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Blast Disease in Rice : A Review

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ABSTRACT

Rice blast caused by the fungal pathogen, *Magnaporthe grisea* (anamorph: *Pyricularia grisea*) limits rice yield in all major rice-growing regions of the world and the rice blast fungus, *Magnaporthe oryzae*, is responsible for the most serious disease of rice and is a continuing threat to ensuring global food security. The fungus has also, however, emerged as a model experimental organism for understanding plant infection processes by pathogenic fungi. This hemibiotrophic pathogen penetrates in epidermal cells and causes lesions on leaves, leaf collar, culm, culm nodes and panicle neck causing failure of seed filling. After successful penetration, the invasive hyphae grow rapidly in the host cells and caused blast lesions. in 5 to 7 days, the pathogen produces numerous conidia from the lesions and initiates a new infection cycle. A number of signal transduction pathways are implicated in appressorium-mediated plant infection and have been characterised as a potential means of developing new chemical intervention strategies for disease control. With the advent of new technologies like marker-assisted selection, molecular mapping, map-based cloning, marker-assisted backcrossing and allele mining, breeders have identified more than 100 Pi loci and 350 QTL in rice genome responsible for blast disease.

Keywords: Rice Blast, Magnaporthe Oryzae, Lesions, Marker-Assisted Selection

I. INTRODUCTION

Magnoporthe oryzae Heb.(anamorph: *Pyricularia oryzae* Cav. Or *Pyricularia grisea* Sacc.) causes the rice blast disease (Wu et al., 2006). M. oryzae is a hemibiotrophic, ascomycetous fungus that has been reported to infect more than 50 grass species (Pennisi 2010). Rice blast disease is one of the most devastating of all cereal diseases worldwide and causes harvest losses of 10–30 % of the global rice yield annually (Talbot 2003) and economic losses over $70 billion of dollar (Scheuermann 2012). *M. oryzae* is listed as number one of the ten most important fungal pathogens in molecular biology (Dean et al., 2012). In the first phase of infection, the conidia form an injection apparatus (appressorium), which penetrates the leaf cuticle (Wilson and Talbot 2009). Invasive hyphae then rapidly grow in the rice leaf and stem. The fungal biomass soon reaches over 30% and the rice plant succumbs to necrosis (Wilson and Talbot 2009). This hemibiotrophic pathogen penetrates in epidermal cells and causes lesions on leaves, leaf collar, culm, culm nodes and panicle neck causing failure of seed filling. Blast-resistant rice cultivars (cvs) have normally a short field life due to the plasticity of the *M. oryzae* genome, that is able to evolve new race by mutation of the avirulence (Avr) genes, causing a breakdown of the deployed plant resistance conditioned by R genes (Dean et al., 2005; Valent and Khang 2010). Many studies indicated that the genetic control of blast resistance is complex and involves both major and minor resistance genes with complementary or additive effects, as well as environmental interactions (Wang et al., 1994; Wu et al., 2005; Li et al., 2007, 2008a, b; He et al., 1989; Bonman 1992). Thus, the discovery and use of novel R genes and development of broad-spectrum resistant varieties are

urgent goals in breeding for blast resistance in rice. Since the idea of indirect selection using genetic markers was first reported by Sax (Sax et al., 1923) over 80 years ago, and particularly in the last few decades, new technologies have emerged that allow breeders to more easily select changes at the DNA level. Much of the progress to date has centered on marker-assisted backcrossing or the pyramiding of genes against rice blast (Torres, 2010). Molecular markers are essential for mapping genes of interest, marker-assisted breeding, and cloning genes using mapping-based cloning strategies (Hayashi et al., 2004).

II. METHODS AND MATERIAL

A. Plant Penetration

The heterothallic ascomycete *Magnaporthe oryzae*, is the most destructive disease of cultivated rice worldwide and can lead to severe losses of annual rice yield (Valent et al., 1991; Talbot, 2003). Under normal conditions, the fungus uses a highly specialized infection structure appressorium generated from a conidium for plant penetration (Howard et al., 1991; Jong et al., 1997). After successful penetration, the invasive hyphae grow rapidly in the host cells and caused blast lesions. In 5 to 7 days, the pathogen produces numerous conidia from the lesions and initiates a new infection cycle.

