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Studies on inheritance of resistance and allelic relationships for strain 2 of pigeonpea sterility mosaic pathogen

By T SRINIVAS[1,2]*, M V REDDY[1], K C JAIN[1] and M S S REDDY[2]

[1]*ICRISAT Asia Center, Patancheru 502 324, Andhra Pradesh, India;* [2]*Andhra Pradesh Agricultural University, Hyderabad 500 030, Andhra Pradesh, India*

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Summary

Inheritance of resistance and allelic relationships were studied in three resistant pigeonpea sources for strain 2 of sterility mosaic pathogen. The resistant genotypes (ICP 7035, ICP 7349 and ICP 8850) were crossed with susceptible genotypes (BDN1 and LRG30) to determine the inheritance of resistance. The resistant and susceptible genotypes were also crossed among themselves to obtain information on their allelic relationships. Parents, F_1 and F_2 generations were sown in pots and screened using infector-hedge technique. Observations in parents, F_1 and F_2 generations, indicated dominance of resistance in certain crosses and the dominance of susceptibility in others. Disease reaction appeared to be governed by two independent non-allelic genes, with at least three multiple alleles, at one of the loci.

Key words: *Cajanus cajan*, pigeonpea, sterility mosaic, inheritance, strain, resistance, susceptibility

Introduction

Sterility mosaic, an important disease of pigeonpea, is known to occur in almost all the major pigeonpea growing areas of India (Kannaiyan *et al.*, 1984) and at times can cause yield losses up to 95% (Reddy & Nene, 1981). An annual loss of 205 000 tonnes of grains valued at US$76.9 millions is estimated from the sterility mosaic disease (Kannaiyan *et al.*, 1984). It was first reported from Pusa in Bihar, India (Mitra, 1931) and is presently a serious problem in north eastern and southern states of India. The disease is characterised by proliferation, mosaic symptoms, cessation of reproductive growth and a reduction in the size of the leaflets (Kandaswamy & Ramakrishnan, 1960). It is transmitted by an eriophyid mite, *Aceria cajani* Channabasavanna (Seth, 1962). Chemical methods of control, while effective, are not considered economical (Nene *et al.*, 1989). Therefore, breeding of resistant varieties, recognised as the most effective and economic method of reducing crop losses, has received high priority for the disease.

Development of pigeonpea cultivars resistant to the disease was first reported by Alam (1931). Systematic resistance breeding was later initiated at the International Crops Research

*Corresponding author

Institute for the Semi-Arid Tropics (ICRISAT) in 1975, and several resistant and tolerant source(s) for the disease were identified (Amin *et al.*, 1993; Nene, Kannaiyan, Reddy & Remanandan, 1981). The genetics of resistance for the disease was also worked out (Sharma, Gupta, Rai & Reddy, 1984; Singh *et al.*, 1983). However, the task of developing resistant varieties has been complicated in view of the reported variability in the pathogen. The presence of strains of SM pathogen of varying virulence was reported by Nene *et al.* (1989) based on the results of multi-location pigeonpea trials. Lines resistant at ICRISAT, Patancheru, Andhra Pradesh became susceptible when grown at other locations within India. A comprehensive study of variability in the sterility mosaic pathogen (Reddy *et al.*, 1993), using a set of seven differentials, at nine different locations in India, revealed the occurrence of five different variants of the sterility mosaic pathogen of pigeonpea in India.

The dynamic nature of the sterility mosaic pathogen has warranted the identification and use of strain-specific sources of resistance in the crop improvement programme. Further, it has also necessitated studies on genetics of strain-specific resistance to aid resistance breeding programmes. The earlier studies on genetics of resistance for the disease (Singh *et al.*, 1983; Sharma *et al.*, 1984) have very little significance in the wake of reports on the existence of several strains of the pathogen. Hence, the present investigation was undertaken to elucidate the inheritance of resistance and allelic relationship of a few resistant sources for strain 2 of the sterility mosaic pathogen.

Materials and Methods

A set of 153 genotypes, earlier reported as resistant or tolerant (Nene *et al.*, 1981) were screened at the ICRISAT Asia Center, Patancheru during May 1994, against strain 2 of the sterility mosaic pathogen, identified by Reddy, Raju & Nene (1991). The screening was carried out using the infector-hedge technique (Nene & Reddy, 1976). Ten seeds of each genotype were sown in plastic pots of 15 cm diameter filled with alfisols (60% sand, 33% clay and 7% silt) and placed beside the infector-hedge. Genotypes with less than 20% disease incidence were re-evaluated, in three replications, to identify promising resistant sources for the strain. Genotypes were classified as resistant when disease incidence was less than 10%.

