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Editorial

Rice susceptibility Genes: Major targets of *Xanthomonas oryzae* pv. *oryzae* to circumvent *R* gene–mediated resistance

Bacterial blight (BB) of rice, caused by the gram-negative bacterium *Xanthomonas oryzae* pv. *oryzae (Xoo)*, continues to pose a formidable threat to global rice production. Rice feeds more than half of the world's population, and any disruption in its productivity has direct consequences for food security, farmer livelihoods, and socio-economic stability, particularly in Asia. Despite decades of breeding efforts and the deployment of resistant cultivars, BB remains persistent and, in many regions, resurging due to the remarkable evolutionary adaptability of the pathogen. A central reason for this persistence lies in the pathogen's ability to subvert host immunity by exploiting rice susceptibility (*S*) genes, thereby undermining classical *R* gene–mediated resistance.

In contrast to the traditional view of plant–pathogen interactions as a battle between resistance (*R*) genes in the host and avirulence (*Avr*) genes in the pathogen, recent discoveries have shifted attention toward susceptibility genes as critical determinants of disease outcome. Rather than merely evading or suppressing host defenses, *Xoo* actively reprograms host cellular processes to create an environment conducive to bacterial proliferation. Among the best-characterized and most influential susceptibility targets are the rice *SWEET* (Sugars Will Eventually be Exported Transporters) genes, which encode sugar efflux transporters that play essential roles in plant growth, development, and carbon allocation.

TALEs as Molecular Weapons: Precision Targeting of Host Susceptibility Genes

Xoo employs a highly sophisticated infection strategy centered on the delivery of transcription activator-like effectors (TALEs) into rice cells via the type III secretion system (T3SS). The T3SS is a syringe-like molecular apparatus conserved among many gram negative plant and animal pathogens. In *Xoo*, this system is encoded by clusters of *hrp* (hypersensitive response and pathogenicity) and *hrc* (HR and conserved) genes, which collectively orchestrate the secretion, translocation, and functional deployment of a diverse repertoire of effector proteins.Once injected into the host cell cytoplasm, TALEs rapidly translocate to the nucleus, where they function as transcriptional activators. Unlike most bacterial effectors that interact with host proteins, TALEs directly bind to host DNA. This unique capability allows *Xoo* to hijack the host transcriptional machinery with extraordinary specificity and efficiency. Each TALE recognizes a distinct DNA sequence, known as the effector-binding element (EBE), located in the promoter regions of target genes.The specificity of TALE–DNA interactions is dictated by the **repeat-variable di-residues** (RVDs) present in the central repeat region of the TALE protein. Each repeat typically corresponds to one nucleotide in the target DNA sequence, establishing a simple and predictable code. This modularity endows Xoo with immense evolutionary flexibility, enabling it to rapidly generate new TALE variants capable of targeting alternative host genes or promoter alleles.

OsSWEET Genes: Central Nodes of Pathogen Exploitation

The rice genome encodes 21 *SWEET* genes, which collectively mediate sugar transport across cellular membranes, including phloem loading, seed filling, pollen nutrition, and responses to abiotic stresses. However, only a subset of these genes has been repeatedly and independently co-opted by *Xoo* during infection. Chief among them are*OsSWEET11 (Xa13/Os8N3), OsSWEET13 (Xa25),* and *OsSWEET14 (Os11N3)*.

OsSWEET11 (Xa13): *The Prototypical Susceptibility Gene*

OsSWEET11 was the first susceptibility gene identified in the rice–*Xoo* pathosystem and remains the most extensively studied. It plays a crucial role in sucrose transport and is normally expressed in vascular tissues. During *Xoo* infection, TALEs such as AvrXa7 and PthXa1 bind to EBEs in the *OsSWEET11* promoter, leading to its ectopic and elevated expression. The resulting efflux of sucrose into the apoplast provides an abundant carbon source that fuels bacterial multiplication and systemic colonization.

Natural mutations in the *OsSWEET11* promoter, collectively referred to as ***xa13***, disrupt TALE binding without impairing the physiological function of the gene. These promoter variants confer **recessive but durable resistance,** illustrating how subtle regulatory changes can decisively alter disease outcomes.

OsSWEET13 and OsSWEET14: Redundant but Critical Targets

As rice breeding programs increasingly deployed *xa13*-containing cultivars, *Xoo* populations adapted by evolving TALEs that target alternative *SWEET* genes. *OsSWEET13 (Xa25*) and *OsSWEET14* emerged as prominent secondary targets. TALEs such as PthXa2, TalC, and Tal5 specifically recognize EBEs in their promoters, ensuring continued access to host-derived sugars even when OsSWEET11 induction is blocked.This functional redundancy among SWEET genes underscores a key challenge in resistance breeding: blocking a single susceptibility pathway may be insufficient for durable protection. The pathogen's ability to shift its effector repertoire highlights the need for multi-target strategies that address the broader susceptibility network.

