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Banded leaf and sheath blight: A menacing disease of maize (*Zea mays* L.) and its management

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Abstract: Maize (*Zea mays* L.) crop is attacked by number of fungal, bacterial and viral diseases, out of which banded leaf and sheath blight (BLSB) caused by anastomosis group 1-IA of *Rhizoctonia solani* f. sp. *sasakii* Exner. is one of the most widespread and destructive disease of maize in Southeast Asian countries. The occurrence of this disease has also been reported from other parts of the world, which causes significant yield loss up to 100%. *R. solani* can survive in the soil for several years and able to infect plants belonging to more than 32 families, including many economically important monocots and dicots plants. The severity of the disease favoured by humid weather with temperature around 28°C, poses challenge to maize growers due to its soil borne nature and lack of resistance cultivars. It is indicated that none of the disease management approaches are effective against BLSB. Banded leaf and sheath blight is difficult to control through either fungicide or crop rotation alone. A number of quantitative trait loci (QTLs) controlling BLSB have been identified that would help the development of maize hybrids resistance to this disease. Management of BLSB requires an integrated approach based on the knowledge of each stage of the disease and molecular aspect of maize defence responses against *R. solani*. Mention conclusion statement and novelty of the work. The present review summarizes consolidated information on distribution, yield loss, symptoms, pathogen life cycle, epidemiology, genetic structure of the pathogen population, molecular aspect of pathogenicity and its integrated management through cultural, biological, chemical and genetic means. The consolidated knowledge presented in this review should help better disease management and reduce crop yield loss due to banded leaf and sheath blight pathogen.

Keywords: Banded leaf and sheath blight, Biological control, Genetic variability, *Rhizoctonia solani* f. sp. *sasakii*, *Zea mays*

INTRODUCTION

Maize (*Zea mays* L.) is one of the most important cereal crops in the world agricultural economy as food, feed and industrial products. As compare to rice and wheat, maize contains approximately 72% starch, 10% protein, and 4% fat, supplying an energy density of 365 Kcal/ 100 gm (Nuss and Tanumihardjo, 2010). Being a C_4 cereal crop, it is cultivated widely throughout the world and has the highest production among all the cereals. It is estimated that in 2014, the total world production of maize was 1021.6 million tons (FAO, 2015), with the United States, China, and Brazil harvesting 35%, 24%, and 8% of the total production of maize respectively. India ranked 6th with the total maize production 23.7 million tons and share 2.3% of the total worldwide maize production. In India, maize is the third most important cereal crop after rice and wheat, grown in a wide range of environments extending from extreme semi-arid to sub-humid and humid regions. Traditional maize growing areas, includes Bihar, Madhya Pradesh, Rajasthan, Uttar Pradesh and Gujarat, whereas, non-traditional maize areas are Karnataka and Andhra Pradesh (Joshi *et al.*, 2005).

Despite very high yield potential of maize, one of the major deterrents to high grain yield is its sensitivity to several diseases. From different parts of the world, about 112 diseases of maize have been reported, of these, 65 are known to occur in India (Saxena, 2002). Seed rot and seedling blight, leaf spots and blights, downy mildews, stalk rots, banded leaf and sheath blight, and smut and rots are the most important diseases of maize crop (Hafiz, 1986). Among different fungal diseases affecting maize production, banded leaf and sheath blight (BLSB) induced by *Rhizoctonia solani* f. sp. *sasakii* causes significant gain yield loss from 11% to 40%, even to 100% on some cultivars in some warm and humid regions, where the conditions are favourable for the pathogen (Madhavi *et al.*, 2011, Izhar and Chakraborty 2013; Gao *et al.*, 2014).

The pathogen: The causal agent of banded leaf and sheath blight (BLSB) is *Thanatephorus sasakii* (Shirai) Tu and Kimbrough (St. Imp. *Rhizoctoniz solani* Kühn

