SESSION 9B

ALTERNATIVES TO MERCURY FOR DISEASE CONTROL

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THE FORGOTTEN DISEASES: WHY WE SHOULD REMEMBER THEM

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ABSTRACT

Evidence is reviewed which shows the organomercury seed-treatments to have been so effective that they reduced to insignificance a number of previously very damaging cereal diseases. Recent experience has shown, however, that these diseases are by no means extinct in the UK and that they could rapidly build up again were the loss of the cheap organomercury materials to dissuade farmers from using seed-treatments on their cereal crops.

INTRODUCTION

In the early 1920s bunt (Tilletia caries) was so common a disease in UK wheat crops that in one year (1921) 22% of the samples examined at the Official Seed Testing Station at Cambridge contained bunt balls (Marshall, 1960). Covered smut (Ustilago hordei) of barley was causing losses of up to 25% in occasional crops (Cotton, 1922), barley leaf stripe (Drechslera graminea) was responsible for serious losses in many seasons (Cotton, 1922; Pethybridge, 1926) and in Northern Ireland the smuts (U. hordei and U. avenae) and leaf spot (Pyrenophora avenae were amongst the factors implicated in causing frequent depreciations in the yield of the oat crop (Muskett & Cairns, 1933).

Within little more than a generation all of these diseases had become so rare in Britain that their occasional occurrence was a cause for mild excitement amongst plant pathologists. The pathogens concerned had fallen victims to one of the most effective disease control strategies in the history of crop pathology: the almost universal use of seed treatments so cheap and so effective that their routine use was accepted without question by the majority of farmers.

Of course, seed treatments active against bunt had been around well before the 1920s. Steeping seed in brine and then drying it with lime had been practised since the days of Jethro Tull (Woolman and Humphrey, 1924), and the use of arsenic for this purpose in the early 1800s had led to the first recorded instance of bird deaths caused by agrochemicals (Young, 1804). Treatments based on copper compounds provided late 19th and early 20th century farmers with more effective control of bunt, and treatment with formalin offered, for the first time, an effective method of combatting the oat smuts and covered smut of barley. It was, however, with the development of the organomercury compounds that the real breakthrough came.

interest but which was seldom, if ever, encountered in farm practice. In the 1980s, however, ADAS advisers began to record the disease more and more frequently. Some of these cases were in seed stocks that had been home-saved for up to six years without treatment but in other cases the seed had been home-saved for a much shorter period.

In attempting to explain how these cases could arise, two facts need to be remembered:-

(i) although it had a good activity against bunt, organomercury could not be relied upon invariably to eliminate the disease,

(ii) any bunt surviving treatment could build up extremely rapidly if seed from the stock were saved and used for a few years without treatment.

The first point may be illustrated by data, presented in Table 1, from trials carried out by ADAS in association with the Morley Research Station (Jones, unpublished).

TABLE 1. Effect of organomercury seed-treatment on bunt of wheat

Seed stock	Site	% bunted ears		Square root transformation			
		Control	Mercury	Control	Mercury	SED	df
1	Trumpington	7.1	0.8	2.5	0.5	0.54	15
	Morley	7.7	0.0	2.4	0.0	0.35	25
2	Trumpington	22.2	1.5	4.4	0.8	0.82	15
	Shelford	72.8	5.7	8.5	2.3	0.27	25

It will be noted that even in the case of the less severely infected stock, control was not complete at the Trumpington site. The Shelford and Trumpington sites were only about a mile apart and were on the same soil type. The differences in disease severity at these two sites appeared to be associated with a difference in sowing date: early December at Trumpington, late December at Shelford. Seedlings are susceptible to infection only until the emergence of the first leaf and it is likely that those in the Trumpington trial grew through this stage more rapidly than those in the later sown plots at Shelford. Differences in soil moisture content may also have influenced susceptibility to infection at the two sites. Whatever the cause of the differences, the trials illustrate very clearly that organomercury could not be relied upon to give 100% control of the disease, especially when conditions were very favourable for infection. Gaudet et al. (1989) have shown similar site to site variations in the effectiveness of carboxin in controlling bunt.

Of course, seed would not normally be saved from crops as badly infected as those from which the seed used in the trials was taken. In farm practice, seed crops would never have more than the occasional infected ear in them and the very low levels which would survive the seed treatment would be of little significance in the first few years of home-saving. Once a critical level of infection was reached, however,

Seed treatment with mercury compounds (initially mercuric chloride) was first advocated in Germany as a protection against soil-borne Fusarium (Hiltner, 1910, cited by Woolman & Humphrey, 1924). It was soon realised, however, that these materials were more effective than either copper or formalin against the seed-borne diseases of cereals.

The first organomercury seed treatment to be developed commercially was mercury chlorophenate, which was being sold in Germany, under the trade name 'Uspulun', as early as 1912. Other mercury based products soon followed, including 'Ceresan' (developed by I.G. Farbenindustrie AG) which was introduced into the UK in the late 1920s.

The one gap in the new seed treatments' spectrum of activity was Ustilago nuda (the loose smut of wheat and barley) which, lying hidden within the embryo itself, remained inviolable to fungicides until the advent of the systemic material carboxin in the 1960s (Ventura et al. 1968). Carboxin based materials came as a godsend to the seed industry as they provided an effective alternative to the laborious hot water steeps previously used for the control of loose smut. They were, however, considerably more expensive than the organomercury compounds and offered no real challenge to the use of these materials on grain crops.

The first real chink in organomercury's armour appeared in the late 1960s when resistant strains of Pyrenophora avenae began to appear in oats (Noble et al., 1966). At the time it was regarded as remarkable that any pathogen could overcome a poison as broadly based as mercury. P. avenae proved not to be unique in this respect, however, and in the late 1980s organomercury resistance also began to appear in the related pathogen P.graminea on spring barley (Jones et al., 1989)

It was, however, environmental concerns rather than resistance which finally led to the demise of organomercury fungicides. The EEC Council Directive 79/117/EEC (Anon, 1979) banned the use of all mercury products in agriculture and horticulture though in the UK and Ireland a derogation allowed their limited use (including their use as cereal seed treatments) until affective alternatives could be found. That derogation has now been rescinded and in autumn 1992 UK farmers have faced their first seed-time since the 1930s without these singularly cheap and effective compounds.

A range of effective alternatives is, of course, available but they are considerably more expensive than organomercury. Not surprisingly, farmers facing increases of at least 300% in their seed treatment bills are beginning to question the need for routine seed treatment. Organomercury has been so effective for so long that comparatively few present day farmers have ever seen the disease it controlled - they have justifiably been called the 'forgotten diseases'. Surely by now levels of inoculum must be so low that it will take years for them to build up to significant proportions even if we do not use seed treatments. In evaluating this argument we need to consider not only the past history of the 'forgotten diseases' but also our recent experience of their incidence.

BUNT OF WHEAT

In the 1960s and 1970s, bunt was generally regarded as one of those 'text-book' diseases which was of general biological and historical

build-up in the stock would be very rapid. Dillon Weston and Engeldow (1932) calculated that saving seed from a crop in which one ear in 8,500 was bunted could result in one ear in 450 being infected in the subsequent year and one ear in four the year after that. This pattern of development would explain cases such as that encountered by one of the authors in the early 1980s (Yarham, unpublished). Seed of cv Maris Huntsman had been home-saved for six years and, despite the fact that no seed treatment had been used, no problems had been noticed. In the seventh year, however, bunt was so severe that it tainted the eggs of poultry fed on the contaminated grain.

In the late 1980s, however, a series of cases was encountered in which it was difficult to see how bunt could have built up so quickly in stocks which were only a year or two from certified and treated seed. In 1989, for example, an infection levels of 50% ears infected was recorded in a crop of cv. Galahad grown from seed which had been home-saved for only two years on a south Norfolk farm. In that same year, an even more remarkable case was encountered in Cambridgeshire where a high level infection was encountered in a stock of wheat in the first year after home-saving from a parent crop grown from certified organomercury treated seed. In 1990, the Cambridgeshire farmer again experienced a very severe attack of bunt despite the fact that all his seed had been organomercury treated. The distribution of the affected wheat on the farm suggested very strongly that soil-borne spores from the previous year had provided the source of inoculum for the 1990 crop. In 1991, the farmer solved his problem by having his seed treated with triadimenol + fuberidazole ('Baytan'), but small untreated plots sown on the previously affected field showed that soil-borne inoculum would have presented a hazard for unprotected crops sown up until late October (Yarham, 1992).

There is little doubt that, in the recent dry years, soil-borne inoculum has been an important source of bunt in the eastern counties of England. It is significant that recent outbreaks of the disease have been mainly confined to these counties, few cases have been reported from the wetter west or from Scotland.

An indication of just how widespread the disease is in our wheat stocks was provided by tests carried out in 1991 by OSTS, Cambridge. While no bunt balls were found in any of the samples examined (a marked contrast to the pre-war years), the use of a newly introduced centrifugation technique showed over 60% to have low levels of bunt spores on the seeds (S. Ball. OSTS, Cambridge, pers. comm.).

