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*Meeting Review*

# Molecular Mechanism & Structure—Zooming in on Plant Immunity

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**The first of three International Society for Molecular Plant–Microbe Interactions (IS-MPMI) eSymposia was convened on 12 and 13 July 2021, with the theme "Molecular Mechanism & Structure—Zooming in on Plant Immunity". Hosted by Jian-Min Zhou (Beijing, China) and Jane Parker (Cologne, Germany), the eSymposium centered on "Top 10 Unanswered Questions in MPMI" number five: Does effector-triggered immunity (ETI) potentiate and restore pattern-triggered immunity (PTI)—or is there really a binary distinction between ETI and PTI? Since the previous International Congress of IS-MPMI in 2019, substantial progress has been made in untangling the complex signaling underlying plant immunity, including a greater understanding of the structure and function of key proteins. A clear need emerged for the MPMI community to come together virtually to share new knowledge around plant immunity. Over the course of two synchronous, half days of programming, participants from 32 countries attended two plenary sessions with engaging panel discussions and networked through interactive hours and poster breakout rooms. In this report, we summarize the concerted effort by multiple laboratories to study the molecular mechanisms underlying ETI and PTI, highlighting the essential role of plant resistosomes in the formation of calcium channels during an immune response. We conclude our report by forming new questions about how overlapping signaling mechanisms are controlled.**

*Keywords*: calcium signaling, defense signaling pathways, ETI, IS-MPMI, plant immunity, plant responses to pathogens, PTI, resistance genes, resistosome, Top10MPMI

## STRUCTURAL INSIGHTS INTO NOD-LIKE RECEPTOR ACTIVATION AND SIGNALING

Plants are equipped with nucleotide-binding leucine-rich repeat (LRR) or NOD-like receptor (NLR) proteins that sense the presence of pathogen effector proteins, either directly or indirectly, to elicit immune responses. In the past several years, laboratories from around the world have been working to understand how the complex structures of NLRs, and the downstream signaling components, dictate responses leading to pathogen resistance. NLRs are classified into two general groups based upon their N-terminal structure, which consists of either a coiled-coil domain or a Toll/interleukin-1 receptor (TIR) domain, termed CNLs and TNLs, respectively. Jiji Chai (University of Cologne and Max Planck Institute for Plant Breeding Research, Germany) provided an overview of key NLR structural characteristics that emerged from the resistosome structure of the *Arabidopsis* CNL ZAR1, first reported in 2019 (Wang et al. 2019). More recently, Chai's group found that TNLs also form resistosomes but, unlike the CNLs, they have NADase activity following oligomerization. The *Arabidopsis* TNL, RPP1, directly recognizes the downy mildew effector ATR1 (Krasileva et al. 2010), forming the active RPP1-ATR1 resistosome, an NAD-hydrolyzing holoenzyme (Ma et al. 2020). Based on conservation with the Roq1 resistosome in wild tobacco (Martin et al. 2020), Chai speculates that the effector-induced tetramerization leading to assembly of two asymmetric TIR dimers is likely a conserved mechanism of TNL activation. Jian-Min Zhou (Institute of Genetics and Developmental Biology, Chinese Academy of Sciences, China) also discussed structural characteristics of resistosomes, specifically focusing on constitution of the CNL ZAR1 resistosome pore (Bi et al. 2021). Unlike the NADase activity that results in TNLs, Zhou confirmed that ZAR1 has calcium ion ($Ca^{+2}$) channel activity, dependent on a conserved residue, Glu11, known to be required for CNL-triggered cell death. Zhou showed that the ZAR1 resistosome channel is permeable to cations and, upon activation, triggers channel-dependent $Ca^{+2}$ influx.

