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FULL RESEARCH PAPER

Comparison of the epidemiology of ascochyta blights on grain legumes

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Abstract Asochyta blights of grain legumes are caused by fungal pathogens in the genus *Ascochyta*. Different species infect the different legume species, and in pea three species including *Phoma medicaginis* var. *pinodella* have been implicated in ascochyta blight. The impact of the diseases varies between crops, countries, seasons and cropping systems, and yield loss data collected under well-defined conditions is scarce. However, ascochyta blights are considered major diseases in many areas where legumes are grown. Symptoms appear on all aerial parts of the plant, and lesions are similar for most of the species, except for *M. pinodes* and *P. medicaginis* var. *pinodella*. Infected seed, stubble and/or air-borne ascospores are major sources of primary inoculum. Their importance varies between species and also between regions. All *Ascochyta* spp. produce rain-splashed conidia during the cropping season which are responsible for the spread of the disease within the crop canopy. Only in pea are ascospores involved in secondary disease spread. Limited data suggests that *Ascochyta* spp. may be hemibiotrophs; however, toxins characteristic for necrotrophs have been isolated from some of the species. Modelling of ascochyta blights is still in the developmental stage and implementation of such models for disease forecasting is the exception.

Keywords Pea · Faba bean · Chickpea · Lentil · *Ascochyta* · *Mycosphaerella pinodes* · *Phoma medicaginis* · *Didymella* · Life-cycle

Introduction

Grain legumes, also referred to as pulse crops (faba bean, chickpea, pea, lentil and lupin), play an important role in farming systems worldwide (Halila et al. 1990; Kelley et al. 1997). Their seed protein content is high ranging from 22% in pea to 45% in lupin, and they are used for human and animal consumption in the southern and northern hemispheres. Most of the genotypes of food and feed legume species are characterised by an indeterminate growth habit: the reproductive structures are not initiated at the same time along the stem, but flowers and pods develop continuously on the plants. As a consequence, all plant organs (vegetative aerial parts, nodules, roots, reproductive organs) are competing for assimilates at any given time.

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Pulse crops are infected by fungal diseases such as grey mould or chocolate spot, ascochyta blights, anthracnose, powdery and downy mildews and rusts. The relative importance of these diseases and their effect on yield vary among countries. However, ascochyta blights affect large areas in many countries where pulses are cultivated and cause considerable losses in seed quality and quantity. In crops seeded in the autumn and winter, a practice associated with significantly higher seed yield due to the crop flowering and producing seeds in a cooler and more humid environment during the second part of the crop's life, ascochyta blights can be of particular importance. These diseases which can occur on all above-ground parts of the plant are the main foliar disease on grain legumes except for lupin.

The primary objective for this review is to highlight similarities and differences in the epidemiology across the species and across countries. The intention is to promote thinking across systems and to explore the potential of adapting successful research strategies from one agricultural system to another, and from one *Ascochyta* spp. to other species of this genus.

The pathogens

All pathogens responsible for ascochyta blights belong to the genus *Ascochyta*, with the different species *pisi* and *pinodes* (on pea), *rabiei* (on chickpea), *lentis* (on lentil), *fabae* (on faba bean), and *viciae* (on vicia). Another closely related species is *Phoma medicaginis* var. *pinodella* which frequently occurs on pea and causes symptoms on internodes and leaves similar to those induced by *A. pinodes*. It is one of the three species implied in the ascochyta blight complex of pea (Hare and Walker 1944; Wallen 1965), which differentiates ascochyta blight of pea from that on lentil, faba bean and chickpea, all of which are caused by a single fungal species. An understanding of the interrelationship between the three pathogens of the pea ascochyta complex is still lacking, but appears to involve interspecies competition and possibly different climatic optima as well as resistance in pea cultivars to particular species. In Australia, Bretag et al. (1995a) observed that in 436 seed lots tested 94.8% of isolates were *M. pinodes*, 4.2% *P. medicaginis* and 1.0% *A. pisi*. Skolko et al. (1954) reported that 85% of Canadian ascochyta blight-infected seed lots were infected by *A. pisi*. However, since the introduction of resistance to *A. pisi* in the mid 1960s *M. pinodes* has become the dominant species found on Canadian pea seed (R.A.A. Morrall, Department of Biology, University of Saskatchewan, Canada, pers. comm.). In France, *M. pinodes* is the dominant pea pathogen, but *A. pisi* can be found in southern France (C. Le May, INRA, France, pers. comm.). Comparison of these pathogens in terms of culture characteristics, symptoms, and disease cycle are given by Jones (1927) and Hare and Walker (1944).

Reproduction

All of the *Ascochyta* spp. produce pycnidia with hyaline, straight or slightly curved conidia (pycnidiospores), and generally with one septum except those from *P. medicaginis* var. *pinodella* which are in most cases unicellular.

The teleomorph of these fungi belong to the genera *Mycosphaerella* (*A. pinodes*) or *Didymella* (*A. lentis*, *A. rabiei*, *A. fabae*) with the exception of *A. pisi* for which the teleomorph has not been described to date (Table 1). The teleomorph of *P. medicaginis* var. *pinodella* was described as *M. pinodes* by Bowen et al. (1997), but this was not supported by subsequent morphological studies and RAPD analysis (Onfroy et al. 1999). *Mycosphaerella pinodes* is homothallic and forms its pseudothecia on the senescent stipules during the second part of the cropping season. In contrast, all *Didymella* species are heterothallic with two mating types (Wilson and Kaiser 1995; Kaiser et al. 1997; Barve et al. 2003; Peever et al. 2004) and pseudothecia, if present, develop at the end of the cropping season on dead plants. These sexual fruiting structures permit the fungi to overwinter and are considered to play an important role in generating pathogen variability (Kaiser 1997).

