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Rice versus *Xanthomonas oryzae* pv. *oryzae*: a unique pathosystem

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Bacterial blight, caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*), is a devastating disease of rice worldwide. The qualitative or pathogen race-specific resistance to this pathogen conferred by major disease resistance (*MR*) genes has been widely used in rice improvement. Accumulating genetic and molecular data have revealed that the molecular mechanisms of rice qualitative resistance to *Xoo* are largely different from those of qualitative resistance in other plant–pathogen pathosystems. In this review, we focus on the unique features of rice qualitative resistance to *Xoo* based on *MR* genes that have been identified and characterized. The distinctiveness of the rice–*Xoo* interaction provides a unique pathosystem to elucidate the diverse molecular mechanisms in plant qualitative resistance.

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Introduction

A large number of microorganisms cause diseases in crops, which results in serious production loss worldwide each year. Plants have developed the ability to resist diseases during the long course of evolution. Genetically, plant disease resistance is classified into two types, qualitative or complete resistance and quantitative or partial resistance, based on the speed and strength of plant response to pathogen invasion [1]. Qualitative resistance can be conferred by a single major disease resistance (*MR*) gene and is mostly pathogen race-specific. Quantitative resistance is mediated by multiple genes or quantitative trait loci and is frequently pathogen species-nonspecific or race-nonspecific. Qualitative resistance has been widely used in crop improvement for its high level of resistance and easy manipulation. *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) causes devastating bacterial blight disease worldwide. Accumulating information suggests that the molecular mechanisms of rice qualitative resistance to *Xoo* are largely different from the resistance conferred by *MR* genes in other plant–pathogen pathosystems, although the mechanisms of plant qualitative resistance remain to be elucidated.

Molecular models of plant innate immunity

Plants resist pathogen invasion through a two-tiered innate immune system: pathogen-associated molecular pattern (PAMP)-triggered immunity (PTI) and effector-triggered immunity (ETI) [2]. In plant–pathogen interactions, pathogen-produced PAMPs, which are relatively conserved across genera or within a genus during evolution, or plant-derived damage-associated molecular patterns created by host peptides or cell wall fragments released during pathogen invasion [3••], are directly recognized by plasma membrane-localized plant pattern recognition receptors (PRRs) to initiate PTI. This type of defense response is frequently weak and pathogen species-nonspecific or race-nonspecific, and thus it is also called basal resistance [4••]. Successful pathogens can overcome PTI using effectors that they secrete into plant cells. Plants carrying resistance (R) proteins can initiate ETI by direct or indirect perception of specific effectors. ETI is pathogen race-specific and generally confers a high level of resistance, and thus it is also called race-specific or gene-for-gene resistance [5]. However, weak ETI and strong PTI have also been reported [4••]. Thus qualitative resistance can be mediated by either *R* genes functioning in ETI or *PRR* genes functioning in PTI. To avoid confusion, we use '*MR*' to indicate genes initiating qualitative resistance.

Common structural features of *MR* genes

Approximately 20 types of *MR* genes have been identified, and most of them share common structural features [6]. The majority of cloned dominant *MR* genes from both monocots and dicots encode cytoplasmic nucleotide-binding (NB)-leucine-rich repeat (LRR) proteins [7]. These genes mediate resistance to various types of pathogens including bacteria, fungi, oomycetes, viruses, and nematodes and even resistance to insects and parasitic plants [6,8,9]. NB-LRR proteins directly or indirectly interact with specific pathogen effectors secreted into host cells to initiate ETI [2,10,11]. A number of dominant *MR* genes for resistance to different fungi and nematode, which have only been characterized in dicots (tomato, sugarbeet, and crabapple), encode integral plasma

