SESSION 7A

PEST AND DISEASE PROBLEMS IN COMBINABLE BREAK CROPS

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INVITED PAPERS 7A-1 to 7A-6

PESTS AND DISEASES OF SOME NEW AND POTENTIAL ALTERNATIVE ARABLE CROPS FOR THE UNITED KINGDOM

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ABSTRACT

Overproduction of cereals in the UK and within the EEC has intensified interest in alternative crops. Many broadleaved combinable crops are now being evaluated for their place in UK agriculture e.g. linseed, evening primrose, borage, lupin, sunflower, grain maize and navy bean. None has been grown in the UK for sufficiently long, or on a sufficiently large area, for pests and diseases to have become a major constraint to production. Information on pests and diseases of these crops, hitherto available only to specialists or scattered in the literature, is collected together to supplement current guides to the cultivation of alternative crops. Those pests and diseases which might cause problems in the future, if crop areas expand, are briefly reviewed and those reported on recent commercial crops are noted as a record of the present situation.

INTRODUCTION

In the EEC there is substantial overproduction of several commodities. In the arable sector production of cereals has exceeded demand and surplus stocks, at 15Mt in 1986, are predicted to reach 80Mt by 1991 if current trends continue. Thus there is a clear need to examine alternatives either to meet the need for replacement of imported commodities or to open up new indigenous or export markets. The need for diversification of arable cropping in the UK is probably more acute than elsewhere in Europe and there is, therefore, much interest in new combinable break crops which might offer the farmer maintained income and profitability.

A range of alternative crops has been considered for the UK (Bunting 1974, 1976; Williams 1978, 1986) and a recent review has further widened the scope of what could be considered for cultivation here (Carruthers 1986a,b). In this paper we exclude oilseed rape, peas and field beans on the assumption that they will continue to be the most widely grown broadleaved combinable crops, helped by the EEC oil- and protein-crop subsidies. Our selection of crops is not an endorsement of their status or a judgement of which has the potential to become an established part of British agriculture. Indeed, Carruthers (1986a) concludes that there is no single alternative crop enterprise which will use a substantial area of land, but that there is a range of possibilities whose contribution will depend on the extent of the market development or the availability of suitable cultivars.

Most of the alternative crops require further improvements through breeding, including better adaptation to our climate, and the development of appropriate agronomy. These factors are much greater than any current problems from pests and diseases. Nevertheless, we have reviewed the

possible pest and disease problems which may impose constraints on production in the future.

Many new crops, especially those grown on small isolated areas or those not native to the UK, enjoy an initial period of freedom from pest and disease attack. For this reason there is little well-documented information on some crops. Much of our information has come from personal communication with specialists or from the popular farming press. Our aim is to bring this information together to complement general guides on the cultivation and economics of alternative crops (e.g. Long *et al.* 1984; Cousins, 1985; Dover, 1985, 1986). Few agrochemicals are yet recommended for use in the UK on the crops we mention, although trials are in progress, so to avoid premature endorsement of either efficacy or need we do not consider methods of control.

Some crops considered here have been grown more widely in continental Europe and elsewhere. Experience in these areas may help to anticipate possible pest and disease problems in the UK but we realise that especially in an agricultural context, it is usually profitless and subsequently embarrassing to attempt to foretell the future. This applies as much to the future of some alternative crops as to the problems which may beset them. No truly new field crop has been introduced into European agriculture since Roman times. Some of the crops currently under consideration as alternatives were known in UK agriculture centuries ago, for example, Wilson (1848) commented that there was no reason why quinoa (*Chenopodium quinoa*) should not become as common as barley. Thus a knowledge of the origin and history of such crops, together with records of their pests and diseases, can help give perspective to the present search for viable alternatives to wheat and barley.

CROPS CURRENTLY GROWN OR UNDER INVESTIGATION AND DEVELOPMENT FOR THE UK

Linseed (*Linum usitatissimum*)

Estimated UK area in 1986, 7200ha, and market potential,80,000t.

This crop is one of the oldest in the UK and was grown as flax for fibre, particularly in Northern Ireland, in 1914-18 and 1939-45. Its recent resurgence as an oilseed crop is linked with improved support arrangements within the EEC. Muskett & Colhoun (1947), Keay (1948) and Moore (1959) record the following diseases in the UK: seedling diseases (*Alternaria linicola*, *Colletotrichum linicola*, *Rhizoctonia solani*), foot and stem rots (*Ascochyta linicola*, *Phoma* sp., *Polyspora lini*, *Stemphylium botryosum*), grey mould (*Botrytis cinerea*), wilt (*Fusarium oxysporum*, f.sp. *lini*, *Fusicladium lini*), rust (*Melampsora lini*), powdery mildew (*Oidium lini*). The importance of some diseases has changed with the development of the crop as an oilseed. There are now statutory requirements for certified seed that contamination by *Botrytis*, or by *Alternaria* plus *Colletotrichum*, *Fusarium* and *Phoma* spp. shall not exceed 5%.

Since 1984 *Botrytis* has been widespread, occasionally causing loss of capsules, especially during the wet summer of 1985 and in lodged areas of crops. Incidence of *Alternaria linicola* was low in 1984 but high in some crops in 1985 particularly in the East Midlands, Hereford and Lancashire, leading to the rejection of some seed crops, even after the application of fungicides. It was recorded in 1986 on crops in Oxfordshire and Hampshire where infection was sufficient to cause concern. One crop grown from imported seed was reported to have failed in 1985 due to *Colletotrichum*. A

low incidence of wilt, presumed to have been caused by *Fusarium*, was reported on two crops in Lincolnshire. Powdery mildew was noted late in the season on some crops but caused little damage at this stage. The first authoritative record on mainland Britain of *Sclerotinia sclerotiorum* stem rot on linseed (Mitchell 1986) is of greater concern. Patches of lodged and prematurely senescing plants were seen in August and September 1985 in three locations in Hereford and Lancashire. Incidence ranged from trace amounts to 15% plants affected, with most occurring in an area drilled at much greater than the recommended seed rate. Where symptoms were most severe the seed used for drilling was later found to contain 33 sclerotia/kg. Examination of harvested seed from the three locations revealed an incidence of sclerotia ranging from 33 to 612/kg seed. The expansion of oilseed rape in the UK has led to an increased incidence of *Sclerotinia* stem rot. Its occurrence on linseed indicates the need for continued care in the selection of crop rotations. During past periods of expansion of the linseed crop the incidence of *Ascochyta* foot rot and rust increased. These and others may reappear if the crop continues to expand to the >20,000 ha predicted by some commercial sources.

Jones & Jones (1984) record that leatherjackets (*Tipula paludosa*) and flea beetles (*Aphthona euphorbiae* and *Longitarsus parvulus*) can damage linseed, the latter often abundant but rarely causing crop failure. Since 1984, few pest problems have been reported. At one location in 1986 in Yorkshire a leatherjacket population of 530,000/ha damaged seedlings in a late April-sown crop and decreased plant population from an expected 700 plants/m² to 250/m² in the worst affected areas. An adjacent earlier sown crop was less affected. No damage by flea beetles has been reported. Thrips caused gross distortion on seedlings of one crop in the south east and distortion before flowering in another in 1986. Each year a few plants in several crops have been slightly affected by tortrix moth larvae webbing the flowering heads. Grazing by pigeons and rabbits has occurred and is considered to be the main pest damage at present. Grazing can damage early growth, and fields near woodland may be vulnerable, but unless damage is severe crops normally continue growth by secondary branching.

Evening primose (*Oenothera biennis*)

Estimated UK area in 1986, 1000ha.

This plant, a native of the Americas and related to the willow herbs, is widely known as an ornamental and a weed on waste land, but is new as a field crop. A programme of hybridisation and selection began in the 1960s and the crop is now grown commercially as a high-value oilseed, providing a source of high quality oil rich in the essential fatty acid, gamma linolenic acid.

Twenty-six species of fungi and an unidentified virus are recorded as causing disease on *Oenothera* spp. in the USA; half of these have been recorded specifically on *O. biennis* (Anon 1960). The list comprises *Botrytis cinerea*, a leaf gall, powdery mildew (*Erysiphe polygoni*) and downy mildew (*Peronospora arthuri*), six rust species, seven leaf spots, seven species causing stem and capsule lesions and two root rots. The total number of species recorded on *Oenothera* spp. from many parts of the world exceeds seventy, although many are presumed saprophytic on dead stems or capsules. Walker (1985) mentions that mildew on crops in Europe caused little damage, in contrast to severe infection seen in the USA. In the UK, Moore (1959) records only the leaf spot caused by *Septoria oenotherae*,

although garden plants are known to suffer powdery mildew (*Oidium* spp.) and, on heavy soils, a root rot. Some pathogens on *Oenothera* spp. in the USA have been recorded on related genera or other hosts in the UK e.g. *Peronospora arthuri*, *Stemphylium botryosum* and *Puccinia epilobii*. In Europe there have been records of *Septoria oenotherae*, *Entyloma oenotherae* and *Synchytrium fulgens* on evening primrose. Given the widespread occurrence here of various native willow herb weeds (*Epilobium* spp. and *Chamaenerion angustifolium*) it is possible that these may in future provide a source of pests and diseases for evening primrose crops nearby.

There have been few reports of disease on recent evening primrose crops in the UK. *Septoria oentherae* leaf spot was seen at low incidence in crops in Cambridgeshire in 1984 and was widespread on lower leaves in 1985 usually causing little damage. However, infection caused failure of some crops in the spring of 1985 and symptoms were severe during the summer in other crops in the Midlands. *S. oenotherae* is seed-borne and these attacks are thought to be due to the lack of fungicide seed treatment. Symptoms were recorded in 1986 in Kent, West Sussex and Berkshire, but incidence was generally lower, perhaps because most crops were then sown with fungicide-treated seed. *Botrytis cinerea* occurred in 1986 on stems of crops in Essex and Cambridgeshire causing plant death, and was severe on stems and leaves in lodged patches of a crop in Hertfordshire. The latter crop was densely sown and very tall, perhaps due to use of excess nitrogen fertilizer. These features would increase the likelihood of damage by *Botrytis*. Indeed, when plants have been grown at high density under humid glasshouse conditions, some seedlings have been infected with *Pythium* and some plants have been killed by *Botrytis* prior to transplanting in the field. Those transplanted with *Botrytis* infection usually recovered and grew normally. Both *Botrytis* and powdery mildew have been found on rosette leaves in late summer and autumn and later on mature plants. Powdery mildew occurred on mature plants in four crops in Kent and West Sussex in 1986.

Insect pests have caused damage to crops grown in other parts of the world. Walker (1985) mentions occasional damage from flea beetles in Europe, massive infestations of flea beetles and cantharid ground beetles in the USA and defoliation of some plots by the Queensland grass beetle (*Rhyparida morosa*) in Australia. However, reports so far from the UK are few. Minor damage to leaves has been caused by tortrix moth larvae and flea beetles and to roots by leatherjackets. In one crop in Leicestershire, adjoining a copse filled with rosebay willow herb, froghoppers (mainly *Philaenus spumarius*) caused distortion to some plants in June and feeding by adult flea beetles (*Altica lythri*) and later their larvae (5/plant) caused loss of some plant tops. These pests were also found in the adjacent stand of rosebay willow herb and their populations were largest in the crop nearest this weed. Adults of *A. lythri* were seen on other commercial crops in Cambridgeshire up to 1985 but none were reported in 1986. Bees are normally very active on the crop during flowering and if pest numbers justify control by insecticide this could be difficult to achieve without harm to the bees. Pollen beetles (*Meligethes aeneus*) have caused a very little damage by aborting flowers, but could become a greater problem in areas where populations are maintained on winter and then spring oilseed rape crops. Some growers have reported minor damage from hawk moth caterpillars. Grazing by pigeons and rabbits has not been reported, although rooks have been known to pull up plants transplanted in peat blocks. Sparrows and finches eat seed in mature capsules but there have been no reports from commercial crops. Stem

nematodes can occur on garden evening primroses and root knot nematodes (*Meloidogyne* sp.) have been recorded on related *Epilobium* spp. but there are no known reports of nematode damage in commercial crops of evening primose.

Borage (*Borago officinalis*)

Estimated UK area in 1986, 500ha.

This plant of Mediterranean origin has been grown as a herb in England at least since the 13th century and is also known as a garden-escape. It has also been grown for centuries in Europe and the USA. Since 1983 it has been grown as a field crop in the UK, as an oilseed particularly rich in gamma linolenic acid. Recent experiences with the crop have been discussed (Anon. 1985a,b; Cutting 1985; Long 1985a) and Jones (1986) has outlined the merits and problems of both borage and evening primrose when grown as commercial crops.

There are few records in the literature of either pests or diseases of borage. In the USA there is a single reference to a *Ramularia* sp. causing a leaf spot on borage (Anon. 1960), while other herbaceous boraginaceous genera there can be attacked by rusts, leaf smut, powdery and downy mildew, *Rhizoctonia* damping off, *Phymatotrichum* root rot, *Sclerotinia* stem rot, *Septoria* and *Cercospora* leaf spots, *Sclerotium* blight, *Verticillium* wilt, *Botrytis* grey mould, root nematodes, aster yellows mycoplasma and beet curly top virus. Elsewhere *Sclerotinia sclerotiorum* has been recorded on borage and in Europe there are records of the rust *Puccinia symphiti-bromorum* alternating between *Bromus* spp. and many Boraginaceae, *Fusarium avenaceum* on roots, and the powdery mildew *Erysiphe horridula*. *Cercospora* spp. occur on other boraginaceous genera in Europe and *Ascochyta boraginis* has been recorded on leaves of borage in the Ukraine. In the UK a race of *Entyloma serotinum* leaf spot has occurred on the related genus *Symphytum* and *Erysipha horridula*, although not recorded on borage itself, is likely to be present in this country. Thus there are a number of possible pathogens which may appear in borage crops in the future, although none, other than those with a wide host range such as *Botrytis*, *Fusarium*, *Sclerotinia* and perhaps powdery mildew, seems likely to cause serious damage.

In general there has been no significant pest or disease damage in the UK (Anon. 1985a), although one case of severe powdery mildew caused crop failure in Essex and leatherjackets destroyed 3 ha in the centre of a 5 ha crop in Yorkshire; this damage was associated with a rather loose seedbed in the centre of the field. Black aphids (*Aphis fabae*) have been reported on several crops but were not thought to be damaging. It may be that the impenetrability of a dense, well-grown borage crop with robust, coarse, hairy plants will act as a deterrent to regular inspection for pests and diseases. Very large numbers of bees are attracted to borage flowers and, as with evening primrose, these would present a limitation on use of insecticide should this be necesssary in future.

Lupins (*Lupinus* spp)

Estimated UK area in 1986, 600 ha, and market potential as soya replacement, 1.3Mt.

Lupins have been cultivated for *c.* 3000 years near their centres of

origin in the Mediterranean basin (*Lupinus albus*) and South America (*L. mutabilis*). Other species (mainly *L. angustifolius* and *L. luteus*) have come into cultivation more recently and are still in the process of development. All have been grown for either grain, forage or green manuring of light soils. The latter uses are recorded in the UK in the 1850s. Currently the largest areas are grown in Eastern Europe, Poland, the USSR and Western Australia. Since the 1960s 'sweet' types have been developed with very low alkaloid content and it is these that are being considered as protein-rich alternative crops for Western Europe. The introduction of an EEC subsidy for lupins in 1984 further stimulated intense interest within the farming community (Gladstones 1970; Bélteky & Kovács 1984; Williams 1986).

Over 160 species of fungi have been recorded on *Lupinus* spp. worldwide, many of which may cause disease under appropriate conditions. Indeed, Gladstones (1970) predicted that, should lupins become established in any area as a major crop, there could be little doubt that diseases would become one of their main limiting factors. Those pathogens recorded in Europe and the UK (Moore 1959; Baker 1972; Anon. 1976-1979; Williams *et al.* 1978, 1979; Hale & Kay 1983; Anon. 1984; Bélteky & Kovács 1984) include; brown leaf spot (*Pleiochaeta setosa*, *Phyllosticta* sp.), grey leaf spot (*Stemphylium botryosum*), anthracnose (*Glomerella cingulata*), wilt (*Fusarium oxysporum*, *Verticillium dahliae*, *Cylindrocladium scoparium*), root rots (*Thielaviopsis basicola*, *Pythium debaryanum*, *Rhizoctonia solani*, *Helicobasidium purpureum*, *Aphanomyces euteiches*, *Armillaria mellea*, *Fusarium solani*, *F. avenaceum*, *F. culmorum*), foot rots (*Fusarium* sp., *Phytophthora cryptogea*), stem rot (*Sclerotinia sclerotiorum*), grey mould (*Botrytis cinerea*), powdery mildew (*Erysiphe* spp. *E. polygoni*, *E. trifolii*), rust (*Uromyces lupinicolus*, *U. anthyllidis*); bean yellow mosaic, bean common mosaic, cucumber mosaic, clover yellow vein, alfalfa mosaic, pea mosaic and tomato spotted wilt viruses and clover phyllody. Susceptibility to *Fusarium oxysporum* can be increased by infection with clover yellow vein virus and to *F. solani* and *Rhizoctonia solani* by bean yellow mosaic virus.

With all these possibilities for damage spring sown crops are likely to remain healthier than those sown in the autumn which may suffer unacceptable losses from root-rotting pathogens. Any attempts at wide-scale autumn sowing are also likely to be under threat of attack by the very destructive leaf spot *Pleiochaeta setosa*. To minimise the risk of heavy losses from soil-borne pathogens with wide host ranges all legume species should be regarded as the same crop when planning cropping sequences. In commercial crops in the UK since 1984 few diseases have caused concern; *Botrytis* occurred during flowering and pod set in the wet summer of 1985 and there have been isolated instances of *Sclerotinia* stem rot. During the same period in France *Pleiochaeta* has occurred and there is concern that wide-scale cultivation might bring problems from rust (Dodgson 1985).

Little has been published on pests of lupins. Aphids, thrips, weevils and leatherjackets have been of variable importance in the past, mainly attacking alkaloid-free types. Pigeons, crows, rooks, pheasants, rabbits and hares have caused serious, although sporadic, grazing damage. Root nematodes have been reported, but there is no evidence that they are likely to become a problem in any species. Budworms, cutworms, springtails and earth mites have been recorded in Australia where *Lupinus luteus* has been very susceptible to insect attack (Gladstones 1970, 1982; Anon. 1976-1979;

Bournoville 1979; Michael *et al.* 1982; Bélteky & Kovács 1984).

In UK crops since 1984 there have been reports of pea and bean weevil (*Sitona lineatus*), thrips, aphids and tortrix moth larvae, and grazing by birds and mammals. With the exception of the latter which has been common and often serious in trials, other damage has not been significant. However, symphylids caused severe damage and crop failure in one crop in the cold spring of 1985 in Yorkshire. Since the first appearance in the UK in 1981 of the large American lupin aphid, *Macrosiphum albifrons* (Stroyan 1981), and its subsequent rapid spread here on lupin species (Carter *et al.* 1984), it has been recorded on commercial crops and gives cause for concern in the future. Other likely problems for the future seem to be thrips and adult and larval damage by *Sitona* spp.

Sunflower (*Helianthus annuus*)

Estimated UK area in 1986, 200 ha, and market potential, 77,000t.

Sunflower, a native of North America and first cultivated there *c.* 3000 years ago, was introduced to Europe in the 16th century and its suggested use as a source of oil was recorded in England in 1716. Breeding improvements made in Russia preceded its reintroduction to North America in the late 19th century where it was grown for silage, and since the 1940s for edible oil. Its subsequent successful cultivation in many parts of the world allows it to rank now with soya bean, peanut and rapeseed as a major source of edible oil. Recent interest in sunflower began in the UK in the mid-1970s and has been stimulated by the introduction of an EEC oilseeds subsidy and the succcessful northward spread of cultivation in France. The area grown in France has increased rapidly since 1980 and is now (*c.*850,000 ha) double that of oilseed rape.

Over 90 species of fungi have been recorded on sunflower, a third of them capable of causing disease. Many of the pathogens are common to areas of sunflower production worldwide and have been reviewed recently (Carter 1978; Kolte 1985; McMullen 1985). Understandably, few of these pathogens have been recorded in the past in the UK (e.g. *Botrytis cinerea, Itersonilia perplexans, Sclerotinia sclerotiorum*, beet western yellows group virus (Moore 1959; Baker 1972; Russell *et al.* 1975), although several other unspecialised pathogens capable of causing minor diseases on sunflower elsewhere occur here on *Helianthus tuberosus* or other hosts. Recent experiences in France (Guérin 1984; Lamarque 1985; Anon. 1986) may provide the most relevant guide to pathogens which may occur in the future in the UK. Expansion in sunflower area in France has been accompanied by rapid changes in the disease problems encountered. The following are considered to be important in France: downy mildew (*Plasmopara halstedii*), first seen 1966; wilt, stem and head rot (*Sclerotinia sclerotiorum*); grey mould (*Botrytis cinerea*); wilt (*Verticillium dahliae*); charcoal rot (*Macrophomina phaseoli);* leaf, stem amd head spot (*Alternaria helianthi*), first seen 1982; stem canker (*Phomopsis helianthi*), first seen 1984. Boron deficiency, and malformations of the flowers from a variety of causes, have also been noted.

