Fusarium-sheath rot (FShR) disease of rice: current status

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Recent findings on various aspects of *Fusarium*-sheath rot disease of rice are critically reviewed. Future lines of work are also indicated.

Key words: Rice, Fusarium-sheath rot, review

INTRODUCTION

Fusarium-sheath rot (FShR) of rice, caused by Fusarium spp. is a recently reported problem. Once it was ignored and considered a secondary invader as in several studies, fastgrowing fusaria, which masked the growth of the sheath rot (ShR) fungus, Sarocladium oryzae were isolated consistently from many ShR affected disease samples. However, this is an attempt to examine and review the recent findings on various aspects of this disease.

PATHOGEN

Different species of *Fusarium*, reported to be associated with rice sheath rot disease from different countries are tabulated below.

Fungus	Country	Reported by
F. avenaceum	India	Devi and Singh (1994)
F. equiseti	India	Kang and Rattan (1983)
	Nigeria	Ngala and Adeniji
	11. 2. 11	(1986)
F. graminearum	India	Singh and Devi (1990)
F. moniliforme	India	Kang and Rattan (1983)
		Kang and Kaur (1989)
		Devi and Singh (1990)
		Biswas (1999)
A.	Nigeria	Ngala and Adeniji
Y		(1986)
	Spain	Martin - Sanchez and
		Jumenez - Diaz (1982)
F. proliferatum	USA	Cartwright et al., (1995)
v. Postskinera		Gannon (1996)
		Abbas et al., (1998)
F. semitectum	Spain	Martin - Sanchez and
	•	Jumenez - Diaz (1982)

A disease, almost identical to FShR, caused by *F. moniliforme* and named as panicle rot of rice has also been reported by Bhargava *et al.*, (1978) on cultivar (cv) Ratna from Uttar Pradesh, India.

DISEASE INCIDENCE, INTENSITY AND YIELD LOSS

F. moniliforme was identified as the prime fungus species to cause FShR in Punjab, India. Disease incidence varied from 33.2-83.3 % in affected fields of cv PR 106 with the intensity from 12.8 -38.8% (Kang and Kaur, 1989) while its intensity was more severe (33.1 - 42.4%) in cytoplasmic male sterile (CMS) lines and compared with 15.4 -19.3% in corresponding maintainers, restorers and inbred rice varieties under natural field conditions in Punjab, India during wet season (Sharma et al., 1993). Similar observations were also made by us (unpublished) in West Bengal, India on several CMS lines and thus the high degree of susceptibility of CMS lines to FShR may become a limiting factor in hybrid rice seed production. FShR was found to be well distributed at various parts of Manipur, India affecting all rice cvs upto 45%. Field observations showed that high yielding cvs were severely affected by FShR than local traditional cvs. It was further observed that F. graminearum was most predominant followed by F. moniliforme and F. avenaceum (Devi and Singh, 1997). Kang and Rattan (1983) reported a loss of upto 50% due to FShR in Punjab, India. In Las Marsimus (Sevilla), Southern Spain, a field survey showed that the incidence of the disease due to F. moniliforme varied maximum from 50 to 70%, estimated yield loss amounted to 18-23% while incidence due to *F. semitectum* was as high as 40% with a yield loss of 15% (Martin - Sanchez and Jumenez - Diaz, 1982). Depending on the particular field Gannon (1996) estimated the yield loss of 10 - 70% in some of the FShR affected fields of Arkansas and Texas, USA due to *F. proliferatum*.

DISEASE SYMPTOMS

FShR symptoms resemble almost those produced by S. oryzae (ShR); again different Fusarium spp. produce more or less identical symptoms (Kang and Rattan, 1983; Kang and Kaur, 1989; Singh and Devi, 1990; Cartwright et al., 1995; Gannon, 1996; Abbas et al., 1998; Biswas, 1999). The symptoms of the disease are noticed at the time of ear emergence. Lesions occurred on the upper leaf sheath, particularly the flag leaf sheath. Lesions were initially irregular, oblong, 5-15 mm in length with grey to light brown centres surrounded by a dark reddish brown margin. Finally, lesions enlarged and coalesced and covered most of the boot leaf sheath. In some cases, lower leaf sheaths may eventually develop lesions also, but rarely more than two leaf sheaths show symptoms. Infection reaches the stem and causes browning and rotting of the peduncle. Superficial dense white or pinkish powdery growth of the fungus is visible on all the affected sheaths, especially evident during humid periods. Panicles partially emerge or remain inside the sheath. Emerged panicles have chaffy grains or partially filled, sterile, thin, reddish brown to off-white shrivelled florets or grains often covered with a white or pinkish-white powder which consists of millions of air borne type spores.

