White leaf spot (*Mycosphaerella capsellae*) on oilseed rape in the UK ?

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INTRODUCTION

In the UK white leaf spot on oilseed rape is mostly restricted to crops in the south and south west of England. Outbreaks have sometimes been severe in individual crops but no national epidemic has occurred. More recently the disease has been reported in the main oilseed rape growing areas of central and eastern counties and it is possible that the disease may become increasingly important. However, the potential for white leaf spot to become epidemic nationally and economically important in the the UK has not been assessed. In France the disease is now widespread and yield losses have been reported when the disease has spread from the leaves onto the pods.

Survival of white leaf spot (*Pseudocercosporella capsellae*) between oilseed rape crops has now been attributed to the production of a sexual stage, described as *Mycosphaerella capsellae* sp.nov. (Inman *et al.*, 1990; Inman *et al.*, 1992a), and air-borne ascospores have been shown to be the most likely source of primary inoculum for infecting autumn-sown crops of oilseed rape in the UK (Inman *et al.*, 1992c; Inman, 1993). Ascospores are produced only in the autumn and the sexual stage does not over-winter. Survival by means of "microsclerotia" (Penaud, 1986) is considered unlikely as these stromatic structures do not appear to be sclerotial in character. Rather they appear to be the primordia for spermogonia and ascomata. Even if conidia were produced from stromata previously considered to be "microsclerotia", they would be unlikely to be a source of primary inoculum for infecting distant crops of oilseed rape because they are dispersed only short distances by rain-splash (Fitt *et al.*, 1992).

After the disease is established in the autumn by air-borne ascospores, disease development is dependent on splash-dispersed conidia (Inman *et al.*, 1992a). Of particular importance is the progress of the disease up the crop canopy after the start of stem extension as this will determine whether the disease spreads onto the pods to decrease yields.

After the start of stem extension in the spring, white leaf spot has been shown to progress up the plant by a combination of two inter-dependent mechanisms (Inman *et al.*, 1992c). These are (1) the vertical splashing of conidia from infected lower leaves to infect young upper leaves and (2) the vertical movement of these infected upper leaves by internode growth. Because conidia are typically splashed no higher than 10-20 cm (Inman *et al.*, 1992c; Walklate *et al.*, 1989) vertical disease spread is dependent on young upper leaves remaining within the height to which conidia can be splashed from infected leaves below

them. Disease progress after the start of stem extension is therefore highly dependent on frequent rainfall. Even a single dry period of several weeks can allow young upper leaves to be moved by internode growth above the range of vertical splash, and disease progress is then halted. This not only prevents the disease spreading onto the pods to potentially decrease yield, but also has implications for survival which may be dependent on lesions being established on durable plant organs such as stems and pods.

Information about the life cycle and biology of *M. capsellae* and the development of white leaf spot epidemics in oilseed rape crops can be used to enable the potential for white leaf spot to become epidemic and economically important in the UK to be assessed.

POTENTIAL FOR WHITE LEAF SPOT TO BECOME EPIDEMIC IN THE UK

The potential for white leaf spot to become epidemic nationally in the UK has not been adequately assessed, although it has been suggested that the incidence and severity of the disease may increase if the trend for warmer wetter winters continues due to climate change (Anon., 1988). The disease is not new to the UK but has been recorded on turnips and swedes for most of this century (Anon., 1920-1973). It has never been a common disease of such fodder brassicas and its distribution has mostly been restricted to the south, south west and north of Britain (Anon., 1920-1973; Mylchreest, 1985). This distribution appears to reflect the distribution of rainfall in the UK (Fig.1; Chandler & Gregory, 1976) and possibly the concentration of fodder brassicas in these areas (Gladders *et al.*, 1984; Fig. 1).

