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Inheritance of powdery mildew (*Erysiphe polygoni* DC) resistance in mungbean (*Vigna radiata* L. Wilczek)

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Abstract Detached mungbean (*Vigna radiata* L.Wilczek) leaves were inoculated with a conidial suspension of a local isolate (TI-1) of the powdery mildew pathogen (*Erysiphe polygoni* DC) under controlled environment conditions. Based on the latent period and severity of the infection, a rating scale of 0–5 was used to classify the host pathogen interactions. Reactions 0, 1 and 2 were considered resistant and referred to as R0, R1 and R2 while 3, 4 and 5 were classified as susceptible (S). RUM lines (resistant to powdery mildew) and their derivatives are crossed with several susceptible (reaction types 3–5) genotypes and the inheritance of the resistance was studied in the F_1, F_2 and F_3 generations. The results showed that powdery mildew resistance in mungbean is governed by two dominant genes designated as *Pm-1* and *Pm-2*. When both *Pm-1* and *Pm-2* were present, an R0 reaction was observed after inoculation with TI-1. The resistant reaction was R1 when only *Pm-1* was present and R2 in the presence of *Pm-2*. In the absence of both *Pm-1* and *Pm-2*, susceptible reactions 3, 4 and 5 were observed.

Key words Mungbean · *Erysiphe polygoni* · Inheritance Powdery mildew · Resistance

Introduction

Powdery mildew (*Erysiphe polygoni*) is a common disease of mungbean crops in South East Asia (Park and Yang 1978). It is prevalent in southern and central India (Grewal 1978). The yield losses due to powdery mildew disease in mungbean are reported to be 20–40% (Soria and Quebral 1973; Legaspi et al. 1978; Fernandez and Shanmugasundaram 1987). However, we have observed complete loss of the crop when the disease occurs at the seedling stage. Powdery mildew is also one of the important diseases in barley, wheat, oats, peas, cowpea and cucurbits, causing considerable losses. Biffen (1907) first reported the inheritance of powdery mildew resistance in barley. Since then it has become one of the most thoroughly-investigated host-pathogen interactions and several comprehensive reviews are available (Moseman 1966; Wiberg 1974 a, b; Giese 1981). Powdery mildew resistance has been reported in wheat (Schneider et al. 1991), oats (Moseman 1966), peas (Heringa et al. 1969) beans (Meiners 1981), red clover (Moseman 1966), cabbage (Walker and Williams 1965), cucumber (Moseman 1966) and sunflower (Mccreight et al. 1987) One, two, or more, dominant, incompletely dominant, or recessive, genes governing the resistance have been identified in different crops. A large number of genes conferring resistance have been identified in barley and wheat (Giese 1981; Giese et al. 1981; Heum et al. 1990).

Powdery mildew resistance in mungbean was reported to be predominantly controlled by loci with additive gene effects (Yohe and Poehlman 1975). Monogenic dominant resistance was inferred in ML-3 and ML-5 resistant genotypes (AVRDC 1978). However, these cultivars were found susceptible to the races used in our studies (Reddy et al. 1987). RUM lines, used as sources of resistance to powdery mildew in the present study, were identified in germplasm screened under controlled environmental conditions (Reddy et al. 1987). They showed a high degree of resistance both in the field, at several locations, and under a controlled environment. The mode of inheritance of powdery mildew resistance in mungbean is reported in this paper.

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Materials and methods

Isolation of *Erysiphe polygoni*

The pathogen was isolated from infected leaves of TPM-1 collected from the experimental field at Trombay. Conidia from infected leaves were dusted on the seedlings of TPM-1 grown in pots in a growth chamber maintained at 21°C. Subsequently the fungus was isolated from a single sporulated lesion on infected leaves and maintained on detached leaves (Reddy et al. 1987).

Culture of excised leaves for disease development studies

Fully-expanded third trifoliate leaves were excised from 25-day-old field-grown plants of parents, F_1, F_2 and F_3 populations. They were inoculated by spraying with a spore suspension (3.5×10^6 conidia /ml water). The leaves were kept in plastic trays in controlled environmental chambers maintained at 21°C and 12 h/day illumination of 4,136 lux/m^2 with fluorescent white lights, as described previously (Reddy et al. 1987).

Disease rating scale

As no standard scale was available for scoring the incidence of powdery mildew on mungbean, observations were made on the latent period of the pathogen on different genotypes and on the leaf area infected. Initially, the percent leaf area infected was recorded on the basis of visual observations followed by actual measurements using a Hiyashi-Denko leaf-area meter. The visual scores showed a correlation of 0.9 with the actual measurements. A numerical rating scale of 0–5 (Table 1), based on the latent period of the pathogen and the leaf area infected, scored visually, was used for screening the disease reaction.