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f. sp. *sasakii* Exner). This is one of the most wide spread, destructive and versatile pathogen found in most parts of the world and infecting a vast range of host plants, including maize causing seed decay, damping –off, stem canker, root rot, aerial blight, and seed or cob decay (Ogoshi, 1987). Previously, the causal agent of maize sheath blight was thought to be the anastomosis group (AG) 1 of multinucleate *Rhizoctonia solani* Kühn (teleomorph: *Thanatephorus cucmeris* (A. B. Frank) Donk), which is a soil- born fungal pathogen with a wide host range. Recently, however, some binucleate isolates belonging to AG-Ba and AG-A of group (AG) (Xia *et al.*, 2008; Zhou *et al.*, 2012) and one uninucleate isolate (Zhou *et al.*, 2015) of *Rhizoctonia* spp. have also been identified as pathogens of maize sheath blight. Caesar *et al.* (2010), Fang *et al*. (2013) and Zhou *et al.* (2016) observed the variation in virulence of pathogenic *Rhizoctonia* spp. and found that there was significant difference in virulence among the pathogenic *Rhizoctonia* isolates, with multinucleate isolates the greatest, binucleate isolates moderate and uninucleate *Rhizoctonia* isolate the lowest.

Distribution : This disease was first reported from Sri Lanka in 1927 as Sclerotial disease (Bertus, 1927), and subsequently recorded from Malaysia, under the name of 'banded sheath rot', in the Philippines as 'banded sclerotial disease' and as 'summer sheath blight' in Japan (Wiltshire, 1956). This disease has also been reported in Germany, USA, Nigeria, Venezuela, Sierra Leone, Ivory Coast and England. BLSB is recognized as a serious impediment to maize production in China, South Asia and Southeast Asia (India, Sri Lanka, Indonesia, Cambodia, Bangladesh, Pakistan, Nepal, Myanmar, Japan, Malaysia, Thailand, Laos, Vietnam, Taiwan, and Korea). Surprisingly, in China, yield losses close to 100% have been attributed to BLSB (Singh and Shahi, 2012). In India, the disease has been reported from states of Himachal Pradesh, Uttar Pradesh, Haryana, Punjab, Madhya Pradesh, Rajasthan, West Bengal, Meghalaya, Assam and Orissa (Rani *et al.*, 2013).

Taxonomy Classification: The genus concept *Rhizoctonia* was first described by De Candolle in 1815 (Sneh *et al.*, 1991). *Rhizoctonia solani* is a genetically diverse group of fungi with more than 100 species (Anderson, 1982; Adam, 1988; Binder *et al.*, 2005), that attack all known crops. A method based on anastomosis groups (AGs) has been used for its identification and classification (Parmeter *et al.*, 1969; Ogoshi, 1987). Among the 14 AGs of multinucleate *Rhizoctonia*, AG1 comprises many plant-pathogenic isolates recovered from a range of hosts. AG1 isolates have been divided into three subgroups based on host, symptoms and cultural characteristics: AG1-IA (sheath blight); AG1-IB (web blight); and AG1-IC (damping-off). However, the host range of AG1-IA and AG1-IB overlap (Ogoshi, 1987; Sneh *et al.*, 1991; Liu and Sinclair, 1993a). Although isolates of AG1 (includes *Rhizoctonia solani* causing BLSB) have been reported worldwide (Ogoshi, 1987).

Cultural Characteristics: The young colonies produced by the fungus were fast growing and formed silky white colonies on Potato Dextrose Agar (PDA) medium; growth optimum at 25 and 30°C; which gradually lost their lustre and became dull. Mycelium often is colourless at young stage, while turns to light brown as it matures (Ahuja and Payak, 1985; Sivakumar *et al.*, 2000). Microscopic studies of hyphae revealed, it as multinucleate; branching near distal septum of cells in young vegetative hyphae; formation of septum in the branch near the point of origin; construction of branch; dolipore septum; no clamp connection; no conidium; sclerotium not differentiated in rind and medulla and no rhizomorph (Ogoshi, 1975; Ahuja and Payak, 1988). Sclerotia of *R. solani* were produced abundantly in culture, typically 1-5 µm diameter spherical and dark brown to black in colour (Akhtar *et al.*, 2009).

Symptoms, disease development and epidemiology: The pathogen affects all aerial parts of the maize plant except tassel. The disease manifests itself on leaf, leaf sheaths, stalks and ears as leaf blight, stalk lesion or rind spotting and stalk breakage etc. It was reported that BLSB disease appears at pre-flowering stage on 30 to 40 day-old maize plants, but infection can also occur on young plants which may subsequently result in severe blighting and death of apical region of growing plant (Saxena, 2002). The disease symptoms on leaves as irregularly globular to elongated lesions which appears as water-soaked areas. The affected areas appear bleached, soon they become straw colored and necrotic (Ahuja and Payak, 1982). The lesions enlarge rapidly resulting in discoloured areas alternating with dark bands, apparent on lower leaves after 7-8 days (Rani *et al.*, 2013).

