LEAF BLIGHT *EXSEROHILUM TURCICUM* (PASS.)
OF SORGHUM – A REVIEW

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ABSTRACT

Leaf blight is one of the most widely distributed and at times damaging foliage pathogens of the sorghum. It is caused by *Exserohilum turcicum*, belongs to the class Deuteromycetes and the order *Fungi imperfecti*. It causes large, elongated, spindle-shaped spots, measuring several cm in length and up to few cm in width having straw/grey colored center and surrounded by a deeply pigmented margin, which darken during sporulation. With the advancement of disease several spots coalesce together killing large patches of the leaf blade. It primarily attacks leaf blade but under extended disease conducive environment it may also attack leaf sheaths and total leaf wilting occurs under heavy infected conditions.

Sorghum (*Sorghum bicolor* (L.) Moench) is grown widely in many countries and can be regarded either as the staple food or the second most important source of energy for the population. It is subjected to damage by several major, minor and location specific diseases that reduce grain and stover yields. It is attacked by several pathogens such as fungi, bacteria, viruses and the parasitic weed, *Striga hermonthica* (Del.) Benth.

There are variations in the relative importance of particular sorghum diseases from one area to another and from one season to the next. But certain diseases, such as grey leaf spot are widespread and endemic while others are particularly important in one area like sorghum downy mildew. Some diseases may become important only in a particular year and certain diseases are important at the beginning of the season, where as insignificant at the time of harvest, such as the case with anthracnose (Wall and Meckenstock, 1992).

Foliar diseases in sorghum are caused by viruses, bacteria, fungi and nematodes. Among these, diseases caused by fungi are more predominant than others. There are eight foliar fungal pathogens. These are *Cercospora sorghi*, *Colletotrichum graminicola*, *Exserohilum turcicum*, *Gloeocercospora sorghi*, *Peronosclerospora sorghi*, *Ramulispora sorghi*, *Ramulispora sorghicola* and *Puccinia purpurea*. Of the foliar diseases, anthracnose, leaf blight and grey leaf spot are rampant over in all the humid regions when high rainfall occurs while rust and the sorghum downy mildew are next in importance.

Usually incidence of leaf blight (*Exserohilum turcicum*) is low but every now and then it can flare up to noticeable levels. Many important sorghum-sudan grass hybrids, grown for foliage, are particularly susceptible to leaf blight in Central America. Besides sorghum, johnson grass is one of its hosts. This disease has been serious in certain parts of Mexico in recent years.

Distribution

Leaf blight caused by *Exserohilum turcicum* (Pass.) is commonly or generally found on sorghum grown in the sub-tropics and tropical lowlands during summer (Frederiksen, 1982). Occurrence of this pathogen was less consistent in the cooler temperate and tropical highland or tropical environments. The pathogen is easily wind-disseminated and apparently most consistent in their occurrence and incidence across the diverse sorghum-growing environments. Leaf blight is one of the most widely distributed and at times

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Plant diseases reduce grain and dry matter yields. Loss of dry matter is of some importance because common sorghums are not absolutely resistant to the common leaf diseases. King (1975) and Frederiksen (1986) estimated sorghum yield losses in Africa due to diseases to be from 10 to 15%. Fortunately, the economic importance or losses caused by leaf blight appear to be minor (Olson and Santos 1976; Sundaram et al., 1972).

Nomenclature of the pathogen: Passerini (1876) described the northern leaf blight species as *Helminthosporium turcicum*. Luttrell (1858), described the perfect stage of the fungus as *Trichometasphaeria* while Leonard and Suggs (1974) redescribed the perfect stage as *Setosphaeria turcica*. Regarding the imperfect stage, Dreschler (1934) described *Helminthosporium*, the conidial stage as those having the true *Helminthosporium* characters, including *Helminthosporium turcicum*, and the cylindro *Helminthosporium*. However, Shoemaker (1959) proposed a new genus for the Graminicola spp. with *Bipolaris turcica* as the type species. Leonard and Suggs (1974) removed the species having a protuberant conidial hylum from *Bipolaris* and established *Exserohilum turcicum* (Pass.). Because of this, the generic name *Exserohilum turcicum* is probably the preferred name for the pathogen that causes leaf blight of sorghum. The causal organism, *Exserohilum turcicum* belongs to the class Deuteromycetes and the order *Fungi imperfecti* (Frederiksen, 1978).

