Inheritance of Powdery Mildew Resistance in Sesame (*Sesamum indicum* L.)- a Review

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Abstract

Powdery mildew is an important disease of sesame (*Sesamum indicum* L.) causing upto 50% yield loss. It occurs on epidemic scale in areas of high rainfall and humidity coupled with low night temperature. The disease is generally managed by application of sulphur dusting and other chemicals. Use of host plant resistance is the pragmatic approach to manage the disease. For development of powdery mildew resistant/tolerant varieties/hybrids, identification of sources of resistance/tolerance and knowledge of its inheritance pattern are essential. However, reliable screening procedures are lacking. Even though wild sources like *Sesamum malabaricum*, *Sesamum mulayanum, Sesamum prostratum* etc. possess the tolerance, so far no tangible progress is made. Use of advanced technologies like molecular markers will be useful to map resistance/ tolerance gene(s) against the disease is the prerequisite which can help in the breeding programme.

1. Introduction

Sesame (*Sesamum indicum* L.) also known as sesamum, til, gingelly, simsin, gergelim etc. is the most ancient oilseed crop of the world. It is being cultivated in Asia since last 5000 years. Belonging to the family *Pedaliaceae*, it is regarded as the *Queen of Oilseeds*, the quality of its oil being of high nutritional and therapeutic value. Inherently low yield potential apart, biotic and abiotic stresses constitute the major yield destabilizing factors do not as well realize the full potential of the currently available varieties. Powdery mildew and phyllody among diseases and capsule borer among insect pests contribute significantly to yield losses. Powdery mildew is a devastative important disease in all the sesame growing states of the country and Andhra Pradesh and Tamilnadu in particular. It occurs on epidemic scale in areas of high rainfall and humidity coupled with low night temperature. The disease causes yield losses between 25 and 50% depending on the level of incidence. The present review includes the etiology, symptoms, sources of tolerance and the genetics of powdery mildew tolerance.

2. Causal Organism

It is caused by many species of the fungi viz., *Erisiphe cichorecearum* (Reddy and Haripriya, 1990), *Erisiphe orontii* (Rajpurohit, 1993), *Sphaerothica fuliginea* (Gemawat and Verma, 1972; Lawrence, 1951), *Leveillula taurica* (Patel et al., 1949), *Oidium erysiphoides* (Mehta, 1951 and Roy, 1965), *Oidium* sp. (Venkatakrishnaiya, 1958), *Oidium sesami* (Puzari et al., 2006) and *Oidium mirabilifolii* (Srinivasulu et al., 2003). It occurs in epidemic scale under heavy rainfall conditions followed by low night temperatures and high humidity. Powdery mildew causes yield losses ranging from 25 to 50% depending upon the level of severity. The first report on incidence of powdery mildew in India was by Patel et al. (1949) and Mehta (1951).

3. Fungal Morphology

3.1. Oidium

Maculae infectus, amphigenae, densissimus, mycelium hyalinae, celeriter effusae, 4-7 µm crassa, appressoriae lobatis, conidiophores erecta, cylindraceis, cellulo basalis erevta, conidia catenatis, cylindraceis, ovideis vel doliformiis, fibrosinis absentia, tubis germinativis simplicis, hypophodis gonylodibus. Infection spots on leaves, amphigenous, dense

evanescent to persistent, hyphae branched, septate wide, appressoria lobes, conidiophoires erect, cylindrical, conidia in chains, cylindrical, ovoid-doliform, fibrosin bodies absent, germ tube lobes appressorium at the tip.

3.2. Sphaerotheca

Mycelium is septate, superificial, hyaline, branched and 4-5 µm thickness, conidiophore erect, simple and septate bearing single celled hyaline, oval to elliptical conidia.

4. Fungal Infection

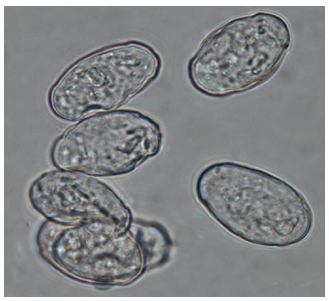
Life cycle of the disease initiates by air borne conidia measuring 25-36 µm in length and 14-18 µm breadth at 25± 1 ^{0}C and 100% R.H. Conidia starts to germinate in about 4 hours and germination is maximum at about 24 hr after incubation. The penetration of the host tissue occurs within 28 hr. The second germ tube is observed after 30 hr and third after 36 hr of incubation. Secondary elongating hyphae, onidiophores initials and abstriction of onidiophores is observed 60, 144 and 156 hr after incubation respectively. The colony produces maximum amount of conidia on ninth day after incubation. The colony remains productive even upto 12 days from the time of inoculation. (Figure 1 and 2)

