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OILSEED RAPE DISEASES

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by

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ABSTRACT

The review considers the diseases of oilseed rape in the context UK Agriculture. The recent and relevant literature, together with the authors experienced observations and results of discussions with plant pathologists involved in research and extension, are reviewed and discussed.

Three principle diseases have been identified as posing the main threat to the UK crop, namely light leaf spot (Pyrenopeziza brassicae), dark leaf and pod spot (Alternaria brassicae and A. brassicicola) and canker (Leptosphaeria maculans). Diseases which are common, but are considered to be less damaging are beet western yellows virus (BWYV), sclerotinia stem rot (Sclerotinia sclerotiorum) and grey mould (Botrytis cinerea). Other diseases, cauliflower mosaic virus (CaMV), turnip mosaic virus (TuMV), club root, (Plasmodiophora brassicae), damping-off caused by Pythium spp. and Rhizoctonia solani, phytophthora root rot (Phytophthora megasperma), powdery mildew (Erysiphe cruciferarum), downy mildew (Peronospora parasitica), white leaf spot (Pseudocercosporella capsellae) and ringspot (Mycosphaerella brassicicola) are considered to be of

relatively minor importance nationally. Little published data exists on some of these diseases and they are reviewed in less detail. The existence of white blister (Albugo candida) and verticillium wilt (Verticillium dahliae) has not yet been confirmed in the UK, but because of their potential for affecting yield of oilseed rape, they are covered in this Review.

The data reviewed show that disease incidence and yield response generally decline as crops are grown further north in England, but increase again in Scotland. The best estimates of yield losses from any disease comes from extrapolation from early work on dark leaf and pod spot, where for every one per cent disease on the pods it was calculated that there was a one per cent loss in yield. When this formula was applied to disease severity as recorded in the ADAS disease surveys for England and Wales for the years 1986-1990, the annual estimated loss due to disease was about five per cent. For an average crop yield of 3.05 t/ha at £240/t for double low cultivars this would be equivalent to £36/ha. National losses due to disease would be in the order of £12 M per year.

In calculating the economics of disease control by using fungicides, it was estimated that the cost of a single fungicide application was equivalent to 0.1 t/ha. For a two-spray programme, including an application following the onset of flowering, 0.3 t/ha would be required, as it is necessary to take into account wheeling losses from the later application. For the average crop this is equivalent to a yield response of nine per cent (current proposals by the European Commission to alter the support system for oilseed rape could radically alter the economics of fungicide treatment for disease control).

Light leaf spot is now considered to be the main fungal disease of oilseed rape in the UK. Disease/yield loss relationships are uncertain, but control can be achieved by fungicides applied in the autumn and early spring. There is resistance to the disease in many current cultivars but, when disease pressures are high, reliance has to be placed on fungicides for control.

Leptosphaeria maculans, the cause of leaf spot and canker, exists in two distinct strains. Systemic infection by the fungus has been identified as a possible problem for disease control with the available protectant fungicides. Sources of genetic resistance are available, but current cultivars generally have low levels of resistance and are potentially at risk.

Alternaria spp. infecting oilseed rape are of worldwide distribution and dark leaf and pod spot is of importance wherever oilseed rape is grown. Yield losses due to the disease can be high and the lack of genetic resistance in all current commercially available cultivars make the disease a potential problem in the UK. Control of the disease is heavily dependent on fungicides. There is a major requirement to develop an effective forecasting system to predict high risk conditions which lead to epidemics of the disease.

Sclerotinia stem rot is widely recognised as an important disease, yet evidence that it causes major losses is limited. It is only of consequence in the UK in about one year in ten and the major outbreaks occur in a limited geographical area in Sussex and Kent. 1991 was considered to be a bad year for sclerotinia. It is possible that outbreaks may be more frequent in future as inoculum levels increase. Experimental evidence suggests that for control measures to be economic a disease incidence of more than 25 per cent of plants infected is required. There are currently no cultivars resistant to the disease and control is achieved by fungicides applied at a time which coincides with ascospore release during the flowering period. A number of forecasting schemes have been devised, including monitoring of depots of buried sclerotia, to predict high risk conditions. These schemes met with limited success under UK conditions, but there is further scope for their evaluation.

There is little published data on grey mould on oilseed rape. The fungus is considered to be an opportunist, colonising senescing tissues or tissues that have been damaged by weather, pest, the passage of machinery or fertiliser scorch. The disease can be damaging under wet conditions. Although stem infections can create the appearance of widespread infection

in crops as plants ripen, specific control measures are not considered to be economic for this disease.

Beet western yellows virus has been found to be ubiquitous and, surprisingly, more common in the north of England in recent years. The disease is almost symptomless and yield loss has been difficult to evaluate. As the rape host constitutes a major reservoir of the virus the threat from the virus to other crops, such as sugar beet and lettuce, requires evaluation. Of recent concern is the dramatic increase in the recorded incidence of the cauliflower and turnip mosaic viruses, recorded on oilseed rape in some parts of the country for the first time in 1991. These two viruses can be severely damaging and their future development in oilseed rape will require close monitoring.

Of the other diseases covered by the Review, the relatively few serious outbreaks of club root are surprising considering the susceptibility of oilseed rape to the disease. Downy mildew, though widespread and causing leaf loss in mild wet autumns and early springs, is not considered to be a major cause of either crop failure or yield loss. Premature senescence of plants within crops has caused concern since the early 1980s. Rhizoctonia has been implicated, but virtually no research into this problem has been undertaken in the UK. Powdery mildew was common in the dry year of 1990, but has not been a problem in 1991, and is likely to remain a sporadic disease. White leaf spot and ring spot are primarily confined to the South West of England and Wales and no general outbreaks have been reported.

The effects of disease on oil quality and glucosinolate content have been evaluated. There was some evidence that control of disease by fungicides can improve oil content and reduce amounts of glucosinolates. However, it was sometimes difficult to separate the effects of the fungicide treatment on diseases from those on crop maturity which also has an influence on glucosinolate content.

Six areas are identified for further research and development: surveys, disease/yield loss relationships, epidemiology, forecasting and thresholds, fungicide spray timing and comparisons and host genetic

resistance. The major area identified for immediate work is that of establishing diseases/yield loss relationships for the main pathogens and the forecasting of disease risk.

The Review includes 250 key references covering all the major aspects of the symptoms, aetiology, epidemiology and control of the diseases of oilseed rape.

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GLOSSARY

abscission	process by which a leaf separates from the stem.
acervulus	cushion-like mass of hyphae producing conidia.
agar	jelly-like medium for culturing fungi and bacteria.
anamorph	asexual spore form of a fungus (imperfect state).
antheridium	a male fungal cell in sexual reproduction.
anthesis	production of pollen during flowering.
apothecium	cup-shaped fungal fruiting body.
appressorium	specialised hyphal cell formed for attachment prior to cell penetration.
ascospore	a sexual spore produced from a flask-shaped receptacle.
ascogonia	fungal reproductive cell of a fungus.
avirulent	strain of a pathogen that does not cause severe disease (also non-virulent).
biotroph	organism only capable of living as a parasite.
canola	Canadian name for oilseed rape.
canker	a sunken lesion, usually found on the stem.
chlamydospore	an asexual thick-walled fungal spore arising from a hypha.
cleistothecium	a fungal fruiting body of the powdery mildews

conidiomata	a fungal structure bearing conidia.
conidium	asexual fungal spore.
cotyledon	leaf forming part of the seed.
cryptic	not producing symptoms.
cultivar	cultivated variety.
cuticle	a skin covering the epidermis.
dithiocarbamate	a group of protectant fungicides with a broad spectrum of activity, eg maneb, mancozeb and zineb.
DMI	a group of fungicides which inhibit the demethylation process in the production of ergosterol, eg propiconazole.
double-low	low in both erucic acid and glucosinolates.
epidemic	a widespread increase in the incidence of a disease.
epidermis	the outer layer of cells covering the leaf and stem.
erucic acid	long-chained fatty acid. Rapeseed oil high in erucic acid is valued as an industrial lubricant.
facultative	living on dead tissue.
foci	discrete patches of disease in a crop.
foot rot	a disease of the stem base.
fungicide resistance	resistance of a fungus to a fungicide.

glucosinolates	a group of sulphur compounds, the breakdown products of which are anti-nutritional.
hypha	thread of a fungus.
hypocotyl	part of a plant between the root and cotyledons.
hypomycelium	mycelium formed within the plant, usually beneath the cuticle.
imperfect state	asexual spore form of fungus (anamorph).
imidazole	group of fungicides including prochloraz.
inoculum	infective material, eg spores or mycelium.
latent	an infection for which symptoms are not obvious at the time of inspection but may show later (cf cryptic).
lesion	dead or damaged area of tissue.
MBC	methyl benzimidazole carbamate - a group of fungicides including benomyl, carbendazim and thiophanate-methyl.
mesophyll	plant cells in a leaf lying between the epidermis.
monocyclic	a disease that arises from inoculum that does not increase in the crop during the growing season.
mycelium	group of hyphal threads.
non-persistent	of viruses - of limited life in the vector.
oogonium	a female sex organ.
oospores	resting spores.

paraphysis	a sterile cell arising from a spore bearing area.
pathogen	a living agent of disease.
pathogenicity	the ability to cause disease.
perfect state	sexual spore form of fungus (teleomorph).
persistent	of viruses - may remain active in the vector for many weeks.
petiole	part of the leaf between the stem and the blade.
phenotype	form of a plant shaped by the environment.
phenylamide	a group of systemic fungicides active against phycomycete fungi, eg metalaxyl.
phyllosphere	the zone around the aerial parts of a plant.
phytoalexin	a chemical produced by a plant in response to fungal infection.
polycyclic	a disease that produces fresh inoculum to infect other plants in a single season.
pseudothecium	a flask-shaped receptacle containing a mass of hyphae producing asci.
pycnidium	a flask shaped receptacle which produces asexual spores.
resistant	the ability of plant, usually under genetic control, to successfully resist infection.
rhizosphere	soil around the roots of a plant.

saprophyte	a fungus living on dead organic matter.
sclerotia	fungal resting bodies.
single low	a cultivar low in erucic acid.
sirodesmins	non-host specific phytoalexins.
spermagonia	a flask-shaped fungal structure producing spores (spermatic).
spermatia	a form of asexual spore.
strain	variant of a pathogen.
stroma	a mass of vegetative hyphae.
systemic	existing within the plant tissues.
teleomorph	sexual spore form of fungus (perfect state).
triazole	a group of systemic fungicides including flutriafol and propiconazole.
trichogyne	female hyphal organ.
umbelliferae	a class of plants including cow parsley (Anthriscus sylvestris L.).
vector	biological agent which transmits a disease eg nematodes and aphids.
virulent	active/aggressive.

INTRODUCTION

The area sown to oilseed rape in the UK has increased rapidly from the early 1970's. Provisional figures from MAFF, DANI and SOAFD indicate that the area sown in 1991 was 443,000 ha. The area grown in the various ADAS Regions, Scotland and Northern Ireland in 1990 (data from the June Census) were as follows:-

	Area (ha)	Percentage of total
Eastern	138,891	36
Northern	74,267	19
M & W	57,951	15
South East	53,409	14
South West	17,934	5
Wales	734	>1
Scotland	45,177	12
Northern Ireland	1,223	>1

The crop suffers from a number of important diseases which can be detrimental to yield. The incidence and severity of the major diseases of oilseed rape varies from season to season and from area to area. Routine application of fungicides against those diseases for which fungicides provide an adequate method of control is not justified (Hardwick and Evans, 1989).

There have been two recent publications detailing the symptoms and control of diseases of oilseed rape (Davies, 1986a; Hawkins, 1985). The purpose of this Review is to describe the main diseases of oilseed rape, to assess the current state of knowledge about the diseases in the UK and to make recommendations where further work is required.

The Review is limited mainly to information from the UK, although data from other countries have been included where relevant. Information for this Review has been obtained from publications (see References section)

and from conversations and correspondence with scientists in the UK and overseas (see Acknowledgements). We have concentrated on data published in the most recent 10 year period, though where earlier work has been judged as still relevant this has been quoted.

We have identified light leaf spot (Pyrenopeziza brassicae), canker (Leptosphaeria malculans) and dark leaf and pod spot (Alternaria brassicae) as being the three most important diseases of oilseed rape in the UK. These are discussed in detail. We have classed the remainder into other important diseases and minor diseases. Data for some of the minor diseases are limited and mainly anecdotal.

We begin the Review with information from the ADAS disease survey, supplemented with data from Northern Ireland and Scotland, in order to provide an overview of the relative incidence and severity of the common diseases of oilseed rape.

Disease incidence and severity

Crops of oilseed rape have been monitored by ADAS on an independent regional basis from the early 1970's (Cook & Evans, 1978). From 1983, all Regions became involved in a selected survey (Evans et al., 1984) and from 1986 this monitoring exercise was conducted along more formal lines (Hardwick et al., 1989). Standard methods of assessment were used, questionnaires were completed to supplement disease data by producing information on husbandry interactions and pesticide usage. The number of crops sampled was generally in proportion to the national area grown based on a total sample size of about 110 crops. Data from Northern Ireland and Scotland are limited.

Details of disease incidence and severity are presented in the Appendix (Tables 1-9). The incidence of disease was generally high, ie most crops carried some disease. However, generally the severity of disease was low.

One feature of the 1990 season which was different from previous seasons was the high incidence and severity of powdery mildew (Tables 3 & 6). In some crops, the whole plant was covered by a grey bloom. The disease

appeared in crops late in the season and anecdotal evidence from farmers who combined such crops was that yield effects were marginal but the dust hazard was considerable.

Beet western yellows virus (BWYV) in 1990 reached an incidence of 98 per cent crops affected with a mean level per crop of 73 per cent plants infected. This makes this disease the most widespread and, in terms of plants infected, common disease of oilseed rape (Table 7).

The variation in disease incidence and severity between Regions has been difficult to determine with great precision as few samples were taken in some areas. However, white leaf spot is a disease mainly confined to the South East, South West and Wales, ringspot to the South West and Wales and sclerotinia is a problem in the counties of Kent and Sussex, although the disease may be occasionally severe in crops throughout England and Wales.

Disease incidence and yield response generally decline as crops are grown further north in England (Hardwick & Evans, 1988; Hardwick et al., 1989; Hardwick, 1990) (Fig. 1) but increases again in Scotland (S J Wale, unpublished).

Yield/disease loss relationships

The best estimates of yield losses from any disease comes from extrapolation from early work on Alternaria where for every one per cent disease on the pods at pod ripening there is a one per cent loss in yield (Gladders, 1988; Hardwick & Evans, 1988). Applying this formula to disease severity for the years 1986-90 the annual estimated losses would be about five per cent for all diseases. For an average crop yield of 3.05 t/ha at £240/t for double low cultivars (Nix, 1989) this would be equivalent to £36/ha. National losses due to disease would therefore be in the order of £12 M per year. This is likely to be an underestimate, as it does not take account of losses due to leaf and stem diseases.

Economics of fungicide application

As a rough guide to the economics of disease control by fungicides, it is estimated that the cost of a single fungicide application is equivalent to a yield response of 0.1 t/ha. For a two-spray programme, including an application following the onset of flowering, a yield response of 0.3 t/ha would be required. The additional yield response is required as it is necessary to take in to account wheeling losses from the later spray application (Ogilvy, 1989). Wheeling losses, post flowering, were reported as 3.4 per cent with a tractor and trailed sprayer and 2.8 per cent for a high clearance self propelled sprayer, based on a 24 m boom (Table 10). A maximum yield loss of 4.8 per cent was recorded in tall crops (1.4 m).

Currently a two-spray programme, which includes a spray applied from the onset of flowering, will require a response of nine per cent. There is a proposal by the European Commission to alter the support system for oilseed rape. As currently envisaged this could reduce the price received by the farmer for rape seed to the world price, estimated to be £130/t, with an additional payment based on the crop area sown paid directly to the producer. This could radically alter the economics of fungicide treatment for disease control on oilseed rape.

Spring sprays with prochloraz (Sportak), MBC or a mixture of iprodione plus thiophanate-methyl (Compass) at early stem extension (March/April) have been evaluated by ADAS Plant Pathologists in nationally co-ordinated trials since 1983. Table 11 gives the mean yield responses of these treatments for the last five years compared with a single spray of Rovral at the end of flowering. Sprays at the end of flowering, as noted above, will be more costly due to the losses caused by wheelings. Sportak applied at stem extension has been the most profitable single treatment in the trial series. In low disease years (1989-1990), there has been little or no economic benefit in applying any spray. There has been a financial loss where Sportak, Compass or Rovral have been used.

Fungicides

Two key timings have been identified for disease control in crops growing in England and Wales - stem extension, aimed primarily at light leaf spot control, and the flowering/pod formation period for sclerotinia and alternaria control (Figs. 2 & 3). In Scotland, fungicide application during the late autumn, aimed at light leaf spot control, has become an essential part of their disease control programme.

In its crop surveys, ADAS has obtained data on fungicide usage since 1986. The proportion of crops receiving stem extension sprays have increased from 17 per cent of crops in 1986 to 52 per cent in 1990. Whilst the proportion receiving flowering sprays has fallen from 41 per cent to 14 per cent, the overall proportion of crops receiving at least one spray has remained fairly constant at around 50 per cent. Sportak and Rovral have established themselves as the principal products for disease control in oilseed rape. In 1990, 65 per cent of spray applications in the spring were with Sportak and 50 per cent of applications in the summer were with Rovral. There has been a rapid decline in aerial applications, from 38 per cent in 1986 to 2 per cent in 1990.

DISEASES OF MAJOR IMPORTANCE

LIGHT LEAF SPOT

Light leaf spot is caused by the fungus Pyrenopeziza brassicae Sutton & Rawlinson (anamorph Cylindrosporium concentricum Grev). The disease can be found on leaves, stems, flowers and pods. P. brassicae occurs throughout the UK and is one of the most common pathogens on oilseed rape (Tables 1, 8 & 9). The disease was first recorded in England on brassicas by Pethybridge (1926).

Symptoms

Light green roughly circular lesions up to 1 cm diameter form on either surface of the leaves. At first, the lesions may be hardly distinguishable from the surrounding tissue. With time the lesions become increasingly pale. Lesions may coalesce to form larger areas. The epidermis of the leaf within the lesion becomes brittle and develops fine cracks. If the leaf is bent larger cracks easily form across lesions. Lesions are usually confined to one or other surface but following extensive infection, cold or frosty conditions the tissue of the lesion may collapse and become bleached, the lesion then being apparent on both sides of the leaf. This symptom can resemble and be easily confused with fertiliser scorch where liquid or solid fertiliser has been trapped on the leaf and 'burnt' out a bleached lesion. Fertiliser scorch, however, usually has a well defined edge to the lesion and does not develop white 'spore droplets' (acervuli) around it.

The principal diagnostic symptom is the development of white 'spore droplets' mainly on green tissue bordering lesions. These spore droplets are spore masses of the imperfect stage of the fungus which develop on acervuli just under the epidermis and burst through. Occasionally, 'spore droplets' may develop before lesions are visible.

Symptoms can develop on any part of a leaf but are rarely found on the mid-rib. Lesions form most commonly where water droplets are trapped on the leaf surfaces. Leaf symptoms are rarely seen before December and

usually develop most rapidly in the new year. With continued weather favourable for the fungus, symptoms may develop at any time before leaf senescence. However, after stem extension, the open nature of the upper canopy and generally drier weather often results in less infection of upper leaves.

In the spring, light leaf spot infection can occur in foci, or uniformly across a field where infection has been widespread. The foci have plants with a bleached or pale appearance and growth may be retarded relative to the adjacent crop.