Table 1 Characteristics of the *Ascochyta* species infecting grain legume species

	A. pisi	*P. m.* var. *pinodella*	*A. fabae*	*A. lentis*	*A. rabiei*	*A. pinodes*
Host	Pea	Pea	Faba bean	Lentil	Chickpea	Pea
Teleomorph	Unknown	Unknown	*Didymella*	*Didymella*	*Didymella*	*Mycosphaerella*
Mating types	–	0	+	+	+	0
Chlamydospores	0	+	0	0	0	0/+

– = no sexual form

0 = absence

+ = presence

Development of the anamorph or teleomorph depends on specific environmental and/or nutritional conditions. Pycnidia are formed generally during the vegetative cycle and pseudothecia at the end of the cropping cycle on senescent tissues (Agrios 2004). Navas-Cortes et al. (1998) determined that humidity levels of 100% were important for the development of pseudothecia of *D. rabiei* whereas low temperatures (5–10°C) were critical for pseudothecial maturation. Roger and Tivoli (1996a) observed that the development of reproductive structures and sporulation of *M. pinodes* were optimal at 20°C, but were sparse at 15°C. The switch from pseudothecial development to pycnidial formation seems to occur as a response to the availability of nutrients, with pseudothecia developing under poor and pycnidia under high nutrient conditions. It was also suggested that a reduction of the phytoalexin concentration (pisatin) is involved in this switch to pseudothecial development (Roger and Tivoli 1996b; Roger et al. 1999a).

Pathogenesis of *Ascochyta* spp.

Type of parasitism

The status of ascochyta blight pathogens in regard to the type of parasitism has not been discussed in detail in the literature. *Ascochyta fabae* was found to develop intracellular hyphae in epidermal tissue of faba bean during the early stages of infection, suggesting an initial biotrophic phase (Maurin et al. 1993). Similarly, Clulow et al. (1991) observed a biotrophic phase lasting for about 48 h in pea epicotyls inoculated with *M. pinodes*. Therefore, fungi responsible for ascochyta blights may be considered as hemibiotrophs characterised by an initial biotrophic phase that is followed by a necrotrophic phase. However, phytotoxins characteristic for necrotrophic pathogens have been isolated from the germination fluid of *A. rabiei* spores and were suggested to be of importance in early ascochyta blight development on chickpea (Höhl et al. 1991). Histological studies for the first critical 48–72 h have not been published for this pathogen to determine whether host invasion follows a biotrophic or necrotrophic strategy.

Infection process

A few studies have been published that describe the infection process by *Ascochyta* spp. on grain legumes at the microscopic level. Direct penetration of the pea cuticle by the germ tube was observed for *A. pisi* (Brewer and MacNeill 1953) and for *M. pinodes* on leaves but not on epicotyls (Clulow et al. 1992). Epicotyl penetration typically occurred through an appressorium which Roger et al. (1999a) also observed for leaf infections by this pathogen. Subsequent colonization by *A. pisi* was characterized by an initial phase of subcuticular development followed by intercellular spread which resulted in the collapse and death of the host cells, beyond which no hyphae were detected (Brewer and MacNeill 1953; Heath and Wood 1969). *Asochyta fabae* was also observed to invade intercellular spaces between epidermis and mesophyll of susceptible faba bean lines within the area of the lesion (Maurin et al. 1993). In contrast, intra- as well as intercellular hyphae in the palisade mesophyll 24 h after inoculation were visible in pea leaves inoculated with *M. pinodes* resulting in a rapid disorganisation and browning of the contents of invaded cells and those adjacent to intercellular hyphae beyond the necrosis (Heath and Wood 1969).

The second phase of infection consists of aggressive mycelium extension which coincides with the diffusion of toxins, enzymes and/or suppressors allowing rapid fungal progression in dead tissues by removing physical barriers (toxins and enzymes) and delaying or suppressing the host defense responses (suppressors). Among toxins, enzymes and suppressors, toxins have received most attention to date, whereas less is known about enzymes and suppressors. All three types of compounds are produced by *A. rabiei* which has been studied most extensively (reviewed by Barz and Welle 1992 and Jayakakumar et al. 2005), and the toxins solanopyrones A, B and C have been isolated from culture filtrates and spore germination fluids (Höhl et al. 1991; Kaur 1995). The role of the phytotoxin ascochitine has been comprehensively studied on faba bean infected with *A. fabae* (Beed et al. 1994), and on pea infected with *A. pisi* (Marcinkowska et al. 1991). Suppressors (reviewed by Shiraishi et al. 1994) and cell wall-degrading enzymes (Heath and Wood 1971a) were isolated and characterised from *M. pinodes* on pea leaves.

Phytoalexins

Phytoalexins with activity against *Ascochyta* spp. have been identified such as medicarpin and maackiain in chickpea (Daniel et al. 1990), and pisatin in pea (Bailey 1969; Heath and Wood 1971b). Experiments have shown that resistant chickpea cultivars infected with *A. rabiei* showed a significantly higher accumulation of phytoalexins than susceptible cultivars (Weigand et al. 1986; Daniel et al. 1990). On pea, Bailey (1969) showed that pisatin concentrations decreased with plant senescence while the tissue became more susceptible to infection by *M. pinodes*, leading Roger and Tivoli (1996b) to conclude that any factor contributing to decreased pisatin concentration and accelerated plant senescence may favour the production of pseudothecia of *M. pinodes*. Roger et al. (1999a) suggested that leaf wetness possibly favours pycnidial development by *M. pinodes* because of a dilution of pisatin. Toyoda et al. (1995) found that exogenously applied plant lectins were able to induce the production of pisatin.

In faba bean, six phytoalexins have been described with activity against germ tubes of primarily *Botrytis cinerea*, and some against *B. fabae* (Hargreaves et al. 1977), but no phytoalexin has been identified with activity against *A. fabae*.

Symptoms

Symptoms develop on all aerial parts of the plant and consist of necrotic lesions. Interestingly, the different ascochyta blight fungi induce similar symptoms except for those of *M. pinodes* and *P. medicaginis* var. *pinodella*. All other *Ascochyta* spp. cause well delineated lesions with clear borders, in the centre of which numerous pycnidia are formed. On the stems, these fungi cause deep necrotic lesions which can lead to breaking of stems and death of plant parts above the affected zone. *Mycosphaerella pinodes* initially produces small lesions in the form of numerous flecks. Leaves with many lesions wither before the lesions become large, especially on the lower portion of the plants. Stem lesions are initiated at the bases of dead leaves and spread above and below that point. They coalesce to encircle the entire lower stem which generally does not break. All species cause necrosis on pods which results in seed infection. Heavily infected seeds have more or less severe discolourations and can shrivel in the most serious cases. *Phoma medicaginis* var. *pinodella* can cause the same necrosis on foliage as *M. pinodes*, but foot-rot is the more typical symptom.