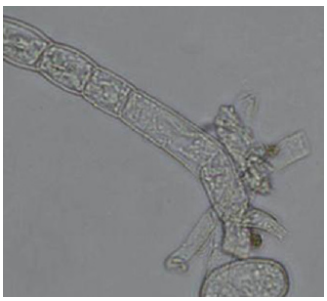
5. Symptoms

Powdery mildew infection on sesame plant has been apparent from seedling stage up to mature plants. All parts of the sesame plant viz., leaves, stem, flower buds and pods were found to be affected by this fungus. Leaves are the most susceptible tissue to fungal attack.

Incidence of mildew starts as small whitish spots on upper surface of the leaves at the age of 40 days or more. Under natural conditions the lower leaves developed infection first and from these the infection spread to the other leaves and finally to other parts of the plant. Depending on the favourable conditions, disease spreads to both the surfaces of lower leaves and lower surface of upper leaves (Figure 3).

These spots coalesce to form larger spots finally covering the entire leaf with dirty white fungal growth. Symptoms include surface leaf necrosis, premature leaf fall, stunted growth of the plant at early stage, yellowing and chlorosis of the leaf at





mature stage and browning of flower buds. Severely infected leaves drop off leaving bare stem. The affected plants produce shrivelled seeds and reducing thereby the yield.

6. Life Cycle of the Fungus

Germination, infection and sporulation follow each other in the life cycle. The life cycle of the fungus is initiated by airborne conidia which are the asexual reproductive spores of the fungus. When the conditions are favourable germination of the fungus is observed as direct germination of conidia. The conidia start to germinate after four hours and produces a single thick germtube from one corner of the conidia. Germinating conidia produces primary hyphae at about 30 hr after incubation, secondary hyphae after 36 hours and tertiary hyphae after 48 hours of incubation. After about 60 hours the other hyphae elongate. The white patches of infection became visible to the naked eye only after 120 hours. The mature conidia gets released about seven days after inoculation. The conidia gets released when the host leaf and the atmosphere are relatively dry during day time. Detached conidia gets carried away by the wind and gets deposited over the fresh host leaves on which they gets germinated again.

7. Favourable Season

Hazarika (1998) studied the influence of sowing dates and varieties on the development of powdery mildew and revealed that PDI in early sowings (August) was higher than late sowing (September). Also highest disease index was recorded in Gouri variety while it was lowest in Pb-Til-No.1. Dinakaran and Dharmalingam (1999) observed the incidence of the disease from 35 to 40 DAS and peak incidence was observed from 65 to 75 DAS. They reported that the incidence of the disease ranged from 60 (Co-1) to 95.8% (JT 7) under unprotected conditions.

Rao and Rao (2000) evaluated five commercially important varieties under natural conditions and concluded that powdery mildew incidence was observed between 53 to 55 DAS. Rao and Rao (2001) conducted a field experiment for three consecutive years using a popular variety Madhavi with two dates of sowing (D_1-first week of January and D_2-third week

of January) and reported that incidence of the disease ranged from 54.32 (D_1) to 62.96% (D_2).

8. Host Plant Resistance

The powdery mildew disease is managed to an extent in the initial stages with chemicals. But often the measure fails to give total protection against the disease. Host plant resistance is the preferred strategy to protect the crop from the disease as it's an ecofriendly approach. Hence, there is an urgent need to study on sources of resistance, genetics of resistance and rapid and reliable screening techniques. Also, information on race spectrum and area specific race(s) is needed. Though few wild sources like *S. malabaricum, S. mulayanum* seem to possess tolerance to powdery mildew, difficulties encountered in recombining such gene sources from wild relatives and lack of reliable screening/selection techniques had taken us nowhere near the targeted goal. The foregoing thus call for expeditious development and deployment of innovative breeding/selection approaches to find meaningful solution to them.

9. Disease Screening Methodology

The genotypes are to be sown in two to three rows of convenient length with recommended spacing. Late sowing has to be taken up as the disease appears in severe form in late planted crop under natural field conditions. Three rows of the susceptible check is to be raised all around the experimental plot to provide the disease inoculum facilitating screening of the entries under field conditions. To make the disease screening still more effective, the entries should be challenged artificially by treating with the inoculum of mycelial spores prepared from the diseased susceptible check. The screening is done (50 days after sowing) when the disease incidence was maximum on the susceptible check. Observations on disease reaction should be recorded on five randomly selected plants in each entry. Nine leaves in each plant, three each from the apical, middle and basal regions are to be scored. The disease intensity is to be scored adopting the following 0-9 grade (TNAU, 1980). Disease grade; 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.

