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Improvement of chickpea resistance to wilt and root rot diseases

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SUMMARY - Breeding chickpeas for resistance to soil-borne fungi inducing wilt and root rot diseases may be hampered by pathogenic variability in the pathogens, the complex nature of genetics of resistance in the host, and the non availability of suitable resistance screening techniques. Races 1-4 of *Fusarium oxysporum* f. sp. *ciceri (FOC)*, the wilting pathogen, were first described in India. Recently, three new races, namely race 0,5 and 6 have been found in southern Spain. Race 0 is the least virulent and might be widespread in the Mediterranean basin. Resistance to *FOC* appears to be confined to desi germplasm. Nevertheless, plant breeders at Culiacan and Hermosillo, Mexico, ICRISAT and ICARDA, have been successful to some extent in developing large-and small-seeded kabuli germplasm with resistance to *FOC*. Results in our laboratory showed that disease reaction of some resistant kabuli types varies depending upon the pathogen race. Resistance to race 1 is conferred by recessive alleles from at least two independent loci. In southern Spain, black collar and root rot is induced either by *Fusarium eumartii* or *F. solani*. *F. eumartii* is more virulent to chickpea and other legumes than *F. solani*. Large-seeded kabuli types are more susceptible to *F. eumartii* and *F. solani* than small-seeded kabuli types. Integrating fungicide seed dressings and advancing sowing dates with moderately susceptible cultivars may contribute to control of wilt and root diseases. However, ascochyta blight-resistant cultivars should be used for fall-winter sowings in order to control blight epidemics.

RESUME - "Amélioration du pois chiche pour la résistance au flétrissement et à la pourriture des racines". La sélection du pois chiche pour la résistance aux champignons du sol est rendue difficile par la variabilité génétique des pathogènes, la complexité de la nature de la résistance génétique chez l'hôte et le manque de technique de criblage adaptée. Les races 1 à 4 de Fusarium oxysporum *f. sp.* ciceri (FOC), *l'agent pathogène du flétrissement, ont été décrites initialement en Inde. Récemment, trois nouvelles races, nommées 0, 5 et 6 ont été identifiées dans le sud de l'Espagne. La race 0 est la moins virulente et pourrait être très répandue dans le bassin méditerranéen. La résistance au* FOC *semble être confinée au type desi. Cependant, les sélectionneurs de Culiacan et d'Hermosillo (Mexique), de l'ICRISAT et de l'ICARDA ont réussi avec un certain succès à développer des génotypes de type kabuli à petites ou grosses graines, résistants au* FOC. *Les résultats de notre laboratoire montrent que la réaction à la maladie de certaines de ces lignées dépend de la race du pathogène. La résistance à la race 1 est liée à des allèles récessifs situés sur au moins deux loci indépendants. Dans le sud de l'Espagne, le "collet noir" et la pourriture des racines sont induits soit par* Fusarium eumartii, *soit par* Fusarium solani. F. eumartii *est plus virulent sur le pois chiche et d'autres légumineuses que* F. solani. *Les types kabuli à grosses graines sont plus sensibles à* F. eumartii *et* F. solani *que les types à petites graines. L'intégration d'un enrobage de fongicide sur la graine et d'une date de semis plus précoce, avec l'utilisation de cultivars moyennement sensibles devrait permettre de contrôler le flétrissement et les maladies des racines. Cependant, pour les semis d'automne et d'hiver, l'utilisation de cultivars résistants à l'anthracnose est indispensable pour le contrôle de cette maladie.*

Introduction

Chickpeas (*Cicer arietinum* L.) can be affected by more than 50 diseases of varied etiology in different parts of the world (Nene, 1980). However, only a few of them are devastating to the crop on a world wide basis, of which some of the most important are induced by soil-borne fungi including fusarium wilt, black collar and root rot, dry root rot, phytopthora root rot and pythium damping-off (Nene and Reddy, 1987).

Most research on chickpea diseases induced by soil-borne fungi has been carried out by scientists from the Indian subcontinent and mainly at ICRISAT. Most

workers agree that control of the chickpea diseases, and in particular of those induced by soil-borne fungi, is achieved best by the use of resistant cultivars (Haware and Nene, 1980; Nene and Haware, 1980; Nene and Reddy, 1987). Because of the importance of the chickpea crop and the use of desi germplasm in the Indian subcontinent, efforts on research and breeding for resistance to wilt and root rot diseases have been placed mainly in desi material. Thus, less information is available on the occurrence and control of those diseases as affecting kabuli chickpeas in the Mediterranean basin (Cabrera de la Colina *et al.*, 1985; Halila *et al.*, 1984; Reddy *et al.*, 1980; Trapero-Casas and Jiménez-Díaz, 1981; 1985), which are the only ones grown in that region.

