

Wheat diseases and their management in Karnataka- An overview

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Abstract

Wheat crop in India, owing to its diverse climatic conditions prevailing throughout the country, suffers more or less due to all three rusts. Among the major constraints of limiting the wheat production in Karnataka is leaf rust followed by leaf blight and foot rot in the rain fed ecosystems. In Karnataka several workers have made an attempt to study the wheat diseases and their spread, mechanism of infection, loss due to individual diseases, and mechanism of resistance, epidemiological factors and management aspects. In the present work it has been tried to compile the work which has been done in Karnataka till date so that people can very easily trace the track at one look.

Key words: Wheat disease, Karnataka, rusts, leaf blight, management

Introduction

Wheat, the second most important food crops of the India, contributes nearly one-third of the total food grain production. In Karnataka, wheat is grown on an area of 0.248 mha with a production and productivity of 0.190 million tons and 766 kg ha⁻¹ respectively during *rabi* 1998-99 (Anonymous, 1999). It has already been proved to be the best component under multiple cropping system of the state. Wheat like any other crop, suffers from many diseases caused by fungi, bacteria, viruses, nematodes and other abiotic disorders. However, the diseases caused by fungi are posing severe constraint on wheat productivity. The major diseases noticed in Karnataka include stem rust or black stem rust (*Puccinia graminis* f.sp. *tritici*), leaf rust or brown rust (*Puccinia triticina*), spot blotch (*Helminthosporium sativum*) and foot rot (*Sclerotium rolfsii*). If diseases alone are managed it is possible to harvest 6 to 8 tons per hectare. Identification of resistance sources and gene pyramiding is supportive step in management of these diseases. Hence, constant efforts were made to screen large number of germplasm and breeding materials for resistance to rusts and leaf blight. The efforts have been made to compile the information on epidemiology, race evolution and management of these diseases in Karnataka.

Rusts

Dynamics of rust disease in Karnataka: It has been demonstrated by Joshi *et al.* (1974) that first build up of

leaf rust take place in plains of Karnataka in South India, generally in the last week of December. Normally, the leaf rust appears first during the last week of December or first week of January followed by stem rust (Patil and Kalappanavar, 1994). The first infection of rust comes from the uredospores received from Nilgiri and Palani hills of Tamil Nadu. In 1983, studies conducted on rust incidence dynamics proved that off season wheat grown during *kharif* season in Chikkamagalur district served as secondary foci of infection (Kulkarni, 1984). Due to the intervention of the government, farmers now have stopped cultivation of wheat during off season in Chikkamagalur and Chitradurga districts.

More number of uredospores per microscopic field was observed by glass rod method compared to glass slid method and developed first order auto-regression, logistic, linear, environmental prediction models for leaf rust of wheat in Karnataka (Naragund, 1989). Navi *et al.* (1991) showed considerable daily fluctuation in leaf rust uredospore with weather factors *viz.*, wind velocity, temperature and relative humidity. The marked difference in the leaf rust severity was more related to the type of variety grown in different localities. The wheat varieties *viz.*, DWR-162 and MACS-2496 having vertical resistance were severely affected. Higher frequency of 121R63-1 (77-5) was due to more cultivated area under matching resistance genes conditioning vertical resistance in DWR-162 and MACS 2496 (Hasabnis, 1998).

Loss due to rusts: Several workers have reported that rust of wheat caused significant reduction in 1000-grain weight and in grain number per ear head. A direct relation between yield and infection of leaf rust was established

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by Nargund (1989). Studies conducted on loss estimation due to rust incidence on grain yield and 1000-grain weight differed with respect to the values of "Area under Disease Progress Curve" (AUDPC) of leaf and stem rusts of wheat. Higher values of AUDPC of leaf rust and stem rust in the highly susceptible variety N 59 resulted in 60 per cent reduction in the yield and 53 per cent in thousand grain weight. Lower values of AUDPC of leaf and stem rust were noticed in HD 2189, NIAW 34 and MACS 2846 with a lower loss in yield ranging from 9-13 per cent and loss in thousand grain weights from 7-18 per cent (Hasabnis, 1998). In general, severe infection with both the rusts resulted in maximum reduction in seed germination and seedling vigour (Kalappanavar *et al.*, 2001).

