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Ascochyta blight of chickpea (*Cicer arietinum* L.): a review of biology, pathogenicity, and disease management*

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Abstract. Ascochyta blight (AB), caused by *Ascochyta rabiei* is a major disease of chickpea (*Cicer arietinum* L.), especially in areas where cool, cloudy, and humid weather persists during the crop season. Several epidemics of AB causing complete yield loss have been reported. The fungus mainly survives between seasons through infected seed and in infected crop debris. Despite extensive pathological and molecular studies, the nature and extent of pathogenic variability in *A. rabiei* have not been clearly established. Accumulation of phenols, phytoalexins (medicarpin and maackiain), and hydrolytic enzymes has been associated with host-plant resistance (HPR). Seed treatment and foliar application of fungicides are commonly recommended for AB management, but further information on biology and survival of *A. rabiei* is needed to devise more effective management strategies. Recent studies on inheritance of AB resistance indicate that several quantitative trait loci (QTLs) control resistance. In this paper we review the biology of *A. rabiei*, HPR, and management options, with an emphasis on future research priorities.

Additional keywords: ascomycete, biotic stress, *Didymella rabiei*, epidemiology.

Introduction

Chickpea (*Cicer arietinum* L.), a self-pollinated, diploid, annual grain legume (pulse), is the third most important food legume in the world after dry bean (*Phaseolus vulgaris* L.) and field pea (*Pisum sativum* L.). It is a major source of high-quality protein in human diets and also provides high-quality crop residues for animal feed. Chickpea maintains soil fertility through biological nitrogen fixation, and contributes to the sustainability of cropping systems in cereal–legume rotations. Among temperate pulses, chickpea is the most tolerant crop to heat and drought and is suitable for production in low fertility soils.

Chickpeas are of 2 types. The kabuli (garbanzo bean) types are usually large seeded, with 'ramsowls-head' shaped seeds, having a smooth surface and a thin cream or beige coloured seed coat. These types are grown in countries of the Mediterranean region, West Asia and North Africa (WANA), Australia, and North America. Desi types are usually small seeded, with angular seeds, reticulated (rough) seed surface, and a seed coat colour varying from yellow to black. Desi cultivars account for about 85% of the world's total production of chickpea, and are mainly grown in the south of Asia, Iran, Ethiopia, and Mexico (Anon. 2002).

In 2002, the world chickpea production was ~7.8 million tonnes from ~9.9 million hectares of land (FAO 2002). This constitutes ~5% of global legume production. Average yields of chickpea vary from <400 kg/ha in Pakistan to >3600 kg/ha in China (FAO 2002). Ascochyta blight (AB), caused by *Ascochyta rabiei* (Pass.) Labrousse, is the most important biotic constraint for chickpea production and causes serious grain yield and quality losses (Gaur and Singh 1996*b*). The disease is devastating in areas where cool, cloudy, and humid weather (15–25°C and >150 mm rainfall) occurs during the crop season (Nene 1982) and can cause complete yield loss.

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Geographical distribution

Following the first report from north-western provinces of India (now in Pakistan), the occurrence of AB has now been reported from most chickpea-growing areas in the world (Kaiser *et al*. 2000*a*, 2000*b*). The disease has been reported from 34 countries across 6 continents and, as recent cultivation in Australia and Canada has shown, it can spread rapidly to new areas of chickpea production. It is the most important biotic factor affecting chickpea cultivation in areas of WANA, southern Europe (Nene 1982; Singh and Sharma 1998; Akem 1999; CAB International 2000), between 31° and 45°N, and is occasionally important between 26° and 30°N. In Australia, chickpea production increased rapidly until 1999 but was then limited by outbreaks of AB because all available commercial varieties were susceptible to the disease (Ackland *et al*. 1998; Knights and Siddique 2002). The disease is currently the most important yield-limiting factor, potentially affecting 95% of the chickpea area in Australia (Knights and Siddique 2002). In Western Canada, the chickpea production area increased rapidly from 800 ha in 1995 to 700 000 ha in 2000 and continued to increase, but the incidence of AB in these areas resulted in >70% yield losses (http://www.pulse.ab.ca/ascoch.pdf). The occurrence of AB has also been reported from Bulgaria (Kaiser *et al*. 1998) and Latin America (Kaiser *et al*. 2000*a*, 2000*b*).

Causal organism

Ascochyta rabiei, the causal agent of AB of chickpea, exists both as an anamorph and a teleomorph. The anamorph, *A. rabiei*, is characterised by the formation of spherical or pear-shaped black fruiting bodies called pycnidia. A pycnidium contains numerous hyaline unicellular and occasionally bicellular spores, pycnidiospores, or conidia, developed on short conidiophores (stalks) embedded in a mucilaginous mass. Pycnidiospores are oval to oblong, straight, or slightly bent at one or both ends and measure 6–12 by 4–6 µm (Punithalingam and Holliday 1972; Nene 1982).

The teleomorph, *Didymella rabiei* (Kovacheski) var.Arx (Syn. *Mycosphaerella rabiei* Kovacheski), is a bipolar heterothallic ascomycete and is characterised by pseudothecia developing on chickpea crop residues that have over-wintered in the field. For successful sexual reproduction, the telomorph requires pairing of 2 compatible mating types (MAT1-1 and MAT1-2), which are widely distributed in several major chickpea-growing areas of the world (Haware 1987; Kaiser 1997; Armstrong *et al*. 2001). Pseudothecia are dark brown to black, subglobose, 120–270 µm in diameter, erupting from the host tissue and without a conspicuous ostiole. Binucleate asci are cylindrical to subclavate surrounded by paraphyses and contain 8 hyaline unequally bicellular ascospores. Ascospores are ellipsoid to biconic with a constriction at the septum and measure 9.5–16 by 4.5–7 µm. The fungus grows readily on a variety of nutrient media, the best being chickpea meal dextrose agar. *Ascochyta rabiei* generally produces a pale cream coloured mycelium in which pale brown to black pycnidia are immersed. Cultures are variable in morphology and colour, with isolates often producing a prevalence of unicellular conidia (CAB International 2000).

The morphological characteristics of *A. rabiei* and *Phoma medicaginis* var. *pinodella* are similar, which makes it difficult to distinguish between the 2 species. However, a PCR test developed by Phan *et al*. (2002) can be used to detect and confirm the identity of *A. rabiei*.

