KSN 2019
Seoul, Korea · May 23 - 26

Abstract Type : Oral
Abstract Submission No. : OR-1548

Therapeutic effects of Dipeptidyl peptidase –IV inhibitors and antioxidant properties on kidney from Trigonella foenum for treatment of type 2 diabetes mellitus in rat; in-vivo; in-silico

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Objectives: A pioneering approach in the treatment of type 2 diabetes mellitus (T2DM), based on gut hormone which regulated by Dipeptidyl peptidase–IV (DPP-IV). As such, we hypothesized that treatment of diabetes with DPP-IV inhibitors isolated from phenolic extracts *Trigonella foenum* (TF) with different approached *in-vivo; in-silico* and kidney histology.

Methods: Effects of DPP-IV inhibitors from phenolic rich fraction of TF in high sucrose diet along with dexamethasone induced T2DM was explored *in-vivo* in rat. Apart from serum glucose; DPP-IV inhibition activity, HbA1c, Insulin and lipid profile. We also examined the antioxidant properties such as lipid peroxidation (LPO), superoxide dismutase (SOD), catalase (CAT) and glutathione (GSH) to kidney tissue along with histology. Docking of inhibitors with DPP-IV enzyme.

Results: High sucrose diet with Dexamethasone administration (1 mg /kg BW 45 days) increased concentration of serum glucose, triglyceride, cholesterol and renal tissue LPO with concomitant initial increase in tissue antioxidant to scavenging free radicals but after some time antioxidants such as SOD, CAT, GSH was decreased. However, after administration of phenolic extract of TF, (*in-vitro*) DPP-IV inhibition increase in TF (71.29%), as compared to Sitagliptin (89.46%) with significant reduction in levels of glucose, TC, TG and histology showed some significant change as compared to diabetes control. Isolated Gallic acid depicts the conformer and affinity energy was – 5.4 and distance from RMSD i.b was 38.669. Sitagliptin depicted the conformer and affinity energy was – 8.9 and distance from RMSD i.b was 3.826.

Conclusions: DPP-IV inhibitors isolated from TF are novel antidiabetic agents with protective effects on kidney in addition to their antioxidant properties. DPP-IV inhibition lower blood glucose by increasing endogenous levels of glucagon-like peptide-1, an incretin with fewer side effects.