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Research Article

Inheritance of sterility mosaic disease resistance to Bangalore and Patancheru isolates in pigeonpea (*Cajanus cajan* (L.) Millsp.)

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Abstract:
Sterility mosaic disease (SMD), is an important biotic constraint in pigeonpea (*Cajanus cajan* (L.) Millsp.) in Indian subcontinent. It is caused by a virus and transmitted by eriophyid mites, *Aceria cajani* Channabasavanna. A comprehensive study of variability in the sterility mosaic pathogen revealed the occurrence of five different isolates in India. Amongst them, three distinct isolates have been characterised, *viz*., Bangalore, Patancheru and Coimbatore. Studies were conducted at Bangalore and Patancheru to determine the inheritance of resistance to Bangalore and Patancheru isolates of the SMD involving a resistant (ICP 7035) and susceptible (TTB 7) genotypes. Observations in parents, F_1 indicated dominance of susceptibility over resistance. The disease reaction of the individual F_2 plant derived F_3 families for Patancheru isolate was controlled by two genes with dominance epistasis and for Bangalore isolate, absence of resistant plants indicate action of two or more genes in controlling resistance to SMD.

Key words:
inheritance, pigeonpea, resistance, sterility mosaic, susceptibility

Introduction

Pigeonpea (*Cajanus cajan* (L.) Millsp.) is an important food legume crop of rainfed agriculture in the semi-arid tropics. Globally, pigeonpea is grown in an area of 4.86 M ha with production of 4.1 million tons and India is the primary pigeonpea growing country in the world, accounting for 3.73 M ha area and 3.07 million tons of production (FAOSTAT 2009: http://www.fao.org). Although, India leads the world both in area and production of pigeonpea, its productivity is lower than the world average. This could be attributed to various abiotic (e.g. drought, salinity and water-logging) and biotic (e.g. diseases like *Fusarium* wilt, sterility mosaic and pod borers) stresses encountered by the crop at different growth stages. Among the diseases of pigeonpea, sterility mosaic disease (SMD) is considered to be the most important and at times can cause yield loss upto 95 per cent (Reddy and Nene, 1981; Kannaiyan *et al.* 1984). The disease is caused by pigeonpea sterility mosaic virus (PPSMV) (Kumar *et al.* 2003) and transmitted by eriophyid mites (*Aceria cajani* Channabasavanna, Seth 1962). The disease is characterized by symptoms such as bushy and pale green appearance of plants, reduction in its size, increase in number of secondary and mosaic mottling of leaves; and finally partial or complete cessation of reproductive structures. The expression of disease on plants is uneven and some parts of the plant may show disease symptoms and other parts may remain unaffected (Kumar *et al.* 2003).

The task of developing resistant varieties of pigeonpea is complicated in view of the genetic plasticity of the pathogen. A comprehensive study of variability in the sterility mosaic pathogen revealed the occurrence of five different isolates of the pigeonpea sterility mosaic virus (Reddy *et al.* 1993) in India. Amongst them, three distinct isolates have been characterised, *viz*., Bangalore, Patancheru and Coimbatore. The Patancheru and Coimbatore isolates are mild strains while the Bangalore isolate is the most virulent (Kulkarni *et al.* 2003). This dynamic nature of the SMD pathogen has warranted the use of strain specific sources of resistance in crop improvement. Knowledge of inheritance controlling the traits is essential for efficient breeding. There are conflicting reports about the genetics of resistance to

sterility mosaic disease claiming both susceptibility and resistance to be dominant. However in most cases, susceptibility was shown to be dominant and resistance to be under the control of recessive genes (Singh *et al.* 2003). The resistance to SMD has been reported to be controlled by single recessive gene (Murugesan *et al.* 1997; Srinivas *et al.* 1997) and oligo-genic (Sharma *et al.* 1984; Nagaraj *et al.* 2004). However, all the above studies were conducted for a particular isolate and relied on F_2 and back cross populations for SMD inheritance in pigeonpea. Thus in the present study, $F_{2:3}$ population were used at Bangalore and Patancheru locations to determine the inheritance of SMD resistance for virulent strain of Bangalore and mild strain of Patancheru isolates.