Disease symptoms

Symptoms of AB can develop on all aerial parts of a plant. Seed-borne infection leads to brown lesions at the stem base of emerged seedlings. Subsequently, the lesions enlarge in size, and girdle the stem causing its breakage and death of the plant. Numerous pycnidia develop on the necrotic lesions. In the field, AB may initially appear as small patches (foci) of blighted plants, but can rapidly spread across an entire crop under favourable temperature and rainfall. Plants are attacked at any growth stage, depending on the inoculum availability. However, AB is most prominent during the flowering to early podding growth stages. Air-borne conidia and ascospores infect younger leaves and produce small water-soaked necrotic spots that rapidly enlarge and coalesce. Conidia may also be water-borne and splash-dispersed to infect foliage tissue on the same or nearby plants. Subsequently, symptoms spread rapidly to all aerial parts including leaves, petioles, flowers, pods, branches, and stems, which leads to rapid collapse of tissues and death of the plant. Development of pycnidia in concentric rings on lesions is the characteristic symptom of *A. rabiei* infection. Lesions that develop on leaves and pods appear circular with brown margins and a grey centre that contains pycnidia, whereas lesions developing on petiole, stems, and branches are elongated. The lesions that develop on apical twigs, branches, and stems differ in size and in later stages girdle the affected plant parts. The regions above the girdled portion are killed and may break off. Diseased pods with visible blight symptoms often fail to develop any seed. Pod infection often leads to seed infection through the testa and cotyledons. Infected seed can be discoloured and possess deep, round, or irregular cankers, sometimes bearing pycnidia visible to the naked eye. Infection during the pod maturation stage often results in shrivelled and infected seed (Nene 1982; Singh and Sharma 1998; Akem 1999).

Host range

Artificial inoculation of *A. rabiei* on lentil, field pea, vetch, common bean, and cowpea revealed that the fungus is pathogenic on all these species (Zachos *et al*. 1963; Nene and

Reddy 1987; Khan *et al*. 1999*b*). *A. rabiei* also infects *Vigna unguiculata*, *P. vulgaris* (Kaiser 1973; Khan *et al*. 1999*a*), *Lactuca serriola*, *Lamium amplexicaule*, *Medicago sativa*, *Melilotus alba*, and *Thlapsi arvense* (Kaiser 1991), which are also grown in chickpea-producing regions. Pycnidial formation occurred in necrotic tissues of *Medicago sativa* and *Melilotus alba*. *Ascochyta rabiei* has also been isolated from *Brassica nigra*, *Descurainia sophia*, *Galium apanine*, *Lamium amplexicaule*, and *Triticum aestivum*, grown in fields where infected chickpea debris of the previous season remained on the soil surface during the off-season (Kaiser 1991).

Pathogen variability

The possible existence of different races of *A. rabiei* was suspected because of the variations in host–pathogen interactions and breakdown of host-plant resistance (HPR) in some cultivars at different locations. The presence of a teleomorph (*D. rabiei*) in the *A. rabiei* life cycle contributes to variability within the pathogen population, which may generate new combinations of virulence genes and thus the development of new pathotypes. However, *A. rabiei* is heterothallic and the 2 mating types are not present in all chickpea-growing areas (Khan *et al*. 1999*b*). Natural occurrence of the teleomorph on chickpea stubble in Australia implies that either both compatible mating types are present or that a low level of homothallic compatibility exists in *A. rabiei* (Galloway and MacLeod 2003). In Canada, many fields had both mating types together (Armstrong *et al*. 2001).

Pathogen variability studies based on morphological, pathogenic, and isozyme patterns, and DNA fingerprinting have been conducted in most of the major chickpea-growing countries. Based on the virulence of *A. rabiei* isolates on different genotypes, the existence of 2–12 races of *A. rabiei* has been proposed by several researchers (Table 1). In India, variations in pathogenicity among a collection of 268 *A. rabiei* isolates have been observed by Vir and Grewal (1974*a*). In Syria and Lebanon, 6 pathotypes of *A. rabiei* were identified using 6 chickpea differential lines (Reddy and Kabbabeh 1985). Recently, Baaya *et al*. (2004), using host differentials and DNA finger printing, identified a new pathotype in Syria, which can overcome the resistance of ICC 12004 and ICC 3996 chickpea lines. However, in several of these studies, no definitive relationships were observed between virulence of the isolates, their geographical origin, and morphological characteristics such as spore size, colony colour, and radial growth *in vitro*. Also, isozyme patterns of esterase and acid phosphatase failed to separate 15 Pakistan isolates of *A. rabiei* according to their aggressiveness (Hussain and Barz 1997).

DNA fingerprinting has been used in an attempt to define differences among all putative races of *A. rabiei*. However, no definitive relationship could be observed among 48 *A. rabiei* isolates belonging to the 2 mating groups collected from India, Pakistan, Spain, the USA, and other countries (Navas-Cortes *et al*. 1998*c*). Randomly amplified polymorphic DNA (RAPD) analysis of *A. rabiei* isolates from Pakistan indicated genetic differences between isolates from the same host plant and similarities

Table 1. Summary of pathogenic and molecular variability studies on *Ascochyta rabiei*
N.B. There are few genotypes in common among the differentials used in various studies

Country	No. of isolates	Variability parameters	Key findings	Reference
Italy	41	Pathogenicity on 13 chickpea genotypes	Three pathogenicity groups suggested	Porta-Puglia *et al*. (1996)
India	348	Pathogenicity on 12 differential genotypes	12 races identified	Singh and Sharma (1998)
India	Different isolates from Jammu region	Pathogenicity on different chickpea genotypes	10 pathotypes identified	Ambardar and Singh (1996)
USA	39	Pathogenicity on 11 differential genotypes	Grouped into 11 virulent forms	Jan and Wiese (1991)
Pakistan	102	Pathogenicity on differential genotypes	Grouped into 8 virulent forms	Jamil *et al*. (1995)
Italy	30	RAPD analysis	No pathotype specific amplification patterns reported	Fischer *et al*. (1995)
India, USA, Syria, Pakistan	47	RAPD analysis	Isolates clustered according to the geographic origin. A DNA marker (ubc756), specific to Indian isolates identified	Santra *et al*. (2000)
Australia, Canada, India, Syria, USA	68	RAPD analysis	Isolates from 4 countries clustered within major groups of Canadian isolates	Chongo *et al*. (2004)
Australia	–	STMS fingerprinting	Diversity of Australian isolates	Phan *et al*. (2003)

between isolates from different plants and cultivars (Sarwar *et al*. 2000). Restriction fragment length polymorphism (RFLP) analysis of *A. rabiei* isolates from the Beja region of Tunisia indicated a low correlation between their virulence and RFLP patterns (Hamza *et al*. 2000). Phan *et al*. (2003), using the sequence tagged microsatellite site (STMS) fingerprinting technique, attempted to study *A. rabiei* diversity and its populations in Australia. All these studies identified specific DNA fragments that may be used as isolate-specific genetic markers in sexual crosses.

In an analysis of micro- and macro-geographical variations of *A. rabiei* using DNA fingerprinting, Morjane *et al*. (1997) observed that 17 different fungal genotypes were distributed at different frequencies in the 5 fields sampled, of which 2 were common in all the fields. In a few instances, more than one fungal genotype was isolated from the same plant. Higher levels of pathogen diversity were found within rather than between locations, and different genotypes from a particular location were not obviously related to each other.

A combination of RAPD analysis and RFLP analysis using an oligonucleotide probe complementary to the microsatellite sequence $(GATA)_4$ distinguished variability within and among the major pathotypes of *A. rabiei*. A combination of microsatellites and RAPD markers distinguished the earlier identified 4 pathotypes of *A. rabiei* (Weising *et al*. 1991) into 5 pathotypes, which were further resolved into several genotypes, indicating that different isolates of a pathotype do not have clonal lineages (Udupa *et al*. 1998). In all the above-mentioned studies, the DNA amplification patterns of *A. rabiei* isolates were not correlated with their grouping into different pathogenic groups. A standard set of international differential lines, which clearly distinguish all *A. rabiei* isolates from a broad geographical area, may help in the identification of different races of *A. rabiei*, if they do indeed exist.