Structural and Functional Architecture of TALEs

TALEs are modular proteins composed of four major domains, each contributing to their remarkable functionality:

1. **N-terminal T3SS Secretion Signal:** This region mediates recognition by the T3SS machinery and is essential for efficient translocation into host cells.
2. **Central Repeat Region:**Comprising tandem repeats of ~34 amino acids, this region contains the RVDs that specify DNA-binding specificity. Minor changes in RVD composition can redirect TALEs to new host targets.
3. **Nuclear Localization Signals (NLSs):** These sequences ensure efficient import of TALEs into the host nucleus, where transcriptional activation occurs.
4. **C-terminal Acidic Activation Domain:** This domain recruits host transcriptional machinery, leading to robust induction of target gene expression.

Prominent TALEs such as AvrXa7, PthXa1, PthXa2, PthXa3, Tal5, and TalC exemplify how variations within this modular framework allow *Xoo* to fine-tune host gene activation across diverse rice genotypes.

Host Countermeasures: Executor Genes and Broad-Spectrum Resistance

Rice has not remained a passive victim in this evolutionary arms race. In addition to classical NLR-type *R genes*, rice has evolved executor (*E*) genes, which function as molecular traps. These genes contain EBEs in their promoters, allowing them to be activated by specific TALEs. Upon induction, executor genes **trigger rapid and strong defense responses**, often culminating in localized cell death that restricts pathogen spread.

The ***Xa1*** gene represents another innovative resistance strategy. Xa1 confers resistance against a wide range of typical TALEs (tTALEs) by recognizing conserved features of these effectors. However, this defense is not foolproof.

Pathogen Evasion: The Role of iTALEs

To overcome executor- and *Xa1*-mediated resistance, Xoo has evolved incomplete or truncated TALEs (iTALEs or pseudoTALEs). These effectors lack functional activation domains but retain nuclear localization signals and DNA-binding capacity. By occupying EBEs or interfering with TALE recognition, **iTALEs effectively suppress the activation of executor genes and neutralize Xa1-mediated resistance.**

The model strain PXO99A exemplifies this strategy, carrying multiple iTALEs such as Tal2h, Tal3a, and Tal3b. The widespread occurrence of iTALEs in virulent *Xoo* populations highlights the dynamic co-evolution between pathogen virulence strategies and host immune systems.

Natural and Engineered Resistance Through Promoter Variation

One of the most significant insights from SWEET gene research is that resistance can arise from promoter polymorphisms rather than changes in protein-coding sequences. Alleles such as *xa13, xa25*, and *xa41* disrupt

TALE binding while preserving the endogenous roles of *SWEET* genes in plant physiology. These naturally occurring variants provide durable resistance with minimal fitness costs.

Building on this principle, modern genome editing technologies have revolutionized resistance breeding. CRISPR–Cas9, TALENs, and zinc-finger nucleases enable precise modification of EBEs within *SWEET* promoters. By editing one or multiple EBEs simultaneously, it is now possible to generate rice lines resistant to diverse *Xoo* strains without introducing foreign DNA.

Susceptibility-Gene Editing: A Paradigm Shift in Resistance Breeding

Traditional resistance breeding has relied heavily on *R gene* pyramiding, which often provides short-lived protection due to rapid pathogen adaptation. In contrast, susceptibility-gene editing targets the host factors that pathogens depend upon, thereby imposing a higher evolutionary barrier to resistance breakdown.

CRISPR–Cas9, in particular, offers unparalleled precision and scalability. Multiplex editing of *OsSWEET11, OsSWEET13*, and *OsSWEET14* EBEs has already demonstrated broad-spectrum and durable resistance under field conditions. Importantly, such edits can mimic naturally occurring alleles, facilitating regulatory acceptance and public trust.

Conclusions and Future Perspectives

The interaction between rice and *Xanthomonas oryzae* pv. *oryzae* epitomizes the complexity and elegance of plant–microbe co-evolution. At the heart of this interaction lie the *OsSWEET* susceptibility genes, which serve as pivotal battlegrounds where pathogen virulence and host defense converge. *Xoo* has refined the art of host manipulation through TALEs, while rice has countered with executor genes, promoter polymorphisms, and broad-spectrum resistance mechanisms.

The growing understanding of susceptibility genes has fundamentally reshaped resistance breeding strategies. By shifting the focus from pathogen recognition to pathogen dependence, researchers and breeders now have powerful tools to achieve durable disease control. Genome editing technologies, particularly CRISPR–Cas9, stand at the forefront of this transformation, offering unprecedented opportunities to engineer resilience into one of the world's most important crops.

As we move forward, integrating susceptibility-gene editing with conventional breeding, genomic selection, and agroecological practices will be essential for sustainable rice production. Continued surveillance of *Xoo* populations, coupled with functional genomics and systems biology approaches, will further refine our ability to stay ahead in this enduring battle between host and pathogen.

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