



Summer Meeting, 10–12 July 2017, Improving Nutrition in Metropolitan Areas

Interaction between lipoprotein lipase and apolipoprotein E gene polymorphisms and dietary factors on lipid traits

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Altered serum lipid level is one of the risk factors for development of cardiovascular disease (CVD)⁽¹⁾. Genetic association studies have identified loci in lipid metabolism-related genes such as lipoprotein lipase (*LPL*) and apolipoprotein E (*APOE*), which have been shown to be associated with lipid traits^(2,3). However, very few studies have offered insight into how diet modifies the effect of *LPL* and *APOE* single nucleotide polymorphisms (SNPs) on lipids^(4,5). Hence, we investigated the association of common *LPL* (rs320 and rs328) and *APOE* SNPs (rs405509, rs439401, rs445925, rs405697, rs1160985, rs1064725, rs7412 and rs429358) with lipids and examined the interactions between the SNPs and dietary factors on lipids in two Caucasian populations. The populations include 664 individuals from the Prevention of Cancer by Intervention with Selenium (PRECISE) and 1,238 individuals from the Caerphilly study. Statistically significant association was detected between *APOE* haplotypes (rs7412 and rs429358) and *APOE* SNP rs445925 and total cholesterol ($P = 0.0004$ and $P = 0.003$, respectively). The carriers of *APOE* E2 allele (5.54 ± 0.97 mmol/L) had lowest total cholesterol levels compared with E3 allele (5.98 ± 1.05 mmol/L) ($P = 0.001$) and E4 allele (6.09 ± 1.06 mmol/L) ($P = 0.0002$) carriers, respectively. The association between *APOE* haplotypes and rs445925 and total cholesterol ($P = 0.000002$ and $P = 0.0003$, respectively) were replicated in the Caerphilly study, in addition to the association with LDL-C ($P = 0.0004$ and $P = 0.001$, respectively). In the Caerphilly study, interaction between fat (% energy) and rs328 at *LPL* on triacylglycerol ($P = 0.02$) and fat (% energy) and *APOE* haplotype on total cholesterol ($P = 0.03$) were observed; however, these interactions were not statistically significant after correction for multiple testing. In summary, we have shown that genetic variations at the *APOE* gene influence plasma lipid concentrations; however, the gene-diet interactions on lipids require confirmation in another larger cohort. The knowledge of combined effect of genetic and dietary factors on lipid profile could help us to a better understanding of the pathogenesis in the CVD.

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