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PLEIOTROPIC EFFECTS OF GLP-1 RECEPTOR AGONISTS: A LITERATURE REVIEW

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ABSTRACT

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) have been transformative in the management of type 2 diabetes (T2DM) and obesity. Advances in drug design have improved their bioavailability, allowing effective glucose control and weight loss with minimal side effects. Studies have demonstrated that GLP-1RAs, beyond their metabolic benefits, exert protective effects in multiple pathologies, including cardiovascular disease, liver disorders, kidney disease and neurodegeneration . Cardiovascular benefits include enhanced myocardial function, reduced atherosclerosis, improved endothelial health and a lower risk of major adverse cardiovascular events, alongside improved insulin sensitivity and lipid metabolism. In the liver, GLP-1RAs ameliorate metabolic dysfunction-associated steatotic liver disease by decreasing hepatic fat, inflammation and oxidative stress. In diabetic kidney disease, they exert renoprotective effects through anti-inflammatory, antioxidative and antihypertensive mechanisms. Emerging evidence also suggests neuropsychiatric benefits, including reductions in depressive and anxiety symptoms, decreased substance use and lower risk of Alzheimer's, Parkinson's and other dementias, likely mediated by modulation of neurotransmitter systems and neuroinflammation. This review summarizes preclinical and clinical evidence demonstrating the multifaceted effects of GLP-1RAs across multiple organ systems.

KEYWORDS

Glucagon-Like Peptide 1 Receptor Agonists, Type 2 Diabetes Mellitus, Obesity, Cardiovascular Diseases, Diabetic Nephropathy, Fatty Liver Disease, Parkinson's Disease, Alzheimer's Disease, Neuropsychiatric Disorders

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Introduction

GLP-1RAs are widely used for the treatment of T2DM and obesity, represent a novel class of antidiabetic agents. In recent years, research has shown that their effects extend beyond glucose-lowering and weight-reducing effects alone. Increasing evidence suggests that GLP- RAs may confer beneficial effects on other organs and systems, including the heart, kidneys, liver and brain, through both glucose-dependent and glucose-independent mechanisms [1].

Methodology

This study aimed to present the effects of glucagon-like peptide-1 receptor agonists (GLP-1RAs) on multiple organ systems. Relevant articles were identified and retrieved from the PubMed database for review. The keywords used in the search included: "Glucagon-like peptide 1 receptor agonists", "type 2 diabetes mellitus", "obesity", "cardiovascular diseases", "diabetic nephropathy", "fatty liver disease", "Parkinson's disease" "Alzheimer's disease" and "neuropsychiatric disorders".

Results

Glucagon-Like Peptide-1 and Its Receptor Agonists

Glucagon-like peptide-1 (GLP-1) is a 30-amino acid hormone produced by intestinal L-cells from proglucagon. It is released in response to meals and acts as an incretin, stimulating insulin secretion, inhibiting glucagon release, slowing gastric emptying and regulating appetite. GLP-1 is rapidly inactivated by the enzyme DPP-IV and some of its effects may be mediated via neuronal signaling in the gut and liver. GLP-1 dysregulation can contribute to obesity and postprandial hypoglycemia [2]. GLP-1 receptors are widely expressed throughout the body, not only in pancreatic α and β cells, but also in the terminal ileum, colon, heart, lungs, kidneys and several brain regions controlling appetite and satiety [1]. GLP- RAs are divided into two main classes based on their molecular structure: human GLP-1-based (dulaglutide, albiglutide, liraglutide, semaglutide) and exendin-4-based (exenatide, lixisenatide). Currently, GLP-1RAs are administered via injections either twice daily (exenatide b.i.d.), once daily (lixisenatide, liraglutide) or once weekly (exenatide weekly, dulaglutide, albiglutide, semaglutide). Recently, a daily oral form of semaglutide was approved, showing efficacy comparable to that of a once-weekly subcutaneous injection [3,4].

Blood glucose level

The initial management of type 2 diabetes typically begins with metformin alone or in combination with other oral agents, with injectable therapies (insulin or GLP-1RAs) introduced if necessary. Meta-analyses comparing basal insulin (with oral agents) and GLP-1RAs show minimal differences in glycemic control, with GLP-1 RAs slightly more effective in lowering HbA1c. They also promote weight loss and cause hypoglycemia, particularly when combined with sulfonylureas or insulin. GLP-1RAs are more convenient owing to their standardized dosing, whereas insulin requires individualized titration. Both therapies are similarly effective in patients with very high baseline HbA1c levels, although reaching conventional targets can be challenging [3].