Disease prevalence and yield losses

Yield losses include both weight and quality losses due to seed infection. The difficulty to precisely determine the impact of ascochyta blights on yield is evident in the large ranges reported for yield losses in published papers. In field pea, Bretag et al. (2000) found that disease severity varied considerably between years, regions and fields in the same region and was attributed to differences in climatic conditions and in the availability of inoculum (Bretag 1991). In chickpea, Nene (1981) quoted yield losses ranging from 10–20% to 50–70% depending upon the country and the year. Similar ranges of yield losses have been published for other ascochyta blight diseases. Very few publications list yield loss assessments as an explicit objective, and consequently there is a lack of data collected under well-characterised conditions. As a result, diseases are frequently classified as important or major with a % loss in the introduction, but without any indication of frequency of such losses, or how and under what conditions they were encountered.

A survey of 68 pea fields in Victoria (Australia) in 1986 by Bretag et al. (1995b) showed that yield losses varied from 3.1 to 26.4% with a mean of 18.1%. In field experiments, Ahmed and Morrall (1996) observed seed infections ranging between 12 and 29% among 10 lentil genotypes, between 35 and 43% among five *A. lentis* isolates, and a significant relationship between Area Under the Disease Progress Curve (AUDPC) and % seed infection. On around 150 commercial lentil seed samples collected from four states of the US Pacific Northwest between 1982 and 1985, the levels of infection with *A. lentis* ranged from 0 to 100% between years, and from 7 and 100% between three states (Kaiser and Hannan 1986). On faba bean, Gaunt and Liew (1981) reported incidence levels of *A. fabae* between 0.3 and 12% from 23 fields in New Zealand in 1977–1978 which were correlated with percentages of infected plants.

Apart from climatic factors and the availability of inoculum, yield losses or variation in seed quality also depend upon the pathogen species and the physiological stage of the plants at the time of infection. For example, in plots artificially infested with *M. pinodes*, *P. medicaginis* var. *pinodella* and *A. pisi*, Wallen (1965) showed that *M. pinodes* caused the greatest reduction in yield (45%). Plots inoculated with *P. medicaginis* var. *pinodella* yielded 25% lower and those inoculated with *A. pisi* 11% lower than control plots. In these plots, seed infection rates were 5.0, 15.6 and 16.6% for *P. medicaginis* var. *pinodella*, *M. pinodes* and *A. pisi*, respectively. For *M. pinodes*, a relationship between yield loss and the growth stage of the pea plants at the time of inoculation was demonstrated experimentally. Xue et al. (1997) observed that *M. pinodes* reduced yield of field pea by 31, 24 and 19% in 1994, and 33, 43 and 30% in 1995 when plants were inoculated at 8–10 nodes, mid-flowering and the pod swell stages, respectively. All inoculations reduced seed weight in both years compared to the control.

Survival and primary inoculum

Four main sources of primary inoculum have been described for ascochyta blight diseases: seeds, plant debris, soil and volunteers.

Seeds

Ascochyta spp. are generally considered to be seedborne pathogens, and infected seed can be the most important source of inoculum for long distance spread. Kaiser (1997) reviewed inter- and intra-national spread of ascochyta blight pathogens of chickpea, faba bean and lentil, and showed that seeds were responsible for the introduction of various *Ascochyta* spp. into, and for dissemination within many countries worldwide. Movement of infected seed of these three crops not only leads to the introduction of virulent pathotypes, but may also spread compatible mating types into new areas which can result in the development of the teleomorph. In the case of Canada, Gossen and Morrall (1986) pointed out that seed-borne inoculum was the means by which ascochyta blights of faba bean, chickpea and lentil initially became established in this country.

The penetration of these pathogens into the inner parts of the seeds permits their survival for several years, mainly when seeds are stored at low temperatures. Corbière et al. (1994) determined that *M. pinodes* was able to survive in pea seed for four years. When pea seeds were highly infected by *M. pinodes*, the pathogen was detected in the embryo and pycnidia were observed in internal cotyledons (Moussart et al.

1998). On faba bean, Michail et al. (1983) observed that *A. fabae* could be isolated from the seed coat, cotyledons and embryo axis at the rate of 100%, 46% and 27%, respectively. *Ascochyta rabiei* was located on or in the seed coat, in a very few cases penetrated into the cotyledons, and rarely grew to the embryo. *Ascochyta lentis* was found in 36% of testae and cotelydons from seeds with more than 12% seed discolouration (Morrall and Beauchamp 1988). The mode of transmission from seed to the foliar plant parts was described as non-systemic (Dey and Singh 1994).

The rate of seed-to-seedling transmission determines how many infected seeds develop into infected seedlings and thus can influence the development of an epidemic. Pea seeds infected by *A. pisi* gave rise to only 40% infected seedlings with lesions on stems and the first two leaves, whereas almost all seedlings developing from seeds infected with *M. pinodes* showed symptoms at or below soil level (Maude 1966; Moussart et al. 1998). Low temperatures were found to increase the frequency of transmission in the case of pea (Moussart et al. 1998), but also in lentil (Gossen and Morrall 1986). Corbière et al. (1994) found seed-to-seedling transmission rates for *M. pinodes* of 100% at 15°C compared to 61.5% at 18°C and 70% at 25°C. Although seed-to-seedling frequencies are high in *M. pinodes*, Bretag et al. (1995a) found there was no correlation between the level of seed infection and the severity of ascochyta blight. This lack of correlation between incidences of seed infection and ascochyta blight severity in pea has been attributed to the importance of air-borne ascospores in the epidemiology of this disease. However, seed infection significantly influences seedling emergence.

Field experiments in lentil showed that seed infection with *A. lentis* had a significant negative effect on germination rates (Gossen and Morrall 1986). In experiments under controlled conditions, lentil seed with no visible discolouration, slight discolouration and large lesions due to *A. lentis* had seed-to-seedling transmission frequencies of 0.07, 0.21 and 0.39, respectively (Gossen and Morrall 1986).

With *A. rabiei*, Weltzien and Kaack (1984) observed that out of 95 infected seeds, 32% produced infected plants. Surface inoculation of seed with spore suspensions of 10^9–10^{10} spores of *A. rabiei* ml^{-1} reduced germination rates to 46% compared to 99% in uninoculated seeds, and resulted in 6% healthy seedlings compared to 99% in uninoculated seed lots (Kaiser et al. 1973).

