Division of Biochemistry
– Molecular Biology –

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Scope of Research

This laboratory aims at clarifying molecular bases of regulatory mechanisms for plant development, especially plant morphogenesis, with techniques of forward and reverse genetics, molecular biology, and biochemistry. Current major subjects are phospholipid signalings in cell morphogenesis, the transcriptional network for cytokinin responses, COP9 signalosome modulating signal transduction in the nuclei, and the endoreduplication cell cycle in cell differentiation.

KEYWORDS

Morphogenesis
Signal Transduction
Phospholipid
COP9 Signalosome
Cytokinin

Selected Publications

Kato, M.; Aoyama, T.; Maeshima. M., The Ca^{2+}-binding Protein PCaP2 Located on the Plasma Membrane is Involved in Root Hair Development as a Possible Signal Transducer, *Plant J*, **74,** 690-700 (2013).

Lin, Q.; Aoyama, T., Pathways for Epidermal Cell Differentiation *via* the Homeobox Gene *GLABRA2*: Update on the Roles of the Classic Regulator, *J. Integr. Plant Biol.*, **54,** 729-737 (2012).

Aki, S.; Nakai, H.; Aoyama, T.; Oka, A.; Tsuge, T., *AtSAP130/AtSF3b-3* Function is Required for Reproduction in *Arabidopsis thaliana*, *Plant Cell Physiol.*, **52,** 1330-1339 (2011).

Taniguchi, Y. Y.; Taniguchi, M.; Tsuge, T.; Oka, A.; Aoyama, T., Involvement of *Arabidopsis thaliana* Phospholipase Dζ2 in Root Hydrotropism through the Suppression of Root Gravitropism, *Planta*, **231,** 491-497 (2010).

Kusano, H.; Testerink, C.; Vermeer, J. E. M.; Tsuge, T.; Shimada, H.; Oka, A.; Munnik, T.; Aoyama, T., The *Arabidopsis* Phosphatidylinositol Phosphate 5-kinase PIP5K3 Is a Key Regulator of Root Hair Tip Growth, *Plant Cell*, **20,** 367-380 (2008).

Menon, S.; Tsuge, T.; Dohmae, N.; Takio, K.; Wei, N., Association of SAP130/SF3b-3 with Cullin-RING Ubiquitin Ligase Complexes and Its Regulation by the COP9 Signalosome, *BMC Biochem.*, **9,** 1 (2008).

Putative MATE Transporter Involved in Plant Architecture *via* Regulating Auxin Biosynthesis

Plant architecture is largely determined by genetic programs and, to some extent, by environmental cues, such as light, humidity, temperature, nutrition, and plant density. To understand how these factors are integrated into plant development, detailed studies have been focused on genetic factors that are crucial for maintenance of shoot apical meristem (SAM), initiation and outgrowth of axillary meristem (AM), proper growth rate for lateral organ development, and correct timing for reproduction and senescence. As a result, a number of gene-mutations, of which cause altered plant architecture, have been revealed to encode factors involved in biosynthesis and/or signaling of plant hormones, including auxin. Auxin is a critical factor controlling a wide variety of developmental processes, including embryogenesis, maintenance of apical dominance, and formation of lateral organs. Active auxin, mainly indole-3-acetic acid (IAA), is reported to be synthesized *de novo* by tryptophan (Trp)-dependent and/or independent pathways in the shoot apex, young leaves, and root apex. After synthesis, auxin is transported by the polar transport machinery, so that an appropriate distribution of auxin is established to maintain normal plant architecture.

We identified a dominant Arabidopsis mutant with an abnormal architecture, which we named *adp1*-D (*altered development program 1*- Dominant). The architecture of *adp1*-D was greatly altered at maturity, with increased number of axillary branches, flowers, and lateral roots (Figure 1). The growth rate of the mutant was accelerated throughout its life cycle. We found that the mutant phenotypes were caused by over-expression of *ADP1* gene, and that the gene encodes an endosome-localizing protein with sequence similarity to the multidrug and toxic compound extrusion (MATE) transporter family (Figure 2). MATE transporters are reported to be involved in a variety of important biological processes, including the exclusion of toxic organic cation and disease resistance and exhibit multi-substrate specificity in prokaryotes and eukaryotes.

Figure 1. Six-week-old wild-type (WT), and *adp1*-D heterozygous (+/-) and homozygous (-/-) plants grown under long-day conditions. The mutation of *adp1*-D caused a bushy phenotype dominantly. Bar = 1 cm.

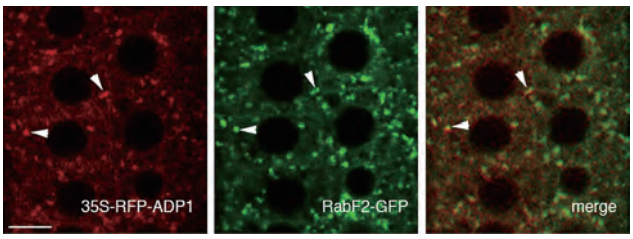

Figure 2. Co-localization of RFP-ADP1 with the endosome marker RabF2a-GFP. Arrowheads indicate Endosomes to which RFP-ADP1 and RabF2a-GFP co-localize. Bar = 15 μm.

Our molecular and genetic evidence demonstrated that the phenotypes of plants over-expressing *ADP1* were caused by reduction of local auxin levels in the meristematic regions. We further discovered that this reduction was probably due to decreased levels of auxin biosynthesis in the local meristematic regions based on the measured reduction in IAA levels and the gene expression data. Overexpression of the gene encoding the auxin-synthesizing enzyme YUCCA1 partly complemented the phenotype of *adp1*-D (Figure 3). Our results indicated that *ADP1*-mediated regulation of the local auxin level in meristematic regions is an essential determinant for plant architecture maintenance by restraining the outgrowth of lateral organs.

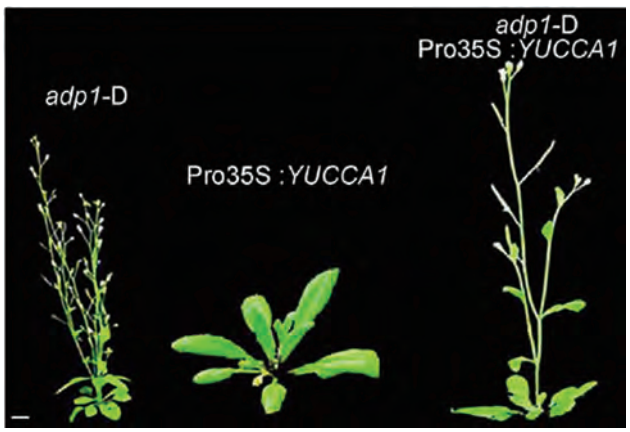

Figure 3. Thirty-five-day-old plants of *adp1*-D, Pro35S:*YUCCA1* and double mutants of *adp1*-D Pro35S:*YUCCA1*. Overexpression of *YUCCA1* partly complemented the phenotype of *adp1*-D. Bar = 1 cm.