DISEASE EPIDEMIOLOGY AND FUNGICIDE ACTIVITY IN WINTER WHEAT

M. J. HIMS

Central Science Laboratory, MAFF, Hatching Green, Harpenden, Herts AL5 2BD

R. J. COOK

Morley Research Centre, Wymondham, Norfolk NR18 9DB

ABSTRACT

Experiments were established in winter wheat crops to test the biological properties of a number of protectant, eradicant or curative fungicides active against leaf diseases. Each fungicide was applied once only to separate plots during a period of seven or eight consecutive weeks in May and June (GS32-75). Disease progress was assessed weekly on unsprayed and sprayed plots up to GS75-81. Leaf blotch caused by *Septoria tritici* occurred most frequently and severely across the 21 sites. Mildew, brown and yellow rust occurred at fewer sites. Plotting the area under the disease progress curves revealed marked differences between fungicides. It was possible to calculate the period of protectant and eradicant activity in thermal time (accumulated day °C above zero) for each of the fungicides and to identify the most effective timing(s) for application in relation to rainfall or the first record of disease.

INTRODUCTION

Disease control in UK winter wheat crops continues to rely on comprehensive spray programmes using broad-spectrum fungicides applied at one or more growth stages. CSL/ADAS National Winter Wheat Disease Surveys (Thomas, 1985-89; Polley, 1990-91) have shown that 89-95% of the crop receives one or more applications of fungicide. These may be applied at specific growth stages (surveys indicate that up to 35% of fungicides applied for leaf disease control may be applied too late for optimum effectiveness), on specific dates or as the weather allows rather than in response to disease thresholds or disease risk (percieved or forecast). This may lead to inadequate control, poor perception of fungicide performance and excessive use of fungicides. Moreover such applications frequently consist of broad-spectrum fungicides designed to achieve control of all the diseases that may occur. This leads to the needless use of one or more active ingredients in multi-component broad-spectrum fungicides or mixtures. In order to help users to make the best possible use of the fungicides currently available in the UK and also to avoid the unnecessary use of inappropriate components in broad-spectrum fungicides or mixtures, the biological properties, in relation to leaf disease control, of a range of fungicides were investigated. Jordan *et al.* (1986) have demonstrated the range of protectant, eradicant and curative properties of triazole fungicides when used for the control of *Septoria tritici* under glasshouse or somewhat artificial 'field' conditions. The series of experiments described here was designed to extend this work to field grown crops of winter wheat and to expand the range of fungicides challenged and leaf diseases encountered.

MATERIALS AND METHODS

During 1988 - 1990, a total of 21 experiments was established in field crops of winter wheat. Crops were chosen for the known disease susceptibility of their cultivars. An unreplicated linear design based on that of Thomas *et al.* (1989) was chosen in an attempt to engineer disease epidemics of increasing severity from both ends of the experiment (which received early applications of candidate fungicides) to the centre which received later applications. Fungicides were applied separately and only once to separate single plots at weekly intervals from early May (GS 32; Tottman, 1987) until GS75. Fungicides were selected from the following list: propiconazole, 125 g AI/ha ('Tilt' 250 EC, Ciba Geigy); fenpropimorph, 750 g AI/ha ('Corbel', BASF or 'Mistral', Rhone Poulenc); chlorothalonil, 1000 g AI/ha ('Bravo 500', ISK Europe); cyproconazole, 80 g AI/ha ('Alto 100' SL, Sandoz); prochloraz, 400 g AI/ha ('Sportak', Schering); triadimenol, 125 g AI/ha ('Bayfidan', Bayer); flusilazole 156.5 g AI/ha + carbendazim 78.25 g AI/ha ('Punch C', Du Pont). Also, in each experiment a series of plots received a sequence of up to four sprays from GS 30-31, in early to mid-April, the first of which was applied to each adjacent plot at successively later weekly intervals. These plots were treated with a mixture of fenpropimorph and prochloraz at the same rate as used for the single treatments except for fenpropimorph as a second or subsequent spray when the rate was reduced to 563 g AI/ha.

To monitor crop growth and leaf development, the topmost fully expanded leaf and each successive leaf was identified from GS31 onwards up to the emergence of the final leaf (leaf 1). Detailed disease assessments were made on unsprayed plots from GS31 onwards until GS75 to provide unhindered disease progress curves. Disease assessments were made on sprayed plots one week after the first and successive applications of each active ingredient and thereafter at weekly intervals until GS75.

Disease progress curves were plotted for each of the topmost four leaves from the date of their emergence within the crop for the unsprayed control plots and those sprayed with each of the fungicides. Plotted curves were examined along with rainfall incidence (imputed infection periods for *Septoria tritici*) or the first appearance of mildew or the rust diseases on each leaf layer in an attempt to identify the length of time for which each of the active ingredients was capable of protecting against or eradicating infection by the causal pathogens. The area under the disease progress curve (AUDPC) was calculated for each fungicide at each application date as well as the sequential sprays. Leaf disease caused by *Septoria tritici* was sufficiently severe to distinguish differences in the biological activity of the fungicides at several of the 21 sites but mildew, brown and yellow rust were sufficiently severe at only three or four sites. The AUDPC permitted the identification of the most effective timing(s) for disease control in terms of crop growth stage, leaf development or thermal time (mean day ^{o}C above zero). Data for each disease are presented from one 'example' site.

RESULTS

All fungicides gave control or partial control of the four leaf diseases. Timing of fungicide application was critical to achieve a degree of control approaching 95-100%. The four sites

varied in terms of crop and disease development with respect to calendar date as did the critical timing(s) for maximum disease control on the upper leaves.

Examination of the disease progress curves for the sprayed and unsprayed plots revealed the date and growth stage when mildew and the rust diseases first appeared as obvious disease on any leaf layer. Examination of the temporal distribution of on-site rainfall indicated the occurrence of one or more 'wet periods' likely to favour upward splash-dispersal of the pycniospores of *Septoria tritici* and subsequent leaf infection. The dates of these events were used as a basis for the calculation of protectant and eradicant activity of the fungicides against the four leaf diseases at the sites described below.

Adisham, Kent : Brown rust (1989) cv. Avalon

Brown rust was first noted in untreated control plots on 6 June (GS65). Thereafter it developed rapidly to colonise a maximum of 45% and 41% of leaves 1 and 2 respectively. Fig. 1 indicates that chlorothalonil gave very useful protection (*c.* 60% reduction in the AUDPC) against brown rust when applied at 370-175 day degrees prior to the first appearance of disease but thereafter the degree of protection declined markedly. The degree of protectant activity of fenpropimorph and the triazole fungicides varied between 95-100% for 370-175 day degrees but with the exception of cyproconazole and propiconazole protectant activity declined markedly at 75 day degrees. There was no indication of significant eradicant activity.

Dorchester, Dorset : *Septoria tritici* (1988) cv. Mercia

Two discrete 'splash events' occurred during late May (24-26: 31 mm and 28-30: 33 mm rain - GS59). This apparently led to the upward dispersal of pycniospores of *Septoria tritici* leading to infection and rapid development of the disease on the flag leaf giving 59% leaf area colonised at GS79-83 (29 June). Fig. 2 indicates that both chlorothalonil and propiconazole gave good protection (70-95% reduction in the AUDPC) when applied at 225-50 day degrees prior to the splash events. Chlorothalonil exhibited some eradicant activity (60% disease control) when applied 100 day degrees after the splash events; thereafter the degree of eradicant activity declined markedly. The excellent eradicant activity (> 75% reduction in the AUDPC) of propiconazole was maintained up to 175 day degrees after infection and even at 275 day degrees it was useful. Fenpropimorph exhibited little protectant activity; eradicant activity was very good at 100 day degrees but declined thereafter. The protectant activity of prochloraz varied between 60-75% at 125-0 and eradicant activity varied between 85-45% at 25-100 day degrees.

Rosemaund, Hereford : Mildew (1989) cv. Apollo

Mildew first appeared on the leaf 1 at GS55 (7 June). Thereafter it developed to colonise 14% at GS75 (5 July). Fig. 3 indicates that chlorothalonil was not active against mildew. Propiconazole exhibited no marked protectant activity unless applied no earlier than 175 day degrees prior to infection; its eradicant activity was *c.* 50% for 150 day degrees. Fenpropimorph and cyproconazole appeared superior to the other two fungicides both giving > 75% protectant activity at 150 day degrees prior to disease appearance and > 50% eradicant activity at 230 day degrees afterwards.

Terrington St Clement, Norfolk : Yellow rust (1989) cv. Slejpner

Yellow rust appeared on the flag leaf at GS59 (16 June). Thereafter, the disease developed rapidly to colonise 35% of the flag leaf at GS75 (30 June). Fig. 4 indicates that all fungicides exhibited a similar pattern of protectant and eradicant activity although the degree of control varied by 15-40%. Chlorothalonil gave excellent protectant control from 380 day degrees onwards but no eradicant control. Propiconazole and cyproconazole were similar or slightly superior to fenpropimorph in their protectant activity particularly at 460-350 day degree stage. However none of these three fungicides exhibited significant eradicant control of yellow rust.

DISCUSSION

CSL/ADAS National Surveys of Winter Wheat Diseases indicate that some crops with the highest final levels of disease are amongst those that received the greatest fungicide inputs; in 1991 24% of surveyed crops received three to seven fungicide applications. This implies that farmers applied the first fungicide too late or chose an inappropriate product and thereafter were attempting to control a disease that was too aggressive and too well established. Surveys also indicate that only 15% of crops received the first or second application of fungicide around flag leaf emergence (GS39) whereas 35% received it around ear emergence (GS59). Results from experiments reported here appear to indicate that for the majority of UK winter wheat crops GS59 may be too late for fungicide use to achieve maximum control on the upper leaves.

Clearly a number of UK winter wheat growers either do not appreciate the importance of correct fungicide timing and, therefore, may need to apply an extra fungicide for satisfactory disease control; or they are relying exclusively but perhaps unwittingly on the eradicant activity of the triazole and morpholine fungicides. This approach may be satisfactory if fungicides were used rationally but advisory experience and surveys indicate that this is infrequent.

For the four sites reported here the critical growth stage(s) at which the fungicides gave the greatest degree of disease control appeared to vary in relation to fungicide activity, the date of disease appearance (rather than infection) or splash events and the rate of epidemic development. With the exception of septoria at the Dorchester site where infection was clearly associated with one or two discrete rain splash events at GS59 the optimum timing of fungicides for the control of mildew and the rust diseases could not be related to infection events *per se*. For the purpose of calculating eradicant and protectant activity it may be more advisable to use the time of leaf emergence assuming the disease is established and active in the

---

Figs. 1-4 Graphs of percentage reduction in the area under the disease progress curve (AUDPC) caused by individual fungicides and the sequential spray programmes at the four sites: 1. Adisham (brown rust); 2. Dorchester (*Septoria tritici*); 3. Rosemaund (mildew); 4. Terrington (yellow rust)

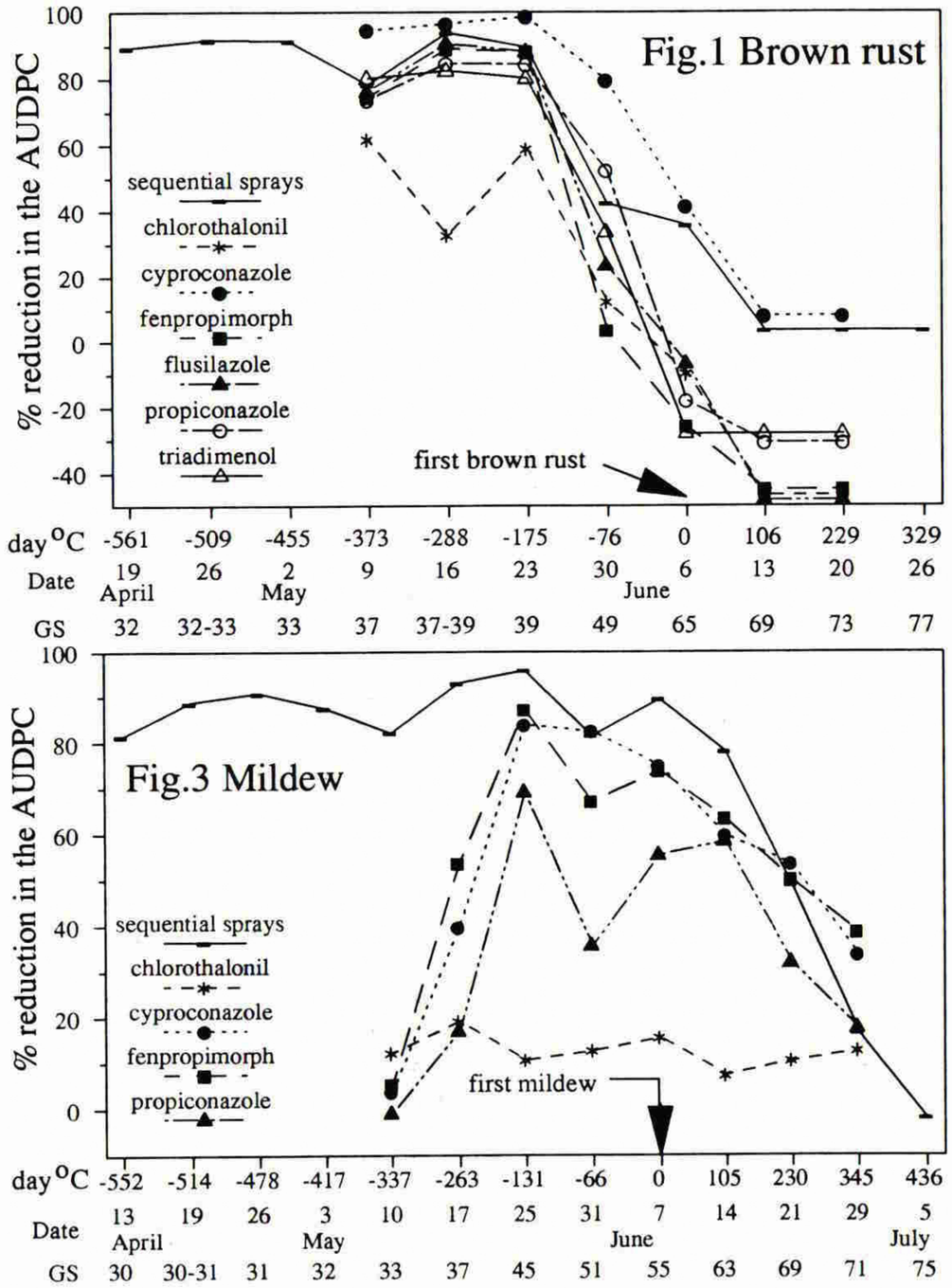
Fig.1 Brown rust
% reduction in the AUDPC
sequential sprays
chlorothalonil
cyproconazole
fenpropimorph
flusilazole
propiconazole
triadimenol
first brown rust
day °C -561 -509 -455 -373 -288 -175 -76 0 106 229 329
Date 19 April 26 2 May 9 16 23 30 6 June 13 20 26
GS 32 32-33 33 37 37-39 39 49 65 69 73 77

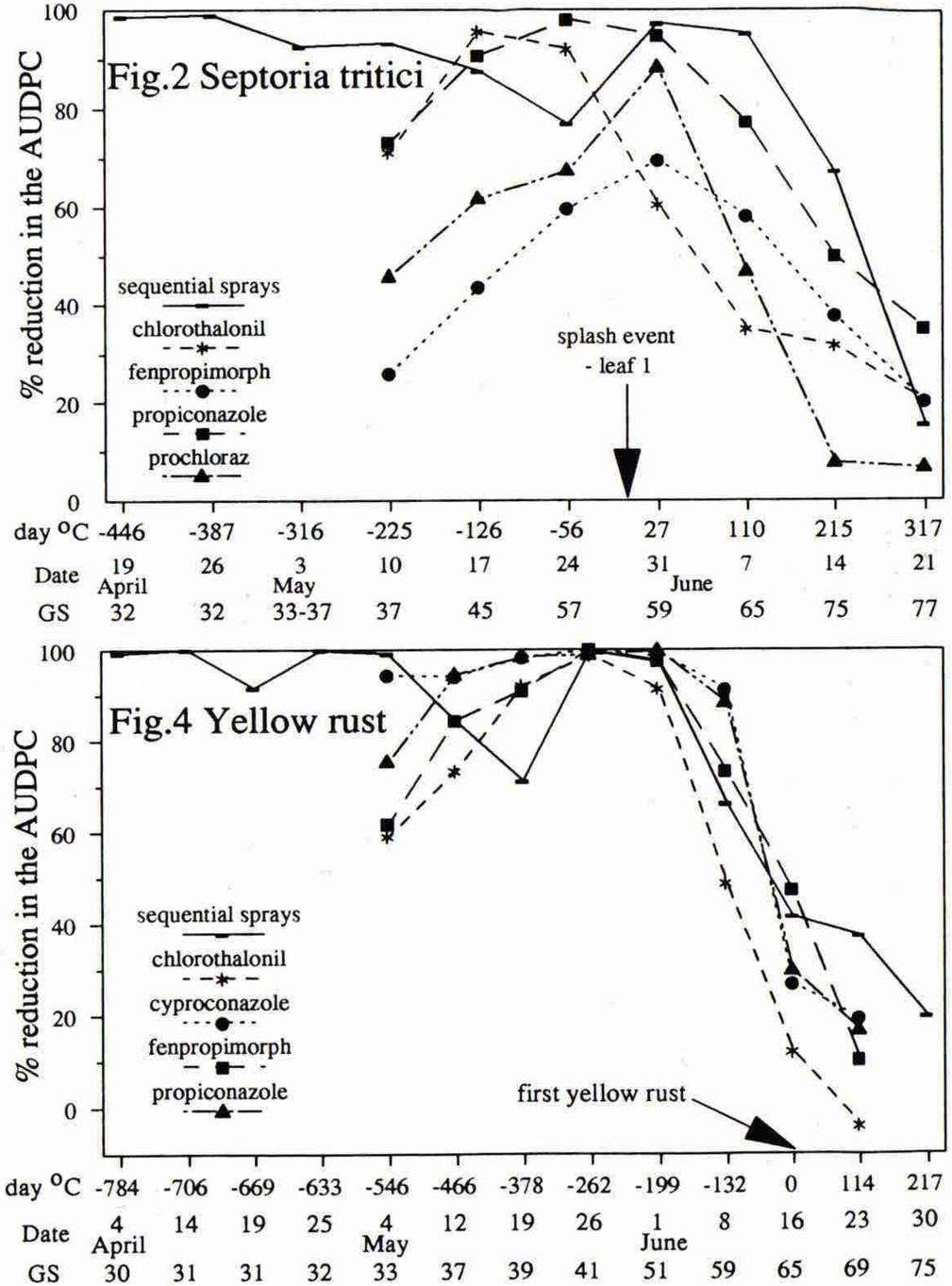
Fig.2 Septoria tritici
% reduction in the AUDPC
sequential sprays
chlorothalonil
fenpropimorph
propiconazole
prochloraz
splash event
- leaf 1
day °C -446 -387 -316 -225 -126 -56 27 110 215 317
Date 19 April 26 3 May 10 17 24 31 7 June 14 21
GS 32 32 33-37 37 45 57 59 65 75 77

Fig.3 Mildew
% reduction in the AUDPC
sequential sprays
chlorothalonil
cyproconazole
fenpropimorph
propiconazole
first mildew
day °C -552 -514 -478 -417 -337 -263 -131 -66 0 105 230 345 436
Date 13 April 19 26 3 May 10 17 25 31 7 June 14 21 29 5 July
GS 30 30-31 31 32 33 37 45 51 55 63 69 71 75

Fig.4 Yellow rust
% reduction in the AUDPC
sequential sprays
chlorothalonil
cyproconazole
fenpropimorph
propiconazole
first yellow rust
day °C -784 -706 -669 -633 -546 -466 -378 -262 -199 -132 0 114 217
Date 4 April 14 19 25 4 May 12 19 26 1 June 8 16 23 30
GS 30 31 31 32 33 37 39 41 51 59 65 69 75

crop. This approach would be satisfactory for mildew since it almost invariably develops steadily within the crop canopy at least up to GS39 when the disease may develop more rapidly in the closed canopy. For the rust diseases this approach is not possible in most situations since they often develop rapidly and without the prior warning of inoculum present on the older leaves. Thus the calculation of the apparent protectant and eradicant activity of fungicides for the rust diseases may be, at least for the present, related to the first appearance of the disease in the crop. Unfortunately this approach inevitably biases the results in favour of protectant activity. It is only too evident from Figs 1 and 4 that there is a period around GS37-51 which is critical for the optimum control of both brown and yellow rust although neither disease was apparent within the crop for a further 14-21days. If this critical period could be identified readily then it would be possible to calculate far more accurately the length of the period of protectant activity and, more important still, the length of the period of eradicant activity; in addition the rate of increase and the rate of decline of this biological activity could be calculated more accurately. Clearly if we are to maximise disease control from fungicide application(s) then we need a far better understanding of disease epidemiology and the influence of meteorological factors both in their own right and also in relation to fungicide activity.

The data presented give a partial illustration of the potential for maximising disease control from a single application of a single fungicide. There are now available fungicides which present considerable opportunity for flexible use either alone or in mixture. However to be used effectively their use must be based on a sound knowledge of their protectant and eradicant properties, disease risk and the influence of meteorological factors on disease development. Disease forecasting systems that also take account of biological activity in relation to infection events and subsequent disease development could be valuable aids to optimise fungicide inputs to winter wheat and possibly other crops.

ACKNOWLEDGEMENTS

We are grateful to MAFF Pesticides Safety Division and HGCA for financial support and to colleagues in ADAS and CSL who conducted each of the experiments cited.

REFERENCES

Jordan, V. W. L., Hunter, T. & Fielding, E. C. (1986) Biological properties of fungicides for control of *Septoria tritici*. *1986 British Crop Protection Conference - Pests and Diseases*, **3**,1063-1069.

Polley, R. W. (1990-1991) *Winter wheat disease survey 1990-1991*. Agicultural Development and Advisory Service, MAFF, Harpenden.

Thomas, M. R. (1985-1989) *Winter wheat disease surveys 1985-1989*. Agicultural Development and Advisory Service, MAFF, Harpenden.

Thomas, M. R., Cook, R. J. & King, J. E. (1989) Factors affecting development of *Septoria tritici* in winter wheat and its effect on yield. *Plant Pathology*, **38**, 246-257.

