Molecular Characterization of the Cytoplasmic Interacting Protein of the Receptor Kinase IRK Expressed in the Inflorescence and Root Apices of *Arabidopsis*

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Meristem maintenance and differentiation is regulated by intercellular communication through receptor-like kinases (RLKs) in plants, but the underlying molecular mechanisms of RLK signaling remain largely unknown. A cytoplasmic interactor for inflorescence and root apices receptor-like kinase (IRK), which is a typical meristematic RLK with leucine-rich repeats in *Arabidopsis*, was identified using a yeast two-hybrid assay and named IRK-interacting protein (IRKI). IRKI is a novel but highly conserved protein found in higher plants. The interaction between IRK and IRKI was confirmed by an in vitro pull-down assay and supported by their simultaneous expression in actively dividing cells in meristems. In the root tip, IRKI expression and localization visualized by green fluorescence protein (GFP) were observed in the quiescent center, initial cells, and immature stele cells. IRKI expression was expanded by exogenous auxin treatment and repressed by inhibitor treatment of polar auxin transport.

Key words: *Arabidopsis thaliana*; auxin; initial cell; meristem; receptor-like kinase

Meristems located in the shoot and root apices have essential roles in plant development. All organs are derived from meristems; for example, shoot apical meristem (SAM) cells produce daughter cells that form leaf primordia and floral meristems in the SAM periphery. Undifferentiated stem cells are maintained through cell division and enable meristems to achieve indeterminate growth. Consequently, SAMs must maintain a balance between differentiated and undifferentiated cells. On the other hand, cells neighboring the quiescent center (QC) in root meristems (RMs), namely the initials, produce the root cap and concentric structure of roots. Cells in the SAMs and RMs recognize and maintain their own identities by positional information. Hence, detailed analysis of intercellular communications and the subsequent intracellular signal transduction in meristems should help to clarify the molecular mechanisms of plant development. Receptor-like kinases (RLKs) such as CLAVATA1 (CLV1) and ERECTA (ER) are necessary for maintaining meristems. *clv1* mutants produce abnormally expanded SAMs that result in stem fasciation and an increased number of floral organs, while *er* mutants produce short internodes that result in clustered buds and compact inflorescences. Plant RLKs are expected to play an important role in intercellular communication during tissue identity maintenance and the regulation of development.

In the past decade, analysis of RLKs and related signaling pathways has proceeded steadily, revealing a few of the molecular components of the pathway. These studies have clarified the functions of various RLKs, including the roles of *S*-receptor kinase (*SRK*) in self-incompatibility, *CLV1*, and *ER* in meristem development, *XA21* in disease resistance, *Brassinosteroid insensitive 1 (*BRI1*) and Phytosulfokine receptor (*PSKR*) in hormone perception, and *Nod-factor receptor kinase* (*NFR5*) in nodule development. More than 400 RLKs are thought to function in *Arabidopsis*, and a leucine-rich repeat (LRR)-type receptor group is known to be the largest family, comprising more than 200 members. Most RLKs are predicted by sequence homology, hence their functions and signaling pathways remain unknown.

The signaling components related to some RLKs have been identified by various methods, including genetic analysis, yeast two-hybrid assay, and microarray experiments. Using biochemical assays, CLV3, kinase-associated protein phosphatase (KAPP), and Rho-related GTPases from plants (Rop) have been

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**Abbreviations:** RLK, receptor-like kinase; IRK, inflorescence and root apices receptor-like kinase; IRKI, IRK-interacting protein; GUS, β-glucuronidase; GFP, green fluorescent protein; SAM, shoot apical meristem; RM, root meristem; QC, quiescent center; KD, kinase domain; GST, glutathione S-transferase; 2,4-D, 2,4-dichlorophenoxyacetic acid; NPA, N-1-naphthylphthalamic acid; PI, propidium iodide; CEI, initial cells of the cortex and endodermis
shown to be CLV1 complex components. In addition, SHEPHERD (SHD) shows a clv-like phenotype when mutated, and has an indispensable role in the formation of CLV protein complexes and/or in CLV protein folding. Furthermore, molecular genetic approaches have shown that WUSCHEL (WUS) establishes a feedback loop with CLV1. BRII-signaling components have also been characterized, and direct interactor BRII-associated receptor kinase 1 (BAK1), and downstream components BIN2, BZR1, and BES1 have been isolated. In a recent study, an LRR-type RLK designated meristematic receptor-like kinase (MRLK) was reported to interact with putative transcription factor AGAMOUS-like 24 (AGL24). MRLK was expected to directly phosphorylate AGL24, although the functional significance of this phosphorylation remains unknown. Although the Arabidopsis genome sequencing project has revealed that a large number of RLKs exist, very few RLK-interacting proteins have been characterized.

