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CURRENT STATUS AND IMPACT OF SHEATH BLIGHT IN RICE (*ORYZA SATIVA* L.) - A REVIEW

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ABSTRACT

Sheath blight (ShB) of rice caused by *Rhizoctonia solani* Kuhn. is a major biotic constraint of rice in most of the rice growing countries of Asia. The pathogen is polyphagous competitive saprophyte and has a wide host range. A crop with a high plant density and closed canopy associated with high nitrogen management favours disease build-up from panicle initiation onwards. Crop losses generally vary from 0 to 50 % depending on severity of the disease and the stage at which the crop is infected and environmental conditions. Sheath blight infection increases peroxidase, chitinase and polyphenoloxidase activity but decreases catalase activity. Infected tissues contain higher levels of phenols than in healthy tissues. Initial symptoms of sheath blight appear in the form of circular, oblong or ellipsoid, greenish, grey, watersoaked spots of about 1cm. long that occur on leaf sheaths near the water line. To date, only partial resistance to sheath blight has been identified, as evidenced by a survey of 6000 rice cultivars from 40 countries, frim which no cultivar exhibiting a major gene for rice sheath blight resistance was identified. Recently, quantitative trait loci (QTL's) analysis identified six QTLs associated with sheath blight resistance. Many bacteria and fungi from rice field soils are antagonistic to sheath blight. Since no commercial variety resistant to sheath blight is available, the strategy of disease management will be to destroy weeds; use of need-based effective chemicals, use of herbicides and balanced fertilizer and nutrient application. More studies will be necessary to evolve a technology using antagonists as biological control measure particularly in upland rice.

Sheath blight (ShB) of rice (*Oryza sativa* L.) caused by *Rhizoctonia solani* Kuhn. is a major biotic constraint of rice in most of the rice growing countries of Asia. The Disease was first recorded from Japan by Miyake in 1910, which had been widespread in the East and South - East Asian countries and, therefore, was popularly known as 'Oriental sheath and leaf blight'. The pathogen is *polyphagous* competitive saprophyte and has a wide host range. Continuous rice cropping favours disease development. A crop with a high plant density and closed canopy associated with high N management favours disease build up from panicle initiation onwards (Biswas, 2001).

Although, the first report of sheath blight on rice from India is comparatively recent (Paracer and Chahal,1963), 'Banded sclerotial disease' of sugarcane caused by the same fungus was recorded by Butler in 1918. This disease is considered second in importance after blast in Japan (Hori, 1984), Taiwan and USA (Roy, 1993). Various estimates of crop losses due to sheath blight have been made, losses generally vary from negligible to 50% depending on the severity of the disease and the stage at which the crop is infected and environmental conditions. According to Lee and Rush (1983) losses occur between 20 to 50% when all the sheaths are infected. Roy (1979) recorded yield loss of 10 to 36% in Assam depending on growth stage of plants when the disease occurs.

Pathogen and Pathogenesis

The fungus causing rice ShB is variously named but the most commonly used one is *Rhizoctonia solani* Kuhn. Teleomorph is identified in *Thanatephorus cucumeria* (Frank) Donk, but Tu and Kimbrough (1978) he

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suggested the name *T. sasakii* (Shirai) Tu and Kimbrough based on differences in morphology and cytological tests. Naiki and Kanoh (1978) classified *T. cucumeris* into 5 anastomosis groups according to their virulence. The ShB isolate in India belongs to AG1group having 3 to 16 nuclei (Ahuja and Payak, 1985).

Basidiospores are very rare in India (Singh and Pavgi, 1969). Primary infection comes from sclerotia. Basidiospores are formed at night. Two types of mycelium – straight and branched, and lobate–are developed, of which only the latter type is infectious. Lesion is covered by lobate mycelium while the straight type may extend beyond it without causing infection (Ou *et al.*, 1973). *Rhizoctonia solani* induce lesion on leaf blades and leaf sheaths of infected plant. *Rhizoctonia solani* produces sclerotia on both abaxial and adaxial leaf sheath surfaces but not in the tissue. *Rhizoctonia solani* forms infection cushions and lobate appressoria on leaf sheath (Kim and Ishii, 1992).

ShB infection increases peroxidase, chitinase and polyphenoloxidase activity but decreases catalase activity in rice . *Rhizoctonia solani* produces phytotoxins, such as phenyl acetic acid (PAA) and its hydroxy derivatives (m-HPAA, o-HPAA and p-HPAA) (Waheeta *et al.*, 1987).

