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The Rice RBRL Protein: The Hub of a Triple Antivirus Network

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1. Introduction

In the context of escalating global climate change and the myriad challenges to food security, it is a necessity for rice—a staple for over half of the world's population—to have stable, high crop yields. However, rice virus diseases, including rice streak virus (RSV) and rice dwarf virus (RDV), present significant threats. This is particularly the case in Asia and Africa, where regional outbreak frequencies exceed 30% annually, resulting in global annual losses surpassing 15 billion USD and establishing these diseases as a substantial biological constraint on production. Traditional breeding strategies aimed at achieving disease resistance rely on natural mutation screening and are often protracted; thus, they may inadequately address the rapid evolution of viruses. Consequently, elucidating the molecular mechanisms underlying resistance has emerged as a critical area of focus. A recent study by Huang et al. [1] systematically analyzed rice antiviral resistance through a novel triadic network encompassing ubiquitination, hormone signaling, and RNA interference (RNAi), establishing a new paradigm for research on crop antiviral resilience.

This article endeavors to provide a critical synthesis of the findings presented by Huang et al. [1], situating the RING1–IBR–RING2-type ubiquitin ligase (RBRL)-centered triple network within the comprehensive framework of plant immunity. The researchers' study delineates a crucial antiviral signaling axis in which recognition of the RSV coat protein (CP) by RBRL activates the jasmonic acid (JA) pathway, resulting in the upregulation and increased activity of the RNAi effector *Oryza sativa* argonaute 18 (OsAGO18). This connection exemplifies a highly integrated sensor–hormone–effector axis in monocot species. A comparative analysis with existing models of JA–RNAi-mediated innate immunity is conducted to highlight the novelty and theoretical significance of this RBRL-mediated pathway. Furthermore, the discussion extends beyond the reported data to explore the implications of this network for growth–defense trade-offs, its applicability in breeding programs, and its stability under field conditions. This independent analysis aims to provide a balanced perspective on both the transformative potential and the practical challenges associated with translating this mechanistic framework into sustainable crop resistance (Fig. 1).

2. RBRL: A double-recognition sensor and ubiquitin ligase

The initial phase of viral infection entails recognition of the pathogen by the host organism. Huang et al. [1] have demonstrated that the RING ubiquitin ligase RBRL in rice specifically interacts with the CP of RSV and the P2 of rice dwarf virus (RDV) through its conserved RING domain. This double-recognition mechanism, which targets distinct viral proteins, endows RBRL with the capability for broad-spectrum virus detection, contrasting markedly with the defense strategies employed by certain dicotyledonous plants that rely on virus-specific receptors. This discovery not only elucidates a novel immune strategy in rice but also introduces a new avenue for researching cross-crop disease-resistance mechanisms. As an E3 ubiquitin ligase, RBRL primarily functions to label viral proteins through ubiquitination modifications, thereby facilitating their degradation via the proteasome pathway. Huang et al. [1] demonstrated that viral infection significantly increases the ubiquitination activity of RBRL. Importantly, the overexpression of *RBRL* in rice results in a marked reduction in viral protein accumulation. This "molecular sentinel" function initiates direct viral elimination while simultaneously activating a complex downstream signal-transduction network, leading to a cascade-amplified immune response. RBRL's role as a frontline sensor bears conceptual similarities to that of pattern-recognition receptors (PRRs) in the animal immune system; however, its mechanism—which directly couples recognition to ubiquitin-mediated degradation—underscores a unique design principle within plant innate immunity.

3. The JA pathway: A central hub for antiviral signaling

The JA pathway serves as a critical regulator of the growth–defense balance in plant stress responses [2–5]. Huang et al. [1] elucidated its complex role in antiviral resistance in rice. Utilizing *rbrl* knockout mutants and *RBRL* overexpression (*RBRL-OE*) rice lines, the researchers demonstrated that RBRL is essential for JA accumulation in response to RSV infection. Transcriptomic analyses revealed that differentially expressed genes were significantly enriched in defense, JA signaling, and reactive oxygen species (ROS) pathways. To further investigate the underlying

