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REVIEW

Integrated disease management of ascochyta blight in pulse crops

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Abstract Ascochyta blight causes significant yield loss in pulse crops worldwide. Integrated disease management is essential to take advantage of cultivars with partial resistance to this disease. The most effective practices, established by decades of research, use a combination of disease-free seed, destruction or avoidance of inoculum sources, manipulation of sowing dates, seed and foliar fungicides, and cultivars with improved resistance. An understanding of the pathosystems and the inter-relationship between host, pathogen and the environment is essential to be able to make correct decisions for disease control without compromising the agronomic or economic ideal. For individual pathosystems, some components of the integrated management principles may need to be given greater consideration than others. For instance, destruction of infested residue may be incompatible with no or minimum tillage practices, or rotation intervals may need to be extended in environments that slow the speed of residue decomposition. For ascochyta-susceptible chickpeas the use of disease-free seed, or seed treatments, is crucial as seed-borne infection is highly effective as primary inoculum and epidemics develop rapidly from foci in favourable conditions. Implemented fungicide strategies differ according to cultivar resistance and the control efficacy of fungicides, and the effectiveness of genetic resistance varies according to seasonal conditions. Studies are being undertaken to develop advanced decision support tools to assist growers in making more informed decisions regarding fungicide and agronomic practices for disease control.

Keywords Chickpea · Faba bean · Fungicide · Field pea · Infected seed · Lentil · Infested residue · Resistance · Rotation · Seed dressing

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Introduction

Ascochyta blight is the most severe foliar disease of cool season pulses, the major crops being chickpea, faba bean, lentil and field pea, and severe epidemics may result in total crop failure. Pathogens that cause ascochyta blight belong to *Ascomycota;* they have worldwide distribution and are predominantly host-specific. *Ascochyta rabiei* (teleomorph: *Didymella rabiei*), *Ascochyta lentis* (syn. *A. fabae* sp. *lentis*) and *Ascochyta fabae* (teleomorph: *Didymella fabae*) infect chickpea, lentil and faba bean, respectively. Ascochyta blight of field pea is caused by a complex of three fungal species; *Ascochyta pinodes* (teleomorph: *Mycosphaerella pinodes*), *Ascochyta pisi* and *Phoma medicaginis* var. *pinodella*, formerly known as *Ascochyta pinodella*. This highly efficient group of pathogens undergo heterothallic sexual reproduction

on infested residue, resulting in air-borne ascospores, which are capable of spread over long distances. Rapid polycyclic spread within crops occurs over short distances through splash-borne asexual conidia (pycnidiospores). The disease affects all above ground parts of the plant and is characterised by necrotic lesions, which on susceptible cultivars in favourable conditions, can girdle stems leading to breakage and severe yield reduction. Seed quality may also be reduced through seed discolouration or retardation of seed development.

Significant improvements in host resistance are being realised in breeding programmes and a greater understanding of integrated disease management options can reduce the incidence, severity or persistence of ascochyta blight. Nevertheless, control of this aggressive disease continues to challenge pulse industries and researchers worldwide, and ascochyta blight epidemics continue to depress yields.

This review summarises the current knowledge of the management of ascochyta blight in the pulse crop. Managing ascochyta blight firstly relies on minimising the onset of disease epidemics by reducing or avoiding primary inoculum, and secondly by suppressing the subsequent epidemic increase using resistance or foliar fungicides. Methods of control include destroying or avoiding inoculum sources, crop rotations, manipulating sowing times, sowing disease-free seed, applying seed and foliar fungicides and adopting cultivars with improved resistance. The combination of strategies is determined by economics, availability of cultivar resistance and disease epidemiology.

Avoiding infested residue and in situ inoculum

Ascochyta fungi survive on infested crop residue lying on the soil surface and for a reduced period of time on buried residue. Asexual reproduction on residue gives rise to pycnidia, which exude pycnidiospores, spread via rain splash, whereas sexual reproduction forms pseudothecia, which discharge ascospores capable of spread over long distances by wind and rain. *Mycosphaerella pinodes* and *P. medicaginis* var. *pinodella* can also produce chlamydospores, long-term soil-borne survival structures that may persist for at least 5 years (Wallen and Jeun 1968), and pea crops become infected if they are planted in soils containing this soil-borne inoculum. The management of infested residue and soils is an important component of controlling ascochyta blight. Where ascospores are the major source of infection, crop rotation is less effective, and crop isolation and residue burial will be more beneficial.

