

Banded leaf and sheath blight: threat to maize

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Abstract: Banded leaf and sheath blight (BLSB) is a destructive disease of maize and it leads to 100 per cent yield losses. The causal organism of BLSB is *Rhizoctonia solani* and the organism grows under favourable humidity (88–90 per cent) and temperature (15–35°C) conditions. The management practices used to control the disease spread are inadequate at this moment due to its broad host range. Therefore, the identification and exploitation of genetic resistance is an economical and sustainable option to control this disease. However, limited genetic variability poses a major challenge for resistance breeding against BLSB in maize. A few resistant sources have been identified and used for the identification of QTLs and candidate genes responsible for BLSB resistance. Here, we summarized the management practices, genetic resources, and different molecular approaches used to identify the QTLs and key candidate genes associated with BLSB resistance in maize.

Keywords: Maize · BLSB · *Rhizoctonia solani* · Resistance · Susceptible

Introduction

Maize (*Zea mays* L.), is one of the most important crop among cereals and it belongs to the family *Poaceae*. It is also known as the ‘Queen of Cereals’ due to its high yield potential (Choudhri, 2019). It hailed from Mexico and Central America. Worldwide production of maize in 2019-

20 was 1147.7 million metric tonnes (FAOSTAT, 2020). The nutritional importance of this crop in millions of people around the world is extensively recognized. It consists of roughly 72 per cent starch, 10 per cent protein, and 4 per cent fat (Ranum *et al.*, 2014). Each part of maize has its industrial importance, be it the grain or the leaves, stalk, tassel, or cob, all are useful for making various products (Kumar *et al.*, 2012; Singh *et al.*, 2021). It is also famous by the name corn that is largely adaptive all over the world and is cultivated in tropical, subtropical, and temperate regions. It is among the most flexible and emerging cash crop having immense adaptabilities under varied climatic conditions (Kaul *et al.*, 2017).

The reduction in the grain yield in maize is due to its susceptibility to various biotic and abiotic stresses. Losses due to the disease may vary over the year and location. There are more than 112 diseases that affect maize production throughout the world. Out of which, 65 diseases were reported in many places in India. Leaf spots, leaf blights, downy mildew, stalk rots, seed rots, and banded leaf and sheath blight are major diseases of maize crop (Saxena, 2002).

Among these, Banded leaf and sheath blight (BLSB) is one of the crucial diseases in maize also known as ear rot, banded sheath rot, sheath blight, banded sclerotial disease, and horizontal banded blight. The fungus responsible for causing BLSB is *Rhizoctonia solani*. It was reported for the first time in 1927 by Bertus in Sri Lanka and was called as ‘sclerotial disease’ (Sharma *et al.*, 2002). In India, the incidence of this disease was first reported by Ullstrup in the Tarai region of UP (Now Uttarakhand). Among the states, Himachal Pradesh, Uttar Pradesh, Haryana, Punjab, Madhya Pradesh, Rajasthan, West Bengal, Meghalaya, Bihar, and Jharkhand experience the regular occurrence of this disease (Chaudhary *et al.*, 2016).

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Economic losses

The disease was earlier reported as a minor disease on maize (Payak and Renfro, 1966). The importance of the disease was only realized in the early 1970s when an epidemic occurred in warm and humid foothill areas in the Mandi district of Himachal Pradesh (Sharad Shroff, 2016). Lal *et al.* (1980) have estimated a loss in grain yield in ten cultivars ranging from 23.9 to 31.9 per cent, whereas Singh and Sharma (1976) estimated 10–40 per cent in other cultivars. Lal *et al.* (1985) had suggested that grain yield loss can go up to an extent of 90 per cent.

