Resistance in Kabuli Chickpeas to Fusarium Wilt

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ABSTRACT

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Fusarium wilt (*Fusarium oxysporum* f. sp. *ciceris*) is a major constraint to production of kabuli chickpeas (*Cicer arietinum*) in the Mediterranean basin. To identify sources of resistance to Fusarium wilt for use in a breeding program, 1,904 lines, including 713 lines tolerant to Ascochyta (*Ascochyta rabiei*) blight and cold bred at the International Center for Agricultural Research in the Dry Areas (ICARDA) and 991 germ plasm lines, were screened in a field plot heavily infested with *F. o. ciceris* at Santaella, Córdoba, Spain, in 1987 and 1989. Nine lines, FLIP 84-43C (ILC-5411), FLIP 85-20C, FLIP 85-29C, FLIP 85-30C, ILC-127, ILC-219, ILC-237, ILC-267, and ILC-513, were highly resistant with 0–9% dead plants. Inoculations of 17 selected lines, including FLIP 84-43 C, ILC-127, ILC-219, ILC-237, and ILC-513, with races 0 and 5 of *F. o. ciceris* and with isolates Fo 8726 and Fo 8733 of the pathogen not characterized to race from affected plants in the field plot, indicated that all lines were resistant to race 0, and only one (FLIP 85-130 C) was resistant to race 5 and isolate Fo 8726. The reaction of breeding lines in the field was correlated primarily to reactions induced by race 0 and isolate Fo 8733. Lines that showed resistance in the field were susceptible to race 5 and to isolate Fo 8726, except for ILC-219, ILC-256, ILC-487, and ILC-513, which were resistant to isolate Fo 8726 but not to race 5.

Fusarium wilt caused by *Fusarium oxysporum* Schlechtend.:Fr. f. sp. *ciceris* (Padwick) Matuo & K. Sato and Ascochyta blight caused by *Ascochyta rabiei* (Pass.) Labrousse are major constraints to chickpea (*Cicer arietinum* L.) production in the Mediterranean region and the Indian subcontinent (9,17,24). Yield losses up to 10% have been reported attributable to Fusarium wilt in India (27) and Spain (31) and up to 40% in Tunisia (1). Yield losses attributable to Ascochyta blight are even higher and have been reported up to 50% in Pakistan (25) and 30% in Syria (5).

Crop rotation, pathogen-free seed, destruction of infected plant debris, and fungicide sprays are recommended control measures for Ascochyta blight and Fusarium wilt. However, the most practical and economical strategy for control of both diseases is the use of resistant cultivars (24). Accordingly, improving chickpea for resistance to these two diseases has been one of the main objectives in chickpea breeding programs (26). Good progress has been made in the identification of sources of resistance to Ascochyta blight (18,30) and Fusarium wilt (10,13,34) in both desi (small, angular, colored seeds) and kabuli (large, ramhead-shaped, beige seeds) types. Also, good progress has been made in the development of wilt-resistant, high-yielding kabuli cultivars at the International Crops Research Institute for the Semiarid Tropics (ICRISAT) (19, 20,34), in Mexico (21,26), and in California (2,3).

Recently, a wilt-resistant cultivar was developed by screening and selection from a local landrace in Tunisia (7,8). However, except for a few cases (22), wilt-resistant kabuli cultivars are susceptible to Ascochyta blight (R. M. Jiménez-Díaz and A. Trapero-Casas, *unpublished*) (8), and cultivars resistant to Ascochyta blight are susceptible to wilt (R. M. Jiménez-Díaz and A. Trapero-Casas, *unpublished*) (15). Nene (22) has emphasized the need to develop chickpea cultivars with combined resistance to several of the important diseases.

This paper reports the results of screening for resistance to Fusarium wilt in kabuli breeding lines with tolerance to Ascochyta blight and cold developed at the International Center for Agricultural Research in the Dry Areas (ICARDA), Aleppo, Syria, and germ plasm lines.

MATERIALS AND METHODS

Screening of the breeding lines was conducted in a field with clay soil, pH 7.8–8.1, with about 1% organic matter at Santaella (latitude 38° north, longitude 5° east), Córdoba, Spain, in 1987 and 1989. The field was heavily infested with *F. o. ciceris*. Wilt incidence in this field was 80% in 1981, and since then, it has been cropped continuously with chickpea to develop a site suitable for studies on the epidemiology and control of Fusarium wilt. Seedbed preparation and application of insecticides and herbicides were done according to farmers' practices (6). Seeding in both years was done in mid-March. For each entry, 40 seeds were sown in single rows 4 m long and 50 cm apart.

