Proceedings of the Nutrition Society (2017), **76 (OCE4)**, E136 doi:10.1017/S0029665117002981

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

I. Shatwan[1,2], B. Ellahi[3], K.H. Winther[4], Y. Ben-Shlomo[5], P.C. Elwood[6], I. Givens[7], M. Rayman[8], J.A. Lovegrove[1] and K.S. Vimaleswaran[1]

[1]*Hugh Sinclair Unit of Human Nutrition, University of Reading, Whiteknights, Reading, UK, RG6 6AP, UK,*
[2]*Food and Nutrition Department, Faculty of Home Economics, King Abdulaziz University, Jeddah, Saudi Arabia,*
[3]*Faculty of Health and Social Care, University of Chester, Chester, CH1 1SL, UK,*
[4]*EFE – Endocrine Elite Research Centre Department of Endocrinology and Metabolism Odense University Hospital Denmark,*
[5]*Department of Social Medicine, University of Bristol, Bristol, BS8 1TH, UK,*
[6]*Department of Epidemiology, Statistics and Public Health, Cardiff University, University Hospital of Wales, Heath Park, Cardiff, CF14 4XW, UK,*
[7]*Centre for Food, Nutrition and Health, University of Reading, Arley Gate, Reading RG6 6AR, UK and*
[8]*Department of Nutritional Sciences Faculty of Health and Medical Sciences, University of Surrey Guildford, GU2 7XH, UK*

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.

1. Musunuru K (2010) *Lipids* **45**, 907–14.
2. Shatwan IM *et al.* (2016) *Int J Mol Sci* **17**, 397.
3. Bennet A.M *et al.* (2007) *Jama* **298**, 1300–11.
4. Carvalho-Wells AL *et al.* (2012) *Am J Clin Nutr* **96**, 1447–53.
5. Nettleton JA *et al.* (2007) *Atherosclerosis* **194**, e131–40.