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

REDUCTION IN THE WHEAT EAR DISEASE COMPLEX WITH TEBUCONAZOLE SPRAYS

A. WAINWRIGHT, J. JEITNER, P. CAZIN-BOURGUIGNON

Bayer plc, Elm Farm Development Station, Great Green, Thurston, Bury St. Edmunds, Suffolk, IP31 3SJ

ABSTRACT

In field trials on winter wheat, sprays containing tebuconazole at 250 g/ha applied after ear emergence resulted in brighter ears than on untreated plots as the crop ripened. After ear samples were taken from plots and washed to remove spores, it could be seen that treatments had reduced spore counts in *Alternaria* spp., *Cladosporium* spp., *Botrytis cinerea, Fusarium* spp. and *Septoria tritici*. The results suggested that these fungi present on ripening ears were directly affected by tebuconazole and their reduction was not solely due to delayed senescence.

INTRODUCTION

Tebuconazole is a broad spectrum azole fungicide, its basic technical data having been given by Reinecke *et al.* (1986) and its chemistry and mode of action described by Berg *et al.* (1987). Various formulations including seed treatments and foliar sprays have been tested in the UK and the results of trials carried out on cereals and oilseed rape between 1984-1986 were reported by Wainwright and Linke (1987). In many of the early trials on winter wheat, a marked increase in ear brightness or cleanliness was observed on the senescing crop following spray treatment with tebuconazole after ear emergence. At that time, it was believed that the effect seen in the field had resulted largely from control of *Leptosphaeria (=Septoria) nodorum* and *Botrytis cinerea* although it was perceived that other fungi of the ear disease complex may have been involved. Subsequent trials with tebuconazole on cereals examined earlier-applied sprays (i.e. before ear emergence) against specific foliar pathogens and the ear cleanliness effect was not normally seen until 1991, when again, later-applied sprays were tested. A simple scoring procedure was used to assess ear brightness in the field. However, a clear estimate of the range and quantities of the main species of the flora was required. This paper describes a spore-washing technique used to estimate fungi present on wheat ears and the effect of tebuconazole treatment.

METHODS AND MATERIALS

Tebuconazole was applied in emulsifiable concentrate formulations containing either tebuconazole alone (250 EC applied at 1 l/ha) or tebuconazole and triadimenol (250 + 125 EC applied at 1 l/ha). In one trial, a treatment of fenpropidin (750 EC) was included at 1 l/ha.

Sprays were applied using pressurised knapsack sprayers with flat fan nozzles, water volumes of 200-300 l/ha and pressures of 200-300 kPa. Normally one, but up to 3 applications were made per treatment, and all included a final spray after ear emergence, between GS 59-79. Of the 9 trials reported, 7 were located in Suffolk with one each in Lincolnshire

and Kent. Plot size was 2-3 m x 15 m except for R3/91 where the 9 m x 12 m plots formed part of a long-term study. Because of the nature of R3/91, a wheat crop followed wheat, and the same plots could be treated and assessed in successive years. Trials were non-replicated or replicated up to four times.

In assessing ear brightness in the field, a 0-3 scale was used where grade 0 = no effect, i.e. plot appeared the same as untreated, and grade 3 = very marked effect.

For ear washing, 10 dry ears were taken from each plot, and five were placed in each of two 100 ml glass jars. A 50 ml quantity of distilled water was added to each jar together with two drops of 'Tween 20' wetting agent. The jars were shaken for a standard 10 min using a Gallenkamp flask shaker set at full speed. Approximately 50 ml of spore suspension was decanted from each jar and stored until assessment. Spores were counted in five samples of 0.1 µl suspension per jar using a counting chamber under the microscope. The combination of counts from two jars thus gave the number of spores present in 1 µl suspension.

Spore numbers for *Botrytis cinerea* in the suspensions tended to be low, hence a sample of 10 ears per plot were incubated for between 2-7 days in a moist chamber under near u.v. light at 18°C prior to the ear washing procedure. For trial FA/28/92, the method was improved by concentrating the spore suspension using a bench centrifuge, obviating the need to incubate ears for *B. cinerea*.

Samples of ears were taken from replicated trials where possible, and where three or more treatments and replicates were assessed (trial FA/27/91 only), an analysis of variance was applied to the data. The length of time taken for spore counting placed a restriction on the number of plots that could be assessed.

RESULTS

In the trials surveyed, spores of *Alternaria* spp. and *Cladosporium* spp. were most consistently found (Tables 1-6) and tebuconazole treatments generally resulted in 40-70% reduction.

Spore counts from three similar trials which each received a three-spray programme are given in Table 1. A correlation between reduced spore numbers (particularly *Alternaria* spp. and *Cladosporium* spp.) and ear cleanliness could be seen. This reduction is probably due to the later (i.e. post-ear emergence) applied sprays and this was borne out by spore counts from trial R3/91 where similar results were obtained regardless of earlier sprays (Table 2).

TABLE 1. Spore counts (spores/µl) from three trials following a programme of sprays applied at GS 32, 39 and 61.

Trial no.,location...	FA32/91, Suffolk		FA33/91, Suffolk		FA34/91, Lincs	
Cv.,replicates...	Avalon,3		Riband,3		Riband,2	
Assessed...	02.08.91		02.08.91		14.08.91	
Treatment*...	Untr	Teb + tri	Untr	Teb + tri	Untr	Teb + tri
Alternaria spp.	110	36	76	37	37	23
Cladosporium spp.	127	63	127	80	135	94
Botrytis cinerea	173	109	118	88	0	0
Fusarium spp.	20	16	26	18	8	26
Septoria nodorum	14	0	2	1	5	0
Septoria tritici	33	4	85	7	11	0
'Brightness' score	0	3	0	3	0	2

* Applied as tebuconazole at 250 g/ha + triadimenol at 125 g/ha.

TABLE 2. Spore counts (spores/µl) from trial R3/91, Suffolk.

Cv., Mercia. Replicates: 2

Assessed...	02.08.91 (45 DAT)		10.07.92 (39 DAT)			
Treatment*...	Untr	Teb	Untr	Teb	Teb	Teb
Applied at GS...		59		21+31+65	31+65	65
Alternaria spp.	67	21	28	5	6	3
Cladosporium spp.	94	40	154	31	54	23
Botrytis cinerea	187	42	31	15	17	15
Fusarium spp.	1	1	1	1	0	0
Septoria nodorum	15	1	1	0	0	0
Septoria tritici	84	3	17	9	3	7
'Brightness' score	0	3	0	3	3	3

* Applied as tebuconazole at 250 g/ha.

Single sprays were applied at GS 59 in trials FA/27/91 and FA/28/92 and again, reductions in spore numbers were observed (Tables 3 and 4). In FA/27/91 growth of *B. cinerea* was encouraged by incubating ears for 7 days, though this may have been too long, allowing over-production of spores leading to great variability. In other trials, the incubation period was less, at between 2-5 days, though latterly the preferred method involved centrifugation, giving a sufficiently concentrated spore suspension without the need for incubation.

Very high levels of *Fusarium* (mostly *F. culmorum*) were present in the wheat cultivars in trial TAS/09/91 (Table 5). Apparent differences in cultivar susceptibility were noted, but not proven statistically, and spore numbers were reduced by tebuconazole treatments.

TABLE 3. Spore counts (spores/µl) from trial FA/27/91, Suffolk.

Cv.: Boxer. Replicates: 4. Assessed: 05.08.91 (49 DAT).

	Untr	Teb*	*P*
Alternaria spp.	35	11	≤0.005
Cladosporium spp.	182	99	≤0.005
Botrytis cinerea	175	120	>0.05
Fusarium spp.	21	9	>0.05
Septoria nodorum	5	1	>0.05
Septoria tritici	11	7	>0.05
'Brightness' score	0	2	-

* tebuconazole applied at 250 g/ha, at GS 59.

TABLE 4. Spore counts (spores/µl) from trial FA/28/92, Suffolk.

Cv.: Apollo. Replicates: 3 Treated: 01.06.92 (GS 59).
Assessed: 20.07.92

	Untr	Tebuconazole (250 g/ha)	Fenpropidin (750 g/ha)
Alternaria spp.	37.9	19.2	30.3
Cladosporium spp.	206.9	101.0	179.1
Botrytis cinerea	4.0	1.4	1.8
Fusarium spp.	50.1	29.2	54.9
Septoria nodorum	0.2	0.1	0.0
Septoria tritici	10.6	3.0	8.1
'Brightness' score	0	3	0

TABLE 5. Spore counts (spores/µl) from non-replicated cultivar trial TAS/09/91, Kent.

Treated: GS 65, assessed: 08.08.91 (35 DAT).

Cv. ...	Beaver		Haven		Hereward		Riband	
Treatments*...	Untr	Teb + tri	Untr	Teb + tri	Untr	Teb + tri	Untr	Teb + tri
Alternaria spp.	101	54	53	20	74	15	75	9
Cladosporium spp.	226	174	241	95	150	93	131	74
Botrytis cinerea	116	36	214	80	32	22	212	96
Fusarium spp.	13125	7125	9000	2019	11750	6856	1103	639
Septoria tritici	130	15	123	10	15	11	121	16
'Brightness' score	0	1	0	3	0	2	0	2

* Applied as tebuconazole at 250 g/ha + triadimenol at 125 g/ha.

In E1/91, (Table 6) tebuconazole/triadimenol was applied at GS 79 when the crop was no longer green. Despite the lateness of the treatment, the ears appeared markedly cleaner than the control and considerable reductions in spore numbers were recorded.

TABLE 6. Spore counts (spores/µl) from trial E1/91, Suffolk.

Cv.: Mercia, non replicated Treated: GS 79
Assessed: 20.08.91 (28 DAT)

	Untr	Teb + tri*
Alternaria spp.	70	34
Cladosporium spp.	86	54
Botrytis cinerea	242	46
Fusarium spp.	11	2
Septoria nodorum	4	0
Septoria tritici	33	8
'Brightness' score	0	2

* Tebuconazole applied at 250 g/ha + triadimenol at 125 g/ha.

DISCUSSION

The performance of fungicides in cereals has been monitored largely against the pathogens which have the greatest impact on crop yield. However, in addition to these organisms, a microflora of weak pathogens and saprophytes can be found on senescing leaves and ears which affect grain quality and may reduce yield (Wiese, 1987; Magan and Lacey, 1986; King, *et al.*, 1981). Complex relationships exist between these organisms in the colonisation of ripening ears (Dickinson, 1981), but clearly the ingress of such fungi would be disrupted by fungicides which reduced the earlier pathogens and prolonged the presence of green tissue. Such an indirect control would most likely result from tebuconazole as its effectiveness against mildew, rusts and *Septoria* spp. is well known (Wainwright and Linke, 1987; Heatherington and Meredith, 1988). However, the outstanding cleanliness of ears would suggest an additional direct effect and this is supported by the reduction in spores seen in E1/91, where sprays were applied too late to delay senescence.

In addition to *Alternaria* spp. and *Cladosporium* spp., which were consistently found in all trials, numbers of spores of *S. tritici* were also reduced by tebuconazole treatment. Unlike *S. nodorum, S. tritici* does not produce distinctive lesions on the glumes (Jones and Cooke, 1969). However, the presence of brown to black pycnidial lines on the glumes would lead to a duller appearance of infected ears.

Botrytis and *Fusarium* spp. tended to give more variable spore counts and larger ear samples together with further replication would have been preferable for assessments. Tebuconazole is known to control *Botrytis* in vines (Kaspers *et al.*, 1987), and the results obtained here confirm

effectiveness on wheat glumes also. *Fusarium* spp. are particularly important pathogens of wheat ears, capable of producing mycotoxins and infecting seed grain (Jenkins *et al.*, 1988). Hutcheon and Jordan (1992) have demonstrated control of *Fusarium* spp. with tebuconazole (as UK264) under glasshouse conditions and the results reported here suggest useful reductions could be obtained in the field.

The results of this work have demonstrated that tebuconazole can, as a side effect, reduce fungi of the late ear disease complex in addition to controlling the major foliar and ear pathogens reported elsewhere. Clearly there is scope for further work, for example the identification of an optimum application time and the checking of harvested grain for mycotoxin levels.

REFERENCES

Berg, D.; Born, L.; Buchel, K-H.; Holmwood, G.; Kauler, J. (1987) HWG 1608 - Chemistry and biochemistry of a new azole fungicide. *Pflanzenschutz-Nachrichten Bayer*, **40**, 111-132 (English edition).

Dickinson, C.H. (1981) Leaf surface micro-organisms as pathogen antagonists and as minor pathogens. In *Strategies for the control of cereal diseases*, J. F. Jenkyn and R. T. Plumb (Eds), Oxford: Blackwell Scientific Publications, pp. 109-121.

Heatherington, P.J.; Meredith, R.H. (1988) Disease control with HWG 1608 on cereals and rape. *Brighton Crop Protection Conference - Pests and Diseases 1988*, **3**, 953-958.

Hutcheon, J.A.; Jordan, V.W.L. (1992) Fungicide timing and performance for Fusarium control in wheat. *Brighton Crop Protection Conference - Pests and Diseases 1992*.

Jenkins, J.E.E.; Clark, W.S.; Buckle, A.E. (1988) *Fusarium Diseases of Cereals. HGCA Research Review No. 4.*, 89 pp.

Jones, D.G.; Cooke, B.M. (1969) The epidemiology of *Septoria tritici* and *S. nodorum*. I. A tentative key for assessing *Septoria tritici* infection on wheat heads. *Transactions of the British Mycological Society*, **53** (1), 39-46.

Kaspers, H.; Brandes, W.; Scheinpflug, H. (1987) Improved control of crop diseases with a new azole fungicide, HWG 1608 (R Folicur, R Raxil). *Pflanzenschutz-Nachrichten Bayer*, **40**, 81-110.

King, J.E.; Evers, A. D.; Stewart, B. A. (1981) Black point of grain in spring wheats of the 1978 harvest. *Plant Pathology*, **30**, 51-53.

Magan, N.; Lacey, J. (1986) The phylloplane microflora of ripening wheat and effect of late fungicide applications. *Annals of Applied Biology*, **109**, 117-128.

Reinecke, P.; Kaspers, H.; Scheinpflug, H.; Holmwood, G. (1986) BAY HWG 1608, a new fungicide for foliar spray and seed treatment use against a wide spectrum of fungal pathogens. *1986 British Crop Protection Conference - Pests and Diseases*, **1**, 41-46.

Wainwright, A.; Linke, W. (1987) Field trials with R Folicur and R Raxil against foliar and seedborne cereal disease, and observations on the control of rape diseases with Folicur in Great Britain (1984-1986). *Pflanzenschutz-Nachrichten Bayer*, **40**, 181-212 (English edition).

Wiese, M.V. (1987) *Compendium of Wheat Diseases*, Second edition 1987, St Paul: The American Phytopathological Society, 112 pp.

MYCLOBUTANIL SEED TREATMENT FOR CEREAL SEED BORNE DISEASE CONTROL IN THE UK

MALCOLM C. E. GREEN

DowElanco Limited, Latchmore Court, Brand Street, Hitchin Herts. SG5 1HZ

NICHOLAS GOOCH

Rohm and Haas (UK) Limited, Lennig House, 2, Mason Avenue, Croydon, Surrey, CR9 3NB

ABSTRACT

The removal of the derogation allowing the use of organo-mercury compounds as a seed treatment in the United Kingdom has given renewed impetus to the consideration of other materials for the control of seed borne diseases of cereals. The paper covers the performance of myclobutanil in controlling the major seed borne diseases of cereals in the UK.

INTRODUCTION

On the 31st of March 1992 the derogation from the EEC that had allowed the continued use of mercury seed treatments for cereals in the UK was finally revoked. This signalled the end of the what has been one of the most successful uses of a crop protection product in the UK. Widespread use of mercury for the control of seed borne diseases of cereals dates back to the 1920s; since then it has successfully controlled the major seed borne diseases of cereals. These diseases, notably common bunt (*Tilletia caries)*, covered smut of barley (*Ustilago hordei)*, and leaf stripe (*Pyrenophora graminea)*, have been so well controlled that they have been referred to as the forgotten diseases of cereals. With the loss of mercury, which has provided exceptionally cost effective control of these diseases, the farmer is faced with a choice of alternative products. Some of these newer products offer levels of disease control comparable to that achieved with mercury with little difference in performance, notably the materials based on guazatine and carboxin. Others that have been introduced more recently control a wider range of diseases, for example loose smut (*Ustilago nuda)*, and offer additional growth regulatory benefits and control of early attack by foliar disease. Such materials tend to be based on triazole chemistry.

Myclobutanil ('Systhane'*) a triazole compound from the Rohm and Haas company had been initially developed in the UK as a foliar applied material for the control of apple powdery mildew (*Podosphaera leucotricha*) and apple scab (*Venturia inaequalis*) (Orpin *et al.*, 1986). It has also been developed in mixtures for the control of seed borne diseases of cereals in several continental countries (Efthimiadis, 1988). In recent years because of the need to find suitable alternatives to mercury based seed treatments the material has been developed as a seed treatment for use in the UK.

* Trade mark of Rohm and Haas

MATERIALS AND METHODS

The results detailed in this paper have been obtained from trials carried out in the UK by DowElanco. The majority of the work was carried out following the experimental guidelines of the International Seed Testing Association (I.S.T.A.), and the European Plant Protection Organisation (E.P.P.O.). All field trials were conducted over the period 1986 to 1992. Trial design was as randomised blocks in all cases with four replicates. RH3866ST (myclobutanil) was used at the proposed recommended rate of 12.5 g AI /100 kg seed, reference materials were used at their relevant UK commercial rate.

Soil borne bunt control was evaluated by mixing 20 g of bunt spores with 1 kg compost. Appropriately treated seed was then placed in this contaminated medium and allowed to grow in the glasshouse.

RESULTS

The tables show the effects of RH3866ST and relevant standards on plant emergence and vigour (Tables 1-4), on plant growth (Table 5) and on disease control (Tables 6-11).

TABLE 1. Plant emergence counts in winter wheat (no. plants / m row).

Treatment	Rate (g AI / 100 kg)	Site 1	Site 2	Site 3
Untreated	0	44.3	9.5	13.2
RH3866ST	12.5	50.3	36.9	37.9
Triadimenol / fuberidazole	37.5 / 4.5	47.5	38.1	40.4
Mercury	2.2	42.9	41.2	43.0

TABLE 2. Plant vigour in winter wheat as a percentage of the untreated, consideration given to plant height, colour and development.

Treatment	Rate (g AI / 100 kg)	Site 1		Site 2		Site 3	
		Zadoks 11	Zadoks 12	Zadoks 11	Zadoks 12	Zadoks 11	Zadoks 12
Untreated	100	100.0	100.0	100.0	100.0	100.0	100.0
RH3866ST	12.5	100.0	106.7	77.5	100.0	85.0	98.5
Triadimenol/ fuberidazole	37.5 / 4.5	85.0	106.7	67.5	100.0	70.0	99.0
Mercury	2.2	113.8	113.3	100	100.0	100.0	100.0

TABLE 3. Plant emergence counts in winter barley (no. plants /m row).

Treatment	Rate (g AI / 100 kg)	Site 1	Site 2	Site 3
Untreated	0	44.3	44.0	44.5
RH3866ST	12.5	43.3	41.8	40.0
Flutriafol / ethirimol / thiabendazole	15 / 200 / 5	42.0	43.3	42.8
Mercury	2.2	43.8	45.0	46.3

TABLE 4. Plant vigour in winter barley as a percentage of the untreated, consideration given to plant height, colour and development.

Treatment	Rate (g AI / 100 kg)	Site 1 Zadoks 11	Site 1 Zadoks 12	Site 2 Zadoks 11	Site 2 Zadoks 12	Site 3 Zadoks 11
Untreated	100	100.0	100.0	100.0	100.0	100.0
RH3866ST	12.5	100.0	97.8	99.62	98.3	99.8
Flutriafol / ethirimol /thiabendazole	15/ 200 / 5	85.0	97.8	67.5	100.0	70.0
Mercury	2.0	113.8	100.0	100.0	100.0	98.7

TABLE 5. Sub-crown internode length (mm) in winter wheat.

Treatment	Rate (g AI / 100 kg)	Site 1	Site 2	Site 3
Untreated	0	8.6	12.4	13.4
RH3866ST	12.5	6.6	7.7	13.7
Triadimenol / fuberidazole	37.5 / 4.5	5.2	4.1	12.9
Mercury	2.2	10.9	15.9	13.0

TABLE 6. Percentage control of seed borne common bunt in winter wheat (Untreated, percentage ears infected).

Treatment	Rate (g AI 100 kg)	Site 1	Site 2	Site 3	Site 4
Untreated	0	80	68.7	74.1	75.8
RH3866ST	12.5	100	100	100	100
Guazatine	60	100	93.3	95.2	98.7
Triadimenol / fuberidazole	37.5 / 4.5	100	100	100	100
Mercury	2.2	100	84.8	93.7	93.0

TABLE 7. Percentage control of soil borne bunt in spring wheat (Untreated, no. of infected ears per pot).

Treatment	Rate (g AI / 100 kg)	Control
Untreated	0	8.3
RH3866ST	12.5	91
Guazatine	60	48
Carboxin / thiabendazole	90 / 5	0
Triadimenol / fuberidazole	37.5 / 4.5	58

TABLE 8. Percentage control of loose smut in winter barley (Untreated, no. of infected ears / m^2).

Treatment	Rate (g AI / 100 kg)	Site 1	Site 2	Site 3	Site 4
Untreated	0	79	56	14.5	11.2
RH3866ST	12.5	100	100	95	95
Flutriafol / ethirimol / thiabendazole	15 / 200 / 5	-	-	100	96
Carboxin / imazalil / thiabendazole	60 / 4 /5	99	98	45	95
Mercury	2.2	42	48	-	-

TABLE 9. Percentage control of leaf stripe in spring barley (Untreated, percentage infection on leaf three).

Treatment	Rate (g AI / 100 kg)	Site 1	Site 2
Untreated	0	73.8	4.6
RH3866ST	12.5	100	99.4
Flutriafol / ethirimol / thiabendazole	15 / 200 / 5	100	-
Guazatine/ imazalil	66 / 5.5	100	-
Mercury	2.2	25	92.5

Seed with varying levels of fusarium infection was treated with a range of seed treatment products and a germination test carried out to the I.S.T.A. guidelines (Table 10).

