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Screening of Common Bean Genotypes for Resistance against Fusarium Wilt

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ABSTRACT: Fusarium wilt is the most serious disease of common bean causing 10 to 15 per cent yield losses each year. It is a soil-borne fungal disease where water conducting (xylem) vessels become blocked. The pathogenicity of the isolated fungus was proving by Koch's postulates. Further 50 common bean genotypes were screened under artificially inoculated controlled conditions among which 4 genotypes (G-257, UK-2, G-341, G-832) showed resistant reaction, 4 genotypes (G-716, G-195, G-2, G-185) showed moderately resistant reaction, 21 genotypes (WB-634, WB-642, WB-1318, WB-969, WB-160, WB-967, WB-923, WB-966, WB-1587, WB-4709, WB-651, WB-1634, WB-1282, WB-6, WB-482, WB-481, G-1313, WB-164, WB-13398, WB-935, WB-25575) showed moderately susceptible reaction, 18 genotypes (WB-1319, WB-1436, WB-1413, PBG-111, WB-662, WB-4564, WB-371, WB-1644, WB-333, WB-206, WB-206, WB-901, WB-956, WB-952, WB-451, WB-222, WB-1429, WB-1187) showed susceptible reaction and remaining 4 genotypes (Arka-Anop, SFB-1, WB-352, WB-1319, SR-1) exhibited highly susceptible reaction.

Keywords: Fusarium, screening, genotypes, resistant, susceptible, common bean, wilt, pathogen.

INTRODUCTION

Common bean (*Phaseolus vulgaris* L.) belong to family Fabaceae and is a native to South Mexico and Central America. It is a major grain legume consumed worldwide for its edible seeds and pods and is an important source of human dietary protein and calories (Pachico, 1993). It is traditionally a basic food crop in many developing countries and serves as a major plant protein source for rural and urban areas. The crop is consumed principally for its dry beans and green pods. It provides 15 per cent protein with high contents of lysine and methionine and 30 per cent caloric requirement to the world's population and represents 50 per cent of the grain legume consumed worldwide (McConnell *et al.*, 2010). The dry pulse bean and green snap bean possess 22 per cent and 6.1 per cent protein, respectively and are increasingly being consumed as an alternative to animal protein by low income families in developing countries (Bhat *et al.*, 2017). Dry beans contain high levels of chemically diverse components like phenols, starch, vitamins and fructo-oligosaccharides giving protection against conditions like oxidative stress, cardiovascular diseases, diabetes, metabolic syndrome and many types of cancers (Camara *et al.*, 2013). Moreover, beans are consumed as boiled, baked, fried or ground into flour. Crop residues such as dried pods, stems and processed by-products are used as fodder. Although common bean is less efficient in fixing N than other legumes, yet it is able to fix up 125 kg N ha^{-1} and nodulates with several rhizobia (Wortmann, 2006). Globally the production of common bean is 26.83 million tonnes in an area of 29.39 million ha with a productivity of 0.91 t ha^{-1}. In India it is grown in an area of 9.47 million ha with a production of 3.90 million tonnes and productivity 0.41 t ha^{-1} (Anonymous, 2017). It is the premier green legume crop of Jammu and Kashmir where its cultivation is mainly confined to rainfed and *karewa* areas covering an area of about 2000 ha with an annual production of about 1600 tonnes and yield of about 0.8 t ha^{-1} (Choudhary *et al.*, 2017). A number of biotic and abiotic stresses like diseases, insect-pests, soil and environmental factors are responsible in reduction of crop yield. Among diseases, Ascochyta blight (*Ascochyta phaseolorum*), bean rust (*Uromyces appendiculatus*), angular leaf spot (*Phaeoisariopsis griseola*), powdery mildew (*Erysiphe polygony*), Bacterial blight (*Xanthomonas phaseoli*), anthracnose (*Colletotrichum lindemuthianum*), Charcoal rot (*Macrophomina phaseolina*), white mold (*Sclerotinia sclerotiorum*) and Fusarium wilt of bean (*Fusarium oxysporum*) are mostly prevalent (Junior *et al.*, 2001

and Schwartz and Harveson 2015). Fusarium wilt is one of the most important economic diseases of common bean worldwide and has caused significant economic losses (Saremi, 2000; Bentley *et al.*, 2006; Okungbowa and Shittu, 2014; Xue *et al.*, 2015).