Proximity to infested residues

Isolation from infested residue is an important strategy in all cool-season pulse crops to avoid ascochyta diseases. Ascospores are wind-dispersed and may spread long distances: at least 400 m in the case of *M. pinodes* (Davidson et al. 2006; Galloway and MacLeod 2003) and 100 m in the case of *A. rabiei* (Trapero-Casas et al. 1996) though the distances may be greater if spores are blown in air currents (Kaiser 1992). In addition to ascospores, infested residue may be blown into neighbouring crops. In Australia, crop residues are considered the most important source of ascochyta inoculum for faba bean (Hawthorne et al. 2004) and field pea crops (Bretag et al. 2006; Carter and Moller 1961). Disease gradients across crops clearly indicated that wind-blown spores or infested debris from neighbouring crop residue acted as primary inoculum in lentil crops in Canada (Morrall 1997) and bean crops in the UK (Bond and Pope 1980). In the latter study, a decreasing frequency of ascochyta blight on beans, from the border to the centre of the field, for a distance of 120–200 m, suggested that spread from adjacent fields was more important than seed infection, whereas previously most outbreaks in the UK and Canada had been attributed to seed infection (Hewett 1973).

Burial of infested residue

Burial of debris hastens residue and pathogen decomposition thereby reducing the inoculum loads. *Ascochyta rabiei* inoculum on buried chickpea residue is no longer viable after 2–5 months. In contrast, inoculum is still viable on residue on the soil surface after 2 years (Gossen 2001; Kaiser 1973; Navas-Cortes et al. 1995; Nene and Reddy 1987). Zhang et al. (2005) found that *M. pinodes* spore production from buried pea residues rarely continued after 11 months regardless of depth of burial, but higher numbers were produced on residues on the soil surface. Similar results were found in Australia

(Davidson et al. 1999). Decomposition is aided by environments of high temperatures and adequate moisture but in extreme environments of less than −40°C or more than +40°C, such as in Canada, residue breakdown is inhibited. In studies examining survival of *A. rabiei* (Gossen and Miller 2004) and *A. lentis* (Gossen 2001) on infested residues, the pathogens were able to survive when buried within the soil profile for more than 4 years, albeit at a low level of pathogen recovery. Hence two or even three non-host crops are needed between successive chickpea or lentil crops to reduce the risk of an epidemic developing (Gossen and Derksen 2003; Gossen and Miller 2004). However, in the Pacific Northwest of USA the pathogens survive for a shorter period of 3 years on buried residue (Kaiser and Hannan 1986). These differences indicate that regional environments influence the speed of residue decomposition, rather than directly impacting on pathogen survival. Nevertheless, burying residue reduces the spread of pycnidiospores and ascospores by preventing exposure for splash or wind dispersal.

Burying residue may reduce spore production and hasten decomposition but it is incompatible with no or minimum tillage practices. In addition, even after several passes with tillage equipment, some residue remains on the soil surface (Gossen and Miller 2004). Burning residue is another tool to effectively destroy inoculum but has also become less popular in many regions due to environmental concerns. The increasing amount of plant residue left on the soil surface with minimum tillage is thought to be a potential hazard for increasing the severity of epidemics, and alternative means of suppressing the pathogens are required. Studies are underway to investigate the potential of using biological control to suppress *A. rabiei* on chickpea residue, concentrating on fungal colonisers such as *Aureobasidium pullulans* and *Clononstachys rosea* (Dugan et al. 2005).

Soil borne inoculum and crop rotation

The recommended interval between like pulse crops to minimise ascochyta infection is governed by the speed of residue breakdown. Crop rotation between 3 and 6 years is recommended in most regions to avoid *in situ* inoculum, while in warm, moist areas of the world, rotations of 1 or 2 years with a non-host is sufficient (Kaiser et al. 2000). The pathogens may survive directly on the residue of previous crops, which in many environments will decompose much quicker if buried.

