© 2015 IJSRST | Volume 1 | Issue 5 | Print ISSN: 2395-6011 | Online ISSN: 2395-602X
Themed Section: Science and Technology

A Review for Rice Sheath Blight Disease

Abdolreza Nokhbeh Zaeim[1], Na-Mirzaie Amirabad[2], Ah-Drakhshan[3], Mo-Noorozi[4], K-Moradi[5]
[1]M.Sc. Dept. of Agronomy and Agroecology, Faculty of Agriculture, Ferdowsi University of Mashhad, Iran
[2]M.Sc. Dept. of Agronomy and Horticultural Plant Breeding, Faculty of Agriculture, Zabol University,Iran
[3]Phd. student of Zabol University and Institute jahad daneshgahi of Kashmar. Iran
[4]Tanin Keshtzar Sabz Agricultural Research, Service and Production Cooperative Company, Iran
[5]Phd. student of Dept. of Agronomy and Plant Physiology, Faculty of Agriculture, Zabol University, Iran

ABSTRACT

Rice is an important food grain and is a staple food for majority of the world's population. However, biotic stresses such as diseases have impeded rice cultivation both in the tropics and subtropics. Of them, Rice sheath blight (ShB), caused by Rhizoctonia solani, leads to severe yield losses in many rice production areas worldwide. little progress has been made in rice breeding for sheath blight resistance. None of the commercially cultivated rice varieties have sufficient level of field resistance, and the disease is presently being managed by chemical pesticides.Within the strobilurins group, azoxystrobin fungicide is widely used as it works effectively against ShB pathogen infestation. The survival and inoculums potential of the R. solani is directly related with severity and incidence of disease. Wild relatives of cultivated rice are a potential source of novel genes for insect and disease resistance, as well as tolerance to several abiotic stresses and a source of yield and yield enhancing traits.
Keywords : Rice, Rhizoctonia Solani, Sheath Blight, Chemical Pesticides.

I. INTRODUCTION

R. solanican infect seed to fully mature plant, causing moderate to significant yield losses depending on the plant part affected. Visible plant disease symptoms include formation of lesions, plant lodging, and presence of empty grains. Large lesions formed on infected sheaths of lower rice leaves may lead to softness of the stem thereby initiating stem lodging (Wu et al., 2012). Rhizoctonia solaniis a common soilborne pathogenic fungus that has worldwide implications on an extensive range of agricultural plants (Anderson, 1982). In rice, it is responsible for causing sheath blight disease, rendering significant declines in crop quality and yield (Su'udi et al., 2013).Wild relatives of cultivated rice are a potential source of novel genes for insect and disease resistance, as well as tolerance to several abiotic stresses and a source of yield and yield enhancing traits (Brar and Singh, 2011; Shakiba and Eizenga, 2014). It has been reported that polygalacturonase inhibiting proteins can inhibit the degradation of the plant cell wall by polygalacturonases from pathogens. Activity assay confirmed the inhibitory activity of OsPGIP1 against the PGase from Rhizoctonia solani. In addition, the location of OsPGIP1 was determined by subcellular localization (Rui et al., 2015). Damage increased substantially in major rice growing regions following introduction of high-yielding semi-dwarf rice cultivars, and the application of excessive nitrogen fertilizers to rice fields. Sheath blight management is difficult due to the low inherent level of resistance among cultivated or wild rice, and broad host range and high genetic diversity of the pathogen (Taheri et al., 2007). Owing to the limitations of conventional breeding and the use of environmentally hazardous chemical pesticides, alternative strategies are being tried globally to enhance protection of crops against invading pathogens (Maruthasalam et al., 2007). Genetic engineering may be proved as a promising alternative strategy for the management of economically important plant disease like rice sheath blight.

II. METHODS AND MATERIAL

A. Mode of Survival of Pathogen

The survival and inoculums potential of the R. solani is directly related with severity and incidence of disease. Sclerotia are the most important source of inoculums (Allison, 1951). Li Shi Dong (2004) reported that 60.9% sclerotia could survive after 265 days of being buried in natural sandy loam under field conditions in Beijing, while colonized rice straw debris (0.5-10 cm long) could not yield the fungus on medium plates after 88 days of being buried under same conditions. Singh and Singh (2008) reported that the infected leaf pieces incubated at 10 and 28°C also showed a steady reduction in fungus survival with an incubation period from an initial 100% to 53.3% and 63.3% respectively after a period of 5 months.