Mechanism of rust resistance: Jalinder (1983) observed the reduction of total and reducing sugars in *P. tritici* affected stem and leaf samples of wheat. Navi (1986) observed considerable reduction in phenol content in susceptible varieties to leaf rust in comparison with resistant varieties. Naragund (1989) reported that higher amount of total phenols in leaf rust affected wheat leaves as compared to healthy leaves at different stages of crop growth. There was higher accumulation of total free phenols and ortho-dihydroxy phenols in the resistant varieties than susceptible ones. At seedling stage, interaction of virulent pathotype 121R63-1 (77-5) with the host showed increased accumulation of phenols. The less virulent pathotype 0R8 (11) and incompatible interaction of HD 2189, HD 4502 and MACS 2846 showed significantly higher accumulation of total free phenols over less virulent pathotype and compatible interactions. The study on peroxidase and polyphenol oxidase on wheat leaf rust interaction revealed that activity of both the enzymes was higher in response to inoculation of the pathogen. HD 4502 variety expressed more activity of peroxidase and polyphenol oxidase as compared to other genotypes (Hasabnis, 1998).

Efforts were made to understand the mechanism of resistance to the leaf rust with resistant varieties such as HD 2189 and HD 4502, which had significantly thicker leaf lamina measuring 153 and 157 μm , respectively as against 147 and 136 μm in susceptible DWR 162 and local red respectively. Epidermal along with cuticular leaf thickness was 20 μm in HD 2189 as against susceptible variety DWR 162 (18 μm) and 18 μm in MACS 2846 as against local red (14 μm). Number of stomata on adaxial leaf surface was significantly higher in DWR 162 (96 /mm²). The breadth of stomata was 39 and 22 μm on abaxil and adaxial surfaces respectively in resistant variety HD 4502 and 38 and 23 μm in MACS 2846 (Hasabnis, 1998). Hegde (1992) reported higher wax content in inoculated and un-inoculated resistant wheat genotypes as compared to susceptible genotypes to leaf rust pathogen.

Sources of resistance: Kulkarni (1979) tested Bijaga Red variety against 15 Indian races of leaf rust and it was found resistant to all except 77B and it was found that *Lr* 9 and *Lr* 19 as resistant source in seedling stage against leaf rust isolates. Gundappa (1983) screened wheat varieties against stem rust of wheat *viz.*, HD 2009, HD 2320, HD 2329, P 2132, Raj 2132, HD 1102, HP 1209, HP 1487, BW 71, BW 75, BW 78, DL 1778, HD 2189, HD 2323, HW 919, HW 888, HW 741, HW 504 and HD 2327 were remained free from infection whereas, terminal severity was 60S on Kalyana Sona and N 59. Jalinder (1983) studied slow rusting mechanism in 16 varieties of wheat. UP 301, DWR 16, HD 2189 and DWR 26 remained free from infection throughout and varieties WH 147, WL 711, Sonalika, C 306 and HD 4502 were identified as slow rusters whereas Agra local, Kalyanasona, Narmada, NI 5439 and Lal bahadur were identified as fast rusters. Navi (1986) while working with slow rusting of leaf rust in bread and durum wheat varieties observed that HD 2278 remained tolerant and C 464, DWR 39, HD 2189 and DWR 16 were identified as slow rusters. Durum varieties (Raj 1555, DWR 137, HD 4502 and DWL 5023) infected later in the season indicating the slow rusting mechanism. Wheat varieties *viz.*, NI 5439, Lal bahadur, Kalyanasona and Sonalika of *Triticum aestivum* and Local red, Agra local, N 59, A-9-30-1 and MACS 1967 of *Triticum durum* were identified as fast rusters. Navi *et al.* (1989) studied slow leaf rusting mechanism in wheat varieties considering parameters *viz.*, long latent period, smaller pustule size, reduced pustule density/cm² of leaf and low ACI. Naragund (1989) identified DWR 39, HD 2189, HI 977, Keerthi, Sonalika, WH 147 and WH 416 of *Triticum aestivum* and Bijaga Yellow, Kiran, MACS 1967 and Raj 1555 of *Triticum durum* as slow leaf rusters on the basis of values of AUDPC. Navi *et al.* (1991) screened 151 promising wheat varieties against *P. recondita* f.sp. *tritici*. Only 10 varieties (HD 2428, PBW 175, HUW 269, NDW 374, HS 207, VL 639, DL 230 7, HD 2402, DL-230-6 and HI 1156) remained free from infection. The slowest leaf rust HD 2189 had long latent period (15 days), smaller pustule size with lower pustule density (6 and 8 number of pustules cm² on 20 and 30 days after inoculation). The leaf rust resistance genes *viz.*, *Lr* 9, *Lr* 19 and *Lr* 24 were found to be effective against virulent race 121R63-1 (Hasabnis, 1998).

