PROJECT REPORT No. 254

LABORATORY AND FIELD TESTING OF FUNGICIDES FOR CONTROL OF ERGOT IN WHEAT AND RYE

JUNE 2001

Price £8.50

PROJECT REPORT 1341

LABORATORY AND FIELD TESTING OF FUNGICIDES FOR CONTROL OF ERGOT IN WHEAT AND RYE

by

P GLADDERS[1], V J EVANS[2], J F JENKYN[2], K D LOCKLEY[3] AND P G MANTLE[4]

[1]ADAS Boxworth, Battlegate Road, Boxworth, Cambridge CB3 8NN
[2]IACR-Rothamsted, Harpenden, Herts. AL5 2JQ
[3]ADAS Mamhead, Mamhead, Exeter, Devon EX6 8HD
[4]Imperial College of Science, Technology and Medicine, London SW7 2AY

This is the final report of a project lasting three years which started in October 1997. The work was funded by a grant of £109,019 from HGCA (project no. 1341). Part of the work carried out forms the basis of a Ph.D. thesis submitted to Imperial College, University of London, by Miss V J Evans.

CONTENTS

ABSTRACT

This project investigated the opportunities for control of ergot infection in wheat and rye with new fungicides and other chemicals under laboratory, glasshouse and field conditions.

Methodology was standardised to include artificial inoculation with a spore suspension at ear emergence so that inoculated ears produced honeydew and hence inoculum for secondary infection. Laboratory and glasshouse experiments used artificial inoculation only.

Tests on agar showed that 23 of the 34 fungicides tested had at least some activity *in vitro*. Strobilurins were not effective on agar, but showed strong activity when mixed with spore suspensions prior to inoculation of wheat ears. In field screening tests of the materials screened on agar, no fungicides were effective against ergot and seven gave significant increases in the mean numbers and weights of ergots. Glasshouse tests where fungicides were painted onto ears at very large rates showed some useful activity but some treatments were phytotoxic and prevented grain formation. Other glasshouse tests failed to provide any evidence of vapour activity against ergot. A range of fungicides applied to glasshouse-grown plants at stem extension significantly decreased ergot. Sprays at GS 31 were significantly better than sprays at GS 33 but there were no benefits of such early timings in subsequent field experiments.

In individual field experiments, fungicide performance was variable with significant control being achieved only occasionally with azole products (e.g. bromuconazole, epoxiconazole, fluquinconazole and tebuconazole) in some experiments. A number of fungicides significantly increased the number and, especially, weight of ergots, and the greatest effects were from strobilurins. There were no benefits from including additional non-ionic wetter in spray solutions, of using an electrostatic sprayer, of increasing spray volumes, of increasing the dose of active ingredient to double rate or of two-spray programmes.

Studies on the movement of radio-labelled carbendazim, tebuconazole and epoxiconazole showed very limited movement of fungicides painted on the flag leaf, injected into the stem below the ear or applied to an absorbent collar placed around the stem below the ear. When fungicide moved to the ear this was mainly to the glumes rather than to the ovary.

Fungicide sprays are unlikely to provide commercially-acceptable levels of ergot control. In some cases, fungicides may even aggravate the problem. Control of ergot will therefore continue to rely on rotations and cultural control, and avoiding susceptible crops on high-risk sites.

SUMMARY

Ergot is caused by the fungus *Claviceps purpurea*. Its spores infect the ovaries of cereals and grasses, and dark sclerotia ('ergots') are subsequently produced in place of the grains and seeds. These ergots contain alkaloids, which are highly toxic to humans and livestock, and hence very low tolerances are set for ergot contamination in grain for feed or milling. In addition, ergots in seed can introduce the disease into a field, and low tolerances also apply to its presence in seed.

HGCA Research Review No. 25 contains a comprehensive summary of the incidence of ergot in traded grain and explains the biology of the disease. Disposal of ergotised grain is becoming increasingly difficult as legal and commercial standards for grain quality become more stringent. Concern about ergot has increased because of demands for 'traceability' which arise from the need to satisfy the requirements of the Food Safety Act. Buyers and consumers wish to be able to trace-back individual consignments of grain to their point (farm) of origin and be reassured that appropriate standards are met in growing the crop, and in handling and storing the grain.

Whilst ergot affects all cereals and many grasses, there are particular problems with open-flowering species of cereals particularly rye, triticale and durum wheat. There is concern that hybrid cereals may be particularly at risk.

A range of cultural measures and seed treatments are available to reduce the risk of ergot attack. However, these are generally only partially effective, particularly where local sources of ergot are common and the weather during flowering favours infection. Recent HGCA-funded studies at ADAS Cambridge identified a number of fungicides with strong activity against the ergot fungus *in vitro* but attempts to test them in the field were unsuccessful. The project described in this report investigated the opportunities for control of ergot infection in wheat and rye with new fungicides and other chemicals under laboratory, glasshouse and field conditions.

The aims of the project were to :

- Investigate the movement of fungicides and other novel disease control compounds within cereal ears and to develop sensitive assays for detecting these compounds within cereal ovaries.
- Determine the optimum dose rate and timing of fungicides and novel disease control compounds for controlling ergot under field conditions.

Standardised methodology included artificial inoculation with a spore suspension at ear emergence. In field experiments, honeydew produced in the inoculated ears provided inoculum for secondary infection, and assessments were done on both inoculated ears and those infected by secondary spread.

1. Laboratory screening of fungicides

At the start of the project, a diverse range of commercial and experimental fungicides was obtained from agrochemical companies to test for their potential to control ergot in cereal crops. Further compounds were obtained during the course of the project, and the final total was 34. A simple method was developed in which filter paper discs were soaked in different dilutions of the test chemicals. These were then placed on 8% potato dextrose agar (PDA) plates that had been uniformly inoculated with conidia of *C. purpurea,* and the diameters of the inhibition zones were measured. Activity of the chemicals was measured in a series of tests, and carbendazim was included in each, as a standard.

Key results

- Twenty-three of the 34 compounds tested showed evidence of at least some activity *in vitro*.
- Azole type fungicides mostly showed good and broadly similar inhibition; prochloraz was active at smaller concentrations than the rest.
- Other chemical groups were less consistent in their activity. Fenpropidin and tridemorph, for example, both morpholine fungicides, had very different effects.
- Four fungicides (prochloraz, tridemorph, chlorothalonil and Exp. 10830A) inhibited growth at the smallest concentration tested (4 mg a.i. l^{-1}).
- Strobilurin fungicides did not show activity and alternative assays were devised (see glasshouse experiments).
- A summary of minimum inhibitory concentrations and activity relative to carbendazim, determined in the agar plate tests is shown in Table S1.

Table S1. Inhibition of the growth of *C. purpurea* by different fungicides measured 8 days after inoculation.

Fungicide	Min. inhibitory concentration (mg a.i.l^{-1})	Inhibition at 500 mg a.i.l^{-1} (% of carbendazim)	Fungicide	Min. inhibitory concentration (mg a.i.l^{-1})	Inhibition at 500 mg a.i.l^{-1} (%of carbendazim)
Carbendazim	20	100	Triticonazole	100	154
Bromuconazole	100	230	Fenpropidin	2500	-
Cyproconazole	100	224	Fenpropimorph	500	93
Difenconazole	100	130	Tridemorph	4	370
Epoxiconazole	100	160	Cyprodinil	100	46
Flusilazole	100	202	Chlorothalonil	4	103
Flutriafol	500	118	Fludioxinil	20	134
Fluquinconazole	100	48	Iprodione	500	7
Prochloraz	4	220	Mancozeb	500	68
Propiconazole	100	215	Spiroxamine	2500	-
Tebuconazole	100	197	Exp. 10830A	4	119
Triadimenol	500	165			

2. Field screening of fungicides

All but three of the chemicals tested in the laboratory screen (experimental compounds that were supplied in very small amounts and/or in an unformulated state) were also tested in fully replicated, small-plot experiments on winter wheat cv. Riband or spring wheat cv. Chablis in 1998 or spring wheat cv. Chablis in 1999. Each experiment also tested effects of applying the fungicides two to three days before or after inoculating with the pathogen at anthesis (growth stage (GS) 59). A disease resistance activator, applied at GS 32 or GS 39, was also tested.

Key results

- Applying the fungicides two to three days before or after inoculating the ears had no significant effect.
- Only five of the compounds tested gave a decrease in the mean number of ergots produced per inoculated ear compared to the respective untreated controls; all decreases were small and were mostly not significant.
- Seven fungicides significantly increased the average number of ergots per inoculated ear and their average weight. The compound that had the largest effect was kresoxim-methyl.

3. Glasshouse evaluation of fungicides

The results of the field screen, which provided no evidence that any of the fungicides gave useful decreases in ergot, contrasted with the results of the laboratory screen, which showed that many fungicides are active against the ergot fungus, *C. purpurea*. The glasshouse experiments were designed partly to investigate the reasons for this but also to seek possible ways to improve the performance of some of the compounds tested. The contrast between the laboratory and field screens could be explained if the inherent activity of fungicides *in planta* is different from their inherent activity *in vitro*. This was tested by inoculating florets of wheat, using a hypodermic syringe, with different concentrations of inoculum that had been mixed with different concentrations of selected fungicides (carbendazim, cyproconazole, azoxystrobin and kresoxim-methyl). A second experiment tested the effects of applying each of the available fungicides, at relatively large concentrations, directly to the ears of wheat, and also of applying them via absorbent collars wrapped around the stems, just below the ears. Effects on ergot of applying fungicides using an absorbent collar could have been a consequence of their movement in the vascular system or as vapour, and so selected compounds were further tested to determine their potential for vapour activity.

Cereals are only at risk of infection by *C. purpurea* during anthesis. Anthesis is, therefore, the logical and usual time to apply fungicides to control this disease. However, there is published evidence showing showed that when flusilazole and tebuconazole were applied at the 1-2 node stage of development (GS 31-32), small

residues of the compounds were found in the flour. In contrast, when the same chemicals were applied at a later growth stage (flag leaf developed; GS 41) no residues could be detected. The last of the four glasshouse experiments, therefore, tested the effects of applying selected fungicides at earlier growth stages than previously tested, *viz.* to the seed or as a soil drench (depending on the formulations available) or as sprays at GS 31 or GS 33.

Key results

- When ears were inoculated with mixtures of inoculum and fungicides, very few ergots developed at the largest fungicide concentration tested (100 mg a.i. l^{-1}), regardless of inoculum concentration.
- When azoxystrobin and kresoxim-methyl were mixed with relatively small concentrations of inoculum (2 x 10^2 and 2 x 10^3 spores ml^{-1}) they were clearly superior to carbendazim. This assay clearly demonstrated that these two strobilurin fungicides have good activity against the ergot pathogen.
- Several compounds tested by painting them on to the ears, and especially those from the azole group, prevented or significantly decreased numbers of ergots formed.
- Three fungicides from the azole group decreased numbers of ergots formed when applied using absorbent collars.
- There was no evidence, for any fungicide, of vapour activity against ergot.
- None of the ten selected fungicides significantly decreased ergot when applied to the seed or to the soil as a drench but all of them significantly decreased ergot when applied as foliar sprays at stem extension. The average effect of a spray at GS 31 was significantly greater than that of a spray at GS 33.

4. Field experiments to study the effects of applying different fungicides at different rates and times, and using different methods

Many fungicides tested in the laboratory and field screens were also tested in other field experiments on wheat or rye at Rothamsted and on commercial farms in Suffolk and Dorset in 1998-2000. The choice of compounds for testing in these experiments was influenced by results of the screening and glasshouse experiments, and also by other, published, results on the same or different pathogens. Most field experiments also included tests of one or more factors that might influence the efficacy of sprays. These included:

(i) Spray timing (pre- or post-inoculation at anthesis, and during stem extension).

(ii) Increasing amounts of active ingredient deposited on the ears (by increasing concentrations in the spray solution, which has a relatively modest effect, or by using an electrostatic sprayer, which can give more than 20-fold increases in amounts of active ingredient deposited).

(iii) Adding extra wetter to the spray solutions to reduce their surface tensions.

(iv) Applying sprays in larger than normal volumes of water (up to 880 l ha^{-1} instead of the usual 220 l ha^{-1} at Rothamsted, and up to 1000 l ha^{-1} in the experiments on commercial farms).

(v) Applying simulated rain immediately after spraying (2mm applied as course droplets using a tractor-mounted sprayer).

Key results

Table S2 summarises effects on numbers of ergots in inoculated ears of those fungicides that were tested in six or more field experiments (including the field screening experiments). These results, which are similar to those for numbers of ergots in ears affected by secondary spread and weights of ergots per ear, show that:

- Effects of fungicides were very variable and, although not always significant, increases and decreases were almost equally common. The largest increases, especially in the weights of ergots, were often associated with strobilurin fungicides.
- Effects of different fungicides in the same experiment were much more consistent than effects of the same fungicides in different experiments.
- Effects were much larger (in percentage but not necessarily in absolute terms) in experiments on commercial farms (Experiment 5.1 - 5.6) than in experiments at Rothamsted (Experiments 2.1 - 2.3 and 4.1 - 4.5) but were still mostly not significant. The explanation is uncertain.

Table S2. Summary of the effects on numbers of ergots (% increase or decrease compared to unsprayed, inoculated control plots) of the fungicides tested in six or more of the field experiments at Rothamsted or on commercial farms (significant effects in bold).

Fungicide	Experiment number													
	2.1	2.2	2.3	4.1	4.2	4.3	4.4	4.5	5.1	5.2	5.3	5.4	5.5	5.6
Carbendazim	+2	+2	-5	+3	-2			+3	**-27**	+10	-21	-82	+22	-3
Cyproconazole	+5			+3		-8	+2	+4	**-23**	-8	0	-40	+33	**-23**
Epoxiconazole	+15			+5		-5			**-35**	+7	+46	-80	+16	-18
Fluquinconazole			+11		-11		+2	-13			+19	0		
Flusilazole	+15				-12			-2			+1	-80	+35	-13
Tebuconazole	+10			+3		-7	+3	+10	**-54**	0	-30	+20	+2	-10
Fenpropimorph	+10					-4		-2			+34	-46	+40	-18
Azoxystrobin	**+20**			+7		+2		-2	**-37**	-3	+33	-80	+2	-5
Kresoxim-methyl + fenpropimorph	**+40**			+8					**-30**	+7	+8	-36		
Chlorothalonil	+18			+2	-8				**-31**	+8	+17	-19	+10	-3

The results also showed that:

- Altering the timing of fungicide sprays (pre- vs. post-inoculation at anthesis or at stem extension) had no significant or consistent effects.
- Increasing the concentration of active ingredient in the spray solutions or using an electrostatic sprayer to apply them had no significant or consistent effects.
- Adding extra wetter to the spray solution or increasing the volume of water in which fungicides were applied had no significant or consistent effects.
- Applying simulated rain immediately after applying the fungicide sprays had no significant or consistent effects.

5. Movement of radio-labelled fungicides

Suspensions of three radio-labelled fungicides (carbendazim, tebuconazole and epoxiconazole) were applied to glasshouse-grown plants of spring wheat cv. Avans using four different methods:

(i) painting the flag leaf at GS 41 (flag leaf sheath extending)
(ii) injecting directly into the stem 5 cm below the ear at GS 59 (ears fully emerged)
(iii) applying to an absorbent collar placed around the stem directly below the ear at GS 59
(iv) painting the glumes at GS 59

For each method, approximately 1.5 million decompositions per minute were applied to each plant, and samples were taken 5, 14 and 28 days after treatment. For plants treated at GS 41, the flag leaf and developing ear were sampled, and for plants treated at GS 59 the ear was sampled and activity measured in the glumes and in the ovaries (or embryos, after the ovaries had been fertilised, or later still, the developing grain).

Key results

- In plants to which the fungicides were applied by painting the flag leaf, there was an increase over time in the amount of activity detected in the developing ear but this never exceeded 0.1% of the total activity applied.
- When the glumes were painted, there was usually more activity in the ovaries than when the fungicides were painted onto the flag leaves but this was still <3% of the total applied.
- When the compounds were applied to collars placed directly below the ear, small amounts of each moved to the ear (never more than 0.18% of the total activity) but activity was mostly in the glumes.
- Compared with the collar treatment, more activity was detected in the ovaries when compounds were injected directly into the stem but never more than 2.6% of the total applied.

Conclusions

While many fungicides tested showed activity against the ergot fungus *in vitro* and in glasshouse tests, field performance was poor and inconsistent. Inoculated ears may have provided an unusually severe test for fungicides but they also generated inoculum that allowed more natural secondary spread. Significant reductions in ergot were sometimes obtained with azole fungicides applied close to flowering but these were too inconsistent to justify commercial use.

Because the ears are at the top of the canopy and, therefore, very accessible, it might be assumed that ergot is an easy target for fungicidal control. However, the fungus actually infects ovaries and so these are the real targets. For most of the time the ovaries are tightly enclosed and protected by the glumes. This probably explains the usually poor and very inconsistent effects of fungicides in the field experiments described. The only time when the ovaries are accessible to fungicide sprays is when the florets are open to allow cross pollination. However, this does not really offer a practical and general solution because the flowers of cereals usually open for relatively short periods and not simultaneously. It may, nevertheless, explain the decreases in ergot that were obtained in some of the field experiments.

Many fungicides tested are systemic but not in the phloem, and so they do not move readily to the ears, as confirmed in the experiment using radio-labelled compounds. Alternative ways to increase amounts of active ingredient delivered to the ovaries (including larger rates, larger volumes, extra wetter and using an electrostatic sprayer) proved unsuccessful. Early timings of fungicides, at GS 31-33, gave significant decreases in ergot in glasshouse tests but not in the one field experiment in which they were tested.

Implications

Fungicide sprays are unlikely to provide commercially acceptable levels of ergot control, and may even aggravate the problem. The identification of fungicides with activity against ergot may, however, help in the development of new seed treatments, and hence limit amounts of inoculum arising from ergots introduced with seed. Apart from this, control of ergot will continue to rely on rotations and cultural control, and avoiding susceptible crops on high-risk sites. Ergot has been a continuing problem in a range of cereal crops during the course of this project and many of the cases on winter wheat were associated with cv. Rialto. This variety should be avoided where ergot is a problem, and, in future, variety evaluation should consider susceptibility to ergot. Grasses are often infected with ergot and whilst the risk from blackgrass is recognised, other species are more important on some farms. Management of grasses in field margins or on set-aside should, therefore, be considered.

TECHNICAL DETAIL

INTRODUCTION

Ergot is caused by the fungus *Claviceps purpurea.* Its spores infect the ovaries of cereals and grasses, and it subsequently produces dark sclerotia ('ergots') in place of the grain. These ergots contain alkaloids which are highly toxic to man and animals, and hence very low tolerances are set for ergot contamination in grain for feed or milling. In addition, ergots in seed can introduce the disease into a field, and low tolerances also apply to its presence in seed. The life cycle of ergot is summarised in Fig. 1. A comprehensive review of its biology and its importance in traded grain is available in HGCA Research Review No. 25 (Yarham, 1993).

There are no national estimates of the economic damage caused by ergot but losses are primarily associated with reductions in value of contaminated grain. Such downgrading frequently results in a loss of £10 per tonne. The development of effective control measures which prevented downgrading of 100,000 tonnes of cereals per annum would represent a benefit of £1 million per annum.

Disposal of ergotised grain is becoming increasingly difficult as legal and commercial standards for grain quality become more stringent. The United Kingdom Agricultural Supply Trade Association (UKASTA) set limits in 1989 of 0.001% in grain for animal feed and a zero tolerance for all other grain (Anon., 1989). Ergot has assumed greater importance because of the demand for 'traceability' which has arisen from the need to satisfy the requirements of the Food Safety Act. Buyers and consumers want to be able to trace-back individual consignments of grain to their point (farm) of origin and be reassured that minimum standards are met in growing the crop, and in handling and storing the grain. Farmers would have to be able to show that they had done this if there was an outbreak of ergot poisoning in humans or livestock, for example. It can be expected that it will become increasingly difficult to trade samples with ergot contamination because all involved with the production and processing of grain will have to be able to demonstrate 'due diligence', and crops will, therefore, have to be better protected from the disease or grain stocks will need to be cleaned more rigorously.

Whilst ergot affects all cereals (Paveley *et al.,* 1996) and many grasses, there are particular problems with open flowering species of cereals especially rye, which was particularly severely affected in 1994 (Paveley *et al*., 1996), triticale and durum wheat. The susceptibility of these crops has been attributed to the longer period available for infection as the glumes gape to allow cross pollination. The disease occurs much less commonly in barley but there is concern that the introduction of hybrid cereals may greatly increase the risk, even in this crop, as demonstrated by studies on male sterile barley (Wood and Coley Smith, 1980b). Hybrid wheat is now a commercial possibility and some have started to appear in National List Trials. Their advantage lies in their higher yields, and a yield increase of 0.5 t ha^{-1} from hybrids grown on 200,000 ha

could produce benefits for the farming industry of up to £10m per annum. Although it is not anticipated that the hybrid wheats themselves will be particularly prone to ergot, because they are self-pollinating, ergot is potentially a major problem in the production of the hybrid from the parent lines because the male sterile parent is more prone to ergot than conventional wheat. Therefore, development of control measures for ergot is likely to be important for the successful introduction of hybrid wheat into commerce.

Blackgrass (*Alopecurus myosuroides*) has been implicated as a source of ergot infection in wheat (Mantle e*t al.,* 1977) and poor control of this grass weed, perhaps associated with increasing herbicide resistance, could also contribute to increasing ergot problems in the future. Pressures to reduce inputs as grain moves to world prices, and reduced EU support could also aggravate grass weed problems still further. Set-aside with grass weeds may also pose a threat to nearby or subsequent cereals. Annual meadow grass (*Poa annua*) can carry ergot throughout the year and is of particular interest as a potential source of the disease.

A range of cultural measures and seed treatments are available to reduce the risk of ergot attack. However, these are generally only partially effective, particularly where local sources of ergot are common and the weather during flowering is conducive to infection. Chemical control using MBC fungicides applied at anthesis can give reductions in ergot infection but not reliably (Wood and Coley-Smith, 1980b), and there is now a need to evaluate alternative chemical treatments. Recent HGCA-funded studies at ADAS Cambridge (HGCA Project Report No. 126; Yarham, 1996) identified a number of fungicides with strong activity against the ergot fungus *in vitro* but failed to demonstrate their effectiveness in crops because ergot did not develop in experimental plots. Since the 1996 report, further new chemistry for disease control, such as the strobilurins, has become available and could present new opportunities for the control of ergot in the field. However, despite the importance of ergot to some individual farmers, its economic importance on a wider scale is smaller than that of many other diseases and it is not, therefore, considered an important target by the agrochemical industry. The purpose of the project described in this report was, therefore, to investigate the potential for improved control of ergot using new fungicides and other chemicals under laboratory, glasshouse and field conditions.

OBJECTIVES:

(a) To investigate the movement of fungicides and other novel disease control compounds within cereal ears and to develop sensitive assays for detecting these compounds within cereal ovaries.

(b) To determine the optimum dose rate and timing of fungicides and novel disease control compounds for controlling ergot under field conditions.

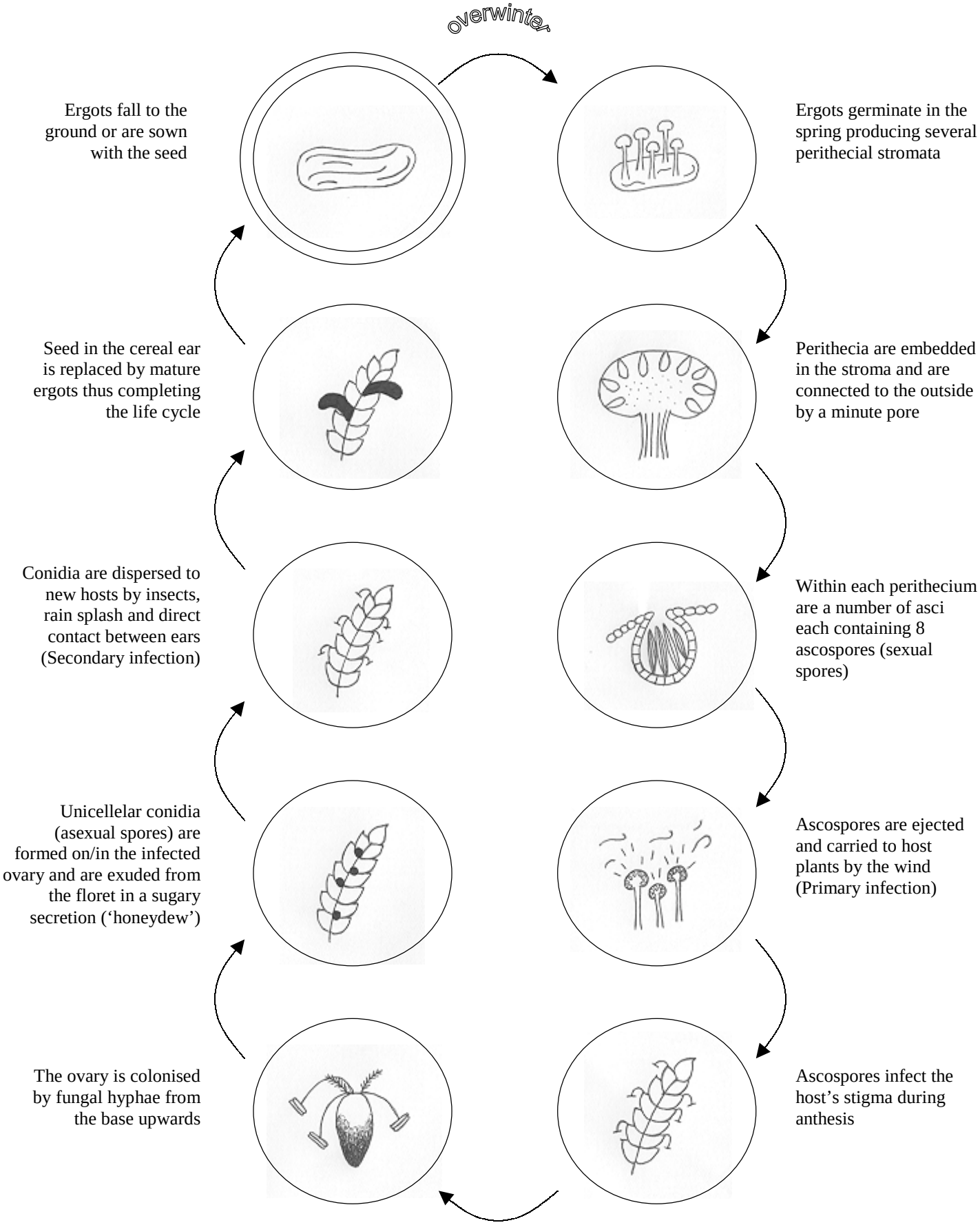

Fig.1 – The life cycle of ergot (*Claviceps purpurea*)

(Modified from Agrios, 1997 and Dillon-Weston, 1948)

CHAPTER 1

LABORATORY SCREEN TO DETERMINE RELATIVE ACTIVITIES OF FUNGICIDES AGAINST *CLAVICEPS PURPUREA*

INTRODUCTION

At the start of this project, substantial numbers of commercial and experimental fungicides were obtained from agrochemical companies to test for their potential to control ergot in cereal crops. Further compounds were obtained during the course of the project, and the final total evaluated was 34. *In vitro* laboratory screens are unlikely to provide results that correlate closely with field performance but they can, in theory, help to identify compounds with inherent activity against the pathogen that can then be subjected to more detailed testing in crops. This chapter describes the initial laboratory-testing phase.

MATERIALS AND METHODS

In vitro activity of fungicides is typically determined by measuring growth of the test fungus on agar media in to which fungicides have been incorporated. This is a reliable and effective procedure, especially when few compounds are being screened against a wide range of potential targets. However, alternative methods may be more efficient when the emphasis is on screening relatively large numbers of compounds against few target species. A simple method was, therefore, developed in which filter paper discs, soaked in appropriate dilutions of the test chemicals (Table 1.1), were placed on agar plates that had been uniformly inoculated with conidia of *Claviceps purpurea,* and the diameters of the inhibition zones measured.

The method used 8% Potato Dextrose Agar (PDA) which supports adequate, but not excessive, growth of the ergot fungus. Various methods of inoculating the plates were tested but the most satisfactory was to spray the plates with an aqueous suspension containing approximately 1×10^6 spores ml^{-1}. This gave reasonably uniform growth, and proved to be quicker and to use less inoculum than flooding the plates with the spore suspension or spreading smaller amounts with a sterile plastic spreader. Some slight improvement in uniformity of growth was achieved by adding a wetter to the spore suspension; 'Citowett' (at 0.05%) proved to be more effective than 'Tween 80'. Sterilisation of the spray head was initially a problem but pre-soaking the head in 10% chloros for 24 hours, and then rinsing with sterile distilled water before use overcame this.

The same bulk isolate was used in all experiments described in this report. It was initially derived from a single ergot (obtained from wheat, and supplied by Nickersons Seeds) which was surface-sterilised before cutting into pieces (*c*. 5 mm long) and plating on to Sucrose Asparagine Agar. Spores obtained from the

original colonies were inoculated into glasshouse-grown wheat to confirm the pathogenicity of the isolate. Honeydew from these plants or similar plants inoculated on subsequent occasions was used as inoculum for the experiments. Spore suspensions kept at 4ºC retained viability for at least 12 weeks. Storage for longer periods was possible by adding glycerol (10% by volume; Mantle *et al.*, 1977) and keeping in a freezer at -20ºC but spores that had been frozen were never used to inoculate the field experiments. Instead, frozen samples were thawed and used to inoculate glasshouse-grown wheat from which honeydew was harvested, as before, to inoculate the plots.

Table 1.1. Fungicides tested in the laboratory screen.

Fungicide	% a.i.	Trade name [1]	Properties [2]	Chemical group
Carbendazim	50	Bavistin	Sys, Pro, Cur	Benzimidazole
Bromuconazole	20	Granit	Sys	Azole
Cyproconazole	10	Alto 100	Sys, Con, Pro, Cur	Azole
Difenconazole	25	Plover	Sys, Pro, Cur	Azole
Epoxiconazole	12.5	Opus	Sys, Pro, Cur	Azole
Flusilazole	40	Sanction	Sys, Pro, Cur, Vap	Azole
Flutriafol	12.5	Pointer	Sys, Con, Pro, Cur	Azole
Fluquinconazole	10	Flamenco	Sys, Pro, Cur	Azole
Prochloraz	45	Sportak	N-Sys, Pro, Cur	Azole
Propiconazole	25	Radar	Sys, Pro, Cur	Azole
Tebuconazole	25	Folicur	Sys, Pro, Cur	Azole
Triadimenol	25	Bayfidan	Sys, Pro, Cur	Azole
Triticonazole	25	Exp 80441B	Sys	Azole
Fenpropidin	75	Mallard	Sys, Pro, Cur	Morpholine
Fenpropimorph	75	Corbel	Sys, Pro, Cur	Morpholine
Tridemorph	75	Calixin	Sys, Pro, Cur	Morpholine
Azoxystrobin	25	Amistar	Sys, Pro, Cur	Strobilurin
Trifloxystrobin	12.5	A9604	Sys, Pro	Strobilurin
Kresoxim-methyl	50	BAS 490 02F	N-Sys, Pro, Cur, Vap	Strobilurin
Cyprodinil	75	Unix	Sys	Anilinopyrimidine
Pyrimethanil	40	Scala	N-Sys, Pro, Cur	Anilinopyrimidine
Chlorothalonil	50	Bravo	N-Sys, Pro	Chlorophenyl
Fludioxinil	2.5	Beret Gold	N-Sys, Con	Cyanopyrrole
Iprodione	25.5	Rovral flo	N-Sys, Con, Pro, Cur	Dicarboximide
Mancozeb	80	Dithane 945	N-Sys, Pro	Dithiocarbamate
Quinoxyfen	50	Fortress	Sys, Pro, Vap	Phenoxyquinoline
Spiroxamine	50	Torch	Sys, Pro, Cur	Spiroketalamine
Silthiofam	12.5	Mon 65507	-	-
Experimental	50	Exp 10623A	-	-
Experimental	50	Exp 10830A	-	-
Experimental	50	Exp 10831A	-	-
Experimental	25	Mon 21250	-	-
Experimental	100	PST 2128	-	-
Experimental	100	PMQ 4534	-	-

[1] Compounds that were experimental when originally supplied are listed using the code number assigned by the manufacturers. Some of them have since been approved and become commercially available but the formulations are not necessarily the same.

