8th ICBPI 2026

THE 8th INTERNATIONAL CONFERENCE ON BIOTIC PLANT INTERACTIONS

第八届植物–生物互作国际会议

ABSTRACT BOOK 摘要汇编

Oct 18-22, 2026 | Nanjing, China

2026年10月18日-22日 | 中国·南京



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Plenary Speech

大会报告

Opsin-based optogenetics: new tools for plant research

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About 50 years ago, Bacteriorhodopsin (BR), a light-powered H+ ion pump in the membrane of a microbe, was discovered. With the advances in DNA technology, further microbial rhodopsins were unearthed; these are light-sensitive active or passive ion transporters, as well as enzymatic modulators of cGMP concentration. By heterologous expression and characterization of BR and other rhodopsins in animal cells we advanced the understanding of these proteins. We first characterized Channelrhodopsin-2 as a light-gated cation channel. Heterologous expression of channelrhodopsins led to previously unimaginable applications in different research fields, now called optogenetics.

For a long time, optogenetics in plants was not possible with rhodopsins, as they do not contain retinal which is necessary for a functional rhodopsin. In addition, as plants need visible light for their growth, white light illumination will activate heterologously expressed rhodopsins. Therefore, we generated transgene tobacco plants with retinal production which express highly functional channelrhodopsins in the plasma membrane. Anion channelrhodopsin-expressing plants grow normally under red light, whereas green illumination simulates drought stress and directs pollen tube growth. A Ca2+-permeable channelrhodopsin illuminated Ca2+ signalling in tobacco. Light-triggered ion flux has the potential to study poorly understood cellular signaling in biotic and abiotic stress situations of plants.

Understanding and exploiting plant immune receptor mechanisms

Jonathan Jones
The Sainsbury Laboratory, Norwich, UK

Plant nucleotide-binding leucine-rich repeat (NLR) immune receptors typically confer resistance through recognition of specific pathogen effectors. The Arabidopsis NLR WRR4A defies this paradigm by recognizing multiple sequence-divergent effectors from Albugo candida, conferring resistance to multiple pathogen races. Despite minimal sequence similarity, these effectors share a conserved N-terminal ferredoxin-like fold. Through cryo-EM structure determination of two WRR4A resistosomes bound to sequence-distinct effectors, combined with AlphaFold modelling, we reveal a shape-based recognition mechanism: WRR4A engages structurally conserved backbone features of the effectors in a mostly side chain-independent manner, enabling recognition of diverse effectors with similar three-dimensional architectures. These insights guided successful engineering of WRR4A to acquire novel recognition specificity. In addition, analysis of the monomeric WRR4A resting state reveals a distinct domain architecture characteristic of C-JID–containing TIR-NLRs and informs their activation mechanism. This work provides insights into NLR-mediated broad-spectrum recognition and the potential for structure-informed engineering of improved crop resistance.

Roles, recognition mechanisms and evolution of stress-regulated secreted signaling peptides

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Plants must constantly adapt to changing environmental conditions. Cell-surface receptor kinases play an important role in the sensing of stimuli originating from the environment itself or that are produced endogenously often in response to such external cues. I will present our recent work on stress-regulated secreted signaling peptides, their perception mechanisms, their diverse functions in adaptive stress responses, and their evolution.

Molecular basis of bacterial pathogenesis: from leaves to flowers

Sheng-Yang He
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Host–pathogen interactions offer many opportunities for the discovery of hidden biological processes across the kingdoms of life, with important basic and practical implications. My lab and collaborators have investigated how bacterial virulence factors manipulate plant cellular functions to promote bacterial colonization of plant leaves. These studies so far have revealed a fascinating array of molecular strategies by which bacteria manipulate plant immunity, stomatal regulation, hormone signaling, and water/nutrient homeostasis. More recently, our research has expanded to examine how climatic conditions influence disease development and how the endogenous plant microbiome shapes plant health and host–pathogen interactions. In this presentation, I will briefly overview our work over the past few years and then discuss our new efforts aimed at gaining insight into the infection biology of Pseudomonas syringae in Arabidopsis leaves at higher resolution, as well as unexpected aspects of plant immunity and bacterial infection biology in the reproductive organs, which have received relatively little attention in the analysis of plant–pathogen interactions.

Origins and mechanisms of TIR enzyme-based immunity in plants

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The Toll-Interleukin-1 Receptor (TIR) domain has a crucial role in bacterial, plant and mammalian innate immunity – working as a signaling adaptor or an NADase enzyme which generates ribosylated nucleotide signals. In recent years, researchers have uncovered the biochemical mechanisms by which TIR catalyzed nucleotide-based second messengers (SMs) turn on host defenses and stop infection. Some striking regulatory parallels between kingdoms, but also lineage-specific mechanisms, have emerged for TIR enzyme activation and nucleotide SM signaling. We're interested in understanding how pathogen-sensing at the cell surface by PRRs and inside cells by NLRs converges on TIR-catalyzed signaling cascades to promote host cell death and restrict pathogen growth in leaves. I'll discuss recent insights into the origins and functions of TIR-SM immunity modules in seed plants and, building on this, an SM engineering strategy to stimulate broad-spectrum disease resistance in crops.

Dissecting exotic resistance to soybean cyst nematodes

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Soybean cyst nematodes cause one of the most economically damaging diseases of any crop worldwide, and available resistance involves non-canonical plant defense mechanisms and some unusual genetics. I will describe presently unpublished findings in three areas. The soybean Rhg1 locus has been a linchpin of SCN control for decades and confers resistance via three disparate genes (and mechanisms) encoded on a ~30kb genomic segment that exhibits ten-fold copy number variation. We had previously deciphered resistance mechanisms for the α-SNAP encoded at Rhg1 and now report mechanistic dissection of the resistance conferred by the Rhg1 amino acid transferase and WI12 proteins. For example, AATRhg1 is tonoplast-localized, modulates cellular levels of Leu, Ile and Tyr and impacts ethylene signaling. WI12Rhg1 fosters salicylic acid production and upregulated expression some canonical defense mediators (BAK1, EDS1, CDPK26, RBOHB) and many receptor-like kinases. WI12Rhg1 protein is also 51% longer than annotated, due to use of an upstream CUG translation start site. Both proteins contribute significant resistance against highly problematic HG 2.5.7 SCN biotypes that partially overcome widely used rhg1-b resistance. Separately, novel SCN resistance has been recruited from wild germplasm. Screening of Glycine soja accessions identified rare occurrence of cqSCN-006 and cqSCN-007, which were introgressed over a decade ago into elite soybean germplasm by Brian Diers and colleagues and are now being integrated into commercial soybean germplasm. In 2021 we published that the cqSCN-006 QTL encodes a unique SNAP protein. We are now identifying the causal gene at cqSCN-007 and will report that this QTL that makes substantial contributions to SCN resistance via an encoded protein that does not resemble α-SNAPs or other previously identified plant disease resistance contributors.

Functionality and evolution of plant pattern recognition receptors

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Plants utilize both cell surface and intracellular immune receptors to detect pathogens. Plasma membrane pattern recognition receptors (PRRs) detect microbe-derived molecules, initiating pattern-triggered immunity (PTI), while intracellular nucleotide-binding leucine-rich repeat receptors (NLRs) recognize microbial effectors, leading to effector-triggered immunity (ETI). Our study reveals novel molecular insights into the EDS1-PAD4-ADR1 node, a crucial hub of immune signaling integrating both surface-resident and intracellular immune receptors.

PTI and ETI synergistically enhance plant resistance against microbial infections. While NLRs exhibit high polymorphism and rapid evolution, little is known about the sequence variation and functional diversification of PRR-encoding genes. Here, we document polymorphic plant pattern recognition receptors evolving in wild populations to recognize fungal or oomycete patterns. These receptors represent rare alleles, unusual for plant PRRs, typically widespread among species accessions, indicating rapid evolutionary adaptation.

Dissecting plant-microbe interactions at the single-cell and spatial resolution

Tatsuya Nobori
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Plant–microbe interactions profoundly influence agricultural productivity and ecosystem dynamics. Decades of research have illuminated the molecular basis of these interactions, from the architecture of the plant immune system to the strategies microbes use to colonize their hosts. Yet how these processes are organized at the level of individual cells remains largely unknown. To address this, we combined single-nucleus multiomics with spatial transcriptomics to dissect immune responses in Arabidopsis thaliana leaves infected with bacterial pathogens. We uncovered a striking diversity of immune cell states, each with distinct transcriptomic and gene-regulatory signatures and a characteristic spatial distribution. Among them is a rare "primary immune responder" (PRIMER) cell state that emerges at the nexus of immune-active regions. Together, these findings support a view in which immune cell states are fundamental building blocks of the plant immune system. In this talk, I will describe how we are mapping the diversity, regulation, and function of these states, and introduce emerging approaches for dissecting plant-microbe interactions at cellular resolution and omics scale.

Signaling Mechanisms of Salicylic Acid Receptors

Yuelin Zhang
Sichuan University

Salicylic acid (SA) is a key phytohormone that activates plant defense responses. In Arabidopsis, NPR1 (also known as NIM1) and NPR3/NPR4 have been identified as dual SA receptors responsible for perceiving SA. However, the mechanisms of how SA binding to the NPR proteins leads to induction of defense gene expression remain unclear. Here, we elucidate how SA triggers transcriptional activation via NPR1 and relieves transcriptional repression mediated by NPR3/NPR4. We identified Mediator Complex Subunit 15A (MED15A) as a bridge between NPR1 and the Mediator complex governing transcription. SA induces direct interaction of NPR1 with MED15A. Structural and functional analysis showed that the binding of NPR1 to MED15A is essential for NPR1-mediated transcriptional activation. Meanwhile, SA relieves transcriptional repression mediated by NPR3/NPR4. NIM1-interacting 1 (NIMIN1) interacts with NPR3/NPR4 and the Topless (TPL) co-repressor, connecting them to Polycomb Repressive Complex 2 (PRC2) to mediate H3K27 trimethylation of SA-responsive genes. SA inhibits the interactions between NPR3/NPR4 and NIMIN1, reduces H3K27 trimethylation levels and increases histone acetylation of the target genes to release NPR3/NPR4-mediated repression. Our study offers a comprehensive view of SA-mediated defense gene activation. These findings lay a foundation for designing more effective SA analogs as agrochemicals and for engineering crop resistance by manipulating SA perception and signaling.

A calcium channel function as a receptor of pathogen effector to trigger rice immunity

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Plants typically recognize pathogen effectors via nucleotide-binding leucine-rich repeat (NLR) receptors that converge on Ca^{2+} signaling to activate immunity. How non-NLR resistance proteins trigger immunity remains unclear. Here we show that the rice non-NLR resistance protein PtrA functions as an endoplasmic reticulum (ER)-localized Ca2+-permeable channel mediating resistance to the blast fungus Magnaporthe oryzae. The PtrA channel activity is suppressed by C-terminal farnesylation, which is relieved by the fungus-derived effector protein AVR-Pita that proteolytically removes the farnesylated C-terminal. This proteolytic switch activates Ca^{2+} release from the ER into the cytosol, triggering PtrA immune signaling. Natural allelic variation alters AVR-Pita cleavage specificity and resistance spectrum. Our findings establish PtrA as a receptor-channel sensing pathogen proteolytic activity to initiate Ca^{2+} signaling, directly coupling effector recognition to ion-channel activation.

Key words: disease resistance proteins, Ca2+ channel, pathogen effector, farnesylation, proteolysis

Designing Plant Immunity

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The convergence of genome editing, directed evolution, and de novo protein design is moving the life sciences from reading and rewriting DNA toward designing it. Crop improvement is following the same path, shifting from breeding that relies on random variation toward a predictable, programmable era of rational design. Centered on the precise improvement of crop genomes, our work pursues original innovation along three fronts. In precision genome editing, we uncovered how type V CRISPR nucleases evolved from transposon-encoded TnpB, showing that the functional splitting of a guide RNA drove the emergence of CRISPR systems. On this basis we created TranC, a compact editor independent of Cas9 with full, self-owned intellectual property. We also developed PrimeRoot for targeted large-fragment DNA insertion in plants and clarified a guide-RNA-independent off-target mechanism of base editing, substantially improving precision and controllability. In AI-guided protein design and chromosome engineering, we established a structure-driven method for protein clustering and AiCE, an AI-guided zero-shot approach to protein directed evolution that efficiently designed a series of novel deaminases and other editing components. By engineering recombinases, we further built Precise Chromosome Editing (PCE), advancing from single-base modification to chromosome-scale restructuring. In in planta directed evolution and programmable immunity, we developed GRAPE, a geminivirus replicon-assisted system that evolves plant proteins within their native cellular context, and a platform for Synthetic Plant Immune Receptors (SPIRs) that shifts disease resistance from discovery to design. Applied to coupling powdery mildew resistance with high yield in wheat and to herbicide-tolerant rice, among other elite germplasm, these tools mark the entry of precision crop breeding into a design-driven, programmable era of life.

Co-option and repurposing of plant pathways by geminiviruses

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Viruses, as obligate intracellular parasites, rely on a host cell for their persistence. To multiply, viruses must manipulate the host cell by hijacking, inhibiting, or repurposing its molecular machinery, thereby creating an environment conducive to viral replication. Typically, this task is achieved using only a handful of viral proteins encoded in the small-sized viral genome. This is evident in geminiviruses, plant viruses with circular single-stranded DNA genomes believed to encode only 4-8 viral proteins. Remarkably, despite their limited genetic arsenal, geminiviruses can establish systemic infections, alter plant development and physiology, manipulate the behaviour of their insect vectors, and cause devastating diseases to crops worldwide. How these viruses invade and manipulate their plant hosts using only a handful of proteins is a long-standing, intriguing question. Recent findings in our research group shed light on some of the strategies utilized by geminiviruses to infect plants: targeting of host hub proteins, repurposing of the host molecular machinery, redirecting of molecular pathways, multifunctionality of viral proteins, and maximization of their coding space. These insights contribute to our understanding of the remarkable efficiency with which geminiviruses manipulate host systems, paving the way for future strategies to combat viral infections in plants.

Local and systemic regulation of symbiotic nitrogen-fixing nodulation in legume plants

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Legume plants adapt their root system architecture to nutrient availability by modulating root growth and branching, as well as by forming root nodules. This organogenesis is induced by rhizobium symbiotic bacteria able to fix atmospheric nitrogen (N) within nodules.

Locally during the formation of root nodules, an extensive transcriptional reprogramming occurs, which is associated with dynamic changes in the 3D genome architecture. High-throughput chromosome conformation capture (Hi-C) indeed revealed large changes in enhancer-promoter interactions linked to symbiotic genes. Notably, a cytokinin (CK)-responsive distal enhancer forms a long-range chromatin loop with the proximal promoter of NODULE INCEPTION (NIN), a master regulator of nodulation, through the CK signaling TYPE-B RESPONSE REGULATOR 3 (RRB3) transcription factor.

In addition to hormones like CK, we characterized the role of signaling peptides in promoting symbiotic nodulation. We notably identified a type-B CLE (CLAVATA-LIKE) signaling peptide, referred to as symbiotic TDIF (sTDIF; symbiotic TRACHEARY DIFFERENTIATION INHIBITORY FACTOR), which is required for increasing locally the root stele diameter in response to rhizobium. This leads to a reprograming of the primary metabolism allowing promoting sugar accumulation in order to favour root nodule accommodation within host roots.

In addition to local regulations occurring in roots, different signaling peptides also participate in whole plant inter-organ long distance (systemic) regulations related to the plant nutritional demand. First, CEP peptides promote root competence for nodulation in response to a N-deficit, through the CRA2 receptor acting systemically in shoots. Second, CLE peptides inhibit systemically from shoots nodule formation through the SUNN receptor when enough nodule organs are already formed or when sufficient mineral N is available in soils. These two antagonistic systemic pathways are dynamically coordinated through the downstream regulation of the miR2111 shoot-to-root mobile effector. More recently, we showed that these systemic pathways allow integrating other environmental factors that N, such as CO2 and phosphate, notably in relation to another root symbiosis, endomycorrhiza, in the latter case.

New insights into plant interactions with pathogenic and beneficial bacteria

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A major focus in my lab has been to understand how plants deploy their immune system to fight off pathogenic bacteria. Our past efforts aimed to understand effector biology and biochemical basis of immune signaling. We recently addressed how cells directly under attack communicate with distal tissues to establish robust disease resistance. To this end, we show that pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) synergistically trigger apoplastic alkalinization, a classical defense response whose biological significance in immunity has remained unclear. The immune-triggered pH cooperates with phytocytokines to form dual signals propagating from local tissues to distal tissues, thereby prime disease resistance.

We additionally show that anti-virulence is a previously overlooked defense strategy that plants employ against bacterial pathogens. For instance, Arabidopsis produces sufarophane to specifically inhibit Pseudomonas syringae transcription of the type III secretion system genes. Moreover, plants accumulate erucamide upon PTI activation, which binds the the conserved HrcC protein in bacteria to block type III injectisome assembly. Importantly, erucamide is critical for anti-bacterial PTI. Neither sulforaphane nor erucamide inhibits bacterial growth at physiological concentrations, challenging the view that defense metabolites primarily kill or inhibit microbial growth.

More recently, we explored whether and how beneficial interactions exist between plants and bacteria during phosphorus (P) limitation, a major stress in the agricultural setting. We found that P-starved Arabidopsis roots actively recruit certain Acinetobacter isolates by secreting malate. At the rhizosphere, the bacteria switch to a new life style in which they actively utilize carbon and nitrogen nutrients in the root exudate to fuel solubilization of inorganic P and mineralize organic P, releasing orthophosphate that is then shared by both bacteria and plants. The study uncovers a new beneficial mutualism between plants and bacteria. We will discuss the potential application of our studies in the field.

Apoplast manipulation by Pseudomonas syringae

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Plants recognize bacterial pathogens through conserved fragments of flagellin (flg22) that bind the cell surface receptor FLS2 in most flowering plants. We previously discovered that a plant-secreted beta-galactosidase (BGAL1) promotes flagellin detection by removing the terminal glycan from glycosylated flagellin polymers, presumably promoting flagellin monomerisation. Here, I will discuss the identity of a BGAL1 inhibitor secreted by the bacterial model pathogen Pseudomonas syringae pv. tomato DC3000 (PtoDC3000). BGAL1 inhibitor 'glycosyrin' is a novel iminosugar produced by a biosynthesis cluster in PtoDC3000 that is regulated by virulence regulator hrpL and occurs in various P. syringae pathovars isolated from diverse plants. We resolved the glycosyrin biosynthesis, regulation and structure and discovered that it not only suppresses flg22 recognition but also dramatically alters the glycobiology of the host plant. Finally, I will give a brief overview of the research pipeline we are using to discover more extracellular suppression mechanisms in the apoplast of P. syringae-infected N. benthamiana plants involving chemical proteomics combined with large scale in silico protein interaction screens using AlphaFold Multimer.

Harnessing effector biology to engineer disease resistance

Wenbo Ma
The Sainsbury Laboratory, UK

The dynamic interplay between a pathogen's virulence factors and a host's immune system determines whether disease occurs. A deep understanding of the molecular mechanisms underlying the host-pathogen arms race is therefore essential for developing durable disease resistance. My lab uses effectors produced by the oomycete pathogen Phytophthora and the bacterial pathogen Pseudomonas syringae as molecular probes to identify key components of plant immunity and microbial pathogenesis, with a recent focus on a battleground centered on small RNAs (sRNAs).

We previously identified Phytophthora effectors that specifically suppress the plant secondary small interfering RNA (siRNA) pathway to promote disease. Further investigation revealed an important role for these siRNAs in plant defense through trans-species silencing of target genes in the invading pathogen, establishing natural host-induced gene silencing (nHIGS) as a bona fide immune mechanism in plants. We also discovered that the plant protein phosphatase 2A (PP2A) is a critical susceptibility factor required for the virulence activity of these effectors. Here, I will discuss our most recent efforts to develop Phytophthora resistance by enhancing nHIGS and disrupting effector manipulation of host PP2A using precision editing. Together, these studies establish a new paradigm for how a deep understanding of effector-host interactions can open new avenues for disease control.

ICBPI 2026 · OCTOBER 18–22, 2026

Session I

Inter-organismal Perception and Recognition

生物间感知与识别

The function and mechanism of the root microbiota in plant growth and health

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Roots of crop plants associated with taxonomically diverse microbiota. Understanding the functions of root microbiota and systematically obtaining the cultured root microbes are crucial steps for harnessing crop microbiome in sustainable agriculture. Recently, we identified root bacterial microbiota members significantly associated with crop growth by performing large scale associations of root microbiota, growth traits and genotypic variations of field-grown crops. Using individual bacteria and synthetic community approaches, we validated the functions of root microbiota members in controlled conditions. In addition, genomes of root-derived bacterial culture collections showed the metabolic and functional capacity of root microbiota. These data and resources allow us to reveal the functions and co-adaptation of root microbiota and field-grown crops.

PRR-phytocytokine modules in legume

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Plant pattern recognition receptors (PRRs) detect pathogen-associated molecular pattern (PAMPs), damage-associated molecular pattern (DAMPs) and stress-associated phytocytokines to signal pathogen threats. However, our understanding of PRRs and their elicitors has been heavily biased towards model organisms. Many ligand-receptor pairs are species-specific or even accession-specific, limiting our understanding of the evolution of PRR-ligand and engineering PRRs for broad-spectrum crop resistance. Here, we employed an innovative approach to identify multiple peptide-receptor pairs in soybean, including many previously uncharacterized ones. I will show the role of PRR-phytocytokine modules in plant-microbe interactions and provides a framework for mining and engineering peptide-receptor modules to strengthen crop immunity.

Leveraging AI-based structural modeling to characterize the interaction interfaces of LRR receptor complexes

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Leucine-rich repeat (LRR) receptor kinases (RKs) and receptor proteins (RPs) are important classes of plant pattern recognition receptors (PRRs) activating pattern-triggered immunity. While classical and AI-based structural approaches have recently provided crucial insights into ligand-LRR-RK binding mechanisms, little is known about how ligands are perceived by LRR-RPs. In contrast to LRR-RKs, many LRR-RPs typically embed one or more loopout regions in their extracellular domains that are crucial for functionality. We here used an AIbased approach to reveal a ligand-binding mechanism shared by the Arabidopsis LRR-RPs RLP23 and RLP42 – the PRRs for the short peptide ligands nlp20 and pg13, derived from NECROSIS- AND ETHYLENE-INDUCING PEPTIDE 1-like proteins (NLPs) and fungal endopolygalacturonases (PGs), respectively. This mechanism relies on a β-strand interaction with the N-terminal part of the island domain (ID) loopout, which adopts an anti-parallel β-sheet conformation. Additionally, we investigated the binding interface of RLP32 – the PRR for proteobacterial TRANSLATION INITIATION FACTOR 1, a folded protein ligand that requires its tertiary structure for recognition. Finally, we describe a mechanistic role of the ID for co-receptor recruitment conserved across LRR-RPs. Together, our results shed light on the ligand-binding mechanisms and receptor complex formation of LRR-RPs, opening avenues for their engineering for crop disease resistance.

What defines a plant's nose? —Volatile perception and plant-plant interactions in maize

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Plants respond to herbivore-induced volatiles from attacked neighbors by upregulating their defenses. How plants perceive these volatiles is unclear. Here, we identifed two G-type lectin receptor kinases that function as green leaf volatile receptors. Using transcriptional screens and gene editing, we identify the maize "Hexenyl acetate insensitive 1" (Haci1) mutant, which shows reduced responsiveness specifically to herbivore-induced green leaf volatiles. HACI1 localizes to the plasma membrane. Its extracellular domain shows strong, specific binding to (Z)-3-hexenyl acetate and (Z)-3-hexen-1-ol. Genomic analyses uncover a functional paralogue, Haci2, which is conserved across different grasses. haci1/2 double mutants are insensitive to green leaf volatiles and are no longer able to mount defenses in response to volatiles from herbivore-attacked neighbors. Thus, cell surface receptors mediate plant stress volatile perception.

N-glycosylation modulates chitin processing and perception in chitin-triggered plant immunity

Jian-Feng Li
Sun Yat-sen University

Fungal pathogens are responsible for over 70% of crop diseases worldwide. Plants detect chitin, a conserved fungal cell-wall component, via plasma membrane pattern-recognition receptor complexes containing the LysM receptor kinase CERK1. This perception initiates intracellular immune signaling and confers broad-spectrum basal resistance against fungal pathogens. Nevertheless, several fundamental questions in this field remain unsolved: (1) how chitin-triggered immunity operates without causing growth penalties; and (2) whether Arabidopsis CERK1, which binds chitin extracellularly and transduces intracellular signals, can mediate chitin signaling independently.

By screening chitin-inducible Arabidopsis chitinases, we identified two apoplastic chitinase paralogs, MIC1 and MIC2. Both enzymes degrade fungal chitin in vitro, but mic1 and mic2 mutants display opposite fungal resistance phenotypes. We found that MIC1 digest chitin eventually into non-immunogenic dimers and monomers, whereas MIC2 produces immunogenic chitin oligosaccharides with longer chains (≥NAG4). Biochemical and genetic assays revealed that an MIC1-specific N-glycosylation at Asn137 dictates its chitinolytic specificity. Rice encodes only an MIC1 ortholog but lacks MIC2. This suggests that MIC1 represents an ancient chitinase, and Arabidopsis MIC2 arose from gene duplication with loss of the Asn137-equivalent glycosylation site. Notably, rice mic1 mutants exhibit enhanced fungal resistance without growth defects.

Using an Arabidopsis mutant lacking all eight membrane-localized LysM receptors, we found that complementation with CERK1 alone only triggers very weak chitin responses. Co-expression of CERK1 with LYK4 fully restores wild-type chitin responses, whereas CERK1-LYK5 co-complementation does not, indicating LYK4 as the primary chitin receptor. Recombinant LYK4 proteins purified from E. coli fails to bind chitin, whereas plant protoplast-expressed LYK4 retains binding capacity, pointing to a requirement for eukaryote-specific post-translational modifications. Indeed, N-glycosylation prediction, mass spectrometry, and site-directed mutagenesis together identified six N-glycosylation modifications on LYK4, which are critical for chitin-binding.

Our findings advance mechanistic understanding of plant chitin signaling and provide promising targets for antifungal crop breeding.

Enhancing rice resistance to a floral disease

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Rice false smut is currently one of the most serious rice diseases. It specifically damages floral organs and produces mycotoxins that threaten human and animal health. High-yield rice varieties are mostly susceptible to false smut; however, the mechanisms underlying this susceptibility remain largely unknown, and no major resistance genes have been cloned. Over the past decade, we have comprehensively investigated rice-false smut fungus interactions at cytological, molecular and genetic levels, and have obtained some clues regarding rice susceptibility/resistance to false smut disease. The pathogen preferentially targets stamens (particularly stamen filaments) and blocks flowering for successful colonization, and probably activates rice grain-filling system for further proliferation. As an adapted pathogen, the false smut fungus also subverts host immunity by utilizing an effector protein to target chitin receptors. This interaction module is highly conserved in the false smut pathosystem, resulting in general susceptibility. Leveraging population genomics, we have identified an ancestral allele of an immune receptor (Ruv1R) conferring robust resistance to false smut. This resistant allele was inadvertently discarded during breeding due to linkage drag with the GS3 grain-shape gene. Intense selection for the long-grain gs3 allele drove the resistant allele to near-extinction while enriching a susceptible allele. To decouple false smut resistance from grain quality, we propose highly actionable genetic strategies: breaking the linkage drag to introduce the superior receptor allele, or engineering the GS3 locus in the Ruv1R-GS3 background using prime editing technology.

Bacterial interference with fungal chemical sensing shapes plant infection

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Conidial dormancy represents a critical survival strategy for fungi under environmental stress, yet the mechanisms governing this adaptive state remain largely unknown. Here, we identify the G protein-coupled receptor Rsd1 as a farnesol-responsive sensor that governs this developmental switch in Fusarium pseudograminearum, the causal agent of Fusarium crown rot (FCR). Under high population density and nutrient limitation, endogenous farnesol activates Rsd1 and engages the cell wall integrity pathway to drive conidial dormancy. Although primarily triggered by self-derived signals, Rsd1 also responds to extracellular farnesol, linking environmental information to an intrinsic dormancy program. This adaptive response enhances survival but creates a fitness trade-off during infection, when rapid germination is required for host colonization. Heterologous expression of RSD1 into Magnaporthe oryzae, which lacks this receptor, delays conidial germination and compromises pathogenicity, supporting conservation of this sensory module across fungi. Microbial communities further tune this developmental decision through farnesol–Rsd1 signaling: germination-promoting bacteria awaken dormant conidia, whereas germination-inhibiting bacteria reinforce dormancy and restrict disease development. Our study reveals a receptor–ligand mechanism that links chemical sensing to fungal life-history decisions and demonstrates how microbial interactions reshape intrinsic fungal regulatory programs governing dormancy and infection.

A natural immune receptor variant restores recognition of toxin-induced condensates

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Plant pathogens deploy diverse strategies to evade host immune surveillance, yet how fungal toxins contribute to immune evasion and whether natural immune receptor variation can overcome this process remain poorly understood. Here, we uncover a toxin-driven immune evasion mechanism in Fusarium species and identify a naturally occurring wheat receptor variant that restores immune recognition. We identified TaRBCG1, a leucine-rich repeat receptor-like protein, as the cell-surface immune receptor recognizing BCG1, a conserved Fusarium immunogenic protein. Genetic association analysis of 825 wheat accessions and reciprocal allele editing revealed a single amino acid polymorphism that determines resistance to Fusarium head blight. Mechanistically, the mycotoxin deoxynivalenol (DON) directly binds BCG1 and promotes its condensation through cysteine-dependent intermolecular disulfide linkage. These condensates evade recognition by the susceptible receptor TaRBCG1-S, thereby attenuating immune activation and facilitating fungal infection. Remarkably, the resistant variant TaRBCG1-R preferentially recognizes DON-induced BCG1 condensates, restores receptor–coreceptor complex formation, and activates immune responses, overcoming toxin-mediated immune evasion. We further demonstrate that chemically distinct virulence-associated toxins produced by diverse Fusarium species induce condensation of their corresponding BCG1 orthologs, revealing a conserved immune evasion strategy across multiple pathosystems. Notably, interfamily transfer of TaRBCG1-R into maize, rice, and tomato enables recognition of toxin-induced condensates and enhances resistance to their corresponding Fusarium pathogens without detectable growth or yield penalties. Together, our findings establish toxin-induced condensation of pathogen-derived immunogenic proteins as a previously unrecognized mechanism of fungal immune evasion. They further reveal how natural variation in a plant cell-surface receptor can restore recognition of otherwise concealed microbial signals, providing a conceptual framework for understanding host–pathogen coevolution and a promising strategy for engineering broad-spectrum disease resistance across crops.

Deciphering the function of LRR-MAL receptor kinases in plant immunity triggered by cell wall derived glycans: from glycans structural recognition to sustainable crop protection

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The plant immune system perceives a diverse set of carbohydrate ligands through the extracellular ectodomains (ECDs) of Pattern Recognition Receptors (PRRs), which activate Pattern-Triggered Immunity (PTI). Among these ligands are oligosaccharides from plant cell wall polymers like cellulose (β-1,4-D-(Glc)3, CEL3), mixed-linked glucans (β-1,4-D-(Glc)2-β-1,3-D-Glc, MLG43), xylans (β -1,4-D-(xylose)4, XYL4) and arabinoxylans (33-α-L-arabinofuranosyl-xylotetraose, XA3XX). The mechanisms behind carbohydrate perception and PTI activation in plants are poorly characterized (Molina et al., 2024a,b; Vilchez-Pinto et al., 2026). We have described the function in the perception of CEL3/CEL5 of a group of receptors with Leucine Rich Repeat-Malectin ECDs (LRR-MAL RKs; IGP1/CORK1, IGP3 and IGP4: Martín-Dacal et al., 2023). We demonstrated that IGP1 (Impaired in Glycan Perception) is the receptor for CEL3/CEL5, whereas IGP3 and IGP4 probably function as co-receptors in this recognition (Martín-Dacal et al., 2023). Notably, IGPs also participate in the perception of MLGs, XYL4 and XA3XX, further demonstrating the relevance of these LRR-MAL RLKs in PTI and disease resistance (Fernández-Calvo et al., 2023; Jimenez-Sandoval et al., 2026). We have determined recently the structural mechanism of IGP1-CEL3/CEL5 recognition (Jimenez-Sandoval et al., 2026). Also, we have identified additional igp mutants impaired in proteins that might be involved in the specific perception of MLGs. Recent progress in the functional characterization of IGP proteins will be presented. Also, our recent progress in the application of IGP-based knowledge to design sustainable crop protection solutions will be discussed.

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A receptor for Gram-positive bacterial recognition in Arabidopsis

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Whether and how plants specifically recognize Gram-positive bacteria has remained an unresolved question in innate immunity. We recently identified glycosylated wall teichoic acid (WTA), a conserved Gram-positive bacterial cell wall glycopolymer, as a microbe-associated molecular pattern perceived by Arabidopsis. WTA triggers salicylic acid-associated responses, defense priming, and non-canonical programmed cell death through the cysteine-rich receptor-like kinases CRK7 and CRK43. Direct binding of glycosylated WTA to the CRK7 ectodomain establishes CRK7 as a receptor for Gram-positive bacteria and reveals specificity for glycosylated WTA. Together, these findings identify the first plant receptor for a Gram-positive bacteria-specific MAMP, establish the first ligand–receptor pair for the CRK family in Arabidopsis, and uncover a molecular mechanism for the recognition of Gram-positive bacteria.

Plant Helper NLRs and TIR-only proteins: Regulatory Mechanisms and Exploitation

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Plant diseases pose a major threat to crop production. Most cloned Resistance genes employed in crop breeding encode intracellular immune receptors known as nucleotide-binding leucine-rich repeat receptors (NLRs). Plant NLRs can be functionally categorized into sensor NLRs, which detect pathogen effectors, and helper NLRs, which transduce immune signals to activate defense responses. Arabidopsis ADR1 helper NLRs have been shown to localize at the plasma membrane (PM) and function as Ca^{2+}-permeable channels to activate immunity. Here, we show that ADR1s relocate from the PM to the nucleus after TIR-type NLRs (TNLs) activation and identified TRANSPORTIN1 (TRN1) as a nuclear transport receptor that mediates nuclear import of ADR1s, thus enabling the formation of TNL-induced EDS1-PAD4-ADR1s complex in the nucleus. TRN1 also suppresses the autoactivity of ADR1s through compromising their PM localization and oligomerization. Nucleus-localized ADR1s impair their autoactivation capacity but the nuclear pool is required for TNL-activated transcriptional reprogramming and disease resistance.

Cross-species transfer of NLRs could broaden recognition capacities that are available for resistance breeding, but transfer of NLRs into taxonomically distinct species to confer resistance has proven to be constrained by restricted taxonomic functionality (RTF). We show that co-delivery of pepper Bs2 and its downstream helper NLR partners NRCs into rice confers resistance against bacterial leaf streak, thus overcoming RTF. The interfamily deployment of sensor NLRs with cognate helper NLRs could broaden the resistance utility of known NLRs and enable breeders to redeploy these NLRs across different plant families.

Finally, I will present an alternative strategy by harnessing the immune signaling amplification function of TIR-only proteins for engineering disease resistance. Through either promoter editing of endogenous OsTIR gene or heterologous expression of Arabidopsis TIR-only gene RBA1 in rice, we show that optimal elevation of TIR-only expression potentiates PTI signaling and enhances resistance to multiple pathogens without compromising yield.

Molecular mimicry of a pathogen virulence target by a plant immune receptor

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Plants and animals respond to pathogen attack by mounting innate immune responses that require intracellular nucleotide binding leucine-rich repeat (NLR) proteins. These immune receptors detect pathogen infection by sensing virulence effector proteins. However, the mechanisms by which receptors evolve new recognition specificities remain poorly understood. Here we report that a plant NLR has evolved the capacity to bind to a pathogen effector by acting as a molecular mimic of a virulence target of the effector, thereby triggering an immune response. The barley NLR Mildew Locus A 3 (MLA3) confers resistance to the blast fungus Magnaporthe oryzae by recognizing the effector Pwl2. Using structural analysis, we show that MLA3 has acquired the capacity to bind and respond to Pwl2 through molecular mimicry of the effector host target HIPP43. We demonstrate that the amino acids at the binding interface of MLA3 and Pwl2 are highly conserved in interface of HIPP43 with Pwl2, and are required to trigger an immune response. We used this discovery to bioengineer SR50—an MLA ortholog in rye that confers resistance to wheat stem rust—by introducing the Pwl2 binding interface of MLA3. The resulting chimeric receptor has dual recognition activities, conferring disease resistance to wheat stem rust and blast in barley transgenic lines. Collectively, these results provide evidence that plant immune receptors have evolved sophisticated mimicry strategies to counteract pathogen attack, and this knowledge can be leveraged to engineer multiple disease resistance by NLRs.

Cell wall can talk: A peptide-receptor pathway links cell wall integrity sensing to pattern-triggered immunity

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Plants employ plasma membrane-localized receptors to perceive non-self or altered-self. As part of this surveillance system, plants monitor their cell wall integrity (CWI). How changes in cell wall integrity (CWI) are sensed and translated into immune signaling, however, remains poorly understood. In my talk, I will present evidence that disruption of CWI promotes pattern-triggered immunity (PTI) through transcriptional reprogramming mediated by the receptor kinase MALE DISCOVERER 1-INTERACTING RECEPTOR LIKE KINASE 2 (MIK2) in Arabidopsis. Upon cell wall damage (CWD), upregulation of the MIK2 ligand SERINE RICH ENDOGENOUS PEPTIDE18 (SCOOP18) activates MIK2-dependent phosphorylation of the receptor-like cytosolic kinase BOTRYTIS-INDUCED KINASE 1 (BIK1). BIK1, in turn, inhibits plasma membrane H+-ATPases (AHA) activity by disrupting AHA-GENERAL REGULATORY FACTOR (GRF) interactions through a conserved phosphorylation event. This phosphorylation-driven extracellular alkalinization is required for effective CWD responses. Collectively, our findings identify the SCOOP18-MIK2 module as a surveillance system that links CWI sensing to plant immunity by regulating apoplastic pH through BIK1-mediated inhibition of plasma membrane H+-ATPases.

Systemic immune activation by bacterial cyclic lipopeptides

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Some Plant Beneficial Bacteria (PBB) isolated from the root-associated microbiome retain a strong potential to protect plants against diseases notably due to their ability to stimulate host immunity and trigger the so-called Induced Systemic Resistance (ISR). Cyclic lipopeptides (CLPs) represent a specific class of secondary metabolites formed by PBB that have been described as potent elicitors of ISR. We are investigating the molecular basis of CLP-mediated priming of plant immunity from perception at the root cell surface to Systemic Immune Activation (SIA) upon subsequent MAMP detection. Using surfactin from beneficial bacilli as model compound, we showed that, in Arabidopsis, CLP perception at the plant cell plasma membrane does not involve the high-affinity protein receptor-mediated surveillance system but relies on a unique lipid-mediated mechanism. This process integrates CLP docking into sphingolipid nano-domains, physical remodelling of the membrane and subsequent activation of mechanosensitive ion channels. This mechanism appears distinct from pattern-triggered immunity (PTI) induced by classical host pattern recognition receptors. Surfactin perception also leads to a specific immune activation signature regarding the type, timing, and amplitude of early defence-related events and transcriptional reprogramming as compared to PTI. We anticipate that this non canonical mechanism of immune activation identified for surfactin also applies for other CLPs and for other microbial membrane-active metabolites, thereby supporting the emerging concept of membrane mechanosensing immunity.

Key words: Lipopeptides; immune priming; plant-beneficial microbe interactions

A pair of chitosan receptors OsFID1-OsFID2 confer broad-spectrum fungal resistance

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Chitosan, a major fungal cell wall component, activates plant immunity, yet the underlying perception mechanism remains unknown. Here, we identified OsFID1, an apoplast-localized DUF26-domain protein, as a chitosan-binding receptor essential for chitosan-triggered immunity. OsFID1 forms a receptor complex with OsFID2, a plasma membrane-localized DUF26-receptor-like kinase, to mediate chitosan sensing and immune activation, requiring OsFID2 phosphorylation for function. The OsFID1-OsFID2 complex confers broad-spectrum resistance to fungal pathogens. OsFID1 homologs are conserved in land plants, showing conserved chitosan-binding activity in monocots and dicots. Ectopic expression of OsFID1 in wheat and maize enhanced fungal resistance without growth penalty. These findings uncover a conserved receptor complex for direct chitosan sensing, advancing our understanding of plant-fungus interactions and providing a potential strategy for engineering durable fungal resistance crops.

Expansion of Immunodiversity during Land Plant Evolution

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Plants utilize cell-surface immune receptors to detect pathogen-derived molecules and activate defense. These receptors have expanded extensively during evolution, but whether this reflects simple gene duplication or diversification of recognition specificity remains unclear. Here, we combine large-scale comparative genomics with analysis of the inner leucine-rich repeat (LRR) surfaces that determine ligand recognition. We find extensive lineage-specific expansion and diversification of LRR receptor kinases across seed plants. Much of this diversity appears to emerge at the family and genus levels, suggesting that different plant lineages have evolved distinct capacities to perceive pathogens. These findings show that receptor number alone underestimates plant immune diversity and provide an evolutionary framework for discovering novel immune receptors with potential applications in crop protection and receptor engineering.

Mechanistic insights into phosphatidic acid signaling in plant resistance to late blight

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Phosphatidic acid (PA) is a key signaling lipid that rapidly accumulates upon activation of pattern recognition receptor (PRR)- or nucleotide-binding leucine-rich repeat receptor (NLR)-mediated immunity. However, how immune signals trigger PA production and, importantly, how PA is sensed in plants remain poorly understood. Here, we show that the plant diacylglycerol kinase DGK5 is required for immune activation-induced PA production. Upon PRR activation, DGK5 undergoes dual phosphorylation that fine-tunes its enzymatic activity for PA production. We further identify two zinc-finger proteins, PA sensing protein 1 and 2 (PASP1 and PASP2), as PA sensors that regulate plant broad-spectrum resistance to late blight and bacteria. Disruption of PASP1 and PASP2 compromises resistance to Pseudomonas and Phytophthora pathogens in Arabidopsis and potato. Mechanistically, PASP1 and PASP2 undergo both basal and immune-induced phosphorylation. A casein kinase-like protein (CKL) mediates their basal phosphorylation, whereas calcium-dependent protein kinases (CPKs) regulate immune-induced phosphorylation. These distinct phosphorylation events coordinately regulate PASP1/2-mediated plant immunity against different pathogen infection. Together, our findings establish a mechanistic framework linking immune-triggered PA activation, PA sensing, and downstream phosphorylation, revealing how PA functions as a second messenger to promote plant immunity.