membrane proteins with an extracellular LRR domain and a transmembrane motif with or without cytoplasmic endocytosis signals. These LRR receptor-like proteins belong to PRR; some of them initiate PTI by recognizing fungal PAMP xylanase or peptide [3,12,13]. Cloned recessive *MR* genes for resistance to different viruses from various crops (rice, pepper, lettuce, pea, barley, tomato, and melon) all encode mutated translation initiation factors of the 4E and 4G families, which cannot be used by viruses to complete the steps of their infection cycle, but do not affect the initiation of host protein translation [14]. Thus a large number of *MR* genes only encode a few types of proteins in most of the well-studied plant–pathogen pathosystems, suggesting that the initiation of host qualitative resistance to different pathogens in these pathosystems appears to share similar mechanisms; the rice–*Xoo* pathosystem, however, is an exception.

Diversified mechanisms of rice qualitative resistance to *Xoo*

The uniqueness of rice–*Xoo* pathosystem arises from the following aspects. First, qualitative resistance to *Xoo* is an important type of defense response in rice. At least 37 *MR* genes for resistance to *Xoo* have been identified [15]. However, only a few *MR* genes, *Bs1–Bs4*, *bs5*, and *bs6*, which confer resistance to *Xanthomonas campestris* pv. *vesicatoria* causing bacterial spot in pepper and tomato, have been reported [16,17]. No *MR* gene against *X. oryzae* pv. *oryzicola* (*Xoc*), which causes bacterial streak disease, has been identified in rice, although maize Rxo1, a NB-LRR-type protein that confers resistance to *Burkholderia andropogonis*, the causal organism of bacterial stripe in maize, can mediate a hypersensitive response to *Xoc* in rice [18]. *MR* genes against the following *Xanthomonas* species are yet to be reported: *X. axonopodis* pv. *citri* and *X. fuscans* pv. *aurantifolii*, which cause canker diseases in citrus; *X. campestris* pv. *campestris* and *X. campestris* pv. *armoraciae*, which cause black rot and leaf spot diseases, respectively, in brassicaceae; *X. translucens* pv. *graminis* and *X. campestris* pv. *vasculorum*, which cause bacterial wilt and gumming diseases, respectively, in grasses; *X. axonopodis* pv. *glycines*, which causes bacterial pustule in soybean; *X. axonopodis* pv. *manihotis*, which causes bacterial blight in cassava; and *X. vasicola* (formerly *X. campestris* pv. *musacearum*), which causes *Xanthomonas* wilt in banana. However, the effectors from phytopathogenic *Xanthomonas* species have been extensively studied [19,20]. It is unclear whether *MR* gene-mediated resistance is simply not a major defense system or if more extensive genetic studies are necessary to identify *MR* genes in these pathosystems.

Second, it is common for *MR* gene-mediated resistance to *Xoo* to be recessively regulated. Fourteen of the 37 reported *MR* genes against *Xoo* are recessive in nature [15]. However, most of the characterized *MR* genes conferring resistance to bacteria, fungi, oomycetes, and nematodes act dominantly in different plant species [7]. Approximately 100 rice *MR* genes conferring resistance to *Magnaporthe oryzae*, which causes fungal blast disease worldwide, have been named and most of them function dominantly [21]. The exception is plant resistance to viruses; about half of the ~200 known virus resistance loci are recessively inherited [22].