B. Regulation of gene expression at the level of transcription

Regulation of gene expression at the level of transcription controls many crucial biological processes. A number of different factors, including transcription factors, are essential for the process of transcription. Transcription factors can recognize DNA in a sequence-specific manner and modulate the frequency of initiation of transcription upon binding to specific sites in the promoter of target genes. The transcription factors can be activators, repressors, or both usually display a modular structure named the DNA-binding domain (Pabo and Sauer 1992). In *M. oryzae*, numerous transcription factors were identified and characterized to be important for proper regulation of infection related morphogenesis (Li and Xu, 2012; Kim et al., 2009). Many transcription factors, including MoCrz1, MoAp1, MoAtf1, MoHac1, MoBzip10, MoSwi6 and MoMsn2 were reported to be involved in hyphal growth, asexual development, stress response, infectious growth and virulence by controlling the expression levels of a series of target genes (Zhang et al., 2009; Tang et al., 2014).

C. Effect of azoxystrobin and kresoxim-methyl on rice blast

During the 1980s, organophosphorus fungicides such as kitazin (EBP), kitazin P (IBP) and isoprothiolane (FJ-one) with different chemical structures but similar mode of action were widely used (Katagiri and Uesugi, 1977; Zhang et al., 2009). The strobilurin-based (QoI; Quinone outside inhibitors) fungicides have been reported to be very effective in controlling rice blast in the USA (Groth, 2006). The specific target of QoI fungicides is the quinol-oxidising (Qo) site of the mitochondrial enzyme cytochrome b (Kim et al., 2003), as these chemicals block electron transport at the Qo site, thereby inhibiting fungal respiration (Bartlett et al., 2000). Azoxystrobin and kresoxim-methyl, belonging to QoI fungicides, are relatively new for controlling rice blast in China. The results of field experiments also suggested that both azoxystrobin and kresoxim-methyl at 187.5 g.a.i. ha−1gave over 73% control efficacy in both sites, exhibiting excellent activity against rice blast. Taken together,azoxystrobin and kresoxim-methyl could be a good substitute for Carbendazim (MBC) or IBP for controlling rice blast in China, but should be carefully used as they were both at-risk (Chen et al., 2015).

D. Methionine Biosynthesis

Some plant amino acids such as cysteine, methionine, tryptophan, histidine and arginine are only present in trace amounts in the leaf apoplast and are likely not available for fungal nutrition (Fernandes et al., 2014; Solomon et al., 2003). Infectious hyphae are expected to synthesize these amino acids from abundant apoplastic amino-acid such as glutamate or aspartate. Genetic studies in *M.oryzae* support this hypothesis for different amino-acids including methionine (Marie et al., 2015).

E. Allele Mining Strategies for Blast Resistance

Allele mining approaches have been intended to identify superior alleles of rice blast resistance genes such as Pita (Yang et al., 2007; Huang et al., 2008; Wang et al., 2008; Ramkumar et al., 2010), Pikh (Ramkumar et al., 2010), Pi54 (Kumari et al., 2013), and Pi-2 (Hittalmani

et al., 2013) from different cultivated rice varieties and wild species. The blast resistance genes Pi9, Pi2 and Piz-ttend to be alleles from different rice resources while physically on the same gene locus on rice chromosome 6, but their level of resistance spectra can be quite different (Zhou et al., 2006; Zhu et al., 2012). In general, there are two approaches available for allele mining and/or identification of sequence polymorphisms for a given gene in a naturally developing population: (i) modified TILLING (Targeting Induced Local Lesions in Genomes) (Comai et al., 2004), called Eco-TILLING and (ii): Re-sequencing (Huang et al., 2009) or sequencing based allele mining.

F. Current advance methods for the identification of blast resistance genes in rice

After the discovery of molecular markers, the selection of target traits becomes easier and many new cultivars have been developed accordingly. Nowadays, breeders are focusing on marker-assisted selection instead of using conventional breeding because it reduces the time for phenotypic selection, saves input costs, brings more reliability to select a desired trait with no influence of environmental factors (Koide et al., 2010). Many DNA markers are directly linked with Pi genes in rice including simple sequence repeats (SSRs), amplified fragment length polymorphisms (AFLPs), and cleaved amplified polymorphic sequences (CAPS), random amplified polymorphic DNAs (RAPDs), restriction fragment length polymorphisms (RFLPs), single-nucleotide polymorphisms (SNPs) and small insertions/deletions (InDels). SSRs and CAPS are PCR-based markers and require only a small amount of DNA for genotyping. These markers are very precise and cost effective and can be applied for the selection of plants containing blast resistance genes in rice at an early stage. Small In Dels and SNPs markers are found in abundance and dispersed widely in the rice genome (Yu et al., 2002). On the basis of information on these markers, (Hayashi and Yoshida, 2006), developed nine PCR-based markers linked with blast resistance in rice. These markers help in finding a gene within the desired target genome regions. Microsatellite markers, also called SSRs, are widely used for screening the blast-resistant and susceptible varieties. The difference between two varieties is based on polymorphism (Miah and rafii 2013). With the advent of new technologies like marker-assisted selection, molecular mapping, map-based cloning, marker-assisted backcrossing and allele mining, breeders have identified more than 100 Pi loci nd 350 QTL in rice genome responsible for blast disease. These Pi genes and QTLs can be intro grassed into a blast susceptible cultivar through marker-assisted backcross breeding. These molecular techniques provide timesaving, environment friendly and labour-cost-saving ways to control blast disease.