Infected tissues contain higher levels of total phenols than in healthy tissues. Phenols in infected tissues are increased with increasing N doses, while the reverse occurred in healthy sheath tissue. Infected tissues also contain higher levels of dry matter and have lower moisture content than those of healthy sheath (Kalita *et al.*, 1984). Phenol content is increased (Premalatha Dath, 1990). Padma Kumari and Menon (1981) reported that inoculated plants accumulate more Ca and Mg but less K and P. Permeability of leaves is increased,which slow down after two days of inoculation (Roy, 1977). Sugar content is reduced but starch content is increased. Respiration during pathogenesis is enhanced but transpiration is reduced (Roy, 1982). Naidu *et al.*, (1981) recorded decrease in chlorophyll content.

Infestation of *Nilaparvata lugens* and *Hirschmanniella oryzae* aggravates sheath blight (Premalatha Dath, 1990).

Disease Development

Environment factors: High temperature (22°C to 35°C) and high relative (RH) humidity are favourable for sheath blight development. Mycelial growth and sclerotia formation are at its higher at 25°C to 30°C and 80 to 95% RH are optimal for disease development (Tiwari and Chaure, 1997). Soil type may also influence disease development (Kannaiyan and Prasad, 1978), disease severity is higher in sandy clay loam than in clayey or sandy soils (Tiwari and Chaure, 1997).

Mycelial growth and sclerotia formation are optimum at pH 6.0 - 7.0, and no growth at pH 3.0 and 9.0 (Tiwari, 1997). The soil microorganism does not affect sclerotial viability, but suppression of *R. solani* may occur by sclerotia inhabiting fungi (Manian and Manibhushan Rao, 1990). Low moisture level (30-60%) of soil encourages seedling infection while waterlogging reduces it (Kannaiyan and Prasad, 1981). ShB is more severe under the shade in rice than in the open (Yoshimura, 1955).

Host susceptibility

ShB is usually more severe in high-yielding dwarf indica cultivars than in traditional tall indica cultivars. A number of moderately resistant cultivars are observed in the medium and late maturity groups (Biswas, 2001). ShB is usually severe on cultivars that are short, highly tillering, more erect and responsive to high fertilizer in comparison to tall cultivars with fewer tillers. Some cultivars may be resistant or susceptible at both seedling and adult stages

while others may be resistant at seedling stage but become susceptible later and *vice versa* (IRRI, 1973).

Plants are more susceptible to infection at booting and flowering. The average percentage of infected tillers and average disease severity are increased as plant age increases (Vanitha *et al.*, 1996). Munshi *et al.* (2000) recorded flowering stage as the most susceptible stage as compared to seedling and tillering stages. No significant difference was found among varieties inoculated at seedling stage (Kozaka, 1961), but observed at flowering or booting stage (IRRI, 1974). Sarkar *et al.*, (1993) stated actively tillering stage as the most susceptible as compared to booting and heading stages. ShB generally reduces kernel bulk density but does not significantly affect head rice yield of cultivars (Candole *et al.*, 2000).

Host nutrition

Heavy doses of nitrogenous fertilizer increase the severity of ShB. However, a slow release nitrogen fertilizer, Crotonilidine di Urea, reduces its attack (Roy, 1986b). Higher N enhances the severity of ShB (Borthakur *et al.*, 1989). More N produces luxuriant growth and drooping of leaves provides a physiological condition conducive to ShB (Shahjahan and Mew, 1989). Potash (Hashioka, 1970) and silica (Mathai *et al.*, 1981) reduce disease severity. B at 500 ppm is the best retardant of mycelial growth and sclerotia formation, followed by Zinc in culture (Lakpale *et al.*, 1997).

Host range

R. solani has a wide host range. Kozaka (1965) in Japan recorded 188 species in 32 families and Tsai (1970) in Taiwan listed 20 sp. belonging to 11 families as potential host upon inoculation. According to Meena and Muthusamy, (1998), host range of *Rhizoctonia solani* are as follows:

Host	Mode of resistance
Cajanus cajan, Capsicum annuum, Curcuma longa, Dolichos biflorus, Lycopersicom esculentum, Panicum miliaceum, Paspalum scrobiculatum, Setaria italica, Sorghum vulgare, Zea mays.	Moderately Susceptible
Brachiaria mutica, Cynodon dactylon, Cyperus rotundus, Echinochloa colona, Eleusine corocana, Phaseolus aureus.	Susceptible
Dolichos lablab var. *typicus, Vigna sinensis*	Most Susceptible

Premalatha Dath (1990) gives a list of plants recorded as hosts from India. Both rice and wheat are hosts of *Rhizoctonia solani*, but wheat as a previous crop, does not favour ShB in rice (Singh *et al.*, 2000). Soybean in rotation with rice is induce heavy incidence of ShB in the southern USA (Lee and Rush, 1983).

