Indian Journal of Agricultural Sciences **83** (6): 617–21, June 2013/Article

Estimation of gene effects for powdery mildew resistance in garden pea (*Pisum sativum*)

S K SANWAL[1], RAJESH KUMAR[2] and M LOGANATHAN[3]

Indian Institute of Vegetable Research, P B 01, P O Jakhini (Shahanshahpur), Varanasi, Uttar Pradesh 221 305

Received: 17 July 2012; Revised accepted: 5 February 2013

ABSTRACT

Generation means analysis was carried out to estimate the nature and magnitude of gene action for identifying the segregants resistance to powdery mildew disease of garden pea (*Pisum sativum* L.). Six generations (P_1, P_2, F_1, F_2, B_1 and B_2) of three crosses, VRP 16 × VRP 22, VRP 16 × VRPMR 9 and VRP 343 × Arka Ajeet were grown for evaluation of disease incidence (DI), per cent disease index (PDI) and area under disease progress curve (AUDPC). AUDPC values revealed differential rate of disease development on various genotypes of pea. Highest rate of disease development was observed on VRP 16 (667.00) and lowest on VRP 22 (74.09). Both additive and dominance gene actions were found to be important in inheritance of powdery mildew resistance (all the three characters) including non-allelic interactions. In view of the parallel role of additive and non-additive gene effects, selection in the segregating generations should be delayed to diminish the dominance gene effects. Duplicate type of epistasis was detected for all the three pathological characters in all the crosses whose effect can be eliminated by following sophisticated selection procedure such as reciprocal recurrent selection and/or biparental mating in early segregating generations for the development of powdery mildew resistant varieties.

Key words: Epistasis, Garden pea, Gene action, Powdery mildew

The garden pea (*Pisum sativum* L.), commonly known as English pea or green pea, is one of the oldest vegetables cultivated in the world. In India, it is mostly grown as winter crop for its green pods in the plains of northern India and as a summer vegetable in the hills. Powdery mildew (*Erysiphe polygoni* DC) is the major limiting factor in pea production and is present in all areas where peas are cultivated (Hagedorn 1985, Smith *et al*. 1996). The disease is favoured by warm days and cool nights where dew forms. In Indian plains, it appears in epidemic form almost every year when the plants are in the pod stage during January to March and turning whole fields white and seriously affecting the photosynthetic activity of the plants leading to substantial losses in yield and reduction in pod quality and seed size (Gritton and Ebert 1975). The losses in yield in a 100% infected crop were estimated to be 21-31% in pod number and 26-47% in pod weight (Munjal *et al.* 1963, Warkentin *et al.* 1996). Leaves, stems and pods may become infected resulting in withering of foliage and occasionally in plant death. Severe pod infection may result in "hollow" peas (Reiling 1984). Use of chemical fungicide involving heavy inputs results in unsafe produce and create environmental pollution. Therefore, cost-effective and environment friendly option is to develop resistant varieties.

No breeding method can achieve the desirable goal without precise understanding of gene action involved for resistance. Powdery mildew resistance in pea is predominantly controlled by both additive and dominance components including epistasis (Tyagi 1999, Tyagi and Srivastava 2000). Moreover, these reports are based only on per cent disease index (PDI), through which the rate of development of disease cannot be quantified. The apparent rate of disease development is a measure of the speed at which an epidemic develops. Despite the presence of virulent pathogen and favourable environment, differences were observed in the rate of disease development on various genotypes culminating in low terminal disease severity. The slowing down of rate of infection has been attributed to the level of host resistance and Van der Plank (1963) considered this rate as horizontal resistance. Wilcoxson *et al.* (1975) quantified the area under disease progress curve (AUDPC) as A-value. Such quantification of disease demonstrates the importance of area under disease progress curve (AUDPC) value as a reliable parameter to estimate and rank the performance of various

[1] Senior Scientist (e mail: satishsanwal@rediffmail.com), [2] Senior Scientist (e mail: rajesh74@gmail.com), [3] Senior Scientist (e mail: logumurga@yahoo.co.in)

host genotypes depending on their ability to retard the rate of disease development. Hence, the present investigation was designed to know the nature of gene action controlling powdery mildew resistance through disease incidence (DI), PDI and AUDPC for formulating further breeding strategy.

