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GLP-1 Receptor Agonists: The Next Multifunctional Class of Drugs

Shubham K. Marbade [1,*]

[1] *Department of Pharmacology, Imperial College of Pharmacy, Tirora, India*

* **Corresponding author:** shubhammarbade@gmail.com

Abstract

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) are well established for the management of type 2 diabetes mellitus and obesity. However, growing evidence suggests that their therapeutic effects extend beyond glycemic control and weight reduction. This review summarizes the emerging pleiotropic effects of GLP-1 RAs on cardiovascular, renal, neurological, inflammatory, dermatological and vascular systems. Evidence indicates that GLP-1 RAs may improve endothelial function, reduce oxidative stress and inflammation, promote natriuresis and vasodilation, and provide cardioprotective and renoprotective effects. Potential benefits have also been reported in heart failure, aging-related cellular dysfunction, psoriasis, neurodegenerative disorders, cancer biology, and vascular smooth muscle dysfunction. These effects are associated with modulation of signalling pathways including cAMP/PKA, PI3K/Akt and AMPK, along with regulation of oxidative and inflammatory processes. Although cardiovascular and renal benefits have comparatively stronger clinical evidence, several other applications remain emerging and require further clinical investigation. Overall, GLP-1 RAs represent a promising multifunctional class of drugs with therapeutic potential beyond diabetes and obesity.

Keywords: GLP-1 receptor agonists; GLP-1; cardioprotection; nephroprotection; neuroprotection; inflammation; obesity; type 2 diabetes mellitus; pleiotropic effects; cardiovascular disease

1. Introduction

WHO defines obesity as the accumulation of excessive fat. It also can be defined in terms of BMI (weight in kg/ height in m^2) [1]. Obesity has an impact on individuals and society, which results in type 2 diabetes mellitus (T2D), hypertension, dyslipidemia, osteoarthritis, sleep apnea, and various cancers and associated disabilities, which leads to decreased productivity [2]. The prevalence of various cardiometabolic diseases has increased alongside the rapid growth of obesity rates, leading to higher morbidity and mortality [3].

Glucagon-like peptide-1 (GLP-1) receptor agonists (GLP-1 RAs) have revolutionized the management of metabolic disorders over the past two decades. Drugs such as liraglutide, semaglutide, dulaglutide, exenatide, and the dual glucose-dependent insulinotropic polypeptide (GIP)/GLP-1 receptor agonist tirzepatide have transformed the therapeutic landscape of obesity, providing outcomes that were previously difficult to achieve with conventional pharmacological therapies [4]. Glucagon-like peptide-1 (GLP-1) receptor agonist, chemically are incretin hormones which are released in the gut in response to food consumption [5]. Therefore, they offer unique treatment for the management of type 2 diabetes mellitus [6]. It induces insulin release from pancreatic β cells [7], inhibits glucagon release from α cells, slows gastric emptying and suppresses appetite by activating specific GLP-1 receptors [5].

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GLP-1 reduce appetite and food intake which leads to weight loss. In obese patients, GLP-1 secretion is impaired [8]. This slow gastric emptying and suppresses appetite by activating specific GLP-1 receptors, which are cell surface GPCRs [5].

Beyond their metabolic effects, growing evidence from large-scale randomized clinical trials and mechanistic studies has revealed that GLP-1 receptor agonists exert pleiotropic effects on multiple organ systems. These drugs possess anti-inflammatory, anti-atherogenic, antioxidant, and cytoprotective properties that extend well beyond glucose regulation and weight reduction. These findings have shifted the perception of GLP-1 receptor agonists from being merely anti-diabetic or anti-obesity medications to multifunctional therapeutic agents capable of modifying disease progression across a wide range of chronic disorders [9].

2. Literature Review

Literature review in this article focuses on recent developments and current approaches of actions of GLP-1 receptor agonists. It follows computerized search methodology to find out different mechanisms of GLP-1 receptor agonists.