Initial symptoms and spread of disease

Initial symptoms of ShB appear in the form of circular, oblong or ellipsoid, green grey water-soaked spots about 1 cm long that occur on leaf sheaths near the water line. The lesions enlarge the centres of which become pale-green or grey and are surrounded by an irregular purple border (Webster and Gunnel, 1992).

Under favourable conditions, the disease may progress:

(1) Inwardly from outer to inner sheath

(2) Vertically from sheath to sheath and lamina, and

(3) Horizontally from tiller to tiller and hill to hill

Heavily infected plants produce poorly filled grains and may die immature panicle (Dasgupta, 1992).

Initial symptoms consist of lesions on the sheaths of lower leaves at late tillering or early inter nodal elongation growth stages. Under favourable conditions of low sunlight, high humidity (≥5%), and warm temperature (28-32°C), the infection spreads rapidly by means of runner hyphae to upper plant parts. Lesions may coalesce to encompass the entire leaf sheath and stem (Rush and Lee, 1992).

Sclerotia may move from one field to another through irrigation water and during movement they may produce mycelia and secondary and tertiary sclerotia (Ou *et al.*, 1973). Lakshmanan and Jagannathan (1984) reported that feeding infected fodder to animals might spread the pathogen.

Disease cycle

The infected rice seeds may produce 4-6.6% seedling infection in India (Ou, 1985; Mathur, 1983). But on transplantation the infected seedlings were unable to develop disease (Naidu, 1992). Disease cycle takes place predominantly through sclerotia in the humid tropics. Sclerotia, the dormant are shed before/or during the harvest operation and remain in soil and survive for a long time. When the buoyant sclerotia tend to accumulate in undisturbed standing water at the plant-water interface, the aerobic fungus creeps up several centimetres in 24 hr and the primary infections are caused in wetland rice. Rain- water runoff and flood irrigation permit good dispersal of floating sclerotia (Lee, 1979), and consequently provide the primary foci of infection through the stretches of rice fields. Further, with the increasing size of sclerotia on their fragments, number and size of lesions also increased (Gangopadhyay, 1983). The pathogen induced lesions on leaf blades and leaf sheaths of infected plants. It produces sclerotia on both abaxial and adaxial leaf sheath surfaces but not in the tissue. The pathogen form infection cushions and lobate appresoria on leaf sheath, and directly penetrate the cuticle or through stomata (Kim and Ishii, 1992).

Once infection occurs, secondary spread takes place through direct contact (role of basidiospores uncertain). Sclerotia may move from one field to another through irrigation water and during movement they may produce mycelia and secondary or tertiary sclerotia (IRRI, 1973).

Management of sheath blight

Varietal resistance: To date, only partial resistance to rice ShB has been identified, as evidenced by a survey of 6000 rice cultivars from 40 countries, from which no cultivar exhibiting a major gene for rice ShB resistance was identified (Hashiba, 1984).

More recently quantitative trait loci (QTL) analysis identified six QTLs associated with ShB resistance on 6 of the 12 rice chromosomes, but only one QTL appeared to be independent of plant height, a morphological trait associated with ShB resistance (Li *et al.*, 1995), and six QTLs, 9SB-2 , 9SB-3 ,9SB-7 ,9SB-9-1,9SB-9-2 and 9SB-11 located on chromosomes 2,3,7,9 and 11, respectively, have been identified for contributing to ShB resistance in rice (Zou *et al.*, 2000) .Additional research suggests that it is feasible to identify major genes conferring high levels of partial resistance (Pan *et al.*, 1999), pyramid these genes and achieve nearly complete sheath blight resistance.

Goita (1985) studied inheritance of resistance to ShB in long grain rice and suggested that two pairs of complementary genes control resistance to ShB with low heritability, possibly owing to epistatic interaction. Xie (1990) inoculated with *R. solani* and screened for sheath blight resistance in long grain rice cultivars, and suggested that inheritance of sheath blight resistance in SC

86-20001-5 was controlled by a single recessive gene. Two independently inherited recessive genes controlled resistance in SC 86-20001-33. Xue and Li (1989) studied inheritance of reaction to artificial sheath blight inoculation in rice. The F1 of four combinations of moderately resistant and susceptible parents showed intermediate reaction to inoculation; the F2 distributions tended to be continuous, that implies that resistance to ShB is controlled by multiple genes.