This paper presents on overview of that part of the research that we have carried out on wilt and root rot diseases of kabuli chickpea in southern Spain for the last 10 years, which might be of relevance for resistance breeding.

The disease complex

Research on the etiology, incidence and distribution of chickpea diseases in southern Spain first indicated that fusarium wilt and root rot diseases occur together with viral infections and iron chlorosis, providing a disease complex with a range of overlapping symptoms (Trapero-Casas and Jiménez-Díaz, 1981; 1985).

Fusarium wilt induced by *Fusarium oxysporum* Schlecht. emend. Snyd. & Hans. f.sp. *ciceri* (Padwick) Snyd. & Hans. was found to be the most prevalent and damaging disease in the complex. Black collar and root rot associated with foliar yellowing and induced either by *F. eumartii* Carpenter or *F. solani* (Mart.) Appl. & Wr. was considered the second in importance. Under conditions of abiotic plant stresses or virus infections, non-vascular isolates of *F. oxysporum* induced a shallow necrosis of roots and collar associated with foliar yellowing. *Macrophomina phaseolina* (Tassi) Goid. caused severe dry root rot in 1981, a year of severe drought and high summer temperatures (Trapero-Casas and Jiménez-Díaz, 1985).

Progress in improving resistance of chickpeas to wilt and root rot diseases may be influenced by the complex nature of the problem, including the genetics of resistance in the host and the pathogenic variability in the pathogen. An additional factor is the screening techniques.

Naturally infested field plots have been used for large scale screening under natural conditions. Use of these sick plots may be hampered by unsuitable inoculum level of the pathogen, lack of uniformity of infestation across sites and the occurrence of multiple infections by several pathogens existing in the soil. Progress and understanding in breeding for resistance to wilt and root rot diseases would be enhanced by use of artificially infested field plots, in which a single pathogen is established at a convenient inoculum concentration. As an alternative, standardized techniques can be developed for greenhouse screening, as showed by scientists at ICRISAT (Nene and Haware, 1980; Nene *et al.*, 1981).

Fusarium wilt (*Fusarium oxysporum* f. sp. *ciceri*)

The existence of races of the pathogen

Isolates of *F. oxysporum* f. sp. *ciceri* from southern Spain induce either a severe leaf chlorosis, flaccidity and plant death by 15-20 days after inoculation (vascular wilt), or a progressive foliar yellowing, which develops 30-40 days after inoculation, and late death of the plant (vascular yellowing) (Trapero-Casas and Jiménez-Díaz, 1981; 1985).

Furthermore, while wilt-inducing isolates are virulent to most chickpea cultivars, yellowing-inducing isolates are not pathogenic to cultivar JG 62 but virulent mainly to local kabuli cultivars (Trapero-Casas and Jiménez-Díaz, 1981; 1985). This suggested the occurrence of races of *F. oxysporum* f. sp. *ciceri* in southern Spain. The existence of races of the pathogen were first shown in India by Haware and Nene (1982), who described races 1, 2, 3 and 4 all of which are pathogenic to JG 62.

Research in our laboratory indicated first that of 61 mass isolates of the pathogen 55 (90%) induce the vascular yellowing syndrome and six (10%) induce the vascular wilt syndrome. When the six wilt-inducing isolates and 11 selected yellowing-inducing isolates were single-spored and together with races 1, 2, 3 and 4 from India were inoculated to race-differential cultivars, three new races of the pathogen namely races 0,5 and 6 were identified (Table 1) (Cabrera de la Colina *et al.*, 1985; Jiménez-Díaz *et al.*, 1988).

Race 0 was proposed to designate all 11 yellowing-inducing isolates nonpathogenic to desi cultivar JG 62 (Cabrera de la Colina *et al.*, 1985) which is susceptible to all other races of the pathogen (Haware and Nene, 1982; Table 1).

Race 0 is mildly to highly virulent to kabuli cultivars C 104, PV 24 and P 2245 and to desi cultivar 12-071/10054.

Race 5 was proposed to designate five wilt-inducing isolates which are pathogenic to desi cultivars CPS 1 and JG 74, and not pathogenic to desi cultivar BG 212 (Cabrera de la Colina *et al.*, 1985, Table 1). Cultivar CPS 1 and JG 74 are highly resistant to race 1, and together with cultivar BG 212 are susceptible to race 2 (Haware

Table 1. Disease reaction of race-differential chickpea cultivars to races of *Fusarium oxysporum* f. sp. *ciceri*[a].

