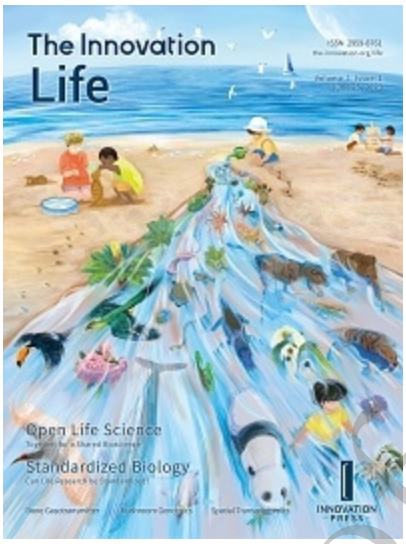

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Signal amplification and immune priming: Emerging principles from strigolactone and apoplastic pH signaling

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A range of pathogens, including viruses, bacteria, fungi, oomycetes, nematodes, and parasitic plants, cause destructive diseases that significantly threaten plant growth and development. Local infections commonly spread to surrounding healthy cells and ultimately extend to entire tissues or the whole plant. Plants deploy diverse immune receptors to perceive pathogens. However, effective amplification of downstream defense responses in infected cells and precise immune priming in adjacent healthy cells are essential for successful pathogen resistance. Two recent studies published in *Cell* have revealed that plant immunity is dynamically intensified through host physiological circuits that convert local immune activation into amplified or primed defense states.[1,2] One study shows that the strigolactone (SL) signaling pathway boosts antiviral immunity in rice by amplifying the production of virus-derived small interfering RNAs (vsiRNAs), which are key core antiviral executors. The other demonstrates that apoplastic alkalization and phytocytokines function as a coordinated signaling module, enabling immune communication between locally infected cells and distally uninfected cells and priming immune responses (Figure 1).

