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Original Research Article

Epidemiology of Sunflower Necrosis Disease

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A B S T R A C T

The field experiment was conducted at Department of Plant Pathology, College of Agriculture, Latur during the year 2011-12. The epidemiological studies *viz*, yield losses, vector population dynamics and disease incidence. The disease causing sunflower necrosis of sunflower has been found to cause heavy yield losses i.e. 95.98 if the infection takes place at early growth period i.e. 30 days after sowing, Vector population level were higher (2.66/ leaf) during first fortnight of September than rest of the season. The first incidence of the disease was recorded 30 days after sowing, the incidence of the disease occurred throughout the growing season of sunflower. However, the outbreak was not uniform throughout the season. Disease incidence was more (11.5%) during second fortnight of September than rest of the fortnight of different months. The correlation between vector population dynamics and disease incidence was positively correlated and found very weak.

Introduction

Sunflower (*Helianthus annus* L.) is an important oilseed crop of India. It is one of the fastest growing annual oilseed crops in India. In India area under sunflower crop was 18.50 lakh hectares with production of 13.50 lakh tones and productivity of 729 kg ha^{-1} (Anon, 2010). Sunflower suffers on the account of many diseases caused by fungi, bacteria and viruses. Among these diseases sunflower necrosis is one of most important in India including Maharashtra. The yield losses caused due to sunflower necrosis disease were reported to the extent of 30-100 per cent (Rao *et al.,* 2000). In Latur district, sunflower necrosis disease was found to occur in severe form causing yield losses to the tune of 20 to 80 per cent (Shirshikar, 2003).

Most of the sunflower hybrids currently under cultivation in India have shown various degrees of susceptibility to the disease. Attempts have been made to manage this disease of viral origin. The epidemiology of causal virus is essential for studying its management. Therefore present investigation has been undertaken keeping in view the economic importance of the crop and yield losses due to necrosis disease in sunflower.

Materials and Methods

To study the epidemiology of sunflower necrosis disease, a non-replicated field experiment was conducted at College of Agriculture, Latur during the year 2011-12.

The losses caused due to infection of sunflower necrosis virus were assessed in *Kharif*, 2011 by comparing average yield of individual 10 healthy plants with virus infected plants.

Plants infected with sunflower necrosis virus at 15, 30, 45, 60, 75 and 90 days after sowing were tagged. Observations *viz.* plant height, head diameter, seeds per head, 100 seed weight and average yield (g/ plant) were recorded from infected plants tagged at different growth stages. Assessment of loss due to disease was done on the basis of average yield per plant and expressed in terms of percentage.

Thrips count was recorded early in the morning before 8 am on top, middle and bottom leaves of 5 randomly selected plants. Thrips count was recorded from 7 to 90 days after sowing with an interval of 7 days as per the method modified by Sipell *et al.*, (1982), The transformation of vector population was done by using Poison formula $\sqrt{x} + 0.5$, where x is the average number of vectors and analyzed statistically.

The plants showing infection of sunflower necrosis disease were observed from 15 to 90 days and per cent disease incidence was calculated.

An apparent infection rate (r) is the speedometer of epidemics of the plant diseases as described by Vander Plank (1963). Apparent infection rate was calculated to measure the epidemics of disease and to determine the vulnerable stage of the crop. To work out apparent infection rate (r) of the disease, observations on disease incidence recorded were used. Based on infections recorded at an interval of 15 days an apparent infection rate r = dx /x (1-x) was calculated according to Vander Plank's equation (1963).

$$r = \frac{2.3}{t_2 - t_1} \log10 \frac{X_2 (1-X_1)}{X_1 (1-X_2)}$$

Where,

r = Apparent infection rate

$t_2 - t_1$ = Days between first observation and subsequent observations

X_1 and X_2= Amount of disease (%/100) on t_1 and t_2 dates, respectively.

To study the effect of climatic factor on vector population dynamics and disease incidence correlation between weather parameter like temperature, relative humidity, rainfall, rainy days and wind velocity on thrips population and disease incidence were worked out.

