

Inheritance of Resistance to Fusarium Wilt in Chickpea

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ABSTRACT

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The inheritance of resistance to chickpea (*Cicer arietinum* L.) wilt caused by *Fusarium oxysporum* f. sp. *ciceri* race 1 (Hyderabad) was studied in a set of diallel crosses among five resistant and two susceptible cultivars at Hyderabad. Studies of F₁, F₂, and F₃ generations of the three crosses involving the resistant parents CPS-1 and WR-315, and the susceptible

parent C-104, indicated that resistance was conferred by a single recessive allele at the same locus in the two resistant parents. The resistance was successfully transferred from WR-315, CPS-1, and other resistant cultivars to a wide range of genetic backgrounds by hybridization and pedigree and bulk selection.

Additional key words: multiple loci, race.

Wilt of chickpea (*Cicer arietinum* L.), caused by *Fusarium oxysporum* Schlecht emend. Snyd. and Hans f. sp. *ciceri* (Padwick) Snyd. and Hans, was first described by Padwick (11) and has since been reported from several countries (10). In India, it is estimated to cause a 10% annual yield loss (13). Although limited screening and identification of resistance sources had been done in India, large-scale systematic screening of the world germ plasm collection has only recently been conducted at the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) (9). The possible existence of at least three races of *F. oxysporum* f. sp. *ciceri* is indicated (4) and over 40 sources of resistance to the race 1 (Hyderabad) have been identified; some of them are also resistant to one of the other races (4).

Only a few reports on the inheritance of resistance, all under field conditions, are available. Ayyar and Iyer (1) indicated that a single gene with incomplete dominance conferred resistance to chickpea wilt. Lopez (8) found that the resistance was governed by one or two recessive genes in crosses involving different strains of chickpea. Pathak et al (12) and Haware et al (3) reported that one recessive gene was responsible for resistance. However, the latter workers pointed out that in some crosses inheritance could not be explained on the basis of one or two genes. They observed that, in field screening, mortality due to other root pathogens and escapes may affect the results of inheritance studies.

The present study was undertaken to investigate the inheritance of resistance under controlled conditions, and its implications with regard to breeding are discussed.

MATERIALS AND METHODS

A set of diallel crosses involving five wilt-resistant parents (WR-315, CPS-1, T-3 [GW], BG-212, and P-436-2) and two susceptibles (C-104 and JG-62) was made in 1978. The F₃ progenies were obtained from random F₂ plants grown under wilt-free conditions. In this paper we report the data from a diallel combination of three parents: WR-315, CPS-1, and C-104. The other crosses either have not been tested in F₃ or gave segregation ratios that were not easily interpreted; these will be reported in detail in a later paper.

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The tests of wilt resistance were conducted in a screenhouse in pots according to the method described by Nene and Haware (9) at temperatures between 25 and 30 C. The fungus culture was derived from a single spore multiplied in 100 g of sand-maize meal in a 250-ml flask incubated for 14 days at 25 C. One hundred grams of the inoculum were mixed thoroughly with 2 kg of a mixture of autoclaved soil (black Vertisol) and riverbed sand (1:1, v/v) in 30-cm earthenware pots. Two successive batches of the susceptible cultivar JG-62 were grown in the pots (10 seeds per pot) and allowed to wilt. The wilt usually appeared about 20 days after planting, and the plant debris were incorporated into the soil.

The wilt resistance of the parents and their F₁, F₂, and F₃ progenies was evaluated in pots in 1979 and 1980. Due to space limitations the tests were conducted in successive batches. Ten to 15 seeds were sown per pot, and the numbers of wilted and healthy plants were recorded at 10-day intervals up to 60 days after emergence. Where two or three sowings were made of the same population, chi-squared tests showed that the results were not significantly different, so the data have been combined for presentation.