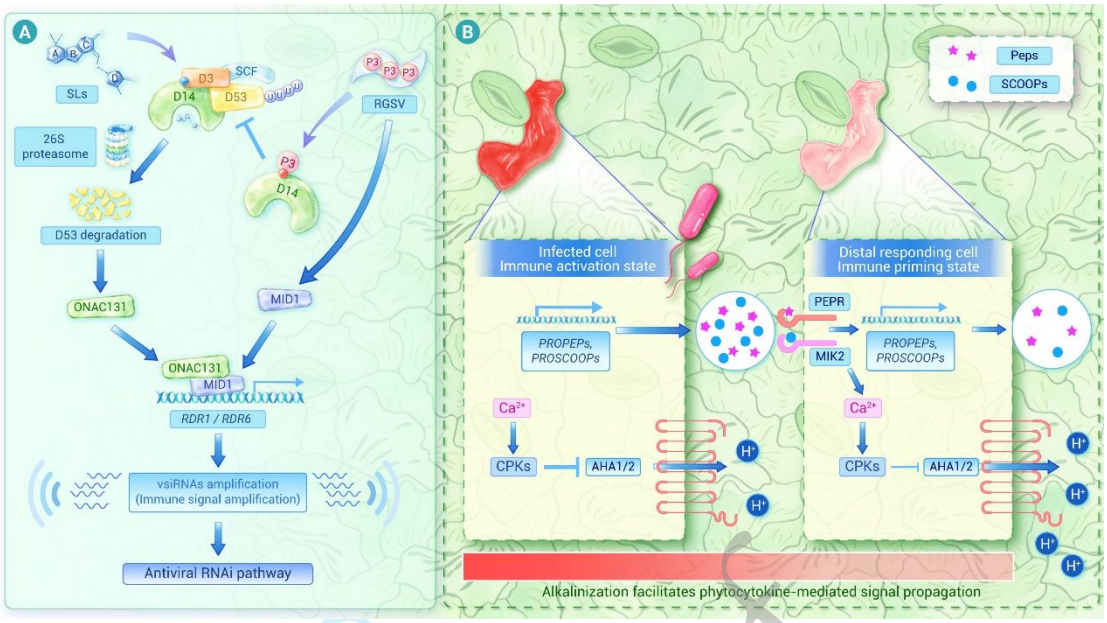

Figure 1. Strigolactone and ITA pathways regulate immune amplification and priming in plants (A) Strigolactone (SL) signaling enhances immune amplification during antiviral defense. In rice, SL perception by D14 recruits the receptor complex, resulting in ubiquitination and degradation of the repressor D53. This process activates SL-dependent transcription of *ONAC131*, which cooperates with the virus-induced MID1 to promote transcription of *RDR1* and *RDR6*. As a result, vsiRNA amplification increases, strengthening the antiviral RNAi pathway in rice. To counteract this SL-dependent immune response, the P3 effector from RGSV binds to D14, disrupts D14-D3 complex formation, and prevents D53 degradation. This inhibition blocks the ONAC131-MID1-RDR1/RDR6 module, reduces vsiRNA amplification, and weakens antiviral RNAi. (B) Working model of ITA-mediated immune priming. Pathogen infection triggers immune responses, including ITA and production of phytocytokines such as Peps and SCOOPs. Apoplastic alkalinization, resulting from CPKs-mediated inhibition of AHA1/2 activity, enhances phytocytokine perception in neighboring uninfected cells and establishes distal immune priming. In immune primed cells, AHA1/2 activity is modestly suppressed and phytocytokine biosynthesis is moderately elevated, supporting continued signal amplification and propagation. Accordingly, immune signals propagate from initially infected sites to distally uninfected tissues through the coupled action of apoplastic alkalinization and phytocytokine diffusion.

SL SIGNALING ACTS AS A KNOB OF IMMUNE AMPLIFICATION IN ANTIVIRAL IMMUNITY

Plants and viruses are engaged in a long-term molecular arms race. In plants, the RNA interference (RNAi) pathway acts as a conserved, broad-spectrum antiviral defense mechanism. Dicer-like proteins (DCLs) recognize and cleave viral double-stranded RNA (dsRNA), generating vsiRNAs. These vsiRNAs are loaded onto an Argonaute protein (AGO) to form RNA-induced silencing complexes (RISCs), which specifically recognize and clear viral RNA, thereby suppressing viral gene expression and establishing RNAi. RNA-dependent RNA polymerases (RDRs) use viral single-stranded RNA as a template to synthesize complementary strands, generating more dsRNA, which is then processed by DCLs to produce abundant vsiRNAs. Thus, RDRs amplify the core antiviral executors and convert a basal antiviral response into a systemic defense signal, bridging local initiation and systemic antiviral silencing in plants.[3]

SLs are carotenoid-derived phytohormones that regulate plant architecture, stress tolerance, symbiosis with arbuscular mycorrhizal fungi and the seed germination of parasitic weeds. The SL signaling pathway centers on the ligand-dependent assembly of the receptors D14/AtD14 with the F-box proteins D3/MAX2 and the transcriptional repressors D53/SMXL6,7,8. In rice, SLs trigger formation of the D14-D3-D53 complex, which induces the ubiquitination and degradation of D53 and a downstream transcriptional response. Although the roles of SLs in regulating plant development and rhizosphere interactions have been well investigated, whether and how SLs regulate plant immunity against pathogen invasion remain largely unknown.

Recently, Yang et al. revealed that SL signaling potentiates antiviral RNAi and amplifies immune response to the rice grassy stunt virus (RGSV) through the ONAC131-MID1-RDR1/RDR6 module.[1] A systematic analysis of viral resistance in plants with altered SL biosynthesis or signaling pathways demonstrated that SL functions as a positive regulator of antiviral immunity. Further studies revealed that SL signaling upregulates the expression of *ONAC131* by inducing the degradation of D53. RGSV infection induces the MYB-like transcription factor MID1, which interacts with ONAC131 to drive the transcriptional activation of RDR1 and RDR6, thereby promoting the amplification of vsiRNAs and antiviral RNAi (Figure 1A). Although

MID1 supports basal *RDR1*/*RDR6* transcriptional activation, MID1 alone is insufficient to elicit a potent antiviral defense. Robust induction of *RDR1*/*RDR6*, necessary for strong antiviral RNAi, depends on ONAC131, which is strictly controlled by SL signaling.[1] These findings suggest that SL signaling acts as a regulatory "gain knob" to promote antiviral RNAi in rice.

To counteract plant immunity, viruses have evolved diverse viral suppressors of RNA silencing (VSRs) that inhibit different stages of the antiviral RNAi pathway. For instance, the nonstructural protein P3 of RGSV is a key virulence factor and its expression alone is sufficient to induce dwarfing and excessive tillering in rice. Yang et al. found that RGSV employs its effector P3 to directly disable the "gain knob" of RNAi. Structural analysis revealed that P3 competitively binds to the SL receptor D14, thereby disrupting D14-D3 complex formation and inhibiting D53 degradation. This interference restricts the function of ONAC131-MID1-RDR1/RDR6 axis, reducing vsiRNA amplification and attenuating antiviral RNAi (Figure 1A).[1] Consequently, the viral counter-defense mechanism can hijack the host's immune regulatory systems to suppress host defense.

Crucially, this study used genome editing to precisely modify the D14 residue necessary for P3 binding. The D14-D102N allele specifically blocks P3 binding while maintaining interaction with D3, which alleviates the RGSV-mediated suppression of SL perception without disrupting normal SL signaling in rice. As a result, the D14-D102N mutant exhibited strong resistance to RGSV infection without a yield penalty.[1] This approach offers a novel strategy for crop antiviral improvement by targeting the signaling interface to prevent viral suppression of the host antiviral pathway without compromising plant growth or yield.