In 1987, 713 lines tolerant to Ascochyta blight and cold were screened. Single rows of line ILC-1929 and cultivars PV 60 and JG 62 were used as repeated susceptible checks. JG 62 and PV 60 were sown in consecutive rows every five test entries, and ILC-1929 was sown every other five test entries alternating with JG 62 and PV 60. JG 62 is a desi cultivar susceptible to races of *F. o. ciceris* described in India (12) but resistant to race 0, which is prevalent in southern Spain (4,16). PV 60 is a Spanish kabuli landrace with large, white seeds and is highly susceptible to Fusarium wilt.

In 1989, 991 new germ plasm lines, together with 14 of the most promising resistant lines identified in 1987, were screened. In addition, lines from the ICARDA North Africa Regional Chickpea Fusarium Wilt Nursery-1989 (INARCFWN-89), which included four of those 14 resistant genotypes, were also screened. For the new germ plasm in 1989, ILC-1929 was sown every 10 test entries with JG 62 and PV 60 sown in consecutive rows every other 10 test entries alternating with ILC-1929. The 14 resistant lines were replicated twice in a completely randomized design, with the same susceptible checks used as for the testing of new germ plasm. Lines from INARCFWN-89 were replicated twice in a randomized complete block design. Susceptible line ILC-4090 was sown every two test entries with JG 62 and PV 60 sown in consecutive rows every six test entries.

In 1987 and 1989, a set of race differential cultivars for *F. o. ciceris* (12) was sown in a randomized complete block design with two replicates in the same plot area.

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Disease reactions were assessed by recording the incidence of dead plants. Stand counts were made 3 wk after sowing. Observations on wilt development were made regularly, and the increase in incidence of dead plants (total cumulative number of dead plants/total plant number at stand count) was obtained by recording and removing dead plants in each entry at 40, 60 (flowering), and 90 (physiological maturity) days after sowing. Disease reactions were classified according to the percentage of dead plants (12) as resistant (0–20%), moderately resistant (21–50%), and susceptible (51–100%). The highest rating in any of the replications was used to categorize the line. At each recording date, isolations were made from a sample of 50 dead plants. Collar and stem tissues were washed in running tap water, cut into pieces 5–10 mm long, surface-disinfested in 1% NaOCl for 2 min, plated on potato-dextrose agar (PDA), and incubated at 23–27 C for 7 days. Fungi growing from the tissues were identified.

The pathogenicity of 17 isolates of *F. oxysporum* from infected plants to cultivars JG 62 (resistant to race 0) and P 2245 (susceptible to all races of the pathogen) (4,16) was tested with mass cultures by the water-culture inoculation method (23,31). Thereafter, two isolates (Fo 8726 and Fo 8733) representative of the range of pathogenicity to JG 62 were selected and single-spored for use in further inoculation experiments. Seventeen lines, some of which had shown resistance in the field screening, were tested for disease reaction to these isolates and to an isolate each of race 0 and 5 of *F. o. ciceris* obtained from previous studies (4,16) by the pot-culture inoculation method (23,31).

Isolates were grown on PDA and incubated at 23 ± 3 C with a 12-hr photoperiod of fluorescent and near-UV light at 36 $\mu E \cdot m^{-2} \cdot s^{-1}$. Inocula for water and pot cultures were increased in potato-dextrose broth (PDB) and a cornmeal-sand (CMS) mixture (23,31), respectively, and incubated under the same conditions as isolates for 2 wk. Liquid cultures containing mostly conidia were diluted to 2.5% with sterile distilled water and distributed into 6-cm-diameter cylindrical glass bottles. Seedlings grown for 7–10 days in sterile sand were removed, washed free of sand, transferred without intentional wounding to the bottles (four per bottle), and the bottles were placed on a rotary shaker run at 110–120 rpm. Control plants were placed in bottles filled with diluted sterile PDB. Sterile distilled water was added to bottles every 3 days to replace the water lost.

For pot-culture inoculations, infested CMS was mixed thoroughly (1:12, w/w) with an autoclaved soil mixture (clay loam/sand/peat, 1:1:1, v/v/v) and germinated seeds were sown in pots filled with that mixture (23,31). Control plants were grown in a comparable mixture of noninfested CMS and autoclaved soil. Plants were grown in a growth chamber at 26 ± 2 C/21 ± 2 C (day/night) and 60–90% relative humidity, with a 14-hr photoperiod of fluorescent light at approximately 252 $\mu E \cdot m^{-2} \cdot s^{-1}$. Plants were fertilized weekly with 100 ml of nutrient solution (14). Plants were observed daily for symptoms.

