Patterns of Resistance and Susceptibility to Races of *Ascochyta rabiei* Among Germ Plasm Accessions and Breeding Lines of Chickpea

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ABSTRACT

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To identify sources of resistance to the six races of *Ascochyta rabiei* reported from Lebanon and Syria, 1,069 germ plasm accessions and breeding lines were screened against the races in the greenhouse at Tel Hadya, Syria, during 1985–1986. Preliminary screening of the germ plasm was done by inoculating 10-day-old seedlings. Lines with little infection were retested in the seedling and podding stages. Of the total lines, 47, 27, 29, 8, 13, and 4 were resistant to races 1, 2, 3, 4, 5, and 6, respectively. Although different lines appeared to carry genes for resistance to several races, none was resistant to all races. Three lines (ILC-202, ILC-3856, and ILC-5029) were resistant to five races and are being used in breeding programs at ICARDA, ICRISAT, and national programs of North Africa, western Asia, southern and eastern Europe, and the Indian subcontinent.

Ascochyta blight, caused by *Ascochyta rabiei* (Pass.) Lab. (teliomorph *Mycosphaerella rabiei* Kov.), is the most damaging disease of chickpea (*Cicer arietinum* L.) in western Asia, North Africa, southern and eastern Europe, and the northwestern region of India and Pakistan. The disease has been reported from 26 countries (11). If environmental conditions result in development of severe disease, yield loss may reach 100%. The use of resistant cultivars is the most effective and economical way to control Ascochyta blight.

The first report of resistance to Ascochyta blight in 1931 (1) has been followed by many additional sources of resistance, summarized by Singh et al (17). At ICARDA, more than 13,000 germ plasm accessions have been screened and a few resistant lines have been identified (14). Promising material from this collection was tested internationally, and many national programs have identified resistant sources (18). In the international evaluation of resistant lines, a differential interaction was found among different locations, indicating variation in the pathogen in different countries (16).

Kovachevski (8) first observed the sexual stage of *A. rabiei* in Bulgaria in 1936 and named it *M. rabiei*. Later, it was confirmed from Greece, Hungary, the United States, and the USSR (4,7,9,20). Early workers (2,10) had not found races, however. In 1969, several races were first reported from the state of Punjab in India (3). Subsequently, two races were reported in India and six in Syria and Lebanon (13,19). New races have also been identified in Italy (12) and Pakistan (B. A. Malik, *personal communication*).

The objective of this study was to identify sources of resistance to the six races reported from Lebanon and Syria through the evaluation of 1,069 germ plasm accessions and breeding lines in the greenhouse.

MATERIALS AND METHODS

Source of germ plasm. Of the 1,069 lines evaluated, 943 were kabuli (charac-

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terized by large ramhead-shaped beige seeds) germ plasm accessions, 76 were desi (characterized by small angular colored seeds) germ plasm accessions, and 50 were ICARDA breeding lines. The breeding lines were developed for Ascochyta blight resistance at ICARDA. The germ plasm accessions included all identified sources of resistance to Ascochyta blight from earlier screenings at ICARDA (14,16–18).

Screening method. Germ plasm accessions were screened in the greenhouse from November 1985 to April 1986 at Tel Hadya, ICARDA's principal experiment station in Syria. The temperature was maintained between 10 and 21 C. Inoculum was produced on chickpea-dextrose broth (40 g of chickpea seed meal, 20 g of dextrose, 1 L of water), and 10-day-old cultures were used (13,14). In the preliminary screening, 10 seeds from each of 20 accessions were sown in an iron tray (45 × 45 × 7 cm). When 10 days old, seedlings were sprayed with a spore suspension (200,000 spores per milliliter) of the fungus until runoff. Groups of trays were incubated in low clear-plastic cages (0.5 × 2 × 2 m) for 10 days at 100% relative humidity. Screening for resistance to the six races was done in two trials because of space limitations in the greenhouse. All lines were screened individually to races 1, 2, and 3 in the first trial and to races 4, 5, and 6 in the second. Each trial was completed in 1 mo.

Lines with resistant reactions were retested. Five seeds of each of these lines were sown in 5-L plastic pots, and each pot constituted one replication. The experiment followed a randomized block design with three replications. Ten-day-old plants were inoculated with a spore suspension in the same manner as described previously. Plants were reinoculated at the podding stage because previous studies have shown that many chickpea lines expressing resistance in the vegetative stage develop severe infection on pods in the podding stage (14).