COVERED SMUT OF BARLEY

Ustilago hordei was a common cause of yield loss in the 1920s, but even before the widespread use of organomercury on barley its incidence had begun to decline (Pethybridge, 1929), probably because of the demise of certain old and highly susceptible varieties (Gray, 1954). Despite this decline, covered smut was still being found in almost 2% of barley seed samples tested by Cambridge OSTS in the early 1940s (Eastham & Brett, 1947) but with the increasing use of organomercury on barley seed the disease declined to almost complete obscurity in the post-war years. However, Rennie et al. (1983) noted that it still occasionally occurred in Scotland, and cases near Ipswich in 1980 and near Cambridge in 1991

served as reminders that the disease was not extinct in England. In the 1991 outbreak about 2% of the ears were infected in a crop of cv. Maris Otter grown from a seed stock which had been home-saved for three years without treatment on a farm in south Cambridgeshire. So effective have seed treatments been in controlling the disease in recent years that there has been no need to breed for resistance to U. hordei. It is not impossible that were we to cease to treat our barley crops we would find some of our modern varieties somewhat wanting in this respect. Covered smut remains a very rare disease but we should not allow its rarity to lull us into complacency. The brief rise to prominence of Cochliobolus sativus (a seed-or soil-borne pathogen causing seedling blight and foot rot) in the early 1970s when the barley cultivar Clermont was being grown (Rennie et al., 1983) provides a good example of how the unwitting introduction of a highly susceptible variety can increase the importance of a previously disregarded and insignificant disease.

BARLEY LEAF STRIPE

In 1941/2, 100 randomly selected barley samples were tested at the Cambridge OSTS for the presence of Pyrenophora graminea. Ninety of these samples were found to carry the pathogen and on 37 infection levels exceeded 50% (Eastham & Brett, 1947). In the early 1980s, Rennie et al. (1983) were able to report that, although levels of up to 40% infection could occasionally be found where uncertified seed was sown without treatment, the pathogen was 'virtually unknown in barley crops grown from certified seed in Scotland since it is so effectively controlled by organomercury seed treatments'. This satisfactory state of affairs was, however, soon to be disturbed by the emergence on spring barley of strains of the pathogen which were resistant to mercury (Jones et al., 1989). These soon became so widespread in both Scotland and England that many barley growers had turned to alternative seed treatments even before the derogation on organomercury was rescinded. Certainly, the late development of resistance to these fungicides had led to our entering the post organomercury era with many barley stocks contaminated with P. graminea. In 1991, just over 50% of the barley samples tested at OSTS, Cambridge were found to be infected with the disease and though in most the levels were low (<4%) the figures clearly show how widespread the disease has become (S. Ball, OSTS pers. comm.)

Few farmers would wittingly save seed from a crop obviously infected with leaf stripe. It would be unwise to assume, however, that seed will necessarily be free of the pathogen simply because the parent crop showed no signs of infection. Observations in Scotland (V. Cockerell, Scottish Agricultural Science Agency, pers. comm.) have shown how wind-blown spores can effectively spread the disease from one field to the next. The same studies revealed how rapidly the disease can build up in a stock once it has been introduced (Table 2)›

TABLE 2. Incidence of infected seeds in a crop of spring barley infected with leaf stripe and in a crop adjacent to it. Gogarbank, 1991.

% plants with leaf stripe in crop A	% seeds with Pyrenophora graminea in grain harvested from						
	Crop A	Adjacent crop (B) at various distances from Crop A					
		0m	5m	10m	50m	100m	200m
8.5	46	28	14	13	7	3	5

NET BLOTCH

In the UK, seed borne inoculum of Pyrenophora teres has been regarded as being of secondary importance to inoculum carried on infected debris. In New Zealand, however, seed has been regarded more seriously as a source of the disease (Hampton, 1980). With good ploughing it is possible substantially to reduce the amount of trash-borne inoculum in a field. Were we to cease to control it by seed treatment, the seed-borne phase of the disease could assume a much greater importance in Britain.

DISEASES OF OATS

In the late 1920s and early 1930s, Muskett & Cairns (1933) carried out a series of experiments on the use of seed treatments on oats in Northern Ireland. They were able to increase yields by 14% by treating with formaldehyde (which effectively controlled both the loose and covered smuts (Ustilago avenae and U. hordei), and by 25% by the use of organomercury. O'Brien & Prentice (1930) who had obtained similar results in Scotland attributed the additional response to organomercury to the control of Pyrenophora avenae. Muskett & Cairns, however, considered that other factors were also involved. The view has subsequently been endorsed by the work of Richardson (1974) whose use of organomercury to control both P. avanae and Micronectriella nivalis (syn. Fusarium nivale) showed that the latter pathogen was far more important than the former as a cause of yield loss in oats, and that yield responses could sometimes be achieved even in the absence of significant seed-borne inoculum of either pathogen.

Mercury resistant strains of the leaf spot pathogen (Pyrenophora avenae) are now so widespread that the absence of mercury is likely to have little effect on the incidence of this not particularly important disease. More serious could be the effect on the oat smuts which could build up if the use of untreated seed were again to become common practice. The significance of F. nivale is discussed below.

FUSARIUM DISEASES

Richardson's work cited above illustrates the complexity of the factors governing response to seed treatment. It also highlights the importance of Fusarium in the pathology of cereal seedlings. Of the dozen

or so Fusarium species recorded on cereals in the UK, F. avenaceum, F. culmorum, F. graminearum, and F. nivale can, under favourable circumstances, cause death of young seedlings. F. nivale is the most important species in this respect, being particularly aggressive in cool, dry soils. The other species are more prone to cause damage when soils are warm and dry. Organomercury treatment, while not eliminating fusarium completely from the seed, certainly reduced it (Bateman, 1976) and also afforded some protection from soil-borne infection (Bateman, 1977). While low levels of tiller death are compensated for by the increased tillering of surrounding plants, severe attacks can occasionally cause marked reduction in yield (Richardson, 1974).

A particularly good example of the effects of Fusarium seedling blight on wheat is provided by the trials in Scotland reported by (Richards, 1990). Varieties being compared for their suitability for organic production were sown without seed-treatment and the seed of one of them (cv. Apollo) was heavily infected with Fusarium. As a result, the ground cover given by this variety at GS 31 was only 16% as compared with 60% for cv. Mercia. On the 1992 NIAB Recommended List the figures for yield as percent of control (unsprayed) are given as 97 for Apollo and 90 for Mercia, yet in Richards' trial Mercia outyielded Apollo by 0.75 t/ha.

Unlike most other seed-borne diseases Fusarium is not always detectable by inspection of the parent crop (high levels of seed infection can occur without any evidence of ear blight). Moreover, since infection can occur from soil-borne inoculum, high levels can occur in a seed stock even if the parent seed was treated. It is thus one of the most likely of the diseases discussed to occur in the first year that a home-saved and untreated stock is used. The risks of severe losses can be reduced by the use of a higher than normal seed rate but in a cool, dry autumn following a warm wet grain-filling period (which would favour seed infection) this is not always sufficient to avoid losses.

In recent years strains of Fusarium (particularly of F. nivale) have arisen which are resistant to the benzimidazole fungicides (Locke et. al. 1987). Since such fungicides (eg fuberidazole) have been included in a number of seed treatment formulations to improve their activity against Fusarium this development has to be viewed with some concern, though in most instances, satisfactory control of the disease is likely to be provided by the other constituents of the formulations (eg triadimenol or carboxin).

DISCUSSION

After carrying out 227 comparisons of treated and untreated stocks of winter wheat and spring barley in Scotland, Richardson (1986) concluded that 'seed-treatment to protect against seed-borne pathogens, other than wheat or barley loose smut if known to be present, is not necessary for certified seed being used to produce a non-seed crop'.

One could endorse this opinion by citing an ADAS trial carried out at Arthur Rickwood EHF in the 1970s (Yarham, 1980). Stocks of wheat cvs Cappelle Desprez and Maris Huntsman were grown on for 6 years with and without organomercury seed treatment and were compared with bought in, treated C2 stocks. Apart from a little loose smut, which occurred in both

the treated and untreated stocks, no build up of seed-borne diseases was observed and in the final year of the trial the yields obtained from the 3 stocks were:-

bought-in organomercury treated stock	5.60 t/ha
home-saved with organomercury treatment	5.80 t/ha
home-saved without treatment	5.81 t/ha

It must be remembered, however, that the ADAS trial consisted of a small area of untreated wheat grown where all other crops, both on the farm and in the region round about, would have been grown from treated seed and where there would thus have been little or no inoculum of seed-borne diseases to infect it. Richardson's observations were also made in situations in which the majority of surrounding crops would have been treated. They were, moreover, made in Scotland, where soil-borne bunt has never been a problem, and before the days when the widespread occurrence of organomercury resistance had led to the build up of Pyrenophora graminea in barley stocks.

It is these two diseases, bunt and leaf stripe, which offer the most compelling arguments for the continued use of seed treatment. Both can spread unseen into fields adjacent to infected crops (the former as wind blown spores to contaminate the soil at harvest, the latter to infect the florets of nearby healthy plants at anthesis), both can build up very rapidly in a stock once they have been introduced into it and, as we enter the post-mercury era, both are at higher levels than they have been for some decades. The new seed dressings afford excellent protection against these diseases and could soon consign them to their former obscurity - unless what is being perceived as the expensiveness the new materials tempts cereal growers not to use them.

Seed treatments should be seen as the first applications in the fungicide programmes now applied almost routinely to cereal crops. As with all other elements in such programmes, the decision as to whether to apply them is to some extent an actuarial one - we cannot be certain of the response they will give us, we use them to insure ourselves against certain risks inherent in growing the crop. What we can do is to try to quantify the risks.