The BLSB disease results in premature death, stalk breakage, and ear rot. About 11–40 per cent losses were reported in 10 different varieties of maize and losses in grain yield were reported up to 97 per cent (Butchaih, 1977) while in another study a yield loss of up to 100 per cent was reported (Sudjono, 1995). In the USA, yearly disease loss estimates for corn production ranges from 2–15 per cent. The expected average economic loss due to corn diseases in the USA, Ontario and Canada, was the US \$76.51 per acre from 2012 to 2015, with the total corn production being nearly 54 billion bushels. In Asia, loss estimates were around 12 per cent. Depending on the severity, grain yield loss varies between 11 and 40 per cent though, it can increase up to 100 per cent when the ear rot phase predominates and where the conditions are favourable for the pathogen. This alarming situation is further worsened by the lack of adequate level of resistance in maize germplasm.

Pathogen

The pathogen is soilborne as it survives in the soil and on diseased crop debris in the form of sclerotia or mycelium (Figure 1). Sclerotia can survive for several years in the soil. The fungi proliferate by irrigation and by the motion of affected soil and debris. The disease starts from the first and second leaf sheath, moves towards the ears, and leads to ear rot. Near the ear, the mycelium of light brown colour and small round black coloured sclerotia were perceived (Singh and Shahi, 2012).

Disease cycle and climatic conditions

The pathogen requires optimum temperature and humidity to grow and spread from diseased plant to healthy plant