Material and Methods

Plant materials: The $F_{2:3}$ progenies for SMD screening was developed at the All India Co-ordinated Research Project on Pigeonpea, University of Agricultural Sciences, Bangalore by crossing a susceptible line TTB 7 with a resistant parent ICP 7035. Molecularly and morphologically (SMD) diverse parents were selected based on the previous reports of Rangaswamy *et al.* (2005) and Ganapathy *et al.* (2010). F_1 plants along with their parents were grown during summer 2007 and were selfed by covering the plants with nylon net to prevent outcrossing through honey bees and other insect pollinators. Seeds from the F_1 plants were collected and used for raising F_2 generation during rainy season. Selfed seeds obtained from F_2 plants were collected to forward $F_{2:3}$ generation for phenotyping against SMD. During rainy season 2008, all the 224 $F_{2:3}$ population with an average of 20 plants per family along with their parents, F_1s and susceptible check (ICP 8863) were raised in poly bags with two replications for phenotyping against SMD.

Resistance screening techniques for SMD: Phenotyping of SMD was done at University of Agricultural Sciences, Bangalore and ICRISAT, Patancheru to screen against Bangalore and Patancheru isolates of SMD following "Leaf Stapling Technique" (Nene and Reddy, 1976). SMD infected leaves containing mites were stapled to leaves of test plants at 2 - 3 leaf stage. As the stapled leaflets from the infected plants dried, the mites from infected leaves migrate to healthy leaf and inoculates the virus. The susceptible check ICP 8863 was included in both sets, at frequent intervals, to monitor the disease spread. At both the locations, plants were scored for incidence of SMD at 15 day intervals up to 75 days by counting the healthy plants (no mosaic symptoms) and diseased plants (with mosaic symptoms) as per the criterion followed in All India Co-ordinated Research Project on Improvement of Pigeonpea. The progenies were classified as resistant (0-10.0 % of plants infected); moderately resistant (10.1-30.0 % of plants infected) and susceptible (30.1-100% of plants infected) (Singh *et al.* 2003). Based on the percentage of susceptible and resistant progenies per family the above score was used to classify them into resistant, moderately resistant and susceptible classes.

Statistical analysis:The analysis of variance was performed for disease incidence obtained at two different locations to test the significance of differences among genotypes. The adjusted mean values of SMD disease reaction were used to estimate coefficients of skewness and kurtosis using 'STATISTICA' software. The Chi-square (χ^2) test was used to test the goodness of fit of the segregating $F_{2:3}$ populations with the expected phenotypic ratios.

Results and Discussion

In the present study, a population of 224 F_2 plants were selfed to obtain the $F_{2:3}$ population and used for phenotyping against SMD at two different locations *viz*., UAS, Bangalore and ICRISAT Patancheru to understand the nature of inheritance to SMD. The mean SMD percentage disease reaction of 15 plants for each $F_{2:3}$ progeny against Bangalore and Patancheru isolates were subjected to ANOVA. The calculated F value was significant at 1% level of significance, indicating that the genotypes under study showed considerable variation for the SMD disease reactions.

Frequency distribution of $F_{2:3}$ segregating population to SMD: The SMD incidence for Bangalore isolate ranged between 4.1 to 100% with a mean of 78.94 %. The coefficient of skewness was -1.45 while that of kurtosis was 2.11. SMD incidence for Patancheru isolate ranged between 0 to 100% with a mean of 55 % disease incidence. The coefficient of skewness was -0.49 while that of kurtosis was -1.09. The variation existed in the $F_{2:3}$ population for SMD incidence is represented graphically using frequency distribution of means for two different isolates (Fig. 1). The per cent disease incidence was plotted on X-axis against genotype frequency on Y - axis with equal class intervals. The resulting histogram showed near normal curves for both the isolates with skewed towards susceptibility for SMD. Although the pattern of frequency distribution of SMD incidence in the $F_{2:3}$ appeared to be some what continuous, large number of plants could be classified into moderately resistant and susceptible categories. Only few plants were classified into resistant group, suggesting

quantitative inheritance for SMD resistance in pigeonpea.