Host range and pathogen variability: *Exserohilum turcicum* is a common pathogen of sorghum, *Teosinte, Paspalum, Zea* and other related grasses in many parts of the world including India. It is prevalent almost everywhere where maize and sorghum are grown. In addition *Triticum, Hordeum, Avena, Saccharum,* and *Oryza* are susceptible to *Exserohilum turcicum* when artificially inoculated (Frederiksen, 1978). Though pathogenic on other crops, naturally occurring isolates from host crops are generally genus specific (Frederiksen, 1980, 1984).

Numerous workers have examined the host range of isolates of *Exserohilum turcicum* from maize, sorghum and Johnson grass. Many workers like Bergquist and Masias (1974), Bhowmik and Prasada (1970), Hamid and Agragaki (1975) and Masias and Bergquist (1974) distinguished races of *Exserohilum turcicum* using isolates capable of differentiating between maize lines with and without *Ht*-
resistance gene and those races which attacked both maize and sorghum (Masias and Bergquist, 1974). Hamid and Agragaki (1975) showed that virulence to maize and sorghum was inherited independently as indicated by a one-to-one-to-one segregation for virulence to both, from a cross between a maize-specific isolate and an isolate virulent to both sorghum and johnson grass.

Stage of the plant: The leaf blight infection usually appeared 30 to 40 days after sowing, when the crop is at 8 to 9-leaf stage and further development is observed when the crop is completing the vegetative stage. Immature plants are more susceptible than the more mature plants (Tuleen, 1975). The pathogen can occur on younger plants, but in the environment favouring disease cause greater damage to older plants due to greater susceptibility in post-anthesis foliage and the concomitant increase in inoculum for infection (Odvody and Hepperly, 1992). Susceptibility to Exserohilum turcicum is reported to decrease with sorghum maturity (Frederiksen, 1980) but the occurrence of leaf blight on leaves of all ages in the field suggest that this host-pathogen interaction is more complex. Highly resistant plants develop some lesions when inoculated in the early stages of seedling growth. Most inoculation sites result in the appearance of the hypersensitive flecks. The ability of the host to form flecks decreases not only with susceptibility but also with maturity. A great increase in the percentage of the plants infected is noticed in the flowering and post flowering periods (Shenoi and Ramalingam, 1983).

Sources of inoculum: The primary survival mechanisms are mycelia and conidia resting in the soil, plant residues and weed hosts. Exserohilum turcicum (Pass.) is known to form chlamydospores within cells of the conidium. These chlamydospores can survive in soil without host tissue but their function as inoculum on sorghum has not been verified (Odvody and Hepperly, 1992). The conidia of Exserohilum turcicum, the initial inoculum, are easily wind disseminated and mostly released in the morning hours (Meredith, 1965). Leach (1980a and 1980b) demonstrated that release of conidia of E. turcicum (and Bipolaris maydis) from conidiophores was a “spore-discharge” influenced by infrared radiation and changes (usually a reduction) in relative humidity and electrostatic forces. Harvested plants with mature spots can also be the potential source of inoculum (Shenoi and Ramalingam, 1983). Increase in temperature, decrease in relative humidity and increase in wind speeds aid in the liberation of conidia and their wind dispersal (Leach, 1975).

Symptoms: Leaf blight like a number of foliar diseases of sorghum, is quite conspicuous. However, leaf blight differs from the symptoms caused by the other foliar pathogens although it can be quite similar to sooty stripe (Frederiksen, 1978). It causes large, elongated, spindle-shaped spots, measuring several cm in length and up to few cm in width having straw/grey colored center and surrounded by a deeply pigmented margin, which darken during sporulation. With the advancement of disease several spots coalesce together killing large patches of the leaf blade. It primarily attacks leaf blade but under extended disease conducive environment it may also attack leaf sheaths.