Molecular Mechanism of Immune Receptor/Signal-Mediated Layered Plant Innate Immunity

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Plants, as sessile organisms, are continuously challenged by invasive pathogens, including bacteria, fungi, and oomycetes. Stomata, formed by paired guard cells, serve as key entry points for pathogens. To restrict invasion, plants perceive pathogen-associated molecular patterns (PAMPs) through membrane-localized pattern recognition receptors (PRRs), triggering signaling pathways that induce stomatal closure, known as stomatal immunity. In response, bacterial pathogens deploy phytotoxins (e.g., coronatine) and effectors to reopen stomata and promote entry, making guard cells a critical battleground in plant-pathogen interactions. Here, we investigate guard cell-mediated defense against bacterial infection. We show that the intracellular nucleotide-binding leucine-rich repeat (NLR) receptor ADR1 forms an immune complex with EDS1 and PAD4, functioning downstream of PRRs to regulate transcriptional responses that drive stomatal immunity. We also identify a guard cell-enriched RNA-binding protein, SAIR1, which forms pathogen-induced condensates and enhances stomatal immunity by modulating translation of defense-related mRNAs. Furthermore, we demonstrate that infected local leaves transmit danger signals to distal, uninfected systemic leaves and trigger stomatal closure as a global defense response, which we term systemic stomatal immunity (SSIM). We identify a uORF-encoded peptide, USIC, as a mobile signal that mediates this process. USIC is perceived by the receptor-like kinase complex SIRK1-KIN7, which activates downstream signaling to establish SSIM. Together, our findings uncover new molecular mechanisms underlying both local and systemic plant innate immunity.

ICBPI 2026 · OCTOBER 18–22, 2026

Session II

Effector Biology in Immunity and Pathogenesis

效应因子免疫调控与致病机理

Evolutionary insights into fungal plant pathogen emergence

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Adaptation of plant pathogens proceeds at speeds that easily overwhelm the rate of resistant cultivar deployment and fungicide development pipelines. Low cultivar diversity and the uniform application of fungicides can exacerbate these dynamics. Understanding the molecular basis of pathogen adaptation is critical to define more sustainable containment strategies. Emerging traits such as virulence on previously resistant cultivars or loss of fungicide sensitivity often appear in a geographically structured manner. Such geographic mosaics can be determined by regional variation in selection pressures and as a consequence of the pathogen's population history. I will show how very large panels of pathogen genomes from the same species are crucial to reconstruct processes of host resistance breakdowns and fungicide resistance emergence. In combination with phenotyping assays, large genome panels enable the tracking of pathogen adaptation across continents and reveal how effector gene loci are undergoing rapid sequence rearrangements. Broadly, this work highlights that pathogen adaptation is bound by numerous adaptive mutations difficult to predict from mutant screenings alone. An emerging theme from work across plant pathogens and beyond is that pathogen adaptation is fueled by hypermutation. This phenomenon is characterized by elevated mutation rates and facilitates the gain of adaptive mutations. I will highlight how recognizing hypermutation is complex but essential for our understanding of pathogen evolution. In conclusion, large-scale genomic investigations of pathogen species are essential to unravel mechanisms of pathogen adaptation and will help mitigate future emergence of new pathogens.

Immune perception of Lepidopteran oral secretions in plants

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Two bursts of reactive oxygen species (ROS) were observed in plant recognition of pathogen-associated molecular patterns, such as flg22. We found that Arabidopsis also experiences two times of ROS burst upon the perception of OS from multiple Lepidopterans including cotton bollworm (Helicoverpa armigera) larvae (oral secretion-induced ROS, ROSOS). Plants use OS to distinguish insect herbivory from mechanical wounding, thereby enhancing the conversion of leaf glucosinolates into toxic bioactive intermediates that deter herbivores. Through a screen of an EMS-mutagenized mutant library, we identified a mutant (for) that specifically fails OS-induced ROS production, while shows unaltered flg22-induced ROS production. This suggested that downstream components of ROS production remained intact in for, and the mutation most likely lies in OS perception. Bulked segregant analysis (BSA) sequencing revealed a single nucleotide mutation in FOR, which leads to a G-to-E substitution at position 967 of the coding protein (FORG967E). FOR is a typical leucine-rich repeat receptor-like kinase (LRR-RK), featuring an extracellular LRR domain for ligand perception and an intracellular kinase domain. Compared to the wild-type protein, the FORG967E variant is less stable and shows impaired OS recognition. Genetic complementation and mutant phenotyping confirmed that FOR is a sensor of OS to trigger plant immune response to insect herbivory.

Assembly and disassembly of pro-immune condensates in the nucleus

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Biomolecular condensates (BCs) are dynamic, membraneless compartments that organize essential cellular processes and play increasingly recognized roles in plant immune signaling. In this talk, we demonstrate how plant karyopherin-β proteins—a family of nuclear transport receptors canonically known for mediating nucleocytoplasmic cargo transport, act molecular chaperones that directly modulate phase separation and BC homeostasis to balance plant immunity. Specifically, we show that the Arabidopsis karyopherin-β KA120 suppresses the spontaneous condensation of the MOS4-Associated Complex (MAC), an essential component of the mRNA splicing machinery. This chaperone activity prevents the ectopic assembly of "MAC-dependent Nuclear Condensates" (MDNCs), pro-immune nuclear BCs that are normally induced only upon pathogen infection. By blocking inappropriate MDNC formation in the absence of pathogens, KA120 suppresses aberrant immune signaling and cell death, safeguarding plants against severe autoimmune disorders that penalize growth and yield. Finally, we contextualize these findings within a broader eukaryotic framework, highlighting karyopherin-β proteins as an ancient, evolutionarily conserved regulators that tune phase behavior to prevent pathological condensation and preserve cellular integrity across diverse biological contexts.

Oomycete Pathogen-Driven Hyperactivation of Host mRNA Decay Reshapes Plant Immunity

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The evolutionarily conserved CCR4-NOT complex governs eukaryotic gene expression by controlling mRNA deadenylation and turnover. Its CCR4/CAF1 subunits catalyze poly(A) tail removal, yet how they orchestrate mRNA homeostasis during plant immunity has remained unclear. Here we show that Phytophthora sojae, a devastating soybean pathogen, secretes a conserved effector, PsAvh360, that directly co-opts the host CCR4-CAF1 deadenylase subcomplex to disarm host defense. PsAvh360 physically associates with both GmCAF1 and GmCCR4, unexpectedly functioning as a molecular enhancer that accelerates their deadenylation activity and mRNA decay. This pathologically driven hyperactivation triggers widespread post-transcriptional reprogramming, selectively destabilizing transcripts linked to hypoxia, salicylic acid signaling, and light responses, thereby rendering soybean highly susceptible to infection. Strikingly, GmCAF1 and GmCCR4 exert opposing effects on immunity: GmCAF1 is a positive regulator, whereas GmCCR4 acts as a susceptibility factor. Overexpression of GmCAF1 confers robust resistance without causing penalties to vegetative growth, making it a promising target for engineering durable control of root and stem rot. Our findings uncover a previously unrecognized virulence strategy-pathogen-driven hyperactivation of host RNA decay-and establish the CCR4-CAF1 complex as a critical battleground in plant–oomycete interactions.

The Hyaloperanospora arabidopsidis effector HaRxL77 is hypermobile between cells and manipulates host defence

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Multicellular organisms require dynamic communication between cells, tissues and organs to integrate responses to external and internal signals. In plants, cell-to-cell communication relies in part on plasmodesmata, which connect adjacent cells and allow the exchange of signals and resources. Upon infection by pathogens, plants act to isolate infected cells from non-infected cells by closing plasmodesmata but pathogens can suppress this defence and maintain plasmodesmata in an open state. To address the question of what a pathogen might gain from keeping plasmodesmata open, we screened effectors from the biotrophic Arabidopsis pathogen Hyaloperonospora arabidopsidis (Hpa) for the ability to move cell-to-cell via plasmodesmata in plant tissues. We quantified the mobility of cytoplasmic effectors and identified six that were hypermobile, i.e., can move further than expected for a protein of that size. Of these, HaRxL77 indirectly modifies plasmodesmatal permeability to facilitate hypermobility and suppresses the flg22-induced ROS burst, suggesting that cell-to-cell mobility of effectors allows defence manipulation ahead of the infection front. Thus, this study provides novel insights into how Hpa exploits plasmodesmata-mediated intercellular connectivity to promote infection, characterising a poorly explored element of plant-pathogen interaction.

Nuclear effector of the rice blast fungus, Magnaporthe oryzae

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Pathogens utilize multiple types of effectors to modulate plant immunity. Although many apoplastic and cytoplasmic effectors have been reported, nuclear effectors have not been well characterized in fungal pathogens. We characterized nuclear effectors of the rice blast pathogen Magnaporthe oryzae. Nuclear effectors are secreted via the biotrophic interfacial complex, translocated into the nuclei of initially penetrated and surrounding cells, and reprogram the expression of immunity-associated genes by binding on effector binding elements in rice. We also found cis-element (TATTTCGT) for in planta specific expression and nuclear localization sequence (PGRSKKE) for a nuclear effector, MoHTR1. Expression of nuclear effector genes in transgenic rice caused ambivalent immunity: increased susceptibility to M. oryzae and Xanthomonas oryzae pv. oryzae, hemibiotrophic pathogens, but enhanced resistance to Cochliobolus miyabeanus, a necrotrophic pathogen. Our findings help remedy a significant knowledge deficiency in the mechanism of M. oryzae-rice interactions and underscore how effector-mediated manipulation of plant immunity by one pathogen may also affect the disease severity by other pathogens.

Key words: Nuclear effector; NLS; Rice blast

Secreted proteins mediate systemic infection of Ralstonia solanacearum

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Ralstonia solanacearum causes bacterial wilt by coordinated secreted virulence proteins that act from root entry to xylem dissemination. Although various secreted proteins are known as key virulence determinants, how these distinct classes of virulence factors coordinate the multi-stage infection process remains poorly understood. Upon root infection, type III effectors suppress basal immunity before massive bacterial multiplication: RipAB translocates into the nucleus, interacts with TGA factors, blocks RNA polymerase II recruitment at immune loci, and suppresses SA-induced genes. Whereas RipAD associates with plasma-membrane aquaporins PIP2;3 and PIP2;4, disrupts their oligomeric assembly and promotes their turnover to weaken ROS accumulation and defense genes transcription, so that early PTI is inhibited at both signaling and membrane-transport levels.

As bacteria advance into vascular tissues, cell-wall degradation and resistance breaking are integrated: the type II-secreted exo-polygalacturonase PehC hydrolyzes pectin-derived oligogalacturonides to dampen DAMP-triggered immunity and releases galacturonic acid as a xylem carbon source, while the hypervirulent-strain effector RipV2 interacts with and ubiquitinates the tomato helper-NLR module SlNRG1 for 26S-proteasome degradation, thereby overriding ETI-based resistance in Hawaii 7996. At late systemic stages, biofilm maturity in xylem is dynamically remodeled by RuvC, an HJ resolvase highly accumulated in aged biofilm that is secreted to cleave Holliday-junction-like structures in extracellular DNA, relieves excessive eDNA lattice retention, induces dispersal, and drives bacterial movement through xylem vessels. Together, these layers explain how distinct secreted proteins stage early immune suppression, mid-vascular invasion and late systemic spread during bacterial wilt.

Keywords: Ralstonia solanacearum; systemic infection, type III effectors; type II secretion; plant immunity, biofilm

Honing the message: planthoppers elicit a rice-specific volatile alarm call that attracts natural enemies

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Plants emit volatile compounds that orchestrate complex ecological interactions, with methylated catabolites of interaction-induced phytohormones being common examples. Salicylic acid (SA) mediates plant antipathogen responses, while its methylated derivative, MeSA, broadly mediates plant-insect interactions without specificity. Here, we identified dimethyl salicylate (DMSA), an unappreciated dimethylated SA catabolite, emitted by rice when attacked by the major destructive pest, the brown planthopper. DMSA biosynthesis requires an O-methyltransferase cascade, BSMT1 (benzoic acid/salicylic acid carboxyl methyltransferase 1)–MSOMT (methyl salicylate O-methyltransferase), which is directly activated by a jasmonate (JA)–responsive MYC2-JAMYB transcriptional cascade. Natural variation in the MSOMT promoter confers its herbivory-induced expression in indica but not japonica cultivars. Functionally, DMSA acts as a specific volatile signal attracting the egg-parasitoid wasps of brown planthoppers (BPHs) without mediating direct resistance, which demonstrably suppresses BPH populations in paddy fields. DMSA is an optimized advance in SA signaling-derived plant “alarm calls” with great potential in sustainable rice pest management.

Key words: dimethyl salicylate; jasmonate signaling; O-methyltransferase; rice; brown planthopper; parasitoid wasp

Chloroplast Immunity in the Interaction Between Wheat and Puccinia striiformis

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Wheat stripe rust, caused by Puccinia striiformis f. sp. Tritici (Pst), is a devastating airborne fungal disease that severely threatens global wheat production. As an obligate biotrophic fungus, Pst relies on living host cells to acquire nutrients while actively suppressing host immune responses. Chloroplasts, vital for photosynthesis and plant immunity, are key targets for pathogen attack. Recent studies have elucidated chloroplast-mediated immune regulation in the wheat-rust pathosystem.

Emerging evidence reveals that Pst secretes multiple effector proteins to target and perturb chloroplast functions. Effectors Pst_12806, Pst_4, and Pst_5 directly target TaISP, a subunit of the cytochrome b6f complex, interrupting the photosynthetic electron transport chain and suppress photosynthesis and reactive oxygen species (ROS) production. Pst_TTP1 hijacks thioredoxin to regulate chlorophyll degradation, delay leaf senescence, and form “green islands” that facilitate pathogen survival. Additionally, Pst exploits the wheat ubiquitination system to degrade host transcription factors, repressing the expression of photosynthesis-related nuclear genes and reducing chloroplast ROS, thereby attenuating host disease resistance.

Meanwhile, wheat has evolved counter-defense mechanisms centered on chloroplasts. The plasma membrane protein TaMP perceive Pst invasion, then translocates into chloroplasts to promote substrate degradation by the Clp protease complex. This process fine-tunes the accumulation of tetrapyrrole intermediates and dampens retrograde signal transduction to the nucleus, preventing excessive activation of chloroplast-mediated immunity. Notably, Pst_SCP disrupts this immune homeostasis by promoting the plasma membrane-to-chloroplast shuttling of TaMP, ultimately enhancing host susceptibility.

Collectively, these findings establish chloroplasts as a critical battleground in plant-pathogen arms races. They advance our mechanistic understanding of the precise, robust regulation of plant immunity, providing a theoretical foundation for developing novel strategies to control wheat stripe rust.

Integrated approaches to understand the molecular bases of bacterial wilt disease

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Ralstonia solanacearum is one of the most destructive plant pathogens worldwide. Despite the importance of this pathogen and the research potential of the R. solanacearum model, the molecular interface between R. solanacearum and host plants remains poorly understood. Most bacterial plant pathogens employ a type-III secretion system to inject type-III effector (T3E) proteins directly inside plant cells. The complex infection process of R. solanacearum is supported by a large number of T3Es, although the function of most of them is still unknown. In order to understand the plant infection by R. solanacearum, we have performed screens aimed at identifying T3Es that target diverse plant functions, including immunity, responses to other environmental cues, and development. We are also trying to identify potential elicitors of immunity in R. solanacearum and their matching plant receptors, in order to generate novel sources of disease resistance against bacterial wilt disease. Finally, our work also aims to characterize the global genome- and proteome-wide changes that take place in plant cells upon R. solanacearum infection, with the goal of understanding general changes associated to bacterial virulence or plant immune responses.

Keywords: Ralstonia, effectors, metabolism, bacterial wilt, virulence.

Epitranscriptomic regulation of a plant DNA virus through destabilizing viral mRNA in wheat

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N6-methyladenosine (m6A) is a pervasive regulator of RNA metabolism and an emerging layer of host–virus interactions, yet its role in plant DNA viruses remains largely unexplored. Here, we used glyoxal and nitrite-mediated deamination of unmethylated adenosine sequencing (GLORI-seq) calibrated with in vitro-transcribed (IVT) RNA controls to quantitatively map m6A at single-nucleotide resolution in transcript of three representative plant DNA viruses: the geminiviruses wheat dwarf virus (WDV) and tomato yellow leaf curl virus (TYLCV), and the pararetrovirus cauliflower mosaic virus (CaMV). Under our detection criteria, we identified a single high-confidence m6A site at A1121 in the WDV coat protein (CP) transcript, whereas no reproducible high-confidence sites were detected in TYLCV or CaMV transcripts. Orthogonal MeRIP-seq analysis supported m6A enrichment within the WDV CP region. Disrupting the DRACH motif at A1121 reduced methylation, stabilized mRNA, and enhanced viral accumulation. Conversely, virus-mediated overexpression of the wheat m6A writer TaMTA increased m6A enrichment on CP mRNA, accelerated CP mRNA decay, and restricted WDV infection, whereas a catalytically inactive TaMTA mutant lacked these effects. BSMV (barley stripe mosaic virus)-mediated silencing of TaMTA reduced CP RNA methylation and enhanced WDV symptoms. Together, these findings reveal that host-installed m6A can selectively restrict a plant DNA virus through site specific destabilization of viral mRNA and underscore the importance of rigorous calibration in defining mechanistically meaningful viral RNA modifications.

Insects manipulates host nuclear trafficking and epigenetic regulation to facilitate herbivory

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Effectors secreted by pathogens or insects manipulate host plant cellular processes depending on their target destination. However, our current knowledge regarding nucleus-localized effectors from herbivorous insects remains limited. Here, we demonstrate that Nilaparvata lugens evolve a nuclear localization signal (NLS)-containing salivary effector NlAMSP that is specialized for targeting host plants. NlAMSP resides in the cytoplasm of insect salivary glands, but, upon secretion, migrates into the nucleus of rice cells. This nuclear translocation is enabled by the cleavage of its signal peptide, allowing the NLS-dependent import via the host importin-α/β pathway. SUMOylation at sites within the NLS is essential for the NlAMSP function, enhancing its nuclear localization and protein stability by preventing autophagy-associated degradation. In plants, NlAMSP interacts with the histone deacetylase OsHDA706 and redirects it from cytoplasm to the nucleus, thereby disrupting its interaction with the JA biosynthesis regulator OsLOX14 in cytoplasm. This interference reduces OsLOX14 accumulation and suppressed the JA-associated defense responses. Furthermore, nucleus-localized OsHDA706 diminishes histone H4K5ac and H4K8ac, thereby suppressing the expression of NLR and WRKY genes essential for rice resistance to N. lugens. Our findings uncover a mechanism by which an insect effector manipulates host nuclear trafficking and epigenetic regulation to facilitate herbivory.

Salivary Effectors Modulate the Interaction Between Planthoppers and Rice

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Piercing-sucking herbivores such as rice planthoppers inject two distinct types of saliva—gel saliva and watery saliva—into host plants during feeding. Gel saliva forms a salivary sheath that protects the stylet, supporting stylet penetration and sustained feeding, whereas watery saliva assists the insects in overcoming host plant defenses. Some proteinaceous effectors present in saliva can be perceived by plants as herbivore-associated molecular patterns, thereby inducing broad-spectrum plant immunity. On the other hand, other effectors can also target host plant resistance factors or mimic host susceptibility factors to suppress rice defense responses and facilitate phloem feeding. With the coordinated action of these two classes of salivary effectors, planthoppers feeding alone does not trigger strong rice immune responses. This finding demonstrates that salivary effector proteins are one of the key factors underlying the successful colonization of planthoppers and their emergence as major rice pests. Collectively, these results systematically delineate the complex attack–defense–counterdefense interaction mechanism between planthoppers and rice. The characterized planthopper effector proteins and their cognate rice resistance/susceptibility genes provide valuable genetic resources for the development of plant immune activators and insect-resistant breeding, respectively.

Key words: Rice planthoppers; Salivary effectors; Plant-insect interaction; Plant Immunity.

Apoplastic Chp family proteins in Clavibacter-plant interactions

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Clavibacter species are Gram-positive bacterial pathogens that infect diverse economically important crops. Among them, Clavibacter michiganensis and C. capsici cause bacterial canker in tomato and pepper, respectively. Unlike Gram-negative plant-pathogenic bacteria Clavibacter species lack both genes for either type III secretion system or type III effectors. Instead, they rely on secreted cellulases and putative serine proteases as major virulence factors. These proteins are secreted into the apoplastic space of plant tissues, where they contribute to disease development. We have shown that plasmid-encoded cellulase CelA and the putative serine protease Pat-1 of C. michiganensis are essential for wilt development in tomato, and ChpG, another putative serine protease, of C. capsici is required for necrosis in pepper. These putative serine proteases are collectively referred to as Chp family proteins, based on their similarity to the known serine proteases, although their enzymatic activities have not been experimentally confirmed. In addition to their roles in virulence, Chp family proteins can trigger a hypersensitive response (HR) in the nonhost plant, Nicotiana tabacum, but only when they are targeted to the apoplast. To date, at least three Chp family proteins - Pat-1, ChpG, and Chp-7 of C. sepedonicus - have been shown to induce HR in N. tabacum. Because three proteins function in both virulence and HR induction, they have been designated hypersensitive response and virulence (Hrv) proteins. Chp family proteins are predicted to be structurally very similar and contain a conserved catalytic triad (His-Asp-Ser) that is required for both virulence and HR induction, suggesting that their protease activity is critical for their biological function. Deletion of one or two chp family gene in either C. michiganensis or C. capsici abolished HR induction in N. tabacum. Unexpectedly, these deletion mutants became pathogenic and caused water-soaking symptoms in N. tabacum, indicating that Chp family proteins act as key avirulence factors in Clavibacter species. Overall, apoplastic Chp family proteins possess dual functions in promoting virulence in host plants while eliciting immunity in nonhost plants, highlighting their central role in Clavibacter-plant interactions.

Effector localization and translocation during rice blast fungus-rice interaction

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Rice is the most important staple food for nearly half of the world's population, yet its production is severely threatened by infection with Magnaporthe oryzae. Effectors are the main weapons used by the blast fungus to attack the rice immune system; therefore, understanding the molecular mechanisms of blast disease, including the identification of distinct effectors and their secretion pathways and transport routes, is essential. Here, we show that a penetration ring composed of the effector Ppe1 forms after appressorium-mediated cell wall penetration and remains at the base of the penetration site during fungal infection. The same persistent Ppe1 ring forms after penetration of neighboring cells by transpressoria. PPE1 is specifically expressed during plant infection, and the Δppe1 mutant is defective in penetration and invasive growth. Blocking penetration peg formation impedes development of the Ppe1 ring. In addition, we found that retromer mediates transport of the cargo protein MoSnc1 (a SNARE protein) from the vacuolar membrane to the surface of invasive hyphae after fungal invasion of rice cells, thereby maintaining effector secretion and promoting colonization. The composition of SNARE complexes and the function of SNARE proteins (21 in total) in M. oryzae, as well as the mechanisms by which they mediate effector secretion, were also investigated.

Plant Bunyaviral Polymerase Recruits Host Fatty Acid Synthase to Promote Palmitoylation-Dependent Ribonucleoprotein Assembly

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Plant negative-sense RNA viruses (NSVs) represent a class of devastating agricultural pathogens that cause substantial global economic losses. The infectivity and survival of these viruses depend on the assembly of ribonucleoprotein (RNP) complexes, comprising viral genomic RNA tightly encapsidated by the nucleocapsid (NP) protein and the viral polymerase; however, the regulatory mechanisms underlying RNP assembly remain elusive. Using the plant bunyavirus tomato spotted wilt virus (TSWV), we have elucidated a lipid metabolism-driven pathway that links host fatty acidsynthase (FASN) activity, palmitoyl transferase, and viral protein palmitoylation to RNP assembly. We demonstrate that the viral RNA-dependent RNA polymerase (RdRp, also known as L protein) recruits the host FASN subunit NbFabF and promotes its selfassociation, a process essential for its proviral activity. NbFabF-dependent palmitate availability promotes zDHHC14-mediated palmitoylation of the NP at residue C194 in planta. Functional analyses reveal that disruption of this modification selectively impairs the NP-L interaction without affecting NP localization, oligomerization, or intracellular trafficking, resulting in defective RNP assembly and reduced viral accumulation. Together, our findings establish a direct mechanistic link between host fatty acid synthesis and viral RNP formation, revealing a previously unrecognized role for lipid metabolic enzymes in regulating negative-strand RNA virus replication.

Aphid effectors suppress plant immunity via recruiting defense proteins to processing bodies

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Aphids are small insects that have developed specialized mouthparts and effector proteins to establish long-term relationships with plants. The peach-potato aphid, Myzus persicae, is a generalist, feeding on many plant species and capable of transmitting numerous pathogens. This study reveals how host-responsive cathepsins B (CathB) in the oral secretions of M. persicae facilitate aphid survival by modulating plant immune responses. Host-responsive CathB localize to plant processing bodies (p-bodies), cytoplasmic ribonucleoprotein granules involved in mRNA storage or decay. Upon localization, these CathB proteins recruit key immune regulators EDS1, PAD4, and ADR1 to these bodies, suppressing plant defenses. A plant protein, Acd28.9 (Hsp20 family), counteracts this CathB activity and contributes to plant resistance to aphids. These findings highlight an unexpected role for p-bodies in plant immunity and uncover a plant resistance mechanism to aphid infestation.

OsRNS4-mediated cross-kingdom RNA interference is inhibited by Ustilaginoidea virens effector SCRE2 to promote infection in rice

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The Ustilaginoidea virens effector SCRE2 has been previously identified to be an essential virulence factor. To elucidate molecular mechanisms underlying SCRE2 virulence function, we identify that SCRE2 interacts with Oryza sativa S-like ribonuclease family member OsRNS4. As a catalytically inactive RNase, OsRNS4 positively regulates rice immunity, depending on its RNA binding ability. OsRNS4 is unconventionally secreted through tetraspanin-positive extracellular vesicles (EVs). OsRNS4 binds to certain groups of rice miRNAs, whereas SCRE2 prevents the binding of OsRNS4 to miRNAs. Especially, OsRNS4-bound miR528-5p in rice is detected in false smut balls and silences the gene UvSfk1 essential for U. virens virulence. Thus, OsRNS4 might act as a carrier to transport miRNAs from rice into fungi, which target the virulence genes in U. virens and thereby contributes to rice resistance against fungal pathogens. As an unidentified counter-defense mechanism, SCRE2 suppresses cross-kingdom RNA interference to promote infection through inhibiting OsRNS4 binding to miRNAs.

Potyvirid 6K1 viroporin enhances viral replication by regulating pH homeostasis within the viral replication complex

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Viroporins are virus-encoded ion channels that play an important role in viral infection. The 6K1 protein encoded by members of the Potyviridae family is the first identified in plant viruses; however, its ion channel activity and pathogenic role remain elusive. Using non-invasive micro-test technology (NMT) and planar lipid bilayer assays, we show that 6K1 functions as a pH-dependent K+channel. Transgenic Arabidopsis overexpressing6K1, but not a loss-of-function 6K1 mutant, exhibited promoted root growth and enhanced K+uptake in roots. Moreover, the 6K2 protein co-localized with viral replication complexes and alkalisedreplication vesicles. Interestingly, NIb, the viral RNA-dependent RNA polymerase, displayed pH-dependent polymerase activity. Transgenic Arabidopsis overexpressing6K1accelerated turnip mosaic virus (TuMV) infection and restored the infectivity of a 6K1-deficient TuMVmutant. Taken together, our findings demonstrate that 6K1 is a pH-dependent K+channel that promotes potyvirus infection by alkalising replication vesicles, providing new mechanistic insight into viroporin-mediated viral pathogenesis.

ICBPI 2026 · OCTOBER 18–22, 2026

Session III

Immune Signaling Networks and Activation

免疫信号网络与激活机制

Structural basis of TIR domain-containing plant NLR signalling: resistosomes and enzymatic activities

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TIR (Toll, interleukin-1 receptor, resistance protein) domains feature in a subset of plant NLRs (nucleotide-binding – leucine-rich repeat receptors; TNLs) that respond to plant pathogen effectors and trigger effector-triggered immunity (ETI). Following the demonstration that the TIR domains from the human protein SARM1 have enzymatic activity - cleavage of NAD+ (nicotinamide adenine dinucleotide), we demonstrated such activity for a number of plant TIR domains (1), and defined a cyclic ADP ribose isomer 2'cADPR as one of the products of NAD+ cleavage (2). We extended this work to defining the enzymatic output of all Arabidopsis TIR domains (the TIRome) and correlated the enzymatic activity to cell-death activity in plants (3). We further report the cryo-EM structures of a canonical TNL protein, M from flax, in the inactive monomeric state and the tetrameric resistosome state bound to an NAD+ mimic (4). Our work provides new information about how TIR domains work, how they produce signaling molecules and how these products signal downstream. These findings will contribute to engineering bespoke plant immune receptors that can protect crops against disease.

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MAPK-mediated intracellular iron redox homeostasis in plant growth and immunity

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The redox state of iron is critical to its uptake, transport, utilization, and chemical reactivity in plants. The Arabidopsis genome encodes eight putative Fe(III) chelate reductases (FROs). Here, we demonstrate that FRO6 and FRO7 are two redundant Fe(III) reductases responsible for producing Fe(II) in plant mesophyll cells. Loss of FRO6 and FRO7 increases plant sensitivity to iron deficiency, resulting in stunted growth, chlorosis, and reduced photosynthetic capacities. FRO6 and FRO7 are localized to the ER instead of plasma membrane and chloroplast based on FRO6/FRO7-GFP fusion reporters and cell fractionation. Their expression is highly induced by iron deficiency, but rapidly suppressed upon MPK3/MPK6 activation in plant immunity. The fro6 fro7 double mutant is more susceptible to Pseudomonas syringae carrying avrRpt2 effector and exhibits delayed hypersensitive response (HR) cell death. Consistent with their compromised resistance, the fro6 fro7 double mutants show reduced accumulations of reactive oxygen species (ROS), highly reactive oxygen species (hROS, including hydroxyl radicals), and malondialdehyde (MDA), a product of lipid peroxidation initiated by hydroxyl radicals generated via the Fenton reaction. Collectively, our findings indicate that FRO6/FRO7 are essential for maintaining cytosolic free Fe(II), which is crucial for iron utilization during normal plant growth. Excess Fe(II) can be detrimental by triggering Fenton reaction, particularly when ROS including hydrogen peroxide are induced during plant immunity. MPK3/MPK6 activation-mediated down-regulation of FRO6/FRO7 may thus represent a mechanism to prevent excessive Fenton reactions, ensuring a balance between cell survival and immunity.

A plant RNA-binding protein senses 2',3'-cNMP produced by insect effectors

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RNA-derived 2',3'-cyclophosphate-terminated nucleotides are signaling molecules in both prokaryotic and eukaryotic immune systems, but how plants sense these signals is elusive. Sucking insects inject digestive enzymes including RNases into plant host to acquire nutrients. We demonstrate that NIB1, a glycine-rich RNA-binding protein (GRP) of rice, senses 2',3'-cAMP/cGMP produced by a salivary T2 ribonuclease of green stink bug during feeding. Binding of the RNase products facilitates NIB1 to form nuclear condensates that absorb the core immune regulator Enhanced Disease Susceptibility 1 (EDS1) and activate defense. Spraying rice plants with 2',3'-cAMP/cGMP elicits NIB1 and EDS1-mediated immune responses. Our findings unveil a conserved pathway in plants where GRPs perceive the nuclease-generated second messengers to launch defense.

Keywords: Plant immunity, EDS1, condensates，insect resistance

Plant antiviral immunity: From mechanistic insights to translational research

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Plant viruses pose a major threat to global crop production and food security. Understanding antiviral immunity and translating mechanistic discoveries into sustainable disease-control technologies are therefore important priorities in plant science. In this talk, I will present our recent progress in dissecting plant antiviral defense mechanisms and developing RNA virus-based tools for crop improvement.

First, I will discuss nucleotide-binding leucine-rich repeat receptor (NLR)-mediated antiviral immunity, highlighting the discovery and functional characterization of antiviral immune receptors in dicot and monocot plants. These studies reveal how immune-receptor activation and resistosome formation initiate effective defense responses against viral infection. Second, I will describe non-NLR-mediated antiviral defense operating at multiple biological levels. Our findings demonstrate that RNA regulation, protein ubiquitination, MAP kinase signaling, endoplasmic reticulum-associated autophagy, and lipid droplets collectively contribute to plant antiviral immunity. We have also investigated antiviral defense at the plant–insect interface, providing insights into how plants respond to viruses and their insect vectors.

To accelerate resistance-gene discovery, we have integrated forward genetics, genome-wide association studies, proximity labeling, and functional genomics to identify host factors involved in virus resistance or susceptibility. These discoveries provide candidate genes and regulatory networks for resistance breeding. Finally, I will present the development and optimization of barley stripe mosaic virus (BSMV)-derived vectors for gene silencing and genome editing in wheat. Through systematic improvement of guide RNA delivery and genome-editing strategies, the BSMV platform has been expanded to support conventional genome editing, base editing, and prime editing, facilitating rapid functional validation and the generation of potentially useful germplasm.

Together, these studies connect fundamental discoveries in plant antiviral immunity with technological innovation. They establish an integrated framework for understanding antiviral defense, identifying resistance resources, and developing virus-enabled genome-editing tools for sustainable crop protection and molecular breeding.

Mass-spectrometry-based cleavagome reveals pervasive proteolytic regulation across the proteome

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Proteolytic cleavage governs diverse cellular processes, yet the complete landscape of protein cleavage—the cleavagome—has remained inaccessible. Although proteolytic cleavage has conventionally been viewed as a targeted mechanism, our analysis indicates that it is in fact widespread, with ~76% of both the Arabidopsis and human proteomes undergoing cleavage; this extent rivals phosphorylation and exceeds ubiquitination, establishing proteolytic cleavage as one of the most pervasive PTM layers. We overturn this view with CleavageFinder, a label-free mass spectrometry workflow that identifies cleavage sites de novo at single-residue re solution without prior protease knowledge. Against curated benchmark datasets, CleavageFinder achieved F1 scores of up to 0.74. In Arabidopsis, CleavageFinder provides unprecedented coverage of cleavage sites and maturation proteases across nearly all peptide hormone families, resolving a long-standing challenge. Time-resolved infection analysis reveals a proteome-wide map of host proteolytic remodeling during DC3000 infection, exemplified by the TMK3-DRL34 immune-regulatory module. This establishes proteolytic cleavage as a fundamental regulatory layer and provides a general framework for post-translational discovery.

Mitochondrial Dynamics in Plant Immunity

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Plant immunity requires not only the activation of signaling pathways but also rapid reorganization of cellular structures and organelles. This presentation summarizes our recent work on how the actin cytoskeleton, mitochondrial dynamics, and endoplasmic reticulum-plasma membrane (ER-PM) contact sites coordinate immune responses in Arabidopsis. Pathogen perception induces dynamic actin remodeling, which controls the spatial organization and activation of cell-surface immune receptor complexes and supports downstream defense responses. In guard cells, immune activation triggers extensive mitochondrial fusion. Actin bundles facilitate mitochondrial encounters and provide structural support for elongated mitochondria, while the MDP25-VDAC3 complex promotes actin bundle formation and actin-mitochondria association. We further identified MIRO1 as a mitochondrial outer-membrane protein that accumulates at mitochondrial contact sites and mediates membrane fusion. This fusion response maintains mitochondrial membrane potential and ATP homeostasis, promotes mitochondrial reactive oxygen species production and organic solute metabolism, and is required for effective stomatal closure and resistance to bacterial entry. In addition, we developed LiMETER, a light-controlled, reversible, and precise tool for labeling ER-PM contact sites, and PDLP1-NEMO, a highly sensitive calcium reporter targeted to plasmodesmata. These tools reveal plasmodesmata-specific calcium signals and their physiological function in regulating intercellular connectivity during plant immune responses. Together, these findings demonstrate that the actin cytoskeleton, organelle dynamics, and membrane contact sites are active components of immune signaling. They provide a cellular framework for understanding how plants coordinate receptor activation, energy metabolism, intercellular communication during pathogen challenge. By linking molecular mechanisms with cellular behavior and whole-plant defense, this work highlights subcellular organization as a central layer through which plants achieve robust immunity under changing environments.

Ubiquitin Ligases RGLG1/2 and ATL31/6 Coordinate Plant Immunity and Development via Targeted Protein Degradation

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Pattern recognition receptors (PRRs) relay immune signals to downstream components, notably the receptor-like cytoplasmic kinase (RLCK) BOTRYTIS-INDUCED KINASE1 (BIK1) to initiate immune responses. Maintaining BIK1 homeostasis is critical for regulating immune signaling. This study elucidates how two pairs of E3 ubiquitin ligases, ARABIDOPSIS TÓXICOS EN LEVADURA 31/6 (ATL31/6) and RING DOMAIN LIGASE 1/2 (RGLG1/2), positively regulate immunity by stabilizing BIK1 protein levels.
We demonstrate that ATL31/6 physically interact with CALCIUM-DEPENDENT PROTEIN KINASE28 (CPK28), a kinase that promotes BIK1 turnover. Flagellin treatment enhances this interaction, leading to the ubiquitination and 26S proteasome-mediated degradation of CPK28. By removing CPK28, ATL31/6 prevent BIK1 degradation, thereby stabilizing BIK1 and enhancing immune signaling. Conversely, RGLG1/2 directly regulate BIK1 stability through a distinct mechanism. RGLG1/2 interact with PUB25, an E3 ligase that mediates BIK1 degradation. RGLG2 represses PUB25 ligase activity, while PUB25 mediates RGLG2 degradation. This mutual antagonism allows RGLG1/2 to suppress PUB25-mediated BIK1 degradation, resulting in BIK1 accumulation and amplified immune responses. Collectively, these findings establish ATL31/6 and RGLG1/2 as positive regulators of plant immunity that converge on BIK1 homeostasis via distinct ubiquitination pathways to ensure robust BIK1 accumulation and potentiate immune responses against pathogens.
Beyond immunity, RGLG1/2 also regulate developmental processes. RGLG1/2 interact with BARELY ANY MERISTEM1/2 (BAM1/2) receptor-like kinases (RLKs). The peptide ligand CLE13 enhances BAM1/2–RGLG2 interaction and RGLG2 ligase activity, resulting in increased BAM1/2 ubiquitination and degradation. Consequently, RGLG1/2 attenuate CLE13-mediated signaling in root meristems. Additionally, ATL31/6 were found to mediate TMK1 degradation, downregulating auxin signaling. Collectively, our findings highlight how distinct E3 ligases fine-tune BIK1 and RLK stability to modulate both immune defense and developmental growth.
Key words: ubiquitination; ubiquitin ligase; RGLG1/2; ATL31/6; BIK1

Identification of soil-borne disease resistance genes and molecular breeding in wheat

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Common wheat is one of the most important crops in the world. However, it is big challenge to directly uncover functional genes from common wheat due to its allohexaploid characterization. In this study, we constructed a pan-genome variant map in common wheat based on 3000 wheat germplasm of worldwide, and selected 800,000 gene-based variants plus 50,000 position-balanced variants as probes, finally developing a Pan800K wheat gene chip. The Pan800K is currently ultra-high-density gene chip with the richest genetic information covering the wheat genome globally, and was transferred to Chengdu Tiancheng Future Sci&Tec Co., Ltd. We also built a wheat multi-omics database including proteome, transcriptome, phosphoproteome, acetylome etc. Soil-borne disease is seriously threatening wheat production in China, especially for fusarium crown rot (FCR) and wheat yellow mosaic virus (WYMV). Through integration of genome-wide association study (GWAS), QTL, RNA-seq etc. we cloned a series of genes modulating FCR and WYMV, including TaCAT2, TaCWI, TaDIR, TaP5CS1, TaRD21a etc. We further dissected several molecular mechanisms regulating FCR resistance for TaSnRK1α-TaCAT2 model by scavenging ROS, TaSRT1-TaHSP18.6 model by mediating auxin content and FpgApple1-TaMyb85-TaCCR by modulating lignin content. Through pyramiding breeding, we developed two new quality varieties Yunong 268 with high zinc content and Yunong 269 with superior bread-making quality.

Specific and Efficient Translation Inhibition-Transcription Activation Resistance Module Against Viruses in Plants

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Positive-sense RNA viruses that generate subgenomic RNAs (sgRNAs) impose tremendous yield losses across global crop production. Unfortunately, most cultivated crop species lack readily deployable natural resistance (R) genes against diverse RNA viruses, leaving limited practical options for viral disease management. Although sgRNA biogenesis represents a highly conserved molecular signature among more than 1200 known plant RNA viruses, this universal viral feature has barely been harnessed for engineering broad-spectrum antiviral immunity.

To address this bottleneck, we engineered an inducible virus-triggered synthetic immune module designated TI-AT-RIP. This tandem-assembled genetic circuit contains three functional components: a translation-inhibitory TI element, a viral subgenomic-promoter-based AT activator, and resistance-inducing RIP effectors. In the absence of viral invasion, the TI domain strongly suppresses downstream translation to keep the module fully silenced and eliminates detrimental growth-defense trade-offs. Upon virus attack, viral replication proteins activate the AT subgenomic promoter to release translational repression and trigger robust RIP-mediated immune responses.