[2] Information taken from the Pesticide Manual (Tomlin, 1997) and The UK Pesticide Guide (Whitehead, 1999).

Sys = systemic; N-Sys = non-systemic; Con = contact; Pro = protectant; Cur = curative; Vap = vapour active.

Chemicals were tested at concentrations of 4, 20, 100, 500 and 2500 mg a.i. l^{-1}. Filter paper discs (Whatman ready cut; 6mm diameter (grade AA)) were soaked in the solutions for 2 minutes before blotting them, to remove surplus liquid, and placing them in the middle of a previously inoculated agar plate. On average, each disc absorbed *c*. 36 mg of solution. Because the number of compounds was too large for them to be screened together, they were divided into five groups that were tested at different times. Carbendazim was included as a standard on each occasion. Four replicate plates of each treatment were incubated for eight days at 25°C before measuring the diameters of the inhibition zones. They were then returned to the incubator for a further five days before measuring a second time. Two measurements, at right angles to one another, were made per plate. A microscope was used to examine spores within the inhibition zones to determine whether compounds had affected spore germination or mycelial growth.

Three of the compounds that showed most activity were subjected to further tests using a range of smaller concentrations (0.03, 0.16, 0.8, 4, and 20 mg a.i. l^{-1}). The method used was, otherwise, as described above, including the inclusion of carbendazim as the standard.

RESULTS

There were significant differences in the effects of the standard, carbendazim, in the different groups but they were small in comparison to the differences between chemicals within groups, especially in the middle of the range of concentrations. They mostly reflected consistency between replicates and consequently small standard errors. The results for carbendazim that are presented in this chapter are, therefore, averages for the five groups. Standard errors for the data relating to other chemicals were, mostly, similarly small in comparison to the differences between the chemicals and the effects of different concentrations (see Appendix A), and are, therefore, not presented in the figures (The maximum SED, for tridemorph after 8 days, was ± 2.41 but most of them were <1.00).

Twenty-three of the 34 compounds tested showed evidence of at least some activity against the fungus *in vitro*. Differences were observed in the responses to changing chemical concentrations with tridemorph, for example, having a very steep dose response and chlorothalonil a very shallow response (Fig. 2). Diameters of inhibition zones at each of the concentrations tested for each of the chemicals that showed activity, at 8 and 13 days after inoculation, are shown in Fig. 3 and, with their SEDs, in Appendix A. Apart from the differences in dose responses, there were also clear differences in intrinsic activity, with the azole-type fungicides mostly showing good and broadly similar inhibition although prochloraz was active at smaller concentrations than the rest. Other chemical groups were less consistent in their activity. Fenpropidin and tridemorph, for example, which are both morpholine-type chemicals, apparently had very different effects.

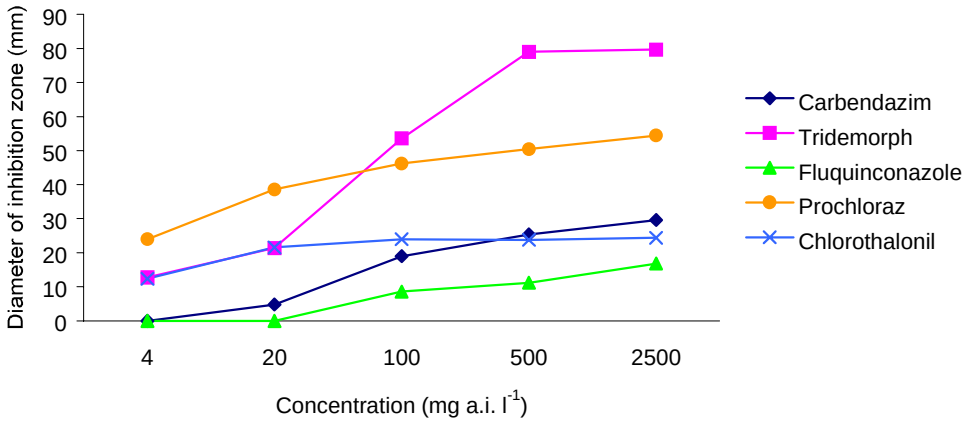

Fig.2. Inhibition of growth of *C. purpurea* by different fungicides, measured 8 days after inoculation.

For some compounds, inhibition zones changed little between the two assessment dates (e.g. cyprodinil) but for others the inhibition zones became smaller or even disappeared, especially at the smaller concentrations. This may have been due to adaptation by the fungus, loss of chemical activity (e.g. by breakdown to less active molecules) or, perhaps most likely, diffusion of the compounds further into the agar so that concentrations within the agar were smaller than those needed to inhibit the fungus, except very close to the assay disc.

Chlorothalonil completely inhibited spore germination at all concentrations tested, and tridemorph did so at concentrations at or above 100 mg a.i. l^{-1}. With other fungicides that exhibited activity, spores were able to germinate but growth of the germ tubes was restricted. In two instances (triadimenol at 500 mg a.i. l^{-1} and bromuconazole at 100 mg a.i. l^{-1}), distinct inhibition zones were observed after 8 days but after 13 days several small colonies had developed within them. Colonies appeared throughout the inhibition zone, even close to the disc where concentrations would still be expected to be fairly high. Resistance to triadimenol has developed in a number of fungi and this may indicate a similar potential in *C. purpurea*.

To simplify comparisons between the fungicides, Table 1.2 shows for each of them, the minimum concentration at which inhibition was detected, and inhibition at 500 mg a.i. l^{-1} expressed as a percentage of the inhibition caused by carbendazim, measured 8 days after inoculation. Carbendazim inhibited fungal growth at concentrations of 20 mg a.i. l^{-1} and above. A few compounds were active at a smaller concentration but most were active only at 100 mg a.i. l^{-1} and above. Fenpropidin and spiroxamine were active only at the largest concentration tested. At 500 mg a.i. l^{-1}, fungitoxicity of most of the twenty-three compounds was comparable to or better than that of carbendazim.

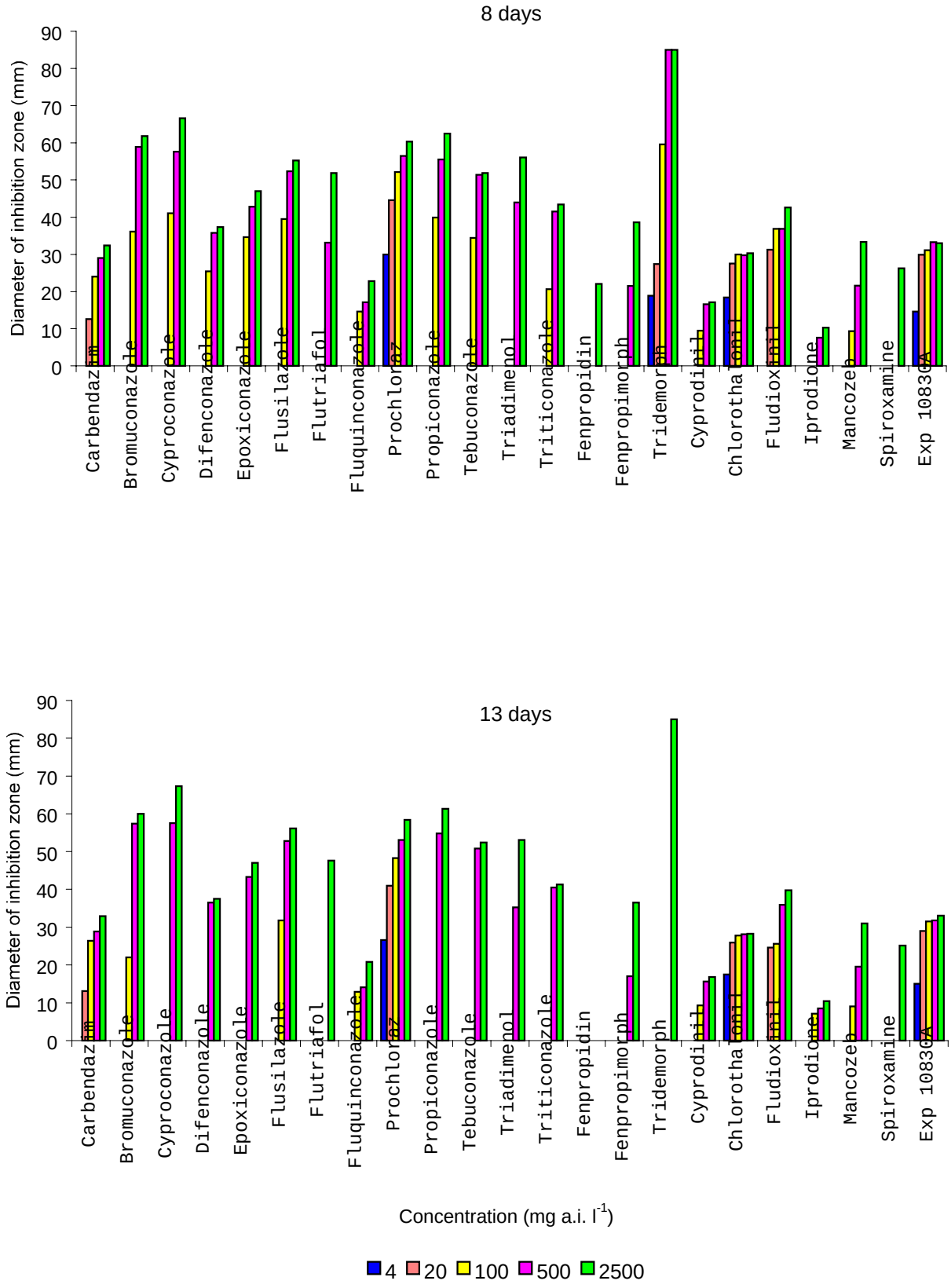

Fig.3. Inhibition of growth of *C. purpurea* by different fungicides, measured 8 and 13 days after inoculation.

Table 1.2. Inhibition of the growth of *C. purpurea* by different fungicides measured 8 days after inoculation.

Fungicide	Min. inhibitory concentration (mg a.i.l^{-1})	Inhibition at 500 mg a.i.l^{-1} (% of carbendazim)	Fungicide	Min. inhibitory concentration (mg a.i.l^{-1})	Inhibition at 500 mg a.i.l^{-1} (% of carbendazim)
Carbendazim	20	100	Triticonazole	100	154
Bromuconazole	100	230	Fenpropidin	2500	-
Cyproconazole	100	224	Fenpropimorph	500	93
Difenconazole	100	130	Tridemorph	4	370
Epoxiconazole	100	160	Cyprodinil	100	46
Flusilazole	100	202	Chlorothalonil	4	103
Flutriafol	500	118	Fludioxinil	20	134
Fluquinconazole	100	48	Iprodione	500	7
Prochloraz	4	220	Mancozeb	500	68
Propiconazole	100	215	Spiroxamine	2500	-
Tebuconazole	100	197	Exp. 10830A	4	119
Triadimenol	500	165			

Four of the fungicides (prochloraz, tridemorph, chlorothalonil and Exp. 10830A) inhibited growth at the smallest concentration tested (4 mg a.i. l^{-1}) when measured 8 days after inoculation (Fig. 3) and all except tridemorph showed a similar effect 13 days after inoculation. These fungicides (except tridemorph which had by then been withdrawn from use), plus carbendazim were, therefore, re-tested using the range of smaller concentrations described above. Chlorothalonil displayed activity at 0.8 mg a.i. l^{-1} but none of the others were active below 4 mg a.i. l^{-1} (Fig. 4).

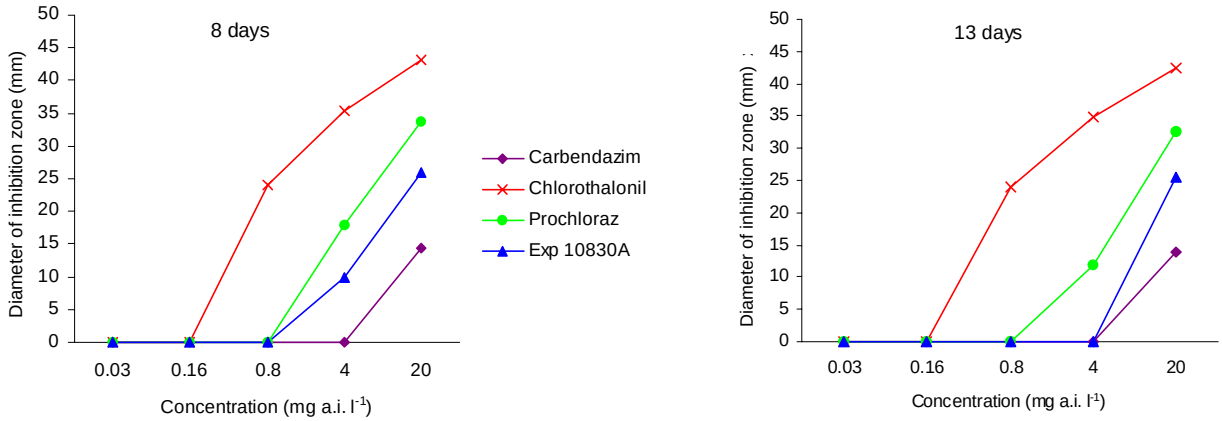

Fig.4. Inhibition of growth of *C. purpurea* by different fungicides, measured 8 and 13 days after inoculation.

DISCUSSION

Although some of the fungicides that were tested showed no activity against *C. purpurea* in the laboratory screen described in this chapter, an unexpectedly large proportion showed activity that was broadly similar to or better than that of carbendazim. Furthermore, it became apparent during the course of the work that agar plate tests are unsuitable for detecting and measuring the activity of at least some of the strobilurin fungicides (Jeremy Godwin, pers. comm.). As a consequence it was not possible to use the results of this laboratory screen, with any confidence, to select a short-list of compounds for more detailed testing under field conditions as had been hoped. It was, therefore, decided that as many as possible of the 34 compounds, including those that showed no activity in the agar plate tests, should also be screened in the field (see Chapter 2).

The results (presented in Appendix A) do, nevertheless, provide much detailed information on the relative activities against *C. purpurea* of different compounds and of the different dose responses both within and between different chemical groups. All of the azoles were active against the fungus but prochloraz was the only one that was active below 100 mg l^{-1}, and it was one of only four compounds that was active at 4 mg l^{-1}. The most active of the compounds was chlorothalonil which, in a supplementary test, inhibited growth of the fungus at a concentration of 0.8 mg l^{-1}.

Fourteen of the fungicides included in the laboratory screen had previously been tested by Yarham (1996) and showed a broadly similar ranking but effective concentrations were different. For example, in the tests described here, the concentration of carbendazim needed to give almost complete inhibition of the growth of *C. purpurea* was 500 mg l^{-1}. In contrast, Yarham found that a concentration of 10 mg l^{-1} was sufficient to inhibit the growth of the fungus when it was incorporated into the agar medium. However, this comparison is potentially misleading because, despite the smaller concentration, amounts of active ingredient needed to achieve almost complete inhibition using Yarham's method were about ten times greater (0.2 mg carbendazim per plate, assuming 20 ml of agar was used per plate) than using the filter paper disc method (only 0.02 mg carbendazim per plate).

CHAPTER 2

FIELD SCREEN TO DETERMINE RELATIVE ACTIVITIES OF FUNGICIDES AGAINST ERGOT

INTRODUCTION

An unexpectedly large number of the 34 fungicides tested in the laboratory screen (see Chapter 1) showed activity against *Claviceps purpurea* (*c.* two thirds of the total) and a number of those that did not, notably the strobilurins, are known to give unreliable results in agar media (Jeremy Godwin, pers. comm.). Differences in the physico-chemical properties (e.g. systemicity or vapour pressure) of those that have activity against the pathogen might also be expected to influence their relative performance in crops, and it was, therefore, decided that as many as possible of the available compounds should be tested on wheat under field conditions at Rothamsted.

MATERIALS AND METHODS

Most of the chemicals tested in the laboratory screen were tested on winter or spring wheat in 1998 (Experiments 2.1 and 2.2, respectively) or spring wheat in 1999 (Experiment 2.3). The exceptions were three experimental compounds (Mon 21250, PMQ 4534 and PST 2128) that were not tested in the field because they were supplied in very small amounts and/or in an unformulated state; none of them showed activity in the laboratory screen. The use of both winter and spring wheat in 1998 was deliberate because it helped to spread the work load.

Each of the three crops was grown on the Rothamsted farm on soils that are mostly flinty silty clay loams. There were 160 plots of winter wheat (cv. Riband) in 1998 that were used to test 18 fungicide treatments, and 80 plots of spring wheat (cv. Chablis) in 1998 that were used to test 9 fungicide treatments and in 1999 that were used to test 8 fungicide treatments. Carbendazim was included in each experiment, as the standard. Among the fungicide treatments in the winter wheat experiment in 1998 (Experiment 2.1) was a formulated mixture of kresoxim-methyl and fenpropimorph but, as both were also tested separately, the results for the mixture are not presented in the summary table (Table 2.1) but are included in Appendix B. The spring wheat experiment in 1998 (Experiment 2.2) also tested a novel resistance activator (Ruess *et al*., 1996) which was not subjected to the laboratory screen. Each of the experiments also tested the effects of applying the fungicides two to three days before or after inoculating with the pathogen at anthesis (growth stage (GS) 59; Zadoks *et al*., 1974). The activator was applied at GS 32 or GS 39. Details of the treatments, and some other basic information about the experiments, are presented in Appendix B. There were four replicate plots

of each treatment, arranged in fully randomised blocks, but 4 unsprayed (inoculated) plots per block in the winter wheat experiment in 1998 and the spring wheat experiment in 1999 (Experiments 2.1 and 2.3, respectively) and 2 unsprayed (inoculated) plots per block in the spring wheat experiment in 1998 (Experiment 2.2). The fungicides were applied at the maximum rates recommended by the manufacturers, using a small-plot hydraulic sprayer that consisted of a hand-held boom connected to a back-pack that propelled the chemical using compressed air. Apart from this, management of the crops was according to standard Rothamsted farm practice.

Each plot measured 3m x 3m and was inoculated with an aqueous suspension of *C. purpurea* conidia (containing *c*. 1.0 x 10^6 spores ml^{-1}; see Chapter 1 for further details) at four points, 0.5 m from each corner. Inoculation was by means of an inoculator developed by Professor P.G. Mantle of Imperial College, London. This consisted of a sprung pad in which were mounted 641 sewing machine needles (with the eyes protruding) and a second felt pad of the same size, which was soaked in the spore suspension. The ears were inoculated by sandwiching them between the two pads. During this process the needles passed through the ear, collected inoculum from the felt pad in the eyes of the needles and then deposited the inoculum in the floral cavity as the needles were withdrawn. Groups of approximately 10 – 15 ears were inoculated at each point, and were marked with a white cable tie.

Shortly before harvest, all inoculated ears were collected and stored in paper bags for later assessment. There was no evidence of secondary spread in the winter wheat in 1998. However, secondary spread did occur in the spring wheat in both years. In 1998, the ergots resulting from secondary spread were mostly large and prominent, and as many as possible of the ears containing them were sampled. In 1999, the ergots resulting from secondary spread were not visible without dissecting the ears and so in that year a random sample of 50 ears was collected from each plot. Inoculated ears were dissected by hand and the ergots counted and weighed. Results were expressed as the mean number and mean weight per ergot. Ears infected by secondary spread were similarly dissected to remove the ergots which were then weighed and the results expressed as the mean weight of ergots per ear (calculated for all visibly infected ears or 50 randomly sampled ears in 1998 and 1999, respectively).

RESULTS

Because of the number involved, the fungicides were tested in three separate experiments in two years. The results for the different fungicides tested in each experiment, including appropriate standard errors, are presented in Appendix B. However, to allow comparisons between treatments tested in different experiments, the results for each treatment in Table 2.1 are expressed as percentages of the corresponding unsprayed, inoculated controls in the same experiment. Applying the fungicides two to three days before or

after inoculating the ears had no significant effect on amount of infection (numbers or weights of ergot) in any of the three experiments.

Table 2.1. Effects of fungicides on numbers and weights of ergots, measured in Experiments 2.1, 2.2 and 2.3, expressed as percentages of their respective unsprayed, inoculated controls [1].

Fungicide	Inoculated ears		Secondarily infected ears
	Mean number of ergots per ear (% of control)	Mean weight per ergot (% of control)	Mean weight of ergots per ear [2] (% of control)
Carbendazim	103	104	78
Bromuconazole	95	99	100
Cyproconazole	105	108	-
Difenconazole	113	129	-
Epoxiconazole	115	161	-
Flusilazole	115	118	-
Flutriafol	108	109	-
Fluquinconazole	111	104	119
Prochloraz	105	111	53
Propiconazole	118	118	-
Tebuconazole	110	120	-
Triadimenol	110	104	-
Triticonazole	121	118	66
Fenpropidin	120	107	-
Fenpropimorph	110	108	-
Tridemorph	93	101	-
Azoxystrobin	120	143	-
Kresoxim-methyl	105	117	111
Trifloxystrobin	118	110	56
Cyprodinil	108	104	-
Pyrimethanil	108	105	100
Chlorothalonil	118	140	-
Fludioxinil	95	109	72
Iprodione	108	116	-
Mancozeb	120	113	-
Quinoxyfen	100	99	111
Spiroxamine	103	101	119
Silthiofam	90	93	111
Exp 10623A	98	112	111
Exp 10830A	100	102	100
Exp 10831A	102	103	122

[1] Data for the three experiments, including SEDs, are presented in Appendix B.

[2] No data for fungicides tested in Experiment 2.1 because there was no secondary spread.

Only five of the compounds tested gave a decrease in the mean number of ergots produced per inoculated ear compared to the respective untreated controls; all of these decreases were small and were mostly not significant (Table 2.1). However, a number of the fungicides tested in Experiments 2.1 and 2.3 significantly increased the average number of ergots per inoculated ear (5 and 2 compounds, respectively; see Appendix Tables B1 and B3). There were broadly similar effects on the average weight of individual ergots in inoculated ears. Thus only one fungicide (silthiofam, in Experiment 2.2) decreased the average weight per ergot but several significantly increased it (and consequently increased the total weight of ergots produced per ear), and especially in Experiment 2.1 where more than half of the compounds tested seemed to have this effect (see Appendix Table B1). The compound that had the largest effect was kresoxim-methyl.

In Experiment 2.3, six of the compounds seemed to decrease the mean weight of ergots per ear in the ears that were infected by secondary spread but the standard errors for these data were very large (58% of the mean) and none of the effects was significant (see Appendix Table B3). None of the increases in mean weights of ergots per ear (Table 2.1) in the ears that were infected by secondary spread, measured in either Experiment 2.2 or Experiment 2.3, was significant.

DISCUSSION

Few of the fungicides tested in the field screen decreased ergot significantly and none of them gave anything approaching commercially-useful levels of control, even when applied before the plants were inoculated with the pathogen. This contrasts with the results of the laboratory screen which showed that a large proportion of the compounds had activity against the fungus that was comparable to or better than carbendazim. The evidence that some fungicides seemed to increase ergot was unexpected and clearly a matter of some practical concern. Glasshouse experiments to help explain some of these results and to investigate some possible ways of improving the performance of fungicides under field conditions are described in the following chapter.

CHAPTER 3

GLASSHOUSE EXPERIMENTS TO INVESTIGATE EFFECTS OF APPLYING FUNGICIDES USING DIFFERENT METHODS AND AT DIFFERENT TIMES

INTRODUCTION

The results of the field screen (Chapter 2), which provided no evidence that any of the fungicides gave useful decreases in ergot, contrasted with the results of the laboratory screen (Chapter 1), which showed that many of the fungicides tested are active against the causal fungus, *Claviceps purpurea*. The glasshouse experiments described in this chapter were designed partly to investigate the reasons for this but mainly to seek possible ways to improve the performance of some of the compounds tested.

The results previously described could be explained if the inherent activity of fungicides *in planta* is different from their inherent activity *in vitro*. Although this seems unlikely, at least for all of the compounds tested, it was considered important to investigate the possibility. It was also considered important to determine whether the increases in ergot that were sometimes detected following the application to wheat of fungicide sprays (particularly of strobilurin fungicides, which can not be tested satisfactorily *in vitro*) were due to stimulatory effects or some other cause. Experiment 3.1, therefore, tested the effects of inoculating florets of wheat with inoculum that had been mixed with selected fungicides (carbendazim, cyproconazole, azoxystrobin and kresoxim-methyl).

Because the ears are at the top of the cereal canopy, they are a very accessible target for fungicide sprays. However, because they are held vertically, they do not intercept as much spray as might be expected (Arnold *et al*., 1984), and perhaps insufficient to achieve effective control when fungicides are applied at normal rates and volumes. Although many of the compounds tested are systemic (see Table 1.1), and might be expected to move readily to the ear, most of them are transported mainly in the xylem and very little, if at all, in the phloem. Experiment 3.2, therefore, tested the effects of applying each of the available fungicides, at relatively large concentrations, directly to the ears of wheat, and also of applying them via absorbent collars wrapped around the stems, just below the ears. Any effects on ergot of applying fungicides using an absorbent collar could have been a consequence of their movement in the vascular system or as vapour. The vapour activity of selected compounds was tested in experiment 3.3.

For practical purposes, cereals are only at risk of infection by *C. purpurea* during anthesis. Infection as a result of artificial inoculation is possible at slightly earlier growth stages but in crops the ovary is not then accessible to natural inoculum. Once pollination has occurred, the embryo quickly becomes resistant to the

fungus. Anthesis is, therefore, the logical and usual time to apply fungicides to control this disease. However, Tanács *et al.* (1998), showed that when flusilazole (as Alert) or tebuconazole (as Folicur Bt EC 225) were applied at the 1-2 node stage of development (GS 31-32), small residues of the compounds were found in the flour. In contrast, when the same compounds were applied at a later growth stage (flag leaf developed; GS 41), no residues could be detected. It has also been reported that loose smut of wheat can be significantly decreased by sprays of triadimefon applied at early stem extension (GS 31), and much more effectively than by sprays applied later, at ear emergence (GS 59) (Jones, 1997). Experiment 3.4, therefore, tested the effects of applying selected fungicides at earlier growth stages than previously tested, *viz.* to the seed or as a soil drench (depending on the formulations available) or as sprays at GS 31 or GS 33.

MATERIALS AND METHODS

Experiment 3.1 Effects of inoculating florets of spring wheat with mixtures of inoculum of *C. purpurea* and fungicides

This experiment tested the effects of adding four fungicides (carbendazim, cyproconazole, azoxystrobin and kresoxim-methyl) to suspensions of *C. purpurea* conidia before injecting them into the florets of wheat. Single pre-germinated seeds of spring wheat (cv. Avans) were sown in a peat-based compost in 11.5 cm pots and grown in a warm glasshouse with supplementary lighting (day/night temperatures of 18ºC/16ºC, and a day-length of 16 h). After the ears had emerged, a single one on each plant was selected (so that all those chosen were at a similar growth stage) and marked. Ten florets on each selected ear were inoculated with the same mixture of inoculum and fungicide using a hypodermic syringe. The experiment tested five concentrations of inoculum (2×10^2, 10^3, 10^4, 10^5 and 10^6 spores ml^{-1}) and five concentrations of each fungicide (0.01, 0.1, 1, 10 and 100 mg a.i. l^{-1}), in all possible combinations. There were four replicates of each of the 25 treatment combinations for each fungicide and of extra plants that were only inoculated, arranged in four fully randomised blocks. Four weeks after inoculation, the numbers of ergots that had formed in each ear were counted.

Experiment 3.2 Effects of applying fungicides directly to the ear or to the stem below the ear

The purpose of this experiment was to test whether control of ergot could be improved by depositing much larger amounts of fungicides on the ear than can usually be achieved with a conventional hydraulic sprayer, and to assess the relative potential of different fungicides to be transported to the ear. Single pre-germinated seeds of spring wheat (cv. Avans) were sown in a peat-based compost in 11.5 cm pots and grown in a warm glasshouse as in Experiment 3.1. Just before anthesis (GS 59) plants at similar growth stages and, where possible, with at least three ears were selected. The fungicides were applied at a concentration of 10 g a.i. l^{-1} using two different methods. One ear on each plant was painted with the fungicide (which, predictably,

deposited much more liquid than would spraying). A collar saturated with the same fungicide was fixed just below the second ear (to test systemic movement to the ear). Pipette filters were used to form the collars, each of which held 1 ml of solution. PVC tape was used to attach the collars below the ears and to seal them in order to reduce evaporation. The third ear (where available) was not treated with fungicide. All ears were inoculated with the ergot fungus (using a hypodermic syringe and a spore concentration of 1×10^6 spores ml^{-1}), either on the day that the chemicals were applied (day 0) or two or four days later (days 2 and 4, respectively). A total of 12 florets was inoculated on each ear; four each at the top, middle and bottom. Because the number of compounds was too large for them to be tested together, they were divided into six groups that were tested at different times. Carbendazim was included as a standard on each occasion. On each occasion, there were three replicates of each treatment, giving a total of nine pots per fungicide, which were fully randomised. Numbers of ergots were counted five weeks after inoculation, and the counts for ears testing paints and collars expressed as percentages of the means for all untreated control ears that were in the same group inoculated at the same time.

Experiment 3.3 Effects of fungicide vapour

In this experiment, eight of the most volatile compounds (carbendazim, cyproconazole, epoxiconazole, flusilazole, fluquinconazole, tebuconazole, kresoxim-methyl and trifloxystrobin) were tested to determine whether vapour activity had the potential to improve field performance. Single pre-germinated seeds of spring wheat (cv. Avans) were sown in a peat-based compost in 11.5 cm pots and grown in a warm glasshouse as in Experiment 3.1. After the ears had emerged, a single one on each plant was selected (so that all those chosen were at a similar growth stage) and the remaining ears removed. Filter papers (Whatman No.1, 5.5 cm diameter) were folded in half and stapled around supporting canes to make flag-like structures. One cane was positioned in each pot so that the 'flag' was above the ear but not touching it. Fungicide (0.5 ml of a 10 g a.i. l^{-1} solution) was pipetted onto the filter paper. A polythene bag was then placed over the ear and saturated filter paper, and sealed at the bottom with a cable tie. After two days, ten florets per ear were inoculated with a suspension of conidia (1×10^6 spores ml^{-1}). At the same time, a further 0.5 ml of fungicide solution was applied to each filter paper. The bags were then re-applied and re-sealed, and left for a further two days before removing them. Numbers of ergots were counted after four weeks. Inoculated ears not exposed to any of the fungicides and sealed in a polythene bag or not, were included as controls. There were four replicates of each treatment arranged in four randomised blocks.

Experiment 3.4 Effect of applying fungicides to the soil or during stem extension

This experiment tested the effects of applying fungicides at much earlier growth stages than previously tested. Fungicides approved for use as seed treatments were applied at the manufacturers' recommended rates (Table 3.1) to seeds of spring wheat (cv. Avans) before sowing in a peat-based compost in 11.5 cm pots and growing in a warm glasshouse as in Experiment 3.1. Three seeds were sown per pot and later thinned to one plant per pot. Other seeds were pre-germinated and sown at one per pot. Plants grown from untreated seed were used to test the effects of applying fungicides as a soil drench at GS 11 (those fungicides not applied as seed treatments) or as foliar sprays at GS 31 or GS 33 (all fungicides). Sprays were applied at concentrations similar to those used in the field (Table 3.1). An average volume of 12.5 ml of spray was used per pot but the smaller amount deposited on the plants was not measured. Concentrations used for soil drenches were 20% of those used for the sprays, and the solutions were applied to the soil surface at 50 ml per pot.

Table 3.1. Fungicide concentrations used for seed treatments and spray applications at GS 31 or 33 in Experiment 3.4.