Plasmopara halstedii is widespread and can be extremely destructive wherever sunflower is grown extensively in temperate regions. At least four physiologic races are known. Most American and European sunflower hybrids that might be grown in the UK are resistant to those races known on each continent up to 1980, but not to all four races, and incomplete

resistance is known in some sunflowers possessing resistance genes. The appearance of the new American races in Europe would give cause for concern (Morice 1984), and since seed production methods may not completely exclude latent infection carried in the seed (Gulya 1986) vigilance even in the early stages of cultivation in the UK is essential. The disease is most serious on heavy soils under conditions of low temperature and high rainfall, such as might be common in the UK. There is a record indicating that the pathogen has occurred in the UK (Leppik 1962), but this is questionable and thought to be based on examination of seed samples rather than plants. Climatic conditions in the UK may also favour *Sclerotinia sclerotiorum* head rot, since the requirement for heads to be wet for an unbroken period of 42 h is unlikely to be a limiting factor here and no current hybrids are resistant. For similar reasons *Botrytis* is likely to be a greater problem in the UK than in France. Moreover, if higher plant populations are used in UK crops this could increase leaf, stem and root diseases generally. In contrast, the diseases caused by *Phomopsis*, *Alternaria* and *Macrophomina*, although important in eastern Europe and recently in France, are favoured by prolonged periods of high temperature, humidity and drought stress, respectively, so seem unlikely problems for the UK. In view of the apparent prevalence of beet western yellows virus in UK oilseed rape crops it is possible that this virus (Russell *et al.* 1975) may become more common in UK sunflower crops.

Since 1984 the only diseases reported in UK sunflower crops were *Botrytis* causing head rot in Avon, Hampshire, Hertfordshire and Leicestershire, particularly following the wet summer of 1985, and isolated plants with *Sclerotinia* head rot in Hampshire, Leicestershire and in trials at Rothamsted in 1985 (Rawlinson *et al.* 1986) and *Sclerotinia* wilt and stalk rot in similar trials in 1986. Many plants in one crop in Hampshire in 1986 suffered serious damage from *Sclerotinia* stalk rot.

Insect pests of sunflower in the USA have been reviewed recently (Carter 1978; McMullen 1985) and may give a guide to the type of damage to be expected in western Europe. Information more pertinent to the UK on insects and other pests is available from France, but so far there has been little damage (Guérin 1984; Ballanger *et al.* 1985; Anon. 1986; Harriot 1986). The range of pests encountered in France at the seedling stage includes: leatherjackets, wireworms, symphylids, millepedes, slugs, nematodes (*Pratylenchus neglectus*) and birds; on young plants, weevils and leaf miners; and on maturing plants, capsid bugs (*Lygus* spp.), leafhoppers, thrips, aphids (*Brachycaudus helichrysi*, *Aphis fabae*), noctuid and pyralid (*Homoeosoma nebulella)* moth larvae. Most insect pests in France seem in balance with their natural predators, but pyralid larvae are seen as a potential danger. Work is in progress in France on aphid control and bird repellents, and in the USA some sunflower breeding is devoted to morphological types less prone to seed eating birds but these seem unlikley to be suitable for UK cultivation.

In UK crops since 1984 the only serious pest problems have arisen from birds grazing young seedlings and eating ripening grain. Three crops in Hampshire, (6 ha in total) and a 6ha crop in Suffolk were completely lost during establishment, with evidence of selective grazing on different cultivars. Minor damage has been caused by tortrix larvae, leaf miners and looper caterpillars in Bedfordshire and thrips in Hampshire. Aphids (*Brachycaudus helichrysi* and others) and pollen beetles (*Meligethes aeneus*) were recorded in trials at Rothamsted but caused little damage. *B. helichrysi*, *Aphis fabae* and *Myzus persicae* were recorded in Oxfordshire in

1986 but again caused little damage.

Grain maize (*Zea mays*)

Estimated UK area, <50 ha, and market potential, 1.3 Mt.

Maize, a native of North America, was first cultivated in England in the 16th century. A rapid expansion of forage maize production began in northern Europe in the 1960s and this was reflected in the UK up to the late 1970s, but the area grown has since declined here. Maize grown for grain in the UK has been a minor interest until revived recently for cultivation on poorer soils (Cooksley 1986).

UK maize crops, whether grown for forage, grain or sweet corn, share the same range of pests and diseases few of which are of any signficance at the northern limits of the crop's range. These have been reviewed by Cook (1978) and little has changed since that review. Frit fly (*Oscinella frit*) and stalk rot (*Fusarium* spp.) remain the most common problems. The latter is becoming more important on sweet corn and may become so on future grain crops, otherwise recent crops have suffered a range of conspicuous but innocuous spots and blotches and remain basically healthy.

Navy beans (*Phaseolus vulgaris*)

Estimated UK area, < 50 ha, and market potential, 80,000t.

Phaseolus spp., native of Central and South America, have been grown in the UK since the 16th century. Navy bean, a small white dry-seeded type developed as a convenience food and source of protein, was successfully trialled here in the 1970s. As a result, breeding programmes were begun to improve the unadapted types then available. A range of cultivars more adapted to UK conditions are now being evaluated in further trials.

Evans & Davis (1978) have reviewed the pests and diseases occurring on navy bean in the UK. Few problems have been seen since that time although three have the potential to be important in future commercial crops; bean seed fly (*Delia platura*) causing loss of seedlings, grey mould (*Botrytis cinerea*) at and after flowering, and halo blight (*Pseudomonas syringae* pv. *phaseolicola*) especially if growers use self-grown seed.

OTHER COMBINABLE CROPS IN CULTIVATION

Mustard (*Brassica juncea* & *Sinapis alba*), currently grown on *c.* 3500 ha in the UK, shares pest and disease problems with oilseed rape and other brassica crops although escapes many of these because it is spring sown. Few problems have been noted recently and apart from beet cyst-nematode (*Heterodera schachtii*), other pests at flowering time and leaf diseases have caused only minor damage (Hemingway 1985). The incidence of dark leaf and pod spot (*Alternaria* spp.) has increased in recent years, coincident with the expansion of oilseed rape. In the late spring of 1986 isolated crops had increased damage by pollen beetle (*Meligethes aeneus*) and seed weevil (*Ceutorhynchus assimilis*).

Vetches (*Vicia* spp.), grown on *c.* 200 ha for seed and bird food, share some pest and disease problems with other cultivated legumes, but none has been reported as significant in recent crops.

Poppy (*Papaver somniferum*) was grown in the UK on *c.* 300 ha in the

1960s for the pharmaceutical industry. A few crops (*c.* 45ha) have been grown in 1986 as a source of high quality oil (Long 1986d) but, apart from infestations of black aphids during flowering, there have been no reports of serious damage by pests or diseases. Of the ten pathogens recorded an poppy in the UK in the past, downy mildew (*Peronospora arborescens*) and black mould (*Cladosporium herbarum* and *Alternaria* sp.) seem most likely to recur in future commercial crops.

PLANTS UNDER CONSIDERATION FOR THE FUTURE

In the search for diversification of cropping and for renewable sources of some commercial commodities a wide range of plants is now being considered in the UK, continental Europe and the USA (Risi & Galwey 1984; Long 1985b, 1986 a,b,c; Hirsinger 1985; Hinman 1986; Cutting 1986). These plants have been selected as possible sources of either oil, protein, fats, cellulose, pharmaceutic products, spice or selected for culinary use. Their pests and diseases are beyond the scope of this paper but the names of those considered for the UK are recorded for completeness and topicality: Quinoa (*Chenopodium quinoa*), lentil (*Lens culinaris*), coriander (*Coriandrum sativum*), fenugreek (*Trigonella* sp.), caraway (*Carum carvi*), chervil (*Anthriscus cerefolium*), oil-rich pea (*Pisum sativum*), meadowfoam (*Limnanthes alba*), *Amaranthus* sp., *Camelina* sp. *Cosmea* sp., *Cuphea wrightii* and spp., *Crambe* sp., soya (*Glycine max*).

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BACTERIAL BLIGHT OF COMPOUNDING PEAS

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ABSTRACT

Bacterial blight (Pseudomonas syringae pv pisi) was detected in the majority of seed stocks tested of the compounding pea cv Belinda grown in Britain in 1985. In all cases, race 2 of the pathogen was isolated. Tests of the resistance/susceptibility of compounding pea cultivars to the five known races of the pathogen indicated that the main Continental cultivars (e.g. Belinda) were susceptible to races 2, 4 and 5 while the main British cultivars (e.g. Progreta) were susceptible only to race 4.

INTRODUCTION

Bacterial blight of peas caused by Pseudomonas syringae pv pisi is a seed-borne disease which has been reported in most of the pea-growing areas of the world although its ocurrence is sporadic. It had not been detected in the field in Britain until 1985 when infection was found during a routine inspection by ADAS in a crop of the compounding pea cv Belinda. The disease is said to cause significant losses when environmental conditions favour its development (Lawyer 1984) and has been particularly troublesome in crops grown in New Zealand under cool wet conditions (Smith & Close 1966, Young et al. 1969). The pathogen is known to occur as five distinct races which are distinguished by the range of pea cultivars they attack.

This paper presents evidence of the distribution of the pathogen in seed stocks of cv Belinda and other cultivars collected by the Plant Health Branch of the MAFF and submitted for testing at Wellesbourne. It also includes a preliminary assessment of the susceptibility/resistance of some of the compounding pea cultivars to the five races of the pathogen.

MATERIALS AND METHODS

Seed samples were collected by MAFF from crops harvested in all the main pea-growing areas. Seed samples of 1 kg were tested in the laboratory by a method originally devised for halo-blight of beans but now adapted for many other seed-borne pathogens, especially Pseudomonas spp. (Taylor 1970, Taylor 1984). Isolates of the pathogen were identified serologically and by host inoculation tests using selected cultivars from the differential series (Table 1). Pathogenicity was tested by stem inoculation of young pea seedlings.

RESULTS

A total of 25 seed stocks was examined for infection. This included 15 stocks of cv Belinda and 10 stocks of other cultivars including Birte, Maro, Progreta and Stehgolt. Two thirds of the Belinda stocks were infected with pv pisi race 2; no other race was identified. Infection was found in seed from Bedfordshire grown from the original Dutch seed, imported in the winter of 1983-84 and from crops as far apart as Wiltshire and Berwickshire. No infection was detected in tests of 10 stocks of other cultivars.

TABLE 1

Race identification of bacterial blight according to pathogenic reaction of pea cultivrs

Differential cultivars	Race of pathogen 1	2	3	4	5
Kelvedon Wonder	+	+	+	+	+
Jade	-	+	+	+	+
Early Onward	+	-	+	+	-
Puget	-	+	-	+	+
Midinette	-	+	+	+	-
Shasta	-	+	-	-	+
Hursts Green Shaft	-	+	+	-	-
Sleaford Triumph	-	-	+	-	-
Vinco	-	-	-	+	-
Partridge	-	+	-	-	-
Fortune	-	-	-	-	-

+, susceptible; -, resistant.

The potential risk to other compounding pea cultivars from infection with race 2 and other races was determined by inoculation tests (Table 2).

In other unpublished studies, resistance to the races has been shown to be controlled by single dominant genes.

TABLE 2

Pathogenicity of races of bacterial blight to compounding pea cultivars

Cultivars	Races of pathogen 1	2	3	4	5
Belinda, Birte, Stehgolt	-	+	-	+	+
Progreta, Maro, Sentinel	-	-	-	+	-

+, susceptible; -, resistant.

DISCUSSION

Until 1985, bacterial blight had not been identified in field crops in Britain although at Wellesbourne we have detected infection in seed of vining peas imported from the USA on 5 occasions over the past 5-10 years. It seems likely that the disease has occurred in vining pea crops but for various reasons has not been detected and has probably not persisted. For example, the symptoms of the disease could be mistaken for fungal infection by Mycosphaerella and, unless imported seeds contained sufficient infection to cause major symptoms, the disease may remain unrecognised. Moreover, vining peas are not usually grown as seed crops in Britain so there would be little chance of disease build-up. In this respect compounding peas differ from vining peas.

Compounding peas are grown from seed multiplied in Britain, if the disease was introduced it might be expected to build up after several cycles of seed multiplication. It is significant that ADAS detected infection in the cv Belinda (an important new cultivar) within 3 years of the cultivation of compounding peas on an extensive scale. Experience in New Zealand has shown that pea blight can reach epidemic proportions in seed crops especially where large areas of a single cultivar are grown and that cultivar becomes infected with the appropriate pea blight race. Pea blight became important in New Zealand when the main compounding pea cv Partridge became infected with a new race (race 2) introduced from Australia.

The British climate in respect of temperature and rainfall is certainly suitable for the development of bacterial blight. However, the severity of infection will depend on rainfall. Conditions conducive to epidemics might be expected at least every 3 to 4 years.

The likely economic significance of pea blight in Britain is difficult to estimate. For an 80,000 ha crop in 1985, valued at approximately £40m and assuming an overall loss in yield of 10% in wet seasons (every 4th year), then the average monetary loss would be £1m per annum. This is a crude estimate, it could be less but it could also be considerably more. In many countries where pea blight is endemic, steps have been taken to reduce seed infection as the primary source of inoculum. Measures included the growth of seed crops under semi-arid conditions as in the USA or the adoption of seed certification schemes as in Australia and New Zealand. The potential risk of pea blight, even to vining peas, in Britain has long been recognised in the MAFF quarantine requirements for this disease. Also in 1970-72, Wellesbourne cooperated with DSIR in New Zealand in a research and screening programme aimed at controlling pea blight in New Zealand and in preventing the importation of infected vining pea seed to the British market.

At the time of the New Zealand studies, only two races of the pathogen were recognised (Taylor 1972) but since then a further three races have been identified. The complex race structure and the widespread distribution of resistance genes in pea cultivars offers the most likely explanation for the sporadic occurrence of this disease on a world scale. The majority of cultivars of both vining and compounding peas contain at least one resistance gene and may contain as many as five. The ability of any race to develop to epidemic proportions would be severely restricted unless a single cultivar or group of cultivars (uniformly susceptible to a

particular race) was grown extensively. This situation occurred with race 2 on Partridge peas in New Zealand. A similar situation would be possible with many of the Continental cultivars of combining peas which are also susceptible to race 2. Fortunately, some of the important British cultivars are resistance to race 2 (e.g. Progreta, which has four resistance genes and is susceptible only to race 4).

The present strategy for the control of this disease is based on disease exclusion through quarantine measures and by seed testing. The use of race 2 resistant cultivars would provide additional control in the short term, while in the longer term breeders could ensure that cultivars contained all the appropriate resistance genes.

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VIRUS DISEASES OF OILSEED RAPE AND THEIR CONTROL

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ABSTRACT

Beet western yellows virus (BWYV) is the most common virus infecting winter oilseed rape (Brassica napus ssp. oleifera) in the UK and cauliflower mosaic virus (CaMV) the second most common. Other viruses found infecting field crops of oilseed rape include turnip mosaic virus (TuMV) and broccoli necrotic yellows virus (BNYV). Symptoms and yield losses caused by these viruses are described. As immunity to TuMV has been found, it may be possible to control this virus and avoid yield losses by breeding immune cultivars. As no immunity has been found to the more prevalent viruses, CaMV and BWYV, alternative control measures are necessary. The new photostable pyrethroid insecticide PP321 gave very good control of BWYV in a field trial at NVRS in 1985-86 but carbofuran granules did not control BWYV.

INTRODUCTION

The large increase in area of oilseed rape (OSR) grown in the UK over the past 10 years has been well documented (Goetz 1979, Ivins 1981). Planted in August or September and harvested in July or August of the following year, the crop provides a long-lived, overwintering host for brassica pests and diseases and already it has been implicated in an increased incidence of fungal diseases of vegetable brassicas (Humpherson-Jones 1984, Gladders 1984). The two most important aphid vectors of brassica viruses Myzus persicae (Sulzer) and Brevicoryne brassicae (L.), are known to infest and overwinter on OSR (Alford & Gould 1975, Setokuchi 1983, Smith & Hinckes 1984).

There have been a few brief reports on viruses infecting oilseed rape since 1979 (Gilligan et al. 1980, Rawlinson & Muthyalu 1979) but the only detailed information is that provided by Walsh & Tomlinson (1985a), Smith & Hinckes (1985) and Walsh (1986).

This paper reviews the virus disease situation in oilseed rape and suggests methods of control.

MATERIALS AND METHODS

Virus isolates

Isolates used were turnip mosaic virus (TuMV) obtained from swede in Warwickshire (Tomlinson & Ward 1978), and beet western yellows virus (BWYV) and cauliflower mosaic virus (CaMV) from OSR (Walsh & Tomlinson 1985b). TuMV and CaMV were maintained in mustard (Brassica juncea) cv Tendergreen and turnip (B. rapa) cv Just Right. BWYV was maintained in Montia perfoliata.

Transmission

For mechanical transmission, virus inocula were prepared by grinding systemically infected leaves in a 1% (w/v) solution of K_2HPO_4 containing 0.1% Na_2SO_3, and the filtered extracts were inoculated to carborundum-dusted leaves of test plants. Reactions of OSR cultivars to viruses were

determined by inoculating young plants (3-5 leaf stage) grown in 9-cm pots of soil in the glasshouse.

For aphid transmissions, M. persicae previously cultured on Tendergreen mustard plants were starved for 2h and then placed on virus-infected leaves. The length of acquisition feeds for BWYV was 24h. The aphids were then allowed a transmission feeding period on healthy OSR plants of at least 24h before being killed with an aphicide.

Classification of virus symptoms in oilseed rape

Reactions of OSR plants to TuMV were classified as susceptible (mosaic and necrotic reaction types) or immune. The severity of the systemic symptoms in plants experimentally infected with CaMV was rated using score categories 0 (no symptoms) - 3 (severe symptoms).

Virus identification

The identity of viruses in naturally, field-infected plants and in plants mechanically inoculated or aphid infected in the laboratory was determined by electron microscopy, immunosorbent electron microscopy (Milne & Luisoni 1975), reactions in various indicator plants, immunodiffusion (gel) test or combinations of these methods. BWYV isolates were experimentally transmitted by aphids from OSR to M. perfoliata, and lettuce.

Field surveys

Crops of OSR cv Jet Neuf grown at five sites in South Warwickshire were examined for plants showing virus symptoms in March 1983. Suspected infected plants were removed and tested for the presence of virus.

In May 1984, seed and ware crops of OSR cvs Bienvenu and Jet Neuf at eight sites near Banbury, Oxfordshire and South Warwickshire were examined and suspected infected plants removed for virus testing. One field crop was monitored throughout the growing season. Estimates of seed yields were made by labelling infected and virus-free plants, removing them just before harvest and cleaning and weighing seeds. Seed yield of each plant was determined and the individual seeds were weighed to determine whether seed weight as well as total seed yield was affected by virus infection. An estimate of the incidence of virus infection was made by examining ten 1 m^2 quadrats at random points in the crop. The total numbers of plants in each quadrat were counted and plants suspected as virus infected were removed and tested for virus presence by electron microscopy and sap inoculation to indicator plants.

Estimates of the incidence of BWYV in crops were made by taking random samples and determining the presence or absence of virus by ELISA (Clark & Adams 1977). Two field crops infected by BWYV were monitored in 1985. Infected and uninfected plants were removed just before harvest and seed yields were determined for the individual plants.

Insecticide trial

A fully replicated trial was set up in 1985 to test the efficacy of the pyrethroid insecticide PP321 ('Karate') and carbofuran ('Yaltox') granules in controlling the spread of viruses in OSR. PP321 was applied at the 1-2 true leaf stage at 7.5g a.i./ha and carbofuran was applied pre-planting at 1kg a.i./ha.

RESULTS

Identity and incidence of viruses in field crops

In March 1983, surveys of field crops of OSR near Stratford-upon-Avon, Warwickshire showed that plants were infected by

CaMV, broccoli necrotic yellows virus (BNYV) and TuMV. All infected plants showed mosaic symptoms with slight leaf distortion.

In 1984, CaMV, BNYV and BWYV were found in OSR crops growing in the Wellesbourne area. Plants infected by CaMV showed the following symptoms before flowering: severe stunting, distortion of stems and leaves and vein clearing. After flowering, stems of infected plants had necrotic streaks and seed pods were twisted and often bore necrotic spots. Most infected plants produced fewer and shorter racemes and the numbers of pods on each raceme were much reduced. Both CaMV and BWYV could be recovered from stems and pods of infected plants. A few infected plants were not stunted and had as many racemes and pods as uninfected plants, but still showed characteristic pod twisting and were slightly chlorotic. Of the 10 quadrats sampled, nine contained infected plants with a maximum of six infected plants/m^2; an infection level of 10%. An estimated 5.3% of all plants were infected by CaMV (Table 1). Assessments were not made of the incidence of BWYV in this field.

TABLE 1

Incidence of cauliflower mosaic virus infected plants in a field crop of oilseed rape

Number of m^2 quadrats sampled	Mean number of plants/m^2	Mean number of plants infected by CaMV/m^2	Mean percentage of infected plants
10	57 ± 3.4	3.0 ± 0.6*	5.3

* determined by inoculations to indicator plants and by electron microscopy.

In May 1985, CaMV incidences of 2-6.5% were recorded in naturally infected plots of OSR in Warwickshire. CaMV was detected in early December 1984 and January 1985 in these plots. Assessments of BWYV incidence in two fields in Warwickshire were made by ELISA in July 1985 and incidences of 25% and 86% were recorded.

BWYV was transmitted to M. perfoliata and identified by serology (immunodiffusion test) and electron microscopy. It was also transmitted to lettuce but attempts to transmit it to sugar beet were unsuccessful. The CaMV isolated from OSR was inoculated to and infected other brassicas including cauliflower and turnip and caused the typical dark green vein banding symptoms.