Obesity

Multiple strategies are available for the treatment of obesity, including lifestyle interventions, pharmacotherapy and bariatric procedures. Anti-obesity medications are recommended for individuals with a BMI ≥30 kg/m² or ≥27 kg/m² with obesity-related comorbidities, particularly when lifestyle modifications alone fail to achieve clinically meaningful weight loss. Among the available pharmacotherapies, GLP-1RAs have demonstrated substantial weight-loss efficacy and are increasingly used because of their favorable balance between effectiveness and invasiveness [5,6]. Evidence suggests that the incretin response is attenuated in individuals with obesity, even in the absence of T2DM, indicating a potential role for incretin-based therapies in this population [7, 8]. The SCALE Obesity and Prediabetes trial demonstrated that liraglutide 3.0 mg daily significantly reduced body weight in adults with obesity or overweight and obesity-related comorbidities without T2DM, achieving a placebo-adjusted weight loss of 5.6 kg over 56 weeks. Consequently, liraglutide has been approved for weight management by major regulatory agencies worldwide. Similarly, the STEP clinical trial program evaluated once-weekly semaglutide 2.4 mg for weight loss in adults with obesity or overweight, predominantly without T2D. The results from STEP consistently showed substantial and clinically meaningful reductions in body weight, leading to the regulatory approval of semaglutide for chronic weight management. Collectively, these findings support the use of GLP-1RAs, particularly liraglutide and semaglutide, as effective pharmacological options for weight management in individuals with and without T2DM [9,10].

Cardiovascular System

The GLP-1 receptor (GLP1R) is expressed in the human atria and ventricles, including cardiomyocytes. However, limitations in the sensitivity and specificity of commercially available antibodies complicate the interpretation of GLP1R+ cardiac cell types that mediate cardiovascular protection [11]. GLP-1RAs have been shown to exert beneficial effects on blood pressure (BP) and cardiovascular function, although these effects vary depending on species, physiological conditions and duration of treatment [12]. In preclinical and clinical studies, chronic administration of GLP-1RAs reduces systolic BP, primarily through natriuresis, vasodilation and modulation of sympathetic activity, whereas diastolic BP is less consistently affected. Cardioprotective effects have also been observed, including improved cardiac output, enhanced post-ischemic recovery and activation of cardioprotective signaling pathways, which appear to be at least partly independent of weight loss. While most human data come from patients with type 2 diabetes, GLP-1RAs are promising adjunctive therapies for hypertension and cardiovascular disease. However, further studies are required to clarify these mechanisms in humans [13-15].

Lipids

GLP-1 plays a key role in lipid metabolism by inhibiting fat synthesis, promoting adipocyte differentiation and browning and enhancing cholesterol metabolism. In line with these actions, GLP-1RAs have been shown to modulate serum lipids, with PEG-loxenatide increasing HDL cholesterol and semaglutide reducing LDL cholesterol and total cholesterol, underscoring their therapeutic potential in metabolic disorders. Overall, GLP-1RAs exert a modest but consistent effect in lowering LDL and total cholesterol, with similar outcomes observed across both short- and long-term treatment. They appear to have minimal impact on triglycerides, VLDL or HDL cholesterol and these lipid-lowering effects do not seem to be influenced by changes in body weight, highlighting that their benefits on lipid metabolism may be at least partly independent of weight reduction [16-18].

Liver

Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most common chronic liver disease worldwide and is closely linked to obesity, insulin resistance and T2DM. Insulin resistance plays a central role in the pathogenesis of MASLD by promoting hepatic lipid accumulation, inflammation and fibrosis, leading to disease progression toward metabolic dysfunction-associated steatohepatitis (MASH). Given the strong metabolic overlap between MASLD and T2DM [19-21].

Incretin-based therapies have emerged as promising therapeutic strategies. GLP-1RAs exert beneficial effects on glucose metabolism, body weight and insulin sensitivity, all of which are critical determinants of hepatic steatosis. By reducing appetite, delaying gastric emptying and improving glycemic control, GLP-1RAs induce sustained weight loss, which is strongly associated with reductions in hepatic fat content and liver volume. Even modest weight loss has been shown to significantly decrease hepatic triglyceride accumulation and improve insulin sensitivity.