MATERIALS AND METHODS

The present investigation was carried out at the experimental farm of Indian Institute of Vegetable Research, Varanasi during 2008-2011. The experimental material comprised of one powdery mildew susceptible (VRP 16) and four resistant (VRP 22, VRPMR 9, VRP 343 and Arka Ajeet) lines. The lines were used to develop three F1's, viz. VRP 16 × VRP 22, VRP 16 × VRPMR 9 and VRP 343 × Arka Ajeet during winter season of 2008-09. The F_1 seed of these three crosses along with their parents were sown during winter season of 2009-10 to develop backcross progenies (BC_1 and BC_2) by crossing each F_1 to both of its parents and F_2 seed was obtained through self pollination. Hence, six generations, viz. P_1 and P_2, first and second parental generations; F_1 and F_2, first and second filial generations; B_1 ($F_1 \times P_1$) and B_2 ($F_1 \times P_2$), backcrosses from three different crosses were planted in the randomized block design with three replications during 2010-11. The parents, F_1s, F_2s and backcross were planted in 3 m × 3 m plot keeping 30 cm distance between the rows and 10 cm between plants. The experimental plot was surrounded by 5-6 rows of susceptible variety Pant Uphar to ensure uniform spread of disease. The disease reaction was recorded on ten plants from parents and F_1s; 20 plants from backcrosses and 30 plants from F_2 generations using all the leaves of selected plants of all the three crosses.

Disease index (DI) was recorded as per Mayee and Datar (1986). PDI was recorded by grading the leaves as per 0-5 scale and calculated using the formula given by Wheerler (1969). The mildew severity was scored at 5 days interval from first appearance of disease to maturity and AUDPC values were calculated as per Wilcoxson *et al.* (1975). The data was subjected to estimate means and variances pooled over replications after its transformation. Six parameters, viz. m (average effect), d (additive), h (dominance), i (additive × additive), j (additive × dominance) and l (dominance × dominance) were estimated as per Hayman (1958) model.

Table 1 Mean performance of parents, F1's, F2's and backcrosses

Cross		Disease incidence	Per cent disease index	Area under disease progress curve
VRP 16 × VRP 22	P1	72.5 (58.37)	64.20 (53.25)	667.00 (25.81)
	P2	9.80 (18.24)	6.42 (14.65)	74.09 (8.61)
	F1	46.50 (42.99)	30.12 (33.21)	361.77 (19.02)
	F2	32.50 (34.76)	24.30 (29.53)	266.5 (16.35)
	BC1	61.20 (51.47)	25.48 (30.33)	455.94 (21.33)
	BC2	24.30 (29.53)	12.10 (20.36)	200.23 (14.15)
VRP 16 × VRPMR 9	P1	72.5 (58.37)	64.20 (53.25)	667.00 (25.82)
	P2	8.20 (16.64)	7.45 (15.89)	77.50 (8.80)
	F1	42.50 (40.69)	28.50 (32.27)	332.68 (18.24)
	F2	34.40 (35.91)	22.60 (28.38)	294.16 (17.15)
	BC1	64.60 (53.49)	27.30 (31.50)	488.10 (22.09)
	BC2	18.90 (25.77)	14.50 (22.38)	188.65 (13.73)
VRP 343 × Arka Ajeet	P1	12.50 (25.77)	4.54 (12.25)	109.05 (10.43)
	P2	17.60 (20.70)	8.50 (19.95)	146.85 (12.12)
	F1	20.25 (24.80)	14.40 (22.30)	172.15 (13.12)
	F2	18.65 (26.92)	11.80 (25.62)	142.12 (11.92)
	BC1	11.15 (25.60)	142.12 (19.55)	142.12 (9.82)
	BC2	21.55 (19.55)	14.90 (22.71)	178.40 (13.36)

Values in parenthesis are transformed values.

RESULTS AND DISCUSSION

Host-parasite interaction in terms of DI (%), PDI and AUDPC was measured and quantified for resistance to powdery mildew. F_2 generation of all crosses showed varying degree of resistance indicating the involvement of quantitative genes governing powdery mildew resistance. The present investigation revealed that VRP 22, VRPMR 9, VRP 343 and Arka Ajeet were resistant while parent VRP 16 was highly susceptible as evident from the score of DI, PDI and AUDPC (Table 1). The F_1's of VRP 16 × VRP 22 and VRP 16 × VRPMR 9 remained intermediate between their respective parents, for all three characters. However, in case of VRP 343 × Arka Ajeet the F_1 did not follow the intermediate pattern, which might be due to mutual cancellation of few gene effects. The above two crosses indicate that resistance is partially dominant over susceptibility. Among the segregating generations, the disease score for backcrosses and F_2 generation in VRP 16 × VRP 22 and VRP 16 × VRPMR 9 was much higher than VRP 343 × Arka Ajeet because in both the crosses the female parent (VRP 16) was highly susceptible. In case of AUDPC, a range of 74.09

(VRP 22) to 667.00 (VRP 16) indicate differential rate of development of disease on various genotypes.

