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Epidemiology in relation to control of white leaf spot *(Mycosphaerella capsellae)*

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INTRODUCTION

White leaf spot of oilseed rape and other crucifers has a world wide distribution but is found primarily in temperate countries. It has been of only minor importance as a disease of oilseed rape in Europe and Canada, although yield losses have occurred in France when the disease developed on the pods (Penaud, 1987).

Little is known about the pathogen (*Pseudocercosporella capsellae* (Ell. & Ev) Deighton) or the epidemiology of the disease. Previous studies have reported the occurrence of both a conidial and a spermatial stage, as well as stromatic structures considered to be sclerotial in character (Penaud, 1987). Conidia are splash-dispersed (Crossan, 1954; Fitt *et al*, 1991) and disease development is therefore dependant on rainfall. Spermogonia and a *Mycosphaerella* sexual stage have now been reported for the first time, on oilseed rape (Inman *et al*, 1991). Spermatia do not have an infective role, but are thought to be responsible for fertilizing the initials of the sexual stage (protoascomata). Survival by microsclerotia is now considered unlikely as they appear to be the primordia for spermogonia and ascomata. In the U.K. the pathogen survives between autumn-sown oilseed rape crops by the production of the sexual stage.

AIR-BORNE SPORES: THE SEXUAL CYCLE

Mycosphaerella capsellae Sp. Nov. Inman & Sivanesan has recently been shown to be the sexual stage of *P. capsellae*, and is distinct from *Mycosphaerella brassicicola* (Duby) Lindau which causes the ringspot disease of Brassicas. In the U.K. ascomata of *M. capsellae* are initiated in pod or stem lesions in late summer and can be found concurrently with spermogonia. In the two seasons studied at Rothamsted, ascospores were produced in the autumn during the period of active vegetative growth of the oilseed rape crop. Ascospores were present in ascomata sampled from infected pod debris as early as September and as late as the end of December. Ascospores were produced one month earlier in 1991 than in 1990. In both years ascomata did not over-winter. Studies of ascospore dispersal using Burkard volumetric air samplers placed over infected pod debris showed that ascospore discharge occurred in response to wetting by rain or heavy dew.

Evidence for the role of ascospores in white leaf spot epidemiology and as a source of primary inoculum was provided by a natural outbreak of the disease in 1990/91 at Rothamsted. Disease gradients of white leaf spot were observed in March 1991, with the disease decreasing in incidence with distance from the edge of an oilseed rape crop. Lesions were present on only the two lowest leaves (leaves 5-9 depending on individual plants) and were estimated to have developed from infections initiated between October and December 1990. The field had grown cereals in the two previous seasons and the source of inoculum appeared to be debris in an adjacent field which had grown oilseed rape in the previous season. White leaf spot incidence and severity were greatest at the edge nearest to this field and decreased with distance from it. The disease was still present at the far edge of the crop, approximately 200 m away. Gradients over such distances suggested that the primary inoculum was air-borne ascospores liberated in the autumn, as dispersal by splashed conidia is limited to short distances.

SPLASH-BORNE SPORES: THE ASEXUAL CYCLE

Disease development was studied during the spring of 1991. Two generations of lesions were produced after stem extension before the disease died out following a period of prolonged dry weather and abscission of infected leaves in May and June. Horizontal disease gradients remained essentially unchanged between March and May despite increases in disease incidence with the production of new generations of lesions. However, the disease did spread vertically. Two increases in the height of the disease resulting from the upward dispersal of conidia by rain-splash were identified:

1) From infected 5th-9th leaves to 9th-15th leaves:

This resulted in the disease progressing from the oldest, lowest leaves on to younger leaves above them. The resulting vertical disease gradient on the 24 April was such that only 5% of lesions were above a height of 23 cm. Mean plant height was 60 cm.

2) From 9th-13th leaves to 13th-16th leaves:

The vertical disease gradients on 15 May were such that only 5% of lesions were above a height of 46 cm. Mean plant height was 118 cm. As there was no increase in leaf height as a result of extension of leaf internodes below leaf number 16 between 24 April and 15 May, this vertical disease spread could be explained only by the vertical movement of conidia by rain-splash.

Vertical disease progress was halted by prolonged dry weather during May and June and by the loss of inoculum as infected leaves senesced and abscissed. The disease failed to become established on the pods and no lesions were found on a sample of 300 stems taken 2 weeks before harvest from an area where the disease incidence had been high (100% plants with leaf lesions). Locally, at least, the disease appeared to have died out.

IMPLICATIONS FOR CONTROL STRATEGIES

At present white leaf spot remains a minor disease of oilseed rape which rarely warrants specific control measures. However, it has become increasingly important in France within the last decade and it is possible that strategies for controlling it may be required in the U.K. in the future. Knowledge of both life and disease cycles is needed to develop such control strategies.

Chemical control:

Genetic recombination within and between pathogen populations during sexual reproduction provides the possibility for the rapid development of fungicide resistance. As a consequence prophylactic fungicide treatment is probably unwise and may quickly become ineffective against white leaf spot, as well as being enviromentally undesirable and uneconomic. The requirement, therefore, is for a control strategy which minimises the use of fungicides and times their application for maximum efficacy.