Table 1 Rating scale for powdery mildew reaction on mungbean

Latent period (days)	% Leaf area infected 20 days after inoculation	Score
10	91–100	5, highly susceptible (HS)
	66– 90	4, susceptible (S)
	31– 65	3, moderately susceptible (MS)
15	6– 30	2, resistant (R2)
20	1– 5	1, resistant (R1)
20	0	0, highly resistant (R0)

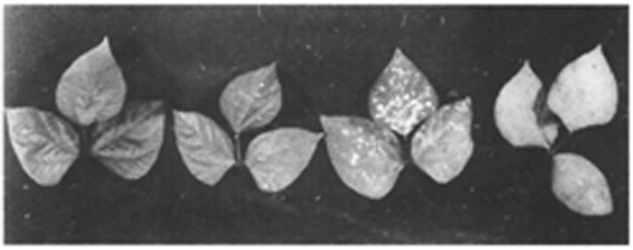

Fig. 1 Excised leaves of mungbean showing resistant R0, R1, R2 and S reactions (left to right).This classification was used for screening segregating population for inheritance studies

Results

Four types of disease reactions, as shown in Fig. 1, were observed on mungbean genotypes. Disease incidence on detached leaves and in the field were scored on the basis of the visual observation of the percent leaf area infected as shown in Fig. 2. A rating scale (Table 1) was developed based on the latent period of the pathogen and the percent leaf area infected 20 days after inoculation (DAI). For inheritance studies, 0, 1 and 2 scores were considered as resistant, whereas 3, 4 and 5 were scored as susceptible types. For convenience the resistant reactions were classified as R0, R1 and R2. In the R0 reaction, no disease symptoms were observed even after 20 DAI. In R1, though no disease symptoms were seen until 15 DAI, small disease lesions appeared at 20 DAI covering less than 5% of the leaf area. In R2, the leaves had no visual lesions upto 10 DAI but thereafter about 10% of the leaf area was covered by the pathogen at 15 DAI, which increased to 20% at 20 DAI. In the susceptible (S) reactions over 50% of the leaf area was infected at 10 DAI and over 75% at 20 DAI.

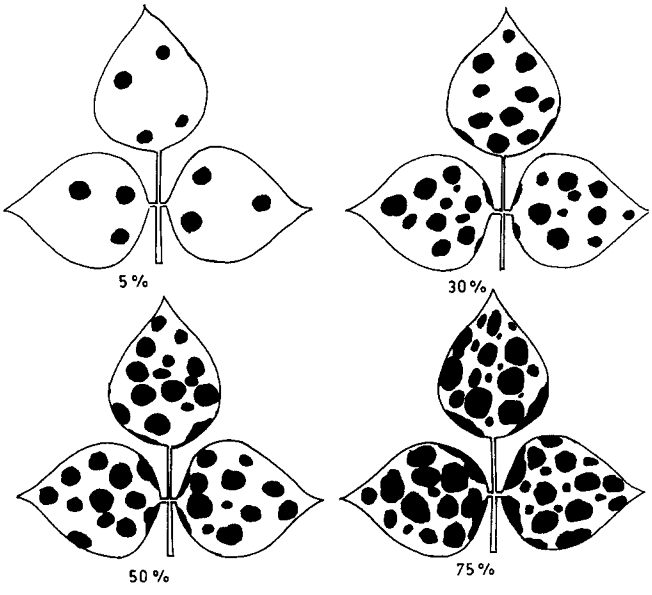

Fig. 2 Standard leaf diagrams of mungbean for assessing powdery mildew disease leaves showing 5, 30, 50 and 75% leaf area infected

Inheritance of powdery mildew resistance

RUM-5 and RUM-22, earlier identified as resistant with an R0 reaction, and TARM-2, a high-yielding resistant selection derived from a cross between RUM-5 and TPM-1, were crossed to a number of susceptible types (TPM-1, VM-202, TAP-7, Kopergaon, K-851 PDM-54 and PDM-116). All the F_1 plants showed an R0 reaction while the F_2 segregated into R0, R1, R2 and S types according to a dihybrid ratio of 9:3:3:1 (Table 2) in all the crosses. This indicated the presence of two dominant resistance genes which have been designated as *Pm-1* and *Pm-2*. When both these genes are present the plants show an R0 reaction. R1

Table 2 Segregation for powdery mildew resistance in the F_2 generation of crosses between resistant lines and susceptible genotypes

Crosses	F_1 plants (no.)[a]	F_2 plants (no.)	Reaction of F_2 plants				χ^2 (9 : 3 : 3 : 1)	*P* value
			R0 (no.)	R1 (no.)	R2 (no.)	S (no.)		
R0×S								
RUM-5×TPM-1	4(R0)	462	260	78	94	30	1.53	50– 70
TPM-1×RUM-5	2(R0)	204	116	35	42	11	0.89	80– 90
VM-202×RUM-22	3(R0)	285	156	49	62	18	1.86	50– 70
TAP-7×RUM-5	4(R0)	186	106	29	37	14	1.62	50– 70
KOPERGAON×TARM-2	7(R0)	450	257	79	88	26	0.72	80– 90
K-851×TARM-2	3(R0)	232	129	42	47	14	0.37	90– 95
PDM-54×TARM-2	5(R0)	442	246	83	87	26	0.33	80– 90
PDM-116×TARM-2	7(R0)	413	234	69	82	28	1.38	50– 70
R1×R2								
S-158-16×S-2-4-1	3(R0)	204	113	38	40	13	0.11	99–100
R1×S								
S-158-16×TPM-1	2(R1)	115	0	83	0	32	0.48[b]	30– 50
R2×S								
S-2-4-1×TPM-1	4(R2)	94	0	0	71	23	0.01[b]	90– 95

