

## Ascochyta blight of broad beans-*Didymella fabae*-*Ascochyta fabae*

Ascochyta blight is the most severe disease of cool-season pulses (Davidson and Kimber, 2007). The species *Didymella fabae* (anamorph *Ascochyta fabae*) that attacks *Vicia faba* can survive and reproduce in and spread from crop debris or be transported in infected seed. Introduction on infected seed occurred in Australia and Canada in the 1970s, and was probably the means for the pathogens original spread to countries outside of southwestern Asia. Ascospores are disseminated by wind from the debris as primary inoculum and secondary cycles are initiated by conidia spread by rain splash from plant lesions. The fungus is host-specific in causing disease but may be able to survive in non-host plants and reproduce on their debris. It is not treated as a phytosanitary risk or listed as an invasive pathogen by major organizations. Seed certification is the primary means of preventing its spread to new areas and the importation of new genotypes of the fungus to areas already infested.

***Didymella fabae*** G.J. Jellis & Punith. 1991 (Ascomycetes, Pleosporales)

Colonies of *Ascochyta fabae* on PDA white to ash-white with sparse to abundant pycnidia; reverse cream to light brown. Colonies more yellow on oat agar. Mycelium abundant, velvety, composed of hyaline to yellowish, smooth, branched, septate hyphae. Pycnidia separate partially immersed, yellow to brown, subglobose to globose, 200-250 µm with usually one papillate ostiole. Conidogenous cells hyaline, short subglobose to cylindrical, arising from innermost layer of cells surrounding pycnidial cavity. Conidia hyaline, straight or slightly curved, base slightly truncated or rounded, one- or sometimes two- or three-septate, 16-24 x 3.5-6 µm, not constricted at septa.

*Didymella fabae*: ascomata on bean straw arranged in rows, immersed, becoming partially erumpent, dark brown, subglobose, single, separate, sometimes in groups, 180-240 x 130-150 µm, with short necks, ostiolate. Pseudoparaphyses hyaline, thin-walled, septate, 1-2 µm diam. Asci in a relatively flat layer, hyaline, cylindrical to subclavate, bitunicate, eight-spored, 55-70 x 10-14 µm, usually constricted near base to form a distinct foot. Ascospores irregularly distichous, hyaline, smooth, slightly biconic, broadly ellipsoid, two-celled, constricted at the septum, with upper cell broader than lower cell, 15-18 x 5.5-6.5 µm. Naturally discharged ascospores on bean straw turn yellowish to brown, sometimes three-septate (Jellis and Punithalingam, 1991). Ascospores produced as a result of some induced crosses between *A. fabae* isolates had a greater range in size than that of the type description (Kaiser et al., 1997), some attaining up to 23.5 in length and some up to 11.8 in breadth.

For additional details, see Punithalingam and Holliday, 1975; Jellis and Punithalingam, 1991.

**Distribution:** Europe, North America (Canada), South America (Brazil, Argentina), Asia (China, Pakistan), Australia.

**Host:** Field (broad) beans, *Vicia faba* (Fabaceae).

*Ascochyta* blight, or leaf and pod spot, of *Vicia faba* is caused by *Didymella fabae* G.J. Jellis & Punith. (anamorph *Ascochyta fabae* Speg.). The fungus was initially known in the asexual state spreading by means of conidia produced in pycnidia. The sexual state (teleomorph) was first reported and described by Jellis and Punithalingam (1991) on overwintering *V. faba* straw in the UK. It was subsequently found in Australia (Jellis et al., 1998), Syria (Bayaa and Kabbabeh, 2000) and Spain (Rubiales and Traperero-Casas, 2002).

Due to a lack of significant differences in their morphology, Gossen et al. (1986) proposed that the host-specific *Ascochyta* pathogens of lentil and broad bean be treated as formae speciales of the same species, *A. fabae*. This taxonomy was used in some subsequent literature (Kaiser et al., 1994; Ahmed and Beniwal, 1998). Discovery and examination of the teleomorphs of both pathogens then lead Kaiser et al. (1997) to confirm the distinction between them as biological species. Both *A. fabae* and *A. lentis* require the pairing of two compatible isolates for the sexual state to develop; cross inoculation of *A. fabae* to lentil failed to induce disease and *A. lentis* failed to induce disease on broad bean. Although the two species were able to interbreed and produce some viable ascospores, most asci were abnormal and the conidia produced in the ascospore cultures failed to infect either host (Kaiser et al. 1997).