Disease reactions were assessed by symptom severity on a 0–4 scale according to percentage of foliage with yellowing or necrosis in acropetal progression (0 = 0, 1 = 1–33, 2 = 34–66, 3 = 67–100%, and 4 = dead plant). Experiments were conducted following a randomized complete block design with three or four replicated bottles or pots (four plants per pot) for each isolate-plant genotype combination.

RESULTS AND DISCUSSION

Symptoms characteristic of Fusarium wilt affected a few plants 40 days after sowing, but an increased incidence of the disease occurred as each season progressed. *F. oxysporum* was the primary fungus isolated from collar and stem tissues, although in a few cases, *F. solani* (Mart.) Sacc. was isolated from collar tissues together with *F. oxysporum*. The highly susceptible check lines ILC-1929 and PV 60 had a nearly uniform susceptible reaction throughout the nursery, which suggests that the pathogen was equally spread throughout the plot (Tables 1 and 2). Wilt developed faster in line ILC-1929 than in line PV 60, although the final incidence of dead plants in the two lines was comparable (Tables 1 and 2). Cultivar JG 62 was less susceptible than line ILC-1929 and cultivar PV 60, as indicated by the mean incidence of dead plants and the percentage of rows with a final incidence of dead plants higher than 50% (Tables 1 and 2). Although wilt incidence in JG 62 varied from 60.9 to 47.8% in the 2 yr of screening, that incidence of dead plants was much higher than 15–16% observed in the same plot in 1983 (R. M. Jiménez-Díaz and A. Trapero-Casas, *unpublished*).

Cultivar JG 62 is considered to be a universal susceptible check for identification of races of *F. o. ciceris* at ICRISAT (12). However, JG 62 is highly resistant to race 0, which is pathogenic to Spanish kabuli chickpeas (4,16). The low wilt incidence in JG 62 in 1983 suggests that race 0 of the pathogen was prevalent in the plot at that time. Therefore, the increase in wilt incidence occurring in JG 62 during 1987 and 1989 compared with that in 1983 might indicate that a shift in the status of the pathogen race present in the plot at Santaella may have occurred.

Disease reaction of race differential cultivars sown in this plot in 1987 and 1989 indicated that cultivars 12-071/10054, JG 62, and C 104 were susceptible, whereas cultivars Annigeri and Chaffa

Table 1. Reaction of breeding and germ plasm lines of chickpea to Fusarium wilt in a field heavily infested with *Fusarium oxysporum* f. sp. *ciceris* at Santaella, Córdoba, Spain, in 1987 and 1989

Breeding line[a]	Number of entries	Percent entries with emergence of 80–100%	Entries (%) with incidence of dead plants[b] after					
			2 mo			3 mo		
			R[c]	MR[d]	S[e]	R[c]	MR[d]	S[e]
1987								
FLIP lines	713	38.8	45.4	30.5	24.1	2.2	2.7	95.1
ILC-1929	83	63.8	13.2	66.3	20.5	0.0	1.2	98.8
PV 60	82	70.7	74.4	21.9	3.7	1.2	0.0	98.8
JG 62	82	65.8	65.8	14.6	19.6	7.3	25.6	67.1
1989								
ILC lines	991	17.2	16.5	14.2	69.3	1.1	3.9	95.0
ILC-1929	55	43.6	1.8	29.1	69.1	0.0	3.6	96.4
PV 60	106	30.5	45.3	21.7	33.0	0.0	0.0	100.0
JG 62	105	9.5	36.2	40.0	23.8	5.7	59.1	35.2

[a] FLIP = breeding lines; ILC = germ plasm lines. ILC-1929, PV 60, and JG 62 are susceptible check lines. PV 60 and JG 62 were sown in consecutive rows every five and 10 test entries in 1987 and 1989.
[b] Total cumulative number of dead plants/total number of plants at stand count 3 wk after sowing.
[c] R = resistant (0–20% plants killed).
[d] MR = moderately resistant (21–50% plants killed).
[e] S = susceptible (51–100%) plants killed.

