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REVIEW

The arms race between *Magnaporthe oryzae* and rice: Diversity and interaction of *Avr* and *R* genes

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Abstract

Rice blast disease, caused by *Magnaporthe oryzae*, threatens global food security. The rice blast pathosystem is a longstanding model system for understanding plant-microbe interactions. In order to elucidate the coevolution of the host and pathogen, and provide the appropriate methods for preventing or controlling rice blast disease, researchers have focused on the evolution of virulence factors and resistance genes. Thus far, more than 30 rice blast resistance (*R*) genes and 12 avirulence (*Avr*) genes have been cloned. This review summarizes the cloned rice blast *R* genes, cloned *Avr* genes of *M. oryzae* and the interaction between them. This discussion also considers some of the major unanswered questions concerning this pathosystem and the opportunities for future investigations.

Keywords: rice, *Maganporthe oryzae*, resistance gene, avirulence gene, co-evolution, genetic diversity

1. Introduction

With the uncertainties associated with climate change and human population growth, food security has become an extremely important global issue. Plant diseases cause significant losses in crop yield and emerge diseases threaten to exacerbate concerns about the global food supply. The breeding and deployment of resistant cultivars is the most economical, effective and environmentally friendly approach to prevent or control crop diseases. Plants rely on innate immunity against microbes to serve as a primary line of defense and systemic signals emanating from infection sites serve to strengthen defense against pathogens (Dangl and Jones 2001; Chisholm *et al*. 2006; Jones and Dangl 2006). This innate immunity relies on recognition of pathogen-associated molecular patterns (PAMPs), which are extracellular molecules resulting from the presence of the microbe. This recognition by extracellular receptors leading to the arrest of pathogen colonization is called PAMP-triggered immunity (PTI). The second layer of defense is due to the recognition of the pathogen's virulence factors and is known as effector-triggered immunity (ETI) (Chisholm *et al*. 2006; Jones and Dangl 2006; Dodds and Rathjen 2010). Pathogens secrete effectors and deliver them into the plant cell to induce host susceptibility, often

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by interfering with components of PTI signaling. Such effectors can be recognized by intracellular plant receptors, resulting in a much stronger resistance response compared to PTI, and often results in a programmed cell death known as the hypersensitive response (HR). The plant receptors are typically nucleotide-binding leucine-rich repeat (NLR) proteins or kinases encoded by the resistance genes (*R* genes). Because the resistance relies on a receptor-ligand interaction, most *R* genes are dominant in genetic crosses between resistant and susceptible cultivars.

Rice is one of the main staple cereal crops in the world. Rice blast disease, caused by the filamentous fungus *Magnaporthe oryzae*, is the most destructive disease in many rice-growing areas, with 10–30% of the annual rice yield lost due to the disease (Skamnioti and Gurr 2009). Understanding the mechanisms for virulence is all the more important in light of the recent emergence of wheat blast (Callaway 2016). Recent advances in understanding the pathogenesis of *M. oryzae* and rice resistance mechanisms have led to a deeper level of understanding of PTI and ETI in this pathosystem.

2. Rice blast resistance genes and the distribution in the rice genome

More than 100 blast *R* genes have been mapped in the rice genome, and 35 of them have been cloned (Table 1). The majority of *R* genes encode NLR proteins. One exception is *Pi-d2,* which encodes a receptor-like kinase with an extracellular B lectin domain and an intracellular serine-threonine kinase domain. Interestingly, this structure is reminiscent of the PAMP recognition receptors involved in PTI responses. A second exceptional *R* gene is the recessive *pi21* gene that encodes a proline-rich protein with

Table 1 Cloned blast resistance (*R*) genes/alleles in rice

R gene	Encoding protein$^{1)}$	Chromosome	Donor	Reference
Pi37	NLR	1	St. No. 1	Lin *et al*. (2007)
Pit	NLR	1	K59	Hayashi and Yoshida (2009)
Pish	NLR	1	Nipponbare	Takahashi *et al*. (2010)
Pi35	NLR	1	Hokkai 188	Fukuoka *et al*. (2014)
Pi64	NLR	1	Yangmaogu	Ma *et al*. (2015)
Pi-b	NLR	2	Tohoku IL9	Wang *et al*. (1999)
pi21	Proline-rich metal binding protein	4	Owarihatamochi	Fukuoka *et al*. (2009)
Pi63/Pikahei-1(t)	NLR	4	Kahei	Xu *et al*. (2014)
Pi9	NLR	6	75-1-127	Qu *et al*. (2006)
Pi2	NLR	6	Jefferson	Zhou *et al*. (2006)
Piz-t	NLR	6	Zenith	Zhou *et al*. (2006)
Pi-d2	B lectin receptor kinase	6	Digu	Chen *et al*. (2006)
Pi-d3	NLR	6	Digu	Shang *et al*. (2009)
Pi25	NLR	6	Gumei2	Chen *et al*. (2011)
Pid3-A4	NLR	6	A4 (*Oryza rufipogon*)	Lü *et al*. (2013)
Pi50	NLR	6	Er-Ba-zhan (EBZ)	Zhu *et al*. (2012); Su *et al*. (2015)
Pigm	NLR	6	Gumei4	Deng *et al*. (2017)
Pi36	NLR	8	Kasalath	Liu *et al*. (2007)
Pi5	NLR	9	RIL260	Lee *et al*. (2009)
Pii	NLR	9	Hitomebore	Takagi *et al*. (2013)
Pi56	NLR	9	Sanhuangzhan No. 2	Liu *et al*. (2013)
Pi54	NLR	11	Tetep	Sharma *et al*. (2005, 2010)
Pikm	NLR	11	Tsuyuake	Ashikawa *et al*. (2008)
Pb1	NLR	11	Modan	Hayashi *et al*. (2010)
Pik	NLR	11	Kusabue	Zhai *et al*. (2011)
Pik-p	NLR	11	K60	Yuan *et al*. (2011)
Pia	NLR	11	Sasanishiki	Okuyama *et al*. (2011)
Pi1	NLR	11	C101LAC	Hua *et al*. (2012)
Pi54rh	NLR	11	*Oryza rhizomatis* (nrcpb 002)	Das *et al*. (2012)
Pi-CO39	NLR	11	CO39	Cesari *et al*. (2013)
Pi54of	NLR	11	*Oryza officinalis* (nrcpb004)	Devanna *et al*. (2014)
PiK-h	NLR	11	K3	Zhai *et al*. (2014)
Pike	NLR	11	Xiangzao143	Chen *et al*. (2015)
Piks	NLR	11	Unknown	GenBank: AET36547.1, AET36548.1
Pi-ta	NLR	12	Yashiro-mochi	Bryan *et al*. (2000)

1) NLR, nucleotide-binding leucine-rich repeat.