Twenty-five seeds each of 18 to 20 F₃ progenies of the three crosses were sown in a row and a susceptible check JG-62 was planted on every fifth row in a plot uniformly infested with *F. oxysporum* f. sp. *ciceri* and located on a deep black Vertisol by repeated incorporation of debris of wilted plants. The numbers of wilted and healthy plants were recorded at 20-day intervals until maturity, and wilted plants were examined in the laboratory for the presence of the pathogen.

RESULTS AND DISCUSSION

All plants of WR-315 and CPS-1 were resistant, and all those of C-104 were susceptible. In crosses of WR-315 and CPS-1 with C-104, the data were consistent with segregation of a single gene with the recessive allele conferring resistance to race 1 of the wilt fungus. The F₁s were susceptible; F₂s segregated in a ratio of 3 susceptible:1 resistant; and F₃ progenies, 1 susceptible:2 segregating:1 resistant (Table 1). However, there was an excess of susceptible progenies in the F₃ generation from WR-315 × C-104 that could have resulted from a failure of occurrence of resistant plants in some progenies because of their relatively small size. These results conform to those of other workers (1,3,8,12) who also showed that resistance to wilt is simply inherited although which race(s) they were working with are not known. The F₁, F₂, and F₃

TABLE 1. Numbers of susceptible and resistant plants or progenies in the F₁, F₂, and F₃ generations of the three crosses, grown in pots and field plots infested with *Fusarium oxysporum* f. sp. *ciceri* in 1979 and 1980

Cross	F ₁ plants		F ₂ plants				F ₃ progenies					
	S	R	S	R	χ^2 (3:1)	Probability	S	Seg	R	χ^2 (1:2:1)	Probability	
WR-315 × C-104	SH ^a	25	0	71	32	2.03	0.10-0.50	11	19	7	1.11	0.50-0.70
	F	NT ^b	NT	NT	NT			8	8	4	2.40	0.30-0.50
CPS-1 × C-104	SH	23	0	24	8	0	1.0	NT	NT	NT		
	F	NT	NT	NT	NT			4	11	3	1.00	0.50-0.70
WR-315 × CPS-1	SH	0	30	0	135	0	1.0	0	0	113	0	1.0
	F	NT	NT	NT	NT			0	0	20	0	1.0

^aSH = plants in pots in a greenhouse; F = F₃ progenies in field plots; S = susceptible; R = resistant; and Seg = segregating. Results from greenhouse plants were based on one to three independent tests.

^bNT = Not tested.

generation plants of the cross WR-315 and CPS-1 were all resistant, indicating that the same locus was involved.

Some crosses did not give good fits to the expected ratios for simple inheritance. Where C-104 was involved as the susceptible parent, the F₂s always segregated in the ratio of 3 susceptible:1 resistant, but with JG-62 there was usually an excess of susceptible plants. Furthermore, susceptible cultivars differ in reaction to the pathogen (5). For example, JG-62 wilts completely within 20 days of emergence, while other cultivars do not wilt until later stages of growth. The observations indicate the existence of other major genes or polygenic complexes acting as modifiers, as suggested for *Fusarium* wilt in flax (6) and *Fusarium* root rot of beans (2).

The situation is further complicated by the occurrence of different races of the pathogen (4). While WR-315 is resistant to race 1 (prevalent in Hyderabad) and race 2 (Kanpur), CPS-1 is resistant only to race 1 but both are susceptible to race 3 (Gurdaspur). Similarly, while C-104 is susceptible to race 1 and race 2, it is resistant to race 3. Therefore, it appears that gene(s) for resistance to different races of *F. oxysporum* may occur in either multiple loci or allelic series.

The possible existence of polygenic complexes and the inheritance of resistance to other races of the pathogen are being studied. Meanwhile, the resistance of WR-315, CPS-1, and other genotypes resistant to race 1 has been successfully transferred to many genetic backgrounds by hybridization and selection based on pedigree and bulk screening in plots infested with *F. oxysporum* f. sp. *ciceri* (7). Their yield potentials are currently being assessed.

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