Race origin and designation	Cultivar										
	12-071 10054	JG 62	C 104	JG 74	CPS 1	BG 212	WR 315	ICCV 2	ICCV 4	PV 24	P 2245
Spain											
0	S[b]	R	M	R	R	R	R	R	R	M	S
5	R	S	S	M	M	R	R	S	S	S	S
6	R	S	M	R	R	R	R	M	M	S	S
India											
1	M	S	M	R	R	R	R	R	R	S	S
2	R	S	S	S	S	S	R	M	S	S	S
3	R	S	S	R	M	M	S	M	S	S	S
4	R	S	S	R	M	M	R	M	S	S	S

[a]/ Pooled results of six experiments with several common race-cultivar combinations. Disease reaction for those combinations were consistent over the experiments. Plants were inoculated by the pot-culture method (Nene and Haware, 1980).
[b]/ Assesed 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%, 4=dead plant) 40-50 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).

and Nene, 1982; Table 1). Furthermore, race 5 is highly virulent to all kabuli cultivars inoculated but not pathogenic to desi 12-071/10054 (Table 1). Isolates of race 5 induce vascular wilt to all suceptible cultivars except CPS 1 and JG 74 which showed a late foliar yellowing.

Race 6 was proposed to designate a wilt-inducing isolate that is pathogenic to desi JG 62 and kabulis ICCV 2 and ICCV 4, and not pathogenic to desis 12-071/ 10054, JG 74, CPS 1 and BG 212 (Jiménez-Díaz *et al.*, 1988; Table 1). Race 6 is distinguished from race 1 by cultivars 12-071/10054 (susceptible), ICCV 2 and ICCV 4 (resistant, Kumar *et al.*, 1985), from race 2 by cultivars JG 74, CPS 1 and BG 212 (susceptible), from race 3 by cultivar WR 315 (susceptible) and from race 4 by cultivars CPS 1 and BG 212 (susceptible) (Haware and Nene, 1982; Table 1).

Race 0, which is the least virulent of the Spanish races, is widespread in southern Spain, while races 5 and 6 have been hitherto found within a restricted area. Isolates of *F. oxysporum* f. sp. *ciceri* nonpathogenic to JG 62 have also been found in Tunisia (Nene and Sheila, 1986), which suggests that race 0 might be widespread in the Mediterranean region.

Resistance to *Fusarium oxysporum* f. sp. *ciceri*

Resistance to the pathogen appeared to be restricted mainly to desi germplasm (Haware *et al.*, 1980; Haware and Nene, 1980; Haware *et al.*, 1981) and much of the efforts in breeding for resistance has been placed in desi types. Recently, however, plant breeders have been successful in developing kabuli germplasm with resistance to *F. oxysporum* f. sp. *ciceri*.

The earliest example is that of plant breeders at Culiacan, Mexico. They developed wilt resistant cultivars Surutato 77 and Sonora 80 by transferring genes for resistance from desi L 41 and L 1186 to kabuli Macarena and Breve Blanco (Singh, 1987) and screening for resistance at an infested field plot (no indication of race status given). Efforts to breed wilt resistant kabuli chickpeas have also been made at Hermosillo, Sonora, Mexico, where resistant cultivars Gavilan, Liz and Tubutana have been developed (Dr. Miguel Jiménez León, personal communication).

Also plant breeders at ICRISAT have developed four short duration kabuli lines, namely ICCV2-5, carrying resistance to race 1 of the pathogen (Kumar *et al.*, 1985).

Our work in screening chickpea germplasm for resistance to *F. oxysporum* f. sp. *ciceri* started in 1979. During 1979-1983, some 600 entries including collections of Spanish land races as well as germplasm from Mexico, Morocco, Turkey and the USA, and disease nurseries from ICARDA and ICRISAT, were tested in field plots heavily infested with *F. oxysporum* f. sp. *ciceri* and to a minor extent with *F. solani* and *M. phaseolina*.

Overall, no resistance was found in kabuli germplasm which represented more than 80% of the entries, although some small-seeded kabuli types were shown to be less susceptible than lage-seeded kabuli types. As an example, disease reaction of some ascochyta blight-resistant ILC lines is given in Table 2.

In 1985, germplasm of the ascochyta blight nursery from ICARDA was screened for resistance to the pathogen in our infested plot at Santaella with similar results (Table 3). All kabuli germplasm showed highly susceptible to *F. oxysporum* f. sp. *ciceri*, except for the small-seeded local land race PV 61. Cultivar JG 62 showed a resistant reaction, which indicates that the race 0 of the pathogen (Cabrera de la Colina *et al.*, 1985) was prevalent in the plot at that time.

