

FIELD EVALUATION OF SESAME GERMPLASM AGAINST SESAME PHYLLODY DISEASE

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Abstract

Phyllody disease, caused by phytoplasma, is a major threat for the successful production of sesame worldwide, including Pakistan. Use of resistant varieties is considered as an economical and durable method of controlling this malady. Therefore, the resistance of 133 sesame genotypes belonging to different regions was evaluated in the field under high inoculum pressure for two consecutive years. During the first year (2007), three genotypes namely NS 98002-04, NS 98003-04 and NS 99005-01 were ranked as highly resistant as they remained symptomless till the harvest of crop while eleven others namely; NS97001-04, NS01004-04, Sumboonkkae, NS940051-04, NS20005-04, NS 11704, NS96019-04, Ahnsankkac, NS 11504, Hansumkkae and NS99006-04 were scored as resistant with percent disease infection (PDI) of 3.12, 3.33, 3.40, 3.45, 5.0, 5.30, 5.88, 7.14, 8.69, 8.70 and 10%, respectively. Other genotypes ranked between moderately resistant to highly susceptible with PDI values ranging from 10.71% to 65.12%. During second year all the tested genotypes were found to be infected with phyllody disease. However, four genotypes viz., NS98002-04, NS98003-04, NS99005-01 and NS01004-04 were resistant with PDIs of 3.25, 3.25, 3.75 and 10.0% respectively. Combined analysis of data also showed that these genotypes could be considered as promising for breeding programmes.

Introduction

Sesame (*Sesamum indicum* L.) is one of the most ancient edible oil crop grown in many parts of the world (Barut *et al.*, 2006; Akbar *et al.*, 2012; Naqvi *et al.*, 2012; Wahid *et al.*, 2012). China, India, Myanmar (Burma) Uganda, Nigeria, Pakistan, Bangladesh, Ethiopia, Thailand, Turkey and Mexico are the major producers of sesame (Anon., 2004). Its seed is a rich source of oil (50%) and protein (20%) (Shyu & Hwang, 2002; Akbar *et al.*, 2011a, 2011b). Sesame seed oil has a long shelf life because of an antioxidant called sesamol and also contains oleic acid. It is used in cooking, salad, margarine and is a raw material for the production of some industrial materials like paints, varnishes, soaps, perfumes, pharmaceuticals and insecticides (Jin *et al.*, 2001; Wang *et al.*, 2013).

Sesame is vulnerable to infection by a number of pathogens that cause considerable yield losses. Among the major diseases, phyllody is a very serious disease, which can inflict up to 80% yield loss with a disease intensity of 61-80% (Kumar & Mishra, 1992; Salehi & Izadpanah, 1992). It has been reported from India, Iran, Iraq, Israel, Burma, Sudan, Nigeria, Tanzania, Pakistan, Ethiopia, Thailand, Turkey, Uganda, Upper Volta and Mexico (Akhtar *et al.*, 2009). Sesame phyllody was first recorded in Pakistan at Mirpur Khas in 1908 (Vasudeva & Sahambi, 1955; Vasudeva, 1961). In Pakistan this disease was found to be caused by a phytoplasma belonging to subgroup 16SrII-D (Akhtar *et al.*, 2008). Phytoplasmas are phloem inhabiting, wall less bacteria in the class Mollicutes which cause devastating damage to crops and are known to infect approximately 1000 plant species worldwide including fruits, vegetables, cereals, trees and legumes (Semuller *et al.*, 1998; Iftikhar & Fahmeed, 2011). Typical symptoms of sesame phyllody include floral virescence, phyllody and proliferation however, sometimes these symptoms are found to be accompanied with yellowing, cracking of seed capsule, germination of seeds in capsules and formation of dark exudates on the foliage (Akhtar *et al.*, 2009).

Sesame phyllody is not seed borne. In nature, disease is mainly spread by leafhopper *Orosius albicinctus* and

survives in alternate hosts (Akhtar *et al.*, 2009). Treatment of infected plants with tetracycline-HCl, destruction of weed reservoirs and use of systemic insecticides to control leafhopper has been unsuccessful. However, the use of resistant genotypes is a long-term solution to control this malady (Weintraub & Jones, 2010). Therefore, the present study was initiated to screen sesame germplasm (representing six countries) against 16SrII-D phytoplasma under field conditions for the first time.

Materials and Methods

A total of 133 white and black seeded sesame genotypes (gene pool maintained at NIAB, Faisalabad) were evaluated for their resistance against sesame phyllody in the field during 2007 and 2008 (Table 1).