Results and Discussion

The data presented in table 1 indicated that the significant reduction in plant height, head diameter, number of seeds per head, test weight, yield per plant and yield loss in the plants were affected at various stages over healthy. The plant height was reduced in infected plants and it was found more in the plants infected at 30 days (52.40 cm) than at 45 (79.42 cm), 60 (90.56cm) and 75 days after sowing (117.10) as against of healthy plants (132.57 cm). The head diameter varied from 5.20, 6.43, 7.22 and 8.89 cm in plants infected at 30, 45, 60 and 75 days after sowing, respectively as compared to 10.13 cm in healthy plants. The number of seeds per head in the plants infected at 30, 45, 60 and 75 days after sowing were 52.41, 391.23, 415.20 and 510.31, respectively in comparison to 640.60 seeds in healthy heads. The seeds in the early infected plants were small, discoloured, shriveled and chaffy as compared to healthy seeds. There was severe reduction in weight of the seeds. The 100-seed weight from early infected heads (30 days after sowing) was 3.20 g as against 6.50 g from healthy ones.

The time of infection affected also the yield per plant. More reduction in yield was evident in plants infected at early growth stages. Per plant yield reduction was decreased with delay in infection. Drastic reduction in yield (1.67 g/plant) was recorded in early infected plants than plants infected at 45 days (14.35 g/plant), 60 days (17.56 g/plant) and at 75 days (27.70 g/plant) as compared to healthy plant (41.63 g/plant). The time of infection has marked effect on yield losses of sunflower. The plants infected at 30 days after sowing evidenced heavy losses i.e. (95.98 per cent). Yield losses were found to reduced with delay in infection. The plants infected at 45, 60 and 75 days after sowing recorded 65.52, 57.81 and 33.46 per cent losses, respectively. Rao and Nagaraju (1999) reported that infection of sunflower at the vegetative stage by necrosis virus lead to complete loss of the crop. The disease causing sunflower necrosis of sunflower has been found to cause heavy yield losses i.e. 95.98 if the infection takes place at early growth period i.e. 30 days after sowing. The losses reduced with delay in infection. The disease has been reported cause losses in yield ranging from 30-100 per cent depending upon time of infection (Rao *et al.,* 2000 and Anon, 2001).

The results table 2 obtained on thrips population dynamics and necrosis disease incidence indicated that the occurrence of the vector was started from 21 days after sowing and was found throughout the season i.e. from August to first fortnight of October. The thrips population dynamics was negligible at initial growth stage and it was increased with age of the crop and decreased in later stage of growth. However, vector population was higher (2.66/ leaf) during first fortnight of September than rest of the season. The first incidence of the disease was recorded at 30 days after sowing and it was occurred throughout the growing season. However, the outbreak was not uniform throughout the season. Disease incidence was more (11.5%) during second fortnight of September than rest of the months.

The correlation between vector population dynamics and disease incidence was found to be positive and weak. Epidemiological studies have been carried out by Directorate of Oilseed Research (DOR), Hyderabad and at Coimbatore centre in August sown crop, the incidence ranged from 2.9 to 13.3 per cent in September sown crop from 2.7 to 16.2 per cent. At Raichur centre incidence ranged from 6.8 to 16.5 per cent and 9.9 to 21.3 per cent during July and August sown crop, respectively (Anon, 2000).

Shirshikar (2002), recorded that sunflower sown in July, January and February had maximum disease incidence. However, September, October, November and December sowings showed relatively lower necrosis incidence (less than 4%).

The data on apparent infection rate (r) presented in table 3 revealed that virus caused infection to sunflower throughout its growing period which it indicated that the crop was vulnerable to the necrosis disease at all growth stages. However, the early growth stage i.e. 30 to 45 days after sowing were found to be highly vulnerable as in this growth period the higher infection rate (r=0.024) was recorded. The infection rate was decreased with increased age of the crop.

