formed a strongly coupled state. They could map out spectroscopically a change of total nuclear spin by a single unit of angular momentum. This change corresponded to a single nuclear magnon, the elementary quantum unit of a collective nuclear wave (see the figure).

Finally, Gangloff *et al.* coherently manipulated the coupled electron-nuclei entity in which a large fraction of quantum dot nuclei are involved, which launched a nuclear magnon by all-optical means. This process corresponds to a coherent, nondissipative exchange between the electron spin and the collective of nuclei—a prerequisite for a quantum memory.

These results could be the first step toward the development of a quantum memory interface with sufficiently long coherence time with quantum dots. The coherence benefits from the many-body nature of the nuclear system, which provides robustness. Moreover, every quantum bit would be associated with its dedicated inherent memory by default. This step has been the missing piece of the puzzle for a semiconductor nanostructure QI platform. As for carrier spin quantum bits, other key demonstrations (*6*) have been provided already, such as efficient initialization and manipulation on time scales of nanoseconds or even shorter as well as the efficient interconversion with photons for information transfer. Also, at a fundamental level, these results are highly interesting because a quantum many-body state for the nuclei has been established that can be coherently manipulated optically through the electron spin. It should be possible to create specific nonclassical nuclear states, such as Schrödinger cat states. ■

"In this regime…the electron spin and nuclear ensemble formed a strongly coupled state."

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PLANT IMMUNITY

A pentangular plant inflammasome

The first plant resistosome structure provides clues to cell death control and immunity

By **Jeffery L. Dangl**[1,2] *and* **Jonathan D. G. Jones**[3]

Like animals, plants require, and have evolved, a robust innate immune system. Plants can detect and respond to pathogen-derived molecules (effectors) through cell surface receptors and intracellular receptors, typically encoded by disease resistance (*R*) genes. Analysis of plant genome sequences reveals hundreds of such nucleotide-binding, leucine-rich repeat (NLR) proteins encoded by putative *R* genes. How such NLR proteins function has long been a matter of speculation. On page 43 and 44 of this issue, Wang *et al.* (*1*) and Wang *et al.* (*2*), respectively, end much of the speculation by defining the mechanism of activation for at least one NLR, the *Arabidopsis thaliana* HOPZ-ACTIVATED RESISTANCE 1 (ZAR1) protein, which activates defense in response to several pathogenic bacterial effectors.

The mechanism of ZAR1 immune response depends on nucleotide triphosphate (NTP)–dependent oligomerization, as broadly anticipated on the basis of mechanisms of mammalian NLR proteins, but fascinating and specific details are revealed in these definitive analyses. Mammalian NLR proteins can form inflammasomes, specialized immunity protein nanomachines, upon detection of intracellular pathogen–derived ligands (*3*). The human genome encodes 22 NLR proteins. Mammalian NLRs mostly carry a NACHT nucleotide binding domain [in contrast to plant nucleotide binding–ARC (NB-ARC) domains (*4*)], which are commonly flanked by carboxyl-terminal leucine-rich repeats (LRRs) and amino-terminal domains of either caspase activation and recruitment domain (CARD) or pyrin domain (PYD). As with plant NLRs, the LRRs are believed to function in ligand sensing and autoregulation, whereas CARD and PYD domains activate downstream signaling. The NACHT domain enables activation of the signaling complex by means of adenosine triphosphate (ATP)–dependent oligomerization.

This mechanism is shared with the mammalian apoptotic protein apoptotic protease-activating factor 1 (APAF1) with its modular structure of CARD, NB-ARC and WD40 repeats. APAF1 promotes caspase activation upon detection of cytochrome c released from mitochondria through its WD40 repeats, leading to apoptosis. Cytochrome c binds and activates APAF1, which results in the formation of an oligomer of seven APAF1 molecules (*5*). This oligomerization imposes induced proximity of the amino-terminal CARDs, resulting in irreversible caspase activation and apoptosis.

