

EDITORIAL

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The XA48–OsVOZ1 immune module: from asymmetric domestication to PTI–ETI pyramiding for yield-penalty-free resistance

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Abstract

To advance modern crop breeding by de novo domestication, Hui Lin and colleagues recently characterized an immune module in rice subspecies *Oryza sativa* subsp. *indica* (*OSI*) and *O. sativa* subsp. *japonica* (*OSJ*). The module involves rice resistance protein XA48 and the downstream transcription factor OsVOZ1. Carrying an alanine (A) to serine (S) variation, OsVOZ1 functions redundantly with OsVOZ2 in suppressing rice resistance to bacterial blight, a worldwide significant crop disease caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*). XA48, identified in an *OSI* cultivar, recognizes the *Xoo* effector XopG, confers resistance and susceptibility to XopG-harboring *Xoo* strains from Northeast and South Asia, respectively, and activates effector-triggered immunity (ETI) by degrading OsVOZ1/2. In resistance spectra, this ETI complements pattern-triggered immunity (PTI) mediated by XA21, a resistance protein previously identified in wild rice. The Xa48–OsVOZ1 module has undergone asymmetric selection: Xa48 is retained in *OSI* and lost in *OSJ*, whereas *OSI* retains both OsVOZ1^{AS} alleles and *OSJ* retains only OsVOZ1^A. Although restoring XA48 to *OSJ* severely reduces grain yield, integrating XA48/ETI and XA21/PTI into modern *OSI* and *OSJ* cultivars rebuilds broad-spectrum resistance without a yield penalty, opening an avenue toward modern crop breeding using immunity sources from wild rice.

Keywords Rice bacterial blight, Immune module (XA48–OsVOZ1), Asymmetric selection, PTI–ETI (XA21–XA48) integration, Broad-spectrum resistance, Growth–defense balance

As a major event in human civilization, wild plants were successfully domesticated into crops by continuing artificial selection (Meyer et al. 2012; Yu et al. 2021; Huang et al. 2022). Artificial selection has greatly shaped agronomic traits while confronting a dilemma

of growth–defense tradeoffs, shown as the development of disease resistance at the cost of yield loss (Fornasiero et al. 2022; He et al. 2022). Rice is a major staple crop and a model system for using genetic resources from wild species to improve agronomic traits, particularly disease resistance (Long et al. 2024; Zheng et al. 2024). Exactly, the first cloned *Xanthomonas* resistance (*Xa*) gene, *Xa21*, originates from the wild rice species *O. longistaminata* (Song et al. 1995). *Xa21* encodes a leucine-rich repeat receptor-like kinase (LRR–RLK) and confers race-specific pattern-triggered immunity (PTI) against the Philippine *Xoo* strain PXO99A but is ineffective against *Xoo* strains from Northeast Asia (Song et al. 1995; Pruitt et al. 2015; Chen et al. 2021). To date, at least 46 *Xa* genes have been identified; among them, 12 *Xas* with different resistance

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spectra have been identified in wild rice, while 44 *Xas* have been retained in landraces or modern crop cultivars following artificial selection during domestication (Tauqeer et al. 2025; Lin et al. 2026). The mechanistic basis of resistance selection and its utility in modern crop breeding to balance growth-defense tradeoffs remain unclear (Fernie and Yan 2019; Jian et al. 2023; Yoshikawa and Sato 2025). Lin et al. (2026) recently answered both questions by identifying *Xa48*, a novel *Xa* gene that complements *Xa21* in resistance spectrum, and by establishing a genetic strategy to combine *XA21* and *XA48* for enhanced *Xoo* resistance without a yield penalty.

