

CYTOKININ-MEDIATED LEAF LONGEVITY  
CONTROL BY PHOSPHORELAY OF AHK3 TO ARR2 IN  
*ARABIDOPSIS*

Hyo Jung Kim<sup>1</sup>, Hojin Ryu<sup>3</sup>, Sung Hyun Hong<sup>1</sup>, Hye Ryun Wool<sup>1,\*</sup>, Pyung Ok Lim<sup>1</sup>, In Chul Lee<sup>1</sup>, Jen Sheen<sup>2</sup>, Hong Gil Nam<sup>1</sup>, and Ildoo Hwang<sup>3</sup>

<sup>1</sup>National Core Research Center for Systems Bio-Dynamics and Division of Molecular and Life Sciences, Pohang University of Science and Technology, Pohang 790-784, Korea

<sup>2</sup>Department of Molecular Biology, Massachusetts General Hospital and Department of Genetics, Harvard Medical School, Boston, MA 02114, U.S.A.

<sup>3</sup>Division of Molecular and Life Sciences, Pohang University of Science and Technology, Pohang, 790-784, Korea.

The longevity of plant organs is limited by senescence. Cytokinins have long been known as anti-senescence plant hormones. However, molecular mechanisms underlying cytokinin-mediated leaf longevity control are largely unknown. Here we identified an *Arabidopsis* mutant, *ore12-1*, that has increased leaf longevity due to a gain-of-function mutation in AHK3, a histidine kinase cytokinin receptor. A loss-of-function mutant had a reduced sensitivity to cytokinin in delaying leaf senescence. We further found that, among three *Arabidopsis* cytokinin receptors, AHK3 plays a major role in leaf longevity control. In mature leaf cells, AHK3 led phosphorylation on a conserved Asp residue of ARR2, a response regulator, in a cytokinin-dependent manner. Furthermore, wild type but not an unphosphorylatable mutant ARR2 led to increased leaf longevity in transgenic plants. Thus, the phosphorelay cascade from AHK3 to ARR2 positively controls cytokinin-mediated leaf longevity.