MOLECULAR CYTOGENETICS

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Effect of *PPAR-γ2* gene Pro12Ala and *ADR-β3* gene Trp64AArg polymorphism on glucose homeostasis in Type 2 diabetes subjects from Western India

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Background

Several studies have shown the effect of Pro12Ala polymorphism of *PPAR-γ2* on insulin sensitivity and Trp64Arg polymorphism in *ADR-β3* gene on obesity and insulin resistance in Type 2 Diabetic (T2D) subjects. The present study was carried out to find the interaction of these two gene polymorphisms and their combined effect on glucose homeostasis (HbA1C) in T2D subjects.

Materials and methods

The present study comprises of 535 subjects (including 235 T2D & 300 controls). Genotyping was carried out for the above mentioned polymorphisms and glycosylated hemoglobin [HbA1C] levels were analyzed for each subject. All T2D subjects were divided into four groups according to their genotype. Group-I: 31 patients with Pro/Pro and Trp/Arg genotype; Group-II: 159 patients with Pro/Pro and Trp/Trp genotype; Group-III: 6 patients with Pro/Ala and Trp/Arg genotype; and Group-IV: 39 patients with Pro/Ala and Trp/Trp phenotype.

Results

It was observed that 12Ala allele frequency was nearly equal in T2D patients and controls (9.0% vs. 9.1%, p>0.05). 64Arg allele frequency was 8.3% in T2D patients and 6.7% in controls (p>0.05). The mean HbA1C level was lower in T2D patients with 12Ala allele compared to patients homozygous for 12Pro allele (7.73±1.42% vs. 8.47±1.92%, p<0.02). However, no significant difference in mean HbA1C levels was observed in T2D patients with 64Arg allele compared to patients homozygous for 64Trp allele (8.50±1.81% vs. 8.31±1.88%, p>0.05). The mean HbA1C levels were higher in Group-I (8.62±1.84%, p<0.0092), Group-II (8.47±1.94%, p<0.001) and Group-III (8.26±1.71%, p>0.05) compared to grouPIV having a mean HbA1C of 7.65±1.37%.

Conclusions

The protective effect of 12Ala allele is likely to be diminished in Group-III T2D patients in the presence of 64Arg allele. Polymorphisms of 64Arg and 12Pro alleles that is likely to play a role in controlling glucose homeostasis by gene-gene interactions.

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