TABLE 10. Percentage germination of fusarium infected winter wheat following application of a range of seed treatment products.

% Fusarium Infection	Untreated	RH3866ST	Guazatine	Triadimenol / fuberidazole	Flutriafol / thiabendazole
27	80.6	96.6	98.6	97.0	97.0
42	66.6	97.0	97.6	97.0	93.0
44	75.6	96.6	97.6	98.0	90.0
47	71.6	94.6	98.6	97.0	89.6
58	43.0	89.0	94.0	95.0	86.6
64	53.6	94.0	97.0	94.0	87.0
70	53.0	95.6	96.0	93.6	87.6

Winter wheat seed with a natural 20 % *Fusarium nivale* infection was drilled at several sites within the UK and assessments were made of level of fusarium infection at the base of the plant at growth stage Zadoks GS 12 (Table 11).

TABLE 11. Percentage winter wheat plants infected with *Fusarium nivale*.

Treatment	Rate (g AI / 100 kg)	Site 1	Site 2	Site 3	Site 4
Untreated	0	36.3	30.0	81.0	74.3
RH3866ST	12.5	1.3	10.0	50.2	44.2
Triadimenol / fuberidazole	37.5 / 4.5	1.3	3.3	22.8	18.1
Mercury	2.2	0	20.0	63.4	42.8

DISCUSSION

Selectivity

In common with the existing triazole based seed treatment products available in the UK RH3866ST exhibited a delay in emergence of the crop. This delay in emergence was associated with a reduction in the length of the sub-crown internode of the emerging seedling (Table 5). The result of this growth regulation is the development of a plant with a thicker stem going into the winter, the so called "spring onion" effect. A potential consequence of this is a reduction in tiller number and a greater potential for over winter survival of the plant.

Efficacy

In recent years there has been an apparent resurgence in the incidence of bunt; this has been largely associated with a reduction in seed treatment by farmers looking to reduce costs where ever possible. This has served to highlight the rapid speed with which this disease will re-appear if control measures are not used as a matter of course by farmers. As the results indicate (Table 6) RH3866ST will give excellent control of seed borne bunt. One potential consequence of non treatment for bunt control is

that of field failure; this will result in the return to the soil of a large number of bunt spores. Whilst still a relatively rare phenomenon in the UK this return of spores to the soil can result in soil borne bunt occurring. This is well documented in Canada (Denis *et al;* 1989) and certain other continental climates. The limited amount of glasshouse testing carried out on the product so far has demonstrated that there is control of soil borne bunt from RH3866ST comparable to that achieved from the "standard" product for this, the triadimenol / fuberidazole mixture (Table 7).

The systemic properties of RH3866ST enable the product to get within the seed embryo and give good control of loose smut, a more deeply seated disease. In the trials (Table 8)the level of control achieved with the product has been consistently good suggesting that the product may be suitable for seed retrieval for this particular disease.

In common with current seed treatments in the UK market based on triazole compounds RH3866ST gives excellent control of leaf stripe in spring barley as the results presented indicate (Table 9). When used for the control of this disease in winter barley the level of control is not as good with around 60 to 70 % being achieved. In recent years strains of leaf stripe resistant to mercury have appeared; the results of the trials with RH3866ST indicate that there is no problem with cross resistance to this compound.

Fusarium is undoubtedly an important disease, particularly in winter wheat, and one that may not have been given the attention it deserves in recent years. Mercury, whilst not necessarily giving complete control of the disease, has given sufficient control to ensure good crop establishment in the majority of cases. Surveys of fusarium in cereal crops have indicated the presence of large proportions of MBC-resistant material. In the post mercury era if widespread problems are to be avoided it is important that the farmer has alternative materials to the MBC generators for the control of fusarium. The results presented indicate that even with a modest 27% infection the associated reduction in germination of the seed takes it below the statutory limits for seed crops. The level of control of the fusarium from RH3866ST was sufficient to ensure that even where the level of infection was substantial the germination was above the requirements for certified seed. (Tables 10 & 11).

Work is currently underway to develop recommendations for the product on other cereal crops. RH3866ST is currently being appraised by the UK registration authorities and it is hoped that a registration will be available to allow sales in 1993.

REFERENCES

Efthimiadis, P. (1988) Myclobutanil seed treatment - A new approach to control cereal diseases. *Brighton Crop Protection Conference - Pests and Diseases* 1988, **3**, 887 - 893

Orpin, C.; Bauer, A.; Bieri, R.; Faugeron, J.M.; Siddi, G. (1986) Myclobutanil, a broad-spectrum systemic fungicide for use on fruit, vines and a wide range of other crops. *1986 British Crop Protection Conference - Pests and Diseases,* **1**, 55 - 62.

Denis, A. G.; Byron, J. P.; Toby, E. (1989) Effect of environment on efficacy of seed-treatment fungicides for control of common bunt in spring and winter wheat. *Pesticide Science,* **26,** 241-252.

FUNGICIDE TIMING AND PERFORMANCE FOR FUSARIUM CONTROL IN WHEAT

J.A. HUTCHEON, V.W.L. JORDAN

Department of Agricultural Sciences, University of Bristol, AFRC Institute of Arable Crops Research, Long Ashton Research Station, Bristol, BS18 9AF.

ABSTRACT

Infection of wheat ears through systemic colonisation of stem vascular tissues by four *Fusarium* spp. resulted in significant reductions in grain number, thousand grain weight and yield losses of between 5% and 19%, in the absence of visible ear symptoms. Most fungicide seed treatments gave initial reductions in incidence and leaf sheath penetration during autumn and early spring, but were insufficient alone to prevent lesion development on mature stems and provide yield benefits. Responses obtained from spray timings in relation to plant growth and disease development showed that early applications, up to GS 31, reduced stem colonisation and lesion severity, whereas those applied later in crop development gave more effective reductions in ear disease. Sprays applied to wheat ears inoculated at flowering with individual *Fusarium* spp. resulted in significant control of subsequent ear disease, with UK264 and prochloraz being the most effective of the eight fungicides evaluated.

INTRODUCTION

Four *Fusarium* spp. commonly attack winter wheat in the UK (*Fusarium avenaceum, F. culmorum, F. graminearum and F. nivale*). These species invade stem bases and ears and cause losses of yield and grain quality. All species can be seed- and soil-borne, and can survive in straw, soil organic matter and plant debris. Infection may arise directly from the seed or soil-borne hyphae, or from ascospores and conidia produced from diseased tissue, which are spread to upper parts of the plant by wind or rain splash (Jenkins *et al.*, 1988). However, there is little evidence to demonstrate the contribution of these various inoculum sources to aetiology and yield loss, thereby adding to the difficulty in formulating disease control strategies.

This paper examines the contribution of systemic stem vascular colonisation to disease development, and evaluates the control efficacy of seed treatments, foliar sprays and sprays applied to ears of wheat.

AETIOLOGY

Winter wheat, cv. Avalon, was grown in sterilised soil medium (6:4:2 loam, peat and grit) to which was added a slow release fertiliser (3g/l Osmocote-type). The soil was contained in plant troughs (57 cm x 14 cm x 13 cm) with seeds planted in two rows (10 cm apart) at 2 cm spacings within rows in October 1989 and grown to maturity with drip-feed irrigation in an unheated house with glass roof and gauze walls, to prevent infection from external sources. In April 1990 (GS 31) all plants received fertiliser ('Nitrochalk', 120 kg N/ha). At GS 21 (November), each plant was inoculated

by placing a 5mm mycelial agar disc of either *F. avenaceum*, *F. culmorum*, *F. graminearum* or *F. nivale*, just below soil level and in contact with each plant. There were nine replicate troughs for each pathogen, eight being used for yield determinations. At maturity, each plant trough was hand harvested, ear populations counted, and grain recovered by threshing in a Labor ear thresher. Grain number, thousand grain weight and yields were determined. One trough of each pathogen was used for destructive sampling as follows. At GS 37, each shoot apex from one row per trough was enclosed in a polythene sleeve, to protect the subsequently emerging ear from exogenous inoculum and to provide a favourable climate for later expression of ear disease. From the remaining row, 30 main stems were taken and all foliage was removed. A 5 mm portion of tissue from each internode and the developing ear excised from each stem, was surface sterilised in 5% hypochlorite solution, rinsed in sterile water and transferred to segmented potato sucrose agar plates. Pathogen recovery was identified following incubation at 18 ° C.

All four species were recovered from excised internode tissue and developing ears at GS 37. Disease developed in mature ears that were enclosed in polyethylene sleeves from GS 37, thereby demonstrating that ears may become infected through internal systemic colonisation (Table 1). All four *Fusarium* spp. caused losses in numbers of grains per ear, thousand grain weight and yield (Table 2), in the absence of visible ear symptoms.

TABLE 1. Pathogen recovery (expressed as % of tissue infected) from wheat stem internodes (basal, int. 2, int. 3, terminal) and developing ears (GS 37), and enclosed ears at GS 75.

Inoculant	Internode colonisation (%)					Enclosed
	basal	int.2	int.3	term.	ear	ear (%)
F. avenaceum	37	33	23	10	3	20
F. culmorum	27	27	10	3	3	23
F. graminearum	37	43	13	3	7	18
F. nivale	43	40	20	13	13	43

TABLE 2. Influence of systemic infection on grain number, thousand grain weight (TGW) and yield/100 ears of winter wheat.

Inoculant	No. grains/ear	Yield (g)	TGW (g)
F. avenaceum	38.97	145.35	36.82
F. culmorum	34.31	150.90	42.59
F. graminearum	37.76	171.54	44.25
F. nivale	34.32	152.38	43.94
Uninoculated	39.48	179.84	45.69
SED (df=28)	0.834	4.175	0.761

FUNGICIDE CONTROL OPTIONS

Evaluation of seed treatments

Seeds, cv. Mercia, treated with four commercially available seed treatments ('Baytan Flowable' - triadimenol 187.5 FS + fuberidazole 23, 'Rappor' - guazatine 300 LS, 'Cerevax' - carboxin 360 FS + thiabendazole 25, 'Vincit L' - flutriafol 25 + thiabendazole 25), four experimental seed treatments ('Sibutol FS'(UK158) - bitertanol 375 FS + fuberidazole 23,'Cereline' (UK160) - triadimenol 75 FS + bitertanol 188 + fuberidazole 23, 'Raxil T'(UK441) - tebuconazole 15 FS + thiram 500 and 'Raxil'(UK487) - tebuconazole 15 FS) and untreated seeds were sown in soil trays (60 cm x 40 cm x 10 cm) at field density in October 1991, and grown outside to maturity. Macerated straw, infected with *Fusarium* spp., was dispersed onto the soil surface after sowing and covered by a fine layer of course sand. Six replicate trays per treatment were arranged in a randomised block design for yield determination; two extra trays per treatment were used for destructive sampling throughout the year.

In February, March, April and May (GS 37), all plants were removed from three 25 cm lengths of row/treatment, and plant and tiller numbers, disease incidence and leaf sheath penetration were recorded; at GS 37, stem lesions and internal colonisation were also monitored. At GS 65 (June), visible ear disease was assessed *in situ*, and 20 ears incubated for confirmation of causal pathogens. At GS 75, internodal symptoms were assessed from a sample of 50 stems per treatment. At maturity, each tray was hand harvested and processed as described previously, for yield determination.

There were no adverse effects of treatments on plant establishment and tiller numbers during early spring. In March (GS 30) greatest reductions in infection occurred with 'Vincit' and UK441 treated seed (Table 3). By April (GS 31), 40% of infections on untreated plants had penetrated at least three leaf sheaths and 6% had reached stem tissues. Least disease was found for 'Cerevax' and UK441; the latter decreased sheath penetration. In May (GS 37), stem lesions had developed on the basal two internodes from all treatments. Most severe lesions developed on stems from untreated seeds (6% basal internodes fully girdled) but treatment differences were not distinct. *F. graminearum* was most frequently recovered, but colonisation had progressed to the developing ear only in stems from untreated seed. *Fusarium* spp. were not isolated from any other treatment above the third internode. At GS 65, all treatments reduced disease compared with untreated seed, but only UK158, UK160 and UK441 gave significant reductions in diseased ear area (Table 3). By GS 85, stem lesions were severe on basal internodes and had progressed to all internodes. Most treatments reduced severity on the topmost three internodes; UK487 was significantly more effective than all other treatments (Table 3). Although there were no significant effects of treatments on yield, most grain was obtained from UK487, the treatment with least stem lesion area at GS 85.

Evaluation of foliar spray timing

In a preliminary glasshouse experiment, with potted winter wheat seedlings, cv.Avalon, inoculated with mixed *Fusarium* spp., an experimental fungicide (UK264 tebuconazole 250EC - triadimenol 125) applied 24h before inoculation, 2 days, 6 days and 2 weeks post inoculation, and prochloraz applied 4 weeks post inoculation reduced stem lesion severity and ear infection, compared with unsprayed plants.

TABLE 3. Effects of seed treatments on post-emergence *Fusarium* infection and yield.

Treatment	Tillers infected		Ear inf(%)	Stem Severity#		Yield
	March	April	GS 65	Basal	Topmost	(t/ha)
UK158	40.2	75.8	5.7a	86.7cd	50.0bcd	4.53
UK160	35.4	65.3	7.6ab	81.9bc	56.3de	4.75
UK441	27.6	42.7	8.0ab	87.8d	50.3cd	4.76
UK487	33.8	73.9	9.4abc	71.8a	33.8a	5.35
'Baytan'	61.7	49.7	10.6abc	81.2b	73.9f	4.84
'Vincit'	28.4	56.0	12.2bc	85.0bcd	52.6d	4.76
'Rappor'	36.9	75.5	13.9bc	84.5bcd	42.0b	5.01
'Cerevax'	41.2	42.7	14.5bc	82.7bc	42.2bc	5.12
Untreated	58.0	79.4	16.7c	86.5bcd	62.6e	0.445
SED(df=40)						

Means followed by the same letter do not differ significantly (P = 0.05).
- mean lesion area (%) from 3 basal and 3 topmost internodes and GS 85.

Supplementary to this experiment, winter wheat plants inoculated as seedlings and grown in large pots (12 plants/pot) were maintained in a protected environment until maturity. UK264 was applied to five replicate batches at GS 49 and GS 69, and to further previously uninoculated batches that were inoculated with a mixed conidial suspension of the four *Fusarium* spp. (15,000 /ml of each species) 3 days before application. At GS 75, all pots were transferred to a misting chamber to receive light rain for 15 s/h for a 6h period per day. Two weeks later, ear disease was assessed, whole pots were hand-harvested and yield components determined. Irrespective of inoculation timing, all spray treatments decreased ear disease. However, significant yield responses were only obtained in plants inoculated at the seedling stage by UK264 applied at GS 49, and from the later (GS 69) spray timing in plants inoculated post ear emergence (Table 4).

TABLE 4. Effects of inoculation and fungicide (UK264) timing on Fusarium ear disease, yield and thousand grain weight (TGW).

Inoculation timing	Spray timing	Ear disease (%)	Yield (g/100 ears)	TGW (g)
seedling	-	62.4	138.8	32.23
seedling	GS 49	33.6	154.3	33.19
seedling	GS 69	18.4	149.8	32.82
SED (df=8)			7.03	0.854
GS 49	-	25.6	119.7	35.18
GS 49	GS 49+3d	8.0	129.1	37.02
GS 69	-	31.2	121.9	31.18
GS 69	GS 69+3d	7.2	149.4	42.76
SED (df=12)			9.02	1.994

UK264 was applied on single occasions at GS31, GS39, GS55, and as a 2-spray programme at GS39 + GS55, to wheat plants, grown in large pots (12 plants/pot) outside which had been inoculated with four *Fusarium* spp. individually at the seedling stage. These treatments were compared with single sprays of prochloraz (400 gAI/ha) or carbendazim (250 gAI/ha) at GS 31, and unsprayed. There were 4 replicates of each combination of species and treatment. Stem lesion severity and incidence of ear infection were assessed at GS 75.

All treatments reduced the severity of stem lesions caused by *F. nivale*, with prochloraz, carbendazim and the 2 - spray programme of UK 264 being the most effective. When disease was caused by *F. avenaceum, F. culmorum or F. graminearum*, GS 31 applications of either prochloraz or UK264 significantly reduced stem lesion severity compared to other treatments and timings, whereas GS 55 applications of UK264 were not effective. Ear disease on unsprayed plants inoculated with *F. nivale, F. avenaceum, F. culmorum and F. graminearum* was 12%, 19%, 26% and 71% respectively. Substantial reductions in ear disease (67%, 79%, 65% and 92% respectively) were obtained only with UK264 treatments that included an application at GS 55.

Evaluation of ear sprays

Potted winter wheat plants, cv. Avalon, were inoculated by atomising ears, at mid-anthesis, with a conidial suspension (100,000/ml) of one of each of the four *Fusarium* spp. Plants were incubated in a glasshouse misting unit for 72h prior to fungicide application. The formulated fungicides (Table 5) were applied at manufacturers' recommended rates for field application in 250 l/ha water. Two days after spray application, all plants were returned to the misting unit and arranged in randomised blocks (5 replicates/treatment). The percentage of each ear diseased was recorded four weeks later.

Under conditions which favoured severe ear disease, all fungicide treatments, except the contact fungicide chlorothalonil which was only effective against *F.culmorum*, significantly reduced ear disease. Overall, UK264 gave the most effective control against all four *Fusarium* spp., significantly better than all other treatments except prochloraz (Table 5).

TABLE 5. Effect of fungicides on the severity of ear disease four weeks after inoculation of ears.

Treatment	Proportion (%) of ear diseased (mean of 50 ears)			
	F. avenaceum	*F. culmorum*	*F. graminearum*	*F. nivale*
carbendazim	28.33 cd	18.48 c	15.16 bcd	20.64 bc
chlorothalonil	53.72 e	21.95 cd	51.83 e	38.58 e
flusilazole + carbendazim	17.34 b	10.50 b	11.19 bc	16.48 b
nuarimol	21.05 bc	12.76 b	16.46 cd	19.26 bc
prochloraz	9.09 a	11.04 b	10.05 ab	5.07 a
propiconazole	22.38 bc	26.21 d	15.65 bcd	31.22 d
triadimenol	35.12 d	24.00 d	18.89 d	35.43 de
UK264	6.08 a	4.94 a	4.16 a	7.80 a
untreated	71.34 f	59.88 e	57.00 e	45.12 f

Means followed by the same letter do not differ significantly ($P = 0.05$).

DISCUSSION

It is known that *Fusarium* ear infection may arise from ascospores or conidia transported to upper plant parts by wind or rain during flowering, and that symptoms may develop at any time after ear emergence when warm wet weather persists prior to harvest. This study has demonstrated that ears may also become infected by *F. avenaceum, F. culmorum, F. graminearum or F. nivale,* through systemic pathogen progress within stem tissue. Although the relative contribution of different infection sources to ear disease in field crops has yet to be determined, all are capable of inducing ear disease and causing yield loss. Outbreaks of ear disease are often erratic, and currently can not be predicted with any degree of reliability to target fungicide intervention for optimal control efficacy. Therefore, until more detailed and reliable knowledge of the biology and epidemiology of *Fusarium* spp. is obtained, and damage quantified, control strategies are likely to be targeted to reduce specific phases of disease. This study has demonstrated that seed treatment, foliar and ear sprays all have merits and limitations for control of *Fusarium.* Seed treatment alone may contain disease in the early stages of development and reduce internal colonisation, but it is unlikely to be effective against disease arising from external inoculum sources later in the season, which can lead to yield loss. Late-season foliar and ear sprays, whilst effectively reducing yield loss from severe ear disease, would not be expected to influence yield losses induced by stem colonisation prior to application. However, a combination of seed treatment and late-season sprays could form the basis for the development of an optimal control strategy for *Fusarium.*

REFERENCES

Jenkins, J.E.E.; Clark, W.S; Buckle, A.E. (1988) *Fusarium Diseases of Cereals. H-GCA Research Review No. 4.* 89 pp.

BIOLOGICAL PROPERTIES OF FLUSILAZOLE CONTRIBUTING TO ITS FIELD PERFORMANCE

C. M. Smith, M. C. Klapproth, D. W. Saunders, L.E.B. Johnson, A. E. Trivellas

E. I. du Pont de Nemours and Company, Du Pont Agricultural Products, Stine-Haskell Research Center, Newark, Delaware 19714-0030 USA

ABSTRACT

The biological properties of flusilazole were investigated in wheat with respect to its systemic movement, resistance to wash-off, redistribution by rainfall, and vapour phase activity. Radiotracer studies showed flusilazole rapidly penetrated foliage, migrated distally, and was well distributed within the plant tissue. A residual quantity of the fungicide also remained on the foliar surface at the point of application. Flusilazole's resistance to wash-off was demonstrated by maintenance of effective powdery mildew control despite exposure of plants to simulated rainfall within a short time of fungicide application. Flusilazole was available for foliar redistribution by rainfall to protect non-fungicide-treated foliage. Through fumigant action, flusilazole provided excellent powdery mildew control of untreated plants placed near fungicide-sprayed ones. These properties of flusilazole are important contributors to its outstanding field efficacy.

INTRODUCTION

Flusilazole is a demethylation-inhibiting fungicide, developed by E. I. du Pont de Nemours and Company, which offers excellent broad-spectrum control of cereal diseases (Fort & Moberg, 1984). A major attribute of this fungicide is its consistent, high level of field performance under a wide range of environmental conditions (Austin, 1986). To investigate the basis of this efficacy, several biological properties of flusilazole were studied in glasshouse experiments on wheat. This paper presents results on flusilazole's uptake and translocation, resistance to wash-off, redistribution by rainfall, and vapour phase activity.

MATERIALS AND METHODS

For all experiments, wheat plants were grown in 7 cm diameter pots of 'Metro-Mix' potting medium in growth chambers at 27°C and 80% r.h. with 16-h day. In studies of resistance to wash-off, redistribution by rainfall, and vapour phase activity, plants were inoculated with *Erysiphe graminis* f. sp. *tritici* (strain sensitive to demethylation-inhibiting fungicides) by allowing a cloud of conidia to settle on the foliage. Inoculated plants were then maintained in growth chambers at 20°C and 80% r.h. with 16-h day. Eight days after inoculation, disease was assessed as the percentage of leaf area with visible powdery mildew symptoms. Disease control was expressed as % control or as ED_{90} (g AI fungicide/ha required to provide 90% control under the conditions described). Three replicates per treatment were used.