Focusing on the events following TNL activation, Jane Parker, (Max Planck Institute for Plant Breeding Research, Germany) addressed the requirement of EDS1 family lipase-like proteins and "helper" NLRs (called RNLs) for TNL-induced plant defense responses. RNLs such as ADR1 and NRG1 are NLRs with an RPW8-like N-terminal domain considered to be critical in TNL signaling. Upon TNL activation, downstream signaling involves two exclusive EDS1 heterodimers with PAD4 or SAG101, creating a cavity that is likely a signaling surface for downstream responses (Bhandari et al. 2019). Specifically, it has been shown that the *Arabidopsis* EDS1-SAG101 heterodimer associates with NRG1-family RNLs to elicit a

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cell death response upon bacterial inoculation in *Nicotiana benthamiana* leaves (Sun et al. 2021). Because the association is TNL activation dependent, it is possible that a product of the NADase activity binds to the EDS1-SAG101 heterodimer which, in turn, recruits NRG1. Alternatively, the TNL NADase activity activates NRG1 to form a stable interaction with the heterodimer. The role of activated NRG1 was addressed by Pierre Jacob (University of North Carolina–Chapel Hill, United States), who characterized structural features and functions of the *Arabidopsis* RNL NRG1.1 (Jacob et al. 2021). Crystallization of NRG1.1's signaling domain revealed its similarity to the mixed lineage kinase-like (MLKL) animal and plant proteins which make pores with cation channel activity (Cai et al. 2014; Mahdi et al. 2020). Like MLKL, active NRG1.1 is enriched at the plasma membrane. Using HeLa cells, Jacob showed that an autoactive NRG1.1 variant oligomerizes, forms pores within the plasma membrane, and drives an influx of cations, likely favoring $Ca^{+2}$ in planta; it is unclear whether activated NRG1.1 in complex with EDS1-SAG101 oligomerizes and forms similar channels. Collectively, these findings set up a model in which RNLs may possess a conserved mechanism for promoting $Ca^{+2}$ permeability, potentially by forming a resistosome, underscoring a role for calcium signaling in CNL- and TNL-triggered immunity.

## RECEPTOR SENSING AND INTERPLAY BETWEEN PATTERN-TRIGGERED IMMUNITY AND EFFECTOR-TRIGGERED IMMUNITY

The second plenary session of the symposium covered broadly how the two host receptor systems of effector-triggered immunity (ETI) and pattern-triggered immunity (PTI) are functionally connected to counteract pathogen attacks. NLRs are one of two primary classes of immune receptors for sensing pathogens, the other being pattern recognition receptors (PRRs), which are associated with PTI instead of ETI. Defense responses induced by PTI include callose deposition, $Ca^{+2}$ influx, reactive oxygen species (ROS) burst, and expression of defense genes that are induced in a stronger manner in ETI (Bigeard et al. 2015). $Ca^{+2}$ influx is one of the earliest cellular PTI events, occurring within 30 s of microbial pattern perception, and can regulate the closure of stomata conferring stomatal immunity to the plant. Cyril Zipfel (University of Zurich, Switzerland) reported that BIK1, an *Arabidopsis* receptor-like cytoplasmic kinase (RLCK) involved in PTI, phosphorylates OSCA1.3 and OSCA1.7 $Ca^{+2}$ channels for pattern-induced stomatal immunity but not for PTI in leaf discs (Thor et al. 2020). Zipfel proposed that PTI-induced $Ca^{+2}$ influx in mesophyll and epidermal cells relies on other BIK1-phosphorylated channels such as glutamate receptor-like channels (GLRs); *GLR2.7/2.8/2.9* are induced as part of a core *Arabidopsis* PTI response (Bjornson et al. 2021).

Aside from the $Ca^{+2}$ influx events that are hallmarks of both ETI and PTI, Rory Pruitt (University of Tübingen, Germany) addressed whether the two PRR families, LRR receptor kinases (LRR-RKs) and LRR receptor proteins (LRR-RPs) rely on the same downstream signaling components or not. By studying LRR-RK and LRR-RP signaling in *Arabidopsis*, divergent regulatory roles of BIK1 were identified between the two receptor types (Wan et al. 2019). Interestingly, other RLCKs such as PBL31 promote LRR-RP signaling, which overlaps with ETI in requiring the PAD4-EDS1 heterodimer and the RNL ADR1 (Pruitt et al. 2020). Association of the LRR-RK coreceptor for LRR-RPs, SOBIR1, with PBL31, PAD4, EDS1, and ADR1 suggests that these components might function at an intersection between ETI and PTI.