If a farmer intends to save seed from a crop which was itself grown from certified and treated seed, if he has inspected it thoroughly and found no evidence of seed-borne disease, if his other crops (and those of his neighbours) are also disease free and grown from treated seed, and if the flowering and grain-fill periods have been so dry that the risk of seed-borne Fusarium is low, then he might be justified in deciding to save seed without treatment. Even so, he would be well advised to 'make assurance double sure' by getting the seed checked for the presence of diseases by one of the official seed testing laboratories. He would need to remember, however, that however healthy the seed, his crop could still be at risk from soil-borne Fusarium against which seed-treatment offers at least partial protection.

If these criteria could not be met then the farmer would be well advised to use a seed-treatment, choosing a material again with due regard to the risks to which the crop is exposed. On wheat, for example, had he no good reason to be concerned about loose smut or soil-borne bunt but

suspected the Fusarium levels might be high, he might choose to use one of the guazatine based materials which have been shown to be particularly effective against Fusarium (Jones, unpublished). If he wished for additional protection against loose smut (some varieties, eg Beaver, have very low scores for resistance to this disease) he might prefer a carboxin based material, or one based on triadimenol or flutriafol which would also provide early season control of foliar diseases. If he suspected that the field where the seed was to be sown might be contaminated with soil-borne bunt then use of triadimenol of flutriafol would be essential.

Any general advocacy of non-treatment of seed would be irresponsible in the extreme. We dare not risk a return to the situation which prevailed before organomercury was introduced. Recent experience of bunt, in particular, has served as a timely reminder of what seed-borne diseases can do. Levels of up to 50% infection have been recorded, and prices as low as £15/tonne have been paid for grain contaminated with the spores. The 'forgotten diseases' have ways of making themselves remembered.

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ALTERNATIVES TO MERCURY FOR CONTROL OF CEREAL SEED-BORNE DISEASES

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ABSTRACT

The use of mercury seed treatments resulted in effective control of wheat bunt, barley leaf stripe and other seed-borne diseases of cereals. The removal of approvals for mercury has resulted in the use of a range of alternatives. The major formulations are based on DMI compounds, carboxamides, phenylpyrroles and guanidines. The efficacy of these compounds against the main seed-borne diseases are described by reference to UK trials conducted in 1990/91 and 1991/92.

INTRODUCTION

Prior to the introduction of mercury, wheat bunt, barley leaf stripe and other seed-borne diseases such as covered smut and seedling blights were widespread and caused important yield and quality loss in cereals.

In 1925 in the UK, before mercury was widely used, microscopic examination of grain samples revealed that most wheat was contaminated by bunt. Mere visual examination of grain at the Official Seed Testing Station revealed that one third of all samples were grossly contaminated with bunt balls (Marshall, 1960). In barley, leaf stripe was endemic and low vigour crops were normal (Large, 1940).

Use of mercury led to a massive reduction in the frequency of both diseases. For the first time, farmers were able to produce crops largely free of seed-borne disease.

In 1978 an EC Directive was issued which has led to the progressive withdrawal of mercury-based seed treatments throughout member states. Use of mercury in the UK, the last country in the EC to withdraw approvals, was not permitted after 31 March 1992. The underlying reasons for the withdrawal are based on the toxicity and environmental persistence of mercury.

Effective alternatives must be widely used if we are to avoid a decline in cereal seed health to the level experienced prior to the introduction of mercury salts. Alternatives to mercury are listed in Table 1. Some compounds are used alone and in mixtures, others are used only in mixture.

TABLE 1. Examples of major fungicidal chemicals approved for use as cereal seed treatments in Europe.

Chemical Group	Active Ingredients
Benzimidazoles	Thiabendazole Fuberidazole
Ergosterol Biosynthesis Inhibitors - DMI group	Flutriafol Triadimenol Diniconazole Imazalil Myclobutanil Bitertanol Tebuconazole Prochloraz
Carboxamides	Carboxin Fenfuram Methfuroxam
Guanidines	Guazatine
Phenylpyrroles	Fenpiclonil
Hydroxypyrimidines	Ethirimol
Dicarboximides	Iprodione
Dithiocarbamates	Maneb/mancozeb Thiram
Copper salts	Copper oxyquinolate
Organophosphates	Ampropylfos

In some European countries narrow spectrum products based on one active ingredient have received approvals while in others the approach has been to register only products with a broad spectrum of action. Only the major compounds are discussed here.

Benzimidazoles have been widely used, mainly for their efficacy against seedling blights. The development of widespread resistance in *Fusarium nivale (= Microdochium nivale)*, has reduced the effectiveness of these compounds against this pathogen (Locke et al, 1987).

Many ergosterol biosynthesis inhibitors have been developed as seed treatments. Most have strong activity against bunt and some control loose smuts, unlike mercury. Imazalil is used in several formulations to enhance control of leaf stripe in barley. These compounds may reduce the speed of crop emergence and, along with some other chemicals, care is needed to ensure accurate treatment of good quality seed and good seed-bed preparation. In addition to controlling seed-borne diseases, flutriafol and triadimenol will control early infections of mildew and yellow rust.

Carboxamides such as carboxin were the first compounds introduced capable of controlling the embryo-borne loose smuts. They are also effective against bunt and seedling blights and have excellent crop safety.

Guazatine and fenpiclonil are recommended against seedling blights and bunt. They have good crop safety.

Copper salts and dithiocarbamates are registered in several European countries. They tend to give less reliable disease control and are less active than the best materials on leaf stripe, bunt and seedling blights. Because of these weaknesses they are often also used in mixtures.

This paper compares the main alternatives to mercury against the principal seed-borne diseases of wheat and barley. Trial results generated in the UK between 1990 and 1992 are used to illustrate their performance.

MATERIALS AND METHODS

The products tested are listed in Table 2. All trials used naturally infected seed stocks and were fully replicated using randomised block designs. Trials were located in England and Scotland. Seed was treated in a laboratory-scale Rotostat machine. Plot sizes were 2m x 12m and drilling was achieved with a Hege small plot drill.

Trial results are generally expressed as '% control' except where stated. Means were compared using the test of Least Significant Difference (LSD) and values followed by a common letter are not significantly different at P = 0.05.

RESULTS AND DISCUSSION

Trial results against wheat bunt, *Fusarium nivale* seedling blight, barley leaf stripe and loose smut are summarised in Tables 3 to 7. The control of other diseases is discussed at the end of this section.

Wheat Bunt

Results of trials conducted in 1990/91 and 1991/92 are summarised in Table 3.

Disease levels in all trials were high, allowing differences in the activity of products to be seen. Mercury gave high levels of control but never completely eradicated all disease.

Higher control, often resulting in complete eradication of the disease was achieved with flutriafol/TBZ, triadimenol/fuberidazole, carboxin/TBZ and fenpiclonil. Guazatine was always inferior to mercury and the other options against this disease.

TABLE 2. Formulations included in trials - active ingredient rates on treated seed.

Product	Active Ingredients	Rates in ppm
'Cerevax'	Carboxin/thiabendazole (TBZ)	900/50
'Cerevax' Extra	Carboxin/TBZ/imazalil	600/50/40
'Vincit'	Flutriafol/TBZ	50/50
'Vincit' IM	Flutriafol/TBZ/imazalil	75/50/40
'Beret'	Fenpiclonil	200
'Beret' Special	Fenpiclonil/imazalil	200/40
'Baytan'	Triadimenol/fuberidazole	375/45
'Ferrax'	Flutriafol/ethirimol/TBZ	150/2000/50
'Ferrax' IM	Flutriafol/ethirimol/TBZ/imazalil	150/2000/50/30
'Rappor'	Guazatine	600
'Rappor' Plus	Guazatine/imazalil	660/55
'Ceresol'	Phenylmercury acetate ammonium complex	22

It is clear, therefore, that four alternative products are available which will at least match the performance of mercury against bunt.

TABLE 3. % Control of wheat bunt (1990/91 and 1991/92) - 4 trials

year:	1990/91	1990/91	1990/91	1991/92
location:	Lincs	Lincs	Suffolk	Suffolk
variety:	Apollo	Champlein	Champlein	Champlein
drilling date:	9/10/90	9/10/90	16/10/90	16/10/91
Untreated, actual level (%)	(38.1) a	(31.7) a	(36.6) a	(32.1) a
Flutriafol/TBZ	100 b	100 c	100 c	100 d
Carboxin/TBZ	100 b	99 bc	100 c	99 d
Fenpiclonil	100 b	99 bc	100 c	99 d
Guazatine	89 b	93 b	91 b	79 b
Triadimenol/fuberidazole	100 b	100 c	100 c	100 d
Mercury	99 b	96 bc	95 b	94 c

Fusarium Seedling Blight

Trials conducted in 1991/92 against *Fusarium nivale* seedling blight are presented in Table 4. A highly infected seed batch of cv Haven from Ireland was used in both trials. Laboratory tests revealed that the seed had greater than 70% contamination with *F. nivale*.

TABLE 4. Control of *Fusarium nivale* seedling blight (1991/92) - 2 trials

location:	Lincs			Warwicks		
drilling date:	8/11/91			22/10/91		
assessment:	plant count	ear count	relative yield (t/ha)	plant count	ear count	relative yield (t/ha)
days after drilling:	63	236		42	270	
Untreated (actual)	(9.1)c	(126)c	(2.03)c	(22.0)c	(113)c	(6.6)a
Carboxin/TBZ	334 a	170 ab	159 a	140 ab	120 ab	105 a
Fenpiclonil	330 a	185 a	176 ab	147 ab	114 b	109 a
Guazatine	338 a	177 a	185 b	159 a	137 a	109 a
Triad./fub.	304 a	151 b	155 a	121 b	116 ab	102 a
Mercury	279 b	168 ab	170 ab	134 ab	113 b	107 a

All assessments related to untreated = 100
Ear counts based on 4 x 0.25m² quadrats/plot.
Plant counts based on 4 x both sides 0.5m rule.