Table.1 Yield and yield contributing parameters as influenced by the time of sunflower necrosis infection in Cv. KBSH-44 during *Kharif*, 2011

Infection stages (DAS)	**Plant height (cm)**	**Head diameter (cm)**	**Seeds per head**	**100 seed weight(g)**	**Yield per plant (g)**	**Yield losses (%)**
30	52.40	5.20	52.41	3.20	1.67	95.98
45	79.42	6.43	391.23	3.67	14.35	65.52
60	90.56	7.22	415.20	4.23	17.56	57.81
75	117.1	8.89	510.31	5.43	27.70	3.46
Healthy	132.57	10.13	640.60	6.50	41.63	--

DAS= days after sowing

Table.2 Thrips population dynamics in relation to incidence of sunflower necrosis in Cv. KBSH-44

Observation number for vector	Days after sowing	Date of observation	Number of thrips / leaf	Observation for disease incidence	Days after sowing	Date of observation	Incidence
1	7	28th July	0	-	-	-	-
2	14	5th Aug	0	1	15	5th Aug	0
3	21	12th Aug	1.37				
4	28	19th Aug	2.12	2	30	19th Aug	2.5
5	35	26th Aug	2.38				
6	49	2 th Sep	2.52	3	45	2th Sep	5
7	56	9th Sep	2.62				
8	63	16th Sep	2.66	4	60	16th Sep	8.5
9	70	23th Sep	2.5				
10	77	30th Sep	1.81	5	75	30th Sep	11.5
11	84	7th Oct	1.71				
12	90	14th Oct	1.44	6	90	14th Oct	3.5

Table.3 Sunflower necrosis development and apparent infection rate (r) at different growth stages of sunflower Cv. KBSH- 44

Disease incidence (%) days after sowing (cumulative)					Apparent infection rate (r)* unit per day at different growth stages			
30	45	60	75	90	30 - 45	45 - 60	60 - 75	75 - 90
2.5	7.5	16	27.5	31	0.024	0.0054	0.0019	0.00065

$$* \quad r = \frac{2.3}{t_2 - t_1} \log 10 \frac{X_2 (1-X_1)}{X_1 (1-X_2)}$$

Table.4a Correlation between weather parameters and thrips population dynamics in sunflower Cv. KBSH-44

Weather parameters	Correlation co-efficient (r)
Rainfall (mm)	0.082
Rainy days	0.017
Temperature (Max)^{o} C	-0.697*
Temperature (Min)^{o} C	-0.334
Relative humidity (%) AM	0.289
Relative humidity (%) PM	0.376
Wind velocity (kmph)	-0.322

* = significant at 5%

Table.4b Correlation between weather parameters and necrosis incidence in sunflower Cv.KBSH-44

Weather parameters	Correlation co-efficient (r)
Rainfall (mm)	-0.297
Rainy days	-0.166
Temperature(Max)^{o} C	0.026
Temperature(Min)^{o} C	-0.547
Relative humidity (%) AM	-0.331
Relative humidity (%) PM	-0.381
Wind velocity (kmph)	-0.519

The sunflower necrosis disease progress curve revealed that the progress of the disease in the season was not uniform.

The disease development in sunflower was initiated from 30 days after sowing and it prevailed throughout the season. The disease progress curve had clearly indicated that the disease developed very slowly at early crop growth stages. Later the rate of development increased with increase in age of the crop.

The results (Table 4a) revealed that all the weather parameters had positive correlation with vector population dynamics except temperature (max and min) and wind velocity (kmph) had negative correlated with vector population dynamics. Among all correlations temperature (max) has significantly negative correlated with thrips population dynamics, it means that the increase in maximum temperature decreases the thrips population dynamics.

The data presented in table 4b revealed that all the weather parameters *viz*; minimum temperatures (^{0}c), per cent relative humidity (am and pm.), rainfall (mm), number of rainy days and wind velocity (kmph) were found negatively correlated with the disease incidence. Maximum temperature had positive correlation with disease incidence. However, both positive and negative correlations between weather parameters and disease incidence were non-conclusive.

Shivasharanayya (2000) and Shirshikar (2002) reported that minimum and maximum temperature, thrips population and disease is positively correlated and thrips population a negatively correlated with rainfall. The correlation matrix on thrips count and weather parameters and virus disease incidence revealed that there was a positive correlation of mean number of thrips with maximum and minimum temperatures, bright sunshine hours and also with the disease incidence (Shivasharanayya and Nagaraju, 2003). Singh (2005) reported positively correlation between thrips population and necrosis incidence and observed highest incidence during the prolonged dry spell prevailed immediately after heavy rains.

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