

CHARCOAL ROT RESISTANCE IN SUNFLOWER CULTIVARS AT VARIOUS PLANT DENSITIES

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A field experiment was conducted on six sunflower cultivars to study the effects of four plant densities (28 405, 42 978, 56 810 and 85 956 plants/ ha) on the incidence of charcoal rot. The results indicated that plant density within the commercial range is not a significant factor affecting disease incidence. None of the sunflower cultivar were completely resistant to the disease. However, two commercial sunflower hybrids, NK-212 and SF-100, had a lower plant mortality of 15.55 and 16.18%, respectively. The sunflower hybrid, Cargill 204 was the most susceptible with 48.25% plants killed.

Key words: Macrophomina phaseolina, Plant density, Resistance to charcoal rot.

INTRODUCTION

Charcoal rot disease, caused by *Macrophomina phaseolina* (Tassi) Goid. [*M. phaseoli* (Maubl. Ashby)], is one of the most important diseases of sunflower in Pakistan and is a potential threat to sunflower growers. Beside continuous cultivation of sunflower, the occurrence of this disease also appears to be related to extremely hot and dry weather conditions [1,2,10]. Charcoal rot affected plants are characterised by a grey-to-black discolouration at the base of the stem, which extends upward thus hollowing the interior portions of the stem. Later the pith become shriveled and discoloured [2,6].

Sunflower cultivars exhibit variable amounts of resistance to *M. Phaseolina* [3,4]. Some interspecific sunflower hybrids derived from *Helianthus tuberosus* x *H. annuus* have been observed to have the highest resistance [10]. However, early sunflower cultivars appeared to be highly susceptible to charcoal rot fungus [8]. The influence of plant density on charcoal rot infection in sunflower has not received proper attention as yet. Therefore, the major objective of this study was to determine the incidence of charcoal rot on different sunflower cultivars planted under various plant densities.

MATERIALS AND METHODS

Six sunflower cultivars were evaluated for their reaction against *Macrophomina phaseolina* fungus under four plant density conditions, i.e., 28 405, 42 978, 56 810, and 85 956 plants/ha. The experiment was planted during the spring 1987 (February 15, 1987).

Four commercial sunflower hybrids viz: NK-212, Hysun-33, SF-100 and Cargill 204; and two selected lines from Shamas and Vniimk sunflower cultivars were used

in the study. The experimental design used was a randomized complete block design in split-plot layout replicated three times. The main plot consisted of the six sunflower cultivars and different plant densities were placed in sub-plots. Each entry was planted as a single row plot 8.84 m long with rows spaced 0.76m apart. During planting, three to four grains of sorghum infested with *M. phaseolina* fungus were dropped per hill along with sunflower seed 2-3 cm below the soil. Plots were over-sown and thinned at the V2 stage [12].

Charcoal rot disease symptoms appeared when the plants were approaching maturity. Therefore, data on disease incidence were recorded only once, at maturity. Susceptibility of sunflower cultivars was based on enumerated data on percent of plants killed. The data were subjected to analysis of variance and Duncan's new multiple range test [13] was used to test for statistical significance among sunflower cultivars.

RESULTS AND DISCUSSION

Mean squares from the analysis of variance (Table 1) indicated that the differences in resistance among sunflower cultivars were highly significant. It could safely be stated that genetic differences in charcoal rot susceptibility occurred among the sunflower cultivars. Similar results were reported by a number of researchers [3,8,9]. However, plant density did not effect disease incidence. A good agreement among plant densities used in this study in estimating the charcoal rot disease reaction was indicated by lack of a significant cultivar x density interaction (Table 1).

The mean disease incidence by *M. phaseolina* ranged from 8.3 to 56.7% across all treatments (Table 2). Whereas among sunflower cultivars the mean plant mortality percentage ranged between 15.55 to 48.25. The present results with regard to percent-plant mortality by this

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Table 1. Analysis of variance for percent plants killed by *Macrophomina phaseolina* on six sunflower cultivars planted at four plant densities.

Source	df	Mean squares	Percent killed plants
Replication	2	68.832	NS
Cultivars	5	2142.433	**
Error (a)	10	153.611	
Density	3	78.298	NS
Cultivar x density	15	106.902	NS
Error (b)	36	69.965	

**Significant at the 1% probability level NS: Non-significant.

Table 2. Percent plants killed by *Macrophomina phaseolina* in six sunflower cultivars under four plant density conditions.

Cultivars	Plant densities (plants/ha.)				Means*
	28 405	42 978	56 810	85 956	
Cargill-204	43.8	38.0	56.7	54.5	48.25d
Hysun-33	15.1	22.9	16.1	23.2	19.33 ab
NK-212	18.3	18.0	17.1	8.8	15.55a
SF-100	8.3	22.1	18.1	16.2	16.18a
Shamas	37.9	29.3	45.6	41.0	38.45cd
Vni imk	28.7	32.8	27.3	29.4	29.55bc

*Means followed by the same letter are not significantly different by Duncan's new multiple range test at the 5% probability level.

fungus during spring planting of sunflower are in close agreement with the results reported by Mirza *et al.* [9]. Three commercial sunflower hybrids, NK-212, SF-100 and Hysun-33 with overall disease incidences of 15.55, 16.18 and 19.33%, respectively, showed the greatest resistance of the six sunflower cultivars that were evaluated (Table 2). The percent plants killed for sunflower hybrids NK-212 and SF-100 were about one-third of that in the highly susceptible cultivar, Cargill 204 (48.25%). In the literature, the favourable effect of different plant spacing not densities on the incidence of charcoal rot in sunflower cultivars

is evident [5] as disease significantly decrease at 20 cm than those at 30cm.

In the present study the symptoms of charcoal rot disease appeared when plants approached maturity. This is in agreement to the findings of Jimenez-Diaz and Blanco-Lopez [7]. Therefore, the present findings suggest the possibility that beside climatic factors, decreased root activity and a greater root volume near crop maturity and particularly after flowering favours infection and spread of the pathogen. However, this idea needs to be further investigated.

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