Keeping the significance of the bean crop in view under Kashmir agro-climatic conditions, screening of common bean germplasm against the Fusarium wilt was done.

MATERIALS AND METHODS

Screening of disease resistance genotypes. Fifty elite common bean genotypes were grown in polybags under controlled conditions in polyhouse.

Screening was performed by using two inoculation techniques

Soil inoculation technique

Root dip inoculation technique

Soil inoculation technique. The soil was inoculated with suspension of fusarium culture after germination of seedlings. Fifty genotypes were grown under controlled conditions replicated three times. There were three rows of polybags in polyhouse and in each row there were fifty polybags. In each row there was one susceptible check of SFB-1, Arka-Anop and SR-1 were planted. After 16-20 days of germination of seedlings suspension of fusarium culture was inoculated into the soil in order to make soil wilt sick. Fusarium wilt symptoms were observed from 20-25 days after inoculation, continuing to 45 days. The numbers of dead and diseased seedlings were recorded weekly. The observations included: no apparent symptoms or disease (0-10 days); chlorosis and early wilting of seedlings (10-15 days); chlorosis, stunting, defoliation of lower leaves and late wilting (15-30 days); and chlorosis, defoliation, stunting, but no wilting (>30 days).

Root dip inoculation technique. Screening for some lines was performed by the root-dip inoculation technique described by Pastor-Corrales and Abawi (1987), later modified by Salgado and Schwartz (1993). The procedure uses 16 to 20 day old seedlings grown in a polybag mixture (soil: sand: vermicompost; 2:1:1 by volume). The seedlings were removed from bags and the root system gently washed to remove excess soil mixture and placed in tap water for 5 to 10 min. The distal 1/3 of the root system was clipped with scissor and the root system was placed in the root-dip inoculum solution for 5 min. After inoculation, plants were transplanted to the same fresh mixture used for germination. The plants were watered 15 to 20 minutes after transplanting and every other day for the first 7 day, then as needed to maintain plant vigor. The plants were grown in a polyhouse maintained at approximately 16/28^{o}C night/day, respectively.

Observations on wilt incidence was recorded using the formula:

$$\text{Disease incidence (\%)} = \frac{\text{Number of diseased plants (n)}}{\text{Total number of plants examined (N)}} \times 100$$

The per cent incidence was categorized into different reaction types as per the scale developed by Haware and Nene (1982):

Wilt (%)	Reaction Type
0.1-10	Resistant(R)
10.1-25	Moderately resistant (MR)
25.1-50	Moderately susceptible (MS)
50.1-75	Susceptible (S)
>75	Highly susceptible (HS)

Data analysis shall be carried out using statistical procedures as per Gomez and Gomez (1984).