Previously, the authors performed differential screening using an equalized cDNA library to search for the RLKs that regulate development in meristems, and identified an inflorescence and root apices receptor-like kinase (IRK). The IRK gene encodes a LRR-type RLK and is expressed in proliferating and expanding tissues, such as shoot meristems, floral buds, and root meristems. In spite of extensive genetic studies, the function of IRK has not been clarified. To obtain a further understanding of IRK and RLK-mediated signaling in Arabidopsis, yeast two-hybrid screening was used to search for the cytoplasmic interactor of IRK. This paper reports the isolation and characterization of IRK interacting protein (IRKI), and shows the link between IRKI/IRK and the multicellular organization of meristems in the shoot and root apices.

Materials and Methods

Plant materials. Arabidopsis thaliana (ecotype Columbia) was used as the wild type. The plants were grown in soil under continuous fluorescent illumination at 22°C. Roots were harvested from plants maintained on Murashige and Skoog plates. Arabidopsis thaliana suspension cells, MM2d, were cultured as described previously.

Yeast two-hybrid screening. As a bait construction, the coding region of the kinase domain (KD) corresponding to the amino acid sequence from 628 to the C terminus of IRK (At3g56370, GenBank: AB076907) was amplified by PCR using original IRK cDNA with the following forward primer: (5'-GGGGATCCGAGTGACTTGTACCTGCAAA-3'). The IRK-KD DNA fragment was digested by BglII and XhoI and fused downstream of the GAL4 DNA binding domain in a yeast vector, pGBT9 (Matchmaker-two-hybrid system, Clontech Laboratory, Palo Alto, CA, U.S.A.). cDNA library construction, yeast transformation, the HIS3 reporter and filter lift assays, and quantitative analysis of β-galactosidase activities were conducted as described previously.

Isolation of full-length cDNA. A full-length IRKI cDNA clone was isolated from a λZLIBOLOX (Invitrogen, Carlsbad, CA, U.S.A.) cDNA library generated from Arabidopsis inflorescence tissues. An IRKI cDNA fragment in pAD-GAL4 was used as a probe for screening. The cDNA fragment inserted in the λ phage vector and screened by the probe was excised to form a phagemid (pZL1), and sequenced with dye terminator using thermo sequenase (Amersham Biosciences, Buckinghamshire, U.K.).

In vitro binding assay. The IRK-KD cDNA fragment used in yeast two-hybrid screening was inserted into the SalI and NotI sites of pGEX4T-3 (Amersham). The glutathione S-transferase IRK-KD fusion protein (GST-IRK-KD) was expressed in Escherichia coli and purified with glutathione Sepharose 4B (Amersham). The IRKI fragment in pAD-GAL4 was amplified by PCR with the following primers: 5'-TACCATTACAATGGATG-3' and 5'-CCGGCTCGAAGGACTTGTAACCTGAAAAG-3' primers, digested with EcoRI and XhoI, and inserted into pET21a (Novagen, Madison, WI, U.S.A.). The [35S]-methionine labeled IRKI was synthesized by coupled transcription translation with reticulocyte lysate according to the manufacturer’s instructions (Promega, Madison, WI, U.S.A.). IRKI was incubated with GST-IRK-KD bound with glutathione Sepharose 4B beads for 30 min at room temperature. The beads were then washed with appropriate volumes of PBS and PBST (PBS + 0.5% Tween-20), and loaded onto 10% gel for SDS–PAGE.

Northern blot analysis. Total RNA was extracted using the guanidine thiocyanate method (TRIZOL Reagent, Invitrogen). Five micrograms of total RNA was loaded onto 1.4% agarose-formaldehyde gel for electrophoresis and blotted onto nylon membranes (Hybond-N, Amersham). P-labeled probes were then synthesized using a Strip-EZ DNA probe synthesis kit (Ambion, Austin, TX, U.S.A.). The IRKI cDNA fragment digested with NcoI at the initiation codon and with SalI at approximately 470 bp was used as a probe. The 235 bp DNA fragment of histone H4 (At5g59690) was amplified from genomic DNA by PCR with the following primers: 5’-AGGTTTCTGAGAGCAACAATCGAAAG-3’ and 5’-GTAAAGAGTCCTCTCTGTCTTG-3’.

