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Enzyme electrophoresis was used to estimate genetic diversity and population structure of maize, *Zea mays* L.(Graminales) in Korea. In thirteen populations, fourteen of the 24 loci (58.3%) showed detectable polymorphism. Genetic diversity (0.130) was lower than average values for species with similar life history traits. The recent cultivated populations were found to have fewer alleles per locus (1.39 vs. 1.61), fewer alleles per polymorphic locus (2.28 vs. 2.41), lower percent polymorphic locus (23.4% vs. 35.8%), and lower diversity (0.122 vs. 0.168) than primitive cultivated populations. These genetic diversity parameters indicated that the recent cultivated populations were genetically depauperate relative to primitive cultivated populations. Analysis of fixation indices showed a substantial deficiency of heterozygotes relative to Hardy-Weinberg expectations suggesting inbreeding in maize. The G_{ST} value of 13 cultivated populations was 0.151. Nearly 85% of the total the genetic diversity in *Zea mays* was apportioned within populations. The indirect estimate of gene flow based on mean G_{ST} was moderate (Nm = 1.41).

F811 Genetic Diversity and Population Structure of Maize, Zea mays in Both Primitive Cultivated and Cultivated Populations

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F812 Development of Expression vector using insulin-like growth factor-α & epidermal growth factor

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Many attempts have been done on various essential protein production by using transformed E. coli system. However, prokaryote system does not equipped the protein maturation mechanisms which are necessary for eukaryotic proteins. In this sense, among the eukaryotes, silkworms have two major merits in overcoming the difficulties. First, the protein maturation mechanisms are available in silkworm. Second, the silkworms have fibroin promoter known as the most powerful and effective promoter which controls the expression of fibroin, one member of silk protein. Insulin-like growth factor-I (IGF-I) and epidermal growth factor (EGF) play roles in neonatal growth, cell reproduction and cell proliferation. In this study, the production of recombinant human IGF-I and

EGF in silkworm system was designed. The strategies for gene cloning, expression vector construction, gene transfer as well as other analytical methods to confirm the gene integration and gene expression will be discussed

F813 Lack of Association between Pro-inflammatory Genotypes of the Interleukin 1(IL-1B -31 T+ and IL-1RN*2/*2) and Gastric Cancer

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Gastric cancer is one of the most common malignant diseases worldwide. Recently El-Omar *et al.*(Nature , *404:* 398-402, 2000) reported that pro-inflammatory genotypes of the interleukin 1 loci (*IL-1B* -31 T+ and *IL-1RN**2/*2) were associated with a significantly increased risk of a chronic hypochlorhydric response to *Helicobactor pylori* infection and gastric cancer, presumably by altering IL-1levels in the stomach. In the present study, we tested an association between *IL-1B* TATA promoter and *IL-1RN* intron 2 VNTR polymorphisms and gastric cancer in 102 gastric patients and 101 healthy controls. The frequencies of *IL-1B* -31C allele were 0.53 and 0.49, and T allele were 0.47 and 0.51 in cases and controls, respectively. The frequency of *IL-1B* 31/TT was decreased in cases (22.5%: 23/102) compared with controls (24.8%: 25/101), and was more frequent than in the Caucasian populations (10.7%: 46/429). When the *IL-1B* CC genotype was used as the reference group, both the CT and TT genotypes were not associated with an increased risk (OR = 0.67, 95% CI = 0.34-1.31; OR = 0.67, 95% CI = 0.31-1.48, respectively). The *IL-1RN**2 genotype was less frequent in Korean (5.4%: 11/202) than in Caucasian (26.9%: 231/858) and *IL-1RN**2 was not a risk genotype for gastric cancer (OR = 1.14, 95% CI = 0.59-2.20). In conclusion, our study did not support the results of previous investigations indicating that *IL-1B* -31T/*IL-1RN**2 polymorphisms were associated with an increased risk of gastric cancer.

F814 SUMO-1 modification of ataxin-1 is mediated by SUMO motif and enhanced by expanded polyglutamine tract

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Spinocerebellar ataxia type 1 (SCA1) is an autosomal dominant neurodegenerative disease characterized by ataxia and progressive motor deterioration. SCA1 is caused by expansion of polyglutamine tract in its gene product, ataxin-1. Using immunofluorescence microscopy, we have found that ataxin-1 is colocalized with the small ubiquitin-like modifier protein-1 (SUMO-1) in transfected HeLa cells. Interestingly, the strength of the interaction between ataxin-1 and SUMO-1 was influenced by the length of the polyglutamine tract in the ataxin-1; stronger interaction was observed in mutant ataxin-1 with longer polyglutamine tract. Yeast two hybrid experiments showed that SUMO-1 interacts with N-terminus region (a.a.1-a.a.196) including a SUMO motif, hinting that ataxin-1 is modified by SUMO-1. Taken together, therefore, our results suggest that SUMO-1 modification of ataxin-1 might be involved in SCA1 pathogenesis.