Further investigation of relationships among *Didymella* and *Ascochyta* legume pathogens by molecular means (Kaiser et al., 1997; Barve et al., 2003; Peever et al., 2007; Chilvers et al., 2009) have shown that *D. fabae* is closely related to *D. lentis* and *A. pisi* and more distantly related to the chickpea pathogen *A. rabiei* (Pass.) Labr. (*Didymella rabiei* (Kovatsch.) Arx). The recognition of these distinct species based on their hosts reflects their phylogeny. According to the work of Hernandez-Bello et al. (2006), isolates of *D. fabae* and the newly identified teleomorph of the pea pathogen, *D. pisi*, are capable of mating with production of viable hybrid progeny. Nevertheless, most of the progeny could not infect either faba bean

or pea under standard conditions, so that host specificity appears to be a character maintaining separation between the species.

Previously, the placement of the genus *Didymella* within the Pleosporales has been unclear. A molecular phylogeny obtained for species of *Phoma* and related genera, including the type species of *Didymella* and *Ascochyta*, placed *A. fabae* and *A. pisi* within a clade for which the new family Didymellaceae was proposed (de Gruyter et al., 2009).

*Didymella fabae* occurs in certain parts of all the inhabited continents (IMI, 1993; Farr and Rossman, 2009). Because it is seedborne, the pathogen is likely to be present wherever the host, *Vicia faba*, is grown (Gaunt, 1983). It is common in Europe and was introduced in seed to parts of Australia and Canada, but has not been reported from the USA. Kaiser (1997) suggests that the fungus originated in, and spread from, southwestern Asia, the center of origin of the host plant. The disease is most prevalent where faba bean is grown as a winter crop in regions with Mediterranean or mild oceanic climates (Stoddard et al., 2010 in press).

### **Similarities to other species/conditions**

The *Ascochyta* species on pea, fava bean and lentil are difficult to distinguish by morphology of isolates in culture (Gossen et al., 1986; Peever et al., 2007; Cherif et al., 2009). Conidia of *A. fabae* observed on the plant are longer (Gossen et al., 1986; Simay, 1988).

Young lesions of *D. fabae* on the leaves are dark brown spots resembling those of chocolate spot caused by *Botrytis fabae* Sardina. However, *B. fabae* lesions do not contain pycnidia but produce large conidia on erect conidiophores. Often both pathogens are present on the plant, and isolates of *B. fabae* have been obtained from lesions caused by *D. fabae* (Gaunt, 1983).

### **DETECTION AND INSPECTION METHODS**

Symptoms of the disease are easily observed on leaves, stems and pods of plants in the field (see Symptoms). Where confirmation of the disease is required, surface-sterilized tissue from the edges of the lesions should be incubated on PDA to produce pycnidia containing one-septate spores. From older lesions, conidia can be obtained for microscopic examination by scraping the surface of lesions containing pycnidia into water on a glass slide.

If no structures are visible in leaf lesions, incubation of the leaves in a moist chamber overnight may allow distinction from chocolate spot caused by *Botrytis fabae*, which should produce characteristic conidiophores and conidia in lesions (Koike et al, 2006).

Seedborne infection can be detected by observing the characteristics of the colonies appearing after incubation of surface-sterilized seed on PDA (see Seed Health Tests).

### **DIAGNOSIS**

Leaves, stems or pods that exhibit symptoms should be incubated in a moist chamber for 24-48 h at 22°C. Pycnidia will produce abundant conidia for identification. The fungus can be isolated by surface-sterilizing the infected parts and dissecting the margin of the lesions area on to PDA or by directly transferring freshly formed pycnidia from the centre of the lesions on to agar. After incubation at 22°C, colonies of white or grey-white mycelium containing pycnidia are produced. Pycnidial formation is greatly stimulated by exposure to continuous fluorescent illumination for 4-6 days (Hewett, 1966). Cherif et al. (2006) provide photographs of conidia of *Ascochyta* species from legumes obtained in culture and a table of measurements reported in the literature. The data of Simay (1988) indicate that conidia from pycnidia in culture may be smaller than those produced on the plant.

