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Interplay of GLP-1 and IL-6: Implications for Pancreatic and Brain Health

At Present, diabetes has no recognized treatment. The treatment of type 2 diabetic mellitus (T2DM) comprises a variety of pharmacological interventions have been licensed; some are undergoing clinical trials, and they are categorised based on how they work. insulin types, sodium-glucose cotransporter type 2 inhibitors, biguanides, thiazolidinediones, alpha-glucosidase inhibitors, meglitinides, sulfonylureas, and incretin-dependent treatments (dipeptidyl peptidase 4 inhibitors, or DPP-4, and glucagon-like peptide-1 receptor agonists, or GLP-1R). While some of the medications currently on the market are useful in the treatment of type 2 diabetes, long-term pharmacological side effects continue to pose a significant problem. Currently, GLP-1R agonists are the prescribed drugs to take in addition to oral metformin when treating type 2 diabetes is not possible. GLP-1 binds specifically to the glucagon-like peptide-1 receptor (GLP-1R) in the body, directly stimulating pancreatic β-cells to secrete insulin, boosting cell differentiation and proliferation, and preventing cell death to lower blood sugar levels. In both human and murine models, the glycaemic controlling impact of GLP-1 and its analogues has been thoroughly investigated in relation to numerous illnesses. According to recent research, GLP-1 can influence the innate immune response in a variety of inflammatory conditions. Hyperglycemic conditions can lead to DNA breaks, Fas up-regulation, and destruction of pancreatic β cells. Elevated levels of ROS, hypoxia, and TNF-α trigger IKKβ and JNK1 signaling pathways, inhibiting insulin receptor substrate and weakening insulin signal transduction. These pathways act as mediators between stress and the inflammatory response, demonstrating the interplay between inflammatory and metabolic signaling pathways. This review provides an overview of the research progress of GLP-1 and its analogs in immunomodulation and their related signal pathways.

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