Asochyta fabae is seed-borne and splash dispersed, and the primary source of inoculum is thought to be infected seeds in commercial seed lots (reviewed by Gaunt 1983). Gaunt and Liew (1981) used six seed lots with incidences of seed infection ranging from 0.2 to 12% in field experiments and found disease incidences ranging from 2.6 to 50.0%, incidence of seed infection from 15.0 to 22.0%, and yield from 4.3 to 2.4 t ha^{-1}, respectively.

Infected stubble

In many countries, crop residue has traditionally been buried in the soil by ploughing, a procedure that generally promotes the decomposition of the stubble and thus the destruction of pathogen structures. In some countries, agronomic practices in this respect have changed dramatically, primarily to avoid soil erosion by wind after harvest, and low- or zero-till agriculture is practised where the stubble remains at the soil surface. This change in cropping practice may have had a significant impact on the importance of stubble in the epidemiological cycle in some systems. Two main cases have demonstrated the epidemiological importance of fungal survival in infected stubbles.

In Washington State, USA, the teleomorph of *A. rabiei* develops on chickpea crop residues that remain on the soil surface during winter. The number of ascospores has been estimated to reach about 15,000 mm^{-2} of tissue surface of infested stubble, and spores are released during the vegetative stage of the following chickpea crop (Trapero-Casas and Kaiser 1992a). In Spain, Navas-Cortes et al. (1995) observed that on chickpea debris left on the soil surface under natural conditions, *A. rabiei* rapidly colonized the tissues, formed abundant pseudothecia and pycnidia, and remained viable throughout the two years of their study. When plant debris was buried, *A. rabiei* was restricted to the original lesions and lost viability within 2–4 months (Kaiser 1973). Although Navas-Cortes et al. (1995) often observed brown, thick-walled, swollen hyphae associated with fruiting structures in the infected tissues, they were unable to distinguish specialized fungal survival structures such as chlamydospores or sclerotia on either surface or buried debris as described for *A. pinodes* and *P. medicaginis* var. *pinodella* (Table 1). These structures are important allowing these species to survive in the soil for more than one year after the complete destruction of host tissues. Gossen and Morrall (1986) observed that *A. fabae*, *A. rabiei* and *A. lentis* survived at the soil surface for at least one winter season. Steep ascochyta blight gradients were modelled in lentil fields by Pedersen et al. (1993) who found that one-year old residue and volunteers present at the field border affected the new crop up to 50 m into the lentil field.

In the case of *M. pinodes* under growing conditions in Australia, pseudothecia are formed on crop residues from the previous year and the concentration of air-borne ascospores is highest in late autumn to early winter when pseudothecia mature and first become productive (Bretag 1991; Peck et al. 2001).

Soil

Few reports have investigated the behaviour of *Ascochyta* spp. in the soil. Some detailed studies have been conducted only on the species involved in the ascochyta blight complex of pea. The ability of soil cultures of *P. medicaginis* var. *pinodella* and *A. pinodes* to form chlamydospores was considered to be a major factor for their survival, whereas no chlamydospores were formed in soil cultures of *A. pisi* (Wallen and Jeun 1968). The authors observed that chlamydospores enabled these fungi to survive for at least 12 months in sterilised soils. Wallen et al. (1967) reported that *P. medicaginis* var. *pinodella* was present in most soils where peas were grown in eastern Canada, and also in some soils where peas had not been grown for one to five years. *Ascochyta pinodes*, in comparison, was isolated less frequently from these soil samples. Studying *A. pinodes* survival in soil and aerial dissemination through the pea cropping season, Peck et al. (2001) observed that infection from stubble was initially high but dropped to low levels after one year, while infection from soil inoculum declined slowly over three years.

Volunteers

Volunteers have sometimes been indicated in the transmission of inoculum from field borders into the fields. The role of the volunteer plants has been well described for faba bean. For this crop, Bond and Pope (1980) found a distinct gradient of ascochyta blight-infected winter bean plants from the border to the centre suggesting that spread from volunteer plants in adjacent fields was a much more important source of infection than infected seed. However, debris from previous crops may also be important (Lockwood et al. 1985).

In general, the importance of the different sources of primary inoculum may vary depending upon the species, cropping practices and climatic factors. For example, based on the distribution of primary symptoms on upper parts of the chickpea plants, Milgroom and Peever (2003) concluded that *D. rabiei* ascospores were the dominant type of primary inoculum, and were more important than infected seeds in the northwest of the USA. In contrast, in Canadian and Australian chickpea fields, infected seeds and chickpea stubble are considered to be the primary sources of inoculum, although airborne ascospores are of relevance for long-distance dispersal (Chongo et al. 2004; Pande et al. 2005).

Disease development

Ascochyta blights are polycyclic and secondary cycles are generally due to a succession of pycnospores released from plant tissue to tissue, or from plant to plant. Researchers have generally described five to ten cycles of pycnidiospore production and re-infection during the cropping season which results in a progression of the disease along the plant from the base to the top. Leaves or leaf axils tend to be the first plant tissues that are infected, followed by stems, pods and seeds. It is only in the case of *M. pinodes* that ascospores are involved in secondary cycles of infections.

Different phases of the disease cycle

The epidemiological development of ascochyta blights can be considered as a race between the host plant which grows and develops depending on environmental conditions, and the pathogen which may infect the plant at the early stage of development at the base of the plant and from there continues to spread to upper plant parts as they develop. As a consequence, for some crops like faba bean and pea, ascochyta blight severity is generally very high on the lower parts of the plants, but can be very low on the uppermost parts where plant tissue has escaped infection until that time.

Maurin and Tivoli (1992) described this epidemiological pattern in three phases for winter faba bean starting with the first lesions caused by *A. fabae* until seed infection has occurred:

(1) Disease initiation is characterised by the appearance of a few lesions on seedlings during the winter. The fungus spreads from plant to plant and disease incidence increases rapidly. The cold, wet weather during crop establishment is usually favourable to pathogen development on slow-growing seedlings.

(2) The subsequent phase of plant infection starts at the end of the winter when plants begin to grow more rapidly. Lesions, initially limited to the foliage, develop on stems and spread to the top of the plant. The epidemic spreads on foliage from node to node. Disease severity may be reduced when the climatic conditions are unfavourable for spreading (very little rainfall) while the number of foliar nodes increases with plant growth. Hence, plants may escape a severe attack temporarily because plant growth is significantly faster than the progression of the pathogen. During this second epidemic phase, the spread of the fungus strongly depends on rainfall, but it is very likely that disease severity during this phase also depends on inoculum pressure, a consequence of the initial infection.

