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Identification of resistant sources and inheritance of *Fusarium* wilt resistance in garden pea (*Pisum sativum* ssp. *hortense*)

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ABSTRACT

Wilt caused by *Fusarium oxysporum* Schlechtend.Fr. f.sp. *pisi* is a devastating disease of garden pea in India and worldwide. The pathogen being soil-borne fungus is quite difficult to manage by chemical methods and the development of resistant varieties seems to be the only alternative. In the present study 34 garden pea genotypes collected from different sources were evaluated during winter 2012-13 and 2013-14 under wilt sick plot as well as artificial controlled conditions to identify genetic sources of resistance against *Fusarium* wilt. Three genotypes (GP-6, GP-55 and GP-942) were found to be highly resistant, four genotypes (GP-17, GP-48, GP-473, GP-941) were resistant and identified as new donor source. All the popular cultivated varieties (Arkel, Pusa Pragati, AP-3, VRP-6, VL-7, VL-10 and Arka Ajit) were highly susceptible to wilt. The inheritance of *Fusarium* resistance gene(s) was also studied using F_1, F_2, B_1 and B_2 progenies of three crosses, *viz*., Pusa Pragati × GP-55, Pusa Pragati × GP-6 and Arkel × GP-17. Chi-square analysis was performed to determine the Mendelian segregation ratios of resistance and susceptibility among the inoculated progenies. The F_1 and backcross progenies to the resistant parents segregated in the ratio of 1:0, while the F_2 progenies segregated in the ratio of 3:1. The backcross progenies to the susceptible parent segregated in the ratio of 1:1. The results obtained revealed that the resistance to *Fusarium* wilt in garden pea is governed by monogenic dominant gene. The resistance gene can easily be introgressed by backcross breeding in desired genotype.

Key words: *Pisum sativum* ssp. *hortense*, *Fusarium oxysporum* f.sp. *pisi*, screening, inheritance, monogenic dominant resistance.

INTRODUCTION

Garden pea (*Pisum sativum* L. ssp. *hortense* (Neilr.) Asch. & Graebn.) is an annual herbaceous legume vegetable of family Fabaceae. The crop is normally grown in the field during mid-October to November in northern plains of the country. Early sown crops fetch high profit but the crop is more vulnerable to *Fusarium* wilt (*Fusarium oxysporum* f.sp. *pisi*) due to high soil temperature and moisture. Wilt caused by *Fusarium oxysporum* Schl. f. sp. *pisi* Snyd. & Hans (*Fop*), is an important fungal disease of peas in most pea growing regions around the world. Six races have been described in the world. Race 1 was discovered in Wisconsin in 1924 (Linford, 10) and was later found in Washington, Idaho and New York (Wade *et al*., 14). Soon after resistance to race 1 was incorporated into cultivars, a second race which overcame resistance to race 1 was discovered (Snyder and Walker, 13). This second race, designated as race 2, was called near wilt since symptoms became noticeable later in the season than for race 1. Races 1 and 2 were the only economically important wilt races in the United States until race 5 appeared in north western Washington in 1963 (Haglund and Kraft, 4). Races 1 and 2 are known to occur throughout the world, while races 5 and 6 are only important in western Washington State (Haglund and Kraft, 5). Race 3 is present in Europe and England, while race 4 is found in Canada (Hagedorn, 3). Wilt caused by *Fop* is an increasing problem in north India. Affected plants may wilt and die either slowly or rapidly soon after the first symptoms appear. Genetic resistance to races 1, 2, 5, and 6 is conferred by different single dominant genes (Hagedorn, 3) and is available in germplasm releases (Haglund and Anderson, 6); control of diseases caused by soil-borne fungi, does not usually rely on the use of chemicals but is achieved mainly by integration of different disease management procedures. Germplasm collections are valuable sources of resistance to numerous plants. Being a soil borne disease, use of resistant cultivars is generally recognized as the safest, most economical and most effective method for protecting crops from disease (Johnson and Jellis, 8). The knowledge of genetic inheritance is essential for formulation of strategy on how to transfer the genes into adapted susceptible varieties. Hence, the present study was undertaken to identify the sources of *Fusarium* wilt resistance and to determine the inheritance of it in different garden pea backgrounds.