A distinctive feature of the leaf blight is the timing of the appearance of the symptoms. Small flecks appear usually 3 or 4 days after a favourable infection period. These small lesions can be seen with a hand lens but the large lesions do not appear until two weeks time. The plugging of nearby vessels causes a localized wilt within the leaf tissues. During successive favourable periods, the fungus continues to colonize the leaf having bands or characteristic zones within the leaf. Total leaf wilting occurs under heavy infestation
conditions (Frederiksen, 1978). After initial infection, hyphae of *E. turcicum* invade leaf vessels and cause a localized wilt as the lesion extends first longitudinally within vessels then laterally to give a fusiform appearance (Frederiksen, 1980).

**Disease development and host interaction:** Initial infection of sorghum by leaf blight requires high moisture condition for specific periods to allow spore germination and penetration of host tissue (Odvody and Hepperly, 1992). The conidium of *Exserohilum* is unique not only in appearance but also in function (Tuleen and Frederiksen, 1977). The conidia are thick walled, spindle-shaped, olivaceous, brown, 3-8 septate and are formed acrogenously with a prominent hilum. They measure 45-132 x 15-28 μm and germinate by protruding germ tubes from the end cells. The perfect stage of the fungus produces black, ellipsoidal to globose ascocarps containing clavate to cylindrical asci bearing three-septate, hyaline and fusoid ascospores that are 3-6 in number.

The conidia by thickening their walls become conidiospores as an overwintering or overseasoning spore. Spores or conidia germinate by the formation of a germ tube which may or may not form an appresorium on the surface of the leaf. Beneath the appresorium a peg will penetrate through the cuticle and form hyphae within host cells. Most individual penetrations result in the appearance of the hypersensitive fleck (Tuleen and Frederiksen, 1977 and Ullstrup, 1952). Infected hyphae slowly pass through living cells with scant disturbance by initially forming rudimentary appresoria as each wall is encountered. Cells of resistant hosts may form pigments at this stage. Maize with monogenic resistance has a chloronemal halo around the infection site shows the typical "Ht" reaction. A similar response has been occasionally observed in some of the more resistant sorghum cultivars. In the absence of resistance, hyphae enter in the vessels, absorb nutrients, and proliferate and damage results from the mycelial plugging of the vessel (Frederiksen, 1978).

Leaf blight is described as a local or localized wilt (Jennings and Ullstrup, 1957). Wilting is actually due to the tyloses or complexes with the polysaccharides released by the digestion of the vessel lumen rather than to the actual physical plugging by the hyphae. Toxins may be partially responsible for the death and the collapse of the host cells (Tuleen and Frederiksen, 1977). Following the development of a major lesion, pathogen fruiting begins.

Hyphae fill the substomatal cavity or epidermal cell and produce a stroma. The conidiophores of *Exserohilum turcicum* develop from the stroma. Conidiophores are olivaceous, slightly bent, measuring 1150-250 x 7-9 μm and produce a conidium at the apex. The conidiophore curves under high humidity conditions. When humidity declines, conidiophore cells dry, bubbles appear inside them and conidiophore becomes upright throwing the conidium away and out of the boundary layer. Most conidia are released on day following rainfall. Forty per cent are discharged between 8.00 and 12.00 hr, as the morning sun dries the foliage (Meredith, 1965). Mycelia within the vessels continue to almost a daily cycle as indicated by the borders of the lesion. The mycelium utilizes a crossover vessel to widen the lesion and forms a larger area of colonization. (Jennings and Ullstrup, 1957). The conidiophore is capable of emitting additional conidia on subsequent days. Tuleen (1975) using five different differentials at six maturity stages, found that plants in the immature stages were more susceptible than more mature plants.

**Mean performance**

Northern corn leaf blight, caused by *Helminthosporium turcicum* Pass., is a major
disease of corn (*Zea mays* L.) in many corn growing areas of the world.