In the Lincolnshire trial the seed was drilled in early November into a light soil at an exposed site. In the Warwickshire trial, the seed was drilled over two weeks earlier into a heavier soil and a more sheltered location.

In both trials, *Fusarium nivale* caused disease on untreated plots but in the Warwickshire trial, early emergence and final establishment were far less affected than in the Lincolnshire trial. This result demonstrates the importance of environment in disease expression.

At the Lincolnshire site all four non-mercurial treatments gave superior control of *Fusarium nivale* seedling blight when compared to mercury. These treatments resulted in plant counts more than 3 times higher than in the untreated plots. Ear counts later in the season confirmed that plant growth compensation had resulted in smaller final differences between treatments. Relative yields ranged from 155% to 185% of the untreated but differences between non-mercurial treatments were not significant. The Warwickshire trial gave plant counts between 121% and 159% of the untreated and all treatments were significantly superior to the untreated. Ear counts were raised to between 113% and 137% of the untreated and again all treatments were superior to the untreated. Final figures for all treatments were higher than for the untreated but were not quite significant at P = 0.05.

The amount of *Fusarium* seed infection varies according to season and locality. For example experience suggests that *Fusarium nivale* seedling blight is more significant in Ireland than England while the later snow mould phase of the disease is a problem in southern Germany and E. Europe. Environmental conditions after sowing are crucial in determining the extent of disease expression. It is interesting to

note, however, that even when a high level of seed contamination and harsh environmental conditions conspire to reduce plant numbers, crops are surprisingly able to compensate with much tillering during the season.

Barley Leaf Stripe

Mercury seed treatments have been used for 60 years to control barley leaf stripe and close relatives such as seed-borne net blotch and oat leaf spot.

Oat leaf spot, caused by *Pyrenophora avenae*, was well controlled by organo-mercurials until the 1960s when resistant strains of the pathogen emerged (Sheridan, 1971). In 1990, mercury failed to control leaf stripe in many crops of spring barley in Scotland due to resistance. Its removal from the market thus came at a time when it was becoming increasingly unreliable against these diseases.

Table 5 presents trials results on winter barley with mercury and a range of alternatives.

TABLE 5. % Control of winter barley leaf stripe (1990/91 and 1991/92) - 5 trials

year:	1990/91	1990/91	1990/91	1991/92	1991/92
location:	Lincs	W. Lothian	Somerset	Lincs	Warwicks
variety:	Plaisant	Ara	Gaulois	Plaisant	Gaulois
drilling date:	4/10/90	24/10/90	14/12/90	31/10/91	22/10/91
Untreated	(13.6)a	(28.6)a	(14.1)a	(15.0)a	(2.0)a
Flutriafol/TBZ	55 b	55 c	39 b	-	-
Flutriafol/TBZ/imazalil	98 c	98 f	97 e	100 c	99 cd
Flut./ethirimol/TBZ	86 bc	63 cd	54 bd	-	-
Flut./ethirimol/TBZ/imaz	100 c	99 fg	98 f	-	-
Carboxin/TBZ	74 bc	70 d	67 cd	-	-
Carboxin/TBZ/imazalil	-	-	-	100 c	100 d
Fenpiclonil	98 c	100 g	98 e	100 c	91 c
Fenpiclonil/imazalil	100 c	100 g	99 e	100 c	98 cd
Guazatine/imazalil	97 c	99 fg	97 e	-	-
Mercury	100 c	100 g	98 e	83 b	96 cd

Mercury gave complete or nearly complete control in four out of five winter barley leaf stripe trials. In the 1991/92 trial on Plaisant, the level of control achieved with mercury (83%) was lower than expected.

The flutriafol/TBZ mixture typically gave approximately 50% control, while carboxin/TBZ treatment resulted in around 70% control. Fenpiclonil gave excellent control in four out of five trials.

Inclusion of imazalil in formulations resulted in excellent control of leaf stripe in all trials.

Spring barley leaf stripe trial results are presented in Table 6.

In all three trials, non-mercurial treatments including carboxin/TBZ and flutriafol/TBZ gave complete control of high levels of leaf stripe infection. Mercury failed completely in the Kelso trial and gave only slight control in the trial at Kinross; effects attributed to resistance.

TABLE 6. % Control of spring barley leaf stripe

year:	1990/91	1990/91	1991/92
location:	Kelso	Kinross	Lincs
variety:	Blenheim	Triumph	Blenheim
drilling date:	16/4/91	8/4/91	5/3/92
Untreated, actual level (%)	(24.0)	(32.0)	(3.6)
Flutriafol/TBZ	100	100	100
Flutriafol/TBZ/imazalil	100	100	100
Flutriafol/ethirimol/TBZ/imazalil	100	100	100
Carboxin/TBZ/imazalil	100	100	100
Fenpiclonil/imazalil	100	100	-
Guazatine/imazalil	100	100	-
Mercury	0	33	100

Data not amenable to statistical analysis due to absence of variation.

Barley Loose Smut

Table 7 presents trial results from barley loose smut trials. As expected, mercury gave little or no control of the disease in all trials. Neither fenpiclonil nor guazatine/imazalil are claimed to control loose smut and neither gave sufficient control of the disease in these trials. The flutriafol mixtures gave excellent control of the disease in all three trials.

TABLE 7. % Control of winter barley loose smut (1990/91 and 1991/92) - 3 trials

year:	1990/91	1990/91	1991/92
location:	Lincs	Lincs	Lincs
variety:	Viva/Panda mix	Plaisant	Panda
drilling date:	28/09/90	04/10/90	09/10/91
Untreated, actual level (%)	(19.2) b	(3.5) a	(3.2) a
Flutriafol/TBZ/imazalil	100 e	98 a	100 c
Flutriafol/ethirimol/TBZ	100 e	100 e	-
Fenpiclonil	93 d	69 c	93 b
Guazatine/imazalil	49 c	48 d	-
Mercury	0 a	39 b	10 a

Carboxin mixtures are not currently recommended for control of winter barley loose smut due to occasional control failures, particularly on cultivars Panda and Viva.

Other Diseases

Several other diseases are controlled by non-mercurial seed treatments. Covered smut of barley, caused by *Ustilago hordei* and *Septoria* seedling blight caused by *S. nodorum* are thought to be controlled by most alternatives.

In addition to the diseases controlled by mercury, some formulations are also active against foliar diseases. The first seed treatment active against mildew was 'Milstem', based on ethirimol; it was used in the UK on barley in the 1970s but was later withdrawn following problems of resistance. The later introduction of 'Baytan' (triadimenol/fuberidazole) gave the cereal farmer a treatment which controlled all the major seed and soil-borne diseases and powdery mildew of both wheat and barley. Subsequently the mixing of ethirimol with flutriafol and TBZ (as 'Ferrax') produced a dual mode of action product against barley mildew (Northwood *et al*, 1984).

Since the late 1980's the triadimenol/fuberidazole mixture has been much used on susceptible wheat cultivars for the management of yellow rust. By removing inoculum in the autumn there is a much lower level of disease build-up in the spring.

CONCLUSIONS

Mercury seed treatments played the major role in maintaining cereal seed health until their removal from agriculture over the last two decades. They were crop safe, highly effective and less prone to pathogen resistance than their replacements. Nevertheless mercury failures have occurred and had it not been withdrawn it is possible that resistance could have threatened its future use. Furthermore, some alternatives have a broader spectrum or superior efficacy of action e.g. loose smut and *Fusarium nivale*.

Against wheat bunt, the DMI's, carboxamides and fenpiclonil are highly effective replacements and it seems logical that as long as they are widely used then bunt will remain a manageable disease. Any widespread failure to treat seed accurately or the use of less effective materials could result in a significant increase in the frequency and severity of this disease.

Leaf stripe of barley is particularly well-controlled by formulations containing imazalil while other DMI compounds such as flutriafol and triadimenol have useful activity, particularly on the spring crop. Carboxin and fenpiclonil are also effective but require imazalil to give reliable control in the winter crop. The reliance on imazalil for this purpose and the known ability of *Pyrenophora* species to develop resistance to fungicides do, however, indicate the need for alternatives of a different mode of action to be developed.

Seedling blights are most significant when heavily contaminated seed is drilled into conditions where germination is slow. While treatments based on carboxin, fenpiclonil and guazatine will give good control of the disease, it is clear that the early drilling of high quality seed into good seed-beds are important cultural means of control. Where *Fusarium* causes poor establishment, the crop will generally compensate with increased tillering. Only very high levels of seed contamination are likely to have a severe effect on final yield. Heavily infected seed batches are best avoided.

Loose smuts of wheat and barley may be controlled using carboxamides such as carboxin or some DMI compounds such as flutriafol and triadimenol. Control of loose smut has been achieved by seed crop inspection as well as through the use of chemical seed treatments. In the future it is likely that an increase in farmer saved seed may result in a greater need for the use of products able to eradicate loose smuts from seed stocks.