On the stems, superficial pink to fawn elongated lesions can develop. Lesions can be several centimetres long or extend the entire distance between internodes. The edges of stem lesions may be dark brown or black and formed from numerous black spots, decreasing in number away from the lesion. White 'spore droplets' are rarely formed on or around stem lesions. The lesions appear only after stem extension and are the result of infection in leaf axils at the rosette stage. There is no evidence that stem lesions affect the stem strength or increase lodging. Stem lesions can be severe even where fungicide programmes have been used. Stem lesions may become colonised by Phoma lingam or P. herbarum.

Flower buds, flowers, pedicels and pods can all be infected. Infection of flower buds at the green bud stage results in small light green lesions developing on outer tissues. 'Spore droplets' may occur around the lesions. At the yellow bud stage or during flowering, infection may result in the formation of 'spore droplets' without other symptoms. On pods and pedicels, light green lesions may develop, turning pale and mealy with age. 'Spore droplets' form around these lesions in humid conditions.

Infection of flower buds can result in a failure to develop flowers and/or pods. Where pods do form from infected buds or flowers or where infection occurs during early pod development, twisting and deformation of pods may occur. Pod infection can lead to premature splitting and some loss of seed but this effect is not as damaging to yield as that caused by Alternaria infection of pods.

Epidemiology

The infection process has been described by Rawlinson et al. (1978b). Spores deposit preferentially on anticlinal walls of epidermal cells. Conidia become septate and germinate terminally producing short germ tubes (less than 40 µm in length) which swell at the tip but do not form typical appressoria. Penetration occurs directly through the cuticle. A hypomycelium is formed after penetration. Long septate infection hyphae spread up to 2 cm between the cuticle and cell walls of epidermal cells. Small, shorter branches from these hyphae ramify beneath the cuticle to form a reticulum which proliferates to produce a more complex plate. Beneath the plate, hyphae penetrate deeper into the leaf but never into cell lumina. Above the plate the hyphae produce conidiophores in acervular conidiomata. The production of conidia continues until the cuticle ruptures exposing a mass of mature white conidia (spore droplet).

Efficient spore germination occurs only if the leaf surface is wetted at or immediately after deposition. Penetration takes five days at 5-15°C. For subsequent hyphal growth, wetting is unnecessary. The cycle from deposition to production of conidiomata was found to take 13-30 days depending on temperature (mean maximum/minimum temperatures of 20.7/10.6°C and 13.6/5.0°C respectively) (cv Eurora; Rawlinson et al., 1978b). Latent periods much longer than this have been suggested by others (Wale et al., 1990). Lacey et al. (1987), using ascospores, found the time from infection to symptom expression was 22 days at 15°C.

Above average rainfall can result in greater amounts of infection (Rawlinson et al., 1978 b). Thus a greater incidence and severity of light leaf spot occurs in Scotland where such favourable conditions are more likely to occur than in the south of England (Brokenshire & Prasanna, 1984).

Any action that decreases the leaf surface wax, thereby increasing leaf wettability, can aid the spread and retention of conidia and increase infection (Rawlinson et al., 1978a). The use of certain herbicides has been shown to reduce the leaf surface wax and result in greater levels of light leaf spot. In particular, dalapon affected the total amount and

form and distribution of leaf wax. This occurred for up to three months after treatment under controlled conditions (Rawlinson et al., 1978a). Some surfactants may have a similar effect. Conditions of relatively higher temperatures and humidity, low light and short photoperiod would also be conducive to less wax on rape leaves than in a normal winter (Whitecross & Armstrong, 1972). Leaf damage by wind or foraging animals or birds may also result in greater infection provided leaf tissue is not extensively removed. Inoculum in the form of naturally infected leaves remained viable for at least three months after air drying or deep freezing (-20°C) (Rawlinson et al., 1978b). Similar results were found by Maddock & Ingram (1981) where conidial germination declined slowly over 19 months from 90 per cent to 1-2 per cent. The decline in viability of spores under field conditions has not been determined but stubble may be a potent source of inoculum for a considerable period after harvest. However, whilst P. brassicae is capable of saprophytic growth on dead or decaying organic material, it is readily overgrown by more vigorous saprophytic micro-organisms (Maddock & Ingram, 1981). This suggests that once buried, after ploughing, survival on crop debris is likely to be limited.

Seed infection has been demonstrated (Maddock & Ingram, 1981), but at a low level. In addition, the viability of seed-borne conidia was lost after about two months. P. brassicae was considered to be associated with seed coats of mature, potentially viable seeds, although the intact testa does provide some barrier to the pathogen. Transmission to seedlings only occurred where the seed coat was split. The significance of seed-borne spread of P. brassicae is unknown but it is likely to be insignificant relative to other forms of dispersal.

Few wild species of crucifer proved susceptible to P. brassicae, all in the genus Brassicae (Maddock & Ingram, 1981). Natural infection on wild hosts has not been reported and this is also an unlikely source of the fungus for the oilseed rape crop.

Cultivars of winter oilseed rape low in both erucic acid and glucosinolate (double low cultivars) may be more susceptible to P. brassicae and other diseases and pests. Indirect evidence for this comes from work showing

that some glucosinolate breakdown products are toxic to a range of pathogens (Rawlinson, 1979; Rawlinson et al., 1985; Mithen et al., 1987). However, 22 cultivars were not found to be consistently more infected than the most popular single low variety, Bienvenu (Rawlinson et al., 1989). Yield responses on individual double low cultivars could not be related to differences in visible symptoms. Thus the degree of infection was not thought to relate to the degree of physiological stress imposed on cultivars. It was proposed that double low cultivars might be less tolerant of infection. Rawlinson et al. (1991) also suggest that double low cultivars benefit more from the use of pesticides than single low cultivars. However, Sweet & Beale (1991) reporting a series of cultivar trials were unable to confirm that double low cultivars gave higher responses to fungicide treatment than single low cultivars.

Recent work (Rawlinson et al., 1991) has been investigating the effect of diseases, including light leaf spot on glucosinolate content of leaves and seed. Glucosinolate concentrations in plant tissue are dynamic but in one season (1988) negative correlations were noted with glucosinolates in leaf tissue from the time of infection to when first symptoms developed. Thereafter, as the incidence and severity of infection increased, correlation with glucosinolates became positive. This may demonstrate the induction of glucosinolates after infection.

In trials using fungicide treatments to obtain differential infection, when light leaf spot was prevalent and severe, it was found to be positively correlated with the glucosinolate concentration in the seed (Rawlinson et al., 1991). But as the fungicide use increased so did the dry matter accumulation and a 'dilution' of the glucosinolates may have occurred as seed mass increases and glucosinolate content remained unchanged.

Glucosinolates are unlikely to be the sole basis for pest and disease resistance mechanisms in oilseed rape; other mechanisms are likely to interact and contribute to resistance (Rawlinson et al., 1991). However, it is possible that plants containing more of the biologically active glucosinolates, or the ability to produce them on demand more effectively,

could have been selected unwittingly by plant breeders during the process of developing cultivars for commerce.

Effect on yield

P. brassicae has been shown to affect many factors influencing yield in oilseed rape. Autumn infections have been clearly demonstrated as most damaging (Rawlinson et al., 1984; Jefferey et al., 1989; Wale et al., 1990). Severe infection can result in plant loss over the winter. Re-growth in early spring can be slowed or retarded and result in stunting. Leaf area index and leaf, stem and total plant dry weight can also be reduced. Ultimately yield is affected by a reduction in pod number per plant primarily but, to a lesser extent, also seed number/pod and mean seed weight.

Epidemics initiated in mid-January also have the potential to reduce growth rate and yield (Jefferey et al., 1989) but as the spring proceeds progressively later initiated epidemics seem to have less effect on yield. Gladders (1990a) has suggested that progress of light leaf in the spring on bracts and buds may reflect infection initiated in the winter. Similarly, Rawlinson & Cayley (1984) found that infection in the winter could affect primordial tissue of leaves and floral structures and still affect yield despite a dry summer.

Light leaf spot infection recorded during February to July or at stem extension has been negatively and significantly correlated with yield (Rawlinson et al., 1989; Sweet & Beale, 1991; Wale et al., 1990).

Life Cycle

McCartney & Lacey (1990) have recently proposed revised life cycles for P. brassicae in the UK. Revised cycles were necessary as a result of the discovery of the sexual stage, first in culture (Rawlinson et al., 1978b) and then in the field (Lacey et al., 1987). Reports of the teleomorph remain limited but confirmation of its widespread occurrence may only require careful observation throughout the UK.

P. brassicae is a heterothallic fungus with two mating types (Ilott et al., 1984). Also two types of apothecia have been identified, namely cup-shaped apothecia 1-2 mm diameter which form on dead petioles and smaller apothecia 0.1 - 0.2 mm diameter which can form on dead leaf lamellae (Lacey et al., 1987). Apothecia were most common on dead leaves lying on the ground adjacent to stems of rape plants. Asci from both types of apothecia were similar in size (33-89 µm x 4.2-8.9 µm) and contained eight ascospores. Hyaline septate paraphyses, as long as asci and 2 µm diameter are present. Ascospores and conidia are virtually indistinguishable, being hyaline, cylindrical, straight or slightly curved with rounded ends and occasionally septate. Conidial size on oilseed rape corresponded with that on other brassicas.

Small apothecia were found on leaves that had been yellow for more than 15 days but larger apothecia were found only on petioles and took 24 or more days to develop after leaves had died (Lacey et al., 1987).

Acervuli and conidia persist on stubble once a crop is harvested and volunteers developing in the stubble become infected either by conidia or ascospores released from apothecia on dead leaf debris. Apothecia may continue to release ascospores for up to three weeks, even under cycles of wetting and drying (McCartney & Lacey, 1990). Large and small apothecia can be found on dead leaves of volunteer seedlings by the end of October. Ascospores released from these volunteers may provide the primary inoculum and infections in crops at a distance. Once established, the fungus spreads within the crop by rain splash of conidia. Ascospores may be released from dead leaves in the crop after the new year but probably represent a small proportion of the total inoculum. Until the discovery of the perfect stage, spore dispersal over long distances was assumed to be due to splash dispersal of conidia. However, spore trapping studies demonstrated steep dispersal gradients and that the conidial concentration in traps was closely related to rainfall (Rawlinson et al., 1978b). This confirmed that splash dispersal of conidia occurs over relatively short distances (Maddock & Ingram, 1981). Run-off of water droplets containing spores was also considered to be an important mechanism of dispersal of conidia within a crop.

Clearly, conidia can play a part in transmission of disease from one crop to the next where a crop is drilled close to or in the same field as a previous crop. However, when trapping above and downwind of rape crops, McCartney et al. (1986) caught spores several hours after rain had fallen. This led to the first record of the natural occurrence of the teleomorph of P. brassicae (Lacey et al., 1987). In their primary studies of the dispersal of ascospores, Lacey et al. (1987) found up to 150 spores m^3 air averaged over a 24 hour period could be trapped from 1 January to 5 February. By mid-April the concentration exceeded 1000 spores m^3 and by mid-May 2000 spores m^3. Most ascospores were airborne during the morning when the crop was drying after rain. Ascospores were discharged only a few millimetres but turbulence within the canopy effected escape into the air. McCartney et al. (1986) contended there was a potential for dispersal many kilometres and this might explain the increase of light leaf spot in vegetable brassicas.

More comprehensive studies by McCartney and Lacey (1989, 1990) re-enforced many of the earlier observations. In infected crops ascospores were trapped in low numbers early in the year reaching peak numbers in May and July, which succeeded death of the rosette leaves and stem extension leaves respectively. Some ascospores were trapped before apothecia were seen in the crop. The production of ascospores coincided with rainfall but they were trapped on both rainy days and days after rain. Few were caught in traps during prolonged dry spells and the numbers caught during rain were less after a prolonged dry period than after a brief dry period. Ascospore production continued for several days after rain, crop wetness being essential in controlling production and release, but the peak of spores trapped followed the peak of rainfall by several hours. The release of ascospores lasted 2-6 hours. There was also a suggestion of diurnal periodicity with spore production maxima during 0600-1000 GMT.

McCartney & Lacey (1989) have estimated that more than 90 per cent of all ascospores released would still be airborne 100 m from a field edge. In addition 600-7000 ascospores s^{-1} were estimated to be released from each metre width of crop but these were considered as likely to be underestimates.

The recent discovery of a saprophytic ascomycete in the genus Unguicularia occurring ubiquitously on rape debris necessitates a review of the contribution of the sexual stage of P. brassicae to the epidemiology of light leaf spot during the season. Ascospores of both fungi are similar in size and shape and are typically dispersed in groups of four (A J Inman, pers. comm.). Due to their close similarity it is difficult to differentiate their ascospores in the field and past work involving the sampling of ascospores of P. brassicae without the concurrent sampling for apothecia must be viewed with caution.

Control

A large number of trials has been carried out in the UK using fungicides or fungicide programmes to evaluate the effect of light leaf spot and other diseases on yield. Because oilseed rape is prone to a variety of diseases which attack the crop at different stages it is often difficult to unravel the effect of one disease. However, in trials where light leaf spot has been the dominant disease the effect on yield has been calculated.

Some very large responses in yield have been found, up to 1.0 t/ha in both England and Wales (Rawlinson et al., 1984; Jeffery et al., 1989) and Scotland (Wale et al., 1990). In each case, infection was initiated in the autumn and winter. In some trials epidemics, were initiated by introducing inoculum but in Scotland autumn and winter infection occurs naturally in almost all crops (Wale et al., 1990) and here routine autumn fungicide treatment is advised (Fig. 9).

By contrast, in a long series of ADAS trials in England and Wales, despite light leaf spot frequently being the most common pathogen on pods, stems and leaves from stem extension onwards (Hardwick et al., 1989), usually no response to autumn and spring applications has been found (eg Evans & Gladders, 1981; Evans et al., 1984). Responses have been found only when infection was early and symptoms developed in the winter. Consequently, the advice issued by ADAS for light leaf spot control has been to apply fungicides when 25 per cent of plants show light leaf spot infection at stem extension (Giltrap, 1986).

Ploughing-in of crop debris after harvest and before the following season's crop has emerged should minimise dispersal. This is particularly relevant to reduce the airborne spread of ascospores. Considerable variation in resistance to P. brassicae exists in oilseed rape cultivars (Maddock et al., 1981) and a range of resistance is available in recommended cultivars (Anon, 1990a; Anon, 1990b). In general, large yield responses to fungicide treatment have been recorded in cultivars with low resistance to light leaf spot (Sweet & Beale, 1991). However, some cultivars with much greater resistance can also give large responses to fungicide treatment. Others have observed that responses of cultivars to infection are not entirely consistent with visual disease scores (Jeffery et al., 1989) or that the effect of light leaf spot infection may be proportionately greater on some cultivars (Wale et al., 1990).

A variation in virulence of isolates was detected by Maddock et al. (1981) but not to the extent that clear physiological races could be determined. The resistance of widely grown cultivars, such as Bienvenu, declined over a period of a few years, suggesting that some adaptation of light leaf spot to Bienvenu may have occurred. However, resistance to light leaf spot does not seem to vary with geographical location in Europe (J B Sweet, unpublished).

Two groups of fungicides have good activity against P. brassicae, namely MBC-generating fungicides and certain ergosterol biosynthesis inhibitors ('triazoles'). A wide range of MBC and two DMI fungicides, prochloraz and propiconazole are approved for control. Other DMI fungicides, approved for use in France, are in the UK registration system. The application of prochloraz in the autumn has been discussed by Scott and Rea (1986).

Widespread use of both groups have led to fears of resistance developing in P. brassicae. However, two surveys failed to detect resistance in field isolates to an MBC fungicide, benomyl (Ilott et al., 1987; Ball et al., 1990). In vitro studies on resistance showed that resistance to MBC's in P. brassicae was conferred by a single gene and resistant strains could be produced in culture. There were indications that resistant strains were less fit as their growth in culture was slower than that of susceptible strains.

Autumn infection of oilseed rape is most damaging yet symptoms may not be obvious until spring. Thus, monitoring inoculum dispersal or detecting infection in the autumn may offer the opportunity to predict when autumn fungicide treatments are justified. Bait plant techniques have been used by Gladders (1990b) and Wale et al. (1990) but the incubation period of 3-4 weeks before symptoms develop on bait plants is too long for practical advice to be given. Spore trapping is too time consuming and early detection of leaf infection by serological methods may be inappropriate where initial infection is sparse in a crop.

Light leaf spot is widespread in the UK and is now considered to be the main fungal disease of oilseed rape. Disease/yield loss relationships are uncertain but control can be achieved by fungicides applied in the autumn and early spring. There is resistance in many current cultivars, but when disease pressures are high reliance has to be made on fungicides for disease control.

PHOMA LEAF SPOT AND CANKER

Leaf spot and canker caused by the fungus Leptosphaeria maculans (Desm.) Ces. & de Not. (the perfect stage of Phoma lingam [Tode ex Fr.] Desm.) is a common disease of winter oilseed rape distributed widely in temperate regions or at high altitudes in the tropics. It causes high yield losses by premature ripening and lodging. Yield losses since 1979 have been largely prevented by the sowing of resistant cultivars (eg Jet Neuf, Rafal, Bienvenu). In the present NIAB Recommended list, however, the new double low cultivars do not generally have a good level of resistance to the disease.

Symptoms

During the autumn-spring period leaf spots can be found on the lower leaves. They range in colour from light green to fawn or white and are from 3-10 mm in diameter. The edge of the lesion may be surrounded by a dark brown margin. In the centre of the lesion can be found numerous dark brown pinhead sized bodies (200-500 µm in diameter), the pycnidia, embedded in the senescent tissue. The central tissue of the lesion is thin and can drop out leaving a hole. When this occurs early in the autumn the damage can resemble that caused by slugs. If conditions remain favourable, lesions continue to be produced on the leaves during the life of the plant. All aerial parts of the plant can be attacked and it is not uncommon for the primary raceme to be killed. Symptoms initially resemble those cause by Botrytis (see page 54), but can be distinguished from them by the lack of a watersoaked appearance to the rot, the absence of fungal growth on the rotting parts and the presence of typical small cankers at the base of the rot. These cankers typically have a dark margin with a buff-coloured centre. Pycnidia are generally present in the centre of the lesion. From flowering onwards, the canker phase can be found as sunken brown areas at the stem base. The fungus can also cause blackening of the roots. Severe canker causes premature ripening and crop lodging. Lesions on the lower 30 cm of the main stem are also common, but usually occur in association with pest damage (eg cabbage stem flea beetle or stem weevil)

(Newman, 1984a) or, as demonstrated by Hammond & Lewis (1986a), are stimulated by products of anthesis.

A related species, Phoma herbarum Westend., can commonly be isolated from leaf and stem lesions. This fungus is entirely saprophytic and generally can be found colonising leaves that have become damaged and stem lesions initially caused by the light leaf spot fungus. P. lingam can be distinguished from P. herbarum by the slightly smaller conidia, 3.5-4.5 x 1.5 µm compared with 4-5 x 1.5-2.0 µm, and in addition the presence of a small oil droplet. A major macroscopic charactersitic which distinguishes the two species is that, in mass, the conidia of P. lingam are amethyst in colour whereas those of P. herbarum are typically buff-coloured (Anon, 1964).

The ascospores of the fungus, which feature in long range transmission of the pathogen, are produced in ascocarps 300-500 µm in diameter. The asci contain eight ascospores which are elongated ellipsoidal, septate with cells containing oil droplets and 35-70 x 5-8 µm in size.

Epidemiology

The main source of infection is stubble debris from the previous harvest. Large numbers of airborne spores (ascospores) are liberated from fruiting bodies (pseudothecia) on such stubbles from autumn through to spring. Autumn infection, before the five to six leaf stage, tends to produce severe cankers with aggressive isolates. Disease gradients are very steep and although airborne spores can travel over a kilometre, significant attacks are usually within 400 m of a stubble source (Gladders & Musa, 1980). The fungus can be seed-borne, existing as resting mycelium between the seed coat and embryo Neergaard (1979). However, the ascospore phase is the major source of inoculum (McGee, 1977).