Blight score scale. Blight severity was recorded on a nine-point scale (14,16). Host response and symptom development on vegetative parts and pods (breakage of branches and pod infection) were classified as: 1 = no infection, 2 = highly resistant (1–5%), 3 = resistant (6–10%), 4 = moderately resistant (11–15%), 5 = intermediate (16–40%), 6 = moderately susceptible (41–50%), 7 = susceptible (51–75%), 8 = highly susceptible (76–100%), and 9 = plants killed. The highest score in any of the tests (not the average score) was used to categorize the line as resistant or susceptible.

Table 1. Frequencies of disease scores of 1,069 chickpea lines inoculated with six races of *Ascochyta rabiei* in the greenhouse at Tel Hadya, Syria, 1985–1986

Disease score[a]	Number of accessions					
	Race 1	Race 2	Race 3	Race 4	Race 5	Race 6
1	0	0	0	0	0	0
2	0	0	1	0	1	0
3	20	9	2	1	6	2
4	27	18	26	7	6	2
5	25	2	15	2	1	0
6	154	74	199	37	75	37
7	76	75	298	51	84	95
8	31	16	173	19	15	52
9	736	875	355	952	881	881

[a] Severity of blight on both vegetative parts and pods, with 1 = no infection, 2 = highly resistant (1–5%), 3 = resistant (6–10%), 4 = moderately resistant (11–15%), 5 = intermediate (16–40%), 6 = moderately susceptible (41–50%), 7 = susceptible (51–75%), 8 = highly susceptible (76–100%), and 9 = plants killed.

Table 2. Chickpea lines showing resistance to three to five races of *Ascochyta rabiei*

Line	Reaction[a]					
	Race 1	Race 2	Race 3	Race 4	Race 5	Race 6
ILC-72	R	R	R	R	S	S
ILC-190	R	S	R	S	R	S
ILC-201	R	R	S	R	R	S
ILC-202	R	R	R	R	R	S
ILC-482	R	R	S	S	R	S
ILC-2506	R	R	S	R	S	R
ILC-2956	R	S	R	S	R	R
ILC-3279	R	S	R	R	R	S
ILC-3856	R	R	R	R	S	R
ILC-5928	R	R	S	R	R	R
FLIP 83-48C	R	R	R	S	R	S
ICC-3996	R	R	R	S	S	S

[a] R = resistant (score of 2, 3, or 4 on a 1–9 disease scale), S = susceptible (score of 6, 7, 8, or 9).

Table 3. Proposed set of differentials to identify races of *Ascochyta rabiei* in chickpea

Genotype	Reaction[a]					
	Race 1	Race 2	Race 3	Race 4	Race 5	Race 6
Pch 15	R	S	S	S	S	S
ILC-194	R	R	S	S	S	S
ICC-3996	R	R	R	S	S	S
ILC-72	R	R	R	R	S	S
ILC-202	R	R	R	R	R	S
ILC-5928	R	R	S	R	R	R
ILC-1929	S	S	S	S	S	S

[a] R = resistant (score of 2, 3, or 4 on a 1–9 disease scale), S = susceptible (score of 6, 7, 8, or 9).

RESULTS

The frequencies of Ascochyta blight scores of the 1,069 chickpea germ plasm accessions and breeding lines for six races of *A. rabiei* are given in Table 1. Lines 47, 27, 29, 8, 13, and 4 were categorized as resistant (scores of 2, 3, or 4) to races 1, 2, 3, 4, 5, and 6, respectively.

The following are lines considered to possess resistance (scores of 2, 3, or 4) to the indicated races. Identification is by ICARDA germ plasm accession and breeding line codes: ILC = kabuli germ plasm accessions, ICC = desi germ plasm accessions, FLIP = kabuli breeding lines, AUG = Agricultural University Gram (Pakistan), Pch = Pois chiche (Morocco), and G = Gram (P.A.U., India).

Race 1: ILC-72, -190, -191, -192, -194, -201, -482, -484, -2506, -2548, -2555, -2956, -3279, -3346, -3856, -4421, and -5928; FLIP 81-41W, 82-93C, 83-7C, 83-12C, 83-21C, 83-46C, 83-47C, 83-48C, and 83-60C; ICC-1069, -2160, -3578, -3737, -3916, -3918, -3940, -3996, -5035, -5127, -6304, -6306, -6336, and -6373; AUG 480; Pch 15.

