Short Communication

International Crops Research Institute for the Semi-Arid-Tropics, Patancheru, Andhara Pradesh (India)

Inheritance of Dry Root Rot (Rhizoctonia bataticola) Resistance in Chickpea (Cicer arietinum)

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With 2 tables

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Abstract

The inheritance of resistance to dry root rot of chickpea caused by Rhizoctonia hataticola was studied. Parental F₁ and F₂ populations of two resistant and two susceptible parents, along with 49 F₃ progenies of one of the resistant × susceptible crosses were tested for their reaction to dry root rot using the blotting-paper technique. All F₁ plants of the resistant × susceptible crosses were resistant; the F₂ generation fitted a 3 resistant: 1 susceptible ratio indicating monogenic inheritance, with resistance dominant over susceptiblity. F₃ family segregation data confirmed the results. No segregation occurred among the progeny of resistant × resistant and susceptible × susceptible crosses.

Key words: Cicer arietinum — Rhizoctonia bataticola — soilborne disease — resistance — inheritance

Among the several soilborne fungal diseases of the chickpea, Cicer arietinum L., dry root rot caused by Rhizoctonia bataticola Taub (Butler) is the most severe in the semi-arid tropics (SAT), with ambient day temperatures around 30 °C. Plants infected by this fungus dry suddenly at flowering and pod setting. The roots are brittle, and minute sclerotial bodies can be seen in the pith cavity and on the outer surface of the tap root through a hand lens. Both field and laboratory techniques to identify resistant genotypes were developed at the International

Crops Research Institute for the Semi-arid Tropics (ICRISAT), India (NENE et al. 1981). Efforts are also being made by ICRISAT chickpea breeders to combine resistance to fungal diseases such as wilt (Fusarium oxysporum f. sp. ciceri) and dry root rot with high yield. Hence, information on the inheritance of resistance to this disease will be useful in transferring the resistance to high-yielding but disease-susceptible chickpea cultivars. The objective of this study was to determine the genetic basis of resistance to dry root rot in chickpea cultivars 'C 104' and 'H 208'.

The selection of the chickpea parents was based on laboratory screening tests. The F_1 and F_2 seeds of resistant \times resistant ('H 208' \times 'C 104'), resistant \times susceptible ('H 208' \times 'P 165' and 'C 104' \times 'P 165'), and susceptible \times susceptible ('K 850' \times 'P 165') crosses, and the F_3 progenies of 'C 104' \times 'P 165' were screened to study the inheritance of resistance to dry root rot.

Surface-sterilized (2.5 % sodium hypochlorite for 5 min) chickpea seed were sown in the greenhouse in polythene bags containing sterilized sand. Temperature was maintained at 20—25 °C. Five-day-old seedlings were carefully removed and washed free of sand for inoculation. The inoculum was prepared by macerating a five-day-old culture of *Rhizoctonia bataticola* that was multiplied in 100 ml potato dextrose broth at 25 °C, with 50 ml sterilized distilled water. Roots of 20 seedlings were dipped at a time in the freshly prepared inoculum for 30 sec. The seed-

lings were then placed between folds of blotting paper so that the cotyledons and roots were covered, while the green tops remained exposed. The blotter ends were stapled to form envelopes, moistened with sterilized distilled water, and placed in polythene bags with the green tops exposed. Each polythene bag contained 7 blotters with seedlings, and an inoculated susceptible check 'P 165'. The seedlings were incubated in a Percival incubator at 35 °C with 12 h artificial light/day for 8 days. The blotters were removed from the polythene bags each day, moistened, and reshuffled to minimize the location effect within the bag. Individual seedlings were scored for the extent of root infection eight days after incubation on a 1-9 scale, where 1 = free from infection and 9 = highly susceptible. The numbers of resistant (rating ≤ 5) and susceptible (rating ≥ 6) plants of parents, F1, F2 and F3 progenies were counted. The goodness of fit of the observed ratios was tested by computing X2 values.