Fungicide	Seed treatment (g a.i. 100 kg^{-1} seed)	Sprays	
		Field rate (g a.i. ha^{-1})	Concentration (g a.i. l^{-1} assuming 220 l ha^{-1})
Carbendazim	-	250	1.14
Epoxiconazole	-	125	0.57
Flusilazole	-	120	0.55
Fluquinconazole	75.0	150	0.68
Triticonazole	45.0	250	1.14
Fenpropimorph	-	750	3.41
Azoxystrobin	-	200	0.91
Trifloxystrobin	-	200	0.91
Fludioxinil	5.0	25	0.11
Silthiofam	12.5	125	0.57

All plants were grown until ear emergence when at least 2 ears per plant were inoculated with a suspension containing 1 x 10^6 spores ml^{-1} using the inoculator described in Chapter 2. There were four replicates of each treatment (including inoculated controls) that were fully randomised. After six weeks, the total numbers of ergots per ear were counted in the two most infected ears on each plant.

RESULTS

Experiment 3.1 Effects of inoculating florets of spring wheat with mixtures of inoculum of *C. purpurea* and fungicides

The results of this experiment, summarised in Figure 5, are presented as percentages of the total number of florets inoculated in which ergots developed. Average numbers of ergots that formed in ears inoculated with the spore suspensions alone increased from *c.* 3 at a concentration of 2 x 10^2 spores ml^{-1} to *c.* 10 (the maximum) at a concentration of 2 x 10^6 spores ml^{-1}. All of the fungicides showed activity against the pathogen, and they were generally more effective as the concentration of fungicide was increased.

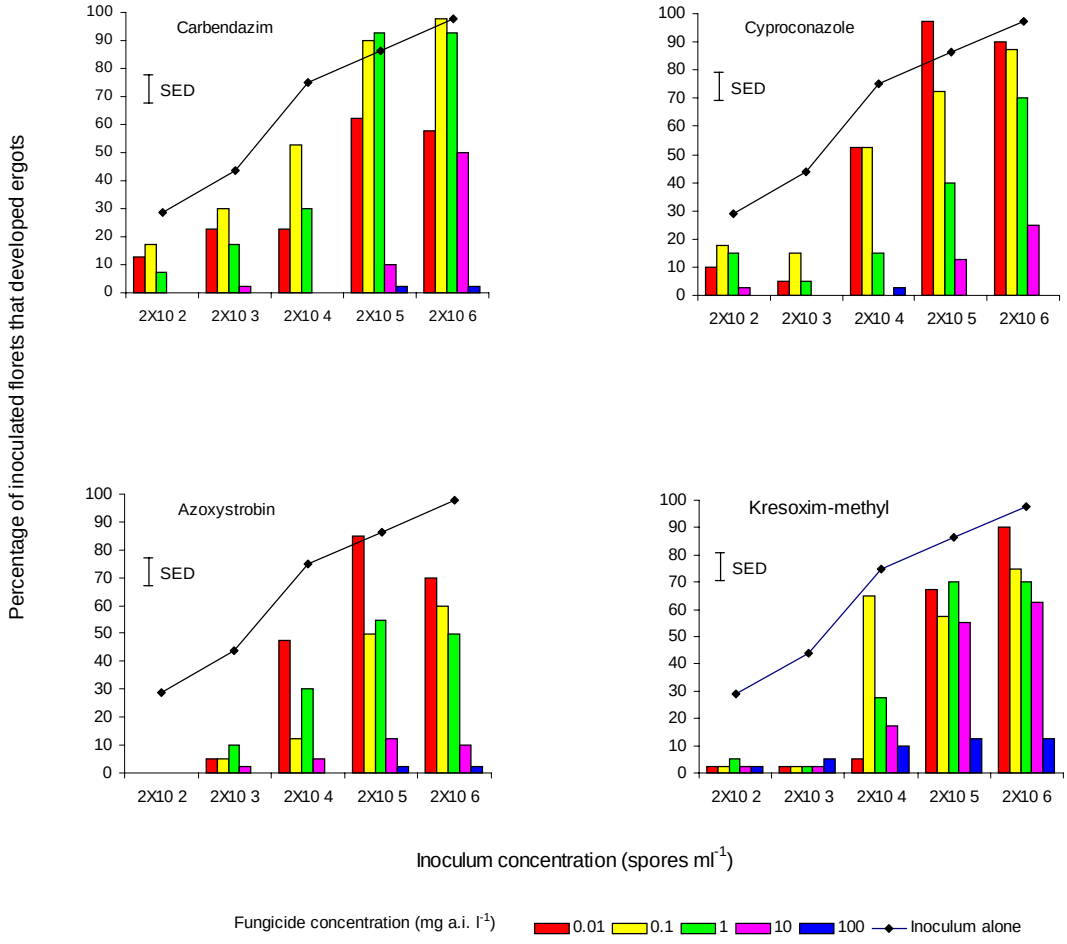

Fig.5. Numbers of ergots (% of inoculated florets) that developed on ears of spring wheat inoculated with suspensions of spores mixed with fungicides (five concentrations of each) compared with the effect of inoculum alone.

At the largest fungicide concentration tested (100 mg a.i. l^{-1}), very few ergots developed regardless of inoculum concentration. Smaller fungicide concentrations were relatively more effective at small inoculum concentrations than at larger inoculum concentrations. At the two smallest concentrations of inoculum (2 x 10^2 and 2 x 10^3 spores ml^{-1}), azoxystrobin and kresoxim-methyl were clearly superior to carbendazim except at the largest fungicide concentrations.

Experiment 3.2 Effects of applying fungicides directly to the ear or to the stem below the ear

Results (i.e. numbers of ergots per ear) for each of the fungicides tested in each of the six groups were expressed as percentages of the numbers of ergots that formed in untreated, inoculated control ears from the same group, and then combined before analysing them. Of the 29 compounds tested in this experiment, several prevented or significantly decreased the number of ergots that were produced. Analyses of the complete data set showed that there were significant differences between fungicides, dates of inoculation and methods of application, and significant interactions between them. However, these results are potentially misleading because when applied directly to the ear, several compounds proved to be phytotoxic to at least some extent, and decreased or prevented the development of normal grain. In extreme cases the ear was bleached as soon as the day after chemical application. Also, there was some evidence that plants were becoming resistant to the pathogen by the last of the three inoculation dates (day 4). The results obtained using the two methods were, therefore, re-analysed separately, omitting the data for day 4 and for the fungicides that proved to be phytotoxic.

The fungicides that proved to be phytotoxic when painted on to the ears were cyproconazole, difenconazole, flusilazole, flutriafol, propiconazole, tebuconazole, fludioxinil, spiroxamine, Exp 10830A and trifloxystrobin. Analysis of the log-transformed data for the remaining fungicides applied using this method showed that differences between the different groups, corresponding to the different occasions on which the materials were tested, were small and unimportant. There were, however, significant differences between the means for the different fungicides (Table 3.2). All but one of the commercially-available compounds from the azole group of chemicals for which data were obtained, significantly decreased the numbers of ergots that were formed compared to carbendazim. One other compound, an experimental material (Exp 10830A, identity not known), also caused a significant decrease. Delaying inoculation until two days after the fungicide treatments had been applied had mostly small and not significant effects on the numbers of ergots that formed.

Only one fungicide, trifloxystrobin, was phytotoxic when applied using an absorbent collar. Analysis of the log-transformed data for other fungicides applied using this method provided some evidence of differences between different groups (occasions on which they were tested). On average, three fungicides from the azole group of chemicals (cyproconazole, epoxiconazole and triadimenol) caused significant decreases in numbers

of ergots compared to carbendazim (Table 3.3). Average numbers of ergots that formed were also decreased by two experimental materials (Exp 10830A and Exp 10831A, identities not known) but effects differed depending on when the ears were inoculated, and not consistently for the two compounds. For most of the other fungicides tested, results were broadly similar whether plants were inoculated on the day that the fungicides were applied or two days later, and this, perhaps, casts doubt on the results for Exp 10830A and Exp 10831A.

Table 3.2. Numbers of ergots (% of untreated) that formed in ears of wheat painted with fungicides and inoculated on the same day or two days later (data are log-transformed values, with back-transformed means in the final column).

Fungicide	Date inoculated		Mean	Back-transformed means
	Day 0	Day 2		
Carbendazim	4.27	4.47	4.37	78.56
Bromuconazole	0.14	1.42	0.78	1.68
Epoxiconazole	-0.43	-0.43	-0.43	0.15
Fluquinconazole	-0.43	1.79	0.68	1.47
Prochloraz	2.01	2.15	2.08	7.52
Triadimenol	0.43	0.67	0.55	1.23
Triticonazole	4.39	4.37	4.38	79.20
Fenpropidin	4.17	3.82	4.00	53.98
Fenpropimorph	4.21	4.00	4.10	59.91
Azoxystrobin	3.91	3.54	3.73	41.06
Kresoxim-methyl	4.95	4.64	4.79	120.30
Cyprodinil	4.57	4.34	4.45	85.10
Chlorothalonil	4.16	4.32	4.24	68.80
Iprodione	4.46	4.40	4.43	83.40
Mancozeb	4.58	4.40	4.49	88.36
Quinoxyfen	4.88	4.89	4.88	131.51
Silthiofam	4.30	4.35	4.32	75.00
Exp 10623A	4.72	4.92	4.82	122.92
Exp 10830A	1.96	2.92	2.44	11.00
SED[1] (101 df)	0.637		0.513 ***	

[1] SEDs are for comparing means for fungicides other than carbendazim with the mean(s) for carbendazim

*** indicates P<0.001

Table 3.3. Numbers of ergots (% of untreated) that formed in ears of wheat plants to which fungicides were applied using absorbent collars, and that were inoculated on the same day or two days later (data are log-transformed values, with back-transformed means in the final column).

Fungicide	Date inoculated		Mean	Back-transformed means
	Day 0	Day 2		
Carbendazim	4.48	4.16	4.32	74.52
Bromuconazole	4.14	4.38	4.26	70.45
Cyproconazole	-1.12	-1.12	-1.12	0.00
Difenconazole	4.28	3.60	3.94	50.77
Epoxiconazole	3.95	2.32	3.13	22.42
Flusilazole	5.57	5.01	5.29	197.41
Flutriafol	4.69	4.93	4.81	121.87
Fluquinconazole	3.86	4.35	4.11	60.13
Prochloraz	4.08	4.25	4.16	63.63
Propiconazole	4.29	4.19	4.24	69.00
Tebuconazole	4.32	3.93	4.12	61.15
Triadimenol	2.94	3.11	3.03	20.13
Triticonazole	4.38	4.39	4.39	79.73
Fenpropidin	2.81	3.94	3.37	28.70
Fenpropimorph	4.26	4.10	4.18	64.83
Azoxystrobin	4.28	4.41	4.34	76.54
Kresoxim-methyl	4.32	4.23	4.28	71.56
Cyprodinil	4.32	4.06	4.19	65.42
Chlorothalonil	5.38	5.58	5.48	239.91
Fludioxinil	4.48	4.42	4.45	85.31
Iprodione	4.45	4.30	4.37	78.88
Mancozeb	4.48	4.23	4.36	77.45
Quinoxyfen	5.02	4.96	4.99	145.76
Spiroxamine	2.83	4.00	3.41	29.87
Silthiofam	5.58	4.88	5.23	186.18
Exp 10623A	4.35	4.35	4.35	77.06
Exp 10830A	0.63	3.83	2.23	8.77
Exp 10831A	3.44	-0.38	1.53	4.12
SED[1] (137 df)	0.583***		0.470***	

[1] SEDs are for comparing means for fungicides other than carbendazim with the mean(s) for carbendazim

*** indicates P<0.001

Experiment 3.3 Effects of fungicide vapour

In this experiment ergots developed in virtually all of the inoculated florets. There was no evidence, for any of the fungicides tested, of vapour activity against the disease, and so no data are presented.

Experiment 3.4 Effect of applying fungicides to the soil or during stem extension

Some of the soil drenches affected plant growth. Fenpropimorph, which had the greatest effect, caused severe stunting and, although the plants produced tillers, they did not produce ears. Effects of other soil drenches were less severe but because they delayed growth, plants were not ready for inoculation until some time after the plants that were treated with sprays. Similarly, treated seed was not pre-germinated and so these plants were also inoculated later than the sprayed plants. Untreated plants were inoculated on each date. To facilitate comparisons between the different treatment dates, results are expressed as percentages of the appropriate inoculated controls (Fig.6).

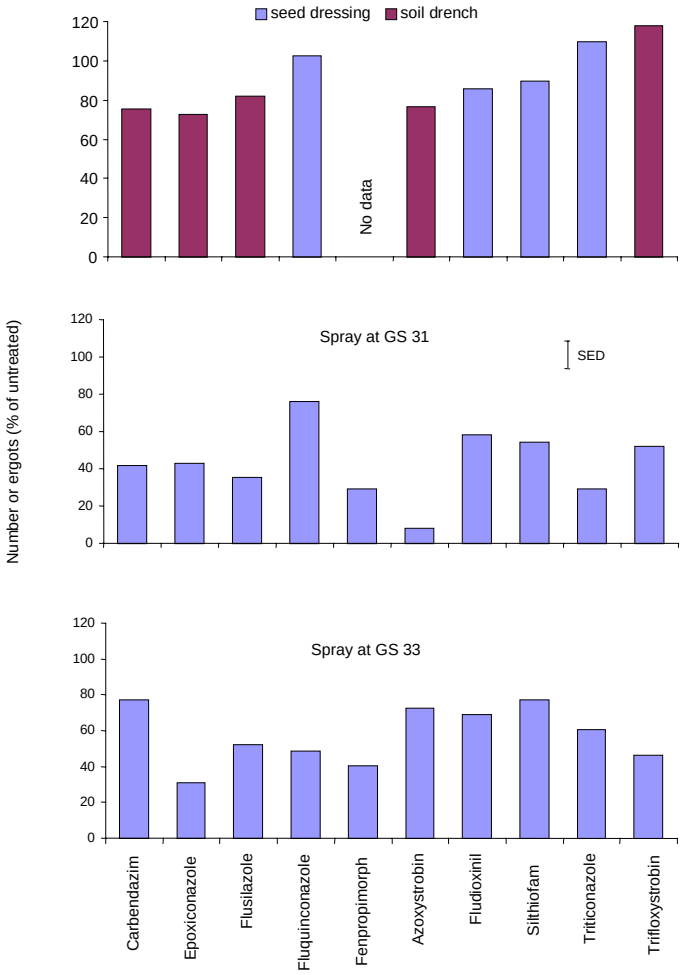

Fig.6. Mean numbers of ergots (% of untreated) that developed in inoculated ears of spring wheat grown from treated seed or after applying fungicides as a soil drench at GS 11 or as foliar sprays at GS 31 or GS 33. (The error bar represents the standard error of differences between means for different fungicides applied at the same or different growth stages)

None of the fungicides significantly decreased ergot when applied to the seed or to the soil as a drench. However, all of them significantly decreased ergot when applied as foliar sprays, and the average effect of the earlier spray (at GS 31) was significantly greater than that of the later spray (at GS 33).

DISCUSSION

The results of Experiment 3.1 suggest that the failure to control ergot in the field screen, using fungicides that had displayed activity against *C. purpurea* in the laboratory screen, was unlikely to have been due to inactivation of the compounds in the plant but probably reflected failure to deliver sufficient amounts of active ingredient to the right place and/or at the right time. Experiment 3.1 also showed that the strobilurin fungicides azoxystrobin and kresoxim-methyl, for which reliable data could not be obtained from the laboratory screen (Jeremy Godwin, pers. comm.), do have fungicidal activity against the disease. The increases in ergot seen in the field screen where these and some other fungicides were applied are, therefore, unlikely to have been due to direct stimulatory effects on the pathogen and must have had some other, indirect, cause.

Because the ears are such an accessible target it might be expected that they would intercept relatively large proportions of sprays that are applied to crops after ear emergence. However, Arnold *et al.* (1984) showed that when sprays were applied to winter wheat using a conventional hydraulic sprayer, the ears intercepted only 5 % of the total. This may usually be sufficient to control diseases affecting the surfaces of the glumes but *C. purpurea* infects the ovaries and so this is, presumably, the real target. For most of the time the ovaries are protected by the glumes. The only exception is when the flowers open to allow cross-pollination, which also provides an opportunity for infection by *C. purpurea*. Fungicide sprays applied at this time may be more effective than those applied when the flowers are closed (Wood & Coley-Smith, 1980a) but it is, unfortunately, difficult to predict when the flowers will open, and they do not do so simultaneously (Wood & Coley-Smith, 1980a). However, the results of glasshouse Experiment 3.2 suggested that, for at least some of the fungicides tested, there might be some scope for improving their performance against ergot under field conditions by increasing the amounts of active ingredient deposited on the ears. Although some of the fungicides tested in this experiment were apparently phytotoxic when painted on to the ears, it is possible that these were effects of the materials in which the fungicides were formulated rather than effects of the active ingredients themselves.

Decreases in ergot when fungicides were applied to absorbent collars fixed around the stem just below the ear, suggested that some compounds have the potential to move systemically to the ears or, perhaps, as vapour. However, Experiment 3.3 provided no evidence that either cyproconazole or epoxiconazole (which decreased ergot in Experiment 3.2 and were also tested in Experiment 3.3) had any useful vapour activity. Experiment 3.4 suggested that applying fungicides at a much earlier growth stage than previously tested

might also contribute to improved control of ergot. The field experiments described in Chapter 4 sought to explore a number of ways in which control of ergot might be improved under field conditions including increasing the amounts of active ingredient deposited on the ears or applying sprays during stem extension rather than at anthesis.

CHAPTER 4

EFFECTS OF APPLYING DIFFERENT FUNGICIDES AT DIFFERENT RATES AND TIMES, AND USING DIFFERENT METHODS, IN FIELD EXPERIMENTS AT ROTHAMSTED IN 1998-2000

INTRODUCTION

The results of the field screen, described in Chapter 2, provided little evidence for useful effects against ergot of applying, at 'normal' field rates, any of the large number of fungicides tested. This is probably because the real targets for the fungicides are the ovaries, which are protected by the glumes and are, therefore, relatively inaccessible. This means that it is difficult to deliver active ingredients to them in the necessary concentrations and/or at the appropriate time. This can, to an extent be overcome by applying fungicides when the glumes are gaping, to allow cross pollination (Wood & Coley-Smith, 1980a), but this does not offer a practical solution because such flowering occurs over an extended period.

The extent to which fungicides can percolate between the glumes to enter the floral cavity is uncertain but this could be the reason why some compounds were so effective when solutions containing relatively large concentrations of active ingredients were painted on to the ears in glasshouse Experiment 3.2. Among the field experiments described in this chapter, were a number that investigated various ways in which the amounts of active ingredient that penetrate the floral cavity, either at the time of spraying or later as a consequence of rain, might, theoretically, be increased. Modest increases in the amounts of active ingredient deposited on the ears can be achieved by increasing their concentration in the spray solution but much larger increases are possible using electrostatic sprayers (Arnold *et al.*, 1984). Both approaches were tested in the experiments described but it can be postulated that penetration will be aided more by increasing the volume of the spray applied than by increasing its concentration. Adding additional wetter to sprays, to reduce their surface tension, might also aid penetration, and has been reported to improve control of ergot in Kentucky bluegrass in Idaho, USA (Schultz *et al*., 1993). Rain falling during or soon after spraying might also help to wash fungicides into the ears, and an experiment testing the effects of simulated rain investigated the potential benefits.

An alternative route for the transport of fungicides to the ovaries, at least theoretically, is by systemic movement through the plant. Apparent activity of some compounds when applied using an absorbent collar wrapped around the stem in glasshouse Experiment 3.2 suggested that some movement can occur in this way. However, the fact that residues of fungicides are seldom detected in grain suggests, perhaps, that it does not normally occur to a significant extent. Other evidence, supported by the results of glasshouse

Experiment 3.4, suggests that fungicides might be more efficiently translocated to the ear while it is still developing than they are during grain filling, and the final experiment described in this chapter, therefore, tested the effects of applying fungicides during stem extension compared to early anthesis.

MATERIALS AND METHODS

In all experiments, individual plots measured 3 m x 3 m, with 1 m paths on each side. For the majority of experiments, and except where otherwise stated, fungicides were applied to plots in 220 l water ha^{-1} using a small-plot hydraulic sprayer that consisted of a hand-held boom connected to a back-pack that propelled the chemical using compressed air. Dates of spray applications and some other basic information about the experiments are presented in Appendix C. Rates of application (g a.i. ha^{-1}), details of which are given below, were the maximum rates approved for use on cereals to control other diseases or those recommended by the manufacturers ('normal' rates) or twice those rates ('double' rates). Each experiment was inoculated at just before anthesis, and on the dates given in Appendix C, with an aqueous suspension of *C. purpurea* conidia (containing *c*. 1.0×10^6 spores ml^{-1}) prepared as described in Chapter 1. Each plot was inoculated at four points, 0.5 m from each corner, using the inoculator described in Chapter 2. Groups of approximately 15 – 20 ears were inoculated at each point, and were marked with a white cable tie. The crop, and the disease, were then left to develop naturally until inoculated and uninoculated ears were sampled at just before harvest.

Experiment 4.1 Winter rye 1998

A number of the fungicides that were tested on winter wheat in the field screen were also tested at the same rates on winter rye, which is much more susceptible to ergot than wheat, in 1998. There were 80 plots (cv. Esprit) that were used to test 9 fungicides (Table 4.1) applied at normal rates two to three days before or after inoculating with the pathogen at just before anthesis (GS 59). There were four replicate plots of each of the 18 treatment combinations, arranged in four randomised blocks, with 2 unsprayed (inoculated) plots per block. Management of the crop was according to standard Rothamsted farm practice.

Assessment methods:

At GS 83 – 87 (dough development) all of the inoculated ears were collected, together with as many as possible of the other ears throughout each plot that had become visibly infected as a result of secondary spread.

Inoculated ears

Inoculated ears were dissected by hand and the ergots counted and weighed. Results were expressed as the mean numbers and weights of ergots per ear.

Secondarily infected ears

Numbers of secondarily infected ears were counted before dissecting them to remove the ergots. Results were expressed as the total numbers of infected ears and the total weights of ergots per plot, and the mean weights of ergots per ear.

Table 4.1. Details of the fungicides tested, and their rates of application to winter rye in 1998.

Active ingredient	Product	Formulation	Application rate per hectare	
			a.i. (g)	Product
Carbendazim	Bavisitin DF	50% w/w WG	275	0.55 kg
Epoxiconazole	Opus	125 g/l SC	125	1.0 l
Tebuconazole	Folicur	250 g/l EW	250	1.0 l
Cyproconazole	Alto 100	100 g/l SL	80	0.8 l
Flutriafol	Pointer	125 g/l SC	125	1.0 l
Azoxystrobin	Amistar	250 g/l SC	250	1.0 l
Kresoxim-methyl + fenpropimorph	Ensign	150 + 300 g/l SE	105 + 210	0.7 l
Cyprodinil	Unix	750 g/l WDG	750	1.0 kg
Chlorothalonil	Bravo	500 g/l SC	1000	2.0 l

Experiment 4.2 Winter rye 1999

Winter rye (cv. Esprit) in 1999 was used to test 9 different fungicides, all applied at double rates (Table 4.2), with and without the addition of an extra wetter (Arma, a non-ionic wetter, applied at 0.33 l ha^{-1} /1.5 ml l^{-1}). The fungicides were applied two to three days before inoculating with the pathogen at just before anthesis (GS 59). There were four replicate plots of each treatment, arranged in four randomised blocks, with 2 unsprayed (inoculated) plots per block, giving a total of 80 plots. Management of the crop was according to standard Rothamsted farm practice.

Assessment methods:

At GS 83 – 87 (dough development) all of the inoculated ears were collected, together with as many as possible of the other ears throughout each plot that had become visibly infected as a result of secondary spread.

Inoculated ears

Inoculated ears were dissected by hand and the ergots counted and weighed. Results were expressed as the mean numbers and weights of ergots per ear.

Secondarily infected ears

Numbers of secondarily infected ears were counted before dissecting them to remove the ergots. Results were expressed as the total numbers of infected ears and the total numbers and weights of ergots per plot, and the mean numbers and weights of ergots per ear.

Table 4.2. Details of the fungicides tested, and their rates of application (double rate) to winter rye in 1999.

Active ingredient	Product	Formulation	Application rate per hectare	
			a.i. (g)	Product
Carbendazim	Bavisitin DF	50% w/w WG	500	1.0 kg
Bromuconazole	Granit	200 g/l SC	400	2.0 l
Difenconazole	Plover	250 g/l EC	150	0.6 l
Flusilazole	Sanction	400 g/l EC	320	0.8 l
Flutriafol	Pointer	125 g/l SC	250	2.0 l
Fluquinconazole	Flamenco	100 g/l EC	300	3.0 l
Prochloraz	Sportak	450 g/l EC	900	2.0 l
Chlorothalonil	Bravo	500 g/l SC	2000	4.0 l
Trifloxystrobin	-	250 g/l EC	500	2.0 l

Experiment 4.3 Winter wheat 1999

Winter wheat (cv. Riband) in 1999 was used to test 9 different fungicides each applied at normal (Table 4.3) or double rates using either the hydraulic sprayer or an electrostatic sprayer. The latter consisted of a 3 m hand-held boom with three electrostatically charged rotary atomisers mounted 1 m apart. Sprays were applied in a water volume of 10.4 l ha^{-1} and the drop size VMD (volume median diameter) was between 80 and 100 µm (Cayley *et al*., 1985).

There were four replicate plots of each of the 36 treatment combinations, arranged in four randomised blocks, with 4 unsprayed (inoculated) plots per block, giving a total of 160 plots. Management of the crop was according to standard Rothamsted farm practice.

Assessment methods:

All of the inoculated ears were collected at GS 83 – 87 (dough development). Because ergots in the ears affected by secondary spread were small and mostly not visible without dissecting the ears, 50 uninoculated ears were sampled at random at the same growth stage.

Inoculated ears

Inoculated ears were dissected by hand and the ergots counted and weighed. Results were expressed as the mean numbers and weights of ergots per ear.

Secondarily infected ears

The randomly sampled ears were dissected to remove the ergots which were counted and weighed. Results were expressed as the mean numbers and weights of ergots per ear.

Table 4.3. Details of the fungicides tested, and their normal rates of application to winter wheat in 1999.

Active ingredient	Product	Formulation	Application rate per hectare	
			a.i. (g)	Product
Cyproconazole	Alto 100	100 g/l SL	80	0.8 l
Epoxiconazole	Opus	125 g/l SC	125	1.0 l
Tebuconazole	Folicur	250 g/l EW	250	1.0 l
Fenpropimorph	Corbel	750 g/l EC	750	1.0 l
Azoxystrobin	Amistar	250 g/l SC	250	1.0 l
Kresoxim-methyl	-	50% w/w	125	0.25 kg
Cyprodinil	Unix	750 g/l WDG	750	1.0 kg
Exp 10623A	-	500 g/l EC	150	0.3 l
Silthiofam	-	12.5% w/w	6.25	50 g

Experiment 4.4 Winter wheat 2000

Winter wheat (cv. Riband) in 2000 was used to test 4 different fungicides, and four treatments that represented different rates, volumes and methods of application (*viz.* normal rate (Table 4.4) and normal volume (220 l ha^{-1}), normal rate and double volume, double rate and normal volume, all applied using the

hydraulic sprayer, and double rate applied using the electrostatic sprayer as in Experiment 4.3). Each of the treatment combinations was followed, or not, by simulated rain which was applied using a tractor-mounted sprayer fitted with coarse nozzles that delivered water at 4000 l ha^{-1}. This is equivalent to 0.4 mm rain and so the five consecutive passes that were made over each plot delivered 2 mm of simulated rain per plot. The simulated rain was applied on the same day as the fungicide sprays, but not until after the last spray had been applied. An extra treatment tested the effects of applying the fungicides at normal rates but in four times normal volume using the hydraulic sprayer. No simulated rain was applied to this treatment. There were four replicate plots of each treatment, arranged in four randomised blocks, with 4 unsprayed (inoculated) plots per block. Management of the crop was according to standard Rothamsted farm practice.

Assessment methods:

All of the inoculated ears were collected at GS 83 – 87 (dough development). Because ergots in the ears affected by secondary spread were small and mostly not visible without dissecting the ears, 50 uninoculated ears were sampled at random at the same growth stage.

Inoculated ears

Inoculated ears were dissected by hand and the ergots counted and weighed. Results were expressed as the mean numbers and weights of ergots per ear.

Secondarily infected ears

The randomly sampled ears were dissected to remove the ergots which were counted and weighed. Results were expressed as the mean numbers and weights of ergots per ear.

Table 4.4. Details of the fungicides tested, and their normal rates of application to winter wheat in 2000.

Active ingredient	Product	Formulation	Application rate per hectare	
			a.i. (g)	Product
Cyproconazole	Alto 240	240 g/l SL	80	0.33 l
Fluquinconazole	Flamenco	100 g/l EC	150	1.5 l
Tebuconazole	Folicur	250 g/l EW	250	1.0 l
Triadimenol	Bayfidan	250 g/l EC	125	0.5 l

Experiment 4.5 Spring wheat 2000

Spring wheat (cv. Chablis) in 2000 was used to test 9 fungicides all applied at double rates (Table 4.5) either at GS 31-32 (1-2 nodes detectable) or GS 59 (ear emergence). There were four replicate plots of each treatment, arranged in four randomised blocks, with 2 unsprayed (inoculated) plots per block. Management of the crop was according to standard Rothamsted farm practice.

Assessment methods:

All of the inoculated ears were collected at GS 83 – 87 (dough development). Because ergots in the ears affected by secondary spread were small and mostly not visible without dissecting the ears, 50 uninoculated ears were sampled at random at the same growth stage.

Inoculated ears

Inoculated ears were dissected by hand and the ergots counted and weighed. Results were expressed as the mean numbers and weights of ergots per ear.

Secondarily infected ears

The randomly sampled ears were dissected to remove the ergots which were counted and weighed. Results were expressed as the mean numbers and weights of ergots per ear.

Table 4.5. Details of the fungicides tested, and their rates of application (double rate) to spring wheat in 2000.

Active ingredient	Product	Formulation	Application rate per hectare	
			a.i. (g)	Product
Carbendazim	Bavisitin DF	50% w/w WG	500	1.0 kg
Cyproconazole	Alto 240	240 g/l SL	160	0.66 l
Fluquinconazole	Flamenco	100 g/l EC	300	3.0 l
Flusilazole	Sanction	400 g/l EC	320	0.8 l
Tebuconazole	Folicur	250 g/l EW	500	2.0 l
Triadimenol	Bayfidan	250 g/l EC	250	1.0 l
Fenpropimorph	Corbel	750 g/l EC	1500	2.0 l
Azoxystrobin	Amistar	250 g/l SC	500	2.0 l
Silthiofam	-	12.5% w/w	12.5	100 g

Statistical analyses

The field experiments described in this chapter, were all of factorial design. All of them included unsprayed, inoculated controls as extra plots (which were not part of the factorial structure) and of which there were always more replicates than of the individual treatment combinations. One of the experiments (Experiment 4.4) tested a much more complex set of extra treatments. As a consequence of these design features, different standard errors of the differences between means (SEDs) are appropriate for making different comparisons; for example between means for the different fungicide treatments or between means for any one fungicide treatment and the corresponding untreated control. For simplicity the SEDs presented in the tables are the largest of the two or more that could be presented. Analyses were done on the untransformed data, and also after doing log transformations, mainly to stabilise variances. However, the range of variation in the results was mostly relatively small so that transformations conferred little benefit. Therefore, in this chapter only the untransformed data are presented. The results of the analyses are shown in the tables of results as the level of significance in the F-tests (*, ** or *** to indicate probabilities of <0.05, 0.01, 0.001, respectively).

RESULTS

Experiment 4.1 Winter rye 1998

Inoculated ears

Average numbers of ergots in the inoculated ears were always greater in the fungicide-treated plots than in the untreated (Table 4.6) but not significantly so. There were similar effects of the fungicides on mean weights of ergots per ear (Table 4.7), and the average for all fungicides was significantly greater than the untreated ($P<0.05$). In plots treated with azoxystrobin, the mean weight of ergots per ear was 28% greater than in the untreated plots. Timing of sprays had no significant effect on numbers of ergots per ear. Mean weight of ergots per ear was significantly smaller where sprays were applied after inoculation than where they were applied before inoculation but the difference was small (6% less).

Secondarily infected ears

There were relatively large, and significant, effects of the fungicides on numbers of ears that were infected as a result of secondary spread (Table 4.8). However, while epoxiconazole significantly decreased the number compared to untreated, most fungicides increased the number and significantly so in the case of azoxystrobin (23% more than untreated plots). On average, plots sprayed after inoculation had fewer infected ears than those sprayed before inoculation but the difference was not significant.