Kalappanavar and Hegde (2001) screened 100 genotypes out of which genotypes HS 365, WH 542 and HW 2045 were having less than 5 ACI values to both stem and leaf rusts over years. Kalappanavar and Julie Nicol (2003) screened 140 wheat genotypes received from the CIMMYT, Turkey. They reported that 25 genotypes were resistant to stem rust, leaf rust and leaf blight. Hasabnis and Srikant Kulkarni (2002) evaluated 17 wheat genotypes for slow leaf rusting characteristics. The genotype HD 2189 had lowest (96.35) value of AUDPC as compared to

2173.05 of Agra local. Among the genotypes tested over the years, PBW 500, HW 2004, HPW 42, HS 365, HS 375, VL 829 and DL-788-2 were found to be the most promising rust resistant sources. However, VL 822 and VL 829 showed resistant reaction. (Kalappanavar *et al.* 2003, Kalappanavar *et. al.*, 2003). Hasabnis *et al.* (2003) evaluated 55 wheat varieties to leaf rust and observed that 34 wheat cultivars expressed hypersensitive type of resistance against the pathogen. Considering lower terminal disease score, (Bijaga Yellow, C 306, GW 173, HD 2189, HD 2502, HD 2687, K 8962, PBW 396, Sujatha and WH 542) smaller values of AUDPC and slow rate of infection, these 10 wheat cultivars have been recommended for future wheat improvement programme as good donors of desirable and durable leaf rust resistance. Hasabnis *et al.* (2004) reported that genotypes DWR 162 and MACS 2496 showed susceptible reaction to leaf rust with the severity of 50 and 40 per cent respectively. The genotypes NIAW 34 and HD 2189 showed moderately susceptible reaction to leaf rust of wheat. Hasabnis and Srikant Kulkarni (2004) reported that in intra specific cross HD 4502 / Amrut, the estimated additive gene effects and dominance gene effects were highly significant. The magnitude of additive x additive gene effects was higher and positive whereas dominance x dominance was negative. In interspecific crosses *viz.*, DDK 1001 / Amrut and NP 200/ Amurt, the estimates of additive and dominance effects were highly significant in the cross NP 200 / Local red. Only additive gene effects were highly significant for average co-efficient of infection. The genotypes *viz.*, HD 2189, DWR 236, DWR 241, DWR 39, DWR 16, HD 2278 and NIAW 34 of *T. aestivum* and MACS 2486 and DWR-137 of *T. durum* showed slow leaf rusting.

Prevalence of rust races in Karnataka: The poor productivity of wheat has been attributed to biotic and abiotic stresses. Further, fast evolving *Puccinia* spp. continues to be problematic. Good amount of debate is being made about new black rust Ug 99 in the country. Further, it is alarming to note that some wheat lines available in India are susceptible to Ug 99 race due to presence of Sr 31 gene. Leaf rust race analysis report indicated that leaf race 21R55 was most prominent followed by race 121R63-1. However, races 17R23, 29R23 and 21R63 & 121R55-1 were also prevalent during 2001-02 and 2002-03 respectively. Race 121R55-1 was new pathotype detected in few samples during 2002-03. In the year 2003-04 and 2004-05 the race 121R63-1 was reported to be most predominant and has combined virulence on Lr 23 and Lr 26. Therefore, popular wheat varieties like DWR 162 and MACS 2496 became highly susceptible. In the year 2004-05 a new pathotype (253R31) was reported from few samples. The other leaf rust races reported during 2004-05 were 21R55, 109R63 and 5R37. During 2005-06, as high as seven leaf rust races were recorded. However, race

253R31 was most predominant followed by 121R63-1. Race 253R31 has the virulence on Lr 19. A new pathotype 5R45 was identified on one sample. During 2003-04 the stem rust race 7G11 was isolated where in 2005-06 two stem rust races were isolated i.e. 58G13 and 62G29. A new pathotype 58G13 was observed on more number of samples collected and it is virulent on Sr 5, Sr 7b, Sr 8a, Sr 9b, Sr 9e and Sr 28 (Kalappanavar *et. al.*, 2006).