Sequences of certain regions of DNA for the species are available in GenBank for comparison (NCBI, 2009). A rapid, specific and sensitive PCR amplification /restriction enzyme digest test for the species in leaf tissue and seed, similar to that developed for *Ascochyta rabiei* (Phan et al., 2002), has not been reported in the literature.

### **CROPS AND OTHER PLANTS AFFECTED**

The fungus is highly specialized to *Vicia faba* (broad bean or faba bean), and inoculations on other legume species have been largely unsuccessful (Yu, 1947; Sepulveda, 1993), although Gaunt (1983) did find that infections could be obtained under certain specific conditions. Despite numerous attempts to cross-infect other legume species, only a very few reports of limited success have been made. Bondartzeva-Monteverde and Vassilievski (1941) found only atypical lesions formed by a broad bean isolate on species other than bean. Sprague (1929) and Beaumont (1950) found that isolates obtained from broad bean induced symptoms on peas (*Pisum sativum*), but the conditions and methods under which the tests were made were not clearly defined. Wallen and Galway (1977) were unable to obtain infection of peas in the greenhouse in Canada.

Hernandez-Bello et al. (2006) found species from legumes to be host-specific in causing disease, but the fungi could be isolated from inoculated, but asymptomatic, non-hosts; *A. fabae* was isolated from pea and lentil plants. The possibility exists, then, that this pathogen could survive in non-host plants and even reproduce on the debris. Autoclaved chickpea stems have been used to obtain teleomorph production from mated *A. fabae* isolates (Kaiser et al., 1997).

*Ascochyta fabae* has been reported as infecting *Onobrychis viciifolia* Scop. (sainfoin) in southwest Asia (Sharifnabi and Fatehi, 1996; Eken, 2003). The identifications based on morphology need to be tested by inoculation of isolates onto *V. faba*.

## SYMPTOMS

Symptoms occur on leaves, stems and pods. Where seedlings have grown from infected seeds, lesions are more obvious on the upper parts of the stem and on the older leaves. Lesions on the leaves are usually circular, dark brown and initially about 1 mm in diameter. After a short time, the lesions become larger and slightly sunken with a pale brown to dark grey centre surrounded by a broad, dark, chocolate-coloured margin. As the spots enlarge, they become more irregular in shape and coalesce to cover larger areas of the leaf. Some zonation may occur within the necrotic area of the lesions, which may cause confusion with lesions of chocolate spot caused by *Botrytis fabae*. A more general browning of the vascular tissue of the leaf may occur as the lesions develop. Prominent, dark pycnidia develop within the lesions, particularly as the leaves age or when conditions are moist. The pycnidia can vary in abundance and are sometimes concentrically arranged.

On the stems, the lesions are usually smaller at the early stages of infection, but elongate up the stem and become markedly sunken. Stem lesions are usually darker than those on leaves, and contain scattered pycnidia. When the lesions are deeply sunken, either the stems of the plants may break at the point of infection, causing the plants to lodge or, if infection occurs at an early stage, the stems may bend upwards producing a kink where the stems regrow vertically. At the seedling stage, when infection originates from the seed, the combination of stem and leaf infection may result in the death of the plant.

As the pods develop, lesions can be produced over the surface. They become very deep with dark brown centres containing abundant pycnidia. In damp conditions, the conidial masses produced are pale pink to yellow. Well-developed lesions may penetrate the pod wall and affect seed set or may blemish the developing seeds within the pod. However, seed staining does not always indicate infection by the pathogen, because other saprobic organisms may invade the damaged tissue of the pod. Colonies of *D. fabae* can also frequently be isolated from unstained seed during routine seed health tests in the laboratory (Biddle, unpublished).