The relative importance of rainfall and host distribution in determining the past distribution of white leaf spot in the UK is unclear. However, the development of oilseed rape in recent decades as a major UK crop, predominantly in central and eastern England (Gladders *et al.*, 1984; Fig.1), has given white leaf spot the potential to spread eastwards into areas where brassica crops had previously been relatively sparse. However, despite the increase in the area of oilseed rape and the potential for long distance dispersal by air-borne ascospores, the disease has not become widespread in UK in the last two decades. It would therefore seem that climatic factors, especially the amount and frequency of rainfall, may be more important in determining the geographical range of the disease than the density of brassica crops. Therefore, although white leaf spot epidemics are likely to continue to occur in crops in the wetter south west and south of England, the disease appears unlikely to develop in the main oilseed rape growing areas in drier central and eastern counties to become epidemic nationally.

Support for this hypothesis can be provided by information about the life cycle and biology *M. capsellae* and the epidemiology of white leaf spot. Comparisons with two other oilseed rape pathogens, *Leptosphaeria maculans* (*Phoma lingam*) and *Pyrenopeziza brassicae*, are also useful in this context. Although all three pathogens have splash-dispersed conidial stages, and are therefore theoretically favoured by the same climatic conditions, differences in biology and life cycle can help explain why *L. maculans* and *P. brassicae* are the major pathogens of oilseed rape in the UK, while *M. capsellae* is limited in distribution and importance (Hardwick *et al.*, 1989). The two most important