Increases in phoma leaf spot and canker have been detected following the use of TCA herbicides (Rawlinson et al., 1978a). Phoma readily colonises damaged tissues and is usually more evident on aerial stems if there is damage from pests such as cabbage stem flea beetle. Premature ripening in

one crop of Ariana in 1987 was caused by an attack of Phoma 30-40 cm above the ground and appeared to be secondary to pest damage.

A key feature of the life cycle is the spread of the fungus from leaf spots to the stem where cankers develop. Hammond et al. (1985) showed that the main pathway for the canker phase is the systemic growth of the fungus through the leaf blade and petiole. The systemic nature of the main line of infection to the canker phase is suggested as a reason for poor control of the disease by fungicides. Field observations in the late 1970's indicated a good correlation between the incidence of leaf spotting in the autumn (November) and the incidence of severe canker at harvest in the following July on susceptible cultivars (Gladders & Musa, 1980). The leaf spot phase was usually more common in the spring during extension growth than in the autumn but infection in the spring gave mainly slight canker lesions. There was a long latent period of several weeks between the initial stem infection and the appearance of canker. Leaf spotting may be apparent in November but cankers were generally not apparent until the end of May. These observations were made on susceptible cultivars such as Primor and Quinta. On more resistant cultivars such as Jet Neuf, cankers were present but generally not severe because their development was restricted by host resistance.

Hammond & Lewis (1986b) demonstrated that epidemics of L. maculans are monocyclic with five developmental sequences following infection of the leaf. These are: latent infection within the leaf, leaf lesion expression followed by symptomless systemic spread in the lamina and petiole through to latent infection of the stem and finally canker development. They suggested that a direct relationship between the incidence of leaf spots and subsequent cankers may be too simplistic as it is likely to be influenced by the timing of infections and the age of leaf. Abscission of the leaf before systemic spread down the petiole occurred would reduce the number of cankers which subsequently developed (Newman & Bailey, 1987a). Hammond & Lewis (1987a) demonstrated the biotrophic phase of L. maculans in oilseed rape. They reported, however, that as the leaf aged the ability of the fungus to establish systemic infections decreased. Hammond & Lewis (1987b) reported different reactions to isolates of Phoma on the cultivar Rapora. They found that

older leaves were more susceptible to infection, and they also found a process of secondary lesion development when mycelium grew over the leaf surface from a delineated lesion.

Strains

L. maculans has virulent and avirulent strains, both of which occur in oilseed rape. These can be distinguished by differences in cultural characteristics. Avirulent strains are faster growing and produce aerial mycelium (Johnson & Lewis, 1991). Avirulent strains also infect a wide range of brassicae hosts (kale, swede, turnip, cabbage) where they can, however, cause severe cankers (Humpherson-Jones, 1983). The strains can be distinguished by pathogenicity tests on seedlings (Humpherson-Jones, 1985), but a more rapid technique using agar media can also be used (McGee & Petrie, 1978).

Johnson & Lewis (1991) have demonstrated DNA polymorphism in L. maculans and have separated two distinct genetic groups, A and B. These groups correspond with those that can be separated on the basis of the accumulation of pigment in liquid culture, group B producing pigment. They do not, however, always correspond with the potential to produce cankers. The non-aggressive pigment forming isolates have a wide host range. The aggressive non pigment forming isolates only infect B. napus and B. rapa (R D Johnson & B G Lewis pers. comm.). Hammond & Lewis (pers. comm.) and Johnson and Lewis (pers. comm.) have shown that the aggressive strain is often symptomless, or produces small lesions on leaves in the autumn but nevertheless develops to produce stem cankers. Aggressive strains also produce ascospores earlier than non-aggressive strains and therefore infect at a time when plants are more susceptible and are thus more likely to develop cankers. Hammond & Lewis (1987b) suggest that the cultivar Rapora may be used to differentiate strains of Phoma.

In addition to the absence of pigment production, Badawy et al. (1991) have shown that sirodesmins are produced only by aggressive strains of L. maculans. They were able to demonstrate that aggressive isolates could be further divided in to five sub-groups based on their reaction to three cultivars of oilseed rape (Quinta, Galcier and Jet Neuf), A0, A1, A2, A3

and A4. Badawy et al. (1991) designated the aggressive isolates 'pathotype group A' and the non-aggressive isolates 'pathotype group NA'. NA isolates only produced weak symptoms on leaves, cotyledons and hypocotyls. The A2 strain was of most concern because of its reaction on Jet Neuf, where it produced severe lesions in hypocotyl tests. Jet Neuf is the source of canker resistance in many new cultivars and they suggested that single ascospore lines should be screened regularly to see if the A2 pathotype is increasing. Badawy et al. (1991) detected resistance to Jet Neuf on leaves and hypocotyls but not cotyledons. They supported the findings of Newman (1981) that cotyledons are not reliable as determinants of resistance. They suggested that the leaf resistance found in Jet Neuf is important because of the systemic pathway from leaves via petioles to stem infection and subsequent cankering. Newman & Bailey (1987a) demonstrated that internal necrosis of stem tissue was as important a factor in assessing resistance as the usual assessment of crown cankering. They devised a scoring system to take account of the different reactions of cultivars and breeding lines. They also demonstrated a good correlation between artificial seedling infections in the glasshouse and field infections, particularly from selections derived from Jet Neuf, in contrast to those from other sources.

While the designation of the two strains differs between the two groups of workers (Badawy et al., 1991; Johnson & Lewis, 1991), the marked differences between non-aggressive and aggressive isolates have led them to consider that the two strains may be different species.

Control

Control of the seed-borne phase has been investigated by Maude et al. (1984) on vegetable brassicas. They discussed the merits of hot water treatment (Bant et al., 1950) and the use of a thiram soak (Maude et al., 1969). Good control of Phoma was achieved by MBC fungicides applied as slurries and dusts. Fenpropimorph was found to be particularly effective for control of both Phoma and Alternaria and is now commercially available in combination with thiram and gamma HCH.

Information on the control of canker with fungicide sprays on leaves and stems is limited and there is little information on the control of severe infection. For the reasons outlined by Hammond et al. (1985) and referred to above, control of canker with fungicides has been variable (Rawlinson & Muthyalu, 1979; Rawlinson et al., 1984). ADAS trials on Jet Neuf during 1981-84 (Evans et al., 1984) did provide information on prochloraz and this is currently considered to be the most effective fungicide for canker control (Fig 6). Many other fungicide products were found to give little (10-20 per cent) or no control of the leaf spot and canker phase of the disease.

The systemic nature of infections and the period of latency reduces the impact of protectant fungicides. Work in Australia (Ballinger et al., 1988), indicated that the systemic triazole fungicide flutriafol applied to soil, either coated on superphosphate fertiliser or as a granular formulation, gave good control of canker and a positive yield benefit.

Resistant cultivars provide a cost-effective means of control, as has been demonstrated by the introduction of the cultivars Jet Neuf and Rafal in 1979. Screening for canker resistance is an essential part of breeding programmes (Newman, 1984b). Observations on ADAS trials in 1989 confirmed that the double low cultivar Cobra may suffer significant loss of yield from severe canker where an autumn threshold of 20 per cent plants affected is exceeded (Gladders, pers. comm.). Severe infection was controlled by a full spray programme (prochloraz in the autumn and spring followed by thiophanate-methyl plus iprodione at the end of flowering). No other cultivar gave a yield response at the same site.

The move to double low cultivars has focused attention on the role of glucosinolates in resistance mechanisms. The effects of glucosinolates on the disease have been investigated by Mithen et al. (1986, 1987) who concluded that plants which produced leaves that restricted lesion development had higher levels of alkenyl glucosinolates than those which produced large lesions or systemic infections.

Much data have accumulated on the infection process. Systemic infection by the fungus is well documented. Two distinct strains have been

identified and shown to vary in pathogenicity and genetic composition. The systemic nature of infection has consequences for fungicide control, although sources of genetic resistance are available. Current cultivars generally have low levels of resistance and are potentially at risk. Data on fungicides and their timing would be required if resistance should fail.

DARK LEAF AND POD SPOT

In the UK, 'dark leaf and pod spot' of oilseed rape is caused mainly by Alternaria brassicae (Berk.) Sacc. and A. brassicicola (Schwein) Wiltshire. A. brassicae is the more important of the two species. Following an increase in dark leaf spot with the intensification of oilseed rape (Evans & Gladders, 1981) it caused serious losses in 1980/81 (Evans et al., 1984) and since then £3-4M has been spent each year on fungicide seed dressings and sprays to control it (Humpherson-Jones, 1985).

Dark leaf and pod spot causes yield loss due to premature ripening and shedding of seed before harvest and by reducing the 1000 grain weight. Severe lesions reduce both the area available for photosynthesis on leaves and pods, and the length of time that they contribute photosynthates before senescing. Infection of inflorescences leads to the abortion of florets, although this does not necessarily affect yield (Louvet, 1958). Seed yield from infected plants is reduced because their pods contain fewer, smaller seeds (McDonald, 1959; Degenhardt et al., 1974; Kadian & Saharan, 1983; Kolte et al., 1987), and more importantly because severely infected pods become shrunken and senescent and dehisce prematurely.

In Europe, epidemics occur on rape about two years in every five and in these years losses may be as high as 60 per cent in individual crops (Domsch, 1957). Weather conditions are critical during May-July when pods are developing and ripening and severe attacks may be expected if prolonged warm, wet weather occurs at this time. Weather must favour the development of severe epidemics before yield is affected. Daebeler & Amelung (1988) reported that yield was affected in German crops only if lesions cover more than five per cent of pod area. Similarly, Kadian & Saharan (1983) found that yield was not reduced in Indian rape seed if pod lesions remained superficial. Once a threshold of disease severity is exceeded, yield reduction occurs in proportion to the severity of Alternaria infection (McDonald, 1959; Bandyopadhya et al., 1974; Degenhardt et al., 1974; Singh & Bhowmik, 1985).

The effects of Alternaria infection on the various determinants of quality in oilseed rape are less clear. Losses of up to 36 per cent in the oil content of infected B. campestris seed have been reported (Kadian & Saharan, 1983; Ansari et al., 1988). But Degenhardt et al. (1974) failed to find any effect of infection on the oil content of B. napus seed. Carbohydrate and protein content of B. campestris seed were affected by infection in the studies of Bandyopadhya et al. (1974) and Degenhardt et al. (1974) respectively, but Stovold et al. (1987) found no relationship between the level of Alternaria infection of samples and the percent oil, percent protein, glucosinolate content or the fatty acid profile of seed.

Alternaria spp. are associated with leaf and pod spot diseases in each of the major areas of rape seed cultivation in Europe, Canada and India. A. brassicae is widespread throughout Europe and has been reported from almost everywhere cruciferous crops are grown. A. brassicicola has been reported from every continent but it is not as widespread. Other Alternaria spp. have been isolated from lesions, but they are usually saprophytic on rape seed. The exact geographical distribution of these pathogens, and comprehensive lists of the countries in which they have been recognised can be found in a review by Neergard (1945) and the Commonwealth Mycological Institute Distribution Maps of Plant Diseases (Anon., 1984b; Anon., 1988b). Both Alternaria spp. attack a wide range of cultivated and wild crucifers. Most, if not all, commercially grown cultivars are susceptible, including vegetable brassicas, forage brassicas and mustard. The increased cultivation of this crop in Europe, has resulted in more cases of serious damage on rape in recent years.

The occurrence, relative importance and nature of the diseases produced by Alternaria spp. vary between regions. McDonald (1959) referred to the diseases on Canadian crops as 'grey leaf spot' and 'black leaf spot', caused by A. brassicae and A. brassicicola respectively. A. brassicicola does not cause significant damage in the prairie provinces, possibly because the generally lower temperatures there often fail to meet the requirement of this species for spore germination (Degenhardt et al., 1982). Artificial inoculation with A. alternata alone results in symptoms on rape seedlings, leaves and inflorescences (Berkenkamp & Vaartnou, 1972; Vaartnou & Tewari, 1972), but A. alternata is present mainly as a

saprophyte on leaves (Tsuneda & Skoropad, 1978a) and seed samples (Petrie, 1974); this species may have both saprophytic and parasitic strains on canola (Tsuneda & Skoropad, 1978a). A. tenuis is also found as a saprophyte on oilseed rape, particularly on swathed crops (Vanterpool, 1963), but is unable to infect Brassica spp. (Morton, 1964; Richardson, 1970).

Symptoms

Both A. brassicae and A. brassicicola cause dark leaf spot and pod spot on oilseed rape, and can cause symptoms on all plant parts at all stages of growth. The symptoms produced by the two species are similar and often indistinguishable. Both fungi are seed-borne and occur superficially on seed as spores and mycelium and also as internal mycelium within the testa and occasionally in the embryo tissues. In transplant beds where seedlings are grown close together in conditions of high soil moisture and high humidity, seed infection by either fungus may cause severe damping-off (McDonald, 1959), but this is not usually a problem in direct-drilled field crops.

Emergent seedlings may have wirestem symptoms on hypocotyls (Rangel, 1945) which can prevent further development (Richardson, 1970). On surviving plants, lesions start on cotyledons and leaves, and eventually on bracts, as small, black spots (diameter 1-3 mm) usually in groups and visible on both the abaxial and adaxial surfaces. On young leaves they remain small, but on mature leaves they expand to form larger (0.5-2.5 cm) brown 'target-like' lesions, with zones according to the density of sporulation, and with distinct margins, surrounded by chlorotic 'halos'. Older lesions are circular, often zonate with a papery, thin centre and may be covered with a mat of spores which are yellow in the case of A. brassicae and dark olive-brown in the case of A. brassicicola. However, differentiation of species cannot reliably be achieved on the basis of symptoms. Lesions eventually coalesce to form larger irregular areas in which the starting points of individual lesions are visible as darker spots. Tissue at the centre of lesions becomes dry and papery, often producing a shot-hole symptom when tissue becomes detached (Neergard, 1945). Severely infected leaves may defoliate prematurely.

Lesions on petioles and stems often take the form of black, elongated streaks, resulting from the deposition of spores by droplets running down them. Inflorescences may become infected, leading to floret abortion. Infection during flowering has the additional effect of elongating the flowering period (Degenhardt, 1973). A 'pod drop' syndrome, attributable to A. alternata (Fries) Keissler and a Cladosporium species, has been reported in Canada (Petrie, 1973b). Blackening of the pedicel at the point of pod attachment causes pods either to be lost or to develop poorly. Spots on the pods themselves are caused by the major Alternaria species. Infected pods are generally smaller and may contain shrunken infected seeds, which often germinate prematurely, in situ (McDonald, 1959). Severely spotted pods become dry and are liable to split prematurely, releasing seed.

A. brassicae is distinguished by its characteristic, very long (75-350 µm), pale olive conidium with a pronounced beak up to half its length. Conidia are usually solitary but may occur in chains of up to four. A. brassicicola has smaller (18-130 µm), darker conidia without a pronounced beak in chains of up to 20 or more. No perfect stage has been identified for A. brassicae or A. brassicicola which are members of the Fungi Imperfecti (class Moniliales). If these fungi are identified in their sexual stage they are likely to be placed with other Alternaria species in the Loculoascomycete genus Pleospora (Webster, 1980). There is no evidence of host specialization or physiological races of these Alternaria species.

Hyphae are septate, branched, up to 8 µm wide, hyaline at first but eventually attaining a colour generally described as 'olive-buff', varying in intensity (Groves & Skolko, 1944; Neergard, 1945; Wiltshire, 1947; Ellis, 1971). Conidiophores are produced in groups of up to twelve, often emerging through stomata as erect, usually unbranched structures which are slightly swollen at the tip and olive-brown in colour. They are up to 11 µm wide, but length varies; conidiophores of A. brassicae reach a maximum length of 170 µm, potentially longer than those of A. brassicicola at 70 µm. Conidia are 'club-shaped', olivaceous, multicellular, thick-walled with both transverse and longitudinal (oblique) septa, and constrictions at the junctions of the septa and the external wall. The appearance of

the fungi in culture depends on the medium used, but cultures are generally cottony, from white to olive-brown. Areas of concentrated sporulation tend to be darker, and where light conditions fluctuate, light and dark concentric rings may be discernable on cultures. A. brassicicola sporulates in culture more easily than the other species. A. brassicae occasionally forms chlamydospores in culture but they are not produced by A. brassicicola.

Under favourable conditions, Alternaria conidia germinate to produce germ-tubes, which grow for a short distance before attempting to penetrate the host. Infection may be achieved by a single, viable conidium. Each cell of a conidium is capable of giving rise to a germ-tube, though usually only a few do so, possibly because of the existence of a mutual inhibition mechanism between cells (Joly, 1964). Penetration is attempted via an infection hypha from the underside of a simple appressorium. Further germ-tube growth can develop from an appressorium, whether or not it has successfully penetrated, and a second penetration attempt may be made at another site (Ruscoe, 1967). Penetration takes place via stomatal apertures or directly through the cuticle. Ruscoe (1967) found that A. brassicae penetrates predominantly via stomata, and A. brassicicola mainly through the cuticle but Tsuneda & Skoropad (1978a) report that these species penetrate by both routes, apparently at random. Stomata may be an important route for penetration as Saharan & Kadian (1983) showed resistance among rape seed and mustard cultivars to be related to the frequency and aperture size of stomata on their leaves. Post-penetrational development of the pathogens has been studied for A. brassicicola and A. brassicae by Ruscoe (1967) and Tewari (1986), respectively. In leaves, both species undergo a short subcuticular phase, followed by colonization of the epidermal and mesophyll layers. Hyphae grow both intercellularly and intracellularly. Eventually the centre of a lesion on an infected leaf contains hyphae ramifying through all parts, except vascular bundles. Finally, groups of conidiophores are produced as branches from mycelium in the epidermis and emerge either via stomatal apertures or by penetrating the cuticle.

Epidemiology

Germination of A. brassicicola conidia is most rapid at around 30°C (Domsch, 1957) but A. brassicae spores germinate best at temperatures from 17-21°C (Rangel, 1945); this lower range means that at lower temperatures more A. brassicae conidia germinate in less time than those of the other species, allowing it to infect rape seed faster and more effectively. At temperatures lower than 17°C only A. brassicae is capable of causing lesions on rape seed (Degenhardt et al., 1982). Germination and penetration occur rapidly at optimal temperatures; at 22°C, A. brassicae can infect rape seed leaves within six hours (Louvet & Billotte, 1964; Degenhardt et al., 1982). Subsequent disease development occurs most rapidly at between 20 and 24°C for A. brassicae (Van Schreven, 1953; Domsch, 1957; McDonald 1959; Louvet & Billotte, 1964) and at 24°C for A. brassicicola (Domsch, 1957). In Europe, lesions caused by A. brassicae in the field expand only if temperatures exceed 18°C (Louvet & Billotte, 1964). Alternaria spore germination is impeded if relative humidity drops below 97 per cent and germination occurs best in the presence of free water.

Sporulation from leaf lesions by A. brassicae and A. brassicicola requires at least twelve hours of continuous high humidity and temperatures above 14°C (Humpherson-Jones & O'Brien, 1986). Sporulation is also affected by light; A. brassicae is inhibited from sporulating in continuous light (Humpherson-Jones & Phelps, 1989) and spores produced at high light intensity and temperature lack viability (Mukadam & Deshpande, 1979). Best sporulation takes place in alternating light and darkness (Gupta et al., 1972; Saharan & Kadian, 1983). Sporulation from lesions is greatest in the field when rain is followed by warm, dry weather.