Management strategies

Navi *et al.* (1986) in a comparative trial with five different sowing dates reported that early sown crop (beginning of November) had less leaf rust. Hunshal *et al.* (1990) compared four different levels of NPK applications, wherein, incidence of leaf rust increased with increase in fertilizer level. Yield also differed significantly among seven wheat cultivars. Optimum dose of nitrogenous fertilizer and seed rate, flat bed method and early planting were found ideal for quantitative resistance. These practices showed lower final disease score (FDS) and lesser value of AUDPC. Gene combination such as Lr 13 + Lr 23 + Lr 24 is most appropriate at the strategic area where inoculation buildup and dissemination takes place. Isolines *viz.*, Lr 13 and Lr 23 help in delaying the epidemics, whereas, Lr 34 in combination with the above genes will slow down the epidemics (Hasabnis, 1998). Kalappanavar and Patil (1998) investigated the ability of fungicides to control *P. recondita* f. sp. *tritici* on wheat. The most effective fungicide was cyproconazole (San 619) whereas, Mencozeb was least effective. The highest yield was observed on the plot treated with Cyproconazole. The fungicide Propiconazole performed best followed by Triadimefon and Hexaconazole. However, neem leaf extract, *Trichoderma harzianum* and *Panchgavya* were the succeeding treatments effective against leaf rust of wheat. The yield of Propiconazole, Triadimefon and Hexaconazole sprayed plots were significantly superior over control indicating marked influence of the leaf rust on yield. The 1000-grain weight was also significant in the above said treated plots compared to other treatments.

Leaf blight

After the impressive progress in managing leaf and stem rust in modern varieties, leaf blight caused by *Helminthosporium sativum* has emerged as the most important disease of second – generation wheat genotypes. Meli (1993) conducted a survey during *rabi* 1992-93 in transitional belt of Karnataka *viz.*, Belgaum, Dharwad and Gadag districts. He recorded maximum disease severity of 47.95 per cent from Saundatti taluk in Belgaum district and 53.46 per cent from Gadag district. Recently leaf blight appeared in severe form in Gokak taluk followed by Belgaum, Raibag, Athani and Dharwad. Over all effects of the disease resulted in reduction in thousand

grain weight (18%), grain yield (31%), and plant height (7%) and biomass (19%) (Patil, 2000).

Morphology: Bidari and Govindu (1980) showed that both unipolar and bipolar germination of conidia of *H. sativum* occurred after two hours of incubation on host leaf surface. Appressorial formation was observed six hours after incubation on host surface. They further, observed that, germ tube may and in a single round to oval brownish appressorium which was on or near the stomata. The penetration peg from appressorium was through stomata or directly through epidermis or between two cells. Kulkarni *et al.* (1985) described *Exerohilum hawaiiensis* on wheat as conidiophores solitary, geniculate, septate measuring 120 x 2.7 µm. The conidia were straight, ellipsoidal, oblong or oval shaped round at the ends, brownish in colour and symmetrical in shape, pale to slight brownish in appearance. The size of conidia varied from 16.18 µm – 80.08 µm x 13.2 µm – 30.2 µm and average size of spore being 39.09 µm x 18.13 µm with pseudoseptatons five to eleven. Hiremath (1985) examined *E. hawaiiensis* on wheat and found that variation in spore size as well as number of pseudosepta. Conidia were straight, ellipsoidal, and oblong or oval shaped round at the ends brownish in colour conidiophores solitary, geniculate and pale to light brown in colour.