Three genotypes (ICP 7035, ICP 7349 and ICP 8850) of medium to late maturity duration, resistant (with no apparent symptoms) to the strain, were selected as resistant parents. These were crossed with two susceptible (severe mosaic symptoms) parents, BDN 1 and LRG 30. The resistant and susceptible parents were also crossed among themselves, to obtain information on allelic relationships.

The resistant parents were sown in four sets at intervals of 15 days in 30 cm pots and placed beside the infector-hedge, while the susceptible parents were raised under disease-free conditions. Confirmed resistant plants were used for crossing with the susceptible parents and sufficient F_1 seed was obtained in each cross combination. Part of the F_1 seed was advanced to the F_2 generation during the rainy season of 1994. The F_1 plants were selfed by covering them with bee-proof nylon cages. Flower initiation, flower colour, pod colour, seed colour, seed size and other contrasting characters among the parents were used as markers to check the identity of F_1 plants. Only true F_1's were advanced to F_2.

Five parents, 10 F_1, and 10 F_2 of the resistant × susceptible (six), resistant × resistant (three) and susceptible × susceptible (one) cross combinations were screened against the strain, during May-December 1995, for their disease reaction using the infector-hedge technique (Nene & Reedy, 1976). Seedlings were raised in 15 cm pots with 10 seedlings per pot. The susceptible control, ICP 8863, was included at frequent intervals, to monitor disease spread. Observations on disease reaction were recorded at 75 days after sowing. The plants

Table 1. *Reaction of parents, F_1 and F_2 generations of the resistant × susceptible crosses of pigeonpea for strain 2 of sterility mosaic pathogen at ICRISAT Asia Center, Patancheru, Andhra Pradesh, India*

		Observed frequencies		Expected frequencies			
Generation	Total plants	Resistant plants (R)	Susceptible plants (S)	Resistant plants (R)	Susceptible plants (S)	Ratio R : S	Probability
ICP 7035 × BDN 1							
F_1	10	10	—	10	—	—	—
F_2	248	199	49	186	62	3 : 1	0.30–0.50
ICP 7035 × LRG 30							
F_1	12	12	—	12	—	—	—
F_2	360	209	151	202.5	157.5	9 : 7	0.30–0.50
ICP 7349 × BDN 1							
F_1	8	8	—	8	—	—	—
F_2	450	329	121	337.5	112.5	3 : 1	0.30–0.50
ICP 7349 × LRG 30							
F_1	9	9	—	9	—	—	—
F_2	327	195	132	183.94	143.06	9 : 7	0.20–0.30
ICP 8850 × BDN 1							
F_1	12	—	12	—	12	—	—
F_2	398	87	311	99.5	298.5	1 : 3	0.10–0.20
ICP 8850 × LRG 30							
F_1	14	—	14	—	14	—	—
F_2	220	49	171	41.25	178.75	3 : 13	0.10–0.20

were classified as resistant (no apparent symptoms) and susceptible (severe mosaic symptoms). The chi-square method (Snedecor & Cochran, 1967) was used to test the goodness of fit of the segregating populations with the expected phenotypic ratios.

Results and Discussion

The susceptible control (ICP 8863), planted along with the test materials (five parents, 10 F_1 and 10 F_2) exhibited 100% infection indicating good spread of the disease. ICP 7035, ICP 7349 and ICP 8850 were recorded as 100% resistant with no apparent symptoms, while BDN1 and LRG 30 exhibited severe mosaic symptoms.

The reactions of F_1 and F_2 generations of the resistant × susceptible crosses are presented in Table 1. Dominance of resistance over susceptibility was observed in the F_1 generation of resistant × susceptible crosses involving the resistant parents ICP 7035 and ICP 7349, while susceptibility was dominant in the F_1 generation of resistant × susceptible crosses involving the resistant parent ICP 8850. The dominance of susceptibility over resistance has also been reported by Singh *et al.* (1983) and Sharma *et al.* (1984). A variation in the dominance relationships of the disease reaction with the cross involved was also noticed by Sharma *et al.* (1984) similar to the findings of the present study.