(3) During the pod infection phase the pathogen infects those pods that develop on infected nodes. Conidial dispersal by splashing during rain facilitates the contamination of the lowest pods. This third phase takes place when plant growth slows down after the flowering stage, and during pod initiation and filling. At this growth stage of the plant, even light disease pressure can induce severe damage because pod infection causes infection of the seeds.

This epidemiological cycle can be generalised for different legume crop species. Regarding the respective roles of ascospores and pycnidiospores, three scenarios of increasing complexity are described among the grain legume species considered.

In the first scenario (Fig. 1), the epidemic is exclusively based on successive pycnidiospore cycles. This scenario is characteristic for epidemics caused by *A. pisi*, *A. lentis*, *A. fabae*, *P. medicaginis* var. *pinodella* and *A. rabiei* (under certain growing conditions). Primary inoculum can be infected seeds (for all of these species), infected debris and in some cases chlamydospores in the soil, or ascospores (for *A. fabae, A. lentis* and *A. rabiei*). For some of the species, the sexual form has been identified but the epidemic role of ascospores is not evident or unknown in the epidemiological cycle. For example, in ascochyta blight on lentil, the commonly described disease cycle observed in Canadian lentil fields is only based on pycnidiospore dispersal during the cropping period and ascospores are not present (Morrall 1997). In contrast, in Australia (Galloway et al. 2004) and in the northwest of the USA (Kaiser and Hellier 1993), the teleomorph has been detected in the field on lentil straw. Similarly, Porta-Puglia (1990) described *A. rabiei* epidemics for the Mediterranean basin caused by pycnidiospores which are dispersed by rainfall and intensified by wind. However, the life-cycle of *A. fabae* on faba bean described by Jellis and Punithalingam (1991) includes the teleomorph *D. fabae*.

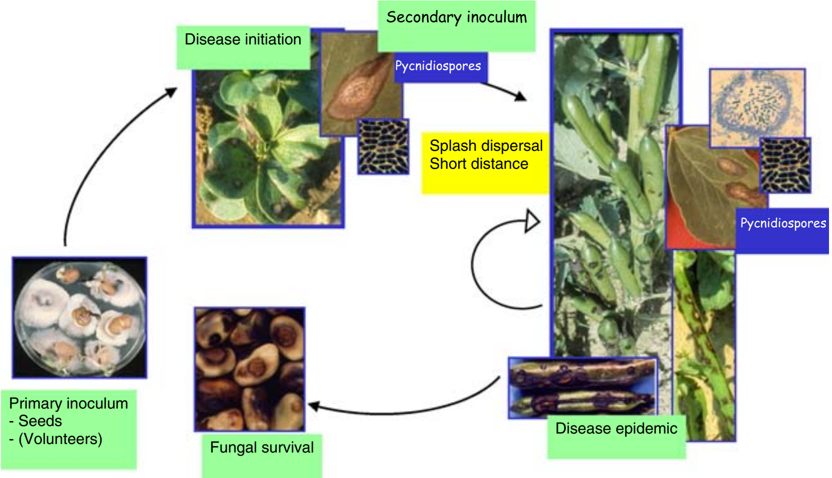

Fig. 1 Disease cycle of ascochyta blight (*Ascochyta fabae*) on faba bean

In the second scenario (Fig. 2) the epidemic is based on both successive ascospore discharges as primary inoculum and successive pycnidiospore cycles. This scenario has been described for epidemics caused by *A. rabiei* and *M. pinodes* when infected stubble remains at the soil surface. Pseudothecia are formed on dead tissues at the end of the cropping season and can constitute an important source of primary inoculum by successive discharges in the following crop at the end of winter and during spring. Trapero-Casas et al. (1996) and Peck et al. (2001) found that ascospores were trapped mostly during winter and spring, thus confirming that this is the period of pseudothecial maturation.

For ascochyta blight on chickpea, Trapero-Casas and Kaiser (1992a) pointed out the important role of ascospores in the disease cycle in the Palouse region of eastern Washington and northern Idaho, USA and stressed the necessity to consider ascospores on chickpea straw. To conserve moisture and reduce soil erosion in those regions, infested chickpea debris remains on the soil surface, thus favouring the development of pseudothecia of *D. rabiei* during the fall and winter months. The first vegetative period of crop development was shown to overlap with the second half of ascospore release. Kaiser (1997) described the life cycle of ascochyta blight of chickpea where both pycnidia and pseudothecia may develop on overwintered chickpea debris.

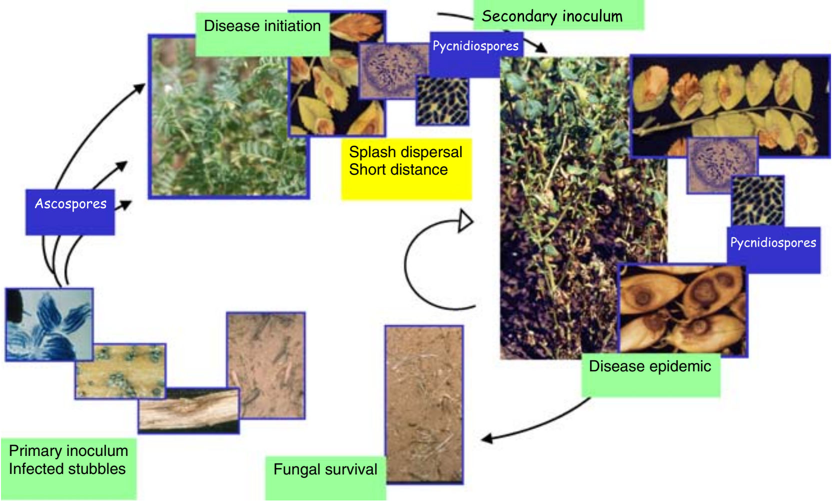

Fig. 2 Disease cycle of ascochyta blight (*Didymella rabiei*) on chickpea

In the third scenario (Fig. 3), the epidemic is based on both ascospores discharges as primary and secondary inoculum, and successive pycnidiospore cycles. This scenario describes epidemics caused by *M. pinodes*. Primary inoculum consists of ascospores in addition to infected seed and plant debris. However, pseudothecia are also formed alongside pycnidia during the cropping season; consequently ascospores are released during the entire season and constitute an important source of secondary inoculum.