Spores, which are the principal means of pathogen spread within and between crops, are typically dispersed by wind, although disease spread is associated with periods of wet weather because water is required for germination and infection. Investigations using spore samplers have shown that conidia are common in the wind-borne air microflora although they can also be dispersed by rain-splash over short distances (Louvet, 1958;

Louvet & Billotte, 1964). Rain can also wash spores from the air above the crop and release them from sporulating lesions by the force of impact of drops on the leaf through the 'puff' or 'tap' mechanisms (Hirst & Stedman, 1963). Transport of spores over large distances is effected mainly by wind. Along with seed infection, wind is the most likely agent for the spread of inoculum to new sowings of rape seed or new areas of cultivation. Spore release, which is passive, is stimulated by falling humidity and restricted by high humidity so that air spore concentrations in a diseased crop exhibit a distinct diurnal periodicity with maximum concentrations occurring in the early afternoon and minimum concentrations in the early morning (Louvet & Billotte, 1964).

Infected seed is a primary source of the fungus. However, crop debris and nearby vegetable and fodder brassica crops will contribute inoculum. In the UK, up to 19 per cent internal seed infection may occur in commercial rape seed and in India 50 per cent seed infection is not uncommon in this crop and in mustard. In B. oleracea seed, A. brassicae is less frequent, most infection in this species being caused by A. brassicicola (Maude & Humpherson-Jones 1980a). Both fungi survive in infected seed for many years in cool, dry conditions. In Europe, Alternaria epidemics on rape seed may begin as infections on leaves during autumn with no further development during winter, followed by a resurgence of new infections in spring and summer as temperatures rise. However, the level of infection in autumn does not necessarily predict the incidence of disease in spring. The most important factors, epidemiologically, are those which influence the yield-reducing pod infection stage of the epidemic. Pod disease may occur as the result of one or a few infections or a continuous series of infections (Louvet 1958). At one extreme, a single, massive infection takes place when favourable conditions coincide for a certain period. At the other extreme, when optimum levels of environmental factors do not coincide, pod infection takes place as the cumulative result of a series of less effective infections. Frequent rain throughout the season would satisfy the requirements for the latter type of epidemic, but in a season of lower rainfall the former type of epidemic may take place only if optimal conditions of temperature, wind and humidity occur at the time of rainfall.

Conditions optimal for an Alternaria epidemic on rape seed in Europe are warm, dry weather to encourage sporulation, followed by wind and rain to distribute spores, then a period of high temperature and humidity to encourage germination, infection and development in the host (Domsch, 1957; Louvet, 1958). Alternating periods of wet and dry weather are particularly effective in encouraging disease (Quak, 1956; Louvet & Billotte, 1964). Severe Alternaria epidemics are associated with seasons of high rainfall in Canada (Vanterpool, 1963) and India (Chahal & Kang, 1979) as well as in Europe (Stankova, 1972).

Seed infection is the main means by which this pathogen survives between crops. Surveys of contamination by Alternaria have revealed a high incidence of the pathogen in commercial seed in each of the major rape seed-growing areas (Petrie, 1974; Randhawa & Aulakh, 1981; Humpherson-Jones, 1985). The incidence of contamination of seed correlates well with the number of lesions on the pods from which they are collected (Tripathi & Kaushik, 1984). Seed infection follows penetration of the pod wall by mycelium from external lesions; the fungus grows well in the humid interior of the pod and spreads to the developing seeds. Internal infection of seeds is largely restricted to the testa (Domsch, 1957), especially the hilum area (Knox-Davies, 1979). Less often the embryo becomes infected if fungal colonization occurs before the hypodermal layer of the testa thickens (Maude & Humpherson-Jones, 1980a). In some circumstances, Alternaria can survive in seed well beyond the normal intercrop period. Superficial inoculum on seed declines rapidly but according to temperature, internal mycelium is more resilient during storage. Internal mycelium of A. brassicicola remains viable for at least twelve years in stored Brassica seed, although Alternaria contamination of rape and mustard seed declines rapidly during storage at high temperatures (Chahal, 1981).

Large numbers of Alternaria spores are released from oilseed rape crops (Petrie 1973a; Humpherson-Jones & Ainsworth, 1983) and may infect alternative hosts, mainly cultivated Brassicas and cruciferous weeds but also including non-cruciferous species. Inoculum generated on these hosts contribute in turn to infections of subsequent oilseed rape crops. Transfer of inoculum between oilseed rape crops and alternative hosts is

facilitated by the lack of physiological specialization and the absence of genetic race structures within rape seed-infecting Alternaria species. Variation exists among isolates in cultural characteristics and pathogenicity on seedlings (Atkinson, 1950; Van Schreven, 1953; Mridha, 1983) but cross-infectivity studies with isolates of Alternaria from a range of cultivated and non-cultivated crucifers (Humpherson-Jones & Hocart, 1983) have revealed no host specificity. Within rape seed, no cultivar/isolate interactions emerge when A. brassicae isolates are inoculated onto various cultivars (Mridha, 1983).

Alternaria species are unspecialized pathogens, capable of survival in the absence of living host tissue. The main substrate for saprophytic survival is moribund Brassica tissue (Humpherson-Jones, 1989) but Alternaria can also be isolated from the surfaces of non-host plants such as flax (Petrie, 1974).

Colonization of rape seed debris is a major means of survival between crops. In Europe, A. brassicae and A. brassicicola can produce viable spores from diseased stubble for up to 22 weeks after harvest - (Humpherson-Jones & O'Brien, 1986) which far exceeds the intercrop period. Specialized structures have been identified which may contribute to long-term survival of inoculum. Conidia of A. brassicae become transformed into microsclerotia on decaying rape seed tissue (Tsuneda & Skoropad, 1977b).

Control

Although Alternaria has been a serious problem in continental Europe for many years, fungicide treatments to control seed-borne and foliar infection have only recently been used extensively, mainly due to the absence of effective chemicals. In the past, soak treatments in hot water or fungicide suspensions have been used to eliminate seed-borne Alternaria. However, problems in drying the seed after treatment and impaired germination in some seed stocks have restricted these treatments to small quantities of high-value vegetable brassica seed. The level of contamination of seed stocks can be limited by applying fungicides to seed-multiplication plots during pod-filling (Maude et al., 1984;

Humpherson-Jones, 1985). Surviving inoculum is present as spores and mycelium at the seed surface and as internally-borne mycelium below the testa. Control of superficial inoculum can be achieved by many treatments but internal mycelium is eradicated only by using systemic fungicides. In comparative studies, tridemorph, thiram and fenpropimorph eradicated seed infection but the dicarboximide iprodione was most effective without decreasing seed viability (Maude & Humpherson-Jones, 1980b). Iprodione is now widely used for this purpose (Wu & Lu, 1984; Kolte, 1985; Stovold et al., 1987) although, in India, carbendazim, captafol and mancozeb are also applied (Kumar & Singh, 1986). Iprodione has traditionally been applied to seeds in the form of a dust or a slurry but Maude & Suett (1986) have recently developed a method for applying the chemical in a polymer seed coating, giving better control of Alternaria than conventional techniques. However, a mixture of fenpropimorph, thiram and gamma-HCH has become the standard seed treatment for the crop.

Seed-treatment alone is often insufficient to exclude Alternaria from crops because wind-borne inoculum arrives from other sources. Control of leaf infections may be required during heavy epidemics, but more usually control of pod infection is important and applications at this stage are more likely to result in economic yield benefit. However, late season fungicide sprays gave a yield benefit in only a third of trials in Scotland between 1984 and 1989 (Sutherland et al., 1990). In Europe, iprodione is widely used in spray treatments as well as seed treatments. It has performed well in comparative tests (Ogilvy, 1984), controlling Alternaria by reducing the number of lesions on pods and limiting their ability to produce spores (Peres & Regnault, 1987) (Fig. 4). Control is most effective when applications are made at the end of flowering; these may increase yield by up to 22 per cent at severely infected sites (Cox et al., 1981; Evans & Gladders, 1981; Evans et al., 1984; Ogilvy, 1984) (Fig. 5). If conditions remain conducive to infection however, a subsequent spray may be necessary (Davies, 1986b). In future, fungicides may be developed by exploiting the toxicity to Alternaria of certain fungus and bacterium-derived antibiotics. For example, mycostatin and griseofulvin are toxic to A. brassicae in vitro (Singh & Rai, 1982) and polyoxins reduce mycelial growth and cause abnormal spore germination in this species (Tewari & Skjoropad, 1979). Griseofulvin and polyoxins B and D

are systemic and they control Alternaria effectively when sprayed on to plants (Husain & Thakur, 1962; Tewari & Skoropad, 1979).

The incidence of Alternaria infection in a rape seed crop is related to its proximity to debris from previous or adjacent crops (Daebeler & Amelung, 1988). Crop rotation reduces the danger of contamination, and cultivations which dispose of or destroy rape seed debris remove a substrate on which inoculum can survive. Similar benefit can be gained by removing from the vicinity cruciferous weed species which act as alternative hosts for Alternaria. Other cultural practices may affect the incidence of Alternaria indirectly. In India, early sowing of rape seed (B. campestris) allows the crop to escape disease (Kolte 1985). But in Europe early, and especially dense sowings, subjected to an application in the autumn, result in crops which have a closed canopy by early spring, under which Alternaria infection is favoured by high humidity (Louvet, 1958). Subsequent fertilizer applications may affect susceptibility to pod infection. While Quak (1956) reported that a full fertilizer input confers resistance to pod infection, Stankova (1972) found that heavy fertilizer application increased pod disease.

As fungi which spend a proportion of their life cycle growing saprophytically on host and other surfaces, Alternaria species are vulnerable to competition for nutrients, antagonism or parasitism by other saprophytes. Various fungi and bacteria, including species isolated from the phyllosphere and rhizosphere of Brassica, have the ability to parasitize rape seed-infecting Alternaria species. For example saprophytic phylloplane fungi such as Aureobasidium pullulans and Epicoccum nigrum parasitize A. brassicicola (Pace & Campbell, 1974). A. brassicae conidia leak nutrients which can be used by other phylloplane microorganisms, including Nectria inventa, which grows tropically towards A. brassicae spores and mycelium, and establishes mycoparasitic contact via appressoria (Tsuneda & Skoropad, 1977a; 1978b).

Pseudomonas fluorescens, isolated from the rape seed rhizosphere, produces antibiotics which are toxic to A. brassicae (Dahiya et al., 1988). Wu & Lu (1984) also isolated 14 fungal, and 27 bacterial species from the seed and rhizosphere of Brassica capable of inhibiting A. brassicicola. In

particular, Penicillium janthinellum mycelium parasitizes A. brassicicola by coiling around conidia and hyphae; several of the other species isolated caused plasmolysis and granulation in conidial and hyphal cells.

Hyperparasites, or the antibiotics produced by antagonists, may in future be incorporated into formulations for biological control of Alternaria on rape seed. One such formulation, 'Mycostop', already exists for protecting cabbage seedlings against A. brassicicola. It consists of a powdery preparation of spores of a Streptomyces species isolated from a Finnish sphagnum peat, suppressive of A. brassicicola (Tahvonen, 1985), and protects seedlings from the pathogen as effectively as thiram (Tahvonen & Avikainen, 1987). Spore suspensions and culture filtrates of Streptomyces are also effective in protecting rape seed from A. brassicae and A. brassicicola (Sharma & Gupta, 1978b, 1979). Other antagonists, capable of protecting cabbage seedlings, were identified by Wu & Lu (1984) and Vannacci & Harman (1987) used antagonists of A. raphani to reduce infection of radish pods. Formulations for biological control of Alternaria on rape seed might in future incorporate antagonists with a wide host range, such as Nectria inventa (Tsuneda & Skoropad, 1980) and Trichoderma spp. (Vannacci & Harman, 1987). Trichoderma is also potentially useful in integrated control as it is comparatively resistant to a wide range of fungicides, including iprodione.

Sources of partial resistance to A. brassicae have been identified in rape (Tewari & Skoropad, 1976) and to A. brassicicola in Brussels sprouts and cauliflower, but all currently available cultivars appear to be susceptible (Domsch, 1957; Dueck & Degenhardt, 1975). Indeed, surveys of large numbers of Indian (Husain & Thakur, 1963) and European (Quak, 1956; Stankova, 1972; Grontoft, 1986) cultivars of rape seed have revealed very little variation in resistance. Petrie (1974) reports small differences in susceptibility among cultivars in field surveys in Canada and Brun et al. (1987) report variation in susceptibility to pod infection among European cultivars. Tests under controlled environmental conditions also reveal subtle differences between cultivars in components of partial resistance such as infection rate, latent period, lesion size and sporulating ability (Saharan & Kadian, 1983). Furthermore, tissue culture experiments have shown differences in susceptibility to toxins in

culture filtrates of A. brassicicola between secondary lines of B. napus (MacDonald & Ingram, 1986). The use of rapid cycling B. napus (M V MacDonald, pers. comm.) and the advent of biotechnology may allow resistance to Alternaria to be transferred to rape seed from potentially useful, but more distantly related species. These include Sinapis alba (Brun et al., 1987), Eruca sativa, which gives a hypersensitive response to infection (Conn & Tewari, 1986) and Camelina sativa and Capsella bursa-pastoris, which are not susceptible to A. brassicae (Conn et al., 1988).

The susceptibility to, and development of, Alternaria in UK cultivars are related to maturity and height of cultivars. Late maturing and tall cultivars tend to have less Alternaria (Sweet et al., 1989).

Rapeseed possesses a layer of epicuticular wax which confers water-repellency to leaves and pods. This limits both the retention of rain-splash droplets containing inoculum and the availability of standing water for germination of spores which do impact successfully (Tewari & Skoropad, 1976). Alternaria lesions are often associated with areas of the leaf where curvature has trapped run-off water. The leaves of cultivars with a thick epicuticular wax layer retain fewer water droplets so that the number of potential infection sites for A. brassicae is reduced. Artificial removal of wax from rape leaves by wiping leads to greater infection by Alternaria (Tewari & Skoropad, 1976), especially on cultivars with comparatively thick waxy coverings (Skoropad & Tewari, 1977). Older leaves, having lost their wax through weathering, are more susceptible to Alternaria.

Besides reducing the retention of inoculum, epicuticular wax appears to provide a barrier both to penetration by Alternaria and to the leaching of various substances from the leaf surface. Oilseed rape leaf leachates stimulate germination of A. brassicae conidia. Wiping leaves of waxy cultivars causes spores inoculated onto their surfaces to germinate more rapidly and to produce more germ-tubes (Conn & Tewari, 1989). In contrast, leachates from leaves of brown sarson (B. campestris L. var. dichotoma), and yellow sarson (B. campestris L. var. sarson) inhibit germination of A. brassicae spores (Sharma & Gupta, 1978a; Sharma et al.,

1985). Gupta et al. (1987) have shown that leaf washings of several Brassica species contain phenolics, the level of which is related to resistance to A. brassicae.

Post-penetrational development appears to be related to the nutritional status of the host and the accumulation of defence-related compounds. Cultivars with lower levels of sugars and soluble nitrogen in leaves tend to be more resistant to Alternaria (Chahal, 1986; Gupta et al., 1987). Several phytoalexins have recently been identified in the Brassicae, of which at least two, cyclobrassinin and methoxybrassinin, are toxic to A. brassicae in vitro (Dahiya & Rimmer, 1989). Susceptibility to A. brassicae among rape seed and other crucifers corresponds well to the type and amount of phytoalexins they are capable of producing (Conn et al., 1988). Brassica phytoalexins are thought to be biosynthetically related to glucosinolates which accumulate in rape seed inoculated with A. brassicae (Doughty et al., 1991).

There is an extensive literature on Alternaria spp. in oilseed rape, more so than for any other disease. The reasons are obvious. The disease is of worldwide distribution and is of importance wherever it occurs. Losses can be high, the lack of genetic resistance in all current cultivars makes the disease a potential problem in all major rape growing areas. Control is heavily dependent on fungicides. A forecasting system is, therefore, required to assess appropriate disease levels and conditions for spread in order to establish criteria for economic control.

OTHER IMPORTANT DISEASES

SCLEROTINIA STEM ROT

Sclerotinia disease is caused by the fungus Sclerotinia sclerotiorum (Lib.) de Bary. Sclerotinia has been found affecting oilseed rape throughout the UK. However, its incidence in surveys is low (Jellis et al., 1984) and severe levels of infection are rare. Small areas around Chichester in West Sussex and Romney Marsh in Kent are the only recorded areas where consistent and severe infections occurs. The disease was more widespread in 1991. Sclerotinia stem rot is a major problem of oilseed rape in parts of France and Germany. In France losses have been estimated at 0.076 t/ha/annum over a 10 year period (Regnault & Pierre, 1984). By contrast, in the UK there have been few severe attacks of S. sclerotiorum recorded (Jellis et al., 1984) (Tables 5 & 8). In Germany, fungicide treatments gave consistent and worthwhile yield responses in trials where more than 25 per cent of plants were affected in untreated plots (Kruger & Stoltenberg, 1983). A similar situation has been found in the UK (J M Ll Davies, pers. comm.). A large return of sclerotia to the soil may, however, present difficulties if susceptible crops are planted in the future in the same field. Where high value crops are grown, such as peas or potatoes, oilseed rape may be the only crop in the rotation where control of S. sclerotiorum can be achieved.

Symptoms

With high soil infestations of sclerotia, infection can occur in the autumn where the disease can cause seedling death. Young plants are killed as mycelium arising directly from sclerotia spreads through the soil. With early summer infection fawn, or bleached lesions develop on the stem. The edges of the lesion may be sharply delineated by a dark margin. The lesions girdle the stem and extend many centimetres up and down frequently from the point of attachment of a petiole. In humid conditions, a white mycelium can develop on the outside of the lesion. Within the stem cavity of the infected area white, firm and later black hard oval to round sclerotia are formed. The girdling of the stem

frequently leads to premature ripening and prematurely senescing plants may be the first indication of this disease when the crop is viewed as a whole. Lodging or breaking of the stem at the site of the lesion may frequently occur.

Where the fungus has invaded a petal lodged on a leaf or petiole, a small greyish-beige spot can develop which develops a white mycelial mat.

Epidemiology

The sclerotia of S. sclerotiorum can persist in soil for many years and this is usually the primary source of inoculum. Sclerotia may also be introduced into fields with seed even after commercial cleaning (Hims, 1979a). They may also be present at or near field boundaries where wild plants particularly members of Umbelliferae can act as hosts (Hims, 1979b). A study of severe outbreaks of S. sclerotiorum in the UK demonstrated that in all instances the greater the number of susceptible hosts in the rotation (eg peas, spring [Vicia] and dwarf [Phaseolus] beans, potatoes) in the ten years prior to oilseed rape, the greater the disease severity (Jellis et al., 1984). Disconcertingly, in several crops surveyed, where peas had been the previous susceptible host, symptoms were not always observed in these crops.

In late spring, sclerotia within the surface layers of soil (upper 2.5 cm) germinate and develop apothecia above soil level. Apothecial development has been recorded in England from mid-April to July (Gladders et al., 1990), peaking between 2 and 8 weeks after the first apothecium was detected. From these fruiting bodies, ascospores are released in large numbers. These are spread in air currents to rape plants where they invade dead or dying tissues, usually fallen petals which have been shed and landed on leaves, stems and petioles (Lamarque, 1983). These tissues are colonised and from this food base mycelium invades host tissues. In field trials, symptoms of infection were first noted 9-15 days after artificial inoculation (Regnault & Pierre, 1984). From the point of infection, mycelium spreads into and along the stem forming the bleached areas diagnostic of the disease. Within the cavity of the stem lesion sclerotia form, initially white but turning black and hard. Infected

plants ripen prematurely and may lodge or break at the stem lesion. Sclerotia fall to the ground or are incorporated into the soil when the crop residues are ploughed in.