RESULTS

Screening of common bean germplasm against the disease. The data on disease incidence of fifty genotypes of common bean at two stages *viz.*, seedling and flowering stage (Table 1). Based on their reaction towards the disease the 50 genotypes were grouped into five categories *i.e.* resistant, moderately resistance, moderately susceptible and susceptible. At seedling stage, 18 genotypes (WB-716, WB-832, WB-371, WB-341, WB-1587, UK-2, WB-651, WB-257, WB-956, WB-482, WB-451, WB-2, WB-481, WB-1313, WB-1643, WB-185, WB-935, WB-25575) were found to be resistant, 20 genotypes (WB-634, WB-642, WB-1436, WB-1318, WB-969, WB-1607, WB-96, PBG-111, WB-966, WB-195, WB-1644, WB-206, WB-4709, WB-1282, WB-901, WB-6, WB-952, WB-222, WB-1429, WB-13398) were found as moderately resistant, 8 genotypes (WB-1413, WB-923, WB-4564, WB-662, WB-333, WB-1634, WB-1319, WB-1187) as moderately susceptible and 4 genotypes (Arka-Anop, SFB-1, WB-352, SR-1) susceptible (Table 2). At flowering stage 4 genotypes (WB-257, UK-2, G-341, WB-832) observed resistant, 4 genotypes (WB-716, WB-195, WB-2, WB-185) moderately resistant, 21 genotypes (WB-634, WB-642, WB-1318, WB-969, WB-160, WB-967, WB-923, WB-966, WB-1587. WB-4709, WB-651, WB-1634, WB-1282, WB-6, WB-482, WB-481, G-1313, WB-164, WB-13398, WB-935, WB-25575) moderately susceptible, 18 genotypes (WB-1319, WB-1436, WB-1413, PBG-111, WB-662, WB-4564, WB-371, WB-1644, WB-333, WB-206, WB-901, WB-956, WB-952, WB-451, WB-222, WB-1429, WB-1187) susceptible and 4 genotypes (Arka-Anop, SFB-1, WB-352, WB-1319, SR-1) highly susceptible (Table-3). It was further observed disease incidence at flowering stage, invariably more in all the genotypes as compared to that at seedling. A considerable variation between genotypes was observed in both stages. In general, the disease incidence ranged from 0 to 54.2 per cent at seedling stage and 8.6 to 100 per cent at

flowering stage.

Table 1: Wilt incidence of common bean genotypes against Fusarium wilt at seedling and flowering stage.

Sr. No.	Genotype	% wilt incidence at seedling stage	Reaction	% wilt incidence at flowering stage	Reaction
1.	WB-716	9.3	R	23.3	MR
2.	Arka-Anop	50.1	S	83.1	HS
3.	WB-634	10.5	MR	26.4	MS
4.	WB-642	14.4	MR	36.8	MS
5.	WB-832	8.4	R	8.6	R
6.	WB-1436	12.2	MR	51.8	S
7.	WB-1318	11.7	MR	32.2	MS
8.	WB-1413	25.7	MS	69.2	S
9.	WB-969	23.1	MR	44.4	MS
10.	WB-1607	20.7	MR	41.2	MS
11.	WB-967	11.5	MR	43.1	MS
12.	PBG-111	23.6	MR	68.1	S
13.	WB-923	31.6	MS	37.5	MS
14.	WB-966	13.2	MR	47.5	MS
15.	WB-195	12.1	MR	24.4	MR
16.	WB-4564	25.6	MS	50.1	S
17.	WB-662	32.1	MS	52	S
18.	SFB-1	50.4	S	100	HS
19.	WB-1644	11.8	MR	56.7	S
20.	WB-371	8.1	R	56.8	S
21.	WB-341	1.6	R	9.5	R
22.	WB-333	35.0	MS	61.4	S
23.	WB-206	13.3	MR	52.2	S
24.	WB-1587	7	R	24.3	MS
25.	UK-2	1.8	R	9.5	R
26.	WB-4709	23.4	MR	48.1	MS
27.	WB-651	7.6	R	26.6	MS
28.	WB-1634	7.1	MS	48.7	MS
29.	WB-1282	19.1	MR	31.6	MS
30.	WB-257	0	R	9.4	R
31.	WB-901	15.8	MR	52.1	S
32.	WB-956	8.3	R	54.1	S
33.	WB-6	12.3	MR	47.2	MS
34.	WB-952	13.2	MR	52.3	S
35.	WB-482	8.9	R	44.3	MS
36.	WB-352	51.1	S	91.8	HS
37.	WB-451	8.0	R	51.6	S
38.	WB-222	24.0	MR	68.2	S
39.	WB-2	4.2	R	24.4	MR
40.	WB-481	11	MR	48.1	MS
41.	WB-1313	7.3	R	25.4	MS
42.	WB-1429	13	MR	51.6	S
43.	SR-1	54.2	S	98.5	HS
44.	WB-1643	6.5	R	46.8	MS
45.	WB-185	9.5	R	23	MR
46.	WB-13398	12.3	MR	25.3	MS
47.	WB-1319	44.0	MS	66.2	S
48.	WB-935	7.2	R	42.9	MS
49.	WB-25575	9.2	R	45.6	MS
50.	WB-1187	25.5	MS	51	S