We performed parallel functional assessments using multiple RIP effectors against viruses belonging to three distinct plant-virus families and multiple genera, including PVX, PepMV, TMV, ToBRFV and CMV. Our synthetic circuit achieves complete immunity or high-level resistance to these viral pathogens, and importantly, it confers cross-resistance against divergent viral species within the same genus. Distinct from conventional antiviral strategies, our work represents a previously unreported synthetic antiviral approach that repurposes native viral subgenomic promoters as molecular switches for immune activation. This programmable strategy greatly expands the toolkit for plant antiviral engineering. Nevertheless, further structural tuning and regulatory optimization are still required to improve module performance for real-world agricultural deployment.

A conserved structural logic underlies sensor–helper NLR communication in the NRC immune receptor network

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NLR immune receptor networks consist of expanded disease resistance proteins (sensor NLRs) that signal via core executors of immunity known as helper NLRs. Although some sensor NLRs are thought to activate their cognate helpers via an activation-and-release mechanism, the structural basis of sensor-helper communication remains poorly understood. Here, we identify and validate sensor-helper NLR interfaces that are critical for immune activation in the NRC network of coiled-coil NLR immune receptors. Using AlphaFold 3 we predicted a high confidence model between the virus resistance protein Rx and its helper NLR NRC2. We validated the interfaces by loss and gain-of-function mutagenesis, including reconstituting a critical salt bridge through reciprocal mutations. We showed that these interfaces are conserved across the NRC network of asterid plants despite over 120 million years of divergence and validated the sensor-NRC interfaces within the common lettuce network. Structure-guided bioengineering of a lettuce sensor NLR enabled expansion of its NRC helper compatibility profile. These results are consistent with the activation-and-release model and point to bioengineering sensor-helper specificity in economically important crop species.

Plant immunity reshapes bacterial lifestyles toward host benefit

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Plants host diverse bacterial communities composed of commensals, beneficial microbes, and potential pathogens. Although collective bacterial colonization is essential for plant health, some community members can cause disease. Thus, a central challenge for plants may not be simply to distinguish friends from foes, but to suppress pathogenic potential in resident bacteria and coordinate microbial behavior to maintain a healthy microbiota. Here, we address this question by monitoring the health of a panel of immunity-related Arabidopsis mutants and screening for bacterial strains that cause disease specifically in these mutants, followed by plant-bacterial co-multiomics and molecular genetics. We show that reactive oxygen species, specialized metabolites, and defense phytohormones suppress pathogenic traits in resident bacteria, thereby maintaining microbiota homeostasis and promoting host plant health. In some cases, this behavioral shift converts a pathogenic bacterium into a host-beneficial microbe. Together, our findings indicate that plant immune pathways reshape bacterial lifestyles by suppressing pathogenicity and promoting host-beneficial functions, thereby supporting plant survival and fitness in natural environments.

The dual functionality of apoplastic small RNAs in tomato defense against Phytophthora pathogens

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Plant small RNAs (sRNAs) are well established as intracellular regulators of gene expression and, more recently, as mediators of trans-kingdom RNA interference against invading pathogens. However, whether extracellular sRNAs can modulate plant immunity through host-intrinsic mechanisms remains largely unexplored. Here, using the tomato–Phytophthora pathosystem, we systematically profiled apoplastic sRNAs during infection and uncovered a selective enrichment of specific sRNAs in the apoplast. Among them, miR408b was strongly induced upon pathogen invasion and preferentially accumulated extracellularly. Rather than entering pathogen cells, apoplastic miR408b functioned as a mobile immune signal, undergoing systemic movement to distal tissues. We identified SlGAUT12, encoding a galacturonosyltransferase required for homogalacturonan biosynthesis, as a direct target of miR408b and a negative regulator of tomato immunity. miR408b-mediated silencing of SlGAUT12 compromised cell wall integrity (CWI) and promoted the accumulation of immunogenic oligogalacturonides (OGs), thereby robustly activating damage-associated molecular pattern (DAMP)–triggered immunity (DTI). In parallel, siR4408, an sRNA derived from a protein-coding gene, directly targeted and cleaved virulence-related mRNAs in Phytophthora, thereby attenuating pathogen virulence. This work reveals a dual function of apoplastic sRNAs: they simultaneously enhance host immunity through an endogenous CWI–DAMP–DTI module and suppress pathogen virulence via trans-species RNAi. These findings advance our understanding of plant defense mechanisms and provide new molecular targets for designing RNA-based biocontrol agents.

Key words: tomato; apoplast; small RNA; systemic immunity; trans-species RNAi

Evolutionary study of plant NLR

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The nucleotide-binding oligomerization domain-like receptors (NLRs) are conserved immune receptors in animal and plant innate immune systems. However, the evolutionary mechanism of plant NLRs remains unclear. Here, we identified the ancestor proteins of plant NB-ARC-type NLRs in the bacterial immune system. The bacteria NLR-like proteins detect phage invasion and form oligomeric complexes, leading to ATP depletion and host immune cell death. The autoimmune mutations found in plant NLRs was also capable of triggering the activation of NLR-like protein in bacteria. Furthermore, we demonstrated that plant Toll-interleukin-1 receptor (TIR)-NLRs (TNLs) were first emerged in the multicellular algae, which the bacterial TIR protein fused with the algal NB-ARC protein via the horizontal gene transfer. Notably, the algal TIR protein possesses the dual enzymatic activities to activate both plant EDS1-dependent immunity and bacterial Thoeris immune system. Our findings reveal an evolutionary history of plant NLRs and highlight that NLRs are conserved immune receptors across the tree of life.
Key words: NLR, NB-ARC, TIR, plant, bacteria.

Effectoromics-Guided Resistance Gene Discovery Against Tobacco Black Shank Disease

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Tobacco black shank (TBS), caused by the oomycete pathogen Phytophthora nicotianae, is a devastating soilborne disease that severely constrains tobacco production. However, the resistance gene resources against TBS remain largely unexplored. To investigate the genetic basis of host and non-host resistance, we collected a panel of 28 cultivated tobacco accessions, 23 wild Nicotiana accessions, and 54 Solanum americanum accessions. Through field and laboratory resistance evaluations, we showed that all S. americanum is immune against TBS. To characterize the P. nicotianae effector repertoire, we generated a near T2T P. nicotianae genome and constructed an effector library containing 528 RxLR effectors. Effectoromics screening reveals the ETI landscape between P. nicotianae and its host/nonhost plants. By integrating GWAS, BSA-RNASeq, and map-based cloning strategies, we cloned 4 new immune receptors and their cognate AVR proteins. This study offers promising resources for resistance breeding and mechanistic studies of plant immunity.

Key words: Tobacco black shank; Phytophthora nicotianae; Effectoromics; Resistance gene; Nonhost resistance

Common Mechanisms of Pathogenesis Among Different Types of Rice Viruses

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Rice is a staple food crop in world, and its safe production underpins national food security. However, viral diseases such as Rice stripe virus (RSV) and Southern rice black-streaked dwarf virus (SRBSDV) cause severe yield and quality losses. Rice has evolved multilayered antiviral defenses, mainly including RNA interference (RNAi), phytohormone signaling, reactive oxygen species (ROS) burst, ubiquitination-mediated degradation, and autophagy. Jasmonic acid (JA) plays key roles in both anti-pathogen and anti-pest defense.

This study investigated OsMYC2, a core transcription factor in the rice JA signaling pathway, and elucidated its downstream antiviral mechanism. OsMYC2 positively regulates resistance to RSV and SRBSDV. Dual-LUC, ChIP-qPCR, and EMSA assays demonstrated that OsMYC2 directly binds the G-box cis-element in the OsRDR1 promoter and activates its transcription. Genetic crossing analysis showed that OsMYC2-mediated resistance depends on OsRDR1. Small RNA sequencing revealed that the OsMYC2-OsRDR1 module amplifies 21-nt phased small interfering RNAs (phasiRNAs) derived from long hairpin-structure non-coding RNA (LHR), predominantly 5-3p. LHR positively regulates RSV resistance via 21-nt phasiRNA-mediated RNAi. The 5-3p mainly targets OsNAC6, which negatively regulates RSV resistance and is regulated by OsRDR1. LHR also confers broad-spectrum resistance to SRBSDV and Magnaporthe oryzae. Field spraying of 5-3p or dsLHR significantly enhanced resistance to both viruses.

This study systematically reveals the OsMYC2-OsRDR1-OsNAC6 module in antiviral immunity, identifies LHR-derived phasiRNAs as mediators of broad-spectrum RNAi-based resistance, and demonstrates the field application potential of phasiRNA spraying.

A pseudo-protease regulates immune responses through translational control and P-body dynamics in rice

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Metacaspases (MCs) are conserved caspase homologs implicated in plant stress responses, immunity, and programmed cell death. However, emerging evidence suggests that some members of this family may function beyond proteolysis. Here, we describe a non-catalytic metacaspase family member in rice, a pseudo-protease termed Os-psMC1, that regulates translation during stress through its association with P-bodies, cytoplasmic condensates involved in mRNA storage, repression, and decay. Os-psMC1 rapidly accumulates in dynamic cytoplasmic condensates following pathogen challenge, activation of immune signaling, and during aging. Despite lacking the catalytic residues required for protease activity, Os-psMC1 influences mRNA abundance and may act through direct RNA binding. Loss of Os-psMC1 results in enhanced global translation during infection and increased disease resistance, indicating that it functions as a negative regulator of immune responses. These findings reveal an unexpected signaling role for a metacaspase pseudo-protease and identify RNA granules as important regulatory hubs linking immune signaling to translational reprogramming in plants.

Transgenerational memory of heat-attenuated immunity in Arabidopsis

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Warming-climate-triggered heat waves exert profound impacts on plant growth, development and immunity. To ensure reproductive success and achieve transgenerational stress adaptation, plants have evolved a sophisticated machinery to deploy transgenerational thermomemory of early flowering at the expense of compromised immunity. Heat drives heat shock transcription factor A2 (HSFA2) and histone H3K27me3 demethylase RELATIVE OF EARLY FLOWERING 6 (REF6) to form a positive feedback loop, which suppresses SUPPRESSOR OF GENE SILENCING 3 (SGS3)-mediated trans-acting siRNA (tasiRNA) biogenesis. The REF6-HSFA2 loop and reduced tasiRNA converge to release HEAT-INDUCED TAS1 TARGET 5 (HTT5). HTT5 is up-regulated upon both elevated temperature and pathogen inoculation, and it modulates transgenerational thermomemory of attenuated immunity, which is likely independent of thermosensitive salicylic acid (SA) biosynthesis. HTT5 encodes an atypical papain-like cysteine protease that lacks the canonical Cys-His-Asn catalytic triad. HTT5 physically interacts with catalase CAT2 and another cysteine protease RESPONSIVE TO DEHYDRATION 21A (RD21A). RD21A positively, while CAT2 negatively modulates plant immunity at the downstream of HTT5. RD21A also interacts with and destabilizes CAT2 protein. By reducing RD21A protein abundance, HTT5 enhances the protein accumulation and enzymatic activity of CAT2. Elevated CAT2 activity consequently suppresses reactive oxygen species (ROS) signaling and the hypersensitive response, ultimately leading to attenuated immune responses against invading pathogens. Our findings thus uncover a previously uncharacterized HTT5-RD21A-CAT2 module that mediates ROS scavenging. This work sheds new light on the essential roles of cysteine proteases in the transgenerational thermomemory of attenuated immunity in plants.

ICBPI 2026 · OCTOBER 18–22, 2026

Session IV

Symbiosis and Microbiome

共生与菌群

The Nitrate Transporter MtNRT2.3 is required for mycorrhiza establishment in Medicago truncatula

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Arbuscular mycorrhizal fungi (AMF) provide their plant hosts with N, P and other nutrients in exchange for a supply of carbon. Nitrate can be provided directly to the host or first assimilated into amino acids. Symbiotic nutrient exchange occurs in arbuscles, specialized fungal structures that form inside host root cells. We investigated Medicago truncatula Nitrate Transporter 2.3 (MtNRT2.3) for its role in symbiotic nitrate acquisition, due to its high expression in arbuscule-containing cells. NRT2.3 was found to localize to the peri-arbuscular membrane and, exhibited nitrate uptake activity when co-expressed in Xenopus laevus oocytes with its interacting partner MtNAR2.1. Knockout of MtNRT2.3 abolished nitrate uptake via mycorrhizal fungi, and, unexpectedly, severely reduced mycorrhizal colonization and resulted in under-developed arbuscules. Provision of high concentrations of nitrate restored the phenotype. Phylogenetic analysis revealed that MtNRT2.3 is absent in liverworts and mosses, clustering separately from MtNRT2.1, which is present in all plant lineages. Furthermore, the mycorrhizal phenotype of nrt2.3 was unable to be complemented by MtNRT2.1 expressed from the MtNRT2.3 promoter, suggesting these transporters have different biochemical activities or regulation. Our findings reveal that MtNRT2.3 plays a critical role in mycorrhiza establishment and has a physiological role distinct from that of MtNRT2.1.

Auxin regulation on soybean nodulation: from mechanisms to opportunities in breeding

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Soybean biological nitrogen fixation (BNF) is a green and efficient nitrogen acquisition strategy with great potential for intercropping systems. However, its agricultural application remains limited by the restricted host range of rhizobia and the suppression of nitrogenase activity under high nitrogen input. Using soybean as a model, we aim to optimize nitrogen fixation efficiency and develop high-efficiency, intercropping-compatible soybean varieties. Our strategy centers on the concept that “auxin governs nodule organogenesis and nitrogen fixation”.

Establishing legume–rhizobium symbiosis requires precise spatiotemporal control of auxin levels, yet the regulatory role of auxin—particularly rhizobia-derived auxin—in this process remains poorly understood. Here, we show that the auxin biosynthesis gene YUC2a is essential for the spatiotemporal control of nodule development in soybean (Glycine max), acting downstream of three transcription factors—NF-YA9, LBD41, and NIN1a—that orchestrate distinct developmental stages. At the early stage, rhizobia-derived auxin induces NF-YA9, which activates YUC2a expression in cortical cells to establish the auxin maximum required for nodule initiation. At the intermediate stage, rhizobia-derived auxin upregulates LBD41, which represses YUC2a to fine-tune auxin levels and ensure proper rhizobial colonization. At the late stage, rhizobia-derived auxin suppresses NIN1a, thereby derepressing YUC2a in nitrogen-fixing symbiosomes to optimize auxin status for nodule maturation. Consistent with this model, disruption of YUC2a and its homologs impairs cell division in nodule primordia, decreasing nodule density and nitrogen fixation capacity, whereas cortex-specific overexpression of YUC2a promotes nodule initiation but compromises rhizobial colonization. These findings reveal a previously unrecognized role of rhizobia-derived auxin as a spatiotemporal coordinator of nitrogen-fixing symbiosis. Together, this dynamic, stage-specific auxin regulatory circuitry optimizes nodule development and nitrogen fixation in soybean, providing a theoretical framework and genetic targets for engineering high-efficiency nitrogen-fixing varieties.

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Plant cell wall sensing integrates symbiotic signaling to enable rhizobial infection

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Root nodule symbiosis, which underpins biological nitrogen fixation in legumes, relies on intracellular colonization of root tissues by rhizobia. Local cell wall remodeling is essential for the transcellular growth of infection threads (ITs). However, how plants integrate these localized cell wall alterations with core symbiotic signaling remains elusive. Here, we identify a membrane-localized receptor kinase, Cell-wall sensing Infection Regulator (CIR), whose expression is specifically induced by rhizobia during infection. CIR physically interacts with the nodulation receptor NFR1α and represses NFR1α phosphorylation. During IT development, CIR senses cell wall pectin remodeling and releases its C-terminal domain, which translocates into the nucleus to propagate symbiotic signaling. Both cir loss-of-function mutants and artificial tethering of the CIR C-terminus to NFR1α block this release and substantially compromise rhizobial infection. Our findings uncover a CIR-mediated cell wall-sensing pathway that bridges localized cell wall remodeling and NFR1α-dependent symbiotic signaling, revealing a mechanism for controlled intracellular microbial invasion in plants.

Extracellular vesicle-mediated plant arbovirus transmission

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Plant viruses are important pathogens that cause serious economic losses worldwide. Most plant viruses depend on insect vectors for transmission. Extracellular vesicle (EV) plays an important role in intercellular and interspecies communication. How EV participates in viral infection and transmission remains unknown. We demonstrated that rice stripe virus (RSV) exploited exosomes, a type of EV, to overcome the salivary gland barrier for transmission from insect vectors to rice. The entry of RSV into exosomes relied on exportin 6, which acted as a vehicle to transport virions into exosome precursors via interactions with the cargo sorting protein VPS37a. At the same time, we successfully isolate rice EVs and found that two EV-enriched rice miRNAs, Osa-miR159a.1-1 and Osa-miR167a, are transported into midgut epithelial cells of insect vectors. Osa-miR159a.1-1 promoted RSV infection in insect vectors through activating insect Notch signaling pathway to inhibit apoptosis. Osa-miR167a directly binds RSV RdRp to suppress viral replication. This differential regulation of EV-mediated cross-kingdom RNA interference contributes to viral homeostasis in insect vectors and the following efficient transmission.

Engineering Honeybee Gut Symbionts by Synthetic Biology to Safeguard Pollinator Health

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Honeybees are the primary pollinators underpinning global food security, yet their populations face growing threats from the widespread application of agrochemicals. Many pesticides act as potent drivers of physiological decline by imposing oxidative stress on the bee gut, compromising epithelial integrity and survival. Because honeybees carry a limited innate antioxidant capacity, fortifying the gut with engineered microbes that produce protective molecules in situ has emerged as a promising strategy for strengthening pollinator resilience.

Here we describe a synthetic biology framework for reprogramming honeybee gut bacteria to counteract pesticide-induced oxidative injury, pursued along two complementary routes. In the first, we developed Escherichia coli Nissle 1917 as an engineering-friendly probiotic chassis, applying genome streamlining and in vivo competitive screening to obtain host-adapted strains with enhanced gut colonization. We then integrated the lycopene biosynthetic pathway into the streamlined chassis and engineered the outer membrane vesicle system to deliver this lipophilic antioxidant extracellularly within the gut. In the second route, we applied parallel synthetic biology tools to the native honeybee gut symbiont Snodgrassella alvi, exploring the extent to which resident symbionts can be repurposed as endogenous delivery platforms.

In honeybee challenge models, the engineered systems reduced gut oxidative damage and improved survival under both DSS-induced intestinal injury and exposure to representative pyrethroid and neonicotinoid pesticides. Together, these results establish engineered gut microbes, from probiotics to native symbionts, as a potential and broadly applicable strategy for safeguarding pollinator health against agrochemical stress, and illustrate the growing role of synthetic biology in microbiome-based host protection.

Genetic basis of rice rhizosphere microbiome for nutrient utilization and stress resistance

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Rhizosphere microbiome is crucial for plant nutrition and fitness, while it is frequently fluctuates with the soil environment, recent studies have demonstrated that some of them is consistently assembled in the rhizosphere of a specific plant genotype regardless of the soil condition, which is determined by host genetics. In this report, I will show that the OsLHT1 gene variation determined the difference of organic nitrogen uptake efficiency and the rhizosphere microbial degradation capability of organic nitrogen between Japonica and Indica cultivars. The OsLEA12 gene was highly associated with the rhizosphere bacteria to endow the rice salt stress tolerance ability. Our results demonstrate that rice can control the assembly of the root-associated functional microbiomes, providing insights for breeding microbiome-assisted crops in modern agriculture.

The mechanism of precise regulation of soybean nodulation, resistance and yield by a domesticated transcription factor GmRR2b

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Soybean, as one of the most significant oil crops, has undergone extensive artificial selection for various traits such as flowering time, pod shattering, seed hardness, blooming, oil content, and dormancy during its domestication process. However, it remains uncertain whether soybean nodulation, a special kind of immune response, has also been subject to artificial selection. Here we employ a bottom-up approach to identify a transcription factor in cytokinin signal pathway, GmRR2b, in the soybean genome that has been selected during soybean domestication and improvement based on its transcriptional activity, which is determined by a various-length InDel in its promoter. GmRR2b precisely orchestrates soybean yield and disease resistance by controlling reactive oxygen species (ROS) burst, as well as negatively regulates soybean nodulation. In human-constructed agroecosystems aimed at maximizing yield, soybean nitrogen fixation might play only a minor role compared with chemical fertilizer inputs. However, when diseases strike the agricultural system, the balance between yield and disease resistance becomes critically important, and the plant's own nitrogen fixation may serve as a shortcut to achieving this equilibrium. This study introduces a novel paradigm for analyzing the mechanisms that maintain the balance between legume nodulation, pathogen resistance and yield under varying conditions, and offers fresh insights into developing environmentally resilient crops capable of adapting to global climate change.

Key words: artificial selection; crop domestication; cytokinin; nodulation; Glycine max; soybean yield; growth-defense trade-off

A novel family of polysaccharide lyase proteins are deployed in rice to negatively control arbuscular mycorrhizal symbiosis

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As the core structure of arbuscular mycorrhizal (AM) symbiosis, arbuscules exhibit an unusually short lifespan, yet the underlying mechanisms remain largely obscure. In this study, a novel family of bacteria-originated polysaccharide lyases (designated as chitin-binding and lyase protein, CBLPs) was investigated in rice. After inoculation with the AM fungus Rhizophagus irregularis, OsCBLP1 was specifically induced in arbusculated cells while OsCBLP2 was expressed in both arbusculated and adjacent cells. Loss-of-function of either OsCBLP1 or OsCBLP2 results in enhanced root colonization, and the double mutant oscblp1oscblp2 exhibits an even more exaggerated phenotype, supporting that the two OsCBLPs exert additive effects on negatively controlling AM symbiosis. Overexpression of OsCBLPs significantly decreased arbuscular abundances in rice and Medicago truncatula roots. Both OsCBLPs were localized to the apoplastic space after plasmolysis, and purified OsCBLP1 can not only efficiently bind and lyse chitin, but also significantly inhibit hyphal elongation of germinated R. irregularis spores. Moreover, OsCBLP2 was induced in rice leaves during symbiosis and could confer effective resistance to blast fungi. Together, this work reveals a common set of polysaccharide lyase genes that are used by plants to control both symbiotic and pathogenic fungi, which also indicates that arbuscule degradation is likely a consequence of active host defense.

Mobile DELLA proteins control ground tissue development to host arbuscular mycorrhiza

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Most land plants can form a symbiosis with arbuscular mycorrhizal (AM) fungi to acquire sufficient nutrients from the soil. During this symbiosis the fungi are hosted as highly branched hyphal structures, called arbuscules, that form a symbiotic interface across which nutrients such as phosphate and nitrogen are exchanged for carbon from the plant. Most plants have multiple cortex layers yet arbuscule formation typically occurs only in the root inner cortex cells, but not in the more outer cortex layers. To investigate what makes inner cortex cells special that they can host arbuscules we focused on the role of DELLA proteins in Medicago truncatula. We demonstrate that DELLA-controlled formation of the AM susceptible inner cortex in Medicago already occurs at the root stem cell niche, in a dose-dependent manner. Genetic analyses suggest that DELLA converges with the well-known ground tissue developmental regulator SHORTROOT to control the number of inner cortex layers. Furthermore, we show that DELLA, like SHORTROOT is mobile and that it moves from the stele/endodermis to the cortex to facilitate arbuscule formation in the mature part of the root. We propose a different identity of outer and inner cortical cell layers, which determines whether AM fungi can be hosted or not.

Wolbachia enhances ovarian development in the rice planthopper Laodelphax striatellus through elevated energy production

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The endosymbiont Wolbachia can both benefit host nutrition and manipulate host reproduction to its own advantage. However, the mechanisms of its nutritional benefits remain unclear. We show that Wolbachia enhances ovarian development in the small brown planthopper Laodelphax striatellus by boosting energy production. Wolbachia-infected females have increased fecundity, accelerated ovarian development, and prolonged oviposition. Enhanced activity of mitochondrial complex I is linked to increased ATP production and the expression of energy metabolism-related genes. We further identify that Wolbachia-synthesized riboflavin is crucial for ATP production and ovarian development. A riboflavin transporter, slc52a3a, positively correlates with Wolbachia density and is required for normal ovarian maturation. In the meantime, we found that LsAtg8 silencing increased Wolbachia levels in embryos and altered its dynamics of density during the development of nymphs without affecting the lifespan or reproductive capacity of the offspring. Our findings demonstrate that Wolbachia-produced riboflavin drives energy production and accelerates ovarian maturation, thus improving host fecundity. This research reveals insights into symbiont-host metabolic interactions and underscores the role of nutrient delivery in symbiosis, and demonstrates that Wolbachia enhances ovarian development and fecundity in the small brown planthopper by synthesizing riboflavin, which drives ATP production and accelerates ovarian maturation. These findings also suggest a multifaceted role for Atg8 in the host–symbiont system and provide insights into pest management strategies targeting endosymbionts.
Keywords: Wolbachia; small brown planthopper; reproduction; pest management

The symbiotic bacteria regulate host reproduction

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The whitefly Bemisia tabaci MED and MEAM1 are two of the most significant and invasive pests in the global agriculture. These whiteflies carry the obligate symbiont Portiera and Hamiltonella in their specialized host cells known as bacteriocytes and harbor Rickettsia in their whole bodies, except bacteriocytes. Hamiltonella induces a female-biased sex ratio in the whitefly B. tabaci. In contrast, Rickettsia influences both fecundity and sex ratio of the whitefly B. tabaci. Encarsia formosa wasps have been used globally for the control of whiteflies and cannot reproduce sexually and rely on obligate parthenogenesis induced by Wolbachia. We revealed that the bacteriocyte symbiont Hamiltonella regulates whitefly sex ratio by modulating the level of histone methylation modifications and the maternal protein Tudor-mediated piRNA pathway. We found that Rickettsia results in increased whitefly fecundity and female-bias by stimulating juvenile hormone synthesis, and it has been integrated into metabolic collaboration between whitefly and obligate symbiont Portiera for synthesis of essential amino acids, which benefits whitefly fecundity. We identified a Wolbachia parthenogenesis-induction feminization factor gene (piff) that targets sex-determining genes and causes female-producing parthenogenesis in the haplodiploid parasitoid E. formosa. Collectively, these findings show that the symbiotic bacteria play an important role in regulating host reproduction.

A quantitative microbiome atlas reveals coevolutionary trade-offs and eukaryotic keystones in plant rhizospheres

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The evolutionary success of land plants is tied to their rhizosphere microbiomes. However, the principles governing how bacteria, fungi, and protists assemble and function across wild plants remain unresolved. Most studies focus only on bacteria in model crops and rely on relative rather than absolute abundance data, obscuring true interkingdom dynamics. Here, we present the One Thousand Plant Microbiomes (1KPM) initiative, a quantitative, multi-kingdom atlas of 1,549 rhizospheres from 590 wild plant species, constituting the largest and most phylogenetically diverse multi-kingdom rhizosphere microbiome atlas to date. We performed spike-in-based quantitative microbiomes profiling and ultra-deep metagenomics, revealing that over half of core bacterial functional genes are phylogenetically conserved across host lineages, alongside unprecedented genomic novelty. We also identified hundreds of phylogenetically conserved microbial functions that coevolved with diverse plant lineages, and uncovered an evolutionary paradox in Fabales, where low taxonomic but high functional diversity reflects symbiotic optimization. Despite bacterial dominance, protists and fungi emerge as keystone hubs of rhizosphere stability, as confirmed by experimental removal of each kingdom. This atlas redefines microbiome assembly rules and establishes rhizospheres as untapped frontiers for microbiome engineering.

Re-evaluating root lesion nematode disease in pepper: the Pratylenchus capsici–microbiome disease complex, their interactions, and temperature-driven dynamics

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ARO VOLCANI

Rising soil temperatures associated with climate change are expected to alter rhizosphere microbial communities, with important consequences for plant–nematode interactions. Here, we investigated temperature-mediated changes in root- and rhizosphere-associated microbial communities in pepper (Capsicum annuum) in response to infection by the root lesion nematode Pratylenchus capsici, using high-throughput metabarcoding. Pepper plants were grown under controlled soil temperatures of 25°C, 30°C, and 33°C, and root tissues and rhizosphere soils were sampled separately. Fungal and bacterial communities were characterized using ITS and 16S rRNA gene sequencing, respectively. In parallel, nematode communities were analyzed using nematode-specific primers to assess plant-parasitic and free-living nematode assemblages. Distinct microbial assemblages were observed between roots and soils across temperatures. Root-associated fungi were dominated by Rhizophagus, with Olpidium consistently enriched in roots and showing higher abundance at 25°C. Co-occurrence analysis revealed an asymmetric interaction in which Pratylenchus was negatively affected by Olpidium occurrence. Soil fungal communities were more diverse, dominated by Aspergillus, Penicillium, Fusarium, and Rhizophagus, showing minimal temperature effects. Root-associated bacteria were dominated by rhizobia and Pseudomonas, with moderate abundances of other genera, while soil bacterial communities were more diverse, with Bacillus as the most prominent genus. Overall, increasing soil temperature reshapes microbial communities in a compartment-specific manner, and asymmetric nematode–fungus interactions may modulate disease development. These findings provide a foundation for climate-smart, microbiome-informed management of root lesion nematodes in pepper.

Influences of Mosquito Gut Microbiota on the Midgut Microenvironment

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Mosquitoes are vectors of major diseases such as malaria and dengue fever, and understanding the mechanisms governing their pathogen transmission capacity will lay the foundation for developing novel mosquito control strategies. The mosquito gut harbors a diverse array of commensal microbiota; however, how these microbes influence the midgut microenvironment and subsequently impact pathogen transmission remains poorly understood. Using the Plasmodium-mosquito system, our study revealed that Anopheline gut bacteria regulate midgut pH and promote the formation of the physical barrier by participating in glucose metabolism, tryptophan metabolism, and immune responses, thereby affecting Plasmodium transmission. These findings broaden our understanding of the tripartite interactions among mosquitoes, gut microbiota, and Plasmodium.

Parasitic castration by a viral protein tyrosine phosphatase targeting the host cell cycle checkpoint protein Rad9A

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Parasitic castration is a widespread strategy where parasites hijack host reproductive resources, yet the key molecular mechanisms driving this phenomenon remain poorly understood. Bracoviruses (BVs), derived from ancestral nudiviruses, are domesticated endogenized viruses (DEVs) that have become permanently integrated into the genome of braconid wasps, most of which parasitize lepidopteran insects. Previous studies have shown that BV is a crucial factor that hinders the development of testes and spermatogonia, leading to parasitic castration in their lepidopteran hosts. However, it is yet unknown which virulence gene is responsible for this phenomenon. Here, we report a strategy whereby parasitic wasps employ their domesticated endogenized virus to induce apoptosis in larval testes of their lepidopteran hosts. A testis-highly expressed viral PTP designated CvBV_22-9 is involved in this phenomenon. Further experiments demonstrated that CvBV_22-9 targets a cell cycle checkpoint protein, Rad9A, without affecting its tyrosine phosphorylation status. Crucially, Rad9A knockout causes embryonic lethality and severe male infertility. Validation in both FAW and Drosophila confirms that CvBV_22-9/Rad9A-mediated testicular apoptosis is evolutionarily conserved in insects. Our findings reveal that parasitoids likely exploit a conserved pathway to induce testicular castration.

Exploration of Symbiotic Nitrogen Fixation

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Symbiotic nitrogen fixation (SNF) is one of the most important biological processes supporting sustainable agriculture, yet its genetic, cellular, and regulatory complexity remains only partially understood. Our research aims to dissect the molecular basis of legume–rhizobium symbiosis by integrating comparative genomics, pangenomics, single-cell and spatial omics, epigenomics, and functional genetics.

In soybean, we constructed genomic resources from diverse tropical and subtropical accessions to investigate structural variation and its contribution to agronomic and symbiotic traits. These analyses identified large chromosomal rearrangements associated with extensive reorganization of chromatin architecture and regulatory landscapes. One such region led to the discovery of SONAR, a lineage-restricted gene generated through exon reshuffling. Functional analyses indicate that SONAR modulates nodulation and symbiotic performance and is also involved in plant responses to heat stress, illustrating how recently evolved genomic features may connect symbiosis with environmental adaptation.

At the cellular level, comparative single-cell analyses across legumes reveal both conserved and species-specific transcriptional programs during nodule development. In Lotus japonicus, single-nucleus transcriptomics identified a rare early symbiotic signaling cell state and enabled the discovery of previously uncharacterized regulators of rhizobial infection and nodule formation. We are further integrating chromatin accessibility, histone modifications, DNA methylation, and long-range chromatin interactions to define regulatory elements controlling nodule development.

Finally, through large-scale comparative genomics across Fabaceae, we are investigating the evolution, conservation, and loss of nodulation-associated genes and regulatory programs. These efforts aim to define the minimal genetic and regulatory requirements for nodulation and ultimately provide a framework for engineering symbiotic nitrogen fixation in non-legume crops

Optimizing Mineral Nutrition to Improve N fixation in Soybean

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Symbiotic nitrogen fixation between legume plants and rhizobia provides a natural and sustainable nitrogen source in agriculture. The nutrient exchange between legumes and rhizobia forms the foundation of symbiotic nitrogen (N) fixation. However, besides providing the carbon source required for N fixation, plants also need to supply essential mineral nutrients for rhizobial growth. Deficiency in mineral nutrients can impede root nodule development and decrease N fixation capacity. However, the specific roles of these mineral nutrients in symbiotic N fixation are not well understood. In soybean root nodules, we discovered that magnesium accumulates in the inner cortex of nodules primarily through the magnesium transporters MGT4/5. This magnesium, by regulating plasmodesmata permeability, controls the C-N exchange in nodules. Iron accumulates in the symbiosome of N-fixing zone primarily through the iron transporter VTL1a, where it serves as a component of nitrogenase, facilitating nitrogen fixation. Under iron-deficient conditions, soybeans can enhance iron transport from uninfected cells to infected cells through NRAMP2, meeting the iron demands of symbiotic nitrogen fixation. Sulfur accumulates in the N-fixing zone of nodules primarily through the sulfur transporters SULTR2.1 and SULTR3.5, and this sulfur is crucial for nodule redox balance. Insufficient sulfur in nodules leads to symptoms such as green nodules and premature senescence. By supplying these minerals to soils, we can effectively cut down chemical N fertilizer use while increasing N fixation ability, offering a promising approach to soil nutrient management.

Mosquito Microbiota and Implications for Disease Control

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The stalling global progress in the fight against malaria prompts the urgent need to develop new intervention strategies. Blocking malaria parasites in the vector mosquito before they are transmitted to humans is a promising strategy to prevent malaria transmission. As part of this effort, we have been developing a promising strategy for blocking the transmission of mosquito-transmitted diseases via populating mosquitoes with anti-pathogen bacteria, known as paratransgenesis or symbiont-based transmission blocking strategy. We demonstrated that engineered symbiotic bacterium Serratia AS1 that secretes anti-Plasmodium effector molecules effectively render mosquitoes resistant to Plasmodium infection. More recently, we identified a natural Plasmodium-blocking symbiotic bacterium, Serratia ureilytica Su_YN1, isolated from the midgut of wild Anopheles sinensis in China that directly inhibits Plasmodium falciparum via secretion of a lipase. We further found Su_YN1 delivers the effector lipase AmLip to Plasmodium parasites via outer membrane vesicles (OMVs), leading to targeted killing of the parasites. Notably, Su_YN1 OMVs incorporate certain serum-derived phospholipids that are important for OMV uptake by Plasmodium parasites via the phosphatidylcholine scavenging pathway, uncovering a novel mechanism by which gut commensal bacteria deliver effector molecules via OMVs to selectively attack the parasites and thus render mosquitoes refractory to malaria parasite infection. We also found that blood meal activates the mosquito symbiotic bacterium Su_YN1 quorum sensing system, inducing the synthesis of C6-HSL molecules that drive OMV biogenesis by activating phenylalanine metabolism. The abundant OMVs facilitate bacterial biofilm-like aggregation and promote Serratia survival and persistence in the gut of blood-fed mosquitoes. Importantly, Su_YN1 rapidly disseminates through mosquito populations by vertical and horizontal transmission, providing a potential tool for blocking malaria transmission in the field.

Genetic Control of Maize Rhizosphere Microbiome Assembly

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Plant-associated microbiomes are increasingly recognized as an extended component of the plant phenotype, yet the genetic mechanisms by which host plants shape rhizosphere microbial communities remain insufficiently understood. Maize provides a powerful system to dissect the genetic basis of microbiome assembly because of its extensive genetic diversity, well-characterized genomic resources and pronounced variation in root architecture, metabolism and environmental responses. Our research integrates maize genetics, quantitative root phenotyping, transcriptomics, metabolomics and microbiome profiling to determine how host genetic variation regulates rhizosphere microbial assembly. Using diverse maize inbred lines, hybrids and mapping populations, we identify reproducible genotype-dependent microbial signatures and link microbial variation to root traits, host gene expression and rhizosphere metabolites. These analyses reveal that host effects on the microbiome are strongly context dependent and are particularly pronounced under nutrient limitation and other environmental stresses. Studies of maize heterosis further demonstrate that hybridization modifies rhizosphere community assembly and that microbial responses are closely associated with heterotic variation in root development. Importantly, functional experiments indicate that these relationships extend beyond correlation. Genetic perturbation of host metabolic pathways, including flavonoid biosynthesis, substantially alters microbial recruitment and plant responses. In parallel, bacterial isolates such as Massilia provide tractable systems for experimentally testing causal relationships between host genotype, microbial colonization and root phenotypes. Together, our results support a model in which maize genetic variation shapes rhizosphere microbiome assembly through coordinated changes in root developmental programs and metabolic outputs. Resolving this genetic architecture provides a foundation for moving from descriptive microbiome profiling toward mechanistic understanding and, ultimately, genetically informed manipulation of beneficial plant-microbiome interactions.

Bacterial allies restore predator control of soil-borne pathogens

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Predators are expected to suppress soil-borne pathogens through top-down control, yet widespread disease outbreaks show that pathogens can evade this regulation. What determines the balance between predator control and pathogen escape remains unclear. Here, we show that the globally distributed soil-borne pathogen Ralstonia pseudosolanacearum impairs nematode predators by producing extracellular polysaccharides that obstruct the pharyngeal lumen and disrupt feeding, thereby weakening top-down control. In response, nematodes shift their foraging toward protective bacteria that counteract pathogen virulence and restore predator feeding and survival. This behavior shift is transient and experience-dependent. Once predator function is restored, nematodes suppress pathogens primarily by reshaping resident bacterial communities rather than through direct grazing. These findings reveal self-organized multitrophic interactions among soil fauna and microbes that emerge under pathogen pressure, expanding our understanding of how biodiversity supports disease suppression.

Molecular Mechanisms Underlying the Efficient Transmission of Southern Rice Black-Streaked Dwarf Virus (SRBSDV) by Its Insect Vector

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Southern rice black-streaked dwarf virus (SRBSDV) is one of the most destructive rice viruses and is transmitted by the white-backed planthopper (WBPH, Sogatella furcifera) in a persistent, circulative, and propagative manner. Its efficient transmission relies on sophisticated molecular interactions among the virus, rice host, insect vector, and vector-associated microbiota. This report integrates recent studies to present a multi-layered mechanistic framework. First, SRBSDV manipulates both host plant physiology and vector behavior to optimize viral acquisition and inoculation. The viral protein P6 regulates rice ethylene signaling in a spatiotemporally distinct manner: cytoplasmic P6 interacts with OsRTH2 during early infection to activate ethylene signaling, promoting viral accumulation while repelling WBPH; nuclear P6 binds OsEIL2 during late infection and blocks its dimerization, suppressing ethylene signaling and attracting WBPH. In parallel, the viral protein P8 directly reprograms the olfactory system of WBPH by competitively binding Pelle, a core Toll pathway kinase, thereby inhibiting DIF phosphorylation and nuclear translocation. This downregulates the E-β-farnesene receptor Or86 and upregulates the β-caryophyllene receptor Or127, shifting viruliferous insects from infected to healthy rice plants. Second, P6 undergoes liquid-liquid phase separation and forms P6-OsTSN1 droplets that co-localize with stress granules. These droplets promote OsTSN1 multimerization and nuclease activity, accelerating the degradation of OsNAC15 and OsLHY transcripts and weakening jasmonate- and autophagy-associated antiviral defenses. Third, the endosymbiont Wolbachia acts as a physical carrier that helps SRBSDV breach the salivary gland barrier. High-transmission field populations harbor high Wolbachia titers; Wolbachia depletion delays virus entry into salivary glands, while repletion restores transmission. The Wolbachia surface protein WSP directly binds the SRBSDV capsid protein P8, and Wolbachia envelops virions during salivary gland crossing. Anti-WSP antibodies reduce salivary gland viral load and transmission efficiency. Collectively, these findings reveal that efficient SRBSDV transmission is achieved through coordinated viral manipulation of rice immunity and ethylene signaling, direct reprogramming of vector olfaction, phase-separation-mediated suppression of host defense, and symbiont-assisted tissue barrier crossing. These mechanisms provide potential targets for sustainable control of SRBSDV and other vector-borne rice viruses.

