Indian J. Genet., **61**(2): 129-131 (2001)

Inheritance of powdery mildew resistance in pea (*Pisum sativum* L.)

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(Received: December 1999; Revised: February 2001; Accepted: March 2001)

Abstract

The inheritance of powdery mildew resistance in pea was studied in 10 crosses, involving 16 different parents. The parents, F_1s and F_2s were grown along with susceptible line L 116 as spreader rows. Natural epidemic in late sown pea crop at Delhi conditions was used to do disease screening. The inheritance pattern studied with χ^2 test suggested that the resistance to powdery mildew was monogenic recessive.

Key words: Pea, powdery mildew, resistance, inheritance.

Introduction

Powdery mildew caused by the ascomycete *Erysiphe pisi* D. C. ex. St-Am can cause severe damage to pea, often becoming epidemic in nature. In early seventies when this disease became a wide spread phenomenon, virtually, this deadly disease engulfed entire country. Consequently, pea area was almost reduced by half [1]. Although fungicidal control is available as an alternative, genetic resistance is preferred because it is more sustainable and cost effective means of disease control.

Hammerlund was first to discover resistance to pea powdery mildew [2]. Since then contradictory reports started pouring in regarding of the inheritance of resistance. A single recessive gene governing resistance to powdery mildew was reported [3]. This was supported by many workers [4, 5, 6]. In contrary to this, involvement of two or more genes were reported by [2, 7, 8]. A recent report [9] also indicated the involvement of two genes. In this context, we have undertaken the present investigation to study the inheritance of powdery mildew resistance using 16 diverse parents in 10 crosses.

Materials and methods

The present study comprised of sixteen parental lines, listed in Table 1 along with their pedigree, source and powdery mildew reaction. The parents used in this study were from different sources and have wide genetic background as given in Table 1. The list of 10 crosses and their parentage are presented in Table 2. Crossing work was undertaken in 1996-97 rabi season at IARI, New Delhi. The failed crosses and in some cases to have good number of F_1 seeds, those crosses were repeated at Directorate of Wheat Research, Summer Wheat Nursery, Dalang Maidan. Lahaul Valley in H.P. in 1997 (May to September). The F_1 were sown in rabi 1997-1998 at IARI, New Delhi to harvest the F_2 seeds and also confirm the phenotype of the crosses. In rabi 1998-1999, the seeds of all F_2, F_1 and parents were sown late in the season (December 10, 1998) at IARI, New Delhi.

Individual F_2 plants of different populations were recorded for their powdery mildew reaction and classified as either PMR (powdery mildew resistant) or PMS (powdery mildew susceptible). Similar observations were also recorded on F_1 and parents. The crop was deliberately sown late to take the advantage of natural epidemic of powdery mildew in pea crop at Delhi conditions. The field was frequently irrigated to keep the plants vegetatively growing to facilitate the disease development. The susceptible spreader rows of L 116 upon primary infection by fungus produced large number of conidia, which served as source of secondary infection and disease spread, finally the disease took an epidemic proportion. Misclassification of genotypes can drastically alter the observed ratio, thus, leading to wrong conclusions [10]. Hence, classification of F_2 genotypes as PMR or PMS is very important. Keeping this in view enough care was taken to see that all the plants receive the inoculum and that the disease development is 100% on susceptible genotypes. Thus, we could classify PMS and PMR plants without and ambiguity.

The χ^2 test for goodness of fit for 3:1 ratio was done using the formulae [10]. If the calculated χ^2 value for 3:1 ratio is non-significant at 1 d.f., it suggests monogenic inheritance. The calculated heterogeneity