Natural infection depends on a complex interaction between the presence and germination of sclerotia, crop development and weather conditions (Morrall & Dueck, 1983). Apothecial development is dependent on the temperature and soil moisture content in spring, moist soil and a temperature of 6-10°C being required in early spring (Kruger, 1984). If the soil dries out, apothecial development is hindered. If the dry weather occurs later in the season, in May or June, then apothecia which have already developed shrivel and release few, if any spores. Ascospore release requires dry and slightly windy conditions. Apothecia production and spore release coinciding with flowering results in higher levels of ascospores on senescing petals. Subsequently leaf wetness or high humidity (above 94 per cent) are required for ascospore germination and infection of petals (Brun et al., 1983; Lamarque, 1983). Germination can take place within 21 hours, but it is inhibited if the humidity is 84 per cent or less. Davies (1986a) considered that infection would be unlikely if high soil moisture for apothecia production, dry and slightly windy weather for ascospore release and subsequent periods of rain to give very high humidity did not occur. Mitchell & Wheeler (1990) showed that apothecia were produced more readily from sclerotia at 0-2 cm soil depth.

Mylchreest & Wheeler (1987) have developed a method for inducing apothecia from sclerotia from isolates obtained from 17 different hosts. This work suggested that a preconditioning period at 4°C, keeping them moist at 10°C and subjecting them to near-UV light at 22°C were the main requirements. This would appear to mirror some of the natural conditions identified as necessary for apothecial production (Kruger, 1984).

Recent studies have confirmed that when the peak of flowering coincides with the peak of ascospore release the likelihood of infection is increased (S J Pope, pers. comm.). A correlation was found between time of flowering and susceptibility of cultivars in particular seasons.

Levels of sclerotial infestation in fields cannot be accurately determined, although previous outbreaks of Sclerotinia in susceptible crops may give some guidance. Where peas and oilseed rape are grown in the same rotation, the examination of cleanings from pea seed can give an indication of infestation (Gladders et al., 1990). The distance which ascospores can be dispersed and remain viable is uncertain. Thus, the significance of inoculum sources outside a crop is not known although it is likely that sclerotia at the margins of fields have the potential to produce inoculum that can infect the crop adjacent to it (Hims, 1979b).

Control

Studies by Price & Colhoun (1975), subsequently confirmed by Jellis et al. (1984), have demonstrated that S. sclerotiorum exhibits no physiologic specialisation. That is, that there are no strains of S. sclerotiorium adapted more to oilseed rape than to other susceptible hosts. Because of the wide host range of S. sclerotiorum and its persistence in soil as sclerotia, susceptible crops should ideally be grown only every five or six years.

Where a crop is known to be infected, deep burial of infected straw and stubble should restrict development and production of apothecia which occurs only in the top layers of soil. Seed and machinery can be contaminated with sclerotia and spread inoculum to land destined for oilseed rape. These pathways of spread should be avoided by cleaning seed and machinery.

Field observations of infection of oilseed rape cultivars in naturally and artificially infested sites have shown highly variable cultivar reactions between and within trials. However, the incidence has been consistently higher on some cultivars than others (Mylchreest, 1984). Recent observations suggest this is may be attributed to earliness of flowering or shortness in stature rather than intrinsic resistance. Using a uniform inoculation technique, Scott (1984) has confirmed that there is no inherent cultivar resistance. However, Newman & Bailey (1987b) suggest that some Japanese selections do show resistance.

In Germany, calcium cyanamide has been used to restrict the development of apothecia. Applications of 0.5-0.6 t/ha at the end of winter are as effective as an application when the plants are 30 cm or more high (Kruger, 1984). This treatment is not used on a commercial scale in the UK.

In the greenhouse, using artificial inoculation, Regnault & Pierre (1984) demonstrated that benomyl, iprodione, prochloraz plus carbendazim, procymidone and vinclozolin were all effective when applied protectively. When tested curatively, up to seven days after inoculation only procymidone and vinclozolin proved effective. In field trials, similar effects were demonstrated although the curative effects were less clear cut. For effective fungicide control, preventative treatments are most appropriate. Since colonisation of fallen petals by S. sclerotiorum is the most usual mode of entry, fungicide treatments are timed so that as many petals as possible receive fungicide. Optimal timing is, therefore, mid-flowering. In ADAS trials, whilst a range of fungicides applied at the optimal timing can reduce incidence of infection by 45-80 per cent, thiophanate-methyl and vinclozolin provided the best control and some flexibility in timing (D D Slawson, pers. comm.) (Fig 7). Yield responses did not reflect control of Sclerotinia as high levels of infection are required before yield losses are recorded and the fungicides used also influenced the development of other diseases.

Thwin & Mitchell (1990) showed that seed treatments applied to rape seed to control seed-borne pathogens inhibited mycelial germination of sclerotia. Fenpropimorph, present in the commonly used product Lindex Plus FS, was the most effective fungicide tested. Fenpropimorph also decreased the production of apothecia. They suggested that the use of Lindex Plus FS as a seed treatment may directly affect the development of epidemics of sclerotinia.

Predictive schemes have been evaluated in the UK and elsewhere. Such schemes are comprised of several components. For example: the occurrence of sclerotia in the field in previous years, the presence of apothecia during early flowering and a predictable sequence of weather. In some instances, 'depots' of sclerotia have been used to monitor the production

of apothecia as a component of fungicide spray warning schemes (Gladders et al., 1990). Because environment within a crop is difficult to measure or forecast, predictive schemes have used disease risk criteria or scores. One such scheme developed by ADAS in England and Wales achieved limited success and required further refinement (D D Slawson, pers. comm.).

GREY MOULD

Grey mould is caused by the ubiquitous fungus Botrytis cinerea Pers. (teleomorph: Botryotinia fuckeliana [de Bary] Whetzel). The stem rot caused by B. cinerea is usually a minor problem (Gladders, 1984) (Table 5). The disease rarely affects more than one per cent of pods (Gladders, 1988) (Table 6), although in eastern Scotland severe pod infection has been recorded in wet seasons (eg 1985, T Brokenshire, pers. comm.) leading to discoloration of harvested seed. Infection of pods can also be increased in crops damaged by pod pests such as seed weevil or brassica pod midge (P Gladders, pers. comm.) and infection of stems, leaves and petioles may be increased after hail damage (N V Hardwick, unpublished) frost, fertiliser scorch or wheeling damage (Anon., 1984a). In wet springs, the primary flower buds have been scorched by lodged fertiliser prills during the top-dressing process, the whole of the primary raceme may be killed by a progressive rot cause by B. cinerea. Because B. cinerea is usually part of a disease complex, estimates of yield loss have not been possible.

Symptoms

On leaves, the fungus produces grey to fawn spots, irregular in outline and frequently developing beneath colonised petals or pollen. On stems and petioles, pale, elongated lesions are produced which may girdle the plant stem or petiole developing a grey fawn mat of sporangiophores over the lesion surface. Margins of lesions are clearly demarcated. Where stems or branches are girdled, upper parts senesce and ripen prematurely. Pod infection produces pale lesions extending from the point of petal attachment along the length of the pod. Grey mould may develop on lesions and pods may split along the seam from the point of infection and shed seed prematurely.

B. cinerea is an opportunistic facultative pathogen invading host tissue through a food base (eg petals or pollen) or wounds. It requires leaf wetness or high humidity for infection and is thus only of significance in wet seasons. Leaf and stem colonisation may occur where fallen petals

become colonised. Pod infection is frequently established after invasion of unshed petals clinging to the developing pod.

Control

The risk of infection can be minimised by reducing or avoiding damage by fertiliser or chemical scorch, mechanical injury or pest infestation.

Resistant cultivars are not available although higher levels of grey mould have been noted in some cultivars (N Fisher, pers. comm.).

Applications of fungicides to control Sclerotinia at mid-flowering or Alternaria at or after 95 per cent petal fall may provide some control of grey mould. The earlier timing is considered most effective by inhibiting colonisation of petals after shedding. Iprodione, prochloraz and vinclozolin are all effective against B. cinerea (Anon., 1984a; Marshall & Harris, 1984). MBC fungicides may also be effective where the fungus has not developed resistance to this group of fungicides.

BEET WESTERN YELLOWS VIRUS (BWYV)

Beet western yellows virus is widely distributed in oilseed rape crops and has been reported in surveys since 1976 (Rawlinson & Muthyalu, 1979), although Gilligan et al. (1980) claim the first published report of BWYV on oilseed rape (Brassica napus L.). The virus was first reported from the USA (Duffus, 1960). Surveys of 16 crops in Warwickshire (Walsh et al., 1989) found 13 to be infected with the virus with two crops where the level of infected plants was 25 and 85 per cent. ADAS surveys, covering c. 110 crops in England and Wales, for 1990 (Hardwick, unpublished) show the virus to be present in 98 per cent of rape crops examined with a mean of 73 per cent of plants infected (Tables 1 & 7).

Symptoms

Deep red discoloration of the margins of the older leaves of young plants can sometimes be evident in the early spring. A chlorotic mottling of the leaves of mature plants may be associated with infection with the virus. Some workers have reported slight stunting and premature ripening of affected plants but the generally high level of infection of crops with this virus and the lack of general comment on its incidence would, however, suggest that in most cases the virus is symptomless.

Epidemiology

The virus is a luteovirus consisting of isometric particles c. 26 nm in diameter (Duffus, 1972). Transmission is in the persistent manner by eight species of aphid, the most important being the peach-potato aphid (Myzus persicae L.). The virus is acquired from infected plants in five minutes, has a latent period of 12-24 hours, persists in the aphid for over 50 days and requires a feeding period of 10 minutes for transmission. Infection takes place in the rape crop in the autumn. The levels of infection will depend upon factors which affect vector activity and population development. Its relationship with sugar beet yellows caused by a closely related strain, beet mild yellowing virus (BMYV), is uncertain. Russell (1965) indicated that BMYV had a more limited host

range than BWYV and may be part of complex of related viruses comprising BWYV. Transmission of the virus from rape to beet is uncertain. Smith & Hinkes (1985) discussed the fact that beet-infecting strains of BWYV occur in the the USA and non-beet infecting strains in England. BMYV occurs in England but not in the USA and Smith & Hinkes (1985) were concerned that oilseed rape might be an overwintering host for the virus that caused virus yellows in sugar beet and also for its vector Myzus persicae. While they demonstrated transmission of BWYV from rape to sugar beet, the poor rate of transmission indicated little risk of significant movement of the virus. Smith (pers. comm.) has suggested that BMYV has changed in virulence since the widespread growing of oilseed rape so that some interaction with BWYV is occurring.

Control

Four years of published surveys have shown that while BWYV is widespread in most years in most areas, it usually reaches its greatest incidence in any year in the south and west (Hill et al., 1989). Smith & Hinkes (1985) showed that seed yields were 10 per cent higher and oil yields 13 per cent higher in plots with low levels of BWYV infection. Read & Hewson (1988) carried out trials at 48 sites betweeen 1985 and 1986, but only 16 sites were replicated experiments. Yield responses were positive but generally low and not always significant. They reported good control of aphids and reductions in virus levels. ADAS experiments (Hill et al., 1989) have shown that single sprays of a pyrethroid insecticide, particularly when applied early in the autumn (September) effectively prevented the build up of aphids in the crop and reduced the incidence of virus. However, the response to such virus control has been difficult to estimate, with significant yield increases obtained in only a proportion of trials (32 per cent where two sprays were applied and 24 per cent for single sprays). At some sites, yield responses were obtained despite low aphid and virus levels, and it is difficult to estimate the contribution to yield responses which arise as a result of pest control as a by-product of virus control. Hill et al. (1989) suggested that while it would probably be cost effective to apply autumn pyrethroid sprays for virus control alone in most years, it is more appropriate to consider control of pests (and incidentally virus) as part of an overall autumn strategy. By

contrast with former years, potentially damaging populations of cabbage stem flea beetle (CSFB) now appear to occur in most areas where oilseed rape is grown. Where early pyrethroid sprays are justified for control of this pest, virus control control would be an additional bonus. In the south, where early sprays for CSFB control may be less frequently warranted, virus incidence is generally higher and may itself justify control measures more often. Walsh et al. (1989) showed that single post emergence sprays of a synthetic pyrethroid insecticide could reduce virus transmission by 73 per cent. Details of the control of aphid vectors of BWYV are to be found in a review by Alford (1991). Walsh et al. (1989) demonstrated that sprays of insecticide increased yield but showed no correlation between yield and virus incidence. They indicated that further work was needed to determine the effect of BWYV on oilseed rape as even if yield reductions could not be demonstrated, its role as an over wintering reservoir of the virus poses a threat to lettuce, sugar beet and other horticultural and fodder Brassicae. Also, the close relationship between BWYV and BMYV and the importance of sugar beet and oilseed rape as alternating hosts for M. persicae, indicate that a potential problem exists if either virus produces strains which are adapted to infecting both sugar beet and oilseed rape.

OTHER VIRUS DISEASES

Two other virus diseases can be found on occasions in crops in most parts of the country, cauliflower mosaic virus (CaMV) and turnip mosaic virus (TuMV). While generally the diseases affect only a few plants in a crop, the incidence appears to be on the increase in the east-central part of England, with the occasional crop showing up to 25 per cent plant infection (J M Ll Davies, pers. comm.). CaMV and TuMV have also been found at high levels in a cultivar trial on the Yorkshire Wolds (Hardwick, unpublished) and throughout the Midlands (C E Sansford, pers. comm.) in the spring of 1991. These two viruses and broccoli necrotic yellows virus (BNYV) were found in oilseed rape by Walsh & Tomlinson (1985), who recorded CaMV at a maximum incidence of 5.3 per cent from a single field from Warwickshire.

Symptoms

CaMV produces a chlorotic mottling and spotting of the leaves, dark brown to black streaks on the stems which may also show a thickening and twisting. Black streaks may occur on pods which may also be distorted. Where infection occurs early the affected plant may be stunted and distorted.

TuMV infected plants are generally severely stunted and may be accompanied by a distortion of the leaves and stem, leaf yellowing or a mosaic pattern, stem necrosis and pod distortion. The virus may be found in the presence of CaMV, when symptoms tend to be even more severe (Walsh & Tomlinson, 1985).

Epidemiology

Both viruses are transmitted by aphids in the non-persistent or semi-persistent manner being acquired and transmitted in less than two minutes without a latent period. Unusually however, once infective with CaMV, the aphid remains able to transmit virus for many hours, whereas with TuMV the aphid only remains infective for less than four hours. Over 30 species of aphid are capable of transmitting the viruses, principal amongst which are the cabbage aphid (Brevicoryne brassicae [Sulzer]) and the peach potato aphid (Myzus persicae [L.]). Plants and vectors can carry mixed infections of BWYV, CaMV and TuMV.

Walsh (1986) reported that seed yields of CaMV virus infected plant was 10 times less than from a virus free plant. Losses were due to reductions in seed numbers per plant and in thousand seed weight. The effects of TuMV were more variable but resulted in lower yields from affected plants.

Control

The incidence of these viruses has been too low to justify specific control measures. Breeding lines of oilseed rape immune to TuMV, but not to CaMV, are available (Walsh, 1986).

Currently the most important virus of oilseed rape is BWYV, if only because it is ubiquitous on oilseed rape. Oilseed rape is the major reservoir for the disease, where it apparently causes little damage. Its importance lies as a threat to other brassicas, where its effects, alone or in combination with other viruses or fungal diseases are of unknown but alarming potential. While common, the other virus diseases were of sporadic occurrence and have generally been of little consequence nationally. However, the recorded high levels of infection in rape crops of CaMV and TuMV in 1991 give cause for concern as these two viruses are capable of causing significant yield loss.

MINOR DISEASES

Minor diseases are so called because their incidence and severity is generally low. Where localised or wider outbreaks do occur, they appear to have little economic damage. However, with minor diseases of oilseed rape this generally means there has been little work on diseases/yield loss relationships and data in the literature are generally restricted to records of occurrence and symptoms.

CLUB ROOT

Club root is caused by the fungus Plasmodiophora brassicae Woronin. The disease is relatively common on horticultural and fodder brassica crops. It is surprising, therefore, that in an analysis of outbreaks in oilseed rape in the six years prior to 1984 only 31 outbreaks were recorded in England, Wales and Scotland (Clarkson & Brokenshire, 1984). The outbreaks were associated with short rotations of brassicas, particularly the fodder type. It is likely that many more cases of slight infection occurred but remained undetected. Despite further increases in area since then, outbreaks of club root remain sporadic (N V Hardwick & S J Wale, unpublished). A number of the confirmed outbreaks were, however, so severe that whole or part of the crops were destroyed in the spring. However, observations suggest that oilseed rape plants can tolerate moderate levels of infection and still produce satisfactory crops provided soil moisture is not limiting.

Club root is found in brassica crops throughout the UK but in Clarkson & Brokenshire's (1984) analysis of outbreaks most occurred in northern and western areas of Britain. In Scotland, of nearly 1400 fields tested for the presence of club root in 1981-1984, 32 per cent were contaminated, demonstrating the widespread occurrence of the pathogen.

The first sign of infection is often wilting of foliage, especially during dry weather. Affected plants are frequently stunted and develop a red

coloration of older leaves. Large irregular swellings or galls occur on the roots, particularly the tap root.

P. brassicae survives in soil as thick-walled resting spores for more than 10 years. The spores are stimulated to germinate by root exudates from a host and zoospores swim towards and infect the roots. Resting spores form in the galls and are released back into the soil when the roots decay. In controlled environment experiments, Williamson (1989) showed that galling could occur with a mean daily temperature of 7°C. In a polyethylene tunnel, however, galling only developed when the mean daily temperature was above 11°C. She also found that day length was a limiting factor, galling developing only when day length was greater than 11 hours. These observations could explain the low incidence of club root in field crops. Crops are sown in the second half of August and early September when soil temperatures are declining. Plants may escape infection where soil temperatures and day length in the autumn (particularly September - Williamson, 1989) fall below critical levels. Similarly crop development in spring is extensive before conditions suitable for infection occur. Observations on a number of infected crops in north-east Scotland in 1989/90 showed that severe infections occurred primarily on the earliest sown crops (S J Wale, unpublished).

All commercially available oilseed rape cultivars are highly susceptible to clubroot (Anon, 1984a; Clarkson & Brokenshire, 1984). Williamson (1989) found some variation in susceptibility in cultivars and suggested that selecting for non-specific resistance may be the most effective way of reducing susceptibility of oilseed rape to club root.

No chemical control is available and the only practical means of control are through crop rotation and avoidance by testing fields for presence of the club root fungus. Increasing the pH by liming and improvements to drainage will reduce the disease risk.

DAMPING-OFF

Soil-borne fungi, particularly Pythium spp. and Rhizoctonia solani, are sometimes associated with rotting of seed or the killing of seedlings soon after emergence.

Symptoms show when the stems or hypocotyl exhibit a brown water-soaked rot at soil level. Plants wilt and collapse ('damp-off'). Late attacks by Rhizoctonia solani, when conditions are not favourable to the fungus, may give rise to 'wirestem'. This is a common seed-bed disease of vegetable brassicas. The symptoms of wirestem are a browning and dry rotting of the stem base. Affected plants may lodge due to the weakened stems. The fungus has been implicated in a disease of the mature plant which causes premature ripening. This phase is discussed in more detail in the section on Rhizoctonia below.

Damping-off is favoured by cool wet conditions which do not allow for rapid germination of the seed and crop establishment. The loss of plants is seldom serious as most crops are drilled at sufficiently high seed rates for any losses to be insignificant.

