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Review Paper

GLP-1 Receptor Agonists: Expanding Pharmacological Horizons Beyond Metabolic Disease

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ABSTRACT

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) were originally developed as glucose-lowering agents for type 2 diabetes mellitus and later established as first-line pharmacotherapy for obesity. Over the past decade, the pharmacological footprint of this drug class has expanded well beyond glycaemic and weight control. This narrative review synthesises current evidence on the expanding non-metabolic indications of GLP-1 RAs, outlines the mechanistic basis for these pleiotropic effects, and appraises associated safety considerations. A literature search was conducted using PubMed/MEDLINE, ScienceDirect, and Google Scholar for articles published through mid-2026, prioritising randomised controlled trials, systematic reviews, and meta-analyses where available. Evidence implicates GLP-1 receptor signalling in cardiovascular protection, renal preservation, hepatic steatosis and fibrosis reduction, modulation of central reward circuitry relevant to substance use disorders, and joint inflammation in osteoarthritis. Evidence in neurodegenerative disease is mixed, with recent large trials in Alzheimer's disease showing no benefit on clinical progression despite biomarker improvement. Overall, GLP-1 RAs have evolved into a multi-organ therapeutic class with robust evidence beyond glycaemic and weight control in cardiovascular and renal disease, and promising but preliminary evidence in hepatic, musculoskeletal, and addiction-related indications. Continued vigilance for thyroid, gastrointestinal, and neuropsychiatric safety signals is warranted as indications expand

INTRODUCTION

The glucagon-like peptide-1 (GLP-1) system began its clinical life in a narrow role: an incretin hormone that could be harnessed to improve glycaemic control in type 2 diabetes mellitus (T2DM). Exenatide, a synthetic analogue of exendin-4 derived from Gila monster saliva, was approved by the US FDA in 2005 as the first agent in this class.1 Since then, the GLP-1 receptor

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agonist (GLP-1 RA) class has grown to include liraglutide, dulaglutide, semaglutide, and the dual GIP/GLP-1 agonist tirzepatide, among others, with progressively longer half-lives, greater potency, and expanding routes of administration, including oral formulations.1

What has changed most dramatically in recent years is not the chemistry of these molecules but the recognition of how broadly GLP-1 receptors are distributed in the body. Beyond pancreatic beta cells, GLP-1 receptors are expressed in the hypothalamus, brainstem, mesolimbic reward circuitry, cardiac tissue, vascular endothelium, renal glomeruli and tubules, hepatocytes and Kupffer cells, and articular tissue.2,3 This wide distribution provides a mechanistic rationale for effects that extend far beyond appetite suppression and insulin secretion. As a result, GLP-1 RAs are increasingly being evaluated, and in several cases already approved, for cardiovascular risk reduction, chronic kidney disease, metabolic dysfunction-associated steatohepatitis (MASH), obstructive sleep apnoea, knee osteoarthritis, and substance use disorders, with more speculative but active investigation in neurodegenerative disease and psychiatric conditions.4

This review aims to consolidate the evidence behind these expanding applications, to describe the underlying mechanisms that connect a single receptor system to such heterogeneous organ effects, and to critically appraise both the promise and the limitations, including safety signals that warrant continued vigilance.

MATERIALS AND METHODS

This is a narrative review rather than a systematic review or meta-analysis; no formal protocol was registered. A literature search was performed in PubMed/MEDLINE, ScienceDirect, and Google Scholar for articles published up to July 2026, using the search term "glucagon-like peptide-1 receptor agonist" in combination with organ- or condition-specific terms, including "cardiovascular," "chronic kidney disease," "MASLD," "MASH," "Alzheimer's disease," "Parkinson's disease," "substance use disorder," "alcohol use disorder," "osteoarthritis," and "adverse effects." Priority was given to phase 3 randomised controlled trials, prespecified trial analyses, and systematic reviews or meta-analyses published in indexed journals. Preclinical and mechanistic studies were included selectively to support biological plausibility. Non-peer-reviewed sources (example, industry pipeline updates) were used only to describe drugs still in development and were clearly identified as such.

RESULTS

Mechanism of Action: One Receptor, Many Systems

Endogenous GLP-1 is secreted by intestinal L-cells in response to nutrient ingestion. It acts on pancreatic beta cells to stimulate glucose-dependent insulin secretion while suppressing glucagon release, slows gastric emptying, and signals centrally to the hypothalamus to promote satiety.1 GLP-1 RAs mimic these actions while resisting degradation by dipeptidyl peptidase-4, thereby prolonging receptor activation.

