

**Z6 13      Molecular Phylogenetic Analysis for Enteroviruses Isolated from Korean Patients with Aseptic Meningitis between 1993 and 1998**

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To analyse the genetic relationship of twelve enteroviruses isolated in Korea, RT-PCR and direct sequencing methods were employed to compare the partial nucleotide sequences in the 5' non-coding region (5'-NCR) of enteroviral isolates. While homologies between two of coxsackievirus group B (CVB) isolates and between two of echovirus (EV) isolates were 83.2% and 85.0% on average, respectively, homology between two randomly selected from isolates of CVB and EV was 84.5% on average. These results demonstrate that all of Korean isolates of CVBs and EVs are closely related to each other irrespective of their serotypes. Moreover, the phylogenetic analysis based on the nucleotide sequence of the 5'-NCR also demonstrate that 7 out of 9 Korean isolates of different enteroviral strains were closely related to each other and could be grouped into B cluster among four different clusters (A-D). Taken together, most but not all enteroviruses that have been isolated in Korea for past 6 years were grouped into the cluster B in respect of genotype based on 5'-NCR sequences, irrespective of the year of isolation and the different of serotypes. [Supported from MOHW, No. HMP-96-D-6-1054]

**Z6 14      Allelic Loss on 7q11.23 in Patients with Supravalvular Aortic Stenosis**

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Supravalvular aortic stenosis (SVAS) is an inherited vascular disease that can cause heart failure and death. SVAS can be inherited as an autosomal dominant trait or as an part of developmental disorder, Williams Syndrome (WS). Previous molecular genetic data have led to the hypothesis that SVAS results from mutations in the elastin gene (ELN). We carried out hemizygotic deletion analysis by FISH using a bacterial artificial chromosome clone 244H3 as a probe, which has the genomic DNA sequence of ELN. FISH showed deletion in one, partial deletion in another and no deletion in the other two patients. We have previously reported that two SVAS patients with no deletion have insertion of CA in codon 297 exon 16 of ELN producing a frame shift to cause premature termination 26 codons downstream. In this study, for deletion mapping, polymorphism analysis of two SVAS patients with ELN deletion and their parents was done with 5 dinucleotide repeat sequence polymorphic markers. The result showed that alleles at loci, ELN and D7S2472 were deleted in the SVAS patients. How much the size of deletion influences the different pathogenesis between WS and SVAS will be presented. [Supported from MOHW, No. HMP-98-M-1-0010]