Reactions of plants to turnip mosaic virus

To examine plant reactions in detail and to search for plants with resistance or immunity to TuMV, glasshouse-grown plants of most commercial cultivars were mechanically inoculated at the 3-5 leaf stage (code 1,01-1,03 of the Sylvester-Bradley & Makepeace (1984) growth stage key). At least 10 plants of each cultivar were inoculated except, for the most common commercial cultivars where 50 plants were inoculated. Most plants developed symptoms after 7-14 days. Some plants developed necrotic local lesions, the numbers of which varied from plant-to-plant both within and between cultivars. In these plants subsequent systemic symptoms consisted of veinal and interveinal necrosis of varying intensity, usually

culminating in apical necrosis and plant death. The most common reaction type was a mosaic. The severity of mosaic symptoms varied from plant-to-plant and from cultivar-to-cultivar. A few plants of cv Rafal showed either no symptoms or, occasionally in some tests, a few large whitish lesions in inoculated leaves only. When back inoculated to indicator plants or examined in the electron microscope, virus could not be recovered from these lesions nor from symptomless uninoculated leaves of the same plants. These plants were vernalised and maintained until the seed had set to determine whether virus symptoms eventually developed and to compare seed yields of inoculated susceptible and inoculated immune plants. No symptoms developed subsequently in these plants.

Reactions of plants to cauliflower mosaic virus

A minimum of 10 glasshouse-grown plants of most commercial cultivars were mechanically inoculated with CaMV at the 3-5 leaf stage. All plants developed symptoms within 20-40 days but these symptoms did not fall into distinct reaction types and consequently were scored for severity rather than type. The most common leaf symptoms were mosaics, dark green vein banding and necrosis. A common feature of infected plants was the production of distorted leaves with short petioles. Some infected plants were vernalised to study symptoms in the later stages. As with TuMV infections, necrotic streaks were observed on the stems of some plants and occasionally, the pods were twisted and distorted and bore necrotic spots. Symptom severity varied within and between cultivars. No immunity was observed but some plants and cultivars were more tolerant than others.

Effect of viruses on the yield of oilseed rape

The OSR (cv Jet Neuf) field containing plants infected with CaMV + BWYV was monitored from April to July 1984. During this period, 21 virus-infected plants and 21 virus-free plants were identified and labelled. One week before harvest, the labelled plants were removed and the seed from individual plants cleaned and weighed. Of the 21 infected plants, 14 were infected with CaMV alone and seven with CaMV + BWYV. Virus-free plants produced more than 10 times as much seed as infected plants. The yield loss of plants infected by CaMV alone was the same as with plants infected by CaMV + BWYV. Not only did virus-infected plants produce less seed than virus-free plants, but the mean weight of individual seeds from infected plants was significantly lower ($P<0.001$) than that of seeds from virus-free plants.

The crops of OSR (cvs Jet Neuf and Bienvenu) containing plants infected with BWYV were monitored throughout the growing season. There was no significant difference between the seed yields of virus-infected and virus-free plants.

Insecticide trial

Levels of CaMV in the trial were very low (<4%). BWYV was common with infection levels of up to 100% in untreated plots. Carbofuran granules (1 kg a.i./ha) did not significantly reduce the infection levels of BWYV. All treatments with the pyrethroid insecticide PP321 (7.5g a.i./ha) significantly reduced transmission of BWYV. The post-emergence PP321 spray reduced BWYV transmission by 73%, the October spray reduced transmission by 47% and the double spray (post-emergence + October) reduced transmission by 86%.

DISCUSSION

BWYV was the commonest and CaMV the second most common virus found in OSR fields examined in Warwickshire. ADAS surveys have shown that nationally in 1985 (Beaton 1985) 60% of crops sampled were infected by

BWYV/beet mild yellowing virus (BMYV) and that within crops virus incidence varied from low to very high. The incidence of CaMV appears to be much lower. According to Gladders (1984) CaMV was much more common in 1984 than usual and some foci of infection were found, particularly in Essex, but most crops sampled had less than 1% of plants infected. These may be underestimates as tests were made only on samples of 25 plants per field.

The spread of brassica viruses is dependent on the abundance and movement of the aphid vectors, mainly M. persicae and B. brassicae. Smith & Hinckes (1984) reported exceptionally large numbers of migrating M. persicae in Suffolk in 1982. The Rothamsted Insect Survey (Taylor et al. 1984) has recorded a sharp rise in the number of B. brassicae alates in Britain; in the summer months of 1975-1984, the maximum monthly numbers of B. brassicae recorded were in 1983 and 1984. Exceptionally high numbers of B. brassicae were also recorded in 1983 in Essex where Gladders (1984) subsequently reported CaMV infection of OSR crops in 1984.

The types of reaction to TuMV infection found in OSR cultivars are similar to those found in TuMV-infected swede (B. napus ssp. napobrassica) (Tomlinson & Ward, 1978; 1982) and include stunting, a symptom which was also seen by Rawlinson & Muthyalu (1975). In the OSR cv Bienvenu, Mikado, Korina and Belinda, losses caused by TuMV infection could be high because infected plants develop lethal systemic necroses and soon die. Plants of cultivars which develop mosaic symptoms (e.g. cv Jet Neuf, Rafal, Doral, Perle and Fiona) are able to survive for much longer and therefore would act as long-lived sources of TuMV for nearby horticultural brassica crops.

Symptoms in OSR plants caused by CaMV were, at some stages in the growth of the plants, similar to those induced by TuMV, and consisted of necrotic streaks in stems and twisting and distortion of seed pods. However, only CaMV caused the development of shortened petioles.

BWYV and BMYV are serologically closely-related luteoviruses (Govier 1984). Both infect M. perfoliata, but European strains differ in host range depending on whether they infect lettuce (BWYV) (Duffus & Russell 1970, Tomlinson 1972) or sugar beet (BMYV) (Duffus & Russell 1970, Govier 1984). As the BWYV isolated from OSR in 1984 was successfully transmitted to lettuce and attempts to transmit it to sugar beet were unsuccessful this confirms its identity as BWYV.

Investigations on the effect of CaMV and TuMV on OSR plants showed that both viruses reduced seed yield. With CaMV infections this resulted from a reduction in the numbers of pods and seeds together with a reduction in the weight of individual seeds. The effects of TuMV were more variable. In one experiment, the weight of individual seeds was affected more than the number of seeds produced but in another experiment, the opposite effect was obtained. In all tests, however, CaMV- and TuMV-infected plants yielded less seed than virus-free plants. The effect of BWYV on the yield of OSR is less clear, yield loss due to BWYV infection could not be demonstrated in the present study but Smith & Hinckes (1985) reported seed yields 10% greater in plots with low levels of BWYV compared with plots with 100% BWYV infection.

The level of control of BWYV given by PP321 was very good although not as good as the 100% control of barley yellow dwarf virus (BYDV) reported by Perrin & Gibson (1985).

It is not only important to control viruses in oilseed rape because of potential yield losses but also because the crop acts as a large overwintering reservoir of these viruses for infection of horticultural and fodder brassicas. It may be possible to control TuMV by producing immune cultivars. Lines of OSR immune to TuMV are now being examined in detail to determine the heritability of the immunity. At present, there seems little prospect of finding immunity to CaMV or BWYV in OSR and the development of CaMV and BWYV tolerant lines, while beneficial in reducing losses in seed yield, is unlikely to reduce the danger that the crop poses as a source of infection to nearby horticultural brassica crops. The control of these two viruses will therefore rest on the use of insecticides.

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PP321 : CONTROL OF MAJOR PESTS OF OIL SEED RAPE IN WEST EUROPE

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ABSTRACT

PP321, a new highly active photostable pyrethroid, was tested against the major pests of oil seed rape in the UK, France and Denmark. Good efficacy is demonstrated against Psylliodes chrysocephela, Ceutorhynchus napi, Melegethes aeneus, Ceutorhynchus assimilis, Dasineura brassicae and Athalia rosae at application rates of 5-7.5g a.i./ha. No phytotoxicity was evident in the trials.

INTRODUCTION

PP321 (international trade name 'Karate', except for the UK), is a novel photostable pyrethroid patented by ICI. It is effective against a broad range of pests at low application rates. Its general properties and efficacy were described by Jutsum et al. 1984.

In the past three years a large number of trials have been undertaken by ICI and official organisations in the oil seed rape-growing countries of Europe against the major pests of this crop. This report gives results of trials from the UK, France and Denmark against Psylliodes chrysocephala (cabbage stem flea beetle), Ceutorhynchus napi (stem weevil), Meligethes aeneus (pollen beetle), Ceutorhynchus assimilis (cabbage seed weevil), Dasineura brassicae (pod midge), and Athalia rosae (turnip sawfly), all of which can be economically damaging. The data presented demonstrate the effectiveness of PP321 and appropriate application rates for the different target pests.

METHODS AND MATERIALS

Plot size was mostly 40-100m^2 replicated four to six times. The Danish trials had two replicates. The UK seed weevil trials had plots of 0.2-0.67 ha with 2 or 3 replicates. In the French pollen beetle and seed weevil trials, a 'mini-cage' technique was employed whereby plots were first sprayed with the test chemical and then randomly selected inflorescences were caged and the target insect introduced by hand. Cages were constructed from polythene bottles of 25cm base diameter and 40cm high. The sides were removed and replaced with mosquito netting. Twenty five adults of the target species were used per cage. Five or six cages were used per treatment. Mortality was assessed over three days. For other trials a minimum of ten plants per plot were inspected as appropriate for the target infestation.

Chemicals were applied to small plots with gas-powered, hand-held sprayers using fan jets and a spray volume of 300 l/ha at 2-4 bar pressure, an exception being 500 l/ha for the 1983 Danish trials. The large-plot UK seed weevil trials were treated by field crop sprayers at 200-300 l/ha.

The formulations used in the trials reported are given below:
PP321 as 25 g or 50 g/l EC. (2.5% WG Denmark 1985)
Fenvalerate/Fenitrothion as 50/250 g/l EC.
Cypermethrin as 100 g/l EC.
Deltamethrin as 25 g/l EC.
Gamma HCH as 800 g/l SC.
Methidathion as 193 g/l EC.
Phosalone as 500 g/l EC.
Fluvalinate as 240 g/l EC.
aCypermethrin as 100 g/l EC.
Cyfluthrin as 50 g/l EC.
Flucythrinate as 100 g/l EC.

RESULTS

1. Psylliodes chrysocephala

Initial trials in England in autumn 1983 (Trials 1-4) which compared adult and larval spray timings showed interesting trends (Table 1). Application was made when adult or larval damage first became obvious.

TABLE 1

Comparison of chemicals and spray timings for control of P. chrysocephala larvae in winter rape - UK, 1983/84

Chemical	Rate (g a.i./ha)	Timing	Live larvae/100 plants in March/April Trial 1	2	3	4
Cypermethrin	25	adult	79 B	72 A	7 B	53 C*
Cypermethrin	25	larval	17 CD	9 C	2 C	-
Cypermethrin	25	adult + larval	5 D	6 C	0 C	-
PP321	10	adult + larval	4 D	1 C	0 C	5 D*
Deltamethrin	7.5	adult + larval	9 D	7 C	0 C	55 C*
gamma-HCH	560	adult + larval	43 BC	28 B	2 BC	195 B*
Untreated			299 A	82 A	108 A	405 A

*only adult spray applied in NA14

Note: different letters within columns indicate significant differences at P=0.05 in this and subsequent Tables (Duncan's Multiple Range Test).

Although pest numbers were low, it is clear that larval sprays were more effective than spraying against adults and that the pyrethroids were superior to the older compound gamma-HCH. The adult spray timing may have been less effective because new crop growth was unprotected against later adult invasion. Also the later larval spray might have given better control of spring hatching larvae. Good control of spring-hatching larvae was achieved with the pyrethroids from autumn applications, particularly with PP321, indicating long persistence in cold winter conditions.

In autumn 1984, four trials (Trials 5-8) were undertaken comparing different rates and timings of PP321 with standard chemicals (Table 2). Larval applications were not markedly more effective than spraying the adults. PP321 at 5 g a.i./ha appeared at least as good as the cypermethrin or deltamethrin standards. Calculation of the PP321 response curve in each of the four trials showed that for the larval application timing on average a rate of about 3 g a.i./ha matched the 25 g a.i./ha standard cypermethrin treatment. Grower trials in 1985 using PP321 at the 5 g a.i./ha rate confirmed that this was a robust treatment even with high pest populations. In a Cambridgeshire trial, a single larval-timed spray in November gave 83% control of larvae in the spring, the untreated plot having a mean infestation level of 15 larvae per plant.

TABLE 2

Comparison of application rates and timing for PP321 against P. chrysocephela larvae in winter rape - UK 1984/85.

Chemical	Rate (g a.i./ha)	Timing	% control of live larvae in April Trial 5	6	7	8	Mean
PP321	2.5	adult	63 BCD	84 BD	90 BD	93 BE	82
PP321	5	adult	82 CE	92 CDE	92 BE	96 DE	91
PP321	7.5	adult	85 DEF	97 CDE	97 EG	99 E	94
PP321	10	adult	86 EF	96 CDE	100 G	99 E	95
Cypermethrin	25	adult	43 AB	88 BE	89 BC	93 BE	78
Deltamethrin	6.25	adult	31 AB	89 BE	92 B	91 BE	73
PP321	2.5	larval	87 EF	55 AB	96 DEF	79 B	79
PP321	5	larval	95 FG	93 CDE	99 FG	85 BD	93
PP321	7.5	larval	95 FG	98 DE	100 G	80 BC	93
PP321	10	larval	100 G	99 E	99 FG	96 CDE	98
Cypermethrin	25	larval	57 AC	82 BC	96 CDEF	77 B	78
Deltamethrin	6.25	larval	85 DEF	91 CDE	99 FG	83 BD	90
Untreated*	-	-	(1.9) A	(2.9) A	(2.8) A	(1.6) A	(2.3)

*Mean number/plant

In trials by ICI Sopra in France (Trials 9-14) both adult and larval damage was assessed (Tables 3, 4) following single sprays at the crop seedling stage aimed at the adults. Applications were made in September when an average of two feeding damage points per plant was counted.

TABLE 3

Control of P. chrysocephala adult feeding damage with autumn sprays. Number of damage points per plant at 14-21 DAT - France 1983/84

Chemical	Rate (g a.i./ha)	Trial	Reims 9	Tours 10	Reims 11	Toulouse 12
PP321	5		0.05 B	1.93 B	0.21 B	2.49
PP321	7.5		0.04 B	2.13 B	0.19 B	2.75
PP321	10		0.03 B	1.78 B	0.10 B	2.63
Deltamethrin	7.5		0.06 B	1.86 B	0.31 B	3.26
Untreated	-		0.33 A	7.45 A	0.91 A	3.26

TABLE 4

Nos. of live P. chrysocephala larvae per plant at the end of the winter - France 1983/84

Chemical	Rate (g a.i./ha)	Trial	Tours 10	Reims 11	Toulouse 12	Tours 13	Tours 14
PP321	5		0.05 B	0.03 B	0.09 B	0.04 B	0.54 B
PP321	7.5		0 B	0.02 B	0.02 B	0.04 B	0.34 B
PP321	10		0 B	0.02 B	0 B	0 B	0.31 B
Deltamethrin	7.5		0.12 B	0.04 B	0.18 B	0.04 B	0.41 B
Untreated	-		6.26 A	0.49 A	1.78 A	2.06 A	5.91 A

Very marked reductions in the numbers of live larvae per plant were achieved at the end of the winter with as little as 5 g a.i./ha of PP321. Significant reductions in the number of plants attacked were also obtained. Five trials in 1984/85 showed a very similar trend.

2. Ceutorhynchus napi

Trials by Sopra in France over three years (Trials 15-19) showed that a single application of PP321 to winter rape in March/April when the crop was at the susceptible beginning of flowering stage (15-20 cm high) will significantly reduce larval attack and plant damage (Table 5). Application was made when ten adults per day were caught in traps. Trials were assessed 6-8 weeks after treatment. There was no significant difference between rates of PP321, but 7.5 g a.i./ha was the preferred treatment based on the numerical trend and favourable comparison with the standards.

TABLE 5

Control of C. napi larvae and plant damage - France, 1984-86.

Chemical	Rate (g a.i./ha)	Trial 15 Live larvae per plant	Trial 15 Yield (q/ha)	% winter rape plants without C. napi damage Trial 16	Trial 17	Trial 18	Trial 19
PP321	5	3.6 B	19.9 A	57 A	79 AB	58 A	58 B
PP321	7.5	3.0 B	20.2 A	49 A	86 AB	62 A	75 AB
Deltamethrin	7.5*	3.2 B	19.1 A	65 A	74 ABC	53 A	76 A
Cypermethrin	25	3.3 B	19.2 A	54 A	70 BC	-	-
Methidathion	289.5	3.4 B	16.8 AB	30 B	64 C	-	-
Untreated	-	5.8 A	12.2 B	12 C	44 D	35 B	16 C

*5 g used in Trials 18 and 19

3. Meligethes aeneus

The contact and residual action of PP321 against M. aeneus adults on winter rape was tested over three years by Sopra in France using the 'mini-cage' technique (Trials 20-25). The percentage mortality was assessed for three days after treatment (DAT), the insects having been introduced on to previously treated inflorescences under field conditions (Tables 6, 7).

TABLE 6

Percentage mortality of M. aeneus - France, 1984

Chemical	Rate (g a.i./ha)	Trial 20 1 DAT	Trial 20 3 DAT	Trial 21 1 DAT	Trial 21 3 DAT	Trial 22 1 DAT	Trial 22 3 DAT
PP321	3.75	63 A	89 A	38 B	81 C	13 A	85 A
PP321	5	71 A	90 A	57 A	94 AB	12 A	85 A
PP321	6.25	60 A	96 A	57 A	97 A	9 A	91 A
Deltamethrin	5	57 A	95 A	40 AB	89 BC	13 A	72 A
Untreated	-	9 B	10 B	3 C	8 D	1 B	13 B

Mortality increased up to three days after treatment resulting in a good final level of control. Action was most rapid during warm conditions. A similar picture was obtained in 1985 and 1986 trials. The dose response to PP321 was shallow and a rate of 5 g a.i./ha is considered a very effective and robust treatment.

TABLE 7

Percentage mortality of M. aeneus - France, 1986

Chemical	Rate (g a.i./ha)	Trial 23		Trial 24		Trial 25	
		1 DAT	3 DAT	1 DAT	3 DAT	1 DAT	3 DAT
PP321	3.75	62 A	75 A	32 B	70 AB	38 B	87 A
PP321	5	52 A	78 A	41 AB	64 B	64 A	81 A
PP321	6.25	59 A	76 A	44 AB	83 A	52 AB	85 A
Deltamethrin	5	60 A	78 A	57 AB	76 AB	45 B	81 A
Fluvalinate	36	61 A	78 A	46 AB	75 AB	49 AB	80 A
Untreated	-	23 B	34 B	9 C	19 C	11 C	15 B

4. Ceutorhynchus assimilis and Dasineura brassicae

Trials in France in 1984 and 1985 using the 'mini-cage' technique, indicated that good control of C. assimilis can be obtained with PP321 (Table 8). The performance of the chemicals was variable and may be related to temperature differences, especially with phosalone in Trial 30 where the weather was cool in the two days following treatment.

TABLE 8

Percentage mortality of C. assimilis adults on winter rape 3 days after treatment - France, 1984/85

Chemical	Rate (g a.i./ha)	Trial 26	Trial 27	Trial 28	Trial 29	Trial 30	Trial 31
PP321	3.75	89 B	93 B	31 C	51 BC	94 A	47 B
PP321	5	100 A	100 A	67 B	44 BC	91 A	52 AB
PP321	6.25	100 A	100 A	68 B	63 B	97 A	64 A
Deltamethrin	5	100 A	99 AB	32 C	29 C	92 A	47 A
Phosalone	1200	99 A	98 AB	98 A	96 A	26 B	46 B
Untreated	-	10 C	12 C	10 C	8 D	26 B	17 C

Across the six trials PP321 at 5 or 6.25 g a.i./ha compared well with the deltamethrin or phosalone standards.

In Denmark PP321 was included in official trials with C. assimilis from 1983 to 85. Table 9 gives results for two trials in 1983 (Trials 32-33), application being at mid-flowering against natural populations. The activity of PP321 compared favourably with the other chemicals, especially on a rate for rate basis. Persistence was good for this type of small-plot trial.

TABLE 9

Percentage mortality of C. assimilis adults on winter rape - Denmark, 1983

Chemical	Rate (g a.i./ha)	Trial 32 1 DAT	Trial 32 3 DAT	Trial 32 5 DAT	Trial 33 1 DAT	Trial 33 3 DAT	Trial 33 6 DAT
aCypermethrin	20	81	97	94	99	65	73
aCypermethrin	10	70	87	91	100	39	0
Fluvalinate	48	46	24	60	97	22	0
Fluvalinate	24	40	25	43	97	4	0
Cyfluthrin	50	91	100	91	99	75	82
Cyfluthrin	25	86	100	95	100	66	36
PP321	12.5	84	100	93	98	75	50
PP321	7.5	72	100	91	100	44	40
Phosalone	1250	10	56	86	80	8	6
Phosalone	1000	0	35	68	76	45	0

Note : statistical analysis not available.

In the UK (Trials 34-37) PP321 gave control of C. assimilis equivalent to the standard triazophos treatment (Lane, 1986). A reduction in D.brassicae damage was also observed (Table 10). Application of PP321 five days after completion of flowering on the main raceme was rather more effective than the earlier timing.

TABLE 10

Control of C. assimilis and D. brassicae - UK, 1986

Treatment	% pods damaged by C. assimilis Trial 34	35	36	37	% pods damaged by D. brassicae 34	35	36	37
A	1.8	0.6	1.5	6.9	0.3	0.3	0.3	2.0
B	0.4	0.3	5.8	3.2	0.3	0	2.2	0.7
C	0.3	0.8	1.8	5.6	0	0	0.3	1.7
D	4.8	0.8	18.3	23.1	1.0	0.2	2.5	4.7

A = PP321 at 7.5 g a.i./ha at flowering complete on main raceme.
B = as for A but 5 days later.
C = Triazaphos at 420 g a.i./ha at flowering complete on all racemes.
D = Untreated control.

Note: statistical analysis not available.