More recently, a screening trial carried out at the infested field at Santaella showed that valuable wilt resistance is present in some kabuli germplasm

Table 2. Reaction of chickpea germplasm in two fields heavily infested with *Fusarium oxysporum* f. sp. *ciceri* in 1983[a].

Germplasm[b]	Incidence of dead plants (%) in nursery plot at[c]	
	Montilla	Santaella
ILC 72	89.3	100
ILC 182	95.7	100
ILC 191	98.2	100
ILC 194	97.8	100
ILC 195	93.1	97.4
ILC 200	97.1	100
ILC 202	90.2	100
ILC 249	96.2	100
ILC 482	99.0	100
ILC 484	97.2	100
ILC 1407	100	100
ILC 2548	95.3	100
ILC 2555	85.4	77.7
ILC 2912	100	100
ILC 3279	96.9	100
P-2245	99.9	100
PV-25	76.9	76.4

[a]/ Mean of four replicated plots. Stand counts were made 3 weeks after sowing.
[b]/ P-2245 is a kabuli cultivar (20 g per 100 seeds) obtained from ICRISAT. PV-25 is a local kabuli cultivar (56 g per 100 seeds) commonly grown in Andalucía.
[c]/ Total cumulative number of dead plants/total cumulative number of plant at stand count 3 months after sowing.

Table 3. Disease reaction of chickpea germplasm in a field heavily infested with *Fusarium oxysporum* f. sp. *ciceri* at Santaella, Cordoba, Spain, in 1985.

Germplam	Number of entries	Emergence (%) (Range)[a]	Incidence (%) of dead plants (range)[b]	
			2 mo.	3 mo.
ILC 72	1	86	0	76.7
ILC 182	1	88	0	100
ILC 215	1	28	0	92.9
ILC 482	1	80	65.0	100
ILC 3279	1	76	0	100
ILC 3856	1	60	33.3	100
ILC 3868	1	60	13.3	100
ILC 4421	1	90	6.7	100
FLIP-81	2	70-78	2.6-5.7	100
FLIP-82	15	46-98	0-2.2	91.4-100
FLIP-83	12	52-92	0-25.0	65.1-100
JG 62	1	86	0	15.6
WR 315	1	82	0	1.1
P 2245	1	62.9	47.4	100
PV 25	1	59.1	4.9	80.8
PV 61	1	59.3	0	10.0

[a]/ Single row plots 4 m long. Stand counts were made 6 weeks after sowing.
[b]/ Total cumulative number of dead plants/total cumulative number of plants at stand count 2 and 3 months after sowing.

developed by Dr. K.B. Singh at ICARDA with resistance to *Ascochyta rabiei* and tolerance to cold (Jiménez-Díaz *et al.*, unpublished).

Of 735 FLIP entries tested, more than 95% showed highly susceptible to fusarium wilt (Table 4) with a final incidence of dead plants higher than 85%. However, some 2.2% (16) and 2.7% (20) of the entries showed highly and moderately resistant, respectively (Table 4). Twenty of these 36 entries are listed in Table 5.

The efforts by plant breeders to develop chickpea cultivars resistant to *F. oxysporum* f. sp. *ciceri* may be curtailed by the occurrence of pathogen races. As an example, recent work in our laboratory indicates that cultivar Surutato is resistant to races 0,5 and 6 but moderately susceptible to race 1 (Table 6). On the contrary, cultivars Gavilán, Liz and Tubutana (kindly provided by Dr. M. Jiménez León, Hermosillo, Sonora, Mexico) show highly resistant reaction to all four races (Table 6).

An additional difficulty in breeding chickpeas for resistance to *F. oxysporum* f. sp. *ciceri* deals with the genetics of resistance. Inheritance of resistance to the

Table 4. Reaction of chickpea germplasm in a field heavily infested with *Fusarium oxysporum* f. sp. *ciceri* at Santaella, Cordoba, Spain in 1987.

Germplasm[a]	Number of entries	Percentage of entries with emergence[b] 80-100%	Percentage of entries with incidence of dead plants[c]					
			2 mo.			3 mo.		
			0-25%	26-50%	51-100%	0-20%	21-50%	51-100%
FLIP Lines	735	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

[a]/ ILC 1292, PV 60 and JG 62 are cultivars suceptible to fusarium wilt and they were sown every 10 FLIP entries.
[b]/ Stand counts were made 3 weeks after sowing.
[c]/ Total cumulative number of dead plants/total number of plants at stand count 2 and 3 months after sowing.

Table 5. Chickpea germplasm with variable level of resistance to fusarium wilt[a].