III. REFERENCES

[1] Bartlett DW, Clough JM, Godwin JR, Hall AA, Hamer M, Parr-Dobrzanski B. 2000. The strobilurin fungicides.Pest Management Science, 58, 649–662.

[2] Bonman JM. 1992. Durable resistance to rice blast disease environmental influences. Euphytica 63: 115–123

[3] Burd C, Cullen PJ. 2014. Retromer: A Master Conductor of Endosome Sorting. Cold Spring Harbor Perspectives in Biology. 6: a016774.

[4] Chen Y,Yang X,Yuan K, Li Y, Zhang AF, Yao Y, Gao C. 2015. Effect of azoxystrobin and kresoxim-methyl on rice blast and rice grain yield in China. doi:10.1111/aab.12202

[5] Comai, L, Young, K, Till, B.J, Reynolds, SH, Greene, EA, Codomo, CA, Enns, LC, Johnson, JE, Burtner, C and Odden AR. 2004. Efficient discovery of DNA polymorphisms in natural populations by Ecotilling. The Plant Journal 37: 778-786.

[6] De Jong JC, McCormack BJ, Smirnoff N, Talbot NJ. 1997. Glycerol generates turgor in rice blast. Nature. 389: 244–5

[7] Dean JA, Van ZA, Pretorius KE, Hammond-Kosack A, Pietro PD, Spanu JJ, Rudd M, Dickman R, Kahmann J, Ellis GD. 2012. Foster, The top 10 fungal pathogens in molecular plant pathology, Molecular Plant Pathology. 13: 414-430

[8] Dean R, Talbot NJ, Ebbole DJ, Farman ML, Mitchell TK, Orbach MJ, Thon M, Kulkarni R, Xu JR, Pan H, Read ND, Lee YH, Carbone I, Brown D, Oh YY, Donofrio N, Jeong JS, Soanes DM, Djonovic S, Kolomiets E, Rehmeyer C, Li W, Harding M, Kim S, Lebrun MH, Bohnert H, Coughlan S, Butler J, Calvo S, Ma LJ, Nicol R, Purcell S, Nusbaum C, Galagan J, Birren BW. 2005. The genome sequence of the rice blast fungus Magnaporthe grisea. Nature. 434: 980–986

[9] Fernandez J, Marroquin-Guzman M, Wilson RA. 2014. Mechanisms of nutrient acquisition and

utilization during fungal infections of leaves. Annual Review of Phytopathology. 52: 8.1–8.20

[10] Groth DE. 2006. Azoxystrobin rate and timing effects on rice head blast incidence and rice grain and milling yields. Plant Disease,90, 1055–1058.

[11] Hayashi K, Hashimoto N, Daigen M, Ashikawa I. 2004. Development of PCR-based SNP markers for rice blast resistance genes at the Piz locus. Theoretical and Applied Genetics 108: 1212-1220.

[12] Hayashi K, Yoshida H, Ashikawa I. 2006. Development of PCR-based allelespecific and InDel marker sets for nine rice blast resistance genes, Theoretical and Applied Genetics. 113: 251–260.

[13] He YY, Li J, Li C, Kiyosawa S, Higashi T, Horisue N. 1989. Gene analysis of blast resistance of Yunnan upland rice variety, Haonaihuan. Oryza. 26:173–182

[14] Hittalmani S, Kahani F, Dhanagond SM and Rao AM. 2013. DNA marker characterization for allele mining of blast and bacterial leaf blight resistant genes and evaluation for grain yield. African Journal of Biotechnology. 12: 2331-2340

[15] Howard RJ, Ferrari MA, Roach DH, Money NP. 1991. Penetration of hard substrates by a fungus employing enormous turgor pressures. Proceedings of the National Academy of Sciences. USA. 88: 11281–11284.