Epidemiology of the disease

Pathogen survival

Ascochyta rabiei survives either on or in seed or plant debris in the form of mycelium, pycnidia, and various teleomorphic stages (Kaiser 1997). *Didymella rabiei* can survive in a free state in the soil. The teleomorph was first discovered on over-wintered infested chickpea debris from a field near Genesee, Idaho, USA, in 1986 (Kaiser 1994). At temperatures of 10–35°C, *A. rabiei* can survive for 8 months in infected chickpea debris (Nene and Reddy 1987), 20 months on infected stem (Kaiser and Hannan 1987) and 5 months on the surface of chickpea seed (Singh *et al*. 1995). However, when infected debris and stems were buried in soil, the pathogen survival was drastically reduced. When the infected seeds were stored at 4°C, *A. rabiei* remained infective for 13 years (Kaiser 1997).

The teleomorph helps in long-term survival of the pathogen, but this stage has never been observed on newly infected plants. However, in many regions, pseudothecia can often be found on infected crop debris. Low temperature and high moisture are essential for initiation and development of pseudothecia (Trapero-Casas and Kaiser 1992*b*; Navas-Cortes *et al*. 1998*a*). The density of asci and ascospore production per pseudothecium, and conidia per pycnidium were much higher in cool climatic conditions than in warmer conditions (Navas-Cortes *et al*. 1998*b*). Ascospores are also important in long-distance dispersal of the pathogen (Trapero-Casas *et al*. 1996). Relative humidity rather than temperature was the critical factor determining the development of psuedothecia and pycnidia on crop debris. Thus, at lower humidities (such as 86% RH) the development of *A. rabiei* on debris was very limited irrespective of temperature (Navas-Cortes *et al*. 1998*a*). When the debris was buried in soil, pycnidia rather than pseudothecia were produced, and the developed pseudothecia were degenerated and contained a reduced number of asci (Navas-Cortes *et al*. 1995). Perpetuation of *A. rabiei* through crop debris in tropical countries may be influenced by the high temperature and low rainfall during out-of-season summer months, which decrease the survival of *A. rabiei* in crop debris. It is notable that the effect of light on *in vitro* pseudothecial development was negligible and had little effect on the pattern and quantity of ascospores discharged.

Disease spread

Seed transmission of *A. rabiei* and air-borne spores can lead to disease spread and establishment of compatible mating types in new areas and thus the development of the teleomorph. Seed transmission ensures random distribution of the pathogen in a field, providing many primary infection foci. Movement of infected chickpea seed is responsible for introducing AB into Canada, Iran, Australia, and the USA (Kaiser 1997). Maden *et al*. (1975) detected *A. rabiei* in 70% of the chickpea seed samples from central Anatalia-Turkey, with seed infection ranging from 1–16%. Conidia and ascospores are responsible for secondary spread of the disease. Subsequent wetting, rain splash, and strong winds disperse conidia developed on diseased plant parts, particularly if conidia are contained in droplets. Ascospore production on highly infected crop residues reaches up to 1.5×10^4 ascospores/mm^2 of tissue surface. Under moist conditions, the asci protrude through the opening of a mature pseudothecium and forcibly discharge ascospores into the air (Trapero-Casas and Kaiser 1992*b*). At 15–25°C, >70% of ascospores were discharged from mature pseudothecia within 2 h of wetting and served as primary inoculum.

Frequency of infection cycles occurring during a growing season is influenced by environmental conditions and cultivar susceptibility (Nene and Reddy 1987).

Disease development

Ascochyta blight infection and disease development occur at a temperature range of 5–30°C with an optimum of 20°C, and 17 h of wetness is essential to produce severe infection. Dry periods (6–48 h) immediately after inoculation sometimes increase disease severity; however, dry periods of >12 h after an initial wetting period of 6 h usually have an adverse effect on disease development (Trapero-Casas and Kaiser 1992*a*). Jhorar *et al*. (1998) observed that dry periods immediately after inoculation followed by a wetness period reduced disease severity, and reduction in disease severity was correlated with an increase in the dry period. Little infection developed without leaf wetness even when the RH was 98% and no infection developed when the RH was <95%. Disease severity increases with increasing periods of darkness after inoculation. When leaf wetness was maintained over an 8-day period, there was an increase in the number of pycnidia and production of conidia on infected leaves (Jhorar *et al*. 1998). Under cool weather, spread and development of AB in a maturing crop can be rapid, with an incubation period as short as 6 days (Pandey *et al*. 1987).

Plant age was observed to have a profound effect on susceptibility of different chickpea genotypes to AB infection, with plants at podding stage being most susceptible (Chongo and Gossen 2001).

Disease prediction models

Disease prediction models based on the climatic factors that favour development of AB have been used to assess the disease risk for various agrogeographical zones and different growth stages. Comparisons between AB incidence and weather variables over a 15-year period, at 2 different locations (one with a regular disease incidence and the other with no reports of disease incidence), showed that maximum temperature and afternoon RH were the 2 most important variables for disease prediction. A ratio of these two weather variables, referred to as humid thermal ratio (HTR), was the best predictor of outbreaks of AB (Jhorar *et al*. 1997).

Although several studies have been conducted to determine epidemiological factors that favour AB development, many gaps still exist in our understanding of disease development and prediction of epidemics. Hence, more systematic evaluations of host-pathogen interactions, disease perpetuation and dissemination, sources of inoculum, host range, and favourable environmental (weather) conditions are needed to fill the knowledge gaps in disease prediction.

Pathogenicity

The infection process of *A. rabiei* on leaves and stems of both resistant and susceptible genotypes has been well studied. Production of toxins, cell wall degrading enzymes, and degradation of host phytoalexins are responsible for pathogenicity of *A. rabiei*.

Histopathology

Germination of *A. rabiei* spores occurs at 12–48 h after inoculation (HAI). Germ tubes further elongate and form ramifications on the leaf surface. Hyphal branches form appressoria-like structures at their tips, which are separated from the germ tube by a septum and the hyphae covered by a mucilaginous exudate (Hohl *et al*. 1990). *A. rabiei* penetrates directly by mechanical force through the cuticle between 2 epidermal cells. For a short distance, hyphae push forwards subcuticularly along the junction of epidermal cells before proceeding inward (Pandey *et al*. 1987; Hohl *et al*. 1990). Near a stoma, hyphae penetrate through a juncture of guard and subsidiary cells, even when a stoma is open (Pandey *et al*. 1987). By using Gus (β-glucuronidase)-transformed *A. rabiei*, which did not differ significantly from the parent strain in production of hydrolytic enzymes and toxins, it was confirmed that fungal penetration occurs directly through the cuticle. Penetration through hydathodes has also been observed (Kohler *et al*. 1995).