In the post-mercury era it is clear that integrated control of cereal seed-borne diseases will remain the correct approach to seed health management. Adherence to seed health standards, crop inspection and widespread use of approved seed treatments should ensure that bunt, leaf stripe and seedling blights remain at low levels in Europe.

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PHENYLPYRROLES - A NEW CLASS OF FUNGICIDES FOR SEED TREATMENT

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ABSTRACT

Phenylpyrroles are a new class of non-systemic fungicides developed by Ciba-Geigy. They are closely related to the natural antibiotic pyrrolnitrin. As a seed treatment, the phenylpyrroles fenpiclonil (20g AI/100kg seed) and CGA 173506 (5g AI/100kg) are very well tolerated and control the major seed-borne pathogens of wheat, rye, triticale and barley.

Both compounds are extremely effective against *T. caries*, giving >99% control of this disease. Efficacy against *G. nivalis* is excellent and includes MBC-resistant strains. Control of other seed-borne diseases of wheat, rye and triticale such as *Fusarium culmorum*, seed-borne *Septoria* and *Urocystis occulta* is also equal to the best commercial standards. As barley seed treatments, fenpiclonil and CGA 173506 show a high activity against *H. gramineum*. Furthermore, both compounds are highly active against *U. hordei*.

CGA 173506 is also being developed as a seed treatment of rice. The compound can be applied by various methods and gives control of *G. fujikuroi* and *C. myabeanus* at least equal to the best current standards with a very useful side activity against seed-borne *P. oryzae*. Both fenpiclonil and CGA 173506 have shown excellent activity against *Ascochyta spp.* on peas.

The broad spectrum of activity of these phenylpyrroles and their new mode of action make them valuable seed treatment products for today and the future, especially in the light of recent legislation against mercury.

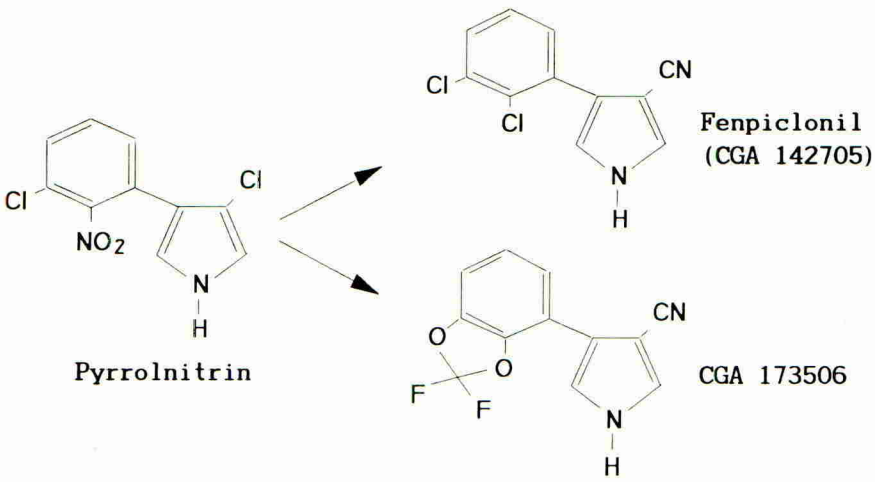
INTRODUCTION

As a method to eliminate seed-borne diseases and to protect against soilborne fungal attack, treatment of seeds with mercury based products has been practised since the beginning of the century. With the ban of organomercury throughout Europe in recent years, new products are needed that show at least a similar spectrum of activity while being safe to both the user and the environment. Ideally, modern seed treatments should combine excellent crop tolerance with a broad spectrum of activity, low use rates and a favourable toxicology. In recent years, some fungicides have lost efficacy due to the development of resistance, e.g. loss of activity against *Fusarium* spp. of benzimidazoles. Consequently, new products should preferably belong to classes of chemicals which do not show cross resistance to currently used seed treatments.

Fenpiclonil and CGA 173506 belong to the new class of phenylpyrrole fungicides developed by Ciba-Geigy. They are non-systemic and control a broad spectrum of fungi among the *Ascomycetes*, *Basidiomycetes* and *Deuteromycetes*. Fenpiclonil has been developed as a seed treatment for cereals and non cereals and is marketed under the tradenames 'BERET' (Nevill *et al.*, 1988) and 'GALBAS'. CGA 173506 is being developed as a foliar fungicide (tradename 'SAPHIRE') as well as a seed treatment (tradename 'CELEST') for cereal and non cereal crops (Gehmann *et al.*, 1990; Leadbeater *et al.*, 1990).

RELATIONSHIP BETWEEN PYRROLNITRIN AND THE PHENYLPYRROLES

The phenylpyrroles are a successful example of a new class of fungicides closely related to a natural bioactive compound. Pyrrolnitrin, a secondary metabolite produced by *Pseudomonas* spp. (Elander *et al.*, 1968) and members of the *Myxobacterales* (Gerth *et al.*, 1982) served as the lead structure for the phenylpyrroles fenpiclonil and CGA 173506.



Pyrrolnitrin has a high activity against various plant pathogenic fungi but has the disadvantage of being very photolabile. The phenylpyrroles fenpiclonil and CGA 173506 are at least as active as pyrrolnitrin and are significantly more stable in light (Nyfeler *et al*, 1990).

BIOLOGICAL ACTIVITY

Overview on crop tolerance and efficacy as a cereal seed treatment

Fenpiclonil and CGA 173506 have been widely tested on commercial varieties of wheat, barley and rye throughout the world without any establishment problems. Delays in emergence, as frequently seen after seed treatment with triazoles, have not been observed.

Both phenylpyrroles control the major seed-borne diseases of wheat, barley, rye and triticale (Table 1). Control of the major pathogens is at least equal to best commercial standards. Where control of *Ustilago tritici* and *Ustilago nuda* is necessary, mixture partners are required, e.g. carboxin or triazoles. Mixtures of phenylpyrroles with difenoconazole provide allround disease control in wheat and give protection against both *Ustilago tritici* and dwarf bunt (*Tilletia controversa*). Addition of a specific leaf stripe compound, e.g. imazalil, is required if reliable, complete control of barley leaf stripe is needed (e.g. crops grown for seed multiplication).

TABLE 1. Activity spectrum of fenpiclonil (20g AI/100kg seed) and CGA 173506 (5g AI) on cereals.

Pathogen(s)	Disease	Crop(s)
Gerlachia nivalis	Snow mould	Wheat, rye, triticale, barley
Fusarium spp.	*Fusarium*-seedling attack	Wheat, rye, triticale, barley
Tilletia caries	Common bunt	Wheat
Septoria nodorum	*Septoria*-seedling attack	Wheat, triticale
Urocystis agropyri	Flag smut	Wheat
Urocystis occulta	Stalk smut	Rye
Helminthosporium sativum (approx. 70 - 90% control)	*Helminthosporium*-seedling attack	Wheat, barley
Helminthosporium gramineum (approx. 85% (-100%) control)	Leaf stripe	Barley
Ustilago hordei	Covered smut	Barley

Control of common bunt (*Tilletia caries*)

Common bunt, caused by *Tilletia caries*, is one of the most serious seed-borne threats to cereal crops in that infection with this disease can lead to total crop rejection. Bunt has been termed a "forgotten disease" because of the effectiveness of mercury for its control. A major concern in the UK is that cereal seed may be left untreated, thus causing widespread problems with the disease. Bunt can build up to damaging levels on wheat in a very short time, just one or two seasons, therefore it is vitally important for any new seed treatment product to reliably give the necessary high level of control of this disease, i.e. in the order of 99-100% control.

Fenpiclonil and CGA 173506 have been exhaustively tested for bunt control throughout the world in trials with naturally infected seed or with seed inoculated with bunt spores (2 - 5g spores / kg wheat). In a series of 31 field trials conducted in Switzerland, Great Britain, France and Germany during 1989-1992, aimed at directly comparing fenpiclonil and CGA 173506, an average level of control of greater than 99.7%

was given by both products (Table 2). It is acknowledged that levels of seed infection in these trials are considerably higher than those likely to be found in commercial practice. The data do, however, show fenpiclonil and CGA 173506 to give outstanding control of bunt even under extreme disease pressure.

TABLE 2. Control of *T. caries* on winter wheat. Means of 31 trials from different European countries in which fenpiclonil and CGA 173506 were tested side by side.

Treatment	Rate (g AI/100 kg seed)	% Efficacy *
Fenpiclonil	20	99.75
CGA 173506	5	99.84

* The mean % attack in untreated was 35.9% and varied between 2.8 and 98%

In a series of 9 trials conducted in Switzerland and Great Britain in 1985 - 1990 in which fenpiclonil (20g AI/100kg seed) was tested in comparison with organomercury (2.2 - 2.4 g AI/100kg), fenpiclonil gave on average 99.7% control, compared to 98.5% control recorded for organomercury. In these trials the lowest efficacy given by fenpiclonil was 99.1%. In Table 3 results of trials from Switzerland and Great Britain are given in which fenpiclonil and CGA 173596 were compared to standard seed treatments. Control by the phenylpyrroles was equivalent to guazatine + fenfuram and triadimenol + fuberidazole, and superior to straight guazatine.