The study on the *Xa48* locus is a sustained endeavor. *Xa* genes are designated sequentially with Arabic numerals, and *Xa48* represents the 48th reported member, following *Xa47(t)* (Xing et al. 2021) and *Xa47* (Lu et al. 2022) characterized by our colleagues. Four years ago, *Xa48* was coded as *Xa47* and predicted to encode a coiled-coil nucleotide-binding and leucine-rich repeat (CC-NLR) protein that might form a resistosome (Xu et al. 2022). To clone *Xa48*, Lin et al. (2026) compared the *OSI* variety Shuangkeza0 (SKZ), which displays strong resistance to the Korean *Xoo* strain J18 but susceptibility to PXO99A, with the *Xa21*-carrying *OSJ* cultivar TP309, which exhibits the reciprocal resistance pattern (Chen et al. 2021). Through crosses of SKZ with the susceptible *OSJ* cultivar Nipponbare (NIPB), the authors mapped the *Xa48* locus and functionally validated its *Xoo* resistance via genome-wide association study and gene overexpression (OE) and editing. *Xa48* mediates resistance to Northeast Asia *Xoo* strains J18, LN2, LN4, and LN18, though not to PXO99A (Lin et al. 2026). Notably, *Xa48* confers resistance throughout the plant life cycle (Lin et al. 2026), unlike most *Xa* genes that typically confer resistance only at the adult stage (Sha et al. 2005; Yang et al. 2022; Wang et al. 2025).

Xa48 is functionally distinct from most of the 45 *XA* proteins reported to date (Tauqeer et al. 2025; Jiang et al. 2020, 2026; Lin et al. 2026). *XA1*, *XA2**, *XA14**, *XA31(t)**, *XA45(t)**, and *XA47* are proteins that carry nucleotide-binding and leucine-rich repeats (NLR); *XA3/XA6/XA26* and *XA21* are LRR-RLKs; *XA4* is a cell wall-associated kinase; *XA5* is a TFIIA γ transcription factor; *Xa7*, *Xa10*, *XA23*, and *XA27* belong to the resistance executors; *XA13*, *XA25*, and *XaA41* are SWEET transporters; *XA46(t)* is a Ser/Thr protein kinase. Notably, all of these function in response to the bacterial transcription activator-like effectors (TALEs) (Tauqeer et al. 2025; Jiang et al. 2026). By contrast, *XA48* recognizes the bacterial effector XopG, a member of the *Xanthomonas* outer protein (Xop) family (Lin et al. 2026). While both Xops and TALEs constitute the type III effector repertoire of *Xanthomonas*, the functional mechanisms of Xops remain

less understood than those of TALEs (White et al. 2009; Li et al. 2026).

XA48 is a typical NLR that forms an ion transport channel permeable to Ca^{2+} , Mg^{2+} , K^{+} , and Na^{+} (Lin et al. 2026). This is consistent with findings that several other NLR proteins interact with pathogen effectors to form resistosomes functioning as Ca^{2+} channels (Wang et al. 2019; Bi et al. 2021; Jacob et al. 2021; Förderer et al. 2022; Liu et al. 2024; Wu et al. 2024). Upon recognition of XopG in J18-inoculated plants, *XA48* mediates Ca^{2+} influx, thereby activating immune responses and cell death (Lin et al. 2026; Fig. 1a). Such hypersensitive cell death often accompanies disease resistance (Chen et al. 2025a, b; Zhang et al. 2026). These defense responses are associated with altered expression of genes involved in salicylic acid (SA), jasmonic acid (JA), and ethylene signaling. Notably, *Xoo* infection does not alter the *in planta* levels of these three hormones from their high basal states; hence, immunity appears to be regulated by hormone signaling pathways rather than by changes in their concentrations (Lin et al. 2026; Fig. 1a).

XopG is an ancient bacterial effector conserved across plant and animal pathogens, whose function depends on a peptidase domain containing a conserved HEXXH motif (Lin et al. 2026). XopG displays *in vitro* peptidase activity but does not cleave *XA48* *in vivo*. Instead, XopG promotes *XA48* oligomerization, which is required for immune activation. Phylogenetic analysis of 38 XopG orthologs revealed three clades; however, only proteins from clades I and II confer avirulence to PXO99A on *Xa48*-carrying rice and interact with *XA48* coiled-coil (CC) domain. This interaction relies on the conserved XopG residues (Ser155 and Ala157), which are required for PXO99A to trigger *XA48*-mediated resistance. Resistance activation additionally requires His142, Glu143, and His146, and mutations at these positions (H142R, E143A, and H146R) render XopG prone to self-cleavage.

Among 76 genome-sequenced *Xoo* strains, XopG orthologs exhibit five structural types. Type I has full-length canonical XopG, and other types either lack the HEXXH motif or are truncated. Northeast Asian strains LN2, LN4, and LN18 contain type I XopG and are avirulent to *Xa48*-carrying rice populations (cultivars, landraces, and wild species), while virulent strains PXO99A and JL28 carry type V XopG. Type I strains originate from Northeast Asia, and strains carrying mutated XopG forms mainly distribute in South Asia. Based on this geographic-structural correlation, the authors propose that the prolonged arms race between *XA48* and XopG has likely exerted selective pressure on *Xa48* evolution (Lin et al. 2026).