Table 2. Incidence of dead plants (%)[a] and reaction of selected chickpea lines to Fusarium wilt in a field heavily infested with *Fusarium oxysporum* f. sp. *ciceris* at Santaella, Córdoba, Spain, in 1987 and 1989[b]

Breeding line	1987[c]		1989[c]		Reaction class[d]	INARCFWN/89[c]		Reaction class[d]	Days to 50% flower (no.)	Plant height (cm)	100-seed weight (g)
	2 mo	3 mo	2 mo	3 mo		2 mo	3 mo				
FLIP 82-78 C[e]	0.0	7.7	6.2	33.0	MR	6.5	35.1	MR	140	64	27
FLIP 84-39 C	4.0	12.0	4.2	14.8	MR-R	...	...	...	149	52	27
FLIP 84-43 C	0.0	2.7	0.0	9.0	R	...	...	...	146	47	29
FLIP 85-20 C	0.0	0.0	0.0	0.0	R	3.7	8.3	R	...	...	...
FLIP 85-29 C	0.0	0.0	0.0	6.2	R	0.0	0.0	R	...	...	...
FLIP 85-30 C	0.0	3.7	2.5	4.2	R	0.0	1.9	R	...	...	...
FLIP 85-36 C	12.0	12.0	17.5	22.1	MR-R	...	...	...	...	...	...
FLIP 85-37 C	0.0	8.0	3.8	12.5	MR-R	...	...	...	...	...	...
FLIP 85-85 C	0.0	0.0	1.8	18.5	MR-R	...	...	...	134	46	27
FLIP 85-130 C	7.1	10.7	0.0	21.1	MR-R	...	...	...	142	53	26
ILC-1929[f]	39.1 ± 14.8	91.2 ± 8.7	61.7 ± 19.7	84.5 ± 12.8	S	56.8 ± 20.9	100	S	134	52	34
PV 60[f]	19.1 ± 15.6	95.5 ± 10.2	34.6 ± 30.9	95.4 ± 6.5	S	21.8 ± 13.1	96.4 ± 2.1	S	...	...	...
JG 62[f]	23.1 ± 24.3	60.9 ± 23.5	31.8 ± 25.5	47.8 ± 21.1	S	8.1 ± 5.9	43.2 ± 7.1	S	...	...	...
ILC-54	...	...	5.9	15.0	R	...	...	...	134	42	37
ILC-127	...	...	0.0	3.7	R	...	...	...	134	66	40
ILC-219	...	...	0.0	0.0	R	...	...	...	138	51	30
ILC-237	...	...	0.0	0.0	R	...	...	...	136	65	38
ILC-240	...	...	13.6	13.6	R	...	...	...	138	58	35
ILC-256	...	...	0.0	12.5	R	...	...	...	138	54	37
ILC-267	...	...	0.0	0.0	R	...	...	...	134	35	20
ILC-336	...	...	0.0	15.6	R	...	...	...	136	60	34
ILC-487	...	...	0.0	12.5	R	...	...	...	130	52	42
ILC-513	...	...	0.0	0.0	R	...	...	...	144	48	47

[a] Total cumulative number of dead plants/total number of plants at stand count 3 wk after sowing.
[b] Each entry of FLIP and ILC lines screened in 1987 and 1989, respectively, were sown in non-replicated rows. FLIP lines tested again in 1989 were replicated twice in a completely randomized design. FLIP lines tested in 1989 as part of the INARCFWN/89 were replicated twice in a randomized complete block design.
[c] Incidence after 2 and 3 mo. INARCFWN/89 = ICARDA (International Center for Agricultural Research in the Dry Areas) North Africa Regional Chickpea Fusarium Wilt Nursery 1989.
[d] Highest disease reaction in two replicated rows. Rating scale: R = resistant (0–20% plants killed), M = moderately resistant (21–50% plants killed), S = susceptible (51–100% plants killed) according to Haware and Nene (12).
[e] FLIP = breeding lines; ILC = germ plasm lines.
[f] Susceptible checks: mean and standard deviation of 82 replicated rows for each entry in the 1987 screening; 55, 106, and 105 replicated rows for ILC-1929, PV 60, and JG 62, respectively, in the 1989 screening; and 26, eight, and eight replicated rows for ILC-4090, PV 60, and JG 62, respectively, in the INARCFWN/89.