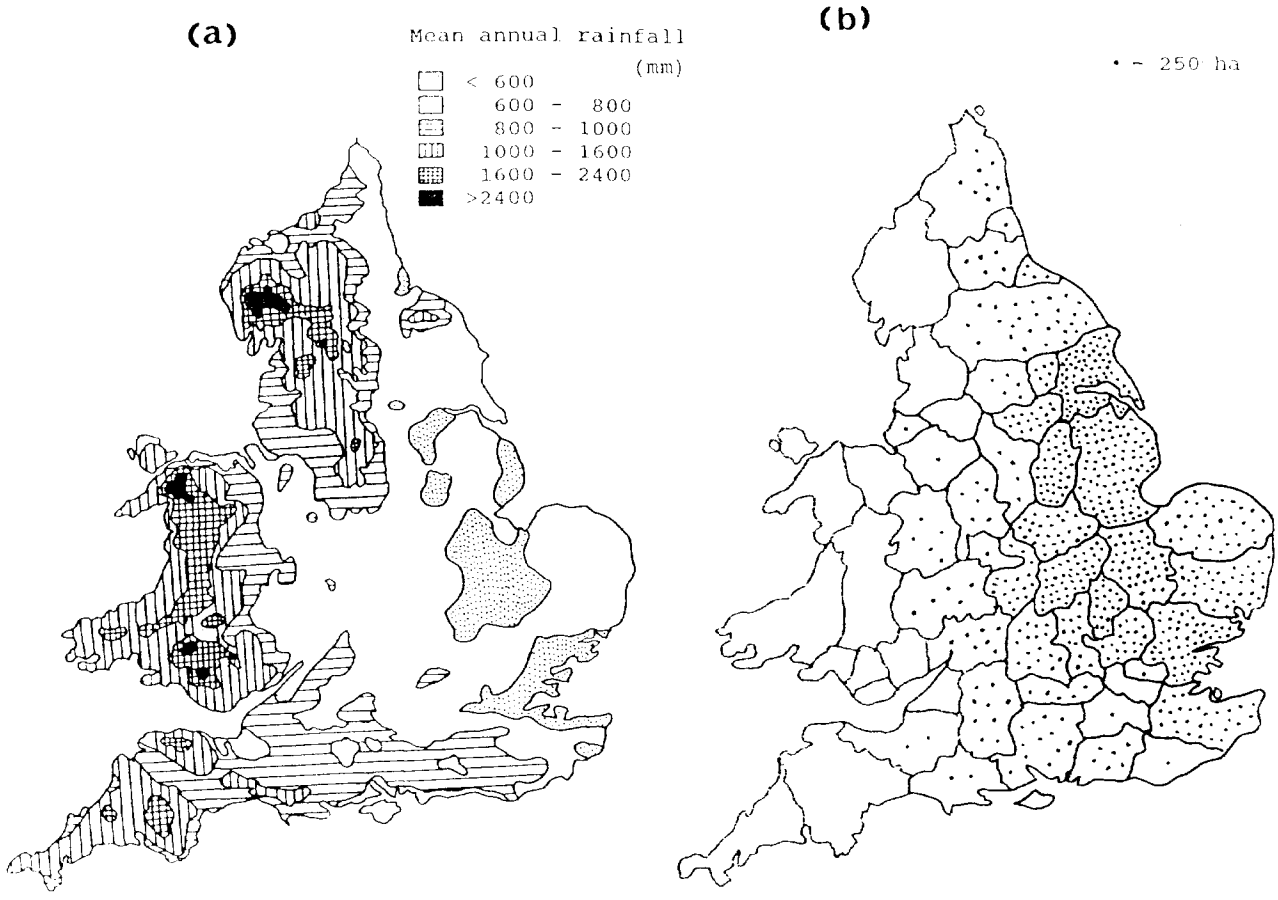

Figure 1. (a) mean annual rainfall in England and Wales, 1931-61 (Chandler & Gregory, 1976). (b) distribution of oilseed rape in England and Wales, 1982 (Aspects of Applied Biology **6**, by kind permission of P. Gladders)

differences relate to the ability to produce air-borne spores that enable effective disease spread within and between crops during the season and to the ability to produce stem lesions that are likely to be the major source of primary inoculum for the next season.

Disease spread between crops

The sexual cycle of *M. capsellae* is monocyclic and air-borne ascospores are produced only in the autumn (Inman *et al.*, 1990; Inman, 1993). These ascospores can potentially be dispersed over relatively long distances and are the most likely source of primary inoculum for infecting autumn-sown crops of oilseed rape at the start of the season. However, once crops are infected by this initial air-borne inoculum subsequent disease spread occurs only by means of splash-dispersed conidia. Unlike ascospores, these are dispersed only short distances by rain-splash (Fitt *et al.*, 1992) and disease spread between crops of oilseed rape during the season is therefore unlikely. The amount of primary inoculum produced each autumn and dispersed to new crops is therefore an important factor in determining the development of white leaf spot epidemics. Seed transmission is unlikely to be an important source of primary inoculum (Crossan, 1954; Petrie & Vanterpool, 1978; Inman, 1993) and conidia produced on infected volunteer oilseed rape plants or cruciferous weeds are unlikely to be dispersed into new crops. Therefore, the only inoculum that a crop of oilseed rape is likely to receive is air-borne ascospores dispersed in the autumn.

This compares with *P. brassicae* which has a polycyclic sexual stage which can potentially produce air-borne ascospores throughout the season (McCartney & Lacey, 1990; Inman *et al.*, 1992b). Disease spread not only occurs by conidia dispersed by splash within an infected crop, but can also occur by air-borne ascospores being dispersed by wind within and between crops. The development of epidemics is therefore not as dependent on the amount of primary inoculum produced in the autumn because there is the potential for crops to receive air-borne inoculum throughout the season.

L. maculans is similar to *M. capsellae* in its life cycle. The sexual stage appears to be monocyclic and is only initiated in debris at the end of each growing season. As a result there is unlikely to be any disease spread between crops during the season as only splash-dispersed conidia are produced in infected crops. However, unlike *M. capsellae*, *L. maculans* can produce air-borne ascospores from debris infected in the previous year not only in the autumn but also the winter and spring, with numbers of ascospores in the UK, declining sharply thereafter (Gladders & Musa, 1980). Therefore crops may potentially receive air-borne inoculum from external sources during most of the season.

Disease spread within crops

Dependence on splash-dispersed conidia not only restricts disease spread between crops of oilseed rape but also limits disease spread within infected crops. This was demonstrated in an epidemic of white leaf spot in a Rothamsted crop in 1991 (Inman *et al.*, 1992c). The gradient in the incidence of white leaf spot across this crop did not appear to be significantly flattened by secondary disease spread between March and May. This appeared to indicate that disease spread

from individual foci by splash-dispersed conidia was limited during this period.

