Review Paper Open Access

Banded leaf and sheath blight: an emerging disease of maize (*Zea mays* L)

Anshuman Singh[1]*, JP Shahi[1]

[1]Department of Genetics and Plant Breeding, Institute of Agricultural Sciences, Banaras Hindu University, Varanasi 221005, Uttar Pradesh, India
*Corresponding author: E-mail: singhanshuman@rediffmail.com

Abstract

The basidiomycetes fungus *Rhizoctonia solani* is a major pathogen of maize worldwide, particularly in China, South Asian and South East Asian countries. It causes banded leaf and sheath blight (BLSB) on plants, which is considered an emerging disease, accompanied by small losses to total wipeout of the crop. This disease is more prevalent in humid weather with temperature around 28°C. The genetics of inheritance of this disease is unclear. Digenic as well as oligogenic inheritance of disease has been reported. A number of QTLs have been identified which will help to expedite breeding program against BLSB. Moreover, various chemical and biological control methods have been developed, but major emphasis is on development of maize cultivars with genetic resistance to BLSB for environment friendly control of the disease.

Keywords: maize, Banded Leaf and Sheath Blight, *Rhizoctonia solani*

Introduction

Maize (*Zea mays* L) is third most important cereal crop in the world agricultural economy and is a relevant source of food, feed, and industrial products. In India maize ranks fifth position in area and fourth in production among the major cereals grown. Being a C4 plant and having very high yield potential, it is called queen of cereals. One of the main deterrents to high grain yield in maize is its susceptibility to several diseases.

Of 112 diseases of maize reported so far from different parts of the globe, 65 are known to occur in India. Banded leaf and Sheath blight (BLSB) is one of them caused by most widespread, destructive and versatile pathogen *Rhizoctonia solani* f. sp Sasakii (teleomorph: *Corticium sasakii*, syn *Thanatephorus cucumeris*) which claims significant yield loss (Saxena, 2002). It was first reported by Bertus (1927) in Sri Lanka under the name Sclerotial disease. Since, it develops on leaf and sheath, the symptoms appear in concentric spots that cover large area of infected leaf and husk. The pathogen spreads from the basal sheath to the developing ear under favorable environmental conditions. The main damage reported in the humid tropics is a brownish rotting of ear, which shows conspicuous, light brown, cottony mycelium with small, round and black sclerotia.

Distribution

This disease has been reported in Germany, USA, Nigeria, Venezuela, Sierra Leone, Ivory Coast and England. In particular, BLSB is recognized as a serious impediment to maize production in China, South Asia and Southeast Asia (Sri Lanka, Indonesia, Cambodia, Bangladesh Pakistan, Nepal, Myanmar, Thailand, Laos, Vietnam, Philippines, Taiwan, Malaysia, Korea and Japan). Surprisingly, in China, yield losses close to 100% have been attributed to BLSB.

Economic importance

This disease causes a considerable reduction of high yielding varieties. In country like India, Lal et al (1980) have estimated in ten cultivars a loss in grain yield ranging from 23.9 to 31.9%, whereas Singh and Sharma (1976) estimated 10-40% in other cultivars. Lal et al (1985) had suggested that grain yield loss can go up to an extent of 90%. In Guangxi province of China, yield losses of 87.5 and 57.8% were recorded under natural conditions in the hybrids Luyu 13 and Guiding planted at Bao Qiao and Chen Xiang (Sharma, 2005). In addition to anastomosis group *R. solani* AG-1 IA, a major pathogen of maize (Gonzalez-Vera et al, 2009), Buddemeyer et al (2004) conducted studies to estimate the damages, caused by *R. solani* AG2-2IIIB, for different maize cultivars under German growing conditions of sugar beet-maize cropping system. They reported that shoot fresh matter and grain yield of infested plants as compared to healthy plants were reduced up to 37 and 12%, respectively. In USA, Sumner and Minton (1989), by planting maize in infested and non-infested plots with high and low inoculum levels, reported yield reduction of 42 and 8% in soils infested with high inoculum level, while the same was 17 and 1% under low inoculum level for a period of three years.

 RECEIVED 11/05/2012

Environmental conditions and disease symptoms

High relative humidity and rain fall significantly favors development and spread of this disease. An optimum temperature about 28°C and high relative humidity (88 to 90%) in the first week of infection favor rapid disease progress. If the relative humidity goes below 70%, disease development and spread becomes slow (Sharma, 2005). Additionally, high crop densities impact disease severity.