Nagaraj (1980) observed maximum mycelial growth at 25°C and the least growth at 10°C in case of *Drechslera sorokiniana*. Meli (1993) studied the effect of different temperature levels on the growth of isolates of *E. hawaiiensis* and reported that the temperature of 30°C was found to support significant growth over temperature levels tested. Patil (2000) observed better growth of mycelium of all isolates of *E. hawaiiensis* at 30°C followed by 25°C. An attempt was made to study the germination of conidia and germination was seen on slide and also in resistant and susceptible genotypes. Temperature of 25°C and pH 7 was found optimum for maximum germination of *H. sativum* (Chandrashekar, 2003). Nagaraj (1980) reported that best growth of *D. sorokiniana* at pH 5.8. However, Patil *et al.* (1964) reported that minimum growth of the *D. sorokiniana* was obtained at pH 6.2 followed by 6.0 which were superior to other pH levels. Raguchandra *et al.* (1988) concluded that, growth was best at pH 6.6 for *Bipolaris sorokiniana*. Hiremath *et al.* (1989) recorded the maximum growth of *D. hawaiiensis* was at pH 6.0 which differed significantly from the other pH levels in supporting the growth of the fungus. Meli (1993) observed maximum growth of different isolates of *E. hawaiiensis* at pH 6.0 and no significant difference in growth at pH levels 5.5, 6.5 and 9.0. Patil (2000) recorded maximum weight of *E. hawaiiensis* at pH levels between 6.0 and 6.5 for all the eight isolates tested.

Epidemiology: In Karnataka, wheat is usually sown during mid October to early December. The leaf blight symptoms

appear during the crown root initiation stage of the plant. The severity of the disease is judged by the age of the seedling. Bidari and Govindu (1975) reported *H. sativum* on wheat was able to infect 16, 32 and 48 days old plants. Later stages of growth were found to be more susceptible than earlier stages. Ten days old seedlings were found most susceptible and maximum infection was noticed at 60 days old plants. Studies on survivability of pathogen indicated short span of few weeks in the soil. Kulkarni *et al.* (1985) for the first time reported its occurrence on wheat in severe form in Karnataka. The pathogen was found to survive under laboratory conditions for 21 months, under natural conditions for 15 months and under refrigerated conditions for 27 months and remains viable in seed for 28 months. Bidari (1973) studied the survival of three isolates of *H. sativum* in wheat seed, plant debris and soil and it was found that all the three isolates survived for more than nine months. The blotter test method conducted on wheat seeds confirmed that black point was due to *H. sativum* and the pathogen was found deeply seated under the seed coat resulting in seed rotting, seedling infection and pre and post emergence death of seedlings. Bidari and Govindu (1975) studied the three isolates of *H. sativum* and compared. The leaf isolates were more virulent than the neck and ear head isolates. The leaf, neck and ear head isolates were grouped as virulent forms respectively. The perfect stage of the pathogen could not be detected although very old infected straw material was carefully observed for perithecia. Much variation in leaf blight pathogen (*H. sativum*) was observed in Karnataka and was designated into eight isolates as A, B, C, D, E, F, G and H. These isolates were grouped into three pathotypes as highly virulent - A, B, E & G from Arabhavi, Kittur, Raichur and Gangavati, moderately virulent - C, D & F from Bagalkot Bijapur and Siraguppa and least virulent - H from Navalagund (Meli, 1993). Subramanyam *et al.* (1990) reported that spores of *E. hawaiiensis* germinated on inoculated leaf pieces of a susceptible cultivar by producing several germ tubes. These germ tubes formed appressoria on the epidermis and over the stomata indicating both direct and indirect penetration

Collateral hosts: The pathogen could infect *Chloris barbata*, *Chloris gayana*, *Dactyloctenium aegyptium*, *Eleusine coracana*, *Oryza sativa*, *Sorghum bicolor*, *Hordium vulgare*, *Secale cereale* and *Zea mays* under artificial inoculations (Hiremath *et al.*, 1984, Raguchander *et al.*, 1988; Subramanyam, *et al.*, 1990; Ramachandra, 2000). The cross inoculation on wheat was also successful, hence they may act as alternative to the pathogen.