Table 2: Reaction of common bean genotypes against Fusarium wilt under controlled condition at seedling stage.

Genotypes	% Rating	Categories	No. of genotypes
WB-716, WB-832, WB-371, WB-341, WB-1587, UK-2, WB-651, WB-257, WB-956, WB-482, WB-451, WB-2, WB-481, WB-1313, WB-1643, WB-185, WB-935, WB-25575	0.1-10% of plants wilted	Resistant	18
WB-634, WB-642, WB-1436, WB-1318, WB-969, WB-1607, WB-96, PBG-111, WB-966, WB-195, WB-1644, WB-206, WB-4709, WB-1282, WB-901, WB-6, WB-952, WB-222, WB-1429, WB-13398	10.1-25% of plants wilted	Moderately resistance	20
WB-1413, WB-923, WB-4564, WB-662, WB-333, WB-1634, WB-1319, WB-1187	25.1-50% of plants wilted	Moderately susceptible	8
Arka-Anop, SFB-1, WB-352, SR-1	50.1-75% or more of plants wilted	Susceptible	4
_____	>75% or more of plants wilted	Highly susceptible	

Table 3: Reaction of common bean genotypes against Fusarium wilt under controlled condition at flowering stage.

Genotypes	% Rating	Categories	No. of genotypes
WB-257, UK-2, WB-341, WB-832	0.1-10% of plants wilted	Resistant	4
WB-716, WB-195, WB-2, WB-185	10.1-25% of plants wilted	Moderately resistance	4
WB-634, WB-642, WB-1318, WB-969, WB-160, WB-967, WB-923, WB-966, WB-1587, WB-4709, WB-651, WB-1634, WB-1282, WB-6, WB-482, WB-481, G-1313, WB-164, WB-13398, WB-935, WB-25575	25.1-50% of plants wilted	Moderately susceptible	21
WB-1319, WB-1436, WB-1413, PBG-111, WB-662, WB-4564, WB-371, WB-1644, WB-333, WB-206, WB-206, WB-901, WB-956, WB-952, WB-451, WB-222, WB-1429, WB-1187	50.1-75% or more of plants wilted	Susceptible	18
Arka-Anop, SFB-1, WB-352, WB-1319, SR-1	>75% or more of plants wilted	Highly susceptible	4

DISCUSSION

Common bean (*Phaseolus vulgaris* L.) is the third most important food legume crop worldwide (Schwartz *et al.*, 2005). It is widely consumed throughout the world and is considered a good source of protein, carbohydrates, dietary fibre and some vitamins and minerals (Campos-vega, 2013). The crop is affected by several disease e.g. Fusarium wilt, bean rust, angular leaf spot, powdery mildew, halo blight, anthracnose, white mold etc. Among various diseases, the crop suffers a huge loss of upto 100 per cent due to Fusarium wilt caused by the necrotrophic fungi *Fusarium oxysporum* f.sp. *phaseoli*. In India, the disease has been reported for the first time by Gupta *et al.* (1993).The symptoms of the disease appear as yellowing and premature senescence of the lower leaves. The chlorotic symptoms progress upwards until all leaves are bright yellow, followed by wilting and discoloration of foliage and when plants are infected at early stage, they remain stunted (Buruchara and Camacho 2000).