Due to absence of suitable donors, information on inheritance of resistance is lacking. Indications are that resistance is a dominant character, and crosses between resistant and susceptible cultivars, majority of F2 populations are susceptible (Premalatha Dath, 1990). Wax thickness is segregated at 3:1 ratio (Lee and Rush, 1983). Cushion formation by the fungus is dominantly inherited in 3:1 or 13:3 ratios. Two pairs of complementary genes have been suggested to control resistance (Premalatha Dath, 1990).

Sheath blight resistance was identified in the *Oryza* sp., *O. minuta* J. S. Presl., ex C.B. Presl. (Amante- Bordeos *et al.*, 1992) and *O. officinalis* wall ex Watt (Lakshmanan, 1991), and transferred into cultivated rice through backcrossing. These studies indicate that *Oryza* sp. are an important source of sheath blight resistance genes, and transferring these genes intro rice cultivars adapted to the production area in the southern US is an important disease management strategy. Two cultivars, Andrewsali and Monoharsali show resistant reactions against ShB disease (Kalita *et al.*, 2000).

Cultural practices: Burning the infected crop debris after harvest, keeping the fields weed free and bunds cleaning is necessary to control the disease. Incorporation of Neem (*Azadirachta indica*) and groundnut cake under dry conditions, and ellupa cake, gingelly cake and neem cake under flooded condition reduced the survival of sclerotia of the rice pathogen (Lakshmanan, 1984).

High density of seed rate and planting encourage the spread of the disease (Mithrasena and Adhikari, 1986). Inorganic fertilizer can also influence saprophytic survival; K and P reduced it (Kannaiyan and Prasad, 1983). The application of silicon to complement host resistance to sheath blight appears to be an effective strategy for disease management in rice, especially when the soil is low or limiting in plant available silicon (Rodrigues *et al*, 2001). Willocquet *et al.*, (2000) reported that incidence -severity relations indicated a less aggregated distribution of the disease in direct -seeded rice crop than in any of the transplanted ones, regardless of spacing.

Biological approach: Many bacteria and fungi from rice field soils are antagonistic to ShB. Chen *et al.* (2000) reported that fermented product of B-916 (*Bacillus subtilis*), Jingangmycin and their combinations are useful for controlling *Rhizoctonia solani.*

Two *Pseudomonas fluorescens* strains *viz.*, PF1 and PF7, which inhibited the mycelial growth of *Rhizoctonia solani* and increased the seedling vigour of rice plants *in vitro* were selected for assessing induced systemic resistance against *R. solani* in rice. The Pseudomonas application as a bacterial suspension or a talc-based formulation through seed, root soil and foliar application, either alone or in combination (seed+root+soil+foliar), effectively reduce ShB (Nandakumar *et al.*, 2001).

Antagonistic fungi *Trichoderma viride, T. koningii* and *Gliocladium virens* were evaluated against *R.solani.* In dual culture technique, *T. viride* (Coimbatore-2 isolate) was superior in inhibiting the growth of pathogen followed by *T. harzianum* (Coimbatore), *G. virens* (Pantnagar) and *T. harzianum*

(Pantnagar). Maximum disease reduction was observed in *T. koningii* (Delhi isolate) followed by *T. viride* (Coimbatore-2), *T. viride* (Delhi) and *T. viride* (Coimbatore-1), when sprayed 24 hours after inoculation (Sudhakar *et al.*, 1998).

Seeds treated with *T. viride* and *T. harzianum* showed significant reduction in sheath infection. Both antagonists exhibited higher efficacy in reducing sheath infection and increased grain yield when they were treated with either 2% (w/v) methylcellulose or 2% [w/v] methylcellulose and 0.1 M $MgSO_4$. *T. harzianum* was more effective than *T. viride* in reducing sheath infection and increase in yield (Das and Hazarika, 2000).

Meena and Muthusamy (1998) recorded that 0.1 and 0.05% palmarosa oil completely inhibited mycelial growth. Palmarosa oil also completely inhibited the sclerotial production of *R. solani*. Extracts of 8 environment friendly plant (*Ocimum basilicum, Citrus reticulata, Syzygium aromaticum, Jasminum officinale, Tagetes tenuifolia, T. erecta, Pyrus pashia* and *Gladiolus sp.*) and were evaluated against *R. solani*, by poisoned food technique at different concentrations. Complete inhibition of fungal growth was observed at 100 ppm concentration by *S. aromaticum* and methyl anthranilate, a chemical constituent of *J. officinale* extract while nonyl alcohol, citral and phenyl ethyl propionate showed 77.7, 88.3, and 83.3% antifungal spectrum at the same concentration. The benzene and acetone extracts of *T. tenuifolia* and hexane and acetone extracts of *T. erecta* also showed complete inhibition at higher concentration (1000 ppm) (Janki-Kandhari *et al.*, 2000).