Roger and Tivoli (1996b) showed that the disease cycle of *M. pinodes* started with the dissemination of ascospores after which pycnidia developed rapidly in lesions on stipules, on green plant tissue or on senescent tissue. The number of pycnidia was highly correlated with disease severity. Pycnidiospores dispersed by rain splash are responsible for secondary infections over short distances and further increases in disease severity, thus accelerating tissue senescence. As a consequence of this early tissue senescence, an early production of pseudothecia is initiated which are only produced on senescent tissues. This also explains why pseudothecia are present predominantly at the bases of pea plants. Their role seems to be essential in the epidemic because they contribute to increased inoculum concentration and disease severity, and thus accelerate the epidemic cycle. After rainfall, pseudothecia release ascospores which are dispersed over long distances by wind. In trials, pycnidiospores were principally trapped in the first 20 cm above the soil surface whereas ascospores were also trapped above the crop canopy. The formation of fruiting bodies progresses from the base to the top of the plants during crop development. Frequently, pycnidia and pseudothecia are simultaneously present on the same stipule. This is unusual because the sexual stage most commonly follows the asexual stage in plant pathogenic fungi (Agrios 2004).

Environmental and climatic factors

Temperature and moisture after inoculation are probably the two key environmental factors in the epidemiology of ascochyta blights on legumes and have a major influence on the length of the incubation and latent periods.

Under optimal temperatures and leaf wetness, the incubation period for *M. pinodes* was shown to be 1–2 days (Roger et al. 1999a), 5 days for *A. pisi* (Heath and Wood 1969) and *A. rabiei* (Trapero-Casas and Kaiser 1992b), and 6 days for *A. lentis* (Pedersen and Morrall 1994). The latent period for *M. pinodes* was 3–4 days (Roger et al. 1999a), 5–6 days for *A. rabiei* (Trapero-Casas and Kaiser 1992b), 6–7 days for *A. lentis* (Pedersen and Morrall 1994), 8–10 days for

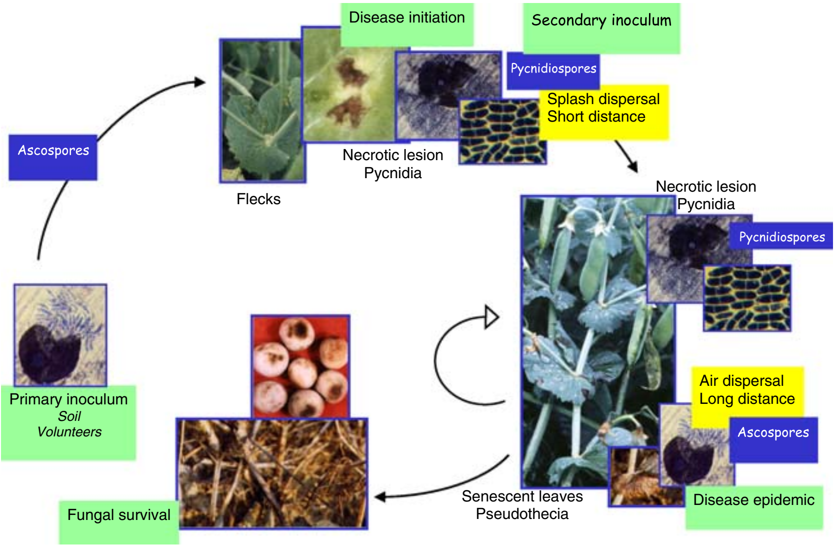

Fig. 3 Disease cycle of ascochyta blight (*Mycosphaerella pinodes*) on pea

A. fabae (Wallen and Galway 1977) and 10 days for *A. pisi* (Heath and Wood 1969).

In lentil, temperature had little effect on lesion size and number of pycnidia per lesion, but infection frequency was higher at 10 and 15°C than at 25°C (Pedersen and Morrall 1994). Trapero-Casas and Kaiser (1992b) observed that at 20°C, 7.6 and 17 h of wetness were required for *A. rabiei* to cause light and severe infection, respectively. At temperatures lower or higher than 20°C, significant infection was only observed after longer periods of wetness. At the optimum temperature of 20°C, pycnidiospores of *M. pinodes* germinated within 2 h, appressoria developed after 6 h and the penetration peg invaded the leaf after 8 h (Roger et al. 1999a). This process was slowed down or stopped as a result of intermittent dry periods (Roger et al. 1999b).

Spore dispersal

It has been recognized that spore dispersal has a major impact on the onset and development of epidemics. Pycnidiospores are dispersed by water-splashing that restricts their spread to short distances, except in cases where fine water droplets containing spores are picked up by air currents and transported over longer distances. However, long-distance spread in general occurs when airborne ascospores are produced and moved by air currents and wind.

Pycnidiospore release by splashing is the common characteristics of spore dispersal for all the ascochyta blight pathogens. This phenomenon has been well described for ascochyta blights of lentil, faba bean, chickpea and pea. For example, Pedersen et al. (1993) found that although less than 50% of pycnidiospores of *A. lentis* were spread further than 15 cm from their place of origin, and only very few to distances up to 70 cm, strong winds could carry them to over distances as far as 100 m. The movement of spores in aerosol droplets was described by Pedersen and Morrall (1995) to explain greater horizontal spread of ascochyta blight when strong winds occurred during rainfall. This led Morrall (1997) to the conclusion that wind had a major influence on the spread of ascochyta blight in lentil despite the absence of air-borne ascospores. On pea, Roger and Tivoli (1996b) showed that pycnidiospores of *A. pinodes* were dispersed by rain-splash to a maximum of 30 cm above the soil surface, with the result that this zone had highest infection levels.

Ascospore release has mainly been described for *D. rabiei* and *M. pinodes*. Trapero-Casas and Kaiser (1992b) reported that new infection foci in chickpea fields were often located at distances of 10–15 km from the nearest chickpea field infected with ascochyta blight suggesting airborne ascospore dissemination as primary inoculum. Spore trapping revealed that ascospores were trapped on rainy days during daylight, and 70% of those were trapped between 12:00 and 18:00 h. This observation was confirmed by Gamliel-Atinsky et al. (2005) who used chickpea plants as living traps in the field to show that ascospore dispersal was dependent on successive rain and dry periods. Extensive spore-trapping experiments in peas infected with *M. pinodes* revealed that the majority of ascospores was captured within the canopy and only a small proportion escaped beyond the boundaries of the canopy (Roger and Tivoli 1996b). This demonstrated that as the canopy develops it becomes a barrier to ascospore dispersal, probably because air circulation in the canopy is reduced.