Phylogenomic study revealed stepwise evolution of the root nodule symbiosis

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Some plants in ten families of the nitrogen-fixing clade of angiosperms form root nodules through symbiosis with nitrogen-fixing bacteria, converting atmospheric inorganic nitrogen into ammonia nitrogen that plants can assimilate. This process plays a critical role in the nitrogen cycle of terrestrial ecosystems. However, whether root nodule symbiotic nitrogen fixation arose once and was subsequently lost multiple times, or arose independently on multiple occasions, remains controversial, and the formation and evolutionary trajectory of the gene regulatory network underlying symbiotic nitrogen fixation are also unclear. By whole-genome sequencing of representative species from ten key nitrogen-fixing lineages, coupled with comparative genomic and phylogenetic analyses of 194 published genomes, this study has constructed, for the first time at the single-gene level, the evolutionary architecture of the gene regulatory network for root nodule symbiosis. The key breakthrough lies in revealing that three 'gene innovations' gave rise to the capacity for nodulation and symbiotic nitrogen fixation. First, the gamma (γ) paleohexaploidy event in core eudicots generated numerous duplicated genes, thereby providing the genetic raw material for subsequent functional differentiation and neofunctionalisation of nodulation-inducible genes and key nitrogen-fixation genes (e.g. NIN, CNGCa, NFP). Second, the common ancestor of the nitrogen-fixing clade modularly recruited and rewired genes from the arbuscular mycorrhizal symbiosis, nitrate response and stress response pathways, thus establishing an initial gene regulatory network for symbiotic nodulation and nitrogen fixation. Finally, legumes evolved efficient root nodule symbioses through convergent evolution, with two key genes, NPL and SymSTK, playing decisive roles. This study not only reconciles the 'single-origin' and 'multiple-origin' debates at the level of gene regulatory network evolution, but also systematically compiles a core 'gene inventory' of the nodule symbiosis network, thereby providing precise targets for engineering nitrogen fixation into non-legume crops.
Keywords: root nodule symbiotic nitrogen fixation; nitrogen-fixing clade; gene regulatory network; symbiosome

A MAP for rhizobial infection in root nodule symbiosis

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Legumes and nitrogen-fixing rhizobia form root nodule symbiosis, which provides an important sustainable nitrogen source for agriculture. A key process in this endosymbiosis is the formation and development of intracellular infection structures, called infection threads (IT), initiating in the root hair and ramifying in developing nodules, for the continuous delivery of rhizobia into root nodule cells. Root hair infection by nitrogen-fixing rhizobia triggers subcellular remodeling in legume hosts, such as cell wall reconstruction, cytoskeleton rearrangement and polarized exocytosis. The legume “infectosome” protein complex is required for IT development but the underlying mechanisms are unclear. Here, we show that a pair of Medicago truncatula Microtubule-Associated Protein 70-5 (MAP70-5) homologs switch their microtubule localization to an infectosome-like punctate pattern upon rhizobial infection. Unlike the bipolar localization of known infectosome components such as VAPYRIN and Lumpy Infections, MAP70-5A/B co-localize only with perinuclear infectosome and interact with Rhizobium-directed Polar Growth (RPG) therein. We further show that the punctate localization of MAP70-5A/B during symbiosis is dependent on RPG. The map70-5a map70-5b mutants exhibit defective infection threads and the organization of endoplasmic microtubules connecting the IT tip and the nucleus is disrupted in map70-5a map70-5b. Our results reveal the function of MAP70-5A/B in the legume-rhizobial symbiosis, where they are recruited by RPG for patterning microtubules to drive successful IT progression. This work provides insights into how symbiotic specific components integrate with and remodel microtubule cytoskeleton in orchestrating the development of intracellular infection structures in root nodule symbiosis.

A plant virus manipulates both its host plant and the insect that facilitates its transmission

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Tomato yellow leaf curl virus (TYLCV), a devastating pathogen of tomato crops, is vectored by the whitefly Bemisia tabaci, yet the mechanisms underlying TYLVC epidemics are poorly understood. Previous studies have shown that TYLCV affects the foraging behavior of the whitefly in a manner that facilitates transmission but the underlying mechanism has been unclear. Here, we demonstrated that TYLCV infection induces the expression of TPS3 and TPS7 in tomato plants, resulting in the biosynthesis and release of β-myrcene which renders the plants attractive to non-viruliferous whiteflies. We demonstrated this in behavioral assays in the laboratory as well as trapping experiments in greenhouses. Importantly, we found that TYLCV also manipulates the odor perception of the whitefly by suppressing the expression of BtMEDOR6, which we identified as a specific olfactory receptor gene for β-myrcene. Our study shows that variations in the amino acid at position 203 of BtOR6 determines the binding capacity of the receptor for β-myrcene, which explains the strong attraction of only the cryptic species B. tabaci MED to TYLCV-infected tomatoes. Further research has revealed that TYLCV suppresses OR6 expression by activating the transcription factor ACJ6. The R50 site of the TYLCV V2 protein directly binds to ACJ6 and activates transcription. ACJ6 then binds to the -1177 site of the OR6 promoter, thereby suppressing OR6 transcription. TYLCV, by repressing the expression of BtMEDOR6, cancels the preference of viruliferous B. tabaci MED for TYLCV-infected tomatoes thus promoting virus transmission to non-infected tomatoes. These findings reveal a sophisticated viral strategy whereby TYLCV modulates both host plant attractiveness and vector olfactory perception to enhance its spread.

Host and microbial control of arbuscular mycorrhiza symbiosis through peptide hormones

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Arbuscular mycorrhiza (AM) symbiosis is a mutually beneficial interaction between plants and soilborne fungi of the Glomeromycotina subphylum. In this association, fungi provide mineral nutrients in exchange for plant-derived carbon, requiring tight control to maintain a balanced mutualism. Because AM fungi are obligate biotrophs and fully dependent on host-derived carbon, most research to date has focused on how plants regulate symbiosis. For example, the host controls the interaction through complex signaling networks that integrate symbiosis status and nutrient demand across colonized root cells and distal tissues. By contrast, the role of the fungal partner in regulating symbiosis has received much less attention. Our work shows that AM fungi actively interfere with endogenous host control pathways for their own benefit.

In summary, we found that several genes encoding secreted CLE (CLAVATA3/EMBRYO SURROUNDING REGION-related) peptides are upregulated in Medicago truncatula roots during AM symbiosis. The AM-induced MtCLE53 acts as a negative autoregulatory signal that restricts fungal colonization through the shoot-acting receptor-like kinase SUNN. We further found that the pseudokinase CORYNE, known to form a complex with SUNN, also functions as a negative regulator of AM symbiosis. However, CORYNE is only partially required for MtCLE53 signaling, suggesting that it integrates multiple signaling pathways. Consistent with this, our data indicate that CORYNE is part of an as-yet-unknown receptor complex that perceives a distinct symbiosis regulator, MtCLE16. In contrast to MtCLE53, MtCLE16 is induced in colonized cortex cells and promotes AM symbiosis by dampening host stress and defense responses. We hypothesize that MtCLE16 acts as a local signal amplifier that enhances symbiosis, in stark contrast to previously characterized plant CLE peptides, which predominantly function as negative regulators in this context.

Intriguingly, some AM fungi also produce signaling peptides resembling those of plants, including CLE-like peptides. Functional analysis of a CLE-like peptide from Rhizophagus irregularis shows that it signals through an as-yet-unknown host receptor complex involving CORYNE to manipulate host responses for microbial benefit. Together, our findings suggest that host and microbial control of AM symbiosis are highly intertwined, with AM fungi mimicking symbiosis-promoting CLE peptides to co-opt host signaling pathways for their own benefit.

Host genetic background shapes the dynamics and phenotypic effects of Rickettsia symbiosis in Bemisia tabaci

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The whitefly Bemisia tabaci harbors a diverse community of facultative bacterial symbionts that can profoundly influence host biology, adaptation, and vector competence. Among these, Rickettsia displays strikingly different infection dynamics across B. tabaci genetic backgrounds and geographic populations. Approximately two decades ago, we detected Rickettsia at frequencies of about 70% in both MEAM1 (formerly B biotype) and MED (formerly Q biotype) populations in Israel. Long-term monitoring, however, revealed strikingly divergent trajectories: Rickettsia progressively disappeared from MEAM1 populations while becoming fixed in MED. Different temporal and geographic patterns have been observed elsewhere. Rickettsia has reached high frequencies in MEAM1 populations in both China and the USA, including rapid spread to fixation in some populations, while more recently it has also increased substantially in some MED populations in China. These contrasting patterns indicate that the dynamics of Rickettsia infection are context dependent and may reflect complex interactions among symbiont genotype, whitefly genetic background, and environmental and ecological factors. Comparative genomic analyses reveal only minor differences among Rickettsia genomes associated with MEAM1 and MED, suggesting that major bacterial genomic divergence alone is unlikely to explain the contrasting infection dynamics. Understanding these interactions is particularly important because Rickettsia has been associated with several ecologically relevant host phenotypes, including thermal tolerance, reproductive traits and sex-ratio bias, and enhanced transmission of Tomato yellow leaf curl virus (TYLCV). To directly investigate the contribution of host genetic background to these interactions, we used a microinjection-based approach that enables experimental transfer of Rickettsia between MEAM1 and MED whiteflies. Reciprocal transinfection lines are being used to determine how introduction of the symbiont into a novel host genetic background affects its establishment, transmission, density, tissue distribution, and effects on whitefly fitness and virus-vector interactions. This experimental system provides a unique opportunity to disentangle host- and symbiont-dependent components of the Rickettsia–Bemisia association and to identify mechanisms underlying the contrasting ecological and evolutionary trajectories of this widespread facultative symbiosis.
Key words: Bemisia tabaci; Rickettsia; facultative symbionts; host genetic background; transinfection; vector competence

ICBPI 2026 · OCTOBER 18–22, 2026

Session V

Plant Resistance and Molecular Design Breeding

植物抗性与分子设计育种

Wheat autoimmunity: a new frontier for multiple-disease resistance

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Wheat (Triticum aestivum L.) is a major staple crop worldwide, and developing durable, broad-spectrum resistance to multiple fungal pathogens is essential for sustainable production. Wheat autoimmunity (WAI) mutants represent a unique class of genetic resources that spontaneously activate immune responses under non-stress conditions, resulting in lesion-mimic or leaf-tip necrosis phenotypes while conferring broad-spectrum resistance to key foliar diseases, including powdery mildew and stripe, leaf, and stem rusts. Increasing evidence indicates that WAI arises from dysregulation of immune homeostasis across multiple regulatory levels, involving receptor activation, cellular homeostasis, and membrane transport processes, ultimately converging on evolutionarily conserved defense outputs such as reactive oxygen species (ROS) accumulation, activation of defense-related gene, ion fluxes, and programmed cell death (PCD). Recent advances in gene cloning and structural biology have identified key WAI-associated loci, including nucleotide-binding and leucine-rich-repeat receptors (NLRs), membrane proteins, regulatory components, and transporters, revealing mechanistically distinct pathways underlying constitutive immunity and its trade-off with growth. I am going to present our recent works in the genetics and molecular mechanisms characterization of wheat autoimmunity, critically assess its roles in broad-spectrum, multi-disease resistance, and propose strategies to genetically decouple disease resistance from growth penalties for the rational engineering of WAI alleles.

A tomato lectin receptor-like kinase senses bacterial lipopolysaccharide to activate plant immunity

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Lipopolysaccharide (LPS) is a major component of the outer membrane of Gram-negative bacteria and elicits plant immune responses. The Arabidopsis lectin receptor-like kinase LORE, a receptor for medium-chain 3-hydroxy fatty acids, contributes to LPS-induced sustained production of reactive oxygen species (ROS). LORE also recruits and phosphorylates the protein phosphatase LOPP, which in turn dephosphorylates LORE to attenuate immune signaling. Loss of LOPP enhances resistance to bacterial wilt in both Arabidopsis and tomato. These findings raise the question of whether a related receptor directly perceives LPS in tomato. Here, we identify LIPOPOLYSACCHARIDE SENSOR 1 (LIPS1), a tomato lectin receptor-like kinase that directly binds LPS and activates plant immunity. LPS induced ROS production, defense gene expression, and callose deposition in tomato leaves and roots, whereas the medium-chain 3-hydroxy fatty acid tested did not elicit these responses under the same conditions. Loss of LIPS1 compromised LPS-induced immune responses and increased susceptibility to Pseudomonas syringae pv. tomato DC3000 and Ralstonia solanacearum. Conversely, LIPS1 overexpression enhanced LPS responsiveness and resistance to both pathogens, with no detectable adverse effects on the growth and yield traits examined. Surface plasmon resonance analysis demonstrated direct binding between LIPS1 and LPS. Furthermore, LPS treatment enhanced LIPS1 phosphorylation and self-association, consistent with ligand-induced receptor activation. Together, these findings identify LIPS1 as a sensor of bacterial LPS that activates plant immunity, advance our understanding of LPS perception in plants, and highlight a potential target for improving bacterial disease resistance in tomato.

Systematic identification of E3 ubiquitin ligase–substrate networks in rice immunity against Magnaporthe oryzae via the UbE3 library

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The ubiquitin–proteasome system (UPS) plays a critical role in the interaction between rice and the blast fungus Magnaporthe oryzae. However, the function of E3 ubiquitin ligases in rice immunity remains largely unknown. In this study, we found that the M. oryzae effector AvrPiz-t targets the rice RING-type E3 ligases APIP6 and APIP10, thereby suppressing host immunity. APIP6 mediates the degradation of OsELF3-2, while APIP10 promotes the degradation of OsVOZ1/2. These proteins act as negative regulators of Piz-t-mediated resistance. Due to the difficulty of identifying E3 ligase substrates, we created a complete rice E3 ligase ORFeome library (named UbE3) using PCR amplification and gene synthesis. Screening with this library enabled the systematic discovery of multiple E3–substrate pairs. Firstly, we found that the F-box E3 ligase OsFBK16 targets phenylalanine ammonia-lyase family members (OsPALs), which are key enzymes in the phenylpropanoid pathway. Secondly, we discovered that the RING-type E3 ligase OsRING113 regulates the transcription factor APIP5, which is a known suppressor of cell death and immunity. Thirdly, the U-box E3 ligase OsPUB45 was shown to ubiquitinate COP9 signalosome subunit 5 (CSN5), which is a core component of the signalosome complex. Most recently, we discovered that the U-box E3 ligase OsPUB20 modulates the 14-3-3 proteins OsGF14f and OsGF14c, thereby expanding the regulatory network to include signaling scaffold proteins. Notably, these substrates span diverse functional classes, from metabolic enzymes and transcription factors to signalosome subunits and scaffold proteins, suggesting that E3 ligases orchestrate rice immunity through the regulation of protein homeostasis at multiple levels. Collectively, our findings demonstrate the power of the UbE3 library as a versatile tool for substrate discovery, and unveil novel mechanisms of protein homeostasis governed by ubiquitination of key disease resistance components in rice.

Pikobodies: bioengineering of ligand recognition in the Pik NLR pair

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Plant pathogens rapidly evolve virulent races that can nullify newly deployed resistance specificities, leading to significant crop yield reduction and threatening global food security. There thus is a need to develop made-to-order synthetic plant immune receptors with resistance tailored to the pathogen genotypes present in the field. Pikobodies are based on bioengineering rice Pikm-1/Pikm-2 NLR immune receptor pair via swapping the integrated HMA domain of Pikm-1 for VHH nanobodies that can bind virtually any protein. We show that Pikobodies (Pikm-1VHH/Pikm-2) engineered with nanobodies that bind fluorescent proteins are specifically activated in presence of their matching targets and confer resistance to plant viruses expressing fluorescent proteins. Furthermore, we show that Pikobodies form a pre-activation complex in Nicotiana benthamiana in the absence of their targets. This suggests that the mode of activation of the Pik NLR pair is different from the activate-and-release model described for other pairs of sensor/helper NLRs. The Pik NLR pair pre-activation complex indicates that Pikobodies form a single unit potentially transferrable to any plant taxa. This model implies that Pikobodies can have wide applications in bioengineering disease resistant crops.

MicroRNA156-IPA1/OsSPL14 integrate GA and SA to boost rice immunity

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IDEAL PLANT ARCHITECTURE1 (IPA1, also named OsSPL14), a target gene of microRNA156 (miRNA156), is a master regulator of rice development. Moderately upregulating of IPA1 reduces inefficient tiller number, promotes panicle branching, and strengthens stem. IPA1 as a new “green revolution” gene, greatly improve rice yield. We previously found that down-regulation of miR156 or overexpression of IPA1 greatly increase disease resistance against bacterial blight caused by Xanthomonas oryzae pv. oryzae (Xoo). IPA1 physically interact with SLR1, the solely DELLA protein in rice, and stabilize SLR1. The disease resistance and retarded growth by overexpression of IPA1 or downregulation of miR156 is partially dependent on SLR1.

In this study, we found that IPA1 physically interact with NON-EXPRESSOR OF PATHOGENESIS-RELATED GENES 1 (OsNPR1), a putative receptor of salicylic acid (SA) in rice. Under normal conditions, OsNPR1 protein levels are low because of OsCULLIN3a (OsCUL3a)-mediated ubiquitination and degradation of OsNPR1. Thus, limits IPA1’s transcriptional ability on defense-related genes. Upon pathogen infection, OsNPR1 in monomer translocates into nucleus, where it associates with IPA1. The OsNPR1-IPA1 physical interaction, on one hand, facilitates IPA1’s binding capacity on the promoters of target genes. OsNPR1 both enhance the activating and repressing activity of IPA1 on transcription. RNA-seq analysis revealed that OsNPR1 is required for IPA1 transcriptional regulation on 32.1% of up-regulated genes and 57.1% downregulated genes. The interaction of OsNPR1 and IPA1, on another hand, interferes with the interaction of OsCUL3a with OsNPR1, reducing ubiquitin-mediated degradation of OsNPR1. Together, these findings demonstrated that OsNPR1 and SLR1 function as a co-transcriptional factor of IPA1. In addition, IPA1 stabilize OsNPR1 and SLR1. Finally, IPA1 integrate GA and SA signaling to enhance rice immunity.

Molecular basis of resistance to Fusarium head blight in wheat

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Fusarium head blight (FHB), primarily caused by Fusarium graminearum, is a devastating disease of wheat and other cereals that causes yield losses and contaminates grain with mycotoxins, particularly deoxynivalenol (DON), posing serious threats to food and feed safety. Genetically, wheat resistance to FHB can be broadly considered at three levels: resistance to initial infection, fungal spread, and mycotoxin accumulation. Here, we uncover the molecular basis underlying these three layers of resistance. For resistance to FHB initiation, we identify immune signaling pathways involving pattern recognition and wheat phytocytokines that strengthen early defense against fungal invasion. For resistance to FHB spread, our studies of the genetic locus Fhb1 reveal that HRC-mediated protein condensation regulates alternative splicing and immune responses, determining FHB resistance. Finally, we establish a ‘‘hijacking-counteracting’’ framework in plant-mycotoxin interactions. DON disrupts host central metabolism by hijacking multiple NAD^+-dependent enzymes, promoting FHB susceptibility. In contrast, plant inositol–PI(4,5)P2 metabolic flux promotes vacuolar sequestration of DON, counteracting DON hijacking and safeguarding central metabolism, thereby enhancing resistance to FHB. Together, these studies reveal distinct molecular principles governing FHB resistance, providing promising targets and strategies for breeding crops with enhanced disease resistance and grain safety.

Key words: Fusarium head blight; plant immunity; condensation; deoxynivalenol; disease resistance

From wild legumes to resistant soybean - discovering and engineering resistance to soybean rust

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Soybean rust, caused by Phakopsora pachyrhizi, threatens soybean production worldwide, affecting both commercial growers and smallholder farmers in Africa. Current control depends largely on costly fungicides with associated environmental impacts. Genetic resistance offers a more sustainable and accessible form of protection, but its deployment is constrained by limited resistance diversity and the pathogen's capacity to overcome individual genes. Cloning and characterising native soybean Rpp genes provides a foundation for understanding their function and deploying them in complementary combinations. Exploration of wild legume relatives broadens the available resistance gene pool beyond cultivated soybean, encompassing both NLR immune receptors and genes with novel modes of action. A resistance gene pair identified in wild Glycine confers strong protection when transferred into soybean, providing a foundation for investigating a new immune mechanism and engineering related non-functional genes in the crop. Together, these approaches connect gene discovery, mechanistic understanding, and engineering to develop durable soybean rust resistance for commercial and smallholder agriculture.

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Session VI

Big Data and Emerging Harmful Organisms

大数据与新发有害生物

A pathogen lncRNA secreted into rice sequesters a host miRNA for virulence

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Plants and animals respond to pathogens using pattern-recognition receptor and Nod-like receptor (NLR) proteins1. Pathogens commonly utilize protein effectors to suppress host immunity for successful infection2. However, the existence of non-protein effector classes remains comparatively understudied. Here, we report an RNA-RNA recognition mechanism governing pathogen-host interaction, mediated by a regulatory RNA-encoding DNA region that separately generates two complementary regulatory RNAs. Specifically, a long non-coding RNA (lncRNA) transcribed from this DNA region in the fungal pathogen Magnaporthe oryzae translocates into host rice cells and sequesters a complementary microRNA (miRNA), derived from a distinct host DNA region, thereby subverting host immunity. In turn, this rice derived miRNA promotes disease resistance by repressing expression of PKR1, a gene encoding a negative regulator of host immunity. Sequestration of the host miRNA by the fungal lncRNA releases PKR1 expression to facilitate fungal infection. We further discover that this regulatory RNA-encoding DNA sequence is likely widely present across diverse life species, mediating interactions between pathogens and their plant hosts. Collectively, our findings provide an approach for effective disease control using miRNAs derived from this important DNA region.
Keywords: Magnaporthe oryzae, lncRNA, miRNA, rice, host-pathogen interaction

Major Newly Emerging Disease of Maize and the Genetic Basis of Resistance

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Maize production is increasingly threatened by major and emerging diseases whose prevalence and geographic distribution are being reshaped by climate change, pathogen migration, and intensive cropping systems. In China, southern corn rust, stalk rots, maize white spot, and banded leaf and sheath blight (BLSB) have emerged as major or increasingly important disease threats, underscoring the urgent need to elucidate their genetic basis and develop durable resistance. To address these challenges, we have integrated large-scale disease phenotyping, genome-wide association studies (GWAS), population genomics, and functional genetics to dissect the genetic architecture of maize resistance to multiple pathogens. For Pythium stalk rot caused by Pythium aphanidermatum, GWAS identified ZmBGLU17, encoding a β-glucosidase that promotes lignin and DIMBOA accumulation and enhances resistance to both pathogens and insect pests, as well as ZmLecRK1, encoding a G-type lectin receptor kinase whose natural variation modulates its interaction with the co-receptor ZmBAK1 and thereby contributes to broad-spectrum disease resistance. For BLSB caused by Rhizoctonia solani, we identified ZmRRS1, encoding a malate dehydrogenase, as a major quantitative resistance gene modulating mitochondria-derived reactive oxygen species and disease resistance. In parallel, GWAS of 362 diverse maize inbred lines identified additional loci associated with resistance to Fusarium stalk rot, providing promising candidates for further functional characterization. Collectively, these studies illustrate how the integration of high-throughput phenotyping with large-scale genomic data can accelerate the discovery of resistance genes and favorable alleles, providing valuable genetic resources and molecular targets for breeding maize with durable resistance to major and emerging disease.

Understanding the molecular dialogue between rice and Magnaporthe oryzae for innovative and sustainable blast disease control

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Blast disease, caused by the fungus Pyricularia oryzae (syn. Magnaporthe oryzae), is one of the most devastating diseases of rice and serves as a model for understanding interactions between plants and their plant-fungal pathogens. Our group investigates the molecular dialogue between P. oryzae and rice, with the goal of translating fundamental insights into innovative and sustainable strategies for disease control. To this end, we study fungal effector proteins, their host targets, and the corresponding immune receptors in rice.

In this presentation, I will focus on our recent work on the MAX (Magnaporthe AVRs and ToxB-like) effectors, a large family of structurally conserved effectors in the rice blast fungus. I will describe our efforts to identify the rice proteins targeted by these effectors and discuss how this work can reveal new susceptibility genes. Finally, I will present how knowledge of effector targets has enabled us to engineer immune receptors with broadened and novel pathogen recognition specificities, providing new opportunities for durable resistance against rice blast disease.

Fusarium pathogenicity: from molecular mechanisms to genus-wide genomic prediction

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Fusarium species threaten crop production and global food safety through destructive diseases and mycotoxin contamination. Mitigating these threats requires both mechanistic understanding of major pathogens and systematic assessment of pathogenic potential in less-characterized species. This presentation integrates our research on the molecular basis of Fusarium head blight with a genus-wide framework for predictive pathogenomics.

Our studies in Fusarium graminearum have elucidated signaling and regulatory mechanisms governing host invasion and deoxynivalenol (DON) biosynthesis. Key findings include signaling pathways controlling infection structure formation, actomyosin-dependent assembly of toxin-producing organelles (toxisomes), and chromatin-mediated regulation of toxin biosynthetic genes. In particular, coordinated H3K36 and H3K27 methylation links chromatin regulation to virulence and secondary metabolism. These findings reveal molecular and cellular processes underlying disease development and toxin contamination, identifying potential targets for their control.

Expanding our focus from a well-characterized pathogen to pathogenic diversity across the genus, we established a comprehensive genomic atlas encompassing 323 species across all 23 Fusarium species complexes. By integrating comparative genomics, standardized phenotyping, and phylogenetically informed machine learning, we connected genome evolution with ecological versatility and plant pathogenicity. Our model inferred plant-pathogenic potential in 90.4% of the surveyed species, while a newly developed Pathogenicity Index classified a subset as high risk, highlighting priorities for further investigation and surveillance. Phylogenetic association mapping further uncovered a conserved virulence-associated gene repertoire. Large-scale functional testing of 201 candidate genes in F. graminearum demonstrated that 198 contribute to virulence, linking cross-species genomic associations to direct evidence of genetic contributions to infection.

Together, these studies connect molecular dissection of a major pathogen with genus-wide genomic prediction, providing a foundation for identifying underrecognized pathogenic potential and prioritizing surveillance of potential emerging fungal threats to crop health.

Keywords: Fusarium; virulence mechanisms; mycotoxin biosynthesis; genus-wide genomics; predictive pathogenomics; emerging plant pathogens

Big Data-Driven Genomic Surveillance and Integrated Management of Emerging Wheat Blast Fungus Magnaporthe oryzae Triticum

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Wheat blast, caused by the hemibiotrophic fungal pathogen Magnaporthe oryzae lineage Triticum (MoT), is a fearsome emerging disease posing a critical threat to global food security. Following its catastrophic 2016 transcontinental jump to Bangladesh, an international open-science framework leveraging genomic big data analytics was rapidly mobilized to pinpoint its South American origin and trace its evolutionary trajectory. To contain its geographical spread and bolster global phytosanitary surveillance, we developed and field-validated a highly sensitive, point-of-care diagnostic assay utilizing MoT-specific primers, enabling rapid detection in both agricultural and quarantine settings. Concurrently, a decade-long research initiative integrating artificial intelligence (AI), molecular breeding, and biocontrol has yielded multi-layered disease resistance frameworks. Utilizing AI-driven structural biology platforms, including AlphaFold, we elucidated the precise conformational dynamics of the newly cloned resistance gene Rmg8 and its interactions with pathogenic effectors, while simultaneously characterizing critical host susceptibility (S) genes. To engineer durable, broad-spectrum immunity, Rmg8 was strategically stacked into elite, high-yielding backgrounds, such as BARI Gom 33, which harbors the protective 2NS chromosomal translocation. To further diversify the resistance arsenal, we deployed targeted CRISPR-Cas genome editing of S-genes alongside advanced physical mutagenesis (incorporating Gamma rays, heavy ion beams, and X-rays) to disrupt MoT infection pathways without compromising agronomic traits. Complementing these host-centric genetic strategies, deep sequencing of the wheat seed microbiome uncovered potent endophytic biocontrol agents—specifically Bacillus subtilis and Bacillus velezensis. These microbial allies effectively suppress MoT proliferation through the secretion of antifungal lipopeptides and the priming of host-induced systemic resistance. Ultimately, the synergistic integration of big data diagnostics, AI-guided structural biology, precise genomic editing, and microbiome-assisted protection provides a scalable, holistic blueprint to not only mitigate the wheat blast crisis but proactively safeguard global cereal production against future fungal pandemics.
Keywords: Wheat Blast, Magnaporthe oryzae Triticum, AlphaFold, Rmg8, CRISPR-Cas, Biocontrol, Endophytes, Big Data Surveillance

Single cell sequencing unravels the infection strategy of rice sheath blight disease

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Rice sheath blight, caused by the necrotrophic fungus Rhizoctonia solani, is one of the devastating diseases on rice. In this study, we utilized the single nuclei sequencing technique to dissect the rice immune responses at cell levels during R. solani infection. We profiled stem and sheath cross-section cells of rice seedlings across six time points (0, 6, 12, 24, 48, and 72 h) after inoculation, in which we captured 320,980 high-quality nuclei that are clustered into 13 cell types. At early infection stage (6-12 h), transcriptional changes were largely confined to epidermal and mesophyll cells, dominated by genes encoding cuticle wax, cellulose biosynthesis and expansin (EXPA) proteins. Genetic analysis of EXPA4 supports a responsible role for susceptibility. Notably, at the middle infection stage (24 hpi), the most upregulated genes are the disease resistance genes, especially those of PRR and NLR, suggesting that the plants mounted immune response at this stage. However, at the late stage (48-72 h), PRR genes were induced at even a higher level. In addition, the genes involved in terpenoids and other secondary metabolite biosynthesis were markedly upregulated in parenchyma cells, which may explain the longitudinal infection along the stem for this pathogen. Transgenic plants expressing the candidate PRRs or NLRs increased disease resistance to sheath blight, indicating that they are functional in resisting against R. solani infection. By analyzing the gene expression profile of the fungi, we found that its transcripts were not globally amplified but selectively enriched in genes encoding cell wall degrading enzymes, oxalate acid metabolism-related enzymes and glyoxylate cycle proteins, indicating that the fungus primarily targets rice cell wall for degradation and inhibits rice immune response probably by deploying oxalate acid. In summary, R. solani manipulates the rice cell wall-related genes and suppresses plant immune gene expression at early infection stage, consequently leading to a successful invasion.

Pathogen Identification and Field Control of Maize White Spot Disease in China

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Maize white spot disease (MWS), first breaking out in Yunnan in 2020, has spread rapidly across 12 provinces in China by 2025, expanding from Southwest China to Northeast China's major maize production area, with its affected area surging from 23,600 ha in 2021 to 127,100 ha in 2022. To clarify its long-controversial causal agent, systematic phyllosphere microbiome analysis, multi-region sampling, morphological identification, phylogenetic assessment and pathogenicity validation confirmed Setophoma maydis as one of the primary pathogen, which was specifically and dominantly enriched in diseased leaves while nearly undetectable in healthy tissues. Field chemical efficacy trials demonstrated that strobilurin- and triazole-class fungicides provided the highest control performance. A developed two-spray control strategy, applied respectively at the 6–8 leaf stage and 2–3 weeks after tasseling, sustained over 60% efficacy 30 days post-treatment and achieved a 14.9% yield increase, which has been widely promoted in Yunnan Province to support maize production.

Whole genome identification and functional analyses of TNL-ID genes in soybean

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Soybean mosaic virus (SMV) causes a serious virus disease in soybean, and host genes conferring SMV resistance are mainly elusive. While most of the disease R proteins comprise the characteristic NLR (NBS-LRR) structure, more and more NLR-like proteins with atypical domains have been identified to play important roles in plant immune responses. Here we comprehensively screened the IDs present in soybean TNL-like genes and validated the immune activity of 27 Gm-TNL-ID genes against various pathogens, and identified several genes involved in calcium signaling, broad-spectrum resistance, and also growth-defense balance. We also characterize a soybean SMV resistance cluster (SRC) that comprises five resistance (R) genes. SRC7 contains a noncanonical BSP domain and gave full resistance to SMV. SRC7 possessed broad-spectrum antiviral activity toward several plant viruses including SMV, plum pox virus, potato virus Y, and tobacco mosaic virus. SRC7 expression was posttranscriptionally regulated by miR1510a and miR2109, and the SRC7-miR1510a/miR2109 regulatory network appeared to contribute to SMV–soybean interactions in both resistant and susceptible soybean cultivars. Two typical TNL genes, SRC3 and SRC13, antagonize SRC7 activity by protein heterodimerization and offset the SRC7-mediated immunity, contributing to maintaining the vegetative growth. The breeding value of these genes and loci were also verified by cultivar selection in field test. In summary, we report soybean R gene cluster and several functional R genes encoding atypical TIR-NBS-LRR (TNL) type R proteins, highlighting its importance in SMV–soybean interaction and plant immunity.

The best of two worlds to combat emerging pathogens: a novel mechanism of PAMP-induced ETI in Arabidopsis

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Among the two most effective plant defense pathways against biotrophs, Pattern-Triggered Immunity (PTI) initiated by recognition of conserved pathogen-associated molecular patterns (PAMPS) such as bacterial flagellin provides broad-spectrum resistance but is frequently suppressed by co-evolved pathogens. In contrast, nucleotide-binding leucine-rich repeat (NLR)-mediated Effector-Triggered Immunity (ETI) triggers robust and rapid defenses often associated with a hypersensitive response (HR). However, ETI is often not durable because of rapid evolution of effectors. Combining the broad efficacy of PTI with the speed of ETI may enable the engineering of effective resistance against broad classes of emerging pathogens. Serendipitously, we discovered an HR response induced by the bacterial flagellin-derived peptide flg22 in the accession RLD with mutations in the negative immune regulator SRFR1. To identify additional examples, we screened 822 Arabidopsis accessions for flg22-induced HR and found 28 accessions that exhibited this response, with Zdrl 1-23 showing the strongest HR. These accessions are not natural SRFR1 mutants and displayed HR only in photosynthetic tissue. Genetic mapping and preliminary CRISPR validation identified a coiled-coil (CC)-type NLR (CNL) we call FLG22-LINKED RESISTANCE1 (FLR1) required for this response. Interestingly, structural analysis of FLR1 across these accessions suggest a correlation between HR strength and the electrostatic potential in the concave side and the ascending loop of the leucine-rich repeat (LRR) domain. Accessions with strong HR possess a highly positively charged surface in this region, whereas nonresponsive accessions show a predominantly negatively charged surface. Thus, we hypothesize that FLR1 functions as a PTI- ETI connector by sensing a negatively charged signaling component linked to the FLS2 pathway and that the positively charged surface in the LRR domain serves as the interaction interface. We propose that it should be feasible to engineer signal connections between PAMP receptors and CNLs using the FLR1 interface to accelerate PTI against broad classes of emerging pathogens.

Tomato T2T super-pangenome empowers the gene discovery and molecular breeding for grey mold resistance

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Tomato, a globally vital vegetable crop, currently exhibits reduced genetic variation and stress tolerance. Wild relatives offer a rich but largely untapped genetic resource for breeding. Here, we construct a genus-wide super-pangenome spanning 16 tomato species by integrating 20 telomere-to-telomere (T2T) and 27 chromosome-scale genomes. Population analyses reveal extensive genetic diversity with restricted inter-clade gene flow. Pan-centromeric analysis uncovers dynamic evolution involving satellite turnover and repositioning. A comprehensive structural variant (SV) catalog identifies extensive rearrangements, especially in wild accessions, and pinpoints salinity-associated markers. SV-GWAS analysis reveals a leucine-rich repeat receptor gene SlGMAK confers gray mold resistance. This T2T super-pangenome will accelerate the exploitation of wild relatives to enhance stress resilience in modern tomato breeding.

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Session VII

New Technology for Plant Biotic Interaction

植物生物互作新技术

Remodelling autoactive NLRs for broad-spectrum immunity in plants

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Remodelling plant immune receptors has become a vital strategy for creating new disease resistance traits to combat the growing threat of plant pathogens to global food security and environmental sustainability1-17. However, current methods are constrained by the rapid evolution of plant pathogens and often lack broad-spectrum and durable protection. Here we report an innovative strategy to engineer broad-spectrum, durable and complete disease resistance in plants through expression of a chimeric protein containing a flexible polypeptide coupled with a single or dual conserved pathogen-originated protease cleavage site fused in frame to the N terminus of an autoactive nucleotide-binding and leucine-rich-repeat immune receptor (NLR) containing a coiled-coil or RESISTANCE TO POWDERY MILDEW 8-like coiled-coil domain. Following invasion, pathogen-originated specific proteases cleave the inactive chimeric protein to form free autoactive NLR, triggering broad-spectrum plant disease resistance. We demonstrate that a single engineered NLR can confer broad-spectrum and complete resistance against multiple potyviruses. Given that many pathogenic organisms across kingdoms encode proteases, this strategy has the potential to be exploited to control viruses, bacteria, oomycetes, fungi, nematodes and pests in plants.

Engineering synthetic Ca2+ input enables tunable multipathogen resistance in plants

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Huazhong Agricultural University

Genetic engineering is an important approach for achieving enhanced immunity in plants. While most immune-receptor engineering strategies focused on pathogen recognition, here we establish the engineering of synthetic Ca2+ signal inputs, downstream of diverse immune receptors as a complementary strategy for enhancing immunity. We uncovered the Ca2+ permeability of a previously designed transmembrane pore,
TMH4C4, and demonstrated that TMH4C4-mediated Ca2+ influx activates plant immune responses. We therefore named the repurposed scaffold DESIGNED PLANT DISEASE RESISTANCE1 (DPR1). In stable transgenic monocots and dicots, DPR1 confers multipathogen resistance. In rice, however, strong DPR1 activity enhanced resistance but reduced growth, which was challenging to optimize transcriptionally. AI-aided protein redesign generated RdDPR1 by occluding the original central axis while creating lateral pores, which reduced Ca2+ conductance while restoring agronomic performance and retaining multipathogen resistance. Together, these results establish the quantitative tuning of a convergent immune signal as a new layer in plant immune engineering.

Nanodomain-localized formin gates symbiotic microbial entry in legume and solanaceous plants

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Colonization of plant roots by symbionts requires substantial morphodynamic reorganization. Examples are actin-scaffolded micro-compartments called infection pockets formed during root nodule symbiosis (RNS) by legumes. Here, we demonstrate that the actin-binding formin SYFO2 is indispensable for rhizobial infection in Medicago truncatula, where it drives actin polymerization in phase-separated and symbiosis-specific nanodomains. Being evolutionarily conserved, SYFO2 also regulates symbiotically active arbuscules formed during mycorrhizal symbiosis (AMS) outside the nodulating clade, indicating that it was additionally recruited to promote rhizobial infections in legumes. Following our aim to enable nitrogen fixation in non-legumes, we additionally succeeded in activating endogenous SYFO2 by stably introducing the RNS master regulator NIN into the natural non-host tomato. This demonstrates the possibility of recruiting AM-related genes into an engineered nodulation-specific pathway.

Virus-induced Genome Editing in Hexaploid Wheat: Toward Crop Breeding Applications

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Virus-induced genome editing (VIGE) has been extensively studied for the transient delivery of editing reagents across different plant species, bypassing the need for transgenes and/or tissue culture. However, VIGE remains largely confined to the proof-of-concept stage in model plants and has yet to be widely applied in agricultural crop breeding. Inefficient delivery continues to be the central hurdle for VIGE application, especially in monocot crops. Plant negative-stranded RNA (NSR) viruses possess high genome stability and large cargo capacity, making them promising tools for transgene-free VIGE. Barley yellow striate mosaic virus (BYSMV) is an economically important member of the genus Cytorhabdovirus and can infect over 26 monocot staple crops through transmission by its specific insect vector, the small brown planthopper (SBPH, Laodelphax striatellus). Recently, we developed reverse genetics systems for BYSMV and established a BYSMV-mediated transient overexpression system in monocot plants. We further established a BYSMV-based virus-induced genome editing in tiller (BYSMV-ViGET) system for VIGE in hexaploid wheat, bypassing the need for transgenes and tissue culture. More intriguingly, this system enables the generation of homozygous mutants within one generation across multiple elite cultivars. Beyond wheat, BYSMV-ViGET is expected to be leveraged for genome editing in the other monocot host plant species. Collectively, our BYSMV-ViGET system provides a powerful platform for precision breeding of Poaceae crops.

Targeted protein-condensate degradation: A strategy toward protein editing in engineering disease-resilient plants?

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Gene expression can be modulated at the DNA, RNA, or protein level. Targeted protein degradation is a well-established strategy for directly reducing total protein abundance. Here, we explore whether targeted degradation of protein condensates could represent a step toward protein editing, by enabling selective removal of specific protein assemblies—such as distinct isoforms, post-translationally modified variants, or subcellularly localized populations—that are otherwise difficult to discriminate. Our investigation is motivated by a biological discovery in plants. During infection by Pseudomonas syringae, we found that HEM1 protein condensation at the endoplasmic reticulum (ER) shapes disease tolerance. These condensates modulate cellular homeostasis and immune-associated tissue damage. Genetic disruption of condensate formation enhances plant resilience, suggesting that selective manipulation of protein assemblies could improve plant health.

Based on this insight, we are developing targeted protein-condensate degradation (TCD) approaches to manipulate endogenous protein assemblies in plant cells. We design genetically encoded degraders that recruit the ubiquitin–proteasome system to selectively eliminate specific condensates. Compared with traditional genetic perturbations, this strategy enables programmable and dynamic control at the post-translational level. We propose that TCD, by virtue of its selectivity among distinct condensate states, may offer a route toward protein editing—a capacity to edit protein populations rather than merely reduce total protein levels. Whether this constitutes genuine protein editing remains an open question. Nevertheless, this work provides a framework for manipulating protein heterogeneity and establishes condensate degradation as a new strategy for restoring cellular homeostasis and improving crop performance, such as enhancing disease resilience in plants.

Precision genome editing and chromosome engineering in plants

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Genome editing has transformed the life sciences by making it possible to move beyond reading genetic information to rewriting genomes with precision and, ultimately, designing them de novo. Targeted deletions, replacements, and insertions enable researchers to modify gene function and generate desired phenotypes, opening new avenues for crop improvement. These capabilities offer a foundation for molecular design breeding, helping overcome the constraints of conventional approaches and accelerating the development of crops with desirable traits. In this study, we combined protein engineering with screening in plant cells to develop a suite of tools for precise nucleotide editing and multiplex genome editing. We further extended these capabilities to large DNA segments by establishing an efficient platform for precise, scarless chromosome engineering in plants. Together, these advances expand the scope of plant genome engineering from nucleotide changes to large chromosomal rearrangements and provide a strategy for generating complex structural variation. They broaden the toolkit for crop breeding and plant synthetic biology, supporting the development of novel traits and laying the groundwork for the design of plants with new combinations of biological functions.

Programmable Epigenome Editing and Gene Regulation in Plants

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Epigenetic modifications and transcriptional regulation are two fundamental mechanisms governing gene expression. In our previous work, we employed reverse genetic screening to identify a series of effector proteins capable of either repressing or activating plant gene expression. These proteins act through diverse mechanisms, including the regulation of DNA methylation, histone modifications, chromatin accessibility, and transcriptional elongation. More recently, we uncovered a molecular mechanism underlying the antagonism between H3K4me3 and DNA methylation in plants. Based on this finding, we combined distinct effectors to simultaneously remove H3K4me3 and introduce DNA methylation at target loci, substantially improving the efficiency of targeted DNA methylation. However, overexpression of regulatory effectors can cause varying degrees of off-target effects. To address this limitation, we developed an effector-free CRISPRi system that enables efficient and specific transcriptional repression in plants. We further established a bidirectional CRISPRai system that enables activation of one target gene while simultaneously repressing another. Together, these studies provide a versatile framework for programmable epigenome editing and gene regulation in plants, with potential applications in engineering complex agronomic traits.