[16] Huang CL, Hwang SY, Chiang YC and Lin TP. 2008. Molecular evolution of the Pi-ta gene resistant to rice blast in wild rice (Oryza rufipogon). Genetics 179: 1527-1538.

[17] Huang X, Feng Q, Qian Q, Zhao Q, Wang L, Wang A, Guan J, Fan D, Weng Q and Huang T. 2009. High-throughput genotyping by whole- genome resequencing. Genome Research. 19: 1068-1076

[18] Katagiri M, Uesugi Y. 1977. Similarities between the fungicidal action of isoprothiolane and organophosphorus thiolate fungicides. Phytopathology,67, 1415–1417

[19] Kim S, Park S-Y, Kim KS, Rho H-S, Chi M-H, Choi J. 2009. Homeobox transcription factors are required for conidiation and appressorium evelopment in the rice blast fungus Magnaporthe oryzae. PLoS Genet. 5: e1000757

[20] Kim YS, Dixon EW, Vincelli P, Farman ML. 2003. Field resistance to strobilurin (Q oI) fungicides inPyricularia grisea caused by mutations in the mitochondrial cytochrome b gene.Phytopathology,93, 891–900

[21] Koide N, Kobayashi D, Xu Y, Fukuta S. 2010. Resistance genes and selection DNA markers for blast disease in rice, Oryza sativa, Japan Agricultural Research Quarterly (JARQ). 43: 255–280.

[22] Koumandou VL, Klute MJ, Herman EK, Nunez-Miguel R, Dacks JB. 2011. Evolutionary reconstruction of the retromer complex and its function in Trypanosoma brucei. Journal of Cell Science 124: 1496–1509.

[23] Kumari A, Das A, Devanna B, Thakur S, Singh P, Singh N and Sharma T. 2013. Mining of rice blast resistance gene Pi54 shows effect of single nucleotide polymorphisms on phenotypic expression of the alleles. European Journal of Plant Pathology 137: 55-65.

[24] Li G, Zhou X, Xu JR. 2012. Genetic control of infection-related development in Magnaporthe oryzae. Current Opinion in Microbiology. 15: 678–84.

[25] Li W, Lei CL, Cheng ZJ, Jia YL, Huang DY, Wang JL, Wang JK, Zhang X, Su N, Guo XP, Zhai HQ, Wan JM. 2008a. Identification of SSR markers for a broad-spectrum blast resistance gene Pi20(t) for marker-assisted breeding. Molecular Breeding. 22: 141–149

[26] Li YB, Wu CJ, Jiang GH, Wang LQ, He YQ. 2007. Dynamic analyses of rice blast resistance for the assessment of genetic and environmental effects. Plant Breed. 126: 541–547

[27] Marie E, Crystel B, Marie J, Océane F, Roland B, Marc H, Michel D. 2015. Methionine Biosynthesis is Essential for Infection in the Rice Blast Fungus Magnaporthe oryzae. PLoS One. 10: e0111108.

[28] Maruzs T, Lorincz P, Szatmari Z, Szeplaki S, Sandor Z. 2015. Retromer Ensures the Degradation of Autophagic Cargo by Maintaining Lysosome Function in Drosophila. Traffic. 10: 1088-1107

[29] Miah G, Rafii MR, Ismail AB, Puteh HA, Rahim KN, Islam MA, Latif S. 2013. A review of microsatellite markers and their applications in rice breeding programs to improve blast disease resistance, International Journal of Molecular Sciences. 14: 22499–22528.

[30] Pabo CO, Sauer RT.1992. Transcription factors: structural families and principles of DNA recognition. Annual Review of Biochemistry. 61: 1053–95

[31] Pennisi E. 2010. Armed and dangerous. Science 327: 804–805

[32] Ramkumar G, Biswal A, Mohan KM, Sakthivel K, Sivaranjani A, Neeraja C, Ram T, Balachandran S, Sundaram R and Prasad M. 2010. Identifying novel alleles of rice blast resistance genes Pik hand Pita through allele mining. Plant Breeding.

[33] Sax K. 1923.The association of size differences with seed-coat pattern and pigmentation in Phaseolus vulgaris. Genetics, 8: 552–560

[34] Scheuermann KK, Raimondi JV, Marschalek R, Andrade A, Wickert E. 2012. Magnaporthe oryzae genetic diversity and its outcomes on the search for durable resistance. INTECH Open Access Publisher

[35] Seaman MN, McCaffery JM, Emr SD. 1998. A membrane coat complex essential for endosome-to-Golgi retrograde transport in yeast. The Journal of Cell Biology 142: 665–681.