Each test entry was planted in three replicates in rows of 3 meter in length following completely randomized block design during month of July at Nuclear Institute for Agriculture and Biology (NIAB), Faisalabad, Pakistan during both years. Plant-to-plant distance was kept at 15 cm and row to row 45 cm. Conventional agronomic practices were followed to keep the crop in good condition, however, no plant protection measures were applied against the vector *O. albicinctus* to have the high inoculum pressure. Data on the incidence of phyllody in each genotype was recorded weekly by counting the number of infected plants and total population before harvest. Resistance or susceptibility of genotypes was based on the average percentage of plants infected by the disease, following a seven point (0-6) rating scale, where 0 = no infection (highly resistant); 1 = 0.1-10 percent plants infected (resistant); 2 = 10.1-20 percent plants infected (moderately resistant); 3 = 20.1-30 percent plants infected (tolerant); 4 = 30.1-40 percent plants infected (moderately susceptible); 5 = 40.1-50 percent plants infected (susceptible) and 6 = more than 50 percent plants infected (highly susceptible).

The appearance of first leafhopper *O. albicinctus*, was detected using yellow sticky traps in sesame fields from 2007-2008. The vector population was monitored as in mass assays.

Table 1. Field response of sesame genotypes to phyllody disease.

Genotype	Origin	Seed colour	2007		2008		Combined	
			Percent disease infection	Response	Percent disease infection	Response	Percent disease infection	Response
Hansumkkae	Korea	White	8.70	R	18.02	MR	13.36	MR
Sumboonkkae	Korea	White	3.45	R	15.15	MR	9.30	R
Kwangasang gac	Korea	White	23.68	T	39.06	MS	31.37	MS
Hansco mggae	Korea	Black	62.50	HS	70.87	HS	66.69	HS
Su weon-21	Korea	White	45.45	S	75.00	HS	60.23	HS
Ahnsankkac	Korea	White	7.14	R	18.90	MR	13.02	MR
Iraq-1	Iraq	Black	40.63	S	79.99	HS	60.31	HS
Iraq-2	Iraq	White	25.93	T	70.63	HS	48.28	S
Iraq-3	Iraq	White	18.00	MR	72.34	HS	45.17	S
Iraq-4	Iraq	White	19.05	MR	29.63	T	24.34	T
S-17-6	Pakistan	White	22.92	T	35.66	MS	29.29	T
K N Shah-2	Pakistan	White	15.78	MR	26.50	T	21.14	T
Kotri-1	Pakistan	White	15.21	MR	21.12	T	18.17	MR
S-70-62	Pakistan	White	13.95	MR	27.90	T	20.93	T
Nagar Parkar-2	Pakistan	White	27.27	T	54.48	HS	40.88	S
S-126	Pakistan	White	31.43	MS	38.04	MS	34.74	MS
K-96	Pakistan	White	29.03	T	46.89	S	37.96	MS
K-509	Pakistan	White	59.38	HS	68.50	HS	63.94	HS
K-690	Pakistan	White	57.58	HS	62.34	HS	59.96	HS
Johi-1	Pakistan	White	23.34	T	78.88	HS	51.11	HS
Johi-2	Pakistan	White	18.76	MR	83.93	HS	51.35	HS
S-2	Pakistan	White	34.62	MS	25.00	T	29.81	T
S-7	Pakistan	White	29.63	T	75.39	HS	52.51	HS
S-17	Pakistan	White	21.88	T	45.05	S	33.47	MS
S-17-21	Pakistan	White	26.83	T	35.01	MS	30.92	MS
S-18-13	Pakistan	White	15.79	MR	31.02	MS	23.41	T
Pr 19-9	Pakistan	White	25.00	T	39.00	MS	32.00	MS
Pr 14-2	Pakistan	White	48.28	S	59.00	HS	53.64	HS
S-20-9	Pakistan	White	20.00	MR	48.00	S	34.00	MS
S-20-18	Pakistan	White	25.72	T	64.43	HS	45.08	S
S-21	Pakistan	White	50.01	HS	60.85	HS	55.43	HS
VC/82 No 205 NS	Pakistan	White	63.19	HS	80.12	HS	71.66	HS
S-21-24	Pakistan	White	14.82	MR	33.55	MS	24.19	T
S-22	Pakistan	White	32.43	MS	51.02	HS	41.73	S
S-30	Pakistan	White	47.06	S	64.02	HS	55.54	HS
S-30-10	Pakistan	White	65.12	HS	70.02	HS	67.57	HS
S-48	Pakistan	White	17.50	MR	52.03	HS	34.77	MS
S-53	Pakistan	White	36.84	MS	45.00	S	40.92	S
Kotri-2	Pakistan	White	21.43	T	58.54	HS	39.99	MS
Kotri-3	Pakistan	White	34.29	MS	42.58	S	38.43	MS
Sanghar-1	Pakistan	White	17.95	MR	33.18	MS	25.57	T
Thana Bula Khan 1	Pakistan	White	21.74	T	78.04	HS	49.89	S
S-68	Pakistan	White	23.81	T	49.00	S	36.41	MS
S-68-1	Pakistan	White	25.00	T	52.00	HS	38.50	MS
S-70	Pakistan	White	21.43	T	46.41	S	33.92	MS