Uptake and translocation

Spring wheat (cv. Fremont) was treated at first node (Zadoks GS 31) with [^{14}C] flusilazole (specific activity 16 μCi/mole, labelled in position 3 of the triazole ring) in an aqueous solution in 10% (V/V) acetone. Five μl of the solution was applied to a spot at the base of one leaf sheath per plant. Plants were then maintained in growth chambers at 20°C at 80% r.h. with 16-h day. At 1, 3, and 6 days after fungicide application, each treated sheath with attached leaf blade was removed from the plant and divided into a sheath and three leaf

segments. Each sheath where flusilazole was applied was rinsed twice in 5 ml methanol. The amount of [^{14}C] flusilazole in sheaths, leaf sections, and washings (surface residue) was measured with a liquid scintillation counter after chemical digestion of plant material.

Resistance to wash-off

Flusilazole ('Capitan', 250 g/litre EC) was sprayed at 100 and 200 g AI/ha at 374 l/ha on 2-week-old winter wheat plants (cv. Stephens) at the two-leaf stage. At 0.5, 1, 2, 4 and 24 h after fungicide application, plants were exposed to 3 cm of simulated rainfall and allowed to dry. Controls included fungicide-sprayed plants which received no exposure to rainfall. At 25 h after fungicide application, all plants were inoculated with *E. graminis* f. sp. *tritici*.

Redistribution by rainfall

Flusilazole or flutriafol ('Impact', 125 g/litre SC, ICI) was sprayed at 1, 5, 20 and 100 g AI/ha at 374 l/ha on two sets of 2-week-old winter wheat plants (cv. Stephens) at the two-leaf stage. The first set was used as a preventive control while the second set was exposed to 3 cm of simulated rainfall 3 h after fungicide application (Fig. 1). The run-off from these plants was collected and sprayed onto a third set of plants. All plants were inoculated 24 h later with *E. graminis* f. sp. *tritici*.

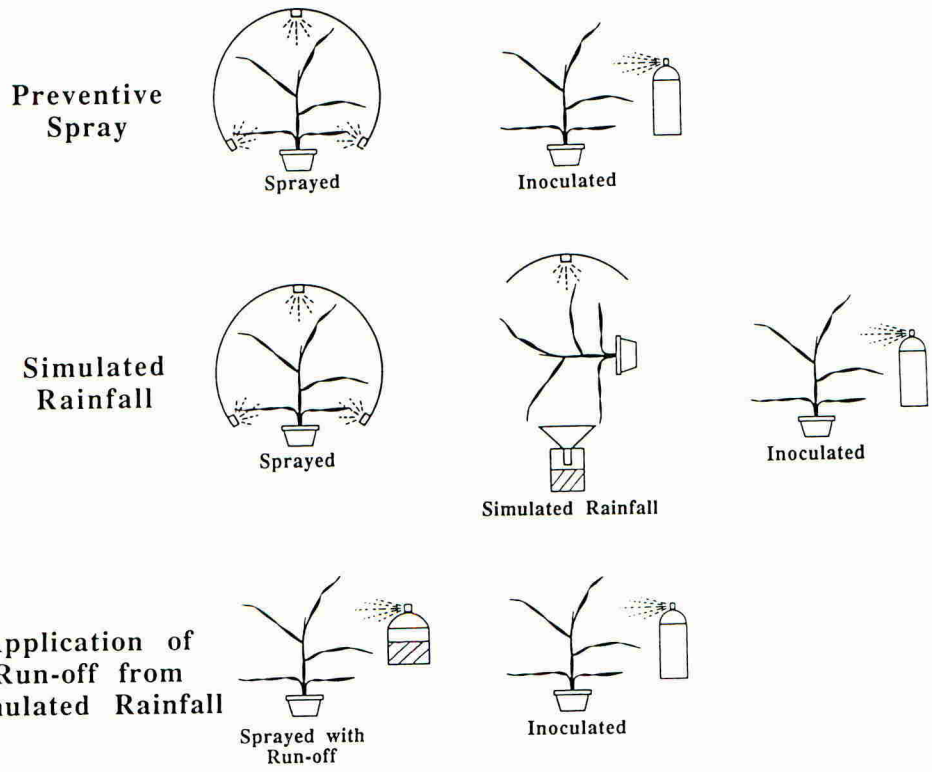

FIGURE 1. Experimental protocol to investigate foliar redistribution of flusilazole by rainfall.

Vapour phase activity

Twenty g AI/ha of flusilazole or tebuconazole ('Horizon', 250 g/litre EC, Bayer) was sprayed at 374 l/ha on 2-week-old winter wheat plants (cv. Stephens) at the two-leaf stage. One treated plant was immediately placed (without contact) within 10 cm of four untreated wheat plants at the two-leaf stage in an enclosed 50 x 50 x 50 cm 'Plexiglas' box. The box was maintained for 24 h in a growth chamber at 20°C with 16-h day. Plants were then inoculated with *E. graminis* f. sp. *tritici* and returned to the closed environment.

RESULTS

Uptake and translocation

In radiotracer studies, [^{14}C] flusilazole rapidly penetrated plant tissue (Fig. 2). Within one day after application, 50% of the flusilazole applied to the sheath surface had penetrated into the sheath and leaf tissues. Six days after application, 88% of the surface-applied flusilazole was present in the plant tissues. A residual amount of flusilazole remained on the sheath surface at the point of application, and ranged from 50% present one day after application to 12% after six days.

Following uptake, flusilazole was transported acropetally. Three to six days after application of a single drop of flusilazole to the base of the sheath, the entire sheath and leaf blade were uniformly labeled. Translocation slowed with time after application.

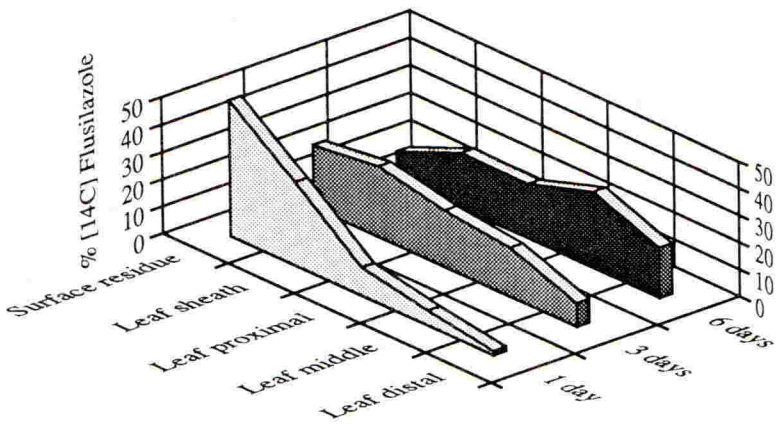

FIGURE 2. Distribution of flusilazole in wheat leaf sheaths, blades, and surface residue at 1, 3, and 6 days following flusilazole application to the base of leaf sheaths.

Resistance to wash-off

Flusilazole provided effective control of *E. graminis* f. sp. *tritici* on wheat plants exposed to simulated rain as soon as 0.5 h following fungicide application (Fig. 3). Exposure to rainfall at 1–4 h after application did not significantly affect flusilazole's performance compared to plants without exposure to rainfall. No difference was observed in disease control on plants that received a rainfall treatment 24 h after flusilazole application relative to non-rainfall-treated controls.

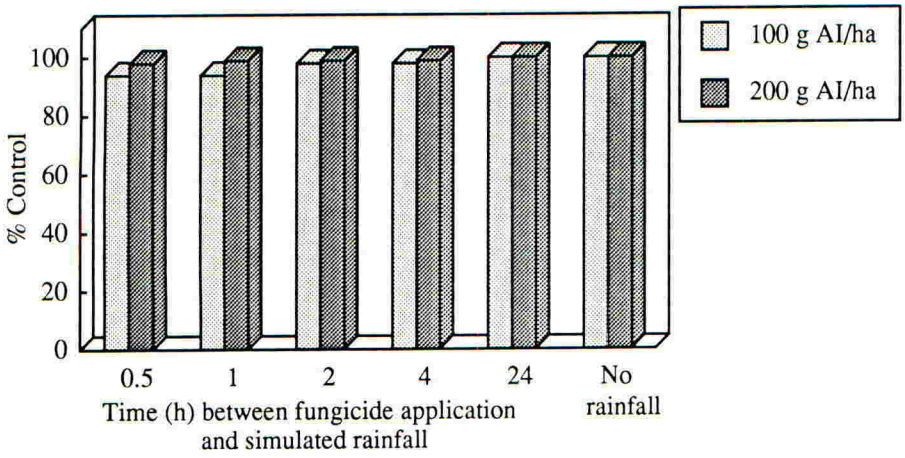

FIGURE 3. Control of *Erysiphe graminis* f. sp. *tritici* on wheat plants exposed to 3 cm of simulated rainfall at 0.5–24 h after flusilazole application at 100 and 200 g AI/ha.

Redistribution by rainfall

The run-off from flusilazole-treated plants exposed to simulated rainfall provided excellent control of *E. graminis* f. sp. *tritici* when applied to untreated plants (Fig. 4). Flusilazole was superior to flutriafol in redistribution performance. In addition, flusilazole provided excellent preventive control of *E. graminis* f. sp. *tritici* and maintained its effectiveness despite simulated rainfall 3 h after fungicide application.

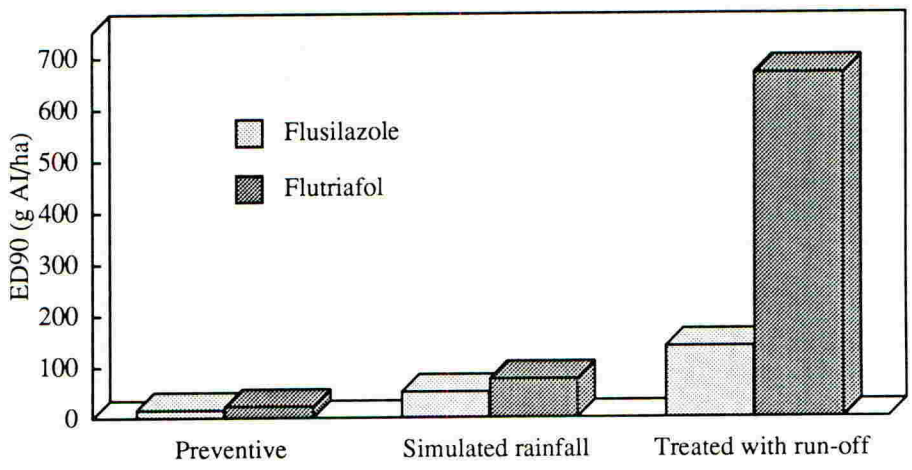

FIGURE 4. Control of *Erysiphe graminis* f. sp. *tritici* on wheat plants which received a preventive application of flusilazole or flutriafol, a simulated rainfall treatment 3 h after fungicide application, or an application of the run-off from the rainfall-treated plants.

Vapour phase activity

When untreated plants were exposed to fungicide-sprayed plants, flusilazole provided excellent control of *E. graminis* f. sp. *tritici* on both treated and untreated plants (Fig. 5). Flusilazole was significantly more effective than tebuconazole in disease control through fumigant action.

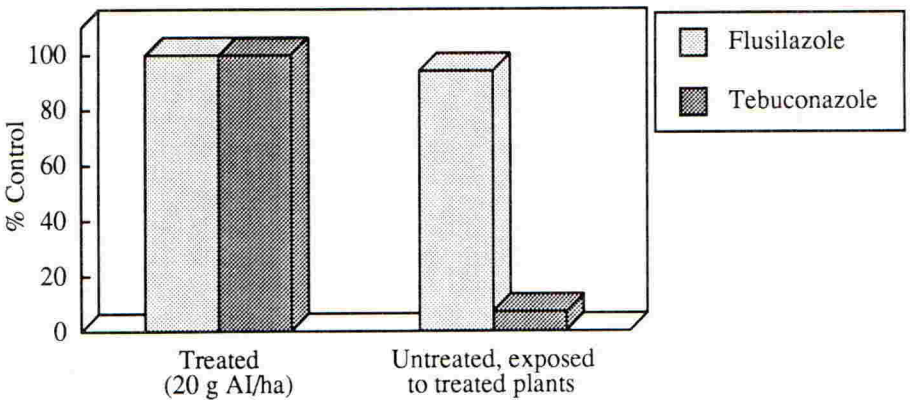

FIGURE 5. Control of *Erysiphe graminis* f. sp. *tritici* on wheat plants treated with 20 g AI/ha flusilazole or tebuconazole and on untreated plants exposed to fungicide-treated plants in an enclosed chamber.

CONCLUSIONS

Flusilazole's systemicity, resistance to wash-off, redistribution by rainfall, and vapour phase activity are important components of its biological activity. Although wheat powdery mildew was used as a bioassay to investigate flusilazole's disease control properties in this paper, the conclusions may be generally applicable to flusilazole's performance on other diseases as well.

As shown in the radiotracer studies, a significant proportion of the flusilazole applied to the leaf sheath rapidly penetrated the plant tissue while the remainder was maintained as a residual on the plant surface. This rapid penetration acts to protect flusilazole from degradation by weathering. Furthermore, flusilazole's surface residues can serve as sources for redistribution by rainfall and vapour phase action, as well as provide disease control at the point of fungicide application.

Following uptake, flusilazole was acropetally translocated via the transpiration stream from a spot on the basal sheath throughout the sheath and blade within three to six days. In contrast to some systemic fungicides (Al-Ayoubi & Shepard, 1990; Shepard, 1985), flusilazole's distribution in plant tissue is relatively uniform over an extended time period. Thus, protection of both fungicide-treated and untreated areas can be achieved with flusilazole.

Flusilazole's uniform distribution in the leaf sheath and blade can contribute to its broad-spectrum control of foot and foliar pathogens. Its residual, remaining at the plant base, acts to protect against the foot disease, eyespot. In addition, some fungicide migrates to the leaf blade for control of foliar diseases such as powdery mildew, *Septoria* leaf spots, and rusts

(Austin, 1986). Thus, through its systemic movement, flusilazole is well suited for control of a wide range of cereal pathogens.

Flusilazole provides excellent disease control despite exposure to rainfall soon after fungicide application. Results demonstrated that control was maintained even with simulated rainfall within 0.5 h of flusilazole application while rain at 24 h had no effect on flusilazole's performance. The rapid penetration of flusilazole into wheat, as indicated in radiotracer studies, is a major contributor to such rainfastness. Flusilazole's resistance to wash-off provides important advantages in flexibility in application timing and ease of use in all areas with variable weather patterns.

Flusilazole is highly effective in disease control through redistribution by rainfall or dew from sprayed to unsprayed foliage and demonstrates superior redistribution properties compared to flutriafol. Such activity complements flusilazole's preventive action and rainfastness. This combined three-way efficacy is due to a balance of flusilazole's uptake by the plant and residues on the foliar surface available for redistribution. Through redistribution, newly emerging leaf areas, such as proximal portions of cereal leaves, can be protected by movement of flusilazole on the leaf surface from distal, sprayed areas. In addition, redistribution can compensate for incomplete spray coverage. Such activity can be critical in providing disease control over extended intervals, particularly during rapid plant growth.

The vapour phase of flusilazole is effective in controlling *E. graminis* f. sp. *tritici* (Al-Ayoubi & Shepard, 1990) and is superior to that of tebuconazole. This fumigant activity can be important in dense cereal canopies with limited air movement, and acts to protect foliage that is untreated due to new growth or incomplete spray coverage.

These attributes of flusilazole—uptake and translocation, resistance to wash-off, redistribution, and vapor phase activity—are important contributors to its outstanding field performance.

ACKNOWLEDGEMENTS

We would like to express our sincere thanks to our many colleagues at Du Pont Agricultural Products worldwide for their support in preparing this paper.

REFERENCES

Al-Ayoubi, L.M.; Shepard, M.C. (1990) The activity of flusilazole against powdery mildew of wheat. *Brighton Crop Protection Conference - Pests and Diseases 1990,* **2**, 855–860.

Austin, J. R. (1986) Broad spectrum disease control in winter cereals with a combined formulation of DPX H6573 plus carbendazim. *1986 British Crop Protection Conference -Pests and Diseases,* **1**, 71-78.

Fort, T.M.; Moberg, W.K. (1984) DPX H6573, a new broad-spectrum fungicide candidate. *1984 British Crop Protection Conference - Pests and Diseases*, **2**, 413–419.

Shepard, M.C. (1985) Fungicide behaviour in the plant: Systemicity. *Fungicides for Crop Protection*, Smith I.M. (Ed.), *BCPC Monograph* No. 31, Croydon: BCPC Publications, **1**, 99–106.

CONTROL OF FUNGAL DISEASES OF ARABLE CROPS USING INHIBITORS OF POLYAMINE BIOSYNTHESIS

D.R. WALTERS, N.D. HAVIS

Department of Plant Science, SAC, Auchincruive, Nr Ayr, KA6 5HW

S.A. FOSTER

Department of Biological Sciences, The University, Dundee, DD1 4HN

D.J. ROBINS

Department of Chemistry, The University, Glasgow, G12 8QQ

ABSTRACT

The polyamine biosynthesis inhibitor α-difluoromethylornithine (DFMO), and the putrescine analogue keto-putrescine, were shown to provide substantial control of powdery mildew (Erysiphe graminis) and brown rust (Puccinia hordei) infections in barley, and rust (Uromyces viciae-fabae) and chocolate spot (Botrytis fabae) infections in broad bean, in glasshouse experiments. In a field trial with spring barley, DFMO used at 375 g/ha controlled mildew early in the season at least as well as a mixture of 120 + 192 g/ha flutriafol + carbendazim. The fungicidal activities of these two compounds appears to be related to a pertubation in polyamine biosynthesis, but although DFMO and keto-putrescine affected polyamine biosynthetic enzymes differently, they both reduced spermidine concentrations in the oat stripe pathogen, Pyrenophora avenae.

INTRODUCTION

Difluoromethylornithine (DFMO) is an enzyme-activated inhibitor of the polyamine biosynthetic enzyme ornithine decarboxylase (ODC) (Metcalf et al., 1978), and depletes intracellular concentrations of putrescine and spermidine in plant pathogenic fungi (Foster & Walters, 1990). This reduces fungal growth, since polyamines are essential for cell division (Walters, 1989). Because higher plants produce putrescine by decarboxylation of either arginine or ornithine, while most fungi appear to synthesize putrescine by ornithine decarboxylation, it has been suggested that specific inhibition of ODC should control fungal diseases without affecting the plant (Rajam et al., 1985; Walters, 1986). Indeed, DFMO has been shown to give substantial reductions in barley powdery mildew infection in pot experiments (West & Walters, 1988).

Polyamine metabolism can also be perturbed by polyamine analogues, with concomitant effects on cell growth. Thus, a variety of polyamine analogues have been shown to alter polyamine metabolism in tumour cells leading to powerful antiproliferative effects (Porter & Sufrin, 1986). Little information exists on the effects of polyamine

analogues on plant pathogenic fungi. Here we examine the possible fungicidal activity of the putrescine analogue keto-putrescine, in comparison with DFMO, and determine whether any such activity was accompanied by altered polyamine biosynthesis.

MATERIALS AND METHODS

Fungicidal effects of DFMO and keto-putrescine

Barley seedlings (Hordeum vulgare L.; cv. Golden Promise) were grown as described previously (West & Walters, 1988). At growth stage 12 (second leaf unfolded, Zadoks scale) they were sprayed to run-off with aqueous solutions of the inhibitors (238 mg/litre DFMO and 102 mg/litre keto-putrescine) in 0.01 % Tween 20, 3 h before or 3 d after inoculation with powdery mildew, Erysiphe graminis f.sp.hordei, or brown rust, Puccinia hordei. Seedlings were inoculated with mildew by dusting conidia onto the leaf surfaces and, for rust inoculation, leaves were painted with a uredospore suspension (25 mg/100 ml water), after which they were loosely covered with clear plastic bags for 48 h. Infection intensity was assessed 10 d after inoculation. For mildew this involved estimating the percentage leaf area infected using a standard area diagram, while for brown rust, the number of pustules on leaves was counted.

Broad beans (Vicia faba L.; cv. Express Long Pod) were grown as described previously (Walters, 1986) and were used when they were 20 d old. Plants were sprayed with the inhibitors and inoculated with the rust, Uromyces viciae-fabae, as described above. Infection intensity was assessed 10 d after inoculation by counting the number of rust pustules on leaves. Plants were inoculated with the chocolate spot pathogen, Botrytis fabae, by painting leaves with a spore suspension (approx. 400,000 conidia/ml) after which they were loosely covered with clear plastic bags for 48 h in order to provide the high r.h. required for spore germination. Infection intensity was assessed 7 d after inoculation by estimating the percentage leaf area infected using a standard area diagram.

Evaluation of DFMO against powdery mildew on barley in a field trial

Spring barley (cv. Golden Promise) was sown on 17 April 1991 at 12 cm row spacing with a seed rate of 190 kg/ha. Seed was sown in plots (4 x 2 m) in a randomized block design with four replicates. Some plots were sprayed with fungicide once, at the first sign of mildew (GS 25; 1 June), while other plots were sprayed twice (GS 25 and 59; 1 June and 1 July). The fungicides used were DFMO (100 %) at 375 g/ha, and a mixture of flutriafol and carbendazim (Early Impact; SC) at 120 + 192 g/ha, respectively. DFMO was made up in water with Agral 90 (0.5 l/ha). Powdery mildew infection was assessed on the third leaf on 7 and 14 June (GS 28 and 30) and on the flag leaf on 9 and 16 July (GS 60 and 61). At the end of the trial, plants were harvested and measurements made of grain weight. Data were analysed statistically using analysis of variance.

Effects of DFMO and keto-putrescine on enzyme activities and polyamine concentrations in Pyrenophora avenae

The activities of ornithine decarboxylase (ODC) and S-adenosylmethionine decarboxylase (AdoMetDC) were assayed, and polyamine levels determined, as described previously (Foster & Walters, 1990).

RESULTS

Fungicidal activity of DFMO and keto-putrescine

DFMO and keto-putrescine, applied either as pre-inoculation or post-inoculation sprays, gave substantial disease control in all plant-pathogen interactions examined (Table 1). Slightly greater disease control was achieved using post-inoculation, rather than pre-inoculation treatments. All treatments were statistically significant.