The pleiotropic effects of this drug class arise because GLP-1 receptors are not confined to the classic incretin axis. Centrally, GLP-1 receptor activation in the hypothalamus and brainstem regulates appetite, while activation within mesolimbic structures such as the ventral tegmental area and nucleus accumbens modulates dopaminergic reward signalling, a pathway relevant to both food intake and substance use.3,19 Peripherally, direct GLP-1 receptor signalling in cardiomyocytes, vascular endothelium, renal tubular and glomerular cells, hepatocytes, and chondrocytes appears to confer anti-inflammatory, antifibrotic, and antioxidant effects that are at least

partly independent of weight loss or glycaemic improvement.2,14 This combination of central and peripheral actions underlies the rationale for the multi-organ therapeutic exploration described below.

Established Metabolic Indications: The Foundation

Before turning to newer applications, it is worth briefly noting the foundation on which this expansion rests. GLP-1 RAs are firmly established for glycaemic control in T2DM and, at higher doses, for chronic weight management in obesity, with agents such as semaglutide and tirzepatide producing weight reductions in the range associated historically only with bariatric surgery.5 Newer triple agonists such as retatrutide, which target GLP-1, GIP, and glucagon receptors simultaneously, have shown even greater efficacy in early-phase trials.6 It is this metabolic efficacy, and the accompanying reduction in adiposity-driven inflammation, that first drew attention to the possibility of benefits in other organ systems.

Cardiovascular Disease

The most mature evidence for a benefit beyond glycaemic control comes from cardiovascular outcome trials. The SELECT trial enrolled over 17,000 adults with overweight or obesity and established cardiovascular disease but without diabetes, and found that semaglutide reduced the composite risk of cardiovascular death, non-fatal myocardial infarction, or non-fatal stroke by approximately 20 percent relative to placebo over a mean follow-up of roughly three years.7,8 This was a pivotal finding because it demonstrated a cardiovascular benefit independent of any diabetes-related mechanism, leading to FDA approval of semaglutide specifically for cardiovascular risk reduction in this population.8

Complementary trials have extended this benefit to heart failure. The STEP-HFpEF and STEP-HFpEF DM programmes showed that semaglutide improved symptoms, physical function, and exercise capacity in patients with heart failure with preserved ejection fraction and obesity, both with and without diabetes.9,10 A prespecified analysis of SELECT has further shown that cardiovascular benefit is preserved regardless of baseline heart failure status, and that benefit does not appear to depend solely on the magnitude of weight or adiposity change, suggesting a direct vascular or anti-inflammatory mechanism alongside the metabolic one.7,11

Chronic Kidney Disease

Renal protection has emerged as one of the more definitively established non-metabolic benefits of this class. The FLOW trial, published in 2024, was the first study specifically powered to test kidney outcomes with a GLP-1 RA, and found that semaglutide reduced the risk of major kidney disease events, including sustained decline in estimated glomerular filtration rate, kidney failure, and kidney-related death, in patients with type 2 diabetes and chronic kidney disease.12,13 Secondary renal analyses from SELECT similarly showed a reduction in a composite renal endpoint in a non-diabetic population, along with a slower rate of eGFR decline overall.14

Proposed mechanisms include reduced renal inflammation and fibrosis, improved endothelial function, modulation of the renin-angiotensin system, and preservation of mitochondrial gene expression in kidney tissue, in addition to indirect benefits from improved blood pressure and glycaemic control.13 Ongoing mechanistic trials are using imaging and histological methods to clarify how much of this renal benefit is direct versus secondary to systemic metabolic improvement.

Hepatic Disease: MASLD and MASH

GLP-1 RAs have also shown efficacy across the spectrum of metabolic dysfunction-associated steatotic liver disease (MASLD), including its more severe inflammatory form, metabolic dysfunction-associated steatohepatitis (MASH, formerly NASH). Interim results from the ESSENCE trial presented in late 2024 showed that semaglutide was superior to placebo on the co-primary endpoints of steatohepatitis resolution without worsening fibrosis, and improvement in liver fibrosis without worsening steatohepatitis.1 Beyond effects on lipid metabolism, GLP-1 RAs appear to exert direct anti-inflammatory and antioxidant actions within the liver, positioning them as a leading pharmacological option for MASLD and MASH.2