The symptoms are more common on sheaths than leaves. A short of wave pattern of disease advancement can be seen not only on leaves but also on sheaths and husk leaves. The disease manifests itself on leaf, leaf sheath, stalks and ears as leaf and sheath blight, stalk lesions or rind spotting and stalks breakage, clumping and cracking of styles and horseshoe shaped lesions with banding of caryopses resulting in ear rots (Knight and Bunil, 1964; Sharma, 1999). In early stages marginal chlorosis and rooting of laminae proceed inwardly, later as the infection becomes older numerous sclerotial bodies are also seen (Saxena, 1997). Buddemeyer *et al.* (2004), observed that *R. solani* caused round to elliptical, yellow to tan or black lesions on seminal crown and brace roots of maize cultivars. Depending on disease severity, crown roots of maize plants were completely rotten and affected plants lodged. Typical BLSB symptoms were observed as small purplish brown lesion or greenish olive brown large continuous patches on leaf sheath and pale olive brown lesions on stalk as well as rotting of ears

(Akhtar *et al.*, 2009).

The fungus is capable of infecting maize plants in all the stages of crop growth right from seedling to maturity. *R. solani* survive in the soil and on infected crop debris as sclerotia or mycelium. Sclerotium serves as primary inoculum. The fungi spread by irrigation, movement of contaminated soil and infected plant debris. At the onset of growing season, in response to favourable humidity and temperature (15 to 35°C), the fungal growth is attracted to rapidly growing seedlings and water soaked seed coats by chemotropic stimulants released by growing plant cells and decomposing plant residue. Secondary spread of this disease occurs by contact of diseased leaves or sheath with healthy plants. High relative humidity (90%), an optimum temperature about 28°C, and rain fall in the first week of infection significantly favours the development and spread of disease. Disease development and spread becomes slow, if the relative humidity goes below 70% (Sharma, 2005). Crop damage is caused by loss of photosynthetic leaf area due to foliar infection and stalk rot which lead to crop lodging (Lu *et al*., 2012). Sheath blight reduced the breaking resistance of lower internodes and consequently resulted in poor lodging resistance (Wu *et al.*, 2012). The maximum damage is caused when ears of maize are infected (Ahuja and Payak, 1982).

Economic Importance: The disease causes a considerable reduction of high yielding maize varieties, resulting in premature death, stalk breakage and ear rot. In India, Singh and Sharma (1976), have estimated 40.5% reduction in maize grain yield with 71% of BLSB disease index, whereas Lal *et al.* (1980) estimated loss in grain yield ranging from 23.9 to 31.9% at disease score levels ranging from 3.0 to 5.0 in ten cultivars. Lal *et al.* (1985), suggested that due to BLSB maize grain yield loss vary to the extent of over 90%. In Guangxi province in South China, maize yield losses of 87.5 and 57.8% have been determined under natural conditions in the hybrids Luya 13 and Guiding planted at Bao Qiao and Chen Xiang countries (Sharma, 2005). Summer and Minton (1989), planted maize in infested and non-infested soils with high and low inoculum levels, estimated 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 in USA. Tang *et al.* (2004), reported that BLSB caused 0-60% loss in maize grain yield under natural conditions. However, the magnitude of grain loss may reach 100%, if the ear rot phase of the disease predominated (Huang *et al.*, 2007).

Host Range: The pathogen has wide host range and infects plants belonging to over 32 families in 188 genera. Isolates of *R. solani* causing BLSB disease in maize infected members of Gramineae (*Cynodon dactylon, Oryza sativa, Saccharum officinarum* and *Sorghum bicolor*), Leguminosae (*Arachis hypogaea, Glycine max, Pisum sativam* and *Vigna radiate*) and Solanaceae (*Lycopersicum esculentum* and *Solanum tuberosum*) (Baruah and Lal, 1981). In artificial inoculations it infects a number of crop plants belonging to families Poaceae, Papilionaceae and Solanaceae: *Paspalum serobiculatum, Pennisetum americanum, P. purpureum, Setaria italic, Panicum miliaceum, Coix lachrymal-jobi, Echinochloa frumentacea, Zea mays, Zea Mexicana, O.sativa, S. officinarun, S. bicolor*), (Ahuja and Payak, 1988; Trivedi and Rathore, 2006).