As the primary source of white leaf spot inoculum in the U.K. appears to be air-borne ascospores produced in the autumn, the application of a fungicide against autumn infections may decrease subsequent disease development. Such a control strategy could be readily incorporated in U.K. disease management programmes as autumn fungicides are often used on oilseed rape against other diseases. However, autumn applications of fungicides to control white leaf spot may not be cost effective since changes in subsidies have increased the disease threshholds at which the use of fungicides is worthwhile. Furthermore, economic yield loss is likely to occur only if white leaf spot reaches the pods, and in most years in the U.K. this does not happen. As the pod canopy is typically 1-1.5 m above ground level it may take 4-5 pathogen generations for the disease to reach the pods if the vertical progress of the disease is restricted to splash dispersal of conidia over vertical distances of 20-25 cm. The potential for internode extension to contribute to the vertical movement of the disease on young infected leaves has yet to be evaluated but this may decrease the number of generations needed for pod infections to occur. However, as disease progress can be halted rapidly by periods of dry weather, fungicide sprays should be used specifically for control of white leaf spot only if the disease becomes well established on the upper sessile leaves (bracts) during flowering and early pod development.

Cultural practices:

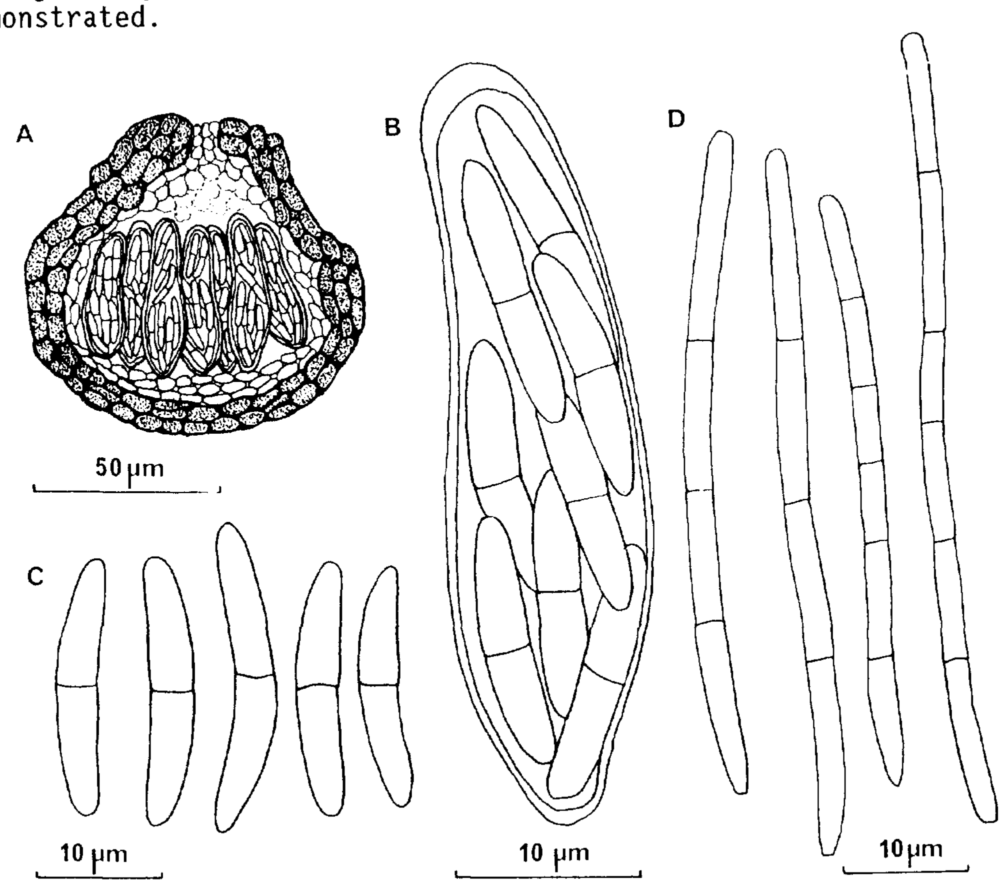
Three main cultural practices have potential for the control of white leaf spot:

1) Incorporation of crop debris after harvest decreases the amount of primary inoculum available for infecting autumn sown oilseed rape crops. The epidemic at Rothamsted occurred because debris from the previous season in the adjacent field had not been buried by ploughing. As a result a concentrated source of air-borne inoculum was present in the autumn to establish the disease on the adjacent new oilseed rape crop.

2) Late sowing may decrease early infection by providing a degree of disease escape. In 1991 most ascospores were dispersed between the beginning of September and the end of October. Plants in late-sown crops emerged only in the middle of September and had produced few, small leaves by December. The initial absence of plants and the small leaf area available for infection by air-borne ascospores later can therefore result in low infection rates. However, reduced crop establishment due to late sowing is likely to make this method unacceptable, except when much inoculum is expected in the autumn.

3) Host resistance is potentially the best way of managing the disease. At present, in breeding for disease resistance, white leaf spot has a low priority by comparison with other rape diseases and there are no approved resistant cultivars in the U.K. However, there do appear to be differences in cultivar susceptibility, although the basis for this is unknown. In France the cultivar Darmor is reported to be only moderately susceptible in both laboratory and field experiments. Results from Rothamsted using 9 test cultivars confirmed that differences in susceptibility occur under laboratory conditions and that Darmor is one of the least susceptible varieties.

In conclusion, although *M. capsellae* remains a minor pathogen of oilseeed rape, new knowledge of its life cycle enables prospective control strategies to be proposed. Air-borne ascospores dispersed in the autumn are the primary inoculum for infecting winter oilseeed rape in the U.K. and probably also in other European countries. In Canada, where only spring sown cultivars of canola are grown, stromatic mycelium of the type described by Petrie and Vanterpool (1978) may have a role in survival. Alternatively, it is possible that protoascomata may over-winter as a result of a shorter growing season and the more rapid onset winter than in Europe. However, the sexual stage has yet to be reported in Canada and such overwintering has not been demonstrated.



Mycosphaerella capsellae. A) Ascoma, B) ascus, C) ascospores, D) conidia.

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