The best studied NLR inflammasome is that formed by mouse NLR family CARD domain–containing protein 4 (NLRC4) with NLR family apoptosis inhibitory proteins (NAIPs), which recognize specific bacterial ligands and consequently activate caspases (*6*). NLRC4 has the modular structure comprising CARD, NACHT, and LRR. Upon detection of bacterial PrgJ or flagellin by NAIP2 or NAIP5, respectively, an oligomer of one NAIP and 10 NLRC4 monomers is formed. Again, induced proximity of the CARD domains upon NACHT- and NTP-dependent oligomerization results in caspase activation. In the complexes of NLRC4 with NAIP2 or NAIP5, the CARD domains are flexible, presumably until stabilized by interaction with downstream signaling caspases.

A. thaliana ZAR1 recognizes the differential biochemical activities of bacterial effector proteins on substrates that belong to the class XII receptor–like cytoplasmic kinase (RLCK) family. Resting state ZAR1 is precomplexed with one of several RLCK class XII pseudokinases: ZED1, ZRK3, and RECEPTOR-LIKE PROTEIN KINASE 1 (RKS1). These three ZAR1-pseudokinase complexes are activated, respectively, by an acetyltransferase (HopZ1a) (*7*), a ribosyltransferase (HopF2) (*8*), and, as studied in the two articles by Wang *et al.*, a uridyltransferase (AvrAC).

These three bacterial effectors all biochemically modify different class VII RLCKs that function to transduce immune signals from the cell surface, thus enhancing pathogen virulence. They also modify decoys (*9*) of

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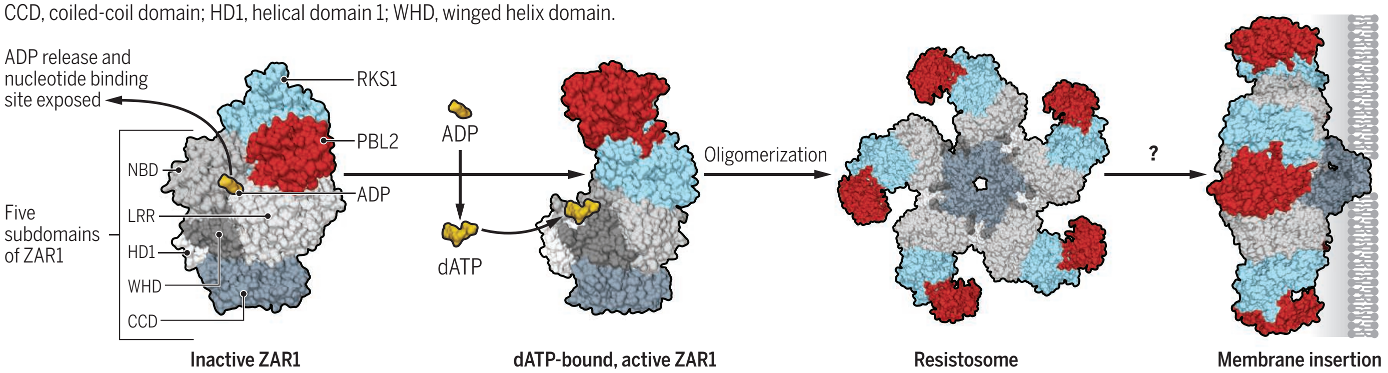
Activation of a plant immune receptor resistosome

Cryo-EM protein structures depict the resting state and activation steps of a pentameric oligomerized plant NLR receptor complex. CCD, coiled-coil domain; HD1, helical domain 1; WHD, winged helix domain.



these "virulence target" kinases; for example, AvrAC uridylylates the *A. thaliana* decoy kinase PBS1-LIKE PROTEIN 2 (PBL2). The authors focus on the mechanism by which uridylylated PBL2 (class VII RLCK) activates the ZAR1-RKS1 (class XII RLCK pseudokinase) complex. Class VII and XII RCLKs are important plant immune regulators. AvrAC uridylylates PBL2, resulting in its association with RKS1, and formation of a complex with ZAR1 that activates innate immune defense (*10*). ZAR1 is a flexible signaling platform that has evolved to monitor the homeostasis of an important immune system battlefield, the RLCK class VII kinases.