APOPLASTIC ALKALINIZATION AND PHYTOCYTOKINES PRIME DISTAL IMMUNITY

Beyond amplifying defense responses through multiple signaling cascades within infected cells, plants must also limit disease spread and prevent secondary infection to resist pathogen invasion. Accumulating evidence indicates that uninfected cells

adjacent to pathogen-contacted cells become immune-primed, likely triggered by immunogenic molecules from the pathogen-contacted cells.[4] However, the molecular identity and functions of these mobile signals in neighboring cells remain unclear. Recently, Wang et al. revealed that apoplastic alkalinization and phytocytokines reinforce each other, serving as signals for local-distal communication and priming plant defenses against pathogens.[2]

Apoplastic alkalinization, defined as immune-triggered alkalinization (ITA), is a recognized response to immune activation, but its precise functions and regulatory mechanisms in plant immunity remain elusive. The authors found that pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) induce ITA in both locally inoculated and distally uninoculated tissues, albeit with distinct magnitudes and durations. Local inoculation with avirulent bacteria also activated defense responses and enhanced disease resistance in distal tissues, indicating that local infection induces distal immune priming. Notably, this immune-primed state was significantly attenuated in the gain-of-function mutant of the plasma membrane H^+-ATPase AHA1, *ost2-2D,* which constitutively acidifies the apoplast and severely impaired both local and distal ITA.[2] The authors further revealed that CPKs-mediated phosphorylation of AHA1 at Ser899, which presumably represses the proton-pumping activity of AHA1, was strongly induced in distal tissues upon local infection. This phosphorylation is necessary for local infection-induced distal ITA and immune priming (Figure 1B).[2] These findings indicate that distal ITA, regulated by CPKs-AHA1/2 module, is essential for establishing immune priming in distal tissues. Recently, Zhai et al. demonstrated that a BIK1-GRF module-mediated reduction in AHA1 Thr211 phosphorylation, which enhances AHA1 proton pumping, is also required for extracellular alkalinization.[5] It is interesting to elucidate whether this process also contributes to the local infection-induced distal ITA and immune priming.

Phytocytokines are immunogenic peptides that are processed and/or secreted upon infection to modulate plant immunity. Genes encoding phytocytokines or their cognate receptors were markedly induced in distal tissues following local infection. Correspondingly, local-infection-triggered responses, such as ITA, AHA1

phosphorylation at Ser899, and disease resistance in distal tissues, were significantly reduced in plants lacking Pep-PEPR or SCOOP-MIK signaling. Moreover, Peps and SCOOPs treatment both induced apoplastic alkalinization through CPKs-AHA1/2 module. These findings suggest that phytocytokines serve as key signaling factors that mediate distal ITA and immune priming, though their limited mobility prevents them from acting as long-distance mobile signals. *PROPEP2* expression was initially induced in leaf segments adjacent to the infection site, followed by more distant segments. Blocking distal ITA completely abolished *PROPEP2* and *PROPEP3* induction and significantly reduced disease resistance in distal tissues upon local pathogen infection. Notably, even low doses of Peps and SCOOPs strongly induced phytocytokine gene expression, such as *PROPEP2* and *PROPEP3*, but only under alkaline conditions. This pH-dependent response likely results from enhanced binding of Peps to PEPRs in alkaline environments (Figure 1B).[2] Overall, ITA facilitates the perception and production of phytocytokines, while phytocytokine sensing further amplifies ITA. This positive feedback loop enables cell-to-cell propagation of apoplastic alkalinization and phytocytokine signaling, thereby priming immune defenses in distal tissues.

CONCLUSION AND PERSPECTIVES

Although these studies investigate different plant-pathogen systems, both demonstrate that plants integrate their innate immune system with key physiological processes to reinforce defense responses and improve disease resistance. The SL signaling pathway limits local infection by increasing antiviral effector molecules, while the ITA-phytocytokine circuit mediates the spread of immune states from infected cells to neighboring healthy cells, thereby restricting pathogen expansion. Moreover, SLs serve as master regulators of shoot branching and root architecture, while apoplastic acidification drives plant cell growth. Understanding how these pathways influence immunity advances our knowledge of plant innate immunity and helps identify core molecular factors that coordinate growth and defense, thereby supporting the development of high-yield, disease-resistant crops.

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AUTHOR CONTRIBUTION

B. W. and W. W. conceptualized the perspective. J. Y., X. W., W. W., and B. W. wrote the manuscript. All authors contributed to the manuscript and approved the final version.

DECLARATION OF INTERESTS

The authors declare no competing interests.