Table 1. (Cont'd.).

Genotype	Origin	Seed colour	2007		2008		Combined	
			Percent disease infection	Response	Percent disease infection	Response	Percent disease infection	Response
S-105	Pakistan	White	24.24	T	54.75	HS	39.50	MS
S-117	Pakistan	White	21.63	T	52.01	HS	36.82	MS
S-122	Pakistan	White	25.57	T	54.24	HS	39.91	MS
S-122-30	Pakistan	White	29.73	T	66.52	HS	48.13	S
Khipro-3	Pakistan	White	25.00	T	83.67	HS	54.34	HS
S-131	Pakistan	White	24.44	T	52.83	HS	38.64	MS
Saeedabad new	Pakistan	White	13.64	MR	29.01	T	21.33	T
S-155	Pakistan	White	15.00	MR	32.01	MS	23.51	T
K-87	Pakistan	White	18.52	MR	52.80	HS	35.66	MS
S-102	Pakistan	White	19.56	MR	39.75	MS	29.66	T
104/3 (Faisalabad)	Pakistan	White	12.12	MR	72.12	HS	42.12	S
K-141	Pakistan	White	15.22	MR	36.36	MS	25.79	T
K-161	Pakistan	White	26.47	T	31.20	MS	28.84	T
K-481	Pakistan	White	10.71	MR	29.01	T	19.86	MR
K-595	Pakistan	White	20.51	T	35.42	MS	27.97	T
K-612	Pakistan	White	33.33	MS	66.36	HS	49.85	S
K-1058	Pakistan	White	22.22	T	49.19	S	35.71	MS
110/5 (Faisalabad)	Pakistan	White	19.36	MR	35.29	MS	27.33	T
Mehar-1	Pakistan	White	17.95	MR	27.31	T	22.63	T
Pungny conggae	Pakistan	White	10.81	MR	30.00	T	20.41	T
Dandackggac	Pakistan	White	28.57	T	61.00	HS	44.79	S
Mehar-2	Pakistan	White	51.52	HS	80.00	HS	65.76	HS
Dadu-1	Pakistan	White	27.02	T	46.38	S	36.70	MS
Dadu-2	Pakistan	White	25.00	T	46.06	S	35.53	MS
Rato dero-2	Pakistan	White	28.13	T	63.46	HS	45.80	S
VCR/82 No % NS	Pakistan	White	37.50	MS	56.36	HS	46.93	S
Sehwan-1	Pakistan	White	33.33	MS	67.33	HS	50.33	HS
Sehwan-2	Pakistan	White	56.76	HS	85.33	HS	71.05	HS
VCR/82 No 14 NS	Pakistan	White	27.78	T	50.03	HS	38.91	MS
VCR/82 No 15 NS	Pakistan	White	34.48	MS	54.00	HS	44.24	S
S-32	Pakistan	White	24.00	T	35.04	MS	29.52	T
VCR/82 No 206 NS	Pakistan	White	39.13	MS	39.03	MS	39.08	MS
S-140	Pakistan	Black	42.31	S	65.56	HS	53.94	HS
S-148	Pakistan	Black	36.59	MS	54.02	HS	45.31	S
K-1034	Pakistan	White	42.11	S	69.05	HS	55.58	HS
K-1064	Pakistan	Black	41.38	S	72.58	HS	56.98	HS
Rato dero-1	Pakistan	Brown black	25.93	T	44.96	S	35.45	MS
VCR/82 No 17 NS	Pakistan	Black	26.92	T	53.00	HS	39.96	MS
S-19	Pakistan	Black	33.34	MS	54.25	HS	43.80	S
S-31-1	Pakistan	Black	33.33	MS	59.01	HS	46.17	S
S-33-10	Pakistan	Black	27.78	T	39.99	MS	33.89	MS
S-65	Pakistan	Black	25.64	T	55.23	HS	40.44	S
Nagar Parkar-1	Pakistan	Black	27.98	T	55.56	HS	41.77	S
S-74	Pakistan	Black	32.27	MS	65.33	HS	48.80	S
S-97	Pakistan	Black	40.54	S	69.33	HS	54.94	HS

Table 1. (Cont'd.).