Cultural factors and host resistance

With the exception of stubble disposal and fungicide applications, which will not be discussed here further, few data have been published on the role of cultural factors such as seeding date and plant density on ascochyta blight development.

Early seeding of Australian pea crops in May (late autumn) resulted in higher infection rates of plants and seeds by *M. pinodes* and lower yields compared to late-seeded peas in July because of higher levels of primary ascospore inoculum and longer periods of leaf wetness (Bretag et al. 1995a, 2000). Similar observations were reported for *A. rabiei* on chickpea (Trapero-Casas et al. 1996). On pea, Tivoli et al. (1996) demonstrated that seed densities of 30, 60, 90 and 120 seeds m^{-2} resulted in percentages of infected stem by *M. pinodes* of 34.7, 41.8, 50.1 and 53.7%, and yield losses of 24.7, 37.4, 40.6 and 46.4%, respectively.

Host resistance is considered to have the greatest effect on ascochyta blight epidemics. Although poorly understood, pulse crops appear to become more susceptible to infection by *Ascochyta* spp. with increasing plant age. For example, *M. pinodes* was shown to be more aggressive on old tissues which was correlated with decreasing phytoalexin concentrations in the tissue (Heath and Wood 1971b). Similarly, partially resistant chickpea cultivars were shown to lose resistance to *A. rabiei* with increasing plant age (e.g., Trapero-Casas and Kaiser 1992b; Singh and Reddy 1993; Chongo and Gossen 2001), but it was also suggested that newly developed plant tissue on these plants showed higher resistance than older tissue (Chongo and Gossen 2001). In lentil, in contrast, Pedersen and Morrall (1994) observed that tissues below the top four or five nodes on the main stem and secondary branches were almost completely resistant suggesting that resistance increased as tissue matured.

Studying the effect of resistance in lentil on the infection process of *A. lentis*, Pedersen and Morrall (1994) and Ahmed and Morrall (1996) found that although the incubation periods were similar among lentil cultivars, AUDPC, the number of lesions, lesion length and width and the number of pycnidia per lesion were negatively correlated with the level of resistance in the cultivars. Similar observations have been made on pea and faba bean (Maurin and Tivoli 1992; Prioul et al. 2003).

Differences in plant architecture have also been implicated in influencing ascochyta blight epidemics by modifying the microclimate in the canopy. For example, faba bean stem length was found to be negatively correlated with pod infection by *A. fabae* and moisture levels of the soil surface, and pods higher up on taller stems escaped infection more readily than pods on short stems (Jellis et al. 1985). Resistance to *M. pinodes* was found to be positively correlated with lodging resistance, and both lodging and mycosphaerella blight were negatively correlated with the proportion of xylem, lignin and fibre content of pea stems (Banniza et al. 2005). Le May et al. (2005) developed a simulation model for the growth of pea infected with mycosphaerella blight by incorporating architectural features such as stem height, branching ability and lodging resistance into the model.

Modelling

Modelling has the objective to formalize by means of mathematical equations the knowledge on disease epidemiology. This leads either to a description of all or some segments of an epidemic with the objective to understand its mechanisms, or to forecast the risk of appearance or development of the disease in relation to factors like temperature, rainfall, and crop rotations. Models can therefore become an important and integral part of Integrated Disease Management (IDM). As a first step, disease epidemics have often been described in terms of temporal and spatial models to describe disease progress. More complex models have been separated into two groups, mechanistic models and empirical models. Mechanistic models are based on a concept or hypothesis about the mechanism of the interaction and therefore require prior knowledge of the variables involved. Empirical models, in contrast, do not require prior knowledge about the variables and look at the best mathematical fit of the model to the data, e.g., temperature, rainfall, and crop rotation. Both types of models have been used to describe diseases of grain legumes.

Quantitative descriptions of temporal and spatial developments of ascochyta blights are few. A comprehensive study of temporal and spatial dynamics of mycosphaerella blight in Canada was published by Zhang et al. (2004). Disease progress in space and time was well described by logistic models. Steepest disease gradients were identified upwind at the end of the growing season. Geostatistical analysis of spatial patterns revealed differences in disease severity depending upon geographic directions attributed to different wind speed and direction.

For ascochyta blight on lentil, Pedersen and Morrall (1994) established regression equations which predict incubation and latent periods at different temperatures. Using a mechanistic approach, ascochyta blight severity of chickpea was described as a function of temperature and of the natural logarithm of the length of the wetness period, predicting that approximately 20°C was the optimum temperature requiring the shortest wetness period (Trapero-Casas and Kaiser 1992b). For severe infection, a minimum of 7.6–10.3 h of wetness was required at a temperature range of 15–25°C based on this model. Similarly, polynomial equations were used to predict incubation period, latency of *M. pinodes* and ascochyta blight severity on pea based on temperature and wetness period (Roger et al. 1999a).

Using and building upon a disease-coupled crop growth model published by Béasse et al. (2000), Le May et al. (2005) developed an improved model to predict the impact of ascochyta blight in pea on yield components by incorporating a combination of disease progression in the canopy (number of nodes affected by the disease) and the structure of the canopy (leaf area index profile). For doing so, they first estimated the contribution of each node to radiation absorption, then calculated the reduction in contribution of each node due to disease and finally combined the individual contributions which allowed them to estimate crop growth. Using data from six varieties they showed a good fit between estimated and observed values.

It is surprising to observe the low number of forecasting models for ascochyta blights on grain legumes. The few forecasting models that have been described only permit prediction of one or two phases of the epidemic, such as disease appearance, disease development, fungal reproduction or spore release. For *M. pinodes* on pea, Salam et al. (2006) established a model using historical weather data to forecast disease development under different weather scenarios. The prediction of ascospore release proved to be critical in determining seeding dates in order to avoid spore deposition on the newly establishing crop. Using data from fifteen pea cultivars, Bretag et al. (1995b) developed empirical crop loss models that allowed yield loss to be estimated based on disease severity.

Gamliel-Atinsky et al. (2005) confirmed that pseudothesial formation and maturation of *D. rabiei* required low temperature and moisture periods, based on which in combination with other published data Shtienberg et al. (2005) developed an empirical model to forecast pseudothecial maturation.