RHIZOCTONIA

Rhizoctonia solani Kuhn (teleomorph: Thanatephorus cucumeris [Frank] Donk) has been isolated from the stem bases of oilseed rape showing symptoms of premature ripening. During flowering superficial canker-like lesions occur near the stem base. The characteristic dark fungal mycelium can be seen through a hand lens growing over the surface of the buff-coloured area of the lesion. This lesion is frequently bounded by a dark margin as in canker. Occasionally a ring of grey-brown fungal growth of the teliomorph is present at soil level. Incorporating chopped stem base material into soil-less compost and transplanting seedlings in to the inoculated compost produced symptoms of damping-off. The use of isolates of R. solani on agar medium failed to produce similar symptoms (Hardwick, unpublished). The symptoms in the mature plant are similar to those described by Sippell et al. (1985) where they report some 80-100 per cent

of plants infected by the disease in the Peace River region of Alberta, Canada, with reported yield losses in the region of up to 30 per cent. A similar figure was reported from a crop of oilseed rape in the UK in 1988 (D D Slawson, pers. comm.). Most of the data on Rhizoctonia on oilseed rape are of its occurrence in Canada (Berkenkamp & Vaartnou, 1972; Berkenkamp & Degenhardt, 1974; Hwang et al., 1986; Teo et al., 1988). There is no published information in the UK and even reports are few. Daebeler et al. (1985) reported the disease as being widespread in Germany in 1984, where in the later stages of infection cortical tissues of the stem base were destroyed.

The diameter of the hyphae of Rhizoctonia is 5-12 µm and they show the characteristic 90 degree branching of the fungus with a septum just beyond the branch. The fungus is a common soil inhabitant with a wide host range and survives in the soil as sclerotia and on weed species.

R. solani is a group species, that is there is evidence that it exists in a number of genetically different forms. This has significance for plant breeders who must determine the composition of isolates with which to challenge new lines. R. solani isolates can be grouped according to their ability to fuse with one another. These have been called anastomosis groups (AGs). There are currently about 12 AGs with AG 1 and AG 2 further divided into two sub-groups. Commonly AG 3 occurs on potatoes and AG 1 on cereals. AGs have been reviewed by Anderson (1982) where he concluded that it is important for scientists to consider each AG as an individual unit in order to make progress in devising control methods. AG 2 and AG 4 were found to predominate in oilseed rape (Hwang et al., 1986) with AG 2 being more pathogenic than AG 4. Yitbarek et al. (1988) showed that AG 2-1 was more active at low temperatures and AG 4 at higher temperatures. This fits with findings that AG 2-1 is mainly responsible for seedling blights and AG 4 is isolated from mature plants (Teo et al., 1988). Control of the seedling phase of the disease was effected by the by use of seed treatments (Kataria & Verma, 1989).

Further investigation is required to determine the cause of premature death of oilseed rape in the UK. From the Canadian evidence it is

possible to surmise that one of the possible causes is due to infection by R. solani.

PHYTOPHTHORA ROOT ROT

Root rot caused by the soil-borne fungus Phytophthora megasperma (Drechsl.) occasionally causes problems in oilseed rape. Details of the fungus are given by Waterhouse & Waterson (1966).

Affected plants frequently occur in patches, are stunted and generally found to be wilting. The first indications of infection may be premature ripening in late June/early July. Examination will show a rot to the roots and stem base. The outer layers of tissue may have sloughed off.

The disease is favoured by wet, compacted soil conditions. These are frequently to be found on headlands and low lying areas of the field where water is likely to accumulate. The fungus is particularly active in the temperature range 16-25°C. Control is by cultural methods only.

POWDERY MILDEW

This disease caused by Erysiphe cruciferarum (Opiz ex Junell) varies considerably in incidence from season to season (Tables 3, 4 & 6). It can be found in crops early in the autumn, is generally absent from crops in the spring but is resurgent following flowering. It is considered to be rarely damaging.

Powdery mildew occurs on stems, leaves and pods. On leaves it is generally to be found on both the upper and lower surface where it exhibits the typical white granular and fluffy growth of a powdery mildew. Unlike downy mildew, there is no corresponding yellowing of the upper leaf surface. A fine black speckling may be produced on leaves, stems and pods by the plant in response to infection. In late infections, black cleistotheia may be apparent. Under summer conditions, post flowering,

lesions may spread rapidly to cover the entire plant producing a grey-white bloom.

Conidia are barrel-shaped and produced singly or in short chains, with dimensions of 30-40 x 12-16 µm. The acscospores are oval, 19-22 x 11-13 µm. The asci are contained in thick walled cleistothecia, 95-125 µm in diameter; these develop late in the season and become dark brown to black with age. They are to be found scattered within the mycelial bloom.

Infection is favoured by warm (17-20°C) humid weather. Sources of infection are wind dispersed conidia from volunteer rape plants, which frequently carry the disease in the autumn, and other nearby susceptible and infected brassica crops. Severe infections can produce very high levels of spores at harvest, creating physical conditions which impede harvest.

Crops sown before 20 August often show extensive powdery mildew infection during September/October, whereas late August sowings are scarcely affected. General epidemics which affect stems or pods occur in hot dry summers (1976, 1981, 1989, 1990) but are thought to have little effect on yield because the main development is late in the season (July) after pods have formed. Early sown crops tend to be affected first, presumably because of overwintering inoculum. Powdery mildew is also common and occasionally very severe in spring rape.

Cultivars appear to vary in their susceptibility but further work is required in order to establish differences with any degree of consistency.

Control measures have been restricted to late applications of fungicides effective against the powdery mildews but with limited effect. Current data are limited to observations on the effects of standard fungicide treatments applied primarily to control other diseases. At a single site in Nottinghamshire in 1990, an ADAS experiment showed that prochloraz applied at stem extension reduced the area of pod infection from 13.2 per cent to 0.3 per cent on the cultivar Cobra. Similar reactions were obtained for the cultivars Tapidor and Libravo. Yield was not affected by reductions in disease (C E Sansford, pers. comm).

DOWNY MILDEW

Downy mildew (Peronospora parasitica Pers Fr.) has been the most frequently recorded fungal disease of winter oilseed rape (Brassica napus ssp. oleifera) in the UK (Brokenshire & Prasanna, 1984; Evans et al., 1984; Gladders, 1987). In recent years, however it has been replaced by light leaf spot (Hardwick et al., 1989; Hardwick, unpublished) (Tables 1, 3, 4, 6, 8 & 9). Cultivars are given scores for resistance to downy mildew in the National Institute of Agricultural Botany lists of recommended cultivars because some cultivars are very susceptible at the seedling stage (Anon., 1987, 1988). However, the disease is not thought to limit yield except, rarely, when seedlings are killed. If F1 hybrid cultivars of oilseed rape are introduced, downy mildew may become a more important problem, by analogy with the situation in vegetable brassicas in the UK (J B Sweet, unpublished). Moreover, if winters in the UK became milder and wetter as a result of the predicted climate change (Parry et al., 1989), it would also favour downy mildew. Completely reliable information on crop losses by downy mildew is not available.

Symptoms of disease appear on all above-ground plant parts, particularly on leaves and inflorescences. Small angular translucent light green lesions may first appear on the cotyledons or first leaves at the seedling stage a few days after sowing. Such lesions later enlarge and develop into greyish-white, irregular necrotic patches on the leaf, bearing downy growth of the fungus (conidia and conidiophores) on its under-surface. The presence of the disease is often first noticed by the yellowing of the upper leaf surface. In a severe attack, the affected leaf dries up and shrivels. Often the downy mildew symptoms are restricted to cotyledons or first true leaves, and leaves subsequently produced on the same plant do not show the symptoms. However, small necrotic lesions bearing the downy growth of the fungus may appear on well-developed pods of plants which are otherwise healthy quite late in the season (Kolte, 1985).

The causal fungus is an obligate parasite. Presently a single species, P. parasitica, is recognised as affecting all crucifers but isolates from different species are most virulent on their species of origin (Sherriff & Lucas, 1990). The fungus infects from conidia by forming a germ tube with

an appressorium; an infection peg penetrates directly through the anticlinal walls of the epidermal cells, in which the first haustoria are produced. Subsequently, the intercellular hyphae spread into the mesophyll tissues where large lobed haustoria are formed within cells. After the growth of the mycelium, erect branched conidiophores emerge vertically through the stomata singly or in groups. The oval conidia are hyaline, and measure 24-27 x 12-22 µm. A single conidium is borne at the tip of each branch. Detachment of conidia is possibly caused by twisting of the conidiophore related to changes in humidity. The resting spores (oospores) are produced mostly in the hypertrophic tissues. The spherical oogonia and tendril-like antheridia are produced on separate hyphae. Both the antheridium and oogonium are at first multinucleate. Nuclear division precedes fertilization and chromosome reduction occurs in the oogonium and in the antheridium. The oospores are globose and measure 26 to 45 µm in diameter and look yellow-brown enclosed in crest-like folds (Kolte, 1985).

The fungus survives as oospores in host debris in the soil and primary infections probably originate from this source. It is also reported to survive on alternative hosts (Kolte, 1985) although susceptible brassica crops are growing throughout the year. Conclusive experimental verification of host infection from oospores is not yet available. In localities where continuous cropping of brassicas ensures carry-over of inoculum, the oospore stage, while not necessary to initiate epidemics, may enhance variation in the pathogen population. Seed transmission of infection has also been suggested but is rare if it occurs at all (Lucas, 1988a). Primary infection might result in the development of systemically infected plants, but such plants are observed less frequently under natural conditions. As a result of primary infection in the seedling stage, the conidial growth of the fungus appears on cotyledons, which acts as a source for secondary infection. Usually the true leaves are infected through wind-borne conidia, resulting in spread of the disease through secondary infection. Dispersal over short distances in water droplets can also occur. There is no exact information on the relationship between leaf and floral infection. Under natural conditions, plants showing leaf infection may or may not show floral infection (Kolte, 1985). The disease is most prevalent under cool, moist conditions. The sexual cycle is extremely rapid and is completed in 3-4 days at 20°C and high relative

humidity. A temperature of around 15°C seems to be most favourable for epidemic development as this favours sporulation, germination and the infection process. Conidia survive for only a few days on leaves under typical field conditions, although at low temperatures in the absence of moisture they may remain viable for more than 100 days. The fungus may survive in a quiescent vegetative form within systemically infected plants (Lucas, 1988a).

The main requirement for chemical control is at the seedling stage. Traditionally, the dithiocarbamates have given satisfactory control but these have been largely superseded by other fungicides such as chlorothalonil and dichlofluanid. Several sprays are necessary to ensure good control. The phenylamide fungicides such as metalaxyl have given a good systemic control. Formulations of metalaxyl with dithiocarbamates have been recommended on rape at the establishment stage when disease is severe (Lucas, 1988a). A formulation of metalaxyl for application to seed is now commercially available. In two ADAS trials, (1982-83), no beneficial effects were obtained from autumn applied fungicides (metalaxyl + mancozeb). A combination of autumn and spring or spring treatments gave good disease control, but did not significantly increase yield (Evans et al., 1984). Similar results have been found in ADAS and SAC trial sites where downy mildew infection has been high and fungicides have been applied in November and March.

Host resistance is the most economical and environmentally acceptable method for disease control. Sources with major gene(s) for resistance have been identified (Lucas, 1988a; Nashaat & Rawlinson, 1990). Whether the gene(s) in these sources will prove to be of value in protecting oilseed rape from the disease depends on the frequency with which matching virulence occurs in the pathogen population. It is also possible that these resistance factors could be useful in programmes of integrated control if they were deployed together with fungicides, thus potentially prolonging the effectiveness of both control procedures (Crute et al., 1987).

Cultural control measures are usually aimed at reducing humidity and persistent moisture films in plants by reduction in planting density.

Removal of crop debris and a rotation with non-brassica crops may restrict sources of inoculum. The widespread cultivation of one or only a few rape cultivars may have favoured the disease (Lucas, 1988a; Kolte, 1985).

WHITE LEAF SPOT

White leaf spot on oilseed rape is caused by the fungus Pseudocercosporella capsellae (Ell. & Ev.) Deighton. In the UK, although the disease has been recorded in Scotland in the autumn, the disease is mostly restricted to crops in the south of England (Tables 4 & 9). Outbreaks have sometimes been severe in individual fields, but no general epidemics have occurred (Inman et al., 1991). If warmer, wetter winters occur in the UK as a result of predicted climate change, it is possible that white leaf spot may become more important.

In France, the disease decreased yields significantly when it spread on to the pods in 1985 (Penaud, 1986) and it has also been reported in Germany (Amelung & Daebler, 1988). P. capsellae has a worldwide distribution on brassicas in temperate climates (Anon., 1986) but most of the published information is descriptive and does not refer to the UK (eg Barbetti & Sivasithamparam, 1981; Penaud, 1986, 1987; Petrie & Vanterpool, 1978).

Symptoms on oilseed rape occur on all the aerial parts of the plant and may be found in autumn, spring and summer on the leaves. Lesions are round to angular, 5-10 mm in diameter, papery, white/beige to brown, and may be surrounded by a dark margin. Occasionally narrow line zonation may be seen within the lesion (Petrie & Vanterpool, 1978). They are visible on both sides of the leaf. Darkly pigmented stromatic knots may form in lesions in senescent leaf tissue. These are not sclerotial but are the initials of spermogonia and pseudothecia. Stem lesions are elongated and at first entirely brown, becoming scattered with white spots. The entire lesion is surrounded by a chestnut brown margin, and the demarcation between diseased and healthy tissue is discrete. Lesions may coalesce to form a continuous brown band scattered with white spots. Pod lesions are at first irregular in shape and chestnut brown to black in colour. They progress to become angular spots and the centres lighten from brown to

grey and eventually become white and bleached. Spermogonia and pseudothecia develop in mature stem and pod lesions. In severe infections a mosaic of spots may form as they coalesce in a similar way to those on the stem. In such cases spermogonia and pseudothecia are not seen in the brown areas but are restricted to the white areas of tissue (Inman et al., 1992).

Symptoms on cultivars of B. campestris appear similar to those on rape. Leaf lesions are typically bleached and papery with well defined margins. Dark stromatic knots are reported under a variety of synonymous terms (Miller & McWhorter, 1948; Campbell & Greathead, 1978). On oleaceous hosts, however, lesions are typically much smaller and darker (Miller & McWhorter, 1948; Campbell & Greathead, 1978).

P. capsellae is found on a variety of brassica crops, but has also been reported on a number of other cruciferous species (Crossan, 1954; Petrie & Vanterpool, 1978). Of the brassica crops, cultivars of Brassica campestris and Brassica napus are better hosts than those of Brassica oleracea (Crossan, 1954; Petrie & Vanterpool, 1978). There is little evidence for the existence of any physiological races, and isolates from one brassica host are commonly able to infect others (Crossan, 1954). The few cross-infection studies reported show that isolates from cultivars of B. campestris and B. oleracea readily infect cultivars belonging to either group (Campbell & Greathead, 1978; Miller & McWhorter, 1948).

Infection from germinating spores takes place through open or closed stomata. If stomata are closed, the germ tube enlarges to form an appressorium-like swelling at its tip, but there is apparently no direct penetration of the cuticle (Crossan, 1954). Growth is primarily intercellular, and there is evidence for the role of a toxin in causing cell necrosis (Petrie & Vanterpool, 1978). The toxin is a red pigment which can be readily produced in culture, is insoluble in water, and toxic to rape. It is identical in nature to cercosporin, a toxin common to many Cercospora species (Lynch & Geoghegan, 1977). P. capsellae is reported to possess two types of mycelium in host tissues: i) Hyaline hyphae, 2.5-5.0 µm wide, mostly intercellular, and typically found in young lesions and at the margins of older ones, and ii) dark-brown hyphae, 4-6 µm wide,

vacuolated, which commonly form stromatic mats below the plant epidermis in the older parts of lesions. Smooth, hyaline conidiophores emerge through stomates or are erumpent through the cuticle. Conidia are hyaline, cylindrical, smooth, straight or slightly curved, obtuse at the apex and with a truncate, thickened hilum, 1-7 septate, 30-90 x 2-3 µm depending on host, environment and age (Crossan, 1954). Spermogonia are reported in culture and in stems of Capsella bursa-pastoris (L.) Med., but are not described (Petrie & Vanterpool, 1978). A Mycosphaerella teleomorph for P. capsellae has now been reported (Inman et al., 1992). Spermatia, produced in spermogonia at the end of the season, infect the receptive hyphae and pseudothecia develop. In the autumn when these are mature, they release wind-borne ascospores which can infect the new growing crop, producing typical white leaf spot symptoms.

P. capsellae is widely accepted as being a splash dispersed pathogen and therefore favoured by high rainfall. This is reflected in its geographical distribution (Anon., 1986). Crossan (1954) first demonstrated the importance of water in its dispersal on irrigated turnip plots, and others have subsequently confirmed his observations. Fitt et al. (1989) have used the pathogen as a model system to investigate spore dispersal in splash droplets. Spores were dispersed up to one metre from infected leaves in still air and most spores were carried in largest droplets (diameter over 1 mm) although some spores were carried in droplets less than 200 µm in diameter, which might become air-borne in wind.

At 18-19°C, spore germination requires at least eight hours of 100 per cent relative humidity with free water on the leaf surface (Mestre, 1989) and all spore compartments are capable of producing a germ tube. Infection can occur at temperatures from 5-20°C. At 5°C, the length of the latent period between infection and the appearance of symptoms is 25 days, whereas it is eight days at 20°C. Sporulation on lesions requires high humidity and near ultraviolet light, but spores may be produced in less than 48h under favourable conditions. Thus, several disease cycles can occur during the season, with white leaf spot spreading from initial infections on lower leaves, which senesce and drop off, on to stems and pods under favourable conditions.

The teleomorph is likely to be the main mechanism by which this disease survives between oilseed rape crops. Pseudothecia are initiated at the end of the growing season and survive on crop debris. They become mature and release ascospores in early autumn when new crops are established. A gradient of white leaf spot has recently been observed in a crop of oilseed rape at Rothamsted, with decreasing amounts of disease with increasing distance up to 100 m from a field in which oilseed rape was grown in 1990 (A J Inman, pers. comm.); such a gradient provides good evidence for the spread of inoculum as air-borne ascospores.

Volunteer oilseed rape may also be a means of survival between crops. Wild cruciferous hosts and seed contamination are also considered potential sources of inoculum, although not thought to be important. Crossan (1954) failed to show seed transmission with turnip, but reports a four per cent contamination of commercial collard seed. Petrie & Vanterpool (1978) also considered seed transmission to be negligible, although they could culture the fungus from turnip seed. There are no reports of seed transmission on rape seed.

Due to the potential for transmission from infected crop debris, Crossan (1954) recommended deep ploughing and crop rotation as two means of reducing initial infection. Good chemical control using prochloraz or prochloraz plus carbendazim is well documented (Penaud, 1986) (Fig. 8). There are no recommended resistant cultivars in the UK, although limited data from NIAB trials have shown that cultivars differ in susceptibility. In France, the cultivar Darmour has shown some resistance to white leaf spot (Mestre, 1989). The cultivar Rafal, which was also considered resistant in France, developed a high incidence of systemic infection in NIAB trials in the 1980s, suggesting that environmental factors and or differences in virulence may be of importance in the development of the disease in the UK. The basis of resistance is not known.

RINGSPOT

Ringspot, caused by Mycosphaerella brassicicola (Duby) Lindau is a common, serious disease of vegetable Brassicas in the UK (Dring, 1961). While it

is generally rare on oilseed rape (Davies, 1986a), a few recent, but severe outbreaks in the south west (Tables 3 & 4) suggest that it may become more important in the UK (Parry, 1990) as in Germany (Zornbach, 1991). It has been recorded in Europe, Africa, North and South America and Australasia and is a widespread disease on Brassicas grown in cool moist areas of the world, especially being a problem in coastal regions (Anon, 1981; Dixon, 1981)

Symptoms of ringspot can develop on all aerial tissues of plants, although it is most commonly seen on leaves of oilseed rape (Parry, 1990). Ringspots start as small dark specks which enlarge into roughly circular lesions (up to 2 cm in diameter) with definite edges, often surrounded by chlorotic zones (McKay, 1956). The growth of lesions is often marked by the development of a series of concentric rings. When lesions are numerous, the whole leaf becomes yellowish with cracked and ragged edges and serious attacks can cause premature defoliation. The ring-like appearance of lesions on leaves is enhanced by the concentric development of spermagonia, then pseudothecia, both visible as black spots in the lesions. On stems and pods, lesions are elongated and irregular in size and shape (McKay, 1956). Early infections may encircle and kill pods whilst later pod infections may lead to infection of seed.