2.1. Effects on Coronary Artery Disease

The study performed suggests that in states of high cardiovascular risk, the body attempts to limit disease progression by increasing the secretion of protective agents, such as GLP-1. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) directly mitigate coronary atherosclerosis through several mechanisms. They improve endothelial function and reduce oxidative stress in the coronary arteries. Agonists protect arteries against lipoapoptosis. GLP-1 RAs decrease endoplasmic reticulum stress by downregulating unfolded proteins. Exenatide is found to reduce inflammation by reducing activation of nuclear factor kappa light chain enhancer (NF- κβ) of activated β- cells and vascular cell adhesion molecule (VCAM) [10]. A study showed that GLP- 1 RAs lowered the adherence of monocytes to endothelial cells of the thoracic aorta by downregulating adhesion molecules such as intracellular adhesion molecule (ICAM-1) and VCAM [11].

Both endogenous GLP-1 and GLP-1 RA can diminish cardiovascular inflammation via direct and indirect means. GLP-1 infusion decreases elevation in microvascular permeability during inflammation caused by lipopolysaccharide. Liraglutide inhibits NADPH oxidase, protein kinase C (PkC), and nuclear factor kappa B (NF- κβ) signalling and reduces tumor necrosis factor (TNF- α)-induced inflammation [12].

2.2. Effect on Hypertension

Researchers have suggested that GLP-1 receptors are present in the glomerulus and proximal convoluted tubule (PCT), where they modulate Na+ reabsorption and contribute to its excretion. In blood vessel receptors are present on both endothelial and smooth muscle cells [13]. Receptor agonists inhibit natriuresis by inhibiting the $Na^{+}H^{+}$ exchanger in the PCT, thereby lowering cardiac preload, vascular resistance, and systemic blood pressure. GLP-1 agonists enhance nitric oxide (NO) bioavailability, reduce oxidative stress, and contribute to vasodilation. They also stimulate atrial natriuretic peptide with vasodilator properties. These supress sympathetic outflow lower noepinephrine (NE) in blood to reduce vascular resistance [14]. Liraglutide had shown a significant decrease in systolic blood pressure. Also it had shown dose dependent effect in diastolic blood pressure when used at higher doses [15]. GLP-1 RAs may modulate the renin-angiotensin-aldosterone system to lower blood pressure [16].

2.3. Effect on Heart Failure

Studies have reported that GLP-1 receptors are present in the SA node and atrial tissue [17]. GLP-1 RAs show strong potential in lowering clinical heart failure events and improving symptoms in heart failure with preserved ejection fraction (HFpEF) [18]. GLP-1 RAs activate cyclic adenosine monophosphate (cAMP)- PkA (protein kinase A) and restore physiological calcium transient. They reduce arrhythmic sarcoplasmic reticulum calcium leaks and enhance myocardial contractile performance. Agonists supress NF- κβ activation, downregulates NLR family pyrin domain containing 3 (NLRP3) inflammation reducing cardiac inflammation and interstitial fibrosis. Agonists enhance nitric oxide (NO) production to promote vasodilation, inhibit renal sodium hydrogen exchanger 3 (NHE3) channels, and promote natriuresis to reduce volume overload [17].

GLP-1 RAs lower total cholesterol, low-density lipoprotein, and triglycerides to provide athoprotection and improve the symptoms of heart failure with reduced ejection fraction (HFrEF) [19]. Agonists inhibit proliferative remodelling of vascular smooth muscles, preventing plaque formation and stabilizing vascular lesions. In addition, they enhance cardiac mitochondrial efficiency under hypoxic conditions and promote the use of alternative energetic substances, such as ketones and lactate [20].

2.4. Effect on Aging

GLP-1 RA activation stimulates apurinic/ apyrimidinic endonucleases (APE1) expression which actively aids in DNA repair. These modulates body's endogenous antioxidants defence system, protecting against H_2O_2 induced cellular damage and senescence. Receptor stimulation modulates critical metabolic signalling pathways including sirtuin 1 (SIRT1), AMP activated protein kinase (AMPK) and Phosphoinositide 3-kinase (PI3K) which regulates cellular survival, nutrient sensing and autophagy. These mechanisms helps to resolve mitochondrial energy crisis, restore normal mitochondrial dynamics and protect cells from mitochondrial dysfunction [21].

