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.