Evidence from multiple assays demonstrated the functional interplay among XopG, *XA48*, and OsVOZ1/2

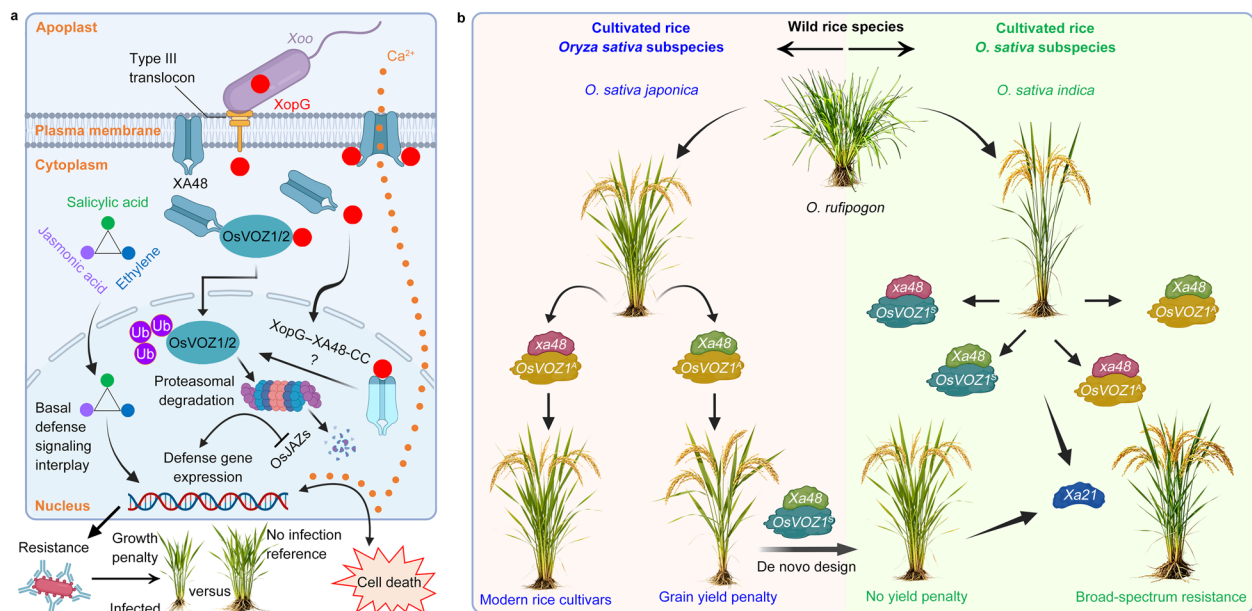


Fig. 1 Subspecies-specific selection of the XA48–OsVOZ1 module and its de novo design for broad-spectrum resistance without yield penalty in rice. **a** The functional network of XopG, XA48, and OsVOZ1/2 consists of three interconnected immune pathways. First, XA48 recognizes translocated XopG and activates Ca^{2+} signaling. Second, the XA48–XopG complex facilitates proteasome-dependent degradation of OsVOZ1/2, derepressing immunity regulators that are otherwise suppressed by OsVOZ1/2-mediated activation of jasmonic acid receptors (OsJAZs). Third, interplay among salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) is required for both basal defense and PTI. These pathways cooperate to confer Xoo resistance and cell death with a growth penalty in rice. Furthermore, XopG binds to XA48–CC in the nucleus, suggesting the possibility of the protein translocation for the interaction to occur and to promote OsVOZ1/2 degradation. **b** De novo design of the immune module based on asymmetric subspecies selection. The wild progenitor *O. rufipogon* gave rise to two cultivated subspecies: *O. s. japonica* (OSJ) and *O. s. indica* (OSI), which harbor the *OsVOZ1^{A/S}* variation. *OSJ* inherits only *OsVOZ1^A* while *OSI* retains both alleles. The *Xa48*–*OsVOZ1^A* combination causes reproductive penalty in *OSJ* but not in *OSI*, leading to *Xa48* functional loss in *OSJ*. *Xa48* has been retained in *OSI* with either the *OsVOZ1^A* or *OsVOZ1^S* allele, historically cultivated in Southeast Asia, where Xoo outbreaks are frequent. De novo introduction of *Xa48*–*OsVOZ1^S* into modern OSJ cultivars achieves both high yield and enhanced resistance, eliminating the reproductive penalty. Integrating the XA48-mediated ETI and XA21-mediated PTI in modern cultivars of either OSI or OSJ reconstitutes broad-spectrum resistance

