

Screening of Sesame (*Sesamum indicum* L.) Genotypes for Powdery Mildew Resistance

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Abstract: Powdery mildew is the most devastating disease in Sesame throughout India, causing considerable yield loss. So an experiment was conducted to identify the most resistance genotype to powdery mildew because, host plant resistance is the cheapest, and most effective disease management strategy. In the present investigation 37 genotypes along with a susceptible local checks were screened against powdery mildew under natural conditions following infector row technique. Nineteen genotypes showed susceptible and ten showed moderately resistant reaction. Only eight genotypes (SSD-4, SSD-7, SSD-19, SSD-20, VRI-1, Co-1, T-12 and N-32) showed resistant reaction. None of the genotypes recorded immune response. The resistant genotypes can be utilized in breeding program to evolve resistant varieties

Keywords: Sesame, powdery mildew, screening, resistance.

INTRODUCTION

Sesame (*Sesamum indicum* L.) is considered as 'Queen of oilseeds' as the quality of its oil is of high nutritional and therapeutic value. High stability of its oil with distinct sweet flavor and oil meal rich in protein, have made it an obvious choice for domestic and confectionary uses, respectively. Sesame is inherently low yielding plant type. Its yield is further limited by various biotic and abiotic stresses. Among all the pests and diseases, powdery mildew is a devastating disease in all the sesame growing states in general, and Telangana, Andhra Pradesh and Tamil Nadu in particular. The first report on incidence of powdery mildew in India was by Patel *et. al.* (1949) and Mehta (1951). Infection first appears as small white patches on upper surface of mature plants after 40 DAS.

Further it causes surface leaf necrosis, premature leaf fall, stunted plant growth, chlorosis of leaves and browning of flower buds. It is caused by many species of fungi, *viz.* *Erisiphe cichoreacearum*

(Reddy and Haripriya, 1990), *Erisiphe orontii* (Rajpurohit, 1993), *Leveillula taurica* (Patel *et. al.*, 1949), *Oidium erysiphoides* (Mehta, 1951; Roy, 1965), and *Oidium sesami* (Puzari *et. al.*, 2006). It occurs on epidemic scale in areas of high rainfall and humidity coupled with low night temperature. In Telangana the low temperatures coupled with crop flowering stage is causing higher losses both in late kharif and rabi.

The farmers of telangana state cultivate sesame as main and also contingency crop especially in failure of main crops like cotton, maize and paddy due to inconsistent monsoon. The disease causes yield loss between 25 and 50% depending on the level of severity. Use of pesticides to control the disease will increase the cost of cultivation and also hazardous to the human health. Apart from this use of pesticides on sesame limits exports. Host plant resistance is a most reliable and permanent disease management strategy, very little is known on gene sources and their level of tolerance. Though few wild sources like *S. malabaricum* and *S. mulayanum*

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seems to possess tolerance to powdery mildew and phyllody, difficulties encountered in recombining such gene sources from wild relatives and lack of reliable screening/selection techniques and their low yield potentials and suitability to all regions. In the present investigation all efforts were made to identify sources of resistance to powdery mildew under field conditions at All India Coordinated Research Project, Regional Agricultural Research Station, Jagtial, Telangana State.

MATERIALS AND METHODS

Thirty seven genotypes comprising germplasm accessions from research station, Indian bred improved varieties and advanced breeding lines (AVT/IVT), and a susceptible local check constituted the experimental material for screening of resistance against powdery mildew. The experimental material was screened during late *Kharif* at Regional Agricultural Research Station at Jagtial, Telangana State in India. Infector row technique was followed to spread the disease intensively. Infector rows were sown in 15 days advance of screening material at after every fourth row with susceptible check so as to establish and continuously supply of the powdery mildew pathogen inoculums on to germinating genotypes.

In addition four rows of the susceptible check was also raised all around the experimental plot to provide the disease inoculum facilitating screening of the genotypes under field conditions.. Each of the genotypes was sown in two rows of 3 m length with 30 × 15 cm spacing and replicated thrice. The crop was raised adopting the all recommended package of practices. The screening disease data was recorded at 50 to 60 days after sowing (DAS) when the disease incidence was maximum on the susceptible check. Observation on disease reaction was made on five randomly selected plants in each entry. Nine leaves were scored in each plant, three each from the apical, middle and basal regions, and all of them were graded. The disease intensity was scored (Table 1) adopting the following 0-9 grade (TNAU, 1980).

Level of resistance/susceptibility of the entries to the disease was determined by Percent Disease

Table 1
Grading of powdery mildew disease intensity

| <i>Disease grade</i> | <i>Description</i> |
|----------------------|---|
| 0 | No lesions or specks |
| 1 | Small sized powdery specks infecting less than 1% leaf area |
| 3 | Enlarged irregular powdery growth covering 1-5% leaf area |
| 5 | Powdery growth to form big patches covering 5-25% leaf area |
| 7 | Powdery growth covering 25-50% leaf area followed by yellowing |
| 9 | 100% leaf area covered with powdery growth, yellowing and dropping of infected leaves |

Index (PDI) following the formula of Mc Kinney (1923).

Per cent disease index (PDI)

$$= \frac{\text{Sum of grades}}{\text{Total number of leaves analyzed} \times \text{maximum disease grade}} \times 100$$

Sum of grades is the sum of disease grade on nine leaves on which observation was recorded and maximum disease grade was nine in 0-9 scale (Table 2). On the basis of the PDI, the entries were grouped into four categories (Raja Ravindran, 1990).

