Resistance to Ascochyta blight in chickpea (*Cicer arietinum* L.)

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Abstract: Ascochyta blight (AB), caused by *Ascochyta rabiei* is a major disease of chickpea (*Cicer arietinum* L.), capable of causing complete yield losses in areas where cool, cloudy, and humid weather persists during the crop season. 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* has not been clearly established. Deploying resistant cultivars of chickpea along with seed treatment and foliar application of fungicides are commonly recommended for AB control. However, host plant resistance is the most economical and sustainable AB management option. Therefore, in this paper we focus on HPR as the major component for integrated management of AB, with emphasis on future research priorities.

Key words: *Ascochyta rabiei*, resistance, screening techniques, epidemiology, chickpea

The Aschochyta blight

Ascochyta blight (AB), caused by fungal pathogen *Ascochyta rabiei* (Pass.) Labrousse, is the most devastating disease of chickpea and can cause up to 100% grain yield and quality losses in areas where cool, cloudy and humid weather (15-25 °C and > 150 mm rainfall) occurs during the crop season (5). The disease has been reported from 34 countries across six continents (3). The recent cultivation of chickpea in Australia and Canada has shown it can spread rapidly to new production areas. In Australia, chickpea production increased rapidly until 1999 but was then limited by outbreaks of AB. The disease is currently the most important yield limiting factor, potentially affecting 95% of the chickpea area in Australia (4). 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.

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Sign and symptoms of Ascochyta blight

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, 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 favorable temperature and rainfall. Plants are attacked at any growth stages, depending on the inoculum availability. However, AB is most prominent during the flowering to early podding growth stages. Airborne 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 lead 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, while 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 shriveled and infected seed (6, 7).

Survival, development and spread of Aschochyta blight

The causal agent of AB (*Ascochyta rabiei*) survives either on or in seed or plant debris in the form of mycelium, pycnidia and various teleomorphic stages (2). Seed transmission of *A. rabiei* and airborne 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 USA (2). 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 (1).

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. Disease severity increases with increasing periods of darkness after inoculation.

Identification and deployment 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 (5). Resistance screening using cut-twig and detached leaf techniques correlated with greenhouse screening (6). Disease rating scale commonly followed is a 1–9 scale, where 1 = no visible lesions on any plants and 9 = profuse lesions on all plants, stem girdling on more than 50% of the plants and many plants killed (6).

Deployment of resistant genotypes is the most effective way to minimize yield losses due to AB. In several studies conducted in different chickpea growing areas of the world, few sources of resistance to AB were identified (Table 1). The development of AB resistant genotypes (Table 2) has made it possible to sow the crop during winter in the Mediterranean region and reintroduce the chickpea cultivation in Australia thereby increasing the chickpea production potential. 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 utilized. 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.

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.

Table 1. Ascochyta blight reaction of 29 resistant breeding lines to *Ascochyta rabiei* in controlled environment and field screening (6)

