, there was only a moderate correlation between the two factors (Fig. 7). A better correlation was found between yield and disease severity (Fig. 8).

When green leaf area index was correlated against either biomass or yield (Figs. 9 and 10), the relationship was reasonable (r = 0.73 and 0.69) but failed to account for as much of the total variation as that of the relationship between accumulated light interception and dry matter increase.

Fig. 6. Dry weight increase in relation to accumulated light interception between GS 30 and GS 75. Rosemaund 1997

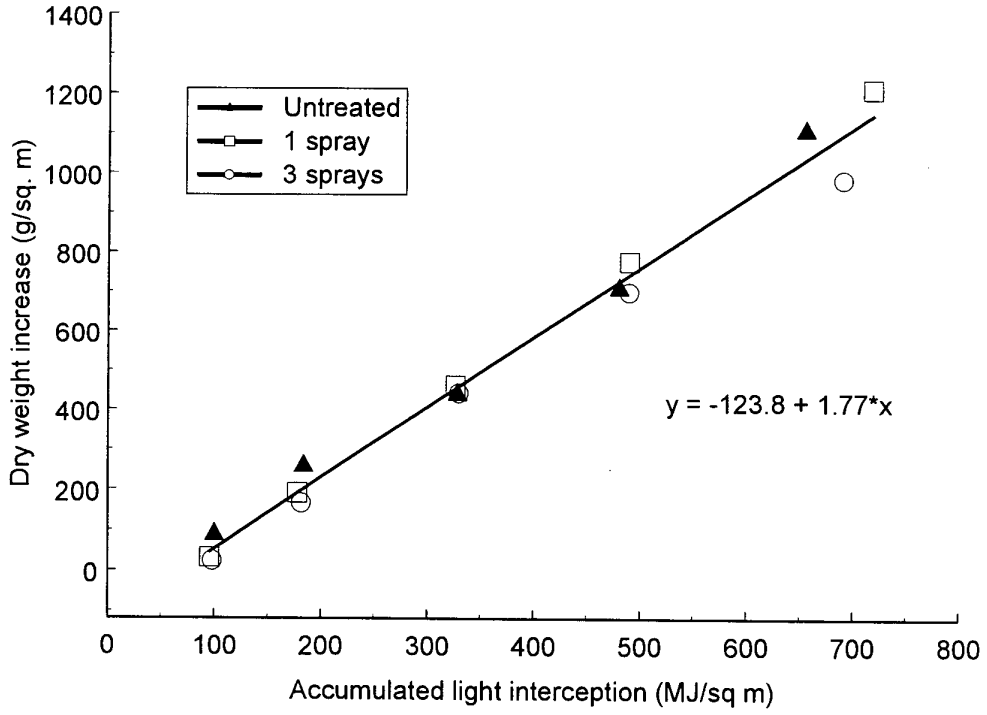


Fig. 7. Relationship between disease on the top 3 leaves and biomass at GS 75. Rosemaund 1997

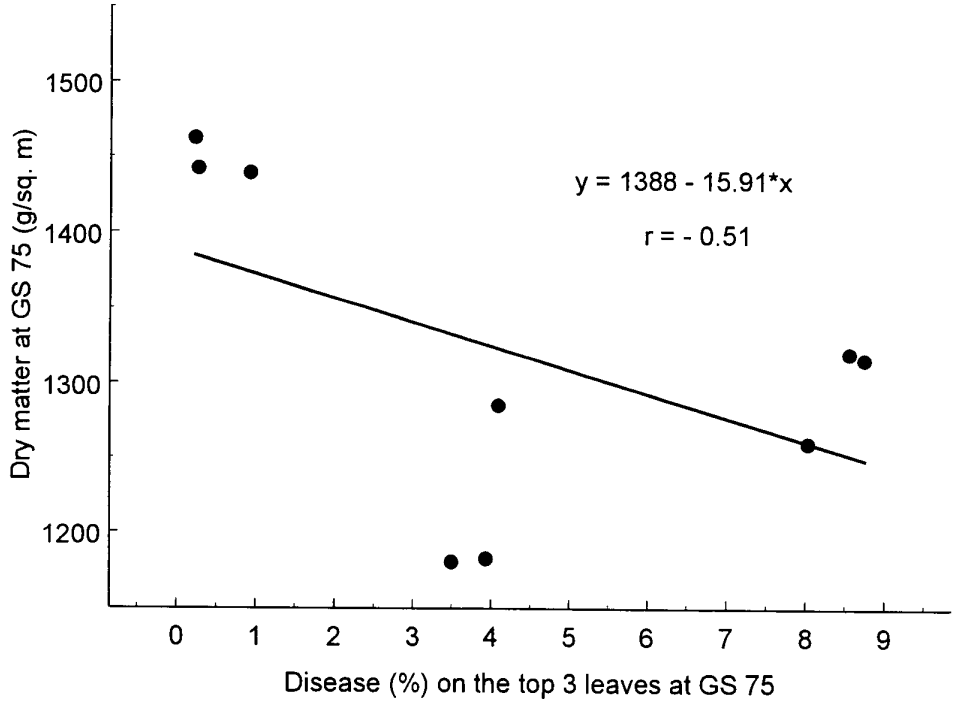


Fig. 8. Relationship between disease on top three leaves at GS 75 and yield Rosemaund 1997

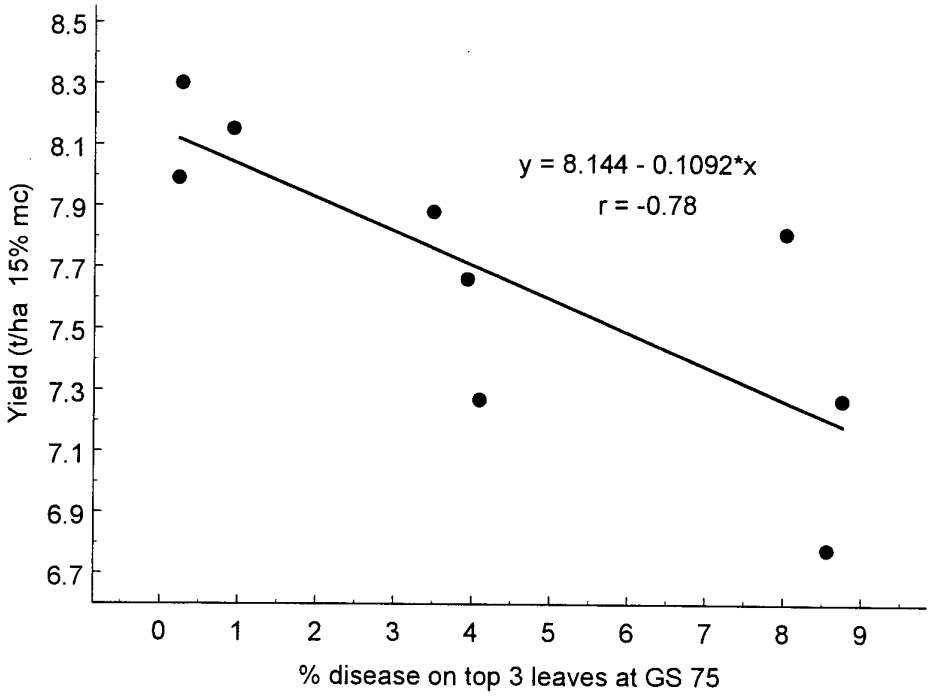


Fig. 9. Relationship between green leaf area index and biomass. Rosemaund 1997

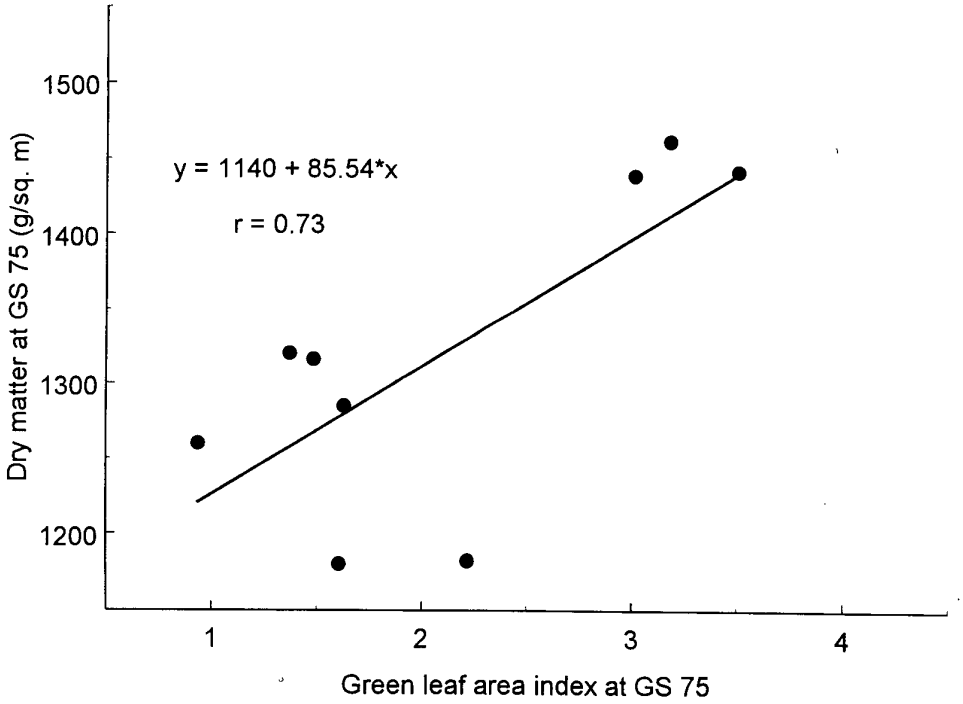
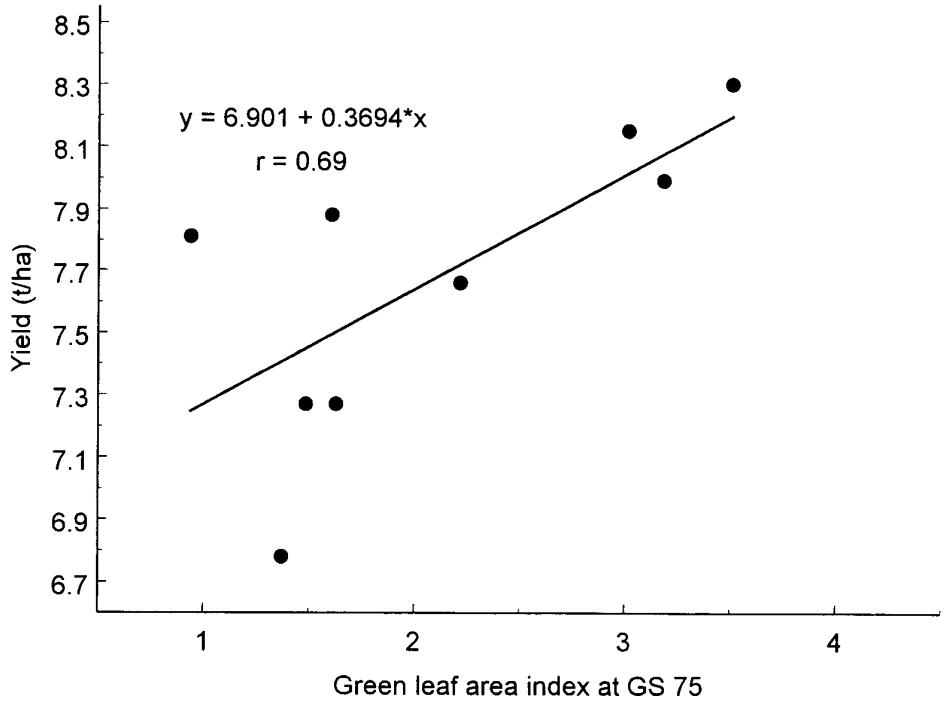


Fig. 10. Relationship between green leaf area index at GS 75 and yield. Rosemaund 1997



Grain yield of the three spray programme was significantly greater than the untreated control but not greater than the one spray programme (Table 1). Although there were no significant differences between thousand grain weights, the extra yield appeared to arise mostly from extra grain weight in the three spray programme. There were no significant differences in specific weight, grain number per ear or number of fertile ears per m^2. Low values in a single replicate of the one spray programme with respect to quality characteristics and harvest parameters increased variability and may have resulted in significant differences failing to be detected. Despite a significant difference in yield, when a financial analysis was made there were no significant differences and the three spray programme gave the lowest return. Stem soluble carbohydrates were measured on one occasion, at full ear emergence. Whilst the three spray programme had the highest percentage content this was not significantly greater than the other two treatments. When converted to tonnes per hectare there were, once again, no significant differences (Table 1).

Table 1. Rosemaund - Yields, quality characteristics, harvest parameters, stem soluble carbohydrates and margin over costs

	Yield	Specific weight	Thousand grain weight	Harvest index	Fertile ears	Grain number per ear	Stem soluble carbohydrates 19 May - GS 59		Margin over costs (+)
Treatment	t/ha @ 15%mc	kg/hl	g	%	no./m^2	no.	% dm	t/ha	£/ha
UT	7.29	71.0	43.5	48.1	968.0	18.0	26.7	0.00217	510.1
1 spray	7.60	70.9	39.0	44.9	873.0	15.1	26.5	0.00230	504.4
3 sprays	8.15	70.4	46.4	49.4	852.0	18.9	29.3	0.00260	486.3
s.e.d.	0.446	0.527	4.12	3.10	38.8	2.36	3.52	0.00043	13.18
significance	*	ns	ns	ns	ns	ns	ns	ns	ns

(+) Margin over costs based on a barley price of £70/tonne, Tilt @ £11.50 per full dose and Aura/Corbel @ £16.50 per 3/4 dose

Aberdeen

By contrast to ADAS Rosemaund, disease levels were severe throughout the spring and summer at Aberdeen. Mildew was the main disease but Rhynchosporium also reached moderate levels. By GS 71-75 (JD 175) there was 45% leaf area infection by mildew on the second top leaf and 10% Rhynchosporium. By contrast, the three spray programme kept mildew infection below 5% and Rhynchosporium below 2% on the same leaf. The one spray programme had initially kept disease levels low but by GS 75, disease on this treatment was approaching that on the untreated.

Significant differences were recorded in GLAI at GS 59 and GS 75 (JD's 153 and 175). At GS 59, GLAI of all three treatments were significantly different (Fig. 11). At GS 75, when disease control on the one spray programme was beginning to decline, the three spray programme was significantly greater than each of the other two treatments. This pattern was repeated when the GAI was considered (Fig. 12).

Fig. 11. Changes in green leaf area index with time. Aberdeen 1997

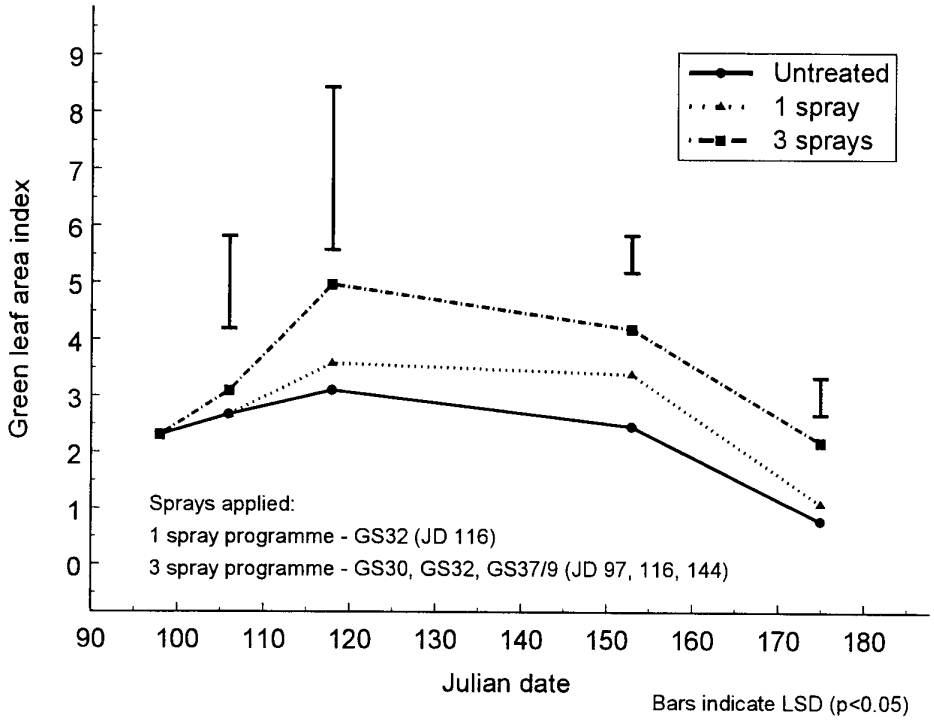
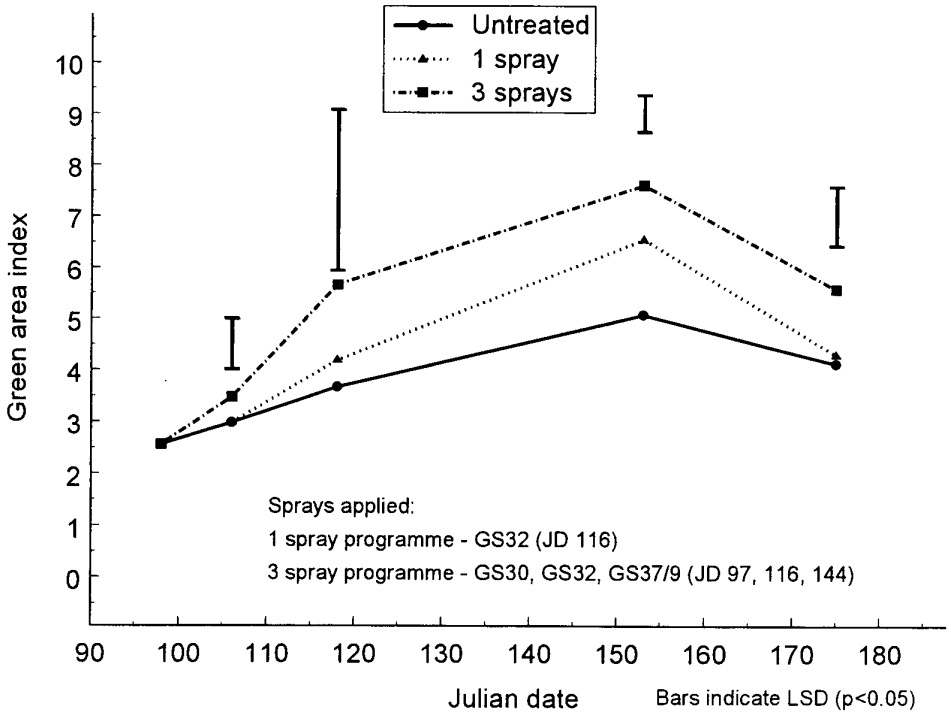
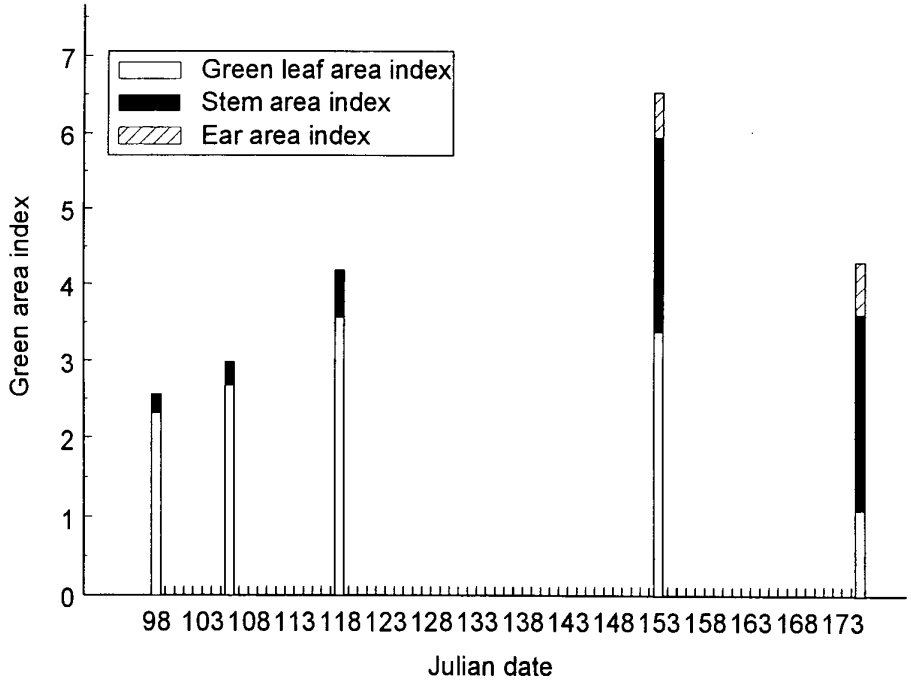


Fig. 12. Changes in green area index with time. Aberdeen 1997



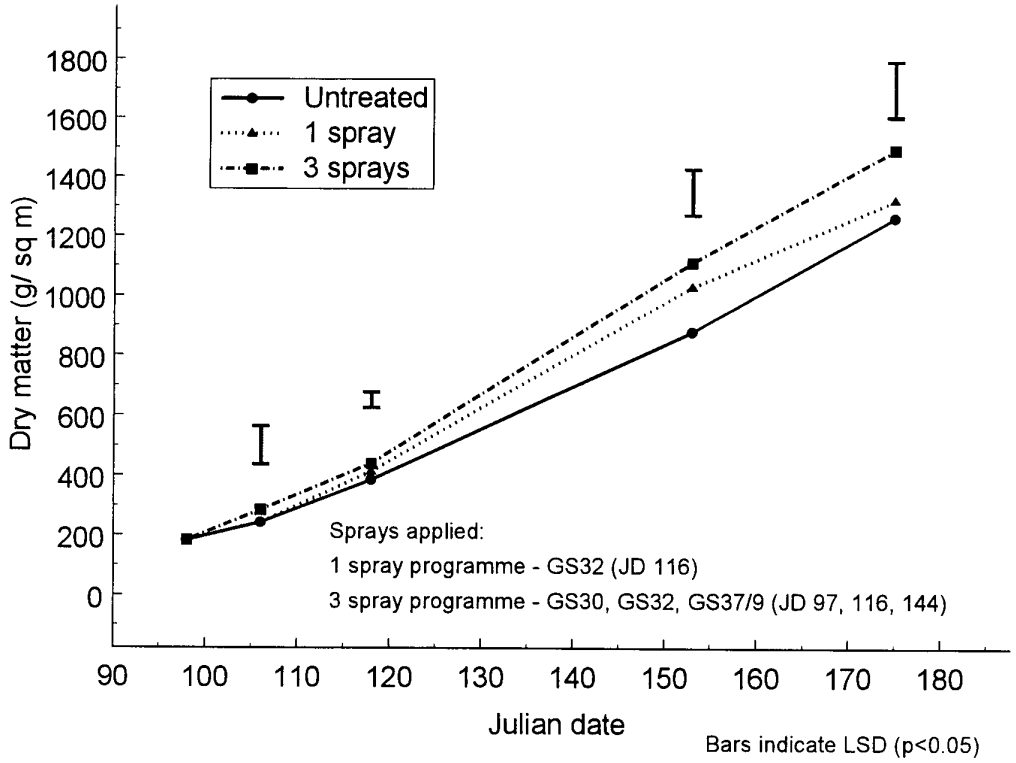
GLAI, once again constituted the greatest portion of the GAI (Fig. 13). However, unlike Rosemaund, the stem green area at Aberdeen formed about 40% of the total GAI at GS 59 and 60% GAI at GS 71-75 with the one spray programme.

Fig. 13. Green area index of leaves, stem and ear with time for one spray fungicide programme. Aberdeen 1997.



The increase in dry matter with time at Aberdeen was similar to that at Rosemaund, although the difference between the untreated and the three spray programme was greater at Aberdeen than Rosemaund (Fig. 14). Significant differences occurred at the later growth stages. At GS 51-59 (JD 153), the 3 spray programme resulted in significantly greater dry matter than the untreated control. At GS 71-75 (JD 175), the same significant difference was recorded. Whilst the three spray programme was not significantly different from the one spray programme, disease build up on the latter resulted in its dry matter approaching that of the untreated control

Fig. 14. Changes in dry matter with time. Aberdeen 1997



When accumulated light interception was plotted against dry weight increase as described above a close relationship with a straight line fit was confirmed (Fig. 15). The separation of points at the last sampling date was greater than for Rosemaund. The regression line passes closer to the origin than with that for Rosemaund. The correlation coefficient for the two variables is 0.948 when all data are used.

The relationship between disease at GS 71-75 on the top 2 leaves and biomass (Fig. 16) accounted for more of the total variation than that between disease on the top 2 leaves at GS 71-75 and yield (Fig. 17). Because regular disease assessments were made at the Aberdeen site, it was possible to calculate the Area under the Disease Progress Curve (AUDPC) for the top 4 leaves. Plotting these values against biomass (Fig. 18) or yield (Fig. 19) had no effect on the correlation coefficient.

Fig. 15. Dry weight increase in relation to accumulated light interception between GS 30 and GS 71-75. Aberdeen 1997

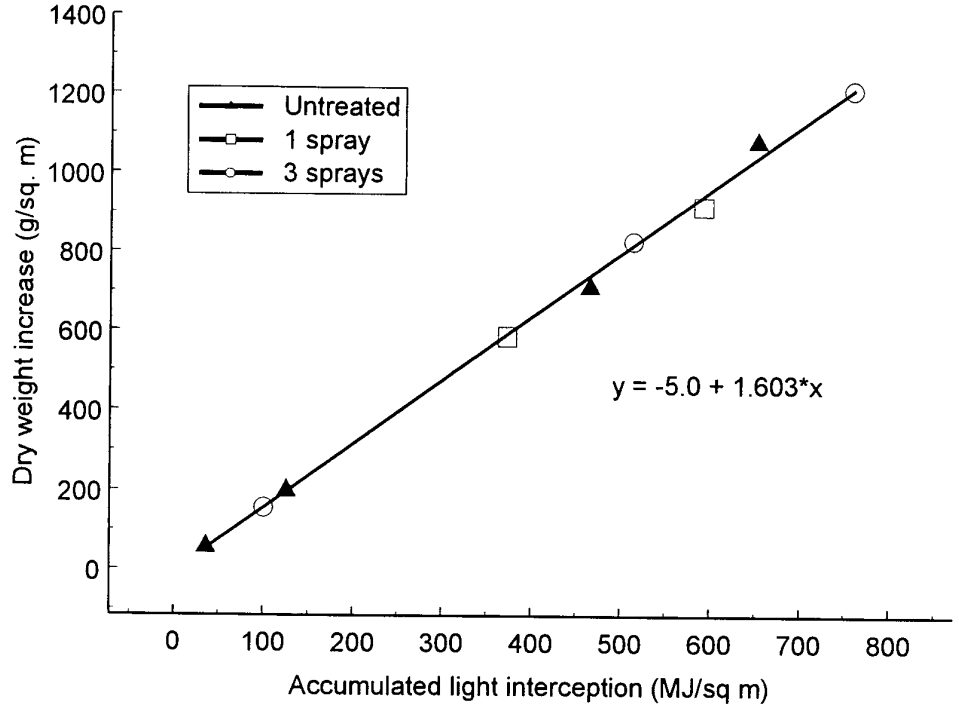


Fig. 16. Relationship between disease on the top two leaves and biomass. Aberdeen 1997

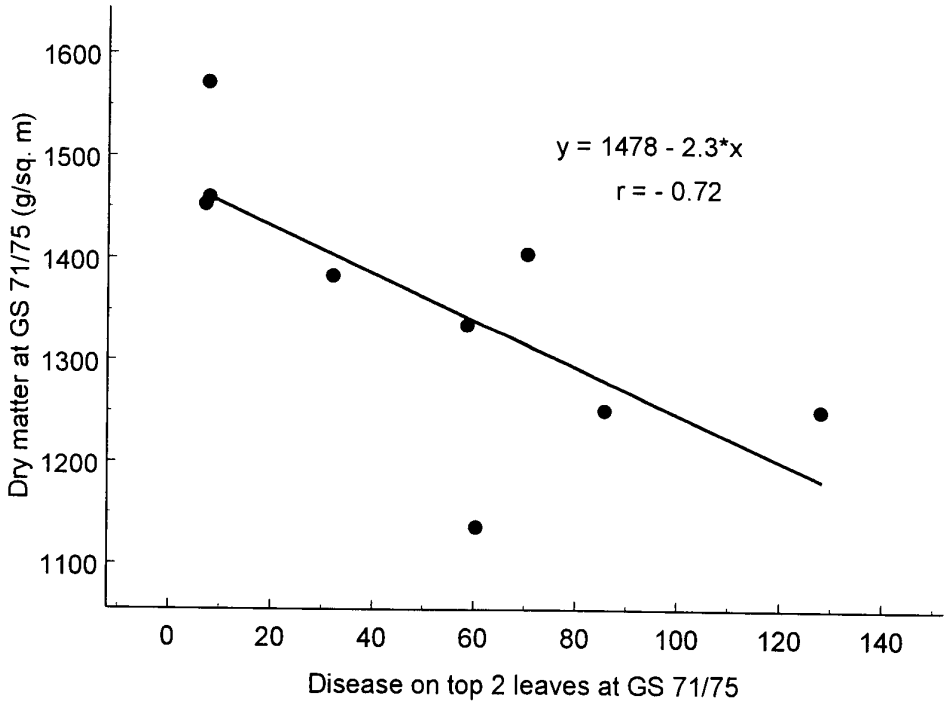


Fig. 17. Relationship between disease on top two leaves at GS 75 and yield Aberdeen1997

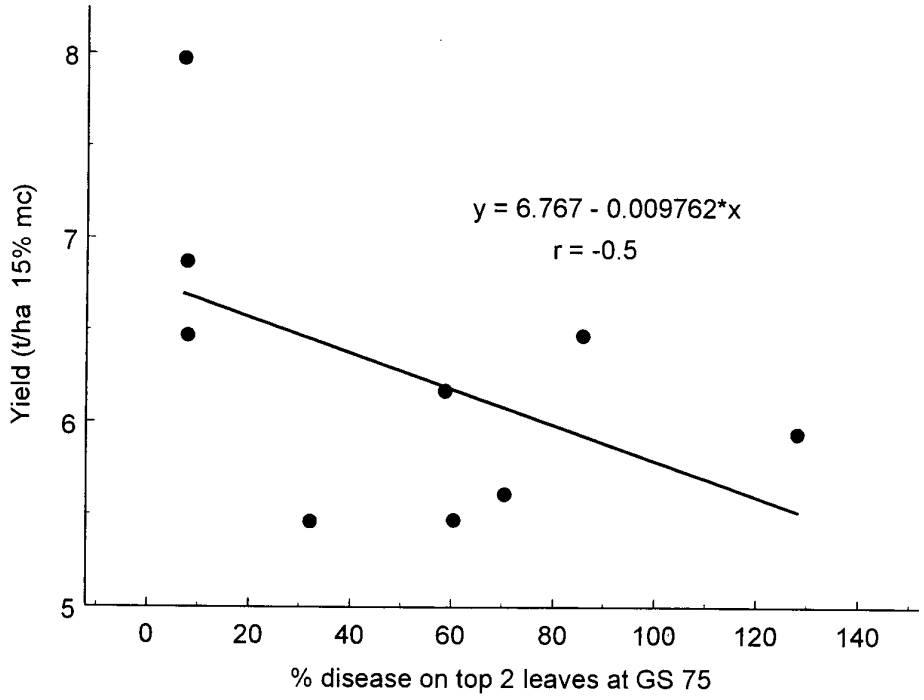


Fig. 18. Relationship between disease (AUDPC) and dry matter at GS 71/75. Aberdeen 1997

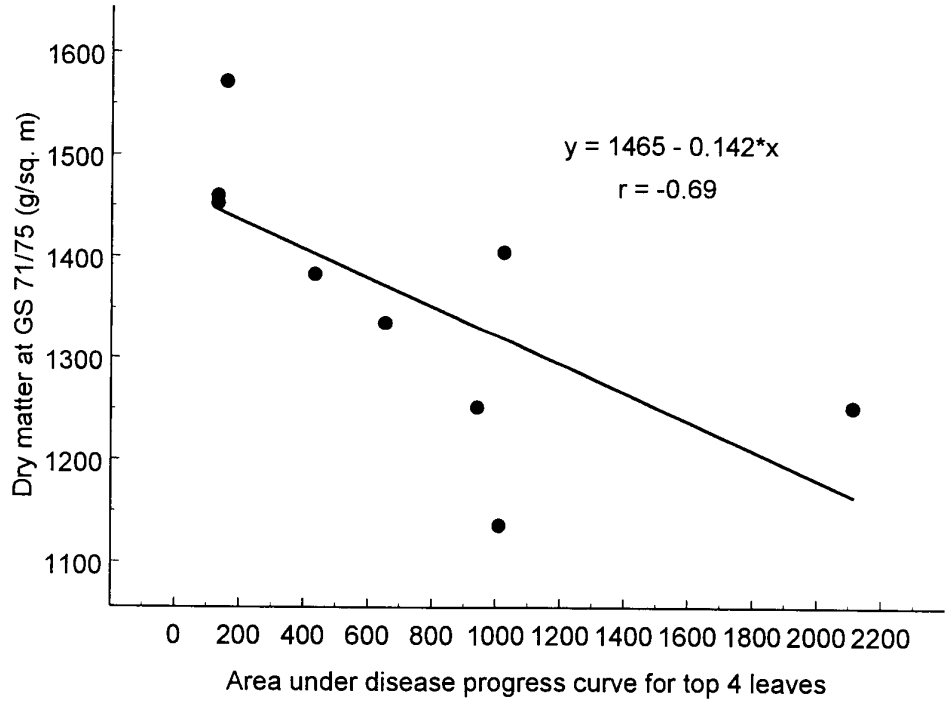
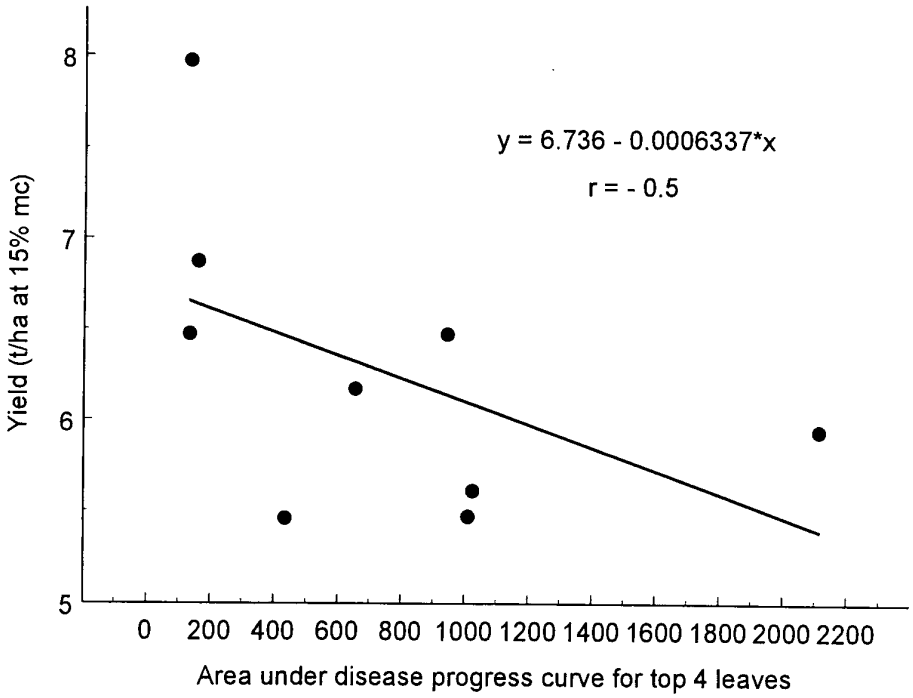


Fig. 19. Relationship between disease (AUDPC) and yield. Aberdeen 1997



The relationships between GLAI and biomass (Fig. 20) or yield (Fig. 21) showed greater correlation coefficients than disease and biomass or yield.

