

Genetics of resistance to P₃ isolate of *Phytophthora* blight in pigeonpea

A.K. Gupta¹, I.S. Singh^{1,*}, M.V. Reddy² & G.C. Bajpai¹

¹ Department of Genetics and Plant Breeding, G.B. Pant University of Agriculture and Technology, Pantnagar – 263 145, India; ² Crop Protection Division, ICRISAT Asia Centre, Patancheru – 502 324, India; (* author for correspondence)

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Summary

Seedlings from six crosses between susceptible × resistant and two between susceptible parents were screened against P₃ isolate of *Phytophthora drechsleri* f. sp. *cajani* in the glasshouses. Disease reaction was scored on a rating scale of 1–5. Resistance was found to be controlled by one dominant gene and some minor genes and is affected by ontogeny. The resistance gene appeared to be different from the one reported earlier (Pd₁) and is designated here as Pd₃.

Introduction

Phytophthora blight (PB) first recognised by Williams et al. (1968) and caused by *Phytophthora drechsleri* Tucker var. *cajani* Pal, Grewal, and Sarbhoy leads to a drastic yield reduction in short-duration pigeonpeas (Reddy et al., 1990) and of fungal pathogens it is second only to fusarium wilt in incidence (Kannaiyan et al., 1984). At present, no early maturing cultivar is known to have resistance/tolerance to this disease. The possibility of occurrence of more than one race(s) of PB has been indicated Sharma et al. (1982) among others. Subsequently the presence of different isolates of the pathogen has been confirmed by Reddy et al. (1990).

The presence of a new and virulent P₃ isolate and its severity on pigeonpea prompted us to determine the number of gene(s) in a resistant line (KPBR 80-2-1).

Materials and methods

The present research work was conducted at Pantnagar and ICRISAT Asia Centre (IAC), Patancheru, India. The F₁s were developed at Pantnagar and IAC while

final screening of test material was done exclusively in the glasshouse at IAC.

Parents and crosses

Six PB susceptible early maturing pigeonpea lines viz., UPAS 120, Pant A3, ICPL 84023, ICPL 87119, ICPL 90005 and ICPL 90035 were selected. A single late maturing line (KPBR 80-2-1) having field resistance to PB was used as resistant parent.

All six susceptible lines were crossed with the resistant line, and two crosses between susceptible parents (UPAS 120 × ICPL 84023 and Pant A3 × ICPL 84023) were made. Since there were huge differences in flowering time of the susceptible and the resistant parents a ratoon crop of the susceptible parents was used for synchronising flowering time. The F₁ plants were selfed with muslin cloth bags to prevent outcrossing. The F₁s were also back-crossed to both parents (P₁ and P₂) to obtain BC₁P₁ and BC₁P₂, respectively.

Pathogen isolate

The pathogen isolate was isolated from a small piece of stem 3 mm in length including portions of lesions on healthy tissues. The stem piece was washed in run-

Table 1. Seedling reaction of parents, F₁s, F₂s and their backcrosses to P₃ isolate of *Phytophthora drechsleri* f. sp. *cajani* in eight crosses of pigeonpea

Parents and crosses	Generations	Observed number of plants		Expected ratio, tested	χ^2 value
		Resistant	Susceptible		
UPAS 120		12	181	–	–
Pant A3		16	169	–	–
ICPL 84023		15	171	–	–
ICPL 87119		21	168	–	–
ICPL 90005		11	152	–	–
ICPL 90035		10	154	–	–
KPBR 80-2-1		183	12	–	–
UPAS 120 × KPBR 80-2-1	F ₁	12	0	–	–
	F ₂	308	82	3:1	3.285
	BC ₁ P ₁	7	5	1:1	0.333
	BC ₁ P ₂	14	0	–	–
Pant A3 × KPBR 80-2-1	F ₁	13	0	–	–
	F ₂	286	101	3:1	0.249
	BC ₁ P ₁	19	23	1:1	0.381
	BC ₁ P ₂	12	0	–	–
ICPL84023 × KPBR 80-2-1	F ₁	20	0	–	–
	F ₂	273	93	3:1	0.033
	BC ₁ P ₁	10	7	1:1	0.529
	BC ₁ P ₂	15	1	–	–
ICPL87119 × KPBR 80-2-1	F ₁	18	0	–	–
	F ₂	292	81	3:1	2.146
	BC ₁ P ₁	11	14	1:1	0.360
	BC ₁ P ₂	14	0	–	–
ICPL90005 × KPBR 80-2-1	F ₁	16	2	–	–
	F ₂	293	86	3:1	1.077
	BC ₁ P ₁	10	8	1:1	0.222
	BC ₁ P ₂	17	1	–	–
ICPL90035 × KPBR 80-2-1	F ₁	8	1	–	–
	F ₂	275	102	3:1	0.850
	BC ₁ P ₁	11	8	1:1	0.474
	BC ₁ P ₂	18	0	–	–
UPAS 120 × ICPL84023	F ₁	2	11	–	–
	F ₂	16	361	–	–
	BC ₁ P ₁	3	15	–	–
	BC ₁ P ₂	4	14	–	–
Pant A3 × ICPL84023	F ₁	4	34	–	–
	F ₂	26	346	–	–
	BC ₁ P ₁	5	14	–	–
	BC ₁ P ₂	3	9	–	–

