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Dipeptidyl Peptidase-IV (DPP-IV) Inhibitory Activity of Parotid Exudate of Bufo melanostictus

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ABSTRACT

Type 2 diabetes arises as a result of beta-cell failure combined with concomitant insulin resistance. Glucagon-like peptide-1 is a gastrointestinal hormone that is released postprandially from the L cells of the gut and exerts a glucose- dependent and direct insulinotropic effect on the pancreatic beta cell. Which activate adenylate cyclase and enhances insulin secretion. GLP-1 is rapidly degraded by DPP-IV to GLP-1(9-37) amide following release from gut L cells. GLP-1 directly enhances glucose-dependent insulin secretion via an increase in beta-cell cAMP. Dipeptidyl peptidase IV (DPP-IV) is a plasma membrane glycoprotein ectopeptidase. In mammals, DPP-IV was widely expressed on the surface of endothelial and epithelial cells and highest levels in humans have been reported to occur in the intestine, bone marrow and kidney. Inhibiting DPP-IV reduces its rapid degradation of GLP-1, increasing circulating levels of the active hormone in vivo and prolonging its beneficial effects. The IC50 value of parotid exudate was found to be 9.4 g/ml. The maximum % inhibition (61.8) was showed at a concentration of 12 g/ml. Parotid exudate through inhibition of DPP-IV, improves glucose tolerance and enhances insulin secretion. DPP-IV inhibitors are a novel class of oral hypoglycemic agents with a potential to improve pancreatic beta cell function and the clinical course of type 2 diabetes.

KEYWORDS

DPP-IV . GLP-1 . parotid exudate . type 2 diabetes

REFERENCES