However, the causal pathogens of ascochyta blight on field pea (*M. pinodes* and *Phoma medicaginis* var. *pinodella*) can survive in soil as mycelium or chlamydospores (Hare and Walker 1944; Wallen and Jeun 1968) and *M. pinodes* is a moderately successful saprophyte (Dickinson and Sheridan 1968). The longevity of these structures influences the period that is required between pea crops. In Australian farming systems, rotation interval between pea crops has recently increased from 3 to 5 years, to avoid infection from in situ inoculum. Disease severity was greater in crops sown on shorter rotations compared to those on longer rotations and yield, based on grower data, was consistently lower in the shorter rotation crops (Davidson and Ramsey 2000). Bretag et al. (2001) monitored changes in populations of soil-borne ascochyta blight fungi, following different cropping sequences of field pea and barley. Inoculum levels were twelve times higher following 3 years of field pea compared to 3 years of barley. Yield losses of field pea sown in the fourth year were highly correlated to the level of soil borne fungi. Similar studies in the USA found that *P. medicaginis* var. *pinodella* could be isolated from soil that had not been sown to pea for up to 5 years, while *M. pinodes* was isolated from soils that had not grown pea for over 20 years (Wallen and Jeun 1968). These results bring into question the effectiveness of a three-year rotation between pea crops to reduce ascochyta blight. Davidson et al. (2001) investigated survival of ascochyta blight pathogens in soils of commercial pea-cropping paddocks. While soil populations were found to degrade over time, the pathogen population levels varied widely between paddocks with the same paddock history. Hence relying on a simple paddock rotation may not be sufficient since crops could be planted in soils with potentially damaging levels of pathogens. It is likely that the level of pathogen populations in the soil is related to the severity of the epidemic in the last pea crop grown.

Studies on the survival of *A. fabae*, from soil samples taken to a depth of 5 cm, concluded that this pathogen does not survive for even a few months directly in field soil (Wallen and Galway 1977). This is probably due to the inability of the pathogen to

form chlamydospores, making it dependent upon the presence of infested residue for survival. While viable inoculum remains on infested residue in the field, rotations are still a primary means of disease control in faba bean and a three-year rotation is recommended in Australian conditions (Hawthorne et al. 2004). Residue is also regarded as an important source of inoculum for *A. fabae* in Iraq (Michail et al. 1983).

Sowing date

Ascospores are released into the air from infested residue at certain times of the year, depending on environmental conditions, and sowing date of crops can be manipulated to avoid the maximum risk period when airborne ascospore are at their highest numbers.

In Australia, pea crops are sown two to three weeks after the agronomic optimum to avoid the peak period of ascospore release which occurs at the beginning of the growing season (Bretag 1991). Earlier sown crops have the most ascochyta and the highest percentage of infected grain at harvest (Bretag et al. 2000) particularly in the most intense pea cropping areas (Davidson and Ramsey 2000). In higher rainfall areas later planting has less impact on yield (Davidson and Ramsey 2000), but this practice risks yield loss in short growing seasons and regions where spring rain is limiting, with losses as high as 40% in some later-sown crops (Bretag et al. 2000).

This situation also occurs in chickpea where the maximum ascospore numbers may coincide with emergence of chickpea crops (Trapero-Casas et al. 1996). In southern Spain a delay in sowing date reduces the disease risk to emerging crops from airborne ascospores. However, as with field pea, delayed sowing can adversely affect yield if it compromises the optimum agronomic sowing date (Gan et al. 2005).

Where ascospores are not the primary source of inoculum, or ascospore release does not coincide with sowing date, delayed sowing of susceptible cultivars of chickpea and lentil is still often recommended to reduce the window of protection required by fungicides to keep ascochyta under control (Gan et al. 2005; Materne et al. 2001). Due to the polycyclic nature of ascochyta, later sowing lowers the epidemic intensity by limiting the number of pycnidiospore cycles.