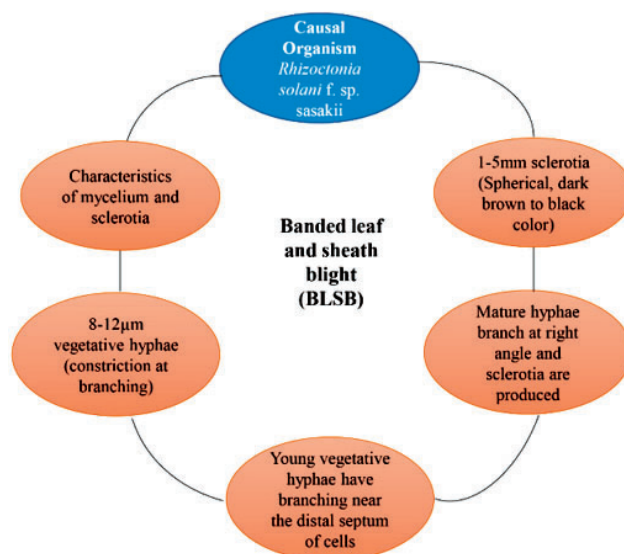


Figure 1. Characteristics of BLSB Disease

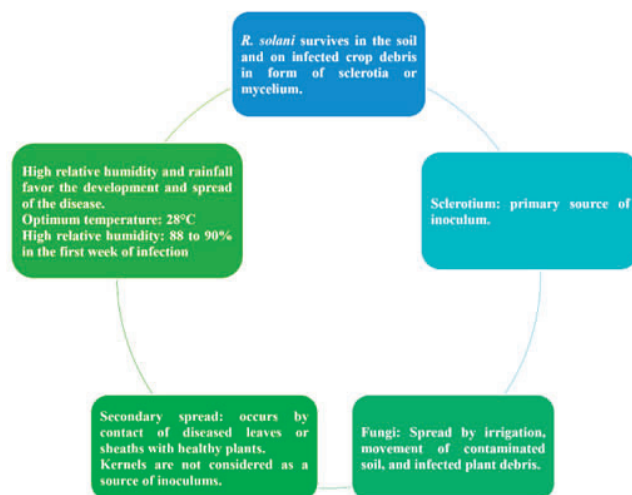


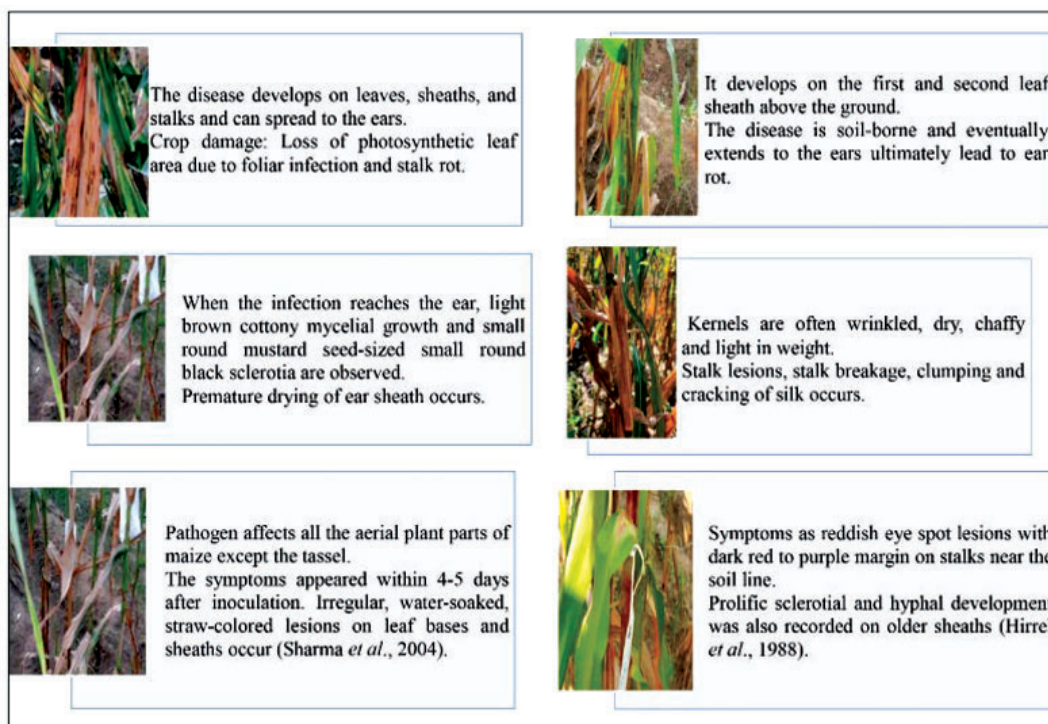
Figure 2. Disease cycle and climatic conditions

by a different mode of migration as explained in Figure 2. The favourable temperature for this disease to occur is 28°C and it also requires a relatively high humidity of about 88–90 per cent (Sharad Shroff, 2016). Grain yield losses can reach up to 100 per cent if the ear rot phase predominates. The pathogen propagates further when favourable conditions prevail (Madhavi *et al.*, 2011). The disease completely destroys plants as it affects photosynthetic rate initially and then yield at later stages. The symptoms are discussed in Figure 3.

Need to identify resistant sources

The development of resistant hybrids with the help of classical plant breeding is further worsened due to the

Figure 3. Symptoms of Banded leaf and sheath blight



limited availability of genetic resistance (Pan and Rush, 1997; Han *et al.*, 2002). The wild grass named teosinte (*Z. mays* spp. *Parviglumis*) is postulated as an ancestor of maize. Teosinte can also cross-breed with modern maize varieties and as a result, fertile maize-teosinte hybrids can be formed that can reproduce naturally (Singh *et al.*, 2017). It has been reported that wild species are very significant sources of genetic variability and these can be exploited in breeding programs. In teosinte, the genetic variability for different important agronomic traits for disease and insect resistance has been identified (Pasztor and Borsos, 1990; Srinivasan and Brewbaker, 1999). It is a valuable source of novel and diverse alleles and thus resistant to various fungal diseases. Various studies have shown that teosinte lines are resistant sources to *Ustilago maydis*, *Gray leaf spot*, and Southern leaf blight caused by *C. heterostrophus* (Chavan *et al.*, 2014; Lennon *et al.*, 2017). It consists of different loci resistant to northern corn leaf blight, corn leaf spot, *H. maydis*, corn smut diseases (Sahoo *et al.*, 2021), whereas different teosinte derived maize population has been screened and found to be resistant to *Fusarium*, downy mildew, and BLSB resistance (Maazou *et al.*, 2017; Adhikari., 2019). Teosinte accession has been screened at PAU, Ludhiana for BLSB by artificial inoculation method and it was reported that teosinte accessions were moderately susceptible to BLSB (Garg *et al.*, 2019).