Differences between the fungicides in the mean weights of ergots per ear (Table 4.9) were relatively small and not significant and so the total weights of ergots per plot (Table 4.10) tended to reflect the differences in numbers of ears with ergot. Both mean weights of ergots per ear and total weights per plot were significantly smaller where sprays were applied after inoculation than where they were applied before inoculation but, as with the inoculated ears, the differences were relatively small (9 and 17%, respectively).

Table 4.6. Mean numbers of ergots per inoculated ear of winter rye sprayed with different fungicides either before or after inoculation in 1998.

Fungicide	Sprayed		Mean
	Before inoculation	After inoculation	
Untreated	11.8		11.8
Carbendazim	11.3	12.9	12.1
Epoxiconazole	13.0	11.8	12.4
Tebuconazole	12.4	11.8	12.1
Cyproconazole	13.2	10.9	12.1
Flutriafol	12.5	11.1	11.8
Azoxystrobin	12.4	12.8	12.6
Kresoxim-methyl + fenpropimorph	12.3	13.0	12.6
Cyprodinil	12.9	12.2	12.6
Chlorothalonil	12.2	11.9	12.0
Mean (excl. untreated)	12.5	12.1	12.3
SED (58 df) Fungicides			0.76
Spray timing	0.59		
Interaction	1.07		

Table 4.7. Mean weights (mg) of ergots per inoculated ear of winter rye sprayed with different fungicides either before or after inoculation in 1998.

Fungicide	Sprayed		Mean
	Before inoculation	After inoculation	
Untreated	201.1		201.1
Carbendazim	203.8	234.0	218.9
Epoxiconazole	248.5	211.4	230.0
Tebuconazole	245.3	211.4	228.3
Cyproconazole	243.7	217.2	230.4
Flutriafol	219.7	183.9	201.8
Azoxystrobin	270.5	245.9	258.2
Kresoxim-methyl + fenpropimorph	233.5	228.5	231.0
Cyprodinil	223.4	233.2	228.3
Chlorothalonil	216.6	205.5	211.1
Mean (excl. untreated)	233.9	219.0	226.4
SED (58 df) Fungicide			15.53
Spray timing	12.14 *		
Interaction	21.97		

Table 4.8. Total numbers of secondarily infected ears per plot in winter rye sprayed with different fungicides either before or after inoculation in 1998.

Fungicide	Sprayed		
	Before inoculation	After inoculation	Mean
Untreated	229.9		229.9
Carbendazim	239.7	273.8	256.8
Epoxiconazole	168.5	145.3	156.9
Tebuconazole	278.8	243.0	260.9
Cyproconazole	256.0	215.0	235.5
Flutriafol	191.3	204.2	197.7
Azoxystrobin	289.3	278.8	284.0
Kresoxim-methyl + fenpropimorph	296.5	233.0	264.8
Cyprodinil	254.5	211.7	233.1
Chlorothalonil	204.3	237.0	220.6
Mean (excl. untreated)	242.1	226.9	234.5
SED (58 df) Fungicide			24.28 ***
Spray timing	18.98		
Interaction	34.34		

Table 4.9. Mean weights (mg) of ergots per secondarily infected ear of winter rye sprayed with different fungicides either before or after inoculation in 1998.

Fungicide	Sprayed		
	Before inoculation	After inoculation	Mean
Untreated	85.3		85.3
Carbendazim	79.4	67.4	73.4
Epoxiconazole	88.1	82.1	85.1
Tebuconazole	86.1	87.5	86.8
Cyproconazole	104.3	64.0	84.1
Flutriafol	85.0	74.5	79.7
Azoxystrobin	80.6	92.8	86.7
Kresoxim-methyl + fenpropimorph	91.1	86.1	88.6
Cyprodinil	88.8	90.2	89.5
Chlorothalonil	87.0	78.5	82.8
Mean (excl. untreated)	87.8	80.3	84.1
SED (58 df) Fungicide			7.29
Spray timing	5.70 *		
Interaction	10.31		

Table 4.10. Total weights (g per plot) of ergots in secondarily infected ears of winter rye sprayed with different fungicides either before or after inoculation in 1998.

Fungicide	Sprayed		Mean
	Before inoculation	After inoculation	
Untreated	20.6		20.6
Carbendazim	19.1	16.6	17.9
Epoxiconazole	15.3	13.0	14.2
Tebuconazole	26.1	20.3	23.2
Cyproconazole	27.4	12.8	20.1
Flutriafol	16.2	16.2	16.2
Azoxystrobin	20.2	26.2	23.2
Kresoxim-methyl + fenpropimorph	27.9	20.3	24.1
Cyprodinil	24.3	18.9	21.6
Chlorothalonil	19.2	19.2	19.2
Mean (excl. untreated)	21.7	18.2	20.0
SED (58 df) Fungicide			3.47
Spray timing	2.71 *		
Interaction	4.91		

Experiment 4.2 Winter rye 1999

Inoculated ears

There were no significant effects of the fungicides on average numbers of ergots that formed in the inoculated ears (Table 4.11) but there were significant differences in the mean weights of ergots per ear (Table 4.12) that reflected differences in the mean weight per ergot (data not shown). However, the largest effect was of trifloxystrobin, which significantly increased mean weight of ergots per ear, and none of the fungicides gave significant decreases compared to the untreated. There were no significant effects of the wetter on either the numbers or weights of ergots.

Secondarily infected ears

All of the fungicides except carbendazim and chlorothalonil significantly decreased the number of secondarily infected ears per plot, compared to the untreated, but the most effective were fluquinconazole and bromuconazole (51 and 46% less than the untreated, respectively; Table 4.13). There were no significant effects of the fungicides on either the mean numbers of ergots per infected ear (Table 4.14) or the mean weights of ergots per infected ear (Table 4.16). There were significant effects of the fungicides on the numbers of ergots per plot (Table 4.15) and the total weights of ergots per plot (Table 4.17) but these reflected the differences in numbers of infected ears. Adding a wetter to the fungicide sprays had no significant effects on numbers of infected ears or on the numbers or weights of ergots in those ears.

Table 4.11. Mean numbers of ergots per inoculated ear of winter rye sprayed without or with the addition of a wetter in 1999.

Fungicide	Wetter		
	Without	With	Mean
Untreated	4.65		4.65
Carbendazim	4.68	4.40	4.54
Bromuconazole	4.17	4.72	4.44
Difenconazole	4.41	4.64	4.52
Flusilazole	4.02	4.18	4.10
Flutriafol	4.15	4.10	4.13
Fluquinconazole	4.15	4.10	4.13
Prochloraz	5.42	4.56	4.99
Chlorothalonil	4.53	4.01	4.27
Trifloxystrobin	5.05	4.67	4.86
Mean (excl. untreated)	4.51	4.38	4.44
SED (58 df) Fungicide			0.341
Added wetter	0.266		
Interaction	0.482		

Table 4.12. Mean weights (mg) of ergots per inoculated ear of winter rye sprayed without or with the addition of a wetter in 1999.

	Wetter		
	Without	With	Mean
Untreated	119.2		119.2
Carbendazim	110.4	111.5	111.0
Bromuconazole	119.1	118.8	119.0
Difenconazole	114.3	121.6	117.9
Flusilazole	123.4	115.7	119.5
Flutriafol	106.1	113.5	109.8
Fluquinconazole	139.4	125.3	132.4
Prochloraz	121.7	131.0	126.4
Chlorothalonil	115.3	103.0	109.2
Trifloxystrobin	160.2	145.3	152.8
Mean (excl. untreated)	123.3	120.6	122.0
SED (58 df) Fungicide			9.62 ***
Added wetter	7.52		
Interaction	13.60		

Table 4.13. Total number of secondarily infected ears per plot in winter rye sprayed without or with the addition of a wetter in 1999.

	Wetter		
	Without	With	Mean
Untreated	103.2		103.2
Carbendazim	102.0	98.0	100.0
Bromuconazole	60.7	51.7	56.2
Difenconazole	64.0	66.5	65.2
Flusilazole	77.0	65.2	71.1
Flutriafol	67.2	73.0	70.1
Fluquinconazole	46.7	54.7	50.7
Prochloraz	79.0	71.0	75.0
Chlorothalonil	88.0	89.7	88.9
Trifloxystrobin	67.0	65.0	66.0
Mean (excl. untreated)	72.4	70.6	71.5
SED (58 df) Fungicide			11.25 **
Added wetter	8.80		
Interaction	15.92		

Table 4.14. Mean numbers of ergots per secondarily infected ear of winter rye sprayed without or with the addition of a wetter in 1999.

	Wetter		
	Without	With	Mean
Untreated	2.17		2.17
Carbendazim	2.47	2.24	2.35
Bromuconazole	1.85	2.12	1.99
Difenconazole	2.04	1.90	1.97
Flusilazole	1.97	2.01	1.99
Flutriafol	2.10	2.26	2.18
Fluquinconazole	1.87	2.17	2.02
Prochloraz	2.29	1.82	2.05
Chlorothalonil	2.05	2.06	2.06
Trifloxystrobin	2.24	2.22	2.23
Mean (excl. untreated)	2.10	2.09	2.10
SED (58 df) Fungicide			0.175
Added wetter	0.137		
Interaction	0.248		

Table 4.15. Total numbers of ergots per plot in secondarily infected ears of winter rye sprayed without or with the addition of a wetter in 1999.

	Wetter		
	Without	With	Mean
Untreated	220.0		220.0
Carbendazim	253.5	220.5	237.0
Bromuconazole	111.3	112.0	111.6
Difenconazole	133.0	124.5	128.8
Flusilazole	148.8	121.8	135.3
Flutriafol	134.3	159.3	146.8
Fluquinconazole	93.0	120.3	106.6
Prochloraz	176.8	130.8	153.8
Chlorothalonil	190.3	198.0	194.1
Trifloxystrobin	154.8	135.5	145.1
Mean (excl. untreated)	155.1	146.9	151.0
SED (58 df) Fungicide			27.43 ***
Added wetter	21.44		
Interaction	38.79		

Table 4.16. Mean weights (mg) of ergots per secondarily infected ear of winter rye sprayed without or with the addition of a wetter in 1999.

	Wetter		
	Without	With	Mean
Untreated	57.2		57.2
Carbendazim	64.3	58.6	61.4
Bromuconazole	53.4	58.4	55.9
Difenconazole	50.8	54.2	52.5
Flusilazole	53.6	64.7	59.2
Flutriafol	53.1	59.8	56.5
Fluquinconazole	49.8	65.4	57.6
Prochloraz	60.2	49.1	54.6
Chlorothalonil	55.6	58.1	56.8
Trifloxystrobin	62.9	56.4	59.6
Mean (excl. untreated)	56.0	58.3	57.1
SED (58 df) Fungicide			5.45
Added wetter	4.26		
Interaction	7.71		

Table 4.17. Total weights (g per plot) of ergots in secondarily infected ears of winter rye sprayed without or with the addition of a wetter in 1999.

	Wetter		
	Without	With	Mean
Untreated	5.93		5.93
Carbendazim	6.83	5.88	6.35
Bromuconazole	3.20	3.15	3.18
Difenconazole	4.23	3.58	3.90
Flusilazole	4.13	3.78	3.95
Flutriafol	3.68	4.40	4.04
Fluquinconazole	2.53	3.53	3.03
Prochloraz	4.88	3.70	4.29
Chlorothalonil	5.20	5.53	5.36
Trifloxystrobin	4.45	3.53	3.99
Mean (excl. untreated)	4.34	4.12	4.23
SED (58 df) Fungicide			0.756 ***
Added wetter	0.591		
Interaction	1.069		

Experiment 4.3 Winter wheat 1999

Inoculated ears

The fungicides tested in this experiment had no significant effects on mean numbers of ergots in the inoculated ears (Table 4.18) but they did have significant effects on the mean weights of ergots per ear (Table 4.19). However, most of the fungicides increased the weight. Azoxystrobin had the largest effect (48% more than the untreated) but the chemically related kresoxim-methyl had no effect. Applying the fungicides at double rate or using an electrostatic sprayer also had no significant effects on mean numbers of ergots per ear but both significantly affected the mean weights of ergots per ear and there was also evidence of a significant interaction between them. However, this suggested, inexplicably, that the mean weight of ergots per ear was increased by applying fungicides at the double rate using the hydraulic sprayer compared to applying fungicides at the normal rate with the hydraulic sprayer or at either rate with the electrostatic sprayer.

Secondarily infected ears

Several of the fungicides increased the very small numbers of ergots per ear, including azoxystrobin, which more than doubled the mean number, but not significantly (Table 4.20). All fungicides except cyproconazole increased the mean weights of ergots per ear (Table 4.21), and the effect of azoxystrobin was large (+203%) and significant. As for the inoculated ears, there were no significant effects on mean numbers of ergots per ear of applying the fungicides at the double rate or of using an electrostatic sprayer. Rate of application did

have a significant effect on weights of ergots per ear but the mean weight was larger at the double rate than at the normal rate (2.55 vs. 1.52 mg per ear).

Table 4.18. Mean numbers of ergots per inoculated ear of winter wheat sprayed with different fungicides applied at normal or double rates using hydraulic or electrostatic sprayers in 1999.

Fungicide	Rate and method of application				Mean
	Hydraulic Normal rate	Hydraulic Double rate	Electrostatic Normal rate	Electrostatic Double rate	
Untreated		5.51			5.51
Cyproconazole	4.37	4.69	5.44	5.81	5.08
Epoxiconazole	5.35	5.80	4.83	4.95	5.23
Tebuconazole	5.09	5.38	5.43	4.65	5.14
Fenpropimorph	5.58	5.20	5.35	4.97	5.27
Azoxystrobin	5.87	6.21	5.18	5.31	5.64
Kresoxim-methyl	4.26	5.04	4.51	4.67	4.62
Cyprodinil	4.94	5.10	4.89	4.97	4.98
Exp 10623A	5.70	5.00	5.51	4.70	5.23
Silthiofam	5.25	6.35	5.05	4.88	5.38
Mean (excl. untreated)	5.16	5.42	5.13	4.99	5.17
SED (119 df) Fungicide					0.346
Rates and methods		0.294			
Interaction		0.693			

Table 4.19. Mean weights (mg) of ergots per inoculated ear of winter wheat sprayed with different fungicides applied at normal or double rates using hydraulic or electrostatic sprayers in 1999.

Fungicide	Rate and method of application				Mean
	Hydraulic Normal rate	Hydraulic Double rate	Electrostatic Normal rate	Electrostatic Double rate	
Untreated		64.0			64.0
Cyproconazole	70.3	69.8	74.2	76.7	72.8
Epoxiconazole	70.5	95.8	70.0	78.3	78.7
Tebuconazole	75.3	89.2	77.9	65.4	77.0
Fenpropimorph	75.6	91.4	75.5	71.1	78.4
Azoxystrobin	78.7	128.9	86.6	83.9	94.5
Kresoxim-methyl	57.0	80.0	54.2	60.2	62.9
Cyprodinil	58.1	78.4	67.1	66.8	67.6
Exp 10623A	74.3	75.9	68.8	65.8	71.2
Silthiofam	57.8	77.1	68.1	54.5	64.4
Mean (excl. untreated)	68.6	87.4	71.4	69.2	74.2
SED (119 df) Fungicide					7.41 **
Rates and methods		6.29 **			
Interaction		14.81			

Table 4.20. Mean numbers of ergots per secondarily infected ear of winter wheat sprayed with different fungicides applied at normal or double rates using hydraulic or electrostatic sprayers in 1999.

Fungicide	Rate and method of application				Mean
	Hydraulic Normal rate	Hydraulic Double rate	Electrostatic Normal rate	Electrostatic Double rate	
Untreated		0.06			0.06
Cyproconazole	0.09	0.02	0.01	0.05	0.04
Epoxiconazole	0.10	0.05	0.01	0.18	0.08
Tebuconazole	0.08	0.01	0.11	0.04	0.06
Fenpropimorph	0.02	0.08	0.12	0.26	0.12
Azoxystrobin	0.05	0.17	0.06	0.22	0.12
Kresoxim-methyl	0.08	0.05	0.01	0.05	0.05
Cyprodinil	0.03	0.12	0.23	0.07	0.11
Exp 10623A	0.04	0.07	0.09	0.09	0.07
Silthiofam	0.10	0.08	0.02	0.05	0.06
Mean (excl. untreated)	0.06	0.07	0.07	0.11	0.08
SED (119 df) Fungicide					0.039
Rates and methods		0.033			
Interaction		0.077			

Table 4.21. Mean weights (mg) of ergots per secondarily infected ear of winter wheat sprayed with different fungicides applied at normal or double rates using hydraulic or electrostatic sprayers in 1999.

Fungicide	Rate and method of application				Mean
	Hydraulic Normal rate	Hydraulic Double rate	Electrostatic Normal rate	Electrostatic Double rate	
Untreated		1.28			1.28
Cyproconazole	2.55	0.25	0.45	0.55	0.95
Epoxiconazole	1.40	1.20	0.25	6.30	2.29
Tebuconazole	1.70	0.25	2.60	0.95	1.37
Fenpropimorph	0.55	1.80	1.85	5.20	2.35
Azoxystrobin	1.85	4.75	2.30	6.60	3.88
Kresoxim-methyl	1.50	1.85	0.40	2.05	1.45
Cyprodinil	0.45	4.15	4.40	2.80	2.95
Exp 10623A	0.75	1.60	1.25	2.25	1.46
Silthiofam	2.05	2.35	1.05	0.95	1.60
Mean (excl. untreated)	1.42	2.02	1.62	3.07	2.03
SED (119 df) Fungicide					0.963
Rates and methods		0.819			
Interaction		1.927			

Experiment 4.4 Winter wheat 2000

Inoculated ears

Average effects of the four fungicides tested in this experiment on mean numbers and weights of ergots in inoculated ears were mostly small and not significant (Table 4.22). The only exception was in the results from the extra plots testing the effects of applying the fungicides in four times the normal volume of water where those treated with tebuconazole or fluquinconazole had greater average weights of ergots per ear (17.02 and 17.05 mg per ear, respectively) than the untreated (12.13 mg per ear) or those treated with triadimenol or cyproconazole (11.77 and 14.71 mg per ear, respectively).

Average effects of using different rates, volumes and methods to apply the fungicides, and of simulated rain were small and not significant. There was some evidence in mean numbers of ergots per inoculated ear of an interaction between simulated rain and fungicides but the differences were very small and not biologically meaningful (Table 4.23). There was also a significant interaction between simulated rain and the different combinations of rates, volumes and methods of applying the fungicides ($P<0.05$) but this was, similarly, difficult to interpret and probably spurious.

Secondarily infected ears

There was relatively little secondary spread in this experiment and the average number of ergots in ears that were not inoculated (number sampled per plot = 50) was only 0.26. Average effects of the four fungicides were mostly small and not significant (Table 4.22). As with the inoculated ears, there was evidence for significant differences between the fungicides in the extra plots testing the effects of applying them in four times the normal volume of water but in contrast to the results for the inoculated ears, it was plots treated with triadimenol or cyproconazole that had most ergots (0.41 and 0.35 ergots per ear, respectively, compared to 0.15 in untreated plots and 0.18 and 0.20, respectively, in plots treated with tebuconazole or fluquinconazole).

There was evidence in mean weights of ergots per ear of a significant interaction between fungicides and the treatments testing different combinations of rates, volumes and methods of applying them (Table 4.24) but, again, the differences were small and difficult to explain.

Table 4.22. Mean numbers and weights of ergots in inoculated and secondarily infected ears of winter wheat sprayed with different fungicides in 2000.

Fungicide	Inoculated ears		Secondarily infected ears	
	Mean number of ergots per ear	Mean weight of ergots per ear (mg)	Mean number of ergots per ear	Mean weight of ergots per ear (mg)
Untreated	2.43	12.1	0.16	0.475
Cyproconazole	2.48	13.3	0.29	0.555
Fluquinconazole	2.48	12.4	0.26	0.322
Tebuconazole	2.51	13.0	0.22	0.486
Triadimenol	2.41	12.1	0.28	0.362
SED (120 df)	0.108	1.02	0.044	0.1778

Table 4.23. Mean numbers and weights of ergots in inoculated and secondarily infected ears of winter wheat sprayed with different fungicides followed by simulated rain or none in 2000.

Simulated rain	Fungicide	Mean number of ergots per inoculated ear	Mean weight of ergots per inoculated ear (mg)	Mean number of ergots per secondarily infected ear	Mean weight of ergots per secondarily infected ear (mg)
Without	Untreated	2.43	12.1	0.16	0.475
	Cyproconazole	2.55	14.4	0.29	0.510
	Fluquinconazole	2.56	12.9	0.28	0.588
	Tebuconazole	2.35	12.5	0.23	0.671
	Triadimenol	2.36	12.5	0.26	0.400
With	Cyproconazole	2.41	12.2	0.28	0.600
	Fluquinconazole	2.41	11.9	0.24	0.456
	Tebuconazole	2.67	13.6	0.22	0.300
	Triadimenol	2.45	11.7	0.31	0.325
SED (120 df)		0.125 *	1.18	0.051	0.2053

Table 4.24. Mean numbers and weights of ergots in inoculated and secondarily infected ears of winter wheat sprayed with different fungicides using different combinations of rates, volumes and methods in 2000.

Rates/volumes/methods [1]	Fungicide	Mean number of ergots per inoculated ear	Mean weight of ergots per inoculated ear (mg)	Mean number of ergots per secondarily infected ear	Mean weight of ergots per secondarily infected ear (mg)
-	Untreated	2.43	12.1	0.16	0.475
NRNV	Cyproconazole	2.56	13.1	0.31	1.050
	Fluquinconazole	2.47	11.7	0.21	0.400
	Tebuconazole	2.64	15.2	0.25	0.275
	Triadimenol	2.38	10.4	0.23	0.225
	Mean	2.51	12.6	0.25	0.488
NRDV	Cyproconazole	2.56	13.9	0.29	0.375
	Fluquinconazole	2.59	12.8	0.25	0.475
	Tebuconazole	2.50	11.4	0.19	0.400
	Triadimenol	2.30	11.6	0.26	0.350
	Mean	2.49	12.4	0.25	0.400
DRNV	Cyproconazole	2.41	14.1	0.24	0.300
	Fluquinconazole	2.54	13.3	0.29	0.412
	Tebuconazole	2.45	12.8	0.24	1.067
	Triadimenol	2.51	11.8	0.29	0.400
	Mean	2.48	13.0	0.26	0.545
DRE	Cyproconazole	2.39	12.0	0.33	0.496
	Fluquinconazole	2.35	11.7	0.30	0.800
	Tebuconazole	2.46	12.8	0.22	0.200
	Triadimenol	2.44	14.7	0.36	0.475
	Mean	2.40	12.8	0.30	0.493
SED (120 df) Fung. x Rates/volumes/methods		0.153	1.44	0.062	0.2515 *
Means for Rates/volumes/methods		0.108	1.02	0.044	0.1778

[1] NRNV = normal rate and normal volume applied using the hydraulic sprayer

NRDV = normal rate and double volume applied using the hydraulic sprayer

DRNV = double rate and normal volume applied using the hydraulic sprayer

DRE = double rate applied using the electrostatic sprayer

Experiment 4.5 Spring wheat 2000

Inoculated ears

The mean number (Table 4.25) and the mean weight (Table 4.26) of ergots per inoculated ear were decreased most by fluquinconazole but not significantly. The mean weight but not the mean number per ear tended to be increased by azoxystrobin but, again, not significantly. Applying the sprays during stem extension instead of at ear emergence had no significant effect.

Secondarily infected ears

There was little secondary spread and the numbers or ergots per ear (Table 4.27) were very small and not significantly affected by the fungicide treatments. Mean weights of ergots per ear (Table 4.28) were similarly very small but tended to be decreased by all fungicides except fluquinconazole but, again, these differences were not significant. As with inoculated ears, there were no significant effects of applying sprays at the two different growth stages.

Table 4.25. Mean numbers of ergots per inoculated ear of spring wheat sprayed with different fungicides at GS 31 or GS 59 in 2000.

Fungicide	Sprayed		
	GS 31	GS 59	Mean
Untreated	2.59		2.59
Carbendazim	2.48	2.87	2.67
Cyproconazole	2.76	2.63	2.70
Fluquinconazole	2.24	2.27	2.25
Flusilazole	2.59	2.46	2.53
Tebuconazole	3.00	2.72	2.86
Triadimenol	2.74	2.52	2.63
Fenpropimorph	2.14	2.94	2.54
Azoxystrobin	2.86	2.25	2.55
Silthiofam	2.66	2.49	2.58
Mean	2.61	2.57	2.59
SED (58 df) Fungicide			0.235
Spray timing	0.183		
Interaction	0.332		

Table 4.26. Mean weights (mg) of ergots per inoculated ear of spring wheat sprayed with different fungicides at GS 31 or GS 59 in 2000.

Fungicide	Sprayed		
	GS 31	GS 59	Mean
Untreated	76.7		76.7
Carbendazim	60.4	85.7	73.1
Cyproconazole	71.1	81.6	76.3
Fluquinconazole	63.0	53.7	58.3
Flusilazole	62.8	76.5	69.6
Tebuconazole	87.7	75.0	81.4
Triadimenol	71.3	73.1	72.2
Fenpropimorph	71.6	82.7	77.2
Azoxystrobin	95.3	79.4	87.4
Silthiofam	67.0	78.3	72.6
Mean	72.2	76.2	74.2
SED (58 df) Fungicide			9.37
Spray timing	7.32		
Interaction	13.25		

Table 4.27. Mean numbers of ergots per secondarily infected ear of spring wheat sprayed with different fungicides at GS 31 or GS 59 in 2000.

Fungicide	Sprayed		
	GS 31	GS 59	Mean
Untreated	0.57		0.57
Carbendazim	0.34	0.48	0.41
Cyproconazole	0.40	0.67	0.53
Fluquinconazole	0.70	0.46	0.58
Flusilazole	0.34	0.36	0.35
Tebuconazole	0.47	0.36	0.41
Triadimenol	0.65	0.60	0.62
Fenpropimorph	0.56	0.39	0.47
Azoxystrobin	0.37	0.49	0.43
Silthiofam	0.45	0.44	0.44
Mean	0.47	0.47	0.47
SED (58 df) Fungicide			0.103
Spray timing	0.080		
Interaction	0.145		

Table 4.28. Mean weights (mg) of ergots per secondarily infected ear of spring wheat sprayed with different fungicides at GS 31 or GS 59 in 2000.

Fungicide	Sprayed		
	GS 31	GS 59	Mean
Untreated	2.58		2.58
Carbendazim	0.65	1.00	0.83
Cyproconazole	0.80	1.95	1.38
Fluquinconazole	2.95	1.90	2.43
Flusilazole	0.90	0.75	0.83
Tebuconazole	1.50	1.45	1.48
Triadimenol	0.90	1.10	1.00
Fenpropimorph	2.70	1.30	2.00
Azoxystrobin	1.45	1.25	1.35
Silthiofam	1.80	1.35	1.58
Mean	1.52	1.34	1.43
SED (58 df) Fungicide			1.030
Spray timing	0.805		
Interaction	1.457		

DISCUSSION

The five field experiments described in this chapter provided no evidence that any of the fungicides tested can be relied upon to decrease ergot to a large enough extent to be commercially useful. Several fungicides apparently decreased the numbers of ears infected by secondary spread in the winter rye experiment in 1999 but all but one of those tested in the winter rye experiment in 1998 increased the number. Although only three fungicides were tested in both experiments (carbendazim, flutriafol and chlorothalonil), several of the other compounds that were tested in the two experiments were closely related. The decreases in numbers of ears infected by secondary spread in 1999 but not in 1998 may have been a consequence of applying the fungicides at double the normal rate in 1999. However, it is difficult to explain why the fungicides that were tested in 1999 had no effect on mean numbers (or weights) of ergots in the infected ears despite, apparently, decreasing the numbers of infected ears.

Generally, the effects of fungicides on average numbers and weights of ergot in inoculated ears and those infected by secondary spread were small and not significant, and increases tended to be more common than decreases. For most fungicides that were tested in two or more experiments, there was little evidence of consistency in their effects. However, the strobilurin and related fungicides did tend to increase ergot to a consistently greater extent than other fungicides or chemical groups. Azoxystrobin increased the number and/or weight of ergots in all three of the experiments described in this chapter in which it was tested (but not always significantly). In the one experiment in which it was tested, trifloxystrobin similarly increased the weight of ergots in inoculated ears. Kresoxim-methyl had no effect in the winter wheat experiment in 1999

but tended to increase numbers and/or weights of ergots in the winter rye experiment in 1998 (and also in the field screening experiment described in Chapter 2). Results from glasshouse Experiment 3.1 confirm that both azoxystrobin and kresoxim-methyl are fungicidally active against *C. purpurea* and so the observed increases in ergot following the application of these and other fungicides, which confirm previously published results (Werner *et al*., 1999), can not be attributed to direct stimulatory effects of the fungicides and must have some other, indirect, cause.

All five experiments also tested whether altering when or how the fungicides were applied might influence their efficacy but provided little or no evidence for biologically significant effects. In the winter rye experiment in 1999 (Experiment 4.2), for example, there were only small benefits of applying sprays 2-3 days after inoculation instead of 2-3 days before inoculation, and in the spring wheat experiment in 2000 (Experiment 4.5) there was, in contrast to the results of glasshouse Experiment 3.4, no significant difference between sprays applied at early stem extension or ear emergence. Results of the glasshouse experiments also suggested that control of ergot might be improved by increasing the amounts of fungicide or volumes of spray deposited on the ears. However, doubling the concentration of active ingredients in sprays applied using a hydraulic sprayer or applying fungicides using an electrostatic sprayer (which can increase deposition more then 20 fold; Cayley *et al.*, 1984) had negligible effects. Increasing the volumes of water in which the sprays were applied, adding an additional wetter to the spray solutions or applying simulated rain were tested in the hope that they might improve control by increasing the chances that some of the fungicides would move between the glumes and enter the floral cavity. However, few of the effects were significant and those that were significant were mostly difficult to explain and probably spurious.

CHAPTER 5

EFFECTS OF APPLYING DIFFERENT FUNGICIDES AT DIFFERENT RATES AND TIMES IN FIELD EXPERIMENTS IN SUFFOLK AND DORSET IN 1998-2000

INTRODUCTION

The activity of commercially-available cereal fungicides against ergot was investigated on winter wheat and winter rye in each year, at single naturally-infected sites in Suffolk and Dorset, respectively. Small numbers of ears were artificially inoculated with the ergot fungus to test fungicidal activity and to provide secondary inoculum for spread within plots. Inoculation was carried out at early ear emergence so that honeydew would be produced during the main flowering period of the crop. Fungicides were first applied immediately after inoculation to provide a field screen of curative activity. This contrasted with field experiments at Rothamsted where pre- and post-inoculation applications were compared. Fungicide treatments tested the main active ingredients in current use, which represented the main types of fungicide chemistry and fungicide properties. Results from the laboratory and glasshouse experiments at Rothamsted were also used to guide treatment selection.

OBJECTIVES

To determine the optimum dose rate and timing of fungicides and novel disease control compounds for control of ergot in cereals under field conditions.

Detailed objectives

1998 To determine efficacy of fungicides applied at ear emergence + flowering with treatments at flowering only.

1999 To identify activity against ergot of fungicides applied at early ear emergence and flowering at double label rates.

2000 To determine efficacy of fungicides applied at the first to second node stage and ear emergence or at ear emergence alone for control of ergot in winter wheat

MATERIALS AND METHODS

Experiment 5.1 Winter wheat 1998

Plots of winter wheat cv. Rialto were established on a site with a history of ergot problems. A 'depot' of 25 rye ergots (1996 harvest) and 25 wheat ergots (1997 harvest) were buried about 1 cm deep in plastic mesh grids, with one ergot per grid square, on 6 January 1998. This was used to monitor germination of ergots during the summer.

The development of ergot was encouraged by use of a chemical partial sterilant (Genesis pollen suppressant) to increase gaping of the glumes and this was applied on 15 May (GS 31) using a tractor mounted sprayer. Ten ears in an untreated area were enclosed in small polythene crossing bags prior to application of the pollen suppressant in order to determine its effect on ear fertility.