Highlighting the relevance of EDS1 and RNL complexes in ETI and PTI, Xin Li (University of British Columbia, Canada) addressed the role of the ADR1 and NRG1 families of helper NLRs in *Arabidopsis* immunity. Reinforcing the remarks from Parker in the earlier plenary session, Li showed that signal from the TNLs can be detected by parallel EDS1-SAG101-NRG1 or EDS1-PAD4-ADR1 modules, which would then facilitate $Ca^{+2}$ influx (Wu et al. 2019, 2021). New data support induction of TIR domain-containing genes, including TNLs, by microbial patterns such as nlp20 that elicit PTI, constituting a part of PTI-ETI crosstalk. Moreover, ETI and PTI overlap was demonstrated in PRR/coreceptor mutant lines by Xiufang Xin (Chinese Academy of Sciences, China). PTI signaling components were found to be critical for full ETI such as the requirement of BIK1 for activation of the NADPH oxidase RBOHD to produce ROS during both ETI and PTI (Yuan et al. 2021). Although Xin used the *Pseudomonas* effector AvrRpt2 to arrive at these conclusions, a complementary study using the *Pseudomonas* effector AvrRps4 reached similar conclusions that ETI strongly induces key PTI components such as BIK1 and RBOHD (Ngou et al. 2021).

## EMERGING CONCEPTS AND NEW INSIGHTS FROM DIVERSE EPOSTER PRESENTATIONS

Virtual ePoster presentations provided many early career researchers with a venue for showcasing their newest results for the rest of the International Society for Molecular Plant–Microbe Interactions (IS-MPMI) community. Many ePosters focused on identifying and functionally characterizing effector proteins secreted by plant pathogens to gain insight into how pathogens induce disease or suppress plant immune responses. For example, Eli Thynne (Christian-Albrechts University, Germany) discussed four effectors from *Zymoseptoria tritici* that suppress the *Pseudomonas syringae* DC3000-induced hypersensitive reaction (HR). Although the molecular and biochemical mechanisms underlying these effector activities are still unknown, investigating how they interfere with HR will likely further reveal HR-signaling components. Boyoung Kim (Seoul National University, Korea) was also interested in uncovering effectors but looked for those that induced a cell death response, by screening 40 effectors from Korean isolates of *Ralstonia solanacearum.* Kim found that the effector protein RipY induced a SGT1-dependent cell death response in *N. benthamiana* that was dependent on the C-terminal region of RipY.

Lauren Hemara (Plant and Food Research, New Zealand) used novel screening methods (Jayaraman et al. in press) to identify *P. syringae* pv. *actinidiae* biovar 3 effectors that trigger HR in kiwiberry (*Actinidia arguta*), including HopZ5. *P. syringae* pv. *actinidiae* is a major issue for kiwifruit in New Zealand, as well as in China, as was mentioned by Jian-Min Zhou in the first plenary session. Interestingly, new data from Zhou suggest that *P. syringae* pv. *actinidiae* HopZ5 could be recognized by the *Arabidopsis* NLR ZAR1. Known to be involved in detection of many effectors, ZAR1 also appears to be required for full recognition of the *P. syringae* effector HopBA1, based on data shown by Sam Ogden (Colorado State University, United States). A truncated immune receptor, RBA1, is also involved in HopBA1-triggered HR (Nishimura et al. 2017), leaving the field with the question: what are the mechanisms underlying HopBA1-triggered HR that involve RBA1 and ZAR1?

Another topic of interest in the ePoster sessions was rationally designing synthetic NLR immune receptors with broad effector recognition specificity as an effective strategy to introduce new disease resistance traits in crop plants. Janina Tamborski (University of California–Berkeley, United States) has been tackling NLR engineering and found that transferring 12 amino acids from the wheat NLR Sr50 to Sr33 was sufficient to confer recognition of AvrSr50 by the synthetic Sr33 immune receptor.

This discovery provides insight into how the LRR domain mediates recognition of effectors, and demonstrates that direct engineering of the Sr33 immune receptor is an effective strategy for introducing new disease resistance traits in wheat.