Fig. 20. Relationship between green leaf area index and dry matter at GS 71/75. Aberdeen 1997

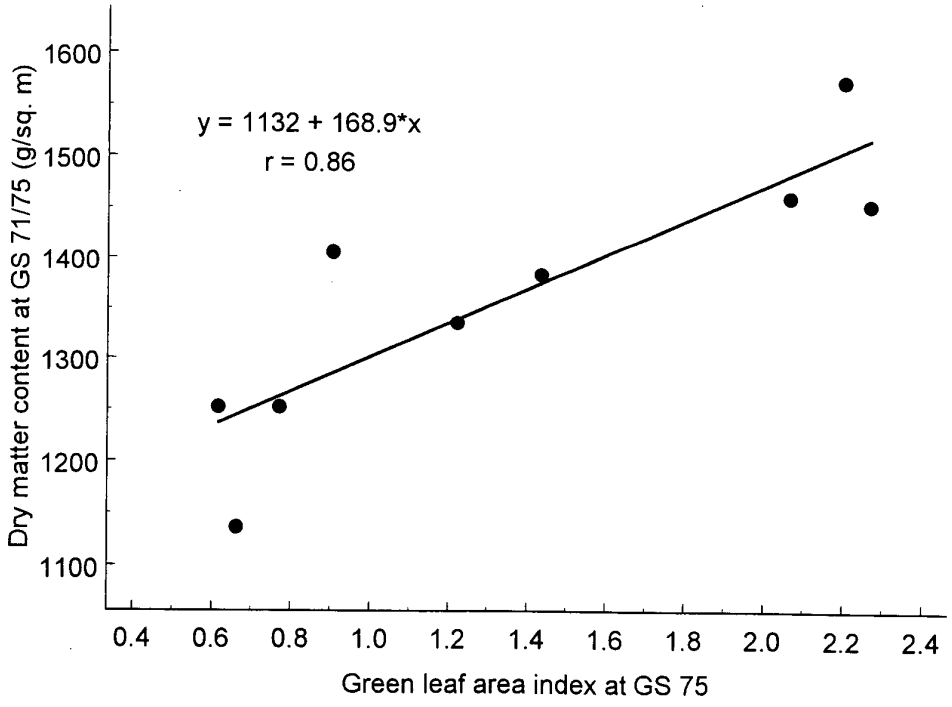
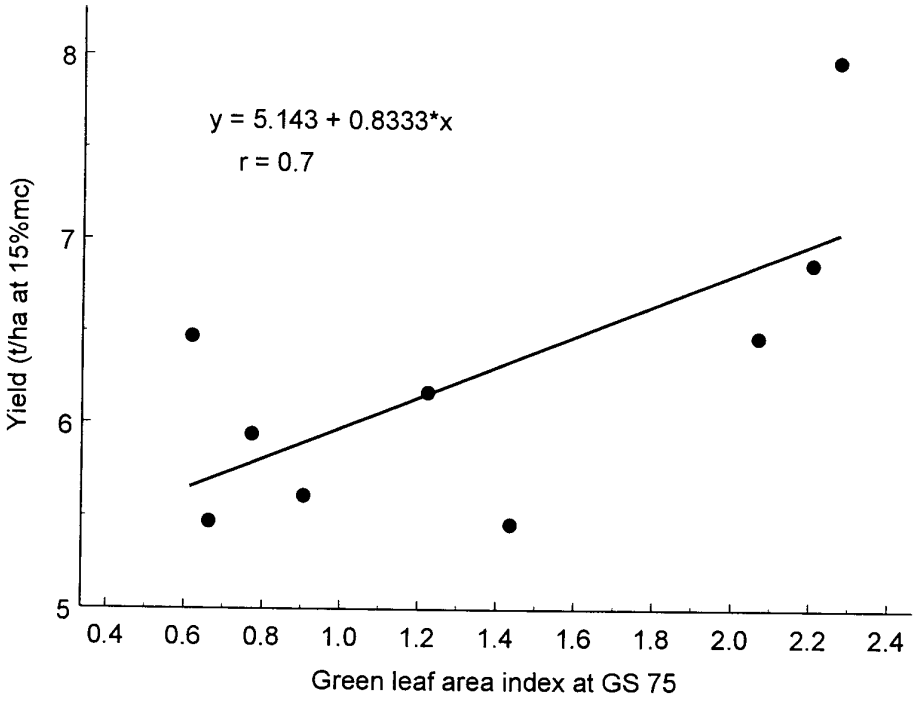


Fig. 21. Relationship between green leaf area index at GS 75 and yield. Aberdeen 1997



The three spray programme significantly increased yield over both the untreated and the one spray programme but there was no significant difference between the latter two treatments. The increase in yield appeared to result from significant increases in thousand grain weight in the three spray programme. Despite the large increase in yield for the three spray programme, the margin over cost was not significantly greater than the other treatments.

Stem soluble carbohydrates were determined on two occasions, at GS 59/61 and GS 85. Whilst the percentage of dry matter that constituted stem soluble carbohydrates increased nearly twenty fold in 52 days, there were no significant differences detected between treatments.

Table 2. Aberdeen - Yields, quality characteristics, stem soluble carbohydrates and margin over costs

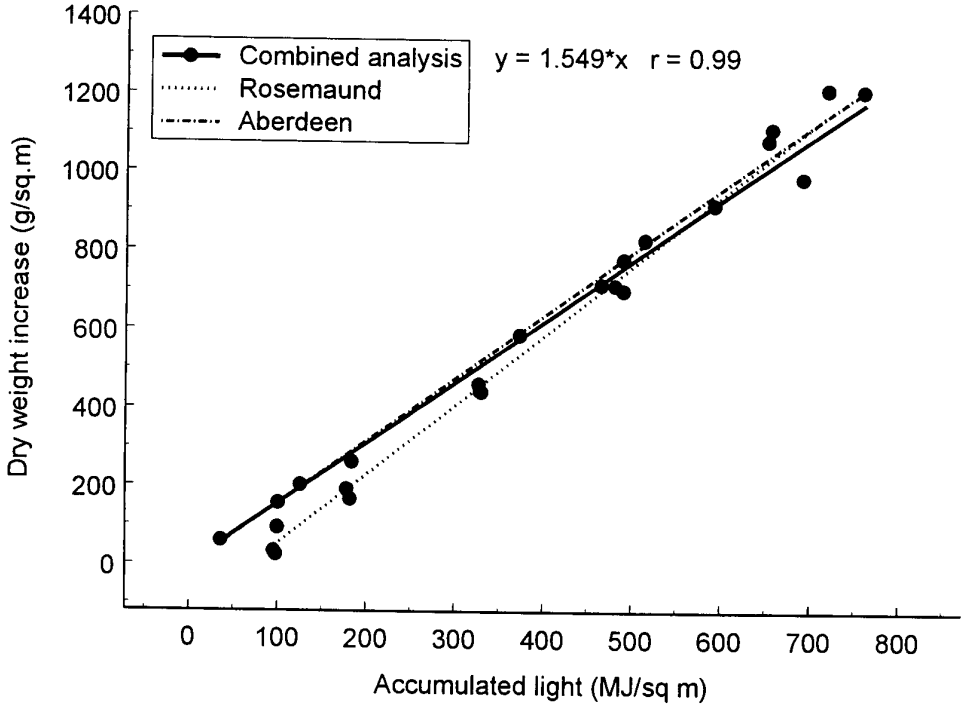
	Yield	Specific Weight	Thousand grain weight	Fertile ears	Stem Soluble Carbohydrates 9 June GS 59/61	Stem Soluble Carbohydrates 31 July GS 85	Margin over costs (+)
Treatment	t/ha @15% mc	kg/hl	g	no./m^2	%dm	%dm	£/ha
UT	5.67	61.4	35.0	892	0.79	21.3	397.1
1 spray	6.03	61.9	35.3	909	0.8	21.2	394.3
3 sprays	7.10	63.6	40.0	900	1.27	20.3	413.2
s.e.d.	0.248	0.32	1.44	62.0	0.356	2.19	17.34
significance	**	***	*	ns	ns	ns	ns

(+) Margin over costs based on a barley price of £70/tonne, Tilt @ £11.50 per full dose and Aura/Corbel @ £16.50 per 3/4 dose

DISCUSSION

There were two key findings from this pilot study. Firstly, there was a close relationship between dry matter accumulation and accumulated light interception. When all the data points are pooled from both sites and a single regression line drawn passing through the origin, it accounted for a large part of the variation (correlation coefficient = 0.99). Figure 22 shows the data points from the two sites and a combined line constrained to pass through the zero point. This graph reflects the period of the crop from GS 30 to GS 75. What happens after GS 75 is uncertain, but it can be expected that dry matter accumulation continues until all green tissue has senesced.

Fig. 22. Dry weight increase in relation to accumulated light interception. Data from two sites.



The importance of this finding, that differences in biomass are achieved through differences in light interception and accumulation, is that the impact of disease is largely mediated through the loss of green tissue and hence light interception. It is important therefore that when fungicide dose is being considered, account is taken of the condition of the crop as well as disease level in the crop (inoculum), weather factors, variety resistance or tolerance to disease and growth stage.

This finding implies that it would be helpful to identify benchmark GAI's at different growth stages. This would permit a grower to determine the degree to which green tissue needs to be enhanced or protected to maintain the optimum. Such benchmark figures cannot be identified from this study, nor were they identified in the literature review.

These results do show how disease can affect canopy and thus light interception, but do not permit judgement as to how to manipulate fungicide dose.

When separate regression lines were fitted to the data for the two sites, the slopes were significantly different. This implies that the Radiation Use Efficiency (RUE) of the crop at Rosemaund was slightly greater than that at Aberdeen.

The second key finding concerns the impact of disease at the two sites. At Rosemaund, low levels of net blotch were present from late winter through most of the growing season. Later, mildew was the most prominent disease but Rhynchosporium was also present. However, disease levels were many times lower than that at the Aberdeen site where mildew primarily (but also Rhynchosporium) were severe. At the same growth stage (i.e. GS 75), the difference in GLAI between the three spray programme and the untreated control was approximately two units at Rosemaund whereas it was only 1.25 units at Aberdeen. The GLAI of all the treatments at Aberdeen were lower than those at Rosemaund, but it is puzzling why the differences in GLAI between three-spray and untreated was greater at Rosemaund when disease levels were so much less.

It is, of course, dangerous to consider only a single point in time, but this disparity does show at earlier growth stages also. It may be that Puffin, the variety used at Rosemaund, is more sensitive to disease.

The similar dry matter contents of treatments at GS 75 at the two sites were not reflected in the same differences in yield at harvest. The difference between the three spray programme and the untreated at Rosemaund was 0.86 t/ha whereas at Aberdeen it was 1.43 t/ha. This is likely to be due to differences in harvest index. Two replicates of the Aberdeen site lodged badly and in consequence their harvest index was probably much lower than at Rosemaund (but not measured).

What is not clear from this study is why an early fungicide application at GS 30 can sometimes give a yield response. The suggestion from the data is that such a fungicide treatment had little effect on the canopy size. That is, where the first fungicide application was made in the three spray programme at GS 30 and compared to the untreated, there was no significant difference in GLAI or GAI measured subsequently at GS 32. Thus it may be that the benefit of an application at GS 30 is due to the reduction of inoculum and the consequential effect on epidemic development.

Also unclear is why the most effective timing for fungicide application in winter barley is at GS 31/2. There is no evidence from this and other studies that this particular timing is crucial to maintain grain numbers as the ear develops within the stem. In most trials, differences in grain number per ear between fungicide treatments and untreated control are non-significant. It may be that effects of disease at this time cause small but significant reductions in tiller survival. It may also be that the timing simply protects the key leaves for light interception against disease.

In terms of crop development, the trial at Aberdeen was consistently 10-14 days later than that at Rosemaund. This may be because the sites had different varieties. Experience from the Appropriate Fungicide Dose Project, however, with the same variety at different sites, suggests this is the norm for the latitudes involved.

Stem length was markedly greater in Pastoral at Aberdeen than in Puffin at Rosemaund and this contributed more to GAI at Aberdeen. There were no indications at either site that different disease epidemics affected accumulation of stem reserves. Consequently, it has not been possible to ascertain whether stem reserves accumulated at one time can compensate for loss of green leaf later.

This trial only had three treatments and three replicates (matching the rest of Experiment 1). It is clear that in future physiological work a minimum of four replicates is needed. In part, this is exemplified by the fact that there was more variability in the untreated and one spray programme physiology data than in the three spray programme data. This is to be expected as foliar disease occurs in a patchy, uneven distribution and this leads to variability in samples. By contrast a three spray programme virtually eliminates disease, and hence sampling occurs in a more uniform crop.

The trial used full fungicide doses in the spray treatments. The cost benefit analysis of the programmes indicated no benefit of fungicide at Rosemaund and only a significant benefit from the three spray programme at Aberdeen. In reality, reduced doses would be expected to give similar levels of disease control to full doses and the separation in profitability between treatments would probably have been greater.

ACKNOWLEDGEMENTS

We would like to thank Dr David Jones and the staff at ADAS Rosemaund for carrying out the study at Rosemaund. The authors would like to acknowledge the assistance given in developing the protocol for these small physiological studies by Dr Rosie Bryson. Thanks are due also to Dr Neil Paveley and other pathology colleagues in ADAS for introducing physiological concepts to other humble pathologists

REFERENCES

Anon. (1997) The Wheat Growth Guide - to improve husbandry decisions. Home-Grown Cereals Authority, 32pp.

Bryson, R J; Sylvester-Bradley, R; Scott, R K; Paveley, N D (1995) Reconciling the effects of yellow rust on yields of wheat through measurement of green leaf area and radiation interception. *Aspects of Applied Biology*, **42**, *Physiological responses of plants to pathogens*, 9-18.

Gaunt R E (1995) The relationship between plant disease severity and yield. *Annual Review of Phytopathology*, **33**: 119-144.

APPENDIX TO PART 1 - SITE DETAILS

	Aberdeen	**Rosemaund**
Field name	26/27/28 Skillydam	Moorfield
Sowing date	26 Sept 96	1 Oct 96
Seed rate	218 kg/ha	350 seeds/m^2
Seed treatment		Panoctine Plus
Soil type	Sandy clay loam	Silty clay loam
Soil series	Pitmedden	Bromyard
Soil association	Tarves	Bromyard
Drainage	Imperfect	Good
Elevation	105 m	100 m
Slope	Slight to north	Slight to south
Soil analysis - date	12 Sept 95	
Mg (mg/l)	198 (Mod)	115 (3)
P (mg/l)	11 (Mod)	17 (2)
K (mg/l)	179 (Mod)	132 (2)
pH (mg/l)	5.9	7.1
Organic matter %	8.2	
Cultivations	Ploughed, Kuhn harrow, sown, rolled after sowing	Ploughed 11 Sept 96 Power harrow 1 Oct 96
Seed drill	Accord	
Row width	13 cm	13.5 cm
Plot size	3 m x 20 m	2 m x 18 m at sowing 2 m x 16 m at harvest
Previous cropping		
96	WOSR	WW
95	WB	WOSR
94	SB	WW
93	WW	WO
Herbicides	Panther 1.5 l/ha 8 Oct 96	Panther 1.0 l/ha + 3.0 l/ha IPU 16 Nov 96 Optica 1.5 l/ha 7 April 97
Fertiliser	0:90:90 2 Sept 96 80 kg/hl 6 March 97 90 kg/hl 18 April 97	No seed-bed fertiliser 49 kg/ha 12 March 97 73 kg/ha 18 April 97
Other treatments		0.25 l/ha Cyperkill 16 Nov 96 2.25 l/ha Holdup 7 April 97 1.25 l/ha Terpal 3 May 97

PART 2

THE PHYSIOLOGY OF RESPONSES OF WINTER BARLEY TO DISEASE

I J BINGHAM, W J THOMSON & S J WALE

SAC, CROPS DIVISION, FERGUSON BUILDING, CRAIBSTONE ESTATE, BUCKSBURN, ABERDEEN AB21 9YA

INTRODUCTION

Disease control decisions are usually made on the basis of the severity of infection and the risk of its spread. However, work on wheat and spring barley, and more recently winter barley (HGCA Winter Barley Appropriate Fungicide Doses), has shown that yield losses cannot be predicted reliably from simple assessments of disease severity. As such measurements of disease severity and forecasts of epidemic development alone are not sufficient to determine the need for fungicide applications. It is now recognized that disease control decisions should be based on an understanding of the effects of pathogens on crop function. This review examines our current knowledge of the effects of pathogens on the physiology of winter barley and considers the implications for disease control.

An extensive search of the literature has revealed a wealth of information on the epidemiology and control of winter barley diseases. In contrast, there have been few studies dealing specifically with the effects of pathogens on the physiology of this crop. Winter wheat and spring barley have been studied more extensively. The review, therefore, draws on information available for cereals in general (winter wheat, spring barley and winter barley) and then identifies potential differences in response between winter wheat and winter barley which may influence decision making with respect to disease control. Only foliar pathogens are considered.

THE REVIEW COMPRISES:

1. An overview of the HGCA cereal physiology review (Physiology in the Production and Improvement of Cereals, HGCA Research Review No. 18; R. Sylvester-Bradley & R. K. Scott 1990) with particular reference to the suggestions made regarding disease physiology.

2. A discussion of more recent developments in understanding of the effects of foliar disease on crop function, with particular reference to winter and spring barley and winter wheat.

3. A summary of the differences between winter barley and winter wheat in terms of growth, development, physiology and disease, to ascertain whether our current knowledge of wheat responses to disease can be readily transferred to winter barley.

4. A short bibliography of additional literature relevant to the effects of foliar disease on the physiology of winter barley, but not covered in 2.

5. A summary of the main findings from work on winter barley physiology reported in Part 1 of this study

6. A discussion of research priorities, based on 1-5 above, to extend our understanding of the effects of disease on the physiology of winter barley, with the aim of improving recommendations for fungicide use.

1. OVERVIEW OF THE CEREAL PHYSIOLOGY REVIEW (HGCA RESEARCH REVIEW No. 18)

The application of physiological principles to the management of cereal crops began in earnest with the publication of the cereal physiology review (Sylvester-Bradley and Scott 1990). The review set out to examine the current knowledge of cereal physiology in relationship to yield formation. With the main emphasis on wheat it considered:

A. The morphology of the cereal plant: growth and development of leaves, roots, tillers, ears and grains.

B. The relevance of physiology to the management of the crop in the field. The sections considered were: variety selection, sowing, nitrogen fertilizer, weed control, disease control, plant growth regulator use, quality, tailoring husbandry to site and season, and the impact of crop improvement through plant breeding and biotechnology.

C. Each section concluded with suggestions for research which would increase our understanding of wheat physiology and ultimately to improve recommendations to farmers for crop management.

D. The cereal physiology review concluded by proposing a strategy to exploit wheat physiology in the future. Seven integrated research strategies were identified:

- Physiological responses and varietal characterisation;
- Match cereal variety selection and husbandry to a wider range of sowing dates;
- Nitrogen fertilizer application strategies based on the way nitrogen governs plant growth and development;
- Estimation of the consequences of damage by disease;
- Assessment of the contributing factors which give rise to lodging;
- Prediction of the quality of cereal grain in advance of harvest;
- Assessment of crop productivity before harvest.

Information contained in the review and further research conducted on the basis of its recommendations have culminated in the publication of the Wheat Growth Guide (Anon 1997). The following section is a summary of how wheat growth and development determines yield. It is based largely on information in the cereal physiology review and Wheat Growth Guide, but supplemented in places with information from other sources. The purpose of the summary is to provide a framework for considering the differences between wheat and barley, which may influence their response to disease.

1.1 Summary of wheat growth and development

1.1.1 Light interception and growth

The objective of crop management is to optimize growth during each phase of development that contributes to grain yield. Growth is the increase in size or weight of the crop, whereas development is the change in crop form (as described by the Zadoks decimal code) as it

progresses through its lifecycle. Maximum yields are achieved when conditions favour rapid growth coupled to slow development.

Growth results from photosynthesis. The rate is determined by the amount of light falling on the crop canopy, the size of the canopy and the efficiency with which the light energy is converted into dry matter. Light availability follows a seasonal pattern with a distinct summer peak between May, June and July, but it also varies unpredictably through changes in weather conditions. Cloudy days typically have less than half the light energy of sunny days.

The size of the canopy is usually measured as GLAI (the green leaf area index) or GAI (the green area index). GLAI is the area of green leaf surface of the crop per unit of ground surface. GAI is the ratio between the total green area (leaves plus stem) and the ground surface. However, as leaves account for a large proportion of the total green area, GLAI is closely related to GAI. As leaves of the mainstem and tillers emerge and unfold, they contribute to the GLAI and GAI. Providing there is an adequate supply of water and N, leaf emergence and expansion is directly related to temperature. Rates of canopy expansion are therefore slow during periods of low temperature immediately after crop establishment. As temperatures rise during the spring, the canopy undergoes a period of rapid expansion reaching a peak between flag leaf emergence and flowering. Thereafter the canopy begins to senesce and GLAI and GAI decline.

Recent work has firmly established the principle of an optimum GAI for crop canopies. In wheat, a GAI of 5-6 is sufficient to intercept 90% of the available light. Further increases in GAI provide progressively diminishing returns in terms of the additional light intercepted. Benefits of additional light interception must be weighed against the costs of producing and maintaining the canopy. Dense canopies are at greater risk from lodging and possibly disease epidemics. Wheat varieties have been found to differ to only a small extent in their optimum GAI. Maximum amounts of dry matter are accumulated when the optimum GAI is established as rapidly as possible and is then maintained for as long as possible.

Dry matter production per unit of total light energy intercepted by cereals averages 1.2-1.4g dry matter/MJ. In general, crop husbandry has relatively little effect on radiation use efficiency (conversion efficiency) and influences yield mostly through effects on the size and duration of the canopy. However, radiation use efficiency can be decreased by some diseases, water stress and extreme nitrogen deficiency through reductions in the rate of photosynthesis per unit leaf area. Radiation use efficiency will also be influenced by changes in canopy structure and degree of light saturation of the upper leaves.

1.1.2 Growth in relation to phases of development.

Key stages in the life cycle of a cereal crop are:

Establishment
Leaf emergence
Tillering
Stem extension & ear development
Deposition of stem storage reserves
Flowering
Grain filling
Ripening

The stages follow each other, but there may be considerable overlap between them. For example, leaf emergence commences shortly after establishment and continues until a little over half way through stem extension.

The rate of development is governed largely by temperature and seasonal changes in photoperiod. Low temperatures tend to slow development and thus increase the potential for growth during each phase. Consequently yield potential is often higher in more northerly latitudes where longer daylengths and cooler temperatures more than offset the lower peak light intensities.

Establishment

Germination and seedling establishment, and in the case of winter cereals, survival over winter, determines the plant population in the spring. Germination and emergence require adequate soil moisture and favourable temperatures, but is also affected by factors such as seed quality, seed bed conditions, sowing depth, disease and pests. Winter-kill may result from damage from frost, frost heave, pests, diseases and waterlogging. Poor establishment does not reduce yield significantly if tillering is able to compensate for plant losses.

Leaf emergence and tillering

Tillering commences when the fourth leaf is unfolding and ceases at the start of stem extension (Zadoks GS 31). Tillers arise from buds in the axils of the lower leaves. Each leaf has a tiller bud, but typically only 5-6 buds grow and emerge. Leaf emergence is completed after the flag leaf has unfolded, and GAI for that shoot is at its maximum. From this point onwards, persistence of GAI is crucial in determining the crop growth rate.

Stem extension, ear development and storage reserves

Rapid stem extension occurs between GS 31 and the start of flowering (GS 61), and is completed by the watery ripe stage (GS71). Tiller emergence ceases by GS 31 and between GS 31 and GS 61 a large proportion of emerged tillers die. It is the smaller, later formed tillers, that tend to die first, as they are unable to compete with expanding mainstem leaves for N. The number of tillers surviving to produce ears and the plant population in spring, combine to set the first yield component, the number of ears per square metre.

Stem extension is also a period during which the number of fertile florets is set. Maximum spikelet number is determined by GS 31 and changes little over a wide range of conditions. Each spikelet produces up to 5 florets. Differences in grain number per ear, the second principal yield component, are the result of the abortion of florets. Floret survival and, therefore grain number depends on an adequate supply of assimilates (C and N) during this phase.

Soluble carbohydrates are accumulated in the stem during stem extension and ear emergence, reaching a maximum just after flowering. Later these are remobilized and contribute to grain filling.

Flowering, grain filling and ripening

Wheat varieties are mostly open-flowering. Ineffective pollination is rarely considered to be a cause of poor yield. After fertilization, the initial period of grain development involves rapid cell division. This determines the number of endosperm cells and their complement of large amyloplasts (in which the bulk of the starch will later be deposited). This period lasts approximately 2-3 weeks from the start of anthesis (flowering GS 61) and is important in setting the potential size of the grain. Cell division requires a supply of assimilates; poor assimilate supply during this phase can reduce cell number and limit the potential size of the grain. A period of grain filling follows in which endosperm cells expand; starch is synthesized and deposited in the amyloplasts and protein is synthesized.

Under normal conditions, the major source of photosynthates for the developing grain are the upper green parts in particular the flag leaf and two lower leaves. Ear photosynthesis contributes relatively little in wheat (ca. 10%). Soluble carbohydrates are mobilized from stem reserves at the mid-late grain filling stage and may contribute up to 50% to the final grain fill. The contribution is greatest if the canopy senesces early e.g. in drought conditions.

Most N (e.g. 85%) in grain protein comes from N remobilized from leaves, stems, roots and glumes as they senesce. The remaining 15% is from uptake after flowering. The relative contribution of these different fractions varies greatly with season and N application rates. The root system remains active after flowering and has a high capacity for N uptake. The relationship between yield and % N in the grain is not simple. Grain N concentration is determined by the balance between starch and N deposition, and there is no direct relationship between these processes.

The final stage of grain growth is known as maturation in which the water content gradually declines, and growth slows. Grains stop growing when their moisture content falls to less than 45%. Ripening then involves a period of more rapid drying to 20-15% MC.

1.2 Points contained within the cereal physiology review relevant to disease

- The risk of disease infection is increased as a result of early sowings due to the 'green bridge' (rusts and mildew survive on volunteers from where they can spread into the crop) and warmer temperatures. The risk of common or sharp eyespot and take-all is greater with early sowings.

- The greater yield potential of early sown crops may be jeopardised by high disease risk.

- Thick crops have a microclimate conducive to most foliar diseases (NB. in the light of recent evidence, this view expressed in the cereal physiology review is now under some doubt).

- Soft tissues with high nitrogen content are more sensitive to infection by many diseases.

- Disease reduces the photosynthetic leaf area with an associated increase in senescence and the rate of respiration. This reduces the level of assimilates available to the roots and the developing grain, and tends to depress tillering and the rate of leaf emergence.

- In some situations control of disease at early stem extension can increase plant growth rates when the number of fertile tillers/plant and number of grains/ear are being formed.

- It is considered important to maintain the health of the last-formed three leaves for as long as possible thereby maximizing the amount of assimilate available to fill the grain.

- Crops with adequate moisture reserves are more likely to benefit from late fungicides applied to protect leaves and ears during grain fill.

- Leaf lifespan may be prolonged with fungicides, growth regulators or nutrients, but grain growth may stop whilst leaves are still green.

1.3 Areas requiring further research highlighted in the cereal physiology review

In order to provide a more rational basis for making disease control decisions, several areas requiring a greater knowledge of physiology were identified.

- Direct effects of fungicides on crop growth in the absence of disease.

  Fungicides can have both damaging and stimulatory effects on crop physiology. A better understanding of these would allow improved choice of product in relation to application conditions.