The rate of disease development in resistant parent (VRP 22, VRPMR 9, VRP 343 and Arka Ajeet) was much lesser than susceptible parent (VRP 16). Similarly, all the six generations of VRP 343 × Arka Ajeet showed the same rate of disease development, i e very slow in the beginning and medium at maturity in contest to the susceptible parent VRP 16. However, in case of segregating generations of VRP 16 × VRP 22 and VRP 16 × VRPMR 9 the speed of disease development in P_1 (VRP 16) was very fast (Fig 1). On the contrary, rate of disease development was slow in P_2 (VRP 22 and VRPMR 9). Intermediate performance of rest of the generations (F_1, F_2, B_1 and B_2) was observed for disease development.

Powdery mildew resistance is a complex character and it depends upon genetic potential of host plant to resist the pathogen. Therefore, gene action study for powdery mildew resistance is of prime importance in peas to formulate breeding strategies for resistance breeding. In the present study, it was assumed that the genetics of DI, PDI and AUDPC implies the genetics of host-parasite interaction between different generations and powdery mildew fungus. Therefore, epistatic model (six parameter model) of Hayman (1958) has to be considered as evident from joint scaling test (Table 2). Among the major gene effects, both additive and dominance gene effects were found to be important in all the three crosses for expression of disease incidence. Predominance of dominance gene effect was observed in VRP 16 × VRP 22 and VRP 343 × Arka Ajeet. The relative magnitude of additive component was greater than dominance component in VRP 16 × VRPMR 9. Thus, both additive and dominance gene actions appeared to govern this trait. The presence of epistasis suggests that this trait is governed by more than one gene, which is in accordance with Sharma *et al.* (2012).

Both additive and dominance components were found to be involved in inheritance of PDI including epistasis. Among the additive and dominance components, the additive gene effects were predominant in VRP 16 × VRPMR 9. However, dominance gene action was relatively greater than additive in VRP 16 × VRP 22 and VRP 343 × Arka Ajeet. Thus, both additive and dominance gene actions were found equally important in control of this character. These findings are in agreement with Tyagi (1999) and Tyagi and Srivastava (2000) in pea. The significance of additive, dominance and epistasis components indicated its importance in control of AUDPC. The magnitude of additive gene effect was greater than dominance in VRP 343 × Arka Ajeet. However, dominance and epistasis were found predominant in VRP 16 × VRP 22 and VRP 16 × VRPMR 9. Duplicate type of epistasis was observed in VRP 16 × VRP 22 and VRP 16 × VRPMR 9. This kind of epistasis generally hinders the improvement through selection as the presence of duplicate epistasis decrease the variation in F_2 and subsequent