It was, generally, reported that this disease appears at pre flowering stage in 40-50 days old plant (Saxena, 2002). The disease develops on leaves, sheaths, and stalks and can spread to the ears. Typically, disease develops on first and second leaf sheath above the ground as this disease is soil borne and eventually extends to the ears that ultimately lead to ear rot. When infection reaches ear, light brown cottony mycelial growth and small round mustered seed sized small round black sclerotia are observed. Premature drying and caking of ear sheath is also observed. Crop damage is caused by loss of photosynthetic leaf area due to foliar infection and stalk rot which lead to crop lodging (Lu at al, 2012). Similarly Ahuja and Payak (1982) found that maximum damage is caused when ears are infected. In addition to ear rots, kernels are often wrinkled, dry, chaffy and light in weight. These symptoms are stalk lesions, stalk breakage, clumping and cracking of silk and horse shoe shaped lesions on caryopsis.

The pathogen (*Rhizoctonia solani*)

R. solani is generally identified by characteristics of the mycelium and sclerotia as it lacks spores formation. Mycelium often is colorless at young stage, while turns to light brown as it matures. The characteristics of hyphae of Rhizoctonia are a) branching near distal septum of cells in young vegetative hyphae; b) formation of septum in the branch near the point of origin, c); construction of branch; d) dolipore septum; e) no clamp connection; f) no conidium; g) sclerotium not differentiated in rind and medulla and h) no rhizomorph (Ogoshi,1975). The diameter of vegetative hyphae is 8-12 µm and is constricted at the point of branching. The mature hyphae branch at right angle and sclerotia are produced abundantly in culture and on infected plant parts. Mostly, sclerotia are 1 to 5 mm in diameter with spherical shape, and dark brown to black colour.

R. solani survives in the soil and on infected crop debris in form of sclerotia or mycelium. Sclerotium acts as primary source of inoculum. Sclerotia are known to survive for several years in the soil. The fungi spread by irrigation, movement of contaminated soil and infected plant debris. At the onset of the growing season, in response to favourable humidity and temperatures (15 to 35°C), the fungal growth is attracted to newly planted crops by chemical stimulants released by growing plant cells.

Secondary spread of this disease occurs by contact of diseased leaves or sheaths with healthy plants. Although horse shoe shaped lesions are caused by the pathogen on kernels, the kernels are not considered as source of inoculum.

Breeding of lines resistant to disease caused by Rhizoctonia has not been extensively studied: Rhizoctonia very likely has a widest host range and differences among isolates are not obvious (Leach and Garber, 1970). However, with the concept of interspecific groups (ISGs) and anastomosis groups (AGs), the potential for breeding resistant varieties has improved. The scheme of anastomosis group was first suggested by Schultz in 1937 and later developed by Richter and Schneider in 1953. Presently at least 14 anastomosis groups have been reported in *R. solani*. Five of 12 AGs have been further divided into sub groups according to culture appearance, pathogenicity, and thiamine requirement. Considering AG classification too general Ogoshi (1987) has introduced the concept of inter specific groups as a more specific category of variation. This recognizes grouping based on combined evidence from anastomosis behavior, pathogenicity, morphology and other ancillary evidence from serological studies (Adam and Butler, 1979), fatty acid analysis (Jhonk and Jones, 1993), protein electrophoresis (Reynolds et al, 1983), and nucleic acid studies. Thus the number of interspecific groups recognized in *R. solani* complex has steadily increased over recent years.

Genetics of resistance to BLSB

In spite of several years of research in this area, the genetics of inheritance of resistance to BLSB is not clear. This causes a major bottle neck in breeding of maize cultivar resistant to BLSB. There are very limited sources of germplasm available which can give high level of tolerance over locations under different environments. Hybrids developed through crossing of tolerant inbred lines show inconsistent performance. This may be attributed to inadequate knowledge about mode of inheritance of resistance, genotype x environment interactions for resistance and possible presence of different races.

Vimla et al (1988) conducted a combining ability analysis for resistance to BLSB. They concluded that both general and specific combining abilities varied significantly for controlled disease resistance but general combining ability variance was predominant. They also identified CM104 as an important donor for resistance against BLSB. Kumar and Singh (2000) studied inheritance of resistance to BLSB on the basis of the analysis of 10 crosses. Eight crosses were made between two resistant (CM104 and CML1) and four susceptible inbred lines, one cross each was made between resistant x resistant and susceptible x susceptible lines. Parents, F_1's, F_2's, and backcrosses, were included in the study. Final evaluation was

made under artificially created epiphytotic conditions. The F_2 segregation pattern for BLSB reaction was approaching a 15:1 mendelian ratio in crosses involving CM104 as the resistant parent, and approaching a 13:3 ratio in crosses involving CML1 as the resistant parent. Based upon F_2 segregation analysis of eight susceptible x resistant crosses they concluded that resistance was governed by two genes. The BLSB reaction in F_2 and backcrosses involving CM104 and susceptible lines suggested that resistance in CM104 was controlled by duplicate dominant genes, while crosses of CML1 showed dominance and recessive interaction pattern of segregation. Yang et al (2005) conducted inheritance analysis and indicated that resistance to BLSB in maize would be controlled by approximately 4-7 pairs of major genes.