Histochemical GUS assays. A 2.7 kb long DNA fragment from the 5’ upstream region of the IRKI coding sequence was amplified from genomic DNA by PCR with the following primers: 5’-CGGGATCCTCAAGGTGTGGGTGGACAT-3’ and 5’-TCCCCC-GGGGAAGAGCCATGAGAA-3’, and inserted
into the pBI101 GUS expression vector (Clontech) for translational fusion with the GUS gene (pIRKI::GUS). Plant transformation, GUS staining, and histochemical analysis were performed as described previously.\textsuperscript{23,25,26}

IRKI expression and IRKI subcellular localization analysis with the GFP reporter. A 5.4 kb genomic sequence including the IRKI coding sequence, an intron, the 55 upstream region, and the 35 downstream region was amplified from genomic DNA by PCR with the following primers: 5'-GGATCCAGACATAGTTT-GCTGTCATCTT-3' and 5'-GGATCCAAAAGGCCGA-ACAGTTCAAGA-3', both containing a BamHI site, and inserted into the pBC vector (Stratagene, La Jolla, CA, U.S.A.). The GFP gene was amplified by PCR from a 3SSG-sGFP-S65T vector\textsuperscript{27} with the following primers: 5'-ATCCCTCAACAATTTAC-3' and 5'-AAGC-CATGACTATGACCTGTCATG-3', the latter of which contains a NcoI site. The digested fragment was inserted into a NcoI site at the IRKI initiation codon, and the full-length IRKI gene containing the GFP insertion was digested with BamHI and inserted into a pBIN19 transformation vector (pIRKI::GFP-IRKI). This construct was transformed into Arabidopsis. The pIRKI::GFP-IRKI plants were grown on MS plates without sucrose for 3 d after germination and used for analysis of IRKI expression in normal roots. To examine the effects of auxin, transformants were grown in the same way for 3 d, then transplanted and grown on MS plates containing 2,4-D or N1-naphthylphthalamic acid (NPA) for 4–7 d. Tissues were stained with propidium iodide (PI, 10 μg/ml) and observed with a confocal laser scanning microscope (LSM 510, Zeiss, Oberkochen, Germany) through an FITC and rhodamine channel for GFP and PI fluorescences respectively.

Results

Screening for the cytoplasmic protein interacting with the IRK kinase domain

To identify the protein interacting with the IRK kinase domain (IRK-KD), yeast two-hybrid screening was performed. Six identical clones from 8.5 × 10\textsuperscript{6} clones were identified using IRK-KD as the bait. The clones grew on plates lacking histidine and showed lacZ activity (Table 1). The IRK-Interacting protein (IRKI) gene was designated, and using the cDNA insert as a probe, full-length IRKI cDNA was isolated from a cDNA library generated from Arabidopsis inflorescence tissues.

Sequence analysis indicated that the IRKI gene corresponding to At5g12900 was 1,689 bp with one intron. The calculated molecular mass of the deduced protein was 63.3 kDa. Although a homology search program, TAIR blast (http://arabidopsis.org/Blast), showed that the limited region of IRKI has weak homology with a kinesin-like protein containing motor domain, IRKI is a novel protein. The BLAST search (http://blast.genome.ad.jp/) showed the existence of 7 IRKI-related proteins, all of which were conserved in the C terminus in Arabidopsis (Fig. 1A and B). The orthologs for IRKI were found in other plants, including potato and soybean (Fig. 1A).

The IRKI protein is specifically bound to but not phosphorylated by the IRK kinase domain in vitro

To confirm the interaction between IRKI and IRK-KD, an in vitro binding assay was performed. The cDNA fragment obtained by yeast two-hybrid screening was subcloned and used to synthesize a 35S-labeled IRKI protein (35S-IRKI) using an in vitro transcription translation system with the reticulocyte lysate. The predicted molecular mass of 35S-IRKI was 48 kDa, and the 35S-IRKI signal bound to IRK-KD was detected (Fig. 2, lane 2). Since the 35S-IRKI protein was not bound with GST (Fig. 2, lane 3), IRKI was shown to bind specifically to IRK-KD in vitro.