Agonists lower persistent, low-grade systemic inflammation by suppressing inflammatory pathways and reducing pro-inflammatory cytokines and oxidative stress. GLP-1 RA therapy lowers systemic glucose, thereby reducing advanced glycation end (AGE) and preventing cross-linking with dermal collagen. These enhance microvascular perfusion in the skin tissue, improving oxygen and nutrient delivery to support wound healing and regeneration [22].

2.5. Effect on Psoriasis

GLP-1 RA showed clinical improvement in psoriasis indicating reduction in psoriatic area and severity index and dermatology life quality index by reduction in dermal T- cells, IL (Interleukin) - 23, IL (Interleukin)- 17 and TNF (Tumour Necrosis Factor)- α expression, alongside inhibition of invariant natural killer T- cells cytokine secretion [23], [24]. Preclinical studies show that agents like liraglutide and exenatide accelerate wound closure, enhance endothelial cell proliferation, increase collagen/fibroblast formation, and promote angiogenesis independently of blood glucose levels [25]. GLP-1 receptors present in psoriatic plaques appear to mediate suppression of the IL-23/Th17 pathway, reduce TNF- and IL-17 levels, and limit inflammatory cell infiltration [26].

2.6. Neuroprotective Effect

Activation of GLP-1 receptors (GLP-1R) on neurons, microglia, and astrocytes triggers multiple signalling pathways (e.g., PI3K/Akt, cAMP/PKA) that provide preservation of dendritic spines and synaptic proteins, improve cognitive, learning, and motor functions, reduce accumulation of Amyloid-Beta (),

hyperphosphorylated Tau, and -synuclein. It also prevents intracellular deregulation, endoplasmic reticulum (ER) stress, oxidative stress, and mitochondrial dysfunction and re-sensitizes impaired brain insulin signalling and restores neuronal glucose energy metabolism [27].

Patients receiving once-weekly exendin-4 showed significant improvements and stabilization in motor control and cognitive function compared to placebo. Improvements persisted for up to 12 months after drug administration ceased, indicating genuine protection rather than temporary symptom suppression. GLP-1 acts as a neurotrophic growth factor in the central nervous system, compensating for impaired insulin signalling in neurodegenerative diseases. GLP-1 receptor activation normalizes cellular energy utilization, restores synaptic function, reduces chronic brain inflammation, and lowers oxidative stress [28].

2.7. Effect on Chronic Kidney Disease

This study evaluated the effects of glucagon-like peptide 1 (GLP-1) receptor agonists on kidney outcomes and overall mortality in patients with chronic kidney disease (CKD) characterized by a baseline estimated glomerular filtration rate (eGFR) of < 60 mL/min/1.73 m^2. GLP-1 receptor agonists were associated with a 15% reduction in the risk of composite kidney outcomes [29].

These reduce glomerular hyperfiltration and single-nephron intraglomerular pressure, leading to a reduction in albuminuria and subnephrotic proteinuria. Agonists **s**uppress chronic intrarenal inflammation, proinflammatory cytokines, and macrophage infiltration, helping attenuate renal tissue fibrosis. In addition, reduces ectopic lipid accumulation ("fatty kidney") and renal sinus fat deposition, diminishing lipotoxic damage to the renal parenchyma [30].

GLP-1 RAs increase intracellular cyclic adenosine monophosphate (cAMP) and activate protein kinase A (PKA). This pathway inhibits NHE3 in the proximal convoluted tubules, reducing sodium reabsorption. As more sodium passes downstream to the macula densa, it triggers tubuloglomerular feedback, which constricts the afferent arteriole, easing glomerular hyperfiltration and intraglomerular pressure [31].

GLP-1 RAs prevent renal oxidative stress by inhibiting nicotinamide adenine dinucleotide phosphate (NADPH) oxidase. This curbs the production of reactive oxygen species (ROS) and limits cellular injury typical of chronic kidney disease (CKD) [32].