B. Host Range

Kozaka (1965), Tsai (1974) observed that rice fungus infected 20 species which are from 11 families and observed that the sclerotia from diseased tissue of weed hosts produced typical symptoms of sheath blight on paddy plants. Singh and Saksena (1980) found that aerial strain causing banded blight disease in bajra infected 22 plants species of both crop and wild plants belonging to 6 different families. Kannaiyan and Prasad (1980) have listed 30 monocot weed species as host of Thanatephorus cucumeries (Rhizoctnia solani).Goswami . (2010) reported that isolate SYL-13 possessed narrow host range and low avirulent while DIN-8 and GAZ-18 had wide host range and considered as virulent isolate of Rhizoctonia solani.

C. Chemical Control

Within the strobilurins group, azoxystrobin fungicide is widely used as it works effectively against ShB pathogen infestation (Groth and Bond, 2006). The fungicide is a derivative of β-methoxyacrylate and was the first registered fungicide from this class of chemistry (Anonymous, 1996). It is sold as Quadris 2.08 SC (Syngenta, Raleigh, NC). Azoxystrobin is considered one of the best fungicides in the U.S. for sheath blight control (Grichar et al., 2004). The mode of action of azoxystrobin is to inhibit electron transport and kill the fungal pathogen Use of fungicide rate and composition is based on intensity of disease and the type of cultivars (susceptible/medium susceptible/ moderately resistant) used. Benefits from fungicide control include lower disease incidence, likely reduction of inoculum, and improved grain and milling yields (Groth, 2008; Groth, 1996).

D. Biological Control

Rhizosphere-isolated, free living soil bacteria with proven plant beneficial properties are known as plant growth- promoting rhizobacteria (PGPR) (Kloepper et al., 1978). Besides, PGPR role in increasing plant or root growth, they directly influence increasedN uptake, phosphate solubilization, phytohormone synthesis, and production of iron chelating siderophores (Lalande et al., 1989; Bowen and Rovira, 1999). Some PGPR are used commercially to enhance plant growth and health. Seed treatment of rice with PGPR resulted in increased root and shoot length of seedlings (Kumar et al., 2009).

Secondary metabolites of rice sheath blight pathogen
A series of metabolites from several types of R. solani have been identified. They included fatty acids (i.e., 9-(Z)-octadecenoic acid and 9,12-octadecadienoic acid) (Aliferis and Jabaji, 2010a), steroids (i.e., ergosterol) (Ma et al., 2004; Aliferis and Jabaji, 2010a), phenolics (i.e., m-hydroxyphenylacetic aicd, m-methoxyphenylacetic acid, p-hydroxybenzoic acid, methyl p-hydroxybenzoate) (Mandavaetal, 1980; Adachi and Inagaki, 1988), alkaloids (i.e., Nb-acetyltryptamine) (Pedrasetal, 2005), cyclopeptides (i.e., cyclo (S-Pro-S-Leu), cyclo (S-Pro-S-Ile), cyclo (S-Pro-S-Val)) (Pedras et al., 2005), saccharides (i.e., RS-toxin) (Vidhyasekaran et al., 1997; Sriram et al., 2000), and glycoprotein (Velazhahan and Vidhyasekaran, 2000). Some metabolites were screened to have phytotoxic activity (i.e., m-hydroxyphenylacetic aicd, m-methoxyphenylacetic acid, p-hydroxybenzoic acid) (Mandava et al., 1980; Adachi and Inagaki, 1988), and elicitation effect on the activities of phenylalanine ammonia-lyase (PAL) and 4-coumarate: CoA ligase (4CL) in suspension-cultured rice cells (i.e., glycoprotein) (Velazhahan and Vidhyasekaran, 2000). The aim of this investigation was to further search for bioactive metabolites from rice sheath blight pathogen R. solaniin order to provide supporting data for reveal their physiological and ecological functions.

E. Role of OsWRKY Transcription Factors in rice Disease Resistance

WRKY transcription factors in plants regulate diverse biological functions including abiotic and biotic stress responses, growth and development, embryogenesis and many other physiological processes.Indica and japonica genotypes of rice were identified to have 111 and 113 WRKY genes respectively in their genomes. Reports on the involvement of some of the WRKY genes in rice disease resistance covering the major diseases like blast and bacterial blight indicate the possibilities of further exploring these genes for the production of disease resistant varieties (John et al., 2015).