Reaction of parents and F_1s to SMD for Bangalore and Patancheru isolates is presented in Table 1. For both the isolates, susceptible control (ICP 8863) exhibited 100% disease incidence. At Bangalore, the resistant parent ICP 7035 showed 6.6 per cent disease incidence where as the susceptible genotype TTB 7 showed 100 per cent disease incidence with severe mosaic symptoms. While, at Patancheru, ICP 7035 exhibited zero per cent disease incidence with no apparent symptoms while the susceptible genotype TTB 7 showed 100 per cent susceptibility with severe mosaic symptoms. All the F_1s screened for SMD infection for Bangalore and Patancheru isolates were found to be susceptible indicating the susceptibility to be dominant over resistance. Similar observations on susceptibility being under the influence of dominant genes have been reported in pigeonpea (Singh *et al.* 1983; Sharma *et al.* 1984; Nagaraj *et al.* 2004 and Ganapathy *et al.* 2009). On the contrary, susceptibility under the influence of recessive genes was reported by Murugesan *et al.* (1997). In another study on inheritance of resistance to two isolates of SMD, Srinivas *et al.* (1997) used three crosses and observed that resistance was dominant in two crosses and susceptibile in the other cross.

The phenotyping against SMD is destructive and was avoided in F_2 generation, so that seeds can be harvested from these plants to obtain the advance generation for QTL mapping. At Patancheru, five families failed to germinate and hence only 219 were considered for evaluation and 0 to 100 per cent SMD incidence was recorded with high variability. Out of 219 $F_{2:3}$ families screened against SMD, 44 were resistant, 11 were moderately resistant and 164 were susceptible. For Patancheru, relatively more number of plants showed resistance to SMD because of lower level of virulence to SMD isolate prevailing in Patancheru location (Kulkarni *et al.* 2003). The observed segregation ratios from Patancheru isolate were 164 (susceptible): 44 (resistant): 11 (moderately resistant). These best fit to digenic model involving duplicate dominance epistasis (12 susceptible: 3 resistant: 1 moderately resistant) with $\chi^2 = 0.862$, $P = 0.649$. While at Bangalore, out of 224 $F_{2:3}$ families screened against SMD, none of the families were resistant, 11 were moderately resistant and 213 families were susceptible. Absence of resistant families indicates higher level of virulence to SMD isolate prevailing in Bangalore location and the same was reported by Kulkarni *et al.* (2003). Resistant families were relatively low for Bangalore isolate as compared to Patancheru isolate. Absence of resistant plant indicates action of two or more genes in imparting resistance to SMD. Considering the disease reaction at two locations, resistance to SMD may be controlled by two or more genes. Recently, Gnanesh *et al.* (2011) for the first time identified four QTLs for Patancheru SMD isolate and two QTLs for Bangalore SMD isolate.

Genetics of SMD has been studied earlier and depending on the resistance source, SMD isolate and scoring method, resistance to SMD in pigeonpea appears to be complex (Saxena, 2008). The present study reveals digenic inheritance of SMD for Patancheru and polygenic for Bangalore isolates. It is therefore postulated that, for both Bangalore and Patancheru isolates, susceptibility is controlled by dominant genes, therefore number of plants with high level of resistance to SMD are fewer in $F_{2:3}$ generation. Resistance to SMD in general is controlled by recessive genes and the causal organism has higher level of virulence and hence it is very difficult to realize plants with resistance to SMD in Bangalore location. There is a need to search sources with high level resistance from either primary or tertiary gene pools.

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References

Ganapathy, K. N., Byregowda, M., Venkatesha, S.C., Rama Chandra, R., Gnanesh, B.N. and Girish, G. 2009. Identification of AFLP markers linked to sterility mosaic disease in pigeonpea *Cajanus cajan* (L.) Millsp. *Int. J. Integr. Biol*., **7**: 145-149.

Ganapathy, K. N., Gnanesh, B. N., Byregowda M., Venkatesha S. C., Sunil S. G. and Channamallikarjuna, V. 2010. AFLP analysis in pigeonpea (*Cajanus cajan* (L.) Millsp.) revealed close relationship of cultivated genotypes with some of its wild relatives. *Genet. Resour. Crop Evol.,* doi 10.1007/s10722-010-9621-1.

Gnanesh, B. N., Abhishek, B., Sarma, M., Wesley V, Byre Gowda, M., Pande, S., Saxena, R., Saxena, K. B. and Varshney, R. K. 2011. Genetic mapping and quantitative trait locus analysis of resistance to sterility mosaic disease in pigeonpea [*Cajanus cajan* (L) Millsp.]. *Field Crop Res*., doi:10.1016/j.fcr.2011.04.011.

Kannaiyan, J., Nene, Y. L., Reddy, M. V., Ryan, J. G. and Raju, T. N. 1984. Prevalence of pigeonpea

disease and associated crop losses in Asia, Africa and America. *Trop. Pest Manage*., **30**: 62-71.