Maize has also been infected by strain of *R. solani* from rice, sugarcane, arrow root and some grasses (Ahuja and Payak, 1985). Rice and maize isolates are, however, indistinguishable on the basis of cross inoculation tests, host range, virulence, number of nuclei per hyphal cell, and other morphological characters including pathogenicity. Comparison studies on cultural and morphological characteristics of *R. solani* isolates from rice, maize, sugarcane, and sorghum revealed that maize and rice are similar than those isolates of sugarcane and sorghum (Saxena, 1997).

Mechanism involved in penetration of host tissue: *R. solani* can infect underlying tissues either through mechanical penetration by means of force or through utilizing natural openings and wounds (Parmeter, 1970; Back *et al.*, 2002). The infection starts when mycelia or hypae of the fungus starts to grow towards a suitable host as a result of attracting chemical exudates, e.g., amino acids, sugars, organic acids and phenols, from the plants (Keijer, 1996a). After the first contact, loose and still unattached hypha starts to grow over the plant and within a few hours the hypha flattens and directional growth over the epidermal cells is initiated, forming T-shaped hyphal branches that can give rise to hyphal aggregates known as infection cushions (Keijer, 1996a; Dodman and Flintje, 1970). Fine infection pegs develop from the infection cushion, enable the fungus to minimize the force needed for penetration. However, also the production of lobate appressoria was observed on rice (Marshall and Rush, 1980). After a peg has penetrated, it continues to grow between the cuticle and the epidermal wall. Finally the cuticle and epidermal wall are penetrated, and the infectious organs may extend growth into the cell lumen (Demirci and Döken, 1998). Penetration is established by using hydrostatic pressure, even though degrading enzymes such as cutinases (Baker and Bateman, 1978), pectinases (Bertagnolli *et al.*, 1996; Jayasinghe *et al.*, 2004) and xylanases (Peltonen, 1995), are most probably also involved in infection and penetration. The production of endopectinlyase has been reported to be associated with the tissue degradation in later stage of infection (Gonzãlez-Garcia *et al.*, 2006). Necrotic lesions on epidermal tissue of shoots, roots and stolons or as damping-off of the young seedlings can be seen, when fungus starts to grow inside the host and degrading the tissue (Demirci and Döken, 1998).

The fungus may also utilize natural openings viz. sto-

mata on stems, cotyledons and leaves or lenticels as entry portals to plant tissue. Wounds can also be used as entry portals, but penetration usually does not occur solely via wounds. The growing hypha first spread and fill out the wound with densely packed hyphae before penetrating into healthy tissues without the formation of infection structure (Parmeter, 1970; Back *et al*., 2002). Plant defence mechanisms may stop the fungal infection at the following establishment stages of *R. solani:* a) attachment of hypha to plant surface, b) formation of infection structure, c) penetration of infection pegs, d) continue invasion of penetration hyphae can be stopped by hypersensitive reaction (Parmeter, 1970; Demirci and Döken, 1998).

Genetic structure of the *R. solani* population: Genetic diversity in *R. solani* AGI-IA population is important for understanding its ecology, pathology, and host specificity. Therefore, by accessing the genetic variability within and among various populations of this phytopathogenic fungus will be useful in disease management. Isozyme and DNA analysis have advanced our understanding of the structure of *R. solani* populations. These molecular tools have easily and distinctly grouped *R. solani* into subgroups of an AG. Isolates of AG1-IA have been subject of different diversity and population studies in which variation has been measured using intra and extracellular enzymes and proteins (Liu and Sinclair, 1993b; Matsuyama *et al.,* 1978; Neeraja *et al.,* 2002a), and various fatty acids (Stevens Johnk and Jones, 1994), as well as various molecular techniques such as restriction fragment length polymorphism (RFLP) (Banniza *et al.,* 1999; Rosewich et al., 1999), amplified fragment length polymorphism (AFLP) (Fiers *et al.,* 2011; Taheri *et al.,* 2007), repetitive element PCR (Rep-PCR) (Linde *et al.,* 2005), simple sequence repeat polymerase chain reaction (SSR-PCR) or microsatellites (Banniza and Rutherford, 2001; Bernardes-De-Assis *et al.,* 2009; Gonzalez-Vera *et al.,* 2010), inter simple sequence repeats (ISSR) (Khodayari et al., 2009), analysis of sequence variation in ribosomal DNA (rDNA) (Fenille et al., 2003; Wang *et al.,* 2015) and random amplified polymorphic DNA (RAPD) markers (Neeraja et al., 2002 b; Gad *et al.,* 2013; Susheela and Reddy, 2013; Chikara *et al.,* 2015). In a population genetic diversity study of *R. solani* from India that was based RFLP and Rep-PCR, results were consistent with small genetic distances among populations and high levels of gene flow (Linde *et al.,* 2005).