A large body of efforts is being diverted towards development of biotechnological tools for identification and tagging of genes conferring resistance to BLSB. The identification of quantitative trait loci (QTL) for resistance to BLSB is considered as an efficient tool in development of disease resistant maize hybrids. The information generated from mapping resistance genes can be used in marker assisted selection (MAS) programmes for development of BLSB resistant lines. In another experiment, results indicate that molecular markers linked to target rQTL can facilitate pyramiding resistance to multiple diseases during early generation of pedigree selection (Asea et al, 2012). Zhao et al (2006) screened a mapping population consisting of 229 F_2 individuals, derived by crossing inbreds R15 (resistant) with 478 (susceptible), against *R. solani* AG1-IA at two locations. They constructed a genetic linkage map, containing 146 single sequence repeat (SSR) markers, on the basis of composite interval mapping, and identified 11 QTLs for resistance to BLSB located on chromosomes 1, 2, 3,4, 5, 6, and 10. However only four QTLs located at chromosomes 2, 6, and 10 were identified across both locations. Lin et al (2008) analyzed digenic epistatic and QTL x environment interactions for resistance to BLSB and detected seventeen QTLs including 12 pairs of digenic epistatic QTLs. These QTLs were distributed on seven chromosomes (2, 3, 4, 6, 7, 9, and 10). Chen et al (2009) identified four QTLs for resistance to BLSB distributed on chromosomes 6, 7, and 10.

Breeding for disease resistance

There is less genetic variability for resistance to BLSB as compared to other diseases. This is a major constrain in resistance breeding programme for BLSB. However, national programmes in India, China, Indonesia, and Philippines are directed in screening for BLSB resistance. In All India co-ordinated maize improvement programme inbreds and hybrids are both evaluated for their reaction against BLSB. Kumar and Singh (2002) reported CM104 and CML1 as resistant inbreds. Among the inbreds, CA00106 showed moderate resistance to all three isolates collected at various locations (i.e. Udaipur, Delhi, and Pantnagar). Previously inbred CM104 was considered as an elite line for BLSB resistance, but it showed susceptibility at Delhi where as CM105 was susceptible at Udaipur and Delhi. Among the CIMMYT inbred lines, CA00310 was moderately resistant at Udaipur and Delhi, while CA00344 and CA00370 were moderately resistant at Pantnagar and Delhi (Garg et al, 2007). This study revealed presence of significant genotype x environment interactions for resistance to BLSB and possible presence of different races at these hot spots (Delhi, Udaipur and Pantnagar). Bhavana and Gadag (2009) also screened 30 inbred lines at these three hot spots under artificial inoculaton conditions and identified inbred lines Pop145 and Suwan-1 as highly tolerant to BLSB. In China, Yang et al (2005) screened maize germplasm during 1997-2000 and identified inbred line CML270 as highly resistant against pathogen *R. solani* AG1-IA collected from different locations. A number of maize inbered lines developed by co-ordination between CIMMYT and national crop improvement programmes of different countries are being utilized to develop resistant lines. Main programme for evaluation of these lines is being conducted in China and India. Sharma et al (2002) reported lines (*viz*, PT 9630 18-1-B-B-B-B-B, Pop 352 co-hs 74-2-1-b-b, Pop145 co-hs-49-1-b-b-b-b, TOO 14901, TOO 14903, TOO 14903, TOO G1 802, CA 14510, CA 14524,CA 14522, TOO 35101, TOO 00310, IPA-2-2-f-1 and Suwan-1 (S) C #-B-B) as tolerant to BLSB on the basis of 2-year study.

Disease management

Due to ambiguity in understanding of inheritance of resistance and non-availability of widely adapted and stable source of resistance to BLSB, control of disease by cultural, biological and chemical procedures is extremely important to minimize the destruction of crop and to prevent economically crop losses.