After penetration, hyphae grow parallel between epidermal and palisade parenchyma cells, disintegrating the inner structure of leaves. Hyphal diameter measures up to 2 µm outside the leaf, and up to 3.5 µm inside the leaf. Subsequently, subepidermal mycelia form dark aggregates. Epidermal cells collapse, and cells of palisade and spongy parenchyma lose their shape and organisation. The entire cortex and part of the pith disintegrate completely by the fifth day after inoculation (DAI). Hyphae aggregate and form pycnidia that emerge by collapse of the surrounding leaf tissues. From pycnidium, conidia ooze out through an ostiole (Pandey *et al*. 1987; Hohl *et al*. 1990). By the seventh DAI, most of the non-lignified tissues are destroyed and necrosis is much more evident. Lignified tissues, particularly xylem tracheary elements, remain unaffected (Pandey *et al*. 1987).

Fungal growth proceeds from the leaflets to stems through petioles. Within the petioles, *A. rabiei* rarely colonises xylem vessels but colonises phloem vessels and the petioles break off (Kohler *et al*. 1995). The fungus invades and colonises xylem and phloem vessels of the stem, but walls of these vessels remain intact. Pycnidia form subepidermally within cortex and pith. In resistant genotypes, a strong autofluorescence typical of a hypersensitive response (HR) was observed in leaf and stem tissues in early stages of infection. As a result, no hyphae could be observed in girdled stems (Hohl *et al*. 1990).

Pathogen toxins and enzymes involved in infection

Extensive disintegration of parenchymatous cortical and pith tissues that occurs in advance of invading fungal hyphae during the infection process indicates involvement of toxins and cell wall-degrading enzymes produced by *A. rabiei* in its pathogenesis (Pandey *et al*. 1987; Hohl *et al*. 1990). The toxins solanopyrone A, B, and C are involved in pathogenicity of *A. rabiei*. Application of purified solanopyrones to chickpea leaves produced visible symptoms in 24 h, followed by contraction of protoplasts of epidermal, palisade, and spongy parenchyma cells (Hohl *et al*. 1991). There was a good correlation between the *in vitro* production of solanopyrones by different isolates of *A. rabiei* and their pathogenicity (Kaur 1995). *Ascochyta rabiei*, when grown on plant sap medium, produced an additional heat-labile toxic polypeptide of 14 amino acids consisting of a glycosidic moiety with a molecular weight of 7.6 k Da. Production of this polypeptide peaked at 4 days, with 166.7 units/mL culture filtrate (Chen and Strange 1994).

Ascochyta rabiei degrade phytoalexins produced in chickpea plants by converting the pterocarpans into 2′-OH isoflavans and 1a-OH pterocarpdiens. These two enzymes required for this conversion, a reductase and a hydroxylase are expressed constitutively in *A. rabiei* (Tenhaken *et al*. 1991). These two enzymes are specific for (–) isomers of phytoalexins such as maackiain and medicarpin (Weltring *et al*. 1995).

Other pathogenic enzymes such as cutinase (Tenhaken *et al*. 1997) and a polygalacturonase that degrades the polygalacturonic acid but not pectin were purified from the culture filtrate of *A. rabiei*. Purified polygalacturonase released no oligo-galacturonides that elicit chickpea plants and trigger a defence response (Tenhaken and Barz 1991).

Host plant resistance

Ascochyta blight resistance of chickpea is determined by a diverse set of anatomical, biochemical, physiological, and genetic characters. Host metabolitic activities that inhibit the pathogen invasion include induction of hypersensitive response (HR), cell wall reinforcement by deposition of callose, lignin, esterbound cinnamic acids/polyphenols, and hydroxyproline-rich glycoproteins, induction of phytoalexins, and proteins that inhibit the pathogen growth or reduce its virulence.

Anatomical characters

Anatomical characters such as thickness of the leaf cuticle, stem cuticle, epithelium, and palisade cells provide a mechanical barrier for pathogen penetration. A higher number of xylem elements and xylem parenchyma cells, and thicker stem epidermis and hypodermis exist in resistant genotypes than in susceptible genotypes (Angelini *et al*. 1993). Susceptible genotypes had a thinner outer cell wall and smaller area of cell lumen in the second outer cell layer (Venora and Porta-Puglia 1993). The cortical region was thinnest in the susceptible genotype Aug 424, and *A. rabiei* caused greater damage to cortical and pith tissues by the third DAI (Sarwar *et al*. 1996).

Host physiology

Ascochyta blight resistance in chickpea genotypes correlates positively with their respiration rate and total carbohydrate content. In a resistant genotype the rate of respiration and total carbohydrate content increased by the second DAI due to a HR, whereas in a susceptible genotype this increase occurred only by the fifth DAI (Dolar and Gurcan 1995). Total and reducing sugars, phosphorous, and potash gradients increased more in resistant than in susceptible genotypes (Khirbat and Jalali 1999).

Hypersensitive response

An incompatible plant–pathogen interaction quite often results in a rapid HR. When infected with *A. rabiei*, a rapid HR-like browning reaction developed in the leaves of resistant genotype ILC 3279 by the second DAI, whereas such changes were not observed in the susceptible genotype ILC 1929 (Hohl *et al*. 1990). Similar responses were observed when crude culture filtrate (CCF) of *A. rabiei* was applied to cell cultures of these 2 genotypes. In cells of ILC 3279, browning became visible 5–7 h after application of CCF. The browned cells did not develop a red stain after treatment with phloroglucinol-HCl, indicating the absence of lignin-specific compounds. Occurrence of HR was further confirmed by determining cell death by fluorescein diacetate staining. Cells of ILC 3279 died rapidly at 12 HAI but there was no cell death in ILC 1929. Lack of HR in cells treated with autoclaved or proteinase K-treated CCF suggested the proteinaceous nature of the HR inducer (Vogelsang *et al*. 1994).

Phytoalexin accumulation

Phytoalexins are metabolic compounds that have an important role in the defence mechanisms of higher plants towards phytopathogenic fungi. In chickpea genotypes resistant to *A. rabiei* infection, pterocarpan phytoalexins, (–) medicarpin and (–) maackiain, were produced rapidly and in higher quantities than in susceptible genotypes in response to attack by *A. rabiei*. Detection of maackiain alone in the resistant genotype indicates its important role in disease resistance (Dolar and Gurcan 1993). Treatment of cell cultures of ILC 3279 with CCF of *A. rabiei* resulted in accumulation of medicarpin as a major ester-bound component in the cell wall 12 h after treatment, whereas there was no accumulation of medicarpin in ILC 1929 cells (Vogelsang *et al*. 1994).

Phenolic compounds

Main constitutive phenolic compounds in chickpea are biochanin A (5,7-dihydroxy-4′-methoxyisoflavone) and formononetin (7-hydroxy-4′-methoxyisoflavone). These isoflavones occur as aglycones (7-O-glucosides), most prominently as 7-O-glucoside-6′-O-malonate esters (Koster *et al*. 1983). When challenged with *A. rabiei* or its CCF, resistant genotypes rapidly accumulated large quantities of phenolic compounds compared with susceptible genotypes (Vir and Grewal 1974*b*; Sindhu *et al*. 1995; Khirbat and Jalali 1997). Also, an elicitor preparation from CCF of *A. rabiei*, enhanced levels of formononetin and biochanin A in sliced cotyledons of chickpea (Kessmann and Barz 1986).