In the F_2 generation, the crosses of the resistant parents, ICP 7035 and ICP 7349 with the susceptible parent BDN1 segregated in the ratio 3 resistant : 1 susceptible, while crosses with the susceptible parent LRG30 segregated in a ratio of 9 resistant : 7 susceptible. However, in the F_2 generation of the crosses involving the resistant parent ICP 8850 and the susceptible BDN1, a ratio of 1 resistant : 3 susceptible was obtained while, in combination with the susceptible parent LRG30, a ratio of 3 resistant : 13 susceptible was obtained. This suggested that ICP 7035, ICP 7349 and ICP 8850 differed with the susceptible BDN1 in respect of a

Table 2. *Reaction of F_1 and F_2 generations of the resistant × resistant and susceptible × susceptible crosses of pigeonpea for strain 2 of sterility mosaic pathogen at ICRISAT Asia Center, Patancheru, Andhra Pradesh, India*

		Observed frequencies		Expected frequencies			
Generation	Total plants	Resistant plants (R)	Susceptible plants (S)	Resistant plants (R)	Susceptible plants (S)	Ratio R : S	Probability
Resistant × Resistant							
ICP 7035 × ICP 7349							
F_1	22	22	—	22	—	—	—
F_2	297	296	1	297	—	—	—
ICP 7035 × ICP 8850							
F_1	14	14	—	14	—	—	—
F_2	457	450	7	457	—	—	—
ICP 7349 × ICP 8850							
F_1	18	18	—	18	—	—	—
F_2	339	339	11	350	—	—	—
Susceptible × Susceptible							
BDN1 × LRG 30							
F_1	20	—	20	—	20	—	—
F_2	472	9	463	—	472	—	—

single gene pair, while with LRG30, they differed in respect of at least two gene pairs. Singh *et al.* (1983) and Sharma *et al.* (1984) also reported a similar variation between different crosses in the number of genes governing resistance. Singh *et al.* (1983) reported the involvement of two genes in crosses involving the resistant parents Pant A3 and ICP 6999, and three genes in crosses involving the resistant parents ICP 3783, ICP 7035 and ICP 7119 with the susceptibles Pant A2, UPAS120 and T21. However, Sharma *et al.* (1984) reported the involvement of two genes governing resistance in ICP 7035 and tolerance in ICP 2376 in cross combinations with the susceptible parent BDN1.

The F_2 segregation ratios of 9 resistant : 7 susceptible in resistant × susceptible crosses involving the resistant parents ICP 7035 and ICP 7349 with LRG30 indicated the presence of two independent non-allelic gene pairs exhibiting complementary gene action. The F_2 segregation ratio of 3 resistant : 13 susceptible in ICP 8850 × LRG30 also indicated the presence of two independent non-allelic gene pairs. The ratios can be explained on the basis of the presence of multiple alleles governing the resistance trait for the strain. The hypothesis of duplicate genes and multiple alleles is combined to explain the disease reactions observed in F_1 combinations and segregation in F_2. A resistance reaction occurs when resistant alleles are present at the two loci while, susceptibility is observed when the susceptible alleles are present even at one locus. At least three allelic forms are present at one of the loci with the dominance relationship of $a_1 > a_2 > a_3$. The alleles a_1 and a_3 are assumed to be responsible for the resistance reaction, while the allele a_2 results in a susceptible reaction. Thus, ICP 7035 and ICP 7349 appear to possess the a_1 allele for resistance (a_1a_1BB), while ICP 8850 possesses the a_3 allele for resistance (a_3a_3BB). The susceptible parent BDN 1 appears to possess the a_2 allele for susceptibility with the genetic constitution a_2a_2BB, while LRG30 appeared to have $a_2a_2$bb genotypic constitution. This would explain the differential reactions of the F_1's and F_2's observed in the resistant × susceptible crosses.

The F_1's of all resistant × resistant crosses (Table 2) were resistant, while the F_1 of susceptible × susceptible cross (Table 2) was susceptible. Further, no segregation was observed in the F_2 generation of either resistant × resistant or susceptible × susceptible crosses. This indicated the role of the same loci for resistance and susceptibility in the parents

studied. The few resistant plants recorded in the susceptible × susceptible cross combinations could be escapes.

A further study to analyse and characterise all available resistant sources for their allelic relationship with regard to strain 2 of the sterility mosaic pathogen would be of immense value in breeding resistant cultivars with a broad genetic base. It would also be useful to investigate the genetics of the host-pathogen interaction by including different strains of the pathogen. Attempts should also be made to combine different alleles to diversify the genetic composition of the lines with regard to the resistance genes. Further, it would be desirable to develop a series with various allelic combinations in a common genetic background to be used as a tester to facilitate proper identification of alleles in different genotypes.

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