

POSTER PRESENTATION

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Effect of *PPAR-γ2* Gene Pro12Ala Polymorphism (Rs1801282) and Vitamin D₃ on Glucose Homeostasis in Type 2 diabetic Subjects from Gujarat-India

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Background

Pro12Ala polymorphism in *PPAR-γ2* gene is known to be involved in insulin sensitization and metabolic deregulation in Type 2 Diabetes (T2D) subject. Considering beneficial effects of Vitamin D₃ in functional regulation of pancreatic-β cells, present population based study was designed to determine the association of Pro12Ala polymorphism with serum Vitamin D₃ level and its effects on glucose homeostasis in T2D and non-diabetic subjects.

Materials & methods

Total 508 subjects (including 210 T2D & 298 controls) were divided into two groups according to serum Vitamin D₃ level. Group-I: included 338 subjects (150 T2D out of 338) having Vitamin D₃ deficiency (≤ 25.0 nmol/l) and group-II: included 170 subjects (60 T2D out of 170) with normal vitamin D₃ level (> 25.0 nmol/l). All cases were investigated for Vitamin D₃, glycosylated hemoglobin (HbA1C) level and Pro12Ala variant of *PPAR-γ2* gene.

Results

It was observed there is 12Ala allele frequency of *PPARγ2* gene in 10.19% of T2D and 9.46% in control subjects ($p > 0.36$). The mean HbA1C was better controlled in group-II T2D subjects with 12Ala allele compared to group-I T2D subjects having same allele ($7.26 \pm 0.44\%$ vs. $8.35 \pm 0.43\%$, $p > 0.014$). In contrary to the above, patients

who were homozygous for 12Pro allele, the mean HbA1c remains high irrespective of the normal Vitamin D₃ levels ($8.59 \pm 0.18\%$ vs. $8.29 \pm 0.28\%$ in group-I and group-II respectively).

Conclusions

Significant decrease in mean HbA1C level was observed in T2D patients with 12Ala allele and normal Vitamin D₃ level compared to the patients having same allele with Vitamin D₃ deficiency. No such effect was observed in T2D patients with homozygous status for 12Pro allele. This indicate that biologically active form of Vitamin D₃ (125(OH)D₃) together with 12Ala allele may affect glucose homeostasis by some gene-nutrition interactions.

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