[36] Seaman MNJ. 2012. The retromer complex—endosomal protein recycling and beyond. Journal of Cell Science 125: 4693–4702.

[37] Solomon PS, Tan KC, Oliver RP. 2003. The nutrient supply of pathogenic fungi: a fertile field for study. Molecular Plant Pathology. 4: 203–210

[38] Talbot NJ. 2003. on the trail of a cereal killer: exploring the biology of Magnaporthe grisea. Annual Review of Microbiology. 57: 177–202.

[39] Tang W, Ru Y, Hong L, Zhu Q, Zuo R, Guo X. 2015. System-wide characterization of bZIP transcription factor proteins involved in infection-related morphogenesis of Magnaporthe oryzae. Environmental microbiology. 17: 1377-1396.

[40] 40Torres AM. 2010. Application of Molecular Markers for Breeding Disease Resistant Varieties in Crop Plants. In Molecular Techniques in Crop Improvement; Jain SM, Brar DS, Eds, Springer Science and Business Media BV. Dordrecht, The Netherlands pp. 185–205

[41] Valent B, Chumley FG. 1991. Molecular genetic analysis of the rice blast fungus Magnaporthe grisea. Annual Review of Phytopathology. 29: 443–67

[42] Valent B, Khang CH. 2010. Recent advances in rice blast effector research. Current Opinion in Plant Biology. 13: 434–441

[43] 42Wang GL, Mackill DJ, Bonman JM, McCouch SR, Champoux MC, Nelson RJ. 1994. RFLP mapping of genes conferring complete and partial resistance to blast in a durably resistant rice cultivar. Genetics 136: 1421–1434

[44] Wilson RA, Talbot NT. 2009. Under pressure: investigating the biology of plant infection by Magnaporthe oryzae, Nature Reviews Microbiology 7: 185-195.

[45] Wu JL, Fan YY, Li DB, Zheng KL, Leung H, Zhuang JY. 2005. Genetic control of rice blast resistance in the durably resistant cultivar Gumei 2 against multiple isolates. Theoretical and Applied Genetics 111: 50-56.

[46] Wu SC, Halley JE, Luttig C, Fernekes LM, Gutiérrez-Sanchez G, Darvill AG, Albersheim P. 2006. Identification of an endo-β-1,4-Dxylanase from Magnaporthe grisea by gene knockout analysis, urification, and heterologous expression. Applied and environmental microbiology 72: 986–993.

[47] Yang MZ, Cheng ZQ, Chen SN, Qian J, Xu LL and Huang XQ. 2007. A rice blast-resistance genetic resource from wild rice in Yunnan, China. Journal of Plant Physiology and Molecular Biology 33: 589-595.

[48] Yang S, Gu T, Pan C, Feng Z, Ding J, Hang Y, Chen JQ and Tian D. 2008. Genetic variation of NBS-LRR class resistance genes in rice lines. Theoretical and Applied Genetics. 116: 165-177.

[49] Yu J, Hu S, Wang J,Wong JKS, Li S, Liu B, Zhang B. 2002. A draft sequence of the rice genome (Oryza sativa L. ssp. indica), Science 296: 79–92.

[50] Zhang C, Huang X, Wang J, Zhou M. 2009. Resistance development in rice blast disease caused byMagnaporthe griseato tricyclazole.Pesticide Biochemistry and Physiology,94,43–47

[51] Zhang H, Zhao Q, Liu K, Zhang Z, Wang Y, Zheng X. 2009. MgCRZ1, a transcription factor of Magnaporthe grisea, controls growth, development and is involved in full virulence. FEMS Microbiology Letters. 293: 160–9

[52] Zhou B, Qu S, Liu G, Dolan M, Sakai H, Lu G, Bellizzi M and Wang GL. 2006. The eight amino-acid differences within three leucine-rich repeats between Pi2 and Piz-t resistance proteins determine the resistance specificity to Magnaporthe grisea. Molecular plant microbe interactions 19: 1216-1228.

[53] Zhu X, Chen S, Yang J, Zhou S, Zeng L, Han J, Su J, Wang L and Pan Q. 2012. The identification of Pi50 (t), a new member of the rice blast resistance Pi2/Pi9 multigene family. Theoretical and Applied Genetics 124: 1295-1304.