In some situations, the main source of inoculum may be produced on early-sown crops, providing inoculum for later-sown crops, which can then become severely affected. Late-sown pea crops in some regions of the Northern Hemisphere suffer more ascochyta for this reason (Hare and Walker 1944).

Diseased seed and fungicide seed treatment

Diseased seed

Infected seed is a means of introducing ascochyta blight to new areas and there are numerous reports of ascochyta blight pathogens being introduced via infected seed (Ali et al. 1982; Bretag et al. 1995; Cother 1977a, b; Galdames and Mera 2003; Gossen and Morrall 1986; Kaiser 1997; Kaiser and Hannan 1986; Kaiser and Muehlbauer 1984; Morrall and McKenzie 1974). The proportion of seeds infected with *A. rabiei* in tested chickpea samples has been recorded as high as 70% in Turkey (Maden et al. 1975), while in the Pacific Northwest, USA, infection of commercial seed lots varied from 0.5 to 31% (Kaiser 1992). High levels of *A. lentis* infection have also been recorded in lentil seed lots, with 20% infection detected in Ethiopian seed lots (Ahmed and Beniwal 1991). Seed testing is a major component of *A. lentis* control in Canada (Morrall 1997) and Australia (Lindbeck et al. 2002). The importance of this was particularly demonstrated in the latter country where 33% of seed lots tested across the nation were infected, with higher incidence on seed harvested from earliest sown crops (Nasir and Bretag 1997). The importance of seed infection as an inoculum source is dependent on several factors; % of seed infection, the rate of seed to seedling transmission, the developmental rate of an epidemic from seedling foci, and the comparative influence of alternative sources of inoculum.

Seed to seedling transmission

Seed transmission rates for *A. rabiei* have been reported as 5% in field conditions (Kimber et al. 2007) to 20–30% in glasshouse conditions (Kimber et al. 2006; Maden 1983). The production of disease-free seed is seen as an important strategy in Pakistan,

in areas free from *A. rabiei* infection (Mitsueda et al. 1997). The use of disease-free seed is crucial for susceptible chickpea cultivars as seedling foci rapidly develop into epidemics in conditions conducive to disease development (Kimber et al. 2007). The rapid spread of ascochyta blight from primary infections in susceptible chickpea cultivars led to the development of a more rigorous PCR-based seed test in Australia. The original seed test (400 seeds on culture medium) was based on a procedure recommended by the International Seed Testing Association (ISTA 1996) and was able to detect as low as 0.25% infection levels. However, even lower levels of infected seed (0.01–0.1%) are sufficient to initiate epidemics when weather conditions are favourable (Kaiser 1992; Kimber et al. 2007). The PCR test uses DNA primers specific to *A. rabiei*, based on sequencing of the internal transcribed spacer region of the ribosomal gene complex. This test can detect DNA from 10 spores in a PCR reaction (Ophel-Keller et al. 1999). Comparisons between the PCR test, which uses 1,000 seed samples, and the plating test, were conducted on 50 seed lots. The PCR test was positive in all 13 cases where the plating test was positive, but it also detected a further 10 cases of *A. rabiei* infection in samples not detected by the plating technique. Some of these 10 cases were associated with severe ascochyta epidemics, where PCR testing was conducted post-sowing (Ophel-Keller et al. 1999), emphasising the need for the more sensitive procedure. Testing revealed that the majority of seed lots in Australia were infected and, in the absence of locally adapted resistant cultivars, the industry rapidly declined in regions conducive to ascochyta epidemics.

The majority of research on ascochyta control in lentil has concentrated on seed treatments and resistant cultivars. In Canada and Australia, stringent seed standards are recommended for lentil. Seed transmission rates in this crop appear to be low (Ahmed and Beniwal 1998) especially in dry soils of more than 15°C, but higher in wet soils at 8°C (Gossen and Morrall 1986). Western Canadian farmers plant ascochyta-infected lentils but levels below 5% seldom cause epidemics (Morrall 1992; Morrall and Sheppard 1981); however in areas of higher rainfall that promote epidemics, pathogen-free seed should be used (Morrall and Bedi 1990).