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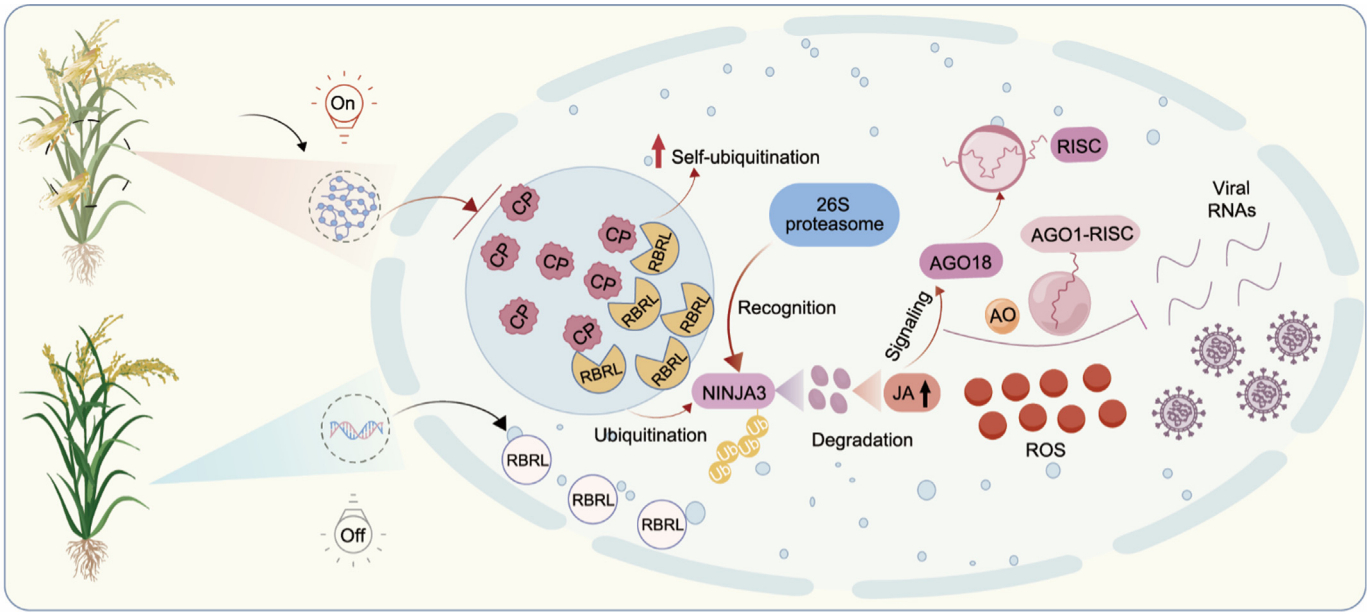

Fig. 1. The RBRL–NOVEL INTERACTOR OF JAZ 3 (NINJA3)–JA pathway: activation of rice antiviral defense through viral CP recognition. Upon virus entry into rice cells, facilitated by insect vectors, the viral CP is recognized and bound by the RING-type ubiquitin ligase RBRL, which subsequently activates its E3 ubiquitin ligase activity. This activation results in the ubiquitination and subsequent degradation of NINJA3, a known inhibitor of the JA signaling pathway. Degradation of NINJA3 alleviates the inhibitor's repression of the JA signaling pathway, facilitating pathway activation, which leads to the upregulation of genes responsible for JA biosynthesis and the expression of downstream antiviral factors, including AGO18. This mechanism also inhibits viral replication through enhanced RNAi, ultimately enabling rice plants to mount a robust antiviral defense. Ub: ubiquitin.

mechanisms, the study identified NOVEL INTERACTOR OF JAZ 3 (NINJA3), a pivotal inhibitor of JA signaling, through co-immunoprecipitation mass spectrometry. The findings indicate that RBRL-mediated degradation of NINJA3 alleviates repression of the JA pathway, resulting in the upregulation of JA biosynthetic genes (e.g., *LOX3*, *AOC*) and downstream defense genes. Analysis of the *ninja3* mutant confirmed that *NINJA3* negatively regulates JA signaling and compromises antiviral defense, with enriched pathways encompassing JA response, RNAi, and ROS metabolism.

