[GM17]

Combined Analysis of the Chloroplast Genome and Transcriptome of the Antarctic Vascular Plant *Deschampsia antarctica* Desv

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Antarctic hairgrass (*Deschampsia antarctica* Desv.) is the only natural grass species in the maritime Antarctic. It has been researched as an important ecological marker and as an extremophile plant for studies on stress tolerance. Despite its importance, little genomic information is available for *D. antarctica*. Here, we report the complete chloroplast genome, transcriptome profiles of the coding/noncoding genes, and the posttranscriptional processing by RNA editing in the chloroplast system

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[GM18]

Molecular regulatory mechanism of BOTRYTIS-INUDCED KINASE1 for antagonistic regulation of the key transcription factor ETHYLENE INSENSITIVE3 in ethylene signaling

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Plant hormone ethylene (ET) affects many aspects of plant physiological and morphological responses to environmental stresses. ET signaling also mediates defense responses to necrotrophic pathogens such as *Botrytis cineria* an *A. brassicicola*. *Arabidopsis* BOTRYTIS-INDUCED KINASE1 (BIK1) is induced upon fungal infections and acts as a crucial player in the immune response pathway for necrotrophic pathogen defense. Surprisingly, *bik1* accumulates EIN3 proteins to an elevated level that is an essential process in ET singling, but the mutant confers ethylene insensitivity in triple response. To unequivocally elucidate BIK1 functions on EIN3 and ET-mediated immunity, we have utilized an integrative approach combining a transient cell-based assay using *Arabidopsis* leaf mesophyll protoplasts as well as a stable genetic analysis using transgenic plants. BIK1 targeted multiple subcellular locations including the plasmamembrane, the cytosol, and the nucleus. A conserved N-terminal myristoylation site of BIK1 plays an important role for its plasmamembrane localization, since BIK1 variants either deleting Met1-Ser7 (ΔBIK1) or point-mutating the 2nd Gly to Ser could target subcellular locations other than plasmamembrane in plant cells. BIK1 negatively regulated an EIN3-binding-site-dependent luciferase reporter (EBS-LUC) activity. Interestingly, both ΔBIK1 and BIK1G2S could not regulate the reporter activity. Whereas BIK1K105A that harbors a point mutation at ATP-binding site for protein kinase activity still maintained its WT activity on EBS-LUC reporter, implicating the plamamembrane-localization of BIK1, but not its kinase activity, is important in the BIK1-mdeiated suppression of EIN3-dependent gene expression. Even so, the phosphorylation of BIK1 is still required as BIK1S33A harboring a point mutation on an autophosphorylation site only partially suppressed EBS-LUC reporter activity. To examine the molecular nature of EIN3-dependent gene suppression by BIK1, a translational fusion construct of EIN3 and firefly luciferase was designed and constructed. BIK1 could down-regulate the EIN3-fLUC activity suggesting a mechanism that BIK1 activity appears to modulate EIN3 protein stability. Since BIK1 suppressed EBS-LUC activity in *ein2*, this BIK1-mediated process would be downstream EIN2 or independent of ET signaling. Currently, genetic analysis is undertaken to verify our cell-based functional mode of BIK1.

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