Indian Journal of Agricultural Sciences **82** (10): 889–91, October 2012

Short Communications

Slow rusting resistance in pea (*Pisum sativum*)*

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Received: 24 december 2010; Revised accepted: 28 July 2012

Key words: Gene action, Six parameter model, Slow rusting resistance

Pea rust caused by *Uromyces fabae* (Pers.) de Bary is one the important limiting factors for the pea production and productivity. Warm and humid conditions favour the outbreak of rust disease and results complete losses when starts early in the season. Pea rust is a major problem under late sown conditions in India. Generally, the infection of the rust begins during the early pod setting stage (Chand *et al*. 2004). Component based resistance in pea genotypes have been reported (Chand *et al.* 2006), the available resistant sources were of slow rusting types. There was a lack of complete resistance in pea (Fondevilla *et al*. 2004). Recently, Vijayalakshmi *et al*. (2005) concluded the role of more than one gene for rust resistance in pea.

The selection of six parents (HUVP 1, JP 4, S 143 as fast rusting and Pant P 11, JPBB 3, FC 1 as slow rusting) were chosen on the basis of diversity in slow rusting traits. The experiment comprising parents (P_1 and P_2), F_1s, F_2s, B_1s, B_2s were planted along with infector rows (Cv. HFP 4) during rabi 2006–2007 with two replications in completely randomized block design (CRBD). Standard agronomic practices were followed to raise a good crop. No fungicide was sprayed. Inoculation was done at the pre-flowering stage (50-55 days after sowing) as per the technique reported by Chand *et al.* (2004). Each plot of P_1, P_2 and F_1 consisted of single row, while for each B_1, B_2, there were two rows and for each F_2 ten rows were planted. The row length was 3 m., row to plant distance was 45 cm × 10 cm. Excluding border plants in each replication data were recorded for slow rusting components, i e number of pustules per cm^2 of leaf area (NP), number of non-bursting pustules per cm^2 of leaf area (NBP), pustule size (PS) on 0-5 Scale, number of aecial cups per pustule (NACP) and disease severity from which AUDPC (area under disease progress curve) values were calculated following the formula given by Shaner and Finney (1977). Data were recorded on ten randomly selected plants from each of the parents and F_1s, 75 plants in each F_2 and 30 plants on each B_1 and B_2. To detect the presence of gene interaction ABCD Scaling Test (Mather 1949) and Joint Scaling Test (Cavalli 1952) were applied. The non-interacting crosses thus detected were analyzed under the three parameter model of Cavalli (1952). The interacting crosses were analyzed following six-parameter model. The observed means for interacting crosses were also subjected to Cavalli's weighted least square analysis for estimation of genetic parameters after eliminating least important epistatic components.

The genetics of rust resistance in pea (*Pisum sativum* L.) is not clearly understood. The monogenic, the polygenic and an oligogenic along with possible involvement of polygenes (Vijayalakshmi *et al.* 2005) have been suggested to control the rust resistance. Five crosses were evaluated for AUDPC and four other slow rusting traits (Table 1). Out of 25 cases (five crosses, five characters) 23 cases were detected as interacting following A B C and D scaling test. However, Joint scaling test detected 24 cases as interacting. All the five crosses showed the epistasis for NP, NBP, PS and AUDPC when A B C D and Joint scaling tests were applied simultaneously. Most of the crosses were found predominantly under the influence of additive gene effect when six-parameter model was applied. For NP, NBP and NACP all the five crosses were under the influence of additive genetic variance. Significant estimate of dominance component (h) was observed for three crosses each for NP and NBP, four crosses for AUDPC, all the five for NACP and only one cross for PS. Stoddard and Herath (2001) also observed that low disease was largely conferred by additive gene action in faba beans. The 'h' was negative in most cases indicating the dominance of alleles imparting resistance over the alleles for susceptibility. For number of non bursting pustules, the sign of dominance × dominance (l) gene effect was negative indicating the dominance of low number of non-bursting pustules which is desirable. Both additive and non-additive gene effects were important for pustule size in most of the crosses

*Short note

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Table 1 Estimates of six-parameter in five crosses of pea

