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PROCEEDINGS ABSTRACT

Biochemical mechanisms and clinical efficacy of glucagon-like peptide-1 receptor agonists in metabolic regulation: a comprehensive review

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The global rise in obesity prevalence has catalysed the development of advanced pharmacological interventions. Amongst these, glucagon-like peptide-1 (GLP-1) receptor agonists have emerged as the result of a critical intersection between metabolic biochemistry and clinical pharmacology. GLP-1 receptor agonists are effective in improving metabolic regulation by enhancing insulin secretion, lowering glycated haemoglobin levels, and promoting weight loss. They also reduce cardiovascular risk, improve lipid and blood pressure profiles, and carry a low risk of hypoglycaemia, making them valuable in managing type 2 diabetes mellitus and metabolic syndrome. They work by mimicking the effects of the incretin hormone GLP-1, which is released from the intestine in response to food intake, thereby enhancing glucose-dependent insulin release, suppressing glucagon release, slowing gastric emptying, and increasing satiety, leading to improved blood glucose control and weight reduction. This review explores the biochemical pathways through which these agents regulate systemic metabolism besides simple appetite suppression. A comprehensive analysis was conducted on current and previous clinical data and biochemical profiles of anti-obesity drugs (GLP-1 receptor agonists) that are designed to be delivered subcutaneously. The focus was on the signalling pathways involved and the role of these drugs in enhancing glucose-dependent insulin secretion and delaying gastric emptying. Clinical data indicate a weight reduction over long-term use of these drugs, accompanied by improved glycaemic control and reduced markers of systemic inflammation. Agents such as semaglutide, liraglutide, and tirzepatide demonstrate significant and sustained weight reduction, representing an effective, minimally invasive option for long-term obesity management. These agents are increasingly being used as primary therapies for obesity, metabolic dysfunction, and established cardiovascular diseases in addition to type 2 diabetes mellitus. Subcutaneously-administered GLP-1 receptor agonists are absorbed into the systemic circulation after injection. In the pancreas, these agents enhance glucose-dependent insulin secretion from β-cells while suppressing glucagon release from α-cells, with indirect inhibition *via* paracrine signalling (somatostatin from δ-cells) leading to improved glycaemic control and reduced hepatic glucose production. In the brain, particularly within the hypothalamus, they activate satiety pathways and reduce hunger-inducing signals, thereby significantly decreasing appetite and food intake. Additionally, these drugs influence reward-related pathways, helping to reduce cravings and hedonic eating. Within the gastrointestinal system, the GLP-1 receptor agonists modulate the release of appetite-related hormones (such as ghrelin and peptide YY)

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and delay gastric emptying, thereby prolonging the user's feeling of satiety after meals and reducing postprandial glucose levels. At the molecular level, GLP-1 receptor agonists activate cAMP-dependent signalling pathways that stimulate insulin secretion, promote β-cell survival, modulate ion channel activity, and affect central appetite-regulating neurocircuits; in essence, these agents exert metabolic and anti-obesity effects. They also have the ability to reduce pro-inflammatory cytokine levels (such as those of tumour necrosis factor-alpha and interleukin-6), thereby contributing to improved serum metabolic profiles. The most common side effects associated with GLP-1 receptor agonist use include nausea, vomiting, and injection-site reactions. Important precautions involve the risk of developing acute pancreatitis and the FDA's boxed warning against the use of these drugs by individuals with a personal or family history of medullary thyroid carcinoma and hereditary conditions associated with this type of cancer. Biochemical evidence suggests that these medications do not merely act as anorexiants, but induce significant changes in metabolic rate and adipose tissue thermogenesis. In conclusion, the integration of GLP-1 receptor agonists into obesity management represents a shift toward targeting the underlying biochemical dysfunctions of metabolic syndrome. Future research studies should focus on the long-term metabolic "reprogramming" induced by these agents and their potential in treating non-alcoholic fatty liver disease.

Keywords

biochemistry; GLP-1 receptor agonists; insulin sensitivity; metabolic pathways; obesity

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Conflicts of interest statement

None to declare.

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