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MATERIALS AND METHODS

Thirty four genotypes obtained from various sources (Table 1) were screened for their level of resistance against *Fusarium* wilt under field conditions in wilt sick plot at Division of Vegetable Science, IARI, New Delhi during 2012-13 and 2013-14. All the genotypes were sown in wilt sick plot (WSP) to screen for disease reaction in comparison to highly susceptible variety 'Arkel' in randomized complete block design with two replications. The wilt susceptible check variety 'Arkel' was sown after every ten test entries in each replication. Seeds of each genotype were sown in first week of October at a spacing of 40 cm × 5 cm apart in 5 m^2 plot size keeping three rows of 4.2 m length each. The crop was fertilized with farmyard manure (10 tonnes/ ha) and NPK (20:60:40 kg/ha), followed by recommended cultural practices. The observations on initial plant stand after germination were recorded at 20 days after sowing and those on final survivality at the time of flowering and harvesting. The difference between the initial and final plant populations of different genotypes was used to calculate the percent disease intensity, which was characterized as per Charchar and Kraft (1).

Wilt severity index was based on a 0-5 scale where, 0 = no disease; 1 = pale yellow green, downward curling of leaf margin sand stipules; 2 = yellowing of leaves from stem base to apex; 3 = stunting and wilting; 4 = drying of lower leaves, yellow to orange brown discoloration of the vascular system in the upper tap root extending up the stem; 5 = completely wilted or plant death. According to Hannan *et al.* (7) if the disease incidence (DI) was 0-10% the crop response was considered highly resistant, while between 11-20% the crop rating was resistant, whereas between 21-30% it was moderately resistant. The crop was declared susceptible when DI was 31-50% and above 50% crop is considered highly susceptible. The angular transformed values of disease intensity for each genotype were computed for statistical analysis. A virulent isolate of *F. oxysporum* f.sp. *pisi* was isolated from the naturally wilted pea plants and identified from the Division of Plant Pathology, IARI, New Delhi. The fungus was cultured, maintained on fresh potato dextrose agar (PDA) and evaluated for host specificity. The sorghum grains were soaked in tap water overnight. Pre-soaked grains were put into conical flasks @ 250 g/ flask. These flasks were autoclaved at 1.1 kg/cm^2 for 20 min. for two subsequent days. The sterilized flasks after cooling were inoculated with 7-day-old actively growing culture of *F. oxysporum* f. sp. *pisi* by adding 4 mm agar plugs with sterile cork borer. These flasks were incubated at 25°C for 10 days. The plastic pots of 15 cm diameter were surface sterilized by 0.1% mercuric chloride and filled with 2.0 kg sterilized soil (1.0% formalin for 15 days) and inoculated with the 15-day-old culture of the pathogen multiplied on sorghum grains (10 g/ kg soil) seven days before sowing as described by Dubey *et al.* (2). Pots with un-inoculated soils were also maintained as control for comparison. Seeds of the susceptible and resistant cultivars were grown in pots (10 seeds per pot) and the plants were allowed to grow. The wilt incidence was recorded at 15-day intervals up to maturity of crop plants. Observations on survival of the plants have been recorded based on wilted plant as wilt incidence.

Based on the previous studies, the F_1 crosses involving five parents namely, susceptible variety Arkel and Pusa Pragati and resistant lines GP-6, GP-55 and GP-17 were selected from existing breeding programme at Division of Vegetable Science, IARI, New Delhi to generate F_2, B_1 and B_2 generations during

Table 1. Garden pea genotypes used for screening against wilt.

Source	Genotype(s)	No. of genotype(s)
ICAR-Indian Agricultural Research Institute, New Delhi, India	Arkel, Pusa Pragati, GP-943, GP-942, GP-945, GP-916, GP-918, GP-917, GP-904, GP-906, GP-910, GP-914, GP-1102, GP-473, GP-941, GP-48, GP-17, GP-55, GP-6	19
ICAR-Vivekananda Parvatiya Krishi Anusandhan Sansthan, Almora, India	VP-248, VL-7, VL-10, VP-525, VP-625, VP266	6
CS Azad University of Agriculture & technology, Kanpur, India	AP-3	1
ICAR-Indian Institue of Vegetable Research, Varanasi, India	VRP-6	1
United States Department of Agriculture, USA	EC-677211, EC-677212, EC-677215, EC-677214, EC-677216, EC-677213	6
ICAR- Indian Institute of Horticultural Research, Bengaluru, India	Arka Ajit (FC1)	1

Table 2. Reaction of garden pea genotypes in two respective years against *Fusarium* wilt disease.