Infected seed is considered a major source of inoculum for *A. fabae* in the UK (Hewett 1973), Iraq (Michail et al. 1983) and New Zealand (Gaunt and Liew 1981). Transmission rate was estimated at 1–3% in Canada (Wallen and Galway 1977), and 4–8% in the UK (Hewett 1973). In the latter country, seed with more than 3% infection is discarded, and 1–3% infection is treated with a seed dressing (Jellis et al. 1998). There are varying reports on the importance of *A. fabae* seed infection in western Canada. Wallen and Galway (1977) found that after sowing seed with 13% infection, only 1% of harvested seed was infected. However, other studies in western Canada found that 1–5% seed infection could result in 27–35% infection on harvested seed (Bernier 1980; Kharbanda and Bernier 1979). Differences are likely to be due to environmental seasonal effects. In New Zealand, a significant yield reduction of 44% was observed due to disease that developed from seed with 12% initial infection. Infected seed affects plant establishment and disease incidence (Gaunt and Liew 1981). Control strategies were recommended for seed production crops including seed testing, a seed treatment of benomyl and captan, followed by a foliar spray of chlorothalonil during podding to prevent seed infection (Gaunt and Liew 1981; Hampton 1980).

Several studies have found no correlation between the level of *M. pinodes* seed infection in field pea and the severity of ascochyta on subsequent foliage (Bretag et al. 1995; Moussart et al. 1998; Xue et al. 1996; Xue 2000). Moussart et al. (1998) concluded that while *M. pinodes* seed infection resulted in disease at the basal parts of the plant as a foot rot symptom, no aerial symptoms were seen and so seed was not regarded as a source of contamination in the epidemiology of the disease. Xue (2000) found a high seed to seedling transmission of *M. pinodes* (70–100%), also leading to foot rot as well as reduced emergence, yield and seed weight. Seed to seedling transmission rate varies depending on environmental conditions (Bretag et al. 1995; Xue 2000) in that in drier regions transmission is of minor concern (Bretag et al. 1995). Seed infection levels >10% significantly reduce emergence (Bretag et al. 1995; Wallen et al. 1967; Xue 2000) but a higher seeding rate can compensate for this loss (Bretag et al. 1995). However, seed infection is important in areas where pea is seldom grown since it introduces the disease to

new areas. Seed infection can be reduced by avoiding seed lots produced from crops with high levels of ascochyta blight, such as early-sown crops, and avoiding late-harvested crops in which the disease has had more time to develop and infect seeds (Bretag et al. 1995).

Fungicidal seed treatments

Seed treatments reduce but do not completely inhibit the transfer of the pathogen to seedlings (Bernier 1980; Kaiser and Hannan 1987, 1988; Demirci et al. 2003). Nevertheless these treatments play an important role in reducing disease, particularly when combined with seed testing to minimise early establishment of the pathogens. Benomyl, carbendazim, chlorothalonil, thiabendazole, thiram and mixtures of these were effective in reducing seed to seedling transmission in pulse crops (Ahmed and Beniwal 1998; Grewal 1982; Kaiser et al. 1973; Kaiser and Hannan 1988; Kimber and Ramsey 2001; Reddy and Kababeh 1984; Rahat et al. 1993). Seed treatments are particularly beneficial for ascochyta control on chickpea and lentil.

Gan et al. (2005) summarised the physical and chemical methods that have been used to treat chickpea seed for *A. rabiei* infection. Excellent control, whereby the fungus was eradicated in laboratory tests and reduced infection to a minimum in field trials, was achieved using benomyl plus thiram, maneb, thiabendazole, or tridemorph plus maneb. Kaiser and Hannan (1988) and Maden (1983) found that benomyl and thiabendazole were the most effective of the fungicides tested and reduced seed infection on *A. rabiei* from 45% incidence to 0%. In laboratory conditions, thiram plus thiabendazole and carboxim plus thiabendazole reduced seed infection from an initial 80% to less than 5% (Kimber and Ramsey 2001).