(Lin et al. 2026). XA48 interacts with OsVOZ1/2, which directly binds both full-length XA48 and its CC domain. XA48 and OsVOZ1/2 localize to the cell periphery and nucleus independently, yet co-localize at these compartments. Upon XopG perception, the XA48–XopG complex assembles without disrupting the co-localization of OsVOZ1/2 with XA48, and promotes the proteasome-dependent degradation of OsVOZ1/2 in the nucleus. Degradation of OsVOZ1/2 relieves suppression of XA48-mediated ETI, XA21-mediated PTI, and basal defense against Xoo (Fig. 1a).

Lin et al. (2026) provided two lines of evidence to elucidate the subspecies-specific asymmetric selection of *Xa48*. First, they investigated the resistance of 1945 accessions against J18 and found that 405 (20.8%) were resistant. Among these resistant accessions, 145 (35.8%) carried a functional *Xa48* allele, whereas the rest harbored loss-of-function mutations, suggesting the involvement of alternative resistance loci. Notably, all rice accessions with a functional *Xa48* allele were exclusively OSI. Second, the authors analyzed *Xa48* sequences in

2451 Asian rice accessions, including wild rice, surveyed GWAS peak single-nucleotide polymorphisms, and determined the dynamic frequency changes of the functional *Xa48* allele during rice domestication and breeding. In the wild rice species *O. rufipogon*, the functional allele frequency is moderate (9.8%). In OSI, its frequency increases from 7.8% in landraces to 24.9% in cultivars. By contrast, in temperate OSJ, the functional allele declined from a rare frequency in landraces (1.0%) to trace (0.3%) levels in improved populations. These data demonstrate that *Xa48* underwent positive selection in OSI but negative selection in OSJ.

A similar selection has been applied to the *OsVOZ1^{A/S}* variation, which in turn dictates the growth penalty associated with the XA48-mediated immunity (Lin et al. 2026; Fig. 1b). The allele frequency of *OsVOZ1^S* is highly differentiated between OSI and OSJ: it occurs at 5.0% in wild rice, increases to 38.6% during OSI domestication, and rises to 85.3% in modern OSI cultivars, but is rare in OSJ (0.3%), which instead carries *OsVOZ1^A*. By contrast, the *Xa48* and *OsVOZ1^S*

are nearly absent from *OSJ*. Notably, the combined frequency of the *Xa48-OsVOZ1^S* alleles increases from 5.1% in landraces to 20.8% in modern *OSI* cultivars, suggesting co-selection of the *XA48-OsVOZ1* module in *OSI*.

Both *OsVOZ1^A* and *OsVOZ1^S* interact with *XA48*. Among 70 randomly tested *OSI* accessions stratified into four genotypic categories (*OsVOZ1^S* with *Xa48*, *OsVOZ1^S* without *Xa48*, *OsVOZ1^A* with *Xa48*, and *OsVOZ1^A* without *Xa48*), seed-setting rates are similar, indicating that the A/S variation itself does not affect the *OsVOZ1-XA48* interaction or seed setting. Similarly, crosses of SKZ (*OSI* harboring *OsVOZ1^S*) with Kasalath (*OSI* harboring *OsVOZ1^A*) and with two other *OSI* varieties, 9311 and TN1 (carrying *OsVOZ1^S* and the mutated *Xa48*), showed no significant differences in seed setting. These findings suggest that the *XA48*-mediated immunity is compatible with either *OsVOZ1* allele in the *OSI* genetic background. By contrast, the *XA48-OsVOZ1^A* combination severely compromises seed setting in *OSJ*.