Character	Crosses	(m̂)	(d̂)	(ĥ)	(î)	(ĵ)	(l̂)	Epi-stasis	Chi-square value
No. of bursting pustules/cm² of leaf area	HUVP 1 × Pant P 11	56.48**±4.08	35.44**±2.67	-86.13**±17.59	54.57**±17.15	-40.42±2.75	43.76±20.99		
	HUVP 1 × JPBB 3	145.61**±3.92	59.20**±4.39	-233.49**±18.42	-56.70**±17.97	6.29±5.39	115.74**±24.90		D
	JP 4 × FC 1	249.86**±4.17	231.48**±6.79	-488.49**±22.38	-308.99**±21.51	3.73±8.54	366.54**±34.19		D
	JPBB 3 × Pant P 11	75.32**±4.32	66.11**±1.91	34.55±17.98	84.25**±17.69	-10.89±10.70	-29.58±19.94		
	S 143 × FC 1	98.03**±4.32	37.03**±4.13	7.23±19.29	-16.27**±4.43	20.88±19.15	-12.78±24.37		
No of non-bursting pustules/cm² of leaf area	HUVP 1 × Pant P 11	19.98**±1.63	15.87**±2.59	14.30±8.50	41.70**±8.32	-11.43±8.75	-67.30**±12.74		
	HUVP 1 × JPBB 3	51.09**±3.54	51.34**±1.37	-25.03±14.58	-25.27±14.40	25.38**±1.77	-32.68*±15.84		
	JP 4 × FC 1	11.70**±0.85	3.92*±1.62	-13.43*±4.89	-4.03±4.69	8.07**±1.73	27.06**±7.81		D
	JPBB 3 × Pant P 11	7.02**±0.67	5.45**±1.15	12.50**±3.76	13.50**±3.51	1.25±1.39	-5.46±5.95		
	S 143 × FC 1	8.29**±1.17	5.40**±1.18	23.45**±5.49	11.40*±5.22	10.90**±1.36	15.76*±7.44		C
Pustule size	HUVP 1 × Pant P 11	3.49**±0.27	-0.17±0.12	3.60**±1.09	3.55**±1.09	-0.17±0.16	-2.58*±1.18		D
	HUVP 1 × JPBB 3	3.00**±0.20	1.17**±0.07	0.53±0.82	1.13±0.81	-0.39**±0.09	-3.26**±0.86		
	JP 4 × FC 1	2.86**±0.02	-1.20**±0.14	0.09±0.31	-0.64*±0.29	0.48**±0.14	2.79**±0.61		
	JPBB 3 × Pant P 11	2.92**±0.09	-0.37**±0.12	0.34±0.42	-0.46±0.42	0.93**±0.14	3.66**±0.61		
	S 143 × FC 1	3.80**±0.23	-0.07±0.19	0.31±1.01	0.01±0.99	0.44*±0.21	2.18±1.23		
No. of aecial cups/pustule	HUVP 1 × Pant P 11	8.37**±0.16	-1.07±1.14	3.44±2.49	2.79±2.37	0.04±1.19	2.24±4.85		0.214
		(6.15**±0.71)	(-1.08**±0.35)	(4.42**±0.13)	(3.83**±0.78)	(0.99±1.31)			
	HUVP 1 × JPBB 3	7.97**±0.18	3.74**±0.17	-1.92**±0.29					5.99
	JP 4 × FC 1	6.67**±0.43	-1.28±0.81	-2.01±2.37	-2.51±2.35	1.83*±0.85	(6.56±3.72)		3.11
		(5.38**±0.79)	(3.04**±0.25)	(1.88*±0.87)	(1.35±0.85)	(3.78*±1.69)			
	JPBB 3 × Pant P 11	6.15**±0.30	-0.37±0.99	2.17±2.35	1.07±2.31	2.79*±1.06	6.46±4.20		2.36
		(3.20**±0.59)	(-3.17**±0.39)	(5.63**±0.85)	(4.44**±0.73)	(8.21**±1.24)			
	S 143 × FC 1	8.34**±0.58	0.10±0.49	4.23±2.63	2.30±2.52	2.00**±0.65	5.09±3.40		2.24
		(4.12**±1.19)	(-1.85**±0.43)	(7.27**±1.67)	(5.52**±1.31)	(3.23*±1.20)			
AUDPC	HUVP 1 × Pant P 11	476.82**±134.51	268.34**±9.97	-336.34±538.49	-54.59±538.43	-124.54**±11.57	-335.08±539.80		0.39
		(895.13**±24.05)	(392.92**±5.87)	(-670.16**±27.67)	(-388.42**±26.11)	(249.87**±23.11)			
	HUVP 1 × JPBB 3	533.52**±14.82	332.51**±28.39	-791.27**±89.16	-456.39**±82.07	95.19**±33.68	715.48**±145.81	D	
	JP 4 × FC 1	-439.16**±14.49	14.59±25.66	-754.45**±79.94	-593.45**±77.42	-250.53**±27.91	826.76**±124.43	D	
	JPBB 3 × Pant P 11	176.09**±16.18	91.00**±5.22	-110.26±66.63	37.62±65.54	-69.13**±9.77	97.13±72.12		1.81
		(170.71**±19.99)	(161.24**±8.21)	(-28.31±27.18)	(119.87**±23.78)	(-138.03**±19.53)			
	S 143 × FC 1	266.47**±4.89	85.75**±12.03	-131.62**±36.01	6.28±31.03	-113.40**±16.82	177.36*±63.53	D	