Defence-related enzymes

Induction of fungal cell wall-degrading hydrolytic enzymes, and enzymes of the phenylpropanoid pathway and cell wall lignification have a role in conferring AB resistance to chickpea. With the addition of CCF of *A. rabiei*, activity of phenylalanine ammonia lyase (PAL) increased by about 20-fold in cell culture of chickpea line ILC 3279, but there was no change in PAL activity of ILC 1929 cell culture (Vogelsang *et al*. 1994). Following *A. rabiei* inoculation, the activity of chitinase in leaves of the resistant genotype (E 100 Y) increased 5-fold by the sixth DAI, compared with the uninoculated control. Further, induction of chitinase was also higher in excised infected pods of resistant genotypes compared with susceptible genotypes (Nehra *et al*. 1994).

Peroxidase is essential for lignosuberisation, which occurs in cell walls in response to pathogen invasion. Diamine oxidase involved in polyamine catabolism is the main source of H_2O_2, which is essential for peroxidase activity. Activities of these 2 enzymes in chickpea stems increased during *A. rabiei* infection, and increase was greater in resistant compared with susceptible genotypes. In lignosuberised barriers set up by cortical and pith parenchyma cells in response to pathogen invasion, apparent histochemical activities of both these enzymes were detected (Angelini *et al*. 1993). Accumulation of peroxidase and β-1,3-glucanase, a fungal cell wall hydrolytic enzyme, was higher in resistant genotypes than in susceptible ones, when grown in the presence of CCF of *A. rabiei* (Sindhu *et al*. 1995).

Disease management

Identification of host plant resistance

The preliminary step for exploiting HPR is the development of reliable and repeatable techniques for large-scale screening of germplasm and breeding lines. Several techniques suitable for AB resistance screening under field and greenhouse conditions have been developed (Nene 1982; Weising *et al*. 1991; Nasir *et al*. 2000; Bretag and Meredith 2002; Bretag *et al*. 2002*a*, 2002*b*). Resistance screening using cut-twig and detached-leaf techniques correlated with greenhouse screening. These quick and reliable methods are useful in screening segregating lines derived from wide hybridisation, since whole plants can then be used to screen for other target traits including seed production (Sharma *et al*. 1995).

Field screening

Field screening of chickpea genotypes for AB resistance as standardised by ICRISAT and ICARDA involved planting the test material with a 40-cm row space and interplanting a susceptible cultivar (e.g. L 550 or Pb 7 or ILC 1929), which serves as an indicator/spreader line, after every 4–8 rows. Infected debris is scattered between rows, and at flowering the plants are inoculated with a spore suspension ($\sim 10^5$ spores/mL) in the evening on cloudy days. Following inoculation, sprinkler irrigation for 15 days is provided during dry weather. The disease rating scale commonly followed is a 1–9 scale, where 1 is no visible lesions on any plants and 9 is profuse lesions on all plants, stem girdling on more than 50% of the plants, and many plants killed (Singh *et al*. 1981; Reddy and Singh 1984).

Controlled environment screening

Different screening techniques have been developed at various research centres, for artificial resistance screening of chickpea germplasm against *A. rabiei*. A controlled-environment facility established at the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, India, facilitates reliable large-scale screening for AB resistance. Ten-day-old chickpea seedlings grown in plastic trays ($35 \times 25 \times 8$ cm) in a mixture of sterilised river sand and vermiculite (3 : 1), were transferred to a plant growth chamber in which air temperature was maintained at 20 ± 1°C. The seedlings were inoculated by spraying a conidial suspension (5×10^4 conidia/mL) of *A. rabiei*. The conidia were produced on chickpea seed and harvested into sterile distilled water. In the growth chamber, leaf wetness was maintained up to 72 HAI and RH maintained at 65–70% during the subsequent 12 days. A 12-h photoperiod was provided with fluorescent lights. Disease severity was scored on a 1–9 rating scale at 14 DAI (Haware *et al*. 1995). Recently, an improved 0–9 rating scale for controlled-environment screening of AB infection has been developed by Chongo *et al*. (2004).

Cut twig method

Long, tender shoots cut from the test plants are wrapped with a cotton plug and transferred to a test tube (15×100 mm) containing fresh tap water. Twigs are inoculated by spraying conidial suspension (4×10^4 conidia/mL) of *A. rabiei* and kept in moist

chambers for 72 h. After 72 h of leaf wetness with >90% RH, infected plants are incubated for another 13 days and then assessed for disease. The results obtained by this method were well correlated with those of greenhouse and field-screening techniques (Sharma *et al*. 1995).

Detached leaf/leaflet technique

Surface-sterilised whole leaves are transferred onto water agar in 90-mm Petri dishes and inoculated by spraying the dishes with a spore suspension. The lids of the Petri plates are sealed with paraffin wax and incubated at 20°C with a 12-h photoperiod. Inoculated leaves are observed for disease development on the eighth DAI (Singh and Sharma 1998).

Leaflets from the most recent fully expanded leaves are collected from 15-day-old chickpea plants. The detached leaflets are floated, lower surface down, on tap water inside 90-mm Petri dishes, and upper surfaces of the leaflets inoculated with 5 µL of *A. rabiei*. The leaflets are incubated for 14 days at 20 ± 2°C, with a 12-h photoperiod. Disease severity scores are based on the number of leaflets infected and lesion size (Dolar *et al*. 1994). The disease ratings obtained from this technique are in correlation with whole-pot screening methods.

Resistant sources

Deployment of resistant genotypes is the most effective way to minimise yield losses due to AB. In several studies conducted in different chickpea-growing areas of the world, several sources of resistance to AB were identified (Table 2). Few of the resistant sources were also released as cultivars (Table 3). Furthermore, development of AB-resistant genotypes has made it possible to sow the crop during winter in the Mediterranean region thereby doubling the chickpea production potential. High levels of AB resistance have been identified among annual wild *Cicer* spp., accessions of *C. bijugum*, *C. judaicum*, and *C. pinnatifidum* (Singh *et al*. 1981; Singh and Reddy 1991; Collard *et al*. 2001) and there is potential to transfer resistance genes from these species into *C. arietinum*. One hundred and twenty-eight wild accessions of chickpea belonging to 8 species were screened for AB resistance under controlled-environment conditions at ICRISAT, Patancheru, India. One accession each of *C. bijugum* and *C. pinnatifidum*, 2 of *C. cuneatum* and 17 of *C. judaicum* were resistant to AB infection, with a mean disease score of ≤3.0 on a 1–9 rating scale. Another 18 accessions of *C. judaicum* and 8 accessions of *C. pinnatifidum* were moderately resistant to AB infection, with a mean disease score of 3.1–5 (Table 4).