PP321 was included in Danish official trials over three years against pod midge on winter rape. Two sprays were used at five-day intervals, the first being applied at four to five days following the official spray threshold warning in the second half of May. Results show that good control is possible (Table 11).

The moderate response in 1985 may be due to a combination of small plot size allowing re-infestation and wet weather which delayed the second application, giving an interval of seven instead of five days.

TABLE 11

Control of D. brassicae on winter rape with two spray programmes - Denmark, 1984/85

Chemical	Rate (g a.i./ha)	1984 % infested pods	1985 % infested pods
Cypermethrin	50	2.4	2.8
Flucythrinate	30	3.5	5.8
Fenvalerate/ fenitrothion	50/250	4.0	-
PP321	7.5	1.8	2.7
FMC54800	7.5	2.3	-
"	15	1.7	-
"	25	1.5	-
aCypermethrin	10	-	4.2
Untreated	-	10.6 mean 3 trials	6.0 mean 3 trials

5. Athalia rosae

Larvae of this pest are occasionally damaging in France. PP321 at 5 g a.i./ha gave very good control in two trials in the Carpentras and Reims areas when applied in the autumn to seedling rape at the start of larval attack.

6. Crop safety

No crop damage was evident with PP321 in any trial up to the highest rate tested.

DISCUSSION

The results demonstrate that effective control of important oil seed rape pests can be achieved with PP321.

Good control of cabbage stem flea beetle can be obtained with single applications of PP321 aimed at adult invasions or timed for the first larval attacks. A similar conclusion was drawn from trials with deltamethrin (Smith & Hewson 1984). Yields tended to be better with the adult timing. Sprays aimed at adult invasions are generally easier from the management viewpoint, but autumn-applied larval sprays may be more effective against spring-hatching larvae. Trials with alpha-cypermethrin (Reed & Nicholls 1984) showed that larval sprays can be more effective when adult invasions are late and this may be related to better control of spring-hatching larvae, but adult timed sprays were more effective with the early-autumn invasions. Work in France with alpha-cypermethrin (Debray & Tipton 1984) also demonstrated the effectiveness of sprays against adult invasions in controlling larval attacks.

It is unlikely that two sprays would be required unless attacks were prolonged and heavy and egg hatch occurred over a protracted period.

An interesting observation (Newman 1984) was that P. chrysocephala and also Ceutorhynchus spp. damage can increase the levels of crown and stem canker (Leptosphaeria maculans) and can be an additional reason for controlling these pests. PP321 applied in the autumn for control of P. chrysocephala has also been shown to reduce Beet Western Yellows virus

infection by controlling the aphid vectors (Walsh, 1986).

The trials against A. rosae and C. napi demonstrate that these pests can be satisfactorily controlled with well-timed application of PP321 at the start of larval invasion. Similarly, M. aeneus is very readily controlled.

Some of the early work with pyrethroids in the late 1970s was disappointing against C. assimilis (Alford et al. 1979). Results are now much improved with the newer more active pyrethroids and a better knowledge of timing. Application has to be made just prior to peak egg laying which is generally fairly late in the flowering stage, flowering at least being complete on the main raceme. Pyrethroids have been used successfully in France and Denmark for several years against pollen beetle, seed weevil and pod midge. The importance of reducing midge damage by controlling seed weevil is well recognised in official recommendations. Work with PP321 in flowering rape (Gough & Wilkinson 1984) showed that a rate of 10 g a.i./ha applied at mid-day had no lethal or sub-lethal effect on foraging honeybees and that use of PP321 is a practical solution against summer pests.

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THE INFLUENCE OF CABBAGE STEM FLEA BEETLE (*Psylliodes chrysocephala* (L.)) ON YIELDS OF OILSEED RAPE

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ABSTRACT

Damage assessment trials to investigate the effects of cabbage stem flea beetle (*Psylliodes chrysocephala*) on yields of winter oilseed rape are described. Results indicate that if an application of an effective insecticide is made in November-December when larval numbers are five per plant or greater, a yield response of 0.34 tonnes per ha (at 92% dry matter) can be expected and use of this population level in late autumn as a treatment threshold will avoid significant yield losses. Preliminary results also indicate that no extra yield increment is obtained from spring treatment if effective autumn control measures have been taken, even if subsequently larval numbers increase to over 13 per plant by April. In the absence of autumn control, spring-applied treatment appears to give a worthwhile yield response if larval numbers in spring increase to at least five per plant or more. The possibility that incidental control of aphid vectors of beet western yellows virus can affect yield response to cabbage stem flea beetle treatments is also discussed.

INTRODUCTION

During the expansion of winter oilseed rape production in England and Wales since the mid-1970s (Anon.1985), cabbage stem flea beetle (CSFB) *Psylliodes chrysocephala* (L.) (Coleoptera: Chrysomelidae) has become a widespread and damaging pest (Alford & Gould 1975, John & Holliday 1984). Early work showed that chemical control of CSFB on overwintering brassica crops could be achieved using gamma-HCH (Williams & Carden 1961) and subsequently more effective organophosphorus, carbamate and pyrethroid insecticides were identified (Alford 1977, John & Holliday 1984). Experience of CSFB attacks in the Agricultural Development and Advisory Service (ADAS), has led to the general adoption of a treatment threshold for advisory purposes of five larvae or more per plant in autumn. In the present paper, data from trials done by ADAS are used to test the general validity of this threshold and more recent work on the pest potential of late-hatched larvae entering plants in spring is reported.

METHODS

Autumn threshold trials

The validity of the currently used autumn damage threshold for CSFB was investigated in six trials done in Hertfordshire and Cambridgeshire between 1982/83 and 1984/85. In each trial, yields following the application of carbofuran granules at the rate of 1.25 kg a.i. per ha immediately after sowing (before crop emergence) were compared with

untreated controls in a randomized block design with either four or five replications. Plots measured 100 m^2 (usually 4 x 25 or 5 x 20 m) and yields were determined by harvesting strips of either 2.23 or 3.66 m width from entire plot lengths. Results were expressed in tonnes per ha at 92% dry matter (d.m.). Larval CSFB numbers were determined by dissection of 20 randomly selected plants per plot in late November/early December and again in February/March.

Spring threshold trials

In three modified trials done in the 1984/85 season, the effect on yield of spring-hatched larvae was investigated using the following treatments:

i HCH - a single application of gamma-HCH at 560 g a.i. per ha in early November, likely to give adequate autumn protection only;

ii HCH+Ph - a single application of gamma-HCH as above followed by the application of phorate granules at 200 g a.i. per ha in February, to give full autumn and spring protection;

iii Ph - phorate alone applied as above in February;

iv U - untreated control.

Each treatment was replicated four times in randomized blocks and larval numbers were assessed as before in untreated plants in December, January, March and April to determine initial population levels and the subsequent extent of spring hatching. Populations on insecticide-treated plots were assessed in January and April only. Yields were determined as previously described.

RESULTS

Autumn threshold trials

Untreated larval populations varied considerably between autumn trial sites and years (Table 1) and ranged from 0.57 to 6.67 larvae per plant at assessments made in late November/early December. No notable increases in untreated populations were detected in February/March and carbofuran treatment was seen to give effective control ($P<0.05$-0.01) at both assessments in all trials (Table 1). Significant yield responses to carbofuran treatment ($P<0.05$) were obtained only in the trials with five or more larvae per plant on untreated plots in December although in all trials, the carbofuran treatment gave a higher mean yield than the control (Table 1).

Figure 1 shows yield responses obtained in a series of similar damage assessment trials done by ADAS in recent years using a range of organophosphorus, carbamate and pyrethroid insecticides applied in autumn. Only data from treatments achieving at least 70% reduction of larval populations in late autumn were included in this analysis and

trials in which substantial spring hatching subsequently occurred were excluded. A significant ($P<0.001$) regression of yield response on untreated larval numbers indicated an expected yield loss of 0.34 tonnes per ha (@ 2% d.m.) at the currently advocated autumn threshold of five larvae per plant. At this threshold, the 95% confidence limits for the yield response obtained in a single trial range from 0.01 to 0.67 tonnes per ha.

Table 1

Summary of larval populations and yield responses obtained in autumn damage threshold trials comparing immediate post-sowing treatment with carbofuran (T) and untreated controls (U), 1982-1985

Year of trial (no. of replicates)	Treatment	Mean larvae per plant Nov.-Dec.	Mean larvae per plant Feb.-Mar.	Yield (tonnes per ha @ 92% d.m.)
1982/83-I	T	0.3**	0.8**	2.19*
(4)	U	5.8	5.4	1.56
1983/84-I	T	0.3	0.5*	4.09
(4)	U	0.6	2.5	4.06
1983/84-II	T	0.4*	0.8*	2.39
(4)	U	2.0	2.1	2.08
1984/85-I	T	0.5*	1.0**	3.59*
(5)	U	5.0	7.3	3.38
1984/85-II	T	0.5*	1.0*	2.76*
(5)	U	6.7	6.5	2.17
1984/85-III	T	1.0**	1.9*	2.71
(5)	U	3.9	4.1	2.50

* and ** indicate significant difference from control mean (U) at $P<0.05$ and 0.01 respectively.

Spring threshold trials

The results of larval assessments made in spring threshold trials are shown in Tables 2-4. In trial I, autumn larval numbers reached the threshold of five per plant but subsequent spring hatching increased this population to 7.5 larvae per plant in April. All combinations of autumn and spring treatments significantly reduced populations at appropriate times. Although no significant yield responses were obtained in this trial, mean yields on autumn treated plots were substantially greater than on untreated controls but the yield difference between autumn treatment alone and combined autumn and spring treatment was relatively small (Table 2).

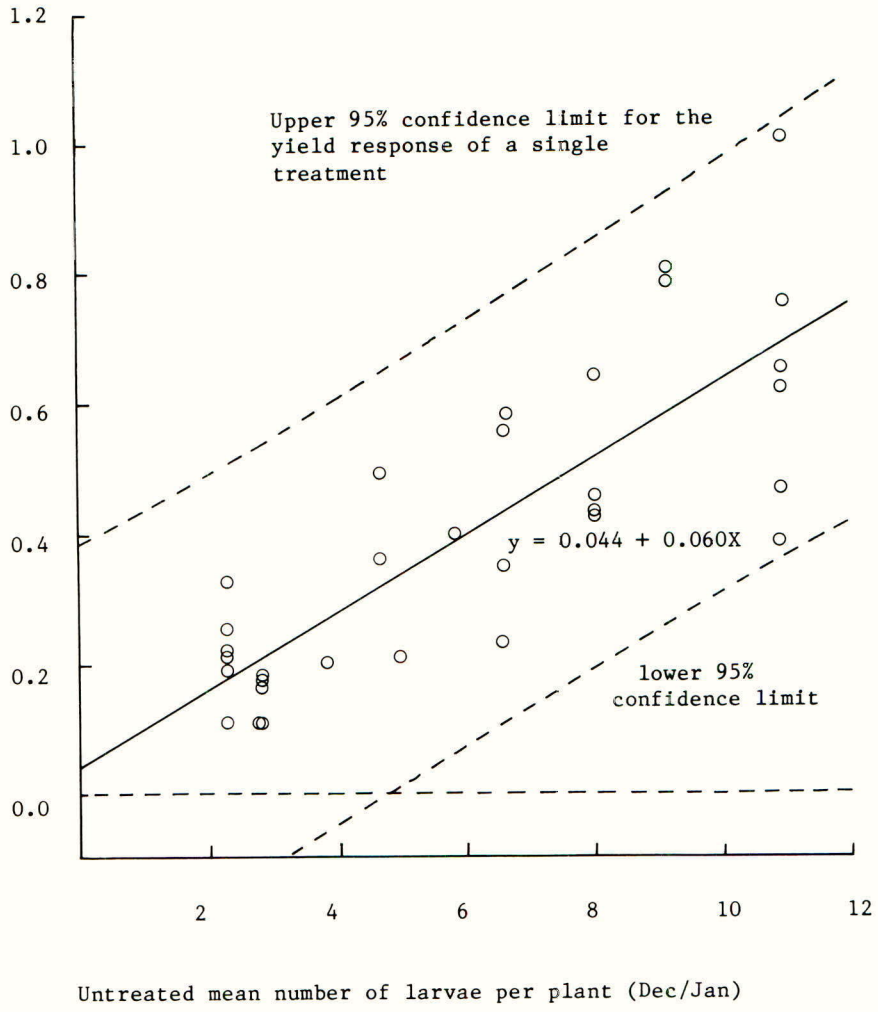

Fig. 1 Relationship between mean larval numbers per plant on untreated plots in December/January and yield response under treatments achieving at least 70 per cent reduction of CSFB populations; regression line and 95 per cent confidence limits for the yield response expected of a single treatment.

Table 2

Summary of larval populations and yield responses obtained in spring threshold trials in 1984/85 comparing combinations of autumn control using HCH (HCH) and spring application of phorate (Ph) with an untreated control (U); trial I (four replications per treatment)

Treatment	Mean larvae per plant				Yield (tonnes per ha @ 92% d.m.)
	December	January	March	April	
HCH	NA	2.1*	NA	1.7***	4.00
HCH+Ph	NA	0.6**	NA	0.3***	3.77
Ph	NA	4.9	NA	1.1***	3.57
U	5.0	5.9	7.3	7.5	3.37
S.E.D. (9 d.f.)	-	1.58	-	0.77	0.20

*, ** and *** indicate significant difference from control (U) mean at $P<0.05$, 0.01 and 0.001 respectively; NA = not assessed.

In trial II, larval numbers exceeded the autumn threshold and were greater than six per plant in December. A substantial spring hatch increased numbers to over 13 larvae per plant by April. HCH effectively controlled the autumn larval attack but failed to control the subsequent spring invasion. Larval numbers were effectively reduced by the phorate application. All treatments gave significant yield responses compared with the untreated control, but relatively little additional response accompanied spring phorate treatment following an autumn application of HCH (Table 3).

Table 3

Summary of larval populations and yield responses obtained in spring threshold trials in 1984/85; trial II (details as Table 2)

Treatment	Mean larval numbers per plant				Yield (tonnes per ha @ 92% d.m.)
	December	January	March	April	
HCH	NA	2.5**	NA	13.8	2.61**
HCH+Ph	NA	3.0*	NA	2.3**	2.63**
Ph	NA	9.7	NA	4.3**	2.28*
U	6.67	9.2	6.5	13.5	1.85
S.E.D. (9 d.f.)	-	1.67	-	2.61	0.23

In a third trial, autumn numbers were below four larvae per plant but subsequent hatching increased this population to almost seven per plant by April. All treatments gave good control and produced signficant yield responses but no additional yield increment was obtained from the phorate treatment following an autumn application of HCH.(Table 4).

Table 4

Summary of larval populations and yield responses obtained in spring threshold trials, in 1984/85; trial III (details as Table 2)

Treatment	Mean larval numbers per plant				Yield (tonnes per ha @ 92% d.m.)
	December	January	March	April	
HCH	NA	1.2***	NA	3.6**	2.91**
HCH+Ph	NA	1.1***	NA	0.5***	3.05***
Ph	NA	3.89	NA	0.9***	2.73*
U	3.8	4.7	4.1	6.8	2.54
S.E.D. (9 d.f.)	-	0.73	-	0.71	0.07

DISCUSSION

Analysis of data available from damage assessment trials on CSFB confirms the general validity of the currently used autumn treatment threshold of five or more larvae per plant in November/December and shows that by acting upon this threshold, signficant yield losses will normally be avoided. Preliminary data on the significance of spring populations enhanced by late hatching indicate that, in the absence of previous autumn treatment, an effective spring treatment will give a worthwhile yield response if larval numbers in February-March exceed five per plant. However, if autumn control has already been achieved subsequent spring hatching at the levels recorded does not appear to warrant the use of further control measures. This probably reflects the greater damage potential of larger instars derived from autumn hatching and the relatively greater susceptability of plants to mining damage prior to the resumption of rapid growth in spring. In consequence, effective autumn treatments which prevent mining by large overwintered larvae before the resumption of spring growth appear to produce the greatest yield benefit.

Early ADAS experience of trials work on CSFB using the then standard and comparatively non-aphicidal treatment gamma-HCH, frequently failed to show signficant yield responses at autumn larval population levels of at least five per plant (D.V. Alford, personal communication). Evidence presented here, however, shows clearly that using materials of wider and improved insecticidal activity, significant yield responses are readily obtainable at this population level. In view of this anomaly, recent

evidence of the widespread occurrence of Best Western Yellows Virus in winter oilseed rape in the UK (Smith & Hincks 1985) is particularly suggestive; at least some of the yield response obtained in CSFB damage-assessment trials using current insecticides may be attributable to the incidental control of the aphid vectors of BWYV. As yet, little is known about the epidemiology of this virus in OSR or about its effect on yield. Further ADAS trials on CSFB have therefore been modified to include a specifically aphicidal treatment which will not affect CSFB and BWYV infection is being measured on all treatments. In this way, it is hoped to quantify any virus component in yield responses to CSFB treatments which may then enable further revision of the treatment threshold for CSFB in those areas where virus is not present.

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NEW FOLIAR INSECTICIDES FOR THE CONTROL OF COWPEA PESTS

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ABSTRACT

Field studies conducted over a period of two years on the efficacy of a wide range of insecticides for the control of cowpea pests are reported. Several of the insecticides evaluated gave good control of most of the pests, with yields being increased 5- to 8-fold. It is concluded that the non-accessibility of insecticides to tropical farmers is the major factor limiting the adoption of insecticide use.

INTRODUCTION

The insect pest problem on cowpea (Vigna unguiculata) is one of the most intractable of any food crop around the world. An overlapping and persistent sequence of pests colonize the crop soon after germination through to crop maturation and subsequent storage. Yield losses due to insect pests are remarkably high, ranging from 20% in locations where the pest problem is slight to well over 60% where it is intense (Jackai et al. 1985). The pest problem and current methods aimed at controlling it has been the subject of several recent reviews, notably those by Jackai et al. (1985) and Jackai & Daoust (1986).

Among the known control options (Jackai et al. 1985) only insecticide use has shown yield increases of over five-fold. Their use and adoption by growers, however, has been very limited. Most cowpea growers in Nigeria and Niger (Africa) and in Brazil (Latin America), which together produce over 70% of the world cowpea, do not spray their crop. This, in part, explains the low yields. The reasons for the slow adoption of spray technology by tropical farmers are all centred around the gross non-accessibility (e.g. non-availability, prohibitive costs, etc.) of appropriate insecticides.

Several individuals and research organizations, particularly in Nigeria, have conducted research on chemical control of cowpea pests over the past two decades, the results of which have led to a reduced number of sprays, from over 10 in the 1960s (e.g. Booker 1965) to less than five today (Raheja 1978; Jackai & Singh 1983). We have also seen a dramatic move from the use of highly persistent products such as the chlorinated hydrocarbons to the use of more biodegradable chemicals. In addition, combined formulations ("insecticide cocktails") are gradually gaining prominence over single products. This paper reports on work conducted at the International Institute of Tropical Agriculture (IITA) on the efficacy of newer insecticide formulations in controlling cowpea pests. These insecticides represent mainly mixtures of safer and more biodegradable products.

MATERIALS AND METHODS

Ten insecticides were screened in 1984, with some of them tested at more than one dosage (Table 1). In 1985, thirteen insecticides were screened, one of them at two dose rates. Six of the products tested in 1985 had also been tested during the previous year (Table 1) and some for over 3 years (Anon. 1984a). Field testing was conducted only during the second cropping season (September to December) during which most cowpea in southwestern Nigeria is grown. Cypermethrin + dimethoate (as product F) was included in both tests as the standard, this material having been tested since 1982 (Anon. 1984a) with good results.

TABLE 1

List of insecticides tested in 1984 and 1985 at Ibadan, southwestern Nigeria

Insecticide	g a.i./l	Application rate (l/ha) 1984	1985
A - biphenate	100	-	0.5, 0.25
B - chlorpyrifos + dimethoate	222 + 278	-	1.0
C - chlorpyrifos + dimethoate	480 + 182	-	1.0
D - cypermethrin + chlorpyrifos	50 + 500	1.0, 0.5	1.0
E - cypermethrin + dimethoate*	30 + 40	0.5	0.5
F - cypermethrin + dimethoate	30 + 250	1.0	1.0
G - cypermethrin + dimethoate	30 + 400	1.0	-
J - cypermethrin + phosalone	30 + 500	1.0	-
K - cypermethrin + profenofos	40 + 400	-	1.0
L - deltamethrin + dimethoate	12.5 + 400	1.0, 0.5	1.0
M - endosulfan + deltamethrin	700 + 10	1.0	-
N - endosulfan + deltamethrin	500 + 10	1.0	0.5
O - flucythrinate	100	0.6, 0.5	0.6
P - flucythrinate + dimethoate	30 + 300	-	1.0
Q - heptenophos	550	1.0	-
R - isofenphos	500	1.0	-
S - PP321 + dimethoate*	10 + 40	-	0.5
T - methamidophos	600	1.0	-
U - profenofos	500	-	1.0

* = oil-based formulation.

Cowpea cv TVx 3236 was used in both trials. Plots had four replications, were planted in a randomized block design and consisted of 7-row plots, ten metres long. Spacing was 1.5 m between plots and 0.75 m between rows. Intra-row spacing was 0.2 m. Insecticides were applied at manufacturer's recommended dosages where only one dosage was tested, in addition to a fraction of this if more than one rate was evaulated. All insecticides were applied as a tank mixture with water, using a CP-3 knapsack sprayer at normal pressure, but the oil-based formulations of cypermethrin + dimethoate and PP321 + dimethoate were applied using an Electrodyne sprayer. During both years there was a control plot which was

sprayed with ordinary tap water. The trials were sprayed at about 35, 45, 50 and 55 days after planting (DAP). These times generally coincided with flower and bud production, full flowering, early podding and mid- to late podding. Previous work has shown these to be the critical periods for controlling flower thrips (Megalurothrips sjostedti Tryb.) (35 DAP), the Maruca pod borer (MPB) (Maruca testulalis Geyer) (45 and 50 DAP) and pod sucking bugs (PSBs), mainly Clavigralla tomentosicollis Stal. (55 DAP and onwards).