Thiabendazole and carbendazim or benomyl have proven to be effective seed treatments on lentil (Bretag 1989; Kaiser and Hannan 1987). Iqbal et al. (1992) found that a range of tested fungicides reduced the recovery of seedborne *A. lentis* but most efficacious were Calixin-M, Benlate and Topsin-M. Lentil seeds with 81% infection had greater emergence when treated with thiabendazole or benomyl and yield was highest in thiabendazole-treated seeds (Kaiser and Hannan 1987).

A number of studies found that the benefits of using seed treatments to control ascochyta infection on faba bean and field pea are inconclusive, possibly because airborne inoculum has a greater influence on the ascochyta diseases on these crops, than does seedborne inoculum. While a range of fungicides are effective at reducing seed-borne inoculum in laboratory assays on both field pea and faba bean (Karbanda and Bernier 1979; Wallen et al. 1967) the treatments have not shown a consistent reduction in plant infection (Karbanda and Bernier 1979; Michail et al. 1983; Thomas et al. 1989; Wallen et al. 1967) or any difference in emergence, disease severity or yield (Walsh et al. 1989). Thiram can increase emergence of infected field pea (Xue 2000) and a mixture of thiram and thiabendazole reduced ascochyta levels in pea at early growth stages (Davidson, unpublished data), but by flowering there was no effective disease control or yield gain at harvest (Davidson, unpublished data; Xue 2000). Conversely, Bretag (1985) demonstrated a small yield gain associated with thiabendazole seed treatment on field pea brought about by reducing the severity of ascochyta blight. Thiabendazole seed treatment is recommended on faba beans but alone does not provide sufficient protection (Jellis et al. 1998).

Foliar fungicides

A range of broad-spectrum foliar fungicides has been tested against ascochyta blight with varying results e.g. Bordeaux mixture, captan, captafol, chlorothalonil, folpet, mancozeb, maneb, metiram, wettable sulphur, zineb (Nene 1982; Sadkovskaya 1970; Warkentin et al. 1996, 2000). These are used as preventative sprays, and need to be applied before disease becomes established, or before rain events during which new infections occur. Chlorothalonil is the most widely used fungicide in ascochyta control and is the most consistent performer in reducing ascochyta blight on pulses (Ahmed and Beniwal 1991; Chongo et al. 2003; Gan et al. 2005; Kimber and Ramsey 2001; McMurray et al. 2006; Shtienberg et al. 2006). For faba bean, lentil and partially-resistant chickpea cultivars, foliar sprays of chlorothalonil are generally effective when applied at early flowering to early pod set (Kharbanda and Bernier 1979; Beauchamp et al. 1986a, b; McGrane et al.

1989; Ahmed and Beniwal 1991). At lower rainfall sites, a single spray during podding may be sufficient to protect against yield loss, reflecting the importance of the environment on epidemiology and disease spread (Beauchamp et al. 1986b). In Australian lentil crops, chlorothalonil or mancozeb are recommended during podding only if the disease is present and conditions are conducive to infection (Lindbeck et al. 2002). Chlorothalonil is applied to faba bean six weeks after sowing in Australia (Hawthorne et al. 2004) to protect against ascospore showers released from neighbouring infested residue (Galloway and MacLeod 2003). Follow-up sprays are applied during flowering and podding if disease is evident and conditions are conducive to disease (Hawthorne et al. 2004). The poor economics of foliar fungicides on field pea usually excludes this practice from commercial cropping.

Some systemic fungicides are also effective e.g. azoxystrobin, benomyl, carbendazim, thiabendazole and tebuconazole (Chongo et al. 2003; Demirci et al. 2003; Shtienberg et al. 2000; Thomas and Sweet 1989; Warkentin et al. 1996). These have the added advantage that they may be applied post-infection, or post rain event, though such applications may have the added complexity of conditions being unsuitable for ground-rig equipment. These fungicides penetrate the host tissue and possess post-infection properties, which enable them to be applied in the three days after infection has occurred (Shtienberg et al. 2000). Application of systemic fungicides post-infection allows for flexibility in management and reduces fungicide applications to real infection events rather than forecast events as with protective fungicides.