Saxena (2002) tested efficacy of chemicals (*viz*, Propaconazole, 0.1%, and Carbendazim, 0.05%), by applying as foliar sprays at 30, 40 and 50th day of planting, alone or in combinations. Effectiveness of Propaconazole was markedly observed when the chemical was applied at initial stages at 30th or 40th day after planting and the second spray at 10 days after first. Foliar sprays of Carbendazim showed the ineffectiveness against BLSB. On *in vitro* evaluation, three often used fungicides, namely Bavistin, Rhizolex, and Thiophenate M, have shown absolute control of mycelial growth with 100% inhibition. It is, therefore, envisaged that under field conditions a high level of control of BLSB could be achieved using these three fungicides (Sharma et al, 2002). The antibiotic Validamycin was able to give only 56.3% inhibition at 30 ppm.

Several micro-organisms are known to parasitize Rhizoctonia species. These are mainly fungus of spe-

cies Trichoderma, Gliocladium, and Laetisaria, bacteria (*Pseudomonas fluorescence*), and nematodes (*Aphelenchus avenae*). Reduction in disease incident of BLSB was observed when *P. fluoresence* was used in seed and soil treatment and in foliar application (Meena et al, 2003). Seed treatment and soil application of this antagonist not only reduces the disease to more than 50%, but additionally Sharma et al (2002) recorded consequent increase in grain yield approximately 1.4-times of the yield of the control.

Another biocontrol agent, named *Trichoderma harzianum*, also provided as high as 68% of inhibition of the mycelia of *R. solani*, under *in vitro* conditions, compared to the control of BLSB (Sharma et al, 2002). Formulations of anti-biotic Validamycin also show good control against BLSB (Jiang et al, 1991) but due to high cost, Validamycin does not appear to be profitable proposition (Sharma et al, 2002).

For the cultural control of *R. solani*, selection of a well drained field and planting on raised beds are important aspects to avoid contact of water with seeds and faster growth of seedlings. Composting of hardwood on Rhizoctonia-infested soil has been found to reduce disease severity, apparently by promoting the growth of Trichoderma and other antagonistic microorganisms (Hoitink, 1980).

Conclusions

All the above mentioned measures of controlling BLSB can be implemented depending upon the conditions. Variability within pathogen should be considered for screening and breeding for resistance, or while testing sensitivity of the pathogen towards different chemicals. An integrated approach using agronomic, nutritive, or chemical controls should be adopted for an effective disease management. Development of resistant varieties using conventional as well as biotechnological methods will help in controlling this menacing disease which is still a challenge even after eighty five years of its discovery.

References

Adams GC Jr, Butler EE, 1979. Serological relationship among anastomosis groups of *Rhizoctonia solani*. Phytopathol 69: 629-633

Ahuja SC, Payak MM, 1982. Symptoms and signs of banded leaf and sheath blight in maize. Phytoparasitica. 10: 41-49

Asea G, Vivek BS, Lipps PE, Pratt RC, 2012. Genetic gain and cost efficiency of marker-assisted selection of maize for improved resistance to multiple foliar pathogens. Mol Breeding 29: 515-527

Bertus LS, 1927. Year book, pp. 44-46. Department of Agriculture, Ceylon

Bhavana P, Gadag RN, 2009. Evaluation of maize genotypes for resistance to banded leaf and sheath blight. Annals of Plant Protect Sci17: 498-499

Buddemeyer J, Pfahler B, Peterson J, Marlander B, 2004. Genetic variation in susceptibility of maize to Rhizotonia solani (AG2-2IIIB)-symptoms and damage under field conditions in Germany. J of Plant Diseases and Protect 111: 521-533

Chen GP, Tan H, Zheng DB, Yang LT, Li YR, 2009. QTL mapping of resistance to banded leaf and sheath blight in maize. Southwest China J of Agric Sci 22: 950-955

Garg A, Prassana BM, Sharma RC, Rathore RS, Saxena SC, Chauhan SVS, 2007. Identification of resistance source to banded leaf and sheath blight (*Rhizoctonia solani* F. sp sasakii) in maize. Indian Phytopathol 60: 162-166

González-Vera AD, Bernardes-de-Assis J, Zala M, McDonald BA, Correa-Victoria F, Graterol-Matute EJ, Ceresini PC, 2009. Divergence between sympatric rice- and maize-infecting populations of *Rhizoctonia solani* AG-1 IA from Latin America. Phytopathol 100: 172-182

Hoitink HAJ, 1980. Composted bark a light weight growth medium with fungicidal properties. Plant Dis 64: 142-147

Jhonk JS, Jones RK,1993. Differentiation of population of AG2-2 of *Rhizoctonia solani* by analysis of cellular fatty acids. Phytopathol 83: 278-283

Jiang HL, Ding X, Ma H, 1991. The occurrence and chemical control of maize sheath blight (*Rhizoctonia solani*) in Ragao of Jiangsu. Plant Prot 6: 11-12