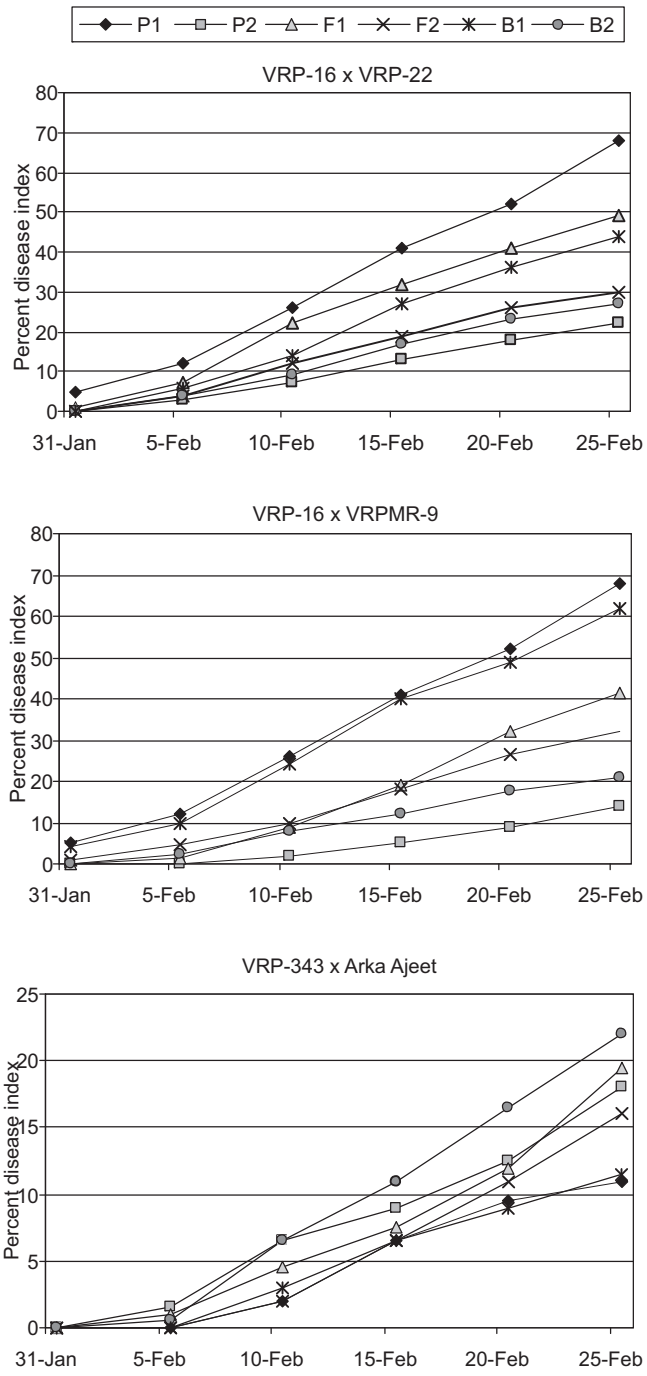

Fig 1 Powdery mildew disease progressive curve in three crosses of garden pea

generations (Tyagi and Srivastava 2001). Therefore, the selection should be delayed until a high level of gene fixation is attained (Tyagi and Srivastava 2001 and Singh *et al.* 2006). Thus, it is evident from present study that, AUDPC is controlled by both additive and dominance gene actions.

Complementary gene action, acts in favour of heterosis causes the increase of heterosis, and duplicates gene action,

Table 2 Estimate of gene effects for disease incidence per cent disease index and AUDPC

Cross		Disease incidence		Per cent disease index		Area under disease progress curve	
VRP 16 × VRP 22	m	34.760**	±0.577	29.530**	±0.577	16.353**	±0.035
	d	21.940**	±0.816	9.970**	±0.282	7.183**	±0.105
	h	27.645**	±2.831	–17.48**	±2.382	7.360**	±0.389
	i	22.960**	±2.828	–16.74**	±2.377	5.553**	±0.254
	j	3.750**	±1.653	–18.66**	±0.589	–2.840**	±0.625
	l	–22.370**	±4.008	49.68**	±2.587	–4.067**	±0.738
	X^2	108.2498**		3 826.52**		712.9607**	
VRP 16 × VRPMR 9	m	35.910**	±0.052	28.380**	±0.058	17.150**	±0.087
	d	27.720**	±0.577	9.120**	±0.294	8.360**	±0.078
	h	18.065**	±1.343	–8.060**	±0.637	3.972**	±0.499
	i	14.880**	±1.173	–5.760**	±0.632	3.040**	±0.380
	j	13.710**	±1.309	–19.120**	±0.606	–0.297	±0.606
	l	–17.010**	±2.663	31.680**	±1.209	–3.583**	±0.798
	X^2	2 100.6441**		6 937.13**		64.7625**	
VRP 343 × Arka Ajeet	m	26.920**	±0.046	25.620**	±0.012	11.920**	±0.046
	d	6.050**	±0.082	–3.160**	±0.578	–3.537**	±0.058
	h	–15.815**	±0.693	–11.760**	±1.161	0.530	±0.229
	i	–17.380**	±0.247	–17.960**	±1.157	–1.313**	±0.218
	j	7.030**	±0.611	1.380 ±1.165	–5.387**	±0.117	
	l	23.150**	±1.349	10.240**	±2.320	3.740**	±0.327
	X^2	5 112.805**		47 132.354**		2 159.132**	

*, ** Significant at 5% and 1% level, respectively

which acts against the heterosis, causes decrease of heterosis (Mather and Jinks 1982). In this study, the dominance (h) and dominance × dominance (l) components had opposite signs in all the crosses indicating the presence of the duplicate type of epistasis. This will reduce the net gain occurring from heterozygosity due to the cancellation of dominance and epistatic effects. In such crosses, the selection intensity should be mild in early and intense in the later generations because it marks the progress through selection (Sharma and Sain 2002).