Yield losses in susceptible hosts are considerable when infection occurs before silking and the environment favors disease. In years of unfavourable blight infection, the highest percentage of leaf tissue blighted was only 37% on susceptible plants. On the other hand, in the years of favourable infection, the susceptible, early maturing hybrids recorded 97% of leaf tissue blighted, the moderately, polygenic-resistant, intermediate maturing had 89% and the chlorotic-lesion-resistant, late maturing had 27% of the blighted leaf area (Raymundo and Hooker, 1981). Grain yield losses in maize due to *H. turcicum* leaf blight has been reported to be 29-91% (Ullstrup, 1952). The most susceptible hybrids exhibited 30-35% leaf area blighted. While most of the hybrids were less severely infected plants under favourable weather conditions for the disease. Many inbreds, including the parents of the most susceptible hybrids showed blight infections of 35% or higher (Hooker, 1975).

In sorghum, resistant sources used as males and male-steriles used as females and their *F*₁ hybrids were evaluated for leaf blight reaction (Hepperly and Rios, 1987). Leaf blight susceptible females (recorded 2.4 lesions leaf⁻¹) showed significantly greater leaf blight than male resistant sources (recorded 0.4 lesions leaf⁻¹) and *F*₁ hybrids (recorded 0.9 lesions leaf⁻¹). Compared to male-steriles, *F*₁ hybrids and the resistant male sources showed 62 and 83% less leaf blight respectively. Increased association of seed astringency with resistant sources, decreased resistance and increased variability of *F*₁ hybrids for leaf blight suggest genetic factors other than a sole dominant resistant allele are influencing leaf blight reaction in sorghum.

In maize, the mean disease ratings for the parental lines ranged from 1.43 to 4.63 and from 1.36 to 4.38 for single crosses. The per cent of heterosis for crosses ranged from -14.35 to 157.34 and from -46.9 to 32.68 over their mid, lower and higher parents, respectively. Hughes and Hooker (1971) reported that lines possessing intermediate resistance contributed to resistance in crosses.

**Genetics**

Resistance or susceptibility to a disease may be governed either by a single (qualitative) gene or multiple (quantitative) genes. Qualitative characters are governed by one or few genes, less influenced by the environment and exhibit discontinuous variation, while the quantitative characters are governed by large number of genes each with a minor effect. Quantitative characters exhibit continuous variation and are highly influenced by the environment.

Qualitative genes: Leaf blight resistance of the sorghum hybrids has been characterized by fewer lesions, low incubation period and lower production of spores per lesion as compared with the susceptible hybrids (Barrera and Frederiksen, 1994).

The utilization of the generalized form of resistance suggests that this disease can be adequately and easily controlled by host resistance (Frederiksen, 1978). Muller (1959) viewed hypersensitivity as the resistant response of the host plant to infection by the pathogen. The rate and the extent of necrosis/hypersensitive reaction in leaf tissue appear to measure quantitatively the resistance to leaf blight (Tuleen and Frederiksen, 1977). Extent and the rate of fleck formation decreases with susceptibility and as each cultivar mature. This phenomenon is suggested as the basis of host plant resistance.

Hypersensitive resistance to *Exserohilum turcicum* in sorghum may be simply inherited and effective over a wide environmental range. Incorporating *Exserohilum turcicum* resistance into hybrid parents reduces the risk of loss (Frederiksen
The hypersensitive reaction is characterized by the formation of the minute necrotic reddish-purple or yellowish-tan flecks within 2-3 days after inoculation. Flecks on resistant plants remain static without sporulation. *Helminthosporium turcicum* isolates from Johnson grass and sorghum produced only flecks on F1 hybrids of resistant sorghum lines crossed to susceptible cytoplasmic male-sterile A-lines, suggesting a dominant gene(s) conditioning resistance. The F2, F3 and backcross populations demonstrated one or two genes conditioning resistance. Different BC2 plants carrying one dominant gene from each of the Indian sorghum selections had similar hypersensitive reactions (Bergquist and Masias, 1973).

Snyder (1949-50) found susceptibility to be dominant in the F1 and F2 and the backcross data suggested that a single factor be involved in the inheritance of leaf blight in Sudan grass.

