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PROCEEDINGS ABSTRACT

The modulatory role of semaglutide on nesfatin-1 in metabolic disorder: a review

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The dysregulation of neuroendocrine circuits that regulate appetite, glucose metabolism, and energy balance leads to metabolic diseases such as obesity and type 2 diabetes mellitus. Semaglutide is a long-acting glucagon-like peptide-1 (GLP-1) receptor agonist capable of enhancing glycaemic management and weight loss by stimulating glucose-dependent insulin secretion, suppressing glucagon release, delaying stomach emptying, and activating central satiety pathways. Moreover, it is known to promote weight loss by suppressing appetite through the hypothalamic GLP-1 receptors, and it affects the brainstem and the hypothalamus; two parts of the brain that regulate satiety. Semaglutide decreases food cravings and caloric intake, while weight loss improves metabolic parameters such as insulin sensitivity and lipid profile. Through weight loss and increased insulin sensitivity, semaglutide improves aspects of the metabolic syndrome, such as central obesity, hyperglycaemia, dyslipidaemia, hypertension, and cardiometabolic disease. On the other hand, nesfatin-1 is an anorexigenic peptide (found in both central and peripheral tissues) that has a role in thermogenesis, glucose homeostasis, lipid metabolism, hunger control, and stress reactions. Its capacity to reduce food intake results from its ability to suppress hunger-promoting neurons and to activate hypothalamic satiety centers in hypothalamic satiety signalling. Nesfatin-1 can act in multiple ways in order to prevent hyperglycaemia. Due to the fact that nesfatin-1 and insulin are co-localized in the β-cells of the pancreatic islets of Langerhans, when blood glucose levels rise, insulin and nesfatin-1 are co-released. In skeletal muscle and adipose tissues, it increases the translocation of the glucose transporter type 4 to the plasma membrane, enabling cells to more effectively remove glucose from the bloodstream. Furthermore, nesfatin-1 can directly inhibit glucagon secretion under hyperglycaemic conditions. It is also known to stimulate sympathetic nervous system outflow, brown adipose tissue thermogenesis, metabolic rate, and heat production, all of which enhance whole-body energy expenditure. Consequently, reduced body weight and negative energy balance are encouraged by this combination of reduced intake and increased expenditure. Peripherally, nesfatin-1 can directly affect the hepatocytes and the adipocytes by downregulating lipogenic factors and by upregulating critical lipolytic enzymes; increased levels of fatty acid oxidation, decreased triglyceride buildup, and improved blood lipid profiles are some of the results of these actions, which have a preventive role against metabolic syndrome and obesity. According to experimental studies, nesfatin-1 seems to increase the intestinal L-cells' production of GLP-1, thereby indicating its potential upstream function in enhancing the effects of the GLP-1-mediated pathway. On the other hand, it has been shown that a semaglutide treatment can increase nesfatin-1 expression in both central and peripheral tissues, suggesting the existence of a positive feedback mechanism between the

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endogenous nesfatin-1-mediated satiety and the GLP-1 receptor activation. Overall, semaglutide and nesfatin-1 seem to function similarly by reducing stomach emptying, suppressing food intake, increasing insulin secretion, and inhibiting glucagon release. These findings emphasize the overlap of the gut–brain and the neuroendocrine pathways in the control of energy balance. The strong appetite suppression and weight loss seen with GLP-1 receptor agonist therapy may be explained mechanistically by nesfatin-1 acting as an endogenous mediator and an amplifier of semaglutide's actions. In conclusion, semaglutide and nesfatin-1 are correlated, indicating the existence of a synergistic interaction between endogenous anorexigenic pathways and drug-induced GLP-1 receptor activation. There is evidence that the GLP-1 signalling and the nesfatin-1 pathways interact functionally, as the systems have similar mechanisms and semaglutide can increase nesfatin-1 expression. In addition to highlighting nesfatin-1 as a possible biomarker and treatment target for obesity and type 2 diabetes mellitus, this review suggests that an understanding of this interplay may offer fresh perspectives on metabolic regulation.

Keywords

GLP-1; nesfatin-1; obesity; semaglutide; type 2 diabetes mellitus

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Conflicts of interest statement

None to declare.

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