2.8. Effect on Cancer

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) influence cancer biology through a combination of direct cellular signaling pathways and indirect metabolic improvements. While initially designed for diabetes and weight management, emerging evidence demonstrates that GLP-1 RAs can suppress tumor progression and reduce the risk of several obesity-related solid malignancies [33], [34], [35]. GLP-1 RAs can blunt the phosphatidylinositol 3-kinase (PI3K)/Akt and mammalian target of rapamycin (mTOR) pathways. Because this axis is notoriously overactive in malignant tissues, suppressing it directly curbs tumor cell proliferation, survival, and metabolic plasticity [33], [36], [37]. Activation of the G-protein-coupled GLP-1R leads to an influx of cyclic adenosine monophosphate (cAMP) and protein kinase A (PKA) activation. This downstream cascade induces apoptosis (programmed cell death) by modulating caspase cascades and downregulating oncogenic cell-cycle drivers, such as cyclins [33], [38], [39].

2.9. Effect on Vascular Smooth Muscle Dysfunction

Activation of the Gαs-coupled GLP-1 receptor spikes intracellular cyclic adenosine monophosphate (cAMP), activating protein kinase A (PKA). This acts as a master brake on abnormal cellular growth. The PKA axis directly blocks pro-proliferative signaling chains, specifically suppressing the ERK1/2 and p38 mitogen-

activated protein kinase (MAPK) pathways. This reduces the expression of critical replication markers, such as PCNA and Cyclin D1 (Ccnd1). Drp1 Phosphorylation: GLP-1 signaling induces phosphorylation at the Ser-637 site of dynamin-related protein 1 (Drp1), which is responsible for mitochondrial fission. Under conditions of high inorganic phosphate (common in diabetic vascular complications), VSMCs transform into bone-like cells, hardening the arteries. GLP-1 RAs block this osteogenic shift by inhibiting activating transcription factor 4 (Atf4), dramatically lowering calcium deposition within the smooth muscle layers [40], [41].

3. Result

The reviewed literature shows that GLP-1 receptor agonists (GLP-1 RAs) have effects extending beyond glycemic control and weight reduction, mediated through cAMP/PKA, PI3K/Akt, AMPK, nitric oxide, and anti-inflammatory and antioxidant pathways. GLP-1 RAs demonstrated potential cardiovascular and blood-pressure benefits through improved endothelial function reduced oxidative stress and inflammation, enhanced natriuresis and vasodilation. They also showed potential benefits in heart failure by improving myocardial function and reducing cardiac inflammation and fibrosis.

Additional evidence suggests possible protective effects in ageing, psoriasis, neurological disorders and chronic kidney disease. These include improved mitochondrial and cellular function, reduced inflammation and oxidative stress, neuroprotection, and reduced renal hyperfiltration and inflammation. A systematic review reported a 15% reduction in composite kidney outcomes.

Emerging studies also indicate potential roles in cancer biology and vascular smooth muscle dysfunction through modulation of PI3K/Akt/mTOR and cAMP/PKA pathways. Overall, GLP-1 RAs show broad pleiotropic effects, although evidence strength varies between established clinical benefits and emerging preclinical findings.

4. Conclusion

GLP-1 receptor agonists have therapeutic potential that extends beyond diabetes and obesity, with promising cardiovascular, renal, neurological, anti-inflammatory, and cellular protective effects. Their diverse actions are associated with modulation of cAMP/PKA, PI3K/Akt, AMPK and oxidative and inflammatory pathways.

Although cardiovascular and renal benefits have stronger clinical support, emerging applications in neurodegenerative disorders, psoriasis, ageing, cancer and vascular dysfunction require further clinical investigation. Overall, GLP-1 RAs represent a promising multifunctional drug class with potential applications across several chronic diseases.

Conflict of Interest

The authors declare no conflict of interest.

AI Usage Disclosure

No generative AI tools were used.

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