SL803 Genetic Dissection of Autonomous Flowering Pathway in Arabidopsis

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Flowering time is regulated by complex genetic networks monitoring environmental conditions and developmental state of plants. To elucidate the molecular mechanism of flowering in Arabidopsis, we adopted two opposite mutagenesis strategies, gain-of-function mutagenesis and loss-of-function mutagenesis. As a wild type, the very late-flowering Arabidopsis plants (*FRI FLC*, the late-flowering behavior is mainly conferred by the interaction of two genes, *FRIGIDA (FRI)* and *FLOWERING LOCUS C (FLC)*) is used. We have obtained two gain-of-function mutants by activation tagging mutagenesis and 40 loss-of-function mutants by fast neutron bombardment.

From activation tagging, we obtained two early-flowering mutants, *fsu1* and *fsu2* (*fsu* stands for *FRI suppressor*). *FSU1* encodes a MADS domain protein, *AGAMOUS-LIKE20 (AGL20)* and plays as a floral activator. *FSU1* integrates both environmental and developmental signals for flowering. *FSU2* locus has two closely related MADS domain genes. The molecular analysis of the fast neutron bombarded early-flowering mutants showed that some of the genes are acting between *FRI* and *FLC* but some of them are acting between *FLC* and *AGL20* in genetic hierachical pathway. The complementation test of the mutants and the cloning of the genes are in progress.

SL804 Two auxin-inducible ACC synthases are differentially regulated during de-etiolation in mung bean.

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It is known that auxin stimulates ethylene production in vegetative tissues by de novo synthesis of ACC synthase, a rate-limiting step in ethylene production. In mung bean, at least seven ACC synthase genes exist and two of them, VR-ACS6 and VR-ACS7, are rapidly induced by IAA in etiolated hypocotyls. By contrast, in green plant tissues only the VR-ACS6 gene expression was induced by auxin. To investigate the detail mechanism of the auxin-regulated VR-ACS6 and VR-ACS7 transcription in light grown plants, the auxin inducibility and ethylene production during de-etiolation of seedlings were monitored. Light transfer of 3-day-old etiolated seedlings caused time dependent decrease in auxin-induced ethylene production and the level of VR-ACS6/7 mRNAs. After 24 hours after transfer, the auxin-induced ethylene production was restored to the initial level. Accordingly, the auxin inducibility of VR-ACS6 was recovered. However, repression of the auxin-induced VR-ACS7 gene expression was not recovered, suggesting that light continuously suppresses the auxin-inducible expression of VR-ACS7. Nuclear run-on analysis showed that the repression of auxin-induced VR-ACS7 expression occurred at the transcriptional level. The repression of transcripts was not fully recovered by the exogenously treated auxin. This data indicates that not the endogenous level of auxin but the sensitivity of the VR-ACS7 gene to auxin is altered in the process of de-etiolation. To localize the cell type specific VR-ACS6/7 transcripts in mung bean hypocotyls, in situ hybridization was

performed. These results and the possible physiological role of the ethylene that is produced via VR-ACS6 and VR-ACS7, respectively, in mung bean seedlings will be discussed.

SL805 Primary Genetic Form of Fish Malodor Syndrome

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Trimethylaminuria (TMAU), also known as the Fish-Odour Syndrome (FOS) is an inborn error of hepatic metabolism whereby environmental factors such as dietary exposures contribute to expression of the disorder by stressing the defective system, deficient flavin-containing monooxygenase (FMO)-mediated N-oxygenation of trimethylamine (TMA). This disorder is characterized by excretion of large amounts of the foul smelling (rotten fish) TMA in sweat, breath and urine. Individuals with this syndrome frequently are associated with rare genetic polymorphisms of a hepatic isoform (*FMO3*) gene such as E305X and P153L.

The primary genetic form of the TMAU (or FOS) is the best understood of the various forms of the disorders. FMO3 of five distinct members of the human FMO isoform family is responsible for the hepatic metabolism of nitrogen-, sulfur-, and phosphorus-containing compounds included in many clinically useful drugs, plant alkaloids and endogenous chemicals. Thus, the FMO3 isoform is most abundant in human liver and appears to be most closely involved in the N-oxidation of TMA. Many studies have shown the human FMO3 to be highly polymorphic and some of the mutations, either alone, or in combination are associated with dysfunctional enzyme activity and the metabolic disorder whereas some mutations appear to be benign.

However, the substantial differences in FMO3-dependent hepatic drug metabolism capacity observed within or among different populations couldnt all be attributed to any one of these rare alleles. Rather, more common mutations producing the altered, but functional FMO3 must be responsible and as such, are of considerable interest.

In our previous studies, we identified two common FMO variants (Glu158Lys and Glu308Gly) that do not inactivate the enzyme and that occur at a relatively high frequency within Korean population (Park et al., 1999; Kang et al., 2000 in *Pharmacogenetics*). These alleles can be regarded as functional variants (a mild form of TMAU), but only among individuals who are homozygous or heterozygous with both mutations in linkage. We have also reported the presence of a large ethnic difference in the frequency distribution of two common FMO3 variant alleles (Park et al., 2001, in *Phamacogenetics*). For these rare and common mutations, thus, because FMO3 plays an important role both in the quality and quantity of drug oxidation carried out by human liver, its genetic and functional polymorphism may have far-reaching implications for individualized therapy of drugs oxidized by FMO.

In addition, during early childhood a transient or mild form of TMAU may occur. Molecular analysis in these children sometimes shows compound heterozygosity for severe mutations and polymorphic amino acid variants of the FMO3 gene.

Therefore, TMAU is an autosomal recessive disorder caused by deficiency of the FMO3 such as null alleles and causative mutations inactivate enzyme activity.