Table 3. Disease reactions of race-differential chickpea cultivars in a field heavily infested with *Fusarium oxysporum* f. sp. *ciceris* at Santaella, Córdoba, Spain, in 1987 and 1989

Cultivar	1987		1989	
	Mean incidence of dead plants (%)[a]	Reaction class[b]	Mean incidence of dead plants (%)[a]	Reaction class[b]
12-071/10054	...	...	100.0	S
JG 62	85.5	S	71.5	S
C 104	46.5	S	55.0	S
JG 74	3.0	R	18.5	M
CPS 1	0.0	R	11.5	R
BG 212	1.0	R	5.5	R
WR 315	0.0	R	11.0	R
Annigeri	5.0	R	24.0	M
Chaffa	17.5	M	27.0	M
L 550	97.5	S	86.0	S
K 850	4.5	R	11.0	R
P 2245	...	...	100.0	S
PV 60	86.6	S	91.6	S
Sonora 80	...	S	4.6	R
Surutato 77	...	...	5.9	R
ICCV 2	...	...	1.5	R
ICCV 4	...	...	1.5	R
ICCV 5	...	...	1.5	R

[a] Total cumulative number of dead plants/total number of plants at stand count 3 mo after sowing. Mean of two replications.
[b] Highest disease reaction in two replications. Rating scale: R = resistant (0–20% plants killed), M = moderately resistant (21–50% plants killed), and S = susceptible (51–100% plants killed) according to Haware and Nene (12).

were moderately susceptible and cultivar K 850 was resistant (Table 3). This pattern of disease reaction is close to that of race 1, except for the resistant reaction of K 850, which is susceptible to this race (12). The susceptible reaction of cultivars JG 62 and 12-071/10054 indicates that neither race 0 nor race 5 were the prevalent races at the area where the race-differential cultivars were sown, because races 0 and 5 are not pathogenic to cultivars JG 62 and 12-071/10054, respectively (4,16). Nevertheless, because the soil was naturally infested, the possibility that more than one race was present in the plot cannot be ruled out.

Of 713 breeding lines and 991 germ plasm lines screened in 1987 and 1989, 95.6 and 96.8%, respectively, were susceptible. Further, some 87.5% of the susceptible lines had an incidence of dead plants exceeding 86.0%. Of the screened lines, 1.3 and 1.1% were resistant in the 1987 and 1989 screenings, respectively. Four lines (FLIP 84-43 C, FLIP 85-20 C, FLIP 85-29 C, and FLIP 85-30 C) had a highly resistant reaction, and six lines (FLIP 82-78 C, FLIP 84-39 C, FLIP 85-36 C, FLIP 85-37 C, FLIP 85-85 C, and FLIP 85-130 C) had a resistant to moderately resistant reaction in replicated tests (Table 2). These lines also have tolerance to cold and Ascochyta blight. Also, four germ plasm lines (ILC-219, ILC-237, ILC-267, and ILC-513) were highly resistant in the unreplicated screening of 1989.

Resistant lines were either free of disease or had a low (0–9%) final incidence of dead plants 3 mo after sowing. In most cases, these lines were also free of disease

Table 4. Reactions of selected chickpea breeding and germ plasm lines to isolates and races of *Fusarium oxysporum* f. sp. *ciceris* in the growth chamber[a]

Entry[b]	Race 0[c]	Race 5	Isolates from the field plot[c]	
			Fo 8726	Fo 8733
FLIP 82-78 C	R	S	M	S
FLIP 83-108 C	R	S	M	S
FLIP 84-39 C	R	S	S	R
FLIP 84-43 C	R	S	M	R
FLIP 84-65 C	R	S	S	R
FLIP 84-66 C	R	S	S	R
FLIP 84-130 C	R	S	S	R
FLIP 85-130 C	R	R	R	M
ILC-54	R	S	S	M
ILC-127	R	S	S	R
ILC-219	R	S	R	R
ILC-237	R	S	S	R
ILC-240	R	S	M	R
ILC-256	R	S	R	R
ILC-336	R	S	S	R
ILC-487	R	S	R	R
ILC-513	R	S	R	R
P 2245	S	S	S	S
JG 62	R	S	S	R
12-071/10054	S	R	R	S

[a] Assessed on a 0–4 scale according to the percentage of foliage with yellowing or necrosis in acropetal progression (0 = 0, 1 = 1–33, 2 = 34–66, 3 = 67–100%, and 4 = dead plant) 40 days after inoculation. Scores ≤ 1 and ≥ 3 were considered as resistant (R) and susceptible (S) reactions, respectively. Scores in between were considered as moderately susceptible reaction (M). Sixteen replicated plants were included per entry. Uninoculated control plants of all entries remained free from infection.