Identification	Emergence[b] (%)	Incidence of dead plants (%)[c]		Disease reaction[d]
		2 mo.	3 mo.	
FLIP 85-20	90.0	0.0	0.0	R
FLIP 85-29	72.5	0.0	0.0	R
FLIP 85-85	70.0	0.0	0.0	R
FLIP 84-43	90.0	0.0	2.7	R
FLIP 85-30	67.5	0.0	3.7	R
FLIP 82-78	65.0	0.0	7.7	R
FLIP 85-37	62.5	0.0	8.0	R
FLIP 84-130	57.5	0.0	8.7	R
FLIP 85-130	70.0	7.1	10.7	R
FLIP 84-39	62.5	4.0	12.0	R
FLIP 85-36	62.5	12.0	12.0	R
FLIP 84-65	90.0	0.0	13.8	R
FLIP 83-108	82.5	0.0	15.2	R
FLIP 84-66	97.5	0.0	15.4	R
FLIP 85-32	60.0	0.0	16.6	R
FLIP 85-35	87.5	11.4	17.5	R
FLIP 84-97	95.0	5.2	21.0	MR
FLIP 82-180	82.5	0.0	21.2	MR
FLIP 82-187	80.0	0.0	25.0	MR
FLIP 84-122	57.5	0.0	26.1	MR
FLIP 85-47	72.5	27.6	27.6	MR
FLIP 84-117	52.5	0.0	28.6	MR
FLIP 84-79	90.0	2.8	30.6	MR
FLIP 84-121	65.0	0.0	30.7	MR
FLIP 84-90	62.5	4.0	32.0	MR
FLIP 84-116	87.5	11.4	37.1	MR
FLIP 82-79	85.0	0.0	38.2	MR
FLIP 84-80	77.5	6.5	38.7	MR
FLIP 85-31	75.7	6.4	38.7	MR
ILC 1929[e]	82.3 ± 11.2	39.1 ± 14.7	91.2 ± 8.7	S
PV 60[e]	83.4 ± 14.2	19.1 ± 15.6	95.5 ± 10.2	S
JG 62[e]	81.6 ± 12.7	23.1 ± 24.3	60.9 ± 23.5	S

[a]/ A total of 735 entries were sown in a field heavily infested with *Fusarium oxysporum* f. sp. *ciceri* at Santaella, Córdoba, Spain, on March 18, 1987.
[b]/ Stand counts were made 3 weeks after sowing.
[c]/ Total cumulative number of deade plants/total number of plants at stant count 2 and 3 months after sowing.
[d]/ R = Resistant (0-20% wilt), MR = Moderately resistant (21-50% wilt), S = Susceptible (51-100% wilt), according to Haware and Nene (1982).
[e]/ Susceptible checks. Data are mean and standard deviation of 82 replicated rows for each entry.

Table 6. Disease reaction of chickpea cultivars to races of *Fusarium oxysporum* f. sp. *ciceri*[a].

Cultivar	Race[b]			
	0	1	5	6
P 2245	S[c]	S	S	S
PV 60	M	S	S	S
Gavilan	R	R	R	R
Liz	R	R	R	R
Surutato	R	M	R	R
Tubutana	R	R	R	R
UC 5	R	S	S	S

[a]/ Plants were inoculated by the pot-culture method (Nene and Haware, 1980).
[b]/ Races 0, 5 and 6 are from southern Spain and race 1 is from India.
[c]/ Assessed on a 0-4 scale according to percentaje of foliage with yellowing or necrosis in acropetal progression (0=%, 1=1-33%, 2=34-66%, 3=67-100%, 4=dead plant) 50 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).

pathogen was first reported monogenic and recessive (López, 1974; Kumar and Haware, 1982; Sindhu *et al.*, 1983). Further work showed that resistance to race 1 is conferred by recessive alleles from at least two independent loci (Upadhyaya *et al.*, 1983a; 1983 b; Singh *et al.*, 1987). Cultivars BG 212, CPS 1, JG 74 and WR 315 carrying recessive alleles at both loci are completely resistant to race 1, while cultivars C 104 and K 850, which are homozygous recessive at one of the loci, show a delayed wilt (late-wilting). Cultivars such as JG 62, which carries neither of the recessive alleles, show early wilt symptoms (early-wilting).

Fusarium root rot

Although several root rot diseases have been reported affecting chickpeas, including mainly black collar and root rot, dry root rot, Phytophthora root rot, collar rot and wet root rot (Nene and Reddy, 1987), only the first is considered of importance in southern Spain (Trapero-Casas and Jiménez-Díaz, 1985).

