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.