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**Editorial**

# INCRETIN BASED DRUGS

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## EDITORIAL

Incretins are gastrointestinal polypeptide hormones that regulate pancreatic beta cell insulin production. These hormones are secreted from the upper gastrointestinal tract in response to food and include Glucagon-like Peptide-1 (GLP-1) and Gastric Inhibitory Peptide (GIP). They have an effect on the pancreas, prompting insulin to be released even before blood glucose levels rise. Both hormones are polypeptides that the enzyme dipeptidyl peptidase-4 rapidly removes from the bloodstream (DPP-4). DPP-4 inhibitors and GLP-1 analogues are the most common incretin pathway targets for type 2 diabetes therapy. These incretin-based hypoglycemic medications are very new, and they have not been linked to drug-induced liver injury as a common cause [1]. Type 2 diabetes mellitus is currently commonly treated using incretin-based medications such as glucagon-like peptide-1 receptor agonists and dipeptidyl peptidase 4 inhibitors. These drugs modulate glucose metabolism through a variety of mechanisms, are associated with low rates of hypoglycemia, and either have no effect on body weight (dipeptidyl peptidase 4 inhibitors) or stimulate weight reduction (dipeptidyl peptidase 4 inhibitors) (glucagon-like peptide-1 receptor agonists) [2].

The success of exenatide and sitagliptin, the first incretin-based medicines in their respective pharmacological classes, has sparked the creation of a slew of novel incretin-based drugs currently in late-stage clinical trials or awaiting FDA approval. Type 2 diabetes is a metabolic condition defined by hyperglycemia, which is caused by a combination of inadequate insulin production and insulin resistance. The incidence and prevalence of type 2 diabetes are gradually increasing, fueled in part by an increase in global obesity rates. There is considerable interest in the treatment of type 2 diabetes, with a focus on the development and use of new agents that exhibit improved efficacy and safety compared to currently available medicines, as longitudinal studies of type 2 diabetes provide evidence linking improved glycemic control with a reduction in the rates of diabetes-associated complications [3].

Glucagon-like Peptide-1 (GLP-1) Receptor (GLP-1R) agonists and dipeptidyl peptidase-4 inhibitors (DPP-4i), the two most recently approved groups of medicinal medicines for the treatment of type 2 diabetes, work by potentiating incretin receptor signalling. GLP-1 and Glucose-dependent Insulinotropic Peptide (GIP) are gut-derived hormones that are produced at low basal levels in the fasting state. Following food consumption, blood levels rise quickly and transiently. Degradation-resistant GLP-1R agonists have been created because natural GLP-1 has a very short circulation half-life due to renal clearance and $NH_2$-terminal degradation by the enzyme DPP-4. Exendin-4, a GLP-1R agonist that is structurally similar to the natural gut peptide, has been approved to treat type 2 diabetes. GLP-1R agonists modulate blood glucose by regulating islet function, primarily by stimulating insulin secretion while inhibiting glucagon secretion. In the absence of concomitant sulfonylurea medication, these GLP-1R–dependent activities are glucose dependent, reducing the risk of hypoglycemia. GLP-1R activation also reduces food intake and inhibits stomach emptying, resulting in weight loss in the majority of patients. In experimental models of coronary artery ischemia, the GLP-1R is expressed in cardiomyocytes and endothelial cells, and preclinical studies show that GLP-1R activation is associated with significant cardioprotection and reduced infarct size [2].

Incretin-based medicines are the first anti-hyperglycaemic drugs that address a known pathophysiological abnormality in T2D. It has various advantages over most other anti-hyperglycaemic medicines, including the fact that it is not linked to an increased risk of hypoglycemia or weight gain. The incretin idea, which opened the door to incretin-based medications as innovative anti-diabetic treatments, was first published over a century ago. GIP is a 42-amino-acid hormone produced by the upper small intestine's K cells. GIP was first discovered in porcine gut. Several challenges had to be handled in order to develop incretin-

based medications. First, DPP-4 catalyses the rapid breakdown of released incretins, which reduces the insulinotropic effects of GIP and GLP. Second, it was first discovered that GIP's insulinotropic effects are reduced in people with type 2 diabetes. GLP-1 has been identified as a major target for therapeutic development. GLP-1 secretion is lower in those with type 2 diabetes, according to research. Incretin-based medicines are commonly utilised in non-obese type 2 diabetes across East Asian countries due to their significant glucose-lowering benefits and low risk of hypoglycemia [5].

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