

**Thursday, September 25, 9:00-9:30 a.m.**

*“Connecting the dots: Linking PAMP perception to receptor kinase-mediated immune outputs”*

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The first layer of plant innate immunity relies on the recognition of microbes via the perception of pathogen-associated molecular patterns (PAMPs) by surface localized receptors called pattern recognition receptors (PRRs). The Arabidopsis leucine-rich repeat receptor kinases (LRR-RKs) FLS2 and EFR are the PRRs for bacterial flagellin (or flg22) and elongation factor Tu (or elf18), respectively. Within seconds of PAMP binding, FLS2 and EFR form a ligand-induced complex with the regulatory LRR-RK SERK3/BAK1 leading to phosphorylation of both proteins. FLS2 (and potentially EFR) also forms a constitutive complex with the membrane-associated cytoplasmic kinase BIK1 that get phosphorylated in a BAK1-dependent manner upon PAMP binding. BIK1 is a positive regulator of several FLS2- and EFR-mediated responses. FLS2 and EFR activation leads to several immune responses, including bursts of  $\text{Ca}^{2+}$  and reactive oxygen species (ROS), activation of mitogen-activated protein kinases (MAPKs) and calcium-dependent protein kinases (CDPKs), and transcriptional reprogramming, ultimately leading to PAMP-triggered immunity. The mechanisms controlling PRR activation at the plasma membrane and regulating intracellular signaling remain however largely unknown. Here, I will present recent work illustrating how activated PRR complexes at the plasma membrane directly engage with downstream signaling, and how these events are tightly regulated by phosphorylation.