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PROCEEDINGS ABSTRACT

The roles of liraglutide and visfatin in metabolic and inflammatory regulation: a review

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Liraglutide is a glucagon-like peptide-1 (GLP-1) receptor agonist that is frequently used for the management of obesity and type 2 diabetes mellitus. Liraglutide attaches to GLP-1 receptors located on pancreatic β-cells, leading to the promotion of insulin release exclusively when blood sugar levels are elevated. At the same time, it suppresses glucagon secretion from α-cells, thereby decreasing blood glucose levels. Additionally, the medication reaches the appetite regulation centers (specifically, the hypothalamus and the brainstem), enhances feelings of fullness and diminishes hunger, resulting in a reduction of energy intake. Furthermore, liraglutide delays gastric emptying, which in turn slows the absorption of nutrients, including glucose, into the bloodstream and mitigates postprandial blood sugar surges. Its impact on inflammatory pathways, adipokine modulation, and the biology of adipose tissue is given special consideration in this review. The adipokine visfatin, which is mostly generated in visceral adipose tissue, is crucial for energy balance, inflammatory signalling, and metabolic control. This review outlines the functions of both intracellular and extracellular visfatin in NAD metabolism, mitochondrial function, and systemic inflammation, and focuses on the possible relationship between liraglutide administration and circulating visfatin levels through enhanced adipose tissue function, weight loss, and the suppression of inflammatory pathways. Liraglutide-induced improvements in adipose tissue physiology may affect circulating visfatin levels because the latter is primarily released by visceral adipose tissue and is linked to metabolic stress and inflammation. Visceral adiposity reduction is one of the main pathways connecting liraglutide to visfatin. In metabolic disorders, visceral adipose tissue is considered as a key source of circulating visfatin. Liraglutide therapy suppresses appetite and modifies central satiety pathways so as to promote weight loss and to decrease the visceral fat mass. Visfatin (as well as other pro-inflammatory adipokines) may undergo decreased secretion when the adipocyte hypertrophy and the adipose tissue inflammation are reduced. Liraglutide affects inflammatory pathways in the adipose tissue in addition to body weight and adiposity. Increased activation of inflammatory signalling pathways, including those of the nuclear factor kappa-light-chain-enhancer of activated B cells and the mitogen-activated protein kinase, can promote the synthesis of cytokines (such as tumor necrosis factor-α, interleukin-1β, and interleukin-6) and is a hallmark of chronic metabolic diseases. It has been observed that visfatin can amplify inflammatory signals in the adipose tissue. Liraglutide may indirectly lessen inflammatory signals linked to high visfatin levels, by inhibiting the generation of inflammatory cytokines and by promoting anti-inflammatory macrophage phenotypes in the adipose tissue. In conclusion, there might be a crucial connection between pharmacological GLP-1 receptor activation and adipokine regulation through the interaction between li-

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raglutide and visfatin. By reducing visceral adiposity, by improving metabolic homeostasis, and by suppressing inflammatory signalling within the adipose tissue, liraglutide may indirectly influence visfatin expression and its circulating levels. Our review also suggests that visfatin should be further explored as a biomarker for treatment responsiveness in obesity and type 2 diabetes mellitus.

Keywords

diabetes; inflammation; liraglutide; obesity; visfatin

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Conflicts of interest statement

None to declare.

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