## BIOLOGY AND ECOLOGY

### Life cycle:

The most common source of infection is through the seed (Hewett, 1973). In the UK, seeds can vary in their infection level depending on the influence of weather conditions on disease development in the parent crop (Biddle, 1994). Dispersal of conidia by rain splash from pycnidia on crop debris or on volunteer plants in the immediate vicinity of the new crop is considered to be important in the localized spread of the fungus in fields (Bond and Pope, 1980; Gaunt, 1983). However, splash dispersal of conidia is usually limited in its extent, although it is possible that some conidia may achieve greater dispersal in aerosols during periods of windy wet weather (Jellis and Punithalingam, 1991). A limited survival of the fungus may occur on crop debris; Dodd (1971) showed survival on buried crop debris for up to 4 months in the UK. No chlamydospore formation was observed in the soil. The fungus also survived over the summer in crop debris in northern Iraq (Michail et al., 1983). The teleomorph *D. fabae*, produced over winter on infected bean straw in stubble fields, is now considered to be a major source of infection through air-borne dispersal of the sexual spores (Jellis and Punithalingam, 1991; Rubiales and Trapero-Casas, 2002).

Spore germination occurs between 15 and 25°C, with an optimum of 20°C at high relative humidity (Wallen and Galway, 1977). A minimum period of leaf wetness of four hours was required for successful infection by conidia at 20 and 25°C (Pritchard et al., 1989). In culture on PDA, the optimum growth temperature was about 25°C (Yu, 1947). However, symptoms are frequently found on leaves and stems in the field during the early spring when temperatures are typically much lower, although the humidity is high for long periods of time. The exact conditions required for the development of the disease are not fully understood, but several authors suggest that periods of wet weather are conducive to disease development (Beaumont, 1950; Dodd, 1971; Hewett, 1973; Wallen and Galway, 1977). The disease appears to be more common on autumn-planted beans where the conditions over the winter may be more favourable for infection (Maurin and Tivoli, 1992).

The cycle of the pathogen in autumn-sown crops can begin in the late autumn or early spring, either from

conidia produced in lesions on seedlings grown from infected seed or with leaf infection by splash-borne conidia from volunteer beans growing in neighbouring fields. Alternatively, airborne ascospores produced by pseudothecia that developed on crop debris in nearby or distant fields may reach the seedlings during the winter (Jellis and Punithalingam, 1991). Once the pathogen is established in the crop, conidia are spread from the pycnidia by water splash, resulting in leaf and stem spotting of the plants. Pod infection can occur at any time during the plant's reproductive phase, although the development of deep lesions is more obvious on mature pods.

Seed infection occurs as a result of mycelial invasion from lesions on the pod wall into the testa, often resulting in black sunken spots (Sprague, 1929), although it has been noted during routine seed testing that colonies can arise from seed with no obvious spotting (Biddle, unpublished). Little is known about the process of seed to seedling transmission. Some laboratory work has demonstrated that high levels of pod infection are critical for seed infection, but seeds from symptomless pods had a low level of infection suggesting the possibility of systemic infection (Garber et al., 1992).

## REPRODUCTIVE BIOLOGY

Kaiser et al. (1997) demonstrated that *D. fabae* is both heterothallic and bipolar, requiring mating between two individuals, each carrying a different mating-type allele for sexual reproduction. Pseudothecia have been observed on non-living plant parts only (Jellis and Punithalingam, 1991; Rubiales and Trapero-Casas, 2002), including autoclaved chickpea (*Cicer arietinum* L.) stems (Kaiser et al., 1997). Mating was successful between isolates of different geographic origins (Hernandez-Bella et al., 2006)

## PHYSIOLOGY AND PHENOLOGY:

Isolates of *Ascochyta fabae* vary greatly in cultural morphology and pathogenicity (Kharbanda and Bernier, 1980; Filipowicz, 1986). Some physiological races have been classified on the basis of the differential interactions between *V. faba* genotypes (Hanounik and Robertson, 1989); Rashid et al., 1991). Jellis et al. (1998) suggest that further work on standardizing methods and differential cultivars is necessary to confirm the existence of specialization. Variation of isolates in southern Australia was too great to permit classification into meaningful pathotypes (Kohpina et al., 1999). Results of inoculations of several plant species with progeny of crosses between *A. fabae* and closely related legume pathogens indicate that pathogenicity is governed by multiple genes (Peever et al., 2007).

## MOVEMENT AND DISPERSAL

**Natural dispersal:** Conidia are dispersed from pycnidia by rain splash from crop debris or infected plants (Hewett, 1973; Bond and Pope, 1980), and ascospores are disseminated in the air from the sexual state produced in weathered debris (Rubiales and Trapero 2002).