The vertical progress of disease after stem extension is also influenced by reliance on splash-dispersed conidia. Studies have shown that white leaf spot progresses vertically within the growing crop by a combination of vertical splash and internode growth (Inman *et al.*, 1992c). For sustained disease spread to occur after the start of stem extension by this mechanism there must be frequent rainfall and leaves must become infected whilst they are young and their internodes unextended. As conidia are not splashed very high, typically less than 10-20 cm vertically (Inman *et al.*, 1992c; Walklate *et al.*, 1989), a period of several weeks dry weather can allow young, upper leaves to be moved by stem extension above the height to which conidia can be splashed from infected leaves below. As a result the contribution of internode growth to vertical disease progress is lost. Sustained disease progress cannot occur wholly by means of vertical conidial splash and disease progress is invariably halted. Older infected leaves are gradually lost by natural senescence and the incidence of the disease declines rapidly. Disease progress during stem extension is therefore susceptible to arrest by periods of prolonged dry weather because it is dependent on splash-dispersed conidia.

However, in the case of light leaf spot vertical disease progress is not wholly dependent on splash-dispersed conidia and is therefore not so greatly influenced by periods of dry weather. Rather, the dispersal of air-borne ascospores vertically within a crop from infected leaf debris (McCartney & Lacey, 1990), or the deposition of air-borne inoculum into a crop from an external inoculum source, can establish the disease on the upper leaves or pods in situations when the potential for vertical disease progress by splash-dispersed conidia has been lost. Therefore, once stem extension has started light leaf spot is not so dependent on frequent rainfall for disease progress as white leaf spot. This may partly explain the successful development of light leaf spot epidemics in the main oilseed rape growing areas in drier eastern and central counties of England.

Like *M. capsellae*, *L. maculans* is predominantly dependent on splash-dispersed conidia for vertical disease spread during stem extension, although some air-borne ascospores may still be dispersed from the previous season's infected debris during the spring (Gladders & Musa, 1980). The progress of *Phoma* leaf spot is therefore similarly susceptible to dry weather after the start of stem extension and is frequently halted in the spring.

Stem lesions and primary inoculum

A final point for consideration in assessing the potential for white leaf spot to become widespread and economically important in the UK is the ability for *M. capsellae* to produce primary inoculum (air-borne ascospores) in the autumn. As leaf debris is ephemeral in character this is likely to be determined by the number of pod, raceme and stem lesions produced during the spring and summer. This type of debris is much more durable than leaf debris and provides a suitable substrate in which the sexual stage can develop and survive between the initiation of sexual primordia in June or July and the production of ascospores, typically from September to November. In most years the disease fails to become established on the pods and therefore the contribution of infected pod debris is

likely to be limited. Therefore, the occurrence of stem lesions would appear to be an important factor in determining the amount of primary inoculum produced in the next season.

However, observations have suggested that white leaf spot stem lesions are generally infrequent in infected crops of oilseed rape and that, where present, they may be initiated quite late in the season (Inman, 1993). This can be explained by the way in which *M. capsellae* infects stems. This occurs by means of conidia which, after germination, must encounter and penetrate stomata in order to infect the host. Stem infections are therefore unlikely to be initiated before stem extension occurs in the spring and the frequency of lesions is likely to be determined by whether the disease development continues on the leaves after the start of stem extension. The frequency of rainfall during stem extension is therefore important in determining the ability of *M. capsellae* to produce stem lesions and thus to produce primary inoculum in the next season.

A further factor likely to decrease the ability of *M. capsellae* to produce stem lesions is the low density of stomata on stems, particularly at the base. The density of stomata on stems is much lower than that on leaves, on average by a factor of five, and the probability of infection on stems is therefore lower (Inman, 1993). The density of stomata also increases with height up the stem. This may possibly increase the importance of vertical disease progress during stem extension in initiating stem lesions. This seemed to be confirmed by observations from an epidemic in the Rothamsted crop (cv. Cobra) in 1991, when disease progress was halted in May by a period of prolonged dry weather. The disease did not progress much higher than 50 cm up the crop and no stem lesions were observed at harvest.