-values in the parentheses are values obtained following sequential elimination of least important component

* Significant at 5% level of significance, ** Significant at 1% level of significance

which is in agreement to with those of Stoddard and Herath (2001). Similar type of inheritance was observed for the number of aecial cups/pustule. Besides additive and dominance, epistatic effects were also found important in the expression of AUDPC. The magnitude of 'h' was greater than 'd' for AUDPC for most crosses that might have been resulted from overdominance or unidirectional dominance or dispersal of genes in the parents that lower the estimates of d type of gene effect. The negative value of 'i' gene effect increases the possibility of obtaining lines with low AUDPC in infinite population. The duplicate type of epistasis was observed for NP in the crosses HUVP 1 × Pant P 11, HUVP 1 × JPBB 3, JP 4 × FC 1, for PS in HUVP 1 × Pant P 11 and for AUDPC in HUVP 1 × JPBB 3, JP 4 × FC 1 and S 143 × FC 1. The complementary type of epistasis was displayed by the cross S 143 × FC 1 for NBP.

Slow rusting is a form of resistance which reduces the epidemic build-up on resistant cultivars compared to highly susceptible standards. Slow rusting resistance cultivar exhibit less infection efficiency, smaller pustule size, less spore production and lower AUDPC than the susceptible cultivars. Since components of slow rusting are quantitative in nature, breeding for rust disease resistance in pea would aim at recovery of superior recombinants by accumulating favourable genes. By making selection in segregating generations for two or more components of slow rusting resistance at least few genes responsible for partial resistance can be accumulated in a single genotype which is expected to be more durable. So the pedigree method of breeding would be the suitable breeding strategy to recover superior recombinants with enhanced level of rust resistance (Srivastava *et al.* 2009). Crosses HUVP 1 × Pant P 11 for NP, S 143 × FC 1 for NBP and HUVP 1 × JPBB 3 for AUDPC exhibited importance of both additive and non-additive components. In such cases biparental mating between the recombinants in early segregating generations would be useful to recover superior recombinants in later generations.

SUMMARY

Genetic studies on pea rust was conducted to assess the nature and magnitude of gene action for slow rusting resistance in pea. Five crosses were made involving six genotypes of pea that were chosen on the basis of diversity in slow rusting traits. Data were recorded on five slow rusting traits and were subjected to the generation mean analysis. The additive types of gene action predominantly controlled the slow rusting resistance in most of the cases. Among interaction components both additive × additive (î) as well as dominance × dominance (Î) kinds of gene action was important for slow rusting resistance; duplicate type of epistasis was predominant in the expression of slow rusting resistance. To recover recombinants with enhanced level of slow rusting resistance, pedigree method of breeding has been suggested.

REFERENCES

Cavalli L L. 1952. *An Analysis of Linkage in Quantitative Inheritance*, pp 135–44. *E C R of Rieve and Waddington* C H (Eds). HMSO, London.

Chand R, Srivastava C P and Kushwaha C. 2004. Screening techniques of pea (*Pisum sativum* L.) genotypes against rust disease (*Uromyces fabae* Pers. de Bary). *Indian Journal of Agricultural Sciences* **74**: 166–7.

Chand R, Srivastava C P, Singh B D and Sarode S B. 2006. Identification and characterization of slow rusting components in pea (*Pisum sativum* L.). *Genetic Resources and Crop Evolution* **53**: 219–24.

Fondevilla S, Barilli E, Sillero J C, Torres A M, Cubero J I, Barragan M and Rubiales D. 2004. Mejora del guisante por resistencia a enfermedades.

Mather K. 1949. *Biometrical Genetics* (I Edin). Methuen, London.

Shaner E and Finney. 1977. The effect nitrogen fertilization on the expression of slow-mildewing resistance in wheat. *Phytopathology* **67**: 1 051–6.

Srivastava C P, Lal S, Kushwaha C, Chand R, Singh A K, Singh V K and Singh B D. 2009. Genetic variability in the segregating generations and the breeding strategies for selection to improve yield and resistance to rust in pea. *Journal of Food Legumes* **22**: 158–61.

Stoddard F L and Herath I H M H B. 2001. Genetic analysis of partial rust resistance in faba beans. *Australian Journal of Agricultural Research* **52**: 73–84.

Vijayalakshmi S, Kusum Yadav, Chanda Kushwaha, Sarode S B, Srivastava C P, Ramesh Chand and Singh B D. 2005. Identification of RADP markers linked rust resistance in pea (*Pisum sativum* L.). *Euphytica* **144**: 265–74.