

Association of cholesteryl ester transfer protein (*TaqIB*) and apolipoprotein E (*HhaI*) gene variants with obesity

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Abstract *Background* The pathophysiology of obesity is known to be influenced by alterations in lipid levels. We aimed to evaluate association of cholesteryl ester transfer protein (CETP) and apolipoprotein (APO) E gene variants with asymptomatic obesity. *Methods* A total of 437 subjects, 159 asymptomatic obese (BMI = 29.29 ± 3.76) and 278 non-obese (BMI = 23.38 ± 1.71) individuals, were included in this case–control study. Lipid levels were estimated using standard protocols. Analysis of *CETP* (*TaqIB*) and *APOE* (*HhaI*) gene polymorphisms was done using PCR–RFLP. *Results* We found significant difference in blood pressure (systolic, $P < 0.0001$ and diastolic, $P < 0.0001$), total cholesterol ($P < 0.0001$), LDL-cholesterol ($P < 0.0001$), and HDL-cholesterol ($P < 0.0001$) in obese as compared to non-obese group. Homozygous *APO E4E4* genotype was only observed in 5.7% of obese individuals and none in non-obese group. *APO E4* allele carriers were also susceptible for obesity ($P = 0.016$, OR = 1.73; 95% CI = 1.12–2.68) than non-carriers. Higher blood pressure (Systolic, $P = 0.001$ and Diastolic, $P = 0.004$) and triglyceride levels ($P = 0.029$) were observed in obese subjects with *APO E4* allele than individuals without *APO E4*. However, *CETP B1* variant allele

carriers did not show alteration in blood pressure and lipid profile in asymptomatic obese subjects. *Conclusions* *APO E4* genotype and allele were found to be associated with asymptomatic obesity, whereas *CETP TaqIB* polymorphism showed no such association in North Indian subjects.

Keywords Metabolism · Lipoprotein · Obesity · Hypertension · Risk

Introduction

Obesity, a multifactorial metabolic disorder, has been highlighted in the latest guidelines of the National cholesterol education program (NCEP) as one of the modifiable factor to prevent morbidity and mortality [1]. Both environmental and host factors play significant roles in development of obesity. Different metabolic determinants are important in the regulation of body mass. Abdominal subcutaneous fat acts as an independent marker of metabolic disorders [2–4]. Pathophysiological sequelae of obesity include hypertension, dyslipidemia, and atherosclerosis [5, 6]. Recent studies have shown increased expression and activation of circulating vasoconstrictor enzymes in adipose tissue of obese individuals [7].

Alterations in proteins involved in lipid metabolism are considered to influence obesity phenotype. Further, the levels of these proteins are influenced by polymorphisms in genes encoding these proteins. Several gene polymorphisms in key proteins, such as apo E, apo B, and CETP, have been identified [8]. CETP facilitates the exchange of triglycerides and cholesteryl esters between lipoprotein particles. The diminished relationship between HDL-cholesterol and CETP *TaqIB* gene polymorphism was observed in obese patients [9]. Furthermore, *B1* allele of

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