[b] Entries are in the chickpea germ plasm collection at the International Center for Agricultural Research in the Dry Areas (ICARDA), Aleppo, Syria, and can be obtained by the designation as listed.

[c] Isolates Fo 8726 and Fo 8733 were obtained from infected chickpeas in the naturally infested field plot at Santaella, Córdoba, Spain, in 1987. Isolates of race 0 (Fo 7802) and race 5 (Fo 8012) were obtained in previous studies from infected chickpeas in different locations in southern Spain. Plants were inoculated by the pot culture method (23,31), with inoculum increased in cornmeal-sand.

or had a low (0–2.5%) disease incidence at flowering, 2 mo after sowing (Table 2). However, disease reactions at 2 and 3 mo after sowing were not necessarily correlated, because many susceptible lines also showed no disease or had a low incidence of dead plants at flowering time. Thus, under our conditions, resistant lines should be identified by a late disease assessment. However, those lines showing a late wilting reaction may be of interest, because yield loss in such lines is expected to be lower than that in early wilting lines (11). Further, late wilting lines may carry useful resistance genes that can confer complete resistance when complemented with other resistance genes.

Research at ICRISAT has indicated that resistance to race 1 of *F. o. ciceris* is controlled by at least three independent loci, so that lines carrying recessive alelles at two loci are completely resistant, whereas lines that are homozygous recessive at one of these loci or dominant at a third locus show a delayed wilt (28,29,32,33). Thus, complete resistance can be obtained from crosses involving late wilting lines (28). Some of the late wilting lines in our study were FLIP 82-180 C, FLIP 84-97 C, FLIP 84-117 C, FLIP 84-121 C, FLIP 84-122 C, FLIP 85-32 C, FLIP 85-35 C, ILC-16, ILC-79, ILC-114, ILC-128, ILC-130, ILC-160, ILC-257, ILC-260, ILC-287, ILC-436, ILC-465, ILC-478, ILC-490, ILC-563, and ILC-568.

All 17 isolates of *F. oxysporum* tested were pathogenic to chickpea in water-culture inoculations, but they differed in their interactions with chickpea cultivars. All isolates were pathogenic to cultivar P 2245, but only 10 were pathogenic to JG 62. In pot-culture inoculations of 17 chickpea lines with two of the above isolates (Fo 8726 and Fo 8733) and with races 0 and 5 of *F. o. ciceris* (Table 4), cultivar P 2245 was susceptible to all races and isolates inoculated and cultivars JG 62 and 12-071/10054 had differential resistant reactions to races 0 and 5, respectively, as expected (16). Reactions of cultivars JG 62 and 12-071/10054 to isolates Fo 8726 and Fo 8733 were similar to those induced by races 5 and 0, respectively.

Large differences occurred in the reactions of the 17 selected chickpea lines to infection by isolates and races of *F. o. ciceris*. All lines were resistant to race 0, but only line FLIP 85-130 C was resistant to race 5. However, reactions of the lines to isolates Fo 8726 and Fo 8733 did not correlate in all cases to those induced by races 5 and 0, respectively (Table 4). Also, disease reaction of lines in the infested field were correlated mainly with reactions induced by race 0 and isolate Fo 8733, although lines FLIP 82-78 C and FLIP 85-130 C, which were moderately resistant to resistant in the field, were susceptible and moderately susceptible to isolate Fo 8733, respectively, in artificial inoculation (Tables 2 and 4). On the contrary, lines that showed high levels of resistance in the field were highly susceptible to race 5 and isolate Fo 8726, except for ILC-219, ILC-256, ILC-487, and ILC-513, which were resistant to isolate Fo 8726 but not to race 5 (Tables 2 and 4).

Resistance to Fusarium wilt has been identified in both desi and kabuli germ plasm (10,13,34), and kabuli cultivars resistant to Fusarium wilt have been developed (2,3,7,8,19–21,34). However, in most cases, wilt-resistant kabuli cultivars are susceptible to Ascochyta blight and cultivars resistant to Ascochyta blight are susceptible to wilt (8,15) (R. M. Jiménez-Díaz and A. Trapero-Casas, *unpublished*).

In this work, we have identified kabuli lines combining tolerance to Ascochyta blight and cold with resistance to Fusarium wilt. These lines should be useful in multiple stress resistance breeding programs of kabuli chickpea suited for countries in the Mediterranean basin in South Europe, North Africa, and West Asia.

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