At the International Center for Agricultural Research in the Dry Areas (ICARDA), Syria, >25 000 chickpea lines have been screened for AB resistance and 14 durable sources of resistance have been identified. ILC 200, ICC 4475, ICC 6328, ILC 6482, and ICC 12004 were found to be resistant to 6 races of *A. rabiei* in repeated field and greenhouse evaluations (Singh and Reddy 1993). Several of the resistant sources identified and breeding lines derived from them have been released worldwide (Table 3). In total, 1584 AB-resistant chickpea

Table 2. Sources of resistance to Ascochyta blight in chickpea germplasm

Genotype	Remarks	Reference
ICC 3634, ICC 4200, ICC 4248, ICC 5124, ICC 6981, ILC 196, ILC 3346, ILC 3956, ILC 4421		Reddy and Singh (1984)
ILC 72, ILC 191, ILC 3279, ILC 3856	Resistant in 8 chickpea-growing countries (including India, Pakistan, and Mediterranean region)	Singh *et al*. (1984)
ICC 76, ICC 187, ICC 607, ICC 1121, ICC 1136, ICC 1416, ICC 1754, ICC 1762, ICC 1903, ICC 7773, ILC 236, ILC 482, ILC 484, ILC 2548, ILC 2956	ILC 482 and ICC 1903 were always rated 1 on a 1–9 scale	Katiyar and Sood (1985)
ICC 4000, 4014	Both foliage and pods of ICC 4000 were resistant to Ascochyta blight	Singh and Kapoor (1985)
ILC 3864, ILC 3870, ILC 4421		Pal and Singh (1990)
ILC 190, ILC 201, ILC 202, ILC 2506, ILC 3856, ILC 5928, ICC 3996, FLIP 83-48	Resistant to 3–6 races of *A. rabiei*	Singh and Reddy (1990)
ILC 5586, ILC 5894, ILC 5926, ILC 6482, ILC 7795, ICC 4475, ICC 6328, ICC 12004	Resistant both in greenhouse and field	Singh and Reddy (1992)
ILC 3287	Rate-reducing phenomenon of Ascochyta blight observed	Reddy and Singh (1993)
CG 715, ACC 76, H 86-8, H 86-100, HK 86-120		Singh and Pal (1993)
ILC 3896, ICC 7514, NEC 123, P 1279-2, P 4268-1		Gaur and Singh (1996*a*)
ICC 8161		Shukla and Pandya (1988)
ICC 1278, ICC 1284, ICC 1285, ICC 1304		Wadud and Riaz (1988)
FLIP 92-262C, FLIP 92-110C, FLIP 92-154C		Toker *et al*. (1999)

Table 3. Some chickpea lines released in different countries, with acceptable level of resistance to Ascochyta blight
Source: Anon. (2002)

Accession	Country of origin	Country of release	Released name	Year of release
ILC 72	n.a.	Italy	Califfo	1990
ILC 72	n.a.	Spain	Fardan	1985
ILC 195	USSR	Egypt	Giza 195	1995
ILC 195	USSR	Morocco	ILC 195	1986
ILC 195	USSR	Turkey	ILC 195	1986
ILC 200	USSR	Spain	Zegri	1985
ILC 202	USSR	China	ILC 202	1988
ILC 237	Spain	Oman	ILC 237	1988
ILC 411	Iran	China	ILC 411	1988
ILC 464	Turkey	Cyprus	Kyrenia	1987
ILC 482	Turkey	Algeria	ILC 482	1988
ILC 482	Turkey	France	TS 1009	1988
ILC 482	Turkey	Iran	ILC 482	1995
ILC 482	Turkey	Iraq	Rafidain	1992
ILC 482	Turkey	Jordan	Jubeiha 2	1990
ILC 482	Turkey	Lebanon	Janta 2	1989
ILC 482	Turkey	Morocco	ILC 482	1986
ILC 482	Turkey	Syria	Ghab 1	1986
ILC 482	Turkey	Turkey	Guney Sarisi 482	1986
ILC 484	Turkey	Libya	ILC 482	1993
ILC 533	Egypt	Georgia	Elixir	2000
ILC 915	Iran	Sudan	Jebel Marra-1	1994
ILC 1335	Afghanistan	Sudan	Shendi	1987
ILC 2548	USSR	Spain	Almena	1985
ILC 2555	Ethiopia	Spain	Alcazaba	1985
ILC 3279	USSR	Algeria	ILC 3279	1988
ILC 3279	USSR	China	ILC 3279	1988
ILC 3279	USSR	Cyprus	Yialosa	1984
ILC 3279	USSR	Iran	ILC 3279	1995
ILC 3279	USSR	Iraq	Dijla	1992
ILC 3279	USSR	Italy	Sultano	1990
ILC 3279	USSR	Jordan	Jubeiha 3	1990
ILC 3279	USSR	Syria	Ghab 2	1986
ILC 3279	USSR	Tunisia	Chetoui	1987
ILC 6188	France	Italy	Ali	1998

n.a., Not available.

Table 4. Screening wild *Cicer* spp. for Ascochyta blight resistance at ICRISAT, Patancheru, India
Rating scale for Ascochyta blight on chickpea seedlings (modified from Jan and Wiese 1991): 1, no symptoms; 2, minute lesions prominent on the apical stem; 3, lesions up to 5 mm size and slight drooping of the apical stem; 4, lesions obvious on all plant parts, and clear drooping of apical stem; 5, lesions obvious on all plants/parts, defoliation initiated and breaking and drying of branches slight to moderate; 6, lesions as in 5, defoliation, broken, dry branches common, some plants killed; 7, lesions as in 5, defoliation, broken, dry branches very common, up to 25% of the plants killed; 8, symptoms as in 7 but up to 50% of the plants killed; 9, symptoms as in 7 but up to 100% of the plants killed. Based on the disease score, the wild accessions were categorized for their reaction to Ascochyta blight infection as follows: 1, immune (I); 1.1–3, resistant (R); 3.1–5, moderately resistant (MR); 5.1–7, susceptible (S); and 7.1–9, highly susceptible (HS)

Cicer species	No. of lines screened	Reaction to Ascochyta blight infection				
		I	R	MR	S	HS
C. arietinum	3	–	–	–	–	3
C. bijugum	18	–	1	–	4	13
C. cuneatum	2	–	2	–	–	–
C. echinospermum	2	–	–	–	2	–
C. judaicum	46	–	17	18	11	–
C. pinnatifidum	26	–	1	8	7	10
C. reticulatum	26	–	–	–	16	10
C. yamashitae	5	–	–	–	1	4
Total	128	–	21	26	41	40

lines were developed with a range of maturity, plant height, and seed size not previously available to growers in the blight-endemic areas in the Mediterranean region. These included 92 lines resistant to 6 races of *A. rabiei* (Singh and Reddy 1996).

Breeding for disease resistance

Conventional breeding

Ascochyta blight resistance breeding commenced in India in the early 1930s and the first resistant cultivar was developed and released about 60 years ago (Luthra *et al*. 1941). Later reports from the Soviet Union (Gushkin 1946) announced the development and release of 3 cultivars, *viz*. Skorospelka, Alpha, and Mogucii, resistant to AB. In contrast, no AB-resistant cultivars were released in the Mediterranean region until 1984. There has been slow progress due to the lack of a simple resistance screening technique, unavailability of germplasm sources with a high level of resistance, and the evolution of new races of *A. rabiei*.