Genotype	Origin	Seed colour	2007		2008		Combined	
			Percent disease infection	Response	Percent disease infection	Response	Percent disease infection	Response
S-100	Pakistan	Black	37.03	MS	57.20	HS	47.12	S
S-109	Pakistan	Black	21.43	T	49.26	S	35.35	MS
S-121-4	Pakistan	Black	52.38	HS	83.00	HS	67.69	HS
S-125	Pakistan	Black	54.55	HS	85.00	HS	69.78	HS
K-699	Pakistan	Black	34.78	MS	65.00	HS	49.89	S
VCR/82 No 8 NS	Pakistan	Black	58.53	HS	82.00	HS	70.27	HS
VCR/82 No 16 NS	Pakistan	Black	16.67	MR	29.99	T	23.33	T
S-31	Pakistan	Black	41.57	S	59.56	HS	50.57	HS
VCR/82 No 207 NS	Pakistan	Black	47.50	S	71.00	HS	59.25	HS
Sinjhoro-1	Pakistan	Black	43.75	S	69.06	HS	56.41	HS
Khipro-1	Pakistan	Black	40.74	S	71.05	HS	55.90	HS
Khipro-2	Pakistan	Black	45.16	S	75.06	HS	60.11	HS
S-132	Pakistan	Black	40.47	S	70.25	HS	55.36	HS
S-135	Pakistan	Black	43.75	S	75.06	HS	59.41	HS
S-147	Pakistan	Black	45.16	S	75.08	HS	60.12	HS
S-67	Pakistan	Black	25.64	T	50.02	HS	37.83	MS
Sanghar-2	Pakistan	White	47.62	S	85.05	HS	66.34	HS
NS940051-04	Pakistan	White	3.45	R	15.02	MR	9.24	R
NS96019-04	Pakistan	White	5.88	R	18.91	MR	12.40	MR
NS97001-04	Pakistan	White	3.12	R	14.67	MR	8.90	R
NS98002-04	Pakistan	White	0	HR	3.25	R	1.63	R
NS98003-04	Pakistan	White	0	HR	3.25	R	1.63	R
NS99005-01	Pakistan	White	0	HR	3.75	R	1.88	R
NS99006-04	Pakistan	White	10.00	R	25.01	T	17.51	MR
NS01004-04	Pakistan	White	3.33	R	9.00	R	6.17	R
NS20005-04	Pakistan	White	5.00	R	16.65	MR	10.83	MR
TS3	Pakistan	White	32.50	MS	54.02	HS	43.26	S
TIL-89	Pakistan	White	22.22	T	23.61	T	22.92	T
NS 303	Pakistan	White	16.97	MR	36.51	MS	26.74	T
NS 3303	Pakistan	White	31.55	MS	37.55	MS	34.55	MS
NS 3403	Pakistan	White	27.68	T	44.03	S	35.86	MS
NS 3503	Pakistan	White	28.85	T	32.41	MS	30.63	MS
NS 4503	Pakistan	White	21.43	T	34.44	MS	27.94	T
NS 5703	Pakistan	White	21.39	T	22.58	T	21.99	T
NS 5903	Pakistan	White	16.20	MR	26.05	T	21.13	T
NS 6303	Pakistan	White	23.26	T	43.70	S	33.48	MS
NS 203	Pakistan	White	20.69	T	25.90	T	23.30	T
NS 1103	Pakistan	White	20.00	MR	50.35	HS	35.18	MS
NS 11404	Pakistan	White	15.06	MR	15.48	MR	15.27	MR
NS 11504	Pakistan	White	8.69	R	35.00	MS	21.85	T
NS 11704	Pakistan	White	5.30	R	20.00	MR	12.65	MR
NS 12004	Pakistan	White	17.71	MR	31.00	MS	24.34	T
NS 12104	Pakistan	White	18.18	MR	39.63	MS	28.91	T

HR = highly resistant; R = resistant; MR = moderately resistant; T = tolerant; MS = moderately susceptible; S = susceptible; HS = highly susceptible