- Monitoring growth and development in relation to disease and fungicide performance, and estimating the consequences of damage by disease.

  Disease control decisions are made on the grounds of disease epidemiology. Less consideration has been given to the way in which disease influences crop function. There is a need to relate disease effects more closely to the size and duration of the crop canopy, and on plant metabolism (e.g. N and C assimilation) and translocation. This would identify the extent to which a canopy can withstand infections without impairing grain formation. Specific pathogens need to be considered as disease organisms differ in their effects.

- Predicting appearance of yield-forming leaves.

  It is suggested that leaf 3 be protected from disease from its emergence, possibly by coinciding this with a fungicide for eyespot control timed at GS 32-33. This *may* mean that only one further fungicide application would be required to provide season long protection. Such a system requires accurate identification of leaf 3 as it emerges and prediction of when it is due to emerge.

Since the publication of the review, progress has been made in these areas. Of particular interest are the advances made in understanding how disease influences crop function and its consequences for yield. These advances are reviewed below.

2. MORE RECENT DEVELOPMENTS IN UNDERSTANDING OF PHYSIOLOGICAL RESPONSES TO FOLIAR DISEASE

2.1 Disease, canopy light interception and yield.

Yield responses to fungicide applications can be high, but disease severity at a particular growth stage is often poorly correlated with yield when crops at different sites and over several seasons are considered (Jenkyn 1984, Wright and Gaunt 1992). For example, Leadbetter and McHale (1987) reported that autumn fungicide applications to winter barley only gave yield increases in Scotland when disease severity exceeded 10%. Other studies have shown significant yield responses at much lower disease severities (Wale 1987). Assessments made at a single growth stage provide no information on how the disease epidemic progressed after assessment.

Estimates of the area under disease progress curves (AUDPC) provide an integrated measure of disease severity over the season. In experiments on yellow rust infected winter wheat there was no clear relationship between yield loss and disease severity on leaves 1-3 (flag leaf = 1) at GS 75. When yield loss was plotted against AUDPC for the same leaves

from GS 33 onwards, a better correlation was found, but the relationship differed markedly between two seasons (Bryson et al. 1995, 1997).
The inability of disease assessments made at single growth stages or as AUDPC to account for variations in yield between crops are because measurements of disease severity are not linked to crop function. Disease severity is usually assessed as the percentage of leaf surface displaying symptoms. No account is made of light interception and radiation use efficiency of the remaining healthy leaves, which contribute to yield formation.

Lim and Gaunt (1986a) reported a closer relationship of yield to green leaf area than to severity of powdery mildew and rust on spring barley. But empirical yield loss-models based on measurements of green leaf area differed between cultivars, sowing dates and seasons. Models combining these different factors explained less of the variation in yield than the individual models (Wright and Gaunt 1992). When the duration of crop growth was included as an estimate of the target yield, fit to the data and the universality of the models was improved.

In the yellow rust experiments of Bryson et al. (1995) a large proportion of the variation in yield response between seasons was explained when yield response was plotted against the area under the green leaf area index progress curves (AUGLAIPC). This provides a measure of the duration of healthy (green area) leaf surface and hence is functionally related to cumulative radiation interception. A further development in the analysis was to estimate the accumulated intercepted radiation by green tissue after GS 39 using the Beer's Law analogy and assuming an extinction coefficient of 0.45. This accounted for slightly more of the total variation in yield response than AUGLAIPC.

By considering radiation interception in terms of canopy structure and light distribution, it is possible to discriminate between canopies having AUGLAIPC values resulting from large GLAI for short duration from those with low GLAI for longer duration. The former will result in lower accumulated radiation interception if the peak GLAI is significantly greater than the optimum.

Thus clear improvements can be made in accounting for the effects of disease epidemics on yield if GLAI (or GAI) duration is measured and its effects on radiation interception determined. A number of techniques are being evaluated for the non-destructive determination of GLAI in crops (Gaunt and Bryson 1995; Bryson et al. 1997).

2.2 Radiation Use Efficiency (RUE)

The efficiency with which light energy intercepted by the canopy is converted into dry matter (conversion efficiency or radiation use efficiency) may be influenced by disease directly through effects on host plant metabolism and canopy architecture, or indirectly through effects on plant water relations.

The effects of biotrophic fungi (which include the rusts and powdery mildews) on host plant metabolism have been studied extensively (Walters 1985; Scholes 1992; Farrar 1995).
The usual response is an increase in respiration rate, a reduction in the rate of photosynthesis and a loss of chlorophyll from the infected leaf (Scholes and Rolfe 1995). However, the dynamics of the response are complex and depend on location within the infected leaf, the susceptibility of the variety, and the type of pathogen. For example, when a tolerant variety of spring barley was inoculated with powdery mildew, the rate of photosynthesis appeared to be increased in symptom-less areas of inoculated leaves immediately following infection. This was succeeded by a slow decrease in photosynthetic rate. A susceptible variety, in contrast, showed no rise in rate and a much faster decline (Kral et al., 1993).

During the more advanced stages of infection by mildew and rusts, islands of green tissue are retained around the fungal mycelium as the rest of the leaf senesces. Green islands of mildewed spring barley leaves exhibited a reduction in the rate of net photosynthesis compared to healthy tissues when expressed on either a unit area (14%) or a chlorophyll basis (32%) and the chlorophyll a:b ratio was substantially reduced. Senescent tissues had no capacity for photosynthesis (Coghlan and Walters 1992). The rate of dark respiration was little affected in green islands or senescent parts compared to healthy tissues (Coghlan and Walters 1992). Similarly no change in respiration rate was found in mildew-infected wheat (Rabbinge et al. 1985), but McAinish et al. (1991) reported an increase in respiration of winter barley leaves infected with powdery mildew.

A non-invasive technique, chlorophyll fluorescence imaging, has recently been used to study spatial patterns of photosynthetic rates during the development of crown rust on oats (Scholes and Rolfe 1995). Photosynthesis was *reduced* in areas of the leaf not invaded by fungal hyphae, though to a lesser extent than invaded tissues. In brown rust pustules, on barley leaves, there was an apparent *increase* in the rate of photosynthesis relative to infected areas of the same leaf (Scholes and Farrar, 1986). However, the effect for the leaf as a whole was a reduction in photosynthetic rate when compared to healthy leaves on a unit area basis. This response of barley brown rust pustules differs to that of rusts of other species (e.g. leek and oats), where a reduction in rate of photosynthesis is usually found within pustules (Scholes and Rolfe 1995).

Several mechanisms have been postulated to account for the observed reductions in photosynthetic rate in diseased leaves. These include altered resistance to diffusion of CO_2 into and within the leaf, damage to chloroplasts, inhibition of the light reactions of photosynthesis, a reduction in activity and amount of Calvin cycle enzymes, removal of Pi from host tissues and repression of photosynthetic gene expression by elevated concentrations of sugar in the tissue (Rabbinge et al. 1985; Scholes 1992). Soluble carbohydrate concentrations (glucose, fructose and sucrose) increased in barley and wheat leaves infected with powdery mildew (Scholes et al. 1994). But in brown rust infected leaves, the amount of sucrose declined, whereas glucose and fructose increased during the early stages of disease development then decreased following sporulation (Owera, et al. 1983; Tetlow and Farrar 1992).

The effects of pathogens on host metabolism depends on the condition of the host. For example, P deficiency decreased the area of oat leaves infected by powdery mildew and delayed the development of the fungus, but photosynthesis was reduced and respiration increased by disease to similar extents in P-deficient and P-sufficient plants (Gunn and Farrar 1995).

Biotrophic fungi represent a significant sink for nutrients. Based on measurements of the nutrient contents of host and fungal cells, Farrar (1995) has calculated that there must be a net influx of nutrients from outside the colony to sustain fungal growth. For carbon this involves movement from non-infected areas of infected leaves. There is little evidence that infected cereal leaves import large amounts of carbon from other leaves. On the contrary they may continue to export photosynthates (Farrar 1995), albeit at a lower rate, as long as the leaves retain some photosynthetic capacity.

Fungal pathogens require substantial quantities of P. Supply of P is probably from direct import in the xylem; export of P in the phloem from infected leaves is negligible compared with healthy leaves (Ahmad et al. 1984). Import of N is increased and export decreased from rusted barley leaves (Ahmad et al. 1982). A greater retention of N has also been found in leaves of barley infected with net blotch (Seidel 1992). Thus cycling of nutrients within the

plant is restricted by disease, which may affect the availability of N for grain filling (but see Carreck and Christian 1991).

A reduction in source:sink ratio, for example through shading or partial defoliation, can sometimes lead to an increase in photosynthetic rate of the remaining leaves (Hay and Walker 1989). The extent to which photosynthetic rates of healthy leaves are influenced by disease elsewhere on the plant is generally not well established. An increase in rate has been demonstrated in healthy leaves in some studies (Williams and Ayres 1981), but others have found no change (Spitters et al. 1990) The magnitude of any effect is likely to depend on the proportion of leaf area infected by disease. If infections are small, any compensatory adjustment in photosynthetic activity of healthy leaves is likely to be limited.

In contrast to the biotrophs, necrotrophic fungi generally have more limited effects on host metabolism. Plant tissues are killed in advance of the pathogen leading to a loss of green area. When necrotic regions are retained on leaves, they continue to intercept light but without contributing to photosynthesis. Fungal lesions may also continue to respire. Necrotrophs may influence light penetration through the canopy if leaf angle is altered through wilting or mechanical weakening.

Foliar pathogens may influence plant water relations in a number of ways depending on the type and severity of infection and the prevailing climatic conditions (Ayres 1978; Ayres and Paul 1986). Stomata of mildew-infected leaves may be immobilized in a partially closed position reducing transpiration rates per unit leaf area (Ayres and Paul 1986). The effects may be largely indirect resulting from an increase in mesophyll CO_2 concentration, itself the consequence of a lower rate of photosynthesis (Rabbinge et al. 1985). Rust infections may initially cause a reduction in stomatal apeture and transpiration, but eventually transpiration is increased when fungal sporulation ruptures the leaf cuticle. In contrast, stomatal apertures may be increased within Rhyncosporium lesions (Ayres 1978). The impact of these responses on plant water balance will depend on the relative effects of disease on leaf area and root length, and the evaporative demand placed on the plant.

The foregoing illustrates the complexity of host leaf responses to pathogens, especially the biotrophs. Whilst a reduction in photosynthetic activity is the usual response, its scale depends on a range of factors including type of pathogen, stage of infection, severity of infection, crop variety, water supply and nutritional status. At the canopy level a reduction in rate of photosynthesis, wasteful light interception (i.e. interception by fungal lesions on leaves overlying healthy leaves) and an increase in respiration of diseased leaves could combine to reduce radiation use efficiency, but their effects might be negated by compensatory increases in photosynthesis by healthy leaves. Furthermore, the impact of diseased leaves on the canopy would be minimal if they were positioned in the lowest leaf layers and shaded by healthy leaves. Relatively few studies have measured the effects of disease on RUE in field crops. Gaunt (1995) suggested that the effects of disease on RUE are negligible. However, most of the supporting evidence comes from work on necrotrophic pathogens and crops other than cereals. Bryson et al. (1997) reported an RUE of 1.41 g DM/MJ of total light intercepted for yellow rust-infected wheat, which is comparable to values for healthy leaves. However, RUE was estimated from the relationship between accumulated light interception and grain yield from GS 61 onwards. A greater contribution of pre-anthesis storage reserves to grain yield in diseased crops (see section 2.4) could have masked possible reductions in current net assimilation per unit of light intercepted.

Further work is needed to establish the effects of disease on RUE over a wider range of growth stages and for a different crop-pathogen combinations in order to develop robust models of disease-yield loss relationships.

2.3 Yield components and the timing of disease epidemics

An analysis of yield components provides information on the key phases in development during which disease may have constrained yield. In wheat, grain development and filling are considered to be at the greatest risk from disease; the yield component most affected under north European conditions being mean grain weight (e.g. Forrer and Zadoks 1983; Wale and Oxley 1992). According to Jordan and Hutcheon (1994) there is no scientific evidence to indicate that foliar disease from stem extension (GS 30) to flag leaf emergence (GS 37) is a constraint to yield of winter wheat. Fungicide applications are timed to coincide with the emergence of the top 3 leaves (GS 32-39) in order to maintain their green area during grain filling. Early application (i.e. during the latent period of infection, but before expression of disease symptoms) gives optimum fungicidal activity (Paveley et al. 1998). Lower leaves are shaded by the top 3 and intercept only a small fraction of the incident radiation. Loss of these leaves to disease will not reduce rates of canopy photosynthesis significantly during the key phase for grain filling.

Although mean grain weight is the yield component of wheat most affected by disease, if disease pressure is high enough at earlier growth stages the other primary yield components, ear number/m^2 and grain number/ear can be reduced (Yang and Xeng 1989; Everts and Leith 1992).

Reductions in the earliest formed yield components are more common in barley. Early and severe infections of powdery mildew on spring and winter barley have been shown to substantially reduce the number of ears and grain size. Less severe or later infections mainly affected grain size (Brooks 1972, Boatman 1992). Similarly, foliar diseases (powdery mildew, brown rust and Rhyncosporium) of winter barley in the spring, reduced fertile tiller numbers and grain weight (Dawson and Hutcheon 1987; Conry and Dunne 1993, Thomson and Wale unpublished results). These and other studies (e.g. Jordan and Stinchcombe 1986) suggest that grain number/ear may be the least sensitive yield component to disease in barley. However, reductions in grain number/ear have been reported in some experiments (e.g. winter barley, Jordan et al. 1982; spring barley, Lim and Gaunt 1986b; Wright and Gaunt 1992) and this component appears to be more sensitive in 6 row than 2 row varieties (Dawson and Hutcheon 1987). The effects on grain number/ear may depend on the timing of the epidemic and the extent of any compensation between yield components. A reduction in tiller production and survival may lead to a greater availability of assimilates for sustaining spikelet/floret survival. However, no evidence of compensation was found between yield components of spring barley (Lim and Gaunt 1986b).

Early epidemics of disease can have persistent effects on GLAI and hence yield formation. A restriction in assimilate supply to leaves during the early stages of their development (prior to emergence of the leaves from their subtending leaf sheaths) can lead to a reduction in the size they ultimately achieve. In this way mildew in spring barley was found to reduce the final size of leaves which emerged *after* the disease had been controlled (Lim and Gaunt 1986a). The effect was only observed with epidemics before GS 31. Those leaves emerging after GS 31 (which includes the top three leaves) were probably sufficiently well developed by this time (GS 31) to be unaffected by reductions in assimilate supply resulting from later (i.e. GS 31 onwards) disease epidemics. In contrast to mildew, no persistent effect was observed with brown rust. This was ascribed to the later development of rust epidemics, rather than any intrinsic difference in effect of the disease, although no data were presented to rule out the latter possibility (Lim and Gaunt 1986a). Similar persistent effects on leaf size have been observed with early infections of net blotch on winter barley (Jordan et al. 1985).

Early disease epidemics can also increase the sensitivity to later infections. Late rust infections (after GS 75) reduced the yield of spring barley only in crops which had

experienced earlier infections of mildew or rust (Lim and Gaunt 1986b). This suggests that the capacity for grain fill was the major constraint to yield in crops kept disease-free during the early stages of development, and not the rate of photosynthesis i.e. yield was sink rather than source-limited. Early disease epidemics appeared to reduce the capacity of the source, so that grain fill became susceptible to reductions in photosynthetic activity by late infections.

Early epidemics may also place constraints on yield by influencing the sensitivity of the crop to other limiting factors such as water availability. Glasshouse and controlled environment studies have shown that disease can substantially reduce root biomass and length (Last 1962; Vizarova and Minarcic 1974; Jordan et al. 1985; Mayfield and Clare 1991). This is understandable since root extension and branching are highly dependent on the supply of carbohydrate from the shoot (Bingham et al. 1996, 1997). There have been relatively few investigations of the effects of foliar pathogens on root growth in field crops, but they are likely to have a profound influence on the architecture of the root system and its distribution through the soil profile. In turn, this is likely to impair the ability of crops to acquire water and nutrients, which could reduce RUE and promote early senescence of leaves. Factors such as these may explain why early fungicide applications (e.g. autumn and GS 30) sometimes give yield responses in winter barley.

2.4 Storage reserves

Assimilates for grain filling come from two sources: storage carbohydrates and current photosynthesis. Storage reserves are considered to be an important mechanism for buffering grain-filling from adverse effects on current photosynthetic activity. The contribution from storage was increased during periods of late season drought (Austin et al. 1980). This has led to recommendations that varieties of wheat with a propensity to accumulate large reserves be grown in drought-prone areas (Foulkes et al. 1998).

Little is known about the effects of disease on the relative contributions of stored and current assimilates to grain-filling. Carver and Griffiths (1981) proposed that mildew reduced the yield of spring barley through a reduction in the amount of stored carbohydrate. Soluble carbohydrate contents were reduced by mildew in proportion to the reduction in green leaf area pre-anthesis (Carver and Griffiths 1982). Inoculation of winter barley plants (grown in hydroponics) with net blotch at GS 31 reduced ear number and mean grain weight (Jordan et al. 1985). As no disease was found on the top three leaves in this treatment, it was suggested that disease at GS 31 may have impaired grain-filling by reducing the amount of stored carbohydrate available for translocation. Disease reduced the storage carbohydrate content of spring barley (Gaunt and Bryson 1995).

However, it must be recognized that measurements of stem storage reserves alone provide no information on their contribution to grain filling. Mobilized reserves may be utilized in other sinks, of which the pathogen may be one, or they may be respired (Schnyder 1993). Direct estimates using isotope-labelling techniques and growth analysis are required (Gaunt and Wright 1992; Schnyder 1993). Using these techniques, disease of spring barley was found to reduce the quantity of carbohydrates stored, whilst the amount used for grain-filling was, in many cases, increased (Gaunt and Wright 1992). However, the effect of disease depended on the time of epidemic development, its duration, and the yield potential of the crop.

The relative contribution of storage reserves seems to depend on the demand for carbohydrate during grain filling, the ability of current photosynthesis to meet the demand and the amount of stored carbohydrate. Consequently, crops most at risk from disease during grain filling are likely to be those with high yield potential and/or low stem reserves (Gaunt and Wright 1992) and where photosynthetic capacity is not greatly in excess of demand. The balance between these different components will be influenced by previous disease (section 2.3) and other environmental factors such as available radiation and water supply.

Experiments in which the crop is shaded at key developmental stages have been used to investigate the sensitivity of yield formation to current photosynthetic activity. From these, deductions have been made concerning source versus sink-limitation of yield.

In wheat, tiller survival is relatively insensitive to reductions in photosynthetic activity during early stem extension (GS 31 - GS 39); the main effect of a low rate of photosynthesis is to restrict the deposition of storage carbohydrates (Scott and Sylvester-Bradley 1998). Reductions in photosynthesis during ear emergence limit the development and fertility of the florets and thus reduce the number of grains per ear, whilst shading during grain filling can reduce mean grain weight (Mimbé and Lafarge 1995; Scott and Sylvester-Bradley 1998).

Few studies have compared the response of wheat and barley to shading in crops grown under the same conditions. Experiments on spring varieties suggest that the species may differ (Willey and Holliday 1971a, b). In spring wheat, yield was most sensitive to shading from ear initiation to anthesis and from anthesis to maturity, where reductions in grain number per ear and mean grain weight occurred respectively (Willey and Holliday 1971b). In contrast, spring barley was most sensitive to shading from ear initiation to anthesis and, to a lesser extent, from establishment to ear initiation; the yield components most affected were grain number per ear and ear number per m^2. Shading had only a small influence on mean grain weight, even when shading was imposed during the period of grain filling (Willey and Holliday 1971a). Thus, when grown under the same conditions, barley, unlike wheat, was able to meet the demands of the grain when rates of photosynthesis were reduced by shading, possibly because its sink capacity was lower and/or its storage reserves were larger.

However, the results of shading experiments are variable. Some studies on spring barley have found large reductions in mean grain weight in response to shading during grain filling (Grashoff and d'Antuono 1997). Such variability is not surprising given that both sink and source capacity during grain filling are a function of the previous history of the plant (Mimbé and Lafarge 1995). Few data are available for winter barley (but see Bonnett and Incoll 1993a, b).

3. DIFFERENCES BETWEEN WINTER BARLEY AND WINTER WHEAT IN TERMS OF GROWTH, DEVELOPMENT, PHYSIOLOGY AND DISEASE INFECTIONS

3.1 Differences in growth, development and physiology.

- The sowing date of winter barley is earlier than winter wheat, and all aspects of husbandry are carried out earlier. Establishment is earlier, tiller and spikelet production is well underway before winter, nitrogen top-dressing and the start of spring growth is earlier, and harvest is complete before the wheat crop is ripe.

- Tiller production is often greater in barley giving denser crop canopies, but will depend on plant density and variety.

- The size of the penultimate three leaves is smaller in barley than in wheat, and the flag leaf in barley is often very small.

- Winter barley has a lower vernalisation requirement than winter wheat (evidence from NIAB variety list for last date of sowing).

- Spikelet and grain production in barley is different from wheat. Grain production is also different between 2-row and 6-row barley varieties.

- Barley varieties are awned whereas wheat varieties are usually awnless. The awns are capable of photosynthesis and there is evidence that the amount of photosynthesis carried out by awns is important in drought conditions. However, the importance of awns under other conditions, including disease, is not understood.

- There has been little work done on root growth in field crops of either wheat or barley. Root growth in wheat is not substantial before winter and, therefore, uptake of nitrogen during this period is limited. Root growth in winter barley occurs earlier and is more extensive in early spring than wheat sown at the same density and on the same date.

- Lodging is more common in winter barley than wheat. There is a lack of information as to the relative importance of stem height, stem-base strength and root lodging as a result of poor anchorage in the soil.

- Shading experiments suggest that source-sink relationships may differ between wheat and barley (spring varieties; section 2.4).

3.2 Differences in disease infection and fungicide use.

- The main diseases of winter barley are different from those found in wheat. The main foliar pathogens in barley are: powdery mildew (*Erysiphe graminis*), *Rhynchosporium* leaf blotch (*Rhynchosporium secalis*), net blotch (*Pyrenophora teres*) and brown rust (*Puccinia hordei*); the main stem-base and root diseases are: take-all (*Gaeumannomyces graminis*), common eyespot (*Pseudocercosporella herpotrichoides*), sharp eyespot (*Rhizoctonia cerealis*), *Typhula* snow rot (*Typhula incarnata*) and *Fusarium* brown foot rot (*Fusarium culmorum*); the main ear and seed diseases are: loose smut (*Ustilago nuda*), barley leaf stripe (*Pyrenophora graminea*) fusarium ear blight (*Fusarium* spp) and ergot (*Claviceps purpurea*); and the three viral diseases are: barley yellow dwarf virus, barley yellow mosiac virus and barley mild mosaic virus.

- The barley diseases, *Rhynchosporium* leaf blotch, net blotch, *Typhula* snow rot and barley leaf stripe do not infect wheat. Mildew and rust pathogens have forma speciales (f.sp.) largely specific to the cereal species.

- The wheat diseases, *Septoria* leaf blotch (*Mycosphaerella graminicola* anamorph *Septoria tritici*), glume blotch (*Stagnospora nodorum* anamorph *Septoria nodorum*), yellow rust (*Puccinia striiformis*) and stinking bunt (*Tilletia caries*) do not infect barley.

- The relative impact of these diseases may be different: common eyespot, sharp eyespot and take-all can be considered greater problems on wheat,

- As winter barley is sown earlier, it is susceptible to foliar disease earlier than wheat. Early-sown crops of winter barley often receive either a seed treatment effective against foliar disease or autumn fungicide application.

- Nitrogen top-dressing is applied earlier to winter barley than wheat. The resultant earlier growth of barley in the spring means that the crop becomes susceptible to disease attack earlier than wheat.

- Greater tiller densities in barley compared to wheat may increase the humidity of the crop microclimate giving rise to conditions more favourable to disease development.

- The ear of wheat is more susceptible to disease than in barley.

- Key fungicide timings for winter wheat and winter barley differ. In order of priority (yield response) they are: Wheat; GS 39, GS 32, GS 59 plus on occasion earlier control if severe epidemics of powdery mildew and yellow rust threaten. Barley; GS 31/32, GS 39-49, plus on occasion an earlier application at the end of tillering/GS 30; winter barley sometimes needs autumn disease control.

4. OTHER RELEVANT WORK ON FOLIAR DISEASES OF WINTER BARLEY

An extensive search of the literature has revealed a lack of information relating to the physiology of diseased winter barley crops. Much of the relevant literature has been reviewed in section 2 along with that for other cereals (wheat and spring barley). Summarized below are a few additional papers relating specifically to winter barley that are of interest.

- White and Jenkyn (1995) studied the production of individual leaves, and the development of powdery mildew on them, in winter barley crops sown on different dates. Leaves formed later in the developmental sequence were more resistant to infection than those formed early. Since early sowing increases the total number of leaves produced on the stem, the flag leaf and penultimate leaf of these crops were more resistant to disease in the spring than those of late sown crops. In autumn and winter, disease severity was *greater* on early sown crops, mainly because of the greater inoculum level, but also because prior to vernalization disease susceptibility was greater.

- Hims and Gladders (1994) reported that fungicide programmes initiated at GS 30/31and continued up to GS 55, were essential to avoid yield loss of winter barley crops. Mildew and brown rust, or net blotch and brown rust, were the main diseases depending on the season. It was estimated that each 1% green leaf area on the top 3 leaves at GS 77 contributed 0.59-0.84% to yield.

- Jordan et al. (1989) investigated the effects of timing of N applications on yield responses to fungicide. Crop structure and the severity of foliar diseases were altered by delaying the main top dressing of N from mid-March to mid-April. A single application of fungicide was then sufficient to give good control of disease and maximum yield response.

- Development of powdery mildew on winter barley was promoted by water–logged soils, sheltered locations, early plant emergence and N application (Kluge 1991)

- The occurrence of mildew, brown rust, net blotch and Rhynchosporium leaf blotch was reduced in mixtures of cultivars with varying levels of resistance to foliar diseases, compared to single cultivar monocultures. The photosynthetically active leaf area was greater, and the tolerance to late frost improved, in the mixtures (Beer 1994).

- Yield increases were also reported by Newton et al. (1997) in mixtures of winter barley containing up to six components, compared with the mean of their monoculture components. The benefits were partly attributable to the increased control of Rhynchosporium leaf blotch with increasing number of components in the mixture.

- Kehlenbeck et al. (1994) investigated the effects of an inducer of resistance produced by *Bacillus subtilis* on field grown winter barley infected with powdery mildew. Plants treated with the inducer produced higher yields than could be accounted for by the observed reduction in mildew infection. Flag leaves of infected inducer-treated leaves had higher assimilation rates, greater sucrose concentrations, and translocated more assimilates to

the ear, than untreated mildewed leaves. It is suggested that mildew colonies on inducer-treated leaves were less able to accumulate sucrose, than those on untreated leaves.

- In winter barley crops, dead leaves infected with mildew, Rhynchosporium leaf blotch and brown rust had a greater N concentration than those that had been treated with fungicide (Carreck and Christian 1991). This suggests that disease impaired the plant's ability to recover N from leaves during senescence. It was argued that this generally does not result in a low grain N concentration, because during the early stages of grain filling, the grain is a strong sink for N and N may be removed from younger green tissue if insufficient is made available from older diseased leaves.

5. SUMMARY OF RESULTS FROM THE WINTER BARLEY PHYSIOLOGY STUDY (Accumulation of Baseline Physiology Data in Contrasting Disease Situations)

Two important findings have emerged from the pilot study conducted on winter barley physiology at SAC Aberdeen and ADAS Rosemaund. Firstly, at each of the sites, a single linear regression was able to describe well the relationship between accumulated light interception and dry matter increase for all disease control treatments. This indicates that the reduction in crop growth resulting from disease infection can be explained by the effects of disease on GLAI and hence light interception. The slope of the regression was significantly greater for Rosemaund compared to Aberdeen, suggesting that RUE was greater at Rosemaund. The results are consistent with reports for the effects of foliar disease on wheat (Bryson et al. 1995, 1997) and imply that disease control strategies for winter barley should, like wheat, be based on maintaining GLAI at the optimum for light interception.

The second important finding was that the crops at Rosemaund and Aberdeen appeared to differ in their degree of response to a given unit of disease. At Rosemaund, disease levels on untreated plants were slight, but they resulted in a reduction of more than 2 units of GLAI compared to plants given a three spray fungicide programme. At Aberdeen, disease infection was more severe, but it resulted in a loss of only 1.25 units of GLAI. There were no data to indicate the cause of the difference in response between these crops. However, in order to predict the likely need for disease control, it will be necessary to establish what factors influence the response of a canopy to a given level of disease.

6. PROPOSED AREAS OF RESEARCH

It is clear from the preceding sections that considerable advances have been made in recent years in understanding the effects of disease on yield. By considering crop growth as a function of radiation interception by healthy leaves and RUE, much of the variation in yield loss between crops can be accounted for. It is also recognized that the ability to withstand the effects of disease can differ between crops. Ideally fungicides should be applied only to crops that are most likely to benefit from disease control. Decisions should be based on a consideration of the potential impact of disease on yield, the risk of disease spread, and the cost of control. The first two factors are not fixed, but depend on the condition of the crop, the type of pathogen and the prevailing weather. Disease control strategies, therefore, need to be flexible and adjusted according to these factors. Potential savings in the cost of control can be made by applying doses appropriate to the risk, or by using fewer, but better timed applications. Disease control strategies for wheat based on an assessment of crop function are currently being developed. Whilst the same general approach may be used in winter barley, the details of the strategy will not apply. Wheat and barley differ in a number of important respects, which will influence their response to disease (section 3).

- The type of diseases affecting wheat and barley, and thus their rate of spread and impact on yield, differ.

- Barley is at greater risk from early disease epidemics. In spring barley early disease has persistent effects on the size of the canopy and on root growth, which may constrain yield after the disease has been controlled (section 2.3). This is likely to be the case for winter barley also.

- Growth and development of wheat and barley differs (e.g. structure of the canopy and apical development), which may influence their ability to withstand the effects of disease during particular growth stages (section 2.3).

There is a general lack of information on the physiology of winter barley crops. Several key areas in which a better understanding would facilitate a more accurate assessment of the potential impact of disease are discussed below.

6.1 Benchmarks for winter barley crops

In any production system, management must set targets, assess progress, adjust inputs and monitor success. These basic principles are outlined in the Wheat Growth Guide. Canopy management is founded on the definition of an optimum GAI for the canopy and a series of growth benchmarks or targets to be met by certain developmental stages if high yields are to be achieved. The progress of a given crop can then be assessed against these benchmarks and subsequent inputs adjusted accordingly.

The same management principles apply to winter barley, but the benchmark values are likely to differ and need to be defined for representative varieties. For example, the optimum GLAI for spring barley has been found to differ from that of wheat (Hoad, personal communication). The most useful benchmarks are:

1. Shoot number in autumn and spring
2. GLAI (or total GAI) at key developmental stages
3. Optimum GLAI or GAI (i.e. GLAI/GAI giving 90-95% light interception)

These benchmarks will provide values against which the condition of a crop can be assessed. In turn, an assessment of the condition of the crop will help predict the likely impact of a disease epidemic on yield (section 6.4).

6.2 Identifying which structures contribute most to yield.

Winter wheat disease control is based upon an understanding of which structures contribute most to yield and thus require the greatest protection (Paveley et al. 1998). Equivalent information for winter barley is lacking.

Research is needed to determine the relative contribution of individual leaves, stem storage reserves and ear photosynthesis to yield under different conditions. Their contribution will vary according to the developmental stage of the crop and the yield component being formed at the time. The importance of the different structures to tiller survival, spikelet development and survival, and grain development and filling, should be determined. This will establish, not only those structures which must be protected from disease, but also the periods during which protection should be provided.