While M. brassicicola can infect a wide range of Brassica species and B. oleracea appears to be the main host (Weimer, 1926), there is no evidence for physiological specialization amongst M. brassicicola isolates (Dixon, 1981). Infection from germinating ascospores takes place through stomata (Dring, 1961). Hyphae enter the substomatal cavity and begin to spread between host cells. They do not enter living cells but a toxin produced by the fungus causes cell death and hyphae rapidly penetrate the dead cells. Typical lesions are produced in 2-3 weeks depending on the temperature (Nelson & Pound, 1959). Spermagonia develop after 3-4 weeks and produce spermatia which cannot germinate or infect plants (Snyder, 1946; Dring, 1961) but serve to fertilize the trichogynes on ascogonia, contained within protopseudothecia which then develop into pseudothecia with ascospores (Dring, 1961). Pseudothecia with ascospores can be produced at 8-20°C and infection by ascospores can occur over the temperature range 12-28°C. The optimum temperature is 16-20°C for both

processes, which also require continuous periods of 100 per cent relative humidity for several days (Nelson & Pound, 1959).

Wind-borne ascospores, which are the principal means of inoculum spread, may be produced throughout the year by this polycyclic pathogen (Zornbach, 1991) and periods of wet showery weather are associated with severe attacks on leaves in autumn and on pods in summer (McKay, 1956). The pathogen may survive between seasons on other Brassica crops (Zornbach, 1991), on oilseed rape debris, stubble and volunteers and on infected seeds (Parry, 1990).

Cultural measures are important for control of ringspot. Rotation and isolation of Brassica crops, and removal of crop debris and volunteers by ploughing should help to decrease amounts of inoculum at the start of the season (McKay 1956; Dixon, 1981). Fungicide sprays with maneb, mancozeb, benomyl or prochloraz can also control the disease and resistance to M. brassicicola has been reported in Roscoff cauliflower cultivars and some Brussels sprout lines (Dixon, 1981).

WHITE BLISTER (White Rust)

White blister, caused by Albugo candida (Pers. ex Chev.) Kuntze, is a common disease of cruciferous species in many countries and is widespread on Brassica oleracea (cabbage etc.) in the UK. While it does not yet occur on oilseed rape in the UK, serious yield losses from the disease have been reported on turnip-rape (B. campestris ssp. oleifera) (Petrie, 1973b; Harper & Pittman, 1974), mustard (B. juncea) (Kumari et al., 1970) and radish (Raphanus sativus) (Williams & Pound, 1963). Some lines of oilseed rape (B. napus spp. oleifera) from China (Fan et al., 1983) and elsewhere (Crute, pers. comm.) are susceptible to A. candida isolates from both B. oleracea and B. campestris. Since the pathogen can cause a phyllody of the floral parts so that infected plants yield no seeds, the effects on oilseed rape could be serious if a form of the pathogen attacking European B. napus cultivars emerged or if susceptible genotypes were selected.

Infected plants become covered with localized white, blister-like pustules on leaves and other tissues. Frequently, the disease causes distortion and abnormal pigmentation of affected tissues. It can also develop a systemic infection of meristem and inflorescences to produce galls known as 'stagheads' later in the growing season.

Physiological specialization of isolates of A. candida has been known for some time and isolates are classified on the basis of host specificity (Pound & Williams, 1963; Pidskalny & Rimmer, 1985; Petrie, 1988). Primary infections are by means of zoospores arising from oospores in soil or plant debris. Thereafter, spread is by means of wind-borne sporangia. On the surface of the host, the sporangia release zoospores, which encyst after 2-3 hours and initiate infection with germ tubes penetrating through stomata (Liu et al., 1989). The fungus, a biotroph, grows as intercellular mycelium and can form small spherical haustoria in palisade mesophyll cells adjacent to substomatal cavities of cotyledons after eight hours. It commonly occurs in close association with downy mildew Peronospora parasitica, which is often found parasitizing galls formed through infection by A. candida (Lucas, 1988b).

The disease can be controlled by preventative sprays of copper-based and dithiocarbamate fungicides, and phenylamides have good eradicant activity against foliar and systemic infections (Lucas, 1988b). Current resistance to A. candida in western oilseed rape cultivars appears to be conditioned by independent dominant genes at three loci, which may explain why it has not been overcome (Fan et al., 1983) and there are also other sources of resistance in Brassica species and Raphanus (Lucas, 1988b). This resistance appears to be expressed after the formation of the first haustorium by necrosis of invaded mesophyll cells (Liu et al., 1989).

VERTICILLIUM WILT

Wilt, caused by Verticillium dahliae Kleb, is considered the most serious disease of oilseed rape in Sweden, where widespread wilt has caused yield losses of up to 50 per cent since 1960 and has also decreased oil content and seed weight (Svensson & Lerenius, 1987). It is also widespread and

locally severe in Germany, especially where oilseed rape has been grown for long periods, or is frequent in rotations (Paul & Kruger, 1990) and occurs on oilseed rape in France (Brun, pers. comm.). The pathogen has been recorded in many temperate and tropical countries on other host species, but there are few records of its occurrence on oilseed rape. Although verticillium wilt has not yet been confirmed on oilseed rape in the UK, V. dahliae is a common soil-borne pathogen here with a very wide host range. Furthermore, it may have been the cause of the early death observed in oilseed rape crops in 1990 when the summer weather was very hot. V. dahliae certainly represents a serious potential threat to UK oilseed rape crops, since many of our single low and double low cultivars are susceptible (Baig, 1990; J Heale, pers. comm.).

While infection may occur early in the season, and V. dahliae may be detected by enzyme-linked immunosorbant assay (ELISA) in six week old inoculated plants (Gunzelmann et al., 1991), specific symptoms are not observed before flowering, when isolated plants in crops may show yellowing and later bronzing on one side of the leaves (Paul, 1988) or wilting symptoms (Paul & Kruger, 1990). Generally, no symptoms are observed until the crop begins to mature, when light brown longitudinal stripes can be seen firstly on the main stem and then on the lateral branches of affected plants, whilst other parts of plants remain green. These stripes extend above infected vessels, and the tissues between them collapse and shrivel so that stems become completely brown and plants ripen prematurely. At this stage it can be difficult to visually distinguish symptoms of Verticillium from those of Phoma on stems and roots (Kruger, 1987). However, infected plants can be pulled up more easily than those infected by Phoma because lateral roots of plants infected by Verticillium frequently decay, whereas those of plants infected by Phoma do not (Paul, 1988). Grey-black streaks can be seen on the infected main roots, which later become black as the fungus produces sub-epidermal microsclerotia. Many black microsclerotia also develop on the stem, especially after harvest, and the surface tissues of affected plants become dark grey. If stems are split open microsclerotia can be seen throughout the stem tissue, unlike the black pycindia of Phoma, which are present only in the outer cell layers.

V. dahliae is characterised by the hyaline, branched conidiophores and elliptical one-celled spores and can be differentiated from Verticillium albo-atrum by the formation of dark brown or black microsclerotia. There is no known sexual stage. V. dahliae has a very wide host range of annual and perennial species of wild and cultivated plants. There is little evidence of host specialization although isolates from one host can cause disease in some crops but only produce symptomless infections in others.

Mycelium from soil-borne microsclerotia is the principal inoculum; infection hyphae penetrate intact roots of young plants, or enter through wounds. The fungus grows inter- or intracellularly through the epidermis, cortex and endodermis to reach the xylem tissue without causing obvious root damage (Paul, 1988). Once the xylem is invaded, fungal growth is limited to the vessels until the surrounding tissues die. Conidia are produced in localized colonies within the vessels and are transported upwards to form new colonies. Although infection of winter oilseed rape may be initiated early in the autumn specific symptoms may not be observed until late spring the following year.

Verticillium wilt is considered to be a monocyclic disease, since inoculum rarely produces new inoculum that could be effective in the same growing period (Tjamos, 1988). Conidia are rarely produced on host surfaces, so contribute little to pathogen spread. V. dahliae can infect seed of oilseed rape although the importance of seed-borne inoculum is not known (Svensson & Lerenius, 1987). The principle source of inoculum, the microsclerotia, are incorporated into the soil after harvest. Microsclerotia can remain viable and infective for several years and severe Verticillium attacks have been reported on oilseed rape in Sweden on fields which had not grown susceptible crops for 8-10 years (Svensson & Lerenius, 1987). The optimum temperatures for growth of V. dahliae isolates and infection are generally in the range 21-27°C (Schnathorst, 1981), but V. dahliae can cause disease in areas where soil temperatures are lower. However, this may explain why verticillium wilt is not known to be a problem on oilseed rape in the UK, since summer soil temperatures are usually lower than the optimum range for the pathogen.

The principal available means for control of V. dahliae is crop rotation. The severity of verticillium wilt was much greater when oilseed rape was grown continuously than when there were four cereal crops between oilseed rape crops in Sweden (Svensson & Lerenius, 1987). Cultural practices such as the destruction of infected debris, volunteer oilseed rape and weeds between successive crops may also help to reduce the amounts of inoculum. There is no realistic method for control of verticillium wilt in oilseed rape by fungicides. Although glucosinolate degradation products can inhibit growth of V. dahliae in vitro and myrosinase activities were greater in incompatible than in compatible reactions between roots and V. dahliae in experiments in both the UK (Baig, 1990) and Germany (A Gunzelmann, pers. comm.), all the cultivars tested were susceptible to the pathogen.

SEED-BORNE PATHOGENS

Several of the pathogens which are common on the leaves, stems and pods of oilseed rape also occur on or in the seed. This is thought to be significant factor in the establishment of early epidemics of disease, eg alternaria. Claims have been made that seed treatments to control alternaria have resulted in a marked reduction of this disease in recent years (Maude & Humpherson-Jones, 1984).

The following diseases are considered to be seed-borne:-

Dark leaf and pod spot (Alternaria brassicae and Alternaria brassicicola) p. 34.

Downy mildew (Peronospora parasitica) p. 67.

Light leaf spot (Pyrenopeziza brassicae) p. 16.

Phoma leaf spot and canker (Leptosphaeria maculans) p. 27.

All the seed-borne pathogens can be controlled in their seed-borne phase by fungicide seed treatment.

Details of the effects of the seed-borne phases of these pathogens are given in the appropriate sections referred to above.

EFFECT OF DISEASE ON QUALITY

The effect of disease on oil content, oil yield and glucosinolate content of seed is not established. A number of trials have been carried out where fungicide treatments have been used to control disease. In some of these, quality has been significantly improved over untreated controls. Sweet & Beale (1991), in a series of cultivar trials, found that fungicide treatments increased the mean oil yield by more than expected from the increase in seed yield on all cultivars. Rawlinson et al. (1989) also found oil content and yield were increased with the use of fungicides. They also found that glucosinolate content of seed was significantly reduced in some cultivars by fungicide treatment but not by the use of insecticides. In their studies, Rawlinson et al. (1989) found that the incidence and severity of light leaf spot was positively correlated with glucosinolate content in seed of double low cultivars but this might be, in part, a dilution effect due to increased yield. Rawlinson et al. (1991) and Bock et al. (1991), using various combinations of autumn, spring and summer fungicide applications, found glucosinolate concentration in the seed was significantly less than the control in fully treated plots only. Other trials comparing fungicide programmes have shown variable or no significant effect on quality parameters (D Drummond, pers. comm.; S J Wale, unpublished). The difficulty in interpreting trials where fungicide treatments are compared to untreated controls is that it is not known whether the fungicide treatments themselves affect quality. Furthermore, treated and untreated plots are all harvested at the same time and no account is taken of differing maturities resulting from differential disease development.

FUTURE R&D PRIORITIES

Six main areas for further study have been identified. We have attempted to identify the appropriate source of funding. We recognise that not all areas are appropriate for HGCA alone. It is possible that joint funding between co-operating bodies would be a way of achieving a comprehensive and integrated programme of R&D. We feel that the HGCA research programme should have the objective of developing fully integrated, environmentally benign and economical disease control procedures. To achieve this, a better understanding is needed of the relationship between oilseed rape and its pathogens. Disease resistance should be fully exploited and integrated with strategies to minimise infection and control disease. We have suggested a project which can achieve some of these aims.

1. SURVEYS

The monitoring of disease incidence and severity in the UK is basic to the determination of priorities for research on diseases of oilseed rape. An annual survey in England and Wales is being supported by Policy Divisions of MAFF.

Further R&D is needed to:

a) determine the incidence and severity of diseases in other parts of the United Kingdom.

R&D Priorities

1) The survey should be extended to Scotland and Northern Ireland.

HIGH PRIORITY (MAFF/SOAFD/DANI/HGCA)

2. YIELD AND QUALITY

There are few data available on disease/yield loss relationships for any disease of oilseed rape.

Such data are needed for the three major diseases, namely dark leaf and pod spot, light leaf spot and phoma leaf spot and canker. Data are also needed to determine the effects of the widespread beet western yellows virus (BWYV) on yield, either alone or in possible synergy with light leaf spot and/or other pathogens.

Further R&D is needed to:

a) more readily identify BWYV and other viruses in the field in order to be able to determine effects of the viruses on yield.

b) develop formulae which relate disease incidence and severity to yield loss, as has been done for cereal diseases.

c) evaluate the effects of the disease on the growth of leaves, stems and pods at different developmental stages in relation to yield production and oil quality including glucosinolates.

R&D Priorities

1) Undertake studies on disease/yield loss relationships for light leaf spot, phoma leaf spot and canker, dark leaf and pod spot, BWYV and other viruses.

HIGH PRIORITY (MAFF/SOAFD/HGCA)

2) Determine possible synergy of BWYV and other viruses with other diseases.

MEDIUM PRIORITY (MAFF/HGCA)

3) Field identification and detection of BWYV and other viruses.

MEDIUM PRIORITY (MAFF/SOAFD)

3. EPIDEMIOLOGY AND DISEASE DEVELOPMENT

It is important to establish the significance of early infection, particularly for light leaf spot and phoma leaf spot and canker, in relation to host resistance and disease severity later in the season.

Further R&D is needed to:

a) develop immunological and molecular methods for early diagnosis of the presence of disease, the identification of pathogens and for the study of the biotrophic phases.

b) study the systemic development of pathogens in rape, the importance of latent biotrophic infections and the subsequent development of diseases in cultivars.

c) establish the sources of inoculum for epidemics and to determine, through spore dispersal studies, the relative importance of within-field splash-borne spores and external air-borne spores as inoculum.

d) study the progress of epidemics in detail for the major pathogens in order to identify which factors are crucial in determining the severity of epidemics and consequent yield loss.

e) study the effect of crop phenotype on disease development.

R&D Priorities

1) To undertake extensive studies of the epidemiology of the major pathogens, including environmental factors.

HIGH PRIORITY (MAFF/SOAFD/HGCA)

2) To devise and evaluate molecular diagnostic methods for studying the early and latent infection stages.

HIGH PRIORITY (MAFF/SOAFD/HGCA)

3) To develop strategies to minimise infection and disease development.

HIGH PRIORITY (MAFF/SOAFD/HGCA)

4. FORECASTING AND THRESHOLDS

Studies of the epidemiology of the diseases and the use of weather information and disease assessments in predicting the severity of epidemics need to be evaluated.

Threshold values of disease incidence and severity at a time when sprays can be applied need to be determined for the major diseases since current forecasting methods rely on subjective assessments.

Further R&D is needed to:

a) analyse weather criteria in relation to important phases in the establishment of disease epidemics (eg inoculum production and dispersal, infection and lesion development).

R&D Priorities

1) Devise and test model systems for the mathematical prediction of disease events.

HIGH PRIORITY (MAFF/HGCA)

2) Develop rapid diagnostic methods and early quantification of disease to facilitate forecasting for rational decision making and optimising inputs.

HIGH PRIORITY (MAFF/SOAFD/HGCA)

5. SPRAY TIMING/FUNGICIDE COMPARISON

The interaction between active ingredients of commercially available fungicides and their timing is necessary to provide optimum disease control.

Reductions in the need for excessive fungicide applications will ensure greater efficiency of production, while minimising the impact of agrochemicals on the environment.

Further R&D is needed to:

a) continue to determine the efficacy of new chemistry in fungicides on disease development.

b) monitor possible resistance problems in pathogens to fungicides.

R&D Priorities

1) Studies of disease development on cultivars under various environmental conditions and the relationship with optimum spray timing and dose.

HIGH PRIORITY (HGCA)

2) Undertake extensive site studies on the efficacy of active ingredients of commercially available fungicides.

MEDIUM PRIORITY (COMMERCE)

3) Survey of pathogens for fungicide resistance.

MEDIUM PRIORITY (MAFF/HGCA/COMMERCE)

4) Effect of disease and disease control measures on glucosinolates.

MEDIUM PRIORITY (HGCA/COMMERCE)

6. GENETIC RESISTANCE

Resistance is currently available in commercial cultivars for downy mildew, phoma leaf spot and canker, light leaf spot and some virus diseases.

Further R&D is needed to:

a) to establish the basis of disease resistance at all stages of development and also to establish the role of glucosinolate compounds in resistance.

b) determine if it is possible to reduce disease risk by alteration of the canopy structure to reduce or eliminate splash dispersed diseases or those associated with petals.

c) investigate conventional and novel means of introducing disease resistance.

d) investigate sources of resistance and variation in pathogenicity.

R&D Priorities

1) Investigate the basis of disease resistance.

HIGH PRIORITY (MAFF)

2) To identify possible sources resistance and their means of introduction.

HIGH PRIORITY (COMMERCE)

3) To study pathogen variability and its implications for the development of durable resistance.

HIGH PRIORITY (HGCA/COMMERCE)

4) To identify aspects of cultivar morphology which will reduce disease severity.

MEDIUM PRIORITY (HGCA/COMMERCE)

5) To study interactions between cultivar, disease, environment, fungicide, yield and quality.

MEDIUM PRIORITY (HGCA/COMMERCE)

A number of the above recommendations could be achieved by a single series of field experiments established across the UK. Using a range of sites would challenge the individual crop with a different disease spectrum and pressure. A standard fungicide spray programme applied sequentially over a range of development stages would manipulate the disease epidemic. Detailed disease assessment would establish disease/yield loss relationship for individual and a complex of diseases. Weather records taken at the sites would assist in the establishment of criteria for forecasting disease risk.