Level of resistance/susceptibility of the entries to the disease is determined by Percentage Disease Index (PDI) following the formula of McKinney (1923).

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

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.

On the basis of the PDI, the entries can be grouped into the following four categories (Raja Ravindran, 1990)

Classification of the entries based on Per cent Disease Index (PDI) where 0 indicates immune (I), 1-30 for resistant (R), 31-50 for moderately resistant (MR)/ tolerant (T) and >51 for susceptible (S).

10. Resistance Sources

Hiremath (1976) evaluated 50 genotypes under natural conditions and reported two genotypes viz., Si-1926 and KRR 2 to be field immune while, 21 moderately resistant, 18 moderately susceptible and 9 susceptible. Studies by Vyas et al. (1983) revealed four accessions viz., TC 160, TC 289, TC 325 and BM 1-2 to exhibit field level tolerance to the disease. Of 32 breeding lines/varieties evaluated by Suresh et al. (1991) and four high yielding genotypes viz., Co-1, DPI 1523, DPI-1-1 and DPI-22-2 to be resistant. Evaluation of 225 genotypes under natural infection by Shadakshari et al. (1989) helped identify two resistant genotypes (ES-277 and IS-401). Dinakaran et al. (1989) studied 34 germplasm lines/cultivars along with five checks under natural field conditions and found six entries viz., SI 3315/11, VS 112, X 791/1/3, R/S 1, R/S 2 and 79-1-1 to be moderately resistant and none resistant.

Ganesh et al. (1992) screened 45 accessions along with susceptible check TMV 6 under rainfed and unprotected conditions for identification of resistant sources. The study revealed that six entries viz., Si 3170, PDK 30, Si 3315/11, Si 2671, Si 3315/5 and 59-1-1 to show field resistance to the disease.

In the subsequent evaluation of 41 accessions by Karunanithi et al. (1993) under natural and screen house conditions OMT 30 was the only entry found moderately tolerant. In his screening of 48 accessions Rajpurohit (1993) found only two entries viz., OMT-30 and DORS-101 to be tolerant. Mehetre et al. (1994) have reported *S. mulayanum*, a wild relative of sesame to be highly resistant to powdery mildew as well as phyllody. Karunanithi and Dinakaran (1996) evaluated a large collection of 600 germplasm under natural field conditions and identified 19 entries to exhibit field level tolerance to the disease. Of them, 5 entries viz., TNAU 17, VS 117, VS 9003, Si 3315/11 and DPI 1588 were found to be tolerant under screen house conditions.

Shamarao et al. (2003) evaluated nine entries for their multiple resistance to diseases and reported four genotypes viz., MT-15, DORS-102, DS-14 and DS-10 to show multiple disease resistance against powdery mildew, alternaria blight

and bacterial leaf blight. In the subsequent evaluation of nine accessions by Jahagirdar et al. (2003) four accessions viz., Mt-15, DORS-102, DS-14 and DS-10) to show multiple disease resistance against powdery mildew, alternaria blight and bacterial leaf blight diseases. Saravanan and Nadarajan (2004) evaluated eight parents (Co-1, TMV-3, VRI 1, SI 3216, YLM 123, SI 42, SVPR1 and AHT 123) and 28 F_1 hybrids for resistance to powdery mildew and observed that the parent Co-1 and two hybrids (Co-1×Si 3216 and Co-1×YLM 123) were moderately resistant recording less than 25% disease incidence.

Gopal et al. (2005) assessed forty two advanced genotypes of sesame for their reaction to powdery mildew under natural infection for two consecutive years. Seven genotypes were found resistant and nine genotypes moderately resistant. Sharmila and Ganesh (2008) evaluated two tolerant lines (VS 9701 and VS 9510), four testers (TMV 3, Co1, SVPR 1 and VRI 1) and eight hybrids for disease tolerance and reported lines VS 950 and Co1 were good general combiners for yield and powdery mildew tolerance and the hybrid VS 9510×Co 1 is highly heterotic and moderately resistant.

Ramana Rao et al. (2011) screened thirty seven genotypes comprising germplasm accessions, improved varieties and advanced breeding lines along with susceptible check Swetha til and reported that 24 genotypes were susceptible and 11 genotypes were tolerant whereas three genotypes (TKG 22, NSKMS-260 and G-55) showed resistant reaction to the disease.