Chemistry and AI-guided crop resistance design

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The extracellular space of plant tissues harbours hundreds of hydrolases that can potentially disrupt microbial colonization. To establish infection, successful pathogens may suppress these hydrolase activities to facilitate disease progression. Here, we investigate the dynamics of extracellular hydrolases in Nicotiana benthamiana upon infection with three Pseudomonas syringae strains: pathovar tomato DC3000 (ΔhopQ1), pv. syringae B728a, and pv. tabaci 6605. Through comprehensive proteomic analysis, we identified 357 apoplastic hydrolases from N. benthamiana and the activity of 23 serine hydrolases and 4 cysteine proteases was consistently suppressed across all three pathogens using activity-based protein profiling. To further investigate potential pathogen-mediated suppression, we are using AlphaFold-Multimer to predict the complex structures of these suppressed hydrolases in interaction with pathogen-secreted proteins. This study demonstrates two powerful approaches for uncovering novel components of extracellular immunity, shedding light on host-pathogen interactions at the apoplastic interface.

Large parallel deletions are associated with massive gene losses and virulence acquisition in the root-knot nematode Meloidogyne incognita

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The root-knot nematode Meloidogyne incognita is a major constraint to crop production worldwide. The resistance gene Mi-1.2 has been widely deployed in tomato varieties to control this parasite. However, virulent nematode genotypes, overcoming the Mi-1.2-mediated resistance, have increasingly emerged in fields. Understanding the genetic mechanisms underlying resistance circumvention is therefore essential towards sustainable crop protection. From two geographically distinct M. incognita avirulent isolates, previous experimental evolution assays have allowed producing two virulent M. incognita near-isogenic lines (NILs) that overcame Mi-1.2 resistance in tomato. Here, we investigated the transcriptomic and genomic changes in the two virulent M. incognita NILs, as compared to their avirulent progenitors controlled by the resistance. Our transcriptomic analyses revealed hundreds of strongly downregulated genes, in common, in both virulent NILs. Most of these downregulated genes were clustered in two main genome regions. Long-read whole-genome sequencing of the four NILs revealed parallel structural variations in both virulent NILs. This included two large genomic deletions spanning ca. 1.3 Mb and 0.6 Mb. These two big deletions encompass clusters of downregulated genes, suggesting they have in fact been lost in both virulent NILs. Short-read genome sequencing and PCR validation confirmed the two parallel deletions in both virulent NILs. The largest deletion includes predicted effector and orphan Meloidogyne-specific genes. The parallel large-scale structural variations identified here are likely to underlie the rapid adaptation of M. incognita to Mi-1.2-mediated resistance. These findings highlight genome structural plasticity as one of the potential key drivers of virulence acquisition in these asexually reproducing parasites.

ICBPI 2026 · OCTOBER 18–22, 2026

Blitz Talk

闪电报告

Blitz Talk 1: Plant-bacteria interactions 植物-细菌互作

A bacterial pathogen deploys peptide and effectors to cooperatively disarm exopolysaccharide-triggered plant immunity

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Plants perceive conserved microbial patterns via transmembrane pattern recognition receptors (PRRs) to initiate immunity. The receptor kinase SlLYK4 recognizes exopolysaccharides (EPS) from the bacterial wilt pathogen Ralstonia solanacearum and confers host resistance. However, how R. solanacearum actively suppresses EPS-triggered immunity to facilitate infection remains elusive. Here, we report the discovery of Rsp3, a secreted peptide from R. solanacearum that functions as a ligand of SlLYK4 to suppress EPS-triggered immunity. Rsp3 directly binds the receptor ectodomain and impedes EPS recognition. Strikingly, Rsp3 also structurally mimics the extracellular LysM domain of SlLYK4 to directly sequester EPS, thereby preventing immune activation. We further demonstrate that two type III effectors, RipAE and RipD, target the intracellular domain of SlLYK4 to disrupt SlLYK4–SlLYK13 receptor complex formation, antagonizing EPS-triggered immune signaling. Simultaneous deletion of Rsp3, RipAE, and RipD markedly attenuates R. solanacearum virulence. Collectively, our findings uncover a co-evolutionarily refined virulence strategy, wherein a bacterium uses a secreted peptide and intracellular effectors to dismantle EPS-triggered immunity at both receptor ectodomain and kinase domain, illustrating how pathogens evolve counter-defense mechanisms against ancient host immune recognition systems.

Nuclear localization controls Xa1–mediated immunity in rice

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Transcription activator-like effectors (TALEs) are key virulence factors of Xanthomonas oryzae, causing bacterial blight and leaf streak in rice and leading to severe yield losses. The zinc-finger BED domain-containing coiled-coil nucleotide-binding leucine-rich repeat (CNL) receptor Xa1 confers resistance by recognizing multiple TALEs. In addition to canonical TALEs, Xanthomonas strains often encode truncated TALEs, termed interfering TALEs (iTALEs), characterized by the absence of the C-terminal activation domain and the presence of N-terminal internal deletions. These iTALEs suppress resistance mediated by Xa1 and its allelic variants Xo1, Xa2, Xa14, Xa31, and Xa45, thereby enhancing pathogen virulence. Despite the significant agricultural value of Xa1, the molecular mechanism by which TALEs activate Xa1-mediated resistance remains poorly understood. Approximately 40% of monocot BED-NLR proteins contain predicted nuclear localization signals (NLS) motifs proximal to the BED domain, a feature conserved in wheat and barley resistance proteins. Although NLSs mediate nuclear localization in some BED-NLRs, direct evidence that nuclear localization is required for BED-NLR immune function remains lacking. Here we demonstrate that nuclear localization of Xa1 is essential for its immune function. Prime-edited rice lines carrying mutations in NLS displayed compromised resistance. Mutating the NLS motifs in either Xa1 or TALEs did not impair their direct physical interaction, suggesting that effector recognition occurs independently of nuclear import. Together, these results confirm that nuclear localization is a precondition for Xa1-mediated immunity and highlight the power of prime editing for mechanistic dissection of plant NLR immune receptors.

Key words: 8th ICBPI; Biotic Plant Interaction; Bacterial blight; Xa1; Nuclear localization signals; TALE

A transcription factor OsWRKY77 orchestrates phytohormone crosstalk to balance growth and defense

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Grain yield and disease resistance, two major agronomic traits, represent a classic antagonistic trade-off in plants, known as the "growth–defense trade-off." Elucidating the regulatory mechanisms that couple immunity to yield-related traits and developing strategies to overcome it remain a central challenge in plant biology. In this study, we identify OsWRKY77 as a pivotal regulatory node that couples immune activation to growth and grain-weight control, thereby shaping the growth–defense trade-off in rice. OsMKK10-2–OsMKP3–OsWRKY77 cascade promotes defense activation while imposing a cost on grain weight. Distinct phosphosites on OsWRKY77 exert opposing effects on DNA-binding activity and its defense- and growth-related transcriptional outputs in planta, providing an endogenous mechanism that prevents excessive defense activation. Additionally, the endosperm-enriched transcription factor OsNF-YB1 enhances OsWRKY77 DNA binding and cooperates with it to directly regulates genes involved in auxin, jasmonate, and salicylic acid signaling, thereby coordinating growth–defense homeostasis during seed development. Leveraging this regulatory insight, we engineered rice lines in two genetic backgrounds with simultaneous knockout of salicylic acid hydroxylase and OsWRKY77, to achieve concurrent gains in blast resistance and yield-related traits, offering a molecular framework to rationally uncouple the growth–defense trade-off.

Key words: 8th ICBPI; Biotic Plant Interaction; Growth–defense Trade-off; Phosphorylation; Phytohormone Homeostasis

Integrative Genomic and Transcriptomic Analyses Identify a Chromosome 4 Locus Associated with Alternaria Brown Spot Resistance in Citrus

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Alternaria brown spot (ABS), caused by the tangerine pathotype of Alternaria alternata, is an economically important fungal disease of susceptible citrus cultivars. The pathogen produces the host-selective Alternaria citri toxin (ACT), a major virulence determinant that promotes host cell death; however, the genetic and molecular basis of citrus resistance to ABS remains poorly understood. Here, we integrated comparative transcriptomics and genome-wide association analysis to identify resistance-associated loci and candidate genes in citrus.

RNA sequencing was performed using leaves of ABS-resistant Clementine and susceptible Dancy mandarin inoculated with the wild-type A. alternata strain Z7, an ACT-deficient ΔΔACTT6 mutant, or mock treatment at 24 and 48 h post inoculation. Resistant and susceptible citrus exhibited distinct transcriptional responses to fungal infection. Genes associated with jasmonic acid metabolism were significantly enriched in Clementine, whereas susceptible Dancy showed extensive transcriptional reprogramming and enrichment of macromolecule degradation-related processes during late infection by the toxin-producing strain.

To dissect the genetic basis of ABS resistance, a genome-wide association study was conducted using 138 Citrus and related accessions. Among 11,246,006 high-quality SNPs, 808 SNPs were significantly associated with ABS resistance, defining 330 candidate genes. A major association region on chromosome 4 contained multiple defense-related genes, including a cluster of pectin methylesterase (PME)/pectin methylesterase inhibitor (PMEI) genes, a pathogenesis-related protein 2 (PR-2), and genes associated with jasmonate signaling and transcriptional regulation. Notably, a PMEI gene remained relatively stable in resistant Clementine but was strongly suppressed in susceptible citrus following infection, whereas a WRKY transcription factor gene showed contrasting expression patterns between resistant and susceptible genotypes.

Together, these results reveal a chromosome 4 resistance-associated region integrating cell-wall regulation and defense signaling and provide candidate genes for functional dissection of citrus resistance to ABS.

Key words: Alternaria brown spot; Citrus; Genome-wide association study; Transcriptome; Disease resistance

Phycosphere Microbiome–Genotype Interactions Amplify Seaweed Phenotypic Genetic Effects

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jimei university

Understanding the formation of complex traits through host–microbe interactions is central to comprehending adaptive evolution. However, the ways in which host genetic variation regulates the phycosphere microbiome of macroalgae, species of significant evolutionary importance and economic value, influences algal phenotypes remain poorly characterized. In this study, we used the representative intertidal macroalga Pyropia haitanensis as the experimental system to investigate the relationships among host genetic background, the phycosphere microbiome, and phenotypic differentiation. The results showed that host genetic background significantly shaped the composition of the phycosphere microbiome, and 12 ASVs significantly associated with host SNP loci were identified. Among these, ASV35 (Sphingomonas) and ASV148 (Flavobacteriaceae) showed opposing associations with thallus growth, and their microbiome–host genome-wide association signals colocalized with a thallus thickness-associated interval at 4.70–5.10 Mb on Chr4. Within this interval, the GDP-D-mannose 3’,5’-epimerase gene (PhGME) and the inositol 2-dehydrogenase gene (PhmIDH) formed a stable linked haplotype module. Different haplotypes of both genes were simultaneously associated with thallus thickness and the abundances of ASV35 and ASV148. Further analysis revealed that Sphingomonas PY271 promoted thallus thinning, increased length, and enhanced biomass growth, whereas Flavobacteriaceae PY291 promoted thallus thickening and suppressed length and biomass growth. The magnitude of the effects of both phycosphere bacteria strains depended on the host genetic background. Moreover, the introduced bacterial strains did not cause substantial restructuring of the overall phycosphere microbiome, but instead primarily reinforced the host’s pre-existing metabolic and phenotypic regulatory programs by inducing host genetic background-dependent transcriptional responses. Collectively, this study proposes an interaction model in which the phycosphere microbiome amplifies host phenotypic genetic effects, providing a new theoretical framework for understanding macroalgae–microbe interactions and the mechanisms underlying complex trait formation in marine environments.

Investigating the Role and Underlying Mechanism of Transcription Activator-Like Effectors in Rice Bacterial Blight Resistance Mediated by the XA14 Gene

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Xanthomonas species colonize many host plants and cause huge losses worldwide. Transcription activator-like effectors (TALEs) are secreted by Xanthomonas and translocated into host cells to manipu-late the expression of target genes, especially by Xanthomonas oryzae pv. oryzae and Xanthomonas oryzae pv. oryzicola, which cause bacterial blight and bacterial leaf streak, respectively, in rice.In this study, we elucidate how transcription activator-like effectors (TALEs) secreted by Xanthomonas oryzae pv. oryzae (Xoo) are recognized by the NLS protein encoded by the rice disease resistance gene Xa14, thereby triggering disease resistance. Concurrently, we demonstrate that Xoo strains harboring mutated TALEs, designated as iTALEs (interfering TALEs), can disrupt this recognition and render the plants susceptible. Furthermore, we have discovered that two distinct iTALE variants, which have evolved and are classified as Type-A and Type-B iTALEs, exhibit differential suppressive capacities. Notably, the Type-B iTALE lacks the ability to suppress XA14-mediated immunity. Deciphering the molecular interactions between TALEs, iTALEs, and the XA14 protein will provide pivotal insights for advancing crop improvement and developing novel strategies for immune prophylaxis.

plasmodesmata-mediated resistance to F. graminearum in wheat

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Pathogen invasion of plant cells is a critical step in establishing successful infection. Many biotrophic and hemibiotrophic pathogens deliver secreted effector proteins into host cells to manipulate cellular processes and promote disease. Previous work from the C. Faulkner laboratory showed that PAMP-triggered plasmodesmata (PD) closure, an important component of plant immunity, is suppressed during infection by certain virulent pathogens. This observation suggests that pathogens may actively counteract immune-induced PD closure to maintain intercellular connectivity and facilitate colonization. However, the molecular mechanisms underlying pathogen-mediated regulation of PD remain largely unknown.Here, we identified Fg36, an effector protein from Fusarium graminearum that localizes to plasmodesmata in wheat cells. Fg36 interacts with Ta-PD1, a wheat PD-localized protein, suggesting that Fg36 may target host PD machinery to modulate plasmodesmal function during infection. Deletion of Fg36 significantly reduced fungal virulence, demonstrating that Fg36 contributes to the successful infection of wheat by F. graminearum.Together, our findings reveal a previously uncharacterized effector-mediated strategy by which F. graminearum targets plasmodesmata during host colonization. This work provides new insights into the molecular mechanisms through which fungal pathogens manipulate intercellular connectivity to overcome host immune barriers and highlights plasmodesmata as an important interface in plant–pathogen interactions.

Disease Tolerance: An Underexplored Dimension of Plant Immunity

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Plant immunity has traditionally been studied through the framework of disease resistance, with major efforts devoted to understanding how plants restrict pathogen growth. However, disease severity is determined not only by pathogen proliferation but also by the extent of host damage during infection. Disease tolerance, defined as the ability of a host to maintain health despite pathogen challenge, remains poorly understood at the molecular level in plants. Using the Arabidopsis–Pseudomonas syringae pathosystem, we found that pathogen-induced protein condensation-associated damage is a key node for disease tolerance. Alleviating this damage substantially reduced disease symptoms without significantly affecting pathogen proliferation, highlighting tolerance as an important and potentially targetable component of plant immunity. Building on this, my research will focus on rice disease tolerance, aiming to identify disease-tolerant rice resources, uncover the underlying molecular mechanisms, and explore how tolerance can be integrated with resistance to achieve durable disease management with minimal yield loss.
Key words: Disease tolerance; Disease resistance; Plant immunity; Rice; Plant health

Characterizing the interactions between the fire blight pathogen Erwinia amylovora with a disease resistant apple cultivar

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Fire blight, caused by the bacterial pathogenErwinia amylovora, is a devastating disease of rosaceous crops, including apple and pear. The identification and deployment of genetic resistance offer a sustainable, long-term strategy for managing fire blight. To date, resistance has primarily been identified in crabapple (Malusspp.) germplasm, where it provides only partial protection against certainE. amylovoralineages and can be overcome by mutations in the corresponding bacterial effector proteins. Here, we report the first fire blight resistance identified in cultivated apple (Malus domestica), represented by the accession N12. Upon pathogen challenge, N12 exhibits a hypersensitive response accompanied by flower abscission, indicative of effector-triggered immunity. Deletion of several Type III effector genes partially restored pathogen virulence, suggesting that these effectors contribute to activation of the resistance response. Using a yeast two-hybrid screen combined with bioinformatic analyses, we identified several candidate host resistance-associated proteins that interact with these effectors. These findings provide promising targets for future functional validation and offer valuable resources for breeding durable fire blight resistance in apple.

A witches' broom phytoplasma effector induces stunting by stabilizing a bHLH transcription factor in Ziziphus jujuba plants

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Jujube witches' broom (JWB) is a bacterial disease associated with Candidatus phytoplasma ziziphi. JWB poses significant challenges to the jujube industry, manifesting in flower alterations (virescence, phyllody), witches' broom, and stunting, ultimately leading to substantial losses in yield and fruit quality. Bacterial pathogens rely on effector proteins to induce diseases. Genome analysis of the JWB phytoplasma has revealed 72 genes encoding secreted effector proteins called JWB phytoplasma proteins (SJPs); however, the molecular mechanisms by which pathogen-derived effectors suppress plant defences are barely known. In this study, we characterized the effector protein SJP39 from JWB phytoplasma and explored its function in host manipulation. Biochemical assays verified that SJP39 directly interacts with the plant transcription factor bHLH87 in the nucleus. SJP39 stabilizes bHLH87 homologs in both Arabidopsis thaliana and jujube, which ultimately induces plant growth defects. Transcriptome data revealed that SJP39 interferes with the gibberellin (GA) signaling pathway in jujube. Further functional validation confirmed that ZjbHLH87 acts as a negative regulator of GA signaling, and SJP39 strengthens this inhibitory effect. Collectively, this study unravels the molecular pathogenesis of JWB disease and identifies SJP39 as an important virulence effector mediating growth abnormalities upon phytoplasma infection. These results provide valuable clues for developing control strategies against JWB and other phytoplasma-associated diseases.

Key words: bHLH transcription factor; effector; jujube witches' broom disease; phytoplasma; plant-pathogen interaction

TALE tails escape recognition by OsSWEET11a for bacterial blight in rice

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Xanthomonas oryzae pv. oryzae (Xoo), the causal agent of bacterial blight (BB) in rice, deploys diverse transcription activator-like effectors (TALEs) that bind specific effector-binding elements (EBEs) in the promoters of host susceptibility (S) or resistance (R) genes. Effector binding occurs through repeat-variable di-residues (RVDs), thereby activating effector-triggered susceptibility or immunity. Previous studies demonstrated that disrupting the TALE-targeted EBEs of SWEET genes conferred broad-spectrum resistance to BB in rice. However, Xoo YN04-5 overcame resistance in simultaneously edited EBEs of OsSWEET11a, OsSWEET13, and OsSWEET14. In the current study, we show that this breakdown is caused by a PthXo1-like TALE, Tal7c, which functions as a major virulence factor by specifically activating OsSWEET11a in rice. Notably, Tal7c carries two additional RVDs upstream of a 24-RVD array which is nearly identical to that of PthXo1, differing by a single substitution at 6th RVD. Using a combination of β-glucuronidase (GUS) and executor Xa10 reporters, together with nucleotide (nt) deletion, insertion and substitution analyses in the Tal7c-bound EBE of OsSWEET11a and RVD deletion analysis of Tal7c, we found that deletion or variations in the 3′-EBE and the C-terminal RVDs do not significantly impair transcriptional activation. Microscale thermophoresis assays confirmed that Tal7c retained higher binding affinity than PthXo1 under specific 3′-EBE deletions, and the key recognition region resides in the first 15 nt of the 5′-EBE. Consequently, deletions downstream of this critical region in rice did not attenuate BB development, revealing a wobble tolerance in the 3′ tail of PthXo1-like TALEs. Genome analysis of Xoo populations revealed the presence of five PthXo1-like variants. Screening of 3K rice genomes uncovered five natural allelic variants in the OsSWEET11a EBE. Among these, three xa13 alleles conferred resistance to at least one of the two TALEs, PthXo1 and Tal7c. Collectively, these findings uncovered a novel arms-race mechanism in the rice-Xoo pathosystem, where TALE-EBE interactions exhibited flexibility beyond the canonical TALE code, particularly through tail wobble. This flexibility highlights the importance of accounting for TALE tail variability when engineering EBE edits in S genes to ensure durable BB resistance.
Key words: Rice; Bacterial blight; Transcription activator-like effector; OsSWEET11a; TALE binding polarity

Latest grapevine resistance mechanism against downy mildew

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Grapevine downy mildew (GDM) is the most damaging disease in the global grape industry and a model plant pathosystem in the history of plant protection research, one that led to the invention of the first modern pesticideBordeaux Mixtureand the establishment of the global plant quarantine system. By multi-omics, genetic and cell biology approaches, my group has significantly advanced understanding of GDM resistance mechanism. At the cellular level, our studies have revealed multi-layered stomatal immunity in resistant grapevines overlooked so far. At the molecular level, we constructed the first Visitsuper pangenome, and identified so-far the largest plant NLR repertoire (over 100,000 grapevine NLR genes) and more than 1,800 eQTLs associated with GDM resistance from 113 grapevine accessions. Regarding environmental cues, we conducted pan-metagenomic and pan-metatranscriptomic analyses of the phyllosphere microbiome from 107 accessions andidentified bacterial strains enhancing GDM resistance. In the end, I willbriefly discuss novel strategies for managing GDM based on the mechanistic insights from these studies, which may inspire advancements in plant protection more broadly.

Blitz Talk 2: Plant-fungus/oomycete interactions 植物-真菌/卵菌互作

A subset of HMAs is cyclic nucleotide synthase required for CNL-mediated immunity in rice

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Zhejiang University (浙江大学) / CAS Center for Excellence in Molecular Plant Sciences (中国科学院分子植物科学卓越创新中心)

Plants usually deploy nucleotide-binding leucine-rich repeat (NLR) receptors to detect pathogen effectors and activate immunity. However, the roles of non-NLR effector targets in NLR-mediated immunity remain poorly understood. Here, we identify a subset of rice heavy metal-associated (HMA) proteins that are targeted by the M. oryzae MAX effector AvrPigm and required for broad-spectrum blast resistance mediated by the CNL PigmR. AvrPigm interacts with functionally redundant HMAs and promotes their translocation into the cytoplasm from plasma membrane (PM). Cryo-EM structure reveals a filament-like oligomer of HPP04HMA containing a single-strand nucleic-acid, which displays non-canonical cyclic nucleotide synthase activity to generate 2,3-cNMP from ssRNA. Genetic evidence functionally associates cytoplasmic HMA-dependent 2,3-cNMP accumulation with PigmR-mediated immunity. The isolated LRR domain of PigmR binds 2,3-cNMP through residues important for PigmR function. Together, our study supports a previously unrecognized HMAs-cNMP-CNL signaling module and reveals an unexpected role for HMAs in NLR-mediated immunity.

Thiamine cross-feeding drives microbial coexistence in the leaf microbiome

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Microbiome homeostasis is crucial for host health and ecosystem function, yet the molecular and ecological mechanisms underlying community assembly and stability remain elusive. In this project, we uncover a conserved yeast-oomycete metabolic mutualism that promotes their coexistence in the leaf microbiome. Using a continental-scale microbiome survey, we identified a mutualistic interaction between two eukaryotic hub microbes: the yeast Dioszegia hungarica and the obligate oomycete Albugo laibachii. We show that Dioszegia facilitates Albugo colonization by supplying thiamine via a dedicated membrane permease, alleviating Albugo's auxotrophy. Genomic and transcriptomic analyses reveal that natural selection has acted on thiamine production in Dioszegia, shaping this mutualistic interaction. In planta assays further demonstrate that cross-feeding enhances Albugo colonization and promotes Dioszegia persistence. Our study illustrates how the evolution of nutrient cross-feeding mediates microbial coexistence and microbiome stability. Targeting microbial nutrient flows offers new strategies for engineering microbiomes and enhancing plant resilience in natural and agricultural systems.

Rhizoctonia Solani Effector Activating SWEET Sugar Transport Proteins to Compete for Sugar Nutrition with Host

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Rhizoctonia solani, the causative agent of the sheath blight disease (ShB), invades rice to obtain nutrients, especially sugars; however, the molecular mechanism via which R. solani hijacks sugars from rice remains unclear. The present study found that the availability of sugars greatly influenced R. solani growth. The study also found that R. solani activated the Sugar will be eventually exported transporter (SWEET) genes, especially SWEET2a. Yeast one-hybrid assay using SWEET2a promoter and the 200 putative secretory proteins from R. solani identified an effector Activator of SWEET2a (AOS2) directly activates SWEET2a. Further analysis showed that AOS2 is localized at the nucleus of tobacco cells and has secretion ability in yeast. AOS2 suppression via spray-induced gene silencing (SIGS) inhibited R. solani-mediated SWEET2a induction and reduced ShB disease severity, suggesting that AOS2 may activate SWEET2a to promote infection. Subsequent analysis based on yeast two-hybrid and bimolecular fluorescence complementation (BiFC) assays indicated that AOS2 interacts with rice WRKY53 and grassy tiller 1 (GT1) to form the AOS2-WRKY53-GT1 transcriptional complex; this complex activated the hexose transporters, SWEET2a and SWEET3a. The comparison of soluble sugar content showed that the gt1, wrky53, sweet2a, and sweet3a mutant strains accumulated less soluble sugar than the wild-type control, while the GT1 OX and WRKY53 OX plants accumulated more soluble sugar than the wild-type control after inoculation. Genetic analysis confirmed that GT1, WRKY53, SWEET2a, and SWEET3a negatively regulate rice resistance to ShB. These data collectively suggest that AOS2 secreted by R. solani interacts with rice WRKY53 and GT1 to form a transcriptional complex that activates SWEETs to efflux sugars to apoplast; This study is beneficial for understanding the process of R. solani invasion and causing host disease, explore the targets of green and efficient plant immunity inducers, and provide theoretical support for achieving sustainable and environment-friendly concepts of green disease prevention.

Keywords: Rhizoctonia solani； Secretory proteins； Transcriptional complex； SWEET；Sugar

Figure1. If the host successfully recognizes the effector protein AOS2 secreted by R. solani, GT1-WRKY53-meidated SWEET2a and SWEET3a induction rises significantly. More hexose flows to the pathogen, increasing the virulence.

Phosphorylation of the RNA-Binding Protein MHZ9 Orchestrates Immune Responses in Rice

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Rice employs sophisticated and multilayered regulatory networks to precisely modulate innate immune responses against diverse pathogens. Although post-transcriptional control has emerged as a vital layer of immune regulation, the mechanisms by which RNA-binding proteins (RBPs) govern rice immune responses remain largely unclear. Here, we report that the GYF-domain RBP MHZ9 acts as a negative regulator of rice disease resistance. MHZ9 directly binds to the mRNAs of the novel nucleotide-binding leucine-rich repeat receptor (NLR) genes and represses their translation, thus preventing excessive NLR accumulation. OsMAPK3/6, the core kinases in PTI signaling, physically interact with MHZ9 and specifically phosphorylate it at Ser50. This modification impairs the RNA-binding and translational repression activities of MHZ9, thus relieving inhibition of NLR translation upon pathogen infection. Conservation of the Ser50 residue across plant species but not in animals suggests that its phosphorylation is critical for plant immunity and likely plant-specific. Genetic analyses demonstrate that RP1 and RP5 are required for the enhanced blast resistance in mhz9, and Ser50 phosphorylation of MHZ9 serves as a critical switch to inactivate MHZ9 and activate immune responses. Our findings uncover a novel regulatory module where OsMAPK3-mediated MHZ9 Ser50 phosphorylation couples PTI signaling to post-transcriptional control of NLRs, shedding light on the molecular basis of immunity-growth balance in rice.

Key-words: GYF domain, RNA-binding protein, NLR, translation, phosphorylation, rice immunity

Structure-based clustering of Phytophthora RxLR effectors identifies a conserved alternating WY architecture shared by multiple Avr effectors

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Phytophthora species are among the most destructive plant pathogens. RxLR effectors constitute the largest effector family in Phytophthora and play central roles in pathogen virulence. (L)WY folds are prevalent structural units within RxLR effectors and contribute to both virulence and avirulence activities. However, the overall distribution of these structural units and the potential existence of additional structural folds within the RxLR effector repertoire remain unclear. We performed large-scale structural prediction of 3,961 RxLR effectors from 11 Phytophthora species and 4 related oomycetes. Structure-based clustering revealed that WY and LWY folds constitute the predominant structural folds within RxLR-WY repertoire. Moreover, we identified alternating WY (AWY) architectures, composed of two distinct WY units arranged in an alternating pattern. AWY-containing RxLR effectors were detected across nearly all major Phytophthora clades, while the arrangements of the two AWY units exhibited substantial diversity. Several P. infestans Avr effectors, including Avr1, Avr8 and Avramr3, exhibited the AWY architecture, and co-expression of multiple AWY-containing proteins with the Solanum NLRs R1 and R8 triggered cell death in Nicotiana benthamiana. Together, our study provides a comprehensive view of RxLR effector architectures and identifies a conserved AWY architecture, providing insights into the evolutionary diversification Phytophthora effectors.

Keyword: Phytophthora, RxLR effector, protein structure, (L)WY fold, Avirulence effector

Photocatalytic Carbon Dots Targeting Fungal Membranes for Sustainable Crop Protection

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Fungicides have played a key role in boosting food production to feed a growing global population, but their overuse has led to severe environmental pollution, harm to non-target species, and the rise of resistant pathogens. To tackle these issues, sustainable alternatives are urgently needed. Here, we used a nanotechnology to synthesize plant-derived amphiphilic carbon dots (CDs) that target the biomembrane system of Fusarium graminearum, one of top 10 fungal plant pathogen threatening global food security, generating reactive oxygen species (ROS) in situ to inactivate fungal spores and reduce pathogenicity in vitro and vivo under natural light illumination conditions. The CDs rapidly adhere to and enter spores via endocytosis, localizing to membranous organelles and causing oxidative damage that disrupts cellular homeostasis. Three years of field trial confirmed their efficacy in controlling the pathogen without adverse effects on wheat growth or the environment, while also demonstrating broad-spectrum antimicrobial activity against various pathogens. Overall, this research introduces a groundbreaking eco-friendly antifungal strategy, offering a sustainable solution to global challenges in food security and ecosystem health.

Key words: Carbon Dots; Fungicides; Fusarium head blight; endocytosis; Sustainable crop production

Effects of the mycovirus on the efficacy of Beauveria bassiana against the whitefly

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Beauveria bassiana is a cosmopolitan entomopathogenic fungus widely used in agricultural pest control, and it can also function as a plant endophyte with important roles in growth promotion and antagonism against plant pathogens. Mycoviruses are common in both phytopathogenic fungi and beneficial biocontrol fungi. BbCV-2 is a double-stranded RNA mycovirus identified in B. bassiana. In this study, we investigated the effects of BbCV-2 on the virulence of B. bassiana against the whitefly directly and indirectly through plant. Our results showed that BbCV-2 significantly enhanced the virulence of B. bassiana against both adult and nymphal stages of whitefly. The presence of BbCV-2 enabled B. bassiana to colonize tomato plants more rapidly and to persist longer following foliar spray inoculation. Endophytic colonization of tomato by BbCV-2-infected B. bassiana led to a marked increase in whitefly mortality and a significant reduction in oviposition. This study supports the broader application of B. bassiana in crop protection and provides a foundation for elucidating the mechanisms by which mycoviruses modulate the activity of entomopathogenic fungi.

Keywords: Beauveria bassiana; mycovirus; tobacco whitefly; plant glandular trichomes

The fungus strategically deploys host protease-activated effectors to target fatty acids and subvert jasmonate immunity

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SCP (sperm-coating protein) domain proteins are evolutionarily conserved lipid-binding proteins. To date, little is known about how their lipid-binding function participates in pathogen-host interactions. Here, we demonstrated that the Magnaporthe oryzae SCP effector MoScp1 employs rice cysteine protease OsCatB3, which cleaves its conserved CNY motif and removes the C-terminal peptide (CAPE), thereby activating its lipid-binding capacity. The processed MoScp1 enters rice chloroplasts, where it suppresses the desaturation of palmitic acid (C16:0) and stearic acid (C18:0), thereby subverting linolenic acid (C18:3) production and dampening JA-mediated immunity. Further mutational analysis identified P207, F212, Q161, and T226 from MoScp1 as essential for fatty acid binding and highly conserved in phytopathogens but not in plant homologs. In addition, genetic analysis revealed that Oscatb3-KO lines confer resistance to Guy11 and the non-cleavable mutant MoScp13A but remain susceptible to MoScp1DCAPE. Conversely, host mutations in fatty acid synthase (OsFAD7) and JA signaling component OsMYC2 rescue the virulence defects of DMoscp1 and MoScp13A mutants, confirming that MoScp1 promotes infection by targeting fatty acid metabolism and JA signaling. Finally, a small molecule inhibitor was selected to restore rice fatty acid metabolism and JA biosynthesis by disrupting the fatty acid-binding capacity of MoScp1DCAPE, conferring effective resistance against rice blast. Taken together, our findings reveal a novel strategy wherein a fungal effector co-opts the host protease system to manipulate host lipid metabolism and suppress JA-related immunity, providing a concept for targeting pathogen-effector interfaces to prevent and manage crop diseases.

Keywords: Magnaporthe oryzae, SCP domain protein, cysteine protease, lipid-binding, JA-related immunity.

Genome reshaping by Helitron transposons in Fusarium oxysporum TR4 affects secondary metabolism but not virulence

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Transposable elements (TEs) are significant drivers of genome evolution, influencing the genome dynamics of clonal fungal pathogens such as those in the Fusarium oxysporum species complex (FOSC) that cause Fusarium wilt in over 100 plant hosts. Among these, Tropical Race 4 (TR4), a clonal lineage within the FOSC, poses a severe threat to global banana production. However, the contribution of TEs to genome variation and functional traits in TR4 remains poorly understood. Here, we investigated Helitron-associated structural variations in a TR4 strain from Mozambique (M1). This revealed two large deletions in core chromosomes associated with an active FoHeli1 Helitron transposon. One of these (464 kb) disrupted 151 genes, including the entire fusaric acid (FA) biosynthetic gene cluster, consequently abolishing FA production, altering secondary metabolite profiles, and increasing sensitivity to exogenous FA. Despite these metabolic changes, infection assays using wild-type, mutant, knock-out, and complemented strains demonstrated that FA production is dispensable for TR4 virulence in banana. Our study highlights the role of FoHeli1 in modulating the genetic and metabolic landscape of TR4, underscoring the broader impact of TEs on fungal genome evolution and functional diversification, especially in clonal lineages.
Key words: Fusarium; Tropical Race 4; Transposable elements; Fusaric acid; Helitron

The Valsa mali Effector VmSR1 Promotes the Degradation of Tudor-SN2 to Suppress RNA Silencing and Plant Immunity

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Valsa mali is the major causal agent of Valsa canker, a devastating disease of apple that causes extensive bark necrosis and canker formation. Although considerable progress has been made in understanding its biology and disease management, the molecular mechanisms underlying V. mali infection remain poorly understood. Pathogens can promote infection by suppressing host RNA silencing, yet fungal RNA silencing suppressors (RSSs) remain largely unexplored. Here, we identified a novel RSS effector, VmSR1, from V. mali and elucidated its role in fungal pathogenicity and host RNA silencing. Using a GFP-based RNA silencing assay in Nicotiana benthamiana 16c, VmSR1 was identified as an effective RSS. Small RNA profiling showed that VmSR1 overexpression predominantly reduced the abundance of multiple miRNAs. Sequence and structural analyses revealed that VmSR1 lacks similarity to previously characterized fungal or oomycete RSSs. VmSR1 expression was strongly induced during infection of apple branches and deletion of VmSR1 significantly impaired V. mali virulence. Moreover, VmSR1 suppressed defense-related gene expression, reduced reactive oxygen species and callose accumulation, and compromised apple resistance to V. mali , demonstrating that VmSR1 is an important virulence effector. Mass spectrometry identified Tudor staphylococcal nuclease 2 as a VmSR1-interacting protein. Interaction assays confirmed that VmSR1 associates with NbTSN2 and its apple homolog MdTSN2 through the first three staphylococcal nuclease domains. VmSR1 promoted TSN2 degradation both in planta and in a cell-free system. RNA immunoprecipitation showed that NbTSN2 associates with multiple miRNAs, while TSN2 silencing reduced their abundance and impaired RNA silencing, supporting a role for TSN2 in maintaining miRNA homeostasis. VmSR1 also underwent liquid-liquid phase separation (LLPS) in vivo and in vitro. Its N-terminal intrinsically disordered region (IDR) contributed to stress-induced droplet formation, whereas its C-terminal IDR containing two low-complexity regions was required for droplet formation and miRNA suppression. Together, our findings identify VmSR1 as a novel fungal RSS and virulence effector that promotes TSN2 degradation and miRNA depletion, revealing a previously uncharacterized mechanism by which V. mali interferes with host RNA silencing and providing new insights into the molecular basis of fungal pathogenicity.
Key words: 8th ICBPI; Biotic Plant Interaction; RNA silencing suppressors; Tudor-SN; Valsa mali

Interaction Mechanisms Between Orchids and Their Symbiotic Fungi

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Orchidaceae, one of the largest plant families with over 28,000 species, forms obligate symbioses with orchid mycorrhizal fungi (OMF), which are essential for completing its life cycle in nature. This dependence is most pronounced during seed germination, since orchid seeds are minute and lack endosperm. They therefore require compatible fungi for germination and seedling establishment. Pre-symbiotic communication begins before physical contact. Seed exudates attract germination-promoting fungi (GPF), which are thought to release signaling molecules that plant receptors perceive, activating symbiotic signaling and suppressing basal immunity. Compatible GPF secrete cell wall-degrading enzymes that facilitate penetration of the testa, allowing the embryo to imbibe water and swell. After penetration, hyphae form intracellular coils called pelotons. Active pelotons mediate nutrient transfer across the symbiotic interface, whereas senescing ones are digested by host cells, releasing carbon and minerals that fuel protocorm development. During germination and the protocorm stage, orchids depend almost entirely on fungi for carbon, nitrogen, and phosphorus. In fully mycoheterotrophic orchids, the trehalase gene family has undergone expansion and is constitutively expressed, hydrolyzing fungal trehalose into glucose, which is then taken up by SWEET and STP transporters, enabling the orchid to hijack fungal carbon. Adult autotrophic and mixotrophic orchids instead return photosynthetic carbon to the fungus, establishing a bidirectional exchange. At the molecular level, orchids partly share the arbuscular mycorrhiza-type common symbiosis signaling pathway, in which calcium signaling and phosphorylation cascades govern fungal recognition and colonization. Gibberellin inactivation and strigolactone secretion contribute to "symbiotic licensing". Orchids also maintain a balance between defense and symbiosis through immune regulation. Given the diversity of orchid species and their symbiotic fungal partners, these symbiotic associations are not constrained to a strict generalist or specialist category but exhibit flexibility. Orchid-fungus interactions thus form a specialized system in which the peloton is the structural interface, trehalase-mediated carbon acquisition the core innovation, and immune-hormonal signaling the basis of compatibility, revealing a continuous evolutionary spectrum from mutualism to mycoheterotrophy. Together, these features offer an integrated model for understanding host recognition, immune-symbiosis balance, nutrient exchange, functional plasticity, and eco-evolutionary dynamics in plant-microbe interactions.

Key words: Orchids; Orchid mycorrhiza; Symbiotic fungi; Interaction; Germination; Nutrient exchange

Precise genome-editing of Dense and Erect Panicle 1 promotes rice sheath blight resistance and yield production in japonica rice

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The primary goals of crop breeding are to enhance yield and improve disease resistance. However, the “trade-off” mechanism, in which signaling pathways for resistance and yield are antagonistically regulated, poses challenges for achieving both simultaneously. Previously, we demonstrated that knock-out mutants of the Dense and Erect Panicle 1 (DEP1) gene can significantly enhance rice resistance to sheath blight (ShB), and we mapped DEP1’s association with panicle length. In this study, we discovered that dep1 mutants significantly reduced rice yield. Nonetheless, truncated DEP1 was able to achieve both ShB resistance and yield increase in japonica rice. To further explore the function of truncated DEP1 in promoting yield and ShB resistance, we generated CRISPR/Cas9-mediated genome-editing mutants, including a full-length deletion mutant of DEP1, named dep1, and a truncated version, dep1-cys. Upon inoculation with Rhizoctonia solani, the dep1-cys mutant demonstrated stronger ShB resistance than the dep1 mutant. Additionally, dep1-cys increased yield per plant, whereas dep1 reduced it. Compared to the full DEP1 protein, the truncated DEP1 (dep1-cys) demonstrated a decreased interaction affinity with IDD14 and increased affinity with IDD10, which are known to positively and negatively regulate ShB resistance through the activation of PIN1a and ETR2, respectively. The dep1-cys mutant exhibited higher PIN1a and lower ETR2 expression than wild-type plants, suggesting that dep1-cys modulated IDD14 and IDD10 interactions to regulate PIN1a and ETR2, thereby enhancing ShB resistance. Overall, these data indicate that precise genome editing of DEP1 could simultaneously improve both ShB resistance and yield, effectively mitigating trade-off regulation in rice.

Key words: DEP1, genome-editing, yield, sheath blight, resistance, rice

A SERK-related extracellular LRR-only protein restrains immune receptor complex assembly in rice

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Cell-surface immune receptors detect microbial molecules and recruit co-receptors, yet how extracellular host proteins tune these assemblies remains poorly understood. Here we characterize a rice immune pathway triggered by MoEIX_07955, a secreted glycoside hydrolase family 11 (GH11) xylanase from the rice blast fungus Magnaporthe oryzae. MoEIX_07955 is expressed during infection and elicits reactive oxygen species production, mitogen-activated protein kinase activation and defence-gene expression in rice. Association assays, biochemical analyses and loss-of-function genetics identify the leucine-rich repeat (LRR) receptor-like protein OsREIX2 as a required perception component. Because OsREIX2 lacks an intracellular kinase domain, we examined somatic embryogenesis receptor kinase (SERK) proteins and found that OsSERK2 associates with OsREIX2. Elicitor treatment enhances this association, whereas disruption of OsSERK2 weakens MoEIX_07955-induced immune outputs.