The autophosphorylation activity of IRK-KD has previously been reported,\textsuperscript{23} hence, the IRK-dependent phosphorylation of IRKI was analyzed to determine if IRKI is an IRK-KD substrate. Full-length IRKI protein was applied to an in vitro phosphorylation assay, but IRK-KD-dependent IRKI phosphorylation was not observed (data not shown).

IRKI expression overlaps with IRK expression

IRKI gene expression was analyzed by northern blotting (Fig. 3) and compared to that of IRK.\textsuperscript{23} Although IRKI expression was detected in all preparations, it was very strong in the shoot apex during the reproductive phase, and relatively strong in the upper part of the stems, flowers, and basal parts of the roots. On the other hand, IRKI expression was very weak in the basal parts of the stems. Since IRK expression was relatively high in the inflorescence apices, open flowers, and roots, and low in the rosette leaves and stems,\textsuperscript{23} it was concluded that the expression patterns of the two genes were similar.

To examine the IRKI expression pattern in detail, we analyzed transgenic plants in which the β-glucuronidase (GUS) gene was expressed under the control of IRKI promoter (pIRKI::GUS line). GUS expression was observed in the leaf primordia and shoot apices of the seedlings (Fig. 4A), and in the root stele (Fig. 4B), leaf vein (Fig. 4C), floral organs such as the stamen

Table 1. Quantitative β-Galactosidase Assay with Yeast Cells Containing IRKI and IRK-KD

<table>
<thead>
<tr>
<th>GAL4BD fusion</th>
<th>GAL4AD fusion</th>
<th>β-gal. activity (MU)\textsuperscript{a}</th>
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<tr>
<td>IRK-KD</td>
<td>IRKI</td>
<td>2.04 ± 0.30</td>
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<tr>
<td></td>
<td>SV40</td>
<td>0.15 ± 0.01</td>
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<tr>
<td>p53</td>
<td>SV40</td>
<td>0.68 ± 0.13</td>
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\textsuperscript{a}As a negative or a positive control, yeast transformants containing only IRKI or company supplied p53 and SV40 in Match-maker two-hybrid system were used. *β-gal. activity is in Miller Units.\textsuperscript{40}
filaments, styles and basal part of the pistils (Fig. 4D), and inflorescence meristems of the adult plants (Fig. 4E). The expression pattern of pIRKI::GUS agreed well with the northern blot analysis results, and overlapped broadly with that of IRK analyzed by transgenic plants carrying the GUS gene fused to the IRK promoter (pIRK::GUS line). This observation supports an interaction between IRKI and IRK in planta.

The IRKI expression in the leaf primordia and shoot apices (Fig. 4A and E) in which cells are actively dividing, was similar to that of several genes such as mitotic cyclin and cyclin dependent protein kinase, which are involved in proliferation and the cell cycle.

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**Fig. 1. Sequence Analysis of the IRKI Protein.**

(A) Deduced amino acid sequences of the IRKI protein (GenBank: AY140302) and At1g2330 (GenBank: AC025416) in *Arabidopsis* were aligned with an IRKI homolog in *Oryza sativa* (GenBank: AP005254) and the ESTs of other plants (in the EST database of TIGR (TIGR Gene Indices)). The accession numbers of the ESTs are as follows: tomato, EST649130; potato, EST615669; lotus, Ljirnpest03-015-a5; soybean, GM700001A20G9. Numbers show the amino acid number of IRKI. Amino acid sequences identical to those of IRKI are shown in bold letters. Bars show the region homologous with Kinesin-like protein. Asterisks indicate invariant amino acids. (B) Phylogenetic tree composed of full-length IRKI protein and related proteins in *Arabidopsis* and the IRKI homolog in *Oryza sativa*. GenBank accession numbers are as follows: At5g58960, AK118548; At2g45260, AY091132; At4g34080, BT011956; At5g14870, BT004334; At1g33380, BT012067; At3g60680, AK117228.