The disease pressure, environmental conditions and coverage achieved by the application, influence the efficacy of foliar fungicides. Foliar fungicides used on susceptible chickpea cultivars in many parts of the world (summarised in Gan et al. 2005) show that even with multiple applications, ascochyta might not be controlled under epidemic situations (Reddy and Singh 1992; Shtienberg et al. 2000). In Canada and Australia, in the presence of *A. rabiei*, the production of chickpea is rarely successful when highly susceptible cultivars are grown, despite multiple fungicide applications (Bretag et al. 2003; Chongo et al. 2003; Kimber and Ramsey 2001). Even under moderate disease pressure, four to six sprays became necessary to significantly reduce disease. Only under dry conditions could fungicide applications be reduced (Chongo et al. 2003). In susceptible chickpea cultivars fungicides are generally uneconomic and impractical (Nene and Reddy 1987) and the rate of disease spread makes it difficult to follow an application schedule.

Preventative sprays are more effective when applied ahead of rain events during which infection occurs. The efficacy of chlorothalonil and mancozeb in Australian chickpea fungicide trials was reduced when the fungicide was not applied in time to protect crops from a rain event (Shtienberg et al. 2006). Analysis of the time of spraying in relation to rain events identified that disease was suppressed when fungicides were applied in time to protect plants from infection, but if plants were not protected during rain events, then control efficacy was low. The coincidence between control efficacy and uncontrolled rain was high i.e. $P < 0.01$, $R^2 = 0.937$ (Shtienberg et al. 2006). Management practices take this into account by encouraging continuous sprays of chlorothalonil every three weeks during the growing season. Simulated analysis of the trial data indicated that rain forecasting, to time fungicide sprays with rain fronts, could reduce the number of applications needed to control the epidemic. Initiating sprays after the presence of disease was confirmed, further reduced the number of sprays required for effective disease control.

Foliar fungicides on field pea have generally been uneconomic despite the reduction in disease and associated yield increases. Highly susceptible cultivars responded more to the fungicides than moderately susceptible cultivars (Warkentin et al. 2000), but even in these crops little spraying of field pea is conducted since multiple applications may be required to achieve significant disease suppression. Multiple sprays, initiated at early to mid-flowering provided some disease control and yield gains (Warkentin et al. 1996; Warkentin et al. 2000). A single application of mancozeb or chlorothalonil at early flowering also increased yields while a single late flowering application generally had no impact (Warkentin et al. 2000). Fungicide trials were conducted in Australia (Davidson, unpublished data) using mancozeb at 6, 9 and 12 weeks after sowing. Neither mancozeb nor chlorothalonil effectively controlled the disease and there were no yield gains in these trials. As breeding programmes develop

higher yielding cultivars, or the economic returns for pea increase, the financial benefit of applying foliar fungicides to field pea may also improve.

Strategic application of fungicides taking into account host resistance

Ascochyta resistance is a major priority in pulse breeding programmes around the world. No cultivars from these programmes have complete resistance, or immunity, to ascochyta due to the complexity of the host–pathogen relationship, but a number of cultivars exhibit partial resistance.

Ascochyta-susceptible pulse cultivars have been reliant on foliar fungicides but integrating enhanced resistance combined with clean seed and wide rotations has reduced foliar sprays and enabled the use of earlier sowing dates to maximise yield. Furthermore the lower input costs associated with reduced fungicide usage has greatly improved the economics of growing these crops. In Australia, foliar fungicides for ascochyta control in lentil crops are applied only at the podding stage since most Australian cultivars have foliar resistance to this disease (Lindbeck et al. 2002).