We next investigated OsLRR13, previously reported as OsLRR2, an apoplastic leucine-rich repeat-only protein that negatively regulates rice defence. Loss and overexpression of OsLRR13 cause reciprocal changes in elicitor-induced responses. Interaction and competition assays indicate that OsLRR13 associates with OsREIX2 and reduces OsREIX2–OsSERK2 complex formation. Ensemble structure predictions further place OsLRR13 and OsSERK2 on a highly overlapping membrane-proximal surface of OsREIX2 and favour mutually exclusive occupancy across predictions, consistent with receptor-side competition. Comparative phylogenomic and ancestral-state analyses suggest that OsLRR13 represents an ancient SERK-related LRR lineage that lost transmembrane and kinase domains while retaining most predicted receptor-contact residues.

Together, our results support a model in which OsLRR13 acts as a receptor-proximal extracellular brake that limits productive OsREIX2–OsSERK2 assembly and attenuates fungal elicitor signalling. This work identifies receptor–co-receptor assembly as a tunable control point in rice surface immunity.

Key words: Pattern-triggered immunity; receptor–co-receptor assembly; extracellular immune regulation; OsLRR13; rice

Coordination of kauralexin biosynthesis by pathogen-induced GGPP supply and a WRKY–VQ regulatory module in maize

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Kauralexins are major diterpenoid phytoalexins in maize that contribute to resistance against fungal pathogens. Although substantial progress has been made in elucidating their biosynthetic pathway, the mechanisms coordinating precursor supply and transcriptional regulation during pathogen infection remain poorly understood. Here, we investigated the biosynthesis and transcriptional regulation of kauralexins in maize. We identified ZmGGPPS2 as a pathogen-inducible geranylgeranyl diphosphate synthase that exhibited robust GGPP-producing activity and was coordinately expressed with key kauralexin biosynthetic genes. The Zmggpps2 knockout mutant showed reduced kauralexin accumulation and compromised resistance to both C. heterostrophus and F. graminearum. To investigate the transcriptional regulation of key kauralexin biosynthetic genes, we identified a WRKY transcription factor that positively regulates multiple genes involved in kauralexin biosynthesis, including ZmGGPPS2, ZmAN2 and ZmKSL2. Genetic and molecular analyses revealed that disruption of this WRKY factor impaired the expression of key kauralexin biosynthetic genes, leading to reduced kauralexin accumulation and increased susceptibility to fungal pathogens. Furthermore, a fungal pathogen-inducible VQ protein was identified as an interacting partner of the WRKY factor. The WRKY–VQ pair synergistically enhanced the transcriptional activation of multiple kauralexin biosynthetic genes. Together, our findings reveal a coordinated regulatory framework in which pathogen-induced GGPP supply and WRKY–VQ-mediated transcriptional regulation converge to promote kauralexin biosynthesis and enhance maize resistance to fungal pathogens.

Key words: Kauralexin; diterpenoid phytoalexin; ZmGGPPS2; WRKY–VQ module

Blitz Talk 3: Plant-virus interactions 植物-病毒互作

Subversion of salicylic acid (SA) signaling pathway by viral proteins promotes begomovirus transmission and infection

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The outbreak of vector-borne plant viruses entails both productive vector-mediated transmission and efficient in planta virus infection. Yet, the factors that regulate these two key viral traits remain understudied. Here we examine the role of βC1 proteins and the salicylic acid (SA)-) signaling pathway in modulating virus transmission and infection. In terms of virus transmission, whitefly infestation induces salicylic acid (SA) accumulation and in turn plant defenses against begomoviruses. Begomoviral betasatellites and βC1 proteins encoded therein interfere with whitefly infestation-induced SA accumulation and SA signaling. Mechanistically, βC1 proteins dampen SA accumulation and signaling by subverting plant EDS1-PAD4 interaction and EDS1-TGA2 association, respectively. Additionally, during virus inoculation on un-infected plants by whitefly, whitefly infestation-induced resistance is inhibited when begomoviruses were co-inoculated with their cognate betasatellites. βC1 proteins encoded by the betasatellites suppress whitefly infestation and SA-induced antiviral immunity by interfering with the signal transduction of SA. Mechanistically, βC1 proteins interact with heat shock protein 90s (NbHSP90s), which binds to NbNPR3 (Nicotiana benthamiana nonexpressor of pathogenesis-related genes 3), a negative regulator of SA signaling. βC1 does not bind directly to NbNPR3, but inhibits SA-induced NbNPR3 degradation via NbHSP90s. In terms of virus infection, Sri Lankan cassava mosaic virus (SLCMV) dramatically induces the accumulation of SA, a positive regulator of antiviral defense. As countermeasures, SLCMV DNA-B and the BV1 protein encoded therein interfere with SA-induced antiviral defenses and SA signal transduction. Mechanistic explorations reveal that BV1 downregulates the transcription of BTB/POZ and TAZ domain-containing protein 1 (BT1), a positive regulator of plant SA signal transduction, plant antiviral defenses. Our findings reveal exquisite mechanisms that facilitate the persistence of vector-borne plant viruses and provide important insights into the intricacies of the virus life cycle.

Keywords: Plant virus transmission and infection; Salicylic acid signaling pathway; βC1.

An unconventional endocytic pathway mediates plasma membrane-to-chloroplast communication in plant immunity and is co-opted by pathogens

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Chloroplasts are emerging as central signaling hubs during plant-pathogens interactions1. However, how plasma membrane (PM)-perceived threat signals are transduced to chloroplasts remains poorly understood2,3. Previous studies have shown that the tomato yellow leaf curl virus-encoded C4 protein and the plant calcium-dependent protein kinase 16 (CPK16) relocalize to chloroplasts upon threats perception and antagonistically regulate SA signaling2. Here, using these two proteins as probes, we uncovered an unconventional defensive endocytic pathway that mediates PM-to-chloroplast protein transport. Upon defense activation, the PM-localized RING-domain E3 ligase RGLG2 physically interacts with and ubiquitinates C4 and CPK16, thereby promoting their internalization through clathrin-mediated endocytosis. Interference with the endocytic pathway using either dominant-negative mutants or chemical inhibitors disrupts the re-localization of C4 and CPK16 from the PM to chloroplasts, indicating that an uncharacterized immune-related membrane trafficking pathway connecting the cell surface to these organelles exists in plants. Strikingly, an ubiquitination-deficient C4 variant fails to re-localize to chloroplast and negatively impacts the viral infection. Taken together, our findings delineate a new endocytic route linking PM and chloroplasts in immune signal transduction exploited by viruses to suppress defence.

Key words: Endocytosis; Membrane trafficking; Signal transduction; Chloroplast; Ubiquitination; Virus

Sugarcane streak mosaic virus P1 protein targets the BES1 transcription factor to subvert DAMP- triggered immunity in plant

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Sugarcane streak mosaic virus (SCSMV) is the major cause of sugarcane mosaic disease. Its P1 protein (P1SCSMV) is crucial for pathogenesis, but how it disrupts plant immunity remains unclear. Here, we found that P1SCSMV directly binds to the core transcription factor of BR signaling, NbBES1, disrupting its binding to the E-box elements of the xyloglucan endotransglucosylase/hydrolase gene 1 (NbXTH1) promoter and suppressing NbXTH1 transcription. NbXTH1 over-expression enhanced the plant resistance to fungal/viral infection, improved the contents of xyloglucan, which directly induced a damage-associated molecular pattern-(DAMP) triggering innate immunity (DTI) and expression of several defense genes, and accompanied with a thickened middle lamellae and primary cell wall. Reduced xyloglucan due to NbXTH1 down-expression further weakened DTI responses and a thinner cell wall. This study reveals how a viral protein subverts cell wall-mediated immunity and identifies NbXTH1 as a broad-spectrum disease resistance factor. Our findings support using xyloglucan or NbXTH1 to engineer broad-spectrum disease resistance in crops.

Key words: Sugarcane streak mosaic virus; P1; wide-spectrum disease resistance; NbXTH1; xyloglucan, cell-wall based innate immunity; DAMP-triggered immunity;

Sugarcane mosaic virus manipulates maize RNA splicing factors to promote infection and pathogenesis

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Sugarcane mosaic virus (SCMV), an important member of the genus Potyvirus, infects maize, sugarcane, and sorghum. It is the primary pathogen causing maize dwarf mosaic disease, can cause 20%–80% yield losses in maize, and seriously threatens global food security. Pre-mRNA splicing is a key step in gene expression regulation; however, the molecular mechanisms by which plant viruses manipulate the host splicing machinery to promote infection and pathogenesis remain largely unclear. Our studies integrated TurboID proximity-labeling proteomics, single-molecule long-read transcriptome sequencing, and other molecular biology, bioinformatics, biochemistry, and genetics approaches to systematically dissect the dual molecular mechanisms by which the SCMV-encoded pathogenicity protein NIa-Pro manipulates maize RNA splicing factors.
On the one hand, NIa-Pro interacts in the nucleus with the conserved splicing factor ZmU2AF65B in a K230/D234-dependent manner, thereby inhibiting the binding of ZmU2AF65B to ZmUPF3 pre-mRNA. This leads to abnormal splicing of ZmUPF3 and a reduction in functional transcripts, which in turn weakens the mRNA surveillance pathway. Genetic evidence shows that a loss-of-function mutant of ZmU2AF65B promotes viral infection and pathogenesis, whereas restoring ZmUPF3-3Flag expression in this mutant background suppresses viral genomic RNA accumulation. In addition, a mutant virus carrying NIa-Pro substitutions that disrupt its interaction with ZmU2AF65B (K230A/D234A) exhibits significantly reduced virulence. On the other hand, we recently found that NIa-Pro hijacks eIF4A3, a core component of the exon junction complex and a DEAD-box RNA helicase, into viral replication complexes. Through steric hindrance, this blocks m6A deposition by the maize m6A methyltransferase ZmMTA at position A6556 of the viral genomic RNA, allowing the viral RNA to evade degradation mediated by the m6A reader ZmECT23, which recruits the ZmCCR4-NOT complex. Silencing ZmeIF4A3 increases viral genomic m6A levels and inhibits infection, whereas its overexpression exacerbates symptoms. Moreover, this mechanism is conserved in potato virus Y and turnip mosaic virus. Together, these findings deepen our understanding of how splicing factors regulate the mRNA surveillance pathway and m6A-mediated antiviral mechanisms, and provide a theoretical basis and potential targets for engineering potyvirus-resistant crops by targeting splicing factors.
Keywords: sugarcane mosaic virus; NIa-Pro; RNA splicing factor; m6A modification

A bacterial symbiont and a plant virus enhance insect fitness by inducing physical defenses against fungal parasites

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The defensive symbiosis that beneficial microbes protect hosts from natural enemies is ubiquitous across animals and plants in nature. However, evidence from the field and underlying mechanisms is scarce. Field surveys and laboratory assays revealed that infection of the invasive whitefly Bemisia tabaci by the bacterial symbiont Rickettsia and plant begomovirus were positively correlated with each other but each negatively correlated with a parasitic fungal infection (Beauveria bassiana) in the host. We show that begomovirus conferred whitefly's resistance to the parasitic fungus by triggering the expression of chitin synthesis pathway genes in whiteflies, reinforcing the cuticle by promoting chitin production. The facultative symbiont Rickettsia facilitated cuticle formation and thereby induced physical defense against entomopathogenic fungus via metabolic cooperation with the obligate symbiont Portiera for the synthesis of phenylalanine and tyrosine in whiteflies, which is used to generate cuticular proteins and pigments. Mutation of chitinase and protease genes in B. bassiana impaired fungal infection of whiteflies. Inhibiting whitefly cuticle formation by repressing chitin and phenylalanine synthesis facilitated fungal infection. Thus, begomovirus and Rickettsia have convergent effects on cuticle defense in whiteflies by impacting distinct molecular pathways. Such defensive symbioses apparently contribute to B. tabaci fitness in the field and our findings reveal that interactions among the host, beneficial microbes, and pathogens have important implications for insect ecology and evolution. This study suggests new avenues for pest management by leveraging defensive microbes and targeting the host cuticle.

Keywords: Defensive symbiosis; Begomovirus; Symbiont Rickettsia; Bemisia tabaci; Parasitic fungal infection

A novel RNase III nuclease confers broad-spectrum antiviral immunity independent of canonical DCL-mediated RNAi pathways in plants

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Nucleases including CRISPR/Cas9 and DCLs play important roles in distinct antiviral defense. Plant viruses often cause devastating damages to crop productions and global food security. Through this study, we found that a novel land-plant-specific RNase III nuclease RTL2 plays a critical role in antiviral immunity in plants. RTL2 dimerizes to form an active RNase III catalytic center which can cleave dsRNA during virus infection. This RTL2 confers a broad-spectrum antiviral resistance independent of the canonical DCL-mediated RNAi pathways. To counteract this defense pathway, diverse viruses have evolved effectors (e.g., TSWV-NSs, TBSV-P19 and others) to interfere with its dimerization and disrupted its dsRNA-cleavage activity. Our phylogenetic analysis further reveals that plant RTL proteins constitute a land-plant-specific RNase III subfamily that deviated from the canonical DCLs. In summary, our finding discovers a previously unrecognized DCL-independent antiviral defense pathway, and diverse plant viruses have evolved mechanisms to evade this immune pathway during co-evolution.

Key words: RNA-targeted antiviral defense; RNase III; RTL2; viral effector; dsRNA

Manipulation of the plant RNA splicing machinery by geminiviruses

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Geminiviruses are a large family of plant DNA viruses that poses a serious threat to agriculture and food security worldwide. Geminiviruses replicate in the nuclei of the infected cells, and due to their limited coding capacity, they rely heavily on the host machinery for replication and spread. While studies in mammalian viruses have uncovered splicing as a key host mechanism hijacked during infection, it remains elusive if geminiviruses generally take advantage of this process to increase their transcriptome and proteome diversity. We have recently found that i) the geminiviral replication-related protein, Rep, physically associates with the plant splicing machinery; ii) geminiviral transcripts are spliced and give rise to new proteins with functional relevance during infection; iii) splicing is required for geminiviral replication and systemic infection; and iv) the geminiviral infection drastically impacts the host splicing landscape. Therefore, splicing emerges as a central process at the interface between geminiviruses and their host plants. Through a functional screen, we identified two host splicing factors, MAC3A (MOS4-Associated Complex 3A) and SMU2 (SUPPRESSORSOF MEC-8 AND UNC-52 2), as essential regulators of TYLCV infection. This requirement extends to other geminiviruses, including chickpea chlorotic dwarf virus (CpCDV) and beet curly top virus (BCTV), suggesting a conserved viral strategy. Together, our findings uncover a novel mechanism where geminiviruses hijack MAC3A and SMU2 to promote infection, offering promising targets for breeding broad-spectrum geminivirus resistance in crops.

Key words: Geminivirus; TYLCV Rep; RNA splicing; Viral infection

Liquid-liquid phase separation of XRN4 contributes to antiviral defense against BSCTV

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The 5′-3′ exoribonuclease XRN4 plays an important role in plant RNA metabolism, yet how its condensation contributes to antiviral defense remains poorly understood. Here, we identify XRN4 as a restriction factor against beet severe curly top virus (BSCTV) and investigate the relationship between its phase separation and antiviral activity. Silencing XRN4 in Nicotiana benthamiana or loss of XRN4 in Arabidopsis thaliana increased susceptibility to BSCTV, whereas AtXRN4 overexpression reduced viral accumulation. AtXRN4 formed dynamic cytoplasmic condensates exhibiting fluorescence recovery after photobleaching and fusion behavior. Recombinant AtXRN4 also formed droplets in vitro, supporting its capacity for liquid–liquid phase separation. Deletion of the C-terminal intrinsically disordered region (IDR) markedly impaired condensate formation and compromised antiviral activity, indicating that this region is important for both processes. Further mutational analysis showed that substitutions at S284 markedly reduced the phospho-Ser/Thr signal detected in immunoprecipitated XRN4. The S284A and S284D variants exhibited reduced punctate condensation, while the S284A variant showed impaired antiviral activity and S284D retained viral restriction. These findings suggest that the phosphorylation state of S284 may influence XRN4 condensation and antiviral function. Together, our findings support a contribution of XRN4 phase separation to antiviral defense and identify its C-terminal IDR and S284 residue as important determinants of its condensation and function. This work provides insight into the spatial regulation of an RNA decay factor during plant antiviral defense.

Key words: XRN4; Liquid–liquid phase separation; Phosphorylation; Beet severe curly top virus

UPR-driven ER-phagy confers antiviral defense against ER-replicating RNA viruses

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Positive-strand RNA [(+)RNA] viruses induce endomembrane remodeling to form viral replication organelles (VROs), which disrupt organelle homeostasis. How hosts restore organelle homeostasis and how these responses influence viral replication remain elusive. Using beet black scorch virus (BBSV), a (+)RNA virus that replicates on the endoplasmic-reticulum (ER) and induces severe deformation of ER membranes, as a model in Nicotiana benthamiana, we demonstrated that BBSV induces ER-phagy, primarily mediated by its auxiliary replication protein p23. p23 interacts with the ER-phagy receptor NbSec62, with phenylalanine at position 48 being critical for this interaction and ER-phagy induction. Upon BBSV infection, the unfolded protein response (UPR) is triggered to promote viral replication. However, the activation of the UPR also induces NbSec62-mediated ER-phagy to suppress BBSV replication. Furthermore, NbSec62 restricts other ER-replicating (+)RNA viruses, including tobacco mosaic virus and turnip mosaic virus. Our findings reveal NbSec62 as a restriction factor that interacts with BBSV VROs to regulate the balance of viral replication and ER homeostasis, providing insights into the UPR-ER-phagy signaling network in virus–host interactions.

A Kunitz-type protease inhibitor restricts potyvirus infection by targeting the viral NIa protease

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The infection of plant viruses is one of the most significant threats to plants. Potyvirus is the largest genus of positive-single-sense RNA plant viruses. Potyviruses (viruses in the genus of Potyvirus) employ a unique polyprotein translation strategy, the polyproteins are precisely cleaved by the virus-encoded proteases to generate mature viral proteins. The interaction between protease inhibitors and proteases is a common defense and counter-defense strategy in the co-evolution of plants and pathogens. Therefore, is there a protease inhibitor present in plants that can inhibit the proteolytic cleavage strategy of Potyviruses? In this study, we used TuMV, a representative virus of Potyviruses, as a model for further investigation. By analyzing the transcriptome data of TuMV non-infected and infected Nicotiana benthamiana plants, we identified an up-regulated gene belonging to the Kunitz protease inhibitor family and named it NbKTI1. Experimental results confirmed that NbKTI1 positively regulates plant resistance to TuMV and potato virus Y (PVY), inhibiting virus infection. Further study revealed that NbKTI1 can specifically inhibit the cleavage activity of NIa, but not P1 or HC-Pro. Using several protein interaction assays, NbKTI1 was found to interact with NIa and NIb encoded by TuMV in vivo. Additionally, NbKTI1 can be recruited into the virus replication complex, further indicating its involvement in regulating TuMV replication.

Together, our findings reveal a plant endogenous Kunitz-type protease inhibitor that targets potyviral NIa protease, thereby suppressing viral polyprotein processing and viral infection. This study provides new insight into protease inhibitor-mediated antiviral immunity and highlights a potential strategy for controlling Potyviruses.

Keywords: Potyvirus, NbKTI1, Protease NIa-Pro, Antiviral immunity

Dual functional plant gene inhibits viral infection and development of systemic necrosis symptoms by distinct mechanisms in Arabidopsis

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Specific targeting of host genes by plant viruses leads to the induction of distinct disease symptoms with devastating consequences for agriculture. However, it is unknown whether plants have evolved genes to specifically inhibit the development of viral disease symptoms. Here we identified the causal gene in 2 Arabidopsis thaliana mutants that develop prominent systemic necrosis after infection with both wild-type cucumber mosaic virus (CMV) and its mutant (CMV-Δ2b), which is defective in the suppression of antiviral RNA interference. We found that antiviral RNAi 5 (AVI5) is located in a large chromosomal deletion in both Arabidopsis mutants. We demonstrate that AVI5 acts to inhibit CMV-Δ2b viral accumulation by promoting the biogenesis of virus-derived small interfering RNAs in the antiviral RNA interference pathway before the host amplifies these small interfering RNAs. We further show that cucumber mosaic virus infection induces systemic necrosis symptoms associated with cell death and a reactive oxygen species burst and that AVI5 potently inhibits them. Unlike its suppression of virus accumulation, however, AVI5 inhibits the development of systemic necrosis symptoms without reducing viral titers or inducing antiviral RNA interference. Together, our study demonstrates that Arabidopsis plants have evolved a dual functional gene for antiviral protection to inhibit viral infection and symptom development by distinct mechanisms.

Key words: AVI5; Antiviral RNAi; vsiRNA; Systemic necrosis

Blitz Talk 4: Plant-insect/nematode/plant parasite interactions 植物-昆虫/线虫/寄生植物互作

Designing experiments for plant resistance screening to two stressors at once

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Tetranychus urticae (the Two-Spotted Spider Mite) is one of the major pests in tomato production in Isreal. It is an economically important pest in numerous crops worldwide and is known for its pesticide-resistance abilities.
T. urticae is particularly problematic during the hot seasons, as it is highly heat resistant while biological control means are not. This means growers must rely solely on chemical control, until this too fails.
A collection of 166 Backcross Introgression Lines between the commercial tomato Solanum lycopersicum and the wild type S. pimpinellifolium has been screened for heat resistance at the Liberman- Lazarovich lab and several lines were identified with improved pollen survival and fruit setting under hot conditions.
We set out to screen the same collection for T. urticae resistance under hot conditions. However, as heat affects both the plants and the mites, we designed several screen methods to try and separate the effect of heat on the mites from its effect on the plants and their interaction.
By doing this, we were able to identify several potential plant properties allowing resistance to T. urticae. Our results indicate the significance of experimental design when examining the effects of more than one stressor, especially when testing both biotic and abiotic stressors with interacting effects.
Key words: Mite Pests; Plant Resistance; Heat; Tomato.

Dynamic molecular responses of Rhopalosiphum padi to host switching between maize and wheat

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The bird cherry-oat aphid, Rhopalosiphum padi, is an important cereal pest with a broad host range and strong host-switching capacity. However, the molecular mechanisms underlying their rapid adaptation to different host plants remain poorly understood. Here, we combined a telomere-to-telomere reference genome of R. padi with transcriptomic and metagenomic profiling to investigate early responses during reciprocal host switching between maize and wheat. Aphids were transferred from maize to wheat and from wheat to maize and sampled at 6 h and 24 h after host transfer. We aim to characterize shared and host-specific transcriptional responses, distinguish early and sustained molecular changes, and evaluate potential contributions of aphid-associated microorganisms to host adaptation. This integrated framework provides a genomic basis for understanding the molecular plasticity of R. padi during plant–insect interactions and may contribute to improved management of cereal aphids.

Mannitol dehydrogenases in nematode infection and soybean resistance

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Two experimental routes to reduce soybean cyst nematode infection. New molecular targets could complement existing SCN resistance. We tested two nematode MTD genes, Hetgly10886 and Hetgly11294. Docking, binding and enzyme assays support MTD activity. Mannitol treatments alter root infection. RNAi reduces movement, root attraction, nematode development and cyst formation. MTD overexpression in soybean reduces nematode development and cyst burden. Infection assays in Wm82 and Forrest, feeding-site imaging, and agronomic evaluation of overexpression lines. MTD genes are candidate targets for RNAi-based control and soybean resistance improvement.

Trichome acylsugar defenses are equally effective against leaf-mining and free-living insects

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Trichomes are epidermal projections, crucial components of plant defense systems. Whether these epidermal defenses are effective against leafminers, which infest the internal mesophyll layers, remains unclear. Using the devasting invasive tomato pest, Phthorimaea absoluta (tomato leafminer), and the trichome-rich wild tomato, Solanum pennellii, we demonstrate that trichome produced acylsugars effectively inhibit the growth of endophagous P. absoluta larvae feeding inside leaves. High spatial resolution leaf- and frass-metabolite analysis by MALDI-MSI and Stimulated Raman Scattering microscopy revealed that trichome-synthesized acylsugars are translocated into the mesophyll tissues when larvae infest leaves. We identified an ATP-binding cassette (ABC) transporter, SpABCB5, as the mediator of this translocation and as an essential component of defense against leafminers. These data reveal that epidermal trichomes not only defend against free-living (ectophagous) herbivores through direct contact or rapid deposition of chemical defenses onto leaf surfaces, but also defend against endophagous leafminers by rapidly depositing chemical defenses into internal tissues. By delivering mines against leaf miners, trichomes offer new avenues for breeding crops with resistance to leafminers.

Key words: Trichome, leafminer, pest resistance, acylsugars, transport, Stimulated Raman Scattering microscopy, MALDI

Nicotiana phytaspase-1 as a tuning node for plant sensitivity to oxidative stress

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Plants face oxidative stress from various subcellular compartments during diverse biotic interactions, requiring precise decision-making between stress adaptation and programmed cell death (PCD). Phytaspases (plant aspartate-specific subtilases) act as crucial regulators of PCD; the activity was first uncovered during the hypersensitive response to tobacco mosaic virus and shown to restrict Agrobacterium T-DNA transfer via VirD2 cleavage. Under normal conditions, phytaspase is secreted into the apoplast; however, upon PCD induction, it is internalized via clathrin-mediated endocytosis (CME), and this re-entry is essential for executing the cell death program. In this work, a conceptual model is proposed. Instead of a universal response to any oxidative stimulus, phytaspase acts as a regulatory tuning node, adjusting cellular sensitivity to reactive oxygen species (ROS) depending on their source compartment. It draws on our previous studies with Nicotiana benthamiana and Nicotiana tabacum phytaspase-1 and identifies 3 types of compartment-specific responses. Mitochondrial ROS trigger CME-dependent internalization. This is a "detect and destroy" strategy leading to PCD. Blocking CME prevents phytaspase-1 entry and death. Chloroplast ROS initiate an opposing mechanism. Under moderate stress, apoplastic phytaspase-1 decreases through degradation. This reduces cellular sensitivity, implementing a "wait it out and survive" strategy. Chloroplast ROS sources such as excess light subside on their own, and desensitization prevents death over a transient stimulus. Severity is therefore set by persistence, not dose: only stress whose source cannot be removed overcomes desensitization and drives phytaspase-1 back into the cell. Apoplastic ROS, generated in response to wounding and other stressful stimuli, systemically increase phytaspase-1 expression and activity. The burst alone is too transient to trigger death, but the expanded pool raises ROS sensitivity, so the next stimulus suffices; this can be interpreted as immune priming. Thus, cell fate is determined by an equation where the decision regarding death is influenced not only by the stimulus (ROS generation) but also by the plant's sensitivity to this stimulus, set by the amount of apoplastic phytaspase-1. By dynamically modulating its localization, degradation, or overproduction in response to specific ROS signatures, phytaspase-1 operates as a highly specialized rheostat, maintaining the balance between adaptation, systemic immunity, and PCD.
Key words: phytaspase; subtilase; programmed cell death; reactive oxygen species; hypersensitive response; wound signaling

Modeling Bemisia tabaci Phenological Shifts Under Global Warming

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Global climate change has progressively elevated ambient temperatures, intensifying extreme thermal events worldwide. These shifts exert direct physiological pressures on major agricultural pests like the whitefly (Bemisia tabaci), a devastating phloem-feeder and highly efficient viral vector in common beans (Phaseolus vulgaris). This study investigated the multi-decadal impact of sustained temperature increments across a 26-year historical window on biological parameters of the whitefly B. tabaci at seasonal intervals (1999, 2000 and 2026) through comprehensive semi-field biological assays. Common bean plants were infested with adult whiteflies (4h), then, the developmental stage of insects (100 in 2025 and 2026; 30 in 1999) on detached leaves was evaluated daily until adult emergence. Nymph length and width were measured at all stages. Air temperature in the greenhouse was recorded daily. We applied Functional Data Analysis (FDA) to model cumulative transition percentages across B. tabaci instars as continuous curves over time. This non-parametric approach outperforms traditional multivariate statistics by incorporating longitudinal dependence and strict monotonicity in nymphal development. Trajectories were estimated using fourth-order cubic B-spline basis smoothing in R software as a function of development days. The thermal regime profoundly influenced biological development, demonstrating that daily temperature peaks and thermal amplitude—rather than isolated heat accumulation—dictate cycle duration. In 2025, extreme maximum temperatures (peaks up to 41.5 °C) accelerated the cycle to just 21 days, ending development with a lower total heat accumulation (162.7 GDD; 7.75 GDD/day). In 1999, mild temperatures (24–30 °C) prolonged the cycle to 39 days with a slower, constant accumulation rate (193.8 GDD; 4.97 GDD/day). Conversely, 2026 recorded the highest heat accumulation (315.3 GDD; 10.17 GDD/day); however, the combination of high daytime temperatures and cold nights increased thermal amplitude, providing optimal developmental conditions. Furthermore, 2025 and 2026 populations exhibited developmental imbalances relative to Dyar's rule, yielding unexpectedly larger final nymphal body sizes under elevated thermal regimes. Multi-decadal warming acts as a strong ecological driver, shifting B. tabaci life history traits by altering developmental pacing, thermal amplitude responses, and structural growth. These empirical findings underscore an urgent imperative to adopt current Integrated Pest Management (IPM) frameworks to address accelerated population turnovers.

Keywords: whitefly; Phaseolus vulgaris; Insect phenology; Growing degree-days; Thermal amplitude; Integrated Pest Management.

Callosobruchus maculatus feeding triggers multilevel defenses in cowpea seeds: from germination regulation to defensive metabolite biosynthesis

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In-depth research has been conducted on plant-herbivore interactions, and most studies on resistance to insects have been focused on plants post-emergence. Currently, little is known about how seeds respond to pest feeding. Understanding how seeds defend against pests is crucial for seed security and agricultural production. Our study revealed that mild infestation by cowpea weevil (Callosobruchus maculatus), a major pest of stored pulses, activated a defense response in stored cowpea seeds. The infestation eliminated the hardseededness and upregulated the germination-prompted genes, thereby increasing the germination rate. Detailed analysis revealed that infestation induced the expression of a series of defense genes, including those involved in the jasmonic acid (JA) signaling pathway and terpenoid synthetic pathway. Transcriptomic and metabolomic analyses identified flavonoid biosynthesis as the primary response in the infested seeds. Specifically, the isoflavonoid biosynthesis was active, with increased expression of corresponding genes and accumulation of metabolites. Further Y-tube olfactometer selection experiments showed that the infested cowpea seeds attracted male insects but repelled females, indicating pest feeding altered the volatile emitted by cowpeas. Our findings thus elucidated the interaction between cowpea seeds and the storage pest and indicated that seeds and plants employ similar defense systems against pests. The study also provides theoretical guidance for controlling C. maculatus.

Overexpression of salicylic acid methyltransferase (ZmSAMT) enhances resistance to Rhopalosiphum padi in maize. Running title: ZmSAMT-overexpressing maize enhances resistance to aphid

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BACKGROUND: Aphids are important pests of maize, including Bt-transgenic maize, and their effective management remains a major challenge. Herbivore-induced plant volatiles (HIPVs), particularly methyl salicylate (MeSA), play a critical role in mediating tritrophic interactions and plant defense in agroecosystems. Exogenous application of synthetic MeSA can be effective against aphids, however, its pronounced volatility and maintenance costs limit large-scale agricultural application.

RESULTS: In this study, we generated ZmSAMT-overexpressing transgenic maize to enable continuous endogenous MeSA emission, establishing a sustainable strategy for aphid management. In vitro enzymatic assays confirmed that ZmSAMT specifically methylated salicylic acid (SA) to form MeSA. Notably, aphid infestation triggered significant upregulation of ZmSAMT expression in wild-type maize. Behavioral assays demonstrated that elevated MeSA emission in transgenic line OE3 significantly repelled Rhopalosiphum padi and actively recruit the predator ladybeetle Harmonia axyridis. Independent of olfactory-mediated effects, aphids feeding on OE3 plants exhibited prolonged developmental duration, a shortened reproductive period, and reduced total fecundity. Furthermore, we observed a significant airborne defense effect that wild type maize situated adjacent to the transgenic plants OE3 harbored fewer aphids.

CONCLUSION: Taken together, our studies indicate that ZmSAMT represents a highly valuable genetic resource for breeding aphid-resistant maize varieties and developing green pest control strategies.

Key words: Herbivore-induced plant volatiles (HIPVs); MeSA; ZmSAMT; aphid resistant; green pest control

Gmγ-ECS2-dependent glutathione metabolism affects soybean cyst nematode development and root redox responses

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Soybean cyst nematode (SCN; Heterodera glycines) infection is associated with changes in host glutathione (GSH) metabolism, but the contribution of GSH biosynthesis to nematode development remains unclear. We investigated CRISPR/Cas9-edited soybean Gmγ-ECS2 (Glyma.05G207600) mutants and evaluated the effects of exogenous GSH on plant growth, nematode infection, thiol metabolites, and antioxidant responses. Compared with Williams 82, gmγ-ecs2#12 and gmγ-ecs2#68 had lower root GSH concentrations, fewer nematodes, a lower proportion of developing juveniles, and fewer cysts. The gmγ-ecs2#68 mutant also exhibited reduced seedling height and cotyledon-to-true-leaf length. Exogenous GSH increased GSH levels and partially improved the growth phenotype of uninfected gmγ-ecs2#68 plants. Following SCN inoculation, root GSH and homoglutathione (hGSH) levels increased markedly in gmγ-ecs2#68, while exogenous GSH-treated plants showed changes in thiol accumulation and growth. The mean number of second-stage juveniles in GSH-treated mutant roots was higher than in untreated mutant roots, although this difference was not statistically significant in the presented data. Biochemical measurements further revealed treatment-dependent changes in hydrogen peroxide (H_2O_2), malondialdehyde, γ-glutamylcysteine, and the activities of catalase, peroxidase, glutathione reductase, and superoxide dismutase. Together, these results link Gmγ-ECS2-dependent GSH metabolism with soybean growth and SCN development and indicate that GSH availability is associated with changes in the root redox response during infection.

Key words: glutathione; Gmγ-ECS2; soybean cyst nematode; homoglutathione; redox homeostasis

Blitz Talk 5: Rhizobium and mycorrhizal symbiosis 根瘤菌与菌根共生

The Medicago SPX1/3-PHR2 network relays phosphate signaling to orchestrate root nodulation-dependent nitrogen acquisition by controlling flavonoid biosynthesis

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The formation of symbiotic associations with rhizospheric microbes is a key strategy by which sessile plants acquire nitrogen and phosphorus from the soil. Root exudates play a central role in shaping the rhizosphere microbiome, and plants can adjust exudate composition in response to their nitrogen or phosphorus demands to recruit appropriate microbial partners. Flavonoids, a major class of secondary metabolites, have been extensively studied for their roles in shaping the root microbiome, particularly in mediating root nodule symbiosis in legumes. However, the mechanisms by which plants coordinate microbe-mediated nitrogen and phosphorus acquisition remain poorly understood. Here, we demonstrate that the Medicago truncatula phosphate starvation response regulatory network SPX1/3–PHR2 controls flavonoid biosynthesis to recruit nitrogen-fixing microbes for nitrogen acquisition. The abundance of nitrogen-fixing microbes, including rhizobia, is reduced in the rhizosphere of the spx1 spx3 double mutant. This reduction is associated with decreased flavonoid levels in root exudates compared with wild-type R108 plants. Further analyses indicate that regulation of flavonoid biosynthesis is mediated by PHR2, a transcription factor that interacts with SPX1/3. Under phosphate-limiting conditions, PHR2 suppresses the expression of flavonoid biosynthetic genes, thereby reducing root nodule symbiosis. By contrast, under phosphate-sufficient conditions, interaction between SPX1/3 and PHR2 relieves this repression, promoting root nodule symbiosis. We further show that PHR2 binds to the promoter regions of flavonoid biosynthetic genes in yeast. Together, these findings support a model in which the SPX1/3–PHR2 network modulates root nodule-dependent nitrogen acquisition in response to phosphate availability. Thus, the SPX1/3–PHR2 module balances microbe-mediated nitrogen and phosphorus acquisition to support optimal plant growth.

Key words: SPX proteins; flavonoids; microbiomes; nitrogen fixation; nitrogen-phosphorus balance

AP2/ERF bifunctional transcription factors regulate arbuscular mycorrhizal symbiosis homeostasis

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By establishing symbiosis with arbuscular mycorrhizal fungi (AMF), plants exchange their own fatty acids for mineral nutrients—such as phosphorus and nitrogen—supplied by the fungi. This represents one of the most important forms of symbiosis in nature and is essential for efficient plant nutrient acquisition and for ecological balance. To safeguard the mutualistic nature of this symbiosis, plants must precisely regulate resource allocation. AP2/ERF transcription factors, including ERM1, WRI5a and LjCBX1, have been shown to act as transcriptional activators that promote symbiosis by positively regulating nutrient exchange during mycorrhizal symbiosis. Interestingly, all of these transcription factors harbor a conserved transcriptional repression motif, the EAR motif, at their C-termini. Our results show that ERM1 interacts with WRI5a through its EAR motif, and that this motif is indispensable for the full transcriptional activation activity of ERM1. At the same time, ERM1 can also recruit, via the EAR motif, the co-repressor TPR4 and histone deacetylases HDAC2/8 to assemble a transcriptional repressor complex, which represses the expression of symbiosis-related genes and thereby exerts a dual function in restraining AM symbiosis. We further found that WRI5a, MtCBX1 and MtCBX2 can likewise recruit TOPLESS co-repressors through their EAR motifs. Together, these findings suggest that AP2/ERF bifunctional transcription factors achieve precise control of AM symbiosis homeostasis through a non-canonical "one target, dual control" mode of action. This study will provide an innovative perspective for the theoretical framework of mycorrhizal autoregulation, and offer a theoretical basis and molecular targets for the genetic improvement of symbiotic benefits in crops.

Key words: arbuscular mycorrhizal fungi; AP2/ERF transcription factor; nutrient exchange; EAR motif; transcriptional repression

The discovery and characterisation of cyclic peptide epichloëcyclins from the filamentous fungal endophyte, Epichloë

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Epichloëcyclins are ribosomally synthesized and post-translationally modified peptides (RiPPs) produced by Epichloë filamentous fungal endophytes that form enduring symbioses with temperate grasses. Epichloëcyclin biosynthesis has been characterised through functional analysis using gene disruption, followed by chemical analysis in planta of four clustered and coregulated genes in Epichloë festucae. All four genes are essential for the biosynthesis of epichloëcyclins and appear conserved in all strains of Epichloë species analysed to date. The grass-induced gene A (gigA) produces one to many RiPPs from a single precursor peptide and strain-specific variant combinations (from A to Z) are widely present across the species. The 8−9-mer core epichloëcyclin peptide can be produced based on a variable number of imperfect repeats of a 27 amino acid sequence encoded in the gigA gene. The other clustered genes required for biosynthesis are gigB, which encodes a DUF3328 domain-containing protein associated with cyclization of epichloëcyclins; gigC, encoding a methyltransferase enzyme responsible for N-methylation of desmethylepichloëcyclins; and kexB, encoding a subtilisin-like enzyme, partly responsible for the pro-peptide cleavage of epichloëcyclin intermediates. Epichloëcyclins are likely to belong to the dikaritin family, but a full structural determination is still required. These RiPPs are not unique to Epichloë, as bioinformatic analyses of the four biosynthetic clustered proteins have led to the discovery of orthologous proteins in Ophiocordyceps sinensis and in seven Aspergillus species. The role of these natural products remains elusive; however, they are not involved in maintaining the symbiotic relationship of Epichloë with its grass host.

Impact of CO2 availability on the systemic regulation of symbiotic root nitrogen-fixing nodulation in legume plants

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Elevated CO2 (eCO2), eg 1000 ppm, corresponding to a possible future of agriculture at the end of the century, promotes symbiotic root nitrogen (N)-fixing nodulation in different legume plants, including Medicago truncatula. In order to determine how N-related systemic pathways regulating nodulation are affected by CO2 availability, we analyzed in eCO2 conditions the root and nodulation phenotypes of the compact root architecture 2 (cra2) mutant. Indeed, CRA2 encodes a receptor that is required for the C-terminally Encoded Peptide (CEP) signaling peptide promotion of root nodulation (Mohd-Radzman et al., 2016). We observed that cra2 mutants do not show the eCO2 increase of nodule number that is observed in wild-type plants. As the CEP/CRA2 pathway systemically promotes root nodulation from shoots, we then tested the regulation of the expression of the miR2111 shoot-to-root effector which expression is induced in shoots through the CEP/CRA2 pathway to promote in roots nodulation (Gautrat et al., 2020), but no strong regulation could be detected in response to eCO2. To identify other candidate shoot-to-root signals that could be involved in the CRA2-dependent N-related regulation of nodulation depending CO2 availability, we performed a RNAseq analysis in wild-type and cra2 mutant plants grown under ambient CO2 and eCO2. This revealed that another already known shoot-to-root N-dependent systemic signal acting downstream of the CEP / CRA2 pathway, named CEP Downstream (CEPD), was rapidly upregulated by eCO2 specifically in shoots depending on CRA2. We then generated cepd1/cepd2 mutants by CRISPR/Cas9 genome editing to investigate CEPD function in nodulation. We found that the increased nodule number in response to eCO2 observed in wild-type plants was lost in cepd1/cepd2 mutants.

Keywords: carbon; systemic regulation; rhizobium; Medicago truncatula; legume; nitrogen-fixing nodule; symbiosis

Host plant identity shapes microbial communities, functional potential and metabolite profiles in the Rhizophagus irregularis hyphosphere

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Most land plants rely on symbioses with arbuscular mycorrhizal (AM) fungi for mineral nutrient acquisition. AM fungal hyphae can recruit diverse microbiomes that contribute to nutrient mobilization, however the role of host plants in shaping hyphosphere community assembly and function remains poorly understood.