## STATUS AND FUTURE DIRECTIONS OF THE MOLECULAR PLANT IMMUNITY FIELD

The latest news at IC-MPMI in 2019 was the discovery that an NLR, ZAR1, formed a pentameric resistosome with potential to be a calcium-channel (Wang et al. 2019). Discussions were swirling around the question: "Does effector-triggered immunity (ETI) potentiate and restore pattern-triggered immunity (PTI)—or is there really a binary distinction between ETI and PTI?" This question ranked as a Top 10 Question in MPMI (Harris et al. 2020), becoming the topic of an MPMI Journal review (Lu and Tsuda 2021) with an accompanying "What's New in MPMI?" Seminar. Recent reports have shown that additional NLRs form resistosomes with channel activity, including other CNLs and likely RNLs, while oligomers of TNLs were found to have NADase activity. One theme that emerged across the presentations at this eSymposium was the use of animal systems to reconstitute protein players and make rapid progress in membrane studies: expressing the autoactive RNL NRG1 in HeLa human cells by Pierre Jacob, the CNL ZAR1 in *Xenopus* oocytes by Jian-Min Zhou, the BIK1 phosphorylated calcium channels in COS-7 mammalian cells by Cyril Zipfel, and more. Animal models have helped overcome the difficulty of studying calcium signaling and membrane dynamics in planta. Another theme was the flexibility of large protein complexes to respond to different signals to tune immune responses, especially the EDS1-SAG101/PAD4-RNL complexes that might contribute to both ETI and PTI.

The past few years have resulted in significant advances connecting and characterizing proteins across the plant immune network, with the key findings presented at this eSymposium summarized in Figure 1. However, attendees and presenters also

