

understand intercellular signaling in radial embryo development. Plants homozygous for Like Kinases (RLKs) RPK1 and TOAD2 type, known as the Toadstool phenotype, are involved in proper radial patterning. TOAD2 have overlapping expression during development, and their Leucine-Rich Repeat domains, which vary considerably, are likely important in intercellular signaling. Our experiments have pointed towards CLE receptors as potential ligands. CLE genes, interacting with RPK1 and/or TOAD2, are expressed in Arabidopsis roots with a CLE peptide inhibits root growth. We are treating roots with the same peptide, not suggesting that the TOAD2 receptor is involved in the response to the CLE peptide. We are also studying RPK1/TOAD2 mediated signaling, and its role in the expression pattern of these genes. Thus, we are using promoter fusions, as well as GUS promoter fusions, to see if they can be expressed during early development. We are using the information from the expression of the CLE gene activity affects radial patterning. We are also studying the potential redundancy within this 32 member family. We are also studying the problem of CLE ligands. To circumvent the problem of CLE ligands, we are using a genetic strain that may allow for detection of CLE ligands. Lastly, we are using a yeast two-hybrid system to study an interaction between the CLE ligands and RPK1 and TOAD2 receptors. Together, we are studying the expression and function of these receptors and how they interact with RPK1 and TOAD2. We are also studying the role of NSF grants in this research.

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Plasmodesmata (PD) are plant-unique intercellular communication channels that provide plants the basis for multicellular or supracellular organization. These fundamental structures are thought to play crucial roles in controlling systemic defense responses against pathogens as well as in orchestrating cellular differentiation and pattern formation during plant development. Here we report the identification of the PD-targeted protein PDLP5, which may play a critical role in orchestrating PD-mediated cell-to-cell communication and defense signaling. Supporting evidence showed that PDLP5 accumulated at the primary pit-fields, its expression was induced by pathogen infection and salicylic acid treatment, and modulation of this gene altered PD permeability and susceptibility to pathogen. Moreover, constitutive overexpression of this protein led to a spontaneous cell death reminiscent of hyper-sensitive response in leaf tissue, formation of autophagy, and hyperinduction of chloroplast stromules. The working model, along with molecular mechanisms potentially underlying this process, that illustrates PDLP5 as a novel molecular link between coordinated regulation of cell communication at the PD level and controlled cell death will be presented.

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In *Arabidopsis* flowers, abscission is triggered by activation of HAES (HSL2), a pair of redundant, leucine-rich repeat receptor-like kinases (Cho et al., 2008). We have identified a novel protein, the ribosylation factor GTPase-activating protein (NEV), that regulates membrane trafficking and is required for the movement of HAES to the abscission site. Through a sensitized assay, we have shown that NEV restores organ shedding in *nev* flowers. Overexpression of three receptor-like kinases—*HAES1*, *HAES2*, and *CAST AWAY* (*CST*)—that function as inhibitors of abscission. The organ shedding is restored prematurely, and enlarged abscission zones are observed. *evr*, *nev serk1* and *nev cst* flowers reported for plants constitutively overexpressing signaling ligand for HAE and HSL2 pathway may be ectopically active in *nev cst* flowers. We will present a model in which EVR, SERK1 and CAST AWAY regulate the region of abscission by regulating the activity of HAE and HSL2.

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**agonistically regulate dynamic auxin interdigitation of leaf pavement cells in Arabidopsis**

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Development of interdigitated lobes and indentations is a previously shown that auxin acts as a coordinate lobing with indenting by activating signaling pathways. In expanding Arabidopsis leaves apparently show tip- and margin-high auxin with the progression of PC interdigitation from primary veins. We found that defects in cytokinin receptors or cytokinin receptor mutant *ahk3cre1* caused intercalation and accumulation of auxin, whereas overexpression of *ahk3* (over-expression), auxin receptors (*TIR1* and *AUX1*) function mutant delayed or abolished PC intercalation. In all these cases the intercalation defects were rescued by auxin, in contrast to the *ahp1-5* and *rop2 rop4* mutants that cannot be rescued by auxin. These results support the idea that auxin signaling and cytokinin counteract to produce intercalation which act as an instructive signal to globally coordinate throughout the entire leaf. Accordingly auxin levels in the level of auxin for a specific cell, and this intercalation and cell-cell coordination of lobing with auxin-dependent pathways. These findings establish a signaling pathway that control a specific intercalation signaling that regulates nuclear gene expression in response to a hormonal signal that modulates a specific plasma membrane/cytoplasmic signaling mechanism.

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**152 Interaction of the receptor-like protein Cf-4 with components of endosomal trafficking and identification of novel interactors**

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The plasma membrane-resident tomato (*Solanum lycopersicum* (Sl)) Cf receptor-like proteins recognize Avr effectors of the fungal pathogen *Cladosporium fulvum*. To date, very few host components involved in Cf-signaling have been identified. Cf-4 contains a typical Yxxø endocytosis motif in its short cytoplasmic domain. These motifs bind µ adaptins involved in clathrin-mediated endocytosis. *Agrobacterium*-mediated co-expression of Cf-4, mutated in the Yxxø motif, with Avr4 in *Nicotiana benthamiana* does not result in a hypersensitive response (HR), in contrast to wild-type Cf-4, suggesting the Yxxø motif is of functional significance.

We here employed a split-ubiquitin-based yeast two-hybrid method to study interaction of full-length Cf-4 with tomato µ adaptins. The adaptins (A-D) were cloned based on homology to the *Arabidopsis* µ adaptins (1). Cf-4 specifically interacts with adaptin SlµC, confirming earlier observations using only the cytoplasmic domain of Cf-4 in a conventional yeast two-hybrid assay. Cf-4 variants altered in the Yxxø motif, Cf-4<sup>Y780A</sup> and Cf-4<sup>W783D</sup>, still bind SlµC in the split-ubiquitin assay, indicating that the motif at positions 780-783 is not required for SlµC binding. There is a second Yxxø motif present at the very C-terminus of the transmembrane domain and we will investigate whether this motif is required for SlµC binding.

A split-ubiquitin tomato cDNA library, enriched for expressed defense genes, was screened for Cf-4 interactors. This library was generated from Cf-4/Avr4 tomato plants mounting the HR (2). A first screen revealed several interesting novel Cf-4 candidate interactors that are currently validated. In addition new screens will be performed.

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**References**  
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**155 Involvement of Arabidopsis RACK1 in ABA signaling and its Regulation by Abscisic Acid**

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Earlier studies have shown that RACK1 is involved in ABA responses in Arabidopsis, but the role of RACK1 in these processes remains unclear. We have revealed that approximately 40% of the RACK1 protein is in a similar manner by the view that RACK1 is a major regulator of protein translation. Co-expression analysis revealed that >10 RACK1 encode ribosome proteins, implicating RACK1's function and the ribosome code in the regulatory role for RACK1 in ABA response. A putative function in protein translation, RACK1 is involved in processes that mammalian and yeast RACK1 homologous proteins are involved in. Three Arabidopsis RACK1 homologous proteins, RACK1C, RACK1D, and RACK1E, complemented the growth defect of a *rack1* mutant. In addition, RACK1 physically interacts with eukaryotic Initiation Factor 6 (eIF6), which is a key regulator of protein translation initiation. Overexpression of RACK1 displayed hypersensitivity to anisomycin and displayed characteristics of impaired protein synthesis and 60S ribosomal subunit biogenesis. Co-expression analysis revealed that ABA signaling pathway and eIF6. Taken together, these results suggest that RACK1 is involved in the control of protein translation and is regulated by ABA.

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