PATHOGENICITY

Pathogenicity test was performed to confirm *Fusarium* spp. association with FShR. Test entries were inoculated by injecting or spraying conidial suspensions or by inserting mycelial bits or fungal disc (grown on potato dextrose agar) into injured boot leaf sheath before panicle emergence. Typical FShR symptoms appeared with all the methods, lesions developed within a week and the sheath completely rotted in three to four weeks (Kang and Rattan, 1983; Grewal and Kang, 1988a; Kang and Kaur, 1989; Devi and Singh, 1997; Biswas, 1999). Percent disease incidence was calculated on the basis of infected tillers and test entries were scored in standard evaluation system (SES) 0-9 scale

proposed by the International Rice Research Institute (IRRI, 1996) for ShR by S. oryzae (Devi and Singh, 1997; Singh and Devi, 1999). A scoring system specifically for FShR has been developed by Grewal and Kang (1988a) in which disease intensity (severity) was scored in a 0-4 scale on the basis of visual observations where 0=no infection and 4 = maximum infection, entire sheath rots, no panicle emergence. This is very useful in case of handling a large number of cvs and CMS lines. Out of 34 rice cvs and lines tested for reaction against FShR (F. moniliforme) under artificial inoculated condition, none was found immune. Jaya and PR 106 were most susceptible in Punjab, India (Kang and Rattan, 1983; Grewal and Kang 1988a; Kang and Kaur, 1989) while cvs like TN 1, IR 20, Vikramarya, Bennibhog and CMS lines like IR 58025 A, IR 62829 A are susceptible to FShR (F. moniliforme) under artificial inoculated condition in West Bengal, India. (Biswas, 1999). Rice cvs KD, 2-6-3, Leima Phou are susceptible to FShR caused by F. avenaceum, F. graminearum and F. moniliforme in Manipur, India (Devi and Singh, 1996, 1997). Singh and Devi (1999) in a field experiment found that none of the 92 rice cvs/lines was immune to FShR under artificial inoculated condition. However, 23 cvs/lines showed high resistance (HR) and 34 resistant (R) against these fusaria. In Las Marsimus (Sevilla), Southern Spain cv Ribello has been reported to be susceptible to F. moniliforme while cvs Bahia, Frances, Girona, Ribello and Sequial are susceptible to F. semitectum (Martin-Sanchez and Jumenez-Diaz, 1982). Two rice cvs Bengal (medium grain) and Cypress (long grain) are most susceptible to FShR (F. proliferatum) in Arkansas and Texas of Southern USA, where rice is a major crop (Cartwright et al., 1995; Gannon, 1996; Abbas et al., 1998). Hence, it may be concluded that difference in susceptibility and resistance may be due to different genetic make up of the rice plants and different Fusaria involved. Highly resistant (HR) and resistant (R) cvs/lines may be utilized as sources of resistance in resistant breeding programme to FShR.

PHYSIOLOGY OF THE PATHOGEN

Devi and Singh (1994) found that *F. avenaceum* produced the most growth at 25°C temperature and *F. moniliforme* and *F. graminearum* at 30°C.