A range of fungicides was selected to represent the main types of commercial active ingredients and with known activity against ergot. Sprays were applied at early ear emergence and flowering or at flowering only (Table 5.1). Formulation details of fungicides are in Table 5.2.

Table 5.1. Fungicides, treatment timings and dose rates, 1998

	Ear emergence (GS 51-55) + pre-flowering (GS 59)		Pre-flowering only (GS59)		
Treatment No.	Product	Dose of product l ha^{-1} (each time)	Treatment No.	Product	Dose of product l ha^{-1} (each time)
1	Untreated control	-	11	Untreated control	-
2	Bavistin DF	1	12	Bavistin DF	1
3	Opus	1	13	Opus	1
4	Bravo	2	14	Bravo	2
5	Folicur	1	15	Folicur	1
6	Alto 100	0.8	16	Alto 100	0.8
7	Pointer	1	17	Pointer	1
8	Amistar	1	18	Amistar	1
9	Ensign + Corbel	0.7 + 0.72	19	Ensign + Corbel	0.7 + 0.72
10	Unix	1	20	Unix	1

Table 5.2. Formulation and rates of active ingredients of fungicide treatments, 1998.

Fungicide active ingredient	Product	Formulation	Rate of active ingredient kg ha^{-1}
Carbendazim	Bavistin DF	50 % w/w WG	0.50
Epoxiconazole	Opus	125 g/l SC	0.125
Chlorothalonil	Bravo 500	500 g/l SC	1.00
Tebuconazole	Folicur	250 g/l EW	0.250
Cyproconazole	Alto 100	100 g/l SL	0.080
Flutriafol	Pointer	125 g/l SC	0.125
Azoxystrobin	Amistar	250 g/l SC	0.250
Kresoxim methyl + fenpropimorph	Ensign	150 + 300 g/l SE	0.105 + 0.210
Fenpropimorph	Corbel	750 g/l EC	0.540
Cyprodinil	Unix	75% w/w WDG	0.75

Fungicides were applied on 28 May (GS 52) and 5 June (GS 59) using an OPS knapsack sprayer.

Inoculation of ears at the late boot-ear emergence stage (GS 47-51) was done on 28 May 1998 using diluted honeydew from a wheat isolate (10^6 spores ml^{-1}) using the inoculator described in Chapter 2. Groups of about 20 tillers were inoculated at two points, two metres in from each end, in each plot (size 6.0 x 1.70 m) and a cable tie used to mark the location of each inoculation point.

Inoculated plants had sticky ears, presumed to be honeydew, by 8 June and this was dispersed using a 2-metre long plastic rod to agitate the ears. The rod was wiped clean at the end of each plot. It was raining heavily during this first agitation, at which time there was 20% germination in the ergot depot. Agitation was repeated on 10 and 11 June, when the crop was at GS 69. The weather was again wet, with prolonged rain and a temperature of 19^0 C. Full site details for all experiments described in this Chapter are given in Appendix D.

Assessment methods:

At GS 75 (7 July) detailed assessments were done on 10 inoculated ears¸ 5 from each inoculated area of each plot. The ears were cut from the tillers and numbers of ergots per ear counted. In addition, 50 ears at 0.3 m from each inoculation area (25 near each of the two inoculation points), and 50 ears at 1.2 m from each inoculated area (25 from each) per plot were examined *in situ*. The number of ears with ergot and the number of ergots in each affected ear were recorded. Ear blight (*Fusarium culmorum*) was recorded where present as number of ears affected per plot.

A second assessment was done at GS 85 (1 August) both on a whole-plot basis to estimate the percentage ears affected in the whole plot and on 50 uninoculated ears in one area from the edge of each plot up to one inoculated area (0.2-1.0 m from inoculation point). At this stage ergots and honeydew were present on late green tillers and these were identified as green tillers in the assessment.

Experiment 5.2 Winter rye 1998

Plots were marked out in a commercial crop of winter rye cv. Esprit at Poyntington, Dorset. Fungicide treatments were as described for winter wheat (Tables 5.1 and 5.2). Ergots were buried on site on 27 November and 12% germination of wheat ergots was noted only on 1 May (GS 39-41) but these did not persist until the next visit on 8 May.

Inoculation of ears at the late boot-ear emergence stage (GS 47-51) was carried out on 8 May 1998 using diluted honeydew from a wheat isolate (10^6 spores ml^{-1}) using the inoculator described in Chapter 2. Groups of about 20 tillers were inoculated at three points in the centre of each plot (size 11.5 x 2.25 m) and a cable tie used to mark the location of each inoculation point. The first fungicide sprays were applied on 8 May (GS 51-57) and the second fungicide sprays on 15 May (GS 59).

Inoculated ears were producing honeydew by 20 May and this was dispersed using a wooden pole to agitate the ears. About 20% ears were at anthesis and the weather was warm, dry and sunny during this 'secondary' inoculation.

Assessments were carried out on 25 inoculated ears and 300 non-inoculated ears per plot at GS 71 (12 June) when the number of infected ears and the number of ergots per ear were recorded.

Experiment 5.3 Winter wheat 1999

Plots of winter wheat cv. Rialto were established on a site with a history of ergot problems at Woolpit, Suffolk on the area used for the experiment in 1998. Because of the ergots left after harvest in 1998, the crop was allowed to flower naturally without resort to pollen suppressant chemical. A range of fungicides was selected to represent the major types of commercial active ingredients and compared with applications of copper or copper + boron which are known to have activity against ergot under deficiency conditions. Sprays were applied at early ear emergence and flowering, at double rate, to identify activity against ergot (Tables 5.3 and 5.4).

Table 5.3. Treatments and product rates per ha, 1999.

Treatment No.	Fungicides applied at ear emergence (GS 51) + pre-flowering (GS 59)	Dose of product l ha^{-1} (each time)
1	Untreated control	-
2	Bavistin DF	2
3	Opus	2
4	Bravo	4
5	Folicur	2
6	Alto 100	1.6
7	Amistar	2
8	Ensign + Corbel	1.4 + 1.44
9	Corbel	2
10	Fluquinconazole	1.5
11	Sportak 45	1.8
12	Sanction	1
13	Twist	2
14[1]	Key Feeds Copper	2
15[1]	Key Feeds Copper + Nitrate Balancer(Boron)	2 + 2

[1] First spray at GS 32
Second spray at GS 51

Table 5.4. Formulation and dose of treatments,1999.

Treatment number	Fungicide active ingredient	Product	Formulation	Rate of active ingredient kg ha^{-1}
1	Untreated			
2	Carbendazim	Bavistin DF	50 % w/w WG	1.00
3	Epoxiconazole	Opus	125 g/l SC	0.25
4	Chlorothalonil	Bravo 500	500 g/l SC	2.00
5	Tebuconazole	Folicur	250 g/l EW	0.50
6	Cyproconazole	Alto 100	100 g/l SL	0.16
7	Azoxystrobin	Amistar	250 g/l SC	0.50
8	Kresoxim methyl / fenpropimorph + fenpropimorph	Ensign + Corbel	150 / 300 g/l SE + 750 g/l EC	0.21 / 0.42 + 1.08
9	Fenpropimorph	Corbel	750 g/l EC	1.50
10	Fluquinconazole	Flamenco	100 g/l EC	0.15
11	Prochloraz	Sportak 45	450 g/l EC	0.81
12	Flusilazole	Sanction	400 g/l EC	0.40
13	Trifloxystrobin	Twist	125 g/l EC	0.25
14	Copper	Key Feeds Copper	6.2% w/v copper	0.124
15	Copper + boron	Keyfeeds Copper + Nitrate Balancer	6.2% w/v copper + 11.8% w/v boron 0.13% w/v molybdenum	0.124 + 0.236 B and 2.6 g Mo

Fungicides were applied on 25 May (GS 51) and 1 June (GS 59) using an OPS knapsack sprayer using Orange ff120 nozzles operated at 2 bars to deliver 250 l ha^{-1}.

Inoculation of ears at the late boot-ear emergence stage (GS 47-51) was done on 25 May 1999 using diluted honeydew from a wheat isolate (10^6 spores ml^{-1}) using the inoculator described in Chapter 2. Groups of about 20 tillers were inoculated at two points, about 1- 1.5 metres in from each end, in each plot (size 6.0 x 1.70 m) and a cable tie used to mark the location of each inoculation point. Inoculation was carried out from 10.45 to 12.00 hours and fungicides were applied from 12.40 to 14.10 hours under hot dry conditions.

Secondary inoculum on inoculated ears was dispersed using a 2 m plastic rod to agitate the ears on 7 June, under showery conditions, and again on 8 June, when traces of honeydew were noticed, but conditions were cool and dry. The rod was wiped clean at the end of each plot.

Assessment methods:

At GS 77 (19 July) detailed assessments were done on 20 inoculated ears¸ 10 from each inoculated area of each plot. The ears were cut from the tillers and numbers of ergots per ear counted. In addition, the total numbers of ears per plot with ergot were counted and the number of ergots per infected ear recorded.

Brown rust was particularly severe in the experiment and a foliar disease assessment was made on the top two leaves on 5 July at GS 75.

Experiment 5.4 Winter rye 1999

Plots were established in a commercial crop of winter rye cv. Esprit on a site at Trent, Sherborne, Dorset. The crop followed a previous crop of rye and maximised the risk of natural ergot inoculum being present.

A range of fungicides was selected to represent the major types of commercial active ingredients and compared with applications of copper or copper + boron as described for winter wheat (Tables 5.3 and 5.4).

Ears were inoculated at the late boot-ear emergence stage (GS 49-57) on 7 May 1999 using diluted honeydew from a wheat isolate (10^6 spores ml^{-1}) using the inoculator described in Chapter 2. Groups of about 20 tillers were inoculated at two points, about 1- 1.5 metres in from each end of each plot and a cable tie used to mark the location of each inoculation point. Inoculation was carried out whilst it was raining.

Inoculated plants had any secondary inoculum dispersed using a wooden broom handle to agitate the ears on 27 May and again on 3 June. Traces of honeydew were first noticed on 27 May and honeydew was more noticeable on 3 June. The broom handle was wiped clean at the end of each plot.

At GS 77 (8 July) detailed assessments were made on a 0.25 m^2 quadrat on each plot. This method was chosen as the inoculated ears could not be located. In each quadrat the total numbers of ears, the numbers of ears infected with ergot, and numbers of ergots per infected ear were recorded.

Experiment 5.5 Winter wheat 2000

Plots of winter wheat cv. Rialto were established on a site with a history of ergot problems at Woolpit, Suffolk on the area used for the experiment in 1998 and 1999. Because of the few ergots left after harvest in 1999, the crop was allowed to flower and was treated with pollen suppressant chemical on 22 May to encourage more open flowering.

A range of fungicides was selected to represent the main types of commercial active ingredients and compared at two application timings. All were used at double commercial rates as high volume sprays (1000 l ha^{-1}). Sprays were applied at second node stage and at early ear emergence or at early ear emergence only to identify activity against ergot (Tables 5.5 and 5.6).

Table 5.5. Treatments and product rates per ha, 2000.

	One to three nodes (GS 31-33) + pre-flowering (GS 55-59)		Pre-flowering only (GS55-59)		
Treatment No.	Product	Dose of product l ha^{-1} (each time)	Treatment No.	Product	Dose of product l ha^{-1} (each time)
1	Untreated control	-	11	Untreated control	-
2	Bavistin DF	2	12	Bavistin DF	2
3	Opus	2	13	Opus	2
4	Bravo	4	14	Bravo	4
5	Folicur	2	15	Folicur	2
6	Alto 100	1.6	16	Alto 100	1.6
7	Amistar	2	17	Amistar	2
8	Corbel	2	18	Corbel	2
9	Sportak 45	1.8	19	Sportak 45	1.8
10	Sanction	1	20	Sanction	1

Table 5.6. Formulation and dose of treatments, 2000.

Treatment number	Fungicide active ingredient	Product	Formulation	Rate of active ingredient kg ha^{-1}
1	Untreated			
2	Carbendazim	Bavistin DF	50 % w/w WG	1.00
3	Epoxiconazole	Opus	125 g/l SC	0.25
4	Chlorothalonil	Bravo 500	500 g/l SC	2.00
5	Tebuconazole	Folicur	250 g/l EW	0.50
6	Cyproconazole	Alto 100	100 g/l SL	0.16
7	Azoxystrobin	Amistar	250 g/l SC	0.50
8	Fenpropimorph	Corbel	750 g/l EC	1.50
9	Prochloraz	Sportak 45	450 g/l EC	0.81
10	Flusilazole	Sanction	400 g/l EC	0.40

Fungicides were applied on 5 May (GS 32) and 5 June (GS 55) using an OPS knapsack sprayer using 03 F110 nozzles operated at 2 bars to deliver 1000 l ha^{-1}.

Inoculation of ears at the ear emerging stage (GS 55) was done on 5 June 2000 using diluted honeydew from a wheat isolate (10^6 spores ml^{-1}) using the inoculator described in Chapter 2. Groups of about 20 tillers were inoculated at two points, about 1- 1.5 metres in from each end, in each plot (size 6.0 x 1.20 m) and a cable tie used to mark the location of each inoculation point. Inoculation was carried from 09.45 to 12.00 hours and fungicides were applied during the early afternoon under warm dry conditions.

Inoculated plants had any secondary inoculum dispersed using a 2 m cane to agitate the ears on the morning of 15 June when anthesis had started (GS 65) and whilst the crop was still damp. Traces of honeydew were noticed. Later in the day, conditions became hot and dry. The cane was wiped clean at the end of each plot.

Assessment methods:

Ergots were apparent in inoculated ears by 11 July and a full assessment was done on 18 July at GS 77-83 on 10 inoculated ears per plot. The ears were cut from the tillers and numbers of ergots per ear counted. In addition, 'natural infection' was assessed on the whole plot by recording the total numbers of ears per plot with ergot and the numbers of ergots per infected ear. Late tillers were still showing honeydew and a second assessment of 'natural infection' was made on 5 August (GS 87) in the central area (2.5 m plot length) of each plot. Ergots in green ears were distinguished from those in mature ears.

Brown rust was particularly severe in the experiment and a green leaf area assessment was made on the top two leaves on 18 July at GS 77.

Experiment 5.6 Winter rye 2000

Plots of winter rye cv. Esprit were established on a site with a history of ergot problems at Trent, Sherborne, Dorset. The crop followed two previous crops of rye and maximised the risk of natural ergot inoculum being present. Plot size was 2.25 m by 11.0 m.

Nine fungicides were selected from those tested in previous years to represent the main types of commercial active ingredients and compared as one and two spray programmes as described for winter wheat (Tables 5.5 and 5.6).

Sprays were applied on 26 April (GS 31) and on 17 May (GS 55-59) using an OPS knapsack sprayer fitted with TeeJet® XR11003VS nozzles operated at 260 kPa to deliver 1000 l ha^{-1}. Sprays were applied at the second node stage and at early ear emergence or at early ear emergence only to identify activity against ergot.

Ears were inoculated at the late boot-ear emergence stage (GS 49-57) on 17 May 2000 using diluted honeydew from a wheat isolate (10^6 spores ml^{-1}) using the inoculator described in Chapter 2. Groups of about 20 tillers were inoculated at two points, about 1- 1.5 metres in from each end of each plot and a white plastic tie and cane used to mark the location of each inoculation point. Inoculated plants had any secondary inoculum dispersed using a wooden broom handle to agitate the ears on 27 May and again on 2 June. Honeydew was noticeable on inoculated ears on 2 June.

Assessment methods:

Twenty-five ergots derived from wheat and 25 from rye were buried just below the soil surface in a grid pattern in the trial area on 2 December. Ergot germination was observed by monitoring the emergence of apothecia at intervals from 22 February to 2 June.

Plots were checked for honeydew production on ears on 30 May (GS 59-61) and on 2 June (GS 61-65). Honeydew was only found on the second inspection when up to 20 inoculated ears per plot were examined and the number of ears producing honeydew recorded. Ergot development was assessed on 13-17 July (GS 80). Inoculated and non-inoculated ears were assessed separately. Up to 25 inoculated ears were examined at each inoculation point in each plot and the number of infected ears and the number of ergots per ear were recorded. Similar assessments were made on 100 non-inoculated ears per plot.

Statistical Analyses

Experiments were randomised block designs with factorial components for time of application in 1998 and 2000. Treatments had three-fold replication in 1998 and 2000 and four replicates in 1999. Results were transformed where appropriate and subjected to analysis of variance using GENSTAT. The results of the analyses are shown in the tables of results as the level of significance in the F-test (*, ** or *** to indicate probabilities of <0.05, 0.01, 0.001, respectively).

RESULTS

Experiment 5.1 Winter wheat 1998

Disease development

Ergot germination was recorded for wheat ergots only as rye ergots stored at ambient from harvest 1996 were not viable. Germination was first seen on 28 May (wheat at the boot stage, GS 52) and reached 8% on 5 June (GS 55) and 15 June (GS 65) with a peak of 40% germination on 22 June (GS 69). There was a similar depot at ADAS Boxworth with the same batch of sclerotia and it showed a peak of only 12% germination of rye sclerotia. The first germination was recorded on 2 May 2000 when winter wheat was at GS 33.

On 8 June 1998, 11 days after inoculation, stickiness attributed to honeydew was noticed on some ears, indicating probable early development of ergot. Ergots were first seen in the ears on 7 July 1998.

Disease control

Ear inoculation was very successful and over 90% of inoculated ears developed ergot (Table5.7). There were no significant differences in the percentages of inoculated ears with ergot (Table 5.7). However, the numbers of ergots per ear (Table 5.8) were reduced by fungicide treatments from 10.07 to as few as 5.68 (tebuconazole). All of the fungicides except flutriafol showed a significant reduction in the numbers of ergots per ear (Table 5.8). There was no interaction between timing and fungicides.

The control of ergot, which spread from the inoculated ears, was examined close to the inoculated area (0.3 m) and at the edge of the plot (1.2 m). The incidence of ergot was 4.47% ears affected at 0.3 m (Table 5.9) with an average of 3.83 ergots per 50 ears (Table 5.10). At 1.2 m, ergot was found in 1.1% ears (Table 5.11) with 0.83 ergots per 50 ears (Table 5.12). No significant treatment differences were detected. When data

from 0.3 and 1.2 m were combined (Tables 5.13 and 5.14), there were again no significant differences between treatments.

A further assessment made at crop maturity estimated total ergot development in the plot (Table 5.15) and this averaged 2.51% ears affected. All fungicide treatments had lower ergot incidence than the untreated control. A specific ear assessment in the main affected zone (0.2 -1.0 m from the inoculated ears) showed 8.5% ears with ergot (Table 5.16) with an average of 19.7 ergots per 50 ears (Table 5.17). There were no treatment differences. Some heavy ergot infection occurred on late tillers at the 1 August assessment, but no treatment affected the proportion of late green tillers, which averaged 3% overall.

Low levels of fusarium ear blight were present and averaged 5 ears per plot in control plots (Table 5.18). Treatment differences were not significant. At this stage, there was slight sooty mould development which was notably less conspicuous in the azoxystrobin treatments, but this was not assessed quantitatively.

Table 5.7. Percentage inoculated wheat ears with ergot, Woolpit 7 July 1998
(Sample size - 10 ears per plot)

Treatment no.	Fungicide	% ears with ergot Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	100.0	100.0	100.0
2	Carbendazim	93.3	90.0	91.7
3	Epoxiconazole	86.7	96.7	91.7
4	Chlorothalonil	90.0	100.0	95.0
5	Tebuconazole	76.7	90.0	83.3
6	Cyproconazole	93.3	90.0	91.7
7	Flutriafol	90.0	96.7	93.3
8	Azoxystrobin	86.7	83.3	85.0
9	Kresoxim methyl / fenpropimorph + fenpropimorph	96.7	93.3	95.0
10	Cyprodinil	93.3	96.7	95.0
Mean		90.7	93.7	92.2
SED (38df)	- Fungicide			5.70
	Timing	2.55		
	Interaction	8.06		

Table 5.8. Number of ergots per inoculated ear, Woolpit 7 July 1998
(Sample size - 10 ears per plot)

Treatment no.	Fungicide	Number of ergots per ear Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	11.27	8.87	10.07
2	Carbendazim	7.70	6.97	7.33
3	Epoxiconazole	6.03	7.07	6.55
4	Chlorothalonil	7.13	6.73	6.93
5	Tebuconazole	4.30	7.07	5.68
6	Cyproconazole	8.60	6.97	7.78
7	Flutriafol	8.13	7.87	8.00
8	Azoxystrobin	7.77	4.90	6.33
9	Kresoxim methyl / fenpropimorph + fenpropimorph	8.73	5.30	7.02
10	Cyprodinil	7.17	7.13	7.15
Mean		7.68	6.89	7.28
SED (38 df)	- Fungicide			1.01 *
	Timing	0.45		
	Interaction	1.42		

Table 5.9 Percentage ears with ergot at 0.3 m from inoculated plants, Woolpit 7 July 1998
(Sample size - 50 ears per plot)

Treatment no.	Fungicide	% ears with ergot Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	7.33	1.33	4.33
2	Carbendazim	1.33	6.67	4.00
3	Epoxiconazole	6.00	4.00	5.00
4	Chlorothalonil	6.67	0.00	3.33
5	Tebuconazole	2.00	6.00	4.00
6	Cyproconazole	4.67	4.00	4.33
7	Flutriafol	4.67	6.00	5.33
8	Azoxystrobin	4.67	3.33	4.00
9	Kresoxim methyl / fenpropimorph + fenpropimorph	4.00	5.33	4.67
10	Cyprodinil	7.33	4.00	5.67
Mean		4.87	4.07	4.47
SED (38df)	- Fungicide			2.01
	Timing	0.90		
	Interaction	2.85		

Table 5.10. Number of ergots at 0.3 m from inoculated plants Woolpit 7 July 1998
(Sample size - 50 ears per plot)

Treatment no.	Fungicide	Number of ergots Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	7.33	1.00	4.17
2	Carbendazim	0.67	4.00	2.33
3	Epoxiconazole	4.33	2.67	3.50
4	Chlorothalonil	6.33	0.00	3.17
5	Tebuconazole	1.00	5.67	3.33
6	Cyproconazole	2.33	3.00	2.67
7	Flutriafol	3.00	13.67	8.33
8	Azoxystrobin	4.00	2.67	3.33
9	Kresoxim methyl / fenpropimorph + fenpropimorph	2.67	2.67	2.67
10	Cyprodinil	6.33	6.33	4.83
Mean		3.80	3.87	3.83
SED (38 df)	- Fungicide			3.23
	Timing	1.446		
	Interaction	4.574		

Table 5.11. Percentage ears with ergot at 1.2 m from inoculated plants, Woolpit 7 July 1998
(Sample size - 50 ears per plot)

Treatment no.	Fungicide	% ears with ergot Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	0.67	1.33	1.00
2	Carbendazim	2.67	0.00	1.33
3	Epoxiconazole	0.67	0.67	0.67
4	Chlorothalonil	1.33	1.33	1.33
5	Tebuconazole	0.00	2.00	1.00
6	Cyproconazole	1.33	0.00	0.67
7	Flutriafol	0.00	2.00	1.00
8	Azoxystrobin	2.00	0.67	1.33
9	Kresoxim methyl / fenpropimorph + fenpropimorph	1.33	2.00	1.67
10	Cyprodinil	1.33	0.67	1.00
Mean		1.13	1.07	1.10
SED (38df)	- Fungicide			1.14
	Timing	0.51		
	Interaction	1.62		

Table 5.12. Number of ergots at 1.2 m from inoculated plants Woolpit 7 July 1998
(Sample size - 50 ears per plot)

Treatment no.	Fungicide	Number of ergots Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	0.33	1.00	0.67
2	Carbendazim	1.67	0.00	0.83
3	Epoxiconazole	0.67	1.33	1.00
4	Chlorothalonil	1.33	0.67	1.00
5	Tebuconazole	0.00	1.33	0.67
6	Cyproconazole	0.67	0.00	0.33
7	Flutriafol	0.00	1.00	0.50
8	Azoxystrobin	1.00	0.33	0.67
9	Kresoxim methyl / fenpropimorph + fenpropimorph	0.67	2.33	1.50
10	Cyprodinil	2.00	0.33	1.17
Mean		0.83	0.83	0.83
SED (38df)	- Fungicide			0.97
	Timing	0.44		
	Interaction	1.39		

Table 5.13. Percentage ears with ergot at 0.3 m and 1.2 m from inoculated ears, Woolpit 7 July 1998
(Sample size 100 ears per plot)

Treatment no.	Fungicide	% ears with ergot Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	4.00	1.33	2.67
2	Carbendazim	2.00	3.33	2.67
3	Epoxiconazole	3.33	2.33	2.83
4	Chlorothalonil	4.00	0.67	2.33
5	Tebuconazole	1.00	4.00	2.50
6	Cyproconazole	3.00	2.00	2.50
7	Flutriafol	2.33	4.00	3.17
8	Azoxystrobin	3.33	2.00	2.67
9	Kresoxim methyl / fenpropimorph + fenpropimorph	2.67	3.67	3.17
10	Cyprodinil	4.33	2.33	3.33
Mean		3.00	2.57	2.79
SED (38 df)	- Fungicide			0.76
	Timing	0.347		
	Interaction	1.077		

Table 5.14. Number of ergots at 0.3m and 1.2 m from inoculated plants combined, Woolpit 7 July 1998 (Sample size - 100 ears)

Treatment no.	Fungicide	Number of ergots Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	7.67	2.00	4.83
2	Carbendazim	2.33	4.00	3.17
3	Epoxiconazole	5.00	4.00	4.50
4	Chlorothalonil	7.67	0.67	4.17
5	Tebuconazole	1.00	7.00	4.00
6	Cyproconazole	3.00	3.00	3.00
7	Flutriafol	3.00	14.67	8.83
8	Azoxystrobin	5.00	3.00	4.00
9	Kresoxim methyl / fenpropimorph + fenpropimorph	3.33	5.00	4.14
10	Cyprodinil	8.33	3.67	6.00
Mean		4.63	4.70	4.67
SED (38 df)	- Fungicide			3.26
	Timing	1.46		
	Interaction	4.61		

Table 5.15. Percentage ears with ergot in whole plot, Woolpit 1 August 1998 (Sample size - whole plot)

Treatment no.	Fungicide	% ears with ergot Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	3.50	3.17	3.33
2	Carbendazim	3.50	2.33	2.92
3	Epoxiconazole	2.67	1.67	2.17
4	Chlorothalonil	3.17	2.00	2.58
5	Tebuconazole	1.67	2.33	2.00
6	Cyproconazole	2.33	2.33	2.33
7	Flutriafol	3.00	2.83	2.92
8	Azoxystrobin	1.83	2.00	1.92
9	Kresoxim methyl / fenpropimorph + fenpropimorph	1.83	3.17	2.50
10	Cyprodinil	2.50	2.33	2.42
Mean		2.60	2.42	2.51
SED (38df)	- Fungicide			0.58
	Timing	0.26		
	Interaction	0.82		

Table 5.16. Percentage ears with ergot from edge of inoculated area to edge of plot, Woolpit, 1 August 1998 (Sample size - 50 ears per plot)

Treatment no.	Fungicide	% ears with ergot Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	8.67	10.00	9.33
2	Carbendazim	12.67	8.67	10.67
3	Epoxiconazole	10.00	11.33	10.67
4	Chlorothalonil	8.67	10.00	9.33
5	Tebuconazole	3.33	11.33	7.33
6	Cyproconazole	8.67	5.33	7.00
7	Flutriafol	6.67	9.33	8.00
8	Azoxystrobin	8.00	5.33	6.67
9	Kresoxim methyl / fenpropimorph + fenpropimorph	6.00	8.67	7.33
10	Cyprodinil	8.00	9.33	8.67
Mean		8.07	8.93	8.50
SED (38 df)	- Fungicide			2.84
	Timing	1.27		
	Interaction	4.02		

Table 5.17. Number of ergots from edge of inoculated area to edge of plot , Woolpit 1 August 1998 (Sample size - 50 ears per plot)

Treatment no.	Fungicide	Number of ergots Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	29.3	23.3	26.3
2	Carbendazim	22.0	10.7	16.3
3	Epoxiconazole	19.3	27.3	23.3
4	Chlorothalonil	18.7	23.3	21.0
5	Tebuconazole	10.7	30.0	20.3
6	Cyproconazole	18.7	10.0	14.3
7	Flutriafol	16.0	23.3	19.7
8	Azoxystrobin	35.0	8.0	21.7
9	Kresoxim methyl / fenpropimorph + fenpropimorph	10.0	23.3	16.7
10	Cyprodinil	16.7	18.7	17.7
Mean		19.7	19.8	19.7
SED (38 df)	- Fungicide			11.01
	Timing	4.92		
	Interaction	15.57		

Table 5.18. Number of wheat ears per plot with fusarium ear blight - 7 July 1998 (Sample size - whole plot).

Treatment no.	Fungicide	Number of ears Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	4.67	5.00	4.83
2	Carbendazim	1.33	0.33	0.83
3	Epoxiconazole	1.33	0.67	1.00
4	Chlorothalonil	2.33	2.00	2.17
5	Tebuconazole	0.67	2.33	1.50
6	Cyproconazole	1.33	9.00	5.17
7	Flutriafol	1.33	2.67	2.00
8	Azoxystrobin	0.67	2.00	1.33
9	Kresoxim methyl / fenpropimorph + fenpropimorph	2.00	0.00	1.00
10	Cyprodinil	1.67	2.67	2.17
Mean		1.73	2.67	2.20
SED (38 df)	- Fungicide			2.07
	Timing	0.93		
	Interaction	2.93		

Experiment 5.2 Winter rye 1998

At Poyntington, Dorset, ergot inocuation at the boot stage was only partially successful with 20-33% ears affected in individual treatments and no significant treatment differences (Table 5.19). Infected ears averaged 2.4 ergots per ear with no evidence of fungicidal control (Table 5.20). There was no secondary spread at this site.

Table 5.19. Percentage inoculated rye ears with ergot, Poyntington, 7 July 1998
(Sample size - 25 ears per plot)

Treatment no.	Fungicide	% ears with ergot Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	28.7	28.2	28.4
2	Carbendazim	28.4	24.9	26.8
3	Epoxiconazole	20.4	26.2	23.3
4	Chlorothalonil	21.8	25.8	23.8
5	Tebuconazole	24.0	33.3	28.7
6	Cyproconazole	27.1	26.2	26.8
7	Flutriafol	26.2	22.7	24.4
8	Azoxystrobin	28.0	34.2	31.1
9	Kresoxim methyl / fenpropimorph + fenpropimorph	25.8	26.7	26.2
10	Cyprodinil	28.4	30.2	29.3
Mean		25.9	27.8	26.9
SED (38df)	- Fungicide			2 .96
	Timing	1.32		
	Interaction	4.19		

Table 5.20. Number of ergots per inoculated rye ear, Poyntington 7 July 1998
(Sample size - 25 ears per plot)

Treatment no.	Fungicide	Number of ergots per ear Spray timing GS 59	GS 52 + GS 59	Mean
1	Untreated control	2.36	2.23	2.30
2	Carbendazim	2.31	2.75	2.53
3	Epoxiconazole	2.55	2.40	2.47
4	Chlorothalonil	2.52	2.45	2.49
5	Tebuconazole	2.18	2.40	2.29
6	Cyproconazole	2.15	2.06	2.11
7	Flutriafol	2.45	2.55	2.52
8	Azoxystrobin	2.24	2.21	2.23
9	Kresoxim methyl / fenpropimorph + fenpropimorph	2.48	2.43	2.45
10	Cyprodinil	2.40	2.63	2.52
Mean		2.37	2.41	2.39
SED (38 df)	- Fungicide			0.186
	Timing	0.083		
	Interaction	0.263		

Experiment 5.3 Winter wheat 1999

Disease development

On 8 June 1999, 7 days after inoculation, stickiness attributed to honeydew was noticed on some ears, indicating probable early development of ergot. Ergots were first seen on the ears on 5 July 1999, when infection was much lower than in 1998.

Ergot was present in annual meadow grass in the plots during the course of the experiment.

Table 5.21. Effect of fungicide treatments on ergot in inoculated ears, Woolpit 1999.