TABLE 3. Control of *T. caries* on winter wheat. Results from Switzerland (1989/90) and Great Britain (1991/92)

Treatment	Rate (g AI/100 kg seed)	% control 1989	1990	1991	1992
Untreated	-	(41.5)*	(18.4)*	(84.0)*	(48.0)*
Guazatine + fenfuram	60 + 30	98.6	99.6	-	-
Guazatine	66	-	-	92.5	73.9
Triadimenol + fuberidazole	37.5 + 4.5	100	100	100	100
Fenpiclonil	20	99.5	100	99.9	99.7
CGA 173506	5	99.2	100	100	99.6
Number of trials		2	2	2	2

* % ears attacked

Control of Fusaria *(Gerlachia nivalis, Fusarium spp.)*

Gerlachia nivalis (syn. *Fusarium nivale;* perfect stage *Monographella nivalis)* is one of the most important and widespread of *Fusarium* species attacking cereals, causing seedling death, stem browning and snow mould. It is of particular importance in Northern Europe and is widespread in the United Kingdom. After harvest 1991, 93% of wheat samples from Scotland tested at the Scottish Official Seed Testing Station in Edinburgh were infected with *G. nivalis* (90 samples were tested). The average level of infection in these samples was 30% seeds infected, with up to 70% seeds infected in the severest cases (personal communication, W. Rennie).

The seed-borne *Fusarium* spp. were only partly controlled by organomercury because the chemical did not eradicate deep-seated infections. Triazole seed treatments currently available (e.g. triadimenol, flutriafol) have only a partial effect against seed-borne *G. nivalis* and as a result are formulated in mixtures with benzimidazole fungicides such as fuberidazole or thiabendazole to improve their control of this disease. In the UK, as well as in many other European countries however, *G. nivalis* is now mainly resistant to benzimidazole fungicides so that reliable control can no longer be expected.

Trials over several years carried out throughout Europe show fenpiclonil at 20g AI/100kg seed and CGA 173506 at 5g AI to give excellent, long-lasting control of seed-borne *Fusarium nivale.* Table 4 shows the high level of control given by both phenylpyrroles in field trials in the UK during 1991/92. Fenpiclonil and CGA 173506 gave clear improvements in crop establishment when evaluated in the December - January following sowing with seed which, without an effective seed treatment, was commercially unacceptable and would lead to a crop failure. This high level of control was seen later at a GS 23-32 evaluation for % crop cover and continued through to the crop harvest.

TABLE 4. Control of *G.nivalis* on winter wheat (Great Britain 1992; means of four trials)

Treatment	Rate (g AI/100 kg seed)	Rel. stand 37-56 days after planting (Untreated = 100)	% control 109-161 days after planting	% plot cover at GS 23-32
Untreated	-	(17.3)*	(13.9)**	21.7
Flutriafol +thiabendazole	5 + 5	125	11.9	38
Guazatine	66	261	68.7	81
Fenpiclonil	20	256	76.0	81
CGA 173506	5	272	83.5	81

(Infected seed, 70% of seed infected with *G. nivalis)*

* Plant number per metre row

** Number of infected plants. Sample size 25 plants.

In parts of Europe where long periods of snow cover occur during winter, it is important for a fungicide to give a high level of control for a prolonged period of time. The excellent long lasting activity of fenpiclonil and CGA 173506 in snow mould trials on wheat and rye as well as the activity of fenpiclonil and CGA 173506 against MBC-resistant *G. nivalis* has been reported (Nevill et al, 1988; Leadbeater et al., 1990). During the season 1990/91, fenpiclonil and CGA 173506 were tested in Switzerland side by side in snow mould trials on rye. In these trials the effficacy of both phenylpyrroles was very similar and superior to the guazatine containing standard (Table 5).

TABLE 5. Control of snow mould *(G.nivalis)* on winter rye - trials in Switzerland 1991

Treatment	Rate (g AI/100 kg seed)	% diseased leaf area in spring, trial FR010*	FR011*	FR012**	FR013**	Mean
Untreated	90.0	90.0	93.3	86.3	75	86.2
Guazatine + fenfuram	60 + 30	14.0	23.3	6.8	9.5	13.4
Fenpiclonil	20.0	12.3	13.0	3.8	4.3	8.4
CGA 173506	5.0	16.7	14.0	11.0	5.3	11.8

* Seed infection 14% (agar plate) ** Seed infection >70% (soil test)

TABLE 6. Control of *F. culmorum* on winter wheat. Results of trials from Switzerland and France

Treatment	Rate (g AI/100 kg seed)	% stand increase at GS 12 Field (CH)[1]	Glasshouse (CH)[2]	Outdoor trials (F)[3]	Glasshouse (F)[4]
Untreated	-	(115.6)[5]	(31.0)[6]	(37.2)[6]	(31.0)[6]
Guazatine + fenfuram	60 + 30	189.8	-	-	-
Thiabendazole	120	-	-	150.1	231.5
Oxine-copper + prochloraz	20 + 20	-	263.4	199.0	229.8
Fenpiclonil	20	198.3	263.4	182.0	235.5
CGA 173506	5	195.5	247.3	182.0	239.5

1 Means of 4 trials (1988 - 1991)
2 1 trial in seed trays. 100 kernels per seed tray, 3 replications (1991)
3 Means of 2 trials in pots. 25 kernels per pot, 6 replications. Pots placed outdoors during germination (October 1991)
4 Means of 2 trials in seed trays. 50 kernels per seed tray, 4 replications. Incubated at 5 - 8 °C before emergence, thereafter at 12 - 15 °C
5 Plants per m^2
6 % emerged, healthy plants

In warm wheat growing areas, *F. graminearum* is the major causal agent of *Fusarium* seedling attack, whereas *F. culmorum* is more common under cooler, temperate climatic conditions. High levels of attack on the seed with *F. culmorum* can lead to substantial reductions in plant stand. Fenpiclonil and CGA 173506 have been tested in field and glasshouse trials on wheat in Switzerland and France. The seed used in these trials carried high levels of infection with *F. culmorum* (90 - 100%). In field trials carried out in Switzerland 1988 - 1991 as well as in seed tray and pot trials both fenpiclonil and CGA 173506 gave excellent stand increases in autumn similar to or better than the best current standards (Table 6).

Control of Barley Leaf Stripe (*Helminthosporium gramineum*)

Leaf stripe of barley, caused by *Helminthosporium gramineum* (syn. *Pyrenophora graminea*), is a widespread seed-borne disease which occurs on both winter and spring barley in most of the barley growing areas of the world. Within Great Britain, barley leaf stripe is particularly important in spring-sown crops, especially in Scotland. The disease built up rapidly in spring barley in the mid to late 80's, mainly due to resistance to mercury seed treatments. With the banning of mercury seed treatments, this has removed a valuable weapon for the control of this potentially damaging disease. At the present time there is a great reliance in European agriculture upon a single active ingredient for the control of barley leaf stripe, i.e. imazalil. This is a situation of some concern and clearly any new active ingredient with efficacy against leaf stripe is important for the long term control of this diseases.

Fenpiclonil and CGA 173506 are effective for the control of leaf stripe, as shown in Table 7. Both phenylpyrroles have reliably given above 86% control of the disease in a large number of trials (28) over several years. In more than a third of these trials a level of control of 95 - 100% was achieved. This high level of control given by the phenylpyrroles is useful on its own for use under normal disease attack situations and where crops are not being grown for seed multiplication. For situations where control higher than 95% is consistently required, mixtures with imazalil have been developed. The combination of these two active ingredients represents a strong anti-resistance strategy for the control of barley leaf stripe.

TABLE 7. Control of *H. gramineum* on winter barley. Means of 28 trials from different European countries in which fenpiclonil and CGA 173506 were tested side by side.

Treatment	Rate (g AI/100kg seed)	% Efficacy	% trials with 95 - 100% efficacy
Fenpiclonil	20	86.3	35.7
CGA 173506	5	87.6	42.9

* The mean % attack in untreated was 27.9% and varied between 1.7 and 99.6%

Control of barley covered smut (*Ustilago hordei*)

Covered smut of barley is a disease rarely seen in modern agriculture, mainly because it is fairly easily controlled by seed treatments, including mercury. The disease is very noticeable in the field and causes a black appearance of infected ears due to replacement of grain sites by teliospores. The membrane of the smut sorus that replaces grains in infected ears does not rupture until harvest and thus infected ears can be readily distinguished from those infected by loose smut *(Ustilago nuda).*

It is essential for any modern seed treatment to control this disease to keep it at the current low level of occurrence. In 7 trials carried out in Switzerland and the UK both fenpiclonil and CGA 173506 gave extremely high levels of control of this diseases as shown in Table 8.

TABLE 8. Control of *U. hordei* on winter barley. Results from Switzerland and Great Britain (1988-92)

% tillers attacked on untreated	Fenpiclonil, 20 g AI		CGA 173506, 5 g AI	
	Number of trials	Mean % efficacy	Number of trials	Mean % efficacy
1 - 3	3	95.6	5	98.1
4 - 7	3	91.9	2	98.7

Efficacy of CGA 173506 against *Gibberella fujikuroi, Cochliobolus myabeanus* and *Pyricularia oryzae* on rice

As a seed treatment of rice, CGA 173506 was shown to be highly active against MBC-sensitive and MBC-resistant *G. fujikuroi.* Performance was better than that of the standard benomyl+thiram (Leadbeater *et al.*, 1990). In Japanese official trials carried out 1989-90, efficacy of CGA 173506 against *C. myabeanus* was also clearly superior to the benomyl-thiram standard, whereas there was no difference in activity against seed-borne *P. oryzae.* CGA 173506 may be applied either as a seed slurry treatment (25g AI/100kg), as a spray onto seeds (20g AI/100kg) or as a dip to pregerminated seeds (10min at 1.25-2.5g/l or 24h at 0.12-0.25g/l). Activity is similar with all application methods. As an example, results of the slurry application are given in Table 9.