At ICARDA, hybridisation work, which was initiated in 1978, attempted to combine high yield with resistance to cold and AB. Using off-season advancement facilities at Terbol in Beqa'a valley in Lebanon, more than 3000 AB-resistant and high-yielding lines have been bred between 1981 and 2002 and freely shared (Malhotra *et al*. 2003). After the initial success, most of the previously released cultivars have succumbed to new races/pathotypes of *A. rabiei*, resulting in short life span for resistant cultivars (Malhotra *et al*. 2003). The bulk-pedigree method to breed AB-resistant chickpeas was in vogue at ICARDA until 1998 when studies revealed that the efficiency of selection for AB resistance and large seed size was improved with single seed descent (SSD) at F_2 and F_3 and pedigree method from F_4 (R. S. Malhotra, ICARDA, pers. comm.). This combination of SSD and pedigree method has resulted in good progress in AB resistance breeding.

Attempts have been made to combine genes that may confer resistance against several races of *A. rabiei* in one line. Chickpea breeders at ICARDA have been successful in pyramiding a few genes from different sources using a stepwise breeding program. A good number of improved

lines, which may posses at least 4 or 5 genes for AB resistance from different genetic backgrounds are now in the final stages of development prior to being tested on a large-scale (Malhotra *et al*. 2003).

ICRISAT has concentrated on development of AB-resistant lines in desi chickpea. Multiple crosses have been used to accumulate resistance genes from diverse sources. Many of the advanced breeding lines developed from this program have shown resistance to all 4 isolates of *A. rabiei* tested under controlled-environment screening (ICRISAT 2003).

Resistance to AB has been one of the major objectives in chickpea breeding programs of many countries, such as Canada, the USA, Australia, Turkey, and Pakistan. Germplasm and breeding lines supplied by ICARDA and ICRISAT have been widely used as sources of AB resistance.

In the USA, development of AB-tolerant kabuli chickpea varieties such as 'Dwelley and Sanford' in early 1990s, helped in reducing damage from AB devastation. In recent years, 2 additional varieties, Evans and Sierra, with good levels of resistance to AB, have been released (http://pwa.ars.usda.gov/pullman/glgp/variety.html). In desi chickpea, an ICRISAT-derived line, ICCV 92809, with early maturity and good level of resistance to AB was released with the name 'Myles'. This variety was also well adapted to western Canada and spread rapidly there. The crop development centre (CDC), Saskatoon, has developed 4 AB-tolerant cultivars in desi type (CDC Anna, CDC Cabri, CDC Desiray, and CDC Nika) and 1 cultivar in kabuli type (CDC Frontier) (Warkentin *et al*. 2004).

In Australia, the first variety with moderate resistance to AB was the desi type cultivar 'Howzat' released in 2001. Australian chickpea breeders have further selected a number of desi and kabuli lines having moderate to high levels of AB resistance. These include breeding lines from ICRISAT (e.g. ICCV 96836) and ICARDA (e.g. FLIP94-508C, FLIP94-90C, FLIP 94-92C, S95362, and S95342) and selections from existing Australian varieties (e.g. Heera, Sona, and Barwon) and breeding lines (Materne *et al*. 2002). Some of these lines are in their final stages of testing and will soon be commercialised to help revive the local chickpea industry. The area cropped to chickpea in Australia is expected to increase to at least 500 000 ha once AB-resistant cultivars become widely available.

Mutation breeding has been successfully used to develop AB-tolerant varieties in Pakistan. The first variety, CM 72 (desi type), from this program was developed in 1983 and helped the chickpea industry to survive. The other mutant varieties later released included CM 88 and CM 98 in desi type and CM 2000 in kabuli type (http://www.niab.org.pk/mutation.htm). Several other AB-tolerant varieties, such as Dashat and NIFA 88, have been developed through conventional breeding methods.

In the absence of highly resistant sources, no single strategy in breeding for AB-resistant cultivars is likely to succeed. A combination of different strategies needs to be developed and utilised. The release of several cultivars, possibly with known reactions in different races/pathotypes, will be useful in case the resistance breaks down in one of the cultivars.

Marker-assisted breeding

Molecular markers linked to major quantitative trait loci (QTLs) contributing resistance have been discovered and may be used in marker-assisted breeding for resistance to AB. The markers will be important in enabling the pyramiding of resistant genes from diverse sources and should significantly reduce the time required in the development of resistant cultivars. Deoxyribonucleic acid markers will also encourage the use of exotic sources of disease resistance by dramatically improving the pace and precision of recovering the recurrent parent genome in backcross programs. Most importantly, DNA markers may help break deleterious linkage drag associated with introgressing resistance genes from wild species.

Considerable progress has been made in mapping of QTLs conferring AB resistance in chickpea. Using a recombinant inbred line (RIL) population from an interspecific cross of *C. arietinum* (FLIP84-92C, resistant parent) × *C. reticulatum* (PI 599072, susceptible parent), Santra *et al*. (2000) identified 2 major QTLs (QTL 1 and QTL 2), which accounted for >45.0% of the estimated phenotypic variation for AB resistance, and mapped these QTLs to linkage groups 6 and 1, respectively. Two RAPD markers flanked QTL 1 and were 10.9 cM apart, whereas 1 inter simple sequence repeat (ISSR) marker and 1 isozyme marker flanked QTL 2 and were 5.9 cM apart. From the same mapping population, Tekeoglu *et al*. (2002) reported that QTL 1 is linked to the microsatellite and ISSR markers GAA 47, ubc 733 and ubc 181, whereas QTL 2 is linked to microsatellite markers Ta 72A, Ta2, Ts 54, and Ta 146.

Genetic basis of host–pathogen interaction

Detailed information on the number, nature, and diversity of genes conferring resistance is a prerequisite for exploiting a particular genotype in resistance breeding programs. Initial studies suggested that AB resistance of chickpea is due to either a single dominant or a recessive gene (Singh and Reddy 1991). Depending on the mode of inheritance of resistance to AB in F_1 and F_2 generations, Singh and Reddy (1983) concluded that the resistance in ILC 72, ILC 183, ILC 200, and ILC 4935 was due to a single dominant gene, and in ILC 191 to a single recessive gene. Allelic studies by Tewari and Pandey (1986) indicated the presence of 3 independently segregating dominant genes for resistance

in P 1215-1, EC 26446, and PG 82-1, and a recessive gene in BRG 8. However, 2 dominant complementary genes were reported to control disease resistance: Arc_1 and Arc_2 in genotype GLG 84038, and Arc_3 and Arc_4 in GL 84099. Similarly, the resistance in ICC 1468 has been reported to be controlled by 1 dominant gene ($Arc_{5(3,4)}$) and 1 recessive gene (Arc_1). In these 3 genotypes, inter-allelic interactions, additive gene effects, and dominance influenced the resistance (Dey and Singh 1993).