Race 2: ILC-72, -185, -186, -187, -194, -201, -202, -482, -3280, -2506, -3001, -3340, -3856, -3864, -3870, and -5928; FLIP 82-144C, 82-239C, 83-12C, 83-28C, 83-48C, and 83-60C; ICC-399 and -6981; G 549.

Race 3: ILC-72, -182, -190, -202, -2956, -3279, and -3856; FLIP 81-41W, 82-26C, 82-91C, 82-150C, 82-259C, 83-13C, and 83-48C; ICC-1467, -1468, -1591, -3912, -3996, -4107, -4192, -4472, -6373, -6981, -6988, and-6989; G 549.

Race 4: ILC-72, -200, -201, -202, -2506, -3279, -3856, and -5928.

Race 5: ILC-190, -200, -201, -202, -249,

-482, -2956, -3279, and -5928; FLIP 83-47C and 83-48C; ICC-5035 and -6988.

Race 6: ILC-2506, -2956, -3856, and -5928.

Lines with resistance to three to five races of *A. rabiei* are listed in Table 2. Three lines (ILC-202, -3856, and -5928) had resistance to five races, six (ILC-72, -201, -2506, -2956, and 3279 and FLIP 83-48C) had resistance to four races, and three (ILC-190 and -482 and ICC-3996) had resistance to three races.

DISCUSSION

A. rabiei is highly variable, and there is a need to study the extent of this variability and its distribution if host-plant resistance is to be a worthwhile control measure. The primary sources of inoculum known for Ascochyta blight are infected seed and diseased debris. However, development of severe epiphytotics of blight in fields where healthy seed has been used and with no history of chickpea cultivation is not uncommon. This raises the possibility of long-distance dispersal of inoculum, with the need for genotypes resistant to the prevailing races of the fungus.

None of the lines that originated from Syria and Lebanon were found to be resistant to any of the six races identified from these countries. Almost all the resistant lines originated from Afghanistan, Iran, Turkey, and the USSR. Also, no variability in plants of a line for resistance to *A. rabiei* was observed. By planting chickpea late in the spring, farmers save their crop from Ascochyta blight damage, but seed yield is greatly reduced because of moisture and heat stresses. Clearly, late planting as a control measure is detrimental to high chickpea production.

Kabuli lines were more resistant than the desi type to races 4, 5, and 6. Except for ICC-3996, which is a desi type, all lines that showed multiple race resistance were kabuli types. One reason for this could be that Ascochyta blight is the major disease of chickpea in western Asia, North Africa, and southern Europe, where kabuli chickpea is grown almost exclusively. This was also true when the world collection of desi and kabuli types was evaluated (14). Another reason could be that Asia Minor, where kabuli types are cultivated, is the primary center of origin for chickpea.

Chickpea is known to differ in reaction to blight, depending on age. Many lines resistant in the vegetative stage show high susceptibility in the podding stage (14). Ascochyta blight disease usually affects chickpea in the flowering and podding stage. Although this study was not set up to develop differentials, seven lines (Pch 15, ICC-3996, and ILC-194, -72, -202, -5928, and -1929) could be used as differentials for identifying the six races of *A. rabiei* in this study (Table 3). Earlier differentials were based on disease reaction in only the vegetative stage (13). The present differentials will be more useful in identifying the races in *A. rabiei* when infected in either the vegetative or the reproductive stage.

Because of the presence of numerous races in *A. rabiei*, it would be difficult to develop cultivars that are resistant across all locations. Breeding efforts have to be race-specific, at least at present. To counter the race situation, mixtures of lines (5) and selection of lines with an intermediate reaction (15) have been proposed in other crop disease situations. We tried both of these methods in the early 1980s and failed to control Ascochyta blight in chickpea. In epiphytotic form, this disease kills the crop within 1 wk. Hence, only highly resistant cultivars can contain this disease. Efforts continue to pyramid genes for resistance to the six races in a single genotype. Fungicide control has been unreliable and uneconomic (6). It is imperative that pathologists and breeders combine efforts to control *A. rabiei* through host-plant resistance.

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