Treatment no.	Fungicides applied at first ear emergence (GS 51) + pre-flowering (GS 59)	Dose of product l ha^{-1}	% inoculated ears affected	Mean number of ergots per inoculated ear
1	Untreated control	-	62.5	1.95
2	Carbendazim	2	52.5	1.35
3	Epoxiconazole	2	68.7	2.84
4	Chlorothalonil	4	70.0	2.28
5	Tebuconazole	2	52.5	1.36
6	Cyproconazole	1.6	62.5	1.95
7	Azoxystrobin	2	70.0	2.59
8	Kresoxim methyl / fenpropimorph + fenpropimorph	1.4 + 1.44	62.5	2.10
9	Fenpropimorph	2	75.0	2.61
10	Fluquinconazole	1.5	67.5	2.33
11	Prochloraz	1.8	63.8	2.25
12	Flusilazole	1	57.5	1.96
13	Trifloxystrobin	2	67.5	2.20
14[1]	Copper	2	62.5	2.14
15[1]	Copper + boron	2 + 2	66.2	1.70
SED (42 df)			10.34	0.468

[1] First spray 28 April at GS 32
Second spray25 May at GS 51

Table 5.22. Effect of fungicides on secondary ergot infection, Woolpit 1999.

Treatment No.	Fungicides applied at first ear emergence (GS 51) + pre-flowering (GS 59)	Dose of product l ha^{-1}	No. ears with ergot per plot[1]	No. ergots per plot
1	Untreated	-	0.00 $^{++}$(0.00)	0.00
2	Carbendazim	2	0.50 (0.35)	0.50
3	Epoxiconazole	2	0.75 (0.60)	0.75
4	Chlorothalonil	4	0.25 (0.25)	0.25
5	Tebuconazole	2	0.50 (0.35)	0.50
6	Cyproconazole	1.6	0.25 (0.25)	0.25
7	Azoxystrobin	2	2.25 (1.27)	2.75
8	Kresoxim methyl / fenpropimorph + fenpropimorph	1.4 + 1.44	1.25 (0.56)	1.25
9	Fenpropimorph	2	2.25 (1.27)	3.75
10	Fluquinconazole	1.5	1.75 (1.06)	4.00
11	Prochloraz	1.8	0.50 (0.50)	0.50
12	Flusilazole	1	1.00 (1.00)	1,25
13	Trifloxystrobin	2	0.50 (0.35)	0.50
14[2]	Copper	2	1.00 (0.68)	1.50
15[2]	Copper + boron	2 + 2	0.75 (0.43)	1.00
SED (42 df)			0.519	1.693

[1] Square root transformed data, in parentheses, were analysed

[2] First spray 28 April at GS 32
Second spray 25 May at GS 51

Table 5.23. Effect of fungicides on brown rust severity and green leaf retention at GS75, Woolpit 1999.

Treatment No.	Fungicides applied at first ear emergence (GS 51) + pre-flowering (GS 59)	Dose of product l ha^{-1}	% brown rust L1 +L2	Mean % green leaf L1 + L2
1	Untreated control	-	35.0	12
2	Carbendazim	2	34.5	13
3	Epoxiconazole	2	0.00	95
4	Chlorothalonil	4	3.30	92
5	Tebuconazole	2	0.00	93
6	Cyproconazole	1.6	0.20	93
7	Azoxystrobin	2	0.00	96
8	Kresoxim methyl / fenpropimorph + fenpropimorph	1.4 + 1.44	0.00	95
9	Fenpropimorph	2	0.10	95
10	Fluquinconazole	1.5	0.00	94
11	Prochloraz	1.8	15.5	59
12	Flusilazole	1	0.03	90
13	Trifloxystrobin	2	1.80	94
14[1]	Copper	2	38.8	9
15[1]	Copper + boron	2 + 2	35.0	10
SED (42 df)			3.43***	7.42***

[1] First spray 28 April at GS 32
Second spray 25 May at GS 51

Disease control

Ear inoculation was successful, and over 60% of inoculated ears developed ergot despite the rather earlier inoculation (at the boot stage) than in 1998 (Table 5.21). There were no significant differences in the percentages of inoculated ears with ergot (Table 5.21), nor in the numbers of ergots per ear (Table 5.21). However, tebuconazole and carbendazim had the lowest numbers of ergot per ear, continuing a trend seen in 1998. There were no effects of copper or boron treatments on ergot.

There was slight foliar scorch from tebuconazole and more serious damage (20-30% area affected on leaf 2) from flusilazole treatments by 7 June.

There was no control of ergot,which spread from the inoculated ears or from natural sources nearby (Table 5.22). It is likely that dry weather after 7 June prevented further secondary spread. Nevertheless the presence of ergot is an indication that fungicides were not completely effective.

Experiment 5.4 Winter rye 1999

The plots at Trent lodged very severely and tags were lost from inoculated groups of tillers. Assessments of secondary infection were made using quadrats. Ergot was present on about one percent tillers overall with 2-3 ergots per infected ear. There were low levels of ergot in epoxiconazole, flusilazole, azoxystrobin and cyproconazole treated plots and no ergot in the fluquinconazole treatment (Tables 5.24 and 5.25). Kresoxim-methyl + fenpropimorph and prochloraz treatments had the highest ergot infection but there were no significant effects of treatments on ergot (Tables 5.24 and 5.25).

Table 5.24. Total number of ears, number of ears with ergot and total number of ergots per 0.25m^2, Trent 1999

Treatment no	Fungicides applied at first ear emergence (GS 51) + pre-flowering (GS 59)	Dose of product l ha^{-1}	No of ears / 0.25m^2	No of ears infected/ 0.25m^2	No of ergots/ 0.25m^2
1	Untreated	-	97.2	1.00	1.25
2	Carbendazim	2	97.5	0.27	0.22
3	Epoxiconazole	2	100.5	0.25	0.25
4	Chlorothalonil	4	113.5	0.94	0.88
5	Tebuconazole	2	100.5	1.25	1.75
6	Cyproconazole	1.6	105.2	0.25	0.75
7	Azoxystrobin	2	116.5	0.25	0.25
8	Kresoxim methyl / fenpropimorph + fenpropimorph	1.4 + 1.44	95.2	1.75	2.00
9	Fenpropimorph	2	111.7	0.60	0.55
10	Fluquinconazole	1.5	117.5	0.00	0.00
11	Prochloraz	1.8	100.0	1.25	2.75
12	Flusilazole	1	96.2	0.25	0.25
13	Trfloxystrobin	2	94.2	1.25	1.50
14[1]	Copper	2	84.0	0.5	0.75
15[1]	Copper + boron	2 + 2	93.0	1.25	1.75
	SED (42 d.f.)		11.36	0.820	1.312

[1] First spray 27 April (GS32), second spray at ear emergence on 7 May (GS 51).

Table 5.25. Mean number of ergots per infected ear, mean number of ergots per ear and percentage of ears infected by ergot, Trent 1999

Treatment no	Fungicides applied at first ear emergence (GS 51) + pre-flowering (GS 59)	Dose of product l ha^{-1}	Mean no of ergots per infected ear	Mean no of ergots per ear	% ears infected
1	Untreated	-	1.25	0.013	1.05
2	Carbendazim	2	0.35	0.002	0.24
3	Epoxiconazole	2	0.25	0.002	0.18
4	Chlorothalonil	4	1.01	0.008	0.87
5	Tebuconazole	2	1.50	0.018	1.28
6	Cyproconazole	1.6	0.75	0.007	0.22
7	Azoxystrobin	2	0.25	0.002	0.18
8	Kresoxim methyl / fenpropimorph + fenpropimorph	1.4 + 1.44	0.80	0.019	1.69
9	Fenpropimorph	2	0.68	0.005	0.53
10	Fluquinconazole	1.5	0.00	0.000	0.00
11	Prochloraz	1.8	0.88	0.028	1.24
12	Flusilazole	1	0.25	0.003	0.30
13	Trfloxystrobin	2	0.56	0.014	1.15
14[1]	Copper	2	0.75	0.009	0.58
15[1]	Copper + boron	2 + 2	0.35	0.022	1.60
SED (42 d.f.)			0.521	0.0138	0.866

[1] First spray 27 April (GS 32), second spray at ear emergence on 7 May (GS 51).

Experiment 5.5 Winter wheat 2000

Disease development

On 15 June 2000, 10 days after inoculation, traces of honeydew were noticed on some inoculated ears, indicating successful early development of ergot. Honeydew was present in inoculated ears generally by 26 June (GS 65-71) and some partially developed ergots were present. Spread of ergot continued up to crop maturity on late tillers and honeydew was still apparent on 5 August.

There were small patches of yellow rust in a few plots on 15 June, but it was clear that brown rust was the most serious foliar disease by 25 June. Subsequently brown rust caused early death of the upper leaves in plots, which had received sprays with limited activity against rusts. Data for green leaf retention indicate treatment differences for rust control.

Table 5.26. Effect of fungicide treatments on percentage of inoculated ears with ergot at GS77, Woolpit 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	% ears with ergot		
Timing			GS 55	GS32 + GS 55	Mean
1	Untreated control	-	81.1	83.9	82.5
2	Carbendazim	2	90.0	90.0	90.0
3	Epoxiconazole	2	76.9	90.0	83.5
4	Chlorothalonil	4	72.3	83.9	78.1
5	Tebuconazole	2	75.0	90.0	82.5
6	Cyproconazole	1.6	81.1	90.0	85.6
7	Azoxystrobin	2	78.9	90.0	84.5
8	Fenpropimorph	2	83.9	81.1	82.5
9	Prochloraz	1.8	83.9	90.0	86.9
10	Flusilazole	1	90.0	90.0	90.0
Mean			81.3	87.9	84.6
SED (38df)	Fungicide				6.89
	Timing			3.08*	
	Interaction			9.74	

Table 5.27. Effect of fungicide treatments on number of ergots per inoculated ear at GS 77, Woolpit 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	Number of ergots per ear		
Timing			GS 55	GS32 + GS 55	Mean
1	Untreated control	-	6.3	6.3	6.3
2	Carbendazim	2	7.7	7.6	7.7
3	Epoxiconazole	2	6.7	8.0	7.3
4	Chlorothalonil	4	6.4	7.4	6.9
5	Tebuconazole	2	5.6	7.2	6.4
6	Cyproconazole	1.6	7.5	9.3	8.4
7	Azoxystrobin	2	6.4	6.5	6.4
8	Fenpropimorph	2	8.9	8.7	8.8
9	Prochloraz	1.8	6.1	8.3	7.2
10	Flusilazole	1	8.5	8.6	8.5
Mean			7.0	7.8	7.4
SED (38df)	Fungicide				1.15
	Timing			0.51	
	Interaction			1.63	

The artificial inoculation of ears with ergot conidia was very successful and overall 85% ears became infected, but no treatment differences were identified between fungicides. There was a significant effect of fungicide timing with 87.9% ears affected after two sprays but only 81.3% affected after a single spray at GS 55 (Table 5.26). All of fungicide treatments showed more ergots per ear than the untreated control but the differences were not statistically significant (Table 5.27).

Table 5.28. Effect of fungicides on the number of ears with secondary ergot infection at GS 77, Woolpit 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	Number of ears per plot with ergot		
Timing			GS 55	GS32 + GS 55	Mean
1	Untreated control	-	26.3	18.3	22.3
2	Carbendazim	2	42.3	23.3	32.8
3	Epoxiconazole	2	22.7	16.0	19.3
4	Chlorothalonil	4	18.0	16.3	17.2
5	Tebuconazole	2	25.3	16.7	21.0
6	Cyproconazole	1.6	19.3	23.3	21.3
7	Azoxystrobin	2	14.3	9.0	11.7
8	Fenpropimorph	2	16.7	20.0	18.3
9	Prochloraz	1.8	24.0	21.0	22.5
10	Flusilazole	1	29.0	24.0	26.5
Mean			23.8	18.8	21.3
SED (38df)	Fungicide				4.95*
	Timing			2.21*	
	Interaction			7.00	

Ergot developed via secondary spread from inoculated tussocks in all plots and most infection was located close to the tussocks. Control plots showed 22.3 ears per plot with ergot, which equates to about 0.8% ears affected. Azoxystrobin reduced the numbers of ears affected from 22.3 to 11.7 ears per plot, whilst carbendazim gave a significant increase in ear infection (32.8 ears per plot) (Table 5.28). There was also a significant difference overall between the two spray programme (18.8 ears affected per plot) and the single spray at GS 55 (23.8 ears affected per plot), suggesting an effect from the early spray at GS 32 (Table 5.28). Whilst these differences were evident when data were examined in relation to the total number of ergots per plot, no significant effects were identified (Table 5.29).

Table 5.29. Effect of fungicides on the number of secondary ergot infections per plot at GS 77, Woolpit 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	Number of ergots per plot		
			GS 55	GS32 + GS 55	Mean
1	Untreated control	-	37.3	26.0	31.7
2	Carbendazim	2	54.0	29.0	41.5
3	Epoxiconazole	2	35.7	27.3	31.5
4	Chlorothalonil	4	27.0	28.0	27.5
5	Tebuconazole	2	40.7	21.3	31.0
6	Cyproconazole	1.6	35.3	37.7	36.5
7	Azoxystrobin	2	21.7	16.3	19.0
8	Fenpropimorph	2	24.0	42.3	33.2
9	Prochloraz	1.8	36.3	31.3	33.8
10	Flusilazole	1	46.7	44.0	45.3
Mean			35.9	30.3	33.1
SED (38df)	Fungicide				9.61
	Timing			4.30	
	Interaction			13.59	

Plots were assessed on 5 August when further ergot infection had developed on late tillers. Green and mature (ripe) ears were distinguished in this assessment to establish if treatment effects had influenced late secondary spread. There were significant differences between fungicides and these were the result of large increases in the numbers of ergots in epoxiconazole and tebuconazole treated plots (Table 5.30). All fungicide treatments showed higher ergot infection than the untreated. There were no differences between the one and two spray programmes. Data for the number of infected ears showed no significant differences between treatments and are not presented.

Table5.30. Effect of fungicides on secondary ergot infection at GS 87, Woolpit 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	Number of ergots per 2.5 m plot		
			GS 55	GS32 + GS 55	Mean
1	Untreated control	-	35.3	28.0	31.7
2	Carbendazim	2	28.7	47.3	38.0
3	Epoxiconazole	2	80.0	101.7	90.8
4	Chlorothalonil	4	35.7	50.0	42.8
5	Tebuconazole	2	78.7	95.7	87.2
6	Cyproconazole	1.6	68.0	61.3	64.7
7	Azoxystrobin	2	51.3	64.0	57.7
8	Fenpropimorph	2	40.0	67.0	53.5
9	Prochloraz	1.8	37.3	34.3	35.8
10	Flusilazole	1	54.0	50.0	52.0
Mean			50.9	59.9	55.4
SED (38df)	Fungicide				17.63*
	Timing			7.88	
	Interaction			24.93	

When the number of green and mature ears with ergot were differentiated, four fungicide treatments (azoxystrobin, cyproconazole, epoxiconazole and tebuconazole) gave highly significant increases in the number of green ears affected. The differences were relatively large when considering ergot as a contaminant of grain, ranging from 15.0 to 18.3 ears per 2.5 m plot compared with 4.2 ears per 2.5 m plot in the untreated control (Table 5.31). There were no significant effects on mature ears (Table 5.32) and although azoxystrobin gave the lowest ergot infection overall, the previous significant reduction was no longer apparent.

Table 5.31. Effect of fungicides on the number of green ears with secondary ergot infection at GS 87, Woolpit 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	Number of green ears with ergot per 2.5 m plot		
			GS 55	GS32 + GS 55	Mean
1	Untreated control	-	5.0	3.3	4.2
2	Carbendazim	2	2.7	14.3	8.5
3	Epoxiconazole	2	13.7	23.0	18.3
4	Chlorothalonil	4	6.3	9.0	7.7
5	Tebuconazole	2	13.3	20.0	16.7
6	Cyproconazole	1.6	15.0	15.0	15.0
7	Azoxystrobin	2	18.0	13.7	15.8
8	Fenpropimorph	2	8.3	8.0	8.2
9	Prochloraz	1.8	5.7	4.3	5.0
10	Flusilazole	1	9.3	6.3	7.8
Mean			9.7	11.7	10.7
SED (38df)	Fungicide				3.21***
	Timing			1.44	
	Interaction			4.54	

Table 5.32. Effect of fungicides on the number of mature ears with secondary ergot infection at GS 87, Woolpit 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	Number of mature ears with ergot per 2.5 m plot		
			GS 55	GS32 + GS 55	Mean
1	Untreated control	-	20.0	14.0	17.0
2	Carbendazim	2	18.3	14.3	16.3
3	Epoxiconazole	2	17.0	18.3	17.7
4	Chlorothalonil	4	13.0	16.7	14.8
5	Tebuconazole	2	18.0	15.3	16.7
6	Cyproconazole	1.6	14.3	17.7	16.0
7	Azoxystrobin	2	10.0	17.0	13.5
8	Fenpropimorph	2	16.0	22.0	19.0
9	Prochloraz	1.8	22.0	16.3	19.2
10	Flusilazole	1	17.7	15.0	16.3
Mean			16.6	16.7	16.6
SED (38df)	Fungicide				4.33
	Timing			1.94	
	Interaction			6.13	

There were large effects of fungicide treatments on green leaf area on the upper two leaves at GS 77. Data were similar for both leaves and only leaf 1 analyses are presented (Table 5.33). The differences reflected control of brown rust, the main foliar disease at this site. Azoxystrobin, cyproconazole, epoxiconazole and tebuconazole had the highest green leaf area (71-84%), whilst untreated control plot were almost dead (0.8% green area). Fenpropimorph and flusilazole also had a significant effect on green area, but carbendazim, chlorothalonil and prochloraz were ineffective. The most effective treatments for green leaf retention were the same as those that gave high numbers of green tillers with ergot (Table 5.31).

A significant correlation between ergots in green tillers on 5 August and green leaf area at GS 77 was identified. The correlations were slightly stronger for numbers of ergots rather than numbers of infected late tillers and for green leaf area on leaf 2 rather than leaf 1. This was most appropriately defined by the linear regression equation, which accounted for 42.7% of the variation in ergot infection of late tillers:

Number of ergots per plot in late tillers = 10.4 + 0.534 (% green leaf area, leaf 2 at GS 77)
(r^2 = 42.7%, P<0.001)

Table 5.33. Effect of fungicides on green leaf retention on leaf 1 at GS77, Woolpit 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	% green leaf area leaf 1		
			GS 55	GS32 + GS 55	Mean
1	Untreated control	-	1.0	0.7	0.8
2	Carbendazim	2	2.3	10.7	6.5
3	Epoxiconazole	2	91.3	73.3	82.3
4	Chlorothalonil	4	18.7	15.0	16.8
5	Tebuconazole	2	84.7	81.3	83.0
6	Cyproconazole	1.6	72.3	70.0	71.2
7	Azoxystrobin	2	86.7	82.3	84.5
8	Fenpropimorph	2	55.0	43.3	49.2
9	Prochloraz	1.8	2.0	1.0	1.5
10	Flusilazole	1	39.3	46.7	43.0
Mean			45.3	42.4	43.9
SED (38df)	Fungicide				9.40***
	Timing			4.20	
	Interaction			13.29	

Experiment 5.6 Winter rye 2000

Ergot sclerotia were buried at Trent on 2 December 1999. The first stroma emerged between inspections of the grid made on 26 April and 4 May. A second stroma had emerged by 9 May. Both were from rye ergots. No further germination of rye ergots occurred, but one wheat ergot germinated on, or shortly before 30 May. Ergot activity therefore coincided with ear emergence of the rye.

Disease development

The first obvious honeydew was seen on inoculated ears on 2 June, 16 days after inoculation. The mean percentages of the inoculated ears producing honeydew on 2 June in each treatment are given in Table 5.34. Low honeydew production was apparent in epoxiconazole, flusilazole and fenpropimorph treatments, particularly after two sprays, but differences were not significant. Ergots had developed in most inoculated ears when examined on 13-17 July (GS80), but only sparsely in non-inoculated ears. Results of the ergot assessment are given in Tables 5.35 and 5.36.

Table 5.34. Effect of fungicide treatments on percentage of inoculated ears with honeydew at GS 61-65, Trent 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	% ears with honeydew		
Timing			GS 55-59	GS31 + GS 55-59	Mean
1	Untreated control	-	33.6	31.3	32.4
2	Carbendazim	2	14.6	22.9	19.5
3	Epoxiconazole	2	16.7	5.9	12.1
4	Chlorothalonil	4	34.3	20.0	27.1
5	Tebuconazole	2	29.7	22.3	26.0
6	Cyproconazole	1.6	24.4	19.6	22.0
7	Azoxystrobin	2	18.2	26.7	22.4
8	Fenpropimorph	2	23.3	7.2	15.3
9	Prochloraz	1.8	15.4	32.3	23.8
10	Flusilazole	1	19.1	11.1	15.1
Mean			22.9	20.5	21.7
SED (38df)	Fungicide				7.83
	Timing			3.45	
	Interaction			11.57	

Table 5.35. Effect of fungicide treatments on percentage of inoculated ears with ergot at GS 80, Trent 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	% ears with ergot		
Timing			GS 55-59	GS31 + GS 55-59	Mean
1	Untreated control	-	81.7	80.0	80.8
2	Carbendazim	2	82.0	80.7	81.3
3	Epoxiconazole	2	76.0	73.3	74.7
4	Chlorothalonil	4	83.3	84.7	84.0
5	Tebuconazole	2	82.7	70.7	76.7
6	Cyproconazole	1.6	74.0	76.0	75.0
7	Azoxystrobin	2	78.7	90.0	84.3
8	Fenpropimorph	2	83.3	74.7	79.0
9	Prochloraz	1.8	82.3	81.3	81.8
10	Flusilazole	1	73.3	80.0	76.7
Mean			79.7	79.1	79.4
SED (38df)	Fungicide				4.30
	Timing			1.92	
	Interaction			6.08	

Table 5.36. Effect of fungicide treatments on number of ergots per inoculated ear at GS 80, Trent 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	Number of ergots per ear		
Timing			GS 55-59	GS31 + GS 55-59	Mean
1	Untreated control	-	3.9	4.0	3.9
2	Carbendazim	2	3.6	4.0	3.8
3	Epoxiconazole	2	2.8	2.7	2.8
4	Chlorothalonil	4	3.8	3.8	3.8
5	Tebuconazole	2	4.2	2.8	3.5
6	Cyproconazole	1.6	3.0	3.0	3.0
7	Azoxystrobin	2	3.4	4.1	3.7
8	Fenpropimorph	2	3.5	2.9	3.2
9	Prochloraz	1.8	4.5	4.0	4.3
10	Flusilazole	1	3.1	3.7	3.4
Mean			3.6	3.5	3.5
SED (38df)	Fungicide				0.42*
	Timing			0.19	
	Interaction			0.60	

The artificial inoculation of ears with ergot conidia was very successful and overall 79% ears became infected, but no treatment differences were identified between fungicides. There was no significant effect of fungicide timing. (Table 5.35). All of the fungicide treatments except prochloraz showed fewer ergots per ear than the untreated control, but the differences were only statistically significant for cyproconazole and epoxiconazole (Table 5.36).

Table 5.37. Effect of fungicides on the percentage of ears with secondary ergot infection at GS 80, Trent 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	% ears with ergot		
Timing			GS 55-59	GS31 + GS 55-59	Mean
1	Untreated control	-	5.7	7.3	6.5
2	Carbendazim	2	3.7	1.7	2.7
3	Epoxiconazole	2	1.3	9.3	5.3
4	Chlorothalonil	4	7.7	6.7	7.2
5	Tebuconazole	2	9.0	6.7	7.8
6	Cyproconazole	1.6	4.7	3.3	4.0
7	Azoxystrobin	2	8.3	8.0	8.2
8	Fenpropimorph	2	7.7	5.0	6.3
9	Prochloraz	1.8	7.3	8.3	7.8
10	Flusilazole	1	2.7	4.0	3.3
Mean			5.8	6.0	5.9
SED (38df)	Fungicide				2.40
	Timing			1.07	
	Interaction			3.39	

Ergot developed via secondary spread from inoculated tussocks in all plots and infection was scattered throughout the plots. The surrounding crop showed about one per cent ears affected, and indicating that local inoculum sources had been active. Control plots showed 6.5% ears with ergot. Carbendazim, cyproconazole and flusilazole all showed low levels of ergot, but differences were not statistically significant. Four treatments had more ergot than the untreated control but these were also non-significant differences. There were no differences between one and two spray programmes (Table 5.37). There were no significant differences in the mean numbers of ergots per ear (data not presented) but fungicide treatments differed in the numbers of ergots per infected ear (Table 5.38). In this case there were differences between cyproconazole (2.5 ergots per affected ear) and prochloraz (4.3 ergots per affected ear), though neither differed from the untreated control (3.6 ergots per affected ear) (Table 5.38).

Table 5.38. Effect of fungicides on the number of secondary ergots per infected ear at GS 80, Trent 2000.

Treatment	Fungicide	Dose of product l ha^{-1}	Number of ergots per infected ear		
			GS 55-59	GS31 + GS 55-59	Mean
1	Untreated control	-	3.2	4.0	3.6
2	Carbendazim	2	3.6	2.1	2.8
3	Epoxiconazole	2	1.6	4.8	3.2
4	Chlorothalonil	4	1.8	4.5	3.2
5	Tebuconazole	2	3.3	3.3	3.3
6	Cyproconazole	1.6	2.2	2.7	2.5
7	Azoxystrobin	2	4.2	3.2	3.7
8	Fenpropimorph	2	4.5	2.5	3.5
9	Prochloraz	1.8	4.2	4.4	4.3
10	Flusilazole	1	2.2	4.7	3.4
Mean			3.1	3.6	3.3
SED (38df)	Fungicide				0.77*
	Timing		0.34		
	Interaction		1.09		

DISCUSSION

Experiment 5.1 Winter wheat 1998

The inoculation technique proved successful with substantial infection on inoculated ears. Showery conditions during June favoured infection and secondary spread was achieved, albeit within about 1 m of inoculated ears. Partial sterility was induced chemically and this is likely to have facilitated secondary infection but it is not possible to quantify its contribution.

Whilst it is possible to examine trends in the data, the only significant effect was on ergots per ear in inoculated ears. All treatments gave fewer ergots per ear than the untreated control, most gave reduction between 20 and 40%. Much higher levels of control are clearly needed in commercial practise.

Despite the lower inoculum pressure in ears affected by secondary spread, there was no clear separation of treatments or differences between one and two spray programmes. Some treatments gave no ergot infection in assessments, but these figures should be treated with caution as secondary spread of ergot was detected in all plots. These results suggest that there may be slight effects on ergot from current fungicides but there are doubts about achieving adequate control.

Experiment 5.2 Winter rye 1998

Artificial inoculation was much less successful on rye than on wheat. This almost certainly reflects the slightly earlier growth stage at inoculation (most ears were in boot) and the lower temperatures at the earlier date of inoculation. Lower temperatures are likely to increase the period from inoculation to honeydew formation and hence reduce the amount of inoculum available for secondary spread. Natural inoculum appears to have shown little activity at this site in 1998. Ergot development was very similar in treated and untreated plots and no significant fungicidal activity was identified.

Experiment 5.3 Winter wheat 1999

The inoculation technique proved successful with substantial infection (60-70% ears, 2 ergots per ear) on inoculated ears. Showery conditions were less prevalent than in 1998 at the critical time during June and very little secondary spread was achieved. Ergot was present in annual meadow grass in the plots and, on other farms, ergot did become a problem on winter wheat. Local weather factors appear to have been unfavourable at this site and use of a partial sterilant to suppress pollen production would seem to be appropriate in future work even where high inoculum is known to be present.

Whilst it is possible to examine trends in the data, much higher levels of control are clearly needed for an ergot spray to be adopted by farmers. Two sprays at double rate clearly were not effective under the test conditions. Indeed most treated plots showed higher ergot infestation than the untreated control. The experimental conditions use high inoculum and provided a severe test for fungicides. Under lower inoculum pressure treatments may be more effective.

There was no effect of copper or boron on ergot. In deficiency situations, flower gaping increases and ears become more susceptible to ergot. The benefits are therefore indirect through flowering habit rather than directly fungicidal. Previous reports related to deficient soils in North America and it is therefore not

surprising that benefits were not shown in this case on non-deficient soil. However, deficiency situations can occur in the UK and reductions in ergot may be one of the benefits of remedial action.

Experiment 5.4 Winter rye 1999

The fungicide evaluation on winter rye failed to detect significant differences in secondary spread. A number of azole fungicides had lower ergot infection than the control plots and none were found in assessments made on fluquinconazole-treated plots. To date, no clear differences have emerged in the effectiveness of fungicides on wheat and rye. This site also had low levels of secondary spread as experienced in the wheat experiment, nevertheless ergot problems were again reported on wheat by farmers.

Experiment 5.5 Winter wheat 2000

The inoculation technique proved successful with substantial infection on inoculated ears (85% ears affected) and secondary spread was more extensive than in 1999. This is likely to have been assisted by the use of a pollen suppressant. The 2000 season was reported to be very favourable for ergot infection in central England and this extended to barley crops for the first time. Discard areas of wheat close to the trial showed about 1% ears affected and demonstrated that low level spread had occurred across the site. There were strong foci of infection around inoculated tillers, which clearly showed the importance of introduced inoculum in relation to natural inoculum.

The experiment in 2000 tested two new approaches, namely the effectiveness of relatively early spray timing (GS 32) and the value of high spray volumes. On inoculated ears the single spray gave 6.6% lower incidence of ear infection than two sprays and this appeared to be consistent pattern across fungicides (only Corbel showed a higher ergot incidence after two sprays than after one spray). The cause of this difference is speculative, possibly implicating effects of the GS 32 sprays on ergot susceptibility directly or indirectly through reducing antagonistic microflora.

There were fewer ears with secondary ergot infection after the two-spray programme than after a single spray at GS 55. The difference was modest with a reduction from 23.8 to 18.8 ears per plot at GS 77. There was a strong effect of Amistar on the number of ears with ergot, providing 48% control. No other fungicide showed significant effects. However, when data for the number of secondary ergots per plot were examined, no significant differences were detected. This was consistent with a later assessment on 5 August on the central portion of each plot, which also showed no fungicide control of ergot.

The late assessment showed that treatments had influenced infection of late tillers, apparently through effects on green leaf retention. Thus plots in which severe brown rust was controlled produced more infected late

tillers. Where plots suffered severe brown rust attacks by GS 77, plants ripened early and did not produce late tillers. These observations are helpful in explaining field observations that ergot can develop at higher levels in tramlines than in unwheeled crops. Tramlines often show late tillering, particularly in wet summers. Late tillers allow ergot infection to occur over a long period and such infection is capable of contributing ergot contamination even if grain is immature at harvest.

These results continue to support previous observations that there may be slight effects on ergot from current fungicides but none is likely to give adequate control. The effects of azoxystrobin are of interest and further observations should be made to establish if control is sensitive to the spray volume applied.

Experiment 5.6 Winter rye 2000

Despite effective inoculation of the plots and considerable wet weather after inoculation, there was limited spread of ergot beyond inoculated ears.

The experiment in 2000 tested two new approaches, namely the effectiveness of relatively early spray timing (GS 31) and the value of high spray volumes. On inoculated ears, epoxiconazole gave a 28% reduction in ergot and is one of few significant effects demonstrated on inoculated ears in this project. Unfortunately similar activity was not detected against secondary infection where carbendazim, cyproconazole and flusilazole treatments had the lowest ergot infection. Unlike the wheat experiment, there were no differences between the one and two spray programmes.

These results continue to support previous observations that there may be slight effects on ergot from current fungicides but none is likely to give acceptable control.

CHAPTER 6

STUDIES ON THE MOVEMENT OF RADIO-LABELLED FUNGICIDES TO CEREAL EARS AND OVARIES

INTRODUCTION

None of the 31 chemicals that were tested in the field gave commercially useful and reliable control of ergot, and control was not improved by using an electrostatic sprayer despite assumed increases in deposition of fungicides on the ears. This probably suggests that, to be effective, fungicides need to penetrate deeper into the ears and reach the ovary. Many of the fungicides tested are known to be systemic in the xylem but there is little published information on the extent to which fungicides applied to the leaves are transported to the ear, and, especially, how readily compounds deposited on, or translocated to, the ears then move to the ovary. To investigate this, three ^{14}C-radiolabelled fungicides were applied, in different ways, to plants that were grown in the glasshouse and that were then sampled over a 28-day period to assess movement to the ears and distribution of the fungicides within them.