Tarumoto and Isawa (1975) also studied the inheritance of resistance in the F2 population of sorghum-Sudangrass hybrid both under field and greenhouse conditions. They reported that resistance to leaf blight in the field is essentially controlled by a single dominant gene as the F1 plants had about the same level of resistance as the most resistant parent and the majority of F2's are as resistant as F1. On the other hand, in the greenhouse the F1 distribution lay between the distribution of both the parents, while the F2 distribution was closer to that of the resistant parent than that of F1, indicating that resistance to leaf blight in the greenhouse is partially dominant. Further, the distribution of the F2's in the field and greenhouse suggest that a few minor or modifier genes may be involved along with the major gene in controlling resistance to leaf blight.

Tarumoto *et al.* (1977) conducted an experiment to study the inheritance of leaf blight reaction in popular hybrid combinations and observed that all of the F1's, F2's and backcrosses were resistant to *Helminthosporium turcicum*. No segregation was observed in the F2's of resistant x resistant parents. In F2's of resistant x susceptible parents, the segregation of resistant and susceptible plants fitted the expected ratio of 3:1 assuming a dominant monogenic inheritance. In the BC2, the segregation of resistant and susceptible plants did not fit the expected ratio of 1:1 indicating the lack of field resistance in most of the BC2 populations.

Quantitative genes: The host-specific genes conditioning the hypersensitivity have been described by several workers (Frederiksen *et al.*, 1975 and Tuleen and Frederiksen, 1977). Frederiksen *et al.* (1978) have provided some evidence to indicate that there are two major genes and some maternal factors conditioning resistance to leaf blight in sorghum. However in maize several hybrids when tested in reciprocal combinations showed similar disease reactions to *Helminthosporium turcicum* suggesting the absence of cytoplasmic effects (Hooker, 1975). Neither maternal nor reciprocal effects were significant for mean lesion area, rate of increase in lesion size and shape of lesion (Sigulas *et al.*, 1988).

Drolsom (1954) observed that the inheritance of the leaf blight reaction in Sudan grass is not simple and is governed by several factors. In the crosses between moderately resistant and resistant parents, few factors governed leaf blight reaction as the parental types were recovered in the progeny suggesting that a small number of genes are involved. However, the occurrence of plants more susceptible than either parent implied that different genes controlled resistance in the two parents or that modifying factors are present. Further, in crosses between susceptible and resistant parents two groups were recovered. In the first group, F1 plants were susceptible
but $F_2$ progenies were somewhat intermediate or susceptible. In the second group, all the plants in the $F_1$, $F_2$, and $F_3$ generations were susceptible indicating the presence of major genes for susceptibility in the female parent. They further suggested that changing environmental conditions from season to season and the probable occurrence of strains differing in pathogenicity could be the other variables involved in the inheritance of leaf blight reaction.

Tarumoto and Isawa (1972) reported that leaf blight resistance would be dominant in *Sorghum* spp. and the number of genes inheriting leaf blight resistance (dominant) would be at least three.

Resistant inbred lines in maize behave differently in their crosses with susceptible line indicating genetic differences exist among them with respect to disease resistance. The $F_1$ plants and the $F_2$’s of susceptible x resistant cross are intermediate between the two parents in mean blight ratings. Similarly the differential reactions of three susceptible lines with resistant lines indicate that resistance is controlled by large number of genes (Jenkins and Robert, 1952). Jenkins et al. (1952) reported that the resistance to the leaf blight caused by *Helminthosporium turcicum* in maize is controlled by large number of genes. Most of these genes have minor effects and their effects are evident at high levels of disease incidence while, a few of them have major effects that are evident at low levels of disease incidence and still others show their effect at widely different levels of disease incidence. Five resistant lines differed in their reaction with the highly susceptible line. Two lines showed dominant reaction with the susceptible line while the others have exhibited intermediate (one) and least dominant (two) reaction with the susceptible line.

In maize, both single-gene and multiple-gene resistance exist in host pathogen system. Monogenic resistance is expressed primarily in the form of lesion type and is a qualitative character. While, multiple-gene resistance is expressed primarily in the form of lesion number and is a quantitative character (Hooker, 1963). Chlorotic-lesion resistance with delayed necrosis and inhibited fungus sporulation expressed both by seedlings and older plants is simply inherited compared to necrotic lesions characterized by abundant fungus sporulation. Cross between resistant parents and the susceptible inbreds expressed chlorotic resistant-type lesions indicating that a single dominant gene with identical alleles condition resistance to *Helminthosporium turcicum*. The resistance of resistant inbreds (H95 and R177) was found to be dominant in hybrids (Hooker, 1975).