Table 1. List of pea strains used in the investigation

S.No.	Name of line	pedigree/ parent line/variety	Source/origin	Powdery mildew reaction
1.	P1746	Wt11777	Weibullsholm Plant Breeding Institute, Sweden	PMR
2.	MD-1-24 (P 1828)	MD-1-24	IARI, New Delhi	PMS
3.	P 1744	Wt 10345	Weibullsholm Plant Breeding Institute, Sweden	PMS
4.	P 1760 (*nana* dwarf)	L 179	I.C. Murfet Tasmania, Australia	PMR
5.	P 1743	Wt. 10102	Weibullsholm plant Breeding Institute, Sweden	PMS
6.	HFP 4	T163 × EC 190196	HAU, Hisar	PMR
7.	P1881	SK 25	Delhi University	PMS
8.	P 1757	NGB 754	Mike Ambrose John Innes Institute, U.K.	
9.	P 1442	IC 37255	Introduction from Sikkim	PMR
10.	PG 3	T 163 × Boneville	PAU, Ludhiana	PMS
11.	P 1746-8-1	Wt. 11777	Weibullsholm Plant Breeding Institute, Sweden	PMR
12.	Pusa 10	Early Suberb × L 993	IARI, New Delhi	PMS
13.	P 1779-4	F4-716-3-2-1 0	IARI, New Delhi	PMR
14.	P 1746-24-1	Wt. 11777	Weibullsholm Plant Breeding Institute, Sweden	PMR
15.	P 1746-1-1	Wt 1777	-do-	PMS
16.	P 1744-1	Wt 10345	-do-	PMS

Note: PMR = powdery mildew resistant, PMS = powdery mildew

Table 2. Crosses made for mapping of morphological markers of chromosome VI

S. No.	Cross identification	Parentage
1.	PC er 51	P 1746 × D 1-24
2.	PC er 6	P 1744-1× P 1760 (nana dwarf)
3.	PC 398	P 1743 × HFP4
4.	PC 439	HFP 4 × P 1881
5.	PC 435	P 1744 × P 1757
6.	PC 400	P 1442 × PG3
7.	PC 436	P 1446-8-1 × Pusa 10
8.	PC 441	Pusa 10 × P 1760 (nana dwarf)
9.	PC 437	P 1746-24-1 × P1746-1-1
10.	PC 15J	P 1779-4 × P 1760 (nana dwarf)

χ^2 was non-significant, hence pooled analysis over ten crosses was also done.

Results and discussion

Powdery mildew develops as white floury specks on both susceptible and resistant genotypes. However, the pods were never infected and stems too were very rarely infected in resistant types. Tissue necrosis beneath the fungal growth was also not observed in resistant genotypes. Hence, there was absolutely no ambiguity in identifying PMS and PMR plants, when screening was done under such heavy infection.

In all the 10 crosses, the F_1s were susceptible like their susceptible parents indicating dominance of susceptible type over resistant type. To test the goodness of fit of the observed segregation with that of expected, the χ^2 values for 3:1 ratio were calculated. The number of PMS, PMR and total plants in each of the 10 crosses and their χ^2 values along with their probability levels (P) for calculated χ^2 at 1 d. f. (3:1 ratio) are given in Table 3 along with the χ^2 values for pooled data over all the crosses with I d.f and heterogeneity with 9 d.f.

In all the 10 crosses the χ^2 calculated was non-significant, indicating segregation F_2 plants into 3 PMS: fPMR type. The heterogeneity χ^2 was found to be non-significant indicating homogeneity among crosses. Thus, PMS and PMR plants were pooled over 10 crosses. The F_2 plants segregated into 2356 PMS: 779 PMR as against 2351 PMS: 784 PMR on the expectation of 3:1 segregation pattern. The pooled χ^2 for 3:1 ratio, which was 0.38, was again non-

Table 3. F_2 segregation for powdery mildew resistance in different crosses of pea

Parents	F_2 segregation			χ^2	P
Female/Male	S	R	Total	(3:1)	(1d.f.)
P 1746/MD 1-24	170	53	223	0.18	0.50
P 1744/P 1760	120	43	163	0.16	0.50
P 1743/HFP 4	310	102	412	0.01	0.90
HFP 4/P1881	281	95	376	0.01	0.90
P 1744/P1757	163	56	219	0.03	0.90
P 1742/PG3	637	208	845	0.06	0.80
P 1746-8-1/Pusa 10	226	75	301	0.00	0.95
P 1760/Pusa 10	188	65	253	0.06	0.80
P 1746/P 1746-1-1	98	30	128	0.16	0.50
P 1773-4/P 1760	163	52	215	0.07	0.70
Pooled analysis	2356	779	3135	0.03	0.90
Heterogenecity χ^2 (9 d.f.)				0.66	>0.99

significant for 1 d.f. with the probability of 0.90. From this, it was concluded that the resistance to powdery mildew in pea was under the control of single recessive gene, named as *er* (*Erysiphe* resistance).

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