Kumar R, Singh IS, 2002. Inheritance of resistance to banded leaf and sheath blight (*Rhizoctonia solani* F. sp sasakii) of maize (*Zea mays* L), pp 356-365. Proceedings of the 8th Asian Regional Maize Workshop, Bangkok, Thailand

Lal S, Baruah P, Butchaiah K, 1980. Assessment of yield losses in maize cultivars due to banded leaf sclerotial disease. Indian Phytopathol 29: 129-132

Lal S, Butchaiah K, Baruah P, 1985. Comparative efficacy of some fungicides and antibiotics in control of banded sclerotial disease of maize. Pesticides, 19: 17-19

Leach LD, Garber RH, 1970. Control of *Rhizoctonia solani*, pp 189-198. In: *Rhizoctonia solani*, Biology and Pathology. Parmeter JR Jr ed.

Lin HJ, Tan DF, Zhang ZM, Lan H, Gao SB, Rong TZ, Pan GT, 2008. Analysis of digenic epistatic and QTL x environment interactions for resistance to banded leaf and sheath blight in maize (*Zea mays*). Int J for Agri and Bio10: 605-611

Lu YL, Xu J, Yuan ZM, Hao ZF, Xie CX, Li XH, Shah T, Lan H, Zhang SH, Rong TZ, Xu YB, 2012. Comparative LD mapping using single SNPs and haplotypes identifies QTL for plant height and biomass as secondary traits of drought tolerance in maize. Mol Breeding 30: 407-418

Meena RL, Rathore RS, Mathur K, 2003. Efficacy of bio-control agents against *Rhizoctonia solani* f.sp sasakii causing banded leaf and sheath blight of maize. J of Mycol Plant Pathol 33 (2): 310-312

Ogoshi A, 1975. Grouping of *Rhizoctonia solani* Kühn

and their perfect stages. Review on Plant Protection Res 8: 93-103

Ogoshi A, 1987. Ecology and pathogenicity of anastomosis and interspecific groups of *Rhizoctonia solani* Kühn. Ann Review of Phytopathol 25: 125-143

Reynolds M, Weinhold AR, Morris TJ, 1983. Comparison of anastomosis groups of *Rhizoctonia solani* by poly acrylamide gel electrophoresis of soluble proteins. Phytopathol 83: 278-283

Richter H, Schneider R, 1953. Untersuchungen zur morphologischen und biologischen Differenzieung von *Rhizoctonia solani* K – Phytopathol Z 20: 165–226

Saxena SC, 2002. Bio-Intensive Integrated disease management of banded leaf and sheath blight of maize, pp. 380-388. In: Proceed of 8th Asian Regional Maize Workshop: New Technologies for the New Millennium, Bangkok, Thailand

Schultz H, 1937. Vergleichende Untersuchungen zur Ökologie, Morphologie und Systematic des "Vermehrungpilzes". Arb Biol Reichanst Land Forstwirtsch Berlin-Dahlem 22: 1-41

Sharma RC, 2005. Banded leaf and sheath blight (*Rhizoctonia solani* F. sp sasakii) of maize, pp159-171. In: Stresses of maize in Tropics. Zaidi PH, Singh NN eds.

Sharma RC, Vasal SK, Gonzalez F, Batsa BK, Singh NN, 2002. Redressal of banded leaf and sheath blight of maize through breeding, chemical and biocontrol agents, pp. 391-397. In: Proceed of the 8th Asian Regional Maize Workshop: New Technologies for the New Millennium, Bangkok

Singh BM, Sharma YR, 1976. Evaluation of maize germplasm to banded sclerotial disease and assessment of yield loss. Indian Phytopathol 29: 129-132

Sumner DR, Minton NA, 1989. Crop losses in corn induced by *Rhizoctonia solani* AG2-2 and nematodes. Phytopathol 79: 934-941

Vimla B, Mukherjee BK, Ahuja VP, 1988. Combining ability analysis for resistance to banded leaf and sheath blight of maize. Indian J of Genet 48: 75-79

Yang JP, Tang HT, Yang JX, Li X, Chen DQ, Zhang B, Shi YG, Huang YX, 2005. Indentification and inheritance of resistance of maize germplasm to sheath blight (*Rhizoctonia solani*). Acta Phytopathologica Sinica 35: 174-178

Zhao MJ, Zhang ZM, Zhang SH, Li W, Jeffers DP, Rong TZ, Pan GT, 2006. Quantitative trait loci for resistance to banded leaf and sheath blight in maize. Crop Sci 46: 1039-1045