Kulkarni, N. K., Reddy, A. S., Kumar, P. L., Vijayanarasimha, J., Rangaswamy, K. T., Muniyappa, V., Reddy, L. J., Saxena, K. B., Jones, A. T. and Reddy, D. V. R. 2003. Broad based resistance to pigeonpea sterility mosaic disease in accessions of *Cajanus scarbaeoides* (L.) Benth. *Indian J. Plant Prot*., **31:** 6-11.

Kumar, P. L., Jones, A. T. and Reddy, D. V. R. 2003. A novel mite transmitted virus with a divided RNA genome closely associated with pigeonpea sterility mosaic disease. *Phytopath*., **93**: 81-91.

Murugesan, S., Murugan, E. and Nadarajan. N. 1997. Inheritance of duration, leaf colour, sterility mosaic disease resistance and growth habit in Pigeonpea. *Madras Agric. J.* **84**: 10 -12.

Nagaraj, K. M., Chikkadevaiah, Kulkarni, R. S. 2004. Inheritance of resistance to sterility mosaic virus in pigeonpea (*Cajanus cajan* (L.) Millsp.). *Indian J. Genet*., **64**: 118 -120.

Nene, Y. L. and Reddy, M. V. 1976. A new technique to screen pigeonpea for resistance to sterility mosaic. *Trop. Grain Legume Bull*., **5**: 23.

Rangaswamy, K. T., Muniyappa, V., Lava Kumar, P., Saxena, K. B., Byregowda, M., Raghavendra, N., Pandurangaiah, K., Vijay Kumar, R., Waliyar, F. and Jones, A. T. 2005. ICP 7035 – A sterility mosaic resistant vegetable and grain purpose pigeonpea variety. *SAT eJournal* **1**: 1-3.

Reddy, M. V. and Nene, Y. L. 1981. Estimation of yield loss in pigeonpea due to sterility mosaic. In: *Proceedings of International workshop on pigeonpeas*. 2: 15-19 December 1980, ICRISAT center, Patancheru, Andra Pradesh, India. **pp.** 305 – 312.

Reddy, M. V., Raju, T. N., Nene, Y. L., Ghanekar, A. M., Amin, K. S., Arjunan, G., Astaputre, J. V., Sinha, B. K., Muniyappa, V., Reddy, S. V., Gupta, R. P. and Gangadharan, K.1993. Variability in sterility mosaic pathogen of pigeonpea in India. *Indian Phytopath*., **46**: 206–212.

Saxena, K.B. 2008. Genetic improvement of pigeonpea-A review. *Trop. Plant Biol*., **1**: 159-178.

Seth, M. L. 1962. Transmission of pigeonpea sterility by an eriophyid mite. *Indian Phytopath*., **15**: 225-227.

Sharma, D., Gupta, S. C., Rai, G. S. and Reddy, M. V. 1984. Inheritance of resistance to sterility mosaic disease in pigeonpea. *Indian J. Genet*., **44**: 84 - 90.

Singh, B. V., Pandya, B. P., Gautam, P. L., Beniwal, S. P. S. and Pandey, M. P. 1983. Inheritance of resistance to sterility mosaic virus in pigeonpea. *Indian J. Genet*., 43: 487- 493.

Singh, I. P., Vishwadhar and Dua, R. P. 2003. Inheritance of resistance to sterility mosaic in pigeonpea (*Cajanus cajan*). *Indian J. Agric. Sci*., **73**: 414-417.

Srinivas, T., Reddy, M. V., Jain, K. C. and Reddy, M. S. S. 1997. Studies on inheritance of resistance and allelic relationships for strain 2 of Pigeonpea sterility mosaic pathogen. *Ann. Appl. Biol.,* **130**: 105-110.