Despite these studies, genetic variability within populations, particularly among isolates of different ISGs of *R. solani* AG1 infecting maize is poorly known. In particular, pathogen populations should be monitored to determine if new genotypes have been introduced into a region. However, understanding of disease epidemiology, host-pathogen interaction, and subsequently successful management of sheath blight disease is really dependent on our knowledge concerning variability of the pathogen populations and the factors affecting genetic structure of these populations.

Molecular aspect of pathogenicity: Currently, molecular aspects of *R. solani* pathogenicity involved in maize leaf sheaths infected by BLSB are poorly known. The lack of molecular information on pathogenicity can be related to the relatively large genome size of the pathogen (Cubeta *et al.,* 2009). *R. solani* isolates have at least 11 chromosomes ranging in size from 0.6 to 6 Mb (Keijer *et al*., 1996b). At present, the genome sequences of AG-IA (Zheng et al., 2013), AG1-IB (Wibberg *et al.,* 2013), AG3 (Cubeta *et al*., 2014), and AG8 (Hane *et al.,* 2014) are available. The genome sizes ranges from 36.9 Mb (AG1-IA, 10,489 gene models), 39.8 Mb (AG8, 13,964 gene models), 47.6 Mb (AG1-IB, 12,422 gene models) to 51.0 Mb (AG3, 12,726 gene models). The resulting databases will allow the comprehensive analysis of developmental processes that are characteristic of this fungus, including the molecular nature of pathogenicity. DNA databases support analysis of the fungal transcriptome, proteome, and metabolome.

Fungi inevitably respond to extracellular signals or stimuli via a wide array of transduction pathways for pathogenicity. One of the most studied pathways in the filamentous fungi is the signalling cascade mediated by membrane-bound heterotrimeric G proteins, composed of Gα from Gβ and Gγ subunits (Li *et al.,* 2007; Wendland, 2001). The Gα subunit containing intrinsic GTPase activity is the key step in controlling the cellular response via the G protein signal transduction pathway. Upon receiving extracellular stimuli, a G protein-coupled receptor (GPCR) interacts with the G protein, inducing replacement of GDP in the Gα subunit by GTP which leads to dissociation of Gα from Gβ and Gγ subunits. The released Gα subunit becomes activated and in turn cyclase, phospholipase, ion transporters, and mitogen activated protein kinase (MAPK) involved in numerous biological processes like regulation of hyphal morphogenesis, infection structure formation, sclerotium formation, regulation of mating, sporulation and spore germination including pathogenicity (Neves *et al.,* 2002). Charoensopharat et al. (2008), demonstrated the function of the Gα subunit gene, *Rga1*, in the rice sheath blight pathogen by target gene disruption and found that disruption of *Rga1* led to decreased vegetative growth and pathogenicity of the sheath blight pathogen *R. solani*. The *Rga1* disruptant showed altered colony morphology, also the sclerotia formation ability of the disruptant was completely lost. Similar results have been observed for the genes encoding G protein subunits in other phytopathogenic fungi, such as *gpa3* in *Ustilago maydis* (Regenfelder *et al.,* 1997), *cpg1* in *Cryphonectria parasitica* (Gao and Nuss, 1996), and *fga1* in *Fusarium oxysporum* (Jain *et al.,* 2002).