Approaches

Different approaches have been used for the management of the disease like the use of toxins specific to fungi, validamycin, topsin, topsin-M, mancozeb, SAAF and carbendazim in different varying concentrations, using bioagents, *Trichoderma harzianum*, *T. viride*, *T. virens* and *Pseudomonas fluorescens* (Akhtar *et al.*, 2010).

Chemical control

Chemical control involves two fungicides, Rhizolex and Thiophenate-M. These fungicides proved effective in preventing damage to the crop and also the yield increase was almost equal (Sharma and Rai, 1999). Seed and soil treatment with carbendazim decreased the disease severity index but caused an elevated increase in plant height and weight of the plant (both fresh and dry weight). Moreover, the seeds treated with *P. fluorescens* gave better results over others in all the parameters (Rani *et al.*, 2013). Propiconazole was found to be effective when initial stages after planting but foliar sprays of Carbendazim was found to be ineffective against the disease (Saxena, 2002). Bavistin, Rhizolex, and Thiophenate M, these chemicals showed high control of BLSB under field conditions (Sharma *et al.*, 2002).

Mechanical controls

Mechanical controls by removing the two lower leaves with leaf sheath (Madhavi *et al.*, 2018). But to date, no method had been of much use to control this disease.

Biocontrol

Biocontrol for controlling the disease involves the treatment of maize seeds with peat-based *Pseudomonas fluorescens* formulation. PF-1 and PF-6 isolates showed a great effect in forbidding the effect of a pathogen in causing the disease. Foliar sprays for controlling the disease can also be used (Sivakumar *et al.*, 2000). *Bacillus subtilis* BNt8 as a biocontrol agent against the disease was used and other different fungi species like the *Glomus* sp, *Acaulospora mellea*, *Trichoderma* sp and *Gliocladium* sp can also be used to reduce the attack of the disease-causing fungi (Djaenuddin *et al.*, 2017). The use of water agar before inoculating it with any of the biocontrol moiety is important to receive an increased control over the pathogen. The leaf sheath inoculation method also helps to reduce the disease growing on the plant (Pascual *et al.*, 2000).

Conventional control measures, through cultural practices or by the use of fungicides, are not enough to control the disease and are not an economically viable option. The development of resistant variety has been slowed down due to the unavailability of disease-resistant sources.

Genetic resistance to BLSB

Association mapping strategy has been used for finding the genes that are responsible for causing disease and identified loci are also connected to drought tolerance (Lin *et al.*, 2013). About four QTLs for BLSB resistance have been located on chromosomes 6, 7 and 10, respectively (Pin *et al.*, 2009). Different studies have been done to identify resistance sources to the disease. Around 20 genotypes from the CIMMYT-Asian Regional Maize Program (CIMMYT-ARMP), seven CM (Coordinated Maize) lines which were developed by various public sector institutions in India, and two LM (Ludhiana Maize) lines were screened. Different sources of resistance were found in different states of India (Garg *et al.*, 2011). About 200 maize lines were screened for 10 of the major diseases

occurring in the plant to find out the resistance sources at 9 different regions of the country. A total of these 18 lines showed resistance to BLSB. These lines can now be used for the mapping of genes resistant to particular diseases in maize plants (Hooda *et al.*, 2012). About 11 QTLs were identified in $F_{2:4}$ and 8 QTLs in $F_{2:3}$ mapping population developed from nine inbred lines were identified as associated resistance to BLSB (Zhao *et al.*, 2006; Garg *et al.*, 2009). Various QTLs were identified for different morphological traits in the teosinte-derived maize population for BLSB resistance (Table 1) (Adhikari *et al.*, 2021). GWAS approach was used to identify a novel allele ZmFBL41 (F-box protein) which is involved in maize resistance to *R. solani* by regulating lignin coding enzyme (Li *et al.*, 2019).

Use of omics

Proteomic analysis reveals two biochemical markers β -1-3-glucanase and peroxidase for screening and resistance breeding against disease (Shamim *et al.*, 2020). RNAseq analysis was performed to study the transcriptional changes in the B73 maize line in response to *R. Solani* and it was concluded that about 388 DEG (Differentially Expressed Genes) were involved in response to *R. solani* and these were considered as core immune genes in maize (Cao *et al.*, 2021).