Results and Discussion

The development of cultivars with durable resistance to phyllody should be an integral component of sesame breeding programmes (Sarwar & Akhtar, 2009; Rajeswari *et al.*, 2010). The leafhoppers *O. albicinctus* appears approximately three weeks after germination on yellow sticky traps and then its population keeps on increasing during the growth period of sesame. As high as 10-15 leafhoppers were counted on individual traps per week. The first appearance of disease was recorded as reduction in leaf size in a few genotypes four weeks after germination, which later on developed in to twisted reduced leaves closely arranged on the top of the stem, with very short internodes and plants become stunted with no capsule formation. Most of the plants for all genotypes got infected during six to eight weeks after germination except the three genotypes namely NS98002-04, NS98003-04 and NS99005-01 (Table 1). All the plants infected during this period showed severe floral phyllody, viriscence and proliferation. The severity of the transformation of floral parts into malformed structures was correlated with time of infection. It was observed that the plants infected before flower initiation had severe symptoms on the entire plant and showed complete sterility. However, plants infected during flowering had severe symptoms on the upper of the plants, occasionally followed by some rudimentary flowers that yielded very small capsules with degenerated seeds as earlier recorded by Akhtar *et al.*, (2009). The percentage of infection during 2007 varied from zero to 65.12% under field conditions. The maximum number of genotypes was tolerant (45) followed by moderately resistant (28), moderately susceptible (18), susceptible (17), resistant (11), highly susceptible (11) and highly resistant (3). Maximum phyllody percentage was observed in genotype S-30-10 (65.12%) and minimum in NS97001-04 (3.12%). Three white seeded genotypes namely NS 98002-04, NS 98003-04 and NS 99005-01 remained symptomless throughout the season. Eleven other white seeded genotypes namely NS97001-04, NS01004-04, Sumboonkkae, NS940051-04, NS20005-04, NS 11704, NS96019-04, Ahnsankkac, NS 11504, Hansumkkae and NS99006-04 were ranked as resistant with percent disease infection of 3.12, 3.33, 3.40, 3.45, 5.0, 5.30, 5.88, 7.14, 8.69, 8.70 and 10% respectively. The findings from present study are in line with the results from previous studies by other researchers in different countries using limited number of entries against unknown phytoplasma (Shambharkar *et al.*, 1997; Selvanarayanan & Selvamuthukumar, 2000; Singh *et al.*, 2007; Rajeswari *et al.*, 2010). However, present study represents a comprehensive evaluation of disease levels of large number of sesame entries with diverse genetic background against 16SrII-D for the first time.

In 2008, maximum number of genotypes was highly susceptible (67), followed by moderately susceptible (24), tolerant (15), susceptible (14), moderately resistant (9) and resistant (4). None of the tested genotypes was found to be highly resistant in 2008. Of the three highly resistant and eleven resistant genotypes during 2007 only four genotypes viz; NS98002-04 (3.25), NS98003-04 (3.25),

NS99005-01 (3.75) and NS01004-04 (10.0) were resistant. Resistant genotypes developed infection at late stages and showed minor severity as disease was restricted at the top portion of plants. The percentage of infection during 2008 varied from 3.25 to 85.33%. The most of the genotypes evaluated in this study during 2007 & 2008 showed a wide variation in response and differed significantly in observed levels of phyllody infection in both seasons. These variable levels may be because of the lack of a single factor or a combination of factors such as spatial and temporal variation in inoculum levels, environmental conditions, vector host preference, host resistance to vector, age of plants, soil conditions etc (Hoogstraten 1992; Akhtar *et al.*, 2010; 2012).

Analysis of combined data showed that there were highly significant differences in the infection of all genotypes (Table 1). On the basis of percent infection values none of the genotype was ranked as highly resistant while 7 were resistant, 9 were moderately resistant, 28 were tolerant, 33 were moderately susceptible, 23 were susceptible and 33 were highly susceptible.

In conclusion, tested genotypes did not show total resistance, however, a clear difference in the degree of resistance was noted between the genotypes. Sources of partial resistance against phyllody disease are available in four genotypes (NS98002-04, NS98003-04, NS99005-01 and NS01004-04). These genotypes were resistant to highly resistant during both years, when inoculum level was high. These genotypes seem to have some significant stability for resistance of infection with phytoplasma. Breeders might consider them as a source of resistance in breeding programme or may directly be prompted after confirming their desirable market types. In future there is a need to continue the search for highly resistant sources in additional sesame germplasm and closely related species.

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