TABLE 1. Comparison of the fungicidal activities of DFMO (238 mg/litre) and keto-putrescine (K-P; 102 mg/litre).

Plant-Pathogen interaction	% leaf area infected			
	Untreated	DFMO	Untreated	K-P
E. graminis/barley				
pre-inoculation spray	42	12	52	13
post-inoculation spray	42	5	52	8
P. hordei/barley				
pre-inoculation spray	27	7	36	7
post-inoculation spray	27	4	36	5
U. viciae-fabae/broad bean				
pre-inoculation spray	18	3	23	7
post-inoculation spray	18	2	23	5
B. fabae/broad bean				
pre-inoculation spray	6	1	3	0.4
post-inoculation spray	6	1	2	0.3

Evaluation of DFMO against barley powdery mildew in a field trial

A single spray of DFMO or of the mixture of flutriafol and carbendazim gave a significant reduction in mildew early in the season, but had no effect on mildew later in the season (Table 2). In plots receiving a second spray at GS 59, DFMO failed to reduce mildew later in the season, whereas the flutriafol + carbendazim mixture gave a significant reduction in mildew infection. A single spray of DFMO or flutriafol + carbendazim did not increase grain yield, although when the fungicides were applied twice, small increases in grain yield were obtained (Table 2).

TABLE 2. Effects of DFMO and a flutriafol + carbendazim mixture on powdery mildew infection and grain yield of barley.

Treatment	Dose (g/ha) in each spray	% powdery mildew infection				Grain yield (kg/ plot)
		7 June	14 June	9 July	16 July	
Single spray						
Untreated	0	9.4	20.2	6.4	18.6	11.3
DFMO	375	3.0	12.3	9.9	24.2	11.7
Flutriafol + carbendazim	120 + 192	4.2	11.3	8.5	17.9	11.7
Two sprays						
Untreated	0	9.4	20.2	6.4	18.6	11.3
DFMO	375	2.1	11.7	7.0	15.2	12.3
Flutriafol + carbendazim	120 + 192	4.8	11.7	2.6	4.8	13.0
L.S.D. (P = 0.05)		2.45	3.80	5.79	6.49	1.65

TABLE 3. Effects of DFMO and keto-putrescine (238 and 102 mg/litre respectively) on ODC and AdoMetDC activities, and polyamine concentrations in Pyrenophora avenae.

(a)

Treatment	Enzyme activity, pmol CO_2 mg/protein/h			
	ODC		AdoMetDC	
	Soluble	Bound	Soluble	Bound
Control	5.3 ± 0.7	5.2 ± 0.6	3.5 ± 0.08	7.8 ± 0.7
DFMO	0.7 ± 0.06b	0.9 ± 0.08b	7.8 ± 0.9b	28.4 ± 1.8b
Keto-putrescine	4.3 ± 0.7	4.2 ± 0.2	1.7 ± 0.3b	2.3 ± 0.5b

(b)

Treatment	Polyamine concentration, nmol g/f.wt			
	Putrescine	Spermidine	Spermine	Cadaverine
Control	89.4 ± 22.2	177.9 ± 6.2	333.1 ± 14.9	22.8 ± 11.4
DFMO	63.0 ± 2.9	55.7 ± 6.1b	331.5 ± 12.5	11.5 ± 1.2
Keto-putrescine	93.1 ± 12.4	102.7 ± 3.8b	416.4 ± 20.6a	171.3 ± 21.8b

Significant differences are shown at P = 0.05 a and P = 0.001 b.

Effects of DFMO and keto-putrescine on ODC and AdoMetDC activities and polyamine concentrations in Pyrenophora avenae

The activities of soluble and bound ODC were increased significantly in P. avenae grown in the presence of DFMO (Table 3a). In contrast, the activities of soluble and bound AdoMetDC were increased significantly in DFMO-treated tissue (Table 3a). DFMO-treated fungal tissue also exhibited a substantial and significant reduction in spermidine concentration (Table 3b). Although the concentrations of putrescine and cadaverine were also reduced in these tissues, the reductions were not significant (Table 3b).

Growth of P. avenae in the presence of keto-putrescine reduced the activities of soluble and bound ODC by 19 % and 20 % respectively, although these reductions were not significant (Table 3a). In contrast, soluble and bound AdoMetDC activities were reduced by 52 % and 71 % respectively (Table 3a). Keto-putrescine had no effect on putrescine concentration in P. avenae, but reduced spermidine concentration by 43 % (Table 3b). Interestingly, concentrations of both spermine and cadaverine were increased in these tissues compared to controls (Table 3b).

DISCUSSION

The ODC inhibitor DFMO has been shown to provide substantial control of powdery mildew and rust infections on barley and rust and chocolate spot infections on broad bean. This confirms and extends previous work which showed that DFMO controlled rust and powdery mildew infections on a range of crops (Rajam et al., 1985; West & Walters, 1988). Moreover, in the field trial, early season control of barley mildew with DFMO was as good as that achieved with a mixture of flutriafol + carbendazim. Furthermore, even though DFMO did not reduce mildew late in the season, grain weights were increased compared to untreated controls. In addition, the putrescine analogue, keto-putrescine, has been shown to possess substantial fungicidal activity. Thus, application of 102 mg/litre keto-putrescine before or after inoculation with fungal pathogens reduced infections by 62-89 %.

Growth of P. avenae in the presence of either DFMO or keto-putrescine resulted in altered polyamine biosynthesis and polyamine concentrations. However, whereas DFMO reduced ODC activities and increased AdoMetDC activities in the pathogen, keto-putrescine had little effect on ODC activities and strongly reduced AdoMetDC activities. Both DFMO and keto-putrescine reduced spermidine concentrations in P. avenae and it is tempting to suggest that this was responsible for the reductions in fungal growth. This is likely to be true for DFMO-treated P. avenae, where the reduction in spermidine was accompanied by reductions in putrescine and cadaverine. However, keto-putrescine treatment had little effect on putrescine and increased the concentrations of spermine and cadaverine. Certainly, the large pool of cadaverine could easily have supported fungal growth. Interestingly, keto-putrescine had considerably less effect on the in vitro growth of P. avenae than on in vivo infection with a range of fungal pathogens. Indeed, Smith et al. (1990) showed that keto-putrescine reduced in vitro growth of Botrytis cinerea by just 27 %. It is possible, therefore, that polyamine metabolism is more sensitive and easily perturbed during fungal development on the leaf surface, or there is a greater uptake and accumulation of keto-putrescine by the developing

fungus.

In conclusion, keto-putrescine possesses considerable fungicidal activity, which may be related to a pertubation of polyamine biosynthesis. To date, most studies of inhibitors of polyamine biosynthesis as possible fungicides have concentrated on use of enzyme-activated inhibitors e.g. DFMO. Perturbation of polyamine biosynthesis using polyamine analogues might provide a useful alternative to the development of new fungicides.

ACKNOWLEDGEMENTS

We are grateful to the British Technology Group plc for generous financial support, and to Dr Richard A'Court of BTG for advice and guidance. SAC receives financial support from the Scottish Office Agriculture and Fisheries Department.

REFERENCES

Foster, S.A.; Walters, D.R. (1990) The effects of polyamine biosynthesis inhibitors on mycelial growth, enzyme activity and polyamine levels in the oat-infecting fungus Pyrenophora avenae. Journal of General Microbiology, **136**, 233-239.

Metcalf, B.W.; Bey, P.; Danzin, C.; Jung, M.J.; Casara, P.; Vevert, J.P. (1978) Catalytic irreversible inhibition of mammalian ornithine decarboxylase (EC 4.1.1.17) by substrate and product analogues. Journal of the American Chemical Society, **100**, 2551-2553.

Porter, C.W.; Sufrin, J.R. (1986) Interference with polyamine biosynthesis and/or function by analogues of polyamines or methionine as a potential anti-cancer therapeutic strategy. Anti-cancer Research, **6**, 525-542.

Rajam, M.V.; Weinstein, L.H.; Galston, A.W. (1985) Prevention of a plant disease by specific inhibition of fungal polyamine biosynthesis. Proceeding of the National Academy of Sciences of the United States of America, **82**, 6874-6878.

Smith, T.A.; Barker, J.H.A.; Jung, M.J. (1990) Growth inhibition of Botrytis cinerea by compounds interfering with polyamine metabolism. Journal of General Microbiology, **136**, 985-992.

Walters, D.R. (1986) The effects of a polyamine biosynthesis inhibitor on infection of Vicia faba L. by the rust fungus Uromyces viciae-fabae (Pers) Schroet. New Phytologist, **104**, 613-619.

Walters, D.R. (1989) Polyamines and plant disease. Plants Today, **Jan-Feb 1989**, 22-26.

West, H.M.; Walters, D.R. (1988) The effects of polyamine biosynthesis inhibitors on infection of Hordeum vulgare L. by Erysiphe graminis f.sp. hordei Marchal. New Phytologist, **110**, 193-200.

DIFENOCONAZOLE: A NEW FUNGICIDE AGAINST *CERCOSPORA BETICOLA* ON SUGAR BEET

G. KNAUF-BEITER, C. FLEISCHHACKER, L. MITTERMEIER

Ciba-Geigy Ltd., Plant Protection Division, Business Unit Disease Control, CH-4002 Basle, Switzerland

R. LIGUORI

Ciba-Geigy SpA, Plant Protection Division, Business Unit Disease Control, I-21047 Saronno, Italy

ABSTRACT

In greenhouse experiments and in field trials against *Cercospora beticola* on sugar beet, difenoconazole, compared to the standards flusilazole, propiconazole + fentin acetate (0.156 + 0.225) and fentin acetate, was shown to be a very flexible compound concerning application timing. It was highly active in protective as well as in curative applications with particular strength when applied as a protective treatment. In the greenhouse, the use of difenoconazole at 2 mg AI/litre resulted in adequate control of *C. beticola* in the protective treatments while 10 mg AI/litre were needed in curative applications. The field trial revealed an excellent activity of difenoconazole at 0.1 kg AI/ha which was superior to that of the standards. With the same application timing, difenoconazole showed benefits over flusilazole at lower rates. Difenoconazole was superior to fentin acetate, which was less flexible in its application timing and which was needed in protective treatments at a higher dosage to reach a high level of *C. beticola* control.

INTRODUCTION

Difenoconazole was introduced by Ciba-Geigy in 1988 (Ruess *et al.*, 1988) as a new triazole ('Score') with a broad spectrum of activity against a large number of cereal and non-cereal pathogens. The range of target crops includes among others wheat, peanuts, potatoes, pome fruit, grapes and vegetables (Dahmen and Staub, 1990, 1992).

In the following, the use of difenoconazole in sugar beet for the control of *Cercospora beticola* which causes *Cercospora* leaf spot disease is described. Results from greenhouse and international field trials are presented.

EXPERIMENTAL CONDITIONS

Glasshouse experiments

Sugar beet plants, cv. Regina, were cultivated under greenhouse conditions (day/night temperatures 20/18°C, light period 16 h) until they reached a growth stage with 6 to 7 leaves. For inoculation, *C. beticola* conidia were harvested freshly from Czapek Dox-V8 agar (20% V8, 4.5% Czapek Dox) and suspended to a final concentration of 120,000 conidia

per ml. Individual treatments are listed in Table 1. Difenoconazole was used as an EC 250 (250 g AI/litre), flusilazole as an EC 400 (400 g AI/litre) and fentin acetate as a WP 60 (600 g AI/kg) formulation. Inoculation was carried out immediately with a DeVilbiss hand spray gun. The plants were incubated under greenhouse conditions for 3 days at 24/22°C with intermittent misting. Afterwards, they were transferred to standard greenhouse conditions without misting.

TABLE 1. Treatments applied in the greenhouse trial.

Protective treatment timing		Rate (mg AI/litre)
21 - 14 - 7 - 3 - 1 days before inoculation	Difenoconazole	0.016 - 0.08 - 0.4 - 2
	Flusilazole	0.016 - 0.08 - 0.4 - 2 - 10
	Fentin acetate	0.01 - 0.1 - 1 - 10 - 100
Curative treatment timing		
1 - 3 - 7 days after inoculation	Difenoconazole	0.016 - 0.08 - 0.4 - 2 - 10
	Flusilazole	0.016 - 0.08 - 0.4 - 2 - 10
	Fentin acetate	0.1 - 1 - 10 - 100 - 500

Disease assessment was carried out 15 days after inoculation by estimating the % diseased area on the individual leaves of each plant. The experiment was carried out twice. The presented results are the mean values over 15 leaves (3 plants per treatment, each with 5 leaves) from one experiment.

Field trials

The presented data are derived from a field trial in 1988 in Serravalle (Italy) with sugar beet, cv. Supra Forte, with natural *C. beticola* infections. The experiment was arranged with 3 replicates in randomized plots each of 12.95 m^2. The first treatments were carried out two weeks after 100% soil covering by the foliage was reached. Application timing and dosage of the compounds are listed in Table 2. Difenoconazole was used as an EC 250 (250 g AI/litre), flusilazole as an EC 100 (100 g AI/litre) and propiconazole + fentin acetate as a WP 30.5 (305 g AI/kg) formulation. The compounds were applied with a tractor equipped with a spray boom and cone nozzles of 1.5 mm in a volume of 600 l/ha.

The evaluation was carried out by estimating the % infected leaf area at four times after application. The intervals between the first application and evaluation were between 21 and 69 days. The results represent the disease amounts from the last evaluation 69 days after the first application. The trial was harvested 76 days after the first application. The sugar content was determined to show the effect of the treatments on the yield quality. Different low case letters following the mean values in Table 6 indicate significant differences at the 95% probability level (Tukey test).

TABLE 2. Treatments applied in the field trial.

Compound	Rate (kg AI/ha)	Protective		Curative	
		No. of applications	Application interval	No. of applications	Application interval
Difenoconazole	0.05/0.1[1]	3	18 d	-	-
	0.05/0.1[2]	3	18 d	-	-
	0.10	3	21 d	2	21 d
	0.10	-	-	3	15 - 18 d
	0.15	2	28 d	2	28 d
Flusilazole	0.10	3	21 d	2	21 d
Propiconazole + Fentin acetate	0.156+0.225	3	21 d	-	-

[1] 1st and 2nd application 0.05 kg AI/ha, 3rd application 0.1 kg AI/ha.
[2] 1st application 0.05 kg AI/ha, 2nd and 3rd application 0.1 kg AI/ha.

RESULTS AND DISCUSSION

In the greenhouse, difenoconazole showed excellent activity against *C. beticola* within the whole application period from 21 days protective to 5 days curative foliar treatments (Tables 3, 4 and 5). It was highly flexible in the application timing with advantages when applied protectively. In later curative treatments (3 or 7 days after inoculation) a higher dosage was needed for a good control of *C. beticola* (10 mg AI/litre in later curative instead of 2 mg AI/litre in protective and 1 day curative treatments).

TABLE 3. Protective activity of difenoconazole, flusilazole and fentin acetate against *C. beticola* in the greenhouse.

Compound	EC 90[1] (mg AI/litre)				
	21 d	14 d	7 d	3 d	1 d
Difenoconazole	1.7	0.4	1.4	0.3	0.3
Flusilazole	2.8	3.2	4.0	1.1	1.5
Fentin acetate	85	70	50	18	6.0

[1] Concentration causing 90% disease control.

TABLE 4. Curative activity of difenoconazole, flusilazole and fentin acetate against *C. beticola* in the greenhouse.

Compound	EC 90[1] (mg AI/litre)		
	1 d	3 d	7 d
Difenoconazole	0.5	1.2	1.9
Flusilazole	0.4	1.8	9.0
Fentin acetate	12	>500	>500

[1] Concentration causing 90% disease control.

In the greenhouse, difenoconazole showed an excellent performance which was superior to that of both standards. Difenoconazole provided the same application timing as flusilazole but in protective trials a higher dosage of flusilazole was needed to reach a comparable reduction of the symptoms (Table 5). Similar rates of difenoconazole and flusilazole reached same levels of disease control in early curative applications, while difenoconazole provided a better activity than flusilazole in the 7 days curative application (Table 5).

TABLE 5. Activity of difenoconazole, flusilazole and fentin acetate at 2 and 10mg AI/litre against *C. beticola* in the greenhouse.

Treatment time	Untreated[1]	Difenoconazole[2] (2 mg AI/litre)	Flusilazole[2] (2 mg AI/litre)	Fentin acetate[2] (10 mg AI/litre)
21 d pro.	19	97	86	26
14 d pro.	19	99	86	52
7 d pro.	21	97	82	74
3 d pro.	20	100	93	87
1 d pro.	18	100	100	93
	Untreated[1]	Difenoconazole[2] (10 mg AI/litre)	Flusilazole[2] (10 mg AI/litre)	Fentin acetate[2] (10 mg AI/litre)
1 d cur.	27	100	100	89
3 d cur.	18	100	100	13
7 d cur.	18	94	83	21

[1] % infected leaf area on the untreated plants.

[2] % disease control.

The application window of difenoconazole was larger compared to that of fentin acetate which exhibited a low flexibility concerning treatment timing. A nearly complete suppression of *C. beticola* symptoms with fentin acetate was reached only at a rate of 100 mg AI/litre in protective and 1 day curative applications. In contrast, in 3 and 7 days curative treatments a dosage higher 500 mg AI/litre of fentin acetate was not sufficient to inhibit further disease development (Tables 3, 4 and 5).

The application of difenoconazole in field trials resulted in an excellent protective and a strong curative activity against *C. beticola* (Table 6). Even with a combined application of 0.05 and 0.1 kg AI/ha the activity of difenoconazole in protective trials was equal or even superior to the standards flusilazole and propiconazole + fentin acetate. Although the curative performance of difenoconazole was slightly inferior to that provided by protective treatments, the curative activity of difenoconazole was better than that of flusilazole. Shortening of the spray interval in the curative application of 0.1 kg AI/ha improved the activity and yield to the level of protective applications.

TABLE 6. Control of *C. beticola* with difenoconazole, flusilazole and propiconazole + fentin acetate in a field trial in Italy 1988.

Treatment	Rate (kg AI/ha)	Interval (days)	% leaf attack	yield (dt/ha)	sugar content (kg/ha)
Protective treatments					
Untreated			90 a	774 a	9467 a
Difenoconazole	0.05/0.1[1]	18	22 c	976 de	13214 bcd
	0.05/0.1[2]	18	17 c	912 abcde	13057 bcd
	0.10	21	10 c	1007 e	13979 d
	0.15	28	22 c	946 cde	13533 cd
Flusilazole	0.10	21	20 c	910 abcde	13862 cd
Propiconazole + fentin acetate	0.156+0.225	21	50 b	888 abcde	11541 abc
Curative treatments					
Difenoconazole	0.10	15 - 18	15 c	928 bcde	13158 bcd
	0.10	21	37 b	845 abcd	12242 bcd
	0.15	28	20 c	872 abcde	12476 bcd
Flusilazole	0.10	21	50 b	807 abc	11005 ab

[1] 1st and 2nd application 0.05 kg AI/ha, 3rd application 0.1 kg AI/ha.
[2] 1st application 0.05 kg AI/ha, 2nd and 3rd application 0.1 kg AI/ha.
At the first curative application approx. 3% leaf attack.

The better performance of difenoconazole than the commercial standards in the control of the leaf spot symptoms of *C. beticola* was also reflected in the increase of the sugar beet yield and sugar content (Table 6). In protective as well as in curative trials, the application of difenoconazole increased the yield up to 30%. Simultaneously, the sugar content of the beets increased up to 48%. The highest increase of yield and sugar content was reached with the application of 0.1 kg AI/ha in 21 day spray intervals (protective) and in 15 - 18 day spray intervals (curative). In both applications, the activity of difenoconazole was superior to the standards.

In addition to the presented results from Italy, field trials in other European countries (Switzerland, Austria, France and Germany) during 1987 and 1991 confirmed the excellent activity of difenoconazole against *C. beticola*, which was superior to that of standards such as fentin acetate and flusilazole (data not shown).

Beside the activity against *C. beticola*, difenoconazole demonstrated also a strong activity against other leaf pathogens on sugar beets, such as *Ramularia beticola*, *Erysiphe betae* and *Uromyces betae*.

ACKNOWLEDGMENT

We would like to thank all Ciba-Geigy colleagues who have contributed to the results presented in this paper.

REFERENCES

Ruess, W.; Riebli, J.; Herzog, J, ; Speich, J.; James, J.R. (1988). CGA 169374, a new systemic fungicide with a novel broad-spectrum activity against disease complexes in a wide range of crops. *Brighton Crop Protection Conference - Pests and Diseases*, **2**, 543-550.

Dahmen, H.; Staub, T. (1990). Biologische Wirkungsweise des neuen Fungizids Difenoconazol an verschiedenen Pathosystemen. *47. Deutsche Pflanzenschutz-Tagung*, 213.

Dahmen, H.; Staub, T. (1992). Biological characterization of uptake, translocation and dissipation of difenoconazole (CGA 169374) in wheat, peanut, and tomato plants. *Plant Disease*, **76**, 523-526.

CONTROL OF TUBER-BORNE DISEASES OF POTATOES WITH FENPICLONIL

A. J. LEADBEATER, W. W. KIRK

Ciba-Geigy Agriculture, Whittlesford, Cambridge, CB2 4QT

ABSTRACT

Fenpiclonil is a novel fungicide of phenylpyrrole chemistry, developed by Ciba-Geigy for the broad-spectrum control of seed-borne diseases in a wide range of crops including potatoes.

Results from field trials conducted in the UK show fenpiclonil to give commercially acceptable control of the major seed tuber-borne diseases of potatoes when applied as a pre-planting seed tuber treatment. The diseases controlled include black scurf and stem canker (*Rhizoctonia solani*), silver scurf (*Helminthosporium solani*), skin spot (*Polyscytalum pustulans*), dry rot (*Fusarium* spp.), gangrene (*Phoma exigua* var. *foveata*) and black dot (*Colletotrichum coccodes*). Fenpiclonil is unusual in its broad-spectrum of disease control in potatoes and, with its novel mode of action and low use rate, is expected to make a significant contribution to potato disease management.