Neurodegenerative Disease

The neurological rationale for GLP-1 RAs rests on the overlapping biology of insulin resistance, chronic neuroinflammation, and protein misfolding that characterises both Alzheimer's disease (AD) and Parkinson's disease (PD). Preclinical work has shown that GLP-1 receptor activation can reduce microglial-driven neuroinflammation, support autophagy-mediated clearance of amyloid-beta, tau, and alpha-synuclein aggregates, and preserve blood-brain barrier integrity.15

Alzheimer's Disease

Despite this mechanistic promise and encouraging observational data, including large electronic health record studies suggesting a substantially reduced risk of incident AD diagnosis among diabetic patients treated with GLP-1 RAs compared with other glucose-lowering drugs, the first large phase 3 disease-modification trials have been disappointing.15 The EVOKE and EVOKE+ trials, which together randomised over 3,800 adults with mild cognitive impairment or mild dementia due to AD to oral semaglutide or placebo, did not demonstrate superiority over placebo in slowing clinical disease progression, despite measurable improvements in some AD-related biomarkers.16,17 Full data presented in March 2026 confirmed this topline result.17,18 This outcome illustrates an important lesson for the field: mechanistic plausibility and favourable observational associations do not guarantee benefit in adequately powered randomised trials, particularly once overt clinical dementia has already developed.

Parkinson's Disease

Evidence in PD has been comparatively more encouraging, though still preliminary. Trials of exenatide and lixisenatide have shown modest motor benefits, with one lixisenatide trial reporting stabilisation of motor symptom scores in treated patients compared with continued decline in the placebo group.3 However, a trial of a pegylated exendin-4 formulation did not meet its primary endpoint, underscoring that results vary by molecule, dose, and trial design.15 Larger and longer trials are needed before conclusions can be drawn.

Substance Use and Addictive Disorders

Perhaps the most mechanistically distinct application of GLP-1 RAs lies in the treatment of substance use disorders (SUDs). GLP-1 receptors are expressed throughout the mesolimbic dopaminergic reward system, including the ventral tegmental area, nucleus accumbens, and nucleus tractus solitarius, regions central to the reinforcement of both food intake and drug-seeking behaviour.19 Preclinical studies spanning several decades have shown that GLP-1 receptor agonism can reduce alcohol, nicotine, cocaine, and amphetamine self-administration and reward

sensitivity in rodents by attenuating dopaminergic signalling.19,23

Clinical evidence is most developed for alcohol use disorder (AUD). Several systematic reviews and meta-analyses, including PRISMA-guided syntheses of randomised trials, report reductions in alcohol consumption and craving with GLP-1 RA treatment, particularly among individuals with comorbid obesity, although effect sizes are heterogeneous and many included trials are small and short in duration.20,24,25 Human neuroimaging studies have correspondingly shown reduced alcohol cue reactivity in the nucleus accumbens, insula, and prefrontal cortex following treatment.24 Evidence for nicotine and other substances remains earlier-stage, drawing mainly on preclinical and small clinical studies, but is conceptually consistent with the alcohol data.19,21,22 Overall, GLP-1 RAs represent a genuinely novel pharmacological angle on addiction, targeting a shared neurobiological substrate across substance classes rather than a substance-specific mechanism.26

Musculoskeletal Disease: Knee Osteoarthritis

Knee osteoarthritis (KOA) is strongly associated with obesity, both through mechanical joint loading and through obesity-associated systemic inflammation. Systematic reviews combining clinical and preclinical studies report that GLP-1 RAs improve pain scores and functional outcomes in KOA and may reduce disease incidence, with proposed mechanisms including both weight-mediated reduction in joint loading and direct anti-inflammatory effects on chondrocytes and synovial tissue.27 Large ongoing phase 3 programmes are further evaluating GLP-1 and dual-agonist therapies specifically for KOA in patients with obesity.28

Other Emerging Indications

A growing list of additional conditions is under active investigation, reflecting the breadth of GLP-1 receptor distribution. These include obstructive sleep apnoea, for which tirzepatide has already received regulatory approval; polycystic ovary syndrome, where weight loss and improved insulin sensitivity may improve reproductive and metabolic parameters; idiopathic intracranial hypertension; and inflammatory bowel disease.29,30 Evidence in these areas ranges from early phase 2/3 trial data to largely mechanistic or observational support, and firm conclusions await larger controlled trials.

