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We have isolated an *am118* mutant showing *phyB* mutant phenotype by activation tagging mutagenesis of winter annual strain of Arabidopsis (*FRI-Col*). T1 generation of *am118* (*FRI-Col*) showed interesting phenotype, such as elongated hypocotyl and petiole, serrated rosette leaves, pale green leaves and early flowering time. T2 progenies showed 2.6:1 segregation ratio indicating that *am118* (*FRI-Col*) is dominant. T2 progenies of *am118* (*FRI-Col*) showed cosegregation of basta resistance and mutant phenotype. For genetic and molecular analyses of mutants regarding phytochrome signaling, we introduced the *am118* (*FRI-Col*) to wild type *Columbia* (*Col*). The mutant *am118* in *Col* showed same phenotypes with *am118* (*FRI-Col*). The *am118* (*Col*) mutant was named as *dhy1* (*dominant long hypocotyl 1*). Because *dhy1* mutant showed similar morphology to *phyB* mutant, we checked if *dhy1* has a defect in *PHYB* gene by DNA gel blot analysis and protein expression. We verified that *dhy1* mutant was not caused by the mutation of *PHYB* gene. DNA gel blot analysis confirmed that there was several copies of T-DNA insert in *dhy1* mutant. The plant DNA flanking the left border of the T-DNA insertion site was isolated by plasmid rescue and used DNA gel blot analysis and RT-PCR. The result of DNA gel blot analysis showed polymorphism between *dhy1* and wild type (*Col*) but the inserted 35S enhancer did not cause overexpression of nearby genes. Genetic and molecular analyses are in progress to gain insights into the function of *DHY1* gene.

F830 Characterization of Activation T-DNA tagging root mutants in Arabidopsis thaliana

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Root is very important in plant survival and productivity. While it is known the importance of studies on root morphology and development, little is known about the principle and mechanism of root development. In this study, we have applied the activation T-DNA tagging strategies. Activation-Tagging vectors that confer resistance to the antibiotic hygromycin have generated several transformed plants exhibiting abnormal root morphology. Some lines were identified on the basis of the short roots and aberrant lateral root formation. These lines represented the inhibition of root growth under hygromycin or cefotaxime sodium, even though they had no effect on the growth without antibiotics. As this response may provide a mechanism for roots to toxic, further studies are aimed at isolation of genes of these mutants.

F831 The First Intron of Petunia Actin-Depolymerizing Factor Gene Enhances GUS Expression in Transgenic Arabidopsis

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Two genomic clones of petunia actin-depolymerizing factor, *PhADF1* and *PhADF2*, which regulate cellular actin dynamics, were isolated and analyzed. It was revealed that the first intron of *PhADF1*(1i1) increases GUS activity, and can induce GUS expression in roots as shown in *PhADF2::GUS* with the intron. To elucidate how the intron enhances GUS expression, transgenic *Arabidopsis* lines harboring constructs with various modifications were generated. It seems that splicing event may play an important role more than sequence element based on results of GUS staining pattern in

transgenic *Arabidopsis*. This suggests that 1il might enhance GUS expression by Intron-Mediated Enhancement(IME). Further analysis will give more informations on regulation of *PhADF* genes.

F832 Genetic Polymorphism of Mitochondrial DNA in Jeju Native Horses Inferred from PCR-RFLP

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We analyzed the mitochondrial DNA in the three populations of Jeju native horses using PCR-RFLP. The partial region, about 2 kilo base pairs, including two mitochondrial polypeptide genes (NADH dehydrogenase subunit 6 gene; *ND6* and cytochrome B gene; *cyt*B) and two mitochondrial tRNA genes (tRNA-Glu gene and tRNA-Thr gene), was amplified by PCR. The RFLP analyses were performed with 10 kinds of restriction enzymes. We found polymorphisms in these digested with four of the 10 enzymes, *Bam*H?, *Hin*f?, *Msp*? and *Rsa*?. Three morph types were detected in those digested with *Msp*? and *Rsa*?, respectively; two morph types with *Bam*H? and *Hin*f?, respectively. They were classified into twelve types, and their frequencies were different among three populations. Also, the patterns of the heteroplasmic digestion were found in some animals. These results showed that mtDNAs, which are maternally inherited, of the Jeju native horses were highly polymorphic. This suggests that hybridization or/and introgression among the populations of the east Asian native horses occurred in the past. These results can be a useful parameter to verify the maternal lineages of the Jeju native horses.

F833 Identification and Characterization of Genes Overexpressed by Hypoxia in Human Synovial Fibroblasts of Rheumatoid Arthritis

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Rheumatoid arthritis (RA) is an immunologically mediated disease characterized by chronic articular inflammation that leads to the destruction of cartilage and bone in the affected joint. The rheumatoid synovium is known to be in hypoxic environment which may result in diverse cellular responses in rheumatoid synovium, especially in synovial fibroblasts which play a central role in the pathogenesis of RA. For this reason, screening of genes overexpressed in RA synovial fibroblasts under hypoxia was performed by suppression subtractive hybridization and differential hybridization with mRNAs extracted from primary cultured human RA or osteoarthritis synovial fibroblasts incubated under hypoxic or normoxic conditions. The procedure resulted in the selection of 38 clones overexpressed in RA synovial fibroblasts and 37 clones overexpressed in hypoxic RA synovial fibroblasts. The selected clones have genes identified as transglutaminase 2, reticulocalbin 1, proteasome 26S unit (n=2), matrin 3, kinesin family member 5B, mitochondrial cytochrome C oxidase subunit 2 (n=2), laminin receptor 1, BPTF, ILF2, BRF1, EF-1 (n=2), ferritin heavy polypeptide (n=3), acid ceramidase, annexin A1, cyclin C, endophilin B1, RAB11A (RAS oncogene family), complement 3 and CTCL tumor antigen se33-1. The present study suggests that hypoxia might influence to the pathogenesis of RA by regulating the expression of various genes in rheumatoid synovial fibroblasts.