The relative yield responses found to different fungicide timings will help guide this research. The critical timings for fungicide application are earlier in winter barley than wheat (section 3.2). For example, empirical studies have shown that application of fungicide at GS 31/32 gives the most consistent yield response in winter barley. The physiological basis for the apparent sensitivity to disease at this particular growth stage is not known and requires investigation. There are several possible reasons.

1. Tiller survival in barley may be particularly sensitive to reductions in assimilate supply during early stem extension. If there is little capacity for compensatory increases in later formed yield components, loss of tillers represents an irreversible reduction in yield potential.

2. The upper leaves of barley, especially the flag leaf, are smaller than those of wheat. This may mean there is a greater reliance on pre-anthesis stem reserves for grain filling in barley, and that fungicide applications at GS 31/2 are needed to protect leaves during the early phases of reserve deposition. Leaf 5, and often leaf 4, have emerged by GS 31. Alternatively, the smaller flag leaf of barley may allow a greater penetration of light into the canopy, so that the lower leaves contribute more to canopy photosynthesis during grain-filling. It may be for this reason that they require the protection provided by early fungicide treatment. However, it should be noted that in general leaf 5, and sometimes leaf 4, senesce *before* anthesis. On the other hand, a relatively small leaf area may be perfectly adequate for grain filling if ear photosynthesis is significant.

3. The requirement for early fungicide application may be simply to prevent inoculum build up which may impair yield forming processes at later growth stages.

In contrast to application of fungicides at GS 31/32, treatment at GS 39-49 or tillering/GS 30 tend to give less consistent yield responses (section 3.2). A greater understanding of the physiology underlying the relationship between the period of disease infection and yield loss, will help identify those conditions in which early or late disease control may be worthwhile.

6.3 Management to maximize the ability of crops to withstand the effects of disease

It is recognized that in some crops yield losses are lower for a given unit of disease. A greater understanding of the physiological basis of this would allow management strategies to be developed which reduce the impact of disease on the crop. Theoretically, the impact of disease will be minimized when the capacity of the source to supply assimilates exceeds the demands of the yield forming sites (in order of development; tillers, spikelets/florets, grains) and other competing sinks. Stem carbohydrate stores act as both a sink and a source, depending on the stage of development.

Two candidate traits associated with an ability to withstand disease have been studied in wheat; they are canopy size and stem storage reserves. In barley, the situation may be more complex because earlier formed yield components (i.e. ear number/m^2) are sensitive to disease (section 2.3).

Crops with a dense canopy (e.g. above the optimum GLAI) will suffer a smaller reduction in light interception for a given loss of GLAI through disease if the infected leaves are low in the canopy (section 2.1). However, the benefits of managing a crop with a large GLAI to reduce disease impact must be weighed against the costs of establishing the canopy and the greater risk of lodging and disease spread. Mixtures of cultivars with differing levels of disease resistance (e.g. Newton et al. 1997) may be a way of minimizing disease spread in large canopies created to reduce disease impact. Research is required to quantify the risks and benefits of manipulating canopy size to improve the ability of crops to withstand disease.

In addition, it may be possible to manage the impact of disease through selection of appropriate varieties. Suitable traits in barley might include.

- *Tiller survival.* Varieties which have a propensity to retain a greater proportion of their tillers may be better able to withstand the effects of disease between GS 31-39 (section 6.2). This may allow fungicide doses to be reduced at this growth stage, or enable a single application to be used at a later growth stage to protect ear emergence and grain filling.

- *Canopy structure.* Varieties differ in the size of their flag leaves, thereby altering light distribution within the canopy. A larger flag leaf may reduce the impact on the canopy of disease on the lower leaves.

- *Storage reserves.* Varieties with a greater propensity for accumulating storage carbohydrates may be better able to withstand disease during grain filling, thus requiring less persistent fungicides. However, any benefit may be lost if: 1) deposition of storage reserves competes strongly with tillers and spikelets for available assimilates as this may lead to greater tiller and spikelet mortality; 2) after deposition, reserves are not remobilized effectively during grain filling. Evidence with wheat suggests these may not be major concerns.

- *Ear photosynthesis.* Varieties with a greater capacity for ear photosynthesis may also be affected less by disease present during grain filling, providing the disease does not infect the ear itself. This could lead to savings in late fungicide use.

Research is needed to investigate the relationship between these varietal traits and the response to given levels of disease and reductions in GLAI, over different sites and seasons.

Results from the study to accumulate baseline physiology data in contrasting disease situations (section 5) have shown that the loss of GLAI for a given amount of disease infection can differ widely between crops. The underlying cause is not known. It may be related to varietal differences in thresholds for leaf senescence, or an interaction between the effects of disease and other factors on GLAI, such as soil moisture availability or the nutritional status of the crop. It is essential to understand the physiological basis for these differences in crop response to disease if we are to predict, with accuracy, the need for fungicide treatment.

6.4 Assessing the potential impact of disease from measurements of crop condition

The impact of disease may be influenced by events occurring earlier in the growth of the crop, for example, the incidence of pests or periods of adverse weather. These factors may influence the ability of a crop to withstand disease by altering the relative capacity of sources and sinks. It would be useful to be able assess the condition of the crop and relate this to the potential impact of disease *during the course of the season*, so that the disease control strategy can be adjusted accordingly.

Research is needed to identify reliable predictors of the ability of the crop to withstand disease at given crop growth stages. These must be based on some assessment of photosynthetic capacity versus sink demand. From empirical studies, key growth stages for assessment are likely to be between GS 30 and GS 49. Systems for measuring GLAI in the field have been developed (Bryson et al. 1997). Sink demand might be assessed by counts of tiller and spikelet numbers. Storage carbohydrate content will be more difficult to assess,

but the possibility of developing rapid field analyses should be investigated. Alternatively, estimates might be derived indirectly from a knowledge of canopy photosynthesis and crop growth.

6.5 Summary of Research Requirements

- Definition of benchmarks for winter barley growth through the season, particularly GLAI or GAI (including optimum GLAI or GAI) and shoot numbers.

- Quantification of the contribution different structures make to yield, with emphasis on the role of individual leaves, stem reserves and ear photosynthesis, during different stages of development. This should encompass an investigation into the physiological basis of the relationship between the period of disease infection and yield loss.

- Investigation of the potential for maximizing the ability of crops to withstand the effects of disease through:

  a) the manipulation of canopy size and the use of variety mixtures with contrasting disease resistance

  b) the identification and selection of appropriate varietal traits.

- Research into the physiological basis for differences between crops in loss of GLAI in response to a unit level of disease.

- Development of a system to assess the potential impact of disease on crop function during the season through in-field measurements of photosynthetic and sink capacity.

REFERENCES CITED

ANON. (1997). *The Wheat Growth Guide.* Home Grown Cereals Authority 32pp.

AHMAD, I, OWERA, S A P, FARRAR, J F & WHITBREAD, R (1982). The distribution of five major nutrient in barley plants infected with brown rust. *Physiological Plant Pathology* **21**, 335-336.

AHMAD, I, FARRAR, J F & WHITBREAD, R (1984). Fluxes of phosphorus in leaves of barley infected with brown rust. *New Phytologist* **98**, 361-375.

AUSTIN, R B, MORGAN, C L , FORD, M A & BLACKWELL R D (1980). Contributions to grain yields from pre-anthesis assimilation in tall and dwarf barley phenotypes in two contrasting seasons. *Annals of Botany* **45**, 309-319.

AYRES, P G (1978). Water relations of diseased plants *In: Water Deficits and Plant Growth*, TT Kozlowski (ed) Vol 5, pp 1-60. Academic Press, London.

AYRES, P G & PAUL, N D (1986). Foliar pathogens alter the water relations of their hosts with consequences for both host and pathogen *In: Water, Fungi and Plants*, P G Ayres & L Boddy (eds) pp 267-285. Cambridge University Press, Cambridge.

BINGHAM, I J, PANICO, A & STEVENSON, E A (1996). Extension rate and respiratory activity in the growth zone of wheat roots: time-course for adjustments after defoliation. *Physiologia Plantarum* **98**, 201-209.

BINGHAM, I J, BLACKWOOD, J M & STEVENSON, E A (1997). Site, scale and time-course for adjustments in lateral root initiation in wheat following changes in C and N supply. *Annals of Botany* **80**, 97-106.

BEER, E (1994). Sowing mixtures of cultivars of winter barley, an element of integrated control. *Gesunde-Pflanzen* **46**, 242-247.

BOATMAN, N D (1992). Effects of herbicide use, fungicide use and position in the field on the yield and yield components of spring barley. *Journal of Agricultural Science, Cambridge*, **118**, 17-28.

BONNETT, G D & INCOLL, L D (1993a). Effects on the stem of winter barley of manipulating the source and sink during grain-filling. I. Changes in accumulation and loss of mass from internodes. *Journal of Experimental Botany* **44**, 75-82.

BONNETT, G D & INCOLL, L D (1993b). Effects on the stem of winter barley of manipulating the source and sink during grain-filling. II. Changes in the composition of water soluble carbohydrates of internodes. *Journal of Experimental Botany* **44**, 83-91.

BROOKS, D H (1972). Observations on the effects of mildew *Erysiphe graminis*, on growth of spring and winter barley. *Annals of Applied Biology*, **70**, 149-156.

BRYSON, R J, PAVELEY, N D, CLARK, W S, SYLVESTER-BRADLEY, R & SCOTT, R K (1997). Use of in-field measurements of green leaf area and incident radiation to estimate the effects of yellow rust epidemics on the yield of winter wheat. *European Journal of Agronomy* **7**, 53-62.

BRYSON, R J, SYLVESTER-BRADLEY, R, SCOTT, R K, & PAVELEY, N D (1995). Reconciling the effects of yellow rust on yields of winter wheat through measurement of green leaf area and radiation interception. *Aspects of Applied Biology*, **42**, *Physiological responses of plants to pathogens*, 9-18.

CARRECK, N L & CHRISTIAN, D G (1991). Studies on the pattern of nitrogen uptake and translocation to the grain of winter barley intended for malting. *Annals of Applied Biology*, **119**, 549-559.

CARVER, T L W & GRIFFITHS, E (1981). Relationship between powdery mildew infection, green leaf area and grain yield of barley. *Annals of Applied Biology* **99**, 255-266.

CARVER, T L W & GRIFFITHS, E (1982). Effects of barley mildew on green leaf area and grain yield in field and glasshouse experiments. *Annals of Applied Biology* **101**, 561-572.

COGHLAN, S E & WALTERS, D R (1992). Photosynthesis in green-islands on powdery mildew-infected barley leaves. *Physiological and Molecular Plant Pathology*, **40**, 31-38.

CONRY, M J & DUNNE, B (1993). Effects of nitrogen timing and frequency of fungicide applications on grain yields of winter barley in Ireland. *Journal of Agricultural Science, Cambridge*, **120**, 181-186.

DAWSON, K P & HUTCHEON, S (1987). The evaluation of growing programmes for winter barley in Scotland with special reference to crop protection. *Proceedings Crop Protection in Northern Britain 1987*, 73-78.

EVERTS, K L & LEATH, S (1992). Effect of early season powdery mildew on development, survival, and yield contribution of tillers of winter wheat. *Phytopathology*, **82**, 1273-1278.

FARRAR, J F (1995). Just another sink? Sources of assimilate fo foliar pathogens. *Aspects of Applied Biology*, **42**, *Physiological responses of plants to pathogens*, 81-89.

FORRER, H R & ZADOKS, J C (1983). Yield reduction in wheat in relation to leaf necrosis caused by *Septoria tritici*. *Netherlands Journal of Plant Pathology*, **89**, 87-98.

FOULKES, M J, SCOTT, R K, CLARE, R W, SPINK, J H & SYLVESTER-BRADLEY, R (1998). Varietal responses to drought, rotational position and nitrogen with implications for variety exploitation. *In: Management Through Understanding Research Into Practice* pp 3/4.1-3/4.17. Proceedings of the sixth HGCA R & D conference on cereals and oilseeds, Cambridge.

GAUNT, R E & WRIGHT, A C (1992). Disease-yield relationship in barley. II. Contribution of stored stem reserves to grain filling. *Plant Pathology*, **41**: 688-701.

GAUNT, R E (1995). The relationship between plant disease severity and yield. *Annual Review of Phytopathology* **33**, 119-144.

GAUNT, R E & BRYSON, R J (1995). Plant and crop yield potential and response to disease. *Aspects of Applied Biology*, **42**, *Physiological responses of plants to pathogens*, 1-7

GRASHOFF, C & d'ANTUONO, L F (1997). Effect of shading and nitrogen application on yield, grain size distribution and concentrations of nitrogen and water soluble carbohydrates in malting barley (*Hordeum vulgare* L.). *European Journal of Agronomy*, **6**, 275-293.

GUNN, S & FARRAR, J F (1995). Source leaf metabolism of oat infected with powdery mildew: effects of phosphate supply. *Aspects of Applied Biology*, **42**, *Physiological responses of plants to pathogens*, 101-108.

HAY, R K M & WALKER, A J (1989). *An Introduction to the Physiology of Crop Yield.* Longman Scientific and Technical, Harlow.

HIMMS, M J & GLADDERS, P (1994). Use of fungicide-manipulated epidemics to determine critical periods for disease control and yield response in winter barley. *Proceedings of British Crop Protection Conference - Pests and Diseases, 1994*, 283-288.

JENKYN, J F (1984). Effects of powdery mildew on grain filling in spring barley in contrasting environments. *Annals of Applied Biology*, **105**, 195-212.

JORDAN, V W L & HUTCHEON, J A (1994). Strategies for optimal fungicide use in less-intensive cereal growing systems. *Proceedings of Brighton Crop Protection Conference - Pests and Diseases - 1994*, 687-694.

JORDAN, V W L & STINCHCOMBE, G R (1986). Interactions between fungicide, plant growth regulator, nitrogen fertiliser applications, foliar disease and yield of winter barley. *Annals of Applied Biology*, **108**, 151-165.

JORDAN, V W L, BEST, G R & ALLEN, E C (1985). Effects of *Pyrenophora teres* on dry matter production and yield components of winter barley. *Plant Pathology* **34**, 200-206.

JORDAN, V W L, STINCHCOMBE, G R & HUTCHEON, J A (1989). Fungicide and nitrogen applications in relation to the improvement of disease control and yield in winter barley. *Plant Pathology*, **38**, 26-34.

JORDAN, V.W.L., TARR, H.S. & MILES, D.M. (1982). Effects of fungicide timing on control of .*Rhynchosporium secalis* in barley plants. *Annals of Applied Biology*, **100**, 305-314.

KEHLENBECK, H, KRONE, C, OERKE, E C & SCHONBECK, F (1994). The effectiveness of induced resistance on yield of mildewed barley. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz* **101**, 11-21.

KLUGE E (1991). Effect of agronomic and cultural factors on the development of mildew in winter barley. *Archiv für Phytopathologie und Pflanzenschutz* **27**, 143-151.

KRAL, G, OERKE, E C & SCHONBECK, F (1993). Effect of powdery mildew the water balance and photosynthesis of spring barley cultivars differing in tolerance. *Zeitschrift fur Pflanzenkrankheiten und Pflanzenschutz*, **100**, 1-10.

LAST, F T (1962). Analysis of the effects of *Erysiphe graminis* DC on the growth of barley. *Annals of Botany* **26**, 279-289.

LEADBEATER, A.J. & McHALE, T. (1987). The effects of fungicide timing on yield responses in winter barley in Northern Britain. *Proceedings Crop Protection in Northern Britain 1987*, 67-72.

LIM, L G & GAUNT, R E (1986a). The effect of powdery mildew (*Erysiphe graminis* f. sp. *hordei*) and leaf rust (*Puccinia hordei*) on spring barley in New Zealand. I. Epidemic development, green leaf area and yield. *Plant Pathology*, **35**: 44-53.

LIM, L G & GAUNT, R E (1986b). The effect of powdery mildew (*Erysiphe graminis* f. sp. *hordei*) and leaf rust (*Puccinia hordei*) on spring barley in New Zealand. II. Apical development and yield potential. *Plant Pathology*, **35**: 54-60.

McAINSH, M R, AYRES, P G & HETHERINGTON, A M (1991). The effects of infection by powdery mildew (*Erysiphe graminis* f.sp. *hordei*) and low temperature on the respiratory activity of winter barley. *Physiological and Molecular Plant Pathology*, **39**, 13-24.

MAYFIELD, A H & CLARE, B G (1991). Effects of *Rhynchosporium secalis* at specific growth stages on barley grown in a controlled environment. *Plant Pathology*, **40**, 561-567.

MIMBÉ, J N & LAFARGE, M (1995). Nitrogen and reduced irradiance effects on ear fertility in winter wheat. *European Journal of Agronomy* **4**, 135-142.

NEWTON, A C, ELLIS, R P, HACKETT, C A & GUY, D C (1997). The effect of component number on *Rhynchosporium secalis* infection and yield in mixtures of winter barley cultivars. *Plant Pathology*, **45**, 930-938.

OWERA, S A P, FARRAR, J F & WHITBREAD, R (1983). Translocation from leaves of barley infected with brown rust. *New Phytologist* **94**, 111-123.

PAVELEY, N D, STEVENS, D B, HIMS, M & AINSLEY, A E (1998). Canopy, disease and fungicide use. *In: Management Through Understanding Research Into Practice* pp 9.1-9.17. Proceedings of the sixth HGCA R & D conference on cereals and oilseeds, Cambridge.

RABBINGE, R, JORRITSMA, I T M & SCHANS, J (1985). Damage components of powdery mildew in winter wheat. *Netherlands Journal of Plant Pathology*, **91**, 235-247.

SCHOLES, J D (1992). Photosynthesis: cellular and tissue aspects in diseased leaves. *In: Pests and Pathogens*. P Ayres (ed) pp 85-105. BIOS Scientific Publishers Limited, Oxford.

SCHOLES, J D & FARRAR, J F (1986). Increased rates of photosynthesis in localized regions of a barley leaf infected with brown rust. *New Phytologist*, **104**, 601-612.

SCHOLES, J D & ROLFE, S A (1995). How do biotrophic pathogens affect the photosynthetic metabolism of their hosts? *Aspects of Applied Biology*, **42**, *Physiological responses of plants to pathogens*, 91-99.

SCHOLES, J D, LEE, P J, HORTON, P & LEWIS, D H (1994). Invertase: understanding changes in the photosynthetic and carbohydrate metabolism of barley leaves infected with powdery mildew. *New Phytologist*, **126**, 213-222.

SCHNYDER, H (1993). The role of carbohydrate storage and redistribution in the source-sink relations of wheat and barley during grain filling - a review. *New Phytologist* **123**, 233-245.

SCOTT, R K & SYLVESTER-BRADLEY R (1998). Crop physiology, assessment and management. *In: Management Through Understanding Research Into Practice* pp 2.1-2.14. Proceedings of the sixth HGCA R & D conference on cereals and oilseeds, Cambridge.

SEIDEL, P (1992). Distribution, host range, taxonomy and genetics of *Drechslera teres* (Sacc.) Shoemaker, the cause of net blotch disease of barley. *Archives of Phytopathology and Plant Protection*, **28**, 29-38.

SPITTERS, C J T, Van ROERMUND, H J W, Van NASSAU, H G M G, SCHEPERS, J & MESDAG, J (1990). Genetic variation in partial resistance to leaf rust in winter wheat: disease progress, foliage senescence and yield reduction. *Netherlands Journal of Plant Pathology*, **96**, 3-15.

SYLVESTER-BRADLEY, R & SCOTT, R K (1990). *Physiology in the Production and Improvement of Cereals*. HGCA Research Review No. 18. pp 155.

TETLOW, I J & FARRAR, J F (1992). Sucrose-metabolizing enzymes from leaves of barley infected with brown rust (*Puccinia hordei* Otth.). *New Phytologist*, **120**, 475-480.

VIZÁROVÁ, G & MINARCIC, P (1974). The influence of powdery mildew upon the cytokinins and the morphology of barley roots. *Phytopathologia Zeitschrift* **81**, 49-55.
WALE, S J (1987). Effect of fungicide timing on yield of winter barley in Northern Scotland. *Proceedings Crop Protection in Northern Britain 1987*, 61-66.
WALE, S J & OXLEY, S (1992). An evaluation of the potential of reduced dose fungicide programmes in winter wheat. *Proceedings of Brighton Crop Protection Conference - Pests and Diseases* 603-608.
WALTERS, D R (1985). Shoot:root interrelationships: the effects of obligately biotrophic fungal pathogens. *Biological Reviews* **60**, 47-79.
WHITE, N & JENKYN, J F (1995). Effects of sowing date and vernalisation on the growth of winter barley and its resistance to powdery mildew (*Erysiphe graminis* f.sp. *hordei*). *Annals of Applied Biology*, **126**, 269-283.
WILLIAMS, G M & AYRES, P G (1981). Effects of powdery mildew and water stress on CO_2 exchange in uninfected leaves of barley. Plant Physiology **68**, 527-530.
WILLEY, R W & HOLLIDAY, R (1971a). Plant population and shading studies in barley. *Journal of Agricultural Science, Cambridge*, **77**, 445-452.
WILLEY, R W & HOLLIDAY, R (1971b). Plant population and shading studies in wheat. *Journal of Agricultural Science, Cambridge*, **77**, 453-461.
WRIGHT A C & GAUNT, R E (1992). Disease-yield relationship in barley. I. Yield, dry matter accumulation and yield-loss models. *Plant Pathology*, **41**: 676-687.
YANG, X B & XENG, S M (1989). Effect of yellow rust on yield components of winter wheat in China. *Plant Pathology*, **38**, 1-8.

Bibliography of some additional references consulted but not cited

BAINBRIDGE, A, FINNEY, M E & JENKYN, J F (1980). The influence of cultural factors and mildew (*Erysiphe graminis*) on yield of winter barley. *Journal of Agricultural Science, Cambridge*, **94**, 389-397.
COOK, R J, POLLEY, R W & THOMAS, M R (1991). Disease-induced losses in winter wheat in England and Wales 1985-1989. *Crop Protection*, **10**, 504-508.
DAVIS, H & FITT, B D L (1992). Seasonal changes in primary and secondary inoculum during epidemics of leaf blotch (*Rhynchosporium secalis*) on winter barley. *Annals of Applied, Biology* **121**, 39-49.
ELABIDINE, F Z, REINHOLD, M & SCHAREN, A L (1992). Effect of early powdery mildew infection on barley under simulated drought stress. *Cereal Rusts and Powdery Mildews Bulletin*, **20**, 1-6
EVANS, S A & HOUGH, A (1984). The effects of several husbandry factors on the growth and yield of winter-sown barley at four sites in each of three years. *Journal of Agricultural Science, Cambridge*, **103**, 555-560.
GLADDERS, P & HIMMS, M J (1994). Improving spring and summer fungicide treatments for winter barley. *Crop Protection*, **13**, 597-606.
GREEN, C F, FURMSTON, D T & IVINS, J D (1985). Time of sowing and the yield of winter barley. *Journal of Agricultural Science, Cambridge*, **104**, 405-411.
GUTTERIDGE, R J, HORNBY, D, HOLLINS, T W & PREW, R D (1993). Take-all in autumn-sown wheat, barley, triticale and rye grown with high and low inputs. *Plant Pathology*, **42**, 425-431.
JENKYN, J F, CARTER, N, DARBY, R J, GUTTERIDGE, R J, MULLEN, L A, PLUMB, R T, ROSS, G J S, TODD, A D, WIDDOWSON, F V & WOOD, D W (1992). Effects of seven factors on the growth and yield of winter barley grown as a third consecutive take-all susceptible crop and of growing barley after oats or a fallow. *Journal of Agricultural Science, Cambridge*, **113**, 303-333.

JENKYN, J F, GUTTERIDGE, R J & TODD, A D (1991). Effects of fungicides, applied in autumn, and a growth regulator, applied in spring, on the growth and yield of winter barley grown in contrasting soil types. *Journal of Agricultural Science, Cambridge*, **117**, 287-297.

JENKYN, J F, GUTTERIDGE, R J & TODD, A D (1992). Effects of sowing winter barley on different dates in autumn on the severity of take-all (*Gaeumannomyces graminis* var. *tritici*) in those and the subsequent crops. *Journal of Agricultural Science, Cambridge*, **119**, 19-25.

JENKYN, J F, GUTTERIDGE, R J & TODD, A D (1995). Effects of incorporating straw, using different cultivation systems, and of burning it, on diseases of winter barley. *Journal of Agricultural Science, Cambridge*, **124**, 195-204.

KIRBY, E J M & APPLEYARD, M (1984). *Cereal Development Guide*, 2nd edn. National Agricultural Centre (Arable Unit), Kenilworth, Warwickshire.

KIRBY, E J M, APPLEYARD, M & FELLOWES, G (1985). Variation in development of wheat and barley in response to sowing date and variety. *Journal of Agricultural Science, Cambridge*, **104**, 383-396.

OERKE, E C & SCHONBECK, F (1990). Effect of nitrogen and powdery mildew on the yield formation of two winter barley cultivars. *Journal of Phytopathology*, **130**, 89-104.

POLLEY, R W, KING, J E & JENKINS, J E E (1993). Surveys of diseases of spring barley in England and Wales, 1976-1980. *Annals of Applied Biology*, **123**, 271-285.

POLLEY, R W, THOMAS, M R, SLOUGH, J E & BRADSHAW, N J (1993). Surveys of diseases of winter barley in England and Wales, 1981-1991. *Annals of Applied Biology*, **123**, 287-307.

SCHMIDT, G, BOYLE, C, AUST, H J & WAGNER, K G (1994). Adenosine/adenine-containing nucleotides in the leaves and roots of young barley plants after infection of the primary leaf by *Erysiphe graminis*. *Journal of Phytopathology*, **141**, 402-414.

TOTTMAN, D.R. (1987). The decimal code for the growth stages of cereals, with illustrations. *Annals of Applied Biology*, **110**, 441-454.

ZADOKS, J C, CHANG, T T & KONZAK, C F (1974). A decimal code for the growth stages of cereals. *Weed Research*, **14**, 415-421.

PART 3
EVALUATION OF FUNGICIDE DOSE AND VARIETY RESPONSE CURVES

S J WALE & F MURRAY

SAC, CROPS DIVISION, FERGUSON BUILDING, CRAIBSTONE ESTATE, BUCKSBURN, ABERDEEN AB21 9YA

SUMMARY

From data obtained in 20 field trials, fungicide dose response curves were determined for a wide range of fungicides against the four main diseases of winter barley (mildew, Rhynchosporium, brown rust and net blotch) using standard methodology. Fungicides were evaluated at five doses (0, 0.25, 0.5, 0.75, 1.0) in a replicated split plot design. One fungicide, Tilt + 0.75 Aura, was used throughout as a standard fungicide. Exponential curves of disease against fungicide dose were fitted to individual and combined data to determine curve parameters for each fungicide in protectant and curative situations for each disease. The curves presented permit relative efficacy and cost effectiveness to be determined for the first time.
In 16 trials using the standard fungicide, the interaction of disease resistance and fungicide dose was evaluated using standard methodology. For each of the four diseases, data from the trials was combined and exponential curves of disease against resistance rating were fitted for each of five fungicide doses. From the curves presented, described as variety response curves, it is possible for the first time to determine the extent to which fungicide dose can be adjusted in relation to disease resistance.

INTRODUCTION

There are almost as many opinions on the 'right' way to grow cereals as there are growers and consultants.

This diversity of opinion exists because of the large number of variable inputs that influence the unit cost of cereal production, the complexity of their interactions and the difficulty of quantifying the effect of changing any **one** of these variables, within the farm system.

One variable input that has a substantial effect on the economic efficiency of production, is the use of fungicides to control foliar diseases. Griffin (1994) reported that fungicides applied to the UK winter cereals crop in 1993 cost the industry in excess of £100m, but prevented losses estimated at £400m. 37 % of the total winter cereal acreage of 2.4 m hectares grown in 1993 was winter barley. More recent survey data suggest that potential losses fluctuate with season, but the fungicide spend remains substantial. Getting disease control 'right' is clearly important.

Growers and consultants use experience to make judgements about fungicide applications. This experience, often accumulated over many years, is a valuable commodity. Nevertheless, consistently good decisions seem more likely where experience is backed up by research information which quantifies responses to changes to individual components of the production system.

This report summarises the results of five years trials of two of the three experiments which comprised the project investigating winter barley appropriate fungicide doses. Experiment 1 investigated fungicide dose response curves and experiment 2, which examined the interaction of fungicide dose and host disease resistance. It supersedes the earlier reports on these two experiments. The third experiment which examined the complex interactions of appropriate fungicide dose programmes has already been reported (Anon.,1998)

Experiment 1. Fungicide dose response curves

The shape of a fungicide dose response curve is influenced by site, season and disease pressure and thus differs from trial to trial. The reduction in disease with increasing dose follows an exponential decrease and thus, for each individual assessment, an exponential curve can be fitted to the data (Paveley *et al*, 1998). Since the untreated (nil fungicide) is the point from which the effect of fungicide on disease control is judged it is not unreasonable to constrain curves through this point. This is only valid, however, when this anchor point is a solid one and confidence can be placed in it. In these trials the untreated disease value was a mean of nine replicates.

Exponential curves have the form

$$y = a + b.\, e^{kx}$$

where y = disease
a = value of lower asymptote
(i.e. disease level at infinite fungicide dose where graph levels out)
b = difference between lower asymptote and untreated
Thus a + b = untreated disease value
x = fungicide dose
k = a value that reflects the shape of the curve

This three parameter function was used to describe the variation in disease response seen in the data. Replicate values for disease were used to determine dose response curves rather than the means derived from replicate values.

For simplicity of fitting the equation was converted to

$$y=a+br^x$$

where $\ln(r) = k$

Where a and b values are large for individual assessment data, they illustrate the amount of constraint required to apply an exponential function to the data.

The objectives of experiment one were:

To determine dose response curves for the principal barley fungicides for economically important winter barley diseases in a range of situations.

To estimate the effect of dose manipulation on curative and protectant properties and persistence of the principal barley fungicides.

Interpretation of dose response curves

In assessing disease levels in trials, although every endeavour is made to minimise variation, both the natural variation of disease within a sample and the subjective nature of disease assessment mean that lines joining the data points of different doses do not always follow a smooth decline. Exponential curves fitted to the data and constrained through the origin do give a smooth decline. This is shown in Fig. 1. where data points for one assessment are plotted together with their exponential curves. Where the scatter of the data points around the curve is small the fit is a good one. By combining assessments the fit becomes increasingly better and more confidence can be placed in the fitted curves.

Clearly there are differences between fungicides in their efficacy. Opus gave a large reduction in disease with just a quarter dose. Thereafter each increment of dose had a diminishing effect. By comparison, the curves of the other triazoles were less steep over the first quarter dose and the maximum control of disease (i.e. the lower asymptote) poorer Parameter estimates of the exponential curves are shown in Table 1.