Weekly flower and pod production was monitored. In addition, two samples of 20 racemes (i.e. flower bud clusters) per plot and up to four samples of 20 flowers per plot were collected weekly. Raceme and flower samples were examined in the laboratory for infestation by thrips and the MPB. Racemes were first collected prior to the first spray; flower samples were first collected at the onset of flowering and, in a number of cases, fewer than 20 flowers were collected during the first sampling. Finally, visual counts were made of PSBs on three alternate rows (i.e. rows 2, 4 and 6) each week, starting with the first sighting of an adult PSB.

Grain yield was measured on the three centre rows and extrapolated to yield per hectare. Data were subjected to ANOVA with subsequent mean separation obtained using Duncan's Multiple Range Test (DMRT).

RESULTS

Control of flower thrips

In 1984, most insecticides gave reasonably good control of thrips in racemes, with the exception of heptenophos which gave higher counts than the control (Table 2). Generally, 2 or more thrips per flower bud are considered the action threshold (Salifu 1982) and, therefore, considering that most racemes averaged 3--5 buds, all treatments (including the control) had a population below this threshold. The flowers had a larger population of thrips than the racemes (Table 2). The smallest number of thrips in 1984 occurred on plots treated with isophenfos, cypermethrin + dimethoate, cypermethrin + chlorpyrifos (at 1 l/ha) and methamidofos. These treatments were all significantly better than the control ($P < 0.01$). Thrips numbers were larger on some treatments than on the control ($P < 0.05$) but most treatments more than doubled the production of fruit structures. This makes it difficult to explain the poor control obtained with such insecticides since thrips prevent/reduce flowering.

It appears that better control of thrips was obtained in 1985 (Table 3) but the lower counts may have been due in part to heavy rainfall. All treatments were better ($P < 0.05$) than the water-treated control, the best result being achieved with the two oil-based formulations which gave more than 30% better control than any of the water-applied products.

Maruca pod borer (MPB)

There was generally good to excellent control of MPB by most insecticides during both years, and even though flucythrinate + dimethoate at 60 g a.i./ha was numerically superior to all other treatments (Tables 2 and 3) there were no significant differences between most treatments. It is not unusual for the control plot to have a smaller number of MPB than some of the insecticide treatments. This was the case during both years (Tables 2 and 3) and can be explained by the fact that flower production on the control plots is generally so severely reduced by the

TABLE 2

Field evaluation of foliar insecticides for control of cowpea pests in 1984

Insecticide (see Table 1)	FTh/20 flowers	MPB/20 flowers	PSBs/6 m	Yield (kg/ha)
D (at 1 l/ha)	83.2 g	0.7 fg	3.0 bcd	1425.3 bcd
D (at 0.5 l/ha)	166.1 de	1.2 efg	4.5 bc	1551.0 abc
E	102.5 efg	2.4 d	1.0 e	1384.0 abc
F	96.7 g	2.2 cd	3.0 bcd	1681.1 ab
G	79.0 g	1.9 de	1.9 cde	1563.3 abc
J	70.1 g	0.9 efg	2.4 de	1628.0 abc
L (at 1 l/ha)	163.3 ef	3.3 bcd	3.9 bcd	1346.3 abc
L (at 0.5 l/ha)	259.0 cd	6.2 a	6.7 b	1217.8 abc
M	163.3 ef	1.0 fg	2.3 cde	1490.0 abc
N	257.4 cd	1.0 ef	6.4 bcd	1758.5 a
O (at 0.6 l/ha)	410.7 b	0.3 g	3.5 bcd	1560.3 bc
O (at 0.5 l/ha)	342.8 bc	1.2 efg	4.5 bc	1290.5 abc
Q	586.0 a	4.5 ab	10.0 a	1098.5 c
R	52.3 g	3.9 bc	3.0 bcd	1207.0 abc
U	97.2 fg	6.1 a	5.0 bcd	1134.5 bc
water-treated control	267.1 c	4.2 ab	4.1 bcd	410.8 d
S.D.	77.4	1.3	-	335.9

FTh = flower thrips; MPB = Maruca pod borer: PSBs = pod sucking bugs.

Figures followed by the same letter are not significantly different at P = 0.05 (DMRT).

Experiment planted on 10 September 1984; sprayed at 35, 45, 50 & 55 DAP.

TABLE 3

Field evaluation of foliar insecticides for control of cowpea pests in 1985

Insecticide (see Table 1)	FTh/20 flowers	MPB/20 flowers	PSBs/2 m row	Yield (kg/ha)
A (at 0.5 l/ha)	63.8 ab	1.9 abc	3.7 abcd	817.9 ab
A (at 0.25 l/ha)	99.5 b	2.2 abcd	7.0 e	787.7 ab
B	43.6 ab	3.1 bcd	6.4 de	637.9 b
C	62.2 ab	4.3 cd	6.8 de	643.4 b
D	66.8 ab	2.0 abc	7.7 e	734.1 ab
E	25.8 a	1.5 abc	2.5 a	905.8 a
F	51.3 ab	1.8 abc	3.4 abc	810.9 ab
K	101.7 b	1.2 ab	6.6 de	742.5 ab
L	62.3 ab	4.5 d	6.5 de	745.8 ab
N	83.4 ab	2.7 abcd	4.8 bcde	763.6 ab
O	87.0 ab	0.5 a	5.2 cde	756.5 ab
P	68.3 ab	1.4 abc	5.3 cde	972.8 a
S	29.3 a	1.4 abc	3.1 ab	932.5 a
T	49.8 ab	1.3 abc	6.2 de	721.9 ab
water-treated control	229.5 c	1.9 abcd	6.1 cde	115.9 c

FTh = flower thrips; MPB = Maruca pod borer; PSBs = pod sucking bugs.

Analysis was performed on transformed data. Values followed by the same letter are not significantly different at P = 0.05 (DMRT).

earlier infestation of thrips that such plants are not sufficiently attractive as oviposition sites for MPB. This results in less oviposition and, therefore, fewer larvae on control plots. Similar observations have been made by earlier workers (Anon. 1984a). Only heptenophos, methamidophos, isophenfos and deltamethrin + dimethoate gave less than satisfactory control of MPB.

Pod sucking bugs (PSBs)

Nine species of PSBs were sampled during both years. The results given in Tables 2 and 3 represent a pooled count of all species, with Clavigralla tomentosicollis the predominant (> 70%) one present. The best results were obtained with cypermethrin + dimethoate (oil-based), which gave significantly better control $P < 0.05$) than the standard. Any insecticide which performed signficantly worse ($P < 0.05$) than the standard was considered unacceptable for PSB control. Only heptenophos fell into this category (Table 2).

In the 1985 trial, seed damage by PSBs was assessed in addition to insect counts. This ranged from 75% for the control to 8% for PP321 + dimethoate. Overall, satisfactory control was obtained with several materials (Table 3). However, only four achieved values less than the action threshold of 2 bugs/row-m (Anon. 1984b). Cypermethrin + dimethoate (oil-based formulation) gave the best control, as in 1984, and was statistically better than most of the other test products. According to the 1985 results, several insecticides were considered unacceptable for PSB control using the criterion established in 1984; these included cypermethrin + profenofos, profenofos, deltamethrin + dimethoate, chlorpyrifos + dimethoate, cypermethrin + chlorpyrifos and biphenate (at 0.25 l/ha).

Yields

Grain yields were almost twice as high in 1984 compared to 1985 (Tables 2 and 3). This may be a reflection of a combination of factors among which larger pest populations and heavy rainfall (which, in a number of cases, coincided with dates of spray application) are noteworthy. The lowest yield in 1984 was obtained with heptenophos (1098 kg/ha), representing an increase of over 2.5 times that of the water-treated control. The highest yield was obtained with endosulfan + dimethoate (4.3 times that of the control). All treatments were statistically better ($P < 0.05$) than the control. Flucythrinate + dimethoate gave the best yield in 1985, followed by PP321 + dimethoate and cypermethrin + dimethoate (oil-based), but these values were not significantly different from thoae of several of the other products. However, all insecticide treatments increased yield ($P < 0.05$) over the control by 5- to 8-fold.

DISCUSSION

Cypermethrin + dimethoate, used as a standard in this study, has been tested for several years in Nigeria (Anon. 1983; 1984a) and is presently being used by a large proportion of those growers in southwestern Nigeria who spray their crop. The test insecticides that gave the same (or better) level of control as the standard are, therefore, the ones considered as having good prospects for use in cowpea insect pest control.

A few of the insecticides tested in this study show a good level of control of all the major pests monitored during 1984 and 1985. These

include cypermethrin + dimethoate and PP321 + dimethoate. These materials can be used with a fair amount of confidence to control the major pests of cowpea. In previous tests (Gowman & Durand 1986) the same results were obtained for the oil-based formulation of cypermethrin + dimethoate and for PP321 + dimethoate (also an oil-based product). A number of other materials gave marginal control of one of the pests but were quite effective in controlling the others. This is particularly so for those that did not control thrips very well. For instance, even though flucythrinate and its mixture with dimethoate, biphenate (at 0.5 l/ha), endosulfan + deltamethrin and deltamethrin + dimethoate did not give excellent control of thrips they were quite effective in the control of the other pests, in particular MPB. This is reflected in the grain yields (see later).

Systemic organophosphates such as dimethoate and monocrotophos are known to give excellent control of thrips (e.g. Anon. 1976). The mixtures of dimethoate with synthetic pyrethroids (each material on its own also controls thrips very well) were not effective in controlling thrips; this may be an indication of partial antagonism insofar as thrips control goes. It is also fairly obvious that the mixture of endosulfan (which does not control thrips effectively) + deltamethrin is also inadequate for the control of thrips at 500 g a.i./ha of endosulfan, suggesting a somewhat antagonistic combination.

Good control of thrips is very instrumental in obtaining high cowpea yields, since damage by this insect will forestall proper production of fruiting structures (Ezueh 1981). It is, therefore, not surprising that heptenophos, which gave rather poor control of thrips (Table 2), also gave the lowest yield of any test insecticide.

Even though the final grain yield is a function of the degree of success in controlling the individual pests, the ultimate yield quality will depend heavily on the degree to which PSBs are controlled, since these pests are the last colonizers of the crop, and can reduce a potentially good yield to nil (Anon. 1984a). Furthermore, since spraying was terminated around 55 DAP only those insecticides with good residual activity would provide a sustained level of PSB control. The data presented here (Tables 2 and 3) support this. In general, those insecticides that gave poor control of PSBs also had a numerically (and at times significantly) lower yield. During years with heavy rainfall (e.g. 1985), those insecticides without substantial rainfastness would get easily washed off, thus reducing their efficacy. This may explain, at least in part, the superior performance of the oil-based formulations across the board. Given this hypothesis a number of conventional products, such as flucythrinate + dimethoate and biphenate (at 0.5 l/ha) appear to possess sufficient ability to withstand easy wash-off. Although not every year can be expected to have heavy downpours, a clear reminder here is that insecticides formulated for use in the tropics should have rainfastness as one of their properties. Heavy rainfall is more the rule than the exception in the high rain forest belt of the tropics, where the insect pest problem is also worse.

In conclusion, our studies show that several new products are available for the control of the most important pests on cowpeas. In formulating them as mixtures, they provide an advantage to the farmer as he does not have to switch from one insecticide to another through the season. While these insecticide mixtures have proven to be more effective, due to their increased spectrum of activity, there are certain combinations (such

as endosulfan + deltamethrin) that will not control aphids, another important pest in the drier areas, and which appear to demonstrate antagonism between the constituent compounds. Systemic materials such as dimethoate or a specific aphicide appear to be necessary either as mixtures or as single formulations to control aphids.

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SESSION 7B

FUNGICIDE RESISTANCE: CURRENT INCIDENCE AND STATUS OF RESEARCH

CHAIRMAN PROFESSOR J. DEKKER

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INVITED PAPERS 7B-1 to 7B-5

STATUS AND HANDLING OF FUNGICIDE RESISTANCE IN PATHOGENS OF GRAPEVINE

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ABSTRACT

As in other crops, the introduction since the early 1970's of single-site fungicides in grapes was followed by the emergence of resistant strains in some of the target pathogens. The three major cases of fungicide resistance in pathogens of grapes have occurred in Botrytis cinerea to benzimidazoles and to dicarboximides, and in Plasmopara viticola to phenylamides. In all three cases resistance emerged most rapidly in areas of highest disease pressure. Resistance to benzimidazoles in Botrytis emerged very rapidly in 1973 and has since lead to their withdrawal in some countries. Botrytis strains resistant to dicarboximides are less fit than sensitive ones. Consequently resistance emerged somewhat more slowly and performance problems occurred only in 1982, several years after resistant strains were detected in monitoring programs. These compounds continue to be useful if used only once or twice in a season. Resistance of P. viticola to phenylamides was first detected in monitoring programs in 1981. Performance problems were rare because these compounds were applied in mixtures with residual fungicides. While it is unlikely that benzimidazoles will be used again against Botrytis where high levels of resistance are present, the dicarboximides and the phenylamides appear to become effective again, after their use has been stopped for some time in areas where resistance has reached high levels in their target pathogens.

INTRODUCTION

Vine growers world-wide are dependent on fungicides for the production of stable yields of grapes with good qualities. The most important diseases of Vitis vinifera are downy mildew (Plasmopara viticola), powdery mildew (Uncinula necator), grey mold (Botrytis cinerea), black rot (Guignardia bidwellii) and excoriose (Phomopsis viticola). The use of fungicides has evolved from copper and sulphur, to organic multi-site fungicides with residual-protective action and finally to specific, single-site compounds, some of which have curative and systemic action.

As in other crops, it was only with the introduction of single-site fungicides, that fungicide resistance became a problem for vine growers (Table 1). Major cases of resistance that affected performance occurred in B. cinerea to benzimidazoles in 1972/73 (Ehrenhardt et al. 1973, Schüepp & Lauber 1977), then in the same fungus to dicarboximides 1979-82 (Schüepp & Siegfried 1983, Löcher et al. 1985) and in P. viticola to phenylamides in 1981 (Staub & Sozzi 1981). An isolated report of Uncinula resistance to benzimidazoles in the US is mentioned here for completeness (Pearson 1980); it will not be considered further since no follow-up information is available. For the other site-specific fungicides no resistance has arisen so far. Indications of resistance in Botrytis to multi-site fungicides have come only from laboratory data

(Barak & Edgington 1984) and lack evidence that such resistance affects field performance.

TABLE 1

Specific fungicides used against the major grape pathogens and first occurrences of field resistance

Fungicide group	Pathogen	Introduction	First reports of field resistance	
			Year	Countries
Benzimidazoles	Botrytis	1971	1973	Germany, Switzerland
	Uncinula	1974	1976	USA
Dicarboximides	Botrytis	1976	1982	France, Germany, Switzerland
Phenylamides	Plasmopara	1978	1981	France, South Africa
Fosetyl-Al	Plasmopara	1978	-	
Cymoxanil	Plasmopara	1978	-	
DMI-compounds[1]	Uncinula	1976	-	
	Guignardia	1976	-	

[1] Inhibitors of sterol biosynthesis

This paper describes the characteristics of the three major cases of resistance in pathogens of grape as they relate to a) the emergence and detection of resistance, b) performance problems due to resistance, c) dynamics of resistant populations and d) fungicide use strategies that were designed to cope with resistance.

DETECTION OF RESISTANCE IN RELATION TO PEFORMANCE PROBLEMS

Each case of fungicide resistance analysed in this paper shows specific relationships between the detection of resistant strains and the occurrence of performance problems in the vineyards. With benzimidazole resistance in Botrytis, monitoring data merely confirmed what was all too evident from performance problems, while in the other two cases resistant strains were detected before major performance problems occurred.

Awareness of the resistance risk

The amount and the nature of information available about the three major cases of fungicide resistance is variable due to the differing degrees of awareness among plant pathologists as to the problem of fungicide resistance. In 1973, when benzimidazole resistance appeared in Botrytis, it was the first case of fungicide resistance in vineyards. No monitoring programs were in place and the rapidity of the emergence of resistance surprised everybody. This precluded detailed studies of the early events in resistance selection. In the case of dicarboximides, the increased awareness of a resistance risk and the slower emergence of resistance allowed several detailed studies on the early events of resistance selection in Botrytis populations before performance problems

evident. With phenylamides, which were used in mixtures with residual compounds from the start, the resistance risk was known in 1981 and again monitoring programs were in place to detect resistant strains early.

Nature of resistant strains from the field

The resistance factor for benzimidazole resistant Botrytis strains is usually in excess of 1'000, while for Botrytis field isolates with dicarboximide resistance, it is usually between 10 - 20 (Leroux and Gredt 1984). A second type of stable resistance to dicarboximides with a much higher resistance level can easily be produced in vitro by prolonged exposure on agar. Although such strains are usually not found in the field, they can mislead inexperienced investigators. Cross-resistance between all related fungicides is the rule within both groups of fungicides. In addition, many strains with double-resistance to both groups can be found. The resistance factor in phenylamide resistant strains of Plasmopara can range from 10 to 10'000. Such strains are basically cross-resistant to all phenylamides, though the resistance factors between some of them are poorly correlated (Clerjeau et al. 1985, Diriwächter et al. 1986).

Background of fungicides use

The technology available for disease control was very different for the two pathogens in question when the fungicides at risk were first introduced. Prior to the benzimidazoles there was no highly effective control of grey mold of grapes. Growers depended on cultural practices that reduced the infection pressure, and on the side-effect of some residual fungicides. The same situation occurred again when the dicarboximides were introduced in areas of high infection pressure where the benzimidazoles had lost their effectiveness due to resistance. Meanwhile many of the growers had come to rely heavily on the benzimidazoles and abandonned some of the cultural measures. The phenylamides (and the other specific fungicides against Plasmopara), on the other hand, complemented the already existing technology for downy mildew control. For reasons of spectrum of activity, reducing the risk of resistance and synergism for higher activity, they were used from the start in combination with residual products. This slowed down the development of resistance in P. viticola. The mixtures also prevented major performance problems where resistance was detected in monitoring tests.

Detection methods used

Detection of resistance is dependent on the execution of a monitoring program and on the methods used to determine the fungicide sensitivity of isolates. Performance failures alone should never be taken as sufficient evidence for resistance. For Botrytis sensitivity to benzimidazoles or dicaboximides in vitro tests on agar plates are most commonly used. Some researchers use ammended agar for spore germination tests (Leroux & Clerjeau 1985) or inhibiton of mycelial growth (Schüepp & Küng 1981), while others use agar diffusion tests with treated filter paper discs (Ehrenhardt et al. 1973) to determine the sensitivity of Botrytis isolates. It is therefore not possible to compile or compare quantitative data from different sources. Rather, each type of study has to be reviewed on its own and the qualitative statements compared. The detection limit for bulk isolates of Botrytis is usually not known. For Plasmopara a leaf disc test is most commonly used; its detection limit for mass isolates is between 1 and 10 % resistant spores (Clerjeau et al. 1985). The detection limit can also be influenced by the number and the size of the samples.

DYNAMICS AND DISTRIBUTION OF FUNGICIDE RESISTANCE

The principal questions here are how the resistant fungal populations are distributed geographically, how they respond to continued use of the selective fungicide and how they compete in nature with the sensitive populations after the use of a selective fungicide group has been discontinued.

Resistance of Botrytis to benzimidazoles

First evidence of resistance in this case was a rapid loss of disease control at the northern fringe of the grape growing area in Europe from 1972 to 1973 (Ehrenhardt et al. 1973, Schüepp & Lauber 1977). This corresponds with the areas of highest infection pressure by this pathogen. Use of benzimidazole fungicides was stopped soon after in these areas, because resistance spread rapidly to all vineyards and loss of disease control was complete. In southern Europe, where the Botrytis pressure is much weaker, the resistance to benzimidazoles emerged more slowly and these fungicides are still used (Gullino & Garibaldi 1985).

There is very little monitoring information available on the events leading to the first cases of resistance. However, subsequent studies showed, that the resistant strains are extremely fit and can compete well with sensitive populations in nature. Schüepp & Küng (1981) compared the resistance in Botrytis isolates collected from Eastern Switzerland in 1974 (at the time of withdrawal of the benzimidazoles) with a similiar collection made in 1978 (Table 2). In 1974 he found one third of the samples from non-treated vineyard to be resistant, which suggests a high mobility and a high fitness of the resitant strains. High fitness of the resistant strains is also indicated by the average level of resistance, that was around 60 % in both years. Similiar results were reported by Leroux & Gredt (1984) for the Alsace and Champagne regions where they found 50 and 90 % resistance, respectively, six years after the use of benzimidazoles had stopped.

TABLE 2

Benzimidazole resistant isolates of Botrytis cinerea from Swiss vineyards in relation to previous use of fungicides from this group (Schüepp & Küng 1981)

Number of benzimidazole applications	% resistant isolates		Number of isolates	Change in % resistant isolates
1971-1973	1974	1978	each year	1974-1978
0	33	47	150	+ 14
1- 4	61	56	80	- 5
5-14	72	61	525	- 11
Overall	62	57	755	- 5

Resistance of Botrytis to dicarboximides

Resistance to dicarboximides in Botrytis on grapes appeared first and lead most rapidly to a reduction in disease control in the same areas that were first involved in resistance to benzimidazoles. With the dicarboximides it took roughly twice as long (from 1976 to 1982) for the compounds to lose activity, and the loss in most cases was not as complete as it had been with the benzimidazoles (Leroux & Besselat 1984, Schüepp & Siegfried 1983).