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**Fig. 2. In Vitro Pull Down Assay for IRKI Protein with IRK-KD.**

A ³⁵S-labeled IRKI protein was synthesized in vitro by coupled transcription translation and incubated with GST or a GST-IRK-KD protein carried by glutathione Sepharose beads. The beads were washed and applied to a gel for SDS–PAGE. Lane 1, direct input of RI-labeled IRKI; lane 2, recovered sample including the GST-IRK-KD protein incubated with RI-labeled IRKI; lane 3, recovered sample including GST incubated with RI-labeled IRKI.
cycle. IRKI expression in an actively proliferating MM2d cell suspension culture suitable for cell cycle studies was therefore examined. In the MM2d cells 4 d after subculturing, up-regulation of histone H4 mRNA was detected (Fig. 5, lane 1). This observation indicates that the MM2d cells used in this study were actively proliferating, but IRKI mRNA was not detected under the same conditions. These results suggest that IRKI is not just a component of common cell proliferation, but that IRKI functions in spatially organized proliferating tissues.

IRKI protein localization in root meristems monitored by GFP

A link between cell proliferation and IRKI expression was expected in plants but not in cultured cells. To understand the details of this expression profile, transgenic plants were produced that express the GFP-IRKI fusion protein under the control of the IRKI own promoter (pIRKI::GFP-IRKI line). Although the localization of GFP fusion proteins can be monitored in RMs and SAMs, GFP-IRKI expression was extensively analyzed in RMs, which are suitable for microscopic observations. GFP fluorescence was observed at the planes in between the following groups of cells: quiescent center (QC) cells, QC cells and the initial cells of the cortex and endodermis (CEI), CEI and endodermis cells, CEI and cortex cells, QC cells and stele initial cells, and immature stele cells (Fig. 6). Weak PI staining was seen in the cells next to the CEI, indicating that these cells had yet fully to divide into endodermis and cortex cells (Fig. 6, left panel, arrow in

Fig. 3. IRKI mRNA Expression during Plant Development.
Two-, nine-, and thirty-five-d-old plants were dissected, and total RNA was extracted from each. Upper lane, signals detected by the IRKI probe; bottom lane, EtBr staining of rRNA.

Fig. 4. Histochemical Analysis of the pIRKI::GUS Line.
(A) Shoot apex of a 5-d-old seedling. The arrowhead indicates a shoot apical meristem and the arrows indicate leaf primordia. (B) Root cross section of a 2-d-old seedling. (C) Aerial view of a 17-d-old plant. (D) Developing flower from stage 10 to 15. (E) Shoot apex of a 22-d-old plant. The arrowhead indicates an inflorescence meristem and the arrows indicate floral primordia. (A), (B), and (E): dark field where the GUS stain is observed as red or white spots (bars = 20 μm); (C) (bar = 10 mm) and (D) (bar = 0.5 mm): bright field.

Fig. 5. IRKI Expression Analysis in an MM2d Cell Suspension Culture.
Total RNAs extracted 4 and 9 d after subculturing of the MM2d cells were analyzed (lanes 1 and 2 respectively). As a positive control, total RNA extracted from the basal part of the roots, as described in Fig. 3, was loaded into lane 3. histone H4 expression shows the proliferating state of the cells and tissue. Bottom lane, EtBr staining of rRNA.
inset), but relatively strong GFP fluorescence was observed in the corresponding positions (Fig. 6, middle and right panels, arrowhead in inset). GFP fluorescence was not observed in the endodermis, cortex, or root cap.

IRKI expression was affected by auxin or auxin transport inhibitor applications

IRKI and IRK were expressed in the SAMs, floral organs, vascular tissues, and RMs. Similar expression patterns have been reported in the auxin-related gene ARGOS,30) CycA2;1,28) and TIR1.31) Phytohormone auxin plays an indispensable role in plant growth and development. For example, RM organization is maintained by the auxin sink below the QC cells.32) Application of excess amounts of auxin or auxin transport inhibitor inhibits normal development and induces morphological changes, and hence the relationship between auxin and IRKI expression was examined.