Partial resistance in chickpea is essential for the success of this crop in many parts of the world though the resistance can still be overcome in regions that have moderate to high inoculum pressure and weather conditions favourable to epidemics (Chongo et al. 2003). Two to four applications of chlorothalonil or azoxystrobin at early and mid-flowering are required under high disease pressure on partially resistant cultivars (Bernier 1980, Chongo et al. 2003; Kharbanda and Bernier 1979; Reddy and Singh 1992). In dry seasons a single spray on a moderately resistant cultivar may be sufficient (Pande et al. 2005). In some cases fungicide applications during podding are maintained to prevent pod infection, seed abortion or seed infection (Hawthorne et al. 2004) since resistance in chickpea is not as effective at flowering and podding (Chongo and Gossen 2001; Singh and Reddy 1993). Fungicide strategies differ according to cultivar resistance (Shtienberg et al. 2000) and the control efficacy of fungicides and effectiveness of genetic resistance vary according to seasonal conditions. When environmental conditions support severe epidemics, foliar fungicides may provide <20% control efficacy on susceptible and moderately susceptible cultivars, but as much as 70% control efficacy on moderately resistant cultivars. In mild epidemics >80% control efficacy is achieved on susceptible cultivars, and >95% on moderately susceptible and moderately resistant cultivars (Shtienberg et al. 2000).

Decision support systems

An understanding of the pathosystems and the interrelationship between host, pathogen and the environment is essential to be able to make correct fungicide and agronomic decisions for disease control. Some studies have been undertaken to develop decision support tools to assist growers in making these decisions.

Jhorar et al. (1997) studied weather data over a 27-year period in association with ascochyta blight of chickpea. Weekly averages of temperature, relative humidity (RH), sunshine duration, and total weekly rainfall and raindays were calculated for the period of vegetative growth to maturity. Disease at time of maturity was correlated with each of these parameters. A ratio of afternoon RH and maximum temperature was calculated to produce a parameter termed the humid thermal ratio and this was highly correlated with disease, $R^2 = 0.90$. This parameter was suggested as a useful model for disease prediction for fungicide applications.

In Israel, a predictive model determined that pseudothecial maturation and ascospore discharge of *A. rabiei* occurs after six rain events of equal to or >10 mm (Shtienberg et al. 2005). Fungicide applications at this time target the primary inoculum of ascospores and should prevent the infection of new crops and possibly the necessity of further fungicide applications in the crop. Subsequent sprays are initiated by monitoring, beginning when ascochyta is first observed in the crop, and are linked to forecasted rain thresholds for different cultivar resistances i.e. 5 mm for highly susceptible cultivars, 10 mm for moderately susceptible, 20 mm for moderately resistant, and 50 mm for resistant cultivars (Shtienberg et al. 2000).

A modelling system for ascochyta in field pea was developed by Salam et al. (2006) to predict time of onset, and progression of ascospore maturity and

spread of spores from the source of infection of *M. pinodes*. This model incorporates effects of rain, temperature and wind on fungal maturation, spore release and spore dispersal. The model Blackspot Manager helps growers to make decisions on when to sow their crop, by using year to date weather data and forward projection of historical data, to predict the likely ascospore load at a particular time of the year. The model also assists growers to select the best fields for field pea location to minimise the risk of ascochyta blight for several years in advance.

In the absence of effective resistance or economic fungicides, agronomic measures must be used to make decisions for ascochyta control in field pea. Multiple regression analysis of disease severity, cropping practices (i.e. sowing date, pea rotation history, proximity to infested residue) and environmental data, including cumulative rainfall and mean temperature, were used to predict ascochyta blight severity in field pea in South Australian cropping systems (Schoeny et al. 2003). The model is aimed at assisting growers to make informed decisions regarding rotations of pea crops and sowing date to minimise ascochyta.

Conclusion

Management of ascochyta is an essential component of successfully growing pulse crops. Where possible, moderately resistant cultivars should be grown but growers will select cultivars depending on yield, seed quality and marketability, not just on ascochyta resistance. Hence cultivars with different levels of ascochyta resistance will be grown and must be managed accordingly.

Integrated disease management includes a combination of cultivar resistance, seed and crop hygiene, seed and foliar fungicides and appropriate sowing dates. Selecting the most effective strategies can be difficult due to the complexity of the pathosystems and the inter-relationship with resistance and the environment. Decision support tools are in their infancy and rely on a good understanding of the epidemiology of the pathogens and the influence of the environment on the development and spread of the disease. As more research is conducted these tools will become more specific to crops, diseases and regions, enabling a good understanding of the forces that drive an epidemic. The challenge will then be to translate this information into a form that is understandable and useable by the grower to make agronomic and disease management decisions that are cost-effective and beneficial to yield and finances.

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