NPR1 and control of cell survival in plant immune responses

In plants, pathogen effector-triggered immunity (ETI) often culminates in cell death, which is restricted by NPR1, an activator of systemic acquired resistance. Recently, we found that NPR1 promotes cell survival by targeting over-accumulated stress proteins for ubiquitination and degradation through formation of salicylic acid (SA)-induced NPR1 condensates (SINCs). SINCs are enriched with defense proteins, including a number of immune receptors, oxidative and DNA damage response proteins, and protein quality control machineries. Transition of NPR1 into condensates is required for formation of the NPR1-Cullin 3 E3 ligase complex to ubiquitinate SINC-localized proteins and promote cell survival. NPR1 not only sequesters stress proteome in SINCs, but also induces transcription of a large number defense genes, including SINC components, by serving as SA-activated transcription cofactor. Our recent structural analysis revealed that NPR1 is a bird-shaped homodimer consisting of a central BTB domain, a BTB- and carboxyterminal Kelch helix bundle (BHB), four ankyrin repeats and a disordered SA-binding domain. In the BTB domain, NPR1 harbors a unique zinc-finger motif essential for interacting with ankyrin repeats and mediating NPR1 oligomerization. We found that SA induces folding and docking of the SA-binding domain onto the ankyrin repeats. Importantly, such docking is required for the transcriptional coactivator function of NPR1 homodimer in complex with TGA3 transcription factor. The new structural information on the NPR1-TGA transcriptional complex together with our analysis of SINCs significantly advance our understanding of NPR1's function in providing plants with protection against multiple stresses.