**Combining ability**

The general combining ability (GCA) variance and the specific combining ability (SCA) variance are very significant for disease scale and three other resistant factors (number and area of lesions and amount of sporogenous per lesion area) indicating the importance of both additive and non-additive gene effects (Wei Ji et al., 1956). They further reported that combining ability effects varied with the resistant types. For general combining ability resistant lines showed negative effect, while susceptible lines showed positive effect. On the other hand, the negative effect of SCA usually occurred in crosses involving resistant and susceptible parents indicating that the resistance is partly dominant. Hughes and Hooker (1971) reported the importance of additive, dominance and epistatic gene actions in the leaf blight resistance. Additive effects are of major importance while the non-additive gene effects are variable in expression and were of smaller magnitude.

In maize, the GCA and SCA variances were highly significant. However, GCA variance was of higher magnitude than SCA
Both additive and dominance components were significant but the additive component was in greater proportion than the dominance component. The direction of positive dominance showed the dominance of susceptibility over resistance. The results from the heterosis studies, combining ability analysis, components of variance analysis and graphic analysis indicated that the gene action for leaf blight in maize was primarily additive with partial dominance and without any interallelic interaction (Ramamurthy et al., 1989).

Significant mean squares due to GCA and SCA were observed for reaction to leaf blight in maize whereas interaction of the combining abilities with environment showed non significant mean squares indicating the presence of significant variation due to GCA as well as SCA in the inheritance of resistance. The SCA component was found to be of greater importance than the GCA component in the inheritance of the disease.

Significant variation between parents was observed for mean lesion area in maize. The mean of the parents was significantly different from the mean of the hybrids for mean lesion area. GCA effects were also significant for mean lesion area. The greatest lesion area and the greatest increase in lesion area are observed in crosses with parent (R4) having no resistant factors. GCA means were greater than zero and lesion area increased with time in crosses with the resistant parents. Significant SCA variation was observed for mean lesion size. Significant SCA effects were attributed to the observation that hybrids with R4 were more susceptible and hybrids with resistant and moderately resistant parents are more resistant than expected on the basis of respective parental averages. The SCA mean squares were smaller than their respective GCA mean squares indicating that GCA is the more important source of variation (Sigulas et al., 1988).

Correlations

The disease scale, number of lesions or the product of lesion number, lesion area, amount of sporulation per lesion area and resistance of F₁ hybrid are highly correlative indicating that the resistance of F₁ hybrids can be known by testing of the parental material (Weiji et al., 1956). Lesion length was significantly correlated with % leaf area infected (r=0.78 and 0.95 at initial and final ratings period). The seedling and adult-plant lesion length exhibited non-significant positive correlation (0.24). Hybrids expressed more resistance to the disease as adult plants than as seedlings.

Area under the disease progress curve was highly correlated with the percentage yield loss in years of favourable disease infection. Significant correlation coefficient values of 0.89 and 0.97 were obtained for quintals per hectare and 500-kernel weight base, respectively. However, in years of unfavourable disease infections, non-significant correlation coefficient values of 0.04 and -0.63 were recorded for quintals per hectare and 500-kernel weight base, respectively (Raymundo and Hooker, 1981).

Variability of resistance

In maize, broadsens e estimates of heritability for disease score ranged from 40 to 70% indicating that selection would be reliable on individual plant phenotype (Hughes and Hooker, 1971).

*Helminthosporium turcicum* leaf blight in maize possessed higher PCV and GCV estimates for disease scores during two subsequent years suggesting that the selection based on this trait facilitates a successful isolation of resistant types. However, the differences between these two were narrow for two years indicating the greater role of genetic factors and the minor role of environmental factors. Further, indicating the expression of
disease inheritance and offering greater scope for selection of desirable resistant parents. Higher GCV values coupled with higher heritability and genetic advance are reported for disease score (Satyanarayana, 1995). The higher genotypic variance to the leaf blight is due to the higher additive gene effects (Johnson et al., 1955 and Panse, 1967).

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