Zheng *et al.* (2013), analysed the genome of *R. solani*

AG1 IA isolate and predicted the likely genetic requirements for the necrotrophic phytopathogen to invade and colonize the rice plant. They concluded that necrotrophy does not require a large number of carbohydrate active enzymes (CAZymes) and secondary metabolites during infection, at least for *R. solani* AG1 IA, which mainly utilizes key pathogenic glycoside hydrolase (GHs) and genes. The novel divergent elements, such as Gα proteins, GPCRs in MAPK signalling pathway, are dedicated to the exclusive parasitic lifestyle and regulate nutrition, reproduction and pathogenicity in the signal transduction pathway. Therefore, they hypothesized that *R. solani* AG1 IA pathogenesis includes key GHs, secondary metabolites and diverse effectors to supress the host defence at the early infection stage. HR and the plant defence can then be activated, which is followed by the progressive expression of specific genes encoding degradation-associated enzymes to damage the rice plant.

Genetics of resistance to BLSB: To date, 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 level of resistance to this disease, under highly epiphytotic conditions. This may be attributed to inadequate knowledge about mode of inheritance of resistance, genotype × environment interactions for resistance and possible presence of different races. Vimla *et al* (1988), used combining ability analysis for resistance to BLSB and concluded that both general and specific combining abilities varied significantly for controlled disease resistance but general combining ability variance was predominant. They also identified inbred line CM104 as the most promising combiner for resistance. Kumar and Singh (2002), 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 line, one cross each was made between resistance × resistance and susceptible × susceptible lines. The BLSB reaction in F2 and backcrosses involving CM104 and susceptible line suggested that resistance in CM 104 was controlled by Duplicate dominant genes while crosses of CML1 showed dominance and recessive interaction.

Recently, genetic and molecular studied on the BLSB and pathogens have been reported in maize (Li *et al*., 2009; Liu *et al.,* 2011; Zhang *et al.,* 2012). These studies revealed that resistance to BLSB is a typical quantitative trait controlled by polygenes and three significant quantitative trait loci (QTL) located on chromosome 2, 6, and 10 to be responsible for resistance to BLSB respectively (Campbell *et al.,* 2002; Chen *et al*., 2000; Zhang *et al.,* 2006). The identification of QTL for resistance to BLSB is considered as an effective tool in development of disease resistant maize hybrid. The information generated from mapping resistance genes can be used in marker assisted selection (MAS) programmes for development of BLSB resistant lines (Singh and Shahi, 2012). In an experiment conducted by Asea *et al.* (2012), results indicate that molecular markers linked to target rQTL can facilitate pyramiding resistance to multiple diseases during early generation of pedigree selection. Zhao *et al* (2006), screened a mapping population consisting of 229 F2 individuals, derived by crossing inbreds R15 (resistance) with 478 (susceptible), against *R. solani* 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. But only four QTLs located at chromosomes 2, 6, and 10 were identified across both locations. Lin *et al*. (2008) analysed digenic epistatic and QTL × environment interactions for resistance to BLSB and detected 17 QTLs including 12 pairs of digenic epistatc 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**.** In India, a F2:3 mapping population was generated using CA00106 (resistant) and CM140 (susceptible) at three geographical locations. This study led to identification of three QTLs on chromosome 6, 8, and 9 with significant epistatic interactions (Garg *et al.,* 2009). It is important to intensify efforts to identify stable and additional sources of resistance to BLSB and improve the disease resistance of present maize hybrids.

***Zea mays- Rhizoctonia solani* interaction:** *R. solani* belongs to a necrotrohic species complex. AG1-IA is one of the largest groups causing the most damages among all other AG groups. Little is known about the pathogenicity and virulence factors of *R. solani*. Maize pathogens have plenty of pathogenicity genes that are required for infection or for enhancing host virulence. The pathogenic capability of an organism is determined by its virulence factors. A specific interaction was governed in plant pathogen interaction that is the pathogen *avr* (avirulence) gene correspond with the resistance *R*-genes of the host plant. When corresponding *R* and *avr* genes are present in both host and pathogen, the result is disease resistance, if either is inactive or absent, disease results (Flor, 1971; Dangl and Jones 2001).