Maximum sporulation for the fungi was at 30°C. F. avenaceum and F. graminearum grew best in complete darkness but F. moniliforme grew best in complete light. F. avenaceum and F. graminearum produced the most spores in complete darkness. Though F. moniliforme produced an appreciable quantity of spores in both complete light and complete darkness, alternate light and darkness had a synergistic effect on spore production. Devi and Singh (1997) found that different pH levels (1.0 -12.5) supported mycelial growth of F. avenaceum, F. moniliforme and F. graminearum. maximum mycelial growth of these three fungi was found at pH 12.0, 7.0 and 12.0 respectively at 25°C. At narrow range of pH (5.5 - 7.5 and 7.5 -9.0), F. avenaceurm and F. graminearum sporulated but F. moniliforme sporulated at wide pH range of 1.0 - 12.5 with maximum at pH 11.0. However, sporulation of F. avenaceum and F.graminearum was found optimum at pH 6.5 and 7.5, respectively. Devi and Singh (1997) recorded maximum mycelial growth of these fusaria in potato dextrose broth out of seven culture media tried. Maximum sporulation of F.avenaceum, F.moniliforme and F. graminearum was found in oat meal, potato dextrose broth and Czapeck - Dox medium respectively. Of the ten carbon sources tesed, sucrose, D (+) xylose and D (-) fructose supported maximum growth of F. graminearum, F.moniliforme and F. avenaceum respectively. Of the fifteen amino acids tested, DL - alpha - alanine, DL-aspartic acid and L-histidine monohydrochloric acid supported maximum growth respectively (Singh and Devi, 1996). Devi and Singh (1997) found that the length of FShR lesion was maximum on cv KD, 2-6-3 inoculated with F.moniliforme followed by F.avenaceum and F. graminearum respectively. When these fungi were inoculated in combinations, the length of lesion was much less than those produced by individual fungus. Mix inoculations showed marked antagonistic effect on lesion development. This antagonistic effect may be due to the production of toxic metabolites by the fusaria. Abbas et al. (1998) first reported the presence of a mycotoxin - "fumonisins" (FB1, FB2, and FB3) produced by F. proliferatum causing FShR in Southern USA.

SURVIVAL OF THE PATHOGEN

F.moniliforme survived in stored rice seeds for 12

months, although incidence was much lower after 9 months. Under natural conditions, maximum survival (>11 months) occurred at 30 cm (depth) in soil. No rainfall was recorded for the first 4 months. Cool and dry conditions favoured the survival of the fungus in plant litter. Under conditions in Punjab, India, the fungus survives on seeds and plant litter in the field and becomes the source of inoculum for the next crop (Grewal and Kang, 1988b). Devi and Singh (1995) studied the survival of FShR fungi under conditions in Manipur, India. They found that F. avenaceum (Gibberella avenacea), F. graminearum (G. zeae) and F. moniliforme (G. fujikuroi) survived in rice seeds for > 13 months. All three pathogens remained viable for 12 months in infected leaf sheaths buried upto a depth of 7 cm, but viability was lost after 10 months at 10 and 15 cm. The fungi was lost after 10 months in diseased leaf sheaths at room temperature (4-29°C). G. avenacea was pathogenic to 12 field weed hosts tested; G. zeae infected 3 and G. fujikuroi only 6.

FACTORS AFFECTING DISEASE DEVELOPMENT

FShR incidence was greater in heavy soils where nitrogen was applied or where *Trifolium alexandrium* was sown in dry season (Kang and Rattan, 1983) Indiscriminate application of chemical fertilizers especially heavy doses of nitrogenous fertilizers is conducive for the development of FShR and more grain chaffiness (Grewal and Kang, 1990a). Grewal and Kang (1990b) observed that rice crop planted before and mid-June had less FShR incidence and grain chaffiness than July plantings. In USA, Gannon (1996) noticed a higher incidence of FShR due to abnormally hot, and dry weather.

DISEASE CONTROL

Gannon (1996) suggested cultural practices like early planting, seed treatment, application of recommended rate of nitrogen and change of susceptible cv for the control of the disease. Devi and Singh (1996) studied the relative efficacy of seven fungicides on FShR, both *in vitro* and *in vivo* conditions. Results of *in vitro* studies revealed that 0.1% Bavistin (Carbendazim), 0.1% Benlate (Benomyl) and 0.2% Dithane M-45 (Mancozeb) were most effective as no growth of *F. avenaceum*,

F.moniliforme and F.graminearum was recorded. In the rice plants (cv Leima Phou) sprayed 15 days interval during the boot stage with 0.1% Bavistin and 0.2% Dithane M-45, the percentage of disease incidence was nil with significant increase in rice yield.

FUTURE RESEARCH NEEDS

Though certain aspects of the disease syndrome have been studied, the information available on rice-FShR system is highly inadequate. More research work is needed on the following aspects: a) mechanism of pathogenesis, b) physiology of host-pathogen interaction, c) factor (s) governing host resistance and their mode of action on the pathogen system, d) disease cycle of the pathogen, e) screening for disease resistance of commercial rice cvs and CMS lines and further breeding programmes, f) integrated approach to disease control through improved cultural practices, soil amendment, biocontrol and chemical control and g) as the fumonisins are heat stable and would not likely be destroyed by cooking, determination of prevalence of fumonisins in rice, grown in countries where rice is the staple food and several food products are derived from rice.