We used a series of in vitro and mesocosm experiments to investigate the effect of different C3 dicot host plants on the hyphosphere microbiome of Rhizophagus irregularis. Metagenomic analyses showed that interactions of R. irregularis with Medicago truncatula or Nicotiana benthamiana produced functionally distinct hyphosphere microbiomes. The Medicago-associated hyphosphere was notably enriched for fungal species, especially Aspergillus and Fusarium, revealing previously overlooked multikingdom interactions in the hyphosphere. The microbial community showed an enhanced capacity to mobilize organic phosphorus and ability to process complex carbohydrates, with Streptomyces identified as key co-drivers. By contrast, the Nicotiana-associated microbiome preferentially utilized simple carbohydrates and inorganic phosphorus sources.

Our findings demonstrate that host plant identity exerts a strong influence on multikingdom interactions in the AM hyphosphere and shapes microbial traits linked to nutrient cycling, supporting an integrated multikingdom network. Plant-specific hyphosphere microbiomes may therefore contribute to adaptive nutrient foraging and plant growth promotion in soil ecosystems.

Key words: Arbuscular mycorrhiza; host-dependent; hyphosphere; Medicago truncatula; metagenome; microbiome; Nicotiana benthamiana; Rhizophagus irregularis

Coordinated responses of root exudates and the rhizosphere microbiome during amelioration of acidic soils

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Plant–microbiome interactions offer a framework for understanding crop responses to acidity and aluminum stress, yet how root exudates, microbial functions and host responses change together during soil amelioration remains unclear. We established a field experiment in a rice–wheat rotation in Fuyang, Hangzhou, China, with 4 modified composite amendments and an unamended control. Plant phenotyping and soil chemical analyses were integrated with untargeted root exudate metabolomics and rhizosphere metagenomics in selected treatments, alongside measurements of crop transporter-related gene expression. Amendment application altered rhizosphere community composition and increased microbial diversity, with treatment-dependent abundance shifts in genera includingBradyrhizobium,MesorhizobiumandAnaeromyxobacter. Changes in the relative abundance of functional genes associated with amino acid metabolism, carbon metabolism and the tricarboxylic acid cycle indicated altered microbial metabolic potential. Concurrently, root exudate profiles shifted, with increased abundances of selected phenolic acids, flavonoids and organic acids, accompanied by upregulation of crop genes involved in nitrogen and phosphorus transport and aluminum detoxification. These biological responses coincided with increased soil pH, 85–93% reductions in exchangeable and soluble aluminum, a 34–56% increase in hydroxyl-bound aluminum, increased crop yield and a 70–85% reduction in grain aluminum concentration. Together, the findings suggest coordinated changes in soil chemistry, root exudates, the rhizosphere microbiome and host nutrient and detoxification responses, consistent with a potential contribution of plant–microbiome interactions to alleviating acidity and aluminum stress. This study provides field evidence for plant–microbiome responses during acidic soil amelioration and informs efforts to promote crop health through rhizosphere management.

Blitz Talk 6 (I): Plant resistance and crop molecular design 植物抗性与作物分子设计

CDK8 coordinates jasmonate-dependent immune responses to sulfur nutrient status

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Plants trigger immune responses by synthesizing defensive specialized metabolites built from carbon, nitrogen, and sulfur. Shunting resources toward such defensive metabolism often comes at the cost of vegetative development, leading to growth-defense tradeoffs. Nevertheless, how plants coordinate growth and immunity when nutrients fluctuate in the environment remains poorly understood. Jasmonate is a central plant hormone that activates resource-intensive defensive pathways while suppressing plant growth. In this work, a genetic suppressor screening was applied to uncover CYCLIN-DEPENDENT KINASE 8 (CDK8) as a regulator that amplifies jasmonate-driven growth repression and yield loss in Arabidopsis thaliana. Combined transcriptome and metabolome assays further show that CDK8 is required for jasmonate-dependent immune activation, especially for generating sulfur-based defensive compounds. When sulfur is abundant, CDK8 strengthens jasmonate signal transduction and boosts the production of sulfur-containing defensive molecules, which is unrelated to the intrinsic sulfur stoichiometry of these metabolites. Notably, low sulfur availability drastically reduces the levels of sulfur-rich defensin peptides and glucosinolates. Meanwhile, plants produce more camalexin, a defensive metabolite that demands less sulfur input. Collectively, these results indicate CDK8 governs the reallocation of defensive resources according to sulfur status, supporting a model where plants maintain immune capacity under nutrient limitation by preferentially deploying defense chemicals with high sulfur-use efficiency.

Key words: CDK8; Jasmonate; Growth-defense tradeoffs; Sulfur

From Biomass to Biostimulant: Synthetic Biology-Driven Xylooligosacharide Production for Crop Growth and Stress Resistance

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Excessive chemical fertilizer application has caused widespread soil acidification, compaction and salinization worldwide. In the meantime, massive crop straw resources remain underutilized, resulting in resource waste and environmental pollution. Xylooligosaccharides (XOS), a novel plant biostimulant derived from lignocellulosic biomass, can trigger plant damage-associated molecular pattern (DAMP) responses to regulate plant growth and stress tolerance. In this study, we adopted synthetic biology strategies to rationally design thermostable xylanase to increase both its activity and thermostability. By optimization and assembly of the expression elements, such as different promoters, molecular chaperones, signal peptides and high-density fermentation, we obtained Pichia pastoris chassis cells with an ultra-high xylanase activity of 46,000 U/mL, which is the highest level reported to date. Using this enzyme, we established a zero-pollution straw enzymatic hydrolysis process, which can produce 93–212 kg XOS from one ton of different straw materials. Field trials demonstrated that foliar spray and drip irrigation of XOS significantly increased the yields of many crops, such as vegetable, rice and corn by 20%–50%, and markedly enhanced cold resistance. Physiological tests confirmed that XOS improved photosynthetic efficiency, regulated endogenous hormones and stress-related enzyme activities, and ameliorated saline-alkali soil properties. Our results demonstrate high valorization of agricultural straw and develop a low-cost, eco-friendly XOS biostimulant, providing a promising solution for green agriculture.

Keywords
Xylooligosaccharides (XOS), biostimulant, synthetic biology, plant signal transduction, straw valorization

Mechanisms of cellooligomer recognition by plant IGP receptors

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Pathogen invasion releases cell wall-derived cellooligomers as damage-associated molecular patterns (DAMPs), but the perception mechanism is unclear. Previous studies have established IMPAIRED IN GLYCAN PERCEPTION 1 (IGP1) as a receptor required for cellooligomer perception. Here, we demonstrate that IGP1 specifically recognizes cellooligomers with a degree of polymerization (DP) ≥ 3. Structural analyses reveal a preformed ligand-binding pocket formed by its leucine-rich repeat (LRR) and malectin domains, with cellotriose as the minimal binding unit. A WYW motif in the LRR domain constitutes the primary binding site, while a potential redox-sensitive CC motif in malectin negatively regulates ligand interaction. Phylogenetic studies highlight deep conservation of the WYW-CC motifs in higher plants. IGP homologs in soybean and wheat enhance resistance to fungal and oomycete pathogens, and exogenous application of cellooligomer broadly boosts disease resistance. This work defines a conserved plant mechanism for sensing cell wall damage and further supports the potential of these molecules as broad-spectrum immune activators with promising agricultural applications.

This article has been accepted recently in Cell Research.

Integrating kmer GWAS and Recombinant Inbred Lines for Gene Cloning in Barley

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A major challenge for gene cloning in cereals lies in their large and complex genomes, which limit the efficiency of traditional map-based approaches. Conventional strategies rely on biparental segregating populations, which provide controlled genetic backgrounds but are limited by the number of recombination events. In contrast, genome-wide association studies (GWAS) use dense markers but require large and diverse natural panels that are time-consuming and labor-intensive to construct. Moreover, both strategies mainly depend on genetic markers that capture SNPs and indels, leaving many presence/absence variations (PAVs) undetected. Here, we show that recombinant inbred lines (RILs), traditionally used for linkage mapping, provide an effective alternative mapping panel when combined with kmer GWAS, which directly captures sequence-level PAVs between the parents. We developed and resequenced 177 barley RILs from the cross Barke × SusBgtSC and phenotyped the population against five fungal pathogens (wheat stem rust, wheat stripe rust, barley leaf rust, barley stripe rust and barley powdery mildew). We compared kmer GWAS with SNP-based GWAS across multiple traits. kmer GWAS recovered all loci detected by SNP-based GWAS, including the cloned Rph3 barley leaf rust resistance locus on chromosome 7H, and additionally detected PAV-associated signals that were absent in SNP-based GWAS. RNA-seq analysis further prioritized candidates within the associated intervals. These results show that kmer GWAS captures a broader range of parental variation than SNP-based GWAS in a RIL population. It bridges linkage mapping and association mapping and provides a practical pipeline for gene cloning in cereals.

Key words: kmer GWAS; Recombinant Inbred Lines (RILs); Gene cloning; Barley;

Effectoromics uncovers a non-canonical TN-TNL receptor module contributing to Phytophthora capsici resistance in pepper

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Pepper (Capsicum spp.) production is severely threatened by blight disease caused by Phytophthora capsici. Although the landrace CM334 is widely used as a resistance source, the underlying resistance genes remain largely unknown. Here, we developed REPAIR (Receptor-Effector PAIRing), a high-throughput transient co-expression platform, and screened 357 P. capsici RxLR effectors against 425 pepper NLRs in Nicotiana benthamiana. This screen identified CaTN180, a membrane-localized TIR-NB protein that contributes to recognition of the effector PC160 and resistance to P. capsici. The CaTN180 locus harbors three tandemly arrayed TIR-NBS paralogs, TN179-TN181, and one full-length TIR-NB-LRR gene, TNL176, all encoding proteins with C-terminal transmembrane helices. Among these, all the TN proteins, but not TNL176, respond to PC160 and trigger EDS1- and NRG1-dependent hypersensitive cell death. TNL176 associates with TN180 and is required for TN-mediated immune activation, revealing a cooperative signaling architecture between LRR-deficient TNs and a full-length TNL partner. Synteny analysis further shows that this TN-TNL locus is conserved across Capsicum but diversified among accessions, with copy-number variation and absence of the transmembrane helix in the susceptible cultivars. Together, these findings uncover a non-canonical immune module in which a membrane-associated TN cooperates with a TNL partner for pathogen recognition and signaling, providing new insights into TNL evolution and a scalable framework for resistance gene discovery in plants.

Keywords: Plant immunity, Effectoromics, Effector-triggered immunity, TIR-NLR, Truncated NLR, Phytophthora capsici

Rational design of promoter editing confers multipathogen resistance in rice*

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In crops, fine-tuned gene expression rather than alterations in the protein-coding region of a gene is responsible for the optimal performance conferred by many elite alleles in crops. Lesion mimic mutants (LMMs), a type of plant mutants with hyperactivated immune responses, often show enhanced resistance but with yield penalties. To fine-tune the expression level of LMM genes using promoter editing is of considerable interest in crop disease control. We here demonstrate the power of predictive promoter editing in optimizing expression of the rice LMM gene RBL1, encoding a CDP-DAG synthase in phospholipid metabolism, by breaking immunity-growth trade-offs. Through bioinformatic analyses of open chromatin accessibility, we identified key cis-regulatory regions in the RBL1 promoter. Guided by these predictions, we efficiently assessed the regulatory role of different cis-regulatory regions in the rice protoplast system and then generated multiple promoter-edited rice lines with varied RBL1 expression levels. Notably, Pro1, an edited line with a 71.0% reduction in gene expression and altered levels of multiple phospholipids, showed broad-spectrum resistance to rice blast without compromising yield in field trials. Similarly, we generated a phenotype-copied PRO1 allele for enhanced disease resistance in another rice cultivar. Our study has generated an edited promoter of RBL1 that confers multipathogen resistance with no yield penalty. Our study demonstrates a framework for predictable promoter engineering in balancing agronomic traits, especially through optimizing LMMs for crop improvement.

Keywords: Rice (Oryza sativa), promoter editing, broad-spectrum disease resistance, fine-tuning gene expression, immunity-growth trade-off

Engineered phage holin for enhanced multipathogen resistance in plants

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Rice is one of the most important cereal crops in the word. Rice diseases are constrained to the yield stability and the food security. Breeding and cultivation of resistance varieties is an efficient method to prevent pathogen infection and avoid loss of cultivar resistance. Many autoactive nucleotide binding and leucine rich repeat immune receptors (NLRs) as a Ca2+ channel forming a plasma membrane pore can trigger immune response and thus confer disease resistance against pathogens. However, immune activation often results in the growth penalties to varying extents. Whether existing a pore-forming membrane protein with appropriate size enable to maintain stable yield and diminished immune response is elusive. Previous studies have demonstrated that bacteriophages enable to specially infect bacterial, parasitize host cytoplasm to replicate, form lethal pores by holins in the plasma membrane, and ultimately attack peptidoglycan by endolysins. Here, a holin named HCR54 (HOLIN COFERRING RESISTANCE 54) was found cell death effects on the leaves of Nicotiana benthamiana, causing increased H2O2 content, enhanced PR levels, lon leakage, Ca2+ signal activation, located in the plasma membrane and endoplasmic reticulum, and forming multimerization, suggesting HCR54 triggered immune response and Ca2+ channel., In addition, HCR54 can confer disease resistance against Pseudomonas syringae pv. tomato DC3000 ΔHopQ1:Luc and Phytophthora capsici after transient expression and stable expression, respectively. Besides, HCR54 keep a good seed germination and confer normal growth phenotype to seedlings and adult plants of N. benthamiana. Furthermore, after stable transformation of HCR54 into rice, HCR54 with no fitness cost confers broad-spectrum disease resistance to Magnaporthe oryzae, Xanthomonas oryzae pv. oryzae (Xoo). These results show HCR54 can offer fresh insight into editing of endogenous R genes for rice disease resistance and stable crop yield.

Keywords: phage holins, broad-spectrum resistance, rice blast, bacterial blight, stable crop yield

Exploration of disease-resistance genes from Roegneria ciliaris for wheat improvement

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Powdery mildew and stripe rust are two devastating fungal diseases that seriously threaten wheat production. Mining resistance genes from wheat wild relatives is an effective approach to breed cultivars with durable disease resistance. Roegneria ciliaris (2n = 4x = 28, ScScYcYc) is a tertiary wild relative of wheat. In this study, a set of wheat-R. ciliaris disomic addition lines were evaluated for disease resistance. The powdery mildew resistance locus tentatively designated PmRc1 was located on chromosome 1Sc short arm bin 1ScS-8. Two compensating translocations T1ScS·1BL and T1ScS-1AS·1AL were generated; T1ScS-1AS·1AL exhibited favorable agronomic traits and breeding prospect. The stripe rust resistance locus tentatively designated YrRc1, conferring high resistance to race CYR34, was mapped to the distal bin of 2ScL. The translocation T4BS·4BL-2ScL was transferred into three elite wheat cultivars by marker-assisted selection, and the progenies displayed enhanced stripe rust resistance without obvious agronomic penalties. Our work demonstrates that R. ciliaris carries valuable resistance gene resources and provides promising translocation materials for wheat disease resistance breeding.
Key words: Roegneria ciliaris; powdery mildew; stripe rust; alien translocation; wheat breeding

Genetically engineered plant endophytes broaden effector-triggered immunity

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Plants utilize nucleotide-binding leucine-rich repeat (NLR) receptors to detect pathogen effectors and initiate a potent immune response called effector-triggered immunity (ETI). However, this defense relies on the presence of recognizable effectors in pathogens, which is often unpredictable during natural infections. To address this, we engineer plant endophytes, termed Sentinels, to heterologously express effectors that are recognized by the host's corresponding NLR. Using an OxyR regulatory circuit, effector expression is activated by reactive oxygen species—a common signal during pathogen infection. This circuit enables ETI activation against pathogens without recognizable effectors. Colonization by the sentinel bacterium slightly alters microbial abundance but maintains overall microbiota diversity and normal plant growth. We demonstrate the strategy's versatility by testing distinct effector-NLR recognition pairs in various plants against a range of pathogens. This strategy exploits the microbiota-host-pathogen interaction network to rapidly engineer a spectrum-expanded ETI, complementing synthetic microbial consortia for plant defense.

Key words: Plant defense; effector-triggered immunity (ETI); nucleotide-binding leucine-rich repeat (NLR) receptor; microbial engineering; genetically modified microbe; microbiota; disease resistance

Epigenetic regulation of TaFIP phytocytokine clusters confers basal resistance via receptor kinase TaFIPR in wheat

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In both plant and animal innate immunity, rapid biosynthesis of cytokinelike peptides is essential for activating immune responses through plasma membrane receptor kinases. Peptide homeostasis is tightly controlled at multiple regulatory levels to avoid adverse effects caused by excessive phytocytokine production. However, the mechanisms underlying the rapid transition between transcriptional activation and repression of phytocytokine genes in response to immune stimuli remain largely unknown. Here we identify previously uncharacterized phytocytokine
clusters in wheat, termed TaFIPs, which are rapidly induced by Fusarium infection. TaFIP genes are epigenetically regulated by H3K4me3 and H3K27me3 bivalent histone marks enabling rapid transcriptional shifts. Functionally, the leucine-rich repeat receptor kinase TaFIPR directly recognizes and binds TaFIPs to activate multiple immune responses, thereby enhancing wheat resistance to Fusarium infection. Our findings reveal an epigenetic mechanism that fine-tunes phytocytokine-mediated immunity in wheat.

Key words: Wheat; Fusarium graminearum; TaFIP phytocytokines; Epigenetic regulation; TaFIPR receptor kinase; Basal resistance

The rice blast effector MoIug12 inhibits OsPAL family enzymes to disrupt the homeostasis of secondary metabolism and promote infection in rice

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Magnaporthe oryzae is one of the most devastating fungal pathogens of rice. To establish successful infection, pathogens secrete effectors that manipulate host immunity by targeting immune signaling and defense-related pathways. However, whether fungal effectors directly target key enzymes involved in plant secondary metabolism and inhibit their catalytic activity remains poorly understood. Here, we investigated the conserved cytoplasmic effector MoIug12. Deletion of MoIug12 reduced fungal virulence, whereas its overexpression in rice increased susceptibility to blast disease. Transcriptomic analysis followed by Gene Ontology (GO) enrichment analysis identified the secondary metabolic process as an enriched biological process, prompting us to investigate the role of phenylpropanoid metabolism in MoIug12-mediated immune suppression. We identified OsPAL6, a key enzyme in phenylpropanoid metabolism, as an interacting partner of MoIug12. Genetic analysis showed that OsPAL6-overexpressing rice lines exhibited enhanced resistance to rice blast, whereas OsPAL6 knockout mutants were more susceptible. Consistently, OsPAL6 knockout mutants showed reduced accumulation of downstream flavonoid metabolites. Biochemical characterization identified R173 and S201 as critical residues associated with OsPAL6 enzymatic activity. In vitro assays using purified proteins demonstrated that MoIug12 directly inhibits OsPAL6 enzymatic activity. Microscale thermophoresis (MST) analysis further revealed that MoIug12 increased the dissociation constant for OsPAL6–substrate binding approximately tenfold, indicating a substantial reduction in apparent substrate affinity. Moreover, yeast two-hybrid and luciferase complementation assays demonstrated interactions between MoIug12 and multiple OsPAL family members, and in vitro enzymatic assays confirmed that MoIug12 inhibits the catalytic activities of multiple OsPAL proteins. Collectively, these findings reveal that MoIug12 suppresses rice defense-associated secondary metabolism by inhibiting OsPAL enzymatic activity and reducing substrate affinity, thereby contributing to M. oryzae infection. This study uncovers a mechanism of effector-mediated suppression of plant defense through direct inhibition of host metabolic enzymes and highlights the MoIug12–OsPAL interaction as a potential target for rice blast resistance breeding.
Key words: Rice blast fungus; effector; rice; host-pathogen interaction; pathogenic mechanism; phenylpropanoid metabolism

Molecular Mechanism of the Receptor-like Kinase OsRLK40 and E3 Ubiquitin Ligase OsRIEL in Coordinated Regulation of Rice Immunity

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Receptor-like kinases (RLKs) generally function as positive regulators of plant immunity, whereas a small subset of RLKs negatively regulate plant immunity. Nevertheless, the molecular mechanisms underlying this negative immune regulation remain poorly understood. In this study, we found that the rice RLK mutant osrlk40 exhibits significantly enhanced resistance to both bacterial and fungal pathogens compared with wild-type plants. Consistently, the osrlk40 mutant displays markedly elevated pathogen-associated molecular pattern (PAMP)-triggered oxidative burst and increased expression of pathogenesis-related (PR) genes, demonstrating that OsRLK40 negatively modulates rice immune responses without affecting grain yield. OsRIEL (Oryza sativa OsRLK40 Interacting E3 Ligase), identified via yeast two-hybrid screening, physically interacts with OsRLK40 and acts as its phosphorylation substrate. OsRLK40-mediated phosphorylation substantially increases the protein stability of OsRIEL. The osriel knockout mutants exhibited enhanced disease resistance, whereas OsRIEL-overexpressing rice plants showed compromised resistance against Magnaporthe oryzae (M. oryzae) and Xanthomonas oryzae pv. oryzae (Xoo). Biochemical assays further revealed that OsRIEL, a RING-type E3 ubiquitin ligase, interacts with and ubiquitinates OsRLCK57. This ubiquitination modification targets OsRLCK57 for degradation via the 26S proteasome pathway, thereby attenuating rice immune responses. Collectively, this study uncovers an uncharacterized negative regulatory cascade of rice immunity, the OsRLK40-OsRIEL-OsRLCK57 module. It highlights the versatile roles of RLKs in broad-spectrum disease resistance and provides valuable theoretical insights and candidate genes for breeding rice germplasm with durable broad-spectrum resistance and no yield penalty.
Key words: Oryza sativa L.; Pathogen-associated molecular pattern-triggered immunity (PTI); Receptor-like kinase; E3 ubiquitin ligase

Blitz Talk 6 (II): Plant resistance and crop molecular design 植物抗性与作物分子设计

Functional Characterization of ACP1 in Suppressing Rice Immunity

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Pathogenic bacteria interfere with immune signaling pathways by secreting effector proteins into host cells. This study aims to explore the biological functions of the effector protein ACP1 (acid phosphatase) from the rice disease fungus (Ustilaginoidea virens). We successfully constructed the plant expression vector of this gene and transiently expressed it in Nicotiana benthamiana through Agrobacterium-mediated methods. Subcellular localization analysis revealed that the protein was simultaneously localized in the nucleus and cell membrane. Western blot verified the successful expression of this protein in tobacco leaves. Functional studies have shown that this effector protein can significantly inhibit the reactive oxygen species burst and cell death induced by INF1 (immunosuppressive experiments). Further co-immunoprecipitation (Co-IP) experiments revealed its interaction with the host target protein HSF. In addition, through the transient expression system of Nicotiana benthamiana mediated by Mycobacterium, combined with the quantitative detection technology of relative electrolyte permeability, it was found that the relative electrolyte permeability of the ACP1-GFP and INF1-GFP co-treatment group decreased. By using aniline blue specific staining combined with laser confocal microscopy observation and ultraviolet spectrophotometry quantitative detection technology, it was found that when ACP1 and INF1 were co-expressed, the callose deposition was lower than that in the INF1 alone treatment group. In conclusion, our research reveals that ACP1 can significantly inhibit INF1-induced cell necrosis and may act as a negative regulatory factor in the PTI immune response of plants.

Keywords: ACP1; plant immunity; Instantaneous expression; bilocalization; immunoprecipitation

Enhancing crop resistance to Phytophthora capsici using resistance genes from Non-Host plants Nicotiana tabacum

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Nonhost resistance (NHR) represents a durable phenomenon where plants resist non-adapted pathogens. While NHR holds significant potential for engineering crops with broad-spectrum and durable resistance, its underlying molecular mechanism remains incompletely understood. The oomycete pathogen Phytophthora capsici is non-adapted to Nicotiana tabacum [Nt] but is virulent on the closely related Nicotiana benthamiana [Nb]. We demonstrated that both species encode the NLR Swordholder (Swh), which recognizes P. capsici AvrNt effector proteins. NtSwh restores NHR in ntswh mutants, whereas NbSwh confers only partial resistance. Mechanistically, NtSwh exhibits weaker autoinhibition mediated by its N-terminal extension domains, enabling rapid immune signaling that prevent pathogen effector-mediated suppression; NbSwh's stronger autoinhibition delays activation, permitting effector suppression. Heterologous expression of NtSwh in tomato enhances field resistance to P. capsici without yield penalty. These findings not only identify a promising resistance gene but also indicate that the kinetics of NLR activation can play a key role in determining NHR, providing insights into strategies for breeding durable crop immunity.

Unveiling Indigenous Beauveria bassiana–Bactrocera zonata Interactions: From Natural Occurrence to Biocontrol Efficacy

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The guava fruit fly, Bactrocera zonata (Saunders, 1842), is a major pest of guava and other soft fruits, with significant economic and quarantine implications. In the past decades, the demand for using environment-friendly pest management has increased the interest in the use of indigenous entomopathogenic fungi (EPF) as biological control agents. This study investigated the biocontrol potential of native Beauveria bassiana against different developmental stages of B. zonata and explored its potential for bioproduct development. EPF were isolated from guava orchard soils using an insect-baiting technique and identified through morphological and molecular characterization. The selected B. bassiana isolates were tested against larvae, pupae and adults at four concentrations (1×10^5–1×10^8 conidia/mL). A concentration- and isolate dependent response was observed with the highest levels of mortality at 1×10^8 conidia/mL, with 94% mortality in larvae, 92% mortality in pupae, and 93% mortality in adults. There was a wide range of virulence among isolates with adult mortality ranging from 55–95%. The isolates FMB-BB-FF04 and FMB-BB-FF02 had the highest pathogenicity and were chosen for further testing. Their efficacy was subsequently investigated through horizontal transmission, greenhouse, and field-cage assays, followed by bio-formulation development. The key novelty of this work is the stepwise exploitation of indigenous fungus–insect interactions, starting from the discovery of natural EPF, molecular characterization, testing of virulence, horizontal transmission, semi-field testing and bioproduct development. The identification of highly virulent locally adapted B. bassiana strains offers a promising basis for the microbial development of alternative insecticide products. This study demonstrates the potential of indigenous EPF for sustainable and environment friendly management of B. zonata in guava agroecosystems and provide a framework to develop locally adapted fungal biocontrol products.

GFP fusion enhances oligomerization of a computationally designed calcium channel to activate plant immunity

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Computationally designed ion channels offer opportunities to engineer plant immunity, but their activity in plant cells may require further optimization. Here, we show that GFP fusion enables the computationally designed Ca^{2+}-permeable channel CalC6 to induce cell death in Nicotiana benthamiana. Whereas untagged CalC6 did not trigger detectable cell death, fusion to GFP, but not His, FLAG or GST tags, enhanced oligomerization and generated the immune-active derivative CSC1. CSC1 assembled into plasma membrane-associated hexamers and exhibited channel activity in Xenopus oocytes. Its cell death-inducing activity was suppressed by Ca^{2+}-channel blockers, supporting a contribution of Ca^{2+} flux. These findings identify GFP fusion as a key modification enabling CalC6-mediated immune activation. Future work will determine how GFP promotes oligomerization and which differences between plant and animal cellular environments may prevent efficient assembly of untagged CalC6 in plants. In addition, transgenic N. benthamiana expressing CSC1 showed enhanced resistance to Phytophthora capsici and reduced turnip mosaic virus accumulation. Pathogen-inducible expression in potato enhanced resistance to Phytophthora infestans, but not Botrytis cinerea, without detectable growth penalties under the conditions tested. Together, these results support GFP-assisted engineering of synthetic channels for plant immunity and crop disease resistance.

The Schizanthus pinnatus genome and EvoNLR uncover the evolutionary history of the Solanaceae NLR repertoire

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Nucleotide-binding leucine-rich repeat receptors (NLRs) underpin disease resistance across Solanaceae, one of the world's most important crop families, yet their evolutionary history remains poorly understood because structurally complex NLR loci are difficult to annotate and genomic resources from early-diverging lineages are lacking. Here we present a chromosome-scale genome assembly of Schizanthus pinnatus, the earliest-diverging extant Solanaceae lineage, revealing that it diverged shortly after the Solanaceae ancestral whole-genome triplication. To reconstruct the evolutionary history of Solanaceae NLR genes, we developed EvoNLR, an evolution-guided annotation tool that substantially improves the identification and structural annotation of complex NLR genes in Solanaceae. Applying EvoNLR to more than 100 Solanaceae genomes generated a comprehensive NLRome resource, and comparative genomic analyses uncovered distinct evolutionary trajectories of different NLR gene families and revealed ancestral NRC lineages. Functional characterization of these ancestral NRCs demonstrated evolutionary diversification of helper NLR activities. Together, the Schizanthus genome and EvoNLR provide an evolutionary framework for understanding the origin and diversification of Solanaceae NLR repertoire, while facilitating genome-wide discovery of functional resistance genes.

Genome-wide Identification of the FER Gene Family and Elucidation of the GhFER-mediated Salt Tolerance Mechanism

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Zhengzhou University

Soil salinity and drought, as major abiotic stresses, severely constrain crop growth, development, and yield formation. To cope with salt stress, plants have evolved multilayered defense mechanisms, including ion homeostasis regulation, accumulation of osmoregulatory substances, and reactive oxygen species (ROS) scavenging. Receptor kinases, acting as the "gateway" for plants to perceive and transmit environmental signals, play a central role in stress signal transduction. FERONIA (FER) receptor-like kinases, as important members of the CrRLK1L subfamily, have been shown to participate in plant hormone signal integration, cell wall integrity surveillance, and biotic and abiotic stress responses. However, the functional mechanisms of key FER gene family members in cotton under salt stress and their upstream transcriptional regulatory networks remain largely unclear. Therefore, by integrating bioinformatics analyses, gene family identification, and promoter analysis with molecular and genetic approaches—including qRT-PCR expression profiling, Virus-Induced Gene Silencing (VIGS) functional validation, physiological measurements, Yeast One-Hybrid (Y1H) assays, and subcellular localization—we systematically dissected the salt stress response and transcriptional regulatory network of the cotton FER gene family and its key member, GhFER3.The main findings of this study are as follows: (1) Six GhFER genes were identified in the Gossypium hirsutum genome. Phylogenetic analysis classified them into multiple clades along with homologs from Arabidopsis and rice, suggesting potential functional divergence. Cis-acting element analysis of the promoters revealed abundant hormone-responsive elements (e.g., ABA, auxin, gibberellin) and stress-responsive motifs (e.g., MBS, TC-rich repeats), indicating the involvement of this gene family in hormone crosstalk and abiotic stress responses. (2) qRT-PCR analysis showed that the expression of GhFER1 and GhFER2 did not change significantly under salt stress, whereas GhFER3, GhFER4, GhFER5, and GhFER6 were significantly downregulated. (3) Following VIGS-mediated silencing of GhFER3, GhFER4, GhFER5, and GhFER6, no obvious phenotypic differences were observed between the silenced plants and the empty vector control (TRV:00) under normal conditions. However, under 200 mM NaCl treatment, the silenced plants exhibited more severe wilting than the control. The GhFER3-silenced lines showed the most pronounced phenotype, accompanied by decreased activities of antioxidant enzymes (CAT, POD, SOD) and proline content, as well as increased malondialdehyde (MDA) content. These results demonstrate that GhFER3, GhFER4, GhFER5, and GhFER6 positively regulate salt tolerance in cotton, with GhFER3 playing a central role. (4) Transcriptomic analysis of the GhFER3-silenced lines revealed that GhFER3 silencing significantly perturbed multiple pathways, including photosynthesis (particularly the light-harvesting antenna complex), hormone transport, and osmotic regulation. (5) Subcellular localization confirmed that GhMYB is localized in the nucleus, and Y1H assays demonstrated that GhMYB can directly bind to the GhFER3 promoter. In summary, the GhMYB–GhFER3 module plays an important regulatory role in the cotton salt stress response.Conclusion:This study reveals that GhFER3, GhFER4, GhFER5, and GhFER6 positively regulate salt tolerance in Gossypium hirsutum, with GhFER3 being the most critical. GhFER3 silencing affects pathways related to photosynthesis, hormone transport, and osmotic regulation. Furthermore, we identified for the first time the crucial role of the GhMYB–GhFER transcriptional regulatory module in cotton's response to salt stress, providing new candidate gene resources for the genetic improvement of salt tolerance in cotton.

An angiosperm tandem kinase atlas reveals lineage-specific diversification of tandem kinases and their association with plant immune receptor evolution

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Tandem kinase proteins (TKPs), characterized by two or more kinase domains within a single protein, have emerged as important components of plant immunity, particularly in grasses. However, their diversity, structural evolution, and evolutionary relationships with canonical immune receptors PRRs and NLRs remain poorly understood. Here, we established the PlantTKPdb (xxx) that includes 17,133 TKPs from 651 angiosperm genomes, revealing extensive variation in their abundance, kinase/pseudokinase architectures, and integrated-domain composition across plant lineages. Most TKPs contained two kinase domains, and nearly two-thirds incorporated non-kinase domains, many of which are associated with plant defense and immune receptor functions. We further identified unusual TKP architectures containing NLR-associated signaling domains or complete NLR modules, revealing structural links between TKPs and NLRs. Comparative genomic analyses showed positive associations between TKP abundance and several major immune receptor families, including NLRs and immune-related PRRs, with these relationships being substantially stronger in monocots than in dicots. Notably, DLSV/DLSV tandem kinases, which encompass most previously characterized immune-related TKPs, showed a pronounced monocot-biased association with CNL repertoires. Together, our findings reveal extensive lineage-specific diversification of plant TKPs and establish an evolutionary connection between tandem kinase and canonical immune receptor repertoires, providing a framework and genomic resource for investigating tandem kinase functions in plant immunity and disease resistance.

Key words: tandem kinase protein (TKP); NLR

Identification and Functional Characterization of a Novel Early-Maturity Factor

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The duration of the growth period is a crucial consideration in cotton breeding. Early-maturing cotton varieties can not only increase the multiple cropping index and alleviate the conflict between cotton and grain production for arable land, but also mitigate the adverse effects of seasonal climates—such as late spring cold spells and early autumn frosts—on cotton yield and quality. Flowering time is one of the most important determinants of early or late maturity in cotton. Manipulating flowering time to achieve early maturity represents a current research hotspot and challenge in the field of cotton molecular breeding. In our recent study published in Nature Communications, we employed multi-omics analyses to identify members of the GhCOL9 gene family as potential key regulators of early maturity in cotton, and confirmed that this family has been conserved throughout the domestication process. Subsequently, by integrating molecular experiments—including CRISPR/Cas9, Yeast One-Hybrid (Y1H), Electrophoretic Mobility Shift Assay (EMSA), and transcriptional activation assays—with genetic approaches, we conducted a comprehensive functional analysis of flowering and investigated the molecular mechanisms of the cotton GhCOL9 family genes (GhCOL9A, GhCOL9B, GhCOL9C, GhCOL9D, and GhCOL9E), aiming to provide theoretical and technical support for the development of early-maturing cotton varieties.The main findings of this study are as follows: (1) Through high-quality genome analyses of representative species from all nine subfamilies of Malvaceae, we finely reconstructed the complex evolutionary history of this family and identified the GhCOL9 gene family, which was completely retained throughout the evolutionary process following polyploidization in Malvaceae. (2) qRT-PCR analysis was performed to detect the expression levels of GhCOL9A, GhCOL9B, GhCOL9C, GhCOL9D, and GhCOL9E in Gossypium hirsutum at different time points. The results revealed obvious diurnal rhythmic variations in the expression of these genes. (3) All five GhCOL9 genes (GhCOL9A, GhCOL9B, GhCOL9C, GhCOL9D, and GhCOL9E) were cloned from G. hirsutum. Transgenic materials, including overexpression lines and mutants, were generated via Agrobacterium-mediated transformation. These plants were grown at the experimental field of the Cotton Research Institute in Anyang, Henan (15 plants per row, 80 cm between rows) and in the large greenhouse of the Institute of Cotton Research, Chinese Academy of Agricultural Sciences (ICR, CAAS). Phenotypic analysis confirmed that overexpression of these genes significantly delayed flowering, whereas the mutants markedly accelerated flowering in cotton. (4) Molecular experiments demonstrated that GhCOL9 significantly suppresses the expression of key flowering genes, including GhFT, GhFPF1, and GhELF4. Furthermore, Y1H and EMSA assays identified GhFT, GhFPF1, and GhELF4 as direct downstream targets of GhCOL9D and GhCOL9E. Transcriptional activation assays confirmed that GhCOL9D and GhCOL9E directly activate the expression of these target genes.Conclusion: The GhCOL9A, GhCOL9B, GhCOL9C, GhCOL9D, and GhCOL9E genes are involved in photoperiod-mediated flowering in cotton by directly binding to the promoters of genes such as GhFT, GhFPF1, and GhELF4 and suppressing their expression. These findings not only enrich the molecular regulatory theory of flowering in cotton but also provide guidance and germplasm resources for the precise breeding of new early-maturing and high-quality cotton varieties.

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Poster

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Poster · Session 1: Inter-organismal Perception and Recognition 生物间感知与识别

P1-01

Integrative Multi-omics Reveals Spatial Metabolic Reprogramming in Cap and Stipe during Pileus Opening in Stropharia rugosoannulata

Hongxiao Yu, Shiyi Liu, Fucheng Lin, Xiaohong Liu
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Pileus opening in Stropharia rugosoannulata involves distinct developmental programs in cap and stipe, yet the underlying metabolic regulatory networks remain unclear. Here, we performed integrative transcriptomic and metabolomic analyses on cap and stipe tissues at mature and opened stages. We show that the cap activates oxidative phosphorylation, fatty acid metabolism, and N-glycan biosynthesis to support energy production and cell wall remodeling for spore dispersal, whereas the stipe induces autophagy, glutathione metabolism, and sphingolipid signaling to execute programmed senescence and nutrient recycling. Metabolomic profiling confirmed these tissue-specific strategies, with galactonate and phloroglucinol accumulating in the opened cap, and dipeptide in the opened stipe. Correlation and regression analyses further identified cytochrome P450 monooxygenase (SR_4BG01369) as a core hub driving coumarin-derived secondary metabolic reprogramming. Collectively, our multi-omics data demonstrate that pileus opening is a spatially coordinated systemic metabolic remodeling, orchestrated by organ-specific functional divergence and robust transcriptional-metabolic coordination.

P1-02

Integrative Multi-Omics Analysis Reveals Transcriptional and Metabolic Reprogramming During the Development of Stropharia rugosoannulata

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Zhejiang university

The development of the economically important edible mushroom Stropharia rugosoannulata relies on the efficient bioconversion of lignocellulosic substrates, yet the underlying molecular dynamics remain largely unexplored. Here, we employed an integrated transcriptomic and metabolomic approach to map the molecular trajectories across its complete developmental lifecycle systematically. Global omics profiling revealed that the transition from vegetative hyphae to primordia triggers the most massive transcriptional reprogramming. Furthermore, temporal clustering (Fuzzy c-means) of multi-omics data identified tightly coordinated, stage-specific expression patterns. Notably, late-stage spiking clusters (Clusters 3 and 4) were significantly enriched in fundamental carbohydrate pathways, accompanied by a dramatic accumulation of key sugar phosphates and osmoprotectants. These findings provide a comprehensive molecular framework for understanding the stage-specific metabolic control driving the programmed maturation of S. rugosoannulata.

P1-05

MoCox6 is a regulator of mitophagy and a druggable target in Magnaporthe oryzae

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Mitophagy is a selective autophagic process that maintains cellular homeostasis by degrading damaged mitochondria and is a promising antifungal target. However, few inner mitochondrial membrane (IMM) regulators of mitophagy are known. Here we identify cytochrome c oxidase subunit 6 (MoCox6) as an IMM regulator in Magnaporthe oryzae that binds MoAtg5 and MoAtg14 following outer mitochondrial membrane rupture to mediate mitophagy. MoSirt5 regulates this process by desuccinylating MoCox6 at K144. Structural analysis revealed that residue D95 at the MoSirt5-MoCox6 interface mediates the dual role of MoCox6 in mitophagy and mitochondrial metabolic competence. Deletion of the COX6 gene significantly reduced vegetative growth and virulence in both M. oryzae and Alternaria alternata. Through high-throughput screening, we identified a small-molecule compound, Pan-RAS-IN-1, which targets MoCox6 to inhibit mitophagy, thereby suppressing M. oryzae virulence. Pan-RAS-IN-1 exhibits broad-spectrum antifungal activity, and its application to rice plants significantly suppressed rice blast incidence.

P1-06

Characterization of Extracellular Vesicles from Phytophthora palmivora and Their Potential Roles in Plant–Pathogen Interactions

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Mahidol University

Extracellular vesicles (EVs) play a crucial role in carrying biomolecular cargo and intercellular communication in eukaryotic organisms. Current studies have demonstrated their involvement in plant-pathogen interactions, pathogen development, and pathogenesis. However, their roles in oomycete–plant interactions remain poorly understood. Phytophthora palmivora is a destructive oomycete pathogen with a broad host range, causing significant diseases in economically important crops such as oil palm, rubber tree, cacao, and durian. This study aims to investigate whether plant host influences EV characteristics and biological activity. P. palmivora was cultured in half-strength potato dextrose broth (PDB) supplemented with either durian (host) or rice (non-host) leaves. EV-enriched fractions were isolated from durian- and rice leaf-supplemented P. palmivora culture filtrates using differential centrifugation and ultracentrifugation. The isolated fractions were characterized using transmission electron microscopy (TEM) and nanoparticle tracking analysis (NTA) to assess vesicle morphology, size distribution, and particle concentration. TEM revealed cup-shaped like vesicular structures in all isolated fractions, while NTA confirmed particle size distributions of 180.8 ± 10.5 nm and 118.0 ± 25.5 nm for durian- and rice leaf-supplemented P. palmivora culture filtrates, respectively. To investigate biological activity, EV-enriched fractions were mixed with P. palmivora zoospores prior to inoculation on detached durian leaves, and pathogen development was assessed after 5 days. Treatment with the EV-enriched fraction from durian leaf-supplemented cultures enhanced P. palmivora development on detached leaves compared to rice leaf-supplemented filtrates and controls, suggesting that P. palmivora-derived EVs may influence pathogen development during host interactions, particularly those from host-induced cultures. These findings provide initial evidence for the biological role of extracellular vesicles in P. palmivora–host interactions and establish a foundation for further investigation of their molecular cargo and mechanisms of action.