11. Genetics of Resistance/Tolerance to Powdery Mildew

It has been studied by several workers both qualitatively and quantitatively in crosses involving known sources of resistance and moderate resistance and susceptible varieties. Krishnaswami et al. (1983) were one of the first to study genetics of resistance to powdery mildew in F_2 progenies of crosses involving susceptible and resistant parents. They concluded resistance to powdery mildew disease to be controlled by two major genes with complementary gene action.

Reddy and Haripriya (1990) reported from their study of a set of 36 F_1 hybrids, ten to show heterosis for tolerance to the disease while, five significant heterobeltiosis. In a series of crosses with RT 54 as resistant parent, all the hybrids were moderately tolerant indicating that it can be a donor parent in breeding for resistance against powdery mildew. Reddy and Haripriya (1993) studied 36 hybrids evolved from a 9×9 diallel cross and observed five F_1s to show significant heterosis and heterobeltiosis for tolerance to the disease. Raja Ravindran and Amritha Devarathinam (1996) studied F_2 progenies of 24 cross combinations involving Co-1 as resistant parent and reported resistant and susceptible plants to segregate in the ratio of 9:7 indicating resistance to be governed by two pairs of dominant genes showing complementary gene action. In a line×tester design, Kumaresan and Nadarajan (2002) studied 12 lines, 4 testers and 48 F_1s for their *per se* performance, heterosis and nature of gene action involved in the inheritance of powdery mildew. They observed that lines Si 3315/11 and OMT 30 and tester Co-1 recorded superior mean performance and desirable *gca* effect for powdery mildew resistance indicating that these three parents could be used as donor parents for transferring powdery mildew resistance.

While the 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; Shadakshari et al., 1989; Dinakaran et al., 1989; Suresh et al.,1991 and Ganesh et al., 1992). Gopal et al. (2005) for instance, have reported that of 42 genotypes screened 7 were resistant, 9 tolerant and the rest susceptible. The contradictory findings could be due to differences in the disease rating methodology, screening method species/and race spectrum as the reports are from different regions of Andhra Pradesh and Tamilnadu. 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. Also, general perception among breeders and pathologists is what the present study concluded on the existence of truly resistant sources against the disease. The differential reaction of genotypes to the pathogen at different regions however, need to be studied by pathologists for racial/species differences. Percentage disease index (PDI) at peak disease level distinguishes well the resistant from tolerant as tolerant types are characterized by slow mildewing. As well additional parameters are required to discriminate resistant from tolerant.

Studies by Shaner (1973), Berger (1981) reveal 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 has been 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 have been found to be more susceptible to the disease as compared to those with acute leaf angle. The higher susceptibility of genotypes with horizontal leaf angle may be due to large exposure of leaf area to conidial spores unlike that of genotypes with acute leaf angle. Based on the

disease reaction in F_1 hybrids involving RT 54, a moderately resistant parent, Reddy and Haripriya (1990) too have reported tolerance to be dominant. Kumaresan and Nadarajan (2002) observed that lines Si 3315/11 and OMT 30 and tester Co-1 recorded superior mean performance and desirable *gca* effect for powdery mildew resistance.

Ramana Rao et al., 2012 studied six populations viz., P_1, P_2, F_1, F_2, B_1 and B_2 of the cross swethatil×PKDS 37 and the inheritance of tolerance to powdery mildew is controlled by two independent recessive genes with complementary epistasis. Sravani et al., 2012 studied F_2 population of a cross between Swethatil and *S. mulyanum*, a wild accession and concluded that and the resistance to disease was governed by two pairs of recessive genes contributed by *S. mulayanum.* The findings that tolerance to the disease is recessive, are to an extent in agreement with studies on other crops that suffer from powdery mildew. Tolerance to the disease in pea has been reported to follow simple Mendelian mode of inheritance governed by recessive genes (Tiwari et al., 1998 and Janila, 1999). Same has been the case with barley, where resistance has been reported to be monogenic recessive against all the tested isolates of the fungus (Buschges et al., 1997).

12. Conclusion

All past efforts to raise the genetic yield level by conventional recombination breeding have hardly yielded anything tangible due to dependence of breeders on narrow cultivar genepool for desired variability and poor understanding of the physiology and genetics of yield related traits. Among the biotic stresses that cause serious crop losses wilt, powdery mildew, phyllody, capsule borer etc. are important. Powdery mildew, an important disease is reported to cause as high as 50% yield losses under favourable weather conditions. Though few wild sources like *S. malabaricum, S. mulayanum* seem 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 had taken us nowhere near the targeted goal. The foregoing thus call for expeditious development and deployment of innovative breeding/selection approaches to find meaningful solution to them.

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