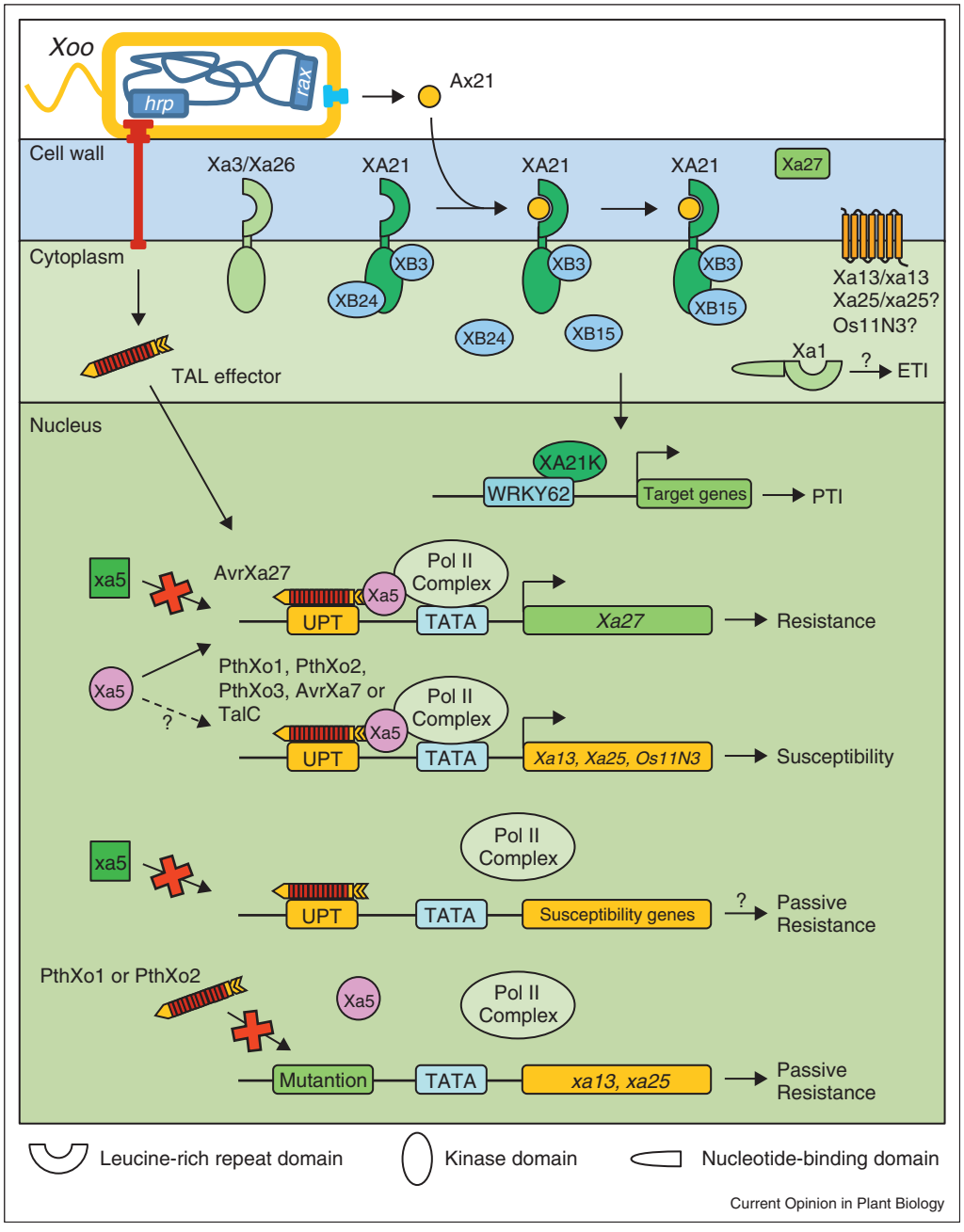
Last, rice qualitative resistance to *Xoo* appears to be regulated by diverse mechanisms. Although only seven *MR* genes (*Xa1*, *Xa3/Xa26*, *xa5*, *xa13*, *Xa21*, *xa25*, and *Xa27*) against *Xoo* have been cloned, these genes encode various types of proteins, indicating the functional diversity in rice–*Xoo* interactions. At least 19 *MR* genes against *M. oryzae* have been cloned; 18 of them encode NB-LRR-type proteins and one (*Pi-d2*) encodes a plasma membrane-localized lectin receptor kinase-type protein [21]. However, only one (*Xa1*) of the seven isolated *MR* genes against *Xoo* encodes an NB-LRR-type protein, although the rice genome contains 623–725 NB-LRR genes [23], suggesting that rice NB-LRR-type proteins play important roles in resistance to *M. oryzae* but not *Xoo*. The noncanonical *MR* genes regulate rice resistance to *Xoo* by different mechanisms.