INTRODUCTION

In most seasons, throughout the potato growing regions of Europe, a wide range of diseases affect the skin quality and viability of progeny tubers grown from infected mother potato tubers. *Rhizoctonia solani* causes emergence and crop development problems (stem canker) as well as serious quality losses on progeny tubers (black scurf). Silver scurf (*Helminthosporium solani*), skin spot (*Polyscytalum pustulans*), gangrene (*Phoma exigua* var. *foveata*), and black dot (*Colletotrichum coccodes*) primarily cause skin quality problems on progeny tubers, although severe infections can affect seed tuber viability. Dry rot (*Fusarium* spp.) causes rotting of infected tubers in the field and in stores. The incidence and importance of many of these diseases is increasing due to increased market quality requirements (e.g. supermarket pre-packs) and increased occurrences of resistance in silver scurf to thiabendazole.

Post-harvest fungicidal treatments for the control of storage diseases of potato tubers are becoming less favoured in seed growing regions since a portion of the crop is often destined for human consumption. Pre-planting fungicide treatments to seed tubers are now perceived as the main option to achieve disease control on progeny tubers (Storey, 1992).

Fenpiclonil is a fungicide of phenylpyrrole chemistry with a broad-spectrum activity for the control of a wide range of seed-borne diseases in cereals and non-cereals, described by Nevill *et al.* (1988). It is related to the natural product pyrrolnitrin (Koch & Leadbeater, 1992) and is chemically unrelated to other fungicides currently marketed. It is currently marketed in several European countries under the trade name 'Beret' and is

awaiting approval by the UK authorities for use on potatoes under the trade name 'Gambit'.

This paper reports the results of field trials conducted in the UK concerning the efficacy of fenpiclonil applied as a pre-planting seed tuber treatment to potatoes, principally against the seed tuber-borne diseases silver scurf, black scurf, stem canker, skin spot, dry rot and black dot. Some results are also reported from trials where fenpiclonil was applied as a post-harvest treatment.

METHODS AND MATERIALS

Replicated field trials were carried out in eastern England and eastern Scotland using naturally infected seed tubers. Infection levels of all seed stocks were determined prior to treatment.

Treatments were applied in a total water volume of 2 l/t using a single hydraulic nozzle above either a roller table or a wire mesh grid where the tubers were turned over by hand after one half of the product had been applied. Seed loading analyses were made by hplc on selected batches following treatment to evaluate the quantity of fenpiclonil reaching the target. The doses achieved (between 32% and 57% of target) are considered representative of the current commercial situation.

Fenpiclonil, formulated as a water based flowable containing 400 g AI/litre, was applied at a product rate of 0.125 l/t in all trials. This equates to a rate of 50g AI/t fenpiclonil.

Whole plot plant counts were made to determine crop emergence and a minimum of five destructive samples were taken to assess stem number and stem canker incidence. Skin disease assessments for *R. solani* and *C. coccodes* were made directly after harvest. *H. solani*, *P. pustulans*, and *F. solani* were assessed after 3-6 months storage under conditions conducive to disease development whilst still being representative of commercial situations. Tubers were assessed for surface area infected with disease using ADAS key 2.4.1 (black scurf) or ADAS key 2.5.1 (silver scurf, skin spot) (Anon., 1976).

RESULTS AND DISCUSSION

Crop tolerance

Field trials carried out during 1989 to 1991 over a wide range of cultivars of commercial importance in the UK confirmed the good crop tolerance of fenpiclonil compared with current commercial standards as measured by plant and stem emergence, subsequent crop vigour and yields.

Control of black scurf and stem canker (*Rhizoctonia solani*)

Field trials using seed potatoes infected with *R. solani* showed fenpiclonil to increase stem number per plant, due to control of stolon blinding caused by this and other

pathogens (results not shown). In field trials carried out in 1990 and 1991, fenpiclonil also gave good control of stem canker on stolons and stems as shown in Table 1.

TABLE 1. Control of stem canker caused by *R. solani* with fenpiclonil (1990).

Treatment	Dose (g AI/t)	Mean % stems with canker 1990	1992
Untreated	-	32	78
Tolclofos-methyl	125	19	13
Pencycuron	150	-	3
Fenpiclonil	50	7	3
Number of trials		6	2

Field trials carried out in 1989, 1990 and 1991 showed fenpiclonil applied as a pre-planting seed treatment to give good control of black scurf on progeny tubers at harvest (Tables 2 and 3). Fenpiclonil reduced both the incidence of progeny tubers infected with sclerotia and the severity of infection on those tubers which were infected, and was at least equal to the standard treatments. The good performance of fenpiclonil is especially remarkable considering the high disease pressure in the trials, largely unrepresentative of commercial conditions. 100% of seed tubers were infected, with a range of severity from 2% to 15% surface area infected.

TABLE 2. Incidence of progeny tubers infected with black scurf at harvest.

Treatment	Dose (g AI/t)	% tubers infected 1989	1990	1991	Mean
Untreated	-	18	86	74	68
Tolclofos-methyl	125	1	64	30	42
Imazalil+ thiabendazole	10+30	-	-	46	(46)
Fenpiclonil	50	0.2	45	31	32
Number of trials		2	5	2	9

TABLE 3. Severity of infection on progeny tubers infected with black scurf at harvest.

Treatment	Dose (g AI/t)	Mean % tuber surface area infected 1989	1990	1991	Mean
Untreated	-	na	5.7	6.3	5.9
Tolclofos-methyl	125	na	4.1	2.4	3.6
Imazalil+ thiabendazole	10+30	na	-	3.9	(3.9)
Fenpiclonil	50	na	1.8	2.7	2.1
Number of trials		-	5	2	8

Control of black dot (*Colletotrichum coccodes*)

Work by Zitter *et al.* (1989) indicated a high activity of fenpiclonil against this disease. This work was supported by field trials at Rothamsted Experimental Station during 1990 and 1991 where fenpiclonil was effective against seed tuber-borne infection (personal communication, P.J. Read).

Control of silver scurf (*Helminthosporium solani*)

Fenpiclonil had a high activity against silver scurf when applied as a seed tuber treatment. This activity is shown both in reduction of the incidence and the severity of infection on progeny tubers after harvesting and storage (Tables 4 and 5). The proportion of tubers with greater than 5% of surface area infected was also greatly reduced by treatment with the product. In all trials to date, fenpiclonil has shown activity against silver scurf equal to or greater than the best standards.

TABLE 4. Incidence of progeny tubers infected with silver scurf after approximately 120 days storage at 2 °C - 4 °C.

Treatment	Dose (g AI/t)	% tubers infected 1989	1990	1991	Mean
Untreated	-	57	84	65	63
Imazalil	10	21	38	-	(28)
Imazalil+ thiabendazole	10+30	-	-	52	(52)
Fenpiclonil	50	16	28	31	24
Number of trials		2	4	4	10

TABLE 5. Severity of infection of progeny tubers infected with silver scurf after approximately 120 days storage at 2 °C - 4 °C.

Treatment	Dose (g AI/t)	Mean % tuber area infected 1989	1990	1991	Mean
Untreated	-	13.7	13.8	9.6	11.7
Imazalil	10	3.3	3.5	-	(3.2)
Imazalil+thiabendazole	10+30	-	-	5.5	(5.5)
Fenpiclonil	50	2.2	1.5	1.9	2.0
Number of trials		2	4	4	10

Control of dry rot (*Fusarium* spp.)

Fenpiclonil has been shown to be very active against *Fusarium* spp. when applied as a post-harvest treatment, giving 98% control of disease in store (Stachewitz *et al.*, 1990). Fenpiclonil was also active against Fusarium in the field when applied as a pre-plant treatment, demonstrated by increased plant emergence where the disease was controlled (Table 6). In this case the pathogen was *F. solani* var. *ceruleum*, confirmed after digging up non-emerged tubers.

TABLE 6. Control of *F. solani* var. *ceruleum* with pre-planting application of fenpiclonil (1992). Evaluation of emergence 61 days after planting (cv. Estima).

Treatment	Dose (g AI/t)	% plants not emerged
Tolclofos-methyl	125	2.3
Pencycuron	150	3.8
Imazalil+thiabendazole	10+30	0.3
Fenpiclonil	50	0.0

Control of skin spot (*Polyscytalum pustulans*)

Post-harvest application in 1990 trials showed fenpiclonil to give good control of this disease, reducing both disease incidence and severity (Table 7). Further field trials are in progress to confirm the activity of seed tuber applications of fenpiclonil against this disease.

TABLE 7. Control of skin spot with pre-storage application of fenpiclonil (1990). Evaluations made after 220 days storage at 5°C.

Treatment	Dose (g AI/t)	% tubers infected	Mean % tuber surface area infected
Untreated	-	94	13.5
Imazalil	10	45	2.7
Thiabendazole	42	54	3.8
Fenpiclonil	50	40	2.0

CONCLUSIONS

The results presented here show fenpiclonil to be highly active against the major seed tuber-borne diseases of potatoes, including one to which there is currently no commercial product available, namely black dot. In the case of this disease however, fenpiclonil is not expected to control soil-borne infections.

Products for the control of potato tuber diseases in the future must take account of social acceptance as well as be highly active. Fenpiclonil, with its high efficacy at low use rates when applied to seed tubers, is ideally suited to future disease management strategies in allowing reduction of post-harvest fungicide treatments, particularly to the subsequent ware crop. In addition, its novel mode of action and activity against benzimidazole resistant strains of fungi, make it a valuable seed treatment product for the future.

REFERENCES

Anonymous (1976) *Manual of plant growth stage and disease assessment keys*, Harpenden: Ministry of Agriculture, Fisheries and Food.

Koch, E.; Leadbeater, A.J. (1992) Phenylpyrroles - a new class of fungicides for seed treatment. *1992 Brighton Crop Protection Conference - Pests and Diseases.*

Nevill, D.; Nyfeler, R.; Sozzi, D. (1988) CGA 142705 - a novel fungicide for seed treatment. *Brighton Crop Protection Conference - Pests and Diseases 1988*, 1, 65-72.

Stachewitz, H. E.; Burth, U.; Leadbeater, A.; van der Maarel, H,; Elmsheuser, H. (1990) Behandlung von Kartoffeln mit Beret (fenpiclonil) gegen Lagerkrankheiten und *Rhizoctonia solani. Mitteilungen aus der Biologischen Bundesanstalt fuer Land- und Forstwirtschaft, Berlin-Dahlem, Heft 266, 47. Deutsche Pflanzenschutztagung*, 192.

Storey, M. (1992) Making effective use of potato seed treatments. *Arable Farming April 1992*, 56-57.

Zitter, T.A.,; Halseth, D.E. (1989) Influence of initial and subsequent seed piece treatments for disease development on potato tubers. *Fungicide and Nematicide Tests* 44, 129-130.

FLUAZINAM: A NOVEL FUNGICIDE FOR USE AGAINST *PHYTOPHTHORA INFESTANS* IN POTATOES

B.P. ANEMA, J.J. BOUWMAN

ICI Agro, Ridderkerk, The Netherlands

T. KOMYOJI, K. SUZUKI

Ishihara Sangyo Kaisha Ltd, Tokyo, Japan

ABSTRACT

Fluazinam (IKF-1216, B-1216, 'Frownside', ICIA 0192, 'Shirlan') is a new preventive fungicide being developed for use against many diseases. Fluazinam had a broad antifungal spectrum and showed good preventive effect against plant diseases caused by *Altenaria, Botrytis, Colletotrichum, Phytophthora, Pseudoperonospera, Sclerotinia* and *Venturia* in a glasshouse test. Fluazinam had little curative and systemic activity; however, it showed good residual effect and rain fastness. Field tests demonstrated excellent activity of fluazinam against potato *Phytophthora infestans*.

INTRODUCTION

Fluazinam (3-chloro-N-(3-chloro-5-trifluoromethyl-2- pyridyl)-α,α,α,-trifluoro-2,6-dinitro-p-toluidine) is a molecule discovered by Ishihara Sangyo Kaisha in Japan and developed for the Dutch market by ICI Agro. The molecule is formulated as a 50% suspension concentrate with the tradename 'Shirlan'.

Fluazinam's mode of action has been demonstrated; it has an uncoupling activity on the oxidative phosphorylation with rat-liver mitochondria (Guo *et al* 1991).

In the past four years a large number of trials have been undertaken by ICI and official organisations against *Phytophthora infestans* (late blight) in potatoes. The compound obtained a registration for this outlet in the spring of 1992. This paper presents results of trials in the Netherlands, and the data presented demonstrates the good efficacy of fluazinam against *Phytophtora infestans* in potatoes.

SPECTRUM OF ACTIVITY

The fungicidal spectrum of fluazinam was examined in a glasshouse test. Several crops grown in plastic pots were sprayed with fluazinam and then inoculated with several plant pathogenic fungi to obtain minumum inhibitory concentrations (MIC) for preventive activity against 14 plant diseases.

Fluazinam had a broad antifungal spectrum and showed especially excellent preventive activity against cucumber grey mould, downy mildew, anthracnose and Sclerotinia rot, apple scab, rice blast and tomato late blight. Fluazinam showed, however, weak activity against powdery mildew and rust diseases.

TABLE 1. Protective activity of fluazinam against several important plant diseases (pot test).

Disease	MIC (mg/l)
Grey mould/*Botrytis cinerea* (cucumber)	4
Downy mildew/*Pseudoperonospora cubensis* (cucumber)	8
Anthracnose/*Colletotrichum lagenarium* (cucumber)	8
Scab/*Venturia inaequalis* (apple)	8
Sclerotinia rot/*Sclerotinia sclerotiorum* (cucumber)	16
Blast/*Pyricularia oryzae* (rice)	16
Late blight/*Phytophthora infestans* (tomato)	16
Sheath blight/*Thanatephorus cucumeris* (rice)	31
Gummy stem blight/*Mycosphaerella melonis* (cucumber)	63
Alternaria leaf spot/*Altenaria mali* (apple)	63
Black spot/*Alternaria kikuchiana* (Japanese pear)	63
Powdery mildew/*Sphaerotheca fuliginea* (cucumber)	250
Crown rust/*Puccinia coronata* (oat)	250
Rust/*Gymnosporangium asiaticum* (Japanese pear)	1000

LABORATORY TESTS

Fungicidal properties

Fungicidal properties of fluazinam (curative activity, systemic activity, residual activity and rain fastness) were investigated against tomato late blight. In this experiment, tomato plants (4-5 leaf stage) were inoculated with spore suspension of *Phytophthora infestans* and lesion area was measured to obtain control values after 4 d of incubation.

Curative activity

Tomato leaves were inoculated with spore suspension of *P. infestans* and after 6-12 h treated with fluazinam in order to examine the curative activity. Fluazinam showed little activity in inhibiting lesion development when treated after the establishment of infection, indicating little curative activity.

Systemic activity

Fluazinam was soil drenched to examine its ability to translocate through roots to leaves of tomato plants. This treatment of fluazinam had little effect in inhibiting lesion development. This indicates that fluazinam has little systemic activity through the roots.

Residual activity

Tomato plants which were sprayed with fluazinam were kept in a glasshouse and the leaves were inoculated with spore suspension of *P. infestans* at 1 and 7 d after the treatment. Loss of activity after 7 d of incubation was found to be relatively small compared to the control value of inoculation 1 d after the treatment, indicating good residual activity of fluazinam.

TABLE 2. Residual activity of fluazinam against tomato late blight.

Days after treatment	Disease control (%)			
	500	125	31	8 (mg/l)
1	100	100	88	25
7	100	75	17	0

Rain fastness

Tomato plants were treated with artificial rain (20 mm/h for 2 h) after spraying fluazinam. Loss of activity after artificial rain was relatively small, showing good rain fastness of the chemical.

TABLE 3. Rain fastness of fluazinam against tomato late blight.

Artificial rain	Disease control (%)			
	500	125	31	8 (mg/l)
-	100	100	88	25
+	100	81	44	6

These results show that fluazinam has little curative and systemic activity with no penetration into leaves or roots, requiring application before infection. It has, however, good properties - residual effect and rain fastness - necessary for preventive fungicides.

Effect of fluazinam on infection process of *P. infestans*

Spore germination

Fluazinam was added to the spore suspension of *P. infestans* from the start of incubation of zoosporangia or after the release of zoospores. Both indirect germination and cystospore germination were inhibited by the addition of fluazinam at concentrations higher than 1 mg/l.

TABLE 4. Effect of fluazinam on indirect germination of zoosporangium and cystospore germination of *P. infestans*.

Fluazinam conc.(mg/l)	Inhibition (%)	
	Indirect germination	Cystospore germination
10	100	99
1	99	89
0.1	3	20
Control	0 (40)	0 (90)

Spores were incubated on a glass slide at 20°C

Sporulation

Fluazinam has little curative activity; treatment with fluazinam after the establishment of infection could not inhibit lesion development of tomato late blight. But numbers of spores formed on the lesion decreased at concentrations higher than 10 mg/l. This indicates that fluazinam has inhibitory activity on spore formation.

TABLE 5. Effect of fluazinam on spore formation of *Phytophthora infestans*.

Fluazinam conc.(mg/l)	Inhibition of spore formation (%)
100	97
10	77
control	0 (108×10^4)

This finding that fluazinam inhibits sporulation on the lesion when treated after infection suggests a potential to suppress secondary infection in field conditions.

FIELD TESTS AGAINST *P. INFESTANS* ON POTATOES

Materials and methods

All trials were laid down using the randomised block design with 4 replicates. Spraying was started when plants in the row touched each other and repeated in a weekly to 10-d schedule up to 1 week before desiccation.

The spray solution was applied with a boom of 2 m width, a spray volume of 500 l/ha and a pressure of 2.5-3.0 bar using 160 perforated nozzles.

Assessments were carried out using the scale of the Dutch Plant Protection Service (0 = crop complete death, 10 = crop is healthy).

Trial results

TABLE 6. Trial results in 1988.

Treatment	rate (g AI/ha)	*P. infestans* infection leaves 3/8/88	leaves 2/9/88	tubers (% wt/wt)
Untreated	-	5.3	-*	2
Maneb + fentin acetate	850+288	9.3	6.3	1
Fluazinam	200	10	9.1	0

* On 3/8/88 untreated plots were desiccated because of too heavy disease pressure.

TABLE 7. Trial results in 1989.

Treatment	rate (g AI/ha)	*P. infestans* infection leaves 14/8/89	leaves 12/9/89	tubers (% wt/wt)
Untreated	-	8.9	-*	20.5
Maneb + fentin acetate	850+288	10	9.6	0.8
Mancozeb + cymoxanil	2400+104	10	9.1	3.7
Fluazinam	200	10	9.1	0.6

* On 22/8/89 untreated plots were desiccated because of too heavy disease pressure.

TABLE 8. Trial results in 1990.

Treatment	rate (g AI/ha)	*P. infestans* infection leaves 14/8/90	tubers (% wt/wt)
Untreated		9.4	8.8
Maneb + fentin acetate	850+288	10	0.7
Mancozeb + cymoxanil	2400+104	10	0.2
Fluazinam	200	10	0

TABLE 9. Trial results in 1990.

Treatment	rate (g AI/ha)	*P. infestans* infection leaves 14/8/90	leaves 1/9/90	tubers (% wt/wt)
Untreated		9.9	9.0	7.9
Maneb + fentin acetate	816+276	10	10	0.3
Mancozeb + cymoxanil	2400+104	10	10	0.2
Fluazinam	200	10	10	0

Also in 1991 trials were carried out where the infection was too low to draw any conclusions. The trials in 1992 showed again that fluazinam gives very good protection against *P. infestans* in the leaves. No tuber details from these trials are available yet.

TABLE 10. Summary of results from the trials in the period 1988-1991.

Treatment	rate (g AI/ha)	Mean *P. infestans* infection leaves (20 trials)	tubers (% wt/wt) (12 trials)
Untreated	-	7.2	5.8
Maneb + fentin acetate	850+288	9.4	0.31
Mancozeb + cymoxanil	2400+104	9.6	0.56
Fluazinam	200	9.5	0.09

Conclusions

In the period 1984-1991 ICI Agro carried out 27 trials (22 in the last four years) to investigate the efficacy of fluazinam against *P. infestans* in potatoes. The results show that fluazinam at a rate of 200 g/ha gives excellent protection of the potato crop against leaf- and tuberblight. This effect is equal to or better than the standard treatments based on maneb + fentin acetate or mancozeb + cymoxanil. The advantage of fluazinam however is that the effect is achieved with less input of active ingredient.

REFERENCES

Guo, Z.; Miyoshi, H.; Komyoji, T.; Haga, T.; Fujita, T. (1991): Uncoupling activity of a newly developed fungicide, fluazinam (3-chloro-N-(3-chloro-2,6-dinitro-4-trifluoromethylphenyl)-5-trifluoromethyl-2-pyridinamine). *Biochemica et Biophysica Acta*, **1056**, 89-92.

EPIDEMIOLOGY IN RELATION TO CONTROL OF GREY MOULD (*BOTRYTIS CINEREA*) ON SUNFLOWER

V.J. CHURCH

AFRC Institute of Arable Crops Research, Department of Plant Pathology, Rothamsted Experimental Station, Harpenden, Herts. AL5 2JQ

ABSTRACT

Grey mould, caused by *Botrytis cinerea*, is potentially the most damaging disease of sunflower crops in the UK. This paper describes the development of strategies for the control of *Botrytis* on sunflower based on studies of the epidemiology of the disease. Controlled environment experiments suggest that the length of the latent period, that is the time between infection and the appearance of grey mould symptoms on the plant, depends on the growth stage at which infection takes place. The latent period decreases as the age of the plant at infection increases, so that symptoms appear at about the same time even though infection may have occurred at different growth stages. A rapid increase in the incidence of grey mould was seen in field crops when maximum spore concentrations in spore trap catches, coincided with the end of flowering and the beginning of maturation. Fungicide spray applications have, generally, not controlled grey mould effectively. The implications and possible strategies for control of grey mould on sunflower are discussed.

INTRODUCTION

Sunflower can be infected by conidia of *Botrytis cinerea* deposited on the flower head when climate and physiological growth stage are favourable. Moisture and relatively high temperatures (18°C - 25°C) are required for germination, although conidia of *B. cinerea* can germinate at temperatures ranging from 7°C - 27°C (Peres, 1986). Free moisture must be present to allow penetration to take place and for infection to become established (Ingold, 1986). Alternating periods of high and low humidity further encourage development of the disease. Pollen can also stimulate the germination of conidia and the fungus may establish itself as a saprophyte on senescing pollen and ray flowers (Lamarque, 1975).