— In present study, 50 common bean genotypes screened against bean wilt under controlled conditions at two stages *viz*., seedling and flowering stages. At seedling stage, 18 genotypes (WB-716, WB-832, WB-371, WB-341, WB-1587, UK-2, WB-651, WB-257, WB-956, WB-482, WB-451, WB-2, WB-481, WB-1313, WB-1643, WB-185, WB-935, WB-25575) were found to be resistant, 20 genotypes (WB-634, WB-642, WB-1436, WB-1318, WB-969, WB-1607, WB-96, PBG-111, WB-966, WB-195, WB-1644, WB-206, WB-4709, WB-1282, WB-901, WB-6, WB-952, WB-222, WB-1429, WB-13398) moderately resistant, 8 genotypes (WB-1413, WB-923, WB-4564, WB-662, WB-333, WB-1634, WB-1319, WB-1187) moderately susceptible and 4 genotypes (Arka-Anop, SFB-1, WB-352, SR-1) susceptible whereas at flowering stage, 4 genotypes (WB-257, UK-2, G-341, WB-832) were observed resistant, 4 genotypes (WB-716, WB-195, WB-2, WB-185) moderately resistant, 21 genotypes (WB-634, WB-642, WB-1318, WB-969, WB-160, WB-967, WB-923, WB-966, WB-1587, WB-4709, WB-651, WB-1634, WB-1282, WB-6, WB-482, WB-481, G-1313, WB-164, WB-13398, WB-935, WB-25575) moderately susceptible, 18 genotypes (WB-1319, WB-1436, WB-1413, PBG-111, WB-662, WB-4564, WB-371, WB-1644, WB-333, WB-206, WB-206, WB-901, WB-956, WB-952, WB-451, WB-222, WB-1429, WB-1187) susceptible and 4 genotypes (Arka-Anop, SFB-1, WB-352, WB-1319, SR-1) highly susceptible. Our results are in conformity with results of Alves-Santos *et al*. (2002); Cramer *et al*. (2003) who studied the pathogenicity and race characterization of *Fusarium oxysporum* f.sp. *phaseoli* isolates from Spain and Greece and found that out of 25 genotypes evaluated; 5 were resistant, 10 were moderately resistant and 10 were susceptible. Our results are in agreement with Brick *et al*. (2006) who studied the reaction to three races of Fusarium wilt in the *Phaseolus vulgaris* core collection. Among accessions evaluated for reaction to race 1 of Fop, 21 were resistant, 47 intermediate and 126 susceptible. Fifteen accessions were resistant to race 4, 61 intermediate and 126 susceptible. Nine

accessions were resistant to both race 1 and 4. Similar studies were conducted by Buruchara and Camacho (2008) who studied the cause of severe vascular wilt in Central Africa and reported that out of the 29 climbing bean genotypes evaluated, 19 were resistant, including 11 of the 15 pre released cultivars. Out of the 44 bush bean cultivars 28 were resistant, 5 were moderately resistant and 11 were susceptible. Similar efforts have also been made earlier to identify common bean germplasm with Fusarium wilt resistance and common bean genotypes with varied levels of resistance identified by various workers (Musoni *et al*., 2010; Immaculee and Uma 2013).

CONCLUSION

Among fifty genotypes, evaluated for their resistance to bean wilt disease under controlled conditions, 18 genotypes were found to be resistant, 20 genotypes, moderately resistant, 8 genotypes moderately susceptible and 4 genotypes susceptible at seedling stage. Whereas, 4 genotypes were observed resistant, 4 genotypes moderately resistant, 21 genotypes moderately susceptible, 18 genotypes susceptible and 4 genotypes highly susceptible at flowering stage. The resistant genotypes at seedling stage may be planted in areas where disease occurs at seedling stage only. Delay in sowing can also help to escape disease from such areas.

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