F. PR Genes Enhance Resistance Against Sheath Blight

Several groups have reported that the introduction of single PR genes such as PR-3 chitinase (Datta et al., 2000, 2001; Lin et al.1995), PR-5(thaumatin-like protein) (Datta et al., 1999) provides resistance against rice sheath blight pathogen. The expression of differentPRgenes in combination such as rice chitinase (CHI11) and tobaccob-1,3-glucanase(gluc) (Sridevi et al., 2008), CHI11and thaumatin-like protein (Kalpana et al., 2006), maize ribosomeinactivating gene MOD1and rice basic chitinase gene RCH10(Kim et al., 2003a) and barleychitinase, and barley b-1,3-glucanasegenes (Jach et al., 1995) that are driven by different constitutive promoters, conferred a higher level of sheath blight resistance than any single PRgene. Moreover, constitutive expression of these genes may provide a metabolic burden and increased energy cost to the plants. Therefore, combined tissue-specific overexpression of PR genes in rice may deliver a novel strategy to control yield losses caused by sheath blight pathogen.

G. OsPGIP1 in rice Sheath Blight Resistance

Cell wall is the first shield for the defense of plant cells against the attack of pathogens (De Lorenzo et al., 2001). During the early stage of pathogen invasion, phytopathogenic fungi produce a series of hydrolytic enzymes to degrade the plant cell wall, such as polygalacturonases (PGases), xylanases and galucanases (Alghisi and Favaron, 1995). In order to resist the invasion of pathogens, the plant also secretes some enzymes such as chitinases and polygalacturonase inhibiting proteins (PGIPs) to prevent the degradation of the plant cell wall by pathogens (Shanmugam, 2005; Vorwerk et al., 2004). PGIP can also maintain the elicitation activity of oligosaccharides for a longer time, prolonging the defense response of the plant (Mishra et al., 2012).

H. Predicting Potential Epidemics of Rice Sheath Blight Using a Rice Disease Epidemiology Model, EPIRICE

EPIRICE is a generic epidemiological model that can be parameterized to address any specific rice disease (Savary et al., 2012). It was recently developed as a general model framework for fungal, viral, and bacterial diseases at different levels of hierarchy in a crop canopy (leaves, sheaths, entire plants) depending on the nature of the disease. Thus, its structure was designed to be as simple as possible, involving a few state variables and a limited number of core parameters and weather variables. Savary et al. (2012) developed EPIRICE to evaluate the potential importance of plant diseases in rice and their intensity and distribution at a global scale, at which very limited actual field data on disease epidemics exist across different locations and years.

III. REFERENCES

[1] Adachi T, Inagaki K. 1988. Phytotoxin producued by Rhizoctonia oryzae Ryker et Gooch. Agricultural and Biological Chemistry, 52, 2625.

[2] Alghisi P, Favaron F. 1995. Pectin-degrading enzymes and plant-parasite interactions. Eur J Plant Pathol 101:365–375

[3] Aliferis K A, Jabaji S. 2010a. 1H NMR and GC-MS metabolic fingerprinting of developmental stages of Rhizoctonia solani sclerotia. Metabolomics, 6, 96–108.

[4] Allison JL. 1951. Rhizoctonia blight of forage legumes and grasses. Pl. Dis. Reptr. 35: 372-373

[5] Anderson NA (1982) The genetics and pathology ofRhizoctonia solani.Annu Rev Phytopathol 20:329–347

[6] Anonymous D. 1996. New fungicide for disease control on fruit and nut crops. Zeneca Incorp. Tech, Information Bulletin, Wilmington, DE.

[7] Bowen GD, Rovira AD. 1999. The rhizosphere and its management to improve plant growth. Advances in Agronomy 66:1-102.

[8] Brar D, Singh K. 2011. Oryza. In: Kole C (ed) Wild crop relatives: Genomic and breeding resources: Cereals. Dordrecht London, New York: Springer Heidelberg, pp 321–336

[9] Datta K, Koukolikova-Nicola Z, Baisakh N, Oliva N, Datta SK. 2000. Agrobacterium-mediated engineering for sheath blight resistance of indica rice cultivars from different ecosystems. Theor Appl Genet 100:832–839.