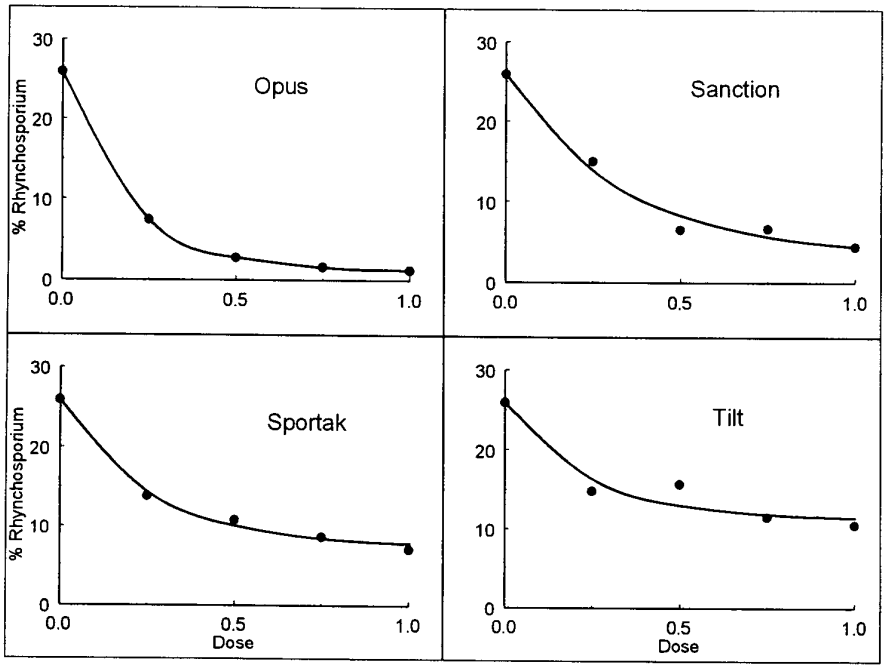
Table 1. Parameter estimates of exponential curves for four triazole fungicides at one assessment. Rhynchosporium, Newquay 1996, leaf 2, 45 days after application.

	Exponential curve parameters			
Fungicide	a	b	a+b	k
Opus	1.16	24.73	25.89	-5.50
Sanction	3.33	22.56	25.89	-3.30
Sportak	7.55	18.34	25.89	-3.95
Tilt	11.26	14.63	25.89	-4.21

The values of 'a' indicate the lower asymptote, the lowest disease achieved by the fungicide if the dose kept on increasing. Full doses of each of the fungicides gave disease control that approached this lowest value.

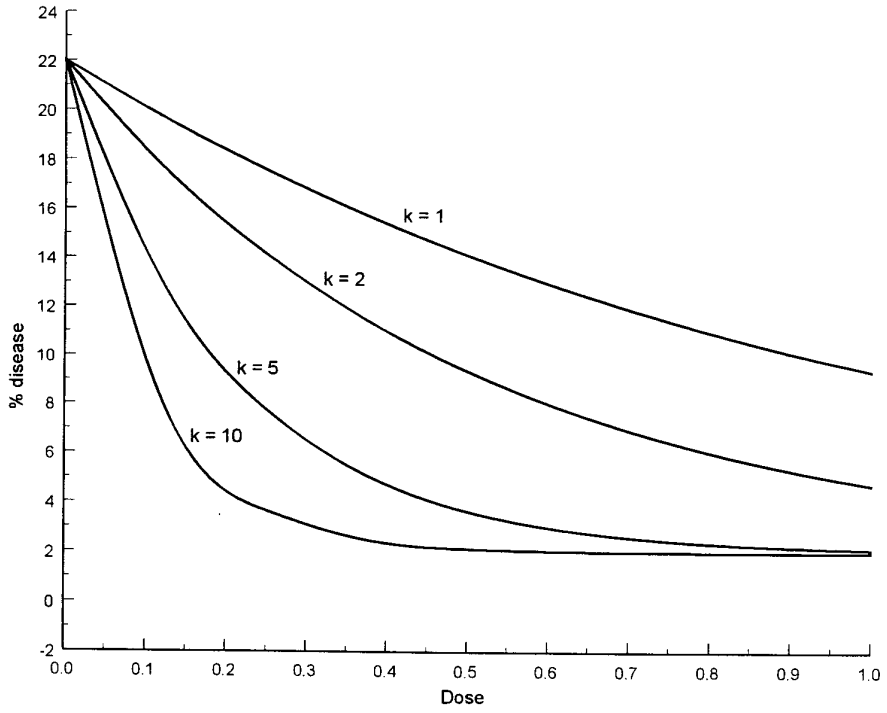
The optimum fungicide dose to achieve maximum disease reduction will vary according to the shape of the curve, that is according to the k value. Fig. 2. shows how the shape of the curve changes with different values of k. As the value of k rises the initial decline becomes steeper and the lower asymptote more rapidly reached. In the example shown in Fig. 1., Opus had the steepest curve and this is evident from the values of k in Table 2.

Fig. 1. Examples of fitted exponential dose response curves for four triazole fungicides at one assessment. Rhynchosporium, Newquay 1996, leaf 2, 45 days after application.



In the same way that the effect of dose on disease can be explained using exponential equations, increases in yield and quality parameters (specific weight and thousand grain weight) with increasing dose can also be described by exponential equations. In these cases, the shape of the curves are the inverse of that for disease. These have been calculated for each individual fungicide at each site. However, because at most sites there was more than one disease present and their relative severity differed from site to site, cross site analysis for individual fungicides has not been attempted.

Fig. 2. Theoretical dose response curves showing how the shape of the curve varies with the value of k



Experiment 2. Variety x fungicide dose interaction

Experiment two set out to examine the effect of host (partial) resistance on disease development and the degree by which fungicide dose could be adjusted when the resistance rating was taken into account.

How resistance ratings are determined

Variety resistance ratings as published in the Recommended List are determined from

a) untreated plots of naturally infected variety trials
b) inoculated tests at NIAB, Cambridge

A large number of winter barley variety trials are carried out each year and the disease on untreated plots is measured at GS 31/2 and between GS 60-80. Data are used only from trials where infection on any variety exceeds 5%.

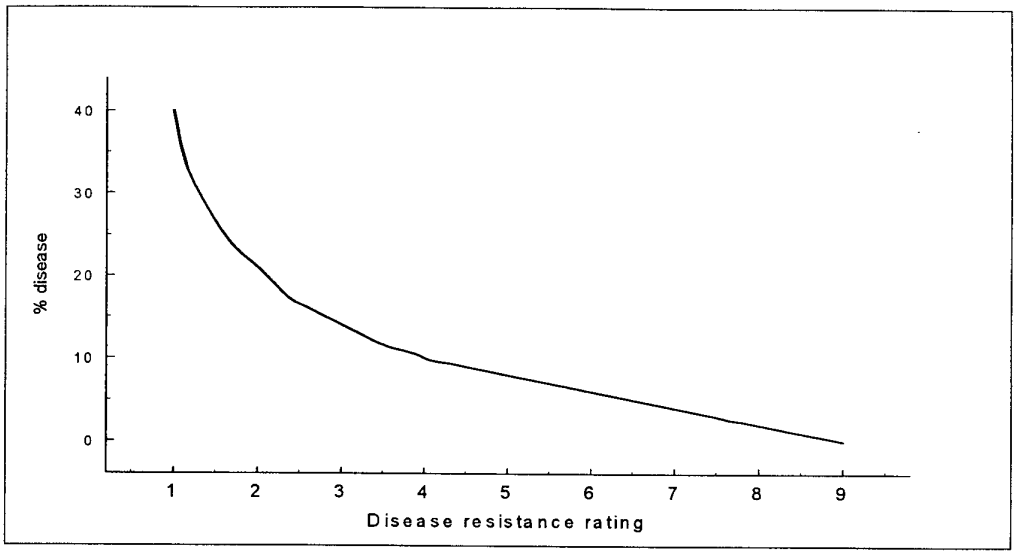
In the inoculated tests, new or prevalent races of pathogens are used to determine the susceptibility of varieties. In determining resistance ratings account is taken of these inoculated tests. Their influence on the rating is least where a disease occurs frequently in naturally infected variety trials and most where disease occurrence is sporadic.

Variety disease resistance ratings are calculated using data from field trials and inoculated tests from the previous three years.

The data for each disease from variety trials and inoculated tests is adjusted relative to control varieties and the adjusted mean % disease converted to a resistance rating using standard tables. Fig. 3 shows the relationship between percentage foliar disease and resistance rating used to determine the rating for the Recommended Cereal Variety List.

The relationship between resistance rating and percentage disease is a direct one for ratings of 4 and above. However, the percentage disease is progressively greater as the resistance declines below 4.

Fig. 3. Relationship between percentage foliar disease and disease resistance rating



Applying five fungicide doses to varieties with different disease resistance ratings, the degree of control achieved by increments of dose at each rating can be determined. Figure 4, the results of a single trial, illustrates that as the resistance increases the optimum dose to achieve adequate control decreases. The parameter estimates for these curves are shown in Table 2.

Fig. 4. Examples of fitted exponential dose response curves for three varieties at one assessment. Rhynchosporium, Aberdeen 1996, leaf 2, 44 days after application.
(Values in brackets after the variety name indicate the Rhynchosporium resistance rating)

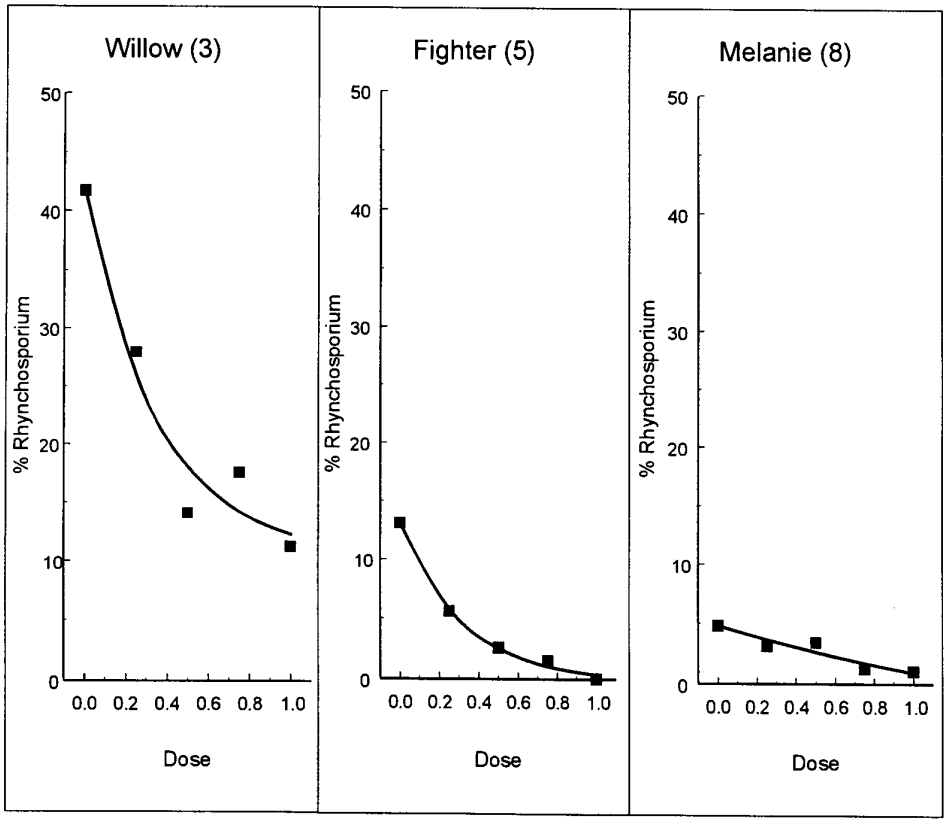


Table 2. Parameter estimates of exponential curves for three varieties at one assessment. Rhynchosporium, Aberdeen 1996, leaf 2, 44 days after application.

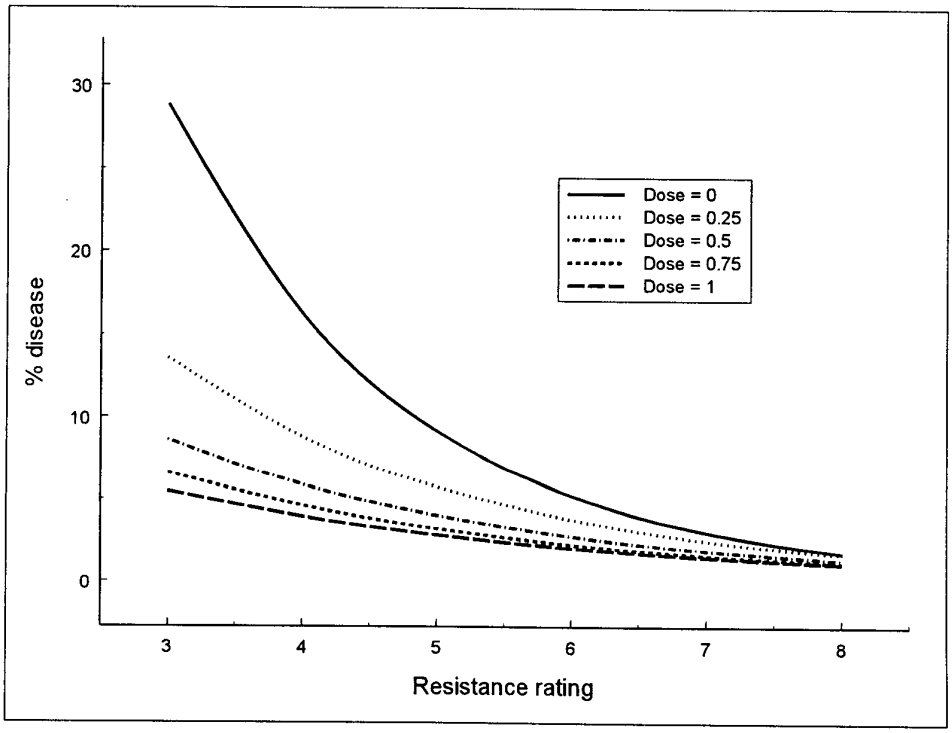
	Exponential curve parameters			
Variety	a	b	a+b	k
Willow	10.38	31.39	41.77	-0.69917
Fighter	-0.163	13.248	13.085	-0.79429
Melanie	-5.2	10	4.8	-0.12217

By using disease resistance rating as the independent variable, exponential disease resistance response curves can be constructed for different fungicide doses that have the same properties as fungicide dose response curves. Such curves can be described as 'variety response curves'. Fig. 5 shows theoretical variety response curves for five doses. In contrast to dose response curves for different fungicides, however, the difference in k values with variety response curves is not usually large. For each of the four principal foliar diseases of winter barley, this experiment set out to determine real values for these curves.

Interpretation of response curves for a single disease at individual sites for experiment 2 have to bear in mind

a) disease pressure present at a site
b) pathogen virulence at a site
c) interaction or competition between pathogens

Fig. 5. Theoretical curves of the effect of disease resistance rating and fungicide dose on infection by winter barley diseases



To produce variety response curves in which confidence can be placed, data from a number of trials are combined . In these multiple assessment variety response curves

y = disease
a = value of lower asymptote (i.e. disease level at infinite resistance rating)
b = difference between lower asymptote and value at minimum resistance rating
Thus a + b = disease value at minimum resistance rating
x = disease resistance rating
k = a value that reflects the shape of the curve

The objectives of experiment two were:

To determine the relationship between host partial resistance and disease development and its effect on yield.

To measure differences in responses of varieties to varying doses of fungicide in a range of disease situations.

To measure the difference in fungicide dose required to compensate for varietal difference in host resistance against the major foliar diseases.

In order to cross link experiments, the fungicide Tilt + ¾ Aura used in this experiment was also used in Experiment 3 and was one of the 8 fungicides evaluated in each trial of Experiment 1.

MATERIALS AND METHODS

Experiment 1. Fungicide dose response curves

Field trials were laid out with three replicates in a split plot design with fungicide as main plots and dose as sub-plots. Plots were drilled at a seed rate appropriate to the variety and local conditions. Plots were either 3m wide, drilled with an Accord seed drill, or 2m wide, drilled with an Øyjord plot drill, with or without guard plots between treatment plots.

Variety at a site was chosen according to it's susceptibility to one of the four target diseases, mildew, Rhynchosporium, brown rust and net blotch. In each case, the variety was chosen from those used in experiment 2, the examination of variety x fungicide dose interaction. A single purpose seed dressing such as Cerevax extra, Rappor plus or Panoctine plus was used in each trial.

Eight widely used or novel fungicides were chosen each year at each site, according to the target disease (Tables 3 and 4). Each fungicide was applied at each of five doses: nil, 0.25, 0.5, 0.75 and manufacturer's full recommended dose. The fungicide mixture Tilt + ¾ Aura/Corbel, was included as one fungicide treatment at all sites and in each season to allow comparison with results obtained in different years and at different sites. Treatments were made at the target timing of GS 31/2 using either an Oxford Precision or AZO sprayer in a single application with water volume of 200-300 l/ha. The timing at sites targeted at net blotch tended to be at GS32-39.

Applications of seedbed fertiliser, fertiliser top dressings and micro-nutrients were made according to standard farm practice for the locality. Herbicides were chosen by site managers as appropriate to the weed flora present. Overall applications of growth regulators were made if the risk of lodging justified their use, and insecticides were used in areas of BYDV risk or where ear aphid infestation reached threshold levels.

Site and husbandry details were recorded for each trial. These are available in previous annual reports.

Disease assessments were carried out on all green leaf layers (i.e. where green leaf area was above 50% of total leaf area on untreated plants) of 25 randomly selected plants from across the trial area at the time of spraying and on ten tillers from each plot at around 3 and 5-6 weeks after application. Leaf layers were tagged on ten indicator plants across trials to allow identification of final leaf layers.

Plots were yielded and a sample of grain taken for determination of dry matter, specific weight and thousand grain weight. Yield and thousand grain weight were adjusted to 15% moisture content.

Initial analysis of data was made by ANOVA using GENSTAT 5. A few extreme outliers were removed from the data sets after consultation with site managers as to the cause. In a few cases residuals were reduced by the use of the plot number as covariate, but in a majority of cases there was no benefit. Transformations of the data may also have been suitable in a small number of cases, but were also rejected as being inappropriate to the majority of the data. Leaving the data untransformed had the advantage of allowing direct comparison of results between sites and seasons.

Disease data were used for the production of dose response curves in this report only in cases where disease reached 5% or higher leaf area infection in untreated plots. Since diseases may interact or compete with one another if present together on a leaf, preference was given to data obtained where one disease showed considerable dominance over others, to reduce the possibility of confutable conclusions being drawn.

Each disease assessment was assigned to either protectant or curative fungicide action according to the following criteria:

Protectant - no disease evident at the time of spraying and no possibility of the leaf carrying, as yet, latent disease. i.e. leaf unemerged at the time of spraying; or if the leaf was emerged, the leaf layer below had no disease present and / or dispersal of inoculum to the leaf was considered not to have occurred.

Curative - leaf showed disease at time of spraying, or if no obvious disease present, then lower leaf layers showed disease and inoculum likely to have spread to and latently infected the assessed leaf.

Table 4. Products, active ingredients and commercial product dose of fungicides evaluated .

Fungicide treatment	Active ingredient	A.I. concentration (g/litre)	Full recommended dose
Amistar	azoxystrobin	250	1.0 l/ha
Aura / Corbel	fenpropimorph	750	1.0 l/ha
Calixin	tridemorph	750	0.7 l/ha
Ensign	kresoxim methyl + fenpropimorph	150 + 300	0.7 l/ha
Folicur	tebuconazole	250	1.0 l/ha
Fortress	quinoxyfen	500	0.3 l/ha
Opus	epoxiconazole	125	1.0 l/ha
Opus + ¾Aura	expoxiconazole+ fenpropimorph	125 + 750	1.0 l/ha + 0.75 l/ha
Patrol / Tern	fenpropidin	750	1.0 l/ha
Sanction	flusilazole	400	0.4 l/ha
Sportak 45	prochloraz	450	0.9 l/ha
Tilt	propiconazole	250	0.5 l/ha
Tilt + ¾ Aura	propiconazole + fenpropimorph	250 + 750	0.5 l/ha + 0.75 l/ha
Tilt + Bavistin	propiconazole + carbendazim	250 + 250	0.5 l/ha + 0.5 l/ha
Tilt + Calixin	propiconazole + tridemorph	250 + 750	0.5 l/ha + 0.7 l/ha
Tilt + Unix	propiconazole + cyprodinil	250 + 75% w/w	0.5 l/ha + 0.67 kg/ha
Unix	cyprodinil	75% w/w	0.67 kg /ha
Untreated	-	-	-

Data were included in the production of combined dose-response curves only where disease criteria were met as described above. Where the criteria were not met or data showed illogical variations an assessment was not included. Where two assessments were made on the same leaf layer at different times after application, because the results would be related only one assessment was taken when combined dose-response curves were calculated.

Disease assessments were combined to produce overall dose response curves. However, because the fungicides evaluated were not present in every trial and varied markedly in the number of times tested, to standardise the data for combined dose response curves, the curve for each fungicide was related to the response curve of Tilt + 3/4 Aura tested in the same trials.

Experiment 2. Variety x Fungicide dose interaction

Trials followed the same format as for experiment one in respect of seed rate, seed treatment, plot size, fungicide application and timing, husbandry, disease assessment, yield and quality measurements. They were laid out with three replicates in a split plot design with varieties as main plots and fungicide dose as sub-plots.

In the first four years, six varieties were chosen with a spectrum of resistance ratings to the four main foliar diseases so that within the six there was a spread of ratings for each disease. Apart from Torrent

the varieties were all on the UK recommended list during the course of the project. Torrent was substituted in years 2, 3 and 4 by Melanie. In the final year, the six varieties were chosen for a range of resistance to net blotch primarily .

The varieties grown and their resistance ratings are shown in Table 5.

Statistical analysis followed the same methodology as for experiment 1. Cross assessment analyses determined the exponential curve of disease against resistance rating for each fungicide dose. These are variety or resistance response curves. For individual assessments the resistance ratings used were those published in the NIAB recommended list for the year following the trial. This figure was used since the rating in the following year is based on disease in the preceding three years and is likely to more accurately reflect the resistance rating of the variety in the previous season. Multiple assessment analyses were carried out using individual assessments that met the disease criteria described for experiment one

Response curves were examined only where a disease on any one variety exceeded 5% on a leaf layer. It is assumed that where disease levels were lower than this a low fungicide dose would be able to control disease satisfactorily.

The spectrum of pathogen virulence was not determined at any site for any disease. It is possible that inconsistencies in the expected levels of disease relative to the disease resistance rating might be attributable to the presence (or absence) of pathogen virulences that can overcome (or not) the host resistance. However, in general, the relationship of disease to resistance rating on untreated plots followed the expected pattern.

Table 3. Summary of site, variety, target disease, and fungicides applied in each year

Site no.	1					2			3				4			5			7	8
Year	'94	'95	'96	'97	'98	'94	'95	'96	'94	'95	'96	'97	'94	'95	'96	'94	'95	'96	'94	'98
Variety	1	1	1	1	3	3	3	3	7	4	4	3	7	4	4	2	2	2	1	3
Target Disease	M	M	M	M	NB	NB	NB	NB	BR	BR	BR	NB	BR	BR	BR	Rh	Rh	Rh	M	NB
Amistar					✓							✓								✓
Aura / Corbel	✓	✓	✓	✓		✓	✓	✓	✓	✓	✓		✓	✓	✓	✓	✓	✓	✓	
Calixin	✓	✓	✓																✓	
Ensign				✓	✓							✓								✓
Folicur						✓	✓	✓	✓	✓	✓		✓	✓	✓	✓	✓			
Fortress				✓																
Opus		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓		✓
Opus Team				✓								✓								
Patrol / Tern	✓	✓							✓	✓	✓		✓	✓	✓				✓	
Sanction	✓	✓	✓		✓	✓	✓	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Sportak 45	✓				✓	✓	✓	✓	✓	✓		✓	✓	✓		✓	✓	✓	✓	✓
Tilt	✓	✓	✓			✓	✓	✓	✓	✓	✓		✓	✓	✓	✓	✓	✓	✓	
Tilt + ¾ Aura	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Tilt + Bavistin																✓	✓	✓		
Tilt + Calixin	✓	✓				✓	✓									✓			✓	
Tilt + Unix			✓	✓	✓						✓				✓			✓		✓
Unix			✓	✓	✓			✓				✓								✓

Site	
1	Aberdeen
2	Morley, Norfolk
3	Rosemaund, Herefordshire
4	Shute, Devon
5	Newquay / Mitchell, Cornwall
7	Blackiemuir, Kincardineshire
8	Starcross, Devon

Variety	
1	Pastoral
2	Willow
3	Puffin
4	Melanie
7	Torrent

Disease	
M	Mildew: *Erysiphe graminis*
Rh	*Rhynchosporium secalis*
BR	Brown rust: *Puccinia hordei*
NB	Net blotch: *Pyrenophora teres*

Table 5 : Varieties used in experiment two and their published disease resistance ratings

	Disease resistance rating																							
	Mildew						Rhynchosporium						Net Blotch						Brown rust					
Variety	94	95	96	97	98	99	94	95	96	97	98	99	94	95	96	97	98	99	94	95	96	97	98	99
Pastoral+	3	3	3	3			7	7	7	7			8	8	8	8			6	6	6	6		
Fighter+	8	8	8	8			6	6	6	6			8	8	8	7			7	7	7	6		
Willow+	8	8	(8)	(8)			4	3	(3)	(3)			8	8	(8)	(8)			8	8	(8)	(8)		
Torrent *	6	6					8	8					7	7					4	4				
Melanie**		6	5	5				8	8	8				6	6	5				4	4	4		
Puffin	5	5	5	6	6	(6)	7	7	6	6	6	(6)	6	5	5	4	4	(4)	8	8	8	8	8	(8)
Intro+	6	6	6	6			6	5	5	5			8	8	8	8			8	8	8	8		
Fanfare***					5	5					8	8					8	7					7	7
Gleam***					6	6					7	7					4	4					7	6
Hanna***					4	4					8	8					7	7					7	5
Manitou***					5	5					8	8					4	4					5	4
Sunrise***					(5)	(5)					(7)	(7)					(5)	(5)					(8)	(8)

Years in which varieties were grown:

+ 1994, 1995, 1996, 1997
* 1994 only
** 1995, 1996, 1997 only
*** 1998 only
Puffin was grown in all five years
( ) Variety was no longer on recommended list. The rating given was the last recorded.

RESULTS AND DISCUSSION

Experiment 1. Fungicide dose response curves

The combined fungicide dose response curves for protectant and curative situations for three diseases, Rhynchosporium, mildew and brown rust are shown in Figs. 6 to 15. Data for net blotch was limited and extremely erratic. In consequence no attempt was made to produce smoothed dose response curves and results are not presented here. For Rhynchosporium and mildew, data was accumulated from trials over five years.

Two sets of results are presented for Rhynchosporium and mildew. In the first set (Figs. 6, 8, 10 & 12) the overall dose response curve for a test fungicide (solid line) is shown against that for Tilt + 0.75 Aura (dotted line) in the same trials. The table following these figures give the pairs of curve parameters including the coefficient of determination (R^2). The data comprising the paired response curves utilise to the maximum the comparison between the standard fungicide (Tilt + 0.75 Aura) and the test fungicide. The pairs of curves are derived from different sets of individual data and thus the disease level of the untreated varies from pair to pair. Comparison between test fungicide can be determined by the control it gives relative to the standard.

A second set of results are presented (Figs. 7, 9, 11 & 13) which show selected data for three or more fungicides analysed together. Again, the tables following these figures give the curve parameters. These sets of results are derived from the same individual data sets and thus initial disease of the untreated is the same for each fungicide.

The data for brown rust (Figs. 14 & 15) and their associated tables comes from data accumulated over the first three years of the project.

With effective fungicides the greatest percentage control was achieved by the first quarter dose. Thereafter, increasing dose gave diminishing degree of further control. As the assessments of disease in the trials took place 3-5 weeks after spraying and often the results came from high disease pressure situations, it is clear that for effective fungicides a low dose can achieve a surprising degree of control. What needs to be decided by the person applying the fungicide is whether the degree of control afforded by a low dose is sufficient to achieve maximum profit. In few of the data sets shown in the figures below does the full dose reach 100% control. Consequently, single applications are rarely sufficient to achieve complete control.

In low disease pressure situations low doses (e.g. 0.25) were usually sufficient for near complete control. This was true even with relatively ineffective fungicides. Often in low disease pressure situations, disease at the time of application is absent or at very low levels and the fungicide is acting protectively. Most fungicides are more effective, dose for dose, in a protectant situation. Difficulty may come for certain diseases, however, in knowing whether infection has taken place. This is particularly true of Rhynchosporium and net blotch where relatively long latent infection periods exist (c. 120 (range 88 - 200) and 75 (range 40 - 100) day^{o}C respectively, J Cooper, personal communication).

The shape of the exponential curve describes some of the characteristics of the fungicide. The greater the value of 'k' (a negative value) the more rapidly disease decreases initially. The lower the value of 'a' the greater the effectiveness of the fungicide at infinite dose. A combination of high a value of 'k' and low value of 'a' indicates an effective fungicide.

Rhynchosporium

Data for recently introduced fungicides (e.g. Unix and the strobilurins) is limited but it appears that they are not as effective in control of Rhynchosporium as the most effective triazoles. Thus control of this disease will continue to rely on the triazoles, particularly in combination with other fungicides active against the disease.

In the protectant situation no fungicide achieved 90% control except in low disease pressure situations. There are no fungicides currently available to the grower that can be used in a high disease pressure situation that will give near complete control. Against this disease, therefore, application of fungicides

protectively or at a very early stage in disease development, combined with multiple applications are essential to prevent significant yield loss where disease pressure is high.

Of the individual and combinations of fungicides tested in the protectant situation, Sanction gave comparable control and Opus and Tilt + Unix slightly better control than Tilt + 0.75 Aura. Of the other 'triazoles' tested Sportak 45 proved better than Folicur or Tilt. Tilt's performance alone was disappointing. In the second and third year of the trials, samples of Rhynchosporium infected leaves were sent to Novartis (formerly Ciba Agriculture) for insensitivity testing from sites where Rhynchosporium developed. Isolates were obtained from the leaf samples and compared for insensitivity to Tilt against standard 'sensitive' and 'resistant' isolates. In both seasons, isolates were detected that were intermediate between the standard isolates. Some sensitive isolates were found each year but the detection of intermediate isolates suggests that the effectiveness of some triazoles, particularly Tilt, may be less than when the fungicide was introduced. Triazoles differ in their performance even where there is a level of insensitivity in the population.

The morpholine fungicide Aura / Corbel was surprisingly effective in controlling Rhynchosporium in the protectant situation. Although claimed only to give partial control, its benefit as a partner to Tilt in the control of Rhynchosporium can be clearly seen from the control achieved by the standard. Another morpholine fungicide, Patrol, proved less effective at Rhynchosporium control. Both the addition of an MBC fungicide, Bavistin, and Unix to Tilt markedly improved the control achieved by Tilt alone. Over the last decade, insensitivity by the Rhynchosporium fungus to MBC's has steadily increased (Jones, 1990; Kendall *et al.*, 1993; Taggart *et al*, 1998) and they have been largely discredited for use on cereals. However, their low cost is attractive and from this data it would appear that whilst control by the MBC component is incomplete their use in combination with other fungicides may remain a useful option for the control of Rhynchosporium.

Disease control in the curative situation rarely exceeded 70%. Once again, Sanction was comparable to, and Opus and Tilt + Unix slightly better than, the standard. Sportak and Tilt performed relatively better in the curative than protectant situation. The addition of Calixin to Tilt resulted in comparable control to the standard. The performance of Aura alone in the curative situation was relatively poorer than in the protectant situation. However, the response curve of Tilt + 0.75 Aura was similar in both situations (compare for example the pairing with Sanction).

Mildew

Until the 1998 growing season, mildew control relied on the Morpholine fungicides (Aura/Corbel, Patrol and Calixin). The introduction of three new fungicides for mildew control from novel chemical groups has provided a much greater diversity at a time when field experience suggested the effectiveness of the morpholines was on the decline. Only limited testing of the new fungicides (Ensign, Fortress and Unix) was possible in the trials but relative to the standard their greater effectiveness against mildew, particularly as protectants, was clear. Most notable was the steep shape of the curves (Fig. 10) demonstrating that provided they are applied prior to infection low doses are as effective as high doses. All three products are recommended for use with other fungicides and it is likely that the control of mildew would be even better where mixtures are concerned.

Fig. 6. Fungicide dose response curves for fungicides in comparison to the standard fungicide (Tilt + 0.75 Aura) Rhynchosporium - protectant situation. Values in bracket indicate the number of comparisons.