**Accidental introduction:** Kaiser (1997) reviewed the record of introduction of *Ascochyta* species on legumes worldwide in seed used for growing crops or as germplasm for selection and breeding. *Didymella fabae* entered Australia and Canada in the 1970s in seed imported from Europe.

## SEEDBORNE ASPECTS

### Incidence

The incidence of seedborne infection of beans varies from year to year depending on the weather conditions, which affect infection in the parent crop (Hewett, 1966, 1973; Biddle, 1994). Infection of seed by *D. fabae* can vary between years and between cultivars grown in the same year. Hewett (1966) found that of 180 seed samples produced from commercial crops during 1964-65, only a small number were infected, whereas of 62 samples tested the following year, just over a third of the samples were infected at levels of 3% to more than 10%. In 1992, a similar survey was carried out on 451 seed lots of winter beans produced in the UK (Biddle, 1994). Of the seed lots tested, 31% contained levels of infection which exceeded 1%, including 3% of seed with more than 10% infection.

The survival of the closely related *Ascochyta fabae* f.sp. *lentis* (teleomorph: *Didymella lentis*) on infected lentil seeds when stored normally or in refrigeration was studied for four consecutive years (1987-90). The pathogen was routinely isolated from seeds kept under both conditions. At the end of the study period, seed germination losses were 44% and 46% for refrigerated and normal storage. Both germination and fungal recovery frequencies dropped with increasing time in storage. The recovery of the pathogen declined by about 50% after 1 year of storage. It is suggested that storing lentil seeds that are to be used for planting for 1 year could be used to reduce the initial inoculum of *D. lentis* under traditional storage (Ahmed and Beniwal, 1998).

## EFFECT ON SEED QUALITY

Seed infection occurs as a result of mycelial invasion through lesions on the pod wall into the testa,

resulting in black sunken spots (Sprague, 1929). Among infected seed in a lot in Iraq, the fungus was found in 100% of the seed coats, 40% of the cotyledons, and 27% of the embryos (Michail et al., 1983). Infected seed may be blemished, making it unsightly and unsuitable for use in human consumption or as an ingredient in racing pigeon food. Infection can result in some pre-emergence mortality, but the main effect on the crop is the production of infected seedlings, which provide the inoculum for later infection by splash-borne conidia.

## **PATHOGEN TRANSMISSION**

Seed transmission was documented by Sprague (1929) (see Biology). Yu (1947) reported that proportion of seedlings grown from infected seed became infected whereas those grown from healthy seed remained uninfected. There has been little work on seed to seedling transmission rates in the field. Hewett (1973) made field sowings of bean seed samples with infection levels of 2-15% and noted that all such seed produced some seedlings with leaf lesions, with the fungus spreading up to 10 m in an average season. Weather conditions had a significant effect on the rate and distance of pathogen spread (see Biology). Wallen and Galway (1977) and Kharbanda and Bernier (1979) reported low levels of transmission from infected seed in spring-sown beans in Canada, with the level of seedling infection much lower than that detected in the seed. Higher levels, up to 50%, in winter-sown beans have been reported from New Zealand (Gaunt and Liew, 1981).

## **SEED TREATMENTS**

Successful control of seedborne infection has not been achieved solely by seed treatments. Maude et al. (1969) reported some reduction of seed infection following a thiram soak at 37°C for 12 h and noted that thiabendazole and benomyl applied as seed treatments gave some improvement in the level of control in the laboratory but not in the field. They suggested that poor penetration by these systemic fungicides reduced the effectiveness of control of deep-seated infection. Other workers have reported similar experiences with systemic fungicides applied in powder or slurry form, although results varied from good to poor levels of control in the field (Maude and Kyle, 1971; Wallen and Galway, 1977; Kharbanda and Bernier 1979; Gaunt and Liew, 1981; Gaunt, 1983; Michail et al., 1983; Jellis et al., 1988). For this reason, current recommendations in the UK are to use and treat the seed only if the level of seedborne infection is below 4% (Knott et al., 1994).

## **SEED HEALTH TESTS**

Hewett (1966) first described a method for routinely assessing the levels of seedborne infection in bean seed using an agar plate technique. The method has not yet been published as an ISTA standard (ISTA, 2009) although the method has been adopted in the UK by Trade Accredited Laboratories and is used for the testing of seeds in the UK Field Bean Seed Certification Scheme.