All the crosses examined in the present study have shown complex genetic behavior. The simple selection procedure in the early segregating generation may not significantly contribute for the improvement of this trait. The complex genetic behavior particularly additive and dominance components could be successfully exploited in later generation. Therefore, breeders should follow relevant method which can accumulate the resistance genes for powdery mildew resistance in one genotype (Singh *et al.* 2008). Though, both the gene actions were involved in control of above three characters, additive and/or additive based interactions were found important in addition to duplicate type of epistasis in VRP 16 × VRPMR 9, dominance and epistatic components were found important in VRP 16 × VRP 22 and VRP 343 × Arka Ajeet. Therefore, selection may be delayed in the case of VRP 16 × VRP 22 and VRP 343 × Arka Ajeet. As far as the importance of additive gene action is concerned in VRP 16 × VRPMR 9, selection may also be postponed due to the presence of epistasis while handling segregating material. On the contrary, both additive and dominance gene effects may be exploited by intermating the plants isolated from early generations in segregating population in order to accumulate favourable genes (Singh *et al.* 2008). This system may ensure full utilization of both additive and dominance gene effects and eventually lead to fixation of the resistance at the desired level.

REFERENCES

Gritton E T and Ebert R D. 1975. Interaction of planting date and powdery mildew on pea performance. *Journal of Amercian Society of Horticultural Science* **100**: 137–42.

Hagedorn D J. 1985. *The Pea Crop*, pp 205–13. Heath M C and Dawkins T C K (Eds). A Butterworths Publication, London.

Hayman B I. 1958. Separation of epistasis from additive and dominance variation in generation means. *Heredity* **12**: 371–90.

Mather K and Jinks J L. 1982. *Biometrical Genetics,* 3rd edn. Chapman and Hall Ltd, London.

Mayee C D and Datar V V. 1986. Phytopathometry. Marathwada Agricultural University, Parbhani, Maharashtra, p 146.

Munjal R L, Chenulu V V and Hora T S. 1963. Assessment of losses due to powdery mildew (*Erysiphe polygoni*) on pea. *Indian Phytopathology* **19:** 260–7.

Reiling T P. 1984. Powdery mildew. *Compendium of Pea Diseases*, pp 21–2. Hagedorn D J (Eds). American Phytopathology Society, St. Paul, MN.

Sharma S N and Sain R S. 2002. Inheritence of tillers for plant in

durum wheat (*Triticum durum* Desf). *Indian Journal of Genetics* **62**(2): 101–3.

Sharma Akhilesh, Kapur Pooja and Katoch Viveka. 2012. Generation mean analysis to estimate genetic parameters for desirable horticultural traits in garden pea (*Pisum sativum* L). *Indian Journal of Agricultural Sciences* **82**(3): 201–6.

Singh H, Singh M, Singh S and Brar P S. 2006. Estimation of gene effects of yield and quality characters in pea (*Pisum sativum* L). *Environment and Ecology* **24**(2): 445–8.

Singh P, Bhadauria Anju and Singh P K. 2008. Combining ability and gene action for alternaria blight and powdery mildew resistance in linseed. *Indian Journal of Genetics and Plant Breeding* **68**(1): 65–7.

Smith P H, Foster E M, Boyd L A and Brown K M. 1996. The early development of *Erysiphe pisi* in *Pisum sativum* L. *Plant Pathology* **45:** 302–9.

Tyagi M K and Srivastava C P. 2001. Analysis of gene effects in pea. *Legume Research* **24**(2): 71–6.

Tyagi M K and Srivastava C P. 2000. Inheritance of powdery mildew and rust resistance in pea. *Annals of Biology* **15:** 13–6.

Tyagi M K. 1999. Generation mean analysis of adult plant resistance to powdery mildew and rust disease in field pea. *Plant Disease Research* **14**: 171–4

Van Der Plank J E. 1963. *Plant Disease Epidemics and Control*, pp 349. Academic Press, New York.

Warkentin T D, Rashid K Y and Xue A G. 1996. Fungicidal control of powdery mildew in field pea. *Canadian Journal of Plant Science* **76**: 933– 5.

Wheerler B E J. 1969. *An Introduction to Plant Diseases*, pp 300–9. John Wiley & Sons Ltd, London.