Table 1. Reaction of parents and F_1 against pigeonpea sterility mosaic disease

	Genotypes	Total no. of plants	Resistant plants	Susceptible plants	Per cent disease incidence	Disease reaction
Bangalore isolate						
P_1	TTB 7	30	-	30	100	Susceptible
P_2	ICP 7035	30	28	2	6.6	Resistant
F_1	TTB 7 X ICP 7035	25	-	25	100	Susceptible
Control	ICP 8863	100	-	100	100	Susceptible
Patancheru isolate						
P_1	TTB 7	40	-	40	100	Susceptible
P_2	ICP 7035	40	40	-	0	Resistant
F_1	TTB 7 X ICP 7035	30	-	30	100	Susceptible
Control	ICP 8863	100	-	100	100	Susceptible

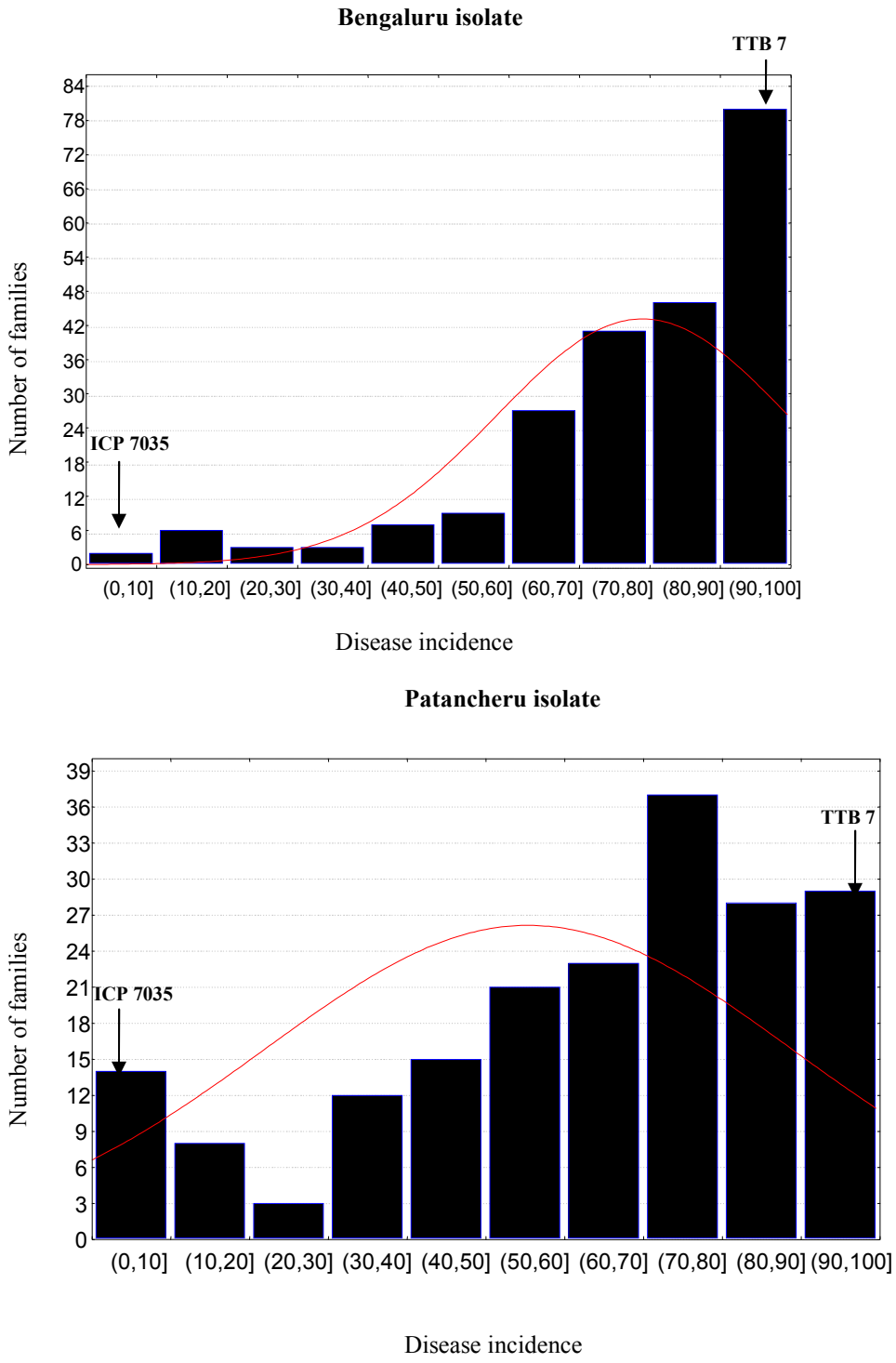

Fig. 1 Frequency distribution of pigeonpea sterility mosaic disease incidence in $F_{2:3}$ families of TTB 7 × ICP 7035.