As with the benzimidazoles there exists a distict gradient of resistance levels in the Botrytis populations from the northern to the southern part of the European grape regions. While resistance approached nearly 100 % in 1982 in Swiss vineyards with high disease pressure (Schüepp & Siegfried 1983), it is still at a very low level in the vineyards of Italy (Gullino & Garibaldi 1985). The same north-south gradient could be described by Leroux & Besselat (1984) for the French vine-growing regions (Fig. 1). Data from the Champagne region for both 1982 and 1983 showed a high level of resistance in the tested isolates and a low performance of the dicarboximides. By contrast, the areas of Southern France showed no resistance and excellent performance by these fungicides.

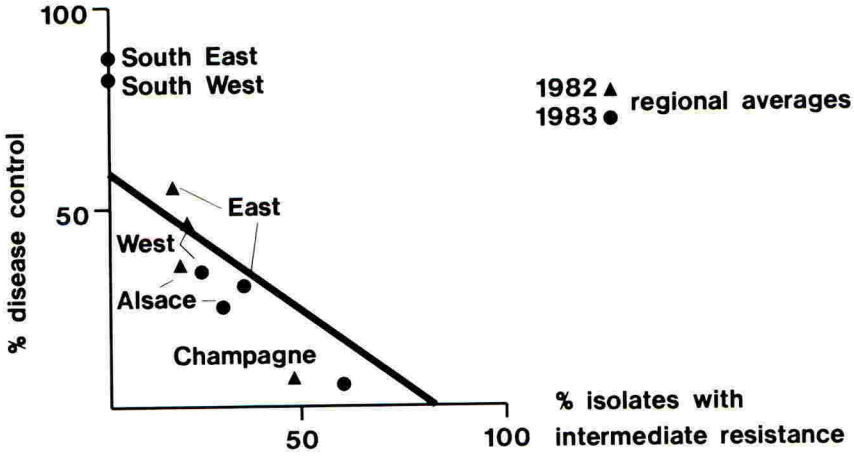

Fig. 1. Control of B. cinerea with 4 dicarboximide treatments in relation to occurrence of dicarboximide resistant strains in French vineyards in 1982 and 1983 (Leroux and Besselat 1984).

The gradient of resistant strains between dicarboximide treated and untreated vineyards is more pronounced than was the case with benzimidazole resistant strains. In addition, dicarboximide resistant strains decreased noticeably after an interruption of treatments with dicarboximides. Data compiled by Moncomble (1986) for the Champagne illustrates these points well (Fig. 2). In 1980 untreated and treated vineyards yielded 5 and 85 % resistant strains, respectively. The resistance in untreated vineyards reached 59 % in 1983, when the use of

dicarboximides was officially dicontinued. Over the next two years the resistant strains decreased again to 26 % in untreated vineyards in 1985. Throughout the study the samples from vineyards with one dicarboximides treatment yielded less resistant isolates than those with two treatments. This suggests a fitness disadvantage of the dicarboximide resistant isolates, that limits their spread into non-treated areas and reduces their proportion when the treatments are discontinued.

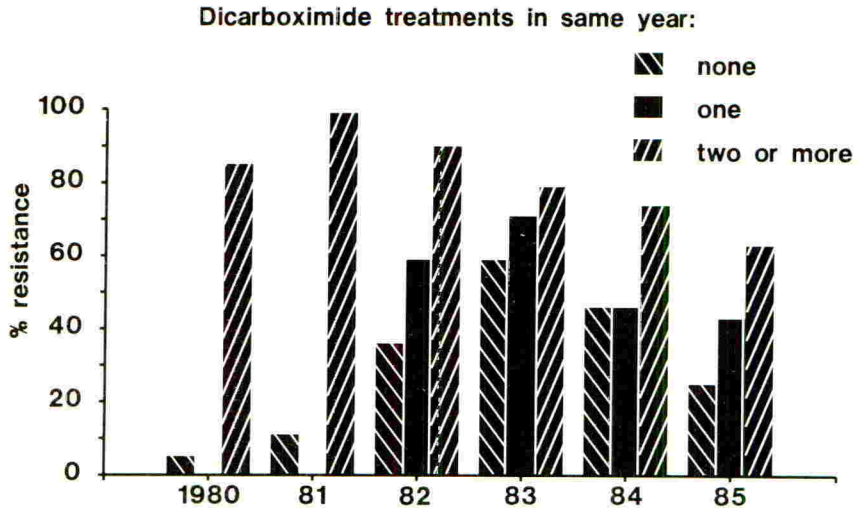

Fig. 2. Development of dicarboximide resistance in B. cinerea in the Champagne in relation to the number of dicarboximide treatments (Moncomble 1986).

Resistance of Plasmopara to phenylamides

Resistance to phenylamides in P. viticola was first detected in samples from routine monitoring programs in South Africa and Southwestern France (Staub and Sozzi, 1981). It developed in spite of the use of mixtures with residual compounds; nurseries with especially high disease pressure or experimental plots with treatments of straight phenylamides may have served as the first foci of resistance in both areas.

Subsequent evolution of fungicide resistance in different regions of France varied, depending on phenylamide usage (Table 3). The Cognac area was the first to show wide-spread resistance and it produced close to 100 % resistant isolates in 1983 and 1984. After the phenylamide treatments had virtually stopped, the first signs of a reduction in the resistant population were evident in 1985 (Moreau et al. 1985). In the south of France, phenylamide resistance remained stable at an intermediate level from 1983 to 1985, while it is still virtually absent in the Alsace and the Champagne. Outside France, resistance was detected in Greece in 1982, in Switzerland in 1983 and in Italy in 1985 (Ciba-Geigy, unpublished monitoring data). The variable resistance levels from year to year and the reduction of resistance where

phenylamide use is stopped indicates a fitness disadvantage of the resistant strains, which makes it difficult for them to become firmly established in the population. One such disadvantage may be the increased sensitivity to high temperatures (Piganeau & Clerjeau 1985).

TABLE 3

Percent sites with phenylamide resistant samples of Plasmopara viticola from vineyards not treated with phenylamides (Moreau et al. 1985)

	1983	1984	1985
Cognac	93	97	74
Bordelais	17	7	6
Armagnac	-	30	39
Val Loire	46	27	44
Bourgogne	6	11	25
Champagne	1	0	0
Alsace	0	0	0

In all these cases, the use of fungicide mixtures prevented severe performance problems and crop losses. It is through the systematic monitoring programs that the distribution of resistance to phenylamides in P. viticola could be described in these areas.

COUNTERMEASURES USED AND OUTLOOK

Benzimidazoles

At the time of their introduction for use against Botrytis on grapes the benzimidazoles were exposed to the worst possible conditions for the development of resistance. No monitoring was in place and no countermeasures were developed because the awareness of the resistance risk was low. In addition, there were no other effective fungicides that could have been used in combination or in alternation with benzimidazoles. The resistant strains proved to be very fit and remained in the populations at high levels even after use had been stopped for 10 years. The outlook is bleak for this group of fungicides to ever play a major role again in Botrytis control in areas of high disease pressure. It will be interesting to see whether the carbamates with negative cross-resistance to benzimidazoles (Suzuki et al. 1984) will be introduced against Botrytis in grapes. Mixtures with benzimidazoles would be required for good performance and three-way mixtures or other countermeasures needed to avoid the emergence of resistance to both agents.

Dicarboximides

The general use strategy for dicarboximides against Botrytis has been defined by the FRAC (Fungicide Resistance Action Committee) working group dealing with this group of fungicides (Löcher et al. 1985; Anonymous 1986a). As in 1985, the use of dicarboximides was not recommended in Champagne for 1986, while in other areas with resistant strains a limitation to two sprays per season is recommended. The

intention is to exploit the reduced fitness of resistant strains by allowing them to fall back to levels where dicarboximides might be used again. For other regions this approach stabilizes resistance at low levels that assure the continued usefulness of the dicarboximides. More specific recommendations are given by officials depending on the level of resistance in individual vineyards and on the dicarboximide used in previous years (Anonymous 1986b). The importance of reducing the Botrytis pressure by cultural measures, such as limited nitrogen fertilization and removal of leaves around the grapes, is also stressed in several countries (Annonymous 1986d). The outlook for dicarboximides is clearly more favorable than for benzimidazoles, and they are likely to retain a significant role in Botrytis control.

Overall the greatest deficiency in attempts to combat resistance in Botrytis is the absence of effective alternatives to the benzimidazoles and the dicarboximides, that could be incorporated into anti-resistance strategies. Until such alternatives are found, cultural practices and the use of multi-site fungicides with side-effects against Botrytis remain the only means to fight the further progress of resistance.

Phenylamides

Here too, the cooperation between the different producers of phenylamides within FRAC has lead to a generally accepted anti-resitance strategy (Urech & Staub 1985). It relies on the use of prepack mixtures with residual fungicides and a limitation on the number of applications per season. In addition, the curative use is not recommended. In France, restricted use of phenylamides was again recommended for the Cognac area in 1986, after the monitoring data of 1985 had indicated a reduction in resistance in that area during non-use of phenylamides (Anonymous 1986c).

The general outlook for downy mildew control is better than that for Botrytis. There are several alternatives to the phenylamide mixtures that give sufficient control in most disease situations. Beside the purely residual multi-site products, these fungicides are available in mixtures with cymoxanil, that has some curative action, and with fosetyl-Al, that also has systemic properties.

Other specific fungicides

Of the other specific fungicides, fosetyl-Al and cymoxanil are both heavily dependent on the residual mixture partners to give satisfactory control of downy mildew. This weakness becomes their strength in the fight against the emergence of resistant strains, which theoretically is also possible to these compounds.

The DMI-compounds are increasingly used against powdery mildew and black rot. They are so potent, especially against powdery mildew, that they excert a strong selection pressure for resistance in U. necator. It would not be surprising, therefore, if in areas of high infection pressure, this pathogen responds with a shift towards decreasing sensitivity, as has been observed in cucumber powdery mildew (Anonymous 1986a). In such areas it would be advisable to use DMI-compounds in combination with other agents active against powdery mildew.

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CURRENT INCIDENCE IN THE UNITED KINGDOM OF FUNGICIDE RESISTANCE IN PATHOGENS OF CEREALS

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ABSTRACT

Resistance to fungicides has occurred in a range of cereal pathogens in the past twenty years. The paper briefly reviews the history of these cases and gives information on the current incidence of fungicide resistance in cereals. Twelve pathogen/fungicide/host combinations are discussed covering mildew, Septoria leaf spot, eyespot, Fusarium spp, Botrytis cinerea, loose smut and barley leaf stripe and leaf spot of oat.

INTRODUCTION

Over the past twenty years there have been a number of reports of the resistance of various cereal pathogens to a range of fungicides. Most of these cases have been of great importance to manufacturers, advisors and farmers. This paper reviews the current situation regarding fungicide resistance in cereals and deals at varying length with twelve pathogen/fungicide/crop combinations (Table 1).

CASE HISTORIES

Pyrenophora avenae

The first case of fungicide resistance in cereals was reported by Noble et al in 1966 and concerned the discovery in Scottish seed oats of mercury-resistant strains of the oat leaf spot pathogen Pyrenophora avenae. Subsequently, similar resistance was reported from Northern Ireland (Malone, 1968) and England and Wales (Dickens & Sharp, 1970). In the latter case resistant isolates of the pathogen were found in 32 of 123 winter oat seed samples and 31 of 33 spring oat samples. There are no recent detailed studies of the status of this pathogen but it is assumed that mercury resistance is common.

Pyrenophora graminea

Leaf stripe, caused by Pyrenophora graminea, occurs sporadically in many barley crops but has seldom caused significant yield losses. Organomercury seed treatment has for many years been a cheap and effective control of this disease. However, in 1984 poor control occurred in spring barley, cv Mazurka, on crops originating from one seed source where a commercial seed treatment containing phenyl mercury acetate (PMA) had been used. Studies showed whilst all 35 isolates from affected seed and from crops grew on agar containing mercury at 2 µg/ml and some at 20 µg/ml, other isolates from untreated seed of cv Athos were inhibited at 2 µg/ml mercury (M R M Clark, pers. comm.). In subsequent field trials carried out in Wiltshire and Scotland in 1986 with cv Triumph, extremely poor control of leaf stripe by PMA was demonstrated. (D R Jones, pers. comm.). Laboratory studies on isolates of P. graminea from 7 sources in England

TABLE 1

Current cases of fungicide resistance recorded in the United Kingdom

Pathogen	Fungicide group	Crop
Pyrenophora avenae	organomercury	oat
Pyrenophora graminea	organomercury	barley
Ustilago nuda	carboxamide	barley
Pseudocercosporella herpotrichoides	benzimidazole	wheat
Pseudocercosporella herpotrichoides	benzimidazole	barley
Fusarium nivale	benzimidazole	wheat
Fusarium culmorum	benzimidazole	wheat
Botrytis cinerea	benzimidazole	wheat
Septoria tritici	benzimidazole	wheat
Erysiphe graminis f. sp *hordei*	sterol C-14 demethylation inhibitor	barley
Erysiphe graminis f. sp *tritici*	sterol C-14 demethylation inhibitor	wheat
Erysiphe graminis f. sp *hordei*	hydroxypyrimidine	barley

and Scotland were carried out in 1985 (D R Jones, pers. comm.). The isolates from 3 sources were sensitive to mercury with ED_{50} values of below 0.2 µg/ml, whereas a resistant isolate had an ED_{50} value of 3.1 µg/ml.

Ustilago nuda

In 1984 there were several reports in the UK that seed treatment of winter barley with carboxin for the control of loose smut (*Ustilago nuda*) did not give satisfactory disease control. In most cases, but not all, the problems were on stocks of cv Panda which originated in France. It was subsequently found that resistant strains of the pathogen had an ED_{50} of 2.0 µg/ml. ADAS trials in 1984/85 and 1985/86 showed unacceptable levels of loose smut control by carboxin in two stocks of cv Panda, whereas it was effective on stocks of cvs Gerbel, Igri and Sonja. (D R Jones, pers. comm.). In field surveys in France in 1985 resistance was found in virtually all samples of cv Viva examined, and a proportion of samples of many other cultivars including Panda, Gerbel and Barberusse (P. Leroux

pers. comm.).

Pseudocercosporella herpotrichoides

From 1974 until the early 1980's there was an increasing use of benzimidazole fungicides for the control of eyespot, caused by Pseudocercosporella herpotrichoides. The first reports of failure to control eyespot occurred in 1981 when two winter wheat crops, in Suffolk and Gloucestershire, suffered severe attacks despite the use of a benzimidazole fungicide at growth stage 30-31 (Griffin et al. 1982). A survey of benomyl resistance in the eyespot pathogen was carried out on over 500 crops of winter barley and winter wheat in England and Wales in 1983 (King & Griffin, 1985). In randomly selected crops sampled in July, 37% of isolates from winter wheat tillers were resistant to benomyl. In winter barley 51% of isolates were resistant. In a smaller survey in the East of England in 1984, carried out jointly by the Plant Breeding Institute at Cambridge and ADAS, 75% of winter wheat isolates and 79% of winter barley isolates were found to be benzimidazole-resistant. In a similar survey in 1985 the proportion of resistant isolates had risen to 84% for wheat and 94% for barley.

Fusarium spp

During May and June 1985 several cases of severe brown foot rot infection of wheat with Fusarium nivale (Microdochium nivale) were seen in Essex and Suffolk (R J Cook, pers. comm.). Initial tests were made on 24 isolates from seven fields for resistance to benomyl.

All isolates grew equally well on unamended agar and on plates containing 5 µg/ml benomyl. A selection of these isolates were further tested at concentrations up to 200 µg/ml and growth still occurred. Resistance of F. nivale to benzimidazole fungicides has previously been reported in West Germany (Harthe & Buchenauer, 1985), Sweden (Olvang, 1984) and Japan (Tanaka et al., 1983). The same year 18 isolates of Fusarium culmorum from a trial in Lincolnshire were tested for benomyl resistance (P Gladders, pers. comm.). Three of these grew on agar amended with 5 µg/ml benomyl.

A survey of 100 wheat crops for Fusarium spp resistance to benzimidazole fungicides is being carried out in 1986 as a component of the ADAS National Cereal Disease Survey of winter wheat. With approximately a third of the laboratory tests completed the main species found is F. nivale and over 90% of isolates are benzimidazole-resistant.

Erysiphe graminis f. sp. hordei

In the early 1970's disease control of Erysiphe graminis on barley was dependent upon two chemicals, ethirimol and tridemorph. After a few years usage, however, disease control by ethirimol became less effective because of decreased sensitivity (Bent, 1978). The fungicide was used as a seed treatment on both autumn and spring sown crops and consequently the pathogen population was exposed to ethirimol throughout most of the year. After the manufacturer withdrew the autumn use recommendation and other types of mildew fungicides were marketed the general use of ethirimol declined. The sensitivity of the pathogen population to ethirimol subsequently increased. Experiments by Hollomon et al. in 1985 showed that following the use of ethirimol either as a seed treatment, or as a seed treatment plus subsequent sprays, the sensitivity of the population rapidly decreased. It will therefore be of interest to see how

E. graminis responds to the mixed triazole-ethirimol seed treatment recently introduced (Northwood et al., 1984).

When the sterol C-14 demethylation inhibitor (DMI) fungicide triadimenol was first marketed as a seed treatment in 1980 the level of mildew control was considerably better than that obtained with any other product (Martin et al., 1981). However, the closely related fungicide triadimefon had already been marketed as a foliar spray for 3 years and some studies were already reporting increasing resistance to the DMI group of fungicides (Fletcher & Wolfe, 1981). Information from ADAS spring barley experiments from 1978 to 1984 showed that the level of mildew control and yields obtained from treatment with triadimefon changed considerably when compared to the data for the morpholine fungicide tridemorph (J E E Jenkins, pers. comm.). In the early years of this series of trials, triadimefon always out-performed tridemorph, but by 1984 the converse was the case. As there is no evidence of a change in sensitivity to morpholines in the mildew population the conclusion is that there is increased resistance to triadimenol and triadimefon.

A similar trend was found in the UK Cereal Pathogen Virulence Survey but by 1983 resistance to DMIs appeared to be declining in Scotland (Wolfe et al., 1983). The reason for this was probably the increasing use of morpholine fungicides in that country as farmers followed the advice of the Scottish Colleges of Agriculture to alternate fungicides from different mode-of-action groups (Gilmour, 1984).

Erysiphe graminis f. sp. tritici

In 1983 it was reported (Bennett & van Kints) that in East Anglia there had been a general decrease in sensitivity of the wheat mildew population to DMI fungicide triadimenol over the previous three years. The pathogen, Erysiphe graminis f. sp. tritici, became more resistant the following year (Summers & van Kints, 1984) but the sensitivity was dynamic in character and levels fluctuated throughout the year due to selection pressure from fungicide applications. Resistance levels increased to a peak in late June and then declined in early August.

The same trend was observed in the South East of England in 1985 (J T FLetcher, pers. comm.). By expressing the results as FD_{50} values (the fungicide dose as a seed treatment which decreases by ca 50% the level of mildew in comparison with untreated test plants) a field survey of 15 crops showed that sensitivity to DMIs varied from FD_{50} 37.5 to FD_{50} 8.0; the values quoted are g. a.i. triadimenol per 100 kg. seed, 37.5 being the commercial rate of use. It would seem from this data that in some cases the standard rate of product only halved the mildew level compared to untreated plants. This finding is consistent with many field observations that this group of fungicides no longer gives effective control of wheat mildew.

Septoria tritici

Studies in 1984 in South West England indicated that benzimidazole-resistant strains of Septoria tritici might be widespread (Griffin & Fisher, 1985). Consequently a survey of 104 winter wheat crops in England and Wales was undertaken in the spring of 1985. The results showed that of 968 isolates tested, 73% were resistant to benomyl at 2 µg/ml, a concentration which controls sensitive isolates (M J Griffin, pers. comm.).

At one time the use of benzimidazole fungicides at growth stages 30-31 for the control of eyespot gave a useful suppression of Septoria leaf spot. It appears that control of this pathogen by this group of fungicides is now generally unlikely.

Botrytis cinerea

Botrytis cinerea can cause ear infections on wheat and occasionally can infect leaves by first colonizing fallen pollen deposits on the leaf surface. In 1985, 68 isolates of B. cinerea from twelve crops were checked for benomyl resistance and 65 grew on 5 µg/ml amended agar (P Gladders, pers. comm.). This result is perhaps not surprising as surveys of the resistance status of this pathogen on horticultural crops have shown that approximately half of isolates tested were resistant to benzimidazoles (Fletcher, 1985).

Other studies

Various plant pathologists are monitoring the effectiveness of other pathogen/fungicide sensitivities on cereals. In the next few years papers might be expected on such combinations as prochloraz and P. herpotrichoides, benzimidazoles and DMIs on Rhynchosporium secalis and benzimidazoles on Leptosphaeria nodorum.

DISCUSSION

The results of the experiments and surveys over the past few years indicate that in many cases useful and often cheap control of some diseases with some fungicides is no longer possible.

Organomercury for the control of oat leaf spot and barley leaf stripe is no longer a certainty. The loss of control of loose smut by carboxamide fungicides has led to the withdrawl of some label recommendations. With stem base diseases, the chances are now slim of getting good control of eyespot with benzimidazoles, and the same may be true of Fusarium brown foot rot.

Of the various leaf diseases of cereals, control of Septoria leaf spot with benzimidazoles has largely been lost, and the same could possibly be true of the minor wheat pathogen Botrytis cinerea. In the case of barley mildew, farmers must alternate the available products and not be too dependent upon one group of fungicides. Only in this way can the usefulness of ethirimol and the DMI compounds be maintained. Lastly, there is now less likelihood of benzimidazoles controlling ear blights caused by Fusarium spp and Botrytis cinerea.