Breeding lines	Ascochyta blight reaction (1-9 scale)[A]									
	Controlled environment				Field[B]					
	Patancheru				Ludhiana			Dhaulakuan		
	2005	2006	2007	Mean	2005	2006	Mean	2008	2009	Mean
ICCV 04524	2.0	2.0	2.0	2.0	3.0	3.0	3.0	2.0	3.0	2.5
ICCV 04525	2.3	2.0	2.6	2.3	3.0	3.0	3.0	3.0	2.0	2.5
ICCV 04526	2.3	2.6	2.0	2.3	2.3	2.7	3.0	3.0	2.0	2.5
ICCV 04537	2.3	2.0	2.6	2.3	3.0	3.0	3.0	3.0	2.0	2.5
ICCV 98811	2.7	2.5	2.9	2.7	3.0	3.0	3.0	3.0	2.0	2.5
ICCV 98816	2.3	2.6	2.3	2.3	2.7	2.7	2.7	-	2.0	2.0
ICCV 04523	2.7	3.0	2.4	2.7	2.0	2.0	2.0	2.0	2.0	2.0
ICCV 05571	2.8	3.0	2.6	2.8	3.0	3.0	3.0	3.0	2.0	2.5
ICCV 04052	3.0	2.0	4.0	3.0	3.0	3.0	3.0	-	-	-
ICCV 04530	3.0	3.0	3.0	3.0	3.0	3.0	3.0	3.0	-	3.0
ICCV 05546	3.7	3.0	2.3	3.0	2.7	2.3	3.0	3.0	-	3.0
ICCV 05514	3.0	2.3	3.7	3.0	3.0	3.0	3.0	2.0	2.0	2.0
ICCV 04505	3.3	3.0	2.7	3.0	2.7	2.3	3.0	3.0	2.0	2.5
ICCV 05502	3.0	3.3	2.7	3.0	3.0	3.0	3.0	3.0	2.0	2.5
ICCV 05512	2.7	4.0	2.3	3.0	3.0	3.0	3.0	3.0	3.0	3.0
ICCV 04509	2.3	4.0	2.7	3.0	3.0	3.0	3.0	3.0	2.0	2.5
ICCV 05547	3.7	3.0	2.3	3.0	3.0	3.0	3.0	3.0	-	3.0
ICCV 05551	3.7	3.0	2.3	3.0	3.0	3.0	3.0	3.0	3.0	3.0
ICCV 05503	2.0	4.0	3.0	3.0	3.0	3.0	3.0	3.0	-	3.0
ICCV 05511	2.3	4.0	2.7	3.0	3.0	3.0	3.0	3.0	2.0	2.5
ICCV 05513	2.7	3.0	3.3	3.0	2.3	3.7	3.0	3.0	2.0	2.5
ICCV 05515	3.0	3.3	2.7	3.0	3.3	2.7	3.0	3.0	2.0	2.5
ICCV 05523	3.0	3.0	3.0	3.0	4.0	2.0	3.0	3.0	2.0	2.5
ICCV 05532	2.7	3.3	3.0	3.0	3.3	2.7	3.0	3.0	2.0	2.5
ICCV 98818	3.0	3.3	2.7	3.0	3.0	3.0	3.0	3.0	3.0	3.0
ICCV 04512	3.0	3.0	3.0	3.0	3.0	3.0	3.0	3.0	2.0	2.5
ICCV 05530	3.0	3.0	3.0	3.0	3.0	3.0	3.0	3.0	2.0	2.5
ICCV 04513	3.0	3.7	2.3	3.0	3.0	3.0	3.0	3.0	2.0	2.5
ICCV 05531	3.0	3.3	2.7	3.0	3.0	3.0	3.0	2.0	2.0	2.0
ICC 4991 (Sus. check to AB)	9.0	9.0	9.0	9.0	9.0	9.0	9.0	9.0	7.0	8.5
SEM	0.25	0.25	0.26		0.25	0.31		0.28	0.34	
SED	0.35	0.35	0.36		0.36	0.44		0.38	0.42	
Cv (%)	13.95	12.71	14.48		13.67	16.19		14.75	15.84	
l.s.d. (5%)	0.71	0.71	0.74		0.73	0.89		0.81	0.71	

Table 2. Some chickpea lines released in different countries, with acceptable level of resistance to Ascochyta blight (5)

Accession	Country of origin	Country of release	Released name	Year of release
ILC 72	N.A.[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

Conclusion

Management of AB using resistant cultivars is essential to provide increased and stable chickpea yields throughout the world. Wherever possible, HPR should be emphasized over chemical control as the most environmentally friendly and economic, AB control strategy. Selection of resistant sources for genetic improvement programs and cultivars 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 multilocation multi-season field trials. Durable resistance may only be possible if arrays of resistance genes are combined providing different mechanisms of resistance against all races in a single cultivar. ■

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