Conclusion

This review on the epidemiology of the various *Ascochyta* spp. in pulse crops highlights common and specific features of the different ascochyta blights. The common features can be summarized as (1) all species produce pycnidia, (2) except for *A. pisi*, the teleomorph is present, (3) the role of infected seed as primary inoculum is significant, (4) they form the same types of symptoms except for *M. pinodes* and *P. medicaginis* var. *pinodella*, (5) plant compounds like phytoalexins and pathogen toxins appear to be involved in the host-pathogen interactions, (6) ascochyta blights are polycyclic diseases and epidemics develop on leaves, stems, pods and finally seeds, (7) temperature and moisture are the two primary environmental factors affecting disease development, (8) pseudothecia generally are formed at the end of the cropping cycle (except for *M. pinodes*) and are implied in inoculum survival. Species-specific features are (1) the type of symptoms caused by *M. pinodes*, (2) the length of incubation and latency periods which are different among the different ascochyta blights, (3) the role of infected stubble as primary inoculum in some parts of the world, (4) the involvement of pseudothecia of *M. pinodes* as secondary inoculum.

Reviewing the literature revealed several areas where there is a clear lack of data:

(1) The exact involvement of the soil as primary inoculum is unclear, but we can expect that molecular detection tools for pathogens from soil will improve our knowledge.
(2) The timing of primary inoculum deposition on the crop is difficult to assess and techniques other than trap plants and spore traps are lacking to easily estimate inoculum quantities above the plant canopy.
(3) There is a general lack of understanding of the host-pathogen interactions, at the microscopic level to some degree, but more so at the biochemical and molecular levels. With the exception of *A. rabiei*, the majority of papers, in particular, on biochemical aspects

of the systems are 20–40 years old, and it can be expected that modern tools of molecular biology and biochemistry could have a significant impact on our ability to investigate these host-pathogen systems.

(4) Information on the role of cultural factors on ascochyta blight epidemics is sparse in the published literature. Surprisingly little information has been published that could improve integrated disease management of these diseases.
(5) Mechanistic modelling for life-cycles of all the pathogens and for the epidemics during the cropping period is missing.
(6) More forecasting models are required that establish simple relationships between climatic and epidemic events and that are easy to implement.

When comparing the well-researched areas in ascochyta blight epidemiologies with those where there are obvious gaps in our knowledge, it becomes obvious that the latter are of equal importance, but appear to have been neglected because of lack of research concepts, tools or resources. Clearly, several of these areas would benefit greatly from the use of molecular tools and the application of modern statistical methodology. It is also apparent that some aspects of ascochyta blight research was conducted decades ago, and although still of considerable importance, would benefit from reassessments using modern tools and techniques. On the other hand, some more traditional research areas seem to have been neglected almost completely in favour of molecular research. For example, it seems surprising that to date there is insufficient data to answer the fundamental question of whether *Ascochyta* spp. are hemiobiotrophs or necrotrophs. There seems to be an urgent need to boost new research initiatives in the area of epidemiology of ascochyta blights which combine traditional epidemiological strategies with new tools provided by molecular biology and biochemistry to elucidate the mechanisms of these host–pathogen interactions. Knowledge and data of that nature are essential to make progress in the development of quantitative mechanistic models, but will also assist in resistance breeding by providing an understanding of the complexity of the interaction. Beyond that, there is an obvious gap in applied research which investigates the role and sources of primary inoculum and cultural factors to develop and improve current integrated disease management strategies.

Research on the epidemiology of *A. rabiei* and *M. pinodes* is most comprehensive and advanced, and may serve as an example and an inspiration for the other species as well as for each other. In *M. pinodes* the most detailed information has been gathered on disease development, the initiation, location and dispersal of various dispersal structures, and the effect of disease development on the plant in terms of photosynthetic activity as well as yield formation. A comprehensive review specifically on the epidemiology and control of ascochyta blight on field pea was recently published by Bretag et al. (2006). Research on this species has greatly benefited from concentrated long-term studies primarily in France and Australia whereas research efforts on other species has been either more limited in scope due to limitations in resources (e.g., *A. lentis*), or has been scattered across various countries, diverse climates and cropping systems which has hampered the transfer and application of research results in a more comprehensive manner (e.g., *A. rabiei*). However, research on the advance of *M. pinodes* in, and its interaction with the host at the microscopic, biochemical and molecular level is sketchy. Ascochyta blight of chickpea, in comparison, is much better understood in those areas whereas comprehensive field epidemiological studies comparable to those conducted for *M. pinodes* are lacking. It can be speculated that filling those gaps for both of these pathogens may lead to major advances in disease management: a better understanding of the host-pathogen interaction of *M. pinodes* on pea may give fresh impetus to the breeding of resistant pea cultivars which appears to have stagnated, but which could significantly simplify asochyta blight control in this crop. On the other hand, a more comprehensive understanding of the epidemiology of *A. rabiei* could result in better disease management strategies that are urgently needed in countries like Canada and Australia. The elegant disease forecasting system developed and utilized in Israel (Shtienberg et al. 2005) could be a starting point for studies in other countries to clarify whether, to what degree and under which conditions ascospores induce ascochyta blight on chickpea. This would then determine whether the underlying model could be adapted and adopted elsewhere to prevent primary infection of the chickpea crop by targeted fungicide sprays to kill the ascospores early in the season. It is obvious that in systems where infected seed, stubble, pycnidia and potentially ascospores can initiate ascochyta blight, disease forecasting is bound to become more complex as exemplified by the models developed for *M. pinodes* in France (Béasse et al. 2000; Le May et al. 2005). It is probably safe to say that for those systems we may not know enough about the relative importance of each of these sources, which may be highly variable depending upon the location and year. It is unlikely that the prevention of primary infection can be achieved in such systems; hence models are required which describe the entire life-cycle of these pathogens in response to environmental factors. Also, some of the species are thought to occur and cause damage in the anamorphic phase only, but experiences with *A. rabiei* and *A. lentis* in various parts of the world have shown that a focused attempt has to be made to truly determine whether ascospores can be excluded from the life-cycle of these organisms.

Among world crops, grain legumes play a minor role and consequently research on these crops and their pathogens is bound to be restricted by fewer resources and researchers. This, in combination with the diversity of skills and knowledge required to tackle those gaps in ascochyta blight epidemiology outlined here, should present a strong incentive for future international collaborations.

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