Chemical control: Common pesticides used earlier against sheath blight were copper, organo-mercury and organo-arsenic compounds (Ou, 1985). Benomyl, carbendazim, edifenphos, kitazin, propaconazole and hexaconazole are the most effective chemicals reported by various Indian workers. Besides, Monzet (iron methane arsenate) and ammonium iron methane arsenate (neoasozin), polyoxin, validamycin and mepronil are effective against sheath blight (Hori, 1984).

Four antibiotics, two developed in Japan *viz.*, validamycin and polyoxin and two developed in China viz. jingganmycin and chingfengmeisu have been found effective against sheath blight (Premalatha Dath, 1990).

Other fungicides found effective are dithane M - 45, deconil, thiobendazole, demosan, captafol, topsin, guazatin, cuman etc. Iprodione was very effective at IRRI (Ou, 1985). Sodium selenite was the most effective in reducing both lesion length and lesion number. Plants exhibited phytotoxicity when sodium selenite was applied by root dip treatment and spray, and disappeared within 3 - 4 days (Ashok Bhattacharyya *et al.*, 2001).

Cycloheximide gave the best disease control and the highest yield followed by ferric chloride and sodium selenite (Sarkar and Sinha, 1991). A few herbicides *viz.*, pentachlorophenol, propanil, Saturn, dursban can reduce sheath blight. Sclerotial viability was reduced by applying herbicides particularly paraquat and thiobencarb under field condition (Pathak, 1990).

Molecular strategies: Inactivation of a host specific toxin, RS toxin, induced by *R. solani* by a putative alpha-glucosidase have been identified based on enzyme assay and Western blot analysis from coconut (*Cocos nucifera* : the only known non host of *R. solani*) leaves (Shammugam *et al.*, 2001).

Genetic transformation has been attempted for management of rice sheath blight disease. APR -3 rice chitinase gene (RC 7) has been isolated from *R. solani* infected rice plants and introduced in indica rice cultivars by the *biolistic* and poly ethylene glycol

mediated transformation system. The transformants synthesized different levels of chitinase proteins constitutively and progeny from the plants containing the chitinase gene showed different levels of enhance resistance when challenged *R.. solani* (Karabi-Datta *et al.*, 2001).

Anti microbial peptides play a role in the immune systems of plants by limiting pathogen infection and growth. The puroindolines (PINs), endosperm-specific proteins involved in wheat seed hardness, are small proteins reported to have in vitro anti microbial properties. Rice normally does not contain PINs. Transgenic rice (cv. M202) plants that constitutively express the PIN genes, pin A and/or pinB, throughout the plants were produced. PIN extracts of leaves from the transgenic plants reduced in vitro growth of *R. solani* by 35-50 %. Puroindolines are effective in vivo in anti fungal proteins and could be valuable new tools in the control of fungal pathogens of crop plants (Krishnamurthy *et al.*, 2001).Pre- inoculation of rice cv. IR26 seedlings with a non- pathogenic binucleate Rhizoctonia (BNR) species (isolate 232-CG) induced resistance to rice sheath blight. A significant reduction of disease severity was observed in BNR- treated plants compared with the non treated ones. Treatment with BNR at least 24 h prior to pathogen challenge resulted in significant protection of rice seedlings from *R. solani* infection. Remarkable increases in the activities of phenylalanine ammonia- lyase and peroxidase, the key enzymes in plant defence responses were observed in binucleate Rhizoctonia treated rice seedling. Elevated levels of phenylalanine and peroxidase were positively correlated with the increase in disease resistance in rice induced by BNR.(He-ChenYang and He-CY, 2001).

The ShB is a major rice disease world wide. It is spreading day by day in the area, where it was unknown. At present, management programs to change this trend consist of integrating options of host resistance, need-based effective fungicides, use of balanced fertilizer and nutrient application, rotation schemes and cultural manipulations. More studies will be necessary to evolve a technology using antagonists as biological control measure particularly in upland rice. Many bacteria and fungi from rice field soils are antagonistic to sheath blight. Screening and evaluation of the resistant rice germplasm pool continues, as do efforts to improve cultivar performance by combining known levels of resistance and by changing plant growth habits favourable for disease development. The search for more efficacious fungicides in conjunction with better method of application or timing is necessary. More emphasis needs to given own the biological control and cultural programs to limit primary inoculum to manage the disease.

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