MATERIALS AND METHODS

Suspensions of the three radio-labelled fungicides (carbendazim, tebuconazole and epoxiconazole) were applied to glasshouse-grown plants using four different methods:

1) painting the flag leaf at GS 41 (flag leaf sheath extending)
2) injecting directly into the stem 5 cm below the ear at GS 59 (ears fully emerged)
3) applying to an absorbent collar placed around the stem directly below the ear at GS 59
4) painting the glumes at GS 59

Single pre-germinated seeds of spring wheat (cv. Avans) were sown in 11.5 cm pots containing a peat-based compost and grown in a warm glasshouse as in Experiment 3.1. A small, artists' brush was used to paint the leaves, and a hypodermic syringe to inject the fungicides into the stem cavity. Absorbent collars were formed as described in Chapter 3. For each method, approximately 1.5 million decompositions per minute (dpm; roughly equivalent to 0.75 µCi (microCurie)) of the labelled compounds were applied to each plant. There were two replicate plants for each method, and samples were taken 5, 14 and 28 days after treatment. For plants treated at GS 41, the flag leaf and developing ear were sampled, and for plants treated at GS 59 the ear was sampled and activity measured in the glumes and in the ovaries (or embryos, after the ovaries had been fertilised, or later still, the developing grain).

The sampled tissues were ground in acetone using a pestle and mortar. After evaporating the acetone, the ground tissue was re-suspended in a few drops of acetone and spotted onto a one-dimensional thin-layer chromatography plate. A different solvent system was used for each labelled compound depending on its solubility characteristics. When the solvent front had reached the top of the plate it was removed from the running tank and from this a 2cm band containing the radiolabel parent fungicide was scraped off and placed in a liquid scintillant. The amount of radioactivity (dpm) was then measured using a liquid scintillation counter for ten minutes.

RESULTS

The counts, corrected by subtracting the background count, were expressed as percentages of the total label applied. In plants to which fungicides were applied by painting the flag leaf, there was a decrease in the amount of radioactivity recovered from the treated leaf over time but this was less marked for carbendazim than for the other two (Fig.7). The decreases may have been partly due to translocation from the leaf but are more likely to have been a consequence of the compounds being metabolised by the plant. There was an increase over time in the amount of activity detected in the developing ear but this never exceeded 0.1% of the total activity applied.

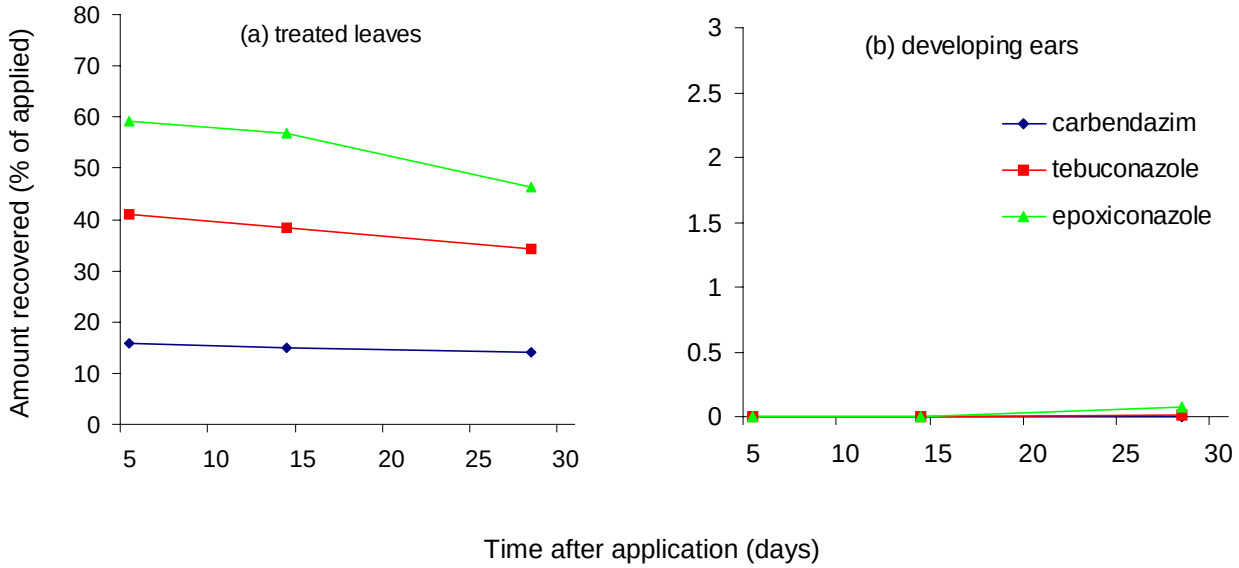

Fig.7. Distribution of radioactive fungicides applied to the flag leaves of wheat at GS 41, measured, 5, 14 and 28 days after treatment (a) treated leaves, (b) developing ears. (Note the different scales used for the two diagrams)

Radioactive parent fungicide in plants treated at GS 59 was measured in the glumes and in the ovaries, embryos or developing grain depending on growth stage but for simplicity the term ovary is used throughout. As in the painted leaves, the amount of fungicide in the painted glumes generally decreased with time but more steeply (Fig.8). With this method of application there was usually more fungicide in the ovaries than when the fungicides were painted onto the flag leaves but still <3% of the total applied. However, this fungicide can probably be attributed to direct movement into the floral cavity during application rather than movement through the plant. When the compounds were applied to collars placed directly below the ear, small amounts of each moved to the ear (never more than 0.18% of the total fungicide applied) this being mostly in the glumes, with a maximum of only 0.02% in the ovary. Compared with the collar treatment, more fungicide moved to the ovaries when application was by direct injection into the stem especially for tebuconazole and epoxiconazole; however never more than 2.6% reached the ovaries, this being less than a quarter of the amount in the glumes (up to 11.6%). The amount of fungicide recovered was consistently smallest for carbendazim, probably because this is the least soluble of the three fungicides and so the target concentration may not have been achieved.

DISCUSSION

None of the fungicides moved readily to the ear, with the small amount of fungicide that did move being concentrated in the glumes. Movement to the ear was increased when the compounds were injected directly into the stem but there was still little movement to the ovaries. All three of the radio-labelled fungicides are known to be systemic but tebuconazole and epoxiconazole are not among the most mobile of the azole group. Related compounds might have moved more readily to the ears but, as none of them gave consistently better control of ergot, it seems unlikely that the differences in movement would have been large. Limited movement of the three fungicides to the ears is consistent with the evidence that these compounds are predominantly transported in the xylem and not in the phloem. Given the usually small effects of fungicides from other chemical groups on ergot in the field experiments (see Chapter 2), it seems unlikely that any of them would move more readily to the ears especially as none of them are believed to be phloem-mobile.

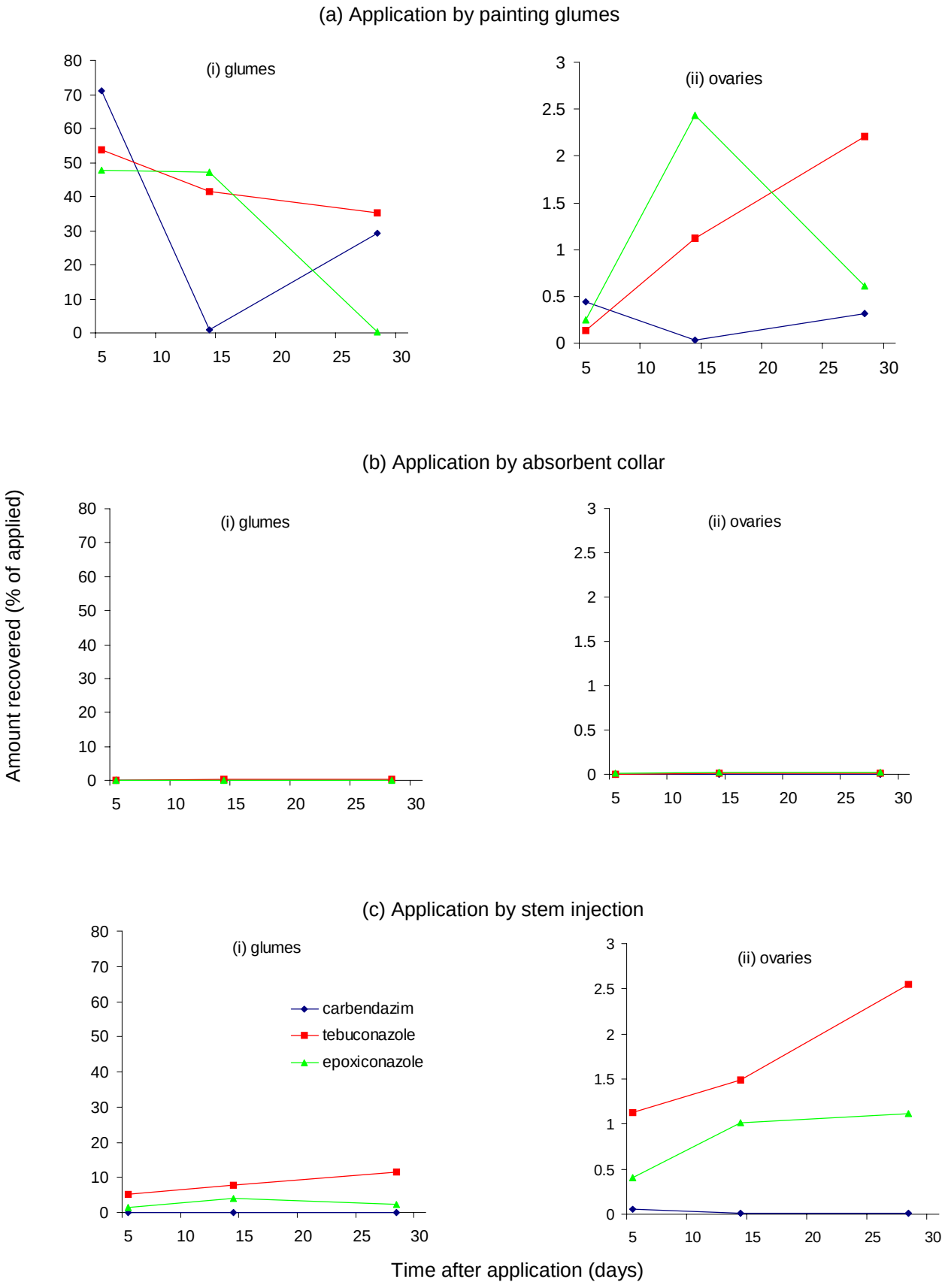

Fig.8. Distribution of radioactive fungicides, applied to wheat plants at GS 59 using different methods, measured 5, 14 and 28 days after treatment (i) glumes, (ii) ovaries. (Note the different scales used for glumes and ovaries)

GENERAL DISCUSSION

Ergot, caused by the fungus *Claviceps purpurea,* can affect all of the main temperate cereals but is more of a problem on wheat and rye than on barley, and seldom seen on oats (Dillon-Weston & Taylor, 1942). It is widely distributed throughout the United Kingdom but tends to occur spasmodically although some areas seem to be particularly prone to it (Long, 2000). It is important not for its effects on yield but because the ergots (sclerotia; dark resting structures that develop in place of the grain, and enable the fungus to survive over winter) contain toxins that are very toxic to humans and livestock (Bradley-Jones, 1965). Very low tolerances are, therefore, set for ergot contamination in traded grain (Anon., 1989), and, with increasing concerns about food safety and 'traceability', merchants and processors are likely to become ever more stringent in imposing these standards. Even if markets can be found for contaminated grain, it will certainly have a reduced value.

Reports received during the course of the project indicated that there were continuing problems with ergot during 1998-2000 both in wheat (particularly cv. Rialto) and in rye. There were also reports of ergot in triticale, where contaminated seed had been sown, and, in 2000, in barley. Volunteer cereals also created problems; in one case, volunteers of wheat cv. Rialto were the source of ergot contamination of a crop of winter barley. Samples of ergot from black grass, brome, ryegrass and wild oats were also received. Problems in wheat predominantly related to cv. Rialto, suggesting that variety evaluation should include measures of ergot susceptibility. This cultivar should be avoided on farms with ergot problems.

Control of the disease mainly relies on cultural methods but these are not completely effective. This is a problem because a partial reduction in ergot contamination is of limited value if the harvested grain still fails to meet the quality standards that are demanded.

Because the ears are at the top of the canopy and are only susceptible to infection over a relatively short period (i.e. during flowering), it might be expected that effective control should be possible using fungicide sprays applied at this time. Research in the 1970s showed that MBC fungicides applied at anthesis can have a useful effect but not consistently (Puranik & Mathre, 1971, Wood and Coley-Smith, 1980a). Since that time, many new fungicides (from a variety of chemical groups) have been developed. Yarham (1996) tested some of these in the laboratory but attempts to test them in the field were unsuccessful because the disease failed to develop. Apart from this few, if any, of the more-recently developed fungicides appear to have been tested systematically for their potential to control ergot caused by *C. purpurea.* Some fungicides have proved effective against ergot of sorghum caused by *C. africana* (Mena *et al*., 1998; Dahlberg *et al*., 1999) but the relevance of this to the control of ergot caused by *C. purpurea* is uncertain. Fungicidal control of ergot has been demonstrated in Kentucky bluegrass (Schultz *et al.*, 1993) but required much higher doses of azole fungicides than are currently approved for cereals in the UK. The research described in this report,

which focussed on wheat and rye, tested a wide range of commercial products and experimental materials for their activity against *C. purpurea*, investigated whether their performance in the field might be improved by alterations in how and when they are applied, and studied their movement to, and distribution within, the ears.

A total of 34 materials (commercial products, including a disease resistance activator (Ruess *et al*., 1996), and experimental compounds), representing all of the main chemical groups currently available, was obtained for testing. All of these were subjected to a laboratory screen with the intention of identifying compounds with inherent activity against the pathogen that could then be subjected to more detailed testing under field conditions. However, 23 of the 34 compounds tested showed evidence of at least some activity against *C. purpurea in vitro*, and many of them were similar to or more active than carbendazim (used as the standard reference material throughout the work described). It also became apparent during the course of the work, that agar plate tests are unsuitable for detecting and measuring the activity of at least some of the strobilurin fungicides (Jeremy Godwin, pers. comm.). Subsequent tests, described in Chapter 3, showed that two compounds from this group (azoxystrobin and kresoxim-methyl) do have fungicidal activity against the fungus, bringing the number with activity to more than two thirds of the total tested. Detecting activity against *C. purpurea* in so many of the fungicides that were tested was an encouraging result but it meant that it was not possible to use the results of the laboratory screen, with any confidence, to select a short-list of compounds for testing in the field. It was, therefore, decided that as many as possible of the 34 compounds, including those that showed no activity in the agar plate test, should also be screened in the field.

Three of the experimental compounds tested in the laboratory screen, were not tested in the field because they were supplied in very small amounts or in an unformulated state but there was no evidence that any of them had inherent activity against the fungus. All of the remaining 31 materials were tested in the field screen at Rothamsted (three experiments in 1998 or 1999; Chapter 2), and many were also tested in one or more of the other 11 field experiments done at Rothamsted or on commercial farms in 1998-2000 (Chapters 4 and 5). Disappointingly, however, and, in contrast to the results of the laboratory screen, there was little evidence of useful or consistent activity against the disease of any of the compounds tested. Effects, judged by comparison with unsprayed but inoculated control plots, were usually small and/or not significant. Larger effects whether or not significant, were seldom, if ever, big enough to be commercially useful, and there was as much evidence for increases in ergot following the application of fungicide sprays as for decreases. For most of the fungicides that were tested in two or more experiments there was little or no consistency in the effects that they had. Results from the field experiments, which generated large volumes or data, are used selectively below to illustrate the evidence on which these conclusions are based. More detailed information is presented in chapters 2 and, especially, 4 and 5.

In all of the experiments, plots were inoculated with the fungus (at about ear emergence) to ensure that the disease was present. It is possible that the inoculated ears themselves represented an unusually severe test of the effectiveness of fungicides but disease in ears affected by secondary spread probably provided a more realistic test and was measured whenever it occurred. Secondary spread was often greater in rye than in wheat probably reflecting the relative susceptibilities to the disease of the two species. Ergots also tended to be much larger in rye than in wheat, which meant that affected ears were often easy to see. This allowed total numbers of ears affected by secondary spread to be counted in individual plots in, for example, the winter rye experiments at Rothamsted in 1998 and 1999. The apparently contradictory results from these two experiments are typical; thus most of the fungicides tested in 1998 increased the number of affected ears whereas most of those tested in 1999 decreased the number. Differences in the fungicides that were tested and the rates at which they were applied in the two experiments mean that real differences in fungicide performance can not be entirely discounted but other evidence from these and other experiments suggests that alternative explanations are more likely.

Numbers of ears affected by secondary spread were counted in some other experiments but in many of them, and especially those on winter wheat, it was difficult to detect infected ears without dissecting them. In these experiments, uninoculated ears were usually sampled at random. Depending on the method used to process these samples, estimates of percentage ears affected were sometimes obtained but not invariably. However, average numbers of ergots, in inoculated ears, were obtained for all experiments. These data, therefore, provide a better basis for comparing the effects of fungicides in different experiments. Numbers of ergots may not, however, tell the whole story because the sizes to which they grow are very variable. Ergots removed from ears sampled from each of the Rothamsted experiments were, therefore, weighed as well as counted.

All of the fungicides that were tested in the field were included in one or other of the three screening experiments at Rothamsted (Experiments 2.1 - 2.3; Chapter 2). Although these experiments provided no evidence that any of the fungicides was outstandingly active against ergot, the results were used to inform the choice of fungicides for testing in subsequent field experiments. The choice was also influenced by the results of the glasshouse experiments (e.g. many of the azoles were apparently effective when painted directly on to ears in Experiment 3.2) and other, published, results on the same (e.g. Schulz *et al.*, 1993) or different (e.g. Jones, 1997) pathogens. As a consequence of this continual re-evaluation of evidence, from the experiments described in this report and other sources, some of the available fungicides were tested in many more experiments than others, and twelve of them were tested only in the field screen.

The first part of Table 6 summarises effects on numbers of ergots in inoculated ears of those fungicides that were tested in six or more of the field experiments. Three points are immediately apparent:-

(i) Effects of fungicides were very variable and, although not always significant, increases and decreases were almost equally common.

(ii) Effects of different fungicides in the same experiment were much more consistent than the effects of the same fungicides in different experiments.

(iii) Effects were much larger (in percentage terms but not necessarily in absolute terms) in the experiments on commercial farms (Experiment 5.1 - 5.6) than in the experiments at Rothamsted (Experiments 2.1 - 2.3 and 4.1 - 4.5) but were still mostly not significant.

Table 6. Summary of the effects on numbers of ergots (% increase or decrease compared to unsprayed, inoculated control plots) of the fungicides tested in six or more of the field experiments at Rothamsted or on commercial farms (significant effects in bold).

Fungicide	Experiment number													
	2.1	2.2	2.3	4.1	4.2	4.3	4.4	4.5	5.1	5.2	5.3	5.4	5.5	5.6
	Inoculated ears													
Carbendazim	+2	+2	-5	+3	-2			+3	**-27**	+10	-21	-82	+22	-3
Cyproconazole	+5			+3		-8	+2	+4	**-23**	-8	0	-40	+33	**-23**
Epoxiconazole	+15			+5		-5			**-35**	+7	+46	-80	+16	-18
Fluquinconazole			+11		-11		+2	-13			+19	0		
Flusilazole	+15				-12			-2			+1	-80	+35	-13
Tebuconazole	+10			+3		-7	+3	+10	**-54**	0	-30	+20	+2	-10
Fenpropimorph	+10					-4		-2			+34	-46	+40	-18
Azoxystrobin	**+20**			+7		+2		-2	**-37**	-3	+33	-80	+2	-5
Kresoxim-methyl + fenpropimorph	**+40**			+8					**-30**	+7	+8	-36		
Chlorothalonil	+18			+2	-8				**-31**	+8	+17	-19	+10	-3
	Ears infected by secondary spread													
Carbendazim	No secondary spread	Numbers not counted	-21	No secondary spread	+8			-28	-38	No secondary spread	+50	-82	+31	-22
Cyproconazole						-34	**+81**	-7	-46		+25	-40	+15	-31
Epoxiconazole						+39			-11		+75	-80	-1	-12
Fluquinconazole			-3		-7		**+63**	+2			+400	-100		
Flusilazole					-8			**-39**			+125	-80	+43	-6
Tebuconazole						0	+38	-28	-23		+50	+40	-2	-8
Fenpropimorph						+98		-18			+375	-56	+5	-3
Azoxystrobin						+111		-25	-17		+275	-80	-40	+3
Kresoxim-methyl + fenpropimorph									-37		+125	+60		
Chlorothalonil					-5				-20		+25	-30	-13	-12

Secondary spread did not occur in all experiments but where it did, and where the ergots were counted, the results obtained from the different experiments were broadly similar to those for inoculated ears except in Experiments 4.3 and 4.5. In the experiments at Rothamsted, the effects did, however, tend to be larger than those calculated for the inoculated ears but this difference was not apparent in the results from the experiments that were sited on commercial farms.

Effects of fungicides on weights of ergots in the inoculated ears (again expressed as percentages of the unsprayed but inoculated controls, Table 7) were generally much larger than the effects on numbers, and they almost invariably caused an increase. Weights of ergots in ears affected by secondary spread were more variable but, as with numbers, effects tended to be relatively consistent within experiments.

It might be suggested that some of the differences between the experiments at Rothamsted and those on commercial farms could have been a consequence of differences in the procedures adopted. On the commercial farms, for example, the fungicide sprays were always applied within 2-4 hours after inoculation whereas in the Rothamsted experiments (some of which also tested the effects of applying sprays before or after inoculation) the interval was never less than 2 days. However, while the short intervals could explain the relatively large decreases in ergot in Experiments 5.1 and 5.4, it is difficult to understand how they could explain the similarly large increases in Experiments 5.3 and 5.5. Indeed it is difficult to think of any explanation for the, sometimes large, increases in numbers of ergots following the application of fungicide sprays. Although such increases were not always significant they occurred too frequently to be dismissed lightly. Control by the fungicides of antagonistic microflora may be a possibility but, if this is the explanation, larger and more consistent differences between the fungicides, as a consequence of their different spectra of activity, might have been expected.

Increases in the weights of ergots per ear (measured only at Rothamsted, and presented in Chapter 4) partly reflect increases in the number. However, effects on weight were typically much larger than effects on number, and increases in weight were sometimes detected when numbers were unaffected or decreased. This implies that individual ergots were often larger in the fungicide-treated plots than in the untreated, which was confirmed by doing the appropriate calculations. Because ergots replace the grains, factors that affect grain filling might also be expected to affect the growth of ergots. As all of the most-frequently tested fungicides have proven activity against leaf diseases, this offers a plausible explanation for the effects of the fungicides on the size of ergots (but probably not their number). Similar increases in the total weights of ergots following the application of fungicides in experiments in Germany have also been reported (Werner *et al*., 1999). The experiments reported here suggest that effects were often larger where strobilurins had been applied compared to fungicides from other, chemically-unrelated, groups, perhaps reflecting the often larger yield responses that they cause (Bayles & Hilton, 2000; Bryson *et al*., 2000). Counter-intuitively, larger ergots might be advantageous if they were readily dislodged from ears before harvest or were easier to

remove from grain by sieving after harvest. In practice, however, the largest ergots often showed signs of splitting and tended to fragment and would, therefore, still be difficult to remove from grain. It is possible that such fragments have a reduced ability to survive in soil but this was not investigated. Fungicides might, conceivably, increase numbers of ergots per unit area or per ear if they caused increases in the number or size of ears, respectively, but in our experiments fungicides were usually applied much too late for such effects to be expected.

Table 7. Summary of the effects on weights of ergots (% increase or decrease compared to unsprayed, inoculated control plots) of the fungicides tested in six or more of the field experiments at Rothamsted (significant effects in bold).

			Experiment number					
Fungicide	2.1	2.2	2.3	4.1	4.2	4.3	4.4	4.5
				Inoculated ears				
Carbendazim	+6	-1	-13	+9	-7			-5
Cyproconazole	+12			+16		+14	+9	0
Epoxiconazole	**+83**			+14		+23		
Fluquinconazole			+12		+11		+2	-24
Flusilazole	**+36**				0			-9
Tebuconazole	**+30**			+14		+20	+7	+6
Fenpropimorph	+16					+23		+1
Azoxystrobin	**+69**			**+28**		**+48**		+14
Kresoxim-methyl + fenpropimorph	**+96**			+15				
Chlorothalonil	**+62**			+5	-8			
				Ears infected by secondary spread				
Carbendazim		+17	-11	-14	+7			-68
Cyproconazole				-1		-26	+17	-47
Epoxiconazole				0		+79		
Fluquinconazole			+6		+1		-32	-6
Flusilazole					+3			-68
Tebuconazole				+2		+8	+2	-43
Fenpropimorph						+84		-22
Azoxystrobin				+2		**+203**		-48
Kresoxim-methyl + fenpropimorph				+4				
Chlorothalonil				-3	-1			

No secondary spread

The results summarised in Table 6 and 7 clearly indicate that the field performance of the fungicides seldom matched the potential that they apparently displayed in the laboratory screen. This may be because they failed to reach the appropriate target. The more consistent differences between experiments than between

fungicides also suggests that the time at which the fungicides were applied (in relation to, for example, the growth stage of the crop or the weather) or, perhaps, the methods used to apply them in the different experiments were more important than inherent differences in the properties of the different fungicides.

Although it might be assumed that ergot is an easy target for fungicidal control (because the ears are at the top of the canopy and, therefore, very accessible), it is the ovaries that the fungus actually infects. The ovaries are, therefore, the real targets but for most of the time they are tightly enclosed and protected by the glumes. The only exception is when the florets are open, to allow cross-pollination. Carbendazim sprays applied at this time to male sterile barley were reported to be more effective than those applied when most of the florets were closed (Wood & Coley-Smith, 1980a). However, this does not really offer a practical and general solution to the problem because the flowers of cereals usually open for relatively short periods of time and do not do so simultaneously. This may, nevertheless, explain why the fungicides decreased ergot in some of the experiments but not in the majority of them. Pollen suppressant was used on wheat in the Suffolk experiments in 1998 and 2000, to encourage more open flowering, and this may have contributed to the relatively large decreases caused by the fungicides in Experiment 5.1. Ergot control may be more reliably achieved in male sterile cereals or their chemically-induced equivalents than in 'conventional' cereals and merits further study. Similarly, ergot may be controlled more effectively in barley than in wheat or rye.

Alternative routes by which fungicides might, theoretically, reach the ovaries are by percolation between the glumes, movement as vapour or movement systemically through the plant. Percolation of fungicides between the glumes is unlikely to occur to more than a limited extent, if at all, but it could be the explanation for the good control of ergot that was achieved when solutions containing relatively large concentrations of some active ingredients were painted onto the ears in Glasshouse Experiment 3.2. However, increasing the amounts of active ingredients deposited on the ears in the field experiments either by doubling their concentrations in the spray solutions (which has a relatively modest effect) or by using an electrostatic sprayer (which can give more than 20-fold increases in amounts of active ingredients deposited; Cayley *et al*., 1984) had no significant effects on control of ergot. Conceivably, the use of a paintbrush to apply the fungicides in the glasshouse experiment aided their penetration by disturbing the glumes.

Alternative ways to increase penetration of the ears that were considered, were applying sprays in larger than normal volumes of water (up to 880 l ha^{-1} instead of the usual 220 l ha^{-1} at Rothamsted, and up to 1000 l ha^{-1} in the experiments on commercial farms), applying simulated rain (2 mm applied as coarse droplets using a tractor mounted sprayer) immediately after spraying and adding an extra wetter to the spray solutions to reduce their surface tension. All of them had mostly negligible effects. The non-significant effect of adding 'Arma' to the sprays in Experiment 4.2 contrasts with results obtained in the USA where adding a different

wetter to the sprays ('Penaturf') was reported to increase the control of ergot in Kentucky bluegrass (Schultz *et al*., 1993). Other types of wetter may prove beneficial and need to be tested.

The fungicides tested differed in their vapour pressures and, theoretically, some had the potential to infiltrate the ear from external deposits. However, an experiment to test for vapour activity failed to demonstrate any effects on ergot. Unless the ovary is able to accumulate fungicide arriving as vapour, this does not seem to be a promising route as volatile products will be short-lived and prone to dispersal into the air rather than into the ear.

Although many of the fungicides tested are known to be systemic, they are predominantly transported in the xylem and not in the phloem. Predictably, therefore, movement to the ears is likely to be very limited and this was confirmed for the three radio-labeled fungicides that were tested in Chapter 6. The same experiment also showed that only a very small proportion of the total reached the ovaries perhaps because of the discontinuity that develops in the vascular tissue at the base of the ovary (O'Brien *et al*., 1985). However, Tanács *et al*. (1998) showed that when flusilazole or tebuconazole were applied at the 1-2 node stage of development (GS 31-32), small residues of the compounds were found in the flour but no residues could be detected when the same compounds were applied at a later growth stage (flag leaf developed; GS 41). This may indicate that fungicides move more readily to the developing ear than they do to ears that are fully-formed. Further support for this is apparently provided by evidence that better control of loose smut of wheat was obtained when triadimefon sprays were applied at stem extension (GS 31) than at ear emergence (GS 59) (Jones, 1997). Results from Glasshouse Experiment 3.4 were, apparently, consistent with these previously-published results in showing significant decreases in ergot when a range of fungicides was applied at early stem extension. Furthermore, the average effect of sprays applied at GS 31 was significantly greater than the average effects of sprays applied at GS 33. In contrast, however, applying fungicides at a similarly early growth stage to plots of spring wheat in the field had no significant effect compared to applying them at anthesis. It is possible that early-applied fungicides were more effective in the glasshouse because the development of the plants was compressed into a much shorter period of time.

The unavoidable conclusion from this work is that, although many fungicides with activity against the ergot fungus (and with different modes of action and physico-chemical properties) are now available, their effectiveness in controlling ergot in the field is limited, as it presumably always has been for carbendazim, by the problem of delivering them to the target (i.e. the ovaries). As was shown for carbendazim more than twenty years ago (Wood and Coley-Smith, 1980a), some control may be achieved if spraying happens to coincide with a significant proportion of the flowers being open but if it does not there will, it seems, be little or no useful effect on ergot. This probably explains the occasional, but always limited, control of ergot in some of the experiments described in this report but not in the majority. The results from the screening experiments may assist in the development of new seed treatments with activity against ergot as previously

shown for Baytan Flowable seed treatments (fuberidazole + triadimenol) (Shaw, 1986). Apart from this husbandry will probably continue to be the principal, if imperfect, option for reducing the impact of this disease for the foreseeable future.

Ergots on the soil surface germinate in late spring but fail to do so if buried more than about 10-15 cm deep. Ploughing can, therefore, help to reduce the amounts of primary inoculum to which crops are exposed (especially where the previous crop was known to be infected) but does not, of course, provide any protection against inoculum emanating from hedgerows and nearby fields. Any potential benefits of ploughing may also be negated if contaminated seed is sown.

Many grasses are also susceptible to the disease, and ergot can easily be found on wild grasses throughout the UK. Black grass growing as a weed in cereal crops is a particular problem because it tends to flower a little earlier than cereals. Spores arising from ergots on the soil surface (the primary inoculum) can, therefore, infect the blackgrass flowers and give rise to another crop of spores (secondary inoculum) that spread infection to the crop. (Secondary inoculum is produced on all hosts and can often be seen as a sticky exudate (honeydew) oozing out of the flowers). Good grass weed control is, therefore, an important element of any strategy to control ergot. Infection is sometimes particularly noticeable in late tillers, perhaps reflecting the fact that they reach a susceptible stage after the fungus has gone through a cycle of multiplication on the earlier-formed ears. In areas where there is a particular concern about ergot, it may be worth looking in the tramlines, where late tillers are often numerous, and if ergot is easily found to consider harvesting the tramlines separately from the bulk of the crop. There is not much evidence of true resistance to infection among cereal varieties but those with the closed-flowering habit tend to escape infection compared to varieties which are more open flowering. In winter wheat, cv. Rialto is a well-known example of the latter and should be avoided, especially in areas where previous experience suggests that the risks of ergot infection are high. If, despite taking these measures, harvested grain is known to be contaminated with ergot, it should be cleaned before sale to avoid rejection or costly penalties.