Genotype	Mean [(x)] wilt incidence (%)			
	2012-13	% of wilt control	2013-14	% of wilt control
Arkel (Check)	95.25 (77.49)[l]	0.0	93.87 (75.73)[j]	0.0
Pusa Pragati	93.25 (75.0) [l]	2.1	93.37 (75.06)[j]	0.5
AP-3	89.75 (71.30)[j,k]	5.8	91.87 (73.55)[i, j]	2.1
VRP-6	87.75 (69.49) [j]	7.9	87.37 (69.16)[i]	6.9
EC-677214	86.25 (68.25)[j]	9.4	85.87 (67.90)[i]	8.5
EC-677216	85.50 (67.64)[j]	10.2	83.5 (66.05)[i]	11.1
Arka Ajit (FC1)	83.50 (66.02)[j]	12.3	83.5 (66.01)[i]	11.1
GP-943	78.50 (62.36)[i]	17.6	78 (62.00)[h]	16.9
VP266	81.25 (64.33)[i]	14.7	83.37 (65.95)[i]	11.2
GP-945	76.50 (61.00)[i]	19.7	76.75 (61.15)[h]	18.2
GP-916	78.25 (62.21)[i]	17.8	78.87 (62.62)[h]	16.0
GP-918	71.50 (57.73) [h]	24.9	73.5 (59.01)[h]	21.7
GP-917	71.00 (57.40)[h]	25.5	72.25 (58.19)[h]	23.0
GP-904	70.75 (57.33)[h]	25.7	72.62 (58.44)[h]	22.6
GP-906	61.25 (51.43)[f,g]	35.7	65.37 (53.97)[g]	30.4
GP-910	58.00 (49.59)[f]	39.1	60.75 (51.20)[g]	35.3
VL-7	61.75 (51.79)[g]	35.2	61.12 (51.41)[g]	34.9
VL-10	52.00 (46.13)[f]	45.4	53.5 (46.99)[f]	43.0
EC-677213	45.25 (42.26)[e]	52.5	45.62 (42.47)[e]	51.4
VP-248	41.25 (39.94)[d,e]	56.7	44.87 (42.03)[e]	52.2
VP-525	38.50 (38.32)[d]	59.6	41.25 (39.94)[d,e]	56.1
VP-625	35.75 (36.70)[d]	62.5	40.12 (39.27)[d]	57.3
GP-914	36.00 (36.85)[d]	62.2	39.75 (39.05)[d]	57.7
GP-1102	32.75 (34.89)[d]	65.6	34.35 (35.88)[c,d]	63.4
EC-677211	27.50 (31.61)[c]	71.1	30.5 (33.48)[c]	67.5
EC-677212	26.00 (30.64)[c]	72.7	27.75 (31.77)[c]	70.4
EC-677215	19.75 (26.37)[b,c]	79.3	23.62 (29.00)[c]	74.8
GP-473	16.25 (23.75)[b]	82.9	15.37 (23.07)[b]	83.6
GP-48	12.50 (20.66)[b]	86.9	15 (22.71)[b]	84.0
GP-17	12.50 (20.66)[b]	86.9	12.00 (20.24)[b]	87.4
GP-941	12.00 (20.24)[b]	87.4	12.75 (20.90)[b]	86.4
GP-942	8.00 (16.39)[a]	91.6	7.5 (15.88)[a]	92.0
GP-55	8.50 (16.94)[a]	91.1	8.75 (17.20)[a]	90.7
GP-6	6.50 (14.75)[a]	93.2	6.25 (14.47)[a]	93.3
CD at 5%	(3.80)		(3.44)	
CV	(4.01)		(3.57)	

*Figures in parentheses are angular transformed values

'x' Means followed by the same letter in a column within each year are not significantly different ($p \leq 0.05$) according to Fisher's protected least significant difference test using values after angular transformation of the proportion of wilt.

the 2012-13 and also to develop new F_1 population for evaluation in the next year. Among these, three crosses (Pusa Pagati × GP-55, Pusa Pragati × GP-6 and Arkel × GP-17) were selected for inheritance study. Thus, the experimental materials comprised of six generations (P_1, P_2, F_1, F_2, B_1 and B_2) were evaluated in field as well as artificially inoculated in pots in greenhouse during 2013-14. Seeds of the parents (Pusa Pragati, Arkel, GP-6, GP-55 and GP-17) and their respective F_1s, F_2s, B_1s and B_2s populations were grown in plastic pots (10 seeds per pot) in artificial condition. Similarly all the parents, F_1s, F_2s and backcrosses generations (B_1 and B_2) were grown in a randomized block design with two replications in the experimental wilt sick plot. Three rows of each parents and F_1s, two rows of each F_1s, F_2s, B_1s and B_2s and 5 rows of F_2 were sown in randomized block design with two replications during first week of October in winter 2013-14. Each row was 4.2 m, while inter- and intra-row plant spacings were 40 and 10 cm, respectively. After disease appearance the number of healthy plants and the total number of wilted plants were counted. The observations were recorded in each of P_1, P_2, F_1, F_2, B_1 and B_2 in generations were used. For Chi square analysis to test the ratio of phenotypically resistant: phenotypically susceptible plants in the F_2, B_1 and B_2 progenies, the total number of plants falling into resistant and susceptible plant was counted and subjected to χ^2 analysis for goodness-of-fit, as suggested by Panse and Sukhatme (11).