Future prospective

As many studies identified QTLs for BLSB resistance but still there is a lack and need to identify more loci for BLSB resistance such that resistant hybrids can be developed or identification make possible to transfer QTLs to susceptible lines through Marker Assisted Selection. Secondly, a pyramiding of QTLs can also be done to develop resistant lines. There is a need to collect more germplasm from CIMMYT and USDA to screen against BLSB. In addition, further research is required in the field of proteomics and transcriptomics as these will help to identify the regions conferring resistance to BLSB. More extensive work needed addressing the genetic inheritance will provide exciting novel insights. Refined genetic approaches and system analysis will be instrumental to get deeper insights into the mechanisms of BLSB resistance in maize.

Table 1. QTLs associated with different morphological traits for banded leaf and sheath blight resistance

Population Type	Trait	Single marker analysis/ QTLs/ Chromosome No.	Marker interval	Reference
F _{2:4} Elite inbreds R15 and 478	BLSB	<i>qBLSB-1</i> <i>qBLSB-2a</i> <i>qBLSB-2b</i> <i>qBLSB-2c</i> <i>qBLSB-3</i> <i>qBLSB-4</i> <i>qBLSB-5</i> <i>qBLSB-6a</i> <i>qBLSB-6b</i> <i>qBLSB-6c</i> <i>qBLSB-10</i>	umc1245-dupssr12 umc1250-bnlgl 1721 bnlg1662-bnlgl 1940 bnlg1036-umc2150 phi19322-umc1010 umc2281-umc1662 phi10918-umc2164 bnlg1600-umc1818 bnlg1006-umc1723 umc1859-bnlgl1759 mmc0501-phi054	Zhao <i>et al.</i> (2006)
Teosinte and inbred line DI-103BC ₁ F ₅	Ear length, kernel rows per ear, & kernels per row Test weight, Ears per plant Plant height	phi10918-linked QTL umc1500-linked QTL umc2000-linked QTL Phi420701 QTL	- - - -	Adhikari <i>et al.</i> (2021)
F _{2:3} developed from 9 inbred lines (CA00106, CA00310, CM104, CM105, CM300, CM139, CM140, LM5 and LM6)	BLSB resistance	Chromosome 4 Chromosome 8 Chromosome 9 Chromosome 2 Chromosome 3 Chromosome 6 Chromosome 7 Chromosome 10	(bnlg252-bnlgl1621) (umc2146-umc1172) (phi108411-umc2346) (umc2363-umc1622) (umc2101-umc1892) (umc1127) (umc1066-bnlgl1792) (bnlg1518-bnlgl1526)	Garg <i>et al.</i> (2008)
CML429 × DM9 F _{2:3}	BLSB resistance, Disease incidence	(2 QTLs) on Chromosome 6 (2 QTLs) on Chromosomes 7&10	bnlg107 and umc1796 bnlg1161 and phi059	Chen <i>et al.</i> (2009)
R 15 (resistant) × Ye 478 (susceptible) w F _{2:4}	BLSB resistance	BLSB2-1 BLSB2-2 BLSB2-3 BLSB2-4 BLSB3-2 BLSB6-1 BLSB6-2 BLSB9-1 BLSB7-1 BLSB3-1 BLSB7-2 BLSB3-2 BLSB4-1 BLSB4-2 BLSB4-3 BLSB10-1 BLSB6-3 BLSB10-1 BLSB10-2	umc1285-nc003 umc2150-bnlgl172 bnlg1662-bnlgl1606 bnlg1606-bnlgl1940 umc1659-umc1052 bnlg1538-umc181 umc1818-umc1083 umc1231-umc2343 bnlg2132-umc1016 bnlg1523-bnlgl1447 umc1125-umc1154 umc1659-umc1052 bnlg2162-umc1051 umc2281-umc1662 bnlg1621-umc1299 phi118-umc1319 umc1723-umc1014 phi118-umc1319 mmc0501-phi054	Lin <i>et al.</i> (2008)

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