Although grey mould on sunflower is likely to be the most damaging disease of sunflower crops in the UK, little is known about the importance of the time of infection or the subsequent development of disease on the heads. This paper reports the results of field and controlled environment experiments which studied the development of grey mould on sunflower in relation to prevailing conditions, concentrations of *B. cinerea* conidia, and the timing of infection, and field experiments on its control by chemical means. The paper also proposes future strategies for grey mould control.

MATERIALS AND METHODS

From 1986 to 1990 fungicide field trials were done at Rothamsted on both late and early maturing cultivars of sunflower (Church *et al.*, 1990; Church & Rawlinson, 1991). The principal fungicide used was carbendazim at the standard rate of 0.25kg/ha plus vinclozolin at 0.5kg/ha in 220 litre, applied at up to five different growth stages using a standard hydraulic sprayer (Wilson, 1972), the APE 80 electrostatic rotary atomizer, and a motorised knapsack mist-blower (Solo Junior 410). In 1986, a range of fungicides was applied, using the electrostatic sprayer, in addition to carbendazim plus vinclozolin. These were benomyl at 0.56kg/ha, chlorothalonil at 1.0kg/ha, iprodione and prochloraz, both at 0.5kg/ha, and propiconazole at 0.125kg/ha. In 1987, carbendazim plus vinclozolin were applied on five different occasions using both hydraulic and electrostatic sprayers. In 1988, up to three times the standard rate was applied by hydraulic sprayer, to study the effect of increasing the rate of application on control of *B.cinerea* head infection. In 1989, prochloraz at 0.5kg/ha was applied with vinclozolin by mist-blower to study the effect of this method of application. In 1990, a similar experiment was done which included application with an hydraulic sprayer with drop leg nozzles. In 1991 fungicides were not applied but the incidence of grey mould in the field was monitored in relation to growth stages, weather conditions and airborne spore concentrations.

In field experiments the early maturing cultivars Avante (Sigco S47) and Allegro were used. Eight plots, 10m x 3.5m, of each cultivar were sown at 12 seeds/m^2. Nitrogen was applied in spring as 'Nitram' at 145kg/ha. The herbicide trifluralin was incorporated into the seed bed, and linuron applied pre-emergence. No fungicides or insecticides were used. The plots were covered with bird-proof netting from emergence to harvest. Rainfall and temperature were recorded at a site less than 1km away. A continuously recording spore trap (Burkard Manufacturing Company, Rickmansworth) was used to measure daily average concentrations of *B. cinerea*, within the plot area, from the beginning of flowering to harvest. Ten plants from within each plot were chosen at random and monitored daily from the beginning of flowering, for growth stage and occurrence of grey mould.

In 1991, in controlled environment experiments, both Avante, and Sunbred 246, a later maturing cultivar, were grown. Plants were sown in 18cm diameter pots filled with a soilless compost of peat, sharp sand and a clay fraction plus a slow release fertilizer. Liquid fertilizer ('Phostrogen') was applied when four pairs of leaves were present and again during bud formation. Day and night temperatures were maintained at 20°C and 13°C respectively with a day length of 16 hours. Light intensity was 280μE/s/m^2 and relative humidity controlled at 85%. Plants were inoculated with a suspension of *B. cinerea* conidia at six different growth stages from the beginning of flowering to the start of maturation. The conidial suspensions were obtained by washing conidia from cultures grown on malt extract agar with distilled water. A haemocytometer slide was used to estimate the concentration of conidia in the suspension. Plants were inoculated by spraying each flower head with 4ml of the suspension containing c.6x10^5 spores/ml, using an aerosol spray-gun. After inoculation

each head was enclosed in a plastic bag for 48 hours and then sprayed daily with water to maintain conditions favourable for fungal growth (Ladsous *et al.*, 1988). Growth stages and disease development on each plant were monitored daily from the beginning of flowering to physiological maturity when seed moisture was at 30%.

RESULTS

In 1986 to 1988 weather conditions favoured infection by *B. cinerea* but little control on late maturing plants was achieved; for example, in 1988, 87% of treated plants were infected by harvest time, compared with 92% untreated plants. However, experiments have shown that early maturing cultivars are more likely to give a positive response to fungicides. In 1986 there was a significant increase in yield of 0.73t/ha after 2 applications of prochloraz + iprodione + chlorothalonil, compared with a decrease of 0.5t/ha on treated plots of a late maturing cultivar. There was a much smaller response in 1987 following 5 applications by both hydraulic and electrostatic sprayers, of carbendazim + vinclozolin on Sunbred 246, with no differences between the two methods of application, or in the incidence of grey mould between treated and untreated plots in which 75% of plants were infected. However, in 1988, when carbendazim + vinclozolin were applied to the earlier maturing cv.Avante on 5 occasions and at up to 3 times the normal rate, the incidence of grey mould at harvest was 53% less than on untreated plots (18% heads infected), when applied at the normal rate, and 72% less when applied at the higher rates. Grain and oil yields increased by 20%, with no significant difference between the rates of application.

In 1989 and 1990 the incidence of grey mould on cv.Avante at harvest was <6%. In these years the concentration of airborne *B.cinerea* conidia during the flowering period was only 10% of that in 1988, while rain-days and rainfall were almost half, and mean air temperatures were about 2.5°C warmer. Little disease developed, and there were no significant differences in grain or oil yields.

In 1991 the progress of the disease during and after flowering, the daily variation in rainfall, mean temperature and numbers of airborne *Botrytis* spores were recorded (Fig.1). Maximum air temperature ranged between 17°C and 27°C with alternating periods of dry and wet weather, conditions which favour the development of grey mould (Lamarque, 1979). Spore concentrations usually increased after rainfall, but there was a gradual increase from 22 July, reaching a maximum on 4 August before declining. The peak spore concentrations corresponded with the end of flowering and the start of maturation for most plants. Grey mould was first noted as early as 5 August but numbers of heads showing signs of infection remained few until 27 August when the rate of incidence increased rapidly. No differences were observed between the two cultivars. The rapid increase in disease occurred about 23 days after the maximum spore concentration.

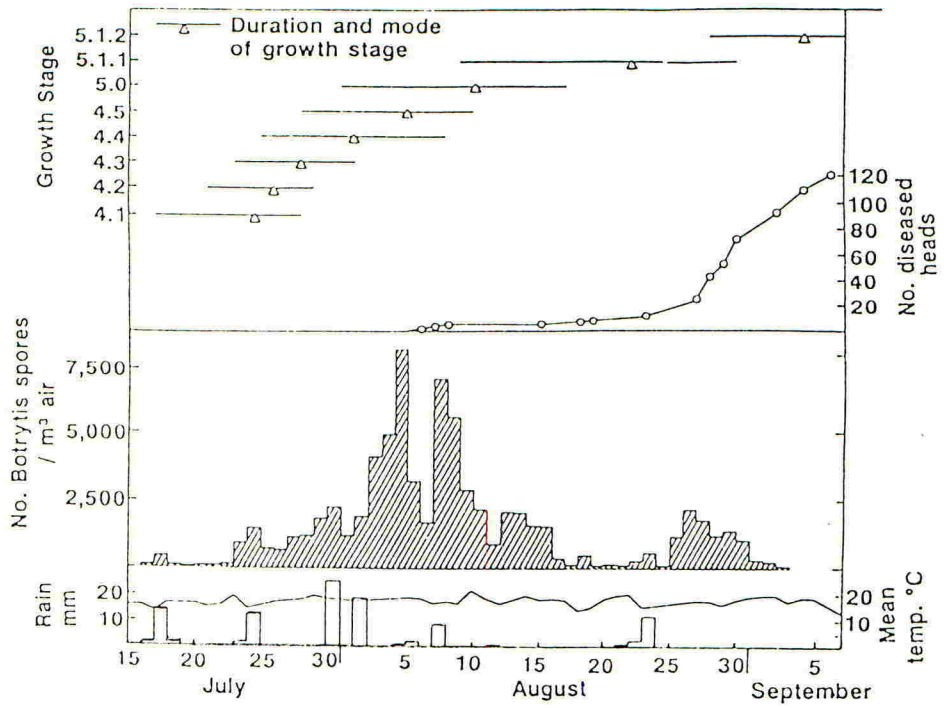

FIGURE 1. Progress of grey mould in relation to weather factors, concentrations of *B. cinerea* conidia and sunflower growth stages (CETIOM, 1984), in the field crop in 1991.

In the controlled environment experiments in 1991 the number of days between inoculation and the appearance of symptoms on plants inoculated at different growth stages is shown in Figure 2. The time from inoculation to the appearance of disease was greater for plants inoculated during the early stages of flowering than for those inoculated at the end of flowering or at the beginning of maturation. The latent period for the later maturing cultivar Sunbred 246, was around 65 days when inoculated towards the end of flowering, GS 4.4 (CETIOM, 1984), but only 35 days when inoculated at GS 5.1.1., the start of maturation, when the moisture content of the seed is *c.*50% and the lower leaves are beginning to senesce. Disease symptoms developed more quickly on cv. Avante, after about 25 days when inoculated at GS 4.4 but as early as 5 days when inoculated at GS 5.1.1. No symptoms appeared on plants inoculated before the production of pollen or towards the end of the maturation period. However, disease symptoms normally appeared at GS 5.1.2 when the moisture content of the seed is *c.*40%, irrespective of when the plants were inoculated (Figure 2).

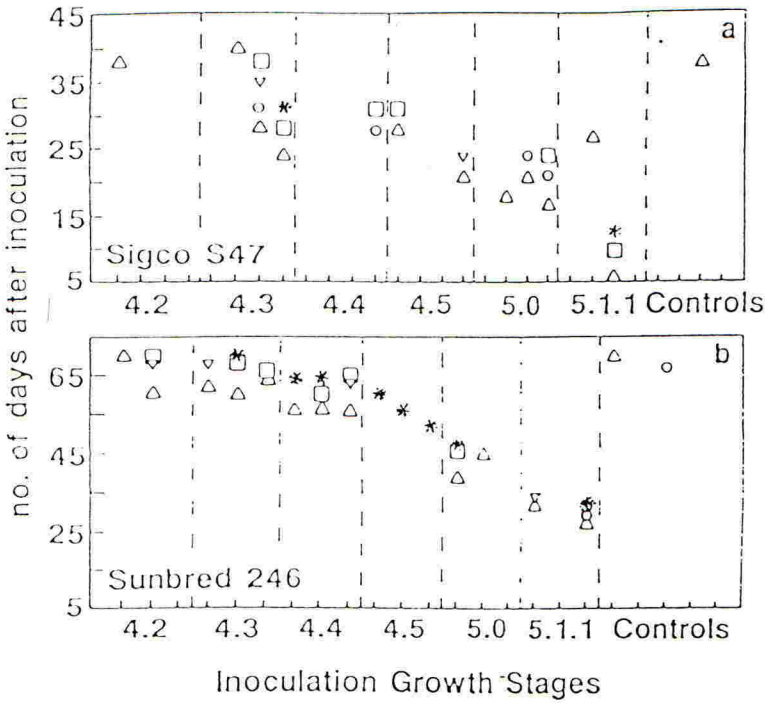

FIGURE 2. Time from inoculation to appearance of symptoms on sunflowers inoculated with *B. cinerea* conidia at different growth stages. Symbols represent the % of severity of infection on individual heads: ▵ 0.1%; ○ 25%; ▿ 50%; □ 75%; ★ 100%

DISCUSSION AND CONCLUSIONS

The results of the controlled environment experiments suggest that the length of the latent period depends on the growth stage at which infection by *B.cinerea* takes place, and that symptoms usually appear at the same time irrespective of the growth stage at infection. The decrease in the observed latent period when heads are inoculated at the end of flowering may be due to the presence of senescing disk florets, ray petals and old pollen which act as a substrate for fungal growth (Rawlinson *et al.*, 1987). In the field crop, the greatest conidial concentrations were recorded when most of the plants were at GS 4.5 and GS 5.0, the end of flowering, and it is probable that most infections took place at that time. Indeed the time between the peak in spore concentration and the appearance of symptoms was about the same as the latent period for cv. Avante when inoculated at GS 4.5. However, it is also possible that some of the late infections may have resulted from conidia deposited at later growth stages when the latent period would have been shorter; or, they could have been caused by conidia deposited earlier when conditions and conidial concentrations were equally favourable, followed by a long latent period.

As infection appears to be directly related to weather conditions and crop maturity it may be possible to devise a forecasting system. However, in the absence of

resistant early maturing cultivars the crop is susceptible to infection over the whole of the flowering period, and consequently only repeated applications of fungicide would be likely to give any degree of control in years when conditions favour infection. Current systemic fungicides give no effective control as they are either absorbed or rapidly degraded before reaching the sites of infection (Peres, 1986). Early maturing cultivars appear to be more responsive to fungicides than late maturing types.

The most damaging symptoms which cause loss of oil yield and quality appear to develop only after physiological maturity (Lamarque, 1985). Other experiments have shown that if a desiccant is applied at this time, when seed moisture is *c.*30%, the effect of the disease is decreased by advancing the harvest date by up to 7 days (Church & Rawlinson, unpublished). Early desiccation and harvesting also maximises the oil yield from early maturing cultivars (Church *et al.*, 1990). As sunflower is grown in the UK as a low input crop desiccation would appear, at present, to be the most efficient and cost effective method of control.

REFERENCES

CETIOM (1984) Les stades repères du tournesol. *CETIOM, INRA, Service de la Protection des Végétaux,* Mars 1984.

Church, V.J.; Rawlinson, C.J.; Fitt, B.D.L. (1990) Development and control of *Botrytis* in UK sunflower crops. *Brighton Crop Protection Conference - Pests and Diseases 1990*, **2**, 739-744.

Church, V.J.; Rawlinson, C.J. (1991) Sunflower - a potential crop for the UK. *Helia*, **14** (15), 117-122.

Ingold, C.T. (1986) *The Biology of Fungi*. Hutchinson, London.

Ladsous, O.; Tourvielle, D.; Says-Lesage, V. (1988) Recherches des sites de pollution, d'infection et de sporulation de *Botrytis cinerea* sur le capitule de tournesol. *Information de la Centre Technique Interprofessionel des Oléagineux Metropolitains*, **105**, 15-20.

Lamarque, C. (1975) Le Botrytis cinerea sur tournesol - Variabilité des symptomes suivant les conditions climatique - Reconnaissance précoce de la maladie. *Défènse des Végétaux*, **173**.

Lamarque, C. (1979) Nécessité de l'alternance de périodes sèches et de périodes humide dans l'extension des épidémies de *Botrytis cinerea* sur capitules de tournesol en France. *Botrytis Symposium*, Amersfoort, Mai 1979.

Lamarque, C. (1985) Evolution permanente de la situation phytosanitaire de tournesol en France. *Phytoma*, April 1985, 20-24.

Peres, A. (1986) *Botrytis cinerea* Pers. Mise au point d'une methode de contamination artificielle et étude de la lutte fongicide sur tournesol. *CETIOM*, Janvier 1986.

Rawlinson, C.J.; Church, V.J.; Inman, A.; Wilson, C. (1987) *Rothamsted Experimental Station Report for 1987*, p.70.

Wilson, J.C. (1972) A spraying machine for small plot experiments. *Experimental Husbandry*, **21**, 25-26.

THE USE OF TEBUCONAZOLE FOR DISEASE CONTROL AND SUBSEQUENT EFFECTS ON LODGING IN OILSEED RAPE

B.J.G. BOLTON, N.M. ADAM

Bayer plc, Crop Protection Business Group, Eastern Way, Bury St Edmunds, Suffolk, IP32 7AH

ABSTRACT

Foliar sprays of tebuconazole were applied to oilseed rape in trials in Great Britain between 1987-91. *Pyrenopeziza brassicae* was well controlled by pre-flowering treatments at all application rates. Control of *Phoma lingam* improved with increasing rate of tebuconazole applied pre-flowering. *Alternaria brassicae* infection on the pods was best controlled by post-flowering applications. The higher the rate of tebuconazole applied pre-flowering, the greater was the reduction in lodging irrespective of which diseases were present. Control of stem diseases and lodging reduction were not so effective from post-flowering applications and yield responses were more variable. Sequential treatments applied pre- and post-flowering demonstrated good overall disease control, lodging reduction and consistent yield increases. Reduced lodging may have resulted from control of stem diseases. In trials where disease levels were low it was often observed that tebuconazole treatments preserved green tissue and delayed natural lodging.

INTRODUCTION

Tebuconazole is a broad-spectrum fungicide developed by Bayer AG. Its chemical and physical properties were described, under the code number HWG 1608, by Reinecke *et al.*, (1986). Early results from trials in Great Britain in which tebuconazole was used to control diseases in oilseed rape were reported by Heatherington and Meredith (1988). This paper summarises the results of further trials carried out in Great Britain and discusses the influence of tebuconazole in reducing crop lodging as a possible consequence of disease control.

MATERIALS AND METHODS

The results from the harvest years 1987-91 are summarised in this paper. In the majority of trials the reported effects on disease control, lodging and yield, were statistically significant (P=0.05) compared with the untreated control. Trials were sited in commercial crops of winter oilseed rape, and were of randomised block design with four replicates and plot sizes of 36-60 m^2.

Tebuconazole was formulated as a 250 g/litre EC or 250 g/litre EW (emulsion in water). Treatments were applied as foliar sprays using knapsack sprayers pressurised with carbon dioxide. Application was

through flat fan nozzles at 250-300 kPa in water volumes of 200-300 l/ha. Rates of use for single treatments were 125, 250 or 375 AI g/ha. In the majority of trials programmes of treatments at two or more timings were included.

Application timing varied between seasons according to changing trial objectives. The main timings were during flower bud development (development stage 3,5-3,7; Sylvester-Bradley, 1985) and post-flowering (4,5-6,1). Additional timings included particularly as components of programme treatments were autumn (1,5-1,10), early spring (2,0), and early petal fall (4,1-4,3). The multiplicity of application rate and timing combinations has necessitated some simplification in summarising these results. Treatment timings have been reduced to three: (1) pre-flowering; (2) post-flowering; (3) sequential treatments combining the two. The pre- or post-flowering timing may have included more than one treatment in that period. For example a pre-flowering treatment could include a programme of autumn, spring, or flower bud development applications. Application rates given in the tables show the total amount of tebuconazole applied in each timing category.

Disease assessments were carried out either by sampling plants or stems followed by grading, or by whole-plot estimates of disease-affected parts of plants.

Estimation of lodging was by visual assessment of whole plots and the calculation of an index based on the degree of lodging and the area of plot affected:

$$\text{lodging index} = \frac{(\%\ \text{area lodged} \times \text{degree of lodging})}{100}$$

The degree of lodging was assessed on the scale: 0 = stems vertical to 100 = stems horizontal. Lodging assessments were typically carried out shortly before harvest.

Trials were harvested using a plot combine-harvester, normally without desiccation of the crop. Seed yield was corrected to 92% d.m.

RESULTS

Pre-flowering applications of tebuconazole were effective in controlling *Pyrenopeziza brassicae*, and there was no dose response up to 375 g/ha (Table 1). When *Phoma lingam* was present disease control was moderate but improved at the higher application rate. Control of *Alternaria brassicae* on the pods was limited. Reductions in the degree of crop lodging appeared to reflect the application rate of tebuconazole. The higher the rate the greater was the reduction of lodging, irrespective of the diseases affecting the trials. Yield results may reflect effects on lodging but were inconsistent.

A different pattern of disease and lodging control was evident from post-flowering treatments (Table 2). *P. brassicae* and *P. lingam* infections on the stems were less well controlled at this timing. *A. brassicae* was more effectively controlled by the later treatments. Where

TABLE 1. Effects of pre-flowering treatments.

Tebuconazole total AI (g/ha)	*Pyrenopeziza brassicae* (stems/pods) 125	250	375	500	*Phoma lingam* (stems) 250	375	*Alternaria brassicae* (pods) 250	375
Disease								
% Reduction	74	71	76	94	28	41	24	17
Unt. (% infection)	16.4	20.6	17.4	20.2	77.5	79.0	4.8	7.6
No. measurements	3	11	21	2	1	1	1	3
Lodging								
% Reduction	45	65	83	91	44	68	64	87
Unt. (index)	44	53	43	20	61	53	36	42
No. measurements	1	6	12	2	1	2	1	3
Yield								
% Increase	2	19	23	18	13	5	17	28
Unt. (t/ha)	2.7	2.6	2.7	1.5	3.6	3.5	3.7	2.9
No. measurements	3	11	21	2	1	2	1	3

Unt. = Untreated

TABLE 2. Effects of post-flowering treatments.

Tebuconazole total AI (g/ha)	*Pyrenopeziza brassicae* (stems/pods) 250	375	500	*Phoma lingam* (stems) 375	*Alternaria brassicae* (pods) 250	375
Disease						
% Reduction	46	38	31	20	74	48
Untreated (% infection)	22.5	27.5	25.3	79.0	4.8	7.6
No. measurements	4	6	2	2	1	3
Lodging						
% Reduction	85	66	29	30	0	34
Untreated (index)	87	59	35	53	36	42
No. measurements	1	3	2	2	1	3
Yield						
% Increase (Decrease)	15	21	6	7	(5)	19
Untreated (t/ha)	2.6	3.1	3.6	3.5	3.7	2.9
No. measurements	4	6	2	2	1	3

TABLE 3. Effects of pre-flowering/post-flowering sequential treatments.

Tebuconazole total AI (g/ha)	*Pyrenopeziza brassicae* (stems/pods)					*Phoma lingam* (stems)			*Alternaria brassicae* (pods)		
	250	375	500	625	750	500	625	750	250	500	750
Disease											
% Reduction	76	100	69	69	77	36	28	48	70	77	75
Untreated (% infection)	37.0	8.0	21.7	10.1	24.1	76.5	74.2	75.7	4.8	4.6	6.2
No. measurements	2	1	13	4	12	5	2	5	1	2	4
Lodging											
% Reduction	100	-	67	41	68	55	41	56	25	55	63
Untreated (index)	87	-	55	62	60	60	62	61	36	41	42
No. measurements	1	0	8	2	8	5	2	5	1	2	4
Yield											
% Increase	23	10	30	26	27	24	23	19	16	32	31
Untreated (t/ha)	3.1	2.7	3.1	3.4	3.2	3.5	3.4	3.7	3.7	3.1	3.3
No. measurements	2	1	13	4	12	5	2	5	1	2	4

P. brassicae occurred on the pods, it too was better controlled at this timing. The variability of lodging results may indicate less reliability of effect at this timing, but results were few. Yield results, though variable, showed increases from the majority of treatments.