Mechanism of infection: The seed borne nature of the disease was confirmed by disease transmission study at this center and the pathogen is transmitted from seed to plant systemically. The histo-pathological studies of the infected leaf revealed that the mode of entry of this pathogen was

both by direct (by producing appressorium on cuticle) and indirect (by penetration of hyphae through stomata) (Subramanyam *et al.*, 1990; Sasalatti and Kalappanavar, 2003). After penetration the mycelial mat was profusely distributed within the host tissue. Due to this, mesophyll cell become disorganized and cause damage to epidermal cells. During severe infection the vascular bundles with bundle sheath were found to be destroyed, causing severe damage to the leaf tissues (Chandrashekar, 2003; Ramachandra and Kalappanavar, 2004).

Mechanism of resistance: Subramanyam *et al.* (1990) studied the influence of inoculation of *D. hawaiiensis* (Bugnicourt) Subram and Hain on bio-chemical parameters of wheat leaves. They found higher total and reducing sugar in resistant cultivars than in susceptible ones and further they observed that both the sugar content decreased after the disease development on both susceptible and resistant cultivars. Nandagopal (1995) reported that the wheat genotypes resistant to *E. hawaiiensis* had lower crude protein and noticed higher content of epicuticular wax in wheat genotypes resistant to infection by *E. hawaiiensis* compared to susceptible genotypes. Nandagopal (1995) found no difference in number of stomata in both resistant and susceptible wheat genotypes to *E. hawaiiensis*. However, he recorded more number of stomata on abaxial surface than the adaxial surface. Naragund *et al.* (1996) studied the histopathological changes of infected leaves of wheat caused by *E. hawaiiensis*. The epidermal cells and mesophyll cells of healthy leaves were compact and the vascular bundles were surrounded by bundle sheaths. In infected leaf the penetration of the fungus took place through the stomatal opening and directed through cuticle. The mycelium was ramified profusely in the host tissue, ultimately destroying the epidermal and mesophyll cells. Under the severe infection the bundle sheath and the vascular bundles were disrupted. In the recently concluded study, it was observed that higher epicuticular wax, phenols, soluble proteins, sugars, cuticular and epidermal cell layer thickness and lesser length, breadth and number of stomata were responsible for resistance to *H. sativum* infection (Sasalatti and Kalappanavar, 2003, Pradeep Kumar, 2005).

Sources of resistance: Hiremath (1985) screened 140 wheat varieties against *H. sativum* and found that majority of varieties showed immune reaction. Forty varieties were found to be moderately resistant 30 and were moderately susceptible and six showed susceptible reaction. Chattannavar *et al.* (1987) evaluated 527 wheat genotypes under artificial epiphytotic conditions against *E. hawaiiensis*. Forty genotypes were found to be highly resistant to the pathogen. Subramanyam (1989) screened 172 wheat genotypes against *E. hawaiiensis* and revealed that none of the varieties had immune reaction. Eight varieties showed resistant reaction, 83 varieties

were moderately resistant, 81 varieties were moderately susceptible while 50 showed susceptible reaction. Disease incidence survey conducted over years revealed that leaf blight incidence was more prominent in dicoccum and durum wheats as compared to bread wheats.

Disease management: Ramachandra (2000) assayed cold aqueous extracts of 16 plant species against *E. hawaiiensis*. *Duranta repens* at 5 and 10 per cent concentration showed significant inhibition of mycelial growth of *E. hawaiiensis* in both laboratory and at field level (62.40 and 68.11%). Spraying 20 per cent aqueous extract of *Duranta repens*, *Azadiracta indica* and *Ocimum sanctum* exhibited significant inhibition of mycelial growth of *H. sativum* to the maximum extent. Chattannavar *et al.* (1983) reported that, RH-2161 was the best followed by Tridemorph / Dithane-M-45. In the management of blight caused by *D. hawaiiensis* obtained best control of the disease was with Fenapanil at 0.1% and Mancozeb at 0.2% (Subramanyam *et al.*, 1990). Seed treatments with Capton or Mancozeb @ 2.5 g/kg of seed or combination of either of them with *T. viride* (4 g/kg seed) provided complete control of the primary seed borne inoculum. The seed treatment also had better impact on seed germination and seedling vigour. Among the different priming agents tested for seed treatment, Poly Ethylene Glycol (40%) with Quintal (Iprodione + Carbendazim) exhibited maximum germination and vigour index with least infection (Manjunatha *et al.*, 2001). Application of Propiconazole, Hexaconazole, Prochloraz and Quintal @ 0.1 per cent gave good control of leaf blight in the field (Chandrashekar, 2003, Ramachandra and Kalappanavar, 2004). Under laboratory conditions, biological agents' viz., *Pseudomonas fluorescens*, *T. viride* and *T. harzianum* were found to have antagonistic property to leaf blight pathogen (Ramachandra, 2000).