RESULTS AND DISCUSSION

The screening of genotypes of garden pea for *Fusarium* wilt resistance in two consecutive years as furnished in Table 2 revealed significant differences between the genotypes for percent disease intensity. The wilt-susceptible check Arkel recorded the highest wilting of 95.25% during 2012-13 and 93.87% during 2013-14 among the genotypes screened, confirming the high and uniform infestation of *Fusarium* in the soil of sick plot and effectiveness of screening the genotypes for resistance to *Fusarium*. During the year 2012-13 the percent disease intensity among the genotypes were ranged from 6.50 to 95.25%. Clear wilt symptoms like yellowing of the lower leaves and stipules and margins of the leaflets curl downward and inward could first be observed in susceptible genotype at 17 days after sowing. All the test genotypes recorded significantly lower disease intensity as compared to the susceptible check Arkel. The line GP-6 recorded the maximum resistant reaction to *Fusarium* as it exhibited the lowest wilting of 6.5%, which was followed by the GP-55 (8.50%), GP-942 (8%) and GP-17 (12.50%). Similarly during 2013-14, the percent disease intensity among the genotypes screened ranged from 6.50 to 93.87%. All the test entries recorded significantly lower disease intensity as compared to the susceptible check Arkel. The line GP-6 recorded the maximum resistant reaction to *Fusarium* as it exhibited the lowest wilting (6.25%), which was followed by the GP-942 (7.50%) and GP-17 (12.00%). The line GP-55 showed comparatively slightly high wilting percentage (8.75%) in 2013-14. According to disease severity (Table 3), three genotypes were recorded as highly resistant (GP-6, GP-55, and GP-942), four resistant (GP-17, GP-473, GP-48, GP-941), three moderately resistant (EC-677215, EC-677211, EC-677212), eight susceptible (EC-677213, VP-248, VP-525, VP-625, GP-914, VP266, GP-945, GP-1102) and sixteen were highly susceptible (Arkel, Pusa Pragati, AP-3, VRP-6, EC-677214, EC-677216, Arka Ajit, GP-943, VL-7,VL-10, GP-916, GP-918, GP-917, GP-904, GP-906, GP-910) genotypes. The overall results indicated that lines GP-6, GP-55, GP-942 and GP-17 were found resistant to *Fusarium* wilt, which could be further used in breeding programme for the development of resistant genotypes. Development of disease is slow in resistant lines and fast in susceptible lines. Similarly, GP 17, GP 207 and GP 473 garden pea genotypes showed high degree of resistance (9-26%) against *Fusarium* wilt, as also reported by Shri Dhar *et al.* (12). It is interesting to note that all the cultivable varieties (Arkel, Pusa Pragati, AP-3, VRP-6, VL-7, VL-10 and Arka Ajit) were highly susceptible to wilt as earlier reported by Shri Dhar *et al.* (12).

Table 3. Grouping of genotypes on the basis of disease reaction scale.

Scale	Reaction	No. of genotypes	Genotypes
0-10%	Highly resistant	3	GP-6, GP-55, GP-942
11-20%	Resistant	4	GP-17, GP-48, GP-473, GP-941
21-30%	Moderately resistant	3	EC-677215, EC-677211, EC-677212
31-50%	Susceptible	8	EC-677213, VP-248, VP-525, VP-625, GP-914, VP266, GP-945, GP-1102
Above 50%	Highly susceptible	16	Arkel, Pusa Pragati, AP-3, VRP-6, EC-677214, EC-677216, Arka Ajit, GP-943 VL-7, VL-10 GP-916, GP-918, GP-917, GP-904, GP-906, GP-910