TABLE 9. Efficacy of CGA 173506 as a rice seed treatment. Results from Japanese official trials 1989-1990.

Treatment	Rate (g AI/100 kg seed)	% efficacy *G. fujikuroi*	*C. miyabeanus*	*P. oryzae*
Benomyl + thiram	100 + 100	84.6 (12)*	60.4 (7)	93.2 (9)
CGA 173506	25	96.8 (18)	97.8 (6)	93.8 (8)

* Number of trials

Fenpiclonil and CGA 173506 as seed treatments for peas and other non cereal crops

Laboratory, greenhouse and field trials conducted in France and Great Britain have shown fenpiclonil and CGA 173506 to be excellent seed treatments for the control of *Ascochyta* spp. on peas. Table 9 shows the results of three field trials from Great Britain. The seed used in these trials was naturally infected with *Ascochyta pisi* (16% seed infection) or *Ascochyta pinodes* (perfect stage *Mycosphaerella pinodes*) (20% seed infection). Metalaxyl, known to be ineffective against *Ascochyta spp.* was included for protection against *Pythium* and downy mildew. Stand increases (probably due to control of *Pythium*) were seen in the *Ascochyta*-trial but not in the two trials with *Mycosphaerella*-infected seed. Standcounts indicated that both fenpiclonil and CGA 173506 were well tolerated. Control of *A. pisi* and *M. pinodes* was comparable or better than the level of control given by the standard (Table 10).

Table 10. Control of *Ascochyta spp.* with fenpiclonil and CGA 173506. Field trials in Great Britain 1989

Treatment		*A. pisi* Rel. stand (Untr.=100)	% control	*A. pinodes* Rel. stand (Untr.=100)	% control
Untreated	-	(23.3)*	(44.0)**	(29.1)*	(49.0)**
Metalaxyl	70	126.2	9.1	94.9	0
Thiabendazole + thiram	36 + 60	130.0	77.3	86.3	93.3
Fenpiclonil + metalaxyl	20 + 70	127.6	81.8	101.2	89.8
CGA 173506 + metalaxyl	10 + 70	120.1	93.2	94.5	91.8
Number of trials		1	1	2	2

* Plants per 3 metres at 2-trifoliate leaf stage

** % plants with symptoms on hypocytol of *A. pisi* or *A. pinodes*

Fenpiclonil and CGA 173506 are also suitable as seed treatments for other non cereal crops such as maize (Leadbeater et al., 1990), cotton, oilseed rape, peanuts, sunflower and others. As a pre-plant treatment of potatoes, fenpiclonil gives commercial control of a wide spectrum of tuber-borne diseases (Leadbeater and Kirk, 1992).

CONCLUSIONS

The banning of mercury based products for seed treatment has meant the removal of a solution for the control of the major seed-borne diseases of cereals in Europe. It is important for the future management of agricultural crops that these diseases are effectively controlled and that the products available to the farmer continue to be effective. The phenylpyrroles, in particular fenpiclonil and CGA 173506, are extremely effective fungicides for seed treatment and offer control of the major seed-borne diseases of cereals and a wide range of seed-borne diseases on non-cereals. Their novel mode of action combined with this high level of efficacy at low use rates make them an essential part of seed-borne disease management for today and the future.

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CONTROL OF CLUBROOT USING CALCIUM CYANAMIDE - A REVIEW

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ABSTRACT

Control of Plasmodiophora brassicae, the causal agent of clubroot ease, has been associated with the use of calcium cyanamide for many years. The availability of granular formulations, particularly 'Perlka', permits easier and more effective applications and should encourage more widespread use especially since calomel (mercurous chloride) has been withdrawn for reasons related to environmental protection. Results of several field studies of the efficacy of calcium cyanamide are discussed in this review. Early investigations, while using widely variable application rates, generally reported very effective control. Studies in 1979-1983, principally in Scotland, indicated that calcium cyanamide was associated with reductions of 30% in clubroot symptom expression. Efficacy increased when 14-21 days elapsed between application and planting. Pre-planting applications of 1500-1600 kg product/ha reduced disease severity and increased the yield of cabbage and cauliflower in a series of trials in 1987-1989. Calcium cyanamide performed at least equal to, if not better than, calomel without any development of phytotoxic symptoms.

INTRODUCTION

Calcium cyanamide has been used for many years as a nitrogenous fertiliser, with liming properties, which possesses herbicidal and pesticidal effects. These include activity as a broad spectrum fungicide for the control of soil-borne pathogens especially Plasmodiophora brassicae, the causal agent of clubroot disease. Karling (1968) cites 17 studies of calcium cyanamide, 14 in Europe and the USA, for this purpose in the period 1928-1963. Only three investigators failed to obtain any controlling effects. Interest has continued in calcium cyanamide as one of the few soil applied fungicidal fertilisers and has been further stimulated by availability of a granular formulation 'Perlka' manufactured by Süddeutsche Kalkstickstoffe-Werke AG (SKW) (Rieder, 1981). This contains 20% nitrogen and 55% calcium oxide with a particle range 0.2-2.00 mm. Field trials in Scotland over the period 1979-1983 indicated a

reduction of 30% in the expression of root galling symptoms over 8 trials (Dixon & Williamson, 1984), these results being supported by other workers such as Mattusch (1978) in Germany, Zvára (1981) in Czechoslovakia and Ryan (1983) in Eire. Studies using artificially infected seedlings of Brassica rapa ssp. pekinensis cv. Granaat (Chinese cabbage) established that calcium cyanamide prevented disease expression using concentrations of 100 mg a.i. per kg compost (Naiki & Dixon, 1987). Investigations of the mode of action of several soil applied fungicides active against P. brassicae showed that only with calcium cyanamide was efficacy increased where planting was delayed. Calcium cyanamide was most effective in seedling tests where the planting was delayed by 14–21 days after application. This may reflect the conversion by micro-organisms of calcium cyanamide to urea. Naiki and Dixon (1987) suggested that calcium cyanamide may affect the viability of resting spores of P. brassicae. Incubation of resting spores for 24 hr in 800 mg/litre of water reduced root hair invasion by 90% and disease symptom expression by 72%. Effective field applied rates of calcium cyanamide have varied widely for example Walker & Larson (1935) 448–897 kg/ha; Renard (1935) 300–500 kg/ha; Shirama (1955) 1130–1500 kg/ha; Zvára & Rod (1967) 600–1200 kg/ha; Horiuchi et al (1982) 1000 kg/ha and Dixon & Wilson (1983) 1000 kg/ha.

There is continued interest in identifying improved methods for the control of P. brassicae. Limited sources of resistance exist, especially in the cole brassicas, and although a degree of control may be achieved by cultural methods based on liming, good drainage and long crop rotations, and by the use of root dipping in a suitable fungicide, these techniques are often impracticable and unreliable. Control through the incorporation of mercurous chloride (calomel) into plant raising compost has been lost as a result of the ban on this material. This paper summarises data from field experiments between 1987–1989 at the Institute of Horticultural Research, Wellesbourne and from Efford Experimental Station, Hampshire in 1989. These experiments determined the level of disease control achieved by calcium cyanamide (Perlka) used at 1000 and 1500–1600 kg product/ha applied 1–3 weeks before planting or as a split applications applied before planting and 2 weeks after planting.

MATERIAL AND METHODS

Experiments at Wellesbourne used Long Meadow field which has a gleyic, brown sandy loam soil and at Efford Field S11 which has a stony sandy silt loam. In each experiment account was taken of the quantity of nitrogen provided by calcium cyanamide and appropriate rates of standard nitrogen fertiliser were applied to raise the total amount in all plots to similar levels. Calcium cyanamide and fertiliser were broadcast by hand on to measured areas to ensure uniformity of application and pre-planting treatments were rotovated to a depth of 6–10 cm. Treatment rates are given in Tables 1–4, a standard calomel treatment of 1.5 kg a.i./m^3 plant raising compost being included in all experiments except Wellesbourne 1987. Plants were raised in commercial 'Hassy' peat filled modules. Basal fertiliser (N:P:K), herbicides and insecticides were applied as husbandry treatments to all experiments in line with standard commercial practice. Plots were irrigated for plant establishment and to encourage disease development. In the Wellesbourne experiments plots consisted of four rows of 12 (1987 and 1988) or 16 (1989) plants at 40–45 cm spacing with a 1.5 m width of bare soil between plots. Each experiment was a randomised block

design with either six (1987 and 1988) or four (1989) replicate plots per treatment. In 1989, when both cabbage and cauliflower were tested, soil treatments were a randomised block design of main plots and each cultivar was randomised as a sub-plot within the main plots. At each assessment 10 plants were removed from the middle two rows of each plot their fresh weight determined and the roots assessed for symptom intensity using a disease severity index (DSI) with ranges of 0-3 in 1987/88 and 0-5 in 1989, zero representing no galling.

In the Efford experiment (1989) treatments were similar to those at Wellesbourne (Table 3) except that the higher rate of calcium cyanamide was 1600 kg product/ha (Table 4). A randomised complete block design was employed with five replicate plots per treatment. Each plot contained three rows of 22 plants with 60 cm spacing between rows and 45 cm spacing within the rows. Plants were assessed for dry weight, and for symptom intensity using a DSI with a range of 0-3.