Foot rot

Foot rot of wheat caused by *Sclerotium rolfsii* is a serious problem mainly in the rainfed eco-system. Survey indicated that foot rot caused by *S. rolfsii* present in rainfed wheat areas of Karnataka (Kulkarni S., 1979; Naragund, 1981; Reddy *et al.*, 1971). The loss due to this disease is in the range of 10-30 per cent. It has been observed that *S. rolfsii* from wheat showed marked increase in population in sterilized sandy loam at all pH levels tested (Chattopadhyay and Mustafee, 1979). Nargund *et al.* (1982) studied the inoculum level of *S. rolfsii* and minimum of two percent inoculum of *S. rolfsii* was necessary to cause the disease. The disease intensity increased with the increase in inoculum.

Resistant sources: Reddy *et al.* (1971) found that Lerma Roja 64A was most resistant and Napo-63 the least against *S. rolfsii*. Kulkarni *et al.* (1978) screened thirty five wheat cultivars against *S. rolfsii*. They found that CC 464 LPP 301 and HD 2189 showed maximum resistance to the

foot rot disease. Nargund *et al.* (1982) screened twenty-five wheat genotypes including varieties and multilines, Lerma Roja showed maximum disease resistance while Agra local was highly susceptible. Among the several varieties tested only few varieties viz., WL 711, HI 977, K 8705, HP 1660, HUW 342, HD 2189, HS 240, NP 200, UP 2565, HS 420, PBW 343, RAJ 3765, and PBW 373 were found to have marked tolerance to foot rot incidence (Kalappanavar *et al.*, 2001).

Biological control: Hegde *et al.* (1980) reported effective control of *S. rolfii* with *Bacillus subtilis* *in vitro*. Manjappa (1979) reported that *T. viride* and *Penicillium sp.* were found to be antagonistic to *S. rolfii*. Wheat bran, paddy hulls, farm yard manure, safflower, oil cake and groundnut oil cakes were tested in artificially infested soil for control of foot rot of wheat. Groundnut and safflower oil cakes were more effective in reducing the mortality of wheat seedling and thus helped in increasing the yield. This was followed by FYM, paddy husk and wheat bran. Among the organisms isolated from amended field *T. viride*, *Penicillium sp.*, *B. subtilis* and *Streptomyces sp.* were antagonistic to *S. rolfii* (Naragund *et al.*, 1982). Harlapur and Kulkarni (1995) and Kalappanavar and Patil (2000) reported that seed treatment with *T. harzianum* @ 4g per kg seed was found effective in controlling the foot rot infection under rainfed situation. *Glomus fasciculatum* a mycorrhiza has also been found to be effective in controlling the disease. The biological agent is cheaper, eco-friendly and no side effect on beneficial soil micro flora unlike inorganic chemicals. Kalappanavar *et al.*, (2001) reported that plant extracts of *Parthenium hysteriphorus* and *Polyalthia pentula* inhibited the mycelial growth of *S. rolfii* effectively.

Chemical control: Several workers reported the effect of fungicides on foot rot organisms (Kulkarni, 1980, Kulkarni and Hegde, 1978). Plantvax (Oxycarboxin) and vitavax (Carboxin) as seed treatment of 2g/kg gave effective control of *S. rolfii* up to 35 days after seeding ((Reddy *et al.*, 1971). Brassicol, Panoram, Panocrine-35, Vitavax and Plantvax were highly effective and Bavistin, Bayleton, RH-124 and Calixin were less effective against *S. rolfii* (Nargund, *et al.*, 1982). Bayton, Vitavax and Brassicol have been found to be effective as soil drenching and seed dressing fungicides (Kulkarni, 1980). In general, seed treatment with Carboxin or Thairam or Capton @ 2.5 g/kg of seed is effective in controlling the disease.

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