Table 2
Classification of the entries based on Percent Disease Index (PDI)

| <i>PDI</i> | <i>Disease reaction</i> |
|------------|--|
| 0 | Immune (I) |
| 1-30 | Resistant (R) |
| 31-50 | Moderately resistant (MR)/tolerant (T) |
| ≥ 50 | Susceptible (S) |

RESULTS AND DISCUSSION

A set of 37 entries of sesame were screened for powdery mildew reaction under field conditions using infector row technique. Out of the 37 entries tested, 19 genotypes were found to be susceptible to powdery mildew (PDI 50.64 to 92.90), ten were tolerant (PDI 30.06 to 48.54%), while, eight were resistant (SSD-4, SSD-7, SSD-19, SSD-20, VRI-1, Co-1, T-12 and N-32) (PDI 7.88 to-16.54%) (Table 3). The

Table 3
Reaction of 37 genotypes to powdery mildew disease

| S. No. | Genotype | Percent Disease Incidence (mean of two seasons) | Reaction |
|--------|------------|---|----------------------|
| 1. | SSD-10 | 33.32 | Moderately resistant |
| 2. | MT-23-3 | 78.03 | Susceptible |
| 3. | AT-231 | 83.21 | Susceptible |
| 4. | MT-10-8-2 | 90.08 | Susceptible |
| 5. | SSD-19 | 7.88 | Resistant |
| 6. | RT-363 | 92.32 | Susceptible |
| 7. | MT-10-8-1 | 64.35 | Susceptible |
| 8. | JLS-301-24 | 58.54 | Susceptible |
| 9. | AT-201 | 56.32 | Susceptible |
| 10. | RT-362 | 67.20 | Susceptible |
| 11. | SSD-20 | 8.54 | Resistant |
| 12. | AT-235 | 70.32 | Susceptible |
| 13. | SSD-7 | 9.70 | Resistant |
| 14. | BSV-1 | 30.56 | Moderately resistant |
| 15. | TKG-431 | 32.60 | Moderately resistant |
| 16. | CST-2001-1 | 92.90 | Susceptible |
| 17. | CST-2008-1 | 84.7 | Susceptible |
| 18. | TKG-412-1 | 32.64 | Moderately resistant |
| 19. | AT-159 | 34.50 | Moderately resistant |
| 20. | JLS-110-12 | 32.22 | Moderately resistant |
| 21. | RT-354 | 88.40 | Susceptible |
| 22. | JLS-9707-2 | 80.26 | Susceptible |
| 23. | SSD-4 | 15.65 | Resistant |
| 24. | SSD-3 | 30.24 | Moderately resistant |
| 25. | HT-9316 | 86.44 | Susceptible |
| 26. | TKG-301 | 52.40 | Susceptible |
| 27. | VS-07-034 | 32.36 | Moderately resistant |
| 28. | CST-2001-9 | 34.54 | Moderately resistant |
| 29. | CST-2008-2 | 50.64 | Susceptible |
| 30. | TKG-22 | 84.26 | Susceptible |
| 31. | RT-54 | 92.36 | Susceptible |
| 32. | Pragathi | 86.70 | Susceptible |
| 33. | VRI-1 | 10.28 | Resistant |
| 34. | Phule Til | 32.60 | Moderately resistant |
| 35. | Co-1 | 12.32 | Resistant |
| 36. | T-12 | 8.60 | Resistant |
| 37. | N-32 | 16.54 | Resistant |

level of resistance and susceptibility varied with the genotypes. Among the susceptibles entries, if the level of disease incidence was more than 80%, susceptible check was found to be highly susceptible

with the incidence level exceeding 90%. Interestingly, none of the entry was found to be immune suggesting lack of strong sources of resistance to the disease and these findings broadly agree with many earlier reports by pathologists and breeders that no reliable source of resistance/immunity could be found (Karunanithi *et al.*, 1993; Rajpurohit, 1993; Karunanithi and Dinakaran, 1996), a few have reported existence of resistant sources (Hiremath, 1976; Suresh *et al.*, 1991; Ganesh *et al.*, 1992, Venkata ramana rao *et al* 2011). The contradictory observations may be due to differences in the disease scaling, screening techniques adopted, species/and race spectrum. The difference in disease rating may be attributed to stringent screening method (spreader row + dusting of spore inoculum artificially) in the present case as against natural infection adopted by Gopal *et al.* (2005).

Studies by Shaner (1973) and Berger (1981) revealed growth rate of plant to be useful in differentiating genotypes with regard to infection rate and disease build up. Duration of the crop is yet another factor that influences the level of susceptibility/tolerance reaction. It was observed in the present study that early maturing genotypes were relatively more susceptible to the disease as compared to the late maturing in conformity with the earlier reports by Kolte (1985) and Hiremath (1976). Also, some agro-botanic traits appear to influence the disease spread. For instance, genotypes having horizontal leaf angle were found to be more susceptible to the disease as compared to those with acute leaf angle. This might be due to large exposure of leaf area to conidial spores unlike that of genotypes with acute leaf angle.

CONCLUSION

In the present investigation, none of the genotypes recorded immune response, and only eight genotypes (SSD-4, SSD-7, SSD-19, SSD-20, VRI-1, Co-1, T-12 and N-32) recorded resistant reaction. Such resistant genotypes can be utilized in breeding program to evolve resistant varieties.

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