RESULTS

Results from the 1987 Wellesbourne experiment are shown in Table 1. At the first harvest, 7 wk after planting, both the low and high rates of calcium cyanamide significantly reduced disease severity. At this time both treatments raised yield and at the high rate the increase was significant.

TABLE 1. Effect of calcium cyanamide on clubroot symptom severity and yield of cabbage cv. Firmhead, Wellesbourne 1987.

Treatment (kg Product/ha)	7 wk Assessment DSI (0-3)	7 wk Assessment Yield per plant (g)	12 wk Assessment DSI (0-3)	12 wk Assessment Yield per plant (g)
Control	2.3	112	2.9	169
1000/ 2 weeks before planting	1.7*	142	2.7	294
1600/ 3 weeks before planting	1.0**	273**	2.1**	956**

* = significantly different from control (P=0.05)
** = significantly different from control (P=0.01)

At the second harvest, 12 wk after planting, plants in the control plots were all severely diseased and stunted. Disease severity was reduced by both calcium cyanamide treatments, but the reduction was only significant in the high rate treatment. This high rate significantly increased yield by a factor of more than five.

Disease pressure in the 1988 experiment at Wellesbourne was less severe than in 1987 (Table 2). At 13 wk after planting all treatments reduced disease severity significantly compared with the control. Yields were greater in all treatments compared with the control.

TABLE 2. Effect of calcium cyanamide on clubroot symptom severity and yield of cabbage cv. Firmhead, Wellesbourne 1988.

Treatment (kg Product/ha)	13 wk Assessment DSI (0-3)	Yield per plant(g)
Control	2.39	1239
Calomel	0.44**	1678**
1000/ 2 weeks before planting	1.56**	1409
1500/ 3 weeks before planting	1.21**	1411

* = Significantly different from control (P=0.05)
** = Significantly different from control (P=0.01)

Severe disease pressure developed in the 1989 Wellesbourne experiment with large clubs forming on untreated plants of both cabbage and cauliflower 3 wk after planting. In the less effective treatments many plants were killed by P. brassicae before the second harvest. As a result of the high disease intensity yields of both cabbage and cauliflower were generally poor.

At the first harvest of cabbage 6 wk after planting, all treatments, except the low rate calcium cyanamide applied 2 wk before planting, reduced disease severity (Table 3). These reductions were significant in the low rate calcium cyanamide treatment applied 1 wk before planting and the high and low rate split treatments. All treatments increased yield with significant increases in the low rate calcium cyanamide treatment applied 1 wk before planting, the high rate treatment applied 2 wk before planting and the high rate split treatment. At the second harvest only 10% of plants in the control plots survived and accordingly no valid statistical comparison was possible between treatments and the control. The low rate calcium cyanamide treatment applied 1 wk and the high rate treatment applied 2 wk before planting and the low rate split treatment gave similar yields to the calomel treatment. The highest yield occurred in the high rate split calcium cyanamide treatment, being greater by a factor of two compared with calomel. The highest proportion of surviving plants was found in the two split calcium cyanamide treatments.

TABLE 3. Effect of calcium cyanamide on clubroot symptom severity and yield of cabbage cv. Firmhead and cauliflower cv. White Rock, Wellesbourne 1989.

Treatment (kg Product/ha)	6 wk Assessment DSI (0–5)	6 wk Assessment Yield per plant (g)	13 wk Assessment DSI (0–5)	13 wk Assessment Yield per plant (g)	13 wk Assessment % survival
Cabbage					
Control	2.5	86	4.9	22	10
Calomel	1.6	142	2.1	424	75
1000/ 1 week before planting	1.0*	187**	3.2	402	73
1000/ 2 weeks before planting	2.9	99	4.4	103	48
1500/ 2 weeks before planting	1.9	235**	3.5	427	85
1500/ 3 weeks before planting	2.3	125	3.9	245	73
1000kg split+	0.9**	149	2.0	596	98
1500kg split+	0.8**	225**	1.6	844	98
Cauliflower					
Control	3.1	26	5.0	0	0
Calomel	1.0**	58*	2.4	261	75
1000/ 1 week before planting	1.7**	72*	3.2	226	78
1000/ 2 weeks before planting	3.0	59*	5.0	0	0
1500/ 2 weeks before planting	1.8*	46	4.0	199	40
1500/ 3 weeks before planting	2.6	48	4.2	143	43
1000kg split+	0.9**	163**	2.6	296	83
1500kg split+	0.6**	175**	2.4	517	83

* = Significantly different from control (P= 0.05)
** = Significantly different from control (P= 0.01)
+ = Split treatments of 500 kg or 750 kg product/ha applied 1 day before planting and again 2 weeks after planting.

At the first harvest of cauliflower (Table 3) calomel and all calcium cyanamide treatments, except the low rate applied 2 wk before planting and the high rate applied 3 wk before planting, significantly reduced disease severity. All treatments increased yield, significantly so in the calomel, low rate calcium cyanamide and split rate treatments. By the second harvest all plants were killed by P. brassicae in the control and low rate calcium cyanamide treatment applied 2 wk before planting. The lowest disease severity values were recorded for calomel and in the low and high rate split calcium cyanamide treatments, the low rate split calcium

cyanamide treatment producing yields similar to those of the calomel treatment. High rate split calcium cyanamide increased yield two fold over the calomel treatment. The highest proportion of surviving plants occurred in the calomel, low rate calcium cyanamide applied 1 wk before planting and two split calcium cyanamide treatments.

At the Efford site all treatments significantly reduced clubroot severity 5 wk after planting with the calomel and high split rate calcium cyanamide treatments being most effective (Table 4).

TABLE 4. Effect of calcium cyanamide on clubroot symptom severity and yield of cauliflower cv. White Fox, Efford 1989

Treatment (kg Product/ha)	DSI (0-3)		Curd Weight(g)
	5 wk Assessment	14 wk Assessment	
Control	2.6	1.9	812
Calomel	0.4*	1.3	853
1000/ 1 week before planting	1.4*	1.1	1000
1000/ 2 weeks before planting	1.6*	0.6*	1035*
1600/ 2 weeks before planting	1.0*	1.0*	1017*
1600/ 3 weeks before planting	1.3*	0.0*	901
1000 Split +	1.3*	0.6*	861
1600 Split +	0.4*	0.5*	883

* = Significantly different from control (P = 0.05)
\+ = Split treatments of 500 kg or 800 kg product/ha applied 1 day before planting and again 2 weeks after planting.

At 14 weeks after planting disease severity was reduced by all treatments, significantly in all of the calcium cyanamide treatments except the low rate applied 1 wk before planting. All treatments increased curd weight but differences were only significant for the calcium cyanamide treatments applied 2 wk before planting.

DISCUSSION

In experiments at Wellesbourne, calcium cyanamide at 1500-1600 kg product/ha almost invariably reduced disease severity and increased yields of cauliflower and cabbage 6-8 wk after planting, this effect persisting to crop maturity in each year. At maturity these differences were statistically significant in most cases. At the lower rate of 1000 kg product/ha disease control was generally less effective. Nevertheless substantial reductions in disease severity and increases in yield resulted

from treatments at this rate in 1987 and 1989 when disease pressure was extreme. In 1989, when most of the plants in control treatments were killed by P. brassicae, several calcium cyanamide treatments gave equivalent or better disease control and yields than calomel. At Efford (1989) all treatments significantly reduced disease severity at the first assessment, the most effective being calomel and the split calcium cyanamide application at 1600 kg product/ha. All calcium cyanamide treatments except the 1000 kg product/ha applied 1 wk before planting, significantly reduced clubroot at assessment 2. Calcium cyanamide at 1000 and 1600 kg product/ha 2 wk before planting significantly increased curd weight of cauliflower.

Unpublished results from similar field studies made at Starcross Experimental Station, Devon, and at Scottish Agricultural College (SAC), Auchincruive were less clear cut. In the Starcross experiments there was inadequate disease control by all treatments including calomel while at Auchincruive disease pressure was relatively low and none of the treatments had a significant effect on yield or disease severity. Nonetheless the Auchincruive experiment demonstrated that in the absence of severe disease no depressing effect on yield resulted from any of the calcium cyanamide treatments.

Field evaluations of chemicals for clubroot control often produce results which are difficult to interpret statistically owing to the patchy nature of disease incidence and the major influence of weather conditions on pathogen development. Results obtained in the above studies consistently showed that pre-planting calcium cyanamide treatments at 1500-1600 kg product/ha provided good disease control and may be as or more effective than previously available standard calomel treatments. These data serve to reinforce previous studies over the past century showing that calcium cyanamide, especially when applied over several seasons, reduces the incidence and severity of clubroot disease. There are also possibilities for integrating the use of calcium cyanamide with other forms of pathogen control. Coulshed & Dixon (1990) reported improved efficacy with combinations of calcium cyanamide and basamid. Treatments of this type are attractive since they may be applied mechanically on a field scale to vulnerable crops such as oil seed rape. Despite being more expensive than calcium ammonium nitrate fertilisers, the slow release of nitrate from calcium cyanamide is attractive since it does not contaminate ground waters and hence there is no environmental pollution which accompanies the use of other forms of nitrogenous fertiliser. When this is allied to its liming value and fungicidal activity calcium cyanamide clearly offers an environmentally acceptable alternative to calomel for clubroot control.

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