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Association of the candidate polymorphisms in *ADAM33* gene with Asthma

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ADAM(a disintegrin and metalloprotease) familes are one of the subfamily of metalloproteinases. *ADAM33* gene was expressed in human lung fibroblasts and highly significant associated with BHR in asthma in linkage analysis and in asthma in Caucasian. So we performed the analysis between 5 candidate SNPs in *ADAM33* gene and asthma phenotype in Korean population.
334 asthmatics and 159 unrelated normal controls were enrolled. 5 candidate SNPs in *ADAM33* gene were genotyped according to previous report by SBE reaction method.

Results

1. One locus was not polymorphic.
2. Distrubution of all 4 SNPs were in Hardy-Weinberg equilibrium.
3. No significant differences of allelic and hyplotypic frequencies between asthma and control groups.
4. Significant associations between T1 SNP or one haplotype and Log(PC20) levels were observed. ($p = 0.03$ and $p = 0.0007$ by co-dominant model, respectively).

In conclusion, we confirm that polymorphism of ADAM 33 is a risk factor for development of BHR in asthma.

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FcΓR IIIb polymorphism and its association with clinical manifestations in Korean lupus patients.

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Backgroud:FcΓR play a prominent role in the clearance of immune complexes in systemic lupus erythematosus (SLE). Polymorphisms of FcΓR have been proposed as genetic factors that influence susceptibility to SLE. FcΓR IIIb polymorphism and its association with systemic lupus erythematosus have been studied in various populations, but the results were inconsistent. In Thais FcΓR IIIb polymorphism was associated with SLE but, in Japanese and Dutch population was not. The aim of this study was to determine the distribution of FcΓR IIIb polymorphism and its associations with clinical manifestations in Korean lupus patients.
Methods : One hundred eighty three SLE patients (17 male, 166 female) meeting 1982 ACR critetia and 300 Korean disease-free control were enrolled. Genotyping for the FcΓR IIIb NA1/NA2 was performed by PCR of genomic DNA using allele-specific primers.
Results:The frequency of FcΓR IIIb genotypes in 183 SLE patients and 300 disease-free controls was as follows:FcΓR IIIbNA1/NA1 27.9% vs 26%,-NA1/NA2 55.2% vs 51.7%,-NA2/NA2 16.9% vs 22.3%, respectively There was no significant skewing in the distribution of the three FcΓR IIIb genotypes between SLE and the controls. The gene frequencies of the FcΓR IIIb-NA1 and NA2 allele were 0.56,0.44 in the SLE and 0.52,0.48 in controls, respectively, and there were no skewing in FcΓR IIIb allele between SLE and controls. We did not find a correlation between FcΓR IIIb genotypes and the clinical manifestations of SLE.
Conclusion:This study shows that in Korean lupus patients, FcΓR IIIb polymorphism was not associated with the development of SLE and does not influence clinical manifestations and the disease course of SLE.