Wang *et al.* (*1*) coexpressed ZAR1 and RKS1 in insect cells. They then purified the complex and solved a cryo–electron microscopy (cryo-EM) structure in the presence of bound adenosine diphosphate (ADP). This defines the autoinhibited ZAR1 resting state. The key features of this complex are that it is heterodimeric—RKS1 interacts exclusively with the ZAR1 LRR domain. The amino acid residues that govern this interaction are conserved in the other class XII pseudokinases, including ZED1 and ZRK1, as predicted, because they form independent preactivation complexes with ZAR1 to monitor the various biochemical modifications of class VII RLCKs by different bacterial effectors.

Wang *et al.* (*1*) expressed AvrAC and PBL2 in *Escherichia coli*, purified the mono-uridylylated PBL2, and added PBL2 to the ZAR1-RKS1 complex. The association of uridylylated PBL2 with RKS1 drives a large conformational change on ZAR1 that allosterically evicts ADP from the ATP binding pocket in the NB-ARC domain. However, in the absence of ATP or deoxyadenosine triphosphate (dATP), this complex persists as a heterotrimer of ZAR1–RKS1–uridylylated-PBL2, likely an intermediate in the activation mechanism. The amino acid residues that govern the interaction of uridylylated PBL2 with RKS1 are not conserved with other class XII RLCKs, explaining why ZAR1 activation by the different effectors is specific in each case.

Wang *et al.* (*2*) beautifully demonstrate how, upon provision of dATP, the ZAR1–RKS1–uridylylated-PBL2 complex oligomerizes into a larger form, which they isolated and subjected to structural analysis by cryo-EM. This form, a pentamer, imposes induced proximity of the amino-terminal region of ZAR1 that contains four α helices in a coiled coil–like domain. In the oligomerized ZAR1, there are substantial reconfigurations of these α helices: The helix directly at the amino terminus "melts," undergoes a fold switch and is replaced by another α helix. A pentamer of these amino-terminal α helices elevated above the pentameric structure in a funnel shape creates the potential to engage with and perhaps create pores in membranes (see the figure). This speculation remains to be validated but will provide the basis for many interesting future experiments.

Activated ZAR1 relocalizes to the plasma membrane, and the amino-terminal α helix is required for this. Mutation analysis and pathology experiments established that amino acids on the outside of the pentameric amino-terminal α-helical funnel alter ZAR1 functions in disease resistance and cell death, but not oligomerization. Additionally, negatively charged residues from the interior of the putative pore forming the α-helical funnel are also required for ZAR1 functions, but not for oligomerization and membrane localization, suggesting that the interior of the funnel provides specific ZAR1 functional attributes.

These important findings substantially advance our understanding of plant innate immune mechanisms. Whether all plant NLRs work by means of NTP-binding-dependent oligomerization of the NB-ARC domain is an open question. The answer seems likely to be yes, because the mechanism shows such profound similarities between plant and animal NLRs. What happens after oligomerization? There are many subcellular localizations reported for plant NLRs that contain amino-terminal coiled coil–like domains, similar to the α-helical domain in ZAR1. Perhaps these also oligomerize to form pores in different plant membrane systems to cause cell death. If pore formation is a general function of NLRs, what ions might flow through those pores and how might they function? For example, calcium influx is a well-known correlate of NLR activation.

Some NLRs relocalize to the nucleus upon activation and are thought to interact with transcriptional machinery to drive defense responses that are associated with immune responses. How can this be reconciled with a general NLR oligomerization model with potential pore formation? The other major structural class of plant NLRs are those with amino-terminal Toll/interleukin-1 receptor/resistance protein homology (TIR) domains function. In plants, TIR-NLRs require "helper" NLRs of the evolutionarily ancient NRG1-ADR1 clade of NLRs that potentially carry a membrane-engaging region at their amino termini. This region shares homology with mammalian mixed-lineage kinase domain–like protein (MLKL), a pore-forming protein (*11*). Additional plant sensor/helper NLR combinations exist (*12*). Different classes of plant NLRs may act through different signaling mechanisms, but the results from the important studies of Wang *et al.* strongly suggest that nucleotide-dependent NLR oligomerization will usually be involved in signal initiation. ■

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