

## Apo E Polymorphism Influence on Plasma Lipid Profiles and LCAT & CETP activities in Normolipidemic Women.

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Apo E polymorphism (e2, e3, e4) was among the first reported genetic polymorphism that explained part of the normal variation in plasma lipid profiles. Both alleles E<sub>2</sub> and E<sub>4</sub> are significantly more frequent in the hyperlipidemia and contribute on the observed differences in CHD risk among different populations. Among 89 healthy females, aged 19 up to 22 years, 70 were E<sub>3/3</sub> (r.f=0.787), 9 were E<sub>3/2</sub> (r.f=0.101), 10 were E<sub>3/4</sub> (r.f=0.112) and no E<sub>2/2</sub> & E<sub>2/4</sub> were found. Weight, BMI and %LBM were elevated in E<sub>2</sub> than those in E<sub>3</sub> & E<sub>4</sub>. No differences in the blood pressure among E<sub>3</sub> isomers, otherwise the pulsation was higher in E<sub>4</sub> than that in the others. There were no differences in plasma total-, total HDL-, HDL<sub>3</sub>-, HDL<sub>2</sub> cholesterol and apo B-100 & apo A-I. However, phenotype means rank E<sub>3/2</sub> > E<sub>3/3</sub> > E<sub>3/4</sub> in average TG levels (p<0.0001) significantly, and rank E<sub>3/4</sub> > E<sub>3/3</sub> > E<sub>3/2</sub> in LDL cholesterol levels. These results were related to the correlation between atherogenic indexes (AI) such as LDL/HDL, (TG-HDL)/HDL & HDL<sub>3</sub>/HDL<sub>2</sub>. The ratio of HDL<sub>3</sub> & HDL<sub>2</sub> were significantly increased in E<sub>2</sub> and E<sub>4</sub> than that in E<sub>3</sub> (P=0.043). LCAT activity did not differ between E<sub>2</sub> and E<sub>3</sub> but were highly increased in E<sub>4</sub> (p<0.0001 among apo E isomers). CETP activity did not differ between apo E isomers. However, E<sub>2</sub> allele impacts on the clearance of apo E mediated lipoproteins because the negative correlation between LCAT and CETP in apo E<sub>2</sub> (r=-0.491) was stronger than that in apo E<sub>3</sub>. In conclusion is that firstly, E<sub>4</sub> allele mediated alteration through LDL or E receptors results in lower TG or higher  $\beta$ -lipoprotein levels and E<sub>2</sub> shows reciprocal effects of E<sub>4</sub>, respectively. Second, E<sub>4</sub> allele was more atherogenic than E<sub>2</sub> allele because the higher levels of AI were criticized. [Acknowledgements: Special thanks are given to the Korean Research Foundation for the financial support.]