Poster · Session 2: Effector Biology in Immunity and Pathogenesis 效应因子免疫调控与致病机理

P2-01

Extracellular vesicle-mediated suppression of rice immunity by the fungal effector MoZmp1

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Rice blast, a globally destructive rice disease prevalent widely in China, leads to severe yield losses. Magnaporthe oryzae penetrates rice via appressoria and delivers effectors through BIC and EIHM to suppress host PTI/ETI immunity and establish susceptibility. Zinc metalloproteases serve as crucial virulence determinants in phytopathogenic fungi by degrading host immune proteins. Although plants deploy proteases for defense, the functions and regulatory pathways of zinc metalloproteases in M. oryzae are largely unknown.

P2-03

A pathogen lncRNA secreted into rice sequesters a host miRNA for virulence

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Plants and animals respond to pathogens through pattern recognition receptor and Nod-like receptor proteins1. Pathogens commonly use protein effectors to suppress host immunity for successful infection2. However, the existence of non-protein effector classes remains comparatively understudied. Here we report an RNA–RNA recognition mechanism governing pathogen–host interaction, mediated by a regulatory RNA-encoding DNA sequence that separately generates two complementary regulatory RNAs. Specifically, a long non-coding RNA transcribed from this DNA region in the fungal pathogen Magnaporthe oryzae translocates into host rice cells and sequesters a complementary microRNA (miRNA), derived from a distinct host DNA region, thereby subverting host immunity. In turn, this rice-derived miRNA promotes disease resistance by repressing the expression of PKR1, a gene that encodes a negative regulator of host immunity. Sequestration of the host miRNA by the fungal long non-coding RNA releases PKR1 expression to facilitate fungal infection. We discovered that this regulatory RNA-encoding DNA sequence is probably widely present across diverse life species, mediating interactions between pathogens and their plant hosts. Collectively, our findings provide an approach for effective disease control using miRNAs derived from this important DNA region.

P2-04

Experimental validation of bioinformatically predicted effector candidates from Cochliobolus miyabeanus during rice infection

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Cochliobolus miyabeanus, the causal agent of rice brown spot disease, secretes effector proteins that facilitate host colonization by manipulating plant immune responses. However, the identification of fungal effectors remains challenging because many effectors exhibit high sequence diversity and lack recognizable functional domains. In this study, we combined bioinformatics prediction, structural modeling, and preliminary experimental analyses to identify and characterize novel effector candidates from C. miyabeanus.Starting from a predicted proteome of 12,007 proteins, candidate secreted proteins were screened using SignalP 6.0, TMHMM 2.0, and EffectorP 3.0. Proteins with annotated homologs in PHI-base and UniProtKB/Swiss-Prot were excluded to prioritize previously uncharacterized candidates. Candidate ranking was further refined based on protein length, cysteine content, sequence conservation, predicted structural features, and potential interactions with rice immune receptors. Four representative candidates were selected for experimental evaluation.Gene-specific primers were designed to amplify the coding sequences of the selected candidates from C. miyabeanus cDNA. PCR products corresponding to the expected sizes were obtained, and the candidate genes were subsequently subjected to cloning and sequence verification for downstream functional analyses. Among these candidates, one particularly small secreted peptide was prioritized because of its conservation among fungal homologs and its predicted structural interactions with the rice chitin receptor components OsLYP6 and OsCERK1.To examine whether this candidate directly activates plant immune signaling, a synthetic peptide corresponding to its predicted mature region was tested using a reactive oxygen species assay. Under the tested conditions, the peptide did not reproducibly induce an early oxidative burst, although delayed increases in luminescence were observed. These preliminary results suggest that this candidate may not function as a directly recognized elicitor and may instead have a more complex role in modulating plant immune responses.Together, this study establishes an integrated workflow connecting computational effector prediction with experimental candidate evaluation. Further analyses, including infection-stage expression profiling, transient expression, subcellular localization, immune suppression assays, protein–protein interaction validation, and fungal gene disruption, will clarify the contributions of these candidates to C. miyabeanus virulence and rice immunity.

P2-06

The non-classical effector MoSprT of *Magnaporthe oryzae* suppresses rice immunity and modulates pathogenicity

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Rice blast, caused by *Magnaporthe oryzae*, is the most destructive fungal disease of rice and seriously threatens global food security. Phytopathogenic fungi secrete effector proteins to evade or suppress host defenses and promote infection and colonization. Existing studies have largely focused on classical effectors bearing N-terminal signal peptides, whereas non-classical effectors without signal peptides remain poorly characterized, especially regarding their nuclear-targeting mechanisms during the rice–*M. oryzae* interaction. Using SignalP 5.0 and SecretomeP, we predicted nonclassically secreted proteins in *M. oryzae* and identified multiple proteomic proteins lacking signal peptides, which were further classified by their nuclear localization signals (NLS). Among them, MoSprT is markedly upregulated during rice infection, and subcellular localization assays show MoSprT accumulates in the nuclei of rice and tobacco cells. Both MoSprT knockout mutants and NLS-deletion mutants display substantially attenuated virulence and lower fungal hyphal biomass relative to the wild-type Guy11. In transgenic rice heterologously expressing MoSprT, chitin-triggered reactive oxygen species (ROS) bursts are suppressed, infection-induced transcription of pathogenesis-related (PR) genes is downregulated, and rice disease resistance is compromised. Collectively, MoSprT functions as a nuclear-targeted non-classical secretory effector and key virulence factor of *M. oryzae*. MoSprT promotes *M. oryzae* infection by suppressing host immune responses, and the NLS domain of MoSprT is critical for protein localization and fungal pathogenicity. We will further dissect the potential secretion pathway of MoSprT and its detailed mechanism underlying rice immune suppression.

Keywords: *Magnaporthe oryzae*; non-classical effector; nuclear; plant immunity

P2-08

A Ralstonia solanacearum effector associates with plant NLRs and induces non-canonical NLR-dependent disease resistance in specific Solanaceae species

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Ralstonia solanacearum is a soil-borne bacterial pathogen that causes devastating diseases worldwide. Upon plant colonization, R. solanacearum injects more than 70 different effector proteins into plant cells to promote infection, ultimately causing plant wilting and death. However, such effectors can also be perceived by intracellular immune receptors in certain plant species, leading to disease resistance. In this work, we characterized a novel R. solanacearum effector, Rip38, which is specifically conserved in certain phylotypes within the R. solanacearum species complex. Inducible expression of Rip38 in Arabidopsis enhanced plant susceptibility to R. solanacearum. Conversely, R. solanacearum mutant strain lacking Rip38 showed stronger virulence upon inoculation in tomato, a natural host for R. solanacearum, suggesting that Rip38 may negatively contribute to R. solanacearum virulence in specific Solanaceae species. To identify the host factors underlying this resistance, we found thatRip38 associates with NLR required for cell death (NRCs) without triggering macroscopic hypersensitive response (HR) or canonical defense genes. In Nicotiana benthamiana, Rip38 restricts R. solanacearum proliferation and suppresses PAMP-triggered immunity (PTI) in an NbNRCs-dependent manner; both growth and PTI suppression were lost in Nbnrc2/3/4 triple mutants and rescued by a single NRC. Resistance, but not PTI suppression, required NRC Ca^{2+} channel activity, as shown by $LaCl_3$ treatment and Ca^{2+} channel impairing NRCs mutants. Rip38 also amplified infection-triggered Ca^{2+} influx. Together, Rip38 reprograms the NRC network into 2 opposing outputs—immune suppression and non-canonical NLR-dependent bacterial restriction.

P2-12

Natural expression variation reveals virulence diversity in wheat powdery mildew

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Obligate biotrophic fungi cannot be cultured independently of living hosts and remain difficult to transform, constraining functional genomics. Here we treated naturally occurring expression differences as molecular perturbations by profiling in planta transcriptomes of 130 genetically diverse isolates of the wheat powdery mildew fungus Blumeria graminis f. sp. tritici on one susceptible wheat genotype. Benefiting from the 34% expansion of the gene-model set enabled by population-derived data, we constructed an atlas that connects pathogen genotype, virulence on diverse host accessions, and the transcriptional state of the infected host via in-planta expression. Integration with virulence phenotypes recovered six characterized avirulence or suppressor genes and prioritized 12 recurrent genomic intervals. Cross-species analysis further connected 1,808 fungal genes to 1,902 wheat genes to dissect Bgt-mediated host regulation. Recovery and recognition assays for AvrWTK4 validated phenotype-linked expression mapping, whereas expression of BgtE-20107 suppressed a wheat defence module. Natural expression variation therefore provides a scalable functional-genomics framework for probing virulence polymorphism in obligate biotrophs.

Key words: Obligate biotroph; population transcriptomics; eQTL; natural expression variation; virulence; host–pathogen interaction

Poster · Session 3: Immune Signaling Networks and Activation 免疫信号网络与激活机制

P3-01

Phosphorylation of OsCBP601 by OsCPK5 and OsCPK13 enhances rice blast resistance

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Calcium (Ca^{2+}) is a pivotal second messenger in plant cells. As Ca^{2+} decoders, calcium-dependent protein kinases (CPKs) sense diverse calcium oscillations and transduce these signals via direct phosphorylation of their substrates, thereby regulating plant growth, development, reproduction, and responses to both biotic and abiotic stresses. Our previous studies demonstrated that OsCPK5 and OsCPK13 positively regulate rice blast resistance, yet they may be guarded by the intracellular coiled-coil (CC)-type nucleotide-binding (NB) leucine-rich repeat (LRR) domain receptor (NLR) protein OsCPK5/13-ASSOCIATING RESISTANCE PROTEIN 1 (OsCARP1), as the oscpk5 oscpk13 double mutant displays enhanced blast resistance in an OsCARP1-dependent manner. However, the precise mechanisms by which OsCPK5 and OsCPK13 modulate rice disease resistance remain largely unknown. Here, we show that both OsCPK5 and OsCPK13 interact with and phosphorylate a calmodulin-binding protein 60 (CBP60) family member, which we provisionally designated OsCBP601. This phosphorylation increases the transcriptional activity of OsCBP601 and potentiates OsCBP601-mediated rice blast resistance, suggesting that OsCPK5 and OsCPK13 may orchestrate immune transcriptional reprogramming through phosphorylation of OsCBP601 during blast fungus infection. Furthermore, prime editing of the phosphorylation site in OsCBP601 enhances blast resistance without compromising normal plant growth, thus providing a promising gene-editing target for breeding disease-resistant rice varieties.

P3-06

PHR1 and PHL1 regulate root colonization by pathogenic and beneficial fungi under phosphate deficiency

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Nara Institute of Science and Technology

Phosphorus is an essential macronutrient for plant growth and development. Plants adapt to inorganic phosphate (Pi) deficiency through the phosphate starvation response (PSR), which is also involved in interactions with root-associated microbes. However, how the master transcription regulators of PSR, PHR1 and homologue PHL1, regulate colonization by pathogenic and beneficial fungi remains unclear. Here, we investigated Arabidopsis thaliana interactions with the pathogenic root fungus Colletotrichum incanum (Ci) and the mutualist C. tofieldiae (Ct). Loss of PHR1/PHL1 increased Ci biomass specifically under Pi deficiency. Genetic and transcriptional analyses supported a role for DDE2-dependent jasmonate (JA) pathway in this resistance. Ci infection was also accompanied by reduced PHR1 protein abundance. PHR1/PHL1 and DDE2 further contributed to limiting Ct colonization and to the timely onset of Ct-mediated plant growth promotion (PGP) under low Pi. Thus, PHR1/PHL1 regulate colonization by both pathogenic and beneficial root fungi during Pi deficiency, contributing to JA-mediated Ci resistance and to the timely establishment of Ct-mediated PGP.

Poster · Session 4: Symbiosis and Microbiome 共生与菌群

P4-01

A wild rice-derived endophytic fungus Magnaporthiopsis oryzae shapes rice microbiome to enhance growth and disease resistance

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Wild rice harbors diverse beneficial endophytic fungi with untapped potential for sustainable agriculture. Here, we identified a novel endophytic fungus, Magnaporthiopsis oryzae MB-9-27, isolated from Oryza granulata, which enhanced rice yield by 12.66% and reduced blast disease by 45.52% under field conditions. MB-9-27 reshaped rice-associated microbiota by enriching beneficial microbial taxa and promoting a simplified interaction network. Functional screening of recruited endophytic fungi identified Mortierella sp. Mortierella sp. as a key beneficial fungus that enhanced rice growth and disease resistance. Metabolomic analysis revealed that Mortierella sp. -associated metabolites, including Indole-3-glycerol phosphate, contributed to plant growth promotion. These findings highlight wild rice-derived endophytes as valuable resources for microbiome-based strategies to improve sustainable rice production.

P4-02

Symbiotic bracovirus of a parasite modulates host ecdysis process

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Parasitoids modulate host development for the survival of their offspring, but the mechanisms underlying this phenomenon remain largely unknown. Here, we found that the endoparasitoid Cotesia vestalis disrupted the larval-larval ecdysis in its host Plutella xylostella by the 20-hydroxyecdysone (20E) synthesis pathway. After parasitization by C. vestalis, the 20E peak of host larvae disappeared before the onset of ecdysis and the expression of ecdysone synthesis genes was sig nificantly downregulated. We further found that a Cotesia vestalis bracovirus (CvBV) gene CvBV_28 − 5 was transiently high-level expressed prior to the host's 20E peak, enabling the precise suppression of this critical developmental signal. Consistently, the knockdown of CvBV_28 − 5 affected the expression of 20E response transcription factors in the cuticle and several ecdysis-related genes. Furthermore, we found that CvBV_28−5 bound directly to the Raf, a MAP3K member of the MAPK pathwaythat functions as a critical regulator of ecdysone synthesis genes in hosts. Collectively, our results provide the first evidence that parasitoids modulate host ecdysis by affecting MAPK-20E signaling during a defined devel opmental window and provide novel insights into the mechanism of parasitoid regulation of host development.

P4-03

Synergistic host-parasitoid antimicrobial peptide interactions ensure the survival of parasitized hosts

Sijie Zhang
Zhejiang University

Host–parasite interaction is always involved in the immune response. It is well known that parasitic wasps introduce various factors to suppress host immune responses, thereby facilitating parasitoid progeny development while compromising host survival. However, little is understood about how parasitic wasps, especially endoparasitoids, survive pathogen threats when they develop in hosts. Antimicrobial peptides (AMPs) are important immune effectors that are active against pathogens and essential for the survival of organisms. Here, we investigated the immune response of the host Plutella xylostella and its endoparasitic wasp Cotesia vestalis during the process of parasitism, focusing on AMPs. We found that the suppression of parasitism on host AMPs diminished as the process went on and selected AMPs responded differently in parasitized hosts following bacterial challenges. We then demonstrated that the host-expressed gloverin was extremely sensitive to pathogen infection after parasitism and was vital for host survival. Furthermore, we found that parasitoid teratocytes expressed antimicrobial peptides, especially hymenopteacin, which were significantly upregulated following bacterial infection. With a broad antimicrobial spectrum, the presence of hymenopteacin in the hemolymph of parasitized hosts significantly decreased bacterial load and increased the survival of the parasitized host. Our study provides a systematic perspective on host-parasitoid immune interaction by highlighting the significance of AMPs.

P4-04

Harnessing a Fungal Endophyte as a Sustainable Alkaloid Factory: Gold Nanoparticles-Elicited ROS Homeostasis for Enhanced Dendrobine

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Dendrobine, a neuroprotective and anticancer sesquiterpenic alkaloid traditionally sourced from endangeredDendrobiumorchids, faces sustainability challenges due to slow plant growth and destructive harvesting. This study establishes a sustainable production platform using the endophytic fungusTrichoderma longibrachiatumMD33 through gold nanoparticle-mediated metabolic engineering. Chemically synthesized gold nanoparticles (CH-AuNPs; ~50 nm) were functionalized with biosynthetic precursors (L-phenylalanine, L-tyrosine, and tyramine) at loading efficiencies of 79-85% and applied to fungal cultures at concentrations ranging from 0.5-20.0 mg/L. Multi-omics analyses (transcriptomics, proteomics, and metabolomics) revealed a dose-dependent hormetic response, with optimal dendrobine production (290.90 ± 14.55 ng/mL; 63.7% increase over control; $p < 0.001$) achieved at 10.0 mg/L CH-AuNPs. Mechanistically, low-to-moderate oxidative stress induced by CH-AuNPs activated redox-sensitive transcription factors (Yap1/Atf1) and stress-responsive MAPK pathways, upregulating key biosynthetic genes including HMGR (+2.5-fold), FPPS (+3.1-fold), TPS (+3.0-fold), CYP71D1 (+4.0-fold), and COMT (+3.3-fold). Metabolomic profiling confirmed nitrogen rechanneling through ornithine decarboxylase pathway activation, with depletion of glutamate/ornithine pools and accumulation of putrescine. Integrated omics enabled construction of a hybrid terpenoid-alkaloid biosynthetic pathway where mevalonate-derived sesquiterpenoid scaffolds merge with ornithine-derived piperidine moieties. Concentrations exceeding 20.0 mg/L induced cytotoxic oxidative damage and metabolic shutdown. This study establishes CH-AuNPs as precision dual-function tools (carriers and elicitors) for fungal metabolic engineering, providing a scalable, eco-friendly platform for dendrobine production while resolving the long-elusive biosynthetic pathway—a critical advancement for pharmaceutical applications in neuroprotection and oncology.

P4-05

Multi-omics of Cinnabar Induced Dendrobine Synthesis in Trichoderma longibrachiatum

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Zunyi Medical University

Dendrobine, a neuroprotective sesquiterpene alkaloid traditionally sourced from endangeredDendrobiumorchids, faces critical supply challenges. The endophytic fungusTrichoderma longibrachiatumMD33 offers a sustainable alternative, yet its biosynthetic pathway and regulatory mechanisms remain uncharacterized. This study investigated cinnabar (HgS)-induced hormesis to elucidate stress-mediated metabolic reprogramming of dendrobine biosynthesis through integrated transcriptomic and metabolomic profiling. Subtoxic HgS concentrations (1.0–4.0 μg/L) triggered controlled reactive oxygen species (ROS) signaling, upregulating the mevalonate pathway (HMGR, FPPS), terpenoid synthases (TPS1/2), and cytochrome P450 monooxygenases, thereby enhancing dendrobine yields by 24% at 4.0 μg/L. Multi-omics integration revealed biphasic regulation: low HgS activated stress-responsive transcription factors (bZIP, Zn-Cys6) and MAP kinase cascades, redirecting resources toward dendrobine production, while cytotoxic doses (6.0 μg/L) induced 73.9% growth inhibition and 73.2% reduction in dendrobine synthesis, correlating with metabolic collapse via disrupted CoA biosynthesis and antioxidant depletion. This study establishes 4.0 μg/L as the hormetic threshold for maximizing dendrobine yields and delineates a fungal-specific biosynthetic architecture combining bacterial-like terpene cyclases with specialized fungal CYP450s—a divergence from the plant-centric model. These findings provide a roadmap for metabolic engineering ofT. longibrachiatumMD33, emphasizing ROS-mediated pathway optimization for sustainable alkaloid production with future CRISPR-based editing of regulatory nodes.

P4-08

Plant host identity shapes the taxonomic composition and functional potential of the Rhizophagus irregularis hyphosphere microbiome

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Most land plants rely on symbioses with arbuscular mycorrhizal (AM) fungi for mineral nutrient acquisition. AM fungal hyphae can recruit diverse microbiomes that contribute to nutrient mobilization, however the role of host plants in shaping hyphosphere community assembly and function remains poorly understood.We used a series of in vitro and mesocosm experiments to investigate the effect of different C3 dicot host plants on the hyphosphere microbiome of Rhizophagus irregularis.Metagenomic analyses showed that interactions of R. irregularis with Medicago truncatula or Nicotiana benthamiana produced functionally distinct hyphosphere microbiomes. The Medicago-associated hyphosphere was notably enriched for fungal species, especially Aspergillus and Fusarium, revealing previously overlooked multikingdom interactions in the hyphosphere. The microbial community showed an enhanced capacity to mobilize organic phosphorus and ability to process complex carbohydrates, with Streptomyces identified as key co-drivers. By contrast, the Nicotiana-associated microbiome preferentially utilized simple carbohydrates and inorganic phosphorus sources.Our findings demonstrate that host plant identity exerts a strong influence on multikingdom interactions in the AM hyphosphere and shapes microbial traits linked to nutrient cycling, supporting an integrated multikingdom network. Plant-specific hyphosphere microbiomes may therefore contribute to adaptive nutrient foraging and plant growth promotion in soil ecosystems.

P4-10

Coordinated responses of root exudates and the rhizosphere microbiome during amelioration of acidic soils

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Plant–microbiome interactions offer a framework for understanding crop responses to acidity and aluminum stress, yet how root exudates, microbial functions and host responses change together during soil amelioration remains unclear. We established a field experiment in a rice–wheat rotation in Fuyang, Hangzhou, China, with 4 modified composite amendments and an unamended control. Plant phenotyping and soil chemical analyses were integrated with untargeted root exudate metabolomics and rhizosphere metagenomics in selected treatments, alongside measurements of crop transporter-related gene expression. Amendment application altered rhizosphere community composition and increased microbial diversity, with treatment-dependent abundance shifts in genera includingBradyrhizobium,MesorhizobiumandAnaeromyxobacter. Changes in the relative abundance of functional genes associated with amino acid metabolism, carbon metabolism and the tricarboxylic acid cycle indicated altered microbial metabolic potential. Concurrently, root exudate profiles shifted, with increased abundances of selected phenolic acids, flavonoids and organic acids, accompanied by upregulation of crop genes involved in nitrogen and phosphorus transport and aluminum detoxification. These biological responses coincided with increased soil pH, 85–93% reductions in exchangeable and soluble aluminum, a 34–56% increase in hydroxyl-bound aluminum, increased crop yield and a 70–85% reduction in grain aluminum concentration. Together, the findings suggest coordinated changes in soil chemistry, root exudates, the rhizosphere microbiome and host nutrient and detoxification responses, consistent with a potential contribution of plant–microbiome interactions to alleviating acidity and aluminum stress. This study provides field evidence for plant–microbiome responses during acidic soil amelioration and informs efforts to promote crop health through rhizosphere management.

Poster · Session 5: Plant Resistance and Molecular Design Breeding 植物抗性与分子设计育种

P5-03

Vacuolar Sugar Transporter SpERD Coordinates Acylsugar Biosynthesis in Glandular Trichome of Wild Tomato

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Glandular trichomes of wild tomato sustain high-intensity acylsugar biosynthesis via leaf-derived carbon and metabolic recycling. In this study, we identify the tonoplast transporter SpERD as a critical regulator of trichome sugar homeostasis. SpERD is enriched in glandular cells, localizes to the vacuolar membrane, and functions as a fructose transporter. SpERD6L1 knockout mutants exhibit increased biomass, sugar accumulation, and elevated acylsugar levels. We demonstrate that fructose acts as a metabolic signal activating acylsugar biosynthetic genes. We propose that SpERD buffers vacuolar fructose loads to modulate carbon flux, its loss triggers a compensatory feedback loop that enhances specialized metabolism and reallocates whole-plant carbon resources. This study links vacuolar transport to carbon recycling in wild tomato.

P5-06

Organ-specific wound-induced activation of phytaspase-1 in Nicotiana benthamiana seedlings

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Rationale: Subtilases play an important role in plant responses to various stresses, one such subtilase is phytaspase, a serine aspartate-specific. Recently, we demonstrated that mechanical wounding of Nicotiana benthamiana mature leaves causes an increase in phytaspase-1 activity and gene expression, and here we aimed to investigate how wounding of different plant organs, such as roots and cotyledons, affects this protease.Methods: As model plants we used N. benthamiana seedlings grown under sterile conditions on low-sugar medium. One-week-old seedlings were subjected to root or cotyledon excision. Five days post-wounding, phytaspase activity was measured fluorometrically in shoot and root tissue extracts and compared to the corresponding tissues in the unwounded control group.Results: Root wounding systemically upregulated phytaspase activity in both root and shoot tissues relative to the control. In contrast, cotyledon injury induced a localized response, with increased phytaspase activity restricted to the shoot. These differential patterns complement previous findings on wound-induced phytaspase activation [1], suggesting that the systemic nature of the stress response depends on the specific organ being injured.Conclusions: Our data indicate that wounding of aerial organs and roots triggers distinct patterns of phytaspase-1 induction. This suggests organ-specific regulatory mechanisms during early ontogeny of N. benthamiana, highlighting a complex systemic signaling network. The study was funded by the Shenzhen Municipal Government and Shenzhen MSU-BIT University.References: 1 Abdullina MA et al. (2025) Int J Mol Sci 26, 7170.

P5-09

A dsRNA-binding protein VdDRB-mediated RNAi regulation in Verticillium dahliae

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RNA interference (RNAi) is a conserved eukaryotic mechanism governing gene silencing through small-RNA (sRNA)-guided transcriptional or post-transcriptional regulation. While core effectors such as Dicer (DCL) and Argonaute (AGO) are well characterized, the auxiliary factors that modulate sRNA processing and loading remain largely elusive, particularly in non-model fungal systems. Here, we established a visual RNAi screening system in Verticillium dahliae V592 and identified a protein VdDRB, which contains a double-stranded RNA binding domain, as an essential component of the silencing system. Deletion ofVdDRBabrogated RNAi-mediated silencing, restoring target proteins to pre-silencing levels. Genetic and biochemical analyses revealed that VdDRB functions in the same pathway as VdAGO2 and VdDCL2, because individual knockouts of eitherVdAGO2orVdDCL2recapitulate the silencing defects observed in Vddrb deletion mutant. Notably, small RNA deep sequencing demonstrated that loss ofVdDRBresults in a global and drastic reduction in sRNA abundance, with a pronounced bias toward the depletion of antisense sRNAs across both exonic and intronic regions. Mechanistically, VdDRB preferentially binds longer dsRNA substrates (50 bp) but not the processed 21-nt products, and its physical interaction with VdDCL2 is RNA-dependent, as evidenced by RNase-sensitive co-immunoprecipitation. Although VdDRB associated with VdAGO2 in vivo, the interaction is likely indirect or transient, as it cannot be detected by standard pull-down assays. Collectively, our findings position VdDRB as a critical upstream scaffold that recognizes longer dsRNA precursors and channels them into the VdDCL2-dependent sRNA biogenesis pathway, thereby ensuring efficient sRNA production and subsequent loading. This work uncovers a previously unrecognized layer of regulatory complexity in the fungal RNAi pathway and provides a conserved mechanistic framework for understanding how dsRNA-binding proteins in sRNA-mediated immunity and epigenetic control.

P5-10

Mechanistic Insights into Melatonin Enable the Development of Broad-Spectrum Antifungal Agents

Renjian Li
Huazhong Agricultural university

Melatonin is a low-cost natural small indole molecule with versatile biological functions. However, melatonin's fungicidal potential has not been fully exploited, and the mechanism remains elusive. Here, we report that melatonin broadly inhibited 13 plant pathogens. In the rice blast fungal pathogen Magnaporthe oryzae, melatonin inhibited fungal growth, formation of infection-specific structures named appressoria, and plant infection, reducing disease severity. Melatonin entered fungal cells efficiently and colocalized with the critical mitogen-activated protein kinase named Mps1, suppressing phosphorylation of Mps1. Melatonin's affinity for Mps1 via two hydrogen bonds was demonstrated using surface plasmon resonance and chemical modifications. To fully exploit melatonin's potential as an antifungal agent, a series of melatonin derivatives targeting the mitogen-activated protein kinase (Mps1) protein of fungal pathogens were synthesized based on the properties of melatonin, among which the trifluoromethyl-substituted derivative Mt-23 exhibited antifungal activity against seven plant pathogenic fungi, and effectively reduced the severity of crop diseases, including rice blast, Fusarium head blight of wheat and gray mold of tomato. Through further systematic modifications, developing an eco-friendly melatonin derivative of commercial value for agricultural applications appears promising.

P5-11

GFP fusion enhances oligomerization of a computationally designed calcium channel to activate plant immunity

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Huazhong Agricultural University

Computationally designed ion channels offer opportunities to engineer plant immunity, but their activity in plant cells may require further optimization. Here, we show that GFP fusion enables the computationally designed Ca^{2+}-permeable channel CalC6 to induce cell death in Nicotiana benthamiana. Whereas untagged CalC6 did not trigger detectable cell death, fusion to GFP, but not His, FLAG or GST tags, enhanced oligomerization and generated the immune-active derivative CSC1. CSC1 assembled into plasma membrane-associated hexamers and exhibited channel activity in Xenopus oocytes. Its cell death-inducing activity was suppressed by Ca^{2+}-channel blockers, supporting a contribution of Ca^{2+} flux. These findings identify GFP fusion as a key modification enabling CalC6-mediated immune activation. Future work will determine how GFP promotes oligomerization and which differences between plant and animal cellular environments may prevent efficient assembly of untagged CalC6 in plants. In addition, transgenic N. benthamiana expressing CSC1 showed enhanced resistance to Phytophthora capsici and reduced turnip mosaic virus accumulation. Pathogen-inducible expression in potato enhanced resistance to Phytophthora infestans, but not Botrytis cinerea, without detectable growth penalties under the conditions tested. Together, these results support GFP-assisted engineering of synthetic channels for plant immunity and crop disease resistance.

Poster · Blitz Poster 闪电报告墙报

PB-01

NAT10-mediated ac⁴C RNA acetylation enhances plant immunity

Hang He
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Plants rapidly reprogram gene expression in response to pathogen attack, but how RNA modifications contribute to immune activation remains poorly understood. Here, we identify N4-acetylcytidine (ac4C) as a pathogen-responsive RNA modification that promotes plant immunity. In Arabidopsis, bacterial and fungal pathogens induce NAT10 expression and global ac4C accumulation, while genetic manipulation of NAT10 alters disease resistance and pattern-triggered immune responses. Integrated ac4C RIP-seq, single-base-resolution ac4C profiling and proteomics revealed extensive pathogen-induced remodeling of the ac4C epitranscriptome, with increased ac4C enrichment associated with elevated protein accumulation of immune-related transcripts.

PB-03

Investigating the Role and Underlying Mechanism of Transcription Activator-Like Effectors in Rice Bacterial Blight Resistance Mediated by the XA14 Gene

Liu Mengting
Hubei University

Xanthomonas species colonize many host plants and cause huge losses worldwide. Transcription activator-like effectors (TALEs) are secreted by Xanthomonas and translocated into host cells to manipu-late the expression of target genes, especially by Xanthomonas oryzae pv. oryzae and Xanthomonas oryzae pv. oryzicola, which cause bacterial blight and bacterial leaf streak, respectively, in rice.In this study, we elucidate how transcription activator-like effectors (TALEs) secreted by Xanthomonas oryzae pv. oryzae (Xoo) are recognized by the NLS protein encoded by the rice disease resistance gene Xa14, thereby triggering disease resistance. Concurrently, we demonstrate that Xoo strains harboring mutated TALEs, designated as iTALEs (interfering TALEs), can disrupt this recognition and render the plants susceptible. Furthermore, we have discovered that two distinct iTALE variants, which have evolved and are classified as Type-A and Type-B iTALEs, exhibit differential suppressive capacities. Notably, the Type-B iTALE lacks the ability to suppress XA14-mediated immunity. Deciphering the molecular interactions between TALEs, iTALEs, and the XA14 protein will provide pivotal insights for advancing crop improvement and developing novel strategies for immune prophylaxis.

PB-04

Functional Characterization of MoPcp1 and Identification of Candidate Interacting Proteins in *Magnaporthe oryzae*

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The rice blast fungus *Magnaporthe oryzae* is a major threat to global rice production and food security, yet the molecular mechanisms underlying its pathogenicity remain incompletely understood. The mitochondrial serine protease Pcp1 is involved in mitochondrial protein processing and homeostasis in yeast, but its function in plant pathogenic fungi remains largely unexplored. Here, we identified the *M. oryzae* homolog MoPcp1 and confirmed its mitochondrial localization. Deletion of *MoPCP1* caused pleiotropic defects in vegetative growth, stress tolerance, conidiation, appressorium formation, and pathogenicity, all of which were substantially restored by reintroducing wild-type *MoPCP1* into the Δ*Mopcp1* mutant, indicating that MoPcp1 is required for normal growth, development, and pathogenicity. To investigate the functional basis of MoPcp1, we generated mutants targeting several conserved residues and constructed a series of transmembrane helix deletion mutants based on predicted membrane-spanning regions. Mutations affecting catalytic residues and all nine transmembrane helix deletion mutants failed to complement the defects of the Δ*Mopcp1* mutant, highlighting the importance of catalytic residues and transmembrane regions for MoPcp1 function. Yeast two-hybrid screening identified several candidate MoPcp1-interacting proteins, and co-localization analysis showed that a subset of these candidates co-localized with MoPcp1 in mitochondria, providing potential clues to the molecular network associated with MoPcp1. Together, our findings establish MoPcp1 as an important regulator of *M. oryzae* growth, development, and pathogenicity and provide initial insights into its structural requirements and potential interacting proteins.

Keywords: *Magnaporthe oryzae*; MoPcp1; mitochondrial serine protease; protein interaction; pathogenicity

PB-05

MVA secreted by the root endophyte Falciphora oryzae promotes plant growth

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Endophytic fungi promote plant growth and adaptation by modulating plant physiology and producing bioactive metabolites. Falciphora oryzae is a beneficial root endophyte with plant growth-promoting activity. However, whether F. oryzae produces bioactive metabolites that contribute to its growth-promoting effects remains unclear. We found that F. oryzae significantly increased Arabidopsis biomass, shortened the primary root, and promoted lateral root formation. Using untargeted metabolomics, we identified compound MVA as a metabolite strongly enriched during F. oryzae–Arabidopsis co-culture. Targeted analysis confirmed that F. oryzae produced MVA and that MVA reached its highest abundance in the co-culture system. We further showed that exogenous MVA promoted Arabidopsis growth, with the strongest effect at 50 μM, and increased tomato and pepper biomass. Together, our findings identify MVA as a candidate F. oryzae-derived metabolite with growth-promoting activity across plant species, advancing our understanding of metabolite-mediated endophyte–plant interactions and highlighting its potential for crop growth promotion.

PB-06

Plasma-Activated Water-Mediated Rhizosphere Microbial Communities for the Control of Fusarium Crown Rot and Promotion of Sustainable Wheat Production

Cancan Niu, Yupan Zhu, Ruonan Ma, Zhen Jiao
Zhengzhou University

Plasma-activated water (PAW) irrigation is aneco-friendly technology for the management of soil-borne diseases, but its efficacy against wheat Fusarium crown rot (FCR) remains unclear. In this study, three PAW application strategies, namely therapeutic treatment, inoculant pretreatment, and preventive treatment, were established to systematically evaluate the control efficiency of PAW against wheat FCR. In vitroantifungal assays demonstratedthat PAW-3 (15 min plasma treatment) completely inhibited pathogen spore germination and mycelial growth by inducing oxidative damage and intracellular content leakage. Pot experiments results revealed that therapeutic application of PAW-2 (5 min plasma treatment) reduced the FCR disease index by 38.5%, while enhancing wheat plant height and aboveground biomass by 11% and 19.3%, respectively. In terms of soil physicochemical properties,soil nitrate-N content increased approximately three-fold, tsoil redox potential was significantly elevated, and soil pH remained in a neutral range. Root microbiome sequencing further indicated that PAW-2 application markedly enriched core beneficial microbial taxa(e.g.,Funneliformisand Pseudomonas),reduced the relative abundance of pathogenic microorganisms, and improved the complexity and stability of the rhizosphere microbial network. Partial least squares path modeling (PLS-PM) further indicated that PAW suppressed wheat FCR through three synergistic pathways: direct antifungal activity, regulation of soil nitrogen and redox conditions, and enrichment of beneficial rhizosphere microorganisms. Overall, PAW combines antifungal activity with soil-improving functions and therefore shows potential as a multifunctional soil amendment for green and sustainable wheat production.

PB-09

Both Jasmonic Acid- and Abscisic Acid-Mediated Signalling Pathways Regulate the Ovicidal Defence of Plants Against Phloem-Feeding Insects

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Plants perceive signals associated with herbivore eggs and in response initiate ovicidal defence. However, which phytohormone pathways regulate this defence and which defensive compounds dominate it remains largely unknown. Here, we found that the hatching rate of eggs of white-backed planthopper (WBPH) Sogatella furcifera was significantly lower on a japonica rice variety P81 than an indica rice variety NB44. When infested by WBPH, P81 plants showed higher jasmonic acid (JA) and abscisic acid (ABA) responses than did NB44 plants; moreover, P81 plants produced the ovicidal compound benzyl benzoate and exhibited higher levels of some flavonoids, phenolamides, and volatiles than were found in NB44 plants. Impairing the ABA-signalling pathway, especially the JA-signalling pathway in P81 plants enhanced the survival of WBPH eggs. Decreasing levels of some flavonoids and phenolamides in P81 plants promoted WBPH egg survival. In vitro bioassays revealed that both naringenin and sakuranetin promote the ovicidal effect of benzyl benzoate on WBPH. The results demonstrate that JA- and ABA-signalling pathways jointly regulate the rice ovicidal defence against WBPH, and that benzyl benzoate, as well as some other compounds, such as naringenin and sakuranetin, contribute to the mortality of WBPH eggs.

PB-10

Mannitol dehydrogenases in nematode infection and soybean resistance

Qiannan Liu
Zhejiang University

These findings identify mannitol dehydrogenases as key regulators of SCN motility, chemotaxis, and infection, revealing a new mechanism underlying nematode parasitism.These two genes thus provide a two-pronged strategy for SCN control by impairing nematode parasitism when silenced and enhancing soybean resistance when overexpressed, making them promising targets for gene silencing-based nematode control and resistance breeding.

PB-11

Characterization of X functions in ABA responses and autophagy

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Being sessile organisms, plants need rapid and finely tuned signaling pathways to adapt their growth and survival to immediate and often adverse environmental conditions. The plant hormone abscisic acid (ABA) plays a crucial role in both biotic and abiotic stress responses. ABA regulates turgor pressure in guard cells, leading to stomatal closure to minimize water loss, while ensuring stomata can open to uptake CO2 for photosynthesis, even under stress. However, the detailed mechanism is still unknown. Here, we identified an uncharacterized gene, X, which regulates stomatal closure via an ABA-dependent pathway. Furthermore, multiple lines of evidence proved that X is related to the plant autophagy pathway. X-GFP forms puncta that increase under nutrient starvation, a hallmark of autophagy induction. These puncta co-localize with ATG1a, ATG6, ATG8i, and ATG12. Complementing the imaging data, yeast two-hybrid assays reveal a strong, direct interaction between X and ATG6 and a weaker interaction with ATG8i. And we also found that the puncta structure of X co-localized with FREE1, which participates in autophagosome trafficking and modulates ABA-mediated stomatal responses. Collectively, our findings uncover a novel dual role of Arabidopsis X in integrating ABA-mediated stress responses and autophagic regulation.

PB-16

Natural expression variation reveals virulence diversity in wheat powdery mildew

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Obligate biotrophic fungi cannot be cultured independently of living hosts and remain difficult to transform, constraining functional genomics. Here we treated naturally occurring expression differences as molecular perturbations by profiling in planta transcriptomes of 130 genetically diverse isolates of the wheat powdery mildew fungus Blumeria graminis f. sp. tritici on one susceptible wheat genotype. Benefiting from the 34% expansion of the gene-model set enabled by population-derived data, we constructed an atlas that connects pathogen genotype, virulence on diverse host accessions, and the transcriptional state of the infected host via in-planta expression. Integration with virulence phenotypes recovered six characterized avirulence or suppressor genes and prioritized 12 recurrent genomic intervals. Cross-species analysis further connected 1,808 fungal genes to 1,902 wheat genes to dissect Bgt-mediated host regulation. Recovery and recognition assays for AvrWTK4 validated phenotype-linked expression mapping, whereas expression of BgtE-20107 suppressed a wheat defence module. Natural expression variation therefore provides a scalable functional-genomics framework for probing virulence polymorphism in obligate biotrophs.

Key words: Obligate biotroph; population transcriptomics; eQTL; natural expression variation; virulence; host–pathogen interaction

PB-17

Virulence evolution basis of a major wheat pathogen inform targeted resistance breeding design

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Rapid pathogen evolution undermines crop resistance, yet its genomic basis remains insufficiently connected to breeding strategies. Here, we integrated population genomics, virulence phenotyping, and effector analyses to investigate wheat powdery mildew and guide resistance deployment. We constructed a genome-wide variation map of 1,415 Blumeria graminis isolates spanning ten grass hosts, 29 countries, and 33 years. Phenotyping 105 isolates across 103 wheat accessions identified 11 association peaks. Integration with published resources defined 69 Avr loci and 16 characterized Avr/Svr genes. Analyses of 605 candidate effectors revealed extensive amino acid substitutions and presence–absence variation under strong host selection. Haplotype networks traced contrasting virulence trajectories involving recurrent mutations in AvrPm8 and predominantly gene loss in AvrPm2. Comparative analyses further revealed extensive sequence and structural diversification within conserved effector scaffolds. Introgression analyses identified cross-forma-specialis genetic exchange involving effector haplotypes, supporting a reticulate arms race across host and pathogen communities. We translated these findings into Bgt-Rstack-Designer, which recommends resistance-gene combinations and crossing schemes based on pathogen Avr prevalence. Across six crosses, F_2 screening identified progeny resistant to two complementary challenge isolates, exceeding parental resistance profiles. These findings connect pathogen virulence evolution with targeted breeding and provide a foundation for surveillance-informed resistance deployment, pending broader phenotypic and genetic validation.

Key words: 8th ICBPI; Biotic Plant Interaction; R-Avr Interaction; Pathogen; Population Genetics; Virulence Evolution; Intelligent Wheat Resistance Breeding.

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