Like other plant species, *Zea mays* employs a diverse array of defence mechanism that minimizes infection during interaction with pathogen. Besides pre-existing physical and chemical barriers, a variety of defence mechanisms are activated upon pathogen attack (Huang *et al.,* 2008). During the past decades, great efforts have been devoted to understand the molecular mechanism of the plants infected by *R. solani*, such as *Oryza sativa* L. and *Zea mays* L. (Liu *et al.,* 2009; Zhang *et al.,* 2010). There are many catalytic enzymes involved in the *R. solani* infective response, including

chitinase, glucanase, and phenylanine ammonia lyase (Anuratha *et al.,* 1996; Jedidah *et al.,* 2000; Liu *et al*., 2009). Furthermore, a few pathogenesis-associated genes transiently exist in maize and resisted the pathogen (Alexander *et al.,* 1993; Datta *et al.,* 1999; Agrawal *et al*., 2001; Zhu *et al.,* 2006). Biochemical changes in many plant-pathogen interactions are accompanied by the rapid increase in phenolic compounds and related enzymes, often termed the hypersensitive response (Mondal *et al.,* 2012). Such changes can be attributed to a variety of mechanisms of defense as exhibited by the host during pathogenesis (Jayaraj *et al.,* 2010). Zhang *et al.,* (2012), identified genes which are differentially expressed in maize during interaction with *R. solani* and found that 15 genes were up-regulated or down-regulated in response to R. solani infection. These genes mainly regulates transcription, protein processing, metabolism, defense, disease response and other functions. Recently, Dahima et al. (2014), estimated total phenol content, peroxidase and polyphenol oxidase content in maize germplasm affected by BLSB and concluded that higher phenol, peroxidase and polyphenol oxidase activities plays a vital role in inducing resistance against BLSB. Gao *et al.* (2014) conducted genome-wide gene expression profiling using Solexa sequencing, to gain insight into the transcriptome dynamics that are associated with BLSB resistance. The most differentially expressed tags were analyzed, representing, 1,476 up-regulated and 1,754 down-regulated genes, except for unknown transcripts, which were classified into 11 functional categories. The most enriched categories were those of metabolism, signal transduction and cellular transport.

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 chemical and biological procedure is extremely important to minimize the destruction of crop and to prevent yield losses (Singh and Shahi, 2012).

Cultural practices: Cultural practices like stripping of the second and third leaf sheaths from the ground level at the age of 35-40 days old maize crop is effective in checking further BLSB development (Mehra *et al.,* 2012). Inter-cropping system of maize with legumes especially with soybean effectively reduced the severity of the pathogen in soil (Kato and Incue, 1995). Maintaining the proper population level and application of cattle compost (FYM) prior to planting, helped in decrease of disease level and its subsequent spread in field (Sharma and Hembram, 1990). Selection of a well-drained field and planting on raised beds are important cultural aspects to avoid contact of excess water with seeds and faster growth of seedlings (Hooda *et al.,* 2015).

Chemical control: Many attempts have been made to control BLSB of maize through fungicides under *in vitro* and field condition. Different fungicides viz. Carbendazim, Benodanil, Thiobendazole, Validamycin, Topsin M, Rhizolex, Propiconazole etc. have been tested and found to be effective in inhibiting growth of the BLSB pathogen under *in vitro* condition. All these fungicides except thiobendazole were effective in reducing BLSB disease severity, also under field conditions (Ahuja and Payak, 1986; Sharma and Rai, 1999). Saxena (2002), tested efficacy of chemicals (viz, Propiconazole, 0.1%, and Carbendazim, 0.05%), by applying as foliar sprays, alone or in combinations. Foliar sprays of Carbendazim showed the ineffectiveness against BLSB. On *in-vitro* evaluation, three often used fungicides, namely Bavistin, Rhizolex, and Thiophenate Methyl, have shown absolute control of *R. solani* mycelial growth with 100% inhibition (Sharma *et al.,* 2002). Meena *et al.* (2003a), evaluated Carbendazim, kitazin and bulb extract of garlic (*Allium sativum*) @ 5 % (w/v) against BLSB, these fungicides and plant extract completely inhibited the mycelial growth of BLSB pathogen at 1 ppm concentration. Rakesh *et al.,* (2011), tested seed dressing fungicides (Bavistin 50WP @ 2.5 g/kg of seed, Vitavax Power 35.5 % + Thiram 37.5 % @ 2.5 g/kg of seed and Thiram 50 WP @ 2.5 g/kg of seed) against BLSB pathogen. These fungicides has been found effective for the management of BLSB. Bavistin was found highly effective with 48.7% disease control and highest maize yield of 64.7 q/ha over control.

