

A novel partial agonist of PPAR γ with excellent effect on insulin resistance and type 2 diabetes

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1. Methods for expression and purification of PPAR γ -LBD

The PPAR γ -LBD (the ligand binding domain of human PPAR γ , residues 206–477) was cloned into the expression vector pET15b. *E.coli* BL21 (DE3) host cells transformed with this expression plasmid were grown in LB at 37 °C to an A₆₀₀ of 1 and induced with 0.1 mM IPTG at 16 °C [13]. The cells were harvested, resuspended in extract buffer (50 mM Tris, pH 8.0, 250 mM NaCl, and 10% glycerol), and lysed by French press. The lysate was centrifuged at 12,000 rpm for 30 min. The supernatant was loaded onto a Ni-affinity column. The column was washed with extract buffer, and the protein was eluted with wash buffer (50 mM Tris, pH 8.0, 250 mM NaCl, 10% glycerol, and 100 mM imidazole). The protein was further purified on a Superdex 200 column.

Fig. S1

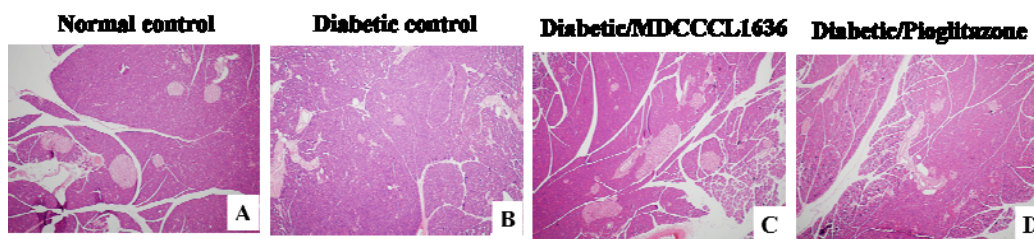


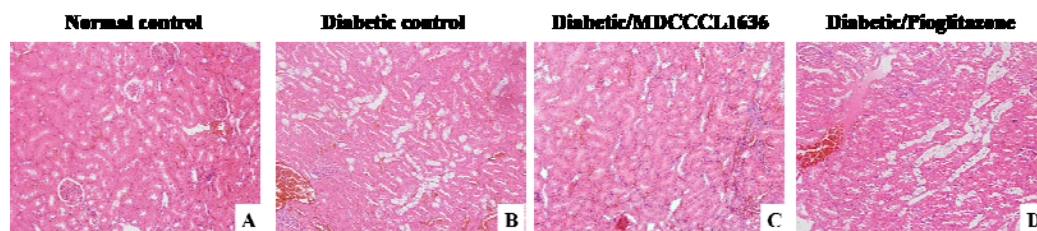
Fig.S2**Figure legends:**

Fig. S1. Histopathological changes in islets of pancreas of experimental groups. (A) Light micrograph of normal control rats showing normal β -cells in pancreatic islet (H & E, 40x), (B) HFD-STZ induced diabetic control rats showing pancreatic islet damage (H & E, 40x), (C) MDCCCL1636 at 7.5 mg/kg/day resulted in the reversal of pancreatic islet damage (H & E, 40x), (D) pioglitazone at 30 mg/kg/day resulted in the reversal of pancreatic islet damage (H & E, 40x).

Fig. S2. Histopathological changes in renal tubular epithelial cells of experimental groups. (A) Light micrograph of normal control rats showing normal renal tubular epithelial cells in pancreatic islet (H & E, 40x), (B) HFD-STZ induced diabetic control rats showing the fatty degeneration of renal tubular epithelial cells (H & E, 40x), (C) MDCCCL1636 at 7.5 mg/kg/day resulted in the reversal of the fatty degeneration of renal tubular epithelial cells (H & E, 40x), (D) pioglitazone at 30 mg/kg/day showing no effect on the fatty degeneration of renal tubular epithelial cells (H & E, 40x).