Indication	Representative agent	Evidence status	Key finding
Cardiovascular disease	Semaglutide	Robust RCT evidence	Reduced major cardiovascular events in selected populations
Chronic kidney disease	Semaglutide	Robust RCT evidence	Reduced major kidney disease events in T2DM with CKD
MASLD/MASH	Semaglutide	Emerging clinical evidence	Improved steatohepatitis-related histologic outcomes
Alzheimer's disease	Oral semaglutide	Phase 3 negative	No slowing of clinical disease progression in EVOKE/EVOKE+
Alcohol use disorder	GLP-1 RAs	Preliminary clinical evidence	Signals of reduced alcohol consumption/craving
Knee osteoarthritis	GLP-1 RAs	Emerging evidence	Potential improvement in pain/function

DISCUSSION

Safety Considerations

The expansion of GLP-1 RA indications makes a clear-eyed view of safety essential, since these drugs are now being considered for much larger and more diverse populations than the original diabetes cohorts.

Gastrointestinal and Biliary Effects

Nausea, vomiting, and other gastrointestinal symptoms remain the most common reasons for treatment discontinuation. GLP-1 RAs have also been associated with an increased risk of gallbladder and biliary disease, although long-term trial data have not confirmed an increased risk of acute pancreatitis, a concern that was prominent in earlier years of the drug class.31,32

Thyroid and Other Cancers

Rodent studies showing GLP-1 receptor-mediated thyroid C-cell proliferation prompted a black-box warning for medullary thyroid carcinoma risk with liraglutide, and pharmacovigilance database analyses have found disproportionate reporting of thyroid cancer across several agents in this class.31,33 Whether this reflects a true causal signal, detection bias from increased medical surveillance in treated patients, or reporting artefacts remains debated, and family or personal history of medullary thyroid carcinoma or multiple endocrine neoplasia type 2 is considered a contraindication.35 Evidence regarding other cancers, including pancreatic and breast cancer, is mixed and inconclusive across meta-analyses of randomised trials.36

Neuropsychiatric Safety

Given the class's central nervous system activity, regulators including the FDA and European Medicines Agency have investigated a possible association with suicidal ideation and behaviour.37 To date, systematic reviews and meta-analyses of available trial and pharmacovigilance data have not found a consistent, significant increase in suicidality risk, and some observational analyses suggest a lower risk in treated patients, but heterogeneity across studies and reliance on spontaneous-reporting pharmacovigilance data mean that continued monitoring, particularly in patients with pre-existing psychiatric conditions, is warranted.34,35

FUTURE DIRECTIONS

The pharmacological trajectory of this class is moving in two directions simultaneously: greater potency and mechanistic breadth, achieved through multi-receptor agonists such as tirzepatide (GLP-1/GIP), retatrutide (GLP-1/GIP/glucagon), survodutide, and co-formulated products like CagriSema (semaglutide plus the amylin analogue cagrilintide); and greater accessibility, through oral non-peptide GLP-1 receptor agonists such as orforglipron, which completed phase 3 trials in 2025, potentially removing the injection-related barriers that have limited uptake.3,28 As these newer agents and formulations progress through late-phase trials for the conditions discussed above, the coming several years should substantially clarify which of the proposed non-metabolic benefits reflect genuine, generalisable disease-modifying effects, as opposed to indirect consequences of weight loss or glycaemic improvement, or associations that do not survive rigorous randomised testing, as appears to have been the case for Alzheimer's disease in the EVOKE trials.

CONCLUSION

GLP-1 receptor agonists have evolved from a niche diabetes therapy into one of the most broadly investigated drug classes in contemporary medicine. Robust randomised trial evidence now supports genuine benefit beyond glycaemic and

weight control in cardiovascular disease and chronic kidney disease, with strong emerging evidence in MASLD/MASH and knee osteoarthritis. Evidence in substance use disorders, particularly alcohol use disorder, is mechanistically compelling and clinically promising but still preliminary. By contrast, the recent failure of semaglutide to slow clinical progression in early Alzheimer's disease is an important reminder that not every plausible mechanism translates into clinical benefit. As next-generation multi-agonists and oral formulations mature, the central task for the field will be distinguishing direct organ-protective effects of GLP-1 receptor signalling from the many downstream benefits of weight loss itself, while maintaining vigilance for thyroid, gastrointestinal, and neuropsychiatric safety signals across an increasingly broad patient population.

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