### **Agar Plate Technique**

- Place seed in a 10% solution of sodium hypochlorite for 5 min.
- The seed is blotted before being placed aseptically onto the surface of plates containing PDA.
- 10 seeds are placed on each plate.
- A minimum of 200 seeds is used in the test: For seed intended for Pre-basic and Basic production, 1000 are used; for first generation (C1) production, 500 seeds are tested.
- The plates are incubated in the dark at 22°C for 7 days.
- Colonies characteristic of *D. fabae* are examined for the presence of pycnidia and microscopic examination of conidia is made to confirm the identity of the pathogen.
- A further incubation for 3-4 days under constant fluorescent light may be necessary to promote pycnidial production.

## **IMPACTS**

Ascochyta blight is a common and occasionally destructive disease that has been reported from all six continents (Gaunt, 1983). The extent of the damage caused by the disease depends on weather conditions (McKenzie and Morrall, 1975; Maurin and Tivoli, 1992) and cultivar resistance. Madeira et al. (1988) reported that the disease reduced the leaf area index and dry matter production and that a significant seed weight reduction of 15% was incurred. Yield losses of 32-41% were reported in several years in the Czech Republic (Ondrej, 1991); similar levels were observed in New Zealand (Hampton, 1980). In the drier areas of eastern England, crop losses are relatively low although pod infection can become severe later in the season, especially in autumn-sown crops, resulting in high levels of seedborne infection. Worldwide yield losses can be as high as 90 to 100% in susceptible varieties (Yu, 1947; Hawtin and Stewart, 1979; Hanounik and Robertson, 1988).

## Prevention - SPS measures: Seed Certification

In the UK, much emphasis has been placed on the production of healthy seed, as the seed has long been considered to be an important source of inoculum. The present UK Seed Certification Scheme is based on standards of maximum levels of seedborne infection permitted (NIAB, 2000) as follows:

Pre-basic seed: 1 infected seed per 1000

Basic seed: 2 infected seeds per 1000

Certified seed first generation: 2 infected seeds per 500

Certified seed second generation: 4 infected seeds per 200

There are no standards applicable to farm-saved seed which is not sold, but it is recommended that the seed should be tested and discarded if infection exceeds 3%. Seed with between 1 and 3% infection may be used following suitable seed treatment (Knott et al., 1994).

Internationally, prevention of the pathogens movement in seed is necessary to prevent not only its introduction into new areas, but also the possible introduction of more virulent races, strains or pathotypes where the disease already occurs as well as the introduction of the second mating type where that is absent, which would allow development of greater variation in the pathogen. A likely danger of importation and storage of broad bean seed in germplasm collections is that it may also maintain viability of *D. fabae* in any infected seed as has done for related legume pathogens (Kaiser, 1997).

## CULTURAL CONTROL

Because of the risk of carryover of infested crop debris it is recommended that faba beans not be grown in the same field in the following season. A rotation of at least four legume-free years is recommended for production of beans and peas in the UK (Knott et al., 1984). Such a break should allow for the natural destruction of any soilborne inoculum. However, rotation plans must take into consideration the results of Trapero-Casas and Kaiser (2009), who found that the related chickpea pathogen, *Didymella rabiei*, can infect non-host plants, including common weeds, without causing disease, and that it could reproduce on the dead material of some of those non-host plants that might be used in the rotation. If *D. fabae* has the same capacity for persistence away from its primary host, then choices of crops used in rotation and weed control practices in those crops may be affected.

Avoidance of fields adjacent to a previous crop of beans and adequate destruction of volunteer plants before sowing will reduce the possibility of splash-borne inoculum, but the discovery of the teleomorph (Jellis and Punithalingam, 1991) suggests a significant risk of infection by wind-blown ascospores from more distant crop debris. The practice of minimum cultivation following a bean crop will allow a greater proportion of crop debris to be left on the soil surface, permitting the possible development of the teleomorph. In Canada, *A. fabae* did not survive over winter in the soil in field plots in which crop debris had been ploughed under the previous year (Wallen and Galway, 1977).

In addition to such measures, Davidson and Kimber (2006) suggest the selection of planting dates that will separate the most susceptible period of crop growth from the likely time of ascospore production, if doing so will not also reduce yield by missing the most agronomically favorable period of the season.