Root rot of chickpeas associated with *Fusarium* spp. has been reported from several countries (ICRISAT, 1980). However, *F. solani* is the major species inducing black collar and root rot (Grewal *et al.*, 1974; Westerlund *et al.*, 1974). In southern Spain, a foliar yellowing associated with collar and root necrosis is induced either by *F. eumartii* or *F. solani* and occasionally by *F. oxysporum*. Distinction between *F. eumartii* and *F. solani* is not widely accepted by *Fusarium* taxonomists. Nevertheless, our isolates of *F. eumartii* differ from those of *F. solani (sensu stricto)* morphological and by their higher virulence to chickpea and other legumes (Trapero-Casas and Jiménez-Diaz, 1985; Trapero-Casas *et al.*, 1986).

Disease reaction of chickpea cultivars to pot and water-culture inoculations (Nene and Haware, 1980) with *F. eumartii, F. solani* and *F. oxysporum* is illustrated with a sample of selected isolates and cultivars (Table 7).

Among kabuli genotypes, large-seeded cultivars such as PV 24 (> 40 g per 100 seeds) were more susceptible than small-seeded ones such as P 2245 (> 20 g per 100 seeds). Desi cultivars were the least susceptible to all three species (Table 7).

Specific field screening for resistance to fusarium root rot has not been carried out except in a few cases (Nain and Agnihotri, 1984). However, screening of wilt-resistant lines developed at ICRISAT for resistance to multiple soil-borne fungi including *F. solani, M. phaseolina, Sclerotium rolfsii* and *Rhizoctonia solani,* indicate that some lines have additional resistance to them and to some foliar fungi (Nene and Reddy, 1987). Also, chickpea lines showing a good level of resistance to fusarium wilt in southern Spain, were screened in field plots infested with *F. eumartii, F. solani, F. oxysporum* (nonvascular), and *M. phaseolina* in addition to *F. oxysporum* f. sp. *ciceri.*

An integrated approach to control wilt and root rot diseases

Control of wilt and root rot diseases of chickpea is best achieved by use of resistant cultivars. Because some germplasm lines with resistance to a single disease have turned out to be highly susceptible to other important diseases, current strategies for resistance breeding are addressed to development of multiple disease resistant cultivars. These strategies include resistance or tolerance to wilt and root rots as well as to major fungal foliar disease, such as ascochyta blight, botrytis gray mould, sclerotinia blight; or viruses (Nene and Reddy, 1987; Reddy and Singh, 1985). Lack of a suitable resistance level in the white large-seeded commercial cultivars prefered in the Mediterranean region, and specially in the Spanish market, force the use of supplementary control measures.

Use of fungicide seed dressings may increase seedling emergence (Shukla *et al.,* 1981; Verma and Vyas, 1977) and delay the epidemics development (Jiménez-Díaz and Trapero-Casas, 1985). However, those treatments do not provide control of the disease complex (Jimenez-Díaz and Trapero-Casas, 1985).

Advancing sowing dates may contribute to control wilt and root rot diseases. Chickpeas in this region are sown in spring time and grown as a short-season crop. Fall to winter sowings provide a more favorable environment that greatly increases plant growth and yield (Saxena and Singh, 1984).

Early results (Trapero-Casas and Jiménez-Díaz, 1986) and current experiments (unpublished), indicate that advancing sowing significantly reduces incidence and severity of fusarium wilt in moderately susceptible cultivars, but not in highly susceptible ones (Table 8). This may relate to avoidance of high temperatures and low soil water content at the end of the growing season, which favor attacks by fusarium wilt and dry root rot diseases.

Winter sowings enhance ascochyta blight epidemics. Thus they require the use of cultivars resistant to ascochyta blight, and in some areas, tolerant to cold (Saxena and Singh, 1984). Also, other agronomic, disease, and weed problems may affect the chickpea crop as related to advanced sowings (Saxena and Singh, 1984). In some experimental fall-winter sown crops at southern Spain, we have observed the occurrence of poor stands, postemergence damping-off and root rot, associated with *Pythium ultimum, P. irregulare, Phytophthora megasperma* and/or *F. solani.* This may be of importance to winter sowings of commercial kabuli types which are highly susceptible to *Pythium* (Kaiser and Hannan, 1983) and *P. megasperma* (Brinsmead *et al.,* 1985).

Table 7. Severity of disease reaction of chickpea cultivars inoculated with isolates of *Fusarium* spp. inducing nonvascular yellowing[a].