The importance of stem lesions to the survival of *M. capsellae* can be illustrated by comparison with *L. maculans*. The life cycle of *L. maculans* is similar to that of *M. capsellae* in that the sexual stage is responsible for producing the primary ascospore inoculum (Gladders & Musa, 1980) and splash-dispersed conidia are important in disease development during the majority of the season. Although the period of ascospore production is longer for *L. maculans*, typically from autumn to spring (Gladders & Musa, 1980), the main difference between these two pathogens is in their ability to infect stems.

Although both pathogens are dependent on splash-dispersed conidia for disease development on leaves during stem extension, *L. maculans* is not dependent on disease progress during this period to initiate stem infections. This is because stem lesions are mainly initiated by the systemic growth of hyphae from leaf lesions through leaf petioles and into the stem (Hammond *et al.*, 1985). Leaf infections initiated as early as the autumn, even before stem extension, can therefore produce stem lesions later in the season. Even if disease progress on leaves is halted in the spring, infected stem debris can still occur at the end of the season and therefore the sexual stage of *L. maculans* can be still be produced. By contrast with *M. capsellae*, the ability of *L. maculans* to produce primary inoculum for infecting new crops of autumn-sown oilseed rape is therefore not wholly dependent on disease progress during the previous spring and summer.

CONCLUSIONS

Recent information about the life cycle of *M. capsellae* and disease

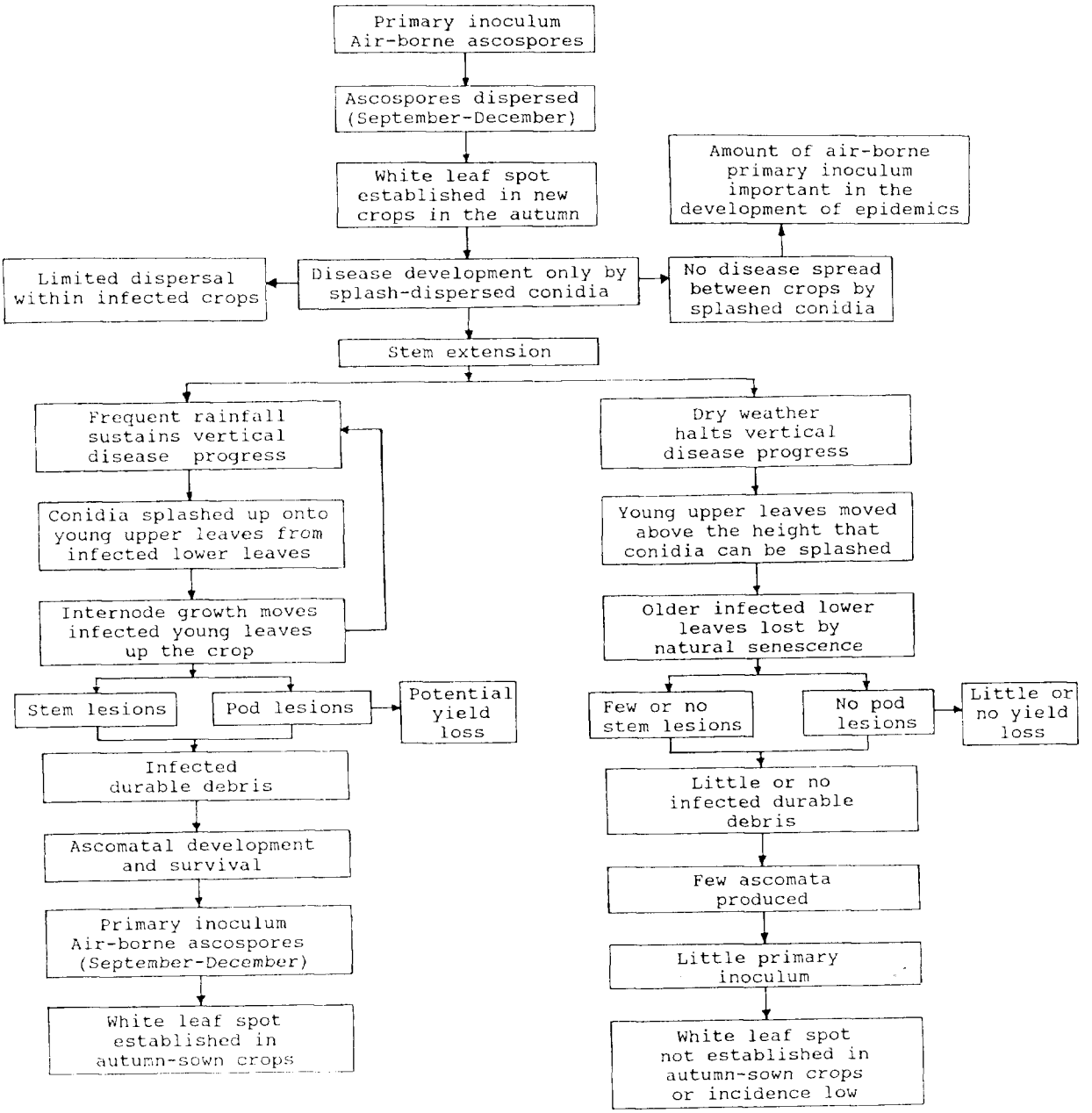

Figure 2. Flow diagram illustrating biological factors likely to limit the ability of white leaf spot to become widespread and epidemic in the UK.

development during the season suggest that white leaf spot is unlikely to become economically important or widespread in the UK under the current conditions of climate and crop distribution (Figs.1 & 2). Long distance dispersal is limited to air-borne primary inoculum (ascospores) in the autumn and there is little possibility for disease spread between crops by splash-dispersed conidia during the season. The amount of primary inoculum is therefore important in determining the development of epidemics in individual crops.