Control of *P. brassicae* with sequential treatments (Table 3) mainly reflects the efficacy of pre-flowering treatments, again with little consistent effect of application rate. *P. lingam* infection too was no better controlled by sequential than pre-flowering treatments. Control of *A. brassicae* was good. Lodging was consistently reduced by treatments of 500-750 g/ha, but the results do not indicate any improvement over pre-flowering treatments. Yield responses were unrelated to application rate, but were generally consistent and higher than from pre- or post-flowering treatments.

DISCUSSION

Disease control in these trials supports previously published results with tebuconazole (Heatherington and Meredith, 1988; Kaspers and Siebert, 1989).

Delayed lodging has been reported following use of tebuconazole (Kaspers and Siebert, 1989). Reduced lodging may result from disease control on the stems giving a robust, more lodging-resistant crop. This would apply to *P. brassicae* and *P. lingam* but it seems unlikely that control of *A. brassicae* on the pods would affect lodging. In some trials the levels of stem diseases were low, resulting in only superficial lesions unlikely to weaken the plant significantly, yet lodging was reduced by tebuconazole treatments. It may be that the fungicide, in preserving green tissue, has delayed senescence of the stems and natural lodging of the crop. Instances were reported from some of the trials of tebuconazole prolonging growth and increasing yield.

The timing and rate of treatment for optimum effects against lodging are not clear from these results, but the early application of tebuconazole (pre-flowering or sequential) appeared to be slightly more reliable. Lodging was not completely controlled by tebuconazole treatments but was reduced, resulting in a leaning crop with the pod canopy supported well off the ground.

Yield responses in these trials will naturally have been influenced by disease control, but reduced lodging is likely to have contributed to the increases. Studies with plant growth regulators have demonstrated the beneficial effect of lodging control on oilseed rape yields (Baylis and Wright, 1990; Bolton and Adam, 1987).

Direct yield increases are not the only benefit offered to the grower through the reduction of lodging. Lodged crops present many difficulties at harvest whatever cutting technique is used. Lodged crops are also prone to disease, ripen unevenly and are at risk of seed losses, weed growth, sprouting and poorer seed quality. Thus the value of tebuconazole to the oilseed rape grower may not be limited to its fungicidal efficacy.

REFERENCES

Baylis, A.D.; Wright, I.T.J. (1990) The effects of lodging and a paclobutrazol-chlormequat chloride mixture on the yield and quality of oilseed rape. *Annals of Applied Biology*, 116, 287-295.

Bolton, B.J.G.; Adam, N.M. (1987) Growth regulation of winter oilseed rape with RSW 0411. *Proceedings Crop Protection in Northern Britain*, 214-219.

Heatherington, P.J.; Meredith, R.H. (1988) Disease control with HWG 1608 on cereals and rape. *1988 Brighton Crop Protection Conference - Pests and Diseases*, 3, 953-958.

Kaspers, H.; Siebert, R. (1989) Folicur (tebuconazole) for the control of rape diseases. *Pflanzenschutz-Nachrichten Bayer*, 42, 121-138.

Reinecke, P.; Kaspers, H.; Scheinpflug, H.; Holmwood, G. (1986) BAY HWG 1608, a new fungicide for foliar spray and seed treatment use against a wide spectrum of fungal pathogens. *1986 British Crop Protection Conference - Pests and Diseases*, 1, 41-46.

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

EPIDEMIOLOGY IN RELATION TO CONTROL OF WHITE LEAF SPOT (*MYCOSPHAERELLA CAPSELLAE*) ON OILSEED RAPE

A.J. INMAN, B.D.L. FITT

AFRC Institute of Arable Crops Research, Department of Plant Pathology, Rothamsted Experimental Station, Harpenden, Herts, AL5 2JQ

R.L. EVANS

School of Biological Sciences, Queen Mary and Westfield College, University of London, Mile End Road, London, E1 4NS

ABSTRACT

The sexual stage of *Pseudocercosporella capsellae*, *Mycosphaerella capsellae*, is produced in the autumn and evidence is presented that the air-borne ascospores are responsible for initiating epidemics of white leaf spot in winter oilseed rape. Subsequent disease spread and development is by means of splash-dispersed conidia. Data from field epidemics show that after stem extension the disease progresses up the crop by two inter-dependent mechanisms: the vertical movement of conidia by rain-splash to infect younger, upper leaves, and the vertical movement of these infected, younger leaves by internode growth. Strategies suggested for the control of white leaf spot are based on information about the life cycle of the pathogen and the epidemiological studies.

INTRODUCTION

In the UK, white leaf spot (*Mycosphaerella capsellae*) on oilseed rape is mostly restricted to crops in the south and south west of England. Outbreaks have sometimes been severe on individual crops but no national epidemic has occurred. More recently the disease has been reported in the main oilseed rape growing areas of central and eastern counties. If warmer, wetter winters occur in the UK as a result of predicted climate change it is possible that white leaf spot may become increasingly important. In France the disease is now widespread and yield losses have been reported when the disease has spread from the leaves on to the pods (Penaud, 1986).

M. capsellae has two infective spore stages: conidia, which are splash-dispersed and which are potentially produced throughout the growing season, and ascospores which are air-borne and produced only in the autumn (Inman *et al.*, 1991). Conidia, dispersed only short distances (Fitt *et al.*, 1992), are unlikely to be the primary inoculum for infecting distant crops of oilseed rape. Air-borne ascospores, however, have the potential to be dispersed over greater distances, and their occurrence in the autumn would indicate that they may be a main source of primary inoculum. Although ascospores of *M. capsellae* can infect oilseed rape plants in the glasshouse (Inman *et al.*, 1991), their epidemiological role has not been demonstrated. Survival of the pathogen as

"microsclerotia" (Penaud, 1986) is now considered unlikely as these stromatic structures are not sclerotial in character but are primordia for spermogonia and ascomata (Inman *et al.*, 1991). Survival in the UK is now thought to be by the production of the sexual stage. Ascospores are produced in the autumn and ascomata do not overwinter. Subsequent disease development is therefore by the dispersal of conidia produced on diseased leaves.

Studies were done to investigate whether patterns of disease development suggested a role for air-borne ascospores in initiating epidemics. Studies were also done to determine how the pathogen moves vertically up the crop during and after stem extension, as yields are only likely to be decreased when the disease spreads upwards on to the pods. Information on epidemic development was used to suggest control stategies, especially relating to the use and timing of fungicide applications.

HORIZONTAL DISEASE GRADIENTS

Two field epidemics of white leaf spot were studied. The first was at Rothamsted in 1991, and the second near North Petherton, Somerset, in 1992. Patterns of disease incidence and severity were studied by sampling plants along transects.

The field at Rothamsted was sown with cv. Cobra on 4 September 1990. It had not grown oilseed rape for 5 years and it was therefore unlikely that inoculum was present within the field. The incidence and severity of white leaf spot was greatest at the edge of the crop adjacent to a field that had grown oilseed rape in the previous season. This was thought to be the source of primary inoculum. Therefore plants were sampled along a transect perpendicular to this edge, up to a distance of 100m, on 26 March, 23 April and 15 May.

In the crop at North Petherton, sown on 6 September 1991 with cv. Libravo, plants were sampled along two transects at right angles to each other. The first was sampled on 17 January in an E-W direction over a distance of 230m, and the second on 28 February in a S-N direction over a distance of 270m. The field had never grown oilseed rape, although it had grown swedes in 1985, and it was again unlikely that inoculum was already present in the field.

The patterns of white leaf spot distribution in oilseed rape crops at Rothamsted and North Petherton is good evidence for the role of air-borne ascospores in initiating epidemics in the autumn. At Rothamsted the incidence and severity of white leaf spot decreased with increasing distance from the edge of the crop that was adjacent to the field that had grown oilseed rape in the previous season. The gradients of both incidence and severity were described equally well by negative exponential models or inverse power law models (Fitt *et al.*, 1987). These models accounted, on average, for 73% and 78% of the variance respectively. Such disease gradients are typical of those produced in crops close to a local source of air-borne spores (Gregory, 1973). The gradients could not have developed over such large distances by means of conidia dispersed only very short distances by rain-splash (Fitt *et al.*, 1992). Therefore, the primary gradient was most probably established by air-borne ascospores of *M. capsellae*. The gradient did not change greatly with time, although it did become slightly flatter. This appeared to reflect the

limited distances that the disease could spread from individual foci by means of conidia dispersed in splash droplets.

The distribution of disease in the epidemic at North Petherton also suggested a role for air-borne ascospores in initiating epidemics in the autumn. The even distribution of disease over distances of 230-270m could not readily have been produced by means of conidia dispersed only short distances in splash droplets. However, unlike the Rothamsted epidemic, the pattern of disease in the crop did not indicate a local source of ascospores. Rather, the even distribution of disease over large distances implied a large "area source", distant from the crop (Gregory, 1973).

VERTICAL DISEASE PROGRESS

At the beginning of stem extension in both crops, white leaf spot was present on only the lowest 1-3 living leaves of each infected plant. These leaves did not increase in height at the start of stem extension as their internodes did not extend. Once stem extension had begun, the disease progressed vertically by a combination of two main inter-dependent mechanisms (Figs 1 & 2): (i) Horizontal and vertical movement of conidia in splash droplets to infect younger leaves which, except for growth stages before stem extension, are invariably above those that have symptoms. (ii) Vertical movement of infected younger leaves by internode growth.

For the disease to progress vertically within the crop during the period of rapid stem extension both of these mechanisms must work in combination. The contribution of vertical splash alone is not sufficient. Walklate *et al.* (1989) showed that the maximum height of splash from oilseed rape plants rarely exceeds 10-20cm. Maximum vertical distances that conidia were splashed were estimated as less than 20cm in the Rothamsted epidemic, and 15-16cm in the North Petherton epidemic (Figs 1 & 2), supporting the conclusions of Walklate *et al.* It is therefore important for the pathogen to continue to progress up the plant as the crop grows, so as not to lose the contribution of internode growth to vertical disease progress. This contribution is potentially much greater than that of splash as internode growth can move leaves vertically by as much as 40-50cm (Fig. 2).

At Rothamsted vertical disease progress had started to lag behind plant growth by mid-April; 85% of the leaves that were diseased on 15 May (leaves number 8-15) had not increased in mean height since 23 April and the remainder (leaves number 16-17) had increased in height by on average only 4cm and 8cm respectively (Fig. 1). The potential for internode growth to contribute to vertical disease movement was therefore lost and disease progress was stopped by prolonged dry weather in May and early June as older, infected leaves were lost from the plant by natural senescence.

At North Petherton the disease progressed steadily up the crop throughout the period of internode extension. Rainfall in late February was important in enabling the pathogen to infect young leaves just prior to internode extension. Internode growth then moved these infected leaves up to a height of 32cm, positioning lesions just below new younger, upper leaves whose internodes had not yet extended (Fig. 2). As a result these new leaves were within the range that conidia could be splashed vertically to inoculate

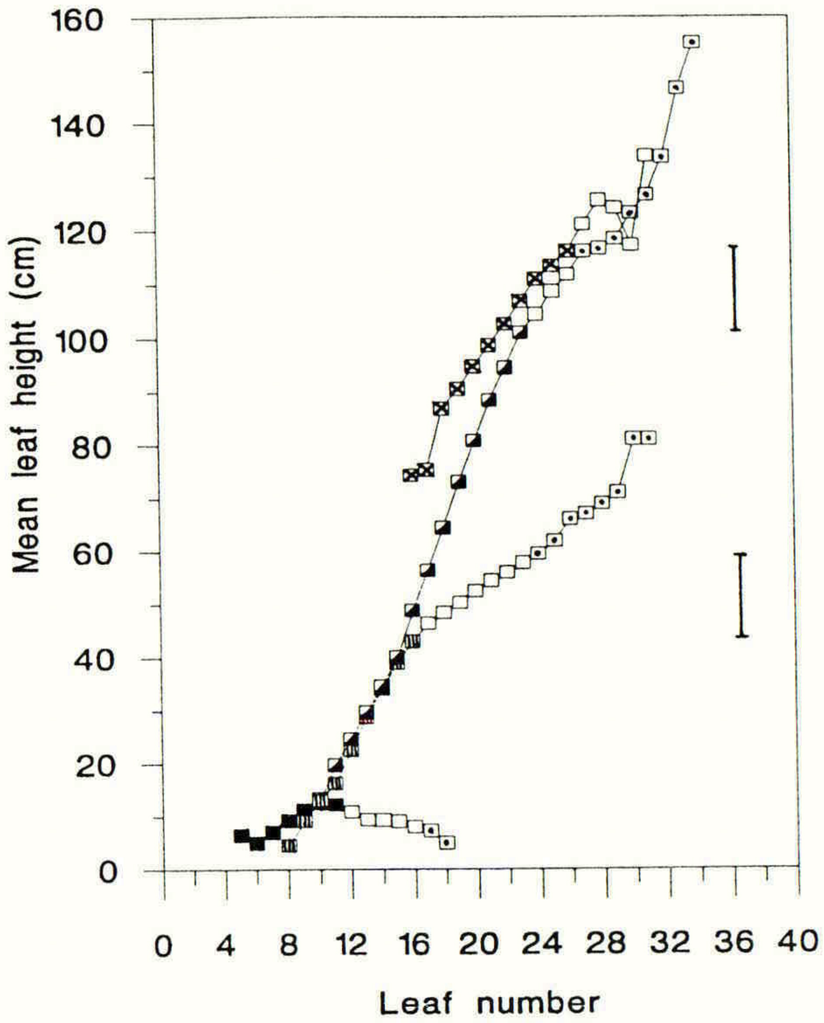

FIGURE 2. Mean heights of oilseed rape leaves with white leaf spot at North Petherton on 28 February (■), 31 March (▥), 29 April (◪) and 28 May (⊠) 1992, or without white leaf spot (□), in relation to leaf number. Heights of leaves present on one assessment date that did not develop white leaf spot at the next (⊡) were used to estimate the maximum height that infective conidia were splashed (vertical bars).

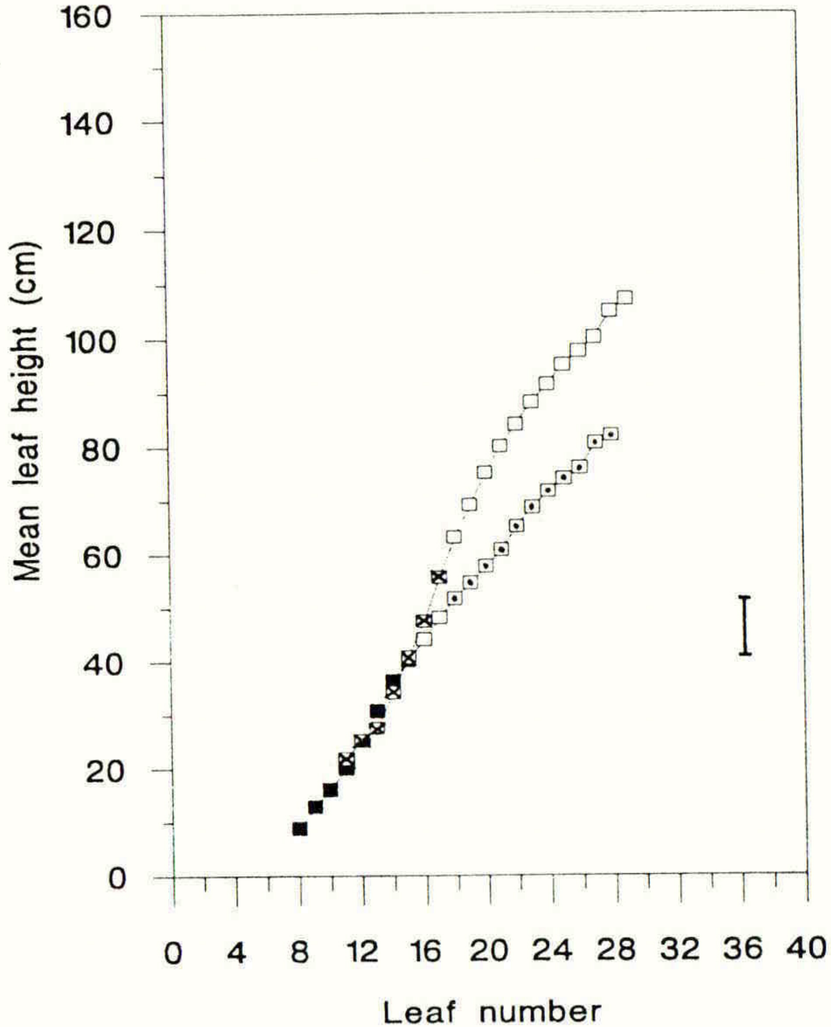

FIGURE 1. Mean heights of oilseed rape leaves with white leaf spot at Rothamsted on 23 April (■) and 15 May (⊠) 1991, or without white leaf spot (□), in relation to leaf number. Heights of leaves present on 23 April which did not develop white leaf spot by 15 May (⊡) were used to estimate the maximum vertical distance that infective conidia were splashed (vertical bar).

and infect them. The potential for internode growth to contribute to vertical disease progress was therefore maintained. Regular rainfall in the following months enabled the disease to continue up the plant by this combination of vertical splash and internode growth. By 28 May, lesions on main stem leaves had reached a maximum height of 119cm, potentially enabling disease spread on to the pod canopy which was at an average height of 130-174cm. Disease progress up to this height had occurred in three pathogen generations from the start of stem extension. However, the number of lesions on the crop was greatly decreased by a carbendazim spray on 6 May, preventing possible spread on to the pods.

CONTROL STRATEGIES

At present white leaf spot remains a minor disease of oilseed rape in the UK and rarely warrants specific control measures. However, it has become increasingly important in France within the last decade and it is possible that strategies for controlling it may be required in the UK in the future. Knowledge of both the life cycle of the pathogen and disease development is important for developing such control strategies.

Cultural practices

Two main cultural practices have potential for control of white leaf spot:

(i) Crop hygiene: Incorporation of crop debris after harvest buries infected debris in which the sexual stage develops and therefore decreases the amount of primary inoculum available for infecting autumn-sown oilseed rape crops.

(ii) Host resistance: In breeding for disease resistance, white leaf spot has only a low priority by comparison with more important diseases of oilseed rape. However, differences in cultivar susceptibility have been observed in both laboratory and field studies in France, and in laboratory experiments at Rothamsted. The cultivars Darmor and Libravo appear to be the least susceptible. Stem resistance to *M. capsellae* may provide a good means of long term disease control. Stem debris is much more durable than leaf debris and provides a viable substrate in which the sexual stage can develop and survive. Furthermore, at the time when sexual primordia are initiated there are few leaves remaining on the plant. The production of stem lesions may therefore be an important link in the life cycle of the pathogen.

Chemical control

Genetic recombination within and between pathogen populations during sexual reproduction provides the possibility for the rapid development of fungicide resistance. As a consequence prophylactic fungicide treatments may quickly become ineffective against white leaf spot. In addition prophylactic treatments are environmentally undesirable and uneconomic. Under the new area payment scheme only one fungicide application per season is likely to be cost-effective, as two-spray programmes will require a 12-15% yield increase to be economic. The requirement is therefore for a control strategy for white leaf spot that minimises the use of fungicides, times applications for maximum efficacy and yield benefit, and is readily incorporated into strategies for control of other oilseed rape diseases.

As the primary source of white leaf spot inoculum in the UK appears to be airborne ascospores produced in the autumn, the application of an autumn fungicide is likely to decrease subsequent disease development. Such a strategy is readily incorporated into disease management programmes which use autumn fungicides against other oilseed rape diseases such as Phoma and light leaf spot. However, in the absence of any other disease problems, autumn applications are unlikely to be the best strategy for controlling white leaf spot. In most years the disease fails to reach the pods and yield losses can be expected only if pod infections occur. After stem extension the disease is highly dependent on frequent and intense rainfall for disease progress to occur up the crop towards the pods. If a period of dry weather enables the younger upper leaves, whose internodes have not yet extended, to be raised above the range that conidia can be splashed from lesions on older, stationary leaves below them, then further vertical disease progress is halted. The older infected leaves are gradually lost by natural senescence and disease progress is halted.

Decisions about the application of fungicides against white leaf spot are therefore best delayed until flowering. If the disease is still present, and is well established on the upper leaves near to the pod canopy, then a summer fungicide treatment may be justified. Sprays applied at flowering have given good control of white leaf spot in France and the UK, and can readily be incorporated into programmes on crops at risk from *Alternaria* and *Sclerotinia*. Procloraz, carbendazim based fungicides and iprodione have all given effective control of white leaf spot (Penaud, 1986; Sumner, 1991).

REFERENCES

Fitt, B.D.L.; Gregory, P.H.; Todd, A.D.; McCartney, H.A.; Macdonald, O.C. (1987) Spore dispersal and plant disease gradients; a comparison between two empirical models. *Journal of Phytopathology*, **118**, 227-242.

Fitt, B.D.L.; Inman, A.J.; Lacey, M.E.; McCartney, H.A. (1992) Splash dispersal of spores of *Pseudocercosporella capsellae* (white leaf spot) from oilseed rape leaves of different inclination, flexibility and age. *Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz*, **99**, 234-244.

Gregory, P.H. (1973) *The Microbiology of the Atmosphere. 2nd Ed.* London: Leonard Hill, pp. 235-262.

Inman, A.J.; Sivanesan, A.; Fitt, B.D.L.; Evans, R.L. (1991) The biology of *Mycosphaerella capsellae* sp.nov., the teleomorph of *Pseudocercosporella capsellae*, cause of white leaf spot of oilseed rape. *Mycological Research*, **96**, 1334-1342.

Penaud, A. (1986) La maladie des taches blanches du colza causée par *Pseudocercosporella capsellae*. *Information techniques*, no. **95-II/1986**, Paris, C.E.T.I.O.M.

Walklate, P.J.; McCartney, H.A.; Fitt, B.D.L. (1989) Vertical dispersal of plant pathogens by splashing. Part II: experimental study of the relationship between raindrop size and the maximum splash height. *Plant Pathology*, **38**, 64-70.

Sumner, D.R.; Bell, D.K.; Lillard, D.A.; Hurst, W.C.; Bush, P.A.; Brenneman, T.B. (1991) Leaf spot control, Maneb, ethylenethiourea residues in processed turnip greens treated with maneb. *Plant disease*, **75**, 878-882.