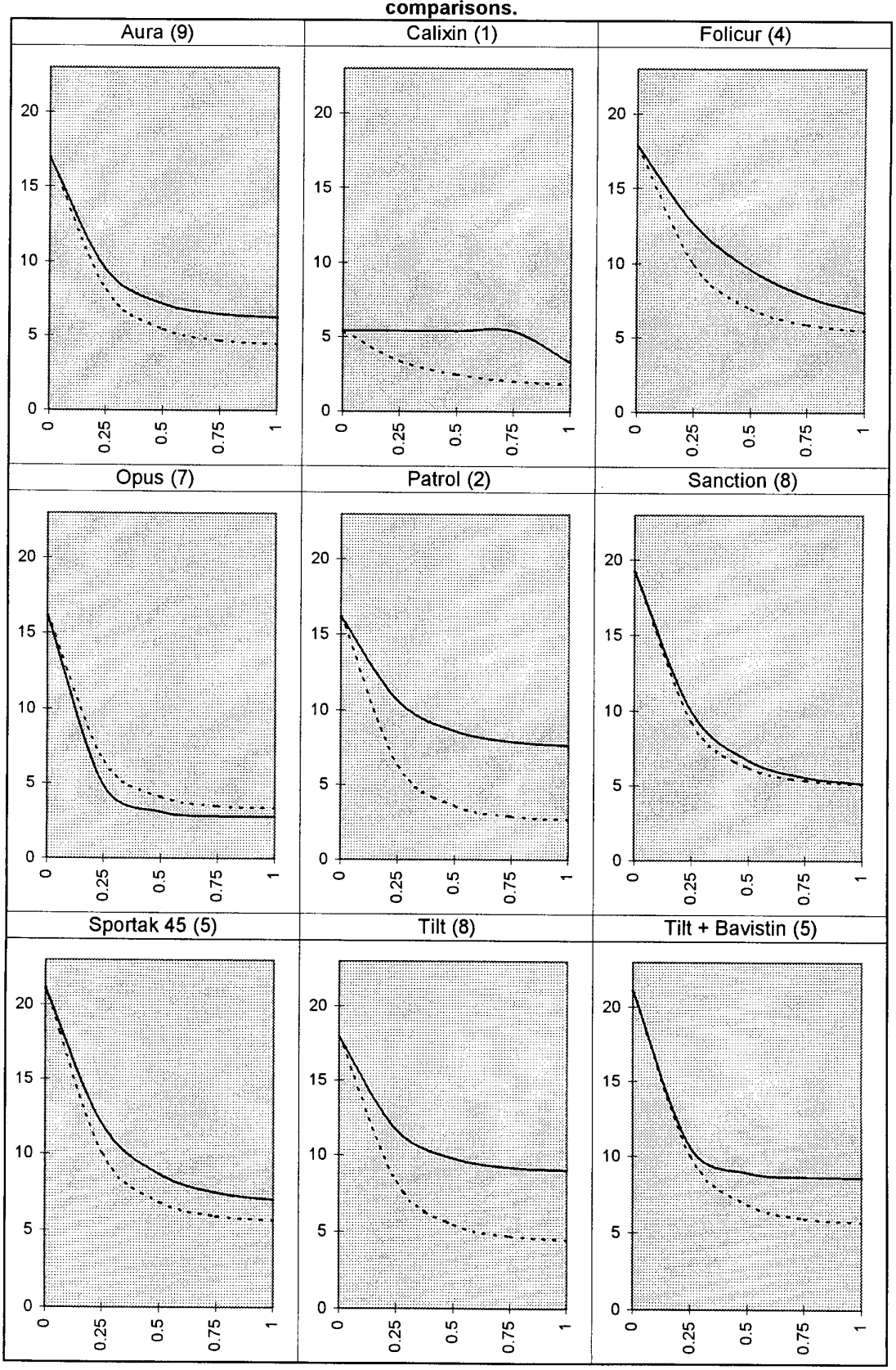


Fig. 6 continued. Fungicide dose response curves for fungicides in comparison to the standard fungicide (Tilt + 0.75 Aura) Rhynchosporium - protectant situation. Values in bracket indicate the number of comparisons.

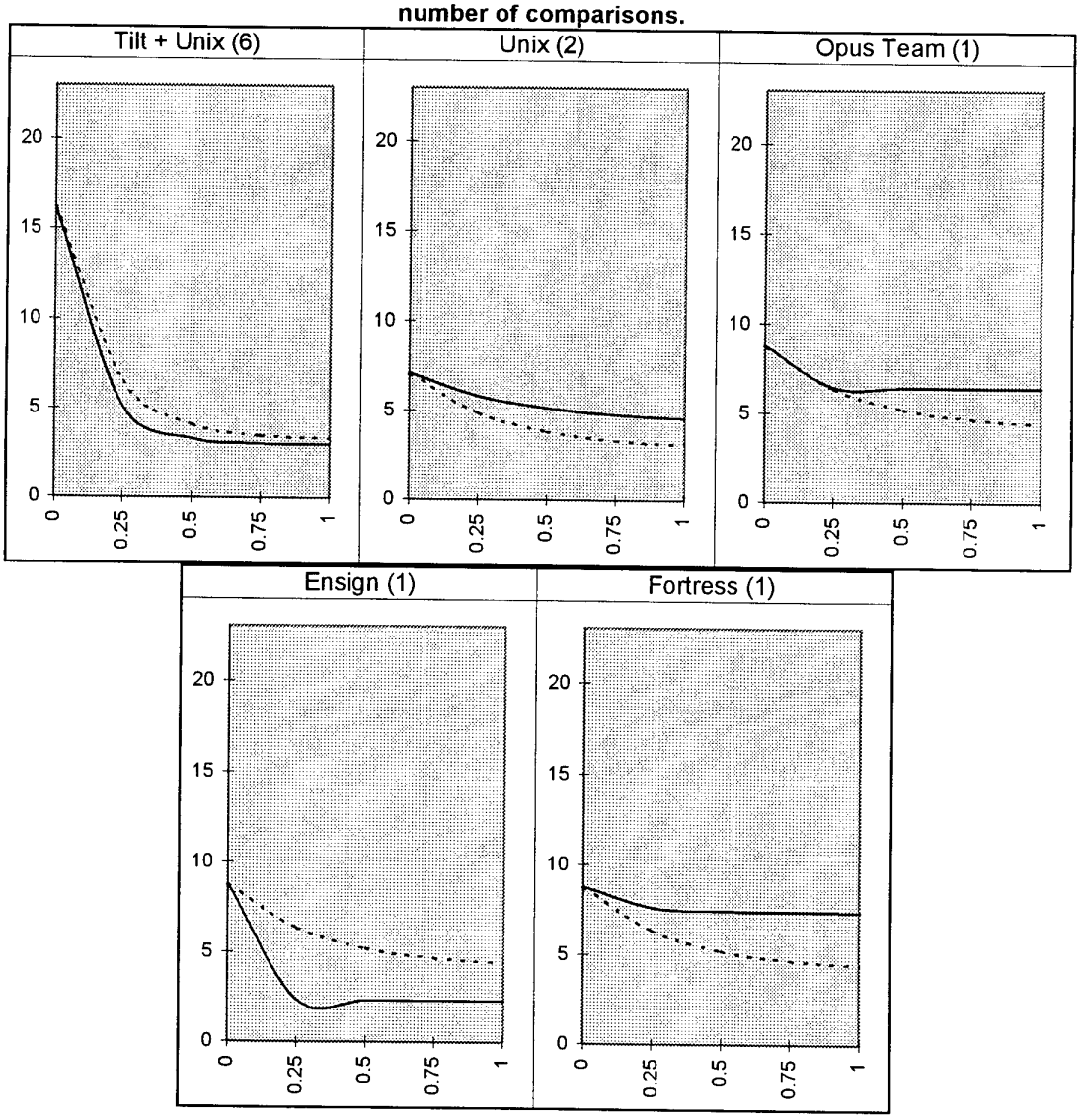


Table 6. Parameters for response curves in Figure 6.

Fungicide	a	b	r	R2
Aura	6.139	10.759	0.306	68.3
Tilt + 0.75 Aura	4.343	12.555	0.2954	81.1
Calixin	5.389	-2.20E-16	9890	80.9
Tilt + 0.75 Aura	1.677	3.712	0.46	25
Folicur	5.169	12.687	0.5875	97.7
Tilt + 0.75 Aura	5.247	12.609	0.3659	95.7
Opus	2.763	13.381	0.155	90.3
Tilt + 0.75 Aura	3.293	12.851	0.247	66.9
Patrol	7.481	8.697	0.3572	87.4
Tilt + 0.75 Aura	2.621	13.557	0.2675	80.3
Sanction	4.961	14.307	0.3476	95
Tilt + 0.75 Aura	5.028	14.24	0.29	57.1
Sportak 45	6.742	14.374	0.3636	97.5
Tilt + 0.75 Aura	5.572	15.544	0.2838	69.1
Tilt	8.914	9.001	0.307	36.4
Tilt + 0.75 Aura	4.374	13.541	0.2835	74.1
Tilt + Bavistin	8.56	12.556	0.1621	37.2
Tilt + 0.75 Aura	5.572	15.544	0.2838	69.1
Tilt + Unix	2.97	13.174	0.1571	66.8
Tilt + 0.75 Aura	3.293	12.851	0.247	66.9
Unix	4.371	2.701	0.528	54.7
Tilt + 0.75 Aura	2.955	4.117	0.462	84.8
Opus Team	6.433	2.323	1.66E-07	-
Tilt + 0.75 Aura	4.236	4.52	0.463	25.2
Ensign	2.3	6.456	1.66E-07	-
Tilt + 0.75 Aura	4.236	4.52	0.463	25.2
Fortress	7.396	1.36	0.15	-
Tilt + 0.75 Aura	4.236	4.52	0.463	25.2

Fig. 7. Fungicide dose response curves for three fungicides.
(Aura, Opus, Tilt+0.75Aura)
Rhynchosporium - protectant situation. Mean of 7 assessments.

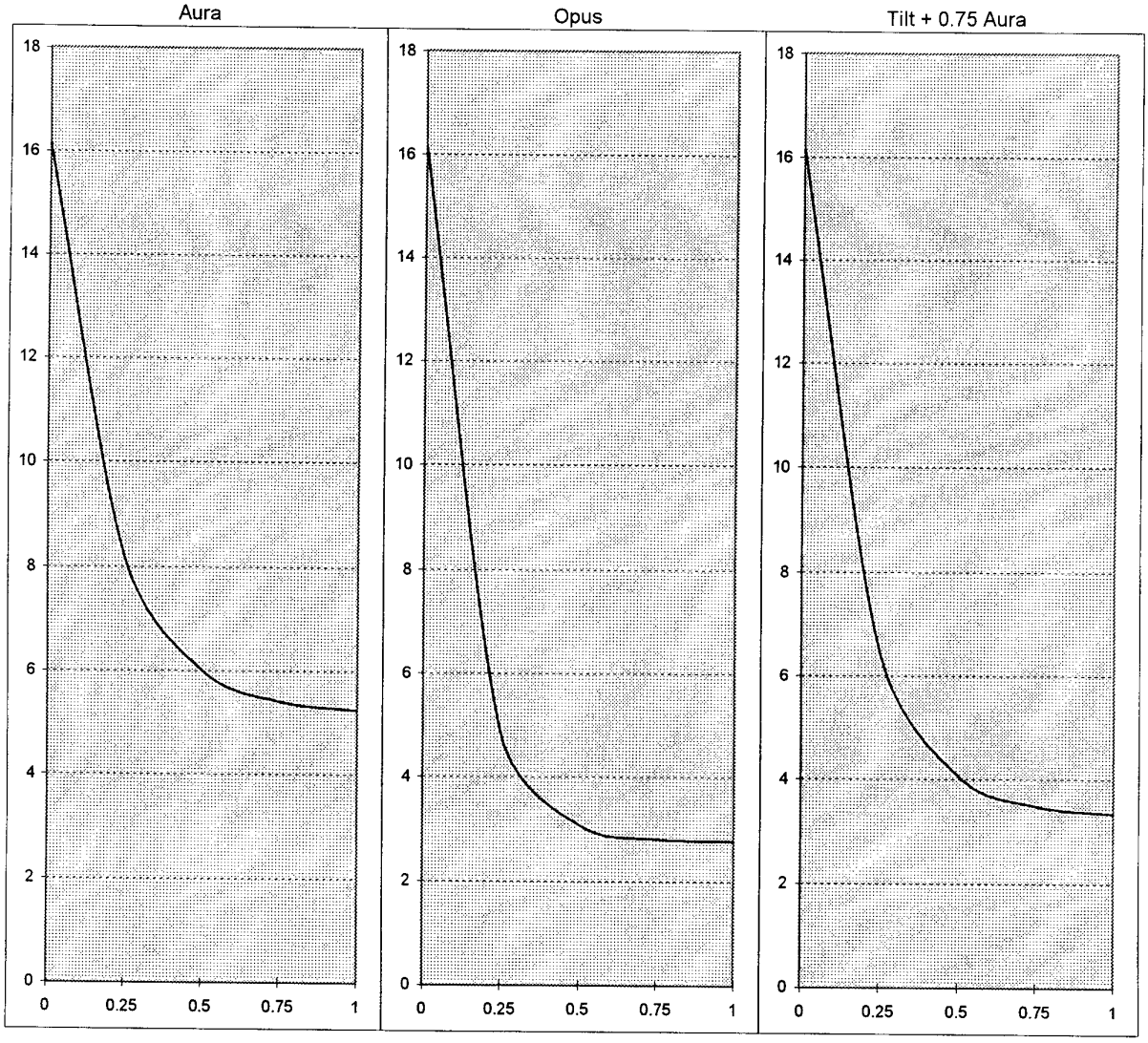


Table 7. Parameters for response curves in Figure 7.

Fungicide	a	b	r	R^2
Aura/Corbel	5.184	10.960	0.275	50.6
Opus	2.763	13.381	0.155	90.3
Tilt + 0.75 Aura	3.293	12.851	0.247	66.9

Fig. 8. Fungicide dose response curves for fungicides in comparison to the standard fungicide (Tilt + 0.75 Aura) Rhynchosporium - curative situation. Values in bracket indicate the number of comparisons.

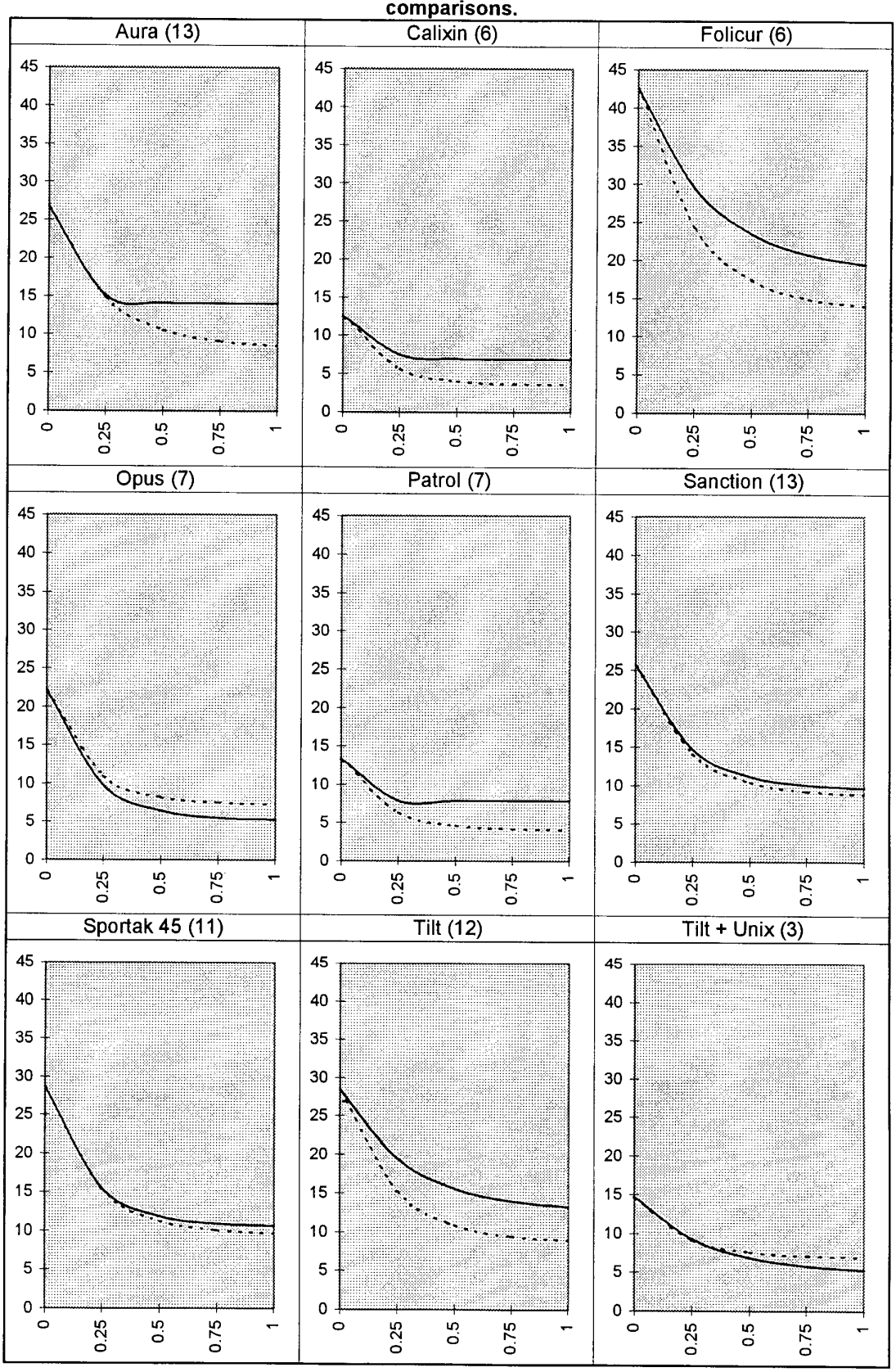


Fig. 8 continued. Fungicide dose response curves for fungicides in comparison to the standard fungicide (Tilt + 0.75 Aura) Rhynchosporium - curative situation. Values in bracket indicate the number of comparisons.

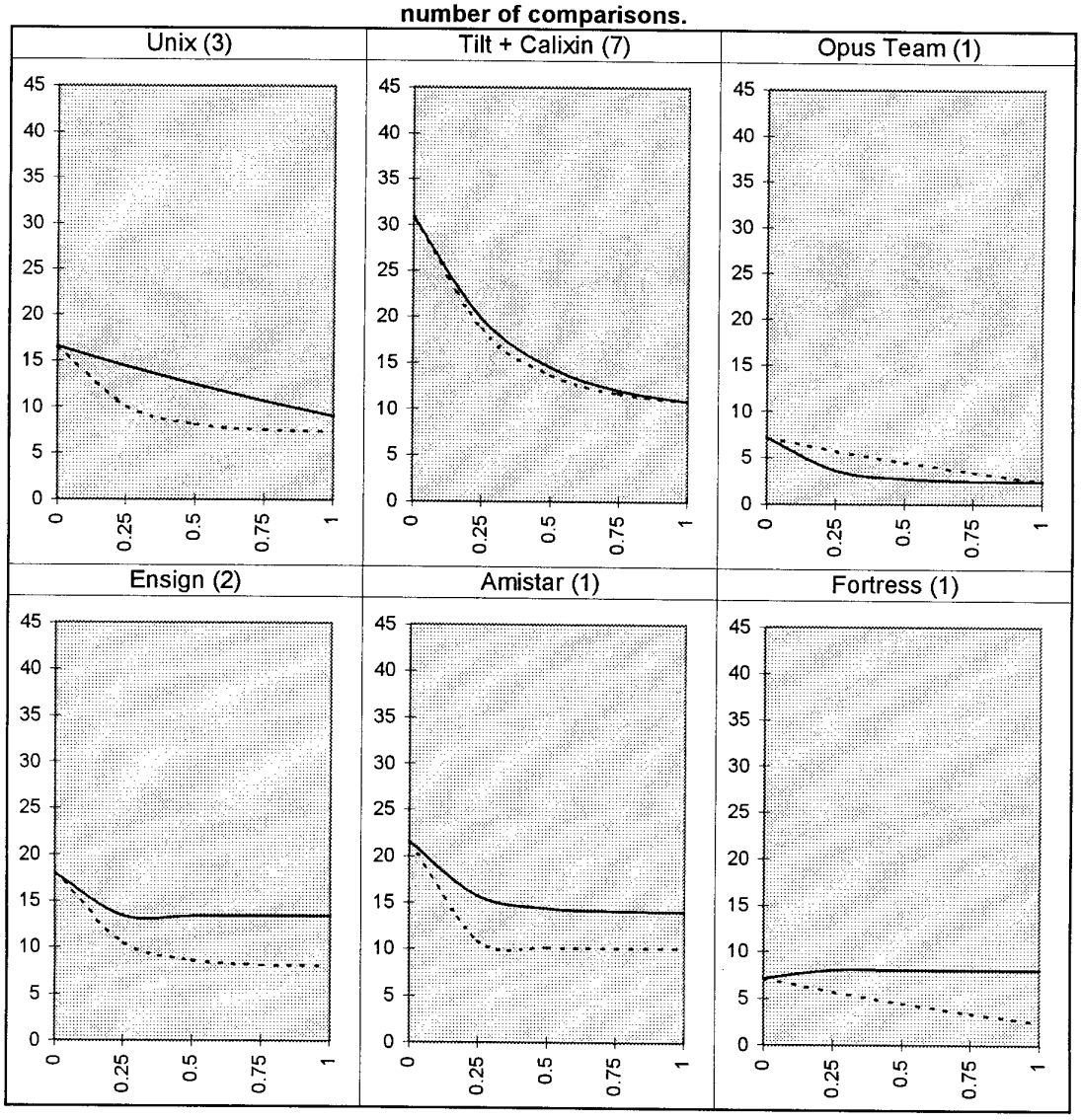


Table 8. Parameters for response curves in Figure 8.

Fungicide	a	b	r	R2
Aura	14.007	12.693	0.0844	79
Tilt + 0.75 Aura	8.273	18.427	0.3527	87.4
Calixin	6.877	5.644	0.101	-
Tilt + 0.75 Aura	3.552	8.969	0.2322	89.4
Folicur	18.35	24.21	0.458	73.3
Tilt + 0.75 Aura	13.45	29.11	0.368	72.1
Opus	5.226	16.858	0.2672	76.5
Tilt + 0.75 Aura	7.315	14.769	0.2468	68.2
Patrol	7.914	5.418	1.66E-07	-
Tilt + 0.75 Aura	4.116	9.216	0.246	88.8
Sanction	9.507	16.183	0.3191	89.5
Tilt + 0.75 Aura	8.681	17.009	0.3209	81.8
Sportak 45	10.54	18.07	0.272	66.2
Tilt + 0.75 Aura	9.55	19.06	0.3027	76.1
Tilt	12.75	15.68	0.426	84.4
Tilt + 0.75 Aura	8.76	19.67	0.3283	82.9
Tilt + Unix	4.839	9.811	0.4492	86.7
Tilt + 0.75 Aura	6.888	7.762	0.292	42.5
Unix	-6.28	22.8	0.906	88.4
Tilt + 0.75 Aura	7.314	9.206	0.302	38.4
Tilt + Calixin	9.766	21.064	0.4736	99.5
Tilt + 0.75 Aura	10.36	20.47	0.403	69.8
Opus Team	2.404	4.752	0.255	53.1
Tilt + 0.75 Aura	-4.344	11.5	0.877	23.5
Ensign	13.392	4.568	1.66E-07	-
Tilt + 0.75 Aura	8.011	9.949	0.251	21.7
Amistar	14.03	7.53	0.219	-
Tilt + 0.75 Aura	10.14	11.42	0.065	-
Fortress	8.0583	-0.9023	1.66E-07	-
Tilt + 0.75 Aura	-4.344	11.5	0.877	23.5

Fig. 9. Fungicide dose response curves for four fungicides. (Opus, Sanction, Tilt+Unix, Tilt+0.75 Aura) Rhynchosporium - curative situation. Mean of 6 assessments.

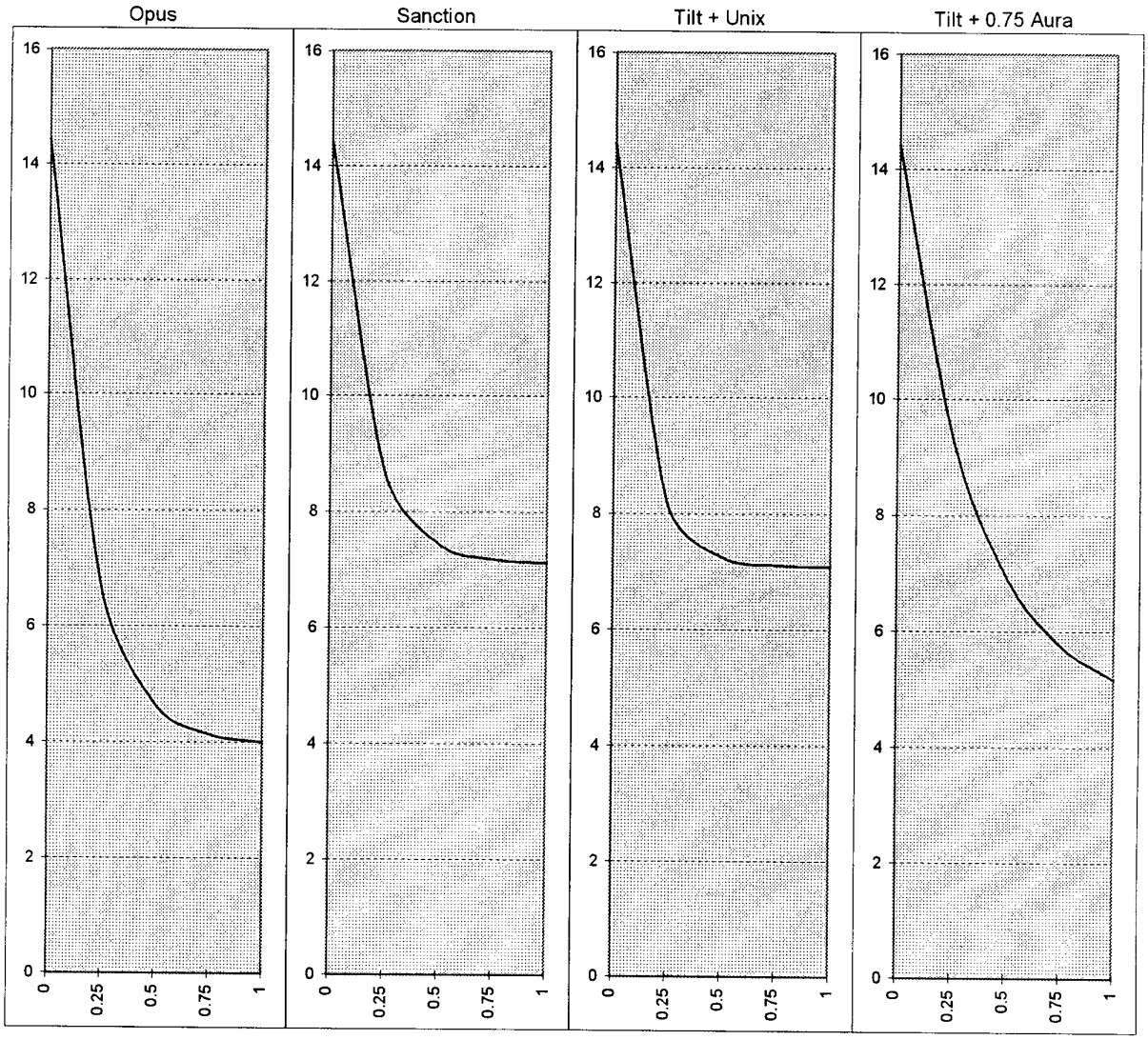


Table 9. Parameters for response curves in Figure 9.

Fungicide	a	b	r	R^2
Opus	3.951	10.469	0.263	85.3
Sanction	7.105	7.315	0.216	-
Tilt + 0.75 Aura	7.086	7.334	0.149	-
Tilt + Unix	4.59	9.83	0.492	85.3

Fig. 10. Fungicide dose response curves for fungicides in comparison to the standard fungicide (Tilt + 0.75 Aura) Mildew - protectant situation. Values in bracket indicate the number of comparisons.

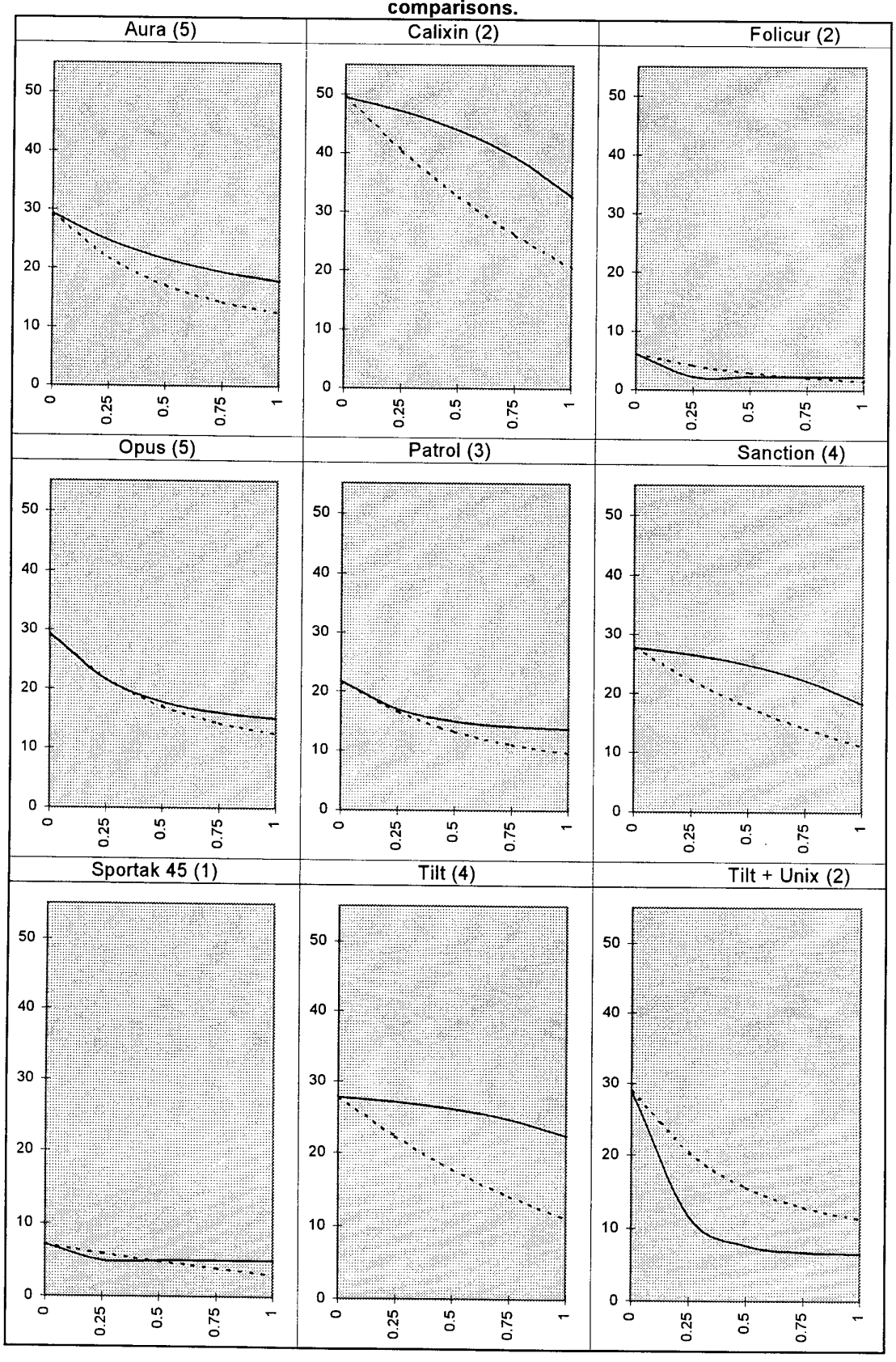


Fig. 10 continued. Fungicide dose response curves for fungicides in comparison to the standard fungicide (Tilt + 0.75 Aura) Mildew - protectant situation. Values in bracket indicate the number of comparisons.