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SENSITIVITY OF PSEUDOCERCOSPORELLA HERPOTRICHOIDES POPULATIONS TO PROCHLORAZ

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ABSTRACT

224 isolates of P. herpotrichoides from 1985 field trials in the UK, France, Germany, Denmark and Eire were evaluated in vitro for susceptibility to a range of concentrations of prochloraz. The levels of sensitivity were similar to those found by other workers for isolates from previous years, confirming that there is no evidence of any decrease in sensitivity since the introduction of prochloraz for eyespot control in 1981.

Isolates with the slow, feathery growth pattern (R types) were significantly more susceptible to prochloraz than those with a fast growth rate and even-edged colony morphology (W types). There was no evidence of a correlation between resistance to benzimidazole fungicides and reaction to prochloraz.

INTRODUCTION

Eyespot of cereals caused by Pseudocercosporella herpotrichoides is a major cause of yield loss in European agriculture. The disease symptoms are characteristic oval lesions on the stem bases of wheat, barley and rye. Yield losses may be due either to premature ripening of diseased plants or to lodging in crops where the stems are weakened by the fungal attack.

Fungicides of the benzimidazole structural type, e.g. carbendazim and benomyl have been used to control eyespot for a number of years and have given excellent results, in terms of both disease control and yield. However, resistance to these compounds is now widespread in many areas of western Europe (Russell and Birchmore, 1986) and prochloraz is the main other commercial treatment that gives acceptable control of this pathogen (Anon., 1986). Prochloraz acts by inhibiting the C 14 demethylation step in fungal ergosterol biosynthesis (Copping et al, 1984), unlike benzimidazoles which affect tubulin synthesis (Davidse, 1973). Prochloraz, therefore, controls both benzimidazole sensitive and resistant strains.

In view of the rapid rise in benzimidazole resistance (King and Griffin, 1985) and the concern that this could also occur with other fungicides which act at a single biochemical site, FBC and latterly Schering, have carried out a survey of sensitivity of P. herpotrichoides to benzimidazoles and prochloraz since 1982. Isolates of the pathogen were obtained from infected stems and tested for sensitivity to single concentrations of each compound incorporated into agar. The concentrations used were 2 mg l^{-1} of carbendazim, with prochloraz initially being used at this rate also. However, the concentration of the latter was reduced to 0.5 mg l^{-1} in 1985 in order to increase the sensitivity of the assay. The results of this programme of work were described by Russell and Birchmore (1986) and showed that, while large numbers of P. herpotrichoides isolates were resistant to carbendazim, none grew on agar containing prochloraz.

Selected isolates of the pathogen have also been evaluated against a series of concentrations of prochloraz in order to determine levels of sensitivity more precisely and to form a base-line for future work. The first report of this investigation was by Gallimore, Knights and Barnes (in preparation) who examined a total of 296 isolates of which 20 were derived from 1985 field trials, 194 from 1984, 50 from 1983 and 32 were isolated at the Plant Breeding Institute, Cambridge between 1956 and 1981. These workers found that the mean concentration required to inhibit colony growth by 50% (IG_{50}) was 0.044 mg l^{-1} prochloraz for isolates from 1984 with no discernible decrease in sensitivity to the compound over the period 1956 to 1986.

Most isolates of the pathogen show either a fast growing, smooth-edged colony morphology or a slow growing, feathery-edged appearance. These have been found to correspond to the W and R infection types of the pathogen, respectively (King & Griffin, 1985; Hollins et al, 1985). Gallimore et al found that R type isolates of the pathogen tended to be more sensitive to prochloraz than W types.

The objective of this paper is to present data on a further 224 isolates, all derived from 1985 trials in the UK, France, Germany, Denmark and Eire, comparing these results with those from the previous studies.

MATERIALS AND METHODS

Isolates of P. herpotrichoides were derived from samples of stems with eyespot lesions collected from treated and untreated plots of field trials. The lesions were cut from the stem bases, re-hydrated by immersion in running tap water for 1 hour and then surface sterilised for 5 minutes in sodium hypochlorite solution. This was removed by washing in sterile distilled water, before a section of tissue of approximately 2 mm^2 was cut from the centre and edge of each lesion. Tissue sections placed on potato dextrose agar (PDA, Oxoid) containing 0.2 mg l^{-1} tolclofos-methyl, 125 mg l^{-1} streptomycin, 10 mg l^{-1} rifampicin and 500 mg l^{-1} ampicillin. The tolclofos-methyl was included to inhibit another common stem base pathogen, Rhizoctonia cerealis, while the purpose of the antibiotics was to inhibit growth of bacterial contaminants.

After 14 days incubation at 20 °C developing colonies of P. herpotrichoides were transferred to unamended PDA, with only one isolate per lesion being retained. When sufficient growth had occurred, 3 mm diameter plugs of agar were taken from the edge of each colony and placed, mycelium downwards, onto PDA containing a range of concentrations of prochloraz 0.0050, 0.0075, 0.010, 0.025, 0.050, 0.075, 0.10, 0.25 and 0.50 mg l^{-1}. Colony diameters were measured after a further 14 days growth at 20 °C and subjected to linear regression against log prochloraz concentration in order to calculate the IG_{50} value for each isolate.

The isolates were also characterised as either W or R types of the pathogen.

RESULTS AND DISCUSSION

The mean IG_{50} value for all of the 224 isolates tested was found to be 0.036 mg l^{-1} prochloraz (Fig. I). This is slightly lower than the 0.044 mg l^{-1} prochloraz obtained by Gallimore et al for 194 isolates, providing further evidence that increased use of the compound has caused no detectable decrease in sensitivity of eyespot populations from Western Europe.

Fig. I shows further that the distribution of IG_{50} values found here was much more compact than that obtained by Gallimore et al (1986), indicating lower levels of variability between the populations sampled during 1985.

FIGURE I

Distribution of sensitivity to prochloraz in 1985 P. herpotrichoides isolates

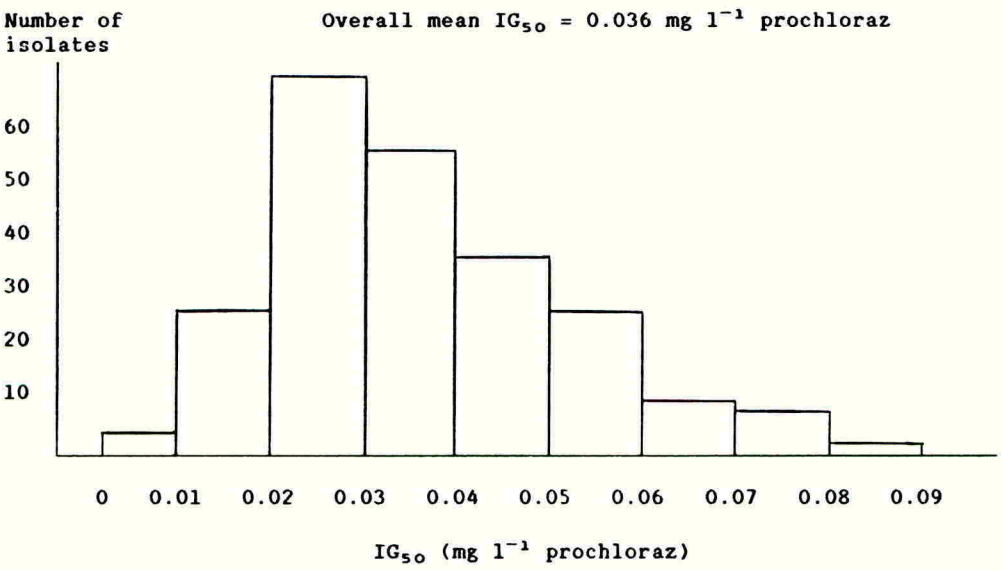

Table I shows the IG_{50} values of isolates derived from each of the five countries which contributed samples in 1986.

TABLE I

Country of origin	Number of isolates tested	Mean IG_{50} values (mg l^{-1} prochloraz)	99% confidence limits ±
France	60	0.034	0.005
UK	69	0.035	0.004
Germany	56	0.039	0.005
Denmark	24	0.042	0.010
Eire	15	0.025	0.005
Total	224		
Overall mean		0.036	0.003

Although the numbers of isolates from Eire were lower than those from the other countries, it is interesting to note that the mean IG_{50} value of these isolates (0.025 mg l^{-1} prochloraz) was markedly lower than the others. This difference was found to be significant at the $p = 0.01$ level when tested by analysis of variance and may reflect the fact that Eire was found to yield many more R than W types in 1985 (Russell and Birchmore, 1986). Gallimore *et al* reported that R types appeared to be more susceptible to prochloraz than W types and this is supported by our findings (Table II).

TABLE II

Country of origin	Mean IG_{50} values (mg l^{-1} prochloraz) W types	R types
France	0.038	0.030
UK	0.038	0.031
Germany	0.040	0.035
Denmark	0.050	0.031
Eire	0.031	0.024
Overall means	0.040**	0.031**

(** significantly different at $p = 0.01$)

R types were found to be consistently the more sensitive of the two types to prochloraz, with the overall means being significantly different. This finding may be important in disease control and the prospects for overcoming resistance since other workers have found that the increase in benzimidazole resistance has coincided with an increase in the proportion of R type isolates (Hollins et al, 1985). Therefore, it is suggested that the two compounds, with different modes of action, are selecting for different sections of the pathogen population. As these two fungicides are frequently used together, this may reduce the chances of resistance to prochloraz arising.

A final comparison which can be made is between the reaction of isolates to carbendazim and to prochloraz. Their ability to grow on agar containing 2.0 mg l^{-1} carbendazim was measured in the study reported by Russell & Birchmore (1986). Mean prochloraz IG_{50} values for isolates of P. herpotrichoides resistant or susceptible to carbendazim are compared in Table III.

TABLE III

Country of origin	Mean IG_{50} values (mg l^{-1} prochloraz) carbendazim resistant	carbendazim sensitive
France	0.033	0.038
UK	0.034	0.035
Germany	0.045	0.035
Denmark	0.040	0.044
Eire	0.026	0.024
Overall means	0.036	0.036

There were no consistent differences between the behaviour of carbendazim sensitive and resistant isolates from different countries.

In conclusion, there is no evidence to suggest that insensitivity to prochloraz is developing in the eyespot population.

Nevertheless, despite the current findings, Schering will continue to monitor eyespot populations in order to detect any possible population shifts in the future.

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SENSITIVITY TO FUNGICIDES OF BARLEY POWDERY MILDEW POPULATIONS IN ENGLAND AND SCOTLAND : STATUS AND IMPLICATIONS FOR FUNGICIDE USE

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ABSTRACT

The sensitivity of barley powdery mildew to triazole fungicides has declined in England over the period 1984 to 1986. The use of morpholine fungicides on cereals in England and Scotland has increased steadily over this period and ethirimol input has increased significantly as a result of the introduction in 1985 of a flutriafol/ethirimol mixture, 'Ferrax', for use on spring and winter barley. No significant response in ethirimol or morpholine sensitivity has been detected in barley mildew populations in England or Scotland.

INTRODUCTION

There are three distinct classes of systemic fungicide available to the farmer to control powdery mildew of barley. These are the C14-demethylation inhibitors (triazoles, imidazoles, pyrimidines and piperazines), the morpholines/piperidines, and the hydroxypyrimidine, ethirimol. In this study triadimenol was used as a representative member of the C14-demethylation inhibitors, fenpropimorph as the morpholine standard and ethirimol as the hydroxypyrimidine standard.

Over the period 1984 to 1986 the extent of use of these three groups has changed significantly. The most notable differences have been an increased tendency to use mixtures, particularly morpholine based mixtures, and the introduction of an ethirimol/flutriafol mixture as a seed treatment in spring 1985.

In order to assess the impact of these changes in chemical input upon population sensitivity, mildew populations were collected from 1984 to 1986 from the spring barley crop. Samples were taken each year in similar areas and at a similar stage in the growing season. Sensitivities were assayed for each of the three major groups of systemic fungicides and data between years compared, in an attempt to identify any trends in sensitivity patterns over this period.

MATERIALS AND METHODS

Collection of field populations

Mildew populations were taken from a representative distribution of spring barley fields in England and Scotland over the period 1984 to 1986. Populations were taken from farm fields, or large field plots, 2000m^2 to 5000m^2 in area. Almost all samples were taken between GS14-32, prior to the application of foliar sprays for mildew control.

The distribution of different mildewicide seed treatments in the sample varied from 1984 to 1986 (Table 1). These differences reflect changing chemical use patterns .

TABLE 1

Seed treatments used in the surveyed fields in England and Scotland:1984-86

Year/Country	Total no. fields sampled	No. of Fields Treated			
		Triazole[1]	Ethirimol[2]	Triazole[3]+ Ethirimol	Untreated[4]
1984 England	162	61	1	0	100
1984 Scotland	12	6	6	0	0
1985 England	77	23	0	23	31
1985 Scotland	14	0	0	14	0
1986 England	72	19	6	31	16

1 Triazole (triadimenol) seed treatment
2 Ethirimol seed treatment
3 Triazole (flutriafol) seed treatment in combination with ethirimol
4 Seed not treated with a triazole or ethirimol

No deliberate attempt was made to sample a fixed percentage of a specified seed treatment. Farmer contacts established in 1984 were maintained in 1985 and 1986. It was assumed that changing use patterns amongst these farmers would be representative of the national situation. In England, East Anglia was sampled more extensively than any other region.

Sampling procedures, disease assessment techniques and methods for laboratory culture of mildew populations have been described previously (Heaney et al. 1984).

Determination of sensitivity to fungicides

Mildew populations were subcultured on untreated barley leaves until sufficient inoculum was produced to assay sensitivity to triadimenol, ethirimol, and fenpropimorph. The majority of populations were subcultured less than three times before being tested.

All plant test material was grown in the following environment: day 20°C, 70% r.h.; night 15°C, 95% r.h.; day length 16 hour, light intensity 7500 lux.

Ethirimol

The detached leaf piece method described by Heaney et al. (1984) was used from 1984 to 1986. The range of ethirimol concentrations used was as follows: 0, 100, 200, 500, 2000, 8000ug a.i./g seed. Populations were graded between 0 (least sensitive) and 20 (most sensitive) based upon their response to the above concentrations.

Triadimenol

The detached leaf piece method was again employed. The lower range of triadimenol concentrations used was; 0, 2, 10, 30, 60 and 100ug a.i./g seed. The higher rate range, used from 1985 onwards, was; 0, 100, 150, 200, 300 and 375ug a.i./g seed. Populations were graded between 0 (least sensitive) and 20 (most sensitive).

Fenpropimorph

Two centimetre sections of 7 day old barley prophylls were floated on solutions of fenpropimorph or water, in Petri dishes. Dishes were 10cm square, 1.7cm deep and divided into 25, $2cm^2$ sections, in five rows of five sections. Two millilitre aliquots of water were placed in sections in the lower four rows. A 5ug/ml solution of fenpropimorph was placed in the top row sections immediately prior to inoculation. Dishes were inoculated using a small settling tower. Standard isolates were completely controlled on treated prophylls, and on 1 or 2 untreated rows nearest to the fenpropimorph treatment. This indicated that the dose-response seen within the test is a function of the vapour action of fenpropimorph. As in other tests, populations were graded between 0 (least sensitive) and 20 (most sensitive).

RESULTS

Changes in fungicide use : 1984 to 1986

Seed treatment

From 1984 to 1986 the major changes in the pattern of systemic fungicide use in England and Scotland was a decline of approximately 15% in the seed treated with a triazole, and in 1985 the introduction of a triazole (flutriafol)/ethirimol mixture which controls powdery mildew and a range of other important seed-borne and foliar pathogens in spring and winter barley. Taken over England and Scotland in 1985 and 1986 the proportion of barley seed treated with this mixture has increased to around 40% of the total seed receiving a triazole input (ICI data).

Accordingly, in England ethirimol input has risen from its previously negligible level in 1984, whereas in Scotland input has remained fairly constant over the period 1984-86. However, the use of triazole/ethirimol mixtures has increased at the expense of ethirimol only treatments in Scotland (ICI data).

Foliar application

Triazole based products continue to dominate the foliar spray market on wheat and barley in the UK. Triazole use has remained steady from 1983 to 1986. The use of morpholine-based mixtures has increased gradually over the last four years as the total use of fungicides has increased and the total market share of morpholine products has steadily risen. Foliar application of ethirimol in the UK has remained negligible. (ICI data).

Changes in fungicide sensitivity

Ethirimol

The mean of the ethirimol sensitivity distribution did not change significantly in England from 1984 to 1986 (Table 2). The distribution range was significantly narrower in 1986 than in previous years, with the least sensitive populations not being detected. No significant changes in sensitivity were demonstrated in Scotland between 1984 and 1985, though in both years sensitivity values were significantly lower than those in England (Table 2). No correlations were established between ethirimol sensitivity and triadimenol sensitivity.

Triazoles

Triazole sensitivity in England declined significantly in barley mildew populations between 1984 and 1985 (Table 3). Sensitivity distributions for these years differed in mean values and range. Highly sensitive populations detected at low frequency in 1984 were not found in 1985. Differences between 1985 and 1986 were not significant, although the sensitivity distribution for 1986 was narrower, with the most sensitive class of isolate (on the high rate test) not being detected. Differences in triazole sensitivity were apparent between England and Scotland in both 1984 and 1985. Populations were more sensitive in Scotland in both years (Table 3).

Morpholines

There were no significant changes in fenpropimorph sensitivity in England between 1985 and 1986 (Table 4). A standard isolate, JB6, isolated prior to 1980 was tested on 13 separate occasions. This isolate recorded a mean sensitivity value of 13.3±1.4, with a range of 12 to 16.7. Thus the variation measured in 1985 and 1986 was almost totally accounted for by experimental variation. Where test variation was exceeded, it was by a single population (sensitivity score 20) which was significantly more sensitive than the standard. There were no significant differences in sensitivity between England and Scotland, though the sensitivity range was narrower in Scotland (Table 4), probably reflecting smaller sample size.

TABLE 2

Ethirimol sensitivity changes in barley mildew 1984-86: England and Scotland

Country	Year	Sample size	Ethirimol Sensitivity		
			Mean sensitivity ± SD	Min. value	Max. value
England	1984	162	15.1±3.0b	6.4	19.6
	1985	77	14.7±2.9b	6.7	20.0
	1986	71	14.8±1.6b	10.9	18.7
Scotland	1984	12	12.6±2.9a	8.0	15.1
	1985	14	12.4±3.2a	6.7	17.4

Values with common letters in the same column are not significantly different at $P = 0.05$

TABLE 3

Triazole sensitivity changes in barley mildew 1984-86: England and Scotland

Country	Year	Sample size	Triazole Sensitivity					
			Low rate test[2]			High rate test[3]		
			Mean sensitivity ± SD	Min. value	Max. value	Mean sensitivity ±SD	Min. value	Max. value
England	1984	162	6.5±2.8b[1]	0.0	17.1	-	-	-
	1985	77	2.2±2.0a	0.0	10.4	9.3±5.1a	0.3	20.0
	1986	71	-	-	-	8.6±3.3a	2.1	15.1
Scotland	1984	12	7.8±1.9c	2.8	12.6	-	-	-
	1985	14	5.8±3.8bc	0.5	10.4	-	-	-

1 P = 0.5 (Students t-test)
2 Triadimenol rate range from 2 to 100ug a.i./g seed
3 Triadimenol rate range from 100 to 375ug a.i./g seed

TABLE 4

Fenpropimorph sensitivity changes in barley mildew 1984-86: England and Scotland

Country	Year	Sample size	Fenpropimorph Sensitivity		
			Mean sensitivity ± SD	Min. value	Max. value
England	1985	77	13.2±1.1a	11.1	17.6
	1986	71	13.6±1.7a	10.0	20.0
Scotland	1985	14	12.5±0.7a	11.0	13.9

Treatment related sensitivity changes

Populations isolated from fields treated with a flutriafol/ethirimol mixture had significantly lower ethirimol sensitivity values than those from untreated fields. Triazole sensitivity values were not significantly influenced by treatment, though values were lower in fields treated with a flutriafol/ethirimol mixture and slightly lower in triadimenol treated fields (Table 5).

Disease control

Triazoles still provided a degree of disease control of powdery mildew populations in 1985, as exemplified by the performance of triadimenol seed treatments at six large plot sites in East Anglia (Table 6).

Powdery mildew control was only apparent at earlier growth stages when disease pressure was relatively low. At later growth stages and when disease pressure was much greater the efficacy of flutriafol/ethirimol mixtures was clearly superior (Table 6).

TABLE 5

Effect of seed treatment on mildew sensitivity 1986: England

Seed Treatment	Mean sensitivity ± SD	
	Triazole	Ethirimol
Triadimenol	9.3±3.5(19[2])a	15.1±1.4(19)ab
Flutriafol + ethirimol	7.4±3.0(30)a	14.2±1.5(31)b
Untreated[1]	9.7±3.3(16)a	15.6±1.4(13)a

1 Not treated with triazole or ethirimol
2 Number of samples

TABLE 6

Control of powdery mildew with triazole seed treatments 1985: England

Seed treatment	% leaf area infected[1]	
	GS14-22	GS30-37
Triadimenol	0.3b	6.0a
Flutriafol + ethirimol	0.1b	1.2b
Untreated	1.5a	7.9a

1 Mean of 6 sites in East Anglia : 1985

DISCUSSION

Despite a marked reduction in the use of triazole seed treatments between 1984 and 1985, triazole sensitivity continued to decline, particularly in England. Triazole foliar sprays, however, continued to be used extensively and in the light of this the results are not unexpected. The broad spectrum action of the leading triazoles ensures that they will continue to be used by the farmer in spite of reduced efficacy against powdery mildew. Although the use of triazole-based mixtures is increasing,

reversal of the trend towards increased resistance to triazoles seems unlikely in the near future. Mixtures may, however, reduce the rate of decline in sensitivity, and it was noticeable that only small differences in sensitivity were apparent between 1985 and 1986.