Disease spread within infected crops is likely to be limited because of the small distances that conidia are dispersed by rain-splash. Dependence on splash-dispersal also has important consequences for disease progress after the start of stem extension. During this period disease progress up the crop is highly susceptible to even a single period of dry weather lasting for several weeks. In most years the disease does not spread onto the pods, except in wetter areas with frequent rainfall, and the risk of yield losses from white leaf spot is therefore low.

Stem lesions appear to be dependent on disease progress after the start of stem extension and therefore rely on frequent rainfall during this period. Rainfall may therefore be an important factor limiting the distribution of white leaf spot in the UK because stem lesions are likely to be the principal source of primary inoculum.

Although it therefore appears unlikely that white leaf spot will develop significantly in the main oilseed rape growing areas of the UK, it is still likely to cause occasional severe epidemics in wetter areas in the south west and south of England. In these areas, control strategies which decrease the amount of primary inoculum in the autumn, including the incorporation of crop debris and the control of stem and pod lesions by cultivar resistance or by fungicides which stop disease progress during stem extension, can be important for long term control. In the short term white leaf spot can easily be controlled by using fungicides integrated into existing programmes for the control of other oilseed rape diseases.

In crops where white leaf spot is the predominant disease problem, the susceptibility of disease progress to dry weather during stem extension means that decisions to apply fungicides are best made at flowering. Fungicides applied before flowering are often likely to be unnecessary because disease progress is frequently stopped in the spring or summer by dry weather. Early applications also restrict the cost effectiveness of fungicides applied later to control *Sclerotinia* stem rot or diseases on the pods such as *Alternaria*. Fungicides applied at flowering have been shown to give effective control of white leaf spot on pods and therefore yield benefits (Penaud, 1986). This therefore appears to be the most efficient and practical strategy for the chemical control of white leaf spot.

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