[10] Datta K, Tu J, Oliva N, Ona I, Velazhahan R, Mew TW,Muthukrishnan S, Datta SK. 2001. Enhanced resistance to sheath blight by constitutive expression of infection-related rice chitinase in transgenic elite indica rice cultivars. Plant Sci 160:405–414.

[11] Datta K, Velazhahan R, Oliva N, Ona I, Mew T, Khush G,Muthukrishnan S, Datta SK. 1999. Over-expression of the cloned rice thaumatin-like protein (PR-5) gene in transgenic rice plants enhances environmental friendly resistance toRhizoctonia solani causing sheath blight disease. Theor Appl Genet 98:1138–1145.

[12] De Lorenzo G, D'Ovidio R, Cervone F. 2001. The role of polygalacturonase-inhibiting proteins (PGIPs) in defense against pathogenic fungi. Annu Rev Phytopathol 39:313–335

[13] Goswami BK, Bhuiyan KA and Mian IH. 2010. Morphological and Pathogenic variations in the isolates of Rhizoctonia solani in Bangladesh. Bangladesh J. Agril. Res. 35:375-380

[14] Grichar WJ, Jaks AJ, Besler BA. 2004. Response of peanuts (Arachis hypogaea) to weather-based fungicide advisory sprays. Crop Protection 24: 349-354.

[15] Groth DE, Bond JA. 2006. Initiation of rice sheath blight epidemics and effect of application timing of azoxystrobin on disease incidence, severity, yield, and milling quality. Plant Disease 90: 1073-1076.

[16] Groth DE. 1996. Two new fungicides to control rice diseases. La. Agric 39: 31-33.

[17] Groth DE. 2008. Effects of cultivar resistance and single fungicide application on rice sheath blight, yield, and quality. Crop Protection 27: 1125-1130.

[18] Jach G, Go ¨rnhardt B, Mundy J, Logemann J, Pinsdorf E, Leah R,Schell J, Maas C. 1995. Enhanced quantitative resistance against fungal disease by combinatorial expression of different barley antifungal proteins in transgenic tobacco. Plant J 8:97–109

[19] John J. Subramanian B. 2015. Role ofOsWRKY transcription factors in rice disease resistance. Trop. plant pathol.40:355–361.

[20] Kalpana K, Maruthasalam S, Rajesh T, Poovannan K, Kumar KK,Kokiladevi E, Raja JA, Sudhakar D, Velazhahan R, Samiyappan R. 2006. Engineering sheath blight resistance in elite indica rice cultivars using genes encoding defense proteins. Plant Sci 170:203–215.

[21] Kannaiyan S and Prasad NN. 1980. Dicot weed hosts of Rhizoctonia solani Kuhn. Agri. Res. J. Kerala. 18:125-127

[22] Kloepper JW, Schroth MN. 1978. Plant growth promoting rhizobacteria on radish.: Proceedings of the Fourth Conference Plant Pathogenic Bacteria. INRA, Angers, France.

[23] Kozaka T. 1961. Ecological studies on sheath blight of rice of rice caused Pellicularia sasakii (Shirai) and its chemical control. Chugoko agric. Res. 20:1-133

[24] Kumar KVK, Reddy MS, Kloepper JW, Lawrence KS, Groth DE. 2009. Sheath blight disease of rice (Oryza sativa L.) -An overview. Biosciences, Biotechnology Research Asia 6: 465-480.

[25] Lalande R, Bissonnette N, Coutlee D, Autoun H. 1989. Identification of rhizobacteria from maize and determination of their plant-growth promoting potential. Plant and Soil 115: 7-11.

[26] Li D. 2004. Population dynamics and survival of Rhizoctonia solani AG-1 in the field soil under ricewheat rotation. Agricultural sciences of China. 3:448-455

[27] Lin W, Anuratha C, Datta K, Potrykus I, Muthukrishnan S, Datta SK. 1995. Genetic engineering of rice for resistance to sheath blight.Nat Biotechnol 13:686–691.

[28] Ma Y, Li Y, Liu JY, Song YC, Tan RX. 2004. AntiHelicobacter pylori metabolites from Rhizoctonia sp. Cy064, an endophytic fungus in Cynodon dactylon. Fitoterapia, 75, 451–456.

[29] Mandava NB, Orellana RG, Warthen JD, Worley JF, Dutky SR, Finegold H, Weathington BC.