In addition to their systemic metabolic effects, GLP-1RAs directly modulate hepatic lipid metabolism. These agents suppress de novo lipogenesis by downregulating key transcription factors, such as SREBP-1c and ChREBP, while enhancing fatty acid β-oxidation through PPAR-α activation. In addition, GLP-1RAs reduce endoplasmic reticulum stress, oxidative stress, and hepatocyte apoptosis, promoting autophagy-mediated lipid clearance. Collectively, these findings support the use of GLP-1RAs as promising therapeutic agents targeting the metabolic, inflammatory and fibrotic pathways underlying MASLD and MASH [1,22].

Kidneys

Diabetic kidney disease (DKD) is the leading cause of morbidity and mortality in patients with T2DM and a major contributor to end-stage renal disease (ESRD). Impaired kidney function affects approximately 40% of individuals with T2DM, underscoring the urgent need for effective strategies to prevent the progression of DKD. While current management relies primarily on glycemic and blood pressure control, accumulating evidence indicates that GLP-1RAs exert renoprotective effects beyond these mechanisms of action. GLP-1RAs activate protein kinase A via cyclic adenosine monophosphate signaling, leading to the inhibition of oxidative stress and inflammatory pathways, including NADPH oxidase and NF-κB, thereby preserving podocyte integrity and preventing mesangial and endothelial dysfunction. In addition, GLP-1RAs promote natriuresis through the inactivation of the sodium-hydrogen exchanger 3 and stimulation of atrial natriuretic peptide secretion, while also attenuating tubular injury and tubulointerstitial fibrosis. Clinical and experimental studies have demonstrated that GLP-1RAs reduce albuminuria and slow the decline in the estimated glomerular filtration rate. Although further studies are required to clarify the differences in renal effects among individual GLP-1RAs and to define their optimal use in clinical practice, current evidence supports GLP-1RAs as a promising therapeutic option for the management of DKD [1,23,24].

Central Nervous System

Recent translational and clinical studies have investigated the potential effects of GLP-1RAs on the central nervous system (CNS). Although only a limited number of GLP-1RAs appear to penetrate the CNS, accumulating evidence suggests their involvement in neurodegenerative and neuropsychiatric disorders [1]. Parkinson's disease (PD) is a progressive neurodegenerative condition characterized by motor and non-motor symptoms and GLP-1RAs are being explored as potential disease-modifying therapies. In a randomized, double-blind, placebo-controlled trial, exenatide treatment led to sustained improvements in motor function in patients with PD, persisting beyond the treatment period, suggesting a neuroprotective effect. Nevertheless, the underlying mechanisms, including direct CNS penetration and modulation of PD-related pathology, remain unclear. In Alzheimer's disease (AD), which is associated with impaired cerebral glucose metabolism and reduced glucose transporter expression at the blood-brain barrier (BBB), GLP-1 and its analog liraglutide have been shown to preserve cerebral glucose utilization and enhance glucose transport. Clinical studies have reported that liraglutide increases BBB glucose transfer capacity in patients with AD to levels comparable to those observed in healthy individuals, although these findings provide only indirect evidence of CNS penetration [25,26]. Additionally, preclinical studies have demonstrated that GLP-1RAs exert antidepressant and anxiolytic effects, likely mediated through anti-inflammatory mechanisms, enhanced neuroplasticity and modulation of neurotransmitter systems such as serotonin and γ-aminobutyric acid. While early clinical observations suggest a reduction in anxiety symptoms among patients treated with GLP-1RAs for metabolic indications, robust human data remain limited. Collectively, these findings indicate that selected GLP-1RAs, including exenatide and liraglutide, may engage CNS targets involved in neuroprotection, cognition and mood regulation; however, definitive evidence of their CNS penetration and direct target engagement in humans is still lacking, highlighting the need for further research [27].

Discussion

GLP-1RAs directly influence multiple organs, including the cardiovascular system, kidneys, liver and brain. Some effects result from improved systemic metabolism, whereas others reflect organ-specific cellular signaling that mitigates disease progression. Expanding our understanding of these pleiotropic actions could support the broader use of GLP-1RAs in promoting healthy aging in diverse populations [28, 29].

Conclusions

GLP-1RAs, initially developed for the treatment of type 2 diabetes and obesity, exert effects that extend beyond glycemic control and weight reduction. Owing to the widespread expression of GLP-1 receptors across multiple organs, these agents have demonstrated beneficial effects on the cardiovascular, renal, hepatic and central nervous systems. Emerging evidence suggests that some of these benefits may occur independently of glucose-lowering or weight loss and may include a potential reduction in the risk of aging-related diseases, highlighting the broader therapeutic potential of GLP-1RAs. However, further studies are needed to clarify the long-term safety, efficacy across diverse populations and the underlying mechanisms driving these broad therapeutic effects [5,28,30].

Disclosure

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