Based on screening results two commercially accepted but susceptible genotypes Arkel and Pusa Pragati and three resistant genotypes GP-55, GP-6 and GP-17 were used for development of breeding population to investigate the genetics of wilt resistance. The reaction of plants to *Fusarium* wilt was analyzed using different generations derived from cross combinations (Pusa Pragati × GP-55, Pusa Pragati × GP-6 and Arkel × GP-17). The results obtained in each of the three crosses are presented in Table 4. All the F_1 population confirmed the dominance nature of *Fusarium* wilt resistance gene due to survival of all the plants. Besides, the stable performance of F_1 for disease resistance during crossing for developing different generations showed consistency of *Fusarium* wilt resistance in parents and F_1s. Each plant was categorized based on *Fusarium* wilt resistance and classified as resistant and susceptible plants. On the perusal of individual plant data (Table 4) it was shown that in cross Pusa Pragati × GP-55, segregation pattern of F_2 population is demonstrated a good fit to one gene model with a ratio of 3:1 (108 resistant: 32 susceptible), with higher confidence level (p = 0.558) after chi-square (χ^2) analysis. The non-significance of the χ^2 test (*P* value ≥ 0.05) indicated close agreement between the observed and expected ratio of resistant: susceptible plants. Backcross generations were also evaluated in each cross combination for confirmation of segregation pattern in F_2 population. The frequency distributions of resistance and susceptibility in plants of the two backcross generations, *i.e.* for BC_1 29R:36S with confidence (p = 0.383) and for B_2 65R: 0S tested for their best-fit with classical Mendelian ratios, *i.e.*, 1:1 in backcross progenies with a susceptible parent and 1:0 backcross progenies with a resistant parent (Table 4). These findings suggested the wilt resistance is governed by monogenic, dominant nature of heritable resistance against *Fop*.

Similarly, in cross Pusa Pragati × GP-6, GP-6 is a highly resistant parent in field as well as in artificial conditions. All the F_1 plants showed resistance as there was no mortality. The segregation of resistant and susceptible plants in the F_2 population was tested for agreement with the Mendelian ratio of

Table 4. Segregation of garden pea genotypes response against *Fusarium* wilt and their probability in the F_2 and back-cross generations.

Cross	Total No. of plants	Observed No. of plants		Expected ratio R:S	Observed ratio R:S	χ^2 value (cal.)	P value (df = 1)
		R	S				
Pusa Pragati × GP-55							
Pusa Pragati (PP)	45	0	45		-	-	-
GP-55	45	45	0		-	-	-
F_1	45	45	0		-	- 0.342	-
F_2	140	108	32	3:1	3.37:1	0.015	0.558
B_1	65	33	32	1:1	1:1		0.902
B_2	65	65	0				
Pusa Pragati × GP-6							
Pusa Pragati (PP)	45	0	45		-	-	-
GP-6	45	45	0		-	-	-
F_1	40	40	0		-	-	-
F_2	110	79	31	3:1	2.54:1	0.600	0.438
B_1	45	24	21	1:1	1:1	0.200	0.654
B_2	45	45	0				
Arkel × GP-17							
Arkel	50	0	50		-	-	-
GP-17	50	50	0		-	-	-
F_1	50	50	0		-	-	-
F_2	160	112	48	3:1	2.33:1	2.130	0.144
B_1	60	28	32	1:1	1:1	0.330	0.565
B_2	60	60	0				

3 (resistant):1 (susceptible) using the χ^2 test (χ^2: 0.60; p = 0.438). Out of 110 F_2 populations, 79 were resistant plants and 31 plants were susceptible. The back-cross (B_1) population, using 'Pusa Pragati' as the recurrent parent, fitted the expected 1:1 ratio of resistant: susceptible and 1:0 was recorded in B_2.

In cross Arkel × GP-17, GP-17 is resistant parent and all plants in the F_1 generation were resistant. The segregation of resistant and susceptible plants in the F_2 population was tested for agreement with the Mendelian ratio of 3 (resistant):1 (susceptible) using the χ^2 test. F_1 backcrossed to resistant parent GP-17 were also resistant. The probability values were indicated that there are no significant differences between the observed and the expected segregation ratios of resistant and susceptible plants (Table 4). The results of these populations confirm that the inheritance of resistance against *Fusarium* wilt disease is determined by a major dominant gene. Wade (14) reported similar results in canning peas. Monogenic resistance in peas to race 1 was also mentioned by Charchar and Kraft (1). The similarity is supported further by Karimi *et al.* (9) that Fusarium wilt was under the control of major gene in pigeon pea. Gene responsible for resistance in genotype GP-17, GP-55 and GP-6 are dominant in nature. This study could identify three genotypes as resistance source against *Fusarium* wilt of garden pea. The inheritance of resistance gene was found to be monogenic dominant in the three genotypes. However, the level of resistance in progenies obtained with GP55 was most consistent as evident from the higher level of confidence in Chi square analysis. The information obtained in this study could be valuable for *Fusarium* wilt resistance breeding and in planning an efficient strategy to incorporate the *Fusarium* wilt resistance in susceptible cultivars.

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