LRR receptor kinase-type *MR* genes

LRR receptor kinases comprise the largest class of receptor kinase-type proteins in PTI [3••]. These proteins have an extracellular LRR domain, a transmembrane motif, and a cytoplasmic kinase domain. Although LRR receptor kinase-type genes have been extensively studied in plant–pathogen interactions, only rice *Xa21* and *Xa3/Xa26* against *Xoo* are reported to confer qualitative resistance in plants (Figure 1). Both genes regulate rice disease resistance dominantly. *Xa21* was first isolated as an *R* gene conferring race-specific resistance to *Xoo*, although it has a more broad-spectrum resistance than most of the identified *MR* genes against *Xoo* [24,25]. A sulfated peptide $axY^{S}22$ derived from Ax21 protein, which is secreted by *Xoo* through its type I secretion system, was found to trigger XA21-mediated resistance by binding to the LRR domain of XA21 [26]. Because this peptide is conserved in many *Xanthomonas* species and even outside the *Xanthomonas* genus, it is considered as a PAMP and XA21 is a PRR [26]. Thus, XA21 mediates a high level of PTI but with race specificity to *Xoo* [25,27].

Several XA21 binding (XB) proteins have been reported to be involved in the rice defense response against *Xoo* by interacting with XA21 *in vivo* (Figure 1). XB24/ATPase promotes autophosphorylation of XA21, which maintains an inactive status for XA21 [28]. Upon recognition of Ax21, XB24 dissociates from XA21, leading to activation of resistance [28]. XB3/E3 ubiquitin ligase is a substrate of XA21 kinase activity and is required for *Xa21*-mediated resistance [29]. XB15/protein phosphatase 2C can dephosphorylate XA21, which results in inactivation of XA21

Figure 1



Different major resistance proteins (Xa3/Xa26, XA21, Xa27, xa13, xa25, and xa5) function in diverse processes to confer resistance against *Xoo* in rice. *Os11N3* is a race-specific susceptibility gene. ETI, effector-triggered immunity; *hrp*, hypersensitive reaction and pathogenicity gene; Pol II, polymerase II; PTI, pathogen-associated molecular pattern-triggered immunity; *rax*, required for AvrXa21 gene; TAL, transcription activator-like; TATA, TATA box; UPT, upregulated by TAL effector; XA21K, kinase domain of XA21; question mark (?), remaining to be determined.

[30]. The transcriptional regulator XB10/WRKY62 negatively regulates *Xa21*-mediated resistance [31]. Cleavage of XA21 and translocalization of its kinase domain to the nucleus, where it interacts with WRKY62, is required for the *Xa21*-initiated defense response [32••]. These results suggest that XA21 may function not only as a receptor to initiate defense signaling but also as a direct regulator to control the activity of transcription factor in rice–*Xoo* interaction.

Xa3/Xa26, present in rice cultivars and hybrid rice lines that are widely planted in China, mediates a race-specific resistance to many strains of *Xoo* but with a resistance spectrum different from *Xa21* [27,33,34] (Figure 1). The

Xa3/Xa26 locus confers a durable resistance to *Xoo* [35•], suggesting that the Xa3/Xa26 protein may recognize not only a conserved but also a stable *Xoo* signature. Kinase and LRR domain swap analyses between Xa3/Xa26 and XA21 indicate that the defense signaling pathways initiated by the two proteins may partially overlap [27]. These results also suggest that Xa3/Xa26 may mediate PTI.