ACKNOWLEDGEMENTS

We wish to thank Mr Bill Angus and Mr Stuart Keavis of Nickersons Seeds for their assistance. Rothamsted farm staff are thanked for their help with the field experiments conducted at Rothamsted as are the farmers who provided other experimental sites and assistance with the project. We are grateful to agrochemical manufacturers for providing products for evaluation, particularly BASF plc and Bayer plc for the radio-labelled fungicides. We also thank B J Pye for providing, and assisting with the use of, the electrostatic sprayer, R H Bromilow for assistance with the experiment using radio-labelled fungicides, and A D Todd for help with statistical analyses. Valuable technical support was also provided by B V Symonds, A Naik, A Clark and Mr G Denton.

REFERENCES

AGRIOS, G.N. (1997). *Plant Pathology (4th Edition).* Academic Press, London.

ANON. (1989). New arrangements for moisture and ergot. *Agricultural Supply Industry* **19** (23), p.4.

ARNOLD, A.J., CAYLEY, G.R., DUNNE, Y., ETHERIDGE, P., GRIFFITHS, D.C., JENKYN, J.F., PHILLIPS, F.T., PYE, B.J., CSOTT, G.C. & WOODCOCK, C.M. (1984). Biological effectiveness of electrostatically charged rotary atomisers. II. Trials with cereals, 1982. *Annals of Applied Biology* **105**, 361-367.

BAYLIS, R.A. & HILTON, G.J. (2000). Variety as a factor in the response of cereals to strobilurins. *Proceedings of the 2000 Brighton Crop Protection Conference - Pests & Diseases* **2**, 731-738.

BRADLEY-JONES, J. (1965). Ergot and Ergotism. *National Agricultural Advisory Service Quarterly Review* **16**, 157-164.

BRYSON, R.J., LEANDRO, L. & JONES, D.R. (2000). The physiological effects of kresoxim-methyl on wheat leaf greenness and the implications for crop yield. *Proceedings of the 2000 Brighton Crop Protection Conference - Pests & Diseases* **2**, 739-746.

CAYLEY, G.R., ETHERIDGE, P., GRIFFITHS, D.C., PHILLIPS, F.T., PYE, B.J. & SCOTT, G.C. (1984). A review of the performance of electrostatically charged rotary atomisers on different crops. *Annals of Applied Biology* **105**, 379-386.

CAYLEY, G.R., ETHERIDGE, P., GOODCHILD, R.E., GRIFFITHS, D.C., HULME, P.J., LEWTHWAITE, R.J., PYE, B.J. & SCOTT, G.C. (1985). Review of the relationship between chemical deposits achieved with electrostatically charged rotary atomisers and their biological effects. BCPC Symposuim on Application and Biology, Monograph No. 28, pp.87-96.

DAHLBERG, J.A., PETERSON, G.L., ODVODY, G.N. & BONDE, M. (1999). Inhibition of germination and sporulation of *Claviceps africana* from honeydew encrusted sorghum with seed treatment fungicides. *Crop Protection* **18**, 235-238.

DILLON-WESTON, W.A.R. (1948). *Diseases of Cereals*. Longmans, Green and Co. London, pp.34-36.

DILLON-WESTON, W.A.R. & TAYLOR, R.E. (1942). Observations on ergot in cereal crops. *Journal of Agricultural Science* **32**, 457-466.

JONES, P. (1997). Control of loose smut (*Ustilago nuda* and *U. tritici*) infections in barley and wheat plants by foliar application of triadimefon. *Plant Pathology* **46**, 946-951.

LONG, E. (2000). Beware the ergot threat! *Arable Farming*, November 11th 2000, pp.6-8.

MANTLE, P.G., SHAW, S. & DOLING, D. A. (1977). Role of weed grasses in the etiology of ergot disease in wheat. *Annals of Applied Biology* **86**, 339-351.

MENA, H., FUENMAYOR, F., TEJERA, J., GEORGES, E. & JIMENEZ, R. (1998). Chemical control of sorghum ergot (*Claviceps africana*) in the field. *International Sorghum and Millets Newsletter* **39**, 105-106.

O'BRIEN, T.P., SAMMUT, M.E., LEE, J.W. & SMART, M.G. (1985). The vascular system of the wheat spikelet. *Australian Journal of Plant Physiology* **12**, 487-511.

PAVELEY, N.D., RENNIE, W.J., REEVES, J.C., WRAY, M.W., SLAWSON, D.D., CLARK, W.C., COCKERELL, V. & MITCHELL, A.G. (1996). HGCA Research Review No.34. *Cereal seed health and seed treatment strategies*. HGCA, London, 131pp.

PURANIK, S.B. & MATHRE, D.E. (1971). Biology and control of ergot on male sterile wheat and barley. *Phytopathology* **61**, 1075-1080.

RUESS, W., MUELLER, K., KNAUF-BEITER, G., KUNZ, W. & STAUB, T. (1996). Plant activator CGA 245704: an innovative approach for disease control in cereals and tobacco. *Proceedings of the 1996 Brighton Crop Protection Conference - Pests & Diseases* **1**, 53-60.

SHAW, S. (1986). Evaluation of Baytan for control of ergot contaminants in cereal seed. *Pflanzenschutz-Nachrichten Bayer* **39**, 48-70.

SCHULTZ, T.R., JOHNSTON, W.J., GOLOB, C.T. & MAGUIRE, J.D. (1993). Control of ergot in Kentucky bluegrass seed production using fungicides. *Plant Disease* **77**, 685-687.

TANÁCS, L., MATUZ, J., CSATLÓS, I. & GERÖ, L. (1998). Study of fungicide residues in the grain of fungicide-treated wheats. III. Flusilazole, carbendazime, tebuconazole and triadimefon-based fungicides. *Cereal Research Communications* **26** (3), 329-336.

TOMLIN, C. (ED) (1997). *The Pesticide Manual (11th Ed.).* The British Crop Protection Council, Farnham.

WERNER, S., HABERMEYER, J. & ZINKERNAGEL, V. (1999). Influence of modern fungicides on the baking quality of winter rye. In: *Modern Fungicides and Antifungal Compounds II*. Intercept Ltd., Hampshire, pp217-223.

WHITEHEAD, R. (ED) (1999). *The UK Pesticide Guide (Revised Edition).* CAB International & The British Crop Protection Council, Cambridge.

WOOD, G. & COLEY-SMITH J. R. (1980a). Observations on the prevalence and incidence of ergot disease in Great Britain with special reference to open-flowering male-sterile cereals. *Annals of Applied Biology* **95**, 41-46.

WOOD, G. & COLEY-SMITH, J.R. (1980b). The effectiveness of fungicides used against *Claviceps purpurea* attacking male-sterile barley in field trials. *Annals of Applied Biology* **96**, 169-175.

YARHAM, D. J. (1993). HGCA Research Review No.25. *Ergot of Cereals: a literature review and survey of incidence in traded grain.* HGCA, London, 96pp.

YARHAM, D. J. (1996). HGCA Project Report No.126. *Screening of fungicides for the control of ergot* (Claviceps purpurea). HGCA, London, 15pp.

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

APPENDIX A

Appendix Table A1. Diameter of inhibition zones (mm) on agar plates resulting from the application of different concentrations of fungicides, measured 8 days after inoculation.

Fungicide	Group in which tested	Concentration (mg a.i. l^{-1})					SED
		4	20	100	500	2500	
Carbendazim	1	0	6.6	18.0	23.0	26.4	0.76
Carbendazim	2	0	5.3	17.8	22.6	28.6	0.76
Carbendazim	3	0	6.4	23.8	28.4	31.5	0.76
Carbendazim	4	0	2.6	19.3	26.0	29.1	0.76
Carbendazim	5	0	2.4	16.0	27.1	32.0	0.76
Carbendazim	Mean	0	4.7	19.0	25.4	29.5	0.34
Bromuconazole	3	0	0	30.1	52.9	55.8	1.19
Cyproconazole	1	0	0	35.1	51.6	60.6	1.02
Difenconazole	1	0	0	19.4	29.8	31.3	0.70
Epoxiconazole	1	0	0	28.6	36.8	41.0	0.65
Flusilazole	1	0	0	33.5	46.4	49.3	0.61
Flutriafol	2	0	0	0	27.1	45.9	2.40
Fluquinconazole	4	0	0	8.6	11.1	16.8	1.15
Prochloraz	4	24.0	38.6	46.1	50.3	54.3	1.24
Propiconazole	1	0	0	33.9	49.5	56.5	0.71
Tebuconazole	1	0	0	28.4	45.4	45.9	0.81
Triadimenol	3	0	0	0	38.0	50.1	0.90
Triticonazole	5	0	0	14.6	35.5	37.4	1.06
Fenpropidin	2	0	0	0	0	16.1	-
Fenpropimorph	3	0	0	0	15.5	32.6	1.00
Tridemorph	5	12.9	21.4	53.6	79.0	79.0	2.41
Azoxystrobin	3	0	0	0	0	0	-
Kresoxim-methyl	3	0	0	0	0	0	-
Trifloxystrobin	5	0	0	0	0	0	-
Cyprodinil	2	0	0	3.5	10.6	11.1	0.61
Pyrimethanil	4	0	0	0	0	0	-
Chlorothalonil	2	12.4	21.5	24.0	23.8	24.3	0.81
Fludioxinil	5	0	25.3	30.9	30.9	36.6	0.88
Iprodione	2	0	0	0	1.6	4.3	0.36
Mancozeb	2	0	0	3.3	15.6	27.4	1.01
Quinoxyfen	5	0	0	0	0	0	-
Spiroxamine	5	0	0	0	0	20.3	-
Silthiofam	5	0	0	0	0	0	-
Exp 10623A	3	0	0	0	0	0	-
Exp 10830A	3	8.6	23.9	25.1	27.3	27.0	0.42
Exp 10831A	3	0	0	0	0	0	-
Mon 21250	4	0	0	0	0	0	-
PST 2128	4	0	0	0	0	0	-
PMQ 4534	4	0	0	0	0	0	-

Appendix Table A2. Diameter of inhibition zones (mm) on agar plates resulting from the application of different concentrations of fungicides, measured 13 days after inoculation.

Fungicide	Group in which tested	Concentration (mg a.i. l^{-1})					SED
		4	20	100	500	2500	
Carbendazim	1	0	7.1	20.4	24.6	26.9	0.71
Carbendazim	2	0	4.9	18.3	25.1	29.3	0.71
Carbendazim	3	0	4.8	20.5	29.1	30.0	0.71
Carbendazim	4	0	1.5	17.4	24.3	30.3	0.71
Carbendazim	5	0	1.8	14.3	26.4	31.4	0.71
Carbendazim	Mean	0	4.0	18.2	25.7	29.6	0.32
Bromuconazole	3	0	0	16.0	51.4	54.0	1.11
Cyproconazole	1	0	0	0	51.5	61.3	1.24
Difenconazole	1	0	0	0	30.5	31.5	1.32
Epoxiconazole	1	0	0	0	37.3	41.0	0.84
Flusilazole	1	0	0	25.8	46.8	50.1	0.56
Flutriafol	2	0	0	0	0	41.6	-
Fluquinconazole	4	0	0	6.9	8.1	14.8	0.97
Prochloraz	4	20.6	35.0	42.3	47.1	52.5	1.06
Propiconazole	1	0	0	0	48.8	55.3	0.69
Tebuconazole	1	0	0	0	44.8	46.4	0.82
Triadimenol	3	0	0	0	29.3	47.1	1.04
Triticonazole	5	0	0	0	34.5	35.3	0.37
Fenpropidin	2	0	0	0	0	0	-
Fenpropimorph	3	0	0	0	11.0	30.5	1.20
Tridemorph	5	0	0	0	0	79.0	-
Azoxystrobin	3	0	0	0	0	0	-
Kresoxim-methyl	3	0	0	0	0	0	-
Trifloxystrobin	5	0	0	0	0	0	-
Cyprodinil	2	0	0	3.3	9.6	10.8	0.59
Pyrimethanil	4	0	0	0	0	0	-
Chlorothalonil	2	11.5	19.9	21.8	22.1	22.3	0.91
Fludioxinil	5	0	18.6	29.6	29.9	33.8	1.14
Iprodione	2	0	0	0	2.5	4.4	0.38
Mancozeb	2	0	0	3.0	13.5	25.0	0.76
Quinoxyfen	5	0	0	0	0	0	-
Spiroxamine	5	0	0	0	0	19.1	-
Silthiofam	5	0	0	0	0	0	-
Exp 10623A	3	0	0	0	0	0	-
Exp 10830A	3	9.0	23.0	25.5	25.8	27.0	0.63
Exp 10831A	3	0	0	0	0	0	-
Mon 21250	4	0	0	0	0	0	-
PST 2128	4	0	0	0	0	0	-
PMQ 4534	4	0	0	0	0	0	-

APPENDIX B

Experiment 2.1. Effects of different fungicides on ergot infection in winter wheat at Rothamsted in 1998.

Rothamsted code	98/R/WW/10
Site	Bones Close
Soil type	Flinty silty clay loam
Previous crop	Winter rape
Cultivar	Riband (treated with Sibutol)
Date sown	22-10-97
Date first spray applied	1-6-98
Date inoculated	3-6-98
Date second spray applied	8-6-98
Date sampled	27-7-98

Appendix Table B1. Effects of fungicides in Experiment 2.1.

	Inoculated ears	
Fungicide	Mean No. of ergots per ear	Mean weight per ergot (mg)
Untreated	4.0	7.3
Carbendazim	4.1	7.6
Cyproconazole	4.2	7.9
Difenconazole	4.5	9.4
Epoxiconazole	4.6	11.8
Flusilazole	4.6	8.7
Flutriafol	4.3	8.0
Propiconazole	4.7	8.6
Tebuconazole	4.4	8.8
Triadimenol	4.4	7.6
Fenpropidin	4.8	7.9
Fenpropimorph	4.4	7.9
Tridemorph	3.7	7.4
Azoxystrobin	4.8	10.5
Kresoxim-methyl + fenpropimorph	5.6	10.3
Cyprodinil	4.3	7.6
Chlorothalonil	4.7	10.3
Iprodione	4.3	8.5
Mancozeb	4.8	8.3
SED (DF=117)	0.33 **	0.53 ***

** indicates P<0.01 *** indicates P<0.001

Experiment 2.2. Effects of different fungicides on ergot infection in spring wheat at Rothamsted in 1998.

Rothamsted code	98/R/WS/1
Site	Bones Close
Soil type	Flinty silty clay loam
Previous crop	Winter rape
Cultivar	Chablis (treated with Sibutol)
Date sown	6-2-98
Date first spray applied	16-6-98
Date inoculated	18-6-98
Date second spray applied	24-6-98
Date sampled	24-8-98

Appendix Table B2. Effects of fungicides in Experiment 2.2.

Fungicide	Inoculated ears		Secondarily infected ears
	Mean No. of ergots per ear	Mean weight per ergot (mg)	Mean weight of ergots per ear (mg)
Untreated	8.1	27.5	92.2
Carbendazim	8.3	26.6	107.5
Bromuconazole	7.7	27.2	93.4
Kresoxim-methyl	8.4	32.2	102.0
Quinoxyfen	8.1	27.3	98.4
Silthiofam	7.3	25.6	102.2
Exp. 10623A	8.0	30.9	101.2
Exp. 10830A	8.1	28.0	89.8
Exp. 10831A	8.3	28.4	106.4
A9180A [1]	8.5	28.6	109.0
SED (DF=58)	0.43	1.60**	7.55

[1] A9180A was an experimental disease resistance activator

** indicates P<0.01

Experiment 2.3. Effects of different fungicides on ergot infection in spring wheat at Rothamsted in 1999.

Rothamsted code	99/R/WS/1
Site	Sawyers II
Soil type	Flinty silty clay loam
Previous crop	Winter wheat
Cultivar	Chablis (treated with Sibutol)
Date sown	17-2-99
Date first spray applied	14-5-99
Date inoculated	16-5-99
Date second spray applied	18-5-99
Date sampled	2-8-99

Appendix Table B3. Effects of fungicides in Experiment 2.3.

	Inoculated ears		Secondarily infected ears
Fungicide	Mean No. of ergots per ear	Mean weight per ergot (mg)	Mean weight of ergots per ear (mg)
Untreated	3.8	23.16	3.59
Carbendazim	3.6	21.17	3.20
Fluquinconazole	4.2	24.03	3.28
Prochloraz	4.0	25.66	1.65
Triticonazole	4.6	27.37	2.05
Pyrimethanil	4.1	24.34	3.15
Fludioxinil	3.6	25.36	2.27
Spiroxamine	3.9	23.44	3.80
Trifloxystrobin	4.5	25.42	1.80
SED (DF=60)	0.32	2.018	1.541

Appendix Table B4. Details of the fungicides tested, and their rates of application, in field Experiments 2.1, 2.2 and 2.3.

Fungicide	Product name	Formulation	Application rate per hectare	
			a.i. (g)	Product
Carbendazim	Bavisitin DF	50% w/w WG	275	0.5 kg
Bromuconazole	Granit	200 g/l SC	250	1.0 l
Cyproconazole	Alto 100	100 g/l SL	80	0.8 l
Difenconazole	Plover	250 g/l EC	75	0.3 l
Epoxiconazole	Opus	125 g/l SC	125	1.0 l
Flusilazole	Sanction	400 g/l EC	160	0.4 l
Flutriafol	Pointer	125 g/l SC	125	1.0 l
Fluquinconazole	Flamenco	100 g/l EC	150	1.5 l
Prochloraz	Sportak	450 g/l EC	450	1.0 l
Propiconazole	Radar	250 g/l EC	125	0.5 l
Tebuconazole	Folicur	250 g/l EW	250	1.0 l
Triadimenol	Bayfidan	250 g/l EC	125	0.5 l
Fenpropidin	Mallard	750 g/l EC	750	1.0 l
Fenpropimorph	Corbel	750 g/l EC	750	1.0 l
Tridemorph	Calixin	750 g/l EC	525	0.7 l
Triticonazole	Exp 80441B	250 g/l EC	250	1.0 l
Azoxystrobin	Amistar	250 g/l SC	250	1.0 l
Kresoxim-methyl + fenpropimorph	Ensign	150 + 300 g/l SE	105 + 210	0.7 l
Kresoxim-methyl	Bas 490 02F	50% w/w	125	0.25 kg
Trifloxystrobin	Twist	250 g/l EC	250	1.0 l
Cyprodinil	Unix	750 g/l WDG	750	1.0 kg
Pyrimethanil	Scala	400 g/l SC	800	2.0 l
Chlorothalonil	Bravo	500 g/l SC	1000	2.0 l
Fludioxinil	Beret Gold	25 g/l FS	25	1.0 l
Iprodione	Rovral flo	255 g/l SC	510	2.0 l
Mancozeb	Dithane 945	800 g/l WP	1600	2.0 kg
Quinoxyfen	Fortress	500 g/l SC	150	0.3 l
Spiroxamine	Torch	500 g/l EW	750	1.5 l
Silthiofam	Mon 65507	12.5% w/w	6.25	50 g
Exp 10623A	-	500 g/l EC	150	0.3 l
Exp 10830A	-	-	-	1.0 l
Exp 10831A	-	-	-	0.8 l
A9180A	-	505 w/w	30	60 g

APPENDIX C

Dates when treatments were applied and the crops sampled, and the most relevant husbandry details of the experiments described in Chapter 4 are given below. No fungicide sprays apart from the treatments tested, were applied to any of the crops. Details of previous cropping before the immediately preceding year, and of the fertilisers and other pesticides etc. that were applied to the crops are omitted. However, all of the experiments described in Chapter 4 were done on the Rothamsted experimental farm by Rothamsted farm staff from whom this information is readily available.

Experiment 4.1 Winter rye 1998

Rothamsted code	98/R/RW/1
Site	Bones Close
Soil type	Flinty silty clay loam
Previous crop	Winter rape
Cultivar	Esprit
Date sown	22-10-97
Date first spray applied	13-5-98
Date inoculated	15-5-98
Date second spray applied	18-5-98
Date sampled	1-7-98

Experiment 4.2 Winter rye 1999

Rothamsted code	99/R/RW/1
Site	Sawyers II
Soil type	Flinty silty clay loam
Previous crop	Winter wheat
Cultivar	Esprit
Date sown	20-10-98
Date spray applied	19-5-99
Date inoculated	21-5-99
Date sampled	5-7-99

Experiment 4.3 Winter wheat 1999

Rothamsted code	99/R/WW/2
Site	Sawyers II
Soil type	Flinty silty clay loam
Previous crop	Winter wheat
Cultivar	Riband
Date sown	16-10-98
Date spray applied	1-6-99
Date inoculated	3-6-99
Date sampled	19-7-99

Experiment 4.4 Winter wheat 2000

Rothamsted code	00/R/WW/2
Site	Sawyers II
Soil type	Flinty silty clay loam
Previous crop	Set-aside
Cultivar	Riband
Date sown	15-10-99
Date spray applied	7-6-00
Date inoculated	9-6-00
Date sampled	2-7-00

Experiment 4.5 Spring wheat 2000

Rothamsted code	00/R/WS/1
Site	Great Knott III
Soil type	Flinty silty clay loam
Previous crop	Winter wheat
Cultivar	Chablis
Date sown	8-3-00
Date GS 31 spray applied	31-5-00
Date GS 59 spray applied	23-6-00
Date inoculated	26-6-00
Date sampled	22-8-00

APPENDIX D

SITE: WINTER WHEAT 1998

Crop details

Location : Woolpit, Suffolk

Field name:	Charity Field
Soil series:	Newport 3
Soil type:	Sandy loam
Drainage:	Good
Soil analysis: (5/8/99)	pH 8.0
	P 95 mg l^{-1} (Index 5)
	K 145 mg l^{-1} (Index 2)
	Mg 50 mg l^{-1} (Index 2)
Previous cropping:	1999 Winter Wheat
	1998 Winter Wheat
	1997 Peas
	1996 Linseed
	1995 Winter Wheat
Previous residues disposal:	Ploughed
Cultivations:	Ploughed
Cultivar:	Rialto - Panoctine treated
Sowing date:	18.11.99
Seed number:	270 seeds m^{-2} (Approx. 150 kg ha^{-1})
Fertiliser:	Seed bed (kg ha^{-1}) None
	Top dressing (kg N ha^{-1}) 86 on 14 April 2000

	Product	Rate	Date applied
Trace elements:	Nil		
Herbicide:	Mentrol Bio	1.5 l ha^{-1}	14/04/00
	Duplosan	1.9 l ha^{-1}	14/04/00
Insecticide:	-		
Molluscicide:	Nil		
Plant growth regulator:	Nil		
Pollen suppressant:	-	-	22/05/00

SITE: WINTER RYE 1998

Location : Poyntington, Dorset

Crop details

Soil series:	South Petherton	
Soil type:	Clay loam	
Drainage:	Well drained	
Soil Analysis:	pH	6.2
	P index	1
	K index	1
	Mg index	1
Previous cropping:	1997	Winter rye
	1996	Winter wheat
	1995	Set-aside
	1994	Winter barley
Previous crop residue:	Baled and removed	
Cultivations:	Ploughed; tine cultivated/crumbler rolled	
Cultivar:	Esprite	
Sowing date:	27 September 1997	
Seed rate:	93 kg/ha	
Fertiliser (kg ha^{-1}):	basal (N:P_2O_5:K_2O):	0:0:125
	top dressings (N):	144
	DFM 300 (magnesium):	1.0 kg ha^{-1}
Herbicides:	Ardent (2.0 l ha^{-1}) - 13 October 1997	
	Grasp (1.4 l ha^{-1}) - 8 May 1998	
	+ Output (1.0 l ha^{-1})	
Insecticides:	Sumi-Alpha (0.15 l ha^{-1}) - 12 November 1997	
Growth regulator:	Meteor (2.0 l ha^{-1}) - 2 April 1998	

SITE: WINTER WHEAT 1999

Crop details

Location : Woolpit, Suffolk

Field name:	Charity Field
Soil series:	Newport 3
Soil type:	Sandy loam
Drainage:	Good

Soil analysis: (7/99)

pH	8.0
P	59 mg l^{-1} (Index 4)
K	177 mg l^{-1} (Index 2)
Mg	74 mg l^{-1} (Index 2)
O/Matter	1.98%

Previous cropping:

1998	Winter Wheat
1997	Peas
1996	Linseed
1995	Winter Wheat

Previous residues disposal: Ploughed

Cultivations: Ploughed

Cultivar: Rialto - Panoctine treated

Sowing date: 06.11.98

Seed number: 270 seeds m^{-2} (Approx. 150 kg ha^{-1})

Fertiliser:

Seed bed (kg ha^{-1}) None

Top dressing (kg N ha^{-1}) 68 on 19 April 1999

	Product	Rate	Date applied
Trace elements:	Nil		
Herbicide:	Ally	30g ha^{-1} + 1.0	16/11/98
	Duplosan	l ha^{-1}	
	Swipe	4.5 l ha^{-1}	27.03.99
Insecticide:	Aphox	280 g ha^{-1}	12.07.99
Molluscicide:	Nil		
Plant growth regulator:	Nil		
Pollen suppressant:	Nil		

SITE: WINTER RYE 1999

Crop details

Location : Trent, Sherborne, Dorset

Soil series:	Curtisden	
Soil type:	Silty clay loam	
Drainage:	Well drained	
Soil Analysis:	pH	7.0
	P index	2
	K index	0
	Mg index	1
Previous cropping:	1998	Winter rye
	1997	Grass
	1996	Grass
	1995	Grass
Previous crop residue:	Baled and removed	
Cultivation's:	Ploughed; pressed; powerharrowed x 2; drilled	
Cultivar:	Esprite	
Sowing date:	5 October 1998	
Seed rate:	115 kg ha^{-1}	
Fertiliser (kg ha^{-1}):	basal (N:P_2O_5:K_2O):	0:60:60
	top dressings (N):	110
	Mantrac (magnesium):	3.5 kg ha^{-1}
Herbicides:	Ardent (2.5 l ha^{-1}) - 7 December 1998	
Insecticides:	Sumi-Alpha (0.15 l ha^{-1}) - 7 December 1998	
Growth regulator:	Meteor (2.5 l ha^{-1}) - 26 March 1999	

SITE: WINTER WHEAT 2000

Crop details

Location : Woolpit, Suffolk

Field name:	Charity Field
Soil series:	Newport 3
Soil type:	Sandy loam
Drainage:	Good

Soil analysis: (5/8/99)

pH	8.0
P	95 mg l^{-1} (Index 5)
K	145 mg l^{-1} (Index 2)
Mg	50 mg l^{-1} (Index 2)

Previous cropping:

1999	Winter Wheat
1998	Winter Wheat
1997	Peas
1996	Linseed
1995	Winter Wheat

Previous residues disposal: Ploughed

Cultivations: Ploughed

Cultivar: Rialto - Panoctine treated

Sowing date: 18.11.99

Seed number: 270 seeds m^{-2} (Approx. 150 kg ha^{-1})

Fertiliser:

Seed bed (kg ha^{-1}) None

Top dressing (kg N ha^{-1}) 86 on 14 April 2000

	Product	Rate	Date applied
Trace elements:	Nil		
Herbicide:	Mentrol Bio	1.5 l ha^{-1}	14/04/00
	Duplosan	1.9 l ha^{-1}	14/04/00
Insecticide:	-		
Molluscicide:	Nil		
Plant growth regulator:	Nil		
Pollen suppressant:		Full	22/05/00

SITE: WINTER RYE 2000

Crop details

Location : Trent, Sherborne, Dorset

Soil series:	Curtisden	
Soil type:	Silty clay loam	
Drainage:	Well drained	
Soil Analysis:	pH	7.0
	P index	2
	K index	0
	Mg index	1
Previous cropping:	1999	Winter rye
	1998	Winter rye
	1997	Grass
	1996	Grass
Previous crop residue:	Baled and removed	
Cultivations:	Ploughed; rolled; power harrowed; drilled	
Cultivar:	Esprite	
Sowing date:	21 October 1999	
Seed rate:	100 kg ha^{-1}	
Fertiliser (kg ha^{-1}):	basal (N:P_2O_5:K_2O):	0:60:60
	top dressings (N):	84 3 April
		85 4 May
Herbicides:	None	
Insecticides:	None	
Growth regulator:	None	

APPENDIX D
CONDITIONS AT SPRAYING

Experiment 5.1 Winter wheat 1998

Spray applications and conditions at spraying

Target GS	Actual *GS*	Date	Weather
GS 52	GS 52	28.05.98	Cloudy Occasional drizzle 19^0 C Wind SW 1-2 mph
GS 59	GS 59	05.06.98	Cloudy, humid 18.5^0 C Wind E 3-7 mph

Spray application equipment

Sprayer: OPS CO_2 powered knapsack sprayer
Nozzles: 50 02 F110
Water volume: 220 l ha^{-1}
Pressure: 200 kPa

Experiment 5.2 Winter rye 1998

Fungicide application details

Target GS	Actual GS	Date	Weather
GS 52	GS 51-57	8.05.98	Cloudy; 16° C; 90% RH; wind speed - 4 kph. Crop foliage was damp as a result of a rain shower occurring immediately before spraying commenced; a Further rain shower, just after spraying finished, again Wetted the foliage but was not sufficient to cause run-off.
GS 59	GS 59	15.05.98	Sunny; 22.5° C; 75% RH; wind - calm; foliage dry

Spray application equipment

Sprayer: OPS CO_2 powered knapsack sprayer
Nozzles: Teejet XR11003
Water volume: 250 l ha^{-1}
Pressure: 240 kPa

Experiment 5.3 Winter wheat 1999

Spray applications and conditions at spraying

Target GS	Actual GS	Date	Weather
GS 31 Mark out plots	GS 31-32	23.04.99	Turned to rain, plots burnt out only
GS 32	GS32	28.04.99	Sunny and warm Wind 3-5 kph Crop dry
GS 52	GS 51	25.05.99	Sunny and warm Wind NW breezy with gusts to 12 kph, slight drift of spray Crop dry
GS 59	GS 59	01.06.99	Warm and dry after thunderstorms on 30 May Wind W 3-5 kph Crop dry

Spray application equipment

Sprayer: OPS CO_2 powered knapsack sprayer
Nozzles: Orange ff 120
Water volume: 250 l ha^{-1}
Pressure: 200 kPa

Experiment 5.4 Winter rye 1999

Spray applications and conditions at spraying

Target GS	Actual GS	Date	Weather
GS 32	GS32-37	27.04.99	Sunny; 19° C; 65% RH; wind speed 5-13 kph. Crop dry
GS 52	GS 49-57	07.05.99	Overcast; 17.5° C; 85% RH; wind speed 2-7 kph. Crop foliage was wet after a rain shower.
GS 59	GS 59	14.05.99	Overcast. Immediately after spraying there was a torrential downpour which must have limited the effectiveness of the treatments.

Spray application equipment

Sprayer: OPS CO_2 powered knapsack sprayer
Nozzles: Teejet XR11003
Water volume: 250 l ha^{-1}
Pressure: 240 kPa

Experiment 5.5 Winter wheat 2000

Spray applications and conditions at spraying

Target GS	Actual GS	Date	Weather
GS 31 Mark out plots	GS 30	19.04.00	Plots burnt out only Breezy, cool and sunny, traces of rain
GS 32	GS32	05.05.00	Sunny and warm Wind 3-5 kph, some stronger gusts, slight spray drift. Crop dry
GS 55	GS 55	15.06.00	Sunny and warm Wind with gusts to <1.5 kph, slight drift of spray Crop dry

Spray application equipment

Sprayer: OPS CO_2 powered knapsack sprayer
Nozzles: 03 F110
Water volume: 1000 l ha^{-1}
Pressure: 200 kPa

Experiment 5.6 Winter rye 2000

Target GS	Actual GS	Date	Weather
GS 31-32	GS31	26.04.00	Dry but cloudy; 12°C; 65% RH; wind speed 7 km/h. Crop foliage dry, but soil very wet.
GS 55	GS 55-59	17.05.00	Overcast but dry after showers; 14.6° C; 62.8% RH; wind speed 10.4 kph.

Spray application equipment

Sprayer: OPS CO_2 powered knapsack sprayer
Nozzles: TeeJet® XR11003VS
Water volume: 1000 l ha^{-1}
Pressure: 260 kPa