All the plants of 'H 208' and 'C 104' showed resistant reaction (rating < 3) eight days after incubation, while 'P 165' and 'K 850' showed susceptible reaction (rating 7—9). The exposed upper portion of seedlings remained green in each case. The disease reaction of the F_1s of resistant \times susceptible crosses was similar to that of the resistant parent, and the F_2 population segregated in a 3 resistant : 1 susceptible

ratio (Table 1). The F3 families of the cross 'C 104' × 'P 165' segregated in a 1 resistant : 2 segregating: 1 susceptible ratio. Among the segregating progenies, resistant and susceptible plants were observed in a 3:1 ratio $(X^2P =$ 0.75—0.95) except in two families (Table 2). The deviations can be ascribed to change factors (STANSFIELD 1983). The F₂ generations of resistant × resistant and susceptible × susceptible crosses did not segregate for disease reaction, although some variation within the population was observed. This discrepancy may be due to the visual scoring, which is done relative to the disease development in the susceptible check 'P 165' in each of the polythene bags. However, the resistant and the susceptible plants could be distinguished easily. The results indicate that resistance to dry root rot is governed by a single dominant gene in the resistant genotypes studied. A similar simple mode of inheritance of resistance to Ascochyta blight in chickpea was reported by Singh and REDDY (1983). Several sources of resistance to dry root rot are available, but their genetic basis has not yet been determined.

Among the parents used in this study, 'C 104' and 'K 850' carry recessive alleles at

Table 1. Disease reactions of chickpeas to inoculation with Rhizoctonia bataticola

Generation	Number of Plants		X ²	
	Resistant	Susceptible	(3:1)	Р
Parents				
H 208	60	. 0		
C 104	60	0		
K 850	0	60		
P 165	0	60		
F ₁ Generation				
C 104 × P 165	20	0		
H 208 × P 165	20	0		
H 208 × C 104	20 ·	0		
K 850 × P 165	0	35		
F ₂ Generation				
K 850 × P 165	0	269		
H 208 × C 104	245	0		
C 104 × P 165	176	69	1.38	0.250.50
H 208 × P 165	172	64	0.56	0.250.50
Pooled	348	133		
Heterogeneity			1.07	0.250.50

Table 2. Reactions of F₃ progenies of cross 'C 104' × 'P 165' segregating for dry root rot infection

	Number of Plants		X^2	
Progeny	Resistant	Susceptible	(3:1)	P
11	16	4	0.27	0.61
25	16	4	0.27	0.61
27	17	3	1.07	0.30
34	16	4	0.27	0.61
39	14	6	0.27	0.61
43	17	3	1.07	0.31
46	13	7	1.07	0.31
52	16	4	0.27	0.61
56	14	6	0.27	0.61
60	16	4	0.27	0.61
63	15	5	0.00	0.95
65	. 12	8	2.00	0.13
66	15	5	0.00	0.95
69	15	5	0.00	0.95
74	11	9	4.27	0.04
77	11	9	4.27	0.04
78	15	5	0.00	0.95
<i>7</i> 9	15	5	0.00	0.95
85	14	6	0.27	0.61
87	15	5	0.00	0.95
89	15	5	0.00	0.95
90	13	7	1.07	0.30
Pooled	321	119	16.98	
Heterogeneity			16.01	0.750.95

two different loci, and 'H 208' carries a dominant gene for delayed wilting caused by F. oxysporum f. sp. ciceri race 1 (UPADHYAYA et al. 1983, SMITHSON et al. 1983). 'P 165' is resistant to Fusarium wilt. Therefore, it may be possible to isolate genotypes resistant to both wilt and dry root rot from the crosses of 'P 165' with 'H 208' and 'C 104', by screening the segregating generations in a disease nursery infested with the two fungal pathogens.

Zusammenfassung

Vererbung der Resistenz gegen Wurzelfäule (Rhizoctonia bataticola) bei der Kichererbse (Cicer arietinum L.)

Junge mit Rhizoctonia bataticola Taub (Butler) inokulierte Keimpflanzen zweier anfälliger und zweier resistenter Kreuzungseltern von Kichererbsen sowie deren F₁- und F₂-Nach-

kommenschaften wurden in gefaltetem Filterpapier kultiviert und hinsichtlich ihrer Reaktion auf die Wurzelfäule-Infektion geprüft. Ebenso wurden 49 F₃-Nachkommenschaften einer dieser Kreuzungen (resistent × anfällig) einer solchen Prüfung unterworfen. Alle F₁-Pflanzen der Kreuzungen resistent × anfällig erwiesen sich als resistent. In der F₂-Nachkommenschaft konnte eine eindeutige 3:1-Spaltung nachgewiesen werden, was auf monogenische Vererbung bei Dominanz der Resistenz hinweist. Die Auswertung der F₃-Familien unterstützte diese Ergebnisse. Nachkommenschaften der Kreuzungen resistent × resistent und anfällig × anfällig spalteten nicht.

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