Differences in triazole sensitivity between England and Scotland seen both in 1984 and 1985 may reflect the reduced frequency of use of triazole applications in Scotland with its lower winter barley acreage and more frequent application of morpholine sprays.

Ethirimol sensitivity levels in English barley powdery mildew populations have remained high over the period 1984 to 1986. Levels of sensitivity are significantly higher than when measured in the early to mid 1970's (mean sensitivity 13.6 compared to 11.9). The increased level of input of ethirimol in England as a result of the introduction of a flutriafol/ethirimol mixture onto spring and winter barley in 1985 has not produced a major population response to date, and the mixture has demonstrated excellent efficacy against powdery mildew. Small selective responses in ethirimol sensitivity can be detected when using the mixture and it will be necessary to closely monitor ethirimol sensitivity changes and flutriafol/ethirimol use patterns in order to maintain a successful balance between efficacy, sensitivity response, and input. Such selective responses are a function of disease control and can be reversed in the absence of selection pressure, as has previously been shown for ethirimol when used alone (Shephard et al. 1975).

It is not yet clear what relative selection pressures are imposed by the mixture as opposed to ethirimol when used alone. The fact that only small selective responses have been measured with the mixture should encourage continued application of this strategy.

Populations remain extremely sensitive to the morpholine/piperidine fungicides and little variation in sensitivity has been detected. Since the morpholines are thought to have a specific mode of action and have been used now since the late 1960's, this state of affairs is at first sight surprising. However, the compounds are not as widely used as the triazoles, nor are they utilised as seed treatments. Furthermore, where their application is becoming more prevalent it is in the form of mixtures. Finally, the morpholines are generally more ephemeral in nature than the triazoles. The combination of these factors may help to explain the current situation and, in the absence of dramatic increases in input, point towards a cautiously optimistic future for this group of fungicides.

An increase in ethirimol and morpholine input over the period 1984 to 1986 has not resulted in major changes in sensitivity to these two groups, and efficacy levels are high. However, the picture may alter as the patterns of chemical input change and as the pathogen population continues to respond to the imposed selection pressure. Under such circumstances accurate information on population sensitivity and chemical input will be essential to effective decision making on fungicide strategy.

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CONTRIBUTION OF FUNDAMENTAL RESEARCH TO COMBATING RESISTANCE

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ABSTRACT

Contributions from biochemistry, genetics and epidemiology have already added to knowledge of fungicide resistance. These findings are critically examined in the context of how they assist in developing effective ways to overcome resistance. Better disease management techniques, which reduce fungicide need and offer a promising way to minimize the impact of resistance, require more information about the biological factors governing spread of resistance. Present knowledge of biochemical mechanisms of resistance is limited but improvements, especially at the molecular level, could allow a more rational approach to synthesis of compounds which specifically interact with altered target sites. This approach requires a detailed understanding of negatively correlated cross-resistance, and offers prospects for the use of appropriate mixtures of site-specific fungicides which restrain selection for resistance to any of the mixture components.

INTRODUCTION

Fungicide resistance is an increasingly important problem for which few solutions are currently available. Considerable effort has been directed towards detecting the levels and extent of resistance, and on fundamental studies of factors which might govern spread within field populations. Often these studies have only examined resistance detected in laboratory studies, and attempts to translate results into effective strategies to combat the spread of resistance, and to evaluate these strategies in field situations have been very few indeed.

The aim of this paper is to assess the contribution made by fundamental studies to understanding the problem of fungicide resistance and to indicate where further work might be beneficial. Contributions from biochemistry, chemistry, genetics and epidemiology will be examined in detail, although other subjects have played a part in generating new ideas and concepts.

BIOCHEMISTRY AND CHEMISTRY

Current status

If resistance is to develop at all, potential biochemical mechanisms must be available which enable the organism to overcome the effects of a particular fungicide.The likelihood that resistance will develop seems to be determined by the chemical rather than the target organism. Discovery of new fungicides with novel modes of action is increasingly difficult and costly. Current strategies of fungicide use depend on suitable companion fungicides with multi-site action, which are generally used at high rates in mixtures. Under these conditions it is difficult for a new multi-site compound to compete with cheaper established chemicals, although environmental pressures may limit future use of existing multi-site inhibitors. The chemist is likely to concentrate on synthesis of

single-site inhibitors, but requires from biochemists and biologists information about the mechanism of resistance, and good predictive tests to establish the likelihood of resistance developing to new compounds. Yet in only a few instances are the mechanisms of fungicide resistance understood (Table 1), especially in field resistant isolates.

TABLE 1

Some mechanisms of fungicide resistance

Fungicide	Site of action	Mechanism of resistance	Comments	Refs
Carbendazim	β tubulin	Altered site action	Negatively correlated cross-resistance with N-phenylcarbamates	1 2
Fenarimol Imazalil	Sterol biosynthesis	Reduced uptake	Energy dependent efflux increased	3 4
IBP Edifenphos	Phospholipid biosynthesis	Metabolism/ detoxification	Cleavage S-C bond to more polar, less toxic derivatives	5
Fenprop-imorph	Sterol biosynthesis	Utilization of intermediates	Unusual sterols accumulate and are used in membranes	6

References: 1 = Davidse & De Waard (1984); 2 = Kato et al. (1984); 3 = De Waard & van Nistelrooy (1980); 4 = Siegel & Solel (1981); 5 = Uesugi & Katagiri (1983); 6 = Leroux & Gredt (1983).

Although one resistance mechanism may have priority, others contribute but are simply not researched because their effects are small. Cross-resistance patterns generally correlate well with mode of action, and biochemical knowledge of the site of action has helped to evaluate the "resistance risk" of candidate fungicides, and to develop suitable fungicide mixtures to combat the spread of resistance. It is suprising that the interaction of such a diverse group of chemicals as C-14 demethylase inhibitors of sterol biosynthesis, with some component of the ATP-dependent proton pump involved in the resistance mechanism, and with the totally separate site of action involving the haeme moiety of cytochrome P450, should correlate so well. However, it is difficult to separate the effects on efflux from altered binding to perhaps membrane components. The lipophilic nature of C-14 demethylase inhibitors suggests that fenarimol and imazalil would bind tightly to the active site as they pass through the membrane, and would not readily be removed from cells they had entered. More detailed biochemical information is clearly needed to elucidate fully this mechanism of resistance to an important group of fungicides; measurement of pH gradients across the membrane might help to explain these effects on efflux.

Successful binding to a target site depends on correct molecular shape and little deviation from the optimum configuration may be tolerated. Amino-acid changes may alter protein conformation around the binding site, and by excluding a fungicide, cause resistance, yet allow a previously excluded analogue with a different structure to bind. Such changes can provide a basis for negatively correlated cross-resistance (De Waard 1984), which offers a potentially powerful tool to combat resistance. Alteration of the succinic dehydrogenase complex in the mitochondrial transport chain seems one mechanism of resistance to carboxin in Ustilago maydis, and some carboxamide analogues were more inhibitory to this altered complex than to wild-type succinic dehydrogenase (White & Thorn 1980). Unfortunately these effects could only be demonstrated in laboratory tests, and the analogues were not sufficiently systemic to be of practical value.

Negatively correlated cross-resistance was also observed between carbendazim and related N-phenylcarbamates for four diseases (Kato et al. 1984), although this may involve binding to α tubulin rather than to the β subunit. This prompted a search for other phenylcarbamates which enhanced these effects, especially against Botrytis cinerea. Only highly carbendazim resistant isolates of Venturia nashicola were sensitive to phenylcarbamates (Ishii et al. 1985), and isolates showing intermediate levels of carbendazim resistance, which may be of practical significance, were not controlled by the phenylcarbamate, S32165. But in eyespot (Pseudocercosporella herpotrichoides), field isolates have been obtained that were highly resistant to both carbendazim and S32165 (Hollomon, unpublished observation). Furthermore, some laboratory mutants of P. herpotrichoides resistant to both these fungicides are sensitive to a second phenylcarbamate, MDPC (Hocart 1986, personal communication). This suggests that the tubulin binding site may differentiate between quite modest changes in molecular configuration and specific carbendazim-phenylcarbamate mixtures may be needed against different diseases. The structural gene for β tubulin in Aspergillus nidulans has recently been cloned (Peberdy, personal communication). Site directed mutagenisis coupled to DNA sequence analysis should provide an amino acid map of the carbendazim binding site, and allow prediction of carbamate structures which might bind more effectively to β tubulin from resistant isolates. Certainly, the biochemical concepts behind negatively correlated cross-resistance offer chemists scope for subtle changes in molecular structure. Many fungicides have asymmetric carbon centres and allow synthesis of isomers which may be active against altered resistance mechanisms (Berg et al. 1987).

Where resistance arises through detoxification or by increased efflux, inhibition of these processes may increase fungicide activity against otherwise resistant organisms. The contribution synergism can make towards combating resistance has been explored in several systems (De Waard 1985), but an effective synergist must have similar properties of systemicity and persistence and be capable of formulation with "at risk" fungicides. These conditions are difficult to fulfill and, consequently, the search for synergists is not appealing for chemists.

Future research

Resistance may emerge gradually through modification of the regulation and specificity of enzymes which normally participate in other

aspects of metabolism. These changes alter the original enzyme function and place mutants at a disadvantage relative to wild-types. Further selection improves effectiveness and specificity, and at the same time separates regulation of the new enzyme function from the control of the old pathway, lessening pleiotropic effects on fitness. Gradual increases in resistance may also reflect gene duplications, which enable organisms to combat foreign chemicals through increased enzyme levels. Biochemical analysis at the enzyme level during evolution of fungicide resistance, might contribute to combating resistance by suggesting ways of retarding development of effective resistance mechanisms. A suitable laboratory system for such analyses might involve yeast, where selection for fungicide resistance can be manipulated in chemostat cultures (Paquin & Adams 1983).

Evolution of fungicide resistance involves changes in gene frequencies within natural populations. Bioassay procedures measure phenotypes, although where qualitative differences unambiguously identify resistant forms, these are often equated with their respective genotypes. Where differences are quantitative, bioassays only provide a measure of fungicide resistance (ED50) for the whole population. Biochemistry offers ways to monitor resistance at the protein level through immunological techniques, or at the DNA level by hybridization. A rapid immunological technique has been developed to monitor insecticide resistance in aphids (Devonshire 1987), but fungicide resistance mechanisms are less well understood and comparable techniques not so advanced. However, knowledge of β tubulin coupled with cloning the benA gene suggest similar techniques for monitoring carbendazim resistance in a wide range of pathogens might soon be possible. These biochemical monitoring techniques would only be possible where resistance mechanisms were understood at the molecular level, but they are likely to be simple, rapid and more sensitive than bioassay. They will be useful, therefore, as diagnostic tools to measure resistance levels where choice of a suitable control strategy by a farmer depends on knowing the level of resistance. Changes in resistance could also be detected at much lower gene frequencies, which would be invaluable when evaluating the effects of various fungicide strategies on the critical early stages of the evolution of resistance.

GENETICS

Many papers report the phenotypic characters of fungicide resistant phytopathogenic fungi, but genotypes of the same isolates are seldom analysed. Handling difficulties, lack of methods to generate recombinant progeny and isolate instability have limited genetic work to 18 plant pathogenic fungi. Consequently, much genetic information on fungicide resistance has been generated through work with Neurospora crassa, A. nidulans and Saccharomyces cerevisiae, but the genetic systems controlling resistance in these fungi seem to apply equally to plant pathogens (Grindle 1987). Resistance is generally controlled by nuclear genes, although cytoplasmic (mitochondrial) genes can be involved in antibiotic resistance. Up to 10 major genes may control resistance to some fungicides (van Tuyl 1977) and each gene may have several alleles. Occasionally, action of each gene may be influenced by unrelated modifier genes or may itself exert effects on other genes. Non-allelic interactions make it difficult to predict resistance levels, and a double mutant may be

no more resistant than one of its mutant parents. Genetic control of resistance can also be exercised through minor genes (polygenes) whose effects are additive but too small to identify individually. In field populations, especially of powdery mildews, polygenically controlled resistance results in a shift from the broad unimodal distribution of wild-type populations towards less sensitive forms. This quantitative response contrasts with the more rapid qualitative one associated with major gene resistance (Georgopoulos 1986).

Several mathematical models have been developed which describe the development of qualitative resistance assuming one locus-two allele genetics. Understanding of the dynamics of quantitative resistance is very limited, and no models explaining evolution of polygenically controlled fungicide resistance exist. A start has been made to address this important challenge (Skylakakis & Hollomon 1987), but substantial genetic information will be required if a meaningful model is to emerge. Estimates from recombinant data of both heritability and the "selection differential" (Via 1986) will be needed, and as resistance evolves, fresh estimates of these parameters will probably be required since recombination and selection may change the expression of resistance.

The selection differential measures the genetic variation in fungicide sensitivity upon which selection can act, and it has been used to predict possible development of resistance to new fungicides. Measurement of the extent of this variation has either involved monitoring base-line sensitivity in untreated populations, or mutation studies since most fungi are haploid and mutants are immediately expressed. The frequency of resistant mutants may vary considerably. In *N. crassa* mutants resistant to captan or chlorothalonil were found in 10^{12} conidia, whereas 2 in 10^{9} conidia were resistant to quintozene and vinclozolin (Grindle 1987). Differences in mutation rates may reflect differences in the number of genes that can mutate to resistance, but not all gene loci are equally mutable and various environmental factors and the choice of mutagen can influence the selection of mutants. Pleiotropic effects of mutant genes are crucial, especially where these affect fitness and competitive ability, and emphasise the need for caution in interpreting behaviour of field populations from data obtained from laboratory mutants. In powdery mildews, where populations are large and difficult to monitor, and mutation studies are difficult, recombination may be the most efficient way of determining the selection differential, especially if quantitative resistance is likely. Recombination between isolates from the mid-point of the wild-type population distribution should contain the maximum genetic variation available, and progeny from a number of appropriate crosses should reveal the extent of this possible variation. Crosses between wild-type isolates of *Erysiphe graminis* f.sp. *hordei* have produced ascospore progeny significantly less sensitive to ethirimol and triadimenol than either parent (Hollomon 1981; Hollomon *et al.* 1984), and outside the range that would have readily been identified through monitoring.

Genetic studies have also provided evidence of dominance arising through interaction of a resistance gene with its wild-type allele. Although many important plant pathogens are haploid, dominance can be of practical significance where heterokaryons and dikaryons exist, since

dominance may ensure that all cells are resistant even though varying proportions of wild-type sensitive nuclei may be present.

EPIDEMIOLOGY

Fungicide resistant individuals will multiply faster in the presence of fungicides than those lacking resistance. It is the need to understand the fundamental factors governing the selection of differences between resistant and sensitive forms that links fungicide resistance and epidemiology. Information on how fungicide exposure interacts with the biology of the disease and cropping practices should provide the basis for practical stategies of fungicide use. So far, the contribution of epidemiology has tended to emphasise mathematical models which might describe the evolution of fungicide resistance, and there have been few attempts to validate models, or their predictions, in field experiments. Nevertheless, models have had a profound influence on the practical advice offered to farmers.

Initial models were simple, took little account of epidemiological factors, and employed poorly defined variables which were difficult to measure (Skylakakis 1982). Following development of theories linking relative fitness to apparent infection rate (MacKenzie 1978; Barrett 1983), a model was developed (Skylakakis 1982) which related measurement of infection rate to the time required for the resistant subpopulation to increase by 2.7 times. This "standard selection time" was used to assess the implications of simultaneous or alternate applications of systemic and protectant fungicides on the build-up of the resistant subpopulation. This model applied only to the exponential phase of disease spread when no competition occurred between or within resistant and sensitive subpopulations for disease free sites. It also assumed that the effects of systemic and protectant fungicides were additive. More realistic assumptions were incorporated into a subsequent model (Levy et al. 1983), which extended consideration to the asymptotic phase of epidemics. Despite varying assumptions, the conclusions drawn from all these models are broadly similar. Resistance spreads rapidly where large, fast-developing, pathogen populations are exposed to persistent and effective chemicals. Fungicide mixtures are likely to be more effective than alternating sequences, especially where spray coverage is poor. However, only fragmentary experimental evidence exists to support these conclusions. A reasonable correlation exists, for three diseases at least, between the time it actually took for fungicide resistance to emerge as a practical problem, and the time predicted by the models (Table 2). In certain instances, fungicide mixtures have been shown to delay the spread of resistance (Staub & Sozzi 1983; Sanders et al. 1985). However, it remains a difficult practical problem to design and monitor field experiments to assess relative fitness through measurement of population size or apparent infection rate.

No models have yet been developed which describe the evolution of quantitative resistance where two subpopulations cannot be identified because classes overlap. Only additive genetic variation (Va) contributes to the evolution of polygenically controlled fungicide resistance and it is necessary to determine what fraction of the total phenotypic variation (Vp) is additive. The ratio Va:Vp measures heritability and the likelihood that a particular level of resistance will be passed on to the

next generation. Heritability is derived from examination of variation between individuals and within daughter clones generated from them. It is likely to be lower in sexual systems than where only asexual reproduction occurs, since epistatic effects associated with resistance, and released through recombination, reduce the value of Va.

TABLE 2

Predicted and observed time for resistant subpopulations of three pathogens to cause resistance outbreaks in the field

Pathogen	Chemical	Standard selection time in days	Selection-pressure duration in time units as indicated (d:days; y:years) Predicted	Observed
Phytophthora infestans	Metalaxyl	3.7-3.8	51-70d	1-2 seasons
Cercospora beticola	Benomyl	9.5-14.3	130-263d	140-200d
Ustilago nuda	Carboxin	158.5	5-7y	12-14y

Data from Skylakakis (1982) and Leroux (1986)

The response of a population ED50 value to selection is the product of heritabilty and the intensity of selection. Changes in ED50 per generation can be given mathematically as: $ED50 = \left(\frac{Va}{Vp}\right)s$

where s is the difference in fungicide sensitivity before and after selection (selection differential). Estimation of s has already been discussed but the genetic parameters may change during evolution of resistance and need re-estimation at the start of each epidemic. Applying this model to the limited data available for evolution of resistance to 2-aminopyrimidines and sterol biosynthesis inhibitors in powdery mildews, shows that resistance has developed considerably more slowly than the model predicts (Table 3, Skylakakis & Hollomon 1987). At present, the model incorporates only genetic data and assumes that apparent infection rate will be constant throughout the range of ED50 values, even though stabilising selection seems to be an important feature of how mildews respond to fungicides. It also assumes that no ingress of sensitive individuals occurs from outside the treated area and that selection pressure is constant throughout. None of these assumptions is realistic and research is urgently needed to explore ways of measuring these parameters in epidemiological terms, and incorporating them into a more robust model. Field experiments with barley powdery mildew have already shown that fungicide mixtures can delay, or even reverse, the spread of quantitative resistance (Hollomon & Brent 1986) and it will be interesting to see if an epidemiologically based model supports these findings.

TABLE 3

Predicted and observed consequences of fungicide selection in powdery mildews using the model of Via (1986)

Powdery Mildew	Fungicide	Heritabilty	Selection Differential given as ED50 in ppm	Time for population to reach upper ED50 value of Selection Differential Predicted (gener-ations)	Observed (years)
Erysiphe graminis f.sp. hordei	Ethirimol	0.60	0.02-0.25	2-3	3
	Triadimenol	0.62	0.001-0.43	3	2
Sphaerotheca fuliginea	Dimethirimol	0.60	0.05- 0.3	2	1
	Imazalil	0.62	0.0003-1.6	2-3	2-3

Table from Skylakakis and Hollomon (1987). Heritability values for S. fuliginea are assumed to be the same as those for E. graminis.

Epidemiology can also make significant contributions towards delaying resistance by defining more clearly the economic benefits of fungicide treatments. Better disease forecasting, more accurate disease assessments and establishing valid threshold levels of disease which cause yield losses, should aid in reducing fungicide use. Where integration with other control measures is possible, this offers the best way available at present to minimize the risk of resistance.

The response of a pathogen to a fungicide may depend on its breeding system. Where reproduction is asexual, linkage disequilibrium between fungicide resistance and other characters are likely to persist and possibly delay evolution of resistance. Linkage between certain virulence characters and fungicide sensitivity have been demonstrated for barley powdery mildew, but the linkage effects have been insufficient to exploit in the form of strategies combining fungicide and cultivar use. In lettuce downy mildew (Bremia lactucae) metalaxyl resistance is associated with the same sexual compatability type, and incompatability on lettuce cultivars with the R-11 resistance gene. Since this gene is present in several widely grown lettuce cultivars, a strategy for delaying resistance has been adopted which uses metalaxyl only on R-11 cultivars (Crute 1984). It will be interesting to see how successful this strategy is in practice since metalaxyl resistance has been detected in sexual progeny able to infect R-11 cultivars.

CONCLUSIONS

No easy solution to the fungicide resistance problem is in sight, but accumulation of fundamental data about resistance suggests two approaches that might succeed. Diseases should be managed so that selection for resistance is minimized. Fungicide use should not only be

integrated with cultivar resistance, but where possible reduced through better disease forecasting, and assessments of disease thresholds likely to cause yield loss or quality deterioration. To do this will require more detailed information on the biology, genetics and population dynamics of resistance. Of particular importance is the development of suitable models which describe the evolution of quantitative fungicide resistance. Above all, models and strategies of fungicide use suggested by them must be validated through specifically designed field experiments.

Chemical synthesis offers an alternative approach. Although some useful novel fungicides may be found by current screening methods, rationally directed synthesis will require considerably more information about the biochemical mechanisms of fungicide resistance. This should allow synthesis of analogues that can be effective in mixtures with the original fungicide. What the chemist must have from from biologists is reliable models and tests which can predict the likely development of resistance to new compounds.

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