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APPENDIX

Tables 1-7 refer to England and Wales

Table 1

The incidence (% crops) of leaf diseases (stem extension)

Disease	1986	1987	1988	1989	1990
Alternaria	38	56	34	50	31
Downy mildew	83	94	77	75	34
Light leaf spot	75	74	85	85	71
Phoma	33	36	34	48	72
Ring spot	1	0	0	0	0
White leaf spot	2	4	3	21	6
BWYV	62	36	43	75	98

Table 2

The incidence (% crops) of stem diseases (pod ripening)

Disease	1986	1987	1988	1989	1990
Alternaria	0	24	15	14	2
Botrytis	24	21	33	18	7
Light leaf spot	94	83	74	98	68
Phoma	76	63	64	49	65
Sclerotinia	12	5	4	4	9

Table 3

The incidence (% crops) of pod diseases (pod ripening)

Disease	1986	1987	1988	1989	1990
Alternaria	21	82	75	56	45
Botrytis	16	36	26	9	61
Downy mildew	23	31	41	33	39
Light leaf spot	94	75	91	64	48
Powdery mildew	*	*	*	*	38
Phoma	0	3	1	12	2
Ring spot	0	2	0	8	2
White leaf spot	2	0	2	0	0

Table 4

The severity (% leaf area) of leaf diseases (stem extension)

Disease	1986	1987	1988	1989	1990
Alternaria	0.1	0.1	tr	tr	0.2
Downy mildew	1.0	2.2	0.8	0.3	0.1
Light leaf spot	1.1	0.8	1.8	0.6	1.2
Phoma	0.1	0.1	tr	tr	0.3
Powdery mildew	*	*	*	*	tr
Ring spot	tr	tr	tr	0	0.4
White leaf spot	tr	tr	0.6	0.1	tr
Total disease	2.3	3.2	3.2	1.0	2.2

tr = trace, generally less than 0.1 per cent

Table 5

The severity (index) of stem diseases (pod ripening)

Disease	1986	1987	1988	1989	1990
Botrytis	0.02	0.03	0.04	0.08	0.15
Canker	0.36	0.12	0.31	0.22	0.68
Sclerotinia	0.02	<0.01	0.01	0.01	0.10

Table 6

The severity (% pod area) of pod diseases (pod ripening)

Disease	1986	1987	1988	1989	1990
Alternaria	0.4	2.0	0.7	1.8	0.2
Botrytis	tr	0.5	0.1	0.2	1.1
Downy mildew	0.3	0.2	0.2	0.2	0.5
Light leaf spot	2.2	3.7	4.2	1.4	1.2
Ring spot	0	tr	0	0	tr
White leaf spot	tr	tr	0	tr	0
Powdery mildew	*	*	*	*	2.3
Total disease	2.9	6.4	5.2	3.6	5.3

Table 7

The Regional percentage plants infected with BWYV

Region	1986	1987	1988	1989	1990
Northern	11	7	4	49	78
Midlands and Western	23	9	14	25	86
Eastern	16	4	8	29	66
South Eastern	28	12	16	27	65
South Western	11	25	46	44	78
Wales	7	45	30	13	65
Mean	16	9	12	33	73

Table 8

Disease Severity* in Northern Ireland

Disease	1987		1988		1989	
(Number of crops)	(7)		(10)		(24)	
Development stage	se	ef	se	ef	se	ef
Light leaf spot	0.6	0	0.5	1.8	1.2	1.1
Alternaria	0	1.1	0	0.6	0	0.3
Canker	0.6	1.1	0	0	0	0
Botrytis	0	0.1	0	0	0	0
Downy mildew	0.3	0	0.8	0	0	0
Sclerotinia	0	0.6	0	0.9	0	0

* based on an index where 1 = slight, 2 = moderate and 3 = severe infection

Data supplied by the Department of Agriculture, Northern Ireland.

Table 9

Scotlands Adopt-A-Crop Survey 1989/90
Oilseed Rape

Timing	LLS		DM		WLS		ALTERNARIA		BOTRYTIS		No
	% crops	sev	% crops	sev	%	sev	% crops	sev	% crops	sev	Crops
Autumn	6	tr	44	0.6	3	tr	-	-	-	-	32
Stem Extension	76	1.3	8	tr	-	-	-	-	-	-	50
Flowering	68	1.0	25	0.3	-	-	-	-	-	-	68
Mid-pod	47	0.5	20	0.3	-	-	2	tr	43	0.5	83

% crops = % crops with symptoms

Severity = mean severity over all crops 0-4 scale

1 = trace 2 = slight 3 = mod 4 = severe

1990 was an exceptional year having a mild dry autumn and winter and warm dry spring and summer.

No Phoma or Sclerotinia recorded in any crop.

Data supplied by the Scottish Agricultural Colleges.

Table 10

Seed yield reductions (% of unwheeled crop) - mean of 3 years

Timing	Site					
	High Mowthorpe		Boxworth		Mean	
	Tract	High cl	Tract	High cl	Tract	High cl
Mid-flower	2.4	1.8	1.8	1.2	2.1	1.5
Post-flower	3.5	2.6	3.3	3.0	3.4	2.8
Desiccation	1.5	2.0	1.2	1.2	1.4	1.6
Post-flo + des	3.8	3.2	3.9	3.9	3.8	3.6

More yield was lost from the taller crops at High Mowthorpe in 1984 and Boxworth in 1985 (Ogilvy, 1989).

Table 11

ADAS National Trial Results - Evaluation of fungicide programmes (1986-90)

		Relative yield (profitability)*				
Early stem extension	End of flowering	1986 (5)	1987 (7)	1988 (5)	1989 (4)	1990 (4)
Sportak	no spray	106 (+£33)	104 (+£16)	106 (+£25)	103 (0)	101 (-£15)
Benlate (+ Agral)	no spray	103 (+£9)	101 (-£5)	101 (-£5)	105 (+£23)	103 (+£10)
Compass	no spray			111 (+£58)	105 (+£14)	101 (-£16)
No spray	Rovral			103 (+£3)	105 (+£14)	101 (-£16)

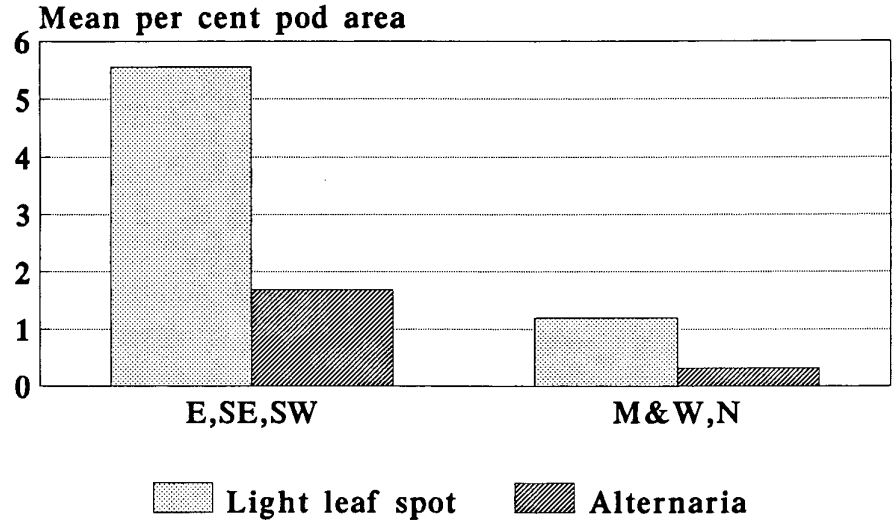
* Profitability - margin over cost (£/ha), based on 1990 prices. Does not include wheeling damage for end of flowering sprays (£22/ha).

Figures in brackets are the number of sites in each year.

Note - the most common cultivars used in these trials were Bienvenu, up to 1988, Cobra and Libravo in 1989 and 1990.

Fig. 1

Winter Oilseed Rape
Severity of disease 1986-1988

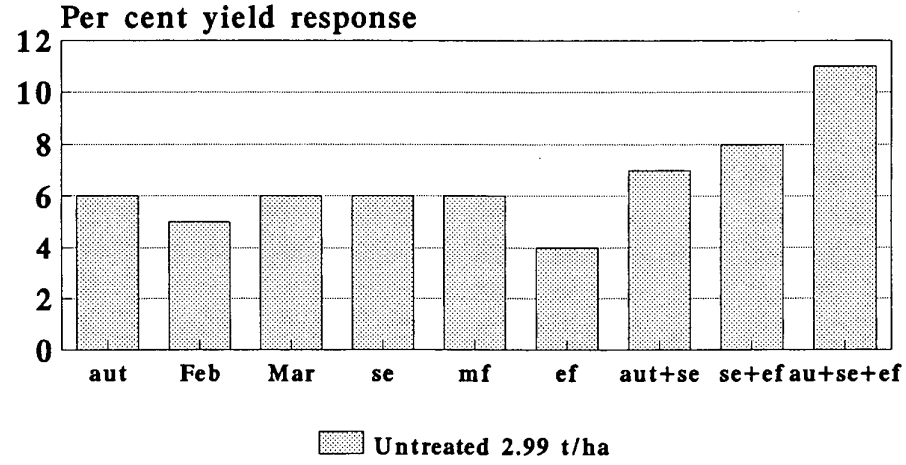


ADAS Regions
E=Eastern, SE=South East, SW=South West
M&W=Mildlands & Western, N=Northern

Hardwick *et al.* (1989)

Fig. 2

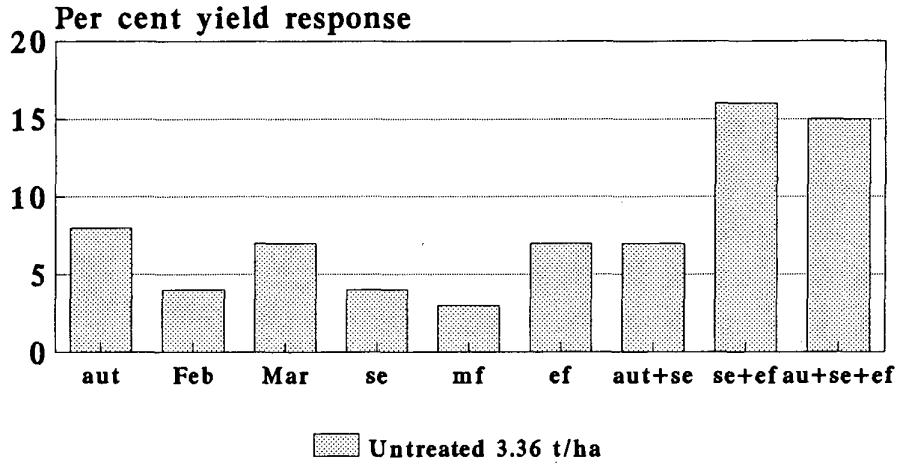
Winter Oilseed Rape
Fungicide programmes



ADAS (National), 1991
(aut - autumn, se - stem extension,
(ef - end of flowering)

Fig. 3

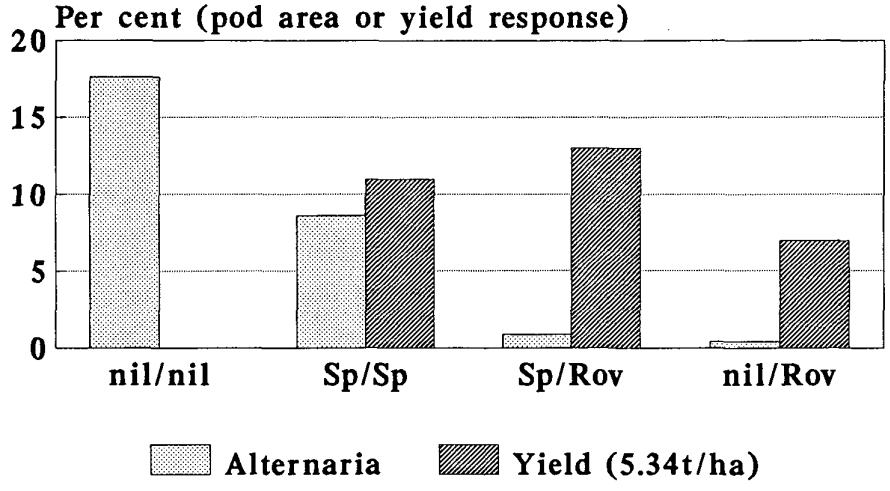
Winter Oilseed Rape
Fungicide programmes



ADAS (Wilts), 1991
(aut - autumn, se - stem extension,
(ef - end of flowering)

Fig. 4

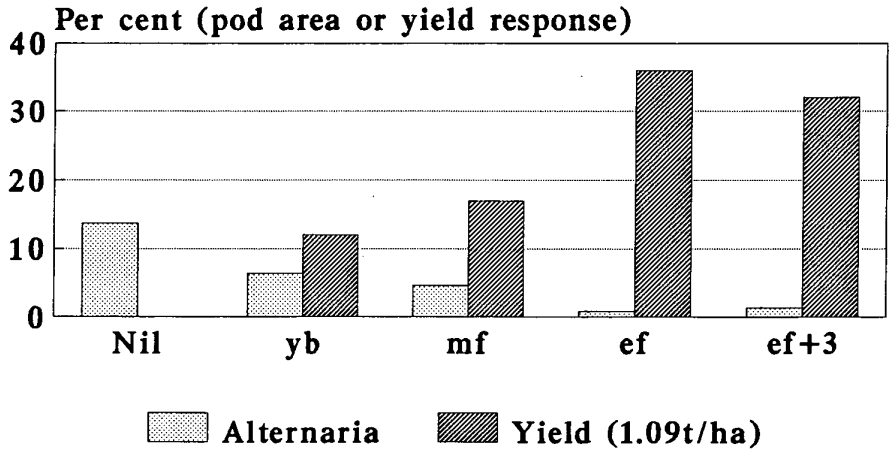
Winter Oilseed Rape
Control of alternaria



ADAS (Somerset), 1987
(Sp=Sportak, Rov=Rovral, stem extension/end of flowering)

Fig. 5

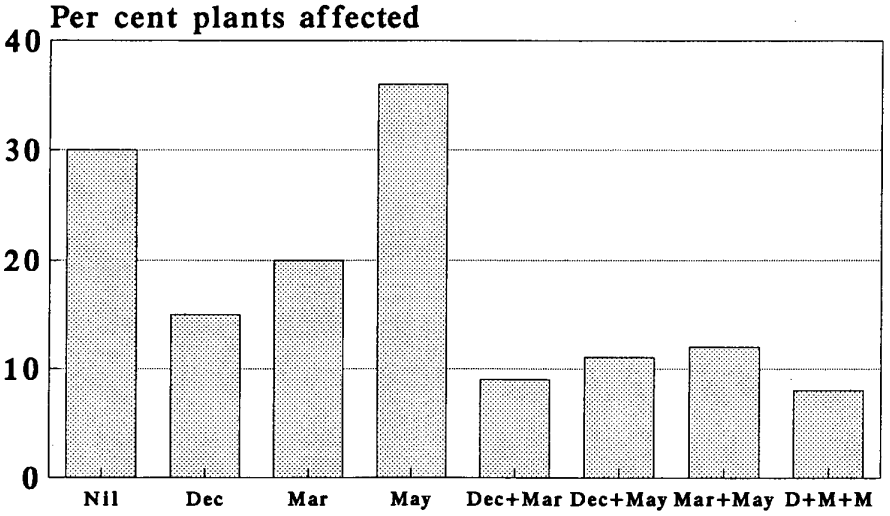
Winter Oilseed Rape
Spray timing for alternaria control



Ogilvy (1984)
(yb=yellow bud, mf=mid-flower,
(ef=end of flowering, +3=3 weeks later)

Fig. 6

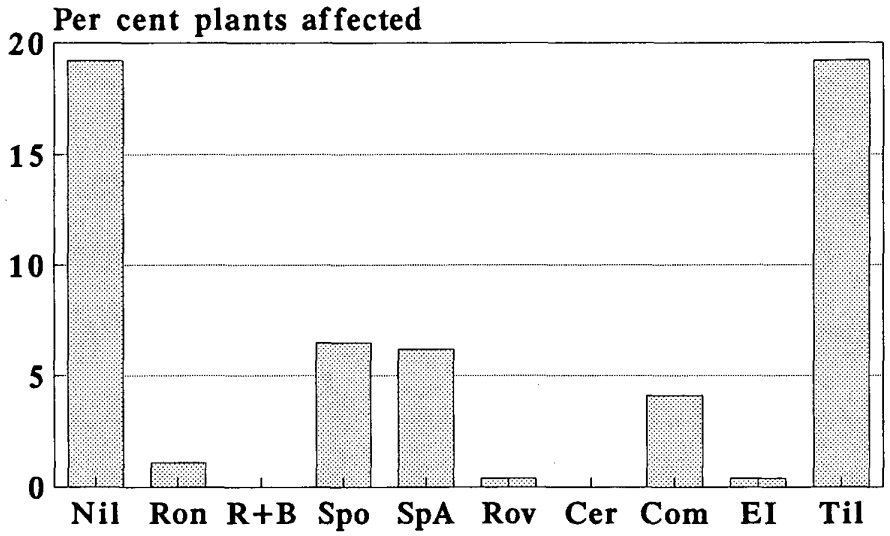
Winter Oilseed Rape
Control of canker



ADAS-Boxworth (1984)
(Sportak at 1.0 l/ha)

Fig. 7

Winter Oilseed Rape
Control of sclerotinia

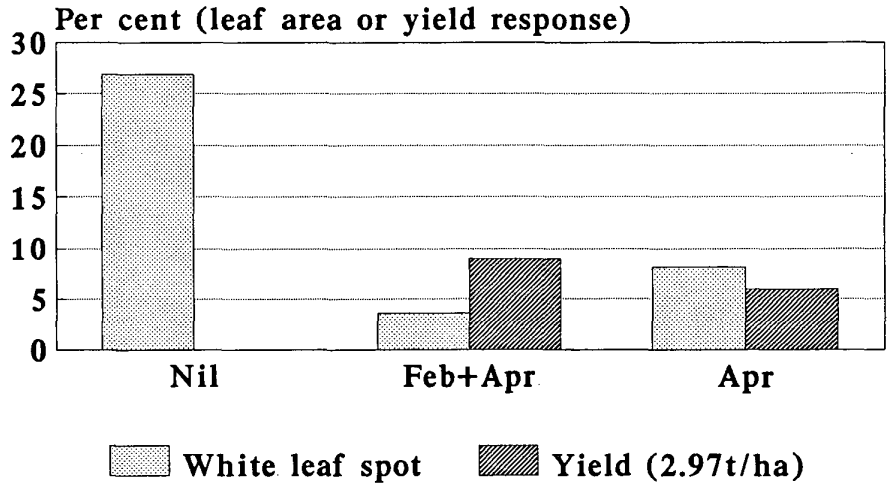


ADAS (Kent), 1990
(spray applied early petal fall)

Ron&R=Ronilan, B=Bavistin, Spo=Sportak
Rov=Rovral, SpA=Sportak Alpha, Cer=Cercobin
Com=Compass, EI=Early Impact, Til=Tilt

Fig. 8

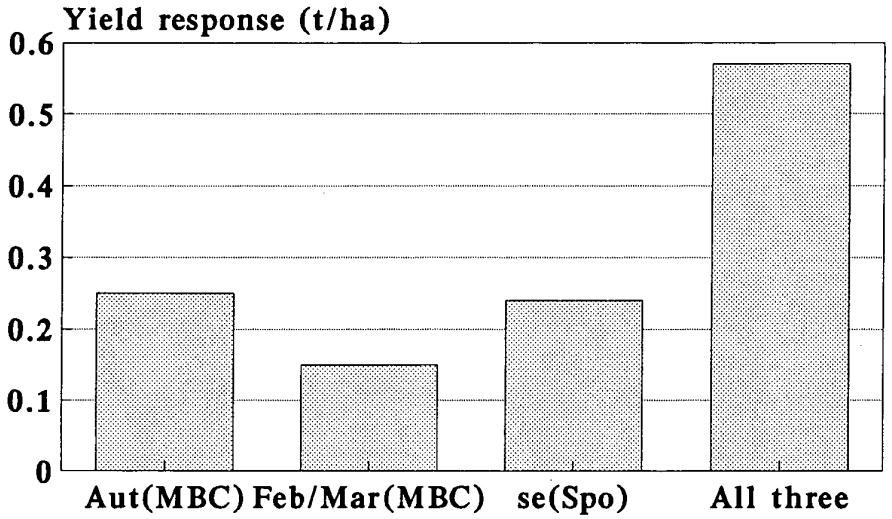
Winter Oilseed Rape
Control of white leaf spot



ADAS (Dorset), 1984
(Sportak, 1.25 l/ha)

Fig. 9

Winter Oilseed Rape
Light leaf spot control, Scotland 1984-9



Wale *et al.* (1990)
(se= stem extension, Spo=Sportak)