Fusarium sp.	Isolates tested (No.)	Syndrome	Cultivar		
			WR 315	P 2245	PV 24
F. oxysporum	3	Foliar yellowing	0.0	1.7	1.6
F. solani	15	Foliar yellowing	0.2	1.2	2.3
		Collar and root necrosis	0.8	0.7	2.1
F. eumartii	5	Foliar yellowing	0.5	2.2	3.8
		Collar and root necrosis	2.9	3.2	3.9

[a]/ Assessed on a 0-4 scale 40 days after inoculation. Scores on foliar yellowing refers to percentage of foliage affected, and scores on collar and root necrosis refers to percentage of tissue necrotic.

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Table 8. Influence of sowing date on development of fusarium wilt in kabuli cultivars PV 25 and P 2245[a].

Sowing date	Incidence of dead plants (%)		Yield of PV 25	
	PV 25	P 2245	g/m^2	g/plant
Early (Feb. 14)	43.3	100	22.6	3.6
Normal (March 28)	77.8	100	7.5	2.9
Late (April 27)	71.1	100	3.6	1.3

[a]/ Cultivar P 2245 is significantly more susceptible to wilt than cultivar PV 25.

References

BRINSMEAD, R.B., RETTKE, M.L., IRWIN, J.A.G., RYLEY, M.J. and LANGDON, P.W. (1985): Resistance in chickpea to *Phytophthora megasperma* f. sp. *medicaginis*. Plant Dis. *69:* 504-506.

CABRERA DE LA COLINA, J., TRAPERO-CASAS, A. and JIMENEZ-DIAZ, R.M. (1985): Races of *Fusarium oxysporum* f. sp. *ciceri* in Andalucia, southern Spain. Int. Chickpea Newsletter *13:* 24-26.

GREWAL, J.S., PAL, M. and KULSHRESTHA, D.D. (1974): A new record of wilt of gram caused by *Fusarium solani*. Current Science *43:* 767.

HALILA, H.M., GRIDLEY, H.E. and HOUDIARD, P. (1984): Sources of resistance to fusarium wilt in kabuli chickpeas. Int. Chickpea Newsletter *10:* 13-14.

HAWARE, M.P., KUMAR, J. and REDDY, M.V. (1980): Disease resistance in kabuli-desi chickpea introgression. Pages 67-69 *in* Proceedings of the International Workshop on Chickpea Improvement. ICRISAT, Hyderabad, India.

HAWARE, M.P. and NENE, Y.L. (1980): Sources of resistance to wilt and root rots of chickpea. Int. Chickpea Newsletter *3:* 11-12.

HAWARE, M.P. and NENE, Y.L. (1982): Races of *Fusarium oxysporum* f. sp. *ciceri*. Plant Dis. *66:* 809-810.

HAWARE, M.P., NENE, Y.L. and RAU, N. (1981): Additional sources of resistance to wilt and root rot of chickpea. Int. Chickpea Newsleetter *4:* 18.

ICRISAT (International Crops Research Institute for the Semi-Arid Tropics) (1980): Proceedings of the International Workshop on Chickpea Improvement, Hyderabad, India.

JIMENEZ DIAZ, R.M. and TRAPERO CASAS, A. (1985): Use of fungicide treatments and host resistance to control the wilt and root rot complex of chickpeas. Plant Dis. *69:* 591-595.

JIMENEZ DIAZ, R.M., TRAPERO CASAS, A. and CABRERA DE LA COLINA, J. (1988): Races of *Fusarium oxysporum* f. sp. *ciceri* infecting chickpeas in southern Spain. Proceedings of the NATO Advanced Research Workshop on the Interaction of Genetic and Environmental factors in the Development of Vascular Wilt Diseases of Plants. Cape Sounion, Athens, Greece (In press).

KAISER, W.J. and HANNAN, R.M. (1983): Etiology and control of seed decay and pre-emergence damping-off of chickpea by *Pythium ultimum*. Plant Dis. *67:* 77-81.

KUMAR, J. and HAWARE, M.P. (1982): Inheritance of resistance to fusarium wilt in chickpea. Phytopathology *72: 1035-1036.*

KUMAR, J., HAWARE, M.P. and SMITHSON, J.B. (1985): Registration of four short duration fusarium-wilt resistant kabuli (*garbanzo*) chickpea germplasms. Crop Sci. *25:* 576-577.

LOPEZ, G.H. (1974): Herencia del carácter "resistencia a rabia" (*Fusarium* sp.) en garbanzo (*Cicer arietinum*) bajo condiciones de campo. Agricultura Técnica en México *3:* 286-289.

NAIN, V.S. and AGNIHOTRI, J.P. (1984): Screening chickpeas for resistance to black root rot disease. Int. Chickpea Newsletter *11:* 34-35.

