

Cell-to-cell trafficking of KNAT1/Brevipedicellus transcription factor

Jin-hee Jung¹, Raju Datla⁴, Alain Joliot⁵, David Jackson³, Jae-Yean Kim^{1,2}

¹Department of Molecular Biology, Division of Applied Life Science, Plant Molecular Biology & Biotechnology Research Center, ²Environmental Biotechnology National Core Research Center², Gyeongsang National University, Jinju 660-701, Korea.

³Cold Spring Harbor Laboratory, 1 bungtown Rd, Cold Spring Harbor NY11724, USA, ⁴Plant Biotechnology Institute, National Research Council of Canada, 110 Gymnasium Place, Saskatoon, SK, Canada S7N 0W9. ⁵Biologie cellulaire des homéoprotéines, CNRS UMR8542 Ecole Normale Supérieure 46 rue d'Ulm F-75005 Paris, France.

ABSTRACT

Brevipedicellus (bp) mutants provide a wonderful system to study the control of plant architecture, especially related with the patterning of stems. In *Arabidopsis*, *bp* mutations result in plants with shorter internodes and pedicel lengths, downward pointing siliques, a compact inflorescence architecture, bends at nodes and an epidermal stripe of disorganized cells along the stem. BP encodes homeodomain protein KNAT1, a member of the KNOTTED1-related HOMEODOMAIN (KNOX) family, which is the strongest *Arabidopsis* homolog of maize KN1. KNOX family play important roles in plant development by regulating cell division and differentiation. KN1 functions non-cell autonomously by cell-to-cell trafficking. Recently, it was also reported that KNAT1-GFP can traffic in the shoot apical meristem when expressed in the L1 layer. Intercellular or inter-tissue communication may be required for the establishment of plant architecture. Thus we intended to investigate the significance of intercellular KNAT1 trafficking associated with its biological function. KNAT1 specifically expressed in the cortical cell layers of the inflorescence stem (peduncle) and pedicel, but its knock-out alleles also displayed disruption in epidermal cell differentiation. This suggests that

KNAT1 functions non-cell autonomously either by its direct cell-to-cell trafficking or the movement of other downstream factors. To dissect this mechanism, we performed complementation assay of *bp* mutant phenotypes (height, pedicel angle and size, epidermal cell pattern) using various KNAT1 fusions under the control of endogenous KNAT1 promoter. Here we report the preliminary results obtained from these analyses.