In *OSJ* NIPB, *OsVOZ1^A*-OE and *OsVOZ1^S*-OE lines exhibited comparable seed-setting rates but weakened resistance. When crossed with NIPB-*Xa48*, both OE hybrids displayed attenuated *XA48*-mediated resistance; however, *OsVOZ1^A*-OE/NIPB-*Xa48* showed reduced seed setting relative to NIPB, whereas *OsVOZ1^S*-OE/NIPB-*Xa48* restored normal seed setting. These results demonstrate that the *XA48-OsVOZ1^A* module specifically impairs reproductive fitness in *OSJ* (Fig. 1b).

In addition, *OsVOZ1/2* contributes to cold tolerance. *OsVOZ1^S*-OE/NIPB is more sensitive to cold stress than *OsVOZ1^A*-OE/NIPB or NIPB, whereas *OsVOZ1/2*-KO lines show enhanced cold sensitivity relative to NIPB. *OsVOZ1^S* undergoes self-oligomerization, a property absent in *OsVOZ1^A*, which may explain its greater role in cold tolerance (Lin et al. 2026).

Based on these findings, the authors established the genetic strategy for pyramiding *XA48*-mediated ETI and *XA21*-mediated PTI to achieve broad-spectrum resistance in modern rice breeding, applicable to both hybrid and inbred lines (Lin et al. 2026). Progeny of the hybrid between TP309-*Xa21* and TP309-*Xa48* displayed broad-spectrum resistance to 30 strains avirulent to either *Xa21* or *Xa48* (Fig. 1b). These hybrid progeny also exhibited enhanced resistance to four strains virulent to either *Xa48* or *Xa21*, along with enhanced expression of *PR* genes, markers of the SA signaling and basal defense with crosstalk to ethylene and JA pathways (Fig. 1a). Notably, transgenic *Xa21Xa48* rice lines achieved a high-level *Xoo* resistance under natural field conditions, even after flooding. The inbred *Xa21Xa48* line derived from the cross between SKZ and the *Xa21*-carrying *OSI* variety BG139

has been widely used in breeding programs. Furthermore, de novo *OSJ Xa48-OsVOZ1^S* lines conferred resistance to J18 without a yield penalty (Fig. 1b).

In conclusion, the functional network of XopG, *XA48*, and *OsVOZ1/2* (Fig. 1a) and the asymmetric selection of the *XA48-OsVOZ1* module underlie the genetic strategy of integrating PTI and ETI into modern rice breeding to rebuild broad spectrum resistance (Fig. 1b). This resistance arises from three integrated mechanisms: (i) the *XA48-XopG* complex-mediated degradation of *OsVOZ1/2*, which derepress defense regulators; (ii) the NLR-type ion channel activity of *XA48* in mediating Ca^{2+} influx, thereby activating cell death and resistance; and (iii) the signaling interplay among SA, JA, and ethylene essential for ETI and basal defense (Fig. 1a).

Thus, beyond its significance for modern crop breeding via de novo domestication (Fernie and Yan 2019; He et al. 2022), the PTI-ETI integration strategy (Fig. 1b) offers valuable heuristic insights for future research in two directions. One is the utility of resistosomes assembled by plant NLR proteins that function as a Ca^{2+} channels (Wang et al. 2019; Bi et al. 2021; Jacob et al. 2021; Förderer et al. 2022; Liu et al. 2024; Wu et al. 2024). Another is to counter recently demonstrated pathogenicity mechanisms involving nutrient manipulation (Wang et al. 2025) and alternative splicing (Chen et al. 2025a, b; Xu and Zhou 2025). Further studies are needed to characterize how *XA48* executes its dual functions, as a Ca^{2+} channel and as a facilitator of *OsVOZ1/2* degradation, in response to XopG. It also remains to be determined whether *XA48* forms a resistosome as hypothesized (Xu et al. 2022) and whether any non-NLR proteins can form Ca^{2+} channels analogous to NLR-derived channels.

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Author contributions

XC, SX, and HD originated the concept for the article, carried out the literature search, and XC drafted the manuscript. XC and HD designed the figure and crafted the figure legend. SX inspected the BioRender-created rice plant images. XC and SX provided feedback and revisions to the manuscript. HD and XC finalized the manuscript.

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Data availability

The BioRender-created graphs and rice plant images are available at request.

Declarations

Ethics approval and consent to participate

Not applicable.

Competing interests

The authors declare no competing financial interests.

Consent for publication

Not applicable.

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