Several components, including positive regulators OsDR8 (thiamine synthesis-related), transcription factors WRKY13 and WRKY45-2, and C3H12 (a CCCH-type zinc finger nucleic acid-binding protein) and negative regulator OsDR10 (a rice tribe-specific protein), that play a role in the Xa3/Xa26-initiated defense transduction pathway have been identified [15]. Interestingly, these positive regulators are also involved in rice resistance to one or two other pathogens, *Xoc* and *M. oryzae*, in addition to the resistance against *Xoo* [15]. Thus, the Xa3/Xa26-mediated resistance pathway against *Xoo* partially overlaps or crosstalks with the defense signaling pathways against other pathogens.

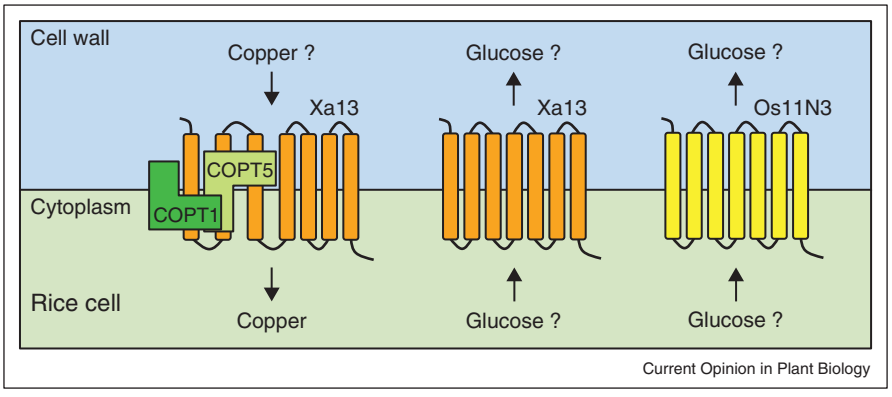
The functions of both *Xa21* and *Xa3/Xa26* are expressionally dose-dependent: as their expression increases, the plant's resistance increases. The expression of *Xa21* and *Xa3/Xa26* is developmentally regulated, and their expression increases gradually with development, which results in rice plants carrying any one of the two *MR* genes being able to mediate full resistance to *Xoo* only at the adult stage [27,36,37]. In addition, *Xa3/Xa26* expression is influenced by genetic background; a *japonica* background more readily facilitates its expression than an *indica* background [37].

MtN3/saliva-type *MR* genes

Two recessive *MR* genes, *xa13* and *xa25*, against *Xoo* belong to the MtN3/saliva multiple gene family [38]. This family encodes membrane proteins and is prevalent in eukaryotes. The only known structure of this protein family is the MtN3/saliva domain that was first identified in root nodulin-related protein of legume [39] and later in the saliva protein of *Drosophila* [40]. This domain is a pair of repeats each spanning two transmembrane helices connected by a loop, which is also named PQ loop repeat, based on the description of the Conserved Domain Database (http://www.ncb.nlm.nih.gov/cdd), but its biochemical function is unknown. Recently, a few MtN3/saliva proteins from animals and plants have been reported as sugar transporters [41••,42].

Rice plants carrying recessive *xa13* have specific resistance to *Xoo* strain PXO99 [43]. This recessive gene is widely used in rice breeding programs in south Asian counties such as India [44]. Promoter swapping analysis confirms that the dominant allele of this recessive gene, *Xa13* (also named *Os8N3* and *OsSWEET11*), is a susceptibility gene specific to PXO99, which secretes the transcription activator-like (TAL) effector PthXo1 [41••,45,46]. PthXol binds specifically to a *cis*-acting element, the UPT_{PthXol} box, in the promoter of dominant *Xa13* to induce its expression [47,48] (Figure 1). *Xa13* encodes a plasma membrane protein that interacts with two plasma membrane-localized copper transporter-type proteins, COPT1 and COPT5, to promote removal of copper from xylem vessels [49••] (Figure 2). Copper is an essential micronutrient of plants and is also an important element for a number of pesticides in agriculture. Copper inhibits *Xoo* growth, and PXO99 is more sensitive to copper than other *Xoo* strains [49••]. Rice transports copper from root to shoot through xylem vessels, where *Xoo* multiplies and spreads to cause disease. In addition, *Xa13* is required for pollen development [43]. Thus, PXO99 overcomes rice defenses by regulating host copper redistribution via transcriptional activation of the host gene that is essential for reproductive development. The