Recent studies on RILs suggest that several QTLs are involved in controlling resistance to AB. Three sets of RILs derived from 2 intraspecific crosses, PI 359075(1) × FLIP 84-92C(2) and Blanco Lechoso × Dwelley, and 1 interspecific cross, FLIP 84-92C(3) × *C. reticulatum* (PI 489777), were developed at ARS-USDA, Pullman, WA (http://www.nps.ars.usda.gov/). Evaluation of disease response in these RILs indicated that 3 recessive complementary major genes with some modifiers conferred AB resistance. Absence of 1 or 2 of the major genes confers susceptibility while presence of the modifiers determines the degree of resistance (Tekeoglu *et al*. 2000). Conversely, 6 QTLs for AB resistance were identified in 3 regions of the genome of an intraspecific population. The major QTLs generally showed additive gene action, as well as dominance inter-locus interaction in the multiple genetic model (Flandez-Galvez *et al*. 2003*a*). Other studies report 2–6 major QTLs with various different effects and interactions (reviewed by Millan *et al*. 2005). These different estimates of the genetic basis of AB resistance result from the use of different fungal isolates and host genotypes. Clearly, AB resistance breeding is a complex endeavour, as any new cultivar needs to carry resistance genes effective against a range of AB isolates. However, these studies seem to suggest that there is a range of different sources of resistance. Pyramiding of different resistance genes may facilitate building up the level of resistance and increasing the durability of that resistance.

Studies conducted in Australia (Collard *et al*. 2003; Flandez-Galvez *et al*. 2003*a*, 2003*b*) also indicated involvement of QTLs for AB resistance. Two sets of mapping populations were used: RILs developed from an intraspecific cross involving a highly susceptible cultivar Lasseter and a resistant line ICC 12004, and an F_2 mapping population derived from a cross between the susceptible cultivar Lasseter and a resistant *C. echinospermum* accession PI 527930. Seven QTLs were identified for AB resistance and mapped on the linkage map. Two QTLs were associated with resistance at the seedling stage and 2 others were associated with adult plant resistance. Resistance Gene Analogue (RGA) and STMS markers closely flanking major resistance QTLs were identified. Two markers (CLRRinv and TA146) flanked the strongest QTL (QTL 3) at an interval of 0.1 cM. QTL 5 and QTL 7 were flanked by STMS markers, which were 1.9 (TS 12, TR 56) and 7.6 cM (M44 sp, TA 28) apart, respectively.

After validation, these flanking markers may be used in marker-assisted selection to breed for elite chickpea cultivars with durable resistance to AB. The tight linkage of RGA markers to the major QTLs will also allow map-based cloning of the AB resistance genes.

Cultural control

Cultural practices that reduce the main sources of inoculum are most important in effective disease management. Planting healthy seed, crop rotation with non-host crops such as cereals, destruction of chickpea stubble, and deep sowing are all important measures to reduce the amount of inoculum and the likelihood of an AB epidemic. Under low disease pressure, agronomic practices such as delayed sowing, lower seed rate, and wider row and plant spacing can reduce the incidence and severity of AB. Application of potassium fertilisers, especially in soils with high nitrogen content, can enhance chickpea yields and retard AB (Kader *et al*. 1990). Tillage can be used to reduce ascospore production, since burial inhibits the teleomorph formation and maturation on infected residues (Navas-Cortes *et al*. 1995). Burning of chickpea stubbles in certain environments can also reduce the inoculum build up but may not be favoured because of negative effects on soil health due to loss of organic matter and essential nutrients.

Chemical control

Although several fungicides have proved effective in control of AB, the need for their repeated application often makes them uneconomical in regions where crop yields are low. In Australia, chickpea varieties susceptible to AB have been successfully grown by strategically applying foliar fungicides such as chlorothalonil and mancozeb several times during the growing season (Bretag *et al*. 2000, 2002*b*, 2003). Seed treatment with Calixin-M (11% tridemorph + 36% maneb) (Reddy *et al*. 1982), systemic methyl benzimidazole fungicides such as benomyl or thiabendazole in combination with captan (Kaiser and Hannan 1988) produced the best results in field trials. Carbendazim and thiram (1 : 1), captan, iprodione, and propiconazole (Singh and Singh 1990; Rauf *et al*. 1992) were all effective in control of seed-borne *A. rabiei* infection.

Foliar application of propineb (Antracol), Bordeaux mixture, chlorothalonil, zineb, ferbam, maneb, captan, captafol, dithianon, propiconazole, penconazole, sulfur, and thiabendazole is also effective in control of AB. Application of these fungicides onto the infected crop is effective in reducing further development and secondary spread of AB (Bashir and Ilyas 1983; Bashir *et al*. 1987; Nene and Reddy 1987; Kaiser and Hannan 1988). Seed treatment combined with 2−3 sprays of captan, mancozeb, or chlorothalonil also effectively manages blight infection. The greatest

benefit for fungicide treatment of AB was obtained when at least one application was made before flowering (Reddy and Singh 1990).

Integrated disease management

Adoption of integrated disease management (IDM) practices is essential for economical and effective control of AB. Moderate levels of HPR can be combined with other cultural practices and/or application of minimum dosage of fungicides for control of AB. The location-specific recommended IDM practices include: (*a*) use of pathogen-free seed, (*b*) seed treatment with fungicides, (*c*) practice of crop rotation, (*d*) deep ploughing of chickpea fields to bury infested debris, (*e*) use of disease-resistant genotypes, and (*f*) strategic application of foliar fungicides.

A combination of a tolerant cv. ILC 482 and 2 sprays of chlorothalonil, one during the seedling stage and another at the early podding stage, provided the most economical field control of AB in Syria (Reddy and Singh 1990). In collaboration with the Syrian national program, ICARDA has developed an IDM package for AB management (Akem *et al*. 2000). The components of this package include use of tolerant cultivars adapted to early sowing, seed dressing with fungicides, single foliar application of chlorothalonil at seedling or early vegetative growth stages, and delayed sowing for lower disease impact. This package resulted in higher chickpea yields compared with the traditional spring plantings using a local variety without seed dressing or fungicide spray (ICARDA 2003).

Conclusions

Management of AB is essential to provide increased and stable chickpea yields throughout the world. Where possible, HPR should be emphasised over chemical control as the most environmentally friendly and economic disease control strategy. Selection of resistant sources for genetic improvement programs should be based on resistance to AB at vegetative, flowering, and podding stages, since many lines resistant in the vegetative stage can be susceptible at the podding stage. Resistance to AB in chickpea cultivars has historically been overcome by new pathotypes of *A. rabiei*, hence the genotypes intended for release to farmers should be selected based on multi-location multi-season field trials. Durable resistance may only be possible if an array of resistance genes is combined providing different mechanisms of resistance against all races in a single cultivar. Studies are underway to determine the genetics and allelic relationships of resistance to AB in different genotypes as an essential precursor to pyramid resistance genes. Knowledge of the variability of *A. rabiei* is also a prerequisite for breeding programs aimed at obtaining durable resistance to AB. Further studies on the ecology of *A. rabiei* and its epidemiology are required to improve the current disease management strategies. Both innovative and conventional approaches should be used to investigate the host–pathogen relationship between *C. arietinum* and *A. rabiei*, and to develop better methods for resistance screening. Development of marker-assisted selection methods will enable rapid screening of different genotypes and breeding populations for disease resistance. Moreover, pyramiding of different sources and/or mechanisms of resistance sharing a similar phenotype will only be possible through the application of molecular breeding tools.

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