1980. Phytotoxins in Rhizoctonia solani: isolation and biological activity of m-hydroxy and m-methoxyphenylacetic acids. Journal of Agricultural and Food Chemistry, 28, 71–75.
[30] Maruthasalam S, Kalpana K, Kumar KK, Loganathan M, Poovannan K, Raja JAJ (2007) Pyramiding transgenic resistance in elite indica rice cultivars against the sheath blight and bacterial blight. Plant Cell Rep 26:791–804.
[31] Mishra AK, Sharma K, Misra RS. 2012. Elicitor recognition, signal transduction and induced resistance in plants. J Plant Interact 7:95–120
[32] Pedras M S, Yu Y, Liu J, Tandron-Moya Y A. 2005. Metabolites produced by the phytopathogenic fungus Rhizoctonia solani: isolation, chemical structure determination, syntheses and bioactivity. Zeitschrift für Naturforschung C (Journal of Biosciences), 60, 717–722.
[33] Rui Wang Liaoxun Lu Xuebiao Pan Zongliang Hu•Fei Ling Yan Yan Yemao Liu Yongjun Lin.2015. Functional analysis of OsPGIP1in rice sheath blight resistance. Plant Mol Biol ,87:181–191
[34] Savary S, Nelson A, Willocquet L, Pangga I, Aunario J. 2012. Modeling and mapping potential epidemics of rice diseases globally. Crop Prot. 34, 6–17.
[35] Shakiba E, Eizenga GC. 2014. Unraveling the secrets of rice wild species. In: Yan WG, Bao J (eds.) Rice—germplasm,genetics and improvement, doi:10.5772/58393
[36] Shanmugam V. 2005. Role of extracytoplasmic leucine rich repeat proteins in plant defence mechanisms. Microbiol Res 160:83–94
[37] Singh PK and Singh AK. 2008. Effect of temperature and soil texture on the survival ability of rice sheath blight pathogen (Rhizoctonia solani). Flora and Fauna (Jhansi). 14:55-56.
[38] Singh SB and Saksena HK. 1980. A new sheath and leaf blight of bajra.Indian Phytopath.33:127-129
[39] Sridevi G, Parameswari C, Sabapathi N, Raghupathy V, Veluthambi K. 2008. Combined expression of chitinase andb-1, 3-glucanase genes in indica rice (Oryza sativaL.) enhances resistance against Rhizoctonia solani. Plant Sci 175:283–290
[40] Sriram S, Raguchander T, Babu S, Nandakumar R, Shanmugam V, Vidhyasekaran P, Balasubramanian P, Samiyappan R. 2000. Inactivation of phytotoxin produced by the rice sheath blight pathogen Rhizoctonia solani. Canadian Journal of Microbiology, 46, 520–524.
[41] Su'udi M, Park J-M, Kang W-R, Hwang D-J, Kim S, Ahn I-P. 2013. Quantification of rice sheath blight progression caused by Rhizoctonia solani. J Microbiol 51:380–388
[42] Taheri P, Gnanamanickam S, Ho¨fte M. 2007. Characterization, genetic structure, and pathogenicity ofRhizoctoniaspp. associated with rice sheath diseases in India. Phytopathology 97:373–383
[43] Tsai WH. 1974. Assessment of yield losses due to rice sheath blight at different inoculation stages. Journal of Taiwan Agricultural Research. 23: 188-194
[44] Velazhahan R, Vidhyasekaran P. 2000. Isolation of an elicitor from Rhizoctonia solani, the rice sheath blight pathogen which activates phenylpropanoid metabolism in suspensioncultured rice cells. Zeitschrift fur Pflanzenkrankheiten und Pflanzenschutz, 107, 135–144.
[45] Vidhyasekaran P, Ponmalar T R, Samiyappan R, Velazhahan R, Vimala R, Ramanthan A, Paranidharan V, Muthukrishnan S. 1997. Host-specific toxin production by Rhizoctonia solani, the rice sheath blight pathogen. Phytopathology, 87, 1258–1263.
[46] Vorwerk S, Somerville S, Somerville C. 2004. The role of plant cell wall polysaccharide composition in disease resistance. Trends Plant Sci 9:203–209
[47] Wu W, Huang J, Cui K, Nie L, Wang Q. 2012. Sheath blight reduces stem breaking resistance and increases lodging susceptibility of rice plants. Field Crops Research 128: 101-108