BC₁P₁ = F₁ × P₁; BC₁P₂ = F₁ × P₂.

ning tap water and surface-sterilized in 2% sodium hypochlorite solution for 1–3 minutes and placed on potato dextrose agar (PDA) slants. On the basis of growth characteristics, slants with the fungus in pure form were identified and confirmed by microscopic examination. In virulence test it was confirmed as P₃ isolate because seedlings of ICP 7119 (susceptible to P₂ and P₃) and ICP 2376 (resistant to P₂ but susceptible to P₃) were killed.

Inoculation technique

After extensive testing of inoculation technique (Drench- vs. spray-inoculation), inoculum concentration, plant age, humidity period, humidity percentage and temperature through a series of experiments, it was found that optimum inoculations resulted with 12-day-old seedlings with one trifoliolate leaf, a temperature range of 25–30 °C at 100% humidity for 36 hrs and an inoculum concentration of 1 g of mycelium/100 ml of water (Gupta, 1995).

Preparation of inoculum

The fungal isolate was grown on solid sterile medium in Petri dishes on v-8 juice agar (v-8 juice, 100 ml; CaCO₃, 2 g; agar, 20 g; and distilled water, 900 ml) [Riberio, 1978]. Then single 5 mm disks of a week-old culture were transferred to v-8 juice broth and incubated at 25–30 °C for 15 days. The mycelial mats were removed, weighed and macerated with a small amount of water in a waring blender. This suspension was diluted to a final concentration of 1 g of mycelium/100 ml of deionized water.

Plant culture and screening procedure

Up to 10 seeds were planted in a glasshouse in plastic pots (15 cm diameter) filled with natural red soil. Seedlings were inoculated using an automizer and the technique described by Nene et al. (1981).

Observational procedure

Observations were recorded, after 10 days of inoculation, on a rating scale of 1–5 for assessing overall disease reaction. The seedlings were classified as resistant (surviving) with infection scores from 1 to 4 and a fully susceptible score of 5 (killed).

Statistical analysis

Simple χ^2 test as described by Snedecor & Cochran (1980) was used to test the goodness of fit of expected segregation ratios.

Results and discussion

Disease reactions of the seedlings of the parents, F₁s, F₂s and back crosses to P₃ isolate of PB are given in Table 1. Nearly 90% of plants were killed in each of the susceptible parents (P₁) verifying the virulence of P₃ isolate while more than 90% plants survived in the resistant line. In the two crosses between susceptible parents, plants from all generations studied were susceptible. Virtually all the F₁s between susceptible and resistant parents were resistant except for 2 of 18 plants in ICPL 90005 × KPBR 80-2-1 and 1 of 9 plants in ICPL 90035 × KPBR 80-2-1. Thus resistance to the P₃ isolate was completely dominant over susceptibility. Dominance of resistance was further confirmed by the segregation ratio in the backcrosses to the resistant parent (BC₁P₂). However, again one susceptible plant was observed in BC₁P₂ of each of the two crosses (ICPL 84023 × KPBR 80-2-1, ICPL 90005 × KPBR 80-2-1). All six crosses segregated 3:1 resistant:susceptible in the F₂ populations and 1:1 in BC₁P₁ (Table 1). Sharma et al. (1982) reported the monogenic dominant control of resistance to P₂ isolate of PB but, one of their resistant parents (Pant A3) was susceptible to the P₃ isolate of PB in the present investigation. Therefore, the resistance gene in KPBR 80-2-1 must be different from that reported earlier (Pd₁) and so here it is designated as Pd₃. Not all the surviving plants were fully resistant and gave scores ranging from 1–4. About 6% of plants were susceptible. Such variation in the disease reaction among the surviving plants indicate that the gene for resistance to PB either had variable penetrance or some minor genes are involved in controlling the resistance. Gene Pd₃ has been found to be stable due to its resistant reaction to PB at several locations (Mishra & Shukla, 1986; Amin et al., 1993).

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