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REFERENCES

- Abbas, H. K.; Cartwright, R. D.; Shier, W.T.; Abouzied, M.M.; Bird, C.B.; Rice, L.G.; Frank Ross, P; Sciumbato, G.L. and Meredith, F.I. (1998). Natural occurrence of Fumonisins in Rice with *Fusarium* Sheath Rot Disease. *Pl. Dis.* 82 (1): 22-25.
- Bhargava, S. L.; Shukla, D. N.; Singh, N. K. and Singh, N (1978). Fusarium moniliforme causing panicle rot of rice. Ind, Phytopath. 31: 367-369.
- Biswas, A. (1999). Occurrence of Fusarium Sheath rot in West Bengal. IRRN 24 (2): 41.

- Cartwright. R.D; Correll, J.C. and Crippen, D.L. (1995).
 Fusarium Sheath rot of rice in Arkansas. (Abstr.)
 Phytopath. 85: 1199.
- Devi, R.K.T. and Singh, N.I. (1994). Effect of temperature and light on growth and sporulation of *Fusarium* Sheath rot. *IRRN* 19(3): 28.
- Devi, R.K.T. and Singh, N.I. (1995). Survival of fugarial sheath rot fungi of rice. *Pl. Dis. Res.* **10**(1): 91-95.
- Devi, R.K.T. and Singh, N.I. (1996). Relative efficacy of fungicides against fusarial sheath rot under in vitro and in vivo conditions. Ind. Phytopath. 49 (4): 378-380.
- Devi, R. K.T. and Singh, N.I. (1997). Distribution, pathogenicity and physiology of Fusarium spp. Causing rice sheath rot in Manipur. In-Recent Researches in Ecology. Environment and Pollution Vol 10:295-303. Ed. by S.C. Sati, J. Saxena and R.C. Dubey. Today and tomorrow's Printers & Publishers New Delhi 5 (India).
- Gannon, R. (1996). Possible new disease found. Rice J. 99 (4)
- Grewal, S.K. and Kang, M.S. (1988a) Screening of rice germplasm against *Fusarium* Sheath rot (ShR) disease. *IRRN* 13 (3): 14.
- Grewal, S.K. and Kang, M.S. (1988b). Seasonal carryover of Fusarium moniliforme. - Sheld.: casual organism of sheath rot of rice, Phytopatho Mediterr. 27 (1): 36-37.
- Grewal, S. K. and Kang, M.S. (1990 a). Influence of nitrogen fertilization of *Fusarium* - Sheath rot and yield of rice. *Pl. Dis. Res.* 5 (1): 47-52.
- Grewal, S. K. and Kang, M. S. (1990 b). Management of Halicas sheath rot of rice by early transplanting. Pl. Dis. Res. 5 (2): 148-153.
- IRRI (1996). Standard Evaluation System for rice. 52 pp.
- Kang, M.S. and Rattan, G.S. (1983), Sheath rot in the Punjab. India. IRRN 8 (3): 7-8.
- Kang, M.S. and Kaur, S. (1989). Sheath rot of rice in Punjab. J. Res. Punjab Agric, Univ. 26 (1): 57-61.
- Martin Sanchez, J.P. and Jumenez Diaz, R. M. (1982). Two new *Fusarium* - species infection rice in southern Spain. *Pl. Dis*, **66**: 332-334.
- Ngala, G. N. and Adeniji, M. O. (1986). Sheath rot disease in tropical Africa. In-Juo, A.S.R. and Lowe, J.A. The wetlands and rice in subsaharan Africa-Ibaban, Nigeria: IITA.
- Sharma, R. C.; Sidhu, G. S., Bharaj, T. S., and Sharma, H.L. (1993). Pathological constraints on hybrid rice production technology. *IRRN* 18 (3):15.
- Singh, N.I. and Devi, R.K.T. (1990). Fusarium graminearum, a casual agent of rice sheath rot disease. Ind. Phytopath. 43 (4): 593
- Singh, N.I. and Devi, R.K.T. (1996). Effect of different carbon sources and amino acids on growth and sporulation of *Fusarium* - species causing sheath rot of rice. *Pl. Dis. Res.* 11 (2): 139-142.
- Singh, N.I. and Devi, R.K.T. (1999). Reaction of rice cultivars/lines to *Fusarium* sheath rot. *Ind. Phytopath.* **52** (2) :172-173.

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