Biological control: Several micro-organisms have been reported to parasitize *Rhizoctonia* species. These are mainly fungus of species *Trichoderma, Gliocladium,* and *Laetisaria,* bacteria (*Pseudomonas* sp., *Bacillus subtilis*), and nematodes (*Aphelenchus avenae*). Application of *Pseudomonas fluorescens* reduces disease incidence in field conditions besides improving plant growth. The biocontrol agent showed production of volatile ammonia and Hydrocyanic acid (HCN) under *in vitro* conditions (Sivakumar *et al.,* 2000). Muis and Quimio (2006), developed a seed treatment formulation of the selected *Bacillus subtilis* to control *R. solani* in corn. Seed treatment with *B. subtilis* BR23 formulation supressed *R. solani* in microplots and increased grain yield by 27% compared to that of the control capton with 14.4 per cent. Madhavi et al. (2011), used *Pseudomonas flurorescens* aganist *R. solani* caused BLSB of maize under *in-vitro* condition. The results showed that Pseudomonads have significantly inhibited the mycelial growth and sclerotial germination of *R. solani* ranging from 48%-92% and 29% -87% respectively over check. *Trichoderma* sp. found to be an effective biocontol agent, 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). Volatile compounds released by *T. harzianum* supress both growth and sclerotial formation of *R. solani*, inhibited 80% and 34% respectively followed by *T. viride* which inhibited 70%

growth and 26% sclerotial formation (Meena *et al.*, 2003b). *Trichoderma* hyphae coiled around *R. solani* hyphae and subsequently caused the cell wall lysis (Yobo *et al.*, 2004). Khan and Sinha (2007), reported *T. harzianum* and its volatile compound inhibited *R. solani* followed by *T. viride* in dual culture techniques. Sahar *et al.* (2009), also reported that *T. hamatum* showed highest reduction in the growth of *R. solani* followed by *B. subtilis*, while it was less effective against *M. phaseolina* and *F. solani*.

Integrated disease management: Integrated disease management (IDM) which covers physical, cultural, chemical, biological and resistance hybrids/varieties, are required for the control of BLSB. This strategy emphasizes prudent use of chemicals in combination with other management practices for maximizing yield with minimum environmental hazards. Dalmacio et al. (1990), conducted three experiments on the mechanical, chemical and biological control of BLSB. In case of mechanical control, the de-leafing of basal portion of maize plants proved to be effective in controlling the upward spread of lesion. Among the chemicals and biological agents, Validamycin gave the best control followed by *T. harzianum.* Akhtar *et al.* (2010), concluded that management of BLSB can be achieved by integrating soil application of *T. harzianum* precolonized farmyard manure (FYM) with foliar application of carbendazim. In an IDM approach, Singh and Singh (2011) found best performance of Validamycin (0.25%) and *T. viride* as foliar spray than the fungicides like Tilt (0.15 %) and Bavistin (0.1%) and bioagent *P. florescence* which contributed higher maize grain yield over check. Carbendazim, neem oil and *T. harzianum* as seed treatment (ST) and combinations of sprays with ST were found effective for managing BLSB of maize in field condition (Bunker *et al.*, 2012). Rani *et al.* (2013), examined fungicides and biocontrol agents viz. benomyl, carbendazim, thiram, *T. viride*, *P. fluorescens* and *B. subtilis* as seed and soil treatments against BLSB. Among all the treatments carbendazim and *T. viride* showed 37.93% and 41.9% respectively, reduction in BLSB disease severity.

Conclusion

The present study concluded that management of maize pathogens is considered very important in the present scenario because the BLSB caused by *R. solani* is most prevalent and serious limiting factors for the successful cultivation of maize worldwide. Studies revealed that none of disease management strategies is absolutely effective against BLSB. Variability within pathogen should be considered for screening and breeding for resistance, or while testing sensitivity of the pathogen towards different fungicides. The integrated management approaches evolved particularly in the present changing climate would provide sustainable management of BLSB. Moreover, inheritance pattern of BLSB resistance in maize verities/hybrids through conventional and/or biotechnological approaches. Additionally, progresses made in the "omics" field will revolutionize the possibilities for improving pathogen identification and investigating host-pathogen interaction, epidemiology and development of novel disease management practices. All these information should lead to more efficient management of this menacing disease.

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