[a] Reaction of F_1 plants is given in parentheses
[b] χ^2 for 3 : 1 ratio

Table 3 Segregation for powdery mildew resistance in the F_3 progenies of crosses between resistant and susceptible genotypes

F_2 reaction	F_3 segregation	Number of F_3 progenies from different crosses			
		R0×S	R1×R2	R1×S	R2×S
R0	R0 (homozygous)	32	3	0	0
	R0, R1 (seg.)	60	7	0	0
	R0, R2 (seg.)	68	6	0	0
	R0, R1, R2, S (seg.)	133	15	0	0
R1	R1 (homozygous)	28	3	18	0
	R1, S (seg.)	58	7	38	0
R2	R2 (homozygous)	41	4	0	9
	R2, S (seg.)	70	8	0	15
S	S (homozygous)	33	3	20	7
Total		523	56	66	31

χ^2 for 1 : 2 : 2 : 4 : 1 : 2 : 1 : 2 : 1 ratio in the cross R0×S = 4.5
χ^2 for 1 : 2 : 2 : 4 : 1 : 2 : 1 : 2 : 1 ratio in the cross R1×R2 = 0.64
χ^2 for 1 : 2 : 1 ratio in the cross R1×S = 0.10
χ^2 for 1 : 2 : 1 ratio in the cross R2×S = 0.29

and R2 reactions are observed when *Pm-1* and *Pm-2* are present individually.

In the F_3 generation, progenies of all 33 susceptible F_2 plants were true breeding for the susceptible reaction while those showing R0, R1, and R2 reactions were either true breeding resistant or else segregated as expected (Table 3). The pooled segregation showed a good fit to the expected ratios of 1:2:2:4:1:2:1:2:1 and 1:2:1. Segregation within the families was as expected for digenic or monogenic ratios.

S-158-16, showing an R1 (*Pm-1 Pm1*) reaction, and S-2-4-1, showing an R2 (*Pm-2 Pm2*) reaction, were crossed with the susceptible genotype TPM-1. The F_1 plants showed an R1 and an R2 reaction respectively. The two F_2 populations segregated in a 3:1 ratio (Table 2). The F_3 families segregated in a ratio of 1 resistant: 2 segregating: 1 susceptible (Table 3). The monogenic dominant nature of *Pm1* and *Pm2* was thus confirmed.

S-158-16 showing R1 reaction was also crossed with S-2-4-1. The F_1 plants showed an R0 reaction while the F_2 population segregated into R0, R1, R2 and S types (Table 2). The F_3 families segregated in a ratio of 1:2:2:4:1:2:1:2:1 This again confirmed that the presence of both *Pm-1* and *Pm-2* gives an R0 resistant reaction.

Discussion

Very little information on powdery mildew resistance in mungbean was available in the literature when the present studies were initiated. Genotypic differences in the severity of infection (percent leaf area infected; the latent period of the pathogen; days for appearance of visual symptoms, namely pustules) have been observed. The above parameters are widely used as a standard practice for assessment of fungal disease reactions (Johnson 1984). They have been employed for assessing reaction to the powdery mildew of cereals (Large 1966) and peas (Pal et al. 1980; Reeser and Hagedorn 1981). Based on the above parameters, a rating scale of 0 – 5 was developed for the powdery mildew of mungbean (Table 1).

RUM lines 5 and 22, and their derivatives investigated in crosses with several susceptible genotypes, indicated that powdery mildew resistance was controlled by two dominant genes (*Pm-1* and *Pm-2*). These lines can be distinguished by host-pathogen interaction phenotypes described as R1 and R2 respectively. Prior to this study, powdery mildew resistance in mungbean was reported to be controlled predominantly by loci with additive gene effects (Yohe and Poehlman 1975).

In the initial experiments, when the F_2 population was classified into only two groups, resistant (R0 and R1) and susceptible (R2 and S) types also showed monogenic dominant inheritance of the resistance trait. With the use of a proper rating scale, R1 and R2 reactions could be individually identified. Crosses between the genotypes showing R1 and R2 reactions with susceptible cultures demonstrated that the genes *Pm-1* and *Pm-2* are independent. Extensive studies on powdery mildew resistance in barley (Jorgensen 1977; Giese 1981; Hossain and Sparrow 1991) and wheat (Reader and Miller 1991; Schneider et al. 1991) show that there are many genes for resistance. In peas only four recessive genes have been reported (van Harmmarlund 1925). There may be additional genes for powdery mildew resistance in mungbean other than the two identified in the present study. The methods reported in this paper will be useful in identifying new resistance genes and developing powdery-mildew-resistant cultivars on a rational basis in this important crop of South East Asia.

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