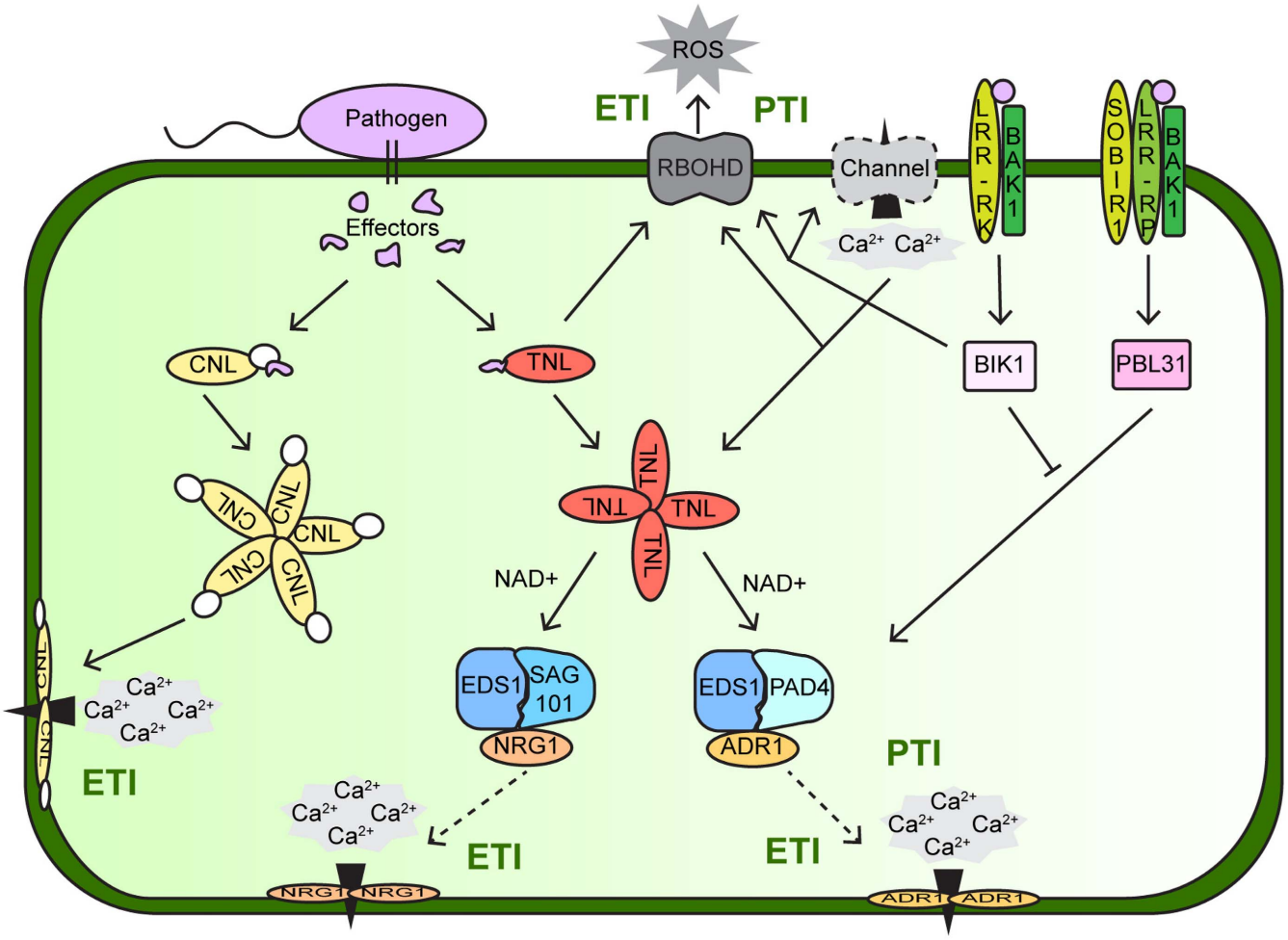


**Fig. 1.** A generalized model summarizing the main findings on molecular plant immunity presented at the first 2021 International Society for Molecular Plant–Microbe Interactions eSymposium. Fuchsia pathogen effectors are indirectly or directly detected by generic coiled-coil domain NOD-like receptors (CNLs) or Toll/interleukin-1 receptor (TIR) domain NOD-like receptors (TNLs). CNLs such as ZAR1 oligomerize into pentameric units that act as calcium ion ($Ca^{2+}$) channels causing effector-triggered immunity (ETI), while TNLs such as RPP8 oligomerize into tetrameric units with NADase activity. The NADase activity of the TNLs triggers either the interaction of the EDS1-SAG101 or EDS1-PAD4 complex with the RPW8-like domain NOD-like receptors (RNLs) NRG1 and ADR1, respectively. Autoactive NRG1 can oligomerize to form a calcium channel leading to calcium ion influx for ETI; ADR1 is proposed to act similarly but is also involved in PTI. A dotted line indicates a gap in understanding of how complex with EDS1-SAG101/PAD4 impacts RNL oligomerization. Extracellular pathogen-derived molecules are detected by generic leucine-rich repeat receptor kinases (LRR-RKs) and leucine-rich repeat receptor proteins (LRR-RPs), both in complex with the coreceptor BAK1. LRR-RPs rely on the LRR-RK SOBIR1 as well. The kinase PBL31 is activated by the LRR-RP complex which connects to the EDS1-PAD4-ADR1 complex as well; there is evidence for a direct interaction between SOBIR1, EDS1, PAD4, ADR1, and PBL31, which is not depicted here for simplicity. Signaling through the LRR-RK and BAK1 complex activates BIK1, which represses LRR-RP signaling, activates some calcium channels, and activates the oxidase RBOHD to create a reactive oxygen species (ROS) burst. Feedback from the calcium ions can also contribute to RBOHD activity as well as TNL signaling, which ultimately lead to PTI or ETI ROS bursts.

identified new questions to be answered. Calcium signaling has long been known to play a role in both ETI and PTI but how, specifically, does $Ca^{+2}$ influx control signaling and cell death for both ETI and PTI? What are the decision-making processes leading to the recruitment of defense pathways such as EDS1 interactions with PAD4 or SAG101? Localization studies bring up the questions of where and how active resistosomes form and whether they have to migrate to the plasma membrane to execute their signaling role? ETI appears to require PTI components for a full response; thus, how does ETI overcome effector suppression of PTI? Furthermore, our understanding of the synergy between ETI and PTI largely comes from studies on model dicot plant species such as *Arabidopsis* and wild tobacco. What variations, conservation, and convergent evolution will be found as studies branch out across crop and other plant species? Questions such as these have replaced that top unanswered question of 2019 as we look forward as a field to the nuances of overlap in proteins and outputs between ETI and PTI.

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