When Arabidopsis plants are cultured on plates containing 0.1–1.0 μM synthetic auxin 2,4-D, abnormal lateral root formation is promoted (Fig. 7A).33,34) When the pIRKI::GFP-IRKI line was cultured with 1.0 μM 2,4-D, the IRKI protein was expressed in the fourth layer and further inside from the epidermis of abnormally proliferating lateral roots, which correspond to the stele of normal roots (Fig. 7A and B). The application of N-1-naphthylphthalamic acid (NPA) auxin polar transport inhibitor induces aberrant cell division in the root tip, resulting in the disruption of meristem organization and inhibition of root elongation.35) When the pIRKI::GFP-IRKI line was cultured in the presence of NPA, IRKI expression was down-regulated and its characteristic localization in the meristem region was lost (Fig. 7C). Considering the expression of IRKI in the stele initials of normal roots (Fig. 6), these results suggest that IRKI is expressed in root cells that possess stele identity and proliferating activity. Auxin influences IRKI expression and subcellular localization, although it is not certain whether directly.

**Discussion**

In meristems, the maintenance of stem cells and differentiation of stem cell descendants is regulated by intercellular communication. During these processes, the RLKs are expected to have essential functions.13) To characterize the RLKs expressed in the meristem and their signaling pathways, IRKI, the interacting protein of IRK, which is one of the typical RLKs,23) was screened. IRKI is a novel protein with C terminus sequences highly conserved among various plant species (Fig. 1A).

IRKI might not function as a substrate for IRK kinase, since IRK-dependent IRKI phosphorylation was not observed. It might function as a regulatory component.
The weak similarity to kinesin (Fig. 1A) might give a clue to IRKI function. The kinesin-like protein, NACK1/2, localizes in the division plane preceding cell wall formation, and is thought to interact with and recruit nucleus- and phragmoplast-localized protein kinase 1 (NPK1) to phragmoplasts.36) On the other hand, in the second division of initial cells of the cortex and endodermis (CEI), GFP-IRKI was detected in the periclinal division plane (Fig. 6 right, inset), but a PI stain was not clear (Fig. 6 left, inset), which might suggest an ongoing cell division. This observation can be explained by the localization of IRKI on the cell division plane. IRKI is possibly involved in IRK subcellular localization, since NACK1/2 recruits NPK1.

IRKI expression was detected in several tissues such as leaf primordia and shoot apices (Fig. 4A and 4E) in which cells are actively dividing. In roots, GFP-IRKI expression was observed preferentially in the stele initials that also divide frequently (Fig. 6), but IRKI expression was not detected in the MM2d cell suspension culture. This suggests that IRKI does not function as a component of common cell proliferation but is linked to the multi-cellular organization of plants.

IRKI and IRK have fairly similar expression patterns (Fig. 4) with TIR1, ARGOS, and CyaA2;1, all of which are thought to be involved in lateral organ or lateral root formation under auxin regulation. SOLITARY-ROOT (SLR) is reportedly a key regulator in auxin-regulated development. The slr mutation blocks pericycle cell division during lateral root initiation, resulting in a loss of lateral roots. Microarray analysis indicated that IRK mRNA expression increased in the wild type, but not in the slr mutants with auxin treatment (H. Fukaki, personal communication). This suggests that IRK, and probably IRKI, are located downstream of SLR and have a function during lateral root formation regulated by auxin.

The regions expressing IRK and IRKI are expanded by auxin. This expanded GFP-IRKI expression was observed in the fourth layer and inside abnormally induced lateral roots, where cells are expected to have stele identity (Fig. 7B), while GFP-IRKI expression decreased and its characteristic localization disappeared with auxin transport inhibitor, NPA (Fig. 7C). In a previous report, auxin distribution in the root tips was monitored by an auxin responsive element, DR5, which was fused to the PEH A reporter gene. Maximum PEH A expression was detected in the columella initials and first columella tier in normal roots, but shifted into the QC cells and vascular initials with NPA treatment. This shift disturbs normal cell division and meristem organization. The disappearance of GFP-IRKI localization by NPA treatment is probably caused by the disorganization of the meristems. These observations suggest a link between IRKI expression and the dividing activity and cellular identity driven by auxin.

The protein interacting with IRK was identified to uncover the mechanisms of RLK-mediated signaling. To analyze in detail when and where the interaction of IRK and IRKI occurs in vivo would be important to understand their function. Although genetic analysis were performed using an over-expressor, a tagged line, and the RNAi of IRKI, no phenotypic changes were observed (data not shown). Since functional redundancy is expected like CLV and ER, analysis of double or triple disruptant lines, or transgenic plants designed to generate some dominant negative effects, would help provide an understanding of the function of IRK and IRKI. Furthermore, these studies could help reveal the molecular mechanisms involved in the meristem development of multicellular plants.

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