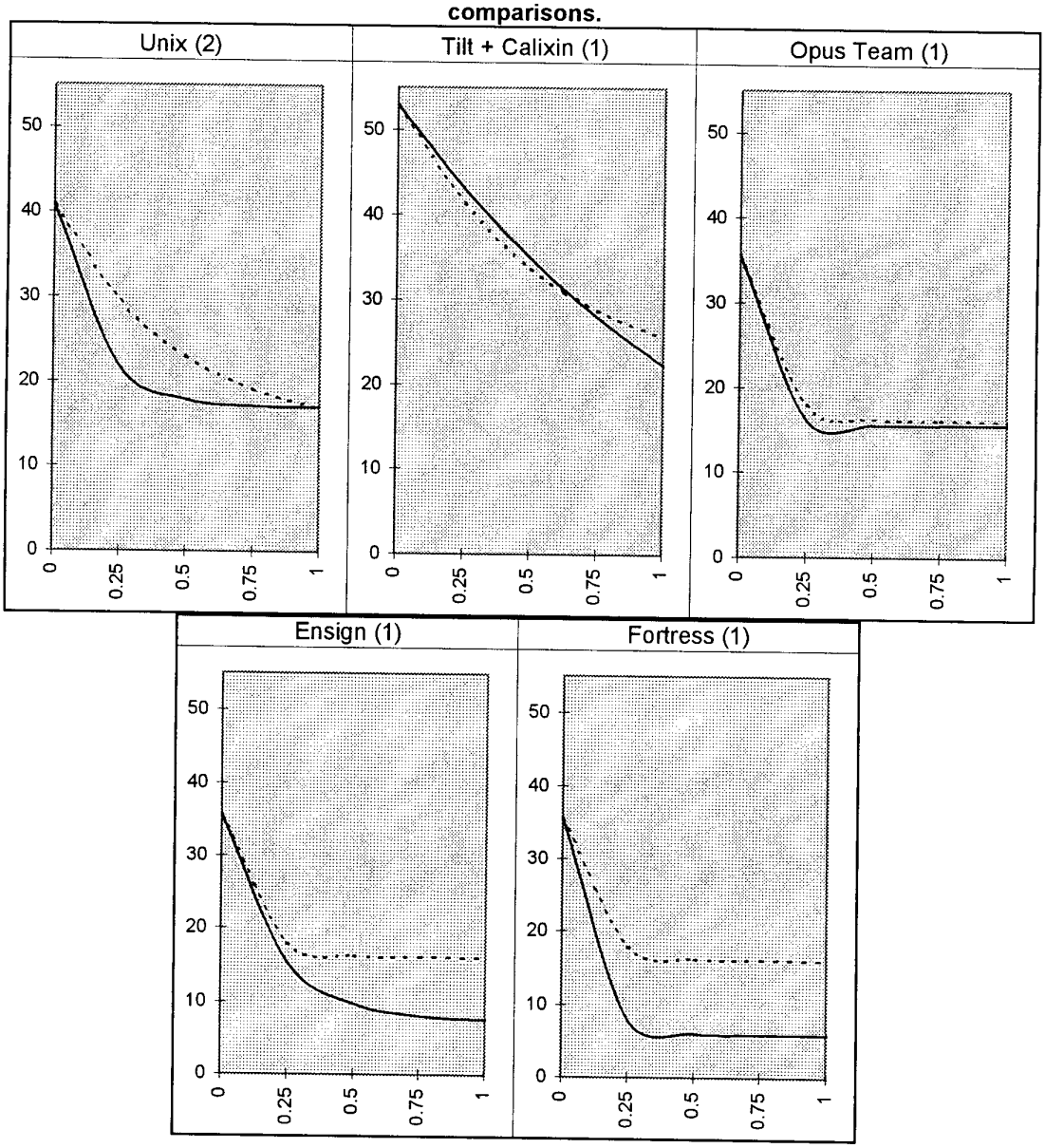


Table 10. Parameters for response curves in Figure 10.

Fungicide	a	b	r	R2
Aura	14.21	15.1	0.698	80.1
Tilt + 0.75 Aura	9.77	19.54	0.611	88.3
Calixin	54.9	-5.49	1.419	88.1
Tilt + 0.75 Aura	-9.59	59	0.846	92.8
Folicur	2.2272	3.8668	0.0063	-
Tilt + 0.75 Aura	0.534	5.56	0.656	70.9
Opus	14.34	14.97	0.481	83
Tilt + 0.75 Aura	9.77	19.54	0.611	88.3
Patrol	13.42	8.29	0.432	55.3
Tilt + 0.75 Aura	6.87	14.84	0.653	90.3
Sanction	30.31	-2.56	1.473	92.3
Tilt + 0.75 Aura	-2.45	30.2	0.818	90.8
Sportak 45	4.917	2.161	1.66E-07	-
Tilt + 0.75 Aura	-3.822	10.9	0.889	74
Tilt	28.91	-1.16	1.54	22.8
Tilt + 0.75 Aura	-2.45	30.2	0.818	90.8
Tilt + Unix	6.44	22.41	0.2273	53.3
Tilt + 0.75 Aura	9.21	19.64	0.571	66.2
Unix	16.98	23.74	0.197	-
Tilt + 0.75 Aura	13.78	26.94	0.582	66.8
Tilt + Calixin	-7.66	60.6	0.838	80.4
Tilt + 0.75 Aura	19.55	33.39	0.652	78.9
Opus Team	15.59	19.95	0.031	-
Tilt + 0.75 Aura	16.101	19.439	0.0956	51.1
Ensign	7.46	28.08	0.2824	73.2
Tilt + 0.75 Aura	16.101	19.439	0.0956	51.1
Fortress	5.879	29.661	0.0705	11
Tilt + 0.75 Aura	16.101	19.439	0.0956	51.1

Fig. 11. Fungicide dose response curves for eight fungicides. (Aura, Opus, Tilt + 0.75 Aura, Tilt + Unix, Unix, Opus Team, Ensign, and Fortress) Mildew - protectant situation. Mean of 6 assessments.

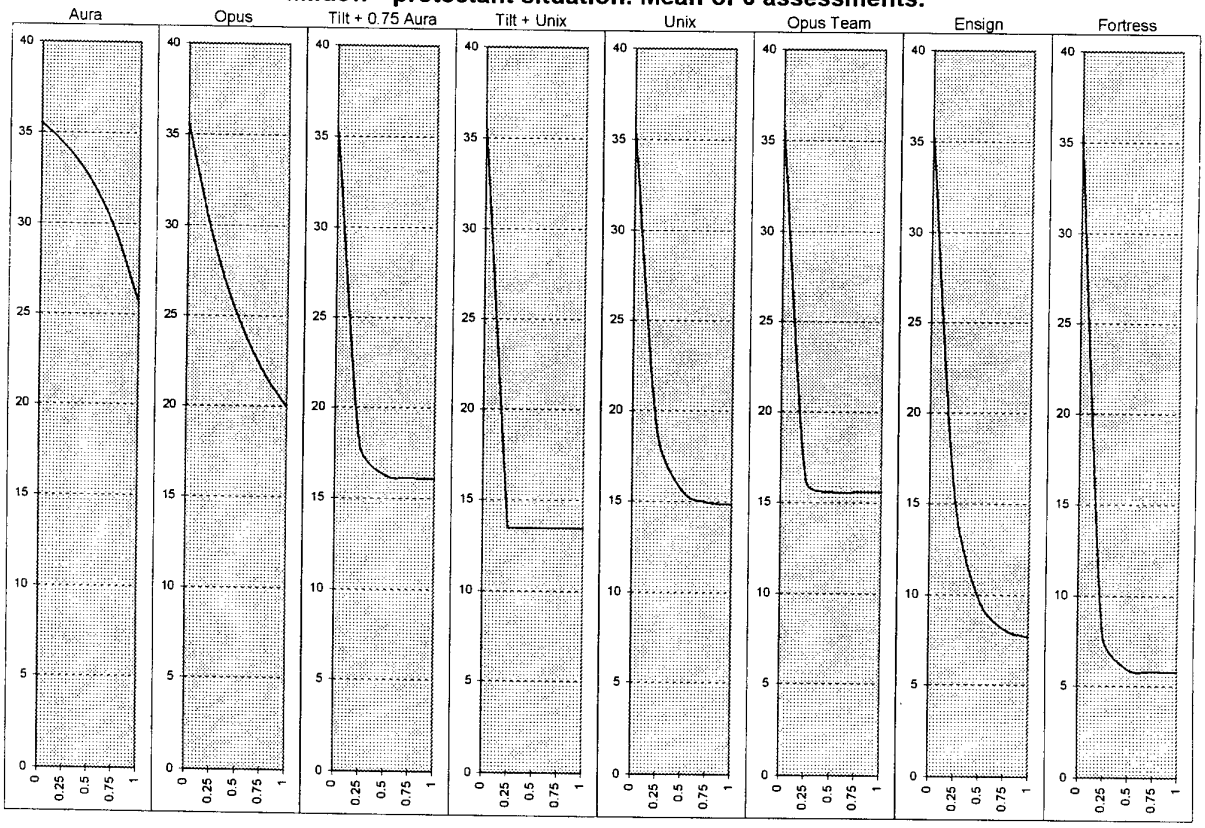


Table 11. Parameters for response curves in Figure 11.

Fungicide	a	b	r	R^2
Aura	38.14	-2.6	1.48	27.7
Opus	15.15	20.39	0.697	81.3
Tilt + 0.75 Aura	16.101	19.439	0.0956	51.1
Tilt + Unix	13.47	22.07	0.166E-06	-
Unix	14.8	20.74	0.196	48.7
Opus Team	15.59	19.95	0.031	-
Ensign	7.46	28.08	0.2824	73.2
Fortress	5.879	29.661	0.0705	11.0

Fig. 12. Fungicide dose response curves for fungicides in comparison to the standard fungicide (Tilt + 0.75 Aura) Mildew - curative situation. Values in bracket indicate the number of comparisons.

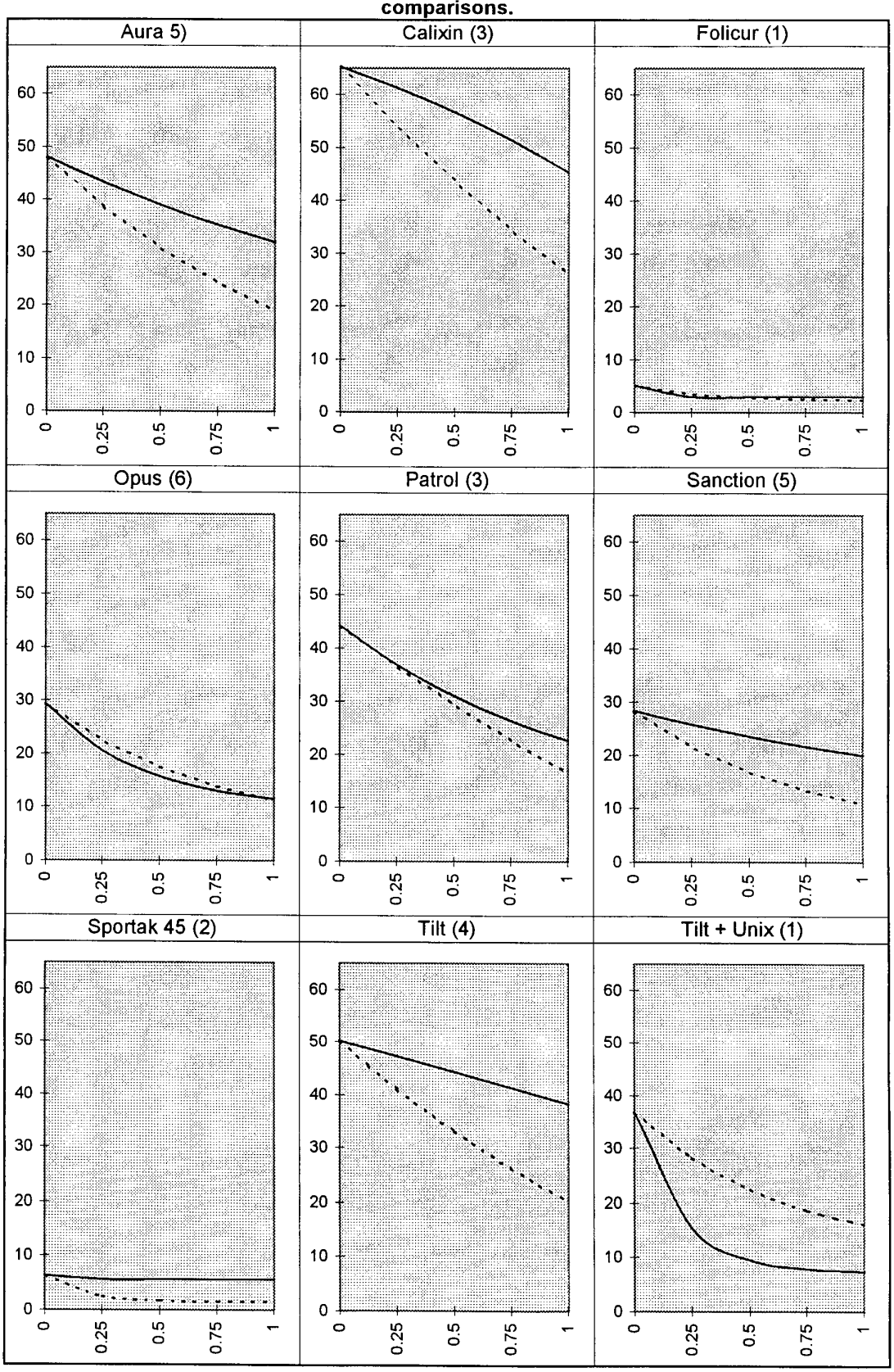


Fig. 12 continued. Fungicide dose response curves for fungicides in comparison to the standard fungicide (Tilt + 0.75 Aura) Mildew - curative situation. Values in bracket indicate the number of comparisons.

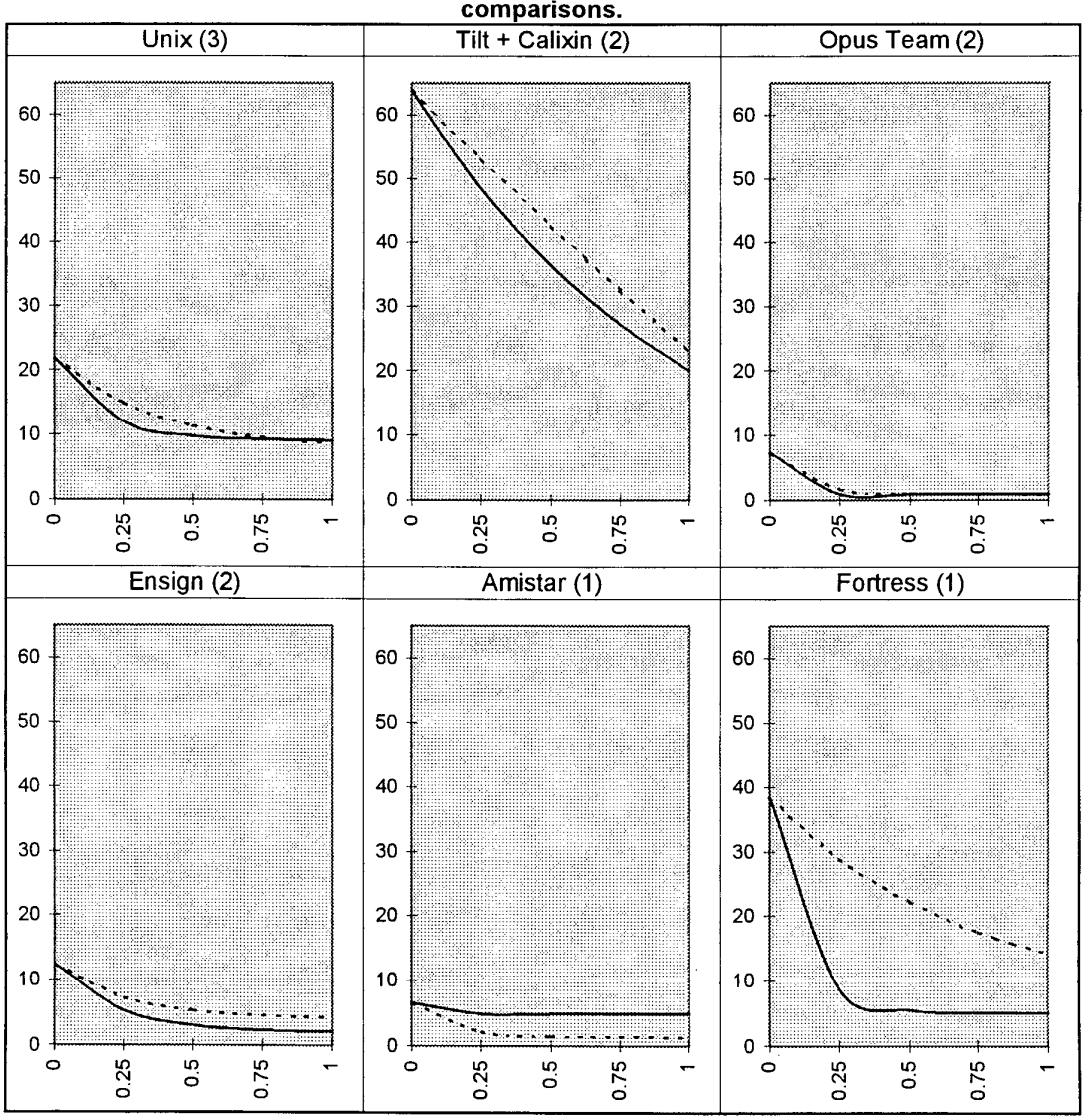


Table 12. Parameters for response curves in Figure 12.

Fungicide	a	b	r	R2
Aura	7.08	41	0.884	65.9
Tilt + 0.75 Aura	-7.92	56	0.833	93.8
Calixin	91.45	-26.2	1.151	89.3
Tilt + 0.75 Aura	-56.75	122	0.908	93.9
Folicur	2.958	2.142	1.66E-07	-
Tilt + 0.75 Aura	2.206	2.894	0.447	78
Opus	9.79	19.63	0.55	84.9
Tilt + 0.75 Aura	4.62	24.8	0.7205	96
Patrol	7.87	36.4	0.799	88.1
Tilt + 0.75 Aura	-78.73	123	0.938	87.8
Sanction	8.41	20	0.872	82
Tilt + 0.75 Aura	4.78	23.63	0.7148	98.6
Sportak 45	5.6104	0.6976	1.66E-07	-
Tilt + 0.75 Aura	1.5433	4.7647	0.1911	98.9
Tilt	342.21	-292	1.01	31.2
Tilt + 0.75 Aura	-17.79	68	0.865	95.7
Tilt + Unix	7.23	29.65	0.273	39.4
Tilt + 0.75 Aura	10.78	26.1	0.6725	99.6
Unix	9.16	12.69	0.222	-
Tilt + 0.75 Aura	7.722	14.128	0.5095	97
Tilt + Calixin	-4.44	68.3	0.774	88.1
Tilt + 0.75 Aura	-214.14	278	0.961	87.6
Opus Team	0.9	6.481	1.66E-07	-
Tilt + 0.75 Aura	0.963	6.418	0.1195	80.3
Ensign	2.016	10.392	0.3113	75.6
Tilt + 0.75 Aura	4.068	8.34	0.391	25.1
Amistar	4.884	1.726	1.66E-07	-
Tilt + 0.75 Aura	1.3474	5.2626	0.1539	91.8
Fortress	5.109	33.391	0.1055	68.5
Tilt + 0.75 Aura	6.3	32.2	0.704	26

Fig. 13. Fungicide dose response curves for five fungicides. (Opus, Tilt + 0.75 Aura, Unix, Opus Team and Ensign) Mildew - curative situation. Mean of 6 assessments.

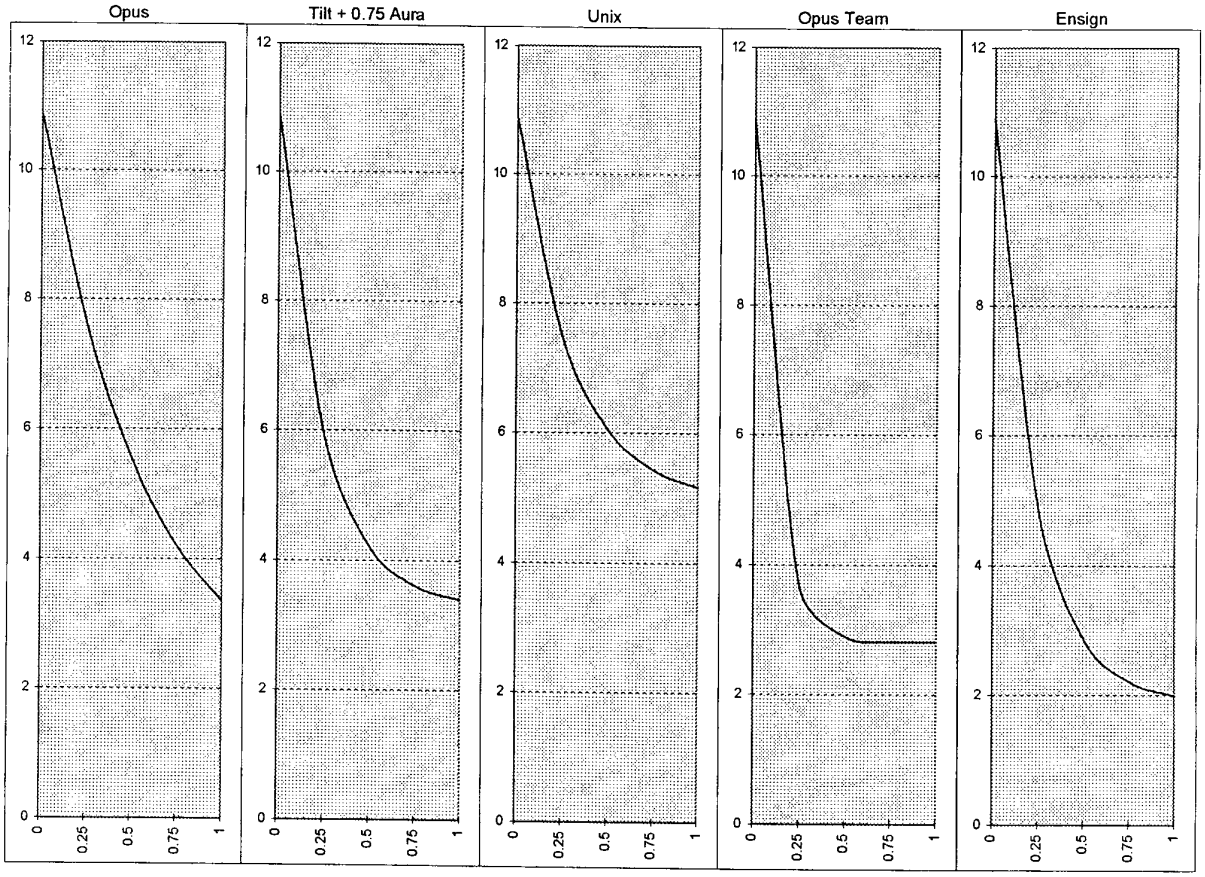


Table 13. Parameters for response curves in Figure 13.

Fungicide	a	b	r	R^2
Opus	1.78	9.09	0.6453	95.5
Tilt + 0.75 Aura	3.277	7.593	0.354	34.4
Unix	4.961	5.909	0.4306	98.9
Opus Team	2.812	8.058	0.1097	56.7
Ensign	1.878	8.992	0.333	77.0

The degree of control achieved by Aura was disappointing although the data were obtained from high disease pressure sites. It is possible that the control achieved was relatively short-lived and mildew, known for its ability to rapidly increase, developed rapidly towards the end of the 5 week assessment period. Comparing Aura to the standard, the majority of control appears to come from the morpholine. Patrol was equivalent if not slightly better than Aura. Calixin alone was not as effective as the other morpholines. It is not known for its persistence. However, when combined with Tilt was it was as effective as the standard.

Although tested in moderate disease pressure situations, the triazole Opus alone provided control comparable to the standard in both protectant and curative situations. Tilt and Sanction, on the other hand, gave relatively poor control, as would be expected from the triazoles. The combination of Tilt + Unix provided control of mildew much greater than the standard with almost all control achieved by the first quarter dose.

Brown rust

Results from trials where brown rust developed were very consistent and may be considered a good representation of the relative performance of those fungicides tested. Brown rust, like mildew has the potential to develop rapidly under favourable conditions. Like mildew also, a high level of control of brown rust is possible with effective fungicides. The results presented here show clear differences in efficacy between the products tested.

The pattern of dose response curves obtained in the protectant situation was broadly the same as that for the curative situation.

The triazoles Folicur and Opus were most effective in both protectant and curative situations, providing 95+% control in the curative situation. In the protectant situation 0.5 doses of Folicur and Opus resulted in control equivalent to full dose. In the curative situation 0.75 dose produce control almost equivalent to a full dose.

The standard, Tilt + 0.75 Aura / Corbel provided approximately 80% control in the curative situation, but other morpholine and triazole fungicides mostly gave less than 70% control.

Halo spot

Halo spot (Selenophoma donacis) is an uncommon disease of barley. Whilst found in only a single trial in the south west of England, the rarity of data on its control was sufficient to note the results. Halo spot was found to be effectively controlled by each of the fungicides tested (Aura, Folicur, Sanction, Sportak, Tilt, Tilt + Bavistin, Tilt + Calixin), the greatest control given by the standard Tilt + 0.75 Aura.

Yield, Thousand grain weight and Specific weight

More than one disease was usually present in the trials and the impact of individual diseases on yield and grain quality could not be determined. Additionally with just a single fungicide application, disease development before application and late in the growing season was not prevented, and these would have affected yield and grain quality rendering interpretation of the effect of specific fungicides meaningless. Whilst individual trial yield and quality data were examined in relation to disease control, no overall analyses were possible and are thus not presented.

Yield, thousand grain weight and specific weight increases with increasing dose also followed an exponential pattern with diminishing extra yield as the dose increased towards full dose. At high disease sites yield responses of 0.5 - 1 t/ha were typically achieved by a single application of the most effective fungicides. The average maximum increase in thousand grain weight was 2.4g (range 0.65 - 3.7 g) and of specific weight was 1.2 kg/hl (range 0.3 - 1.9 kg/hl).

Cost response curves

The comparative dose response curves shown in figures 7, 9, 11, 13, 14 & 15 can be converted into cost response curves by simply substituting the fungicide cost for dose. One example is given in Fig. 16. Data used is that from Fig. 13 and Table 13. Curves such as this enable a grower to establish either the level of control likely at a given cost of fungicide or the cost of achieving a particular level of disease control for a specific fungicide.

With the pairs of dose response curves, evaluation of cost effectiveness between two fungicides has to be determined relative to the standard. Because the pairs of curves do not have the same origin, only the level of control likely at a given cost of fungicide can be readily determined. An example is shown in Fig. 17 where four pairs of fungicide dose response curves in Fig. 8 and Table 8 are converted to cost response curves. A line is drawn on each graph at the £10 fungicide cost. At this cost the control of Rhynchosporium relative to the Tilt + 0.75 Aura control is 12% less for Tilt + Unix, 6% less for Tilt, 15% better for Sanction and 25% better for Opus. At £20 cost the comparable figures are 6% better for Tilt + Unix, 4% better for Sanction and 24% better for Opus. A full dose of Tilt costs less than £20 and at its full dose Tilt gave 17% poorer control than the standard.

Fig. 14. Fungicide dose response curves for eight fungicides. (Aura/Corbel, Folicur, Opus, Patrol, Sanction, Sportak, Tilt, Tilt + 0.75 Aura) Brown rust - protectant situation. Mean of 4 assessments.

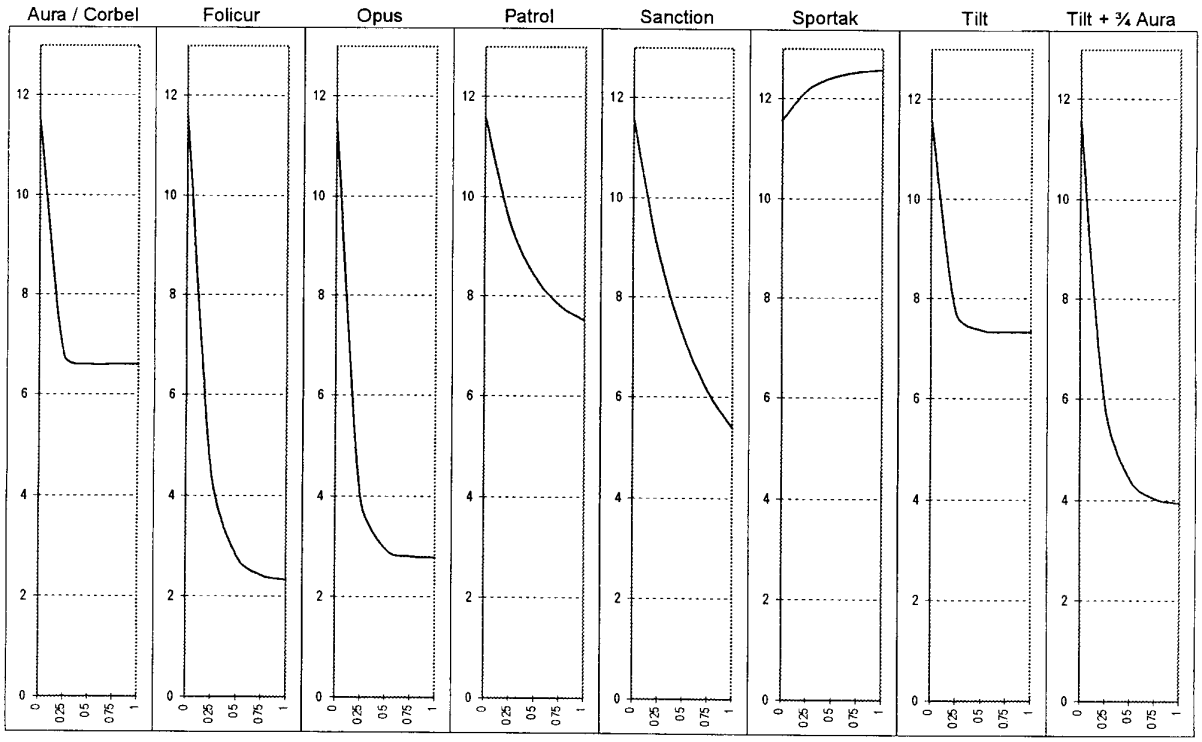


Table 14. Parameters for response curves in Figure 14.

Fungicide	a	b	a + b	k
Aura / Corbel	6.601	4.97	11.571	-13.091
Folicur	2.3	9.271	11.571	-5.7605
Opus	2.7776	8.7934	11.571	-7.8616
Patrol	7.181	4.39	11.571	-2.5774
Sanction	3.991	7.58	11.571	-1.6925
Sportak	12.611	-1.04	11.571	-3.3759
Tilt	7.33	4.241	11.571	-9.6318
Tilt + ¾ Aura / Corbel	3.898	7.673	11.571	-5.3883

Fig. 15. Fungicide dose response curves for fungicides.
(Aura/Corbel, Folicur, Opus, Patrol, Sanction, Sportak, Tilt, Tilt + 0.75 Aura)
Brown rust - curative situation. Mean of 3 assessments.