4. Synergy and crosstalk: Integrating JA signaling with RNAi

A precise synergy exists between the JA and RNAi pathways, mediating broad-spectrum antiviral defense [6,7]. Huang et al. [1] demonstrated that RBRL connects antiviral RNAi with viral CP recognition and JA signaling. A low dose of CP is sufficient to be induced by RBRL to initiate the JA–AGO18 antiviral signaling cascade. This low-threshold induction mechanism likely functions as a specific safety switch, restricting activation of the resource-intensive JA–AGO18 defense to scenarios of confirmed viral invasion. This optimization of energy allocation serves to avoid unnecessary autoimmunity within the plant. Furthermore, deletion of *NINJA3* increases JA biosynthesis and activates signaling response genes, including *OsAGO18*. As a critical antiviral marker, JA facilitates the co-localization of the AGO18 protein with the viral replication complex by regulating its nuclear–cytoplasmic shuttling, which in turn increases small interfering RNA (siRNA)-mediated viral RNA-degradation efficiency. This regulation ensures that the RNAi machinery is precisely recruited to the site of viral replication, representing a key aspect of spatial coordination between hormone signaling and effector execution. The upregulation of *OsAGO18* fortifies host defense by mitigating the inhibition of AGO1 and promoting ROS accumulation [8–10]. This hormone–RNAi cross-regulation establishes a multi-layered defense network.

5. Future perspectives and unresolved challenges

5.1. Balancing resistance and yield: The growth–defense trade-off

While the constitutive activation of the RBRL–JA pathway confers significant antiviral resistance, it may also incur considerable fitness costs, including stunted growth and reduced yield—an established dilemma known as the growth–defense trade-off. To translate this knowledge into effective breeding strategies, future research must prioritize the precise spatiotemporal regulation of this pathway. Promising methodologies include the utilization of pathogen-inducible promoters to regulate *RBRL* expression or the editing of negative regulators such as *NINJA3* to fine-tune JA signaling specifically during infection. The viability of stacking the *RBRL* module with other resistance genes without incurring cumulative yield penalties remains a critical area for further investigation.

5.2. From laboratory to field: Ensuring durability in complex environments

Laboratory findings necessitate validation within complex field environments. The stability of RBRL-mediated resistance is challenged by several factors, including mixed viral infections and abiotic stresses such as drought, which may antagonize JA signaling. Potential suppression by effector proteins present in the saliva of insect vectors, such as the grey planthopper (*Laodelphax striatellus*), poses further complications. The durability of resistance is also a significant concern, as viruses may evolve CP mutations to evade recognition by RBRL. Therefore, long-term field trials and evolutionary studies are imperative to assess the robustness of this resistance. Furthermore, integrating the RBRL module with other immune mechanisms, such as salicylic acid (SA)-mediated defense, could improve resilience and durability under field conditions.

5.3. Expanding the theoretical framework and practical tools

Beyond immediate challenges, the RBRL-centric model warrants comparison with established paradigms. In contrast to many dicot systems that depend on specific nucleotide-binding domain and leucine-rich repeat (NLR) receptors for effector-triggered immunity, RBRL offers broad-spectrum recognition in conjunction with direct ubiquitin-mediated degradation. This constitutes a distinctive strategy in the antiviral defense mechanisms of monocots. Moreover, structural biology approaches can elucidate the three-dimensional architecture of RBRL and its complexes, thereby aiding in the design of small-molecular modulators. The integration of artificial intelligence tools, such as AlphaFold3, may facilitate the simulation of viral protein mutations and the design of "evolution-resistant" resistance alleles, making it possible to explore durability from a molecular evolution perspective.

6. Conclusions

The study by Huang et al. [1] discussed herein presents a comprehensive mechanism blueprint for antiviral immunity in monocotyledonous plants. It delineates the process from the cellular recognition of viral infection—mediated by RBRL double recognition—to the initiation of direct clearance via ubiquitination, and further to the activation of complex signaling networks involving JA and RNAi (Fig. 1). The investigation not only systematically elucidates the core mechanisms in rice but also provides a practical technical framework for antiviral breeding. The findings contribute a novel theoretical framework and methodological approach for improving crop antiviral resistance. The elucidated triple network underscores how plants integrate perception, hormonal signaling, and RNAi into a coherent defense system. Future research that balances efficacy with fitness costs and validates resilience in agroecosystems will be crucial in order to harness this knowledge for sustainable agriculture, with potential implications for other staple cereals such as wheat and maize.

CRediT authorship contribution statement

Jing Liu: Writing – review & editing, Writing – original draft. **Sheng Wang:** Writing – review & editing. **Wishwajith Kandegama:** Writing – review & editing. **Xin Xie:** Writing – review & editing. **Xiangyang Li:** Writing – review & editing, Supervision, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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