Intercropping of broad beans with cereals such as maize or wheat has been suggested as a control measure for the chocolate spot disease caused by *Botrytis* species (Stoddard et al., 2010 in press). The barriers created to aerial spread of *Botrytis* conidia by the non-host plants would seem likely to be effective against rain-splash dispersal of *Ascochyta* conidia as well.

## BIOLOGICAL CONTROL

Early results of work on control of the related legume pathogen, *D. rabiei*, with saprophytic fungi have been positive, presenting a possible alternative for control of *D. fabae*. Application of *Aureobasidium pullulans* (deBary) G. Arnaud to infested chickpea debris reduced suppressed incidence of disease in both the greenhouse and the field (Dugan et al., 2009).

## CHEMICAL CONTROL

Variable results have been reported in field trials evaluating the use of foliar applied fungicides on beans. The timing of application in relation to disease infection periods would seem critical because the principal fungicides available are protectant in their activity. Some success has been reported with MBC (carbendazim) and dicarboximides (Beer et al., 1990), benomyl and mancozeb (Michail et al., 1983), iprodione (Jellis et al., 1988), and chlorothalonil (Kharbanda and Bernier, 1979; Liew and Gaunt, 1980; Knott et al., 1994). A combination of seed treatment and foliar sprays was suggested by Jellis et al. (1988) for protection of valuable breeding material or during early multiplication of cultivars.

## RESISTANT CULTIVARS

Breeding for resistance has been recent in development (Maurin and Tivoli, 1992), but ICARDA have developed a number of resistant breeding lines. The lines BPL 471, 460, 646, 74 and 2485 were resistant to pathogen strains in six countries (Hanounik and Robertson, 1989). In addition, recent introduction of cultivars in the UK have shown good resistance in the field. The autumn-sown winter beans 'Striker', 'Clipper' and 'Silver' are available commercially (NIAB, 2000). Several lines in France (Tivoli et al., 1988; Maurin and Tivoli, 1992) and 'Fioletowy Czyzowskitch' and 'Krasnoyarskii' in Poland (Zakrzewska, 1986) are also reported to have good field resistance to field infection. All the commercial cultivars tested in the Czech Republic were susceptible or highly susceptible, but resistance could be incorporated from the sources 29H, L-8 and Petra (Ondrej and Hunady, 2007). Several resistant to moderately resistant varieties have been developed for Southern Australia (Pulse Australia, 2009). Tivoli et al. (2006) present a table of sources of resistance for breeding.

Resistance to *Didymella fabae* is multigenic and additive (Kharrat et al., 2006; Ondrej and Hunady, 2007), although Kharrat et al. (2006) found it to be largely controlled by dominant alleles. Genes for resistance can be expressed differently in the stems and the leaves of *Vicia faba* (Rashid et al., 1991; Kharrat et al., 1998; Khopina et al., 1999; Avila et al., 2004). Multiple quantitative trait loci for resistance have been identified (Roman et al., 2003; Avila et al., 2004; Diaz-Ruiz et al., 2009).

## IPM

Control of Ascochyta blights of legumes may require a combination of established methods, including use of pathogen-free seed, destruction or avoidance of inoculum sources, manipulation of sowing dates, application of seed treatments and foliar fungicides, and breeding of cultivars with improved resistance (Davidson and Kimber, 2007). Stoddard et al. (2010, in press) note that because the conditions required for infection by the various species are known, forecasting of infection periods can be used strategically, where necessary, to schedule applications of foliar fungicides for greatest effectiveness and least cost.

## GAPS IN KNOWLEDGE/RESEARCH NEEDS

A rapid and specific molecular test for the pathogen in seed and leaves would enhance diagnosis and the prevention of spread both through seed and in the field. A model can be developed and tested for forecasting infection periods and tested for use in timing fungicide applications. The observations by Trapero-Casas and Kaiser (2009) should be applied to this pathogen to determine if it can infect other plants asymptotically and survive through them in the absence of the known host.

## References

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Symptoms in field. Photo courtesy of CABI Crop Protection Compendium.



Pycnidia developing in leaf spots. Photo courtesy of CABI Crop Protection Compendium.



Pycnidia on beans. Photo courtesy of CABI Crop Protection Compendium.



Pycnidia on seeds. Photo courtesy of CABI Crop Protection Compendium.