NENE, Y.L. (1980): Diseases of chickpea. Pages 277-280 *in* Proceedings of the International Workshop on Chickpea Improvement. ICRISAT, Hyderabad, India.

NENE, Y.L. and HAWARE, M.P. (1980): Screenieng chickpea for resistance to wilt. Plant Dis. *64:* 379-380.

NENE, Y.L., HAWARE, M.P. and REDDY, M.V. (1981): Chickpea diseases: resistance screening techniques. Information Bulletin No. 10. ICRISAT, Patancheru, India.

NENE, Y.L. and REDDY, M.V. (1987): Chickpea diseases and their control. Pages 233-270 *in* The Chickpea (Saxena, M.C. and Singh, K.B., eds.). C.A.B. International, Wallingford, U.K.

NENE, Y.L. and SHEILA, V.K. (1986): Fuusarium wilt of chickpea. Fusarium Notes, Vol. 5 (November).

REDDY, M.V., GRIDLEY, H.E. and KAACK, H. (1980): Major disease problems of chickpea in North Africa. Int. Chickpea Newsletter *3:* 13-14.

REDDY, M.V. and SINGH, K.B. (1985): Exploitation of host-plant resistance in the management of ascochyta blight and other diseases of chickpeas. Pages 139-151 *in* Faba beans, kabuli chickpeas, and lentils in the 1980s (Saxena, M.C. and Varma. S., eds.). ICARDA, Aleppo, Syria.

SINDHU, J.S., SINGH, K.P. and SLINKARD, A.E. (1983):Inheritance of resistance to fusarium wilt in chickpea. Journal of Heredity *74:* 68.

SAXENA, M.C. and SINGH, K.B. (eds.) (1984): Ascochyta blight and winter sowing of chickpeas. Martinus Nijhoff/Dr. W. Junk Publishers, The Hague, The Netherlands.

SHUKLA, P., SING, R.R. and MISHRA, A.N. (1981): Search for best seed dressing fungicides to control chickpea wilt (*Fusarium oxysporum*). Pesticides *15:* 15-16.

SINGH, K.B. (1987): Chickpea Breeding. Pages 127-162 *in* The chickpea (Saxena, M.C. and Singh, K.B., eds.). C.A.B. International, Wallingford, U.K.

SINGH, H., KUMAR, J., SMITHSON, J.B. and HAWARE, M.P. (1987): Complementation between genes for resistance to race 1 of *Fusarium oxysporum* f. sp. *ciceri* in chickpea. Plant Pathology *36:* 539-534.

TRAPERO CASAS, A., CABRERA DE LA COLINA, J. and JIMENEZ DIAZ, R.M. (1986): Virulence of *Fusarium oxysporum* f. sp. *ciceri* and *F. solani* infecting chickpeas in southern Spain. Page 12 *in* Poster Abstracts, International Food Legume Research Conference, (O'Keeffe, L.E. and Muehlbauer, F.J., eds.). Spokane, Washington, USA.

TRAPERO CASAS, A. and JIMENEZ DIAZ, R.M. (1981): Occurrence and etiology of chickpea "wilt complex" in southern Spain. Int. Chickpea Newsletter *5:* 10-11.

TRAPERO CASAS, A. and JIMENEZ DIAZ, R.M. (1985): Fungal wilt and root rot diseases of chickpea in southern Spain. Phytopathology *75:* 1146-1151.

TRAPERO CASAS, A. and JIMENEZ DIAZ, R.M. (1986): Influence of sowing date on fusarium wilt and ascochyta blight of chickpea in southern Spain. Page 11 *in* Poster Abstracts, International Food Legume Research Conference (O'Keeffe, L.E. and Muehlbauer, F.J., eds.). Spokane, Washington, USA.

UPADHYAYA, H.D., HAWARE, M.P., KUMAR, J. and SMITHSON, J.B. (1983a): Resistance to wilt in chickpea. I. Inheritance of late-wilting in response to race 1. Euphytica *32:* 447-452.

UPADHYAYA, H.D., SMITHSON, J.B., HAWARE, M.P. and KUMAR, J. (1983b): Resistance to wilt in chickpea. II. Further evidence for two genes for resistance to race 1. Euphytica *32:* 749-755.

VERMA, R.K. and VYAS, S.C. (1977): Effect of seed treatment with systemic fungicides in gram wilt control. Pesticides *11:* 20-21.

WESTERLUND, F.V., CAMPBELL, R.N. and KIMBLE, K.A. (1974): Fungal root rots and wilt of chickpea in California. Phytopathology *64:* 432-436.