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Plant cellular self/non-self discrimination is orchestrated by bacterial pathogens V. Go[°] ehre, T. Spallek, H. Ha[°] weker and S. Robatzek Max-Planck Institute for Plant Breeding Research, Department of Plant-Microbe Interactions, Cologne, GERMANY Introduction: Self/non-self discrimination is the underlying principle by which higher organisms sense microorganisms including potential pathogens. Presence of microbes is monitored by pattern recognition receptors (PRRs) by recognizing so-called microbe-associated molecular patterns (MAMPs). Upon perception of MAMPs, plants initiate a suite of defense responses that lead to plant immunity. To overcome this level of perception, pathogens deliver effector molecules into the plant cell that interfere with immune responses.

Results: We are studying the Arabidopsis thaliana pattern recognition receptor FLS2, which recognizes bacterial flagellin, flg22, and triggers defense reactions such as stomata closure, ROS production, cell wall fortification, and expression of defense genes. Due to recognition at first contact, FLS2 restricts bacteria from entry into plant tissues and limits bacterial proliferation. Therefore, pathogenic bacteria target FLS2 function. AvrPtoB, an effector from Pseudomonas syringae pv. tomato, suppresses immune responses triggered by FLS2. Expression of AvrPtoB in planta leads to degradation of flg22 activated FLS2. AvrPtoB is a modular protein, which carries an E3 ligase activity in its C-terminal part. Interaction of AvrPtoB with FLS2, which does not require the C-terminal domain, was shown in pull-down assays. Furthermore, FLS2 is ubiguitinated by AvrPtoB in vitro.

Fequire the C-terminal domain, was shown in pull-down assays. Furthermore, FLS2 is ubiquitinated by AvrPtoB in vitro. Conclusions: We propose a model, in which AvrPtoB binds to FLS2 independently of the E3 ligase function and prevents acute signaling. Ubiquitination of FLS2 is a subsequent step, which allows degradation of this receptor and thereby permanent down-regulation of defense signaling. Our studies provide mechanistic insights about how plants and microbes interact and how host immune response are subverted by pathogens.