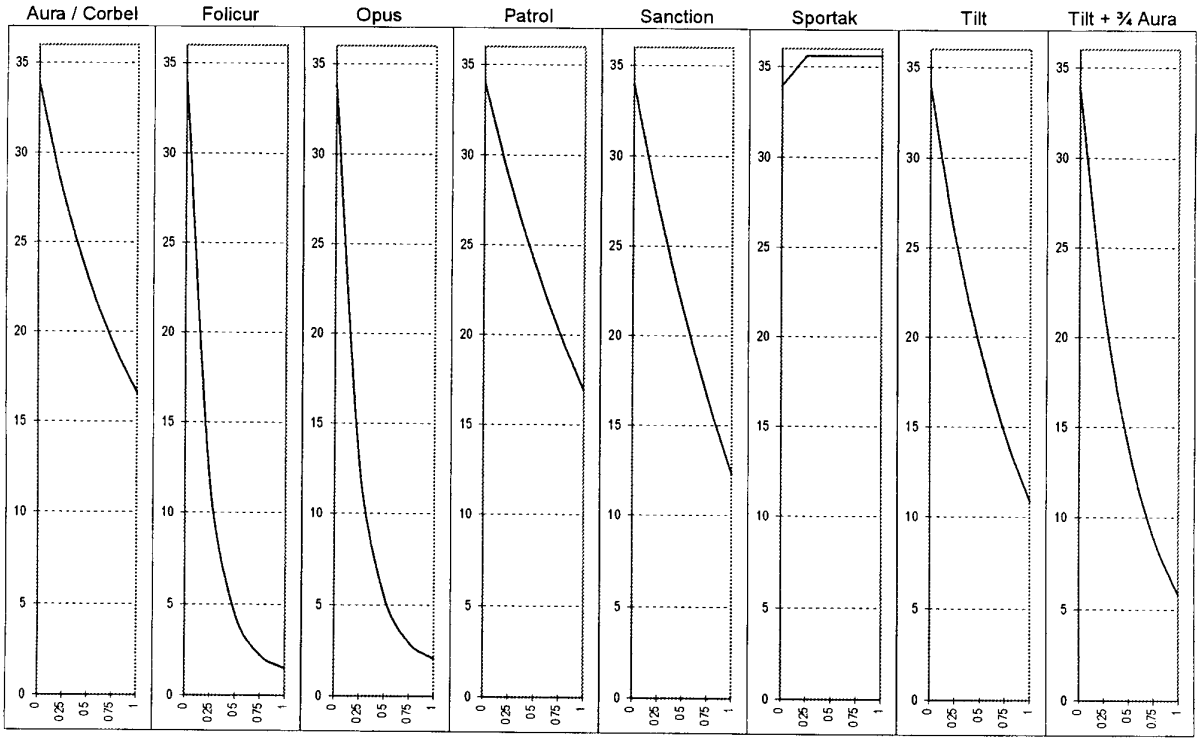


Table 15. Parameters for response curves in Figure 15.

Fungicide	a	b	a + b	k
Aura / Corbel	6.54	27.4	33.94	-1.0041
Folicur	1.149	32.791	33.94	-4.5942
Opus	1.584	32.356	33.94	-4.2568
Patrol	-2.46	36.4	33.94	-0.6313
Sanction	-15.26	49.2	33.94	-0.5801
Sportak	35.608	-1.668	33.94	-62.592
Tilt	-0.86	34.8	33.94	-1.0846
Tilt + ¾ Aura / Corbel	0.58	33.36	33.94	-1.8507

Fig. 16. Cost response curves for fungicides compared at the same sites. Data from Fig. 13 and Table 13. Mildew - curative situation.

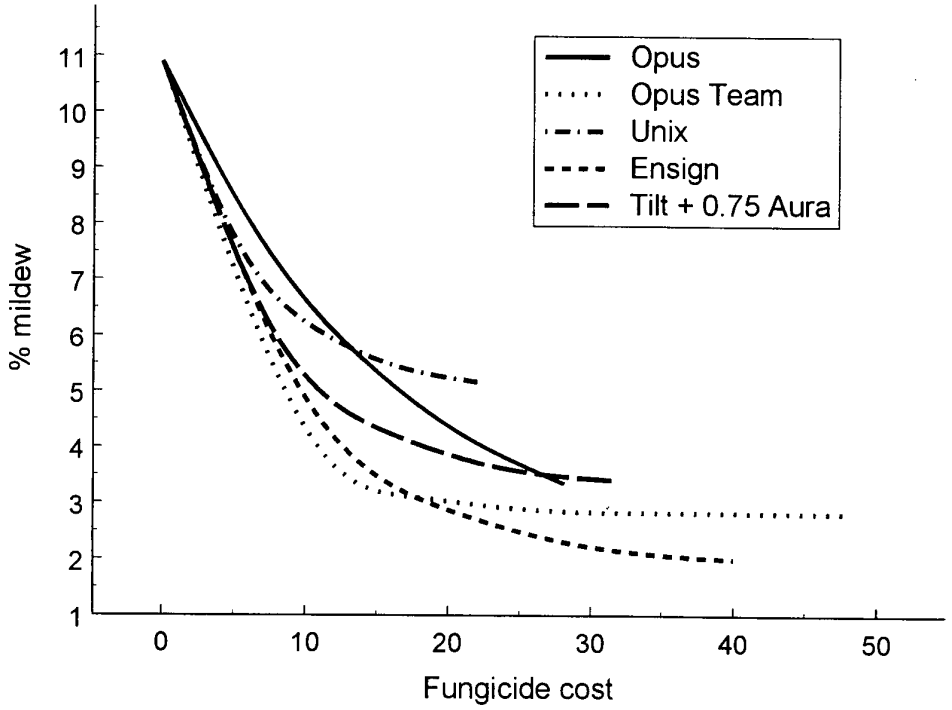
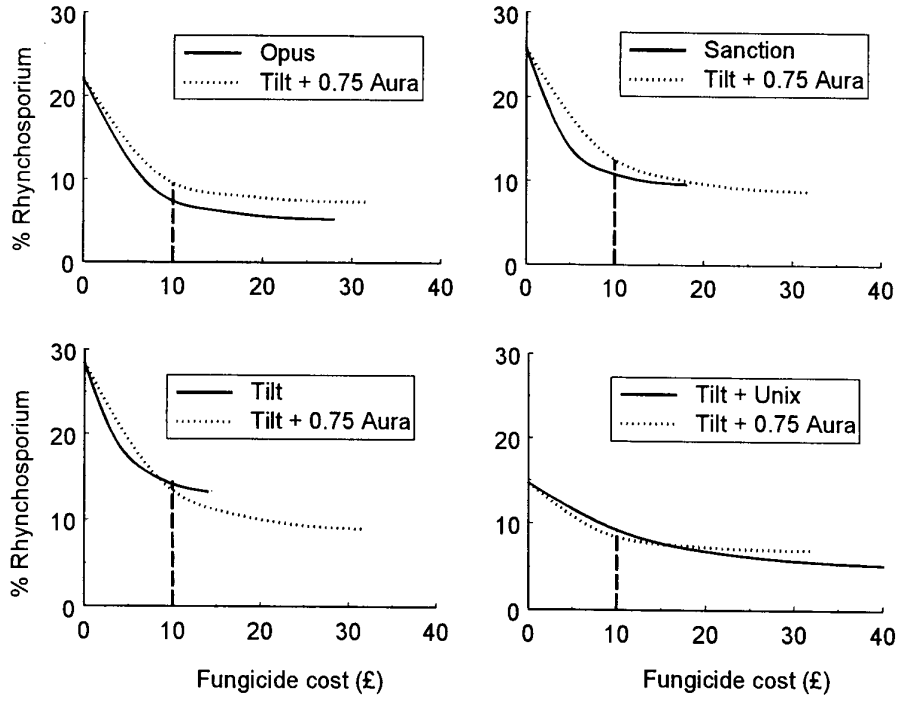


Fig. 17. Cost response curves for four fungicides relative to a standard fungicide using data from Fig. 8 and Table 8. Rhynchosporium - curative situation



Experiment 2. Variety x Fungicide Dose Interaction

Considerably more data was obtained for Rhynchosporium and mildew than brown rust or net blotch. Sets of curves are shown below for each of these four diseases (Figs. 18 to 21) with tables giving the parameters of the fungicide dose equations (Tables 16 to 19). The R^2 values for the response curves for Rhynchosporium and mildew are shown in the tables and are all greater than 80%. By contrast, the R^2 values for brown rust and net blotch are highly variable. The curves for net blotch, in particular, are based on limited data and need to be viewed with considerable caution.

It would be expected that if the varieties used in the trials manifest their resistance to the particular disease as given in published resistance ratings, the curves of the untreated controls for each disease should be similar to the NIAB standard curve for determining resistance (Fig. 3). It is clear, however, that the curves presented here vary from disease to disease and for, mildew do not conform to the NIAB standard curve. The untreated control Rhynchosporium, brown rust and net blotch curves (Figs. 18, 20 & 21) are more or less a straight line between resistance ratings 3 and 8. This mirrors the NIAB curve between these ratings but at a higher level of infection with Rhynchosporium and brown rust. This is encouraging and, in part , validates the NIAB curve. The higher level of disease at lower resistance ratings presented here is probably due to the process of selecting only data sets where the untreated reached 5% area infection.

By contrast the untreated control curve for mildew differs markedly from the NIAB standard curve. Instead of a steady increase in disease as the resistance rating falls from 8 to 4 followed by a small exponential increase in disease as the resistance rating drops to 3 in the NIAB curve, there is a substantial exponential increase in disease as the resistance rating declines below 5. The curve presented here is based on considerably less data than used to produce resistance ratings and uses selected data where infection on the untreated control reached 5% area but the disparity is considerable. By taking the resistance ratings of varieties in the year following experimentation, it would be hoped that changes in the race structure of the mildew population would have been accounted for. However, the results suggest that the varieties used with ratings 3 and 4 are more susceptible to mildew, at least at the sites where trials were carried out, than the resistance ratings suggest.

Of more relevance to this project, the results provide, for the first time, a clear indication of the dose of a competent broad spectrum fungicide required at a particular resistance rating to reduce disease to a desired level. However, each disease needs to be considered in turn as the degree of control that can be achieved by a fungicide varies.

For Rhynchosporium, it is difficult to achieve 100% control on a susceptible variety with the most effective fungicide, even at full dose (Fig. 18). Clearly, with this disease, on varieties with Rhynchosporium resistance ratings up to and including 6, a two or three spray programme is likely to required to maintain the disease at low levels where the disease pressure is high. With resistance ratings above 6, a single well timed application is likely to be sufficient. The dose required for control at any particular resistance rating can be reduced. As demonstrated in Experiment 1, a 0.75 dose gave equivalent control to a full dose at a resistance rating of 3. Even a 0.5 dose provided 85% control of the full dose. As the resistance rating increases so the degree of control achieved by the 0.5 dose approaches that of 0.75 (Fig. 18).

It is possible to obtain an estimate of the value of a unit of resistance from these curves, at least at lower resistance ratings. Thus a 0.25 dose at a rating of 4 was equivalent to a 0.5 dose at a rating of 3, and a 0.25 dose at a rating of 5 equivalent to a 0.5 dose at a rating of 4 (Fig. 18).

With an effective fungicide a high degree of control of mildew can be achieved even at reduced dose. Apart from a variety with a resistance rating of 3, control of mildew in varieties with higher resistance ratings was similar with 0.5, 0.75 or 1.0 doses. The 0.25 dose approached the control of higher doses at ratings of 6 and above. Where the variety was very susceptible (resistance rating of 3), 0.25,0.5 and 0.75 doses only achieved around 60% control compared to 80% for a full dose. Thus, like Rhynchosporium, where susceptible varieties are concerned, multiple reduced doses are required.

In estimating the value of a unit of resistance for mildew, the graph indicates that almost the same degree of control is achieved by a 0.25 dose applied to a variety with a resistance rating of 4 as a full dose to a variety with a resistance rating of 3. Similarly, the same degree of control is achieved by a 0.25 dose applied to a variety with a resistance rating of 5 as a 0.5 dose to a variety with a resistance rating of 4 (Fig. 19).

Fig. 18. Response curves for four fungicide doses and untreated control in relation to Rhynchosporium resistance rating

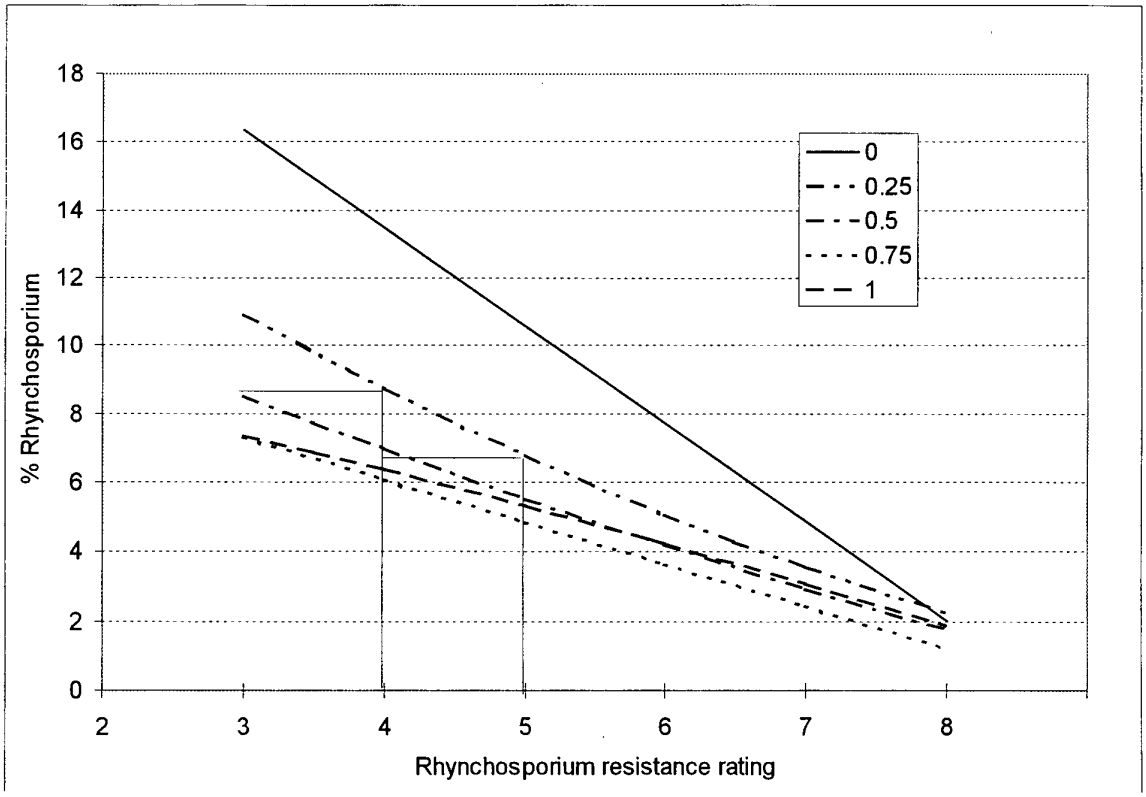


Table 16. Parameters for response curves in Figure 16.

Dose	a	b	r	R^2
0	-1353	1378	0.9979	97.8
0.25	-7.6	27.1	0.881	84.3
0.5	-14.3	28.2	0.932	92.1
0.75	-299	310	0.996	94.4
1.0	28.2	-18.1	1.048	90.1

Fig. 19. Response curves for four fungicide doses and untreated control in relation to mildew resistance rating

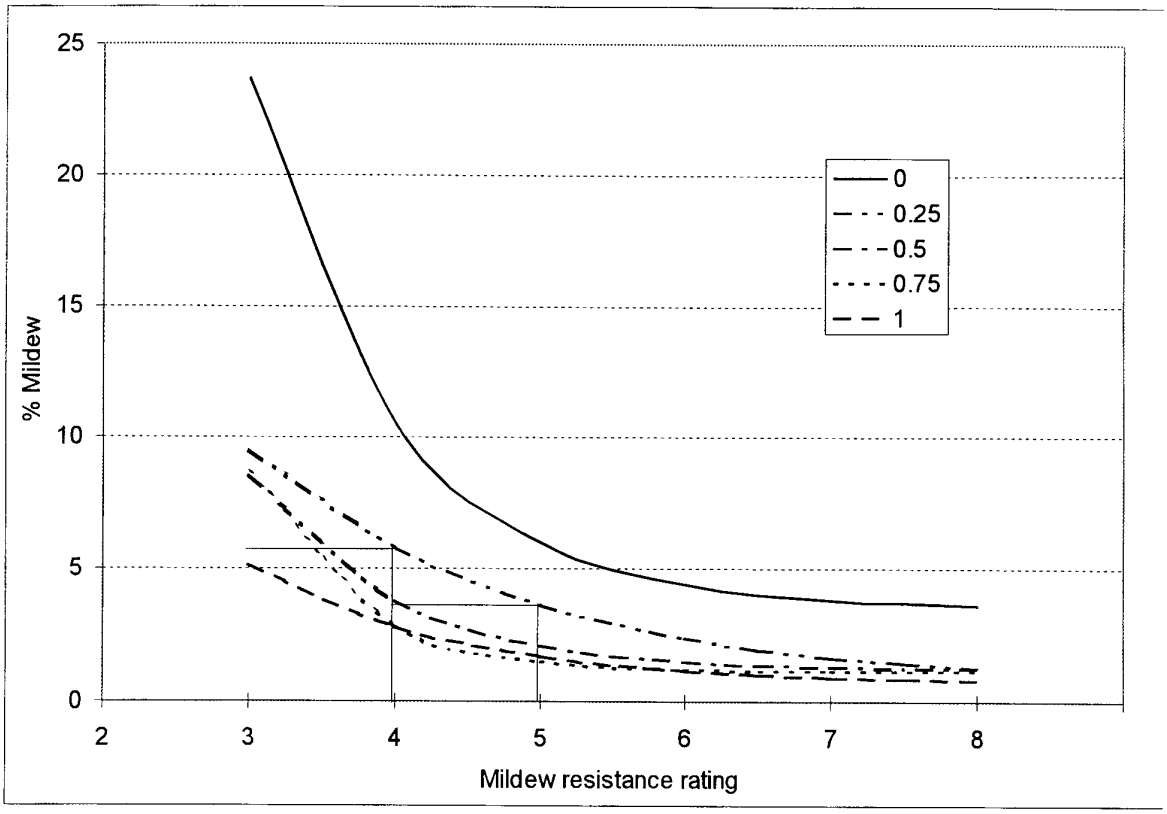


Table 17. Parameters for response curves in Figure 17.

Dose	a	b	r	R^2
0	3.53	469	0.35	85.5
0.25	0.65	46.0	0.578	87.2
0.5	1.180	168	0.353	86.7
0.75	1.121	632	0.228	91.8
1.0	0.701	43.2	0.469	83.7

With brown rust, at a resistance rating of 3, the dose response curve mirrors that found in experiment 1 (Fig. 20). A 0.25 dose provided 52% control, a 0.5 dose 68% control and 0.75 and 1.0 doses 78% control. These relative degrees of control remained fairly consistent across the resistant ratings. Once again, with susceptible varieties, control of brown rust would depend on a programme of fungicides.

In estimating the value of a unit of resistance for brown rust, the graph indicates that almost the same degree of control is achieved by a 0.25 dose applied to a variety with a resistance rating of 5 as a 0.5 dose to a variety with a resistance rating of 3. Similarly, the same degree of control is achieved by a 0.5 dose applied to a variety with a resistance rating of 5 as a 0.75 or 1.0 dose to a variety with a resistance rating of 3. Thus, for a quarter dose of fungicide is equivalent to two units of resistance, which is greater for brown rust than Rhynchosporium or mildew.

Results for net blotch were so inconsistent that interpretation would be inadvisable. The graph (Fig. 21) is included only for the sake of completeness.

For Rhynchosporium, mildew and brown rust, if the disease levels for the four doses and the untreated at a resistance rating of 3 are plotted against dose, they produce dose response curves that mirror that found for Tilt + 0.75 Aura in experiment 1. The fungicide used in experiment 2 was chosen at the start of the project to have all round disease control of the four diseases evaluated here. The results of experiment 1 clearly show that for specific diseases, other fungicides can be more effective. Had a more effective fungicide been used instead of Tilt + 3/4 Aura, it would be expected that the data points at a resistance rating of 3 would reflect this. Similarly, if a high proportion of the control achieved by a full dose of a more effective fungicide is achieved by a low dose (e.g. 0.25), then the response curves of the different doses would be close together as the resistance rating increases.

CONCLUDING REMARKS

The first of the two experiments has provided, for the first time, independent comparative data on fungicide dose response curves. Curves like this are much needed by barley growers to make objective decisions on disease control. They enable not only the relative efficacy of fungicides to be determined but the relative cost effectiveness.

Over the period of testing, new fungicides were introduced onto the market and some of these were evaluated. However, inevitably the degree of testing was considerably less of more recent fungicides than those tested from the start of the project. There is no doubt that further fungicides will be introduced in the future and the data presented here will lose its full relevance. What is needed therefore is a system where dose response curves of new fungicides are require to be determined relative to an agreed standard (or standards) and published before they are released into the market. This evaluation should ideally be the responsibility of the agrochemical manufacturer and become part of the registration and approval process. Only in this way can growers be kept up-to-date and evaluate new fungicides objectively.

The methodologies used in this project and the sister project on appropriate fungicide doses for winter wheat provide a sound basis for future testing although some modifications may need to be introduced. For example, it is essential to be certain of the anchor points of the curves (upper and lower assymptotes). The methodology used here had 9 replicates of the untreated control in each trial. The lower assymptote, however, was based on 3 replicates of the full dose. Additionally the full dose does not always achieve the greatest reduction in disease possible. Thus, inclusion of a higher dose (e.g. 2 x the full dose = 2N) may be required. Such testing of high doses is required by agrochemical companies to ensure crop safety.

It may be possible to reduce the number of doses that require to be evaluated as a reasonably accurate description of the shape of the curve can be obtained with knowledge of just 0, 0.25 and 1.0 doses.

When carrying out comparative tests such as those described here, attention must be given at each site to possible fungicide insensitivity. Ideally, isolates of the pathogens present at each site should be tested for insensitivity to the fungicides evaluated using known susceptible and resistant standards. This is, of course, costly and it may be sufficient just to be aware of trends in insensitivity across the country.

Fig. 20. Response curves for four fungicide doses and untreated control in relation to brown rust resistance rating

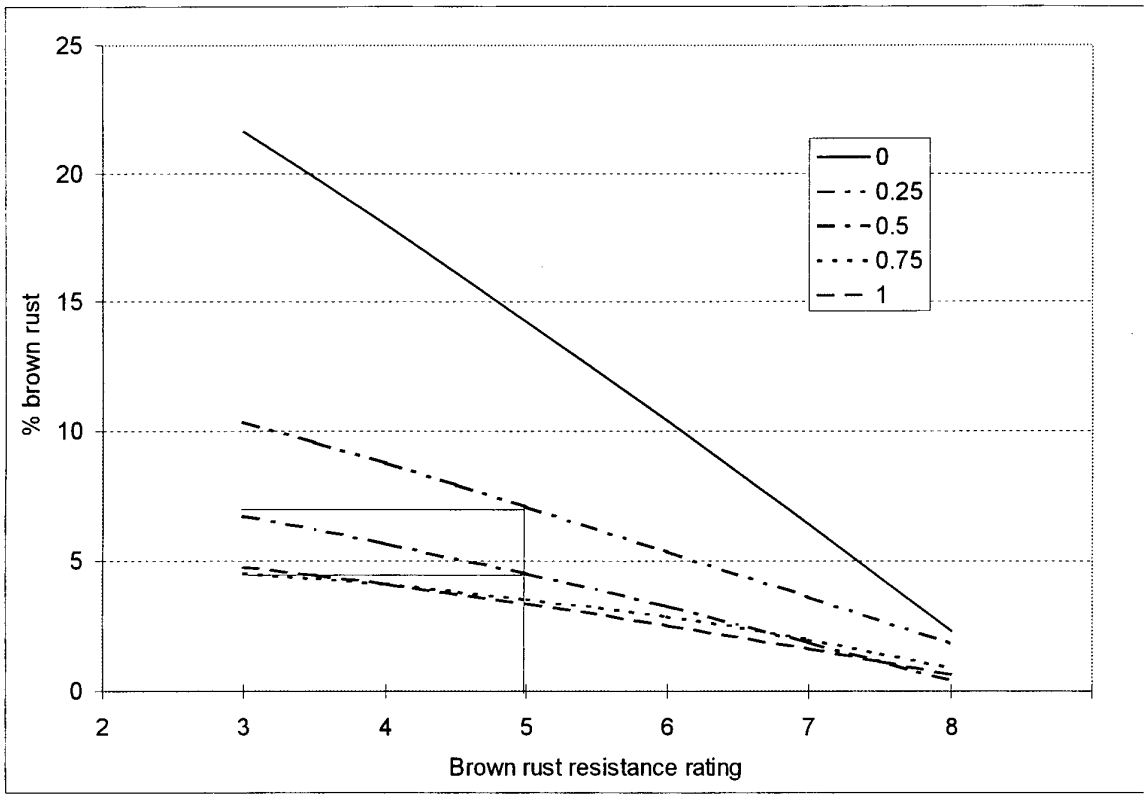


Table 18. Parameters for response curves in Figure 18.

Dose	a	b	r	R^2
0	152	-120	1.028	41.5
0.25	78	-63	1.024	55.5
0.5	17	-7.7	1.101	25.5
0.75	6.2	-0.83	1.261	22.2
1.0	11.9	-5.4	1.096	43.5

Fig. 21. Response curves for four fungicide doses and untreated control in relation to net blotch resistance rating

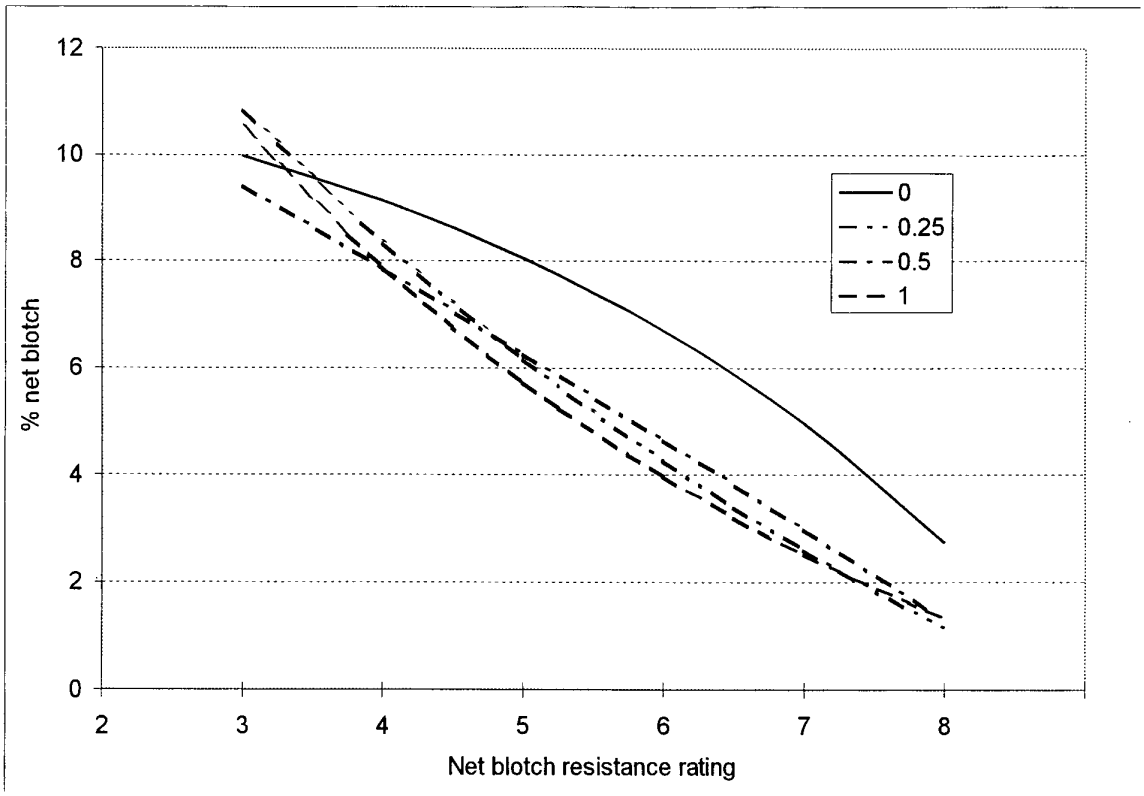


Table 19. Parameters for response curves in Figure 19.

Dose	a	b	r	R^2
0	13.1	-1.52	1.271	55.0
0.25	-8.7	29.48	0.872	85.5
0.5	102	-88	1.017	83.1
0.75	9.5	-0.37	1.45	17.4
1.0	-4.04	26.50	0.819	91.6

The variety response curves produced are also the first to demonstrate the extent to which resistance can be used to reduce fungicide dose in barley. Whilst there are fungicides that are more effective than the standard used, a knowledge of their relative effectiveness obtained from the fungicide dose response experiment should allow the curves to be re-drawn with this in mind.

The degree to which the graphs can be relied on depend on the stability of the pathogen population. If new races develop that overcome resistance, a variety will appear more susceptible than the rating suggests. The role of the UK Cereal Pathogen Virulence Survey in identifying new potentially damaging races is thus pivotal in ensuring growers are aware of potential changes in resistance.

Because there are risks that new races develop that overcome resistances and pathogens may develop insensitivity to fungicides, there will always be the need to regularly inspect crops for disease. Although the data from projects such as this are being incorporated into Decision Support Systems (Wale, 1998), there will always be the need for final decisions on fungicide use to be made with reference to what is found in the field.

REFERENCES

Anon. (1998) HGCA Winter Barley Appropriate Fungicide Dose Project. Final Report Experiment 3.

Griffin, M.J. (1994) The research response to current needs for cost-effective disease control - the latest results. Proceedings of Home-grown Cereals Authority 1994 Conference on Cereals R&D.

Jones, D.R. (1990) Sensitivity of *Rhynchosporium secalis* to DMI fungicides. Proceedings Brighton Crop Protection Conference 1990 - Pests and Diseases, 1135-1140.

Kendall, S.J., Holloman, D.W., Cooke, L.R., Jones, D.R. (1993) Changes in sensitivity to DMI fungicides in *Rhynchosporium secalis*. (Crop Protection **12**: 357-362.

Paveley, N.D., Stevens, D.B., Hims, M.J., Ainsley, A.N. (1998) Canopy, disease and fungicide use. Proceedings of the 6th HGCA R&D Conference. Home-Grown Cereals Authority, London.

Taggart, P.J., Cooke, L.R., Mercer, PC., Shaw,M.W. (1998) Effects of fungicides used to control *Rhynchosporium secalis* where benzimidazole resistance is present. Crop Protection 17, 727-734.

Wale, S.J. (1998) Appropriate fungicide dose selection in a spring barley decision support module. Proceedings Brighton Crop Protection Conference 1998 - Pests and Diseases

Wale, S.J., Murray, Fiona, Paveley, N.D., Jones, D.R., Hims, M.J., Stevens, D.B., Jones, O., Reid, Carol (1999) The use of fungicide dose-response curves in optimising winter barley disease control. Proceedings Crop Protection in northern Britain 1999. 55-60.

ACKNOWLEDGEMENTS

Funding for this work on the appropriate fungicide doses for winter barley by the Home-Grown Cereals Authority is gratefully acknowledged. Thanks are due to colleagues and field staff in the collaborating organisations for all their efforts in carrying out the field trials and reporting so timeously, to the staff at CSL and BioSS for statistical analysis and to colleagues in the parallel project on appropriate fungicide doses for winter wheat for many constructive and valuable discussions on the concept of appropriate fungicide doses.