Fig. 2



The putative functions of Xa13 and Os11N3 proteins in the rice–*Xoo* interaction. Xa13, associated with copper transporter-type proteins, COPT1 and COPT5, to promote the removal of copper from xylem vessels. The copper is predicted to be transported into vascular cells by Xa13-COPT1/5 complex. Xa13 and Os11N3 may also function in glucose efflux.

resistance of rice plants carrying recessive *xa13* to *Xoo* is due to the mutation of UPT_{PthXol} box in *xa13* promoter, which results in PXO99 being unable to induce recessive *xa13* expression [43]. The copper level in the xylem vessels of rice plants carrying recessive *xa13* can inhibit PXO99 growth and plants have passive resistance to *Xoo* [49••].

Rice plants carrying recessive *xa25* show race-specific resistance to *Xoo* strain PXO339 [38]. The dominant *Xa25*, but not the recessive *xa25*, is transcriptionally induced by PXO339, but not other *Xoo* strains that show compatible interaction with rice plants carrying recessive *xa25* [38]. The promoter region of dominant *Xa25* harbors a predicted *cis*-acting element, the UPT_{PthXo2} box, which is proposed for the binding of PthXo2 effector secreted by the type III secretion system of *Xoo* [38,50,51••]. The UPT_{PthXo2} box of the promoter of recessive *xa25* [38] contains a mutation, suggesting that PXO339-induced expression of dominant *Xa25* may be via the UPT_{PthXo2} box (Figure 1). Rice COPT-type copper transporter family consists of seven members [52]. Although coexpression of Xa13, COPT1, and COPT5 is required for influx of copper in yeast, the other five rice COPT proteins can transport copper alone or by forming heterodimers in yeast [49,52]. Xa25 did not interact with COPT1 or COPT5 in yeast cells (M Yuan, S Wang, unpublished data). These results suggest that Xa25 protein may function differently from Xa13 in rice–*Xoo* interaction. Another difference is that PXO339-induced *Xa25* expression is affected by developmental stage. The dominant *Xa25* is more efficiently induced by PXO339 at the seedling stage than at the adult stage; this results in the recessive *xa25* having a feature of dominant reversal, in which the *MR* gene functions as a recessive gene in the seedling stage but as a dominant gene in the adult stage [38].

Another member of rice MtN3/saliva family, *Os11N3* (also named *OsSWEET14*), is also a race-specific susceptibility gene to *Xoo* [41••,53]. *Os11N3* is transcriptionally activated by *Xoo* strains carrying TAL effector AvrXa7, PthXo3, or TalC by binding these effectors to the corresponding UPT boxes in its promoter [53,54] (Figure 1). The Os11N3 and Xa13 proteins function as low-affinity glucose transporters in animal cell lines, which suggests that they may supply sugars to *Xoo* by an efflux mechanism [41••]. It is expected that a recessive *Os11N3* with a mutation in the UPT box of its promoter may have been or will be created by natural selection, just like rice varieties carrying recessive *xa13* and *xa25*. However, an artificial rice mutant in which the UPT_{PthXo3} box is site-mutated has been shown to have qualitative resistance to *Xoo* strains carrying AvrXa7 or PthXo3 [55].

Other types of *MR* genes

The dominant *Xa1* is the only NB-LRR-type *MR* gene isolated so far for resistance to *Xoo* [56] (Figure 1). It confers race-specific resistance to *Xoo* strain T7174. Unlike most of the examined NB-LRR-type *MR* genes, which have constitutive expression patterns, the expression of *Xa1* is induced by both *Xoo* and wounding [56]. Because no other studies have been reported about *Xa1*, it can only be speculated that *Xa1* functions in a typical ETI process.

Dominant *Xa27*-mediated race-specific resistance to *Xoo* depends on its transcriptional activation by AvrXa27, a TAL effector of *Xoo* [57]. The dominant and recessive alleles of *Xa27* encode an identical apoplast protein with no similarity to any known proteins, and they differ from each other only in the promoter regions [57,58]. A *cis*-acting element, the $UPT_{AvrXa27}$ box, for the specific binding of AvrXa27 in *Xa27* promoter is responsible for the *Xoo*-induced expression of *Xa27* [59]. Thus rice resistance to *Xoo* is through employing the UPT box of AvrXa27 in the promoter of *Xa27* (Figure 1). *Xa27*-mediated resistance is associated with obvious secondary cell-wall thickening in vascular bundle elements [57]. According to the molecular models of plant innate immunity, PTI is induced by plasma membrane-localized PRRs and ETI is initiated by cytoplasmic NB-LRR-type R proteins [2,3••,10]. The subcellular localization and sequence specificity of Xa27 protein suggest that it does not appear to initiate defense responses by either a PTI or ETI procedure, although the biochemical function of this protein in rice–*Xoo* interaction remains to be elucidated.

The *xa5*, conferring a race-specific resistance to *Xoo* recessively, encodes a mutated gamma subunit of the basal transcription factor IIA 5 (TFIIAγ5) [60,61]. The TFIIAγ5 encoded by the dominant *Xa5* is predicted to cooperate with TAL effectors of *Xoo* to induce the expression of host susceptibility genes for facilitation of *Xoo* invasion; whereas the mutated TFIIAγ5 encoded by the recessive *xa5* may attenuate TAL effector-activated host gene expression, resulting in passive resistance [62] (Figure 1). This inference is supported by evidence that TFIIAγ5 facilitates the transcriptional activation of Xa27 by AvrXa27 [63]. Pyramiding recessive *xa5* with dominant *Xa27* causes attenuation in the induction of *Xa27*, which leads to susceptibility of rice plants to incompatible *Xoo* strains. On the basis of above results and inference, the dominant *Xa5* may function as a susceptibility gene in the absence of *Xoo* TAL effector-induced *MR* gene, but the recessive *xa5* could also function as a susceptibility gene in the presence of *Xoo* TAL effector-induced *MR* gene.

Conclusions and perspectives

On the basis of the published genetic and molecular data, rice qualitative resistance to *Xoo* appears to be mediated largely by uncommon *MR* genes. Mutation of race-specific susceptibility genes contributes to an important aspect of this type of resistance. In addition, one or more of the PRR-type genes are also involved in the qualitative

resistance. Unlike the qualitative resistance conferred by NB-LRR-type *MR* genes in other plant–pathogen pathosystems, transcriptional activation or suppression of *MR* genes is an important way of regulating rice qualitative resistance to *Xoo*. This transcriptional regulation is commonly regulated by the binding of *Xoo* TAL effectors to the promoters of these *MR* genes or their susceptible alleles. Beyond the diverse protein structures and confirmed or predicted biochemical functions of these MR proteins, rice and *Xoo* provide a unique pathosystem to study the molecular mechanisms of plant–pathogen interactions. In addition to continuing to characterize the nature of *MR* genes against *Xoo*, further studies may focus on whether the unique feature of rice qualitative resistance to *Xoo* is because the TAL effectors of *Xoo* are important players in this type of host–pathogen interactions. It also needs to be concerned why the large number of NB-LRR-type genes in rice genome is not efficiently used in qualitative resistance to *Xoo* and whether this is at least partly due to the fact that most abundant NB-LRR genes do not have conserved binding motifs in their promoters for the *Xoo* effectors. The pathogenic rice bacterium *Xoc* is evolutionarily closely related to *Xoo